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STATISTICAL MODELING: A GENTLE INTRODUCTION

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Introduction

THIS BOOK is about statistical modeling. Some people define this term loosely as “fitting equations to data.” That’s close, but inexact. To do a bit better, let’s take both words in turn.

Statistics is the study of variation among cases: economic growth rates, dinosaur skull volumes, student SAT scores, genes in a population, Congressional party affiliations, drug dosage levels, your choice of toothpaste versus mine . . . really any variable that can be measured.

A model is a metaphor, a description of a system that helps us to reason more clearly. Like all metaphors, models are approximations, and will never account for every last detail. A useful mantra here is: all models are wrong, but some models are useful.¹

Aerospace engineers work with physical models—blueprints, simulations, mock-ups, wind-tunnel prototypes—to help them understand a proposed airplane design. Geneticists work with animal models—fruit flies, mice, zebrafish—to help them understand heredity. We will work with statistical models to help us understand *variation*.

Like the weather, most variation in the world exhibits some features that are predictable, and some that are unpredictable. Will it snow on Christmas day? It’s more likely in Boston than Austin, and more likely still at the North Pole; that’s predictable variation. But even as late as Christmas eve, and even at the North Pole, nobody knows for sure; that’s unpredictable variation.

Statistical models describe both the predictable and the unpredictable variation in some system. More than that, they allow us to partition observed variation into its predictable and unpredictable components—and not just in some loose allegorical way, but in a precise mathematical way that can, with perfect accuracy, be described as Pythagorean. (More on that later.)

This focus on the structured quantification of uncertainty is what distinguishes statistical modeling from ordinary evidence-based reasoning. It’s important to know what the evidence says, goes this line of thinking. But it’s also important to know what it

¹ Attributed to George Box.

doesn't say. Sometimes that's the tricky part.

We will use statistical models for three purposes:

- (1) *to explore* a large body of evidence, so that we might identify predictable features or trends amid random variation.
- (2) *to test* our beliefs about relationships among things we can measure.
- (3) *to predict* the future behavior of some system, and to say something useful about what remains unpredictable.

As Hippocrates counseled doctors in the oath bearing his name:

"Declare the past, diagnose the present, foretell the future."² These are the goals not merely of statistical modeling, but of the scientific method more generally.

² Epidemics Book I, section 11.

What modeling isn't. Many people assume that the job of a statistical modeler is to objectively summarize the facts, slap down a few confidence intervals, and get out of the way.

This view is mistaken. To be sure, statistical modeling demands a deep respect for facts, and for not allowing one's wishes or biases to change the story one tells *with* the facts. But the modeling process is inescapably subjective, in a way that should be embraced rather than ignored. Model-building requires much more than just technical knowledge of statistical ideas. It also requires care and judgment, and cannot be reduced to a flowchart, a table of formulas, or a tidy set of numerical summaries that wring every last drop of truth from a data set. There is almost never a single "right" statistical model for some problem. But there are definitely such things as good models and bad models, and learning to tell the difference is important. Just remember: calling a model good or bad requires knowing both the tool and the task. A shop-window mannequin is good for displaying clothes, but bad for training medical students about vascular anatomy. A big part of your statistical education is to hone this capacity for deciding when a statistical model is fit for its intended purpose.

Second, many people assume that statistical models must be complicated in order to do justice to the real world. Not always: complexity sometimes comes at the expense of explanatory power. We must avoid building models calibrated so perfectly to past experience that they do not generalize to future cases. This idea—that theories should be made as complicated as they need to be,

and no more so—is often called “Occam’s Razor.” A good model will be simple enough to understand and interpret, but not so simple that it does any major intellectual violence to the system being modeled. All models of the world must balance these goals, and statistical models are no exception.

Finally, many people also assume that statistical modeling involves difficult, tedious mathematics. Happily, this isn’t true at all. In fact, virtually all common statistical models are accessible to anyone with a high-school math education, and these days all the tedious calculations are taken care of by computers.

Modeling then and now

On the time scale of important post-Enlightenment ideas, statistical modeling is middle aged. A German astronomer named Tobias Mayer was using something vaguely like linear modeling as early as 1750.³ But most scholars credit two later mathematicians—Legendre, a Frenchmen; and Gauss, a German—with independently inventing the *method of least squares* some time between 1794 and 1805. As you will soon discover (or may already know), the method of least squares is our primary mathematical workhorse for fitting models to data. That makes statistical modeling newer than the invention of calculus (credited jointly to Leibniz and Newton in the late 1600’s), but older than the idea of evolution by natural selection (credited jointly to Darwin and Wallace over a period spanning the 1830’s to the 1850’s).

For most of the nineteenth century, statistical modeling largely remained the concern of a highly specialized group of astronomers and geophysicists. But in our own age—one of fast, cheap computers and abundant data—it has become ubiquitous. The very same principle of least squares proposed by Legendre and Gauss remains, over two hundred years later, an important part of the day-to-day toolkit for solving problems in fields from aeronautics to zoology and everywhere in between. If you’ve ever wondered why your social media accounts are eerily prescient—about your friends, about headlines that might appeal to you, about products you might want to buy—you can thank a statistical model.

Of course, our political and cultural climate still exhibits a streak of distrust toward statistics. Why else would Winston Churchill’s brazen instructions to a young protégé sound so depressingly familiar?

³ Stephen M. Stigler, *The History of Statistics: The Measurement of Uncertainty before 1900*, pp. 16–25. Harvard University Press, 1986

I gather, young man, that you wish to be a Member of Parliament. The first lesson that you must learn is that, when I call for statistics about the rate of infant mortality, what I want is proof that fewer babies died when I was Prime Minister than when anyone else was Prime Minister.⁴

And why else would the famous remark, popularized by Twain and attributed to Disraeli, remain so apt, even a century later?

Figures often beguile me, particularly when I have the arranging of them myself; in which case the remark attributed to Disraeli would often apply with justice and force: ‘There are three kinds of lies: lies, damned lies, and statistics.’⁵

How do you tell the difference between “robust, unbiased evidence,” misleading irrelevance, and cynical fraud? In considering this question, you will already have appreciated at least two good reasons to learn statistical modeling:

- (1) To use data honestly and credibly in the service of an argument you believe in.
- (2) To know how and when to be skeptical of someone else’s damned lies.

For as John Adams put it,

Facts are stubborn things; and whatever may be our wishes, our inclinations, or the dictates of our passion, they cannot alter the state of facts and evidence.⁶

⁴ Quoted in *The Life of Politics* (1968), Henry Fairlie, Methuen, pp. 203–204

⁵ *Chapters from My Autobiography*, North American Review (1907)

⁶ ‘Argument in Defense of the Soldiers in the Boston Massacre Trials’ (1770)

1

Data exploration

SUPPLY AND DEMAND, chocolate and peanut butter, education and income . . . some things just go hand in hand. In each case, a particular idea about how things work turns upon the interpretation of an observed relationship between things we can measure. To do this correctly requires care, judgment—and the right toolkit.

The goal of this chapter is to equip you with some basic visual and numerical tools for exploring multivariate data sets, with an eye towards finding interesting relationships among variables.

Cases and variables. In statistics, we typically refer to the *cases* and *variables* of a data set. The cases are the basic observational units that we’re interested in: people, houses, cars, guinea pigs, etc. The variables are the different kinds of information we have about each case—for example, the horsepower, fuel economy, and vehicle class for a car. We typically organize a data set into a *data frame*. A data frame is like a simple spreadsheet where each case is a row and each variable is a column, like in Table 1.1.

Variables come in two basic kinds. Numerical variables are represented by a number, like horsepower. Categorical variables are described by the answer to a multiple-choice question, like vehicle class. This chapter will describe some strategies for summarizing relationships among both kinds of variables, as well as some further refinements to this basic “numerical versus categorical” distinction.

Table 1.1: A simple example of a data frame. Each case is a car, and there are five variables: horsepower, city gas mileage, highway gas mileage, weight (in pounds), and vehicle class.

| | Horsepwer | CityMPG | HighwayMPG | Weight | Class |
|---------------------|-----------|---------|------------|--------|--------|
| BMW 325xi | 184 | 19 | 27 | 3461 | Sedan |
| Chevrolet Corvette | 350 | 18 | 25 | 3248 | Sports |
| Mercedes-Benz CL500 | 302 | 16 | 24 | 4085 | Sedan |
| Dodge Neon | 132 | 29 | 36 | 2626 | Sedan |
| Acura MDX | 265 | 17 | 23 | 4451 | SUV |

Variation across categories

MANY OF the data sets you'll meet will involve categories: chocolate or vanilla; rap or country; Toyota, Honda, or Hyundai; butcher or baker or candlestick maker. A simple, effective way to summarize these categorical variables¹ is to use a *contingency table*. On the Titanic, for example, a simple two-way table reveals that women and children survived in far greater numbers than adult men:

| | Girl | Woman | Boy | Man |
|----------|------|-------|-----|-----|
| Survived | 50 | 242 | 31 | 104 |
| Died | 22 | 74 | 51 | 472 |

We call this a two-way or bivariate table because there are two variables being compared: survival status versus type of person. The categories go along the rows and columns of the table; the cell counts show how many cases fall into each class. The process of sorting cases into the cells of such a table is often called *cross-tabulation*.

We can also make multi-way tables that show more than two variables at once. Given the constraints of a two-dimensional page, multiway tables are usually displayed as a series of two-way tables. As the following three-way table reveals, richer passengers, of either sex, fared better than others.

| | | Cabin Class | 1st | 2nd | 3rd |
|--------|----------|-------------|-----|-----|-----|
| | | Survived | 139 | 94 | 106 |
| | | Died | 5 | 12 | 110 |
| Female | Survived | 61 | 25 | 75 | |
| | Died | 118 | 146 | 418 | |
| Male | Survived | 61 | 25 | 75 | |
| | Died | 118 | 146 | 418 | |

Tables are almost always the best way to display categorical data sets with few classifying variables, for the simple reason that they convey a lot of information in a small space.²

Ordinal and binary variables. If a categorical variable has only two options (heads or tails, survived or died), we often call it an indicator, binary, or dummy variable. (These names can be used interchangeably.)

¹ Categorical variables are sometimes referred to as *factors*, and the categories themselves as the *levels* of the factor. The R statistical software package uses this terminology.

Table 1.2: A two-way table, because there are two categorical variables by which cases are classified. The data are available in the R package *effects*. Originally compiled by Thomas Cason from the *Encyclopedia Titanica*.

Table 1.3: An example of a *multi-way table*, where counts are classified by cabin class, sex, and survival. NB: passengers of unknown age are included in this table, but not the previous one.

² This animation provides some good guidelines for formatting tables.

Some categories have a natural ordering, like measures of severity for a hurricane, or responses to a survey about consumer satisfaction. (Has your experience with our call center been Atrocious, Merely Bad, Acceptable, Good, or Excellent?) These are called *ordinal variables*. Ordinal variables differ from numerical variables in that, although they can be placed in a definite order, they cannot be compared using the laws of arithmetic. For example, we can't subtract "Good" from "Excellent" and get a meaningful answer, in the way we can subtract \$1000 from \$5000 and get a number.

Relative risk

The **relative risk**, sometimes also called the risk ratio, is a widely used measure of association between two categorical variables. To introduce this concept, let's examine a tidbit of data from the PREDIMED trial, a famous study on heart health conducted by Spanish researchers that followed the lifestyle and diet habits of thousands of people over many years, beginning in 2003.³

The main purpose of the PREDIMED trial was to assess the effect of a Mediterranean-style diet on the likelihood of someone experiencing a major cardiovascular event (defined by the researchers as a heart attack, stroke, or death from cardiovascular causes). But as part of the study, the researchers also collected data on whether the trial participants were, or had ever been, regular smokers. The table below shows the relationship between smoking and whether someone experienced a cardiovascular event during the study period.

| Current or former smoker? | | |
|---------------------------|-----------------------|------------------------|
| | No (<i>n</i> = 3892) | Yes (<i>n</i> = 2432) |
| No event | 3778 | 2294 |
| Event | 114 | 138 |

Let's compare the absolute risk of cardiovascular events for smokers, versus that of non-smokers.⁴ Among the smokers, 138 of 2432 people (5.7%) experienced an event; while among the non-smokers, 114 of 3892 people (2.9%) experienced an event. To compute the relative risk of cardiovascular events among smokers, we take the ratio of these two absolute risks:

$$\text{Relative risk} = \frac{138/2432}{114/3892} = 1.94.$$

³ Estruch R, Ros E, Salas-Salvado J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med* 2013;368:1279-1290. The full text of the article is available at <http://www.nejm.org/doi/full/10.1056/NEJMoa1200303>

⁴ By "absolute risk," we simply mean the chance of an event happening.

This ratio says that smokers were 1.94 times more likely than non-smokers to experience a cardiovascular event during the study.⁵

More generally, for any event (a disease, a car accident, a mortgage default) and any notion of “exposure” to some factor (smoking, driving while texting, poor credit rating), the relative risk is

$$\text{Relative risk} = \frac{\text{Risk of event in exposed group}}{\text{Risk of event in non-exposed group}}.$$

The relative risk tells us how much more (or less) likely the event is in one group versus another. It’s important to remember that the relative risk (in our example, 1.94 for smokers) is quite different from the *absolute risk* (in our example, 0.057 for smokers). This distinction is often missed or elided in media coverage of health issues. See, for example, [this blog post](#) from the UK’s cancer-research funding body about news reports of cancer studies.

⁵ Of course, this doesn’t prove that the smoking caused the cardiovascular events. One could argue that the smokers may have had other systematically unhealthier habits that did them in instead, and the smoking was merely a marker of these other habits. We’ll soon talk about this issue of confounding much more.

Variation of numerical variables

FIGURE 1.1 depicts a histogram of daily average temperatures in two American cities—San Diego, CA, and Rapid City, SD—for every day from January 1995 to November 2011. Temperature is an example of a *numerical variable*, or something for which numerical comparisons are meaningful (twice as far, six times as fast, \$17 cheaper, and so forth). Numerical variables can be *discrete* or *continuous*. Temperature is continuous; we measure it in arbitrarily small increments. Marbles, on the other hand, are discrete; we count them on our fingers and toes.

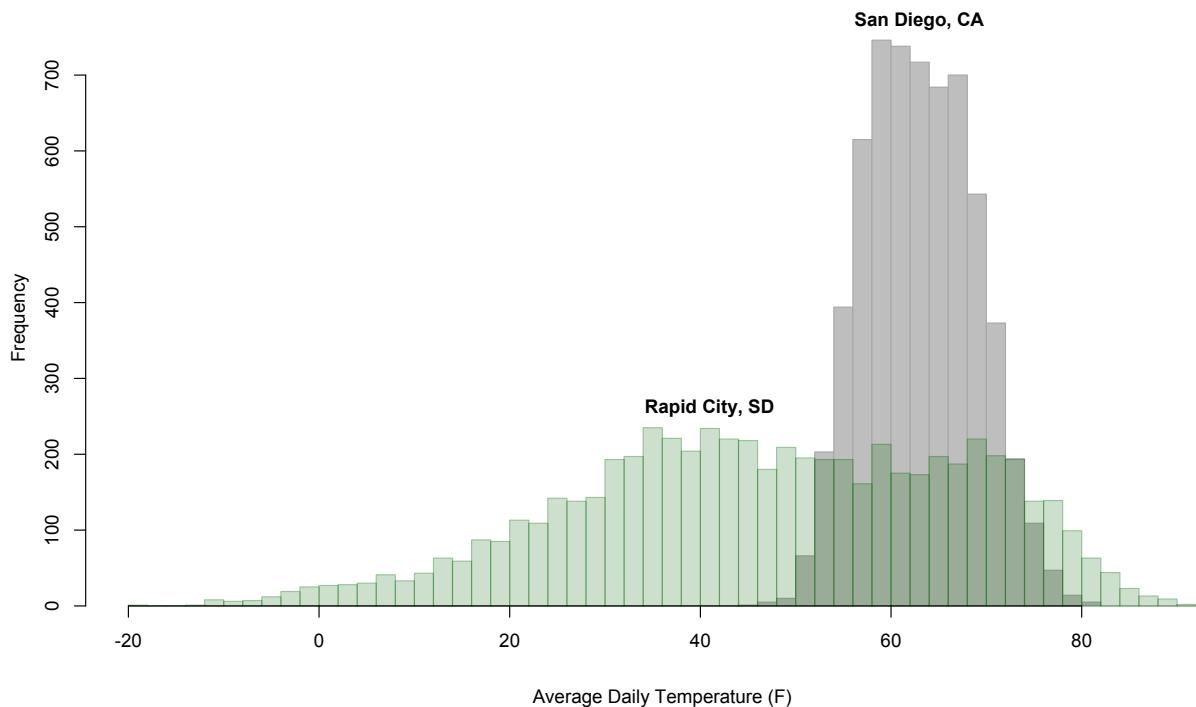
A histogram is a great way to depict the distribution of a numerical variable. To construct one, we first partition the range of possible outcomes (here, temperatures) into a set of disjoint intervals (“bins”). Next, we count the number of cases that fall into each bin. Finally, we draw a rectangle over each bin whose height is equal to the count within each bin.⁶

The histogram in Figure 1.1 suggest two obvious, meaningful questions we can ask about a numerical variable like temperature: where is the middle of the sample, and how much does a typical case vary from the middle?

You’re probably already aware of more than one way to answer the question, “Where is the middle?”

- There’s the sample mean, written as \bar{y} . If we have n data

⁶ Technically this is called a frequency histogram; one could also make a *density histogram* in which the heights of the bars are scaled appropriately so that the total area of all the bars sums to 1.



points $\{y_1, \dots, y_n\}$, then

$$\bar{y} = \frac{1}{n} \sum_{i=1}^n y_i.$$

The subscript i 's run from case 1 to case n , where n is the number of data points in the sample. In many data sets the actual ordering of cases won't matter, and will just reflect the arbitrary ordering of the rows in your data frame.⁷

- There's the median, or the halfway point in a sample.
- There's also the mode, or the most common value.

These different ways of quantifying the middle value all have different properties. For example, the median is less sensitive than the mean to extreme values in your sample; there can be more than one mode in a sample, but only one mean or median.⁸

Sample standard deviation and sample variance

Another important question is, “How spread out are the data points from the middle?” Figure 1.1 drives home the importance

Figure 1.1: Daily average temperatures for San Diego and Rapid City, 1995–2011. These data are visualized in a histogram, which is a simple and effective way to depict the variation of a single numerical variable across many cases.

⁷ An obvious exception is in the analysis of time-series data, where the ordering of observations in time may be highly meaningful.

⁸ For example, consider the data set $\{1, 2, 3, 3, 4, 4, 5\}$.

of dispersion in making useful comparisons. Not only are average temperatures lower overall in Rapid City than in San Diego, but they are also a lot more variable: the coldest days are much colder in Rapid City, but the hottest days are hotter, too.

As with the notion of “middle” itself, there is more than one way of quantifying variability, and each way is appropriate for different purposes. Let’s follow the line of thinking that leads us to the *standard deviation*, which is probably the most common way of measuring dispersion. Suppose we choose to measure the middle of a sample y_1, \dots, y_n using the mean, \bar{y} . Each case varies from this middle value by its *deviation*, $y_i - \bar{y}$. Why not, therefore, just compute the average deviation from the mean? Well, because

$$\begin{aligned}\frac{1}{n} \sum_{i=1}^n (y_i - \bar{y}) &= \frac{1}{n} \sum_{i=1}^n y_i - \frac{n}{n} \bar{y} \\ &= \bar{y} - \bar{y} \\ &= 0.\end{aligned}$$

The positives and negatives cancel each other out. We could certainly fix this by taking the absolute value of each deviation, and then averaging those:

$$M = \frac{1}{n} \sum_{i=1}^n |y_i - \bar{y}|.$$

This quantity is a perfectly sensible measure of the “typical deviation” from the middle. Fittingly enough, it is called the *mean absolute deviation* of the sample.

But it turns out that, for the purposes of statistical modeling, a quantity called the *sample variance* makes more sense:

$$s^2 = \frac{1}{n-1} \sum_{i=1}^n (y_i - \bar{y})^2.$$

That is, we *square* each deviation from \bar{y} , rather than take the absolute value. Remember that when we square a negative number, it becomes positive, so that we don’t have the problem of the positives and negatives cancelling each other out.

The definition of sample variance raises two questions:

- (1) Why do we divide by $n - 1$, when dividing by n would seem to make more sense for computing an average?
- (2) Why do we square the deviations, instead of taking absolute values as above?

To answer the first question: we divide by $n - 1$ rather than n for obscure technical reasons that, despite what you may read in other statistics textbooks, just aren't that important. (It has to do with "unbiased estimators," which, despite the appealing name, are overrated.) Mainly we use $n - 1$ to follow convention.

As for the second question: because sums of squares are special! In all seriousness, there are deep mathematical reasons why we choose to measure dispersion using sums of squared deviations, rather than the seemingly more natural sums of absolute deviations. You'll learn why in a future chapter, but if you want a preview, think about Pythagoras and right triangles. . . .

Of course, computing the sample variance leaves us in the awkward position of measuring variation in the *squared* units of whatever our variable is measured in. This is not intuitive; imagining telling someone that the mean temperature in Rapid City over the last 17 years was 47.3 degrees Fahrenheit, with a sample variance of 402 degrees squared. This is a true statement, but nearly uninterpretable.

Luckily, this is easily fixed by taking the square root of the sample variance, giving us the sample standard deviation:

$$s = \sqrt{\frac{1}{n-1} \sum_{i=1}^n (y_i - \bar{y})^2}. \quad (1.1)$$

Now we're back to the original units, and an interpretable measure of "typical deviation from the middle"—for Rapid City, 20.1 degrees. This looks about right from the histogram below; the blue dot is the sample mean, and the blue line stretches 1 sample standard deviation to either side of the mean.

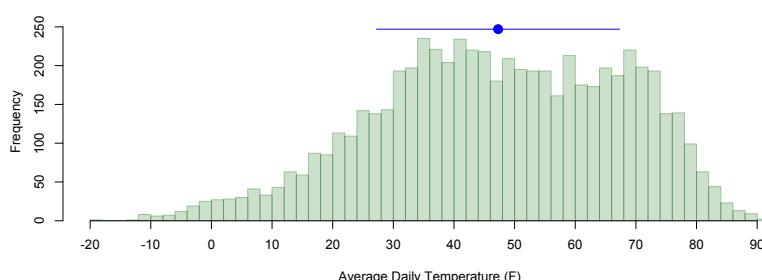


Figure 1.2: The histogram shows average daily temperatures in Rapid City. The blue dot is the sample mean, and the blue line shows an interval encompassing one sample standard deviation to either side of the sample mean.

Two other simple measures of spread are worth mentioning briefly. First, there's the *range*, or the difference between the largest

and smallest values in the sample. There's also the *interquartile range*, or the difference between the 75th and 25th percentiles. This is robust to extreme values, since it involves only the middle 50% of the sample.

Percentiles, quantiles, and coverage intervals

Another useful way to summarize the variation of a numerical variable across cases is to compute a set of percentiles, also called quantiles. A familiar example is the median: it happens that exactly 50% of the daily average temperatures in Rapid City fall below fall below 47.6 degrees, and we call this point the median (or the 50th percentile). Similarly, 10% of days in Rapid City are colder than 20.7 degrees, and 90% of days are colder than 73.2 degrees; these are the 10th and 90th percentiles, respectively. A quantile is just a percentile expressed in terms of a decimal fraction; the 80th percentile and 0.8 quantile are the same number.

A common way to summarize a distribution of a numerical variable is to quote a *coverage interval* defined by two percentiles, like the 10th and 90th percentiles (which covers 80% of the cases) or the 2.5th and 97.5th percentiles (which covers 95% of the cases). So, for example, we might quote an 80% coverage interval for daily average temperatures in Rapid City as (20.7, 73.2), whose endpoints are formed from the 10th and 90th percentiles.

Standardization by z-scoring

Which temperature is more extreme: 50 degrees in San Diego, or 10 degrees in Rapid City? In an absolute sense, of course 10 degrees is a more extreme temperature. But what about in a relative sense? In other words, is a 10-degree day more extreme *for Rapid City* than a 50-degree day is *for San Diego*? This question could certainly be answered using quantiles, which you've already learned how to handle. But let's discuss a second way: by calculating a z-score for each temperature.

The z-score of some quantity x is the number of standard deviations by which x is above its mean. If a z-score is negative, then the corresponding observation is below the mean.

To calculate a z-score for a number x , we subtract the corresponding mean μ and divide by the standard deviation σ :

$$z = \frac{x - \mu}{\sigma}.$$

For a 50-degree day in San Diego, this is:

$$z = \frac{50 - 63.1}{5.7} \approx -2.3.$$

Or about 2.3 standard deviations below the mean. On the other hand, for a 10-degree day in Rapid City, the z-score is

$$z = \frac{10 - 47.3}{20.1} \approx -1.9.$$

Or about 1.9 standard deviations below the mean. Thus a 50-degree day in San Diego is actually more extreme than a 10-degree day in Rapid City! The reason is that temperatures in Rapid City are both colder on average (lower mean) and more variable (higher standard deviation) than temperatures in San Diego.

As this example suggests, z-scores are useful for comparing numbers that come from different distributions, with different statistical properties. It tells you how extreme a number is, relative to other numbers from that same distribution. We often think of the normal distribution as a useful reference here for interpreting z-scores. The normal distribution has the property that about 68% of observations fall within $z = 1$ standard deviation of the mean, and about 95% fall within $z = 2$ standard deviations.

Variation between, and within, groups

A COMMON situation is that we have both categorical and numerical data about each case in a data set. For example, Table 1.4 below shows the average SAT math and verbal scores, stratified by college, for undergraduates in the incoming fall of 2000 freshmen class at the University of Texas at Austin. All 5,191 students who went on to receive a bachelor's degree within 6 years are included; those who dropped out, for whatever reason, are not.

The table tells you something about how the numerical variables (test scores) change depending upon the categorical variable (college), and they are superficially similar to the contingency tables we just encountered. They highlight interesting and useful facts about variation between the groups. Math skills, for example, are probably more important for engineering majors than English majors, and this is reflected in the differences between the group-level means.

| College | Average SAT | |
|------------------|-------------|--------|
| | Math | Verbal |
| Architecture | 685 | 662 |
| Business | 633 | 597 |
| Communications | 592 | 609 |
| Education | 555 | 546 |
| Engineering | 675 | 606 |
| Fine Arts | 597 | 594 |
| Liberal Arts | 598 | 590 |
| Natural Sciences | 633 | 597 |
| Nursing | 561 | 555 |
| Social Work | 602 | 589 |

Table 1.4: Average SAT math and verbal scores, stratified by college, for entering freshmen at UT–Austin in the fall of 2000. Collected under the Freedom of Information Act from the state of Texas.

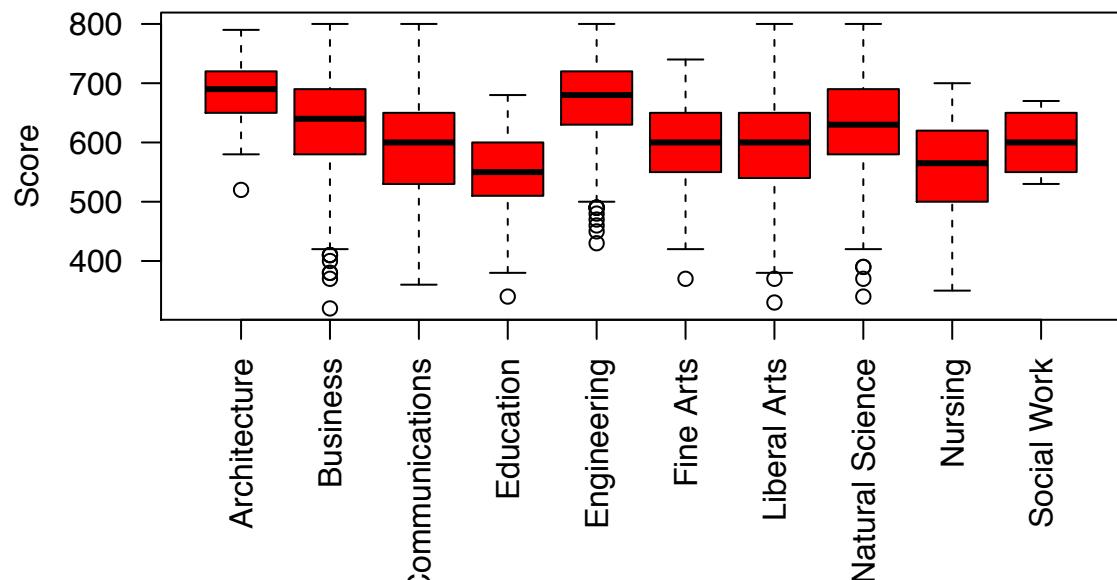
Table 1.4 does differ from a contingency table, however, in one crucial respect: the entries in the table are not counts, but group-level averages. Notice that, to depict between-group variation, the table has reduced each college to a typical case, represented by some hypothetical student who earned the college-wide average SAT scores on both the math and verbal sections. In doing so, it has obscured the underlying variability of students *within* the colleges. But as our example of city temperatures demonstrated, sometimes this variability is an important part of the story as well.

Boxplots

This is where boxplots are useful: they allow you to assess variability both between and within the groups. In a boxplot, like the ones shown in Figure 1.3, there is one box per category. (The top panel shows a boxplot for SAT Math scores; the bottom, for SAT Verbal scores.) Each box shows the *within-group variability*, as measured by the interquartile range of the numerical variable (SAT score) for all cases in that category. The middle line within each box is the median of that category, and the differences between these medians give you a sense of the *between-group variability*. In this boxplot, the whiskers extend outside the box no further than 1.5 times the interquartile range. Points outside this interval are shown as individual dots.

A table like 1.4 focuses exclusively on the between-group variability; it reduces each category to a single number, and shows how those numbers vary from one category to the next. But in

SAT Math Scores by UT College



SAT Verbal Scores by UT College

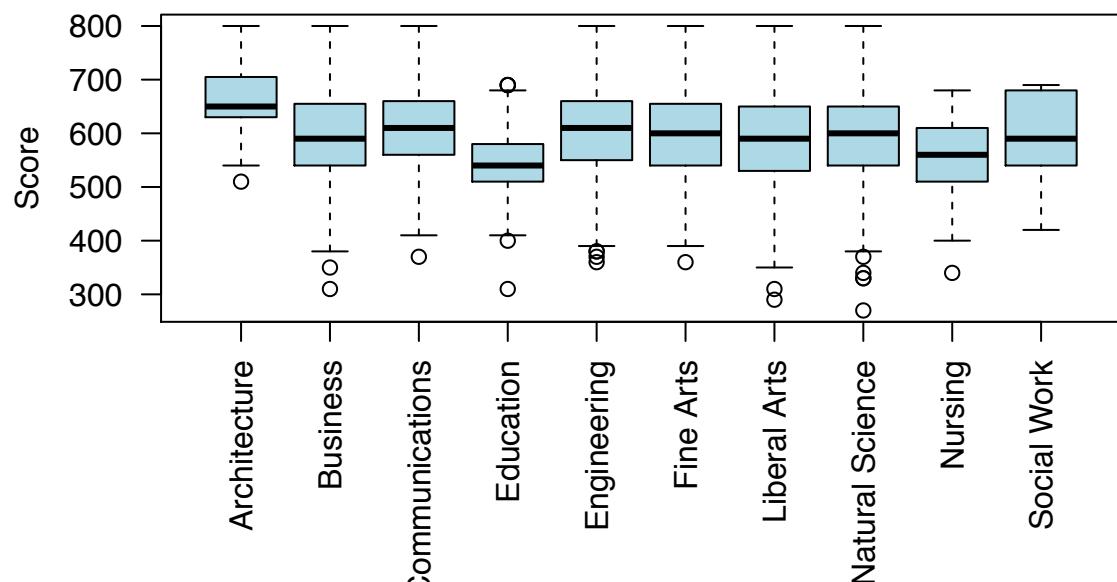


Figure 1.3: Boxplots of the full data set used to form the means in Table 1.4.

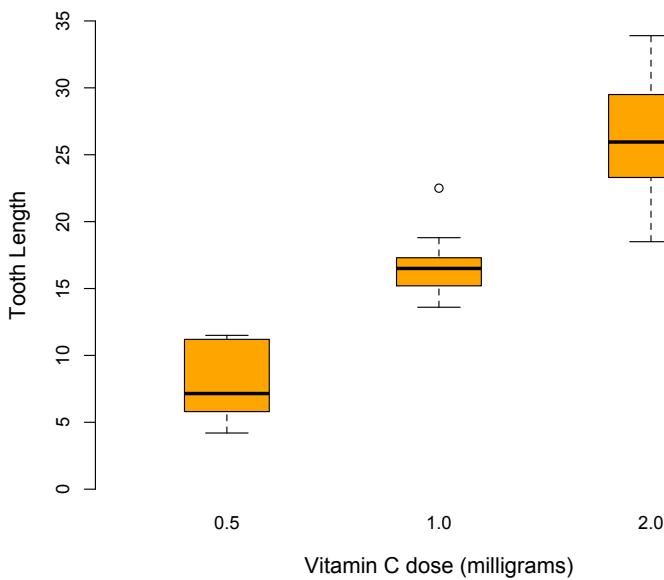


Figure 1.4: For comparison, the table of within-group means is below. Notice how the within-group variability evident in the boxplots at left simply disappears when presented in the form of a summary table, below:

| Dose (mg) | Tooth len. |
|-----------|------------|
| 0.5 | 7.98 |
| 1.0 | 16.77 |
| 2.0 | 26.14 |

many data sets, it is actually the within-group variability that matters most. For example, as Figure 1.3 shows, SAT scores vary much more within a college as they do between colleges. For example, there is 52-point difference in average SAT math scores between Architecture students and Natural Science students. But within Natural Sciences, the interquartile range is nearly twice as large: 100 points.

The situation is quite different Figure 1.4. These boxplots show the growth of guinea pigs' teeth versus their daily dosage of Vitamin C. Like humans, but unlike most other mammals, guinea pigs need Vitamin C to keep rollin', yet they cannot synthesize their own. Their vitamin C intake is strongly predictive of their overall health, measured in this case by the length of their teeth. In this boxplot, we see comparatively more variability between the groups, whose boxplots almost don't overlap.

The same comparison will come up again and again: between-group variability (the differences between typical or average group members) versus within-group variability (the variation of cases within a single group). We'll soon make this comparison mathematically rigorous, but these examples convey the essence of the idea:

- A UT student's college tells you something, though not ev-



erything, about his or her likely SAT scores.

- A guinea pig's Vitamin C regimen tells you something, though not everything, about its tooth growth. But in a relative sense, it tells you more than a UT student's college tells you about his or her SAT scores.

Always remember that a table of group-wise means does not depict "data" as such, but an abstraction of some typical group member. This abstraction may be useful for some purposes. But within-group variability is also important, and may even be the dominant feature of interest. In this case, presenting the group-wise means alone, without the corresponding plots or measures of variability, may obscure more than it reveals.

Dot plots

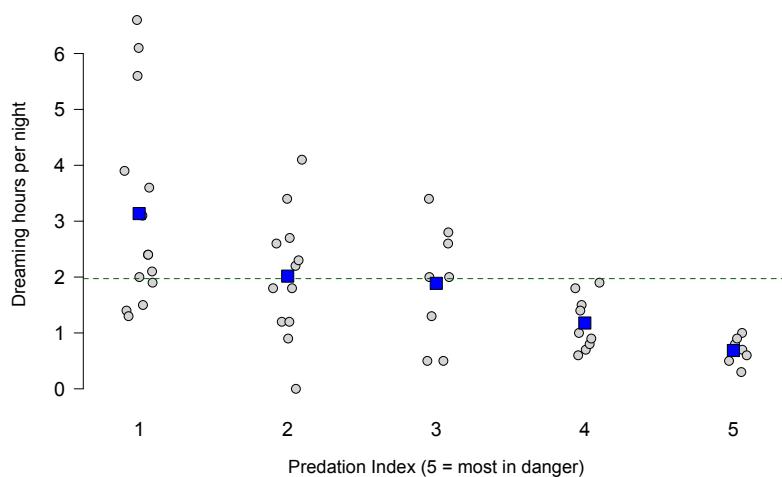
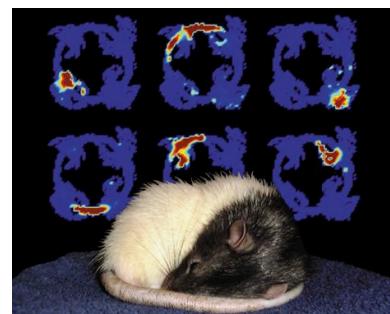
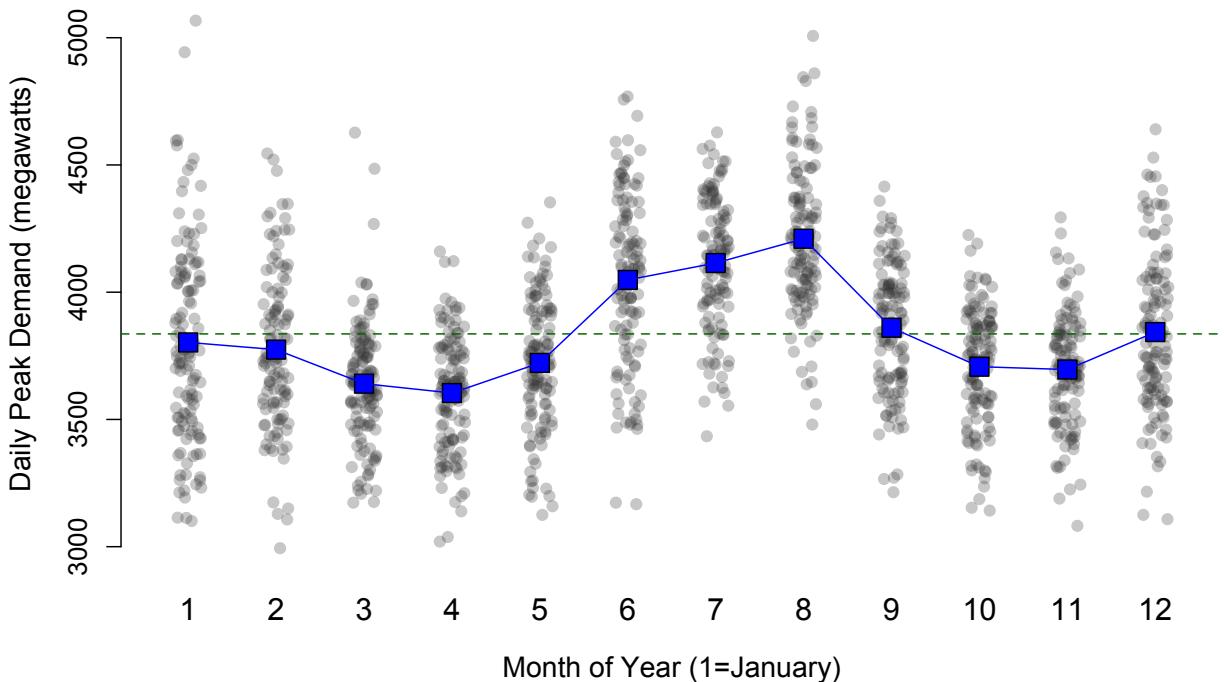


Figure 1.5: Dreaming hours per night versus danger of predation for 50 mammalian species. Data from: "Sleep in Mammals: Ecological and Constitutional Correlates," Allison and Cicchetti (1976). *Science*, November 12, vol. 194, pp. 732-734. Photo of the dreaming critter from the MIT News office (web.mit.edu/newsoffice/2001/dreaming.html).

The *dot plot* is a close cousin of the boxplot. For example, the plot in Figure 1.5 depicts a relationship between the length of a mammal's dreams (as measured in a lab by an MRI machine) and the severity of the danger it faces from predators. Each dot is a single species of mammal—like, for example, the dreaming critter at right. The predation index is an ordinal variable running from 1 (least danger) to 5 (most danger). It accounts both for how likely an animal is to be preyed upon, and how exposed it is when sleeping. Notice the direction of the trend—you'd sleep poorly too if you were worried about being eaten.





As you can see, the dot plot is useful for small data sets, when a boxplot is no simpler than just plotting the cases group by group. Strictly speaking, the points should all line up vertically with their corresponding values of predation index, on the x -axis. But a small amount of artificial horizontal jitter has been added to the dots, which allows the eye to distinguish the individual cases more easily.

Dot plots can also be effective for larger data sets. In Figure 1.6 we see four years of data on daily peak electricity demand for the city of Raleigh, NC, stratified by month of the year. Both the between-group and within-group variation show up clearly.

Group means and grand means

If you looked carefully, you may have noticed two extra features of the dot plots in Figures 1.5 and 1.6. The square blue dots show the *group means* for each category. The dotted green line shows the *grand mean* for the entire data set, irrespective of group identity.

Figure 1.6: Daily peak electricity demand (stratified by month) in Raleigh, NC from 2006–09. The dashed line is the average peak demand for the whole data set, and the blue dots are the month-by-month means.

Notice that, in plotting these means along with the data, we have implicitly partitioned the variability:

$$\text{Individual case} = \text{Group mean} + \text{Deviation of that case}$$

$$\text{Individual case} = \text{Grand mean} + \text{Deviation of group} + \text{Deviation of that case}$$

This is just about the simplest statistical model we can fit, but it's still very powerful. We'll revisit it soon.

More than one numerical variable

Our basic tool for visualizing a bivariate relationship between two numerical variables is the *scatter plot*. Figure 1.7 shows a plot of the daily returns for Microsoft stock versus Apple stock for every trading day in 2015. Every dot corresponds to a day. The location of the dot along the horizontal axis shows the Apple return, and the location on the vertical axis shows the Microsoft return, for that day. In this case, we can see that Microsoft and Apple stocks tend to move up and down together. (Most stocks do.) We can also see the speckling of outliers: those points that are visibly separate

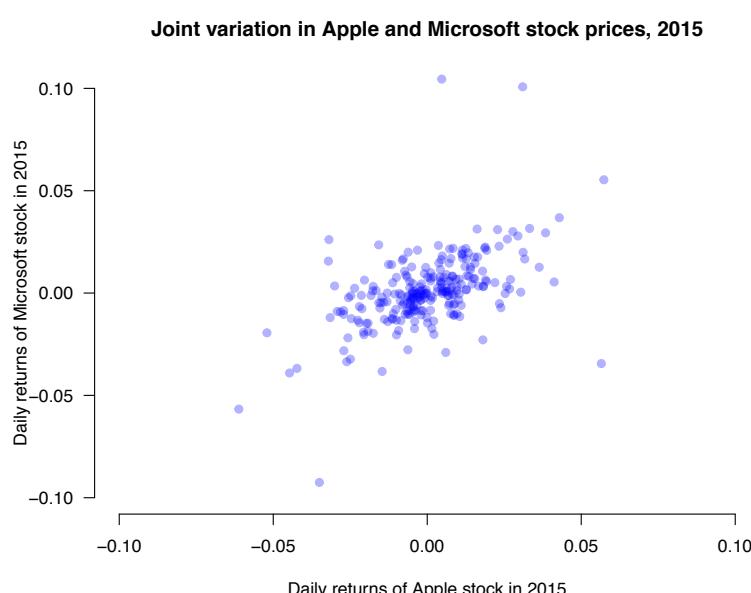


Figure 1.7: A scatter plot of the daily returns for Microsoft stock, versus those of Apple stock, for every trading day in 2015. The daily return is the implied interest rate from holding a stock from the end of one trading day to the end of the next. For example, Apple stock closed at \$105.95 per share on January 7th and at \$110.02 on January 8th. Thus the return for January 8th was

$$\frac{110.02 - 105.95}{105.95} \approx 0.038,$$

or about a 3.8% daily return. On the same day, holders of Microsoft stock enjoyed a 2.9% return.

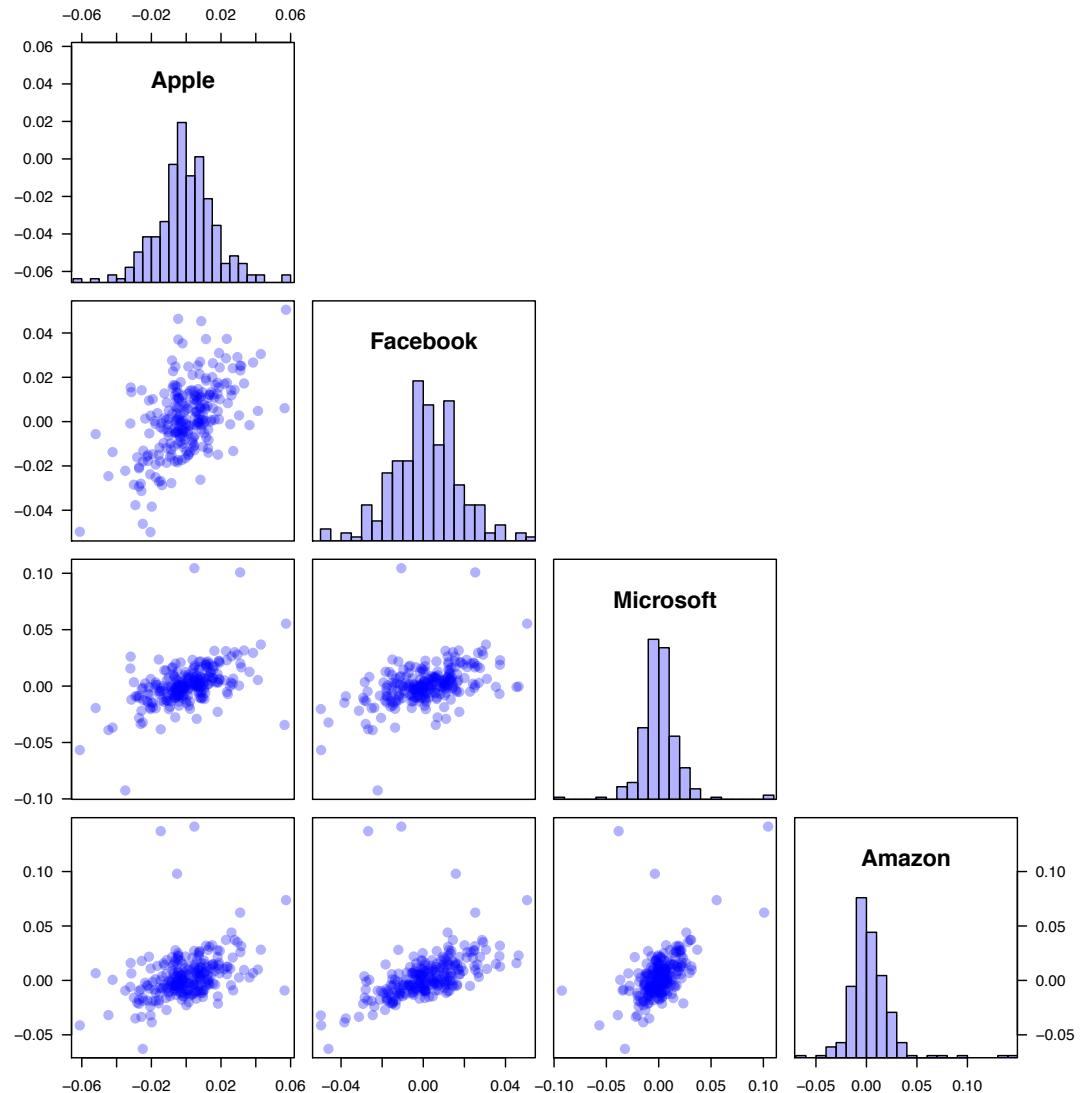


Figure 1.8: A pairs plot: a matrix of four pairwise scatter plots for the daily returns of Apple, Facebook, Microsoft, and Amazon stocks in 2015. The histograms along the diagonal also label the rows and columns of the matrix: e.g. the plot in the second row has Facebook returns along the vertical axis, while the plots in the second column both have Facebook returns along the horizontal axis.

from the main cloud and that represent very good (or bad) days for holders of these two stocks.

A simple way to visualize three or more numerical variables is via a *pairs plot*, as in Figure 1.8. A pairs plot is a matrix of simpler plots, each depicting a bivariate relationship. In Figure 1.8, we see scatterplots for each pair of the daily returns for Microsoft, Facebook, Apple, and Amazon stocks. The histograms on the diagonal serve a dual purpose: (1) they show the variability of each stock in isolation; and (2) they label the rows and columns, so that you know which plots compare which variables.

Sample correlation. The *sample correlation coefficient* is a standard measure of the strength of linear dependence between two variables in a sample. If we label the first variable as x_1, \dots, x_n and the second as y_1, \dots, y_n , then the correlation coefficient is defined as

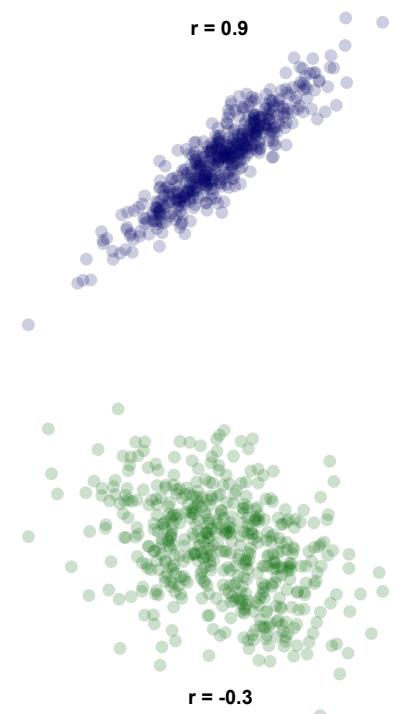
$$r = \frac{\sum_{i=1}^n (x_i - \bar{x})(y_i - \bar{y})}{(n-1)s_x s_y}, \quad (1.2)$$

where s_x and s_y are the sample standard deviations of the X and Y variables. At right you see scatter plots that depict examples of strong positive (top) and weak negative (bottom) correlation. Sample correlation is between 1 and -1 , which are the extremes of perfect positive and perfect negative correlation.

To summarize the correlation among a set of more than two variables, we typically calculate a *correlation matrix* whose entry in row i , column j is the correlation between variable i and variable j . For the four stocks depicted in Figure 1.8, the correlation matrix is below. Notice that the matrix is symmetric and has ones along the diagonal (because a variable is perfectly correlated with itself):

| | Apple | Microsoft | Facebook | Amazon |
|-----------|-------|-----------|----------|--------|
| Apple | 1.00 | 0.52 | 0.55 | 0.36 |
| Microsoft | 0.52 | 1.00 | 0.47 | 0.52 |
| Facebook | 0.55 | 0.47 | 1.00 | 0.50 |
| Amazon | 0.36 | 0.52 | 0.50 | 1.00 |

Caveats. A key fact to remember is that correlation measures the strength of *linear* dependence. If two variables don't fall roughly along a straight line in a scatter plot, then correlation can be misleading. For example, consider Figure 1.9: four different data sets, four different stories about what's going on. Yet all have the same correlation: $r = 0.816$.



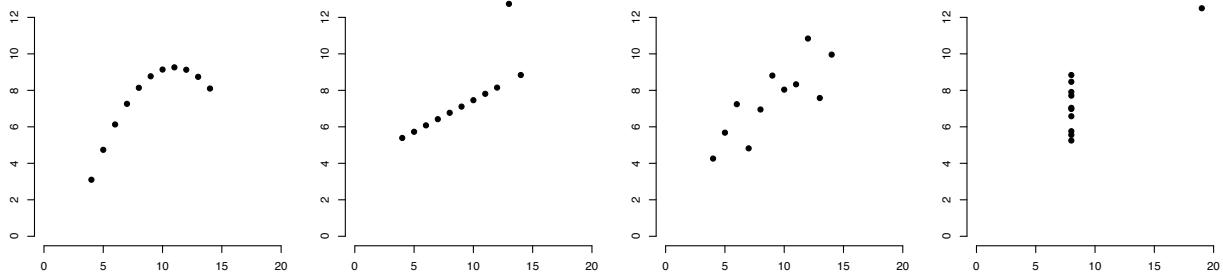


Figure 1.9: above. Data taken from F.J. Anscombe, "Graphs in Statistical Analysis." *American Statistician*, 27 (1973), pp. 17–21

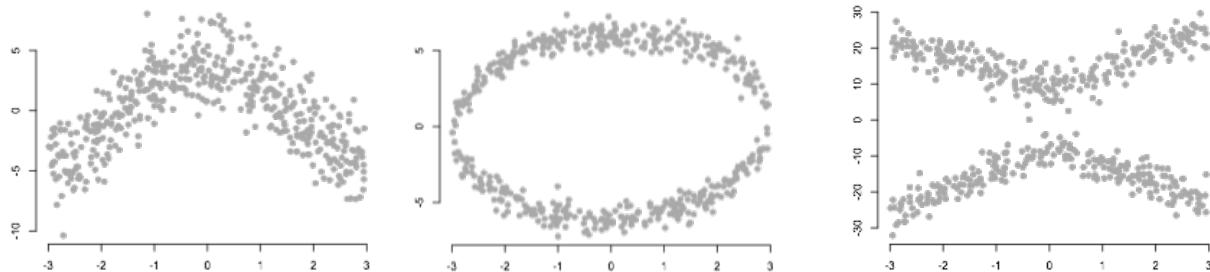


Figure 1.10: left. Each panel shows obvious dependence, but has a sample correlation of $r = 0$.

Another important fact is that a sample correlation of 0 ("uncorrelated") does not necessarily mean that two variables are unrelated. In fact, the correlation coefficient is so intimately tied up with the assumption of a linear relationship that it breaks down entirely when used to quantify the strength of nonlinear relationships. In each of the three plots in Figure 1.10, for example, there is an obvious (nonlinear) relationship between the two variables. Yet the sample correlation coefficient for each of them turns out to be exactly zero.

The lesson of these two plots is that you should always plot your data. After all, a sample correlation coefficient is just one number. It can only tell you so much about the relationship between two variables, and a scatterplot (or boxplot, or dot plot) is a much, much richer summary of that relationship.

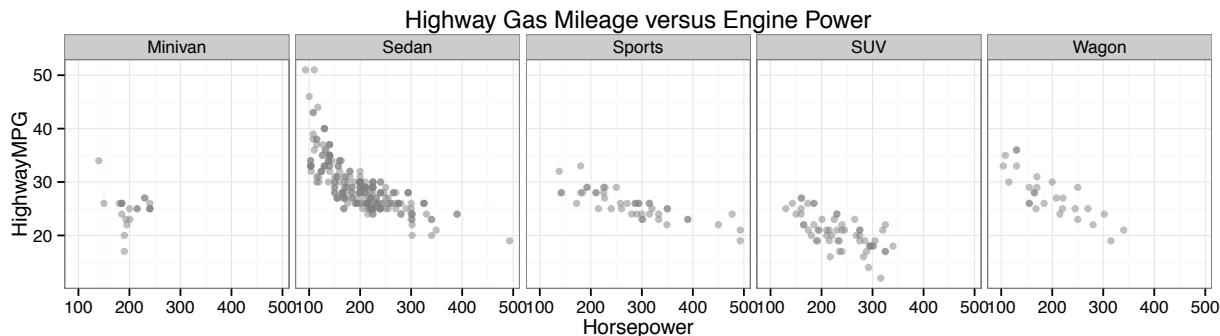


Figure 1.11: Highway gas mileage versus engine power for 387 vehicles in five different classes.

Further multivariate plots

In visualizing data, we are usually constrained by the limitations of the two-dimensional page or screen. Nevertheless, there are many cool techniques for showing more than two variables at once, despite these limitations.

Lattice plots

Figure 1.11 shows three variables from a data set on 387 vehicles: the highway gas mileage, the engine power (in horsepower), and the class of the vehicle (minivan, sedan, sports car, SUV, or wagon). This is done via a *lattice plot*, which displays the relationship between two variables, stratified by the value of some third variable. In this case the main relationship of interest is between mileage and engine power, and the stratifying variable is vehicle class. Notice how figure 1.11 repeats a scatterplot of MPG versus horsepower five times: one plot for the vehicles in each class. To facilitate comparisons across the strata, both the horizontal and vertical axes are identical in each plot.

The figure suggests several facts:

- Nobody makes a powerful minivan.
- The overall MPG–horsepower trend is negative for all classes.
- The SUVs have the worst gas mileage overall, and in particular have worse mileage than the sports cars and wagons despite having similar or lower power. (Compare the average vertical location in the SUV panel versus the others).
- The MPG–horsepower relationship becomes nonlinear for

Another term for a lattice plot is a trellis plot.

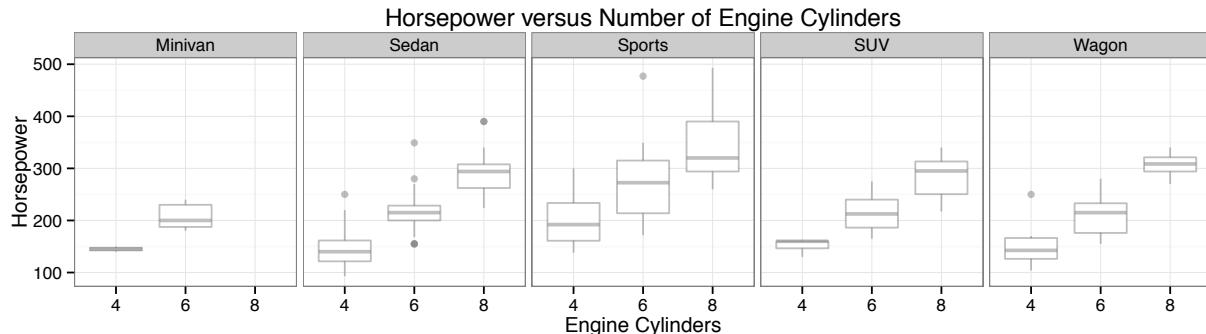


Figure 1.12: Highway gas mileage versus engine power for 387 vehicles in five different classes.

sedans at low horsepower, but perhaps not for wagons.

- As engine power increases, the dropoff in gas mileage looks steeper for SUVs than for sports cars.
- For a fixed level of engine power, there is considerable variability in fuel economy. (Pick a fixed point on the horizontal axis and focus on the cars near there. Now look at the corresponding variability along the vertical axis for those cars.)

We can make a lattice of boxplots as well. For example, Figure 1.12 shows boxplots of engine power versus number of engine cylinders, stratified by vehicle class. This suggests an explanation for the fact that engine power is not a perfect predictor of fuel economy: some cars get more power out of a smaller engine, and are presumably more efficient as a result.

With a numerical variable. In Figure 1.11, the stratifying variable is categorical. But we can also stratify a data set according to a numerical variable, by *discretizing* that variable into bins—much in the same way we do when we make a histogram. Figure 1.13 shows the latitude, longitude, and depth (in kilometers) beneath the earth’s surface for the epicenter of every earthquake recorded since 1963 near Fiji, an island in the South Pacific Ocean. The “depth” variable has been discretized into nine equal-length bins. The nine panels show the latitude and longitude of the quakes whose depths fell in each interval, labeled at the top of each panel.

As depth increases (going left to right, top to bottom), a spatial pattern emerges. The shallower earthquakes are at the intersection of two major tectonic plates. The deeper quakes emanate from the Tonga Trench—35,702 feet below the sea at its deepest point.⁹

⁹ And the final resting place of 3.9 kilograms of radioactive plutonium-238 from the ill-fated Apollo 13 mission.

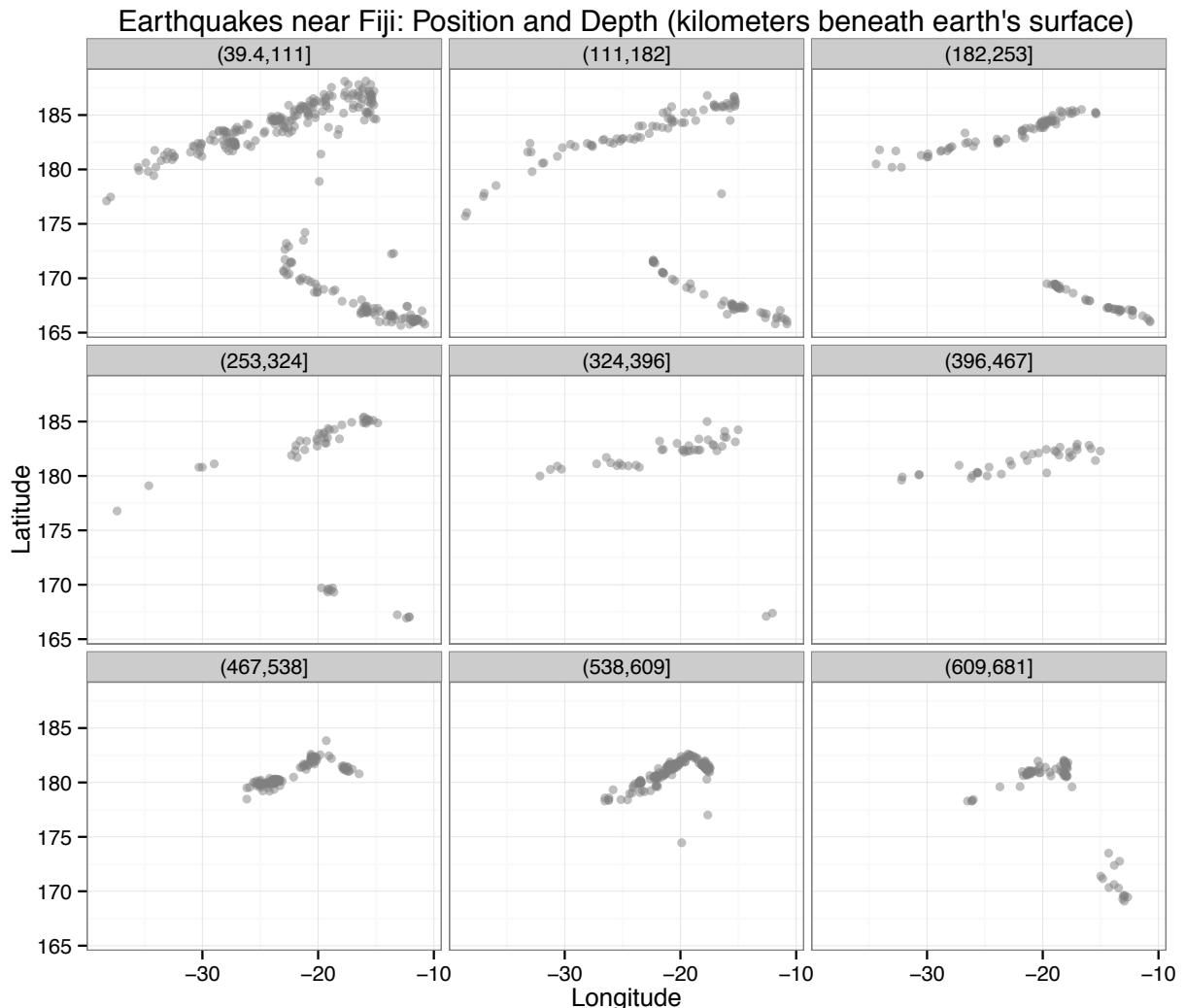


Figure 1.13: Earthquakes in Fiji: latitude versus longitude for quakes within each of nine different depth intervals. Here the range of depths beneath the earth's surface (in kilometers) is labeled at the top of each panel.

2

Fitting equations to data

SO FAR we've concentrated on relatively simple visual and numerical summaries of data sets. In many cases we will want to go further, by fitting an explicit equation—usually called a *regression model*—that describes how one variable changes as a function of some other variables. There are many reasons we might want to do this. Here are four that we'll explore at length:

- to make a forecast;
- to summarize the trend in a data set;
- to make comparisons that adjust statistically for some systematic effect; and
- to quantify the amount of variability in some variable that cannot be predicted, in the context of what *can* be predicted.

This chapter introduces the idea of a regression model and builds upon these themes.

Fitting straight lines

As a running example we'll use the data from Figure 2.1, which depicts a sample of 104 restaurants in the vicinity of downtown Austin, Texas. The horizontal axis shows the restaurant's "food deliciousness" rating on a scale of 0 to 10, as judged by the writers of a popular guide book entitled *Fearless Critic: Austin*. The vertical axis shows the typical price of a meal for one at that restaurant, including tax, tip, and drinks. The line superimposed on the scatter plot captures the overall "bottom-left to upper-right" trend in the data, in the form of an equation: in this case, $y = -6.2 + 7.9x$. On average, it appears that people pay more for tastier food.

This is our first of many data sets where the response (price, Y) and predictor (food score, X) can be described by a linear regression model. We write the model in two parts as " $Y = \beta_0 + \beta_1 X + \text{noise}$." The first part, the function $\beta_0 + \beta_1 X$, is called the *linear predictor*—linear because it is the equation of a straight

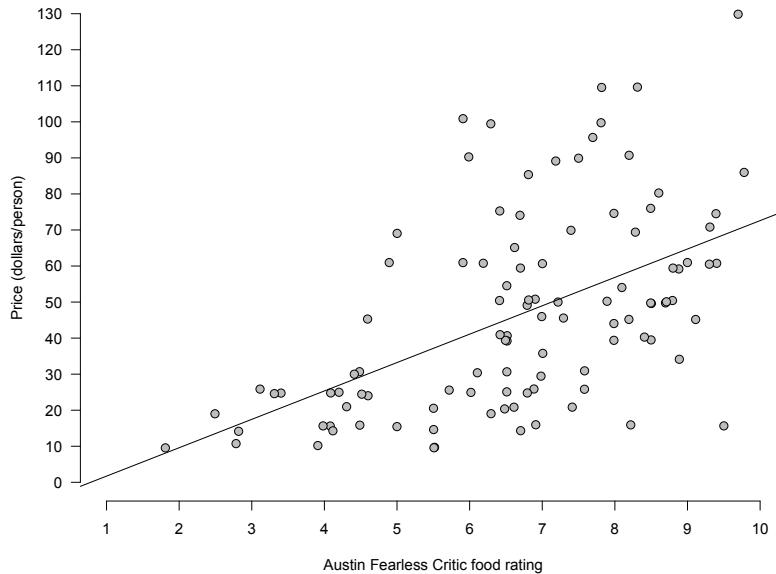


Figure 2.1: Price versus reviewer food rating for a sample of 104 restaurants near downtown Austin, Texas. The data are from a larger sample of 317 restaurants from across greater Austin, but downtown-area restaurants were chosen to hold location relatively constant. Data from Austin Fearless Critic, www.fearlesscritic.com/austin. Because of ties in the data, a small vertical jitter was added for plotting purposes only. The equation of the line drawn here is $y = -6.2 + 7.9x$.

line, predictor because it predicts Y . The second part, the noise, is a crucial part of the model, too, since no line will fit the data perfectly. In fact, we usually denote each individual noise term explicitly:

$$y_i = \beta_0 + \beta_1 x_i + e_i. \quad (2.1)$$

An equation like (2.1) is our first example of a regression model. The *intercept* β_0 and the *slope* β_1 are called the *parameters* of the regression model. They provide a mathematical description of how price changes as a function of food score. The little e_i is called the *residual* for the i th case—residual, because it's how much the line misses the i th case by (in the vertical direction). The residual is also a fundamental part of the regression model: it's what's “left over” in y after accounting for the contribution of x .

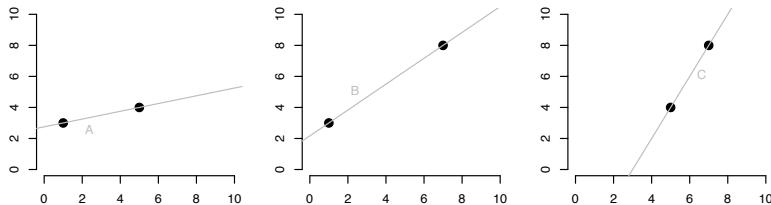
For every two points. . . .

A natural question is: how do we fit the parameters β_0 and β_1 to the observed data? Historically, the standard approach, still in widespread use today, is to use the method of least squares. This involves choosing β_0 and β_1 so that the sum of squared residuals (the e_i 's) will be as small as possible. This is what we did to get the equation $y_i = -6.2 + 7.9x_i$ in Figure 2.1.

The method of least squares is one of those ideas that, once

you've encountered it, seems beautifully simple, almost to the point of being obvious. But it's worth pausing to consider its historical origins, for it was far from obvious to a large number of very bright 18th-century scientists.

To see the issue, consider the following three simple data sets. Each has only two observations, and therefore little controversy about the best-fitting linear trend.



For every two points, a line. If life were always this simple, there would be no need for statistics.

But things are more complicated if we observe three points.

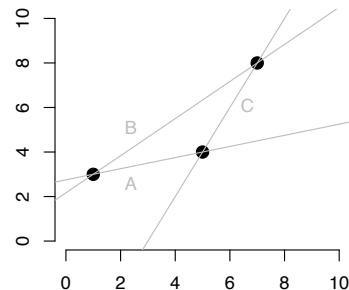
$$\begin{aligned} 3 &= \beta_0 + 1\beta_1 \\ 4 &= \beta_0 + 5\beta_1 \\ 8 &= \beta_0 + 7\beta_1 \end{aligned}$$

Two unknowns, three equations. There is no solution for the parameters β_0 and β_1 that satisfies all three equations—and therefore no perfectly fitting linear trend exists. Seen graphically, at right, it is clear that no line can pass through all three points.

Abstracting a bit, the key issue here is the following: how are we to combine inconsistent observations? Any two points are consistent with a unique line. But three points usually won't be, and most interesting data sets have far more than three data points.

Therefore, if we want to fit a line to the data anyway, we must allow the line to miss by a little bit for each (x_i, y_i) pair. We express these small misses mathematically, as follows:

$$\begin{aligned} 3 &= \beta_0 + 1\beta_1 + e_1 \\ 4 &= \beta_0 + 5\beta_1 + e_2 \\ 8 &= \beta_0 + 7\beta_1 + e_3. \end{aligned}$$



The three little e 's are the residuals, or misses.

But now we've created a different predicament. Before we added the e_i 's to give us some wiggle room, there was no solution

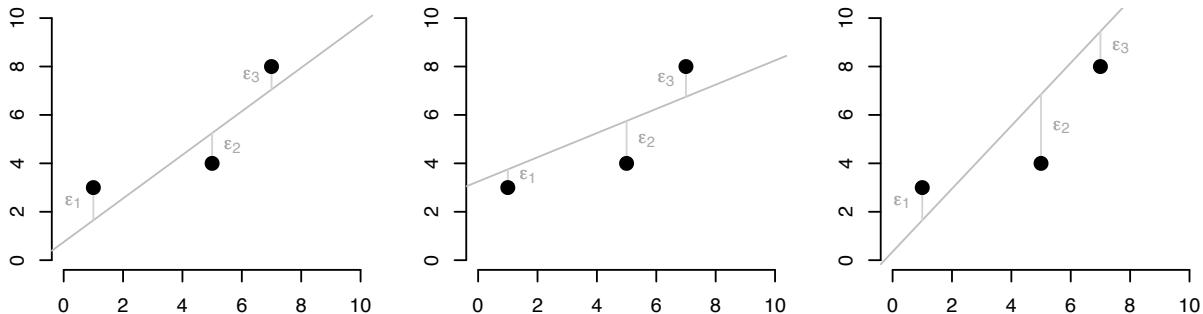


Figure 2.2: Three possible straight-line fits, each involving an attempt to distribute the “errors” among the observations.

to our system of linear equations. Now we have three equations and five unknowns: an intercept, a slope, and three residuals. This system has infinitely many solutions. How are we to choose, for example, among the three lines in Figure 2.2? When we change the parameters of the line, we change the residuals, thereby redistributing the errors among the different points. How can this be done sensibly?

Believe it or not, scientists of the 1700’s struggled mightily with this question. Many of the central scientific problems of this era concerned the combination of astronomical or geophysical observations. Astronomy in particular was a hugely important subject for the major naval powers of the day, since their ships all navigated using maps, the stars, the sun, and the moon. Indeed, until the invention of a clock that would work on the deck of a ship rolling to and fro with the ocean’s waves, the most practical way for a ship’s navigator to establish his longitude was to use a lunar table. This table charted the position of the moon against the “fixed” heavens above, and could be used in a roundabout fashion to compute longitude. These lunar tables were compiled by fitting an equation to observations of the moon’s orbit.

The same problem of fitting astronomical orbits arose in a wide variety of situations. Many proposals for actually fitting the equation to the data were floated, some by very eminent mathematicians. Leonhard Euler, for example, proposed a method for fitting lines to observations of Saturn and Jupiter that history largely judges to be a failure.

In fact, some thinkers of this period disputed that it was even a good idea to combine observations at all. Their reasoning was, roughly, that the “bad” observations in your sample would corrupt

the “good” ones, resulting in an inferior final answer. To borrow the phrase of Stephen Stigler, an historian of statistics, the “deceptively simple concept” that combining observations would improve accuracy, not compromise it, was very slow to catch on during the eighteenth century.¹

¹ *The History of Statistics*, p. 15.

The method of least squares

No standard method for fitting straight lines to data emerged until the early 1800’s, half a century after scientists first entertained the idea of combining observations. What changed things was the *method of least squares*, independently invented by two people. Legendre was the first person to publish the method, in 1805, although Gauss claimed to have been using it as early as 1794.

The term “method of least squares” is a direct translation of Legendre’s phrase “méthode des moindres carrés.” The idea is simple: choose the parameters of the regression line that minimize $\sum_{i=1}^n e_i^2$, the sum of the squared residuals. As Legendre put it:

In most investigations where the object is to deduce the most accurate possible results from observational measurements, we are led to a system of equations of the form

$$E = a + bx + cy + fz + \&c.,$$

in which $a, b, c, f, \&c.$ are known coefficients, varying from one equation to the other, and $x, y, z, \&c.$ are unknown quantities, to be determined by the condition that each value of E is reduced either to zero, or to a very small quantity. . . .

Of all the principles that can be proposed for this purpose, I think there is none more general, more exact, or easier to apply, than that which we have used in this work; it consists of making the sum of the squares of the errors a minimum. By this method, a kind of equilibrium is established among the errors which, since it prevents the extremes from dominating, is appropriate for revealing the state of the system which most nearly approaches the truth.²

The utility of Legendre’s suggestion was immediately obvious to his fellow scientists and mathematicians. Within two decades, least squares became the dominant method throughout the European scientific community.

Why was the principle adopted so quickly and comprehensively? For one thing, it offered the attractiveness of a single best answer, evaluated according to a specific, measurable criterion. This gave the procedure the appearance of objectivity—especially

² Adrien-Marie Legendre (1805), *Nouvelles méthodes pour la détermination des orbites des comètes*. Translation p. 13, Stigler’s *A History of Statistics*.

compared with previous proposals, many of which essentially amounted to: “muddle around with the residuals until you get an acceptable balance of errors among the points in your sample.”

Moreover, unlike many previous proposals for combining observations, the least-squares criterion could actually be applied to non-trivially large problems. One of the many advantages of the least-squares idea is that it leads immediately from grand principle to specific instructions on how to compute the estimate $(\hat{\beta}_0, \hat{\beta}_1)$:

$$\hat{\beta}_1 = \frac{\sum_{i=1}^n (x_i - \bar{x})(y_i - \bar{y})}{\sum_{i=1}^n (x_i - \bar{x})^2} \quad (2.2)$$

$$\hat{\beta}_0 = \bar{y} - \hat{\beta}_1 \bar{x}, \quad (2.3)$$

where \bar{x} and \bar{y} are the sample means of the X and Y variables, respectively. The line $y = \hat{\beta}_0 + \hat{\beta}_1 x$ is the best possible linear fit to the data, in a squared-error sense. That is to say: among the family of all possible straight-line fits to the data, this particular line has the smallest sum of squared residuals. Deriving this solution involves solving a simple mathematical problem involving some calculus and matrix algebra—something that scientists of the nineteenth century could do easily, via pen and paper.

In statistics, a little hat on top of something usually denotes a guess or an estimate of the thing wearing the hat.

Goals of regression analysis

WITH modern computers, the estimation of linear regression models by least squares is now entirely automatic for all but the very largest of data sets.³ It’s so ordinary, in fact, that the method is often abbreviated as OLS: ordinary least squares.

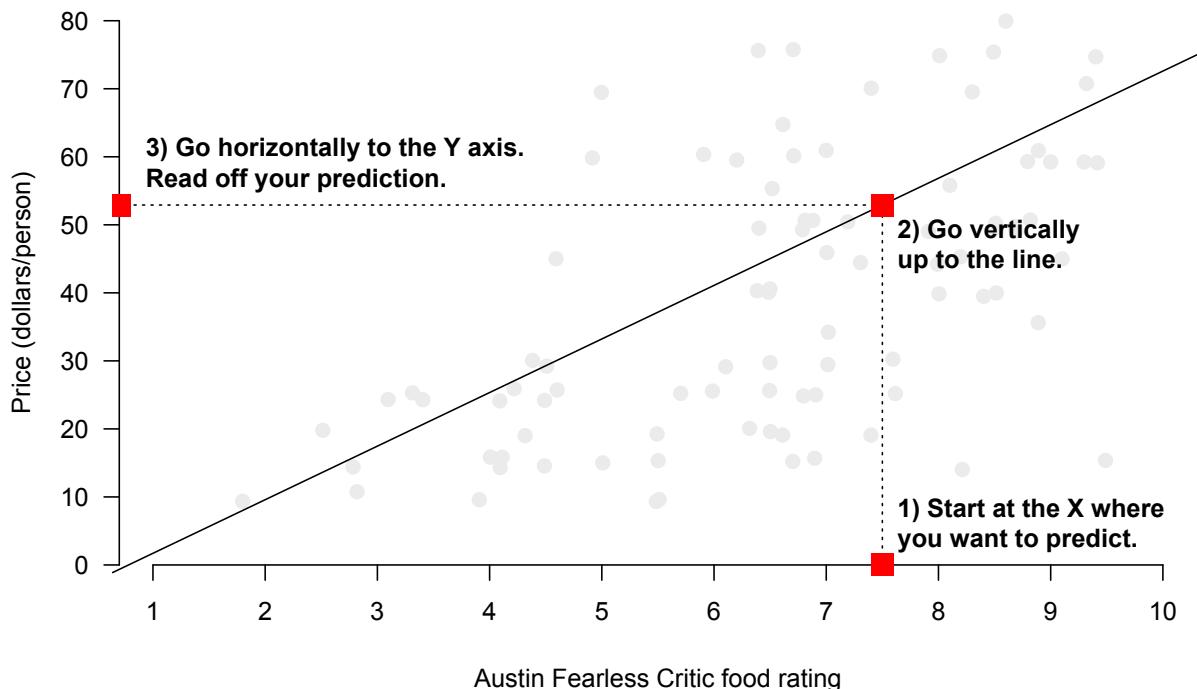
But don’t let the simplicity of the model-fitting step fool you: regression modeling is a wonderfully rich and complex subject. We’ll start by focusing on four kinds of stories one can tell with a regression model. Each is useful for a different purpose.

Story 1: A regression model is a plug-in prediction machine.

One way to interpret a regression model is as is a function $\hat{y} = f(x)$ that maps inputs (x) to expected outputs (\hat{y}). When we plug in the original x values in to the least-squares equation, we get back the so-called *fitted values*, or *model values*, denoted \hat{y}_i :

$$\hat{y}_i = \hat{\beta}_0 + x_i \hat{\beta}_1. \quad (2.4)$$

³ By “very largest,” think: every search that Google has every recorded, every post in the history of Facebook, and so forth. It’s still possible to fit regression models to those data sets, but doing so is far from automatic—and possessing the expertise necessary to do so is a large part of what makes the major Silicon Valley companies so extraordinary (and so valuable).



In this way, the regression model partitions each observed y value into two pieces: $y_i = \hat{y}_i + e_i$, a fitted value plus a residual.

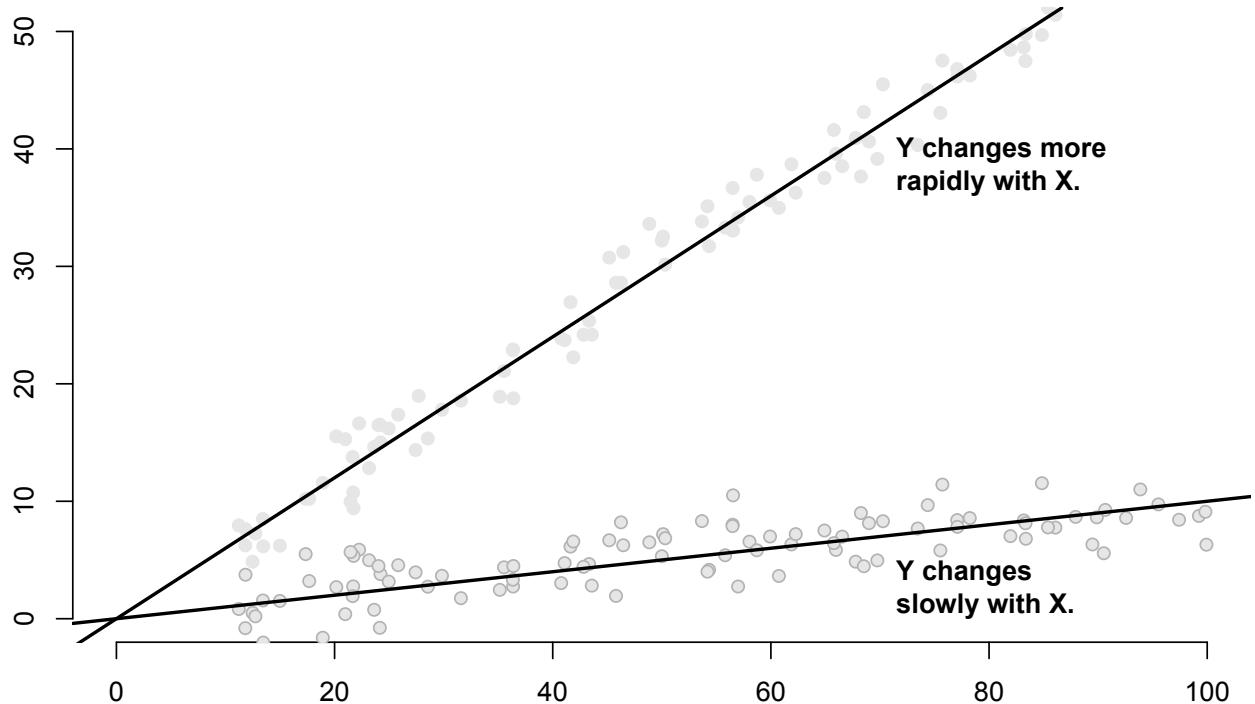
This is especially useful forecasting the response of a new case, where we know the value of the predictor but not the response. Specifically, if we see a new observation x^* and want to predict where the corresponding y^* will be, we can simply plug in x^* and read off our guess for y^* from the line: $\hat{y}^* = \hat{\beta}_0 + x^* \hat{\beta}_1$.

For example, if we know that a new restaurant earned a food rating of 7.5, our best guess for the cost of the meal—knowing nothing else about the restaurant—would be to use the linear predictor: $\hat{y}^* = -6.2 + 7.9 \cdot 7.5$, or \$53.05 per person. (See Figure 2.3). This, incidentally, is where the name *regression* comes from: we expect that future y 's will “regress to the mean” specified by the linear predictor.

Figure 2.3: Using a regression model for plug-in prediction of the price of a meal, assuming a food rating of 7.5.

Story 2: A regression model summarizes the trend in the data.

The linear predictor tells you how Y changes, on average, as a function of X . In particular, the slope β_1 tells you how the re-



sponse tends to change as a function of the predictor:

$$\beta_1 = \frac{\Delta Y}{\Delta X},$$

read “delta-Y over delta-X,” or “change in Y over change in X.”

For the line drawn in Figure 2.1, the slope is $\beta_1 = 7.9$. On average, then, one extra Fearless Critic food rating point (ΔX) is associated with an average increase of \$7.90 (ΔY) in the price of a meal. The slope is always measured in units of Y per units of X —in this case, dollars per rating point. It is often called the *coefficient* of X .

To interpret the intercept, try plugging in $x_i = 0$ into the regression model and notice what you get for the linear predictor: $\beta_0 + \beta_1 \cdot 0 = \beta_0$. This tells you that the intercept β_0 is what we’d expect from the response if the predictor were exactly 0.

Sometimes the intercept is easily interpretable, and sometimes it isn’t. Take the trend line in Figure 2.1, where the intercept is $\beta_0 = -6.2$. This implies that a restaurant with a Fearless Critic food rating of $x = 0$ would charge, on average, $y = -\$6.20$ for the privilege of serving you a meal.

Perhaps the diners at such an appalling restaurant would feel

Figure 2.4: The slope of a regression model summarizes how fast the Y variable changes, as a function of X .

Generally we use a capital letter when referring generically to the predictor or response variable, and a lower-case letter when referring to a specific value taken on by either one.

this is fair value. But a negative price is obvious nonsense. Plugging in $x = 0$ to the price/rating model and trying to interpret the result is a good example of why extrapolation—using a regression model to forecast far outside the bounds of past experience—can give silly results.

Story 3: A regression model takes the X-ness out of Y.

We've seen how a regression model splits up every observation in the sample into two pieces, a fitted value ($\beta_0 + \beta_1 x_i$) and a residual (e_i):

$$\text{Observed } y \text{ value} = (\text{Fitted value}) + (\text{Residual}), \quad (2.5)$$

or equivalently,

$$\text{Residual} = (\text{Observed } y \text{ value}) - (\text{Fitted value}).$$

The residuals from a regression model are sometimes called “errors.” This is especially true in experimental science, where measurements of some Y variable will be taken at different values of the X variable (called design points), and where noisy measurement instruments can introduce random errors into the observations.

But in many cases this interpretation of a residual as an error can be misleading. A regression model can still give a nonzero residual, even if there is no mistake in the measurement of the Y variable. It's often far more illuminating to think of the residual as the part of the Y variable that it is left unpredicted by X .

In Figure 2.1, for example, the positive slope of the line says: yes, people generally pay more for tastier food. The residuals say: not always. There are many other factors affecting the price of a restaurant meal in Austin: location, service, decor, drinks, the likelihood that Matthew McConaughey will be eating overpriced tacos in the next booth, and so forth. Our simple model of price versus food rating collapses all of these other factors into the residuals.

A good way of summarizing this is that the regression model “takes the X -ness out of Y ,” leaving what remains in the residual e_i :

$$\underbrace{y_i}_{\text{Observed } y \text{ value}} = \underbrace{\beta_0 + \beta_1 x_i}_{\text{Predictable by } x} + \underbrace{e_i}_{\text{Unpredictable by } x}.$$

This is easily seen in our example by plotting the residual price (e_i) against food rating (x_i), side by side with the original data, as in Figure 2.5. In the right panel, there is no evident correlation

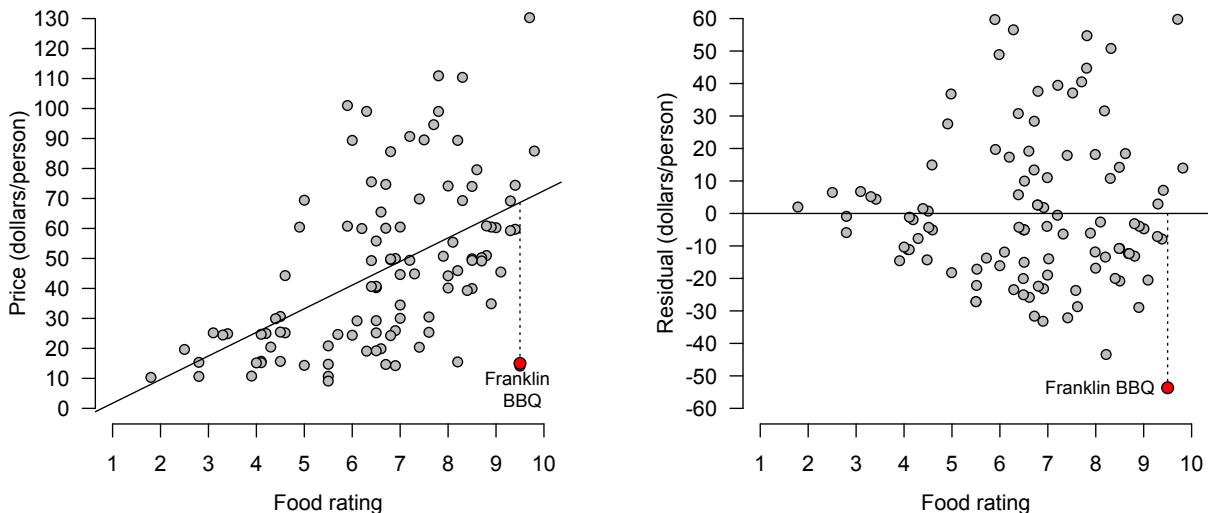


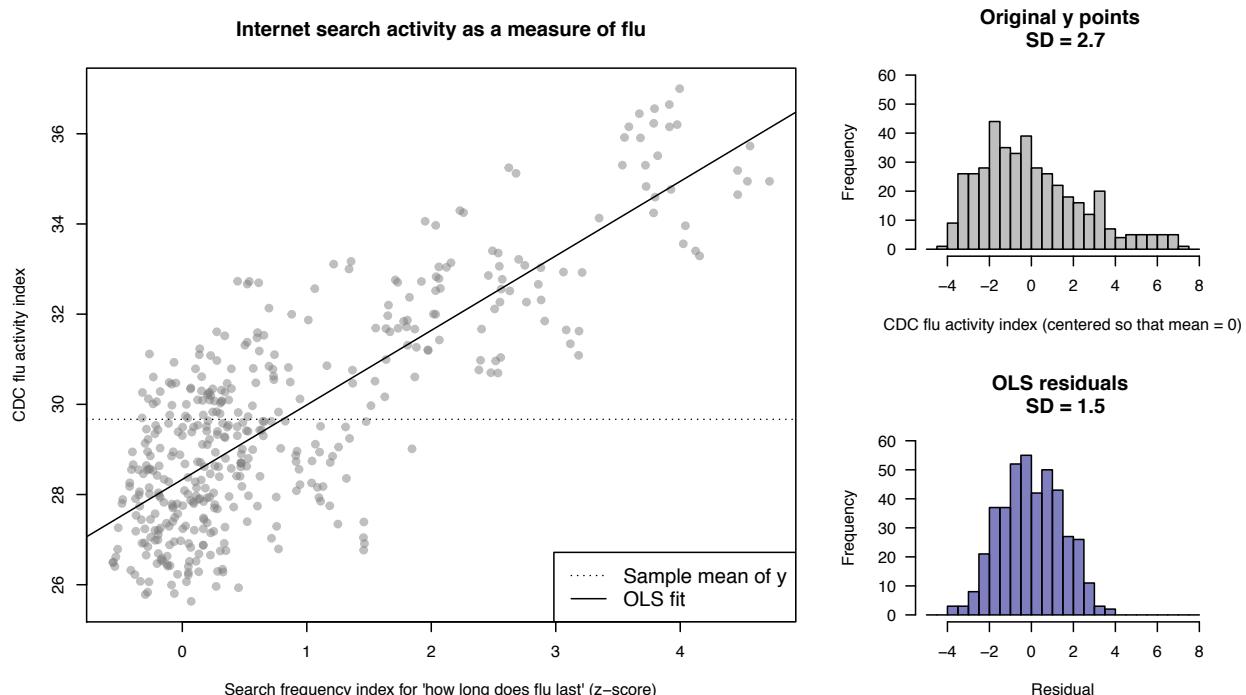
Figure 2.5: Left: the original data on price versus food rating. Right: the residuals from the least squares fit on the left. The residual for Franklin BBQ is the length of the dotted vertical line: $e_i = -\$53.85$.

between food rating and the residuals. This should always be true: a good regression model should take the X-ness out of Y , so that the residuals look independent of the predictor. If they don't, then the model hasn't done its job. (Always plot your residuals to check this.)

You've just seen your first example of *statistical adjustment*. Notice the red dot sitting in the lower right of Figure 2.5, with a low price and a high food rating? This isn't the least expensive restaurant near downtown Austin in an absolute sense. But it is the least expensive *after we adjust for food rating*. To do this, we simply subtract off the fitted value from the observed value of y , leaving the residual—which, you'll recall, captures what's over in the response (price) after the predictor (food score) has been taken into account. The restaurant in question has a food rating of 9.5, good for *Fearless Critic*'s third best score in the entire city. For such delicious food, you would expect to pay $\hat{y}^* = -6.2 + 7.9 \cdot 9.5$, or \$68.85 per person. In reality, the price of a meal at this restaurant is a mere \$15, or $e_i = -\$53.85$ less than expected. That's the largest, in absolute value, of all the negative residuals.

This restaurant is Franklin Barbecue, declared "Best Barbecue in America" by *Bon Appétit* magazine, and undoubtedly the most delicious residual in the city:

Go to Austin and queue up at Franklin Barbecue by 10:30 a.m.
When you get to the counter, Aaron Franklin will be waiting,



knife in hand, ready to slice up his brisket. (Order the fatty end.) Grab a table, a few beers, and lots of napkins and dig in. Take a bite, and don't tell me you're not convinced you've reached the BBQ promised land.

But visitors take note: this article ("A Day in the Life of a BBQ Genius," by food critic Andrew Knowlton) is from July of 2011, and its advice is dated. These days, queueing up at 10:30 would have you last in line!

Story 4: A regression model quantifies the information in a predictor.

The idea behind the Flu Prediction Project, run jointly by IBM Watson and the University of Osnabrück in Germany, is simple.⁴ Researchers combine social-media and internet-search data, together with official data provided by government authorities, like the Centers for Disease Control (CDC) in the United States, to yield accurate real-time predictions about the spread of seasonal influenza. This kind of forecasting model allows public-health authorities to allocate resources (like antivirals and flu vaccines)

Figure 2.6: A scatter plot of the CDC's measure of flu activity versus Google search activity for the phrase "how long does flu last" (z score of search frequency). To the right of the scatter plot, we see two dot plots, both on the same scale: (1) the original deviations from the sample mean, $y_i - \bar{y}$; and (2) the residuals from the regression equation, $y_i - \hat{y}_i$.

⁴ <http://www.flu-prediction.com>

using the most up-to-date information possible. After all, the official government data can usually tell you what flu activity was like two weeks ago. Social-media and internet-search data, if used correctly, have the potential to tell what you it's like right now.

To give you a sense of how strong the predictive signal from internet-search data can be, examine Figure 2.6, focusing first on the scatter plot in the left panel. Here each dot corresponds to a day. On the x -axis is a measure of Google search activity for the term "how long does flu last," where higher numbers mean that more people are searching for that term on that day.⁵ On the y axis, we see a measure of actual flu activity on that day, constructed from data provided by the CDC.

The search activity on a given day strongly predicts actual flu transmission, which makes sense: one of the first things that many people do when they fall ill is to commiserate with a search engine about the depth and duration of their suffering. But just how much information about flu does the search activity for this single term—"how long does flu last"—convey?

In principle, there are many ways of measuring this information content. In fact, you've already met one way to do so: by computing the correlation coefficient between the two variables. Our regression model provides another way, because it allows us to compare our predictions of flu activity both with and without the x variable.

- Without knowing the predictor variable, our best guess for the outcome is just the sample mean, \bar{y} , and the prediction error for each case is $y_i - \bar{y}$. You can think of the sample mean as our "baseline" prediction; it is obviously a pretty simple baseline.
- With the predictor variable, our best guess is given by the regression model, $\hat{y}_i = \beta_0 + \beta_1 x_i$, and the prediction error for each case is the residual, $y_i - \hat{y}_i$.

In each case, we would expect these errors to be distributed around zero. The question is: how much smaller do the errors of the regression model tend to be, compared with the errors we make by predicting the outcome using the sample mean alone? If our predictions errors get a lot smaller with the x variable than without it, then we'll know that this variable conveys a lot of information about response.

To answer this question, return to Figure 2.6. To the right of

⁵ Specifically, it's a z score: how many standard deviations about the mean was the search frequency on that day for that particular term.

LEAST SQUARES THEN AND NOW: AN HISTORICAL ASIDE

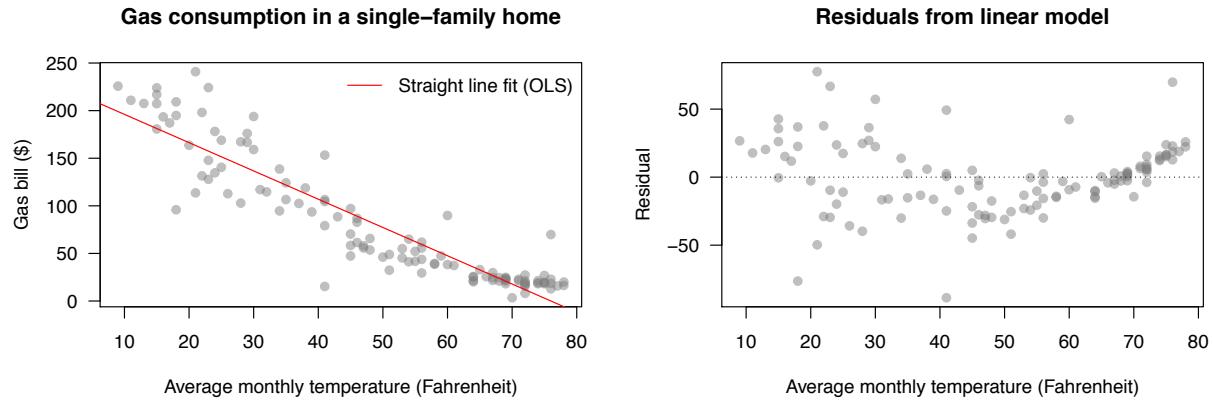
The Ordnance Survey is the governmental body in the United Kingdom charged with mapping and surveying the British Isles. “Ordnance” is a curious name for a map-making body, but it has its roots in the military campaigns of the 1700’s. The name just stuck, despite the fact that these days, most of the folks that use Ordnance Survey maps are probably hikers.



In the days before satellites and computers, map-making was a grueling job, both on the soles of your feet and on the pads of your fingers. Cartographers basically walked and took notes, and walked and took notes, ad infinitum. In the 1819 survey, for example, the lead cartographer, Major Thomas Colby, endured a 22-day stretch where he walked 586 miles—that’s 28 miles per day, all in the name of precision cartography. Of course, that was just the walking. Then the surveyors would have to go back home and crunch the numbers that allowed them to calculate a consistent set of elevations, so that they could correctly specify the contours on their maps.

They did the number-crunching, moreover, by hand. This is a task that would make most of us weep at the drudgery. In the 1858 survey, for example, the main effort involved reducing an enormous mass of elevation data to a system of 1554 linear equations involving 920 unknown variables, which the Ordnance Survey mathematicians solved using the principle of least squares. To crunch their numbers, they hired two teams of dozens of human computers each, and had them work in duplicate to check each other’s mistakes. It took them two and a half years to reach a solution.

A cheap laptop computer bought today takes a few seconds to solve the same problem.



the scatter plot you see two histograms: (1) the original deviations $y_i - \bar{y}$, and (2) the residuals from the regression model. You'll notice that some of the original variation has been absorbed by the regression model: the residuals are less variable (standard deviation 1.5) than the original y points (standard deviation 2.7).

This is how a regression model measures the information content of a predictor: information means reduction in prediction error for the response. The bigger this reduction in prediction uncertainty, the more informative the predictor.

Figure 2.7: Left: a scatterplot of monthly gas consumption (measured in dollars) versus average monthly temperature at a single-family home in Minnesota, together with a linear regression model fit by ordinary least squares. Right: a plot of the residuals from the linear model versus temperature, showing the deficits of the straight line fit. Data source: Daniel T. Kaplan, *Statistical Modeling: A Fresh Approach*, 2009.

Beyond straight lines

UP TO this point, we've talked about fitting straight lines using the principle of least squares. For many data sets, however, a linear regression model doesn't provide an adequate description of what's going on. Consider, for example, the data on monthly gas consumption for a single-family home in Minnesota shown in the left panel Figure 2.7. As the temperature rises, the residents of the house use less gas for heating. But this trend is not well described using a straight line fit by least squares, in this case

$$\text{Gas Bill} = \$226 - 3 \cdot \text{Temperature} + \text{Residual}.$$

For example, consumption levels off when the temperature rises above 65 degrees F, but the straight line keeps going down.

The inadequacy of the linear model is revealed by the residual plot in the right panel. Here, the residuals e_i from the linear fit in

the left panel are plotted versus temperature. Remember, these residuals *should* be unrelated with the predictor if our regression model has done its job right. But here, this is clearly false:

- At very cold temperatures (10-20 degrees), the residuals are almost all positive, suggesting that the regression model made predictions that were systematically too low.
- At cool temperatures (40-60 degrees), the residuals are almost all negative, suggesting that the regression model made predictions that were systematically too high.
- At nice temperatures (65-80 degrees), the residuals are almost all positive, suggesting that the regression model made predictions that were systematically too low yet again.

Thus there is still information left in the temperature variable that can be exploited to do a better job at predicting the gas bill.

In such cases, we need to consider nonlinear regression models. In this section, we'll look at two restricted—but still very useful—families of nonlinear models that can still be fit easily using least squares:

- (1) polynomial models (like quadratic or cubic equations); and
- (2) models involving a simple mathematical transformation of the predictor, the response, or both.

Polynomial regression models

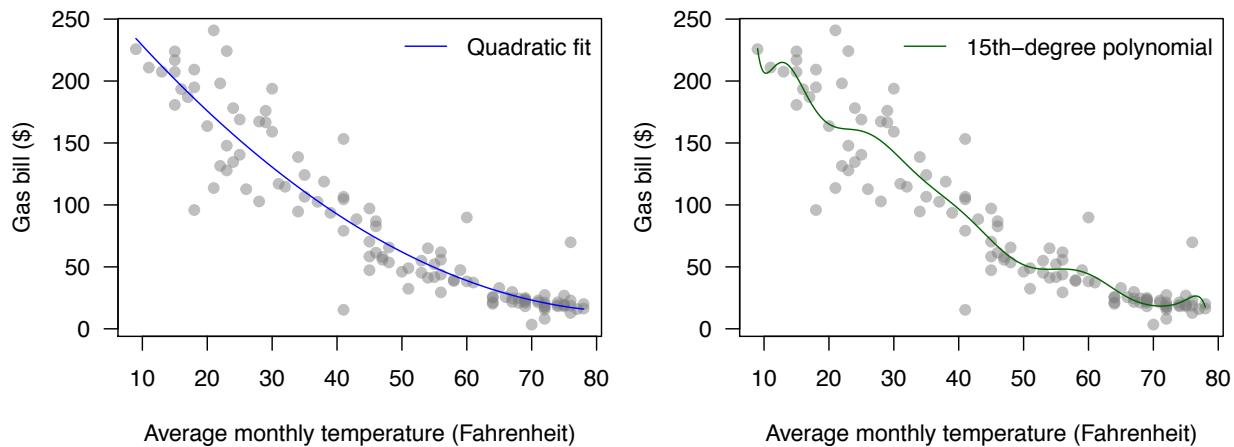
A polynomial is a mathematical function defined by sum of multiple terms, each containing a different power of the same variable (here, as elsewhere, denoted x). A linear function is a special case of a polynomial that only has the first power of x : $y = \beta_0 + \beta_1 x$.

But we can fit other polynomials by least squares, too. For example, the left panel of Figure 2.8 shows the least-squares fit of a quadratic equation (another name for a second-degree polynomial) to the gas-consumption data set:

$$\text{Gas Bill} = \$289 - 6.4 \cdot \text{Temp} + 0.03 \cdot \text{Temp}^2 + \text{Residual}.$$

The quadratic model fits noticeably better than the straight line. In particular, it captures the leveling-off in gas consumption at high temperatures that was missed by the linear model.

Beyond these two families, there is a much wider class of nonlinear models that can still be fit by least squares, but not easily. (That is, Legendre's simple computational method won't work, and we need something fancier.) These are often called nonparametric regression models, and they are the subject of a more advanced course.



Over-fitting. If the quadratic model (a second-order polynomial) fits better than the straight line (a first-order polynomial), why not try a third-, fourth-, or higher-order polynomial to get an even better fit? After all, we can fit polynomial models of any degree by least squares, estimating equations of the form

$$\hat{y} = \beta_0 + \beta_1 x + \beta_2 x^2 + \cdots + \beta_K x^K,$$

for an arbitrary choice of K (the degree of the polynomial).

To want to fit the data as well as possible is an understandable impulse. But for most data sets, if we venture beyond $K = 2$ (quadratic) or $K = 3$ (cubic), we rapidly get into dangerous overfitting territory. *Over-fitting* occurs when a regression model starts to memorize the random noise in the data set, rather than describe the underlying relationship between predictor and response. We see a clear example of overfitting in the right panel of 2.8, which shows the result of using least-squares to estimate a 15th-degree polynomial for gas bill versus temperature. The fitted curve exaggerates minor dips and rises in the data, leading to an absurdly complex function. There's no reason for us to think that gas consumption responds to temperature in the way implied by the green curve on the right of Figure 2.8. For example, why would consumption rise systematically between 10 and 15 degrees, but then drop again between 15 and 20 degrees?

In regression modeling, we want to build models that are only as complex as they must be in order to describe the underlying relationship between predictor and response. But how do we

Figure 2.8: Left: the fit of a quadratic model (2nd-degree polynomial) estimated by least squares. Right: the fit of a 15th-degree polynomial. The model on the left provides an intuitively reasonable description of the underlying relationship, while the model on the right is a clear example of over-fitting.

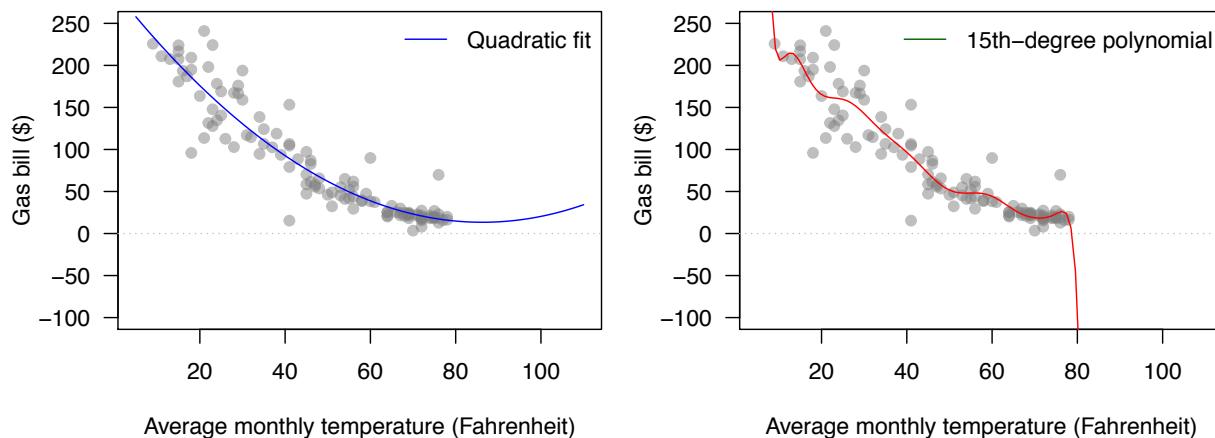


Figure 2.9: Extrapolation in polynomial regression models.

know reliably when we've crossed this line from "fitting well" into over-fitting?

We're still a few chapters away from being able to provide a solid answer to that question. For now, it's fine to let your intuition and your eyes be your guide:

- Does the fitted equation look implausibly wiggly?
- Is there a sound reason, grounded in knowledge of the phenomenon being measured, to believe in the complexity that your model postulates?

With apologies to [Potter Stewart](#): when it comes to overfitting, you'll often know it when you see it. This is one of many reasons why it is always a good idea to plot your data.

Extrapolation. Although the quadratic model fits the data well, its predictive abilities will deteriorate as we move above 80 degrees (i.e. as we use the model to extrapolate further and further beyond the range of past experience). As we can see in the left panel of Figure 2.9, that's because the fitted curve is a parabola: it turns upwards around 85 degrees, counterintuitively suggesting that gas bills would eventually rise with temperature.

This behavior is magnified dramatically with higher-order polynomials, which can behave in unpredictable ways beyond the endpoints of your data. The right panel of Figure 2.9 shows this clearly: notice that the predictions of the 15th-degree polynomial drop off a cliff almost immediately beyond the range of the available data, at 79 degrees. You'll sometimes hear this phenomenon—

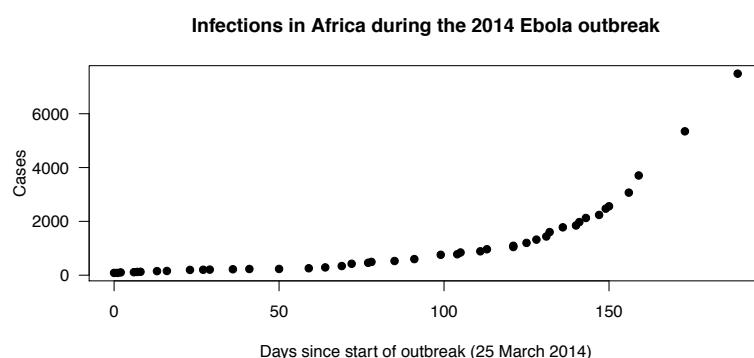


Figure 2.10: Cases of Ebola over time in West Africa, 2014. Compiled from [CDC reports](#) by Francis Smart, as described here.

ridiculous predictions beyond the endpoints of the data—referred to as an “endpoint artifact.”

This example offers a cautionary tale: never extrapolate with a polynomial regression model, unless you really know what you’re doing.

Exponential growth and decay

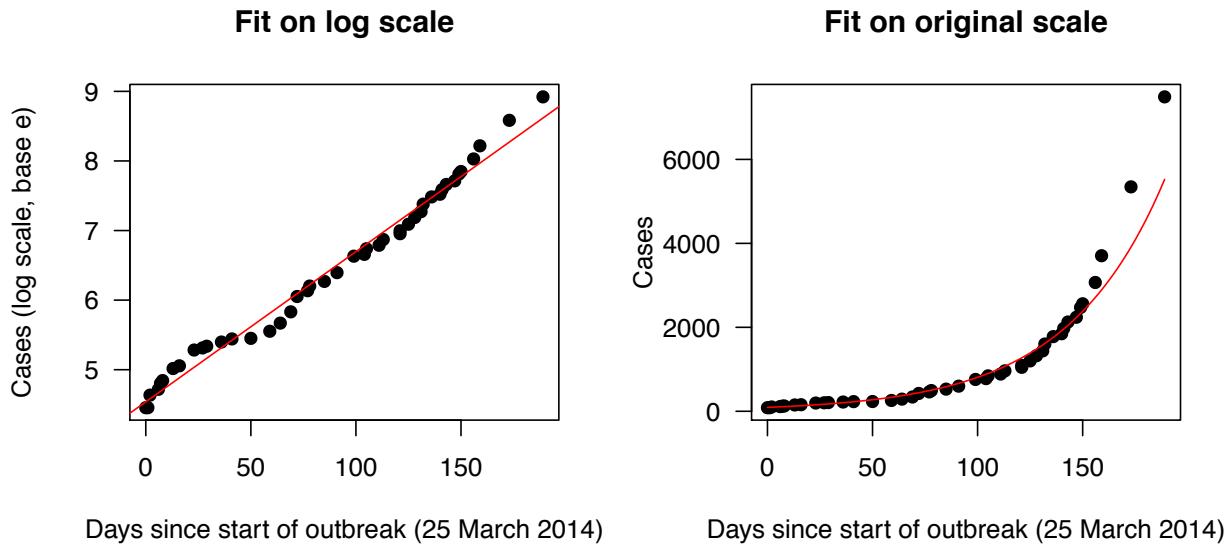
Beginning in March 2014, West Africa experienced the largest outbreak of the Ebola virus in history. Guinea, Liberia, Niger, Sierra Leone, and Senegal were all hit hard by the epidemic. Figure 2.10 shows the number of laboratory-confirmed cases of Ebola in these five countries over time, beginning on March 25.

If we wanted to fit a model to describe how the number of Ebola infections grew over time, we might be tempted to fit a polynomial function (since a linear model clearly won’t work well here). However, basic biology tells us that the transmission rate of a disease through a population is reasonably well described by an exponential growth model: 1 infection leads to 2, which lead to 4, which lead to 8, to 16, and so on. The equation for an exponential-growth model is

$$y = \alpha \cdot e^{\beta t}, \quad (2.6)$$

where y is the expected number of cases and t is the number of time intervals (e.g. weeks or days) since the start of the outbreak.

It turns out that we can use least squares to fit an exponential growth model of this form, using a new trick: *take the logarithm of the response variable* and fit a linear model to this new transformed variable. We can see why this works if we take the logarithm of



y in the equation for exponential growth (labeled 2.6, above). To preserve equality, if we take the log of the left-hand side, we also have to take the log of the right-hand side:

$$\begin{aligned}\log y &= \log(\alpha \cdot e^{\beta_1 t}) \\ &= \log \alpha + \beta_1 t.\end{aligned}$$

The second equation says that the log of y is a linear function of the time variable, t , with intercept $\beta_0 = \log \alpha$ and slope β_1 .

Thus to fit the exponential growth model for any response variable y , we need to follow two steps:

- (1) Define a new variable $z = \log y$ by taking the logarithm of the original response variable.
- (2) Fit a linear model for the transformed variable z versus the original predictor, using ordinary least squares.

Figure 2.11 shows the result of following these two steps for the Ebola data. The left panel shows the straight-line fit on the log scale:

$$\log \text{Cases} = 4.54 + 0.021 \cdot \text{Days}.$$

The right panel shows the corresponding exponential-growth curve on the original scale:

$$\text{Cases} = 93.5 \cdot e^{0.021 \cdot \text{Days}}.$$

Figure 2.11: An exponential-growth model fit to the Ebola data by ordinary least squares, where the y variable is shown on the log scale (left) and on the original scale (right).

The leading constant is calculated from the intercept on the log scale: $93.5 \approx e^{4.54}$. From Figure 2.11, we can see that the exponential-growth model fits adequately, although imperfectly: the rate of growth seems to be accelerating at the right of the picture, and the upward trajectory is visibly nonlinear on the log scale. (Remember: all models are wrong, but some models are useful.)

An exponential model with a negative slope β_1 on the log scale is called an exponential decay model. Exponential decay is a good model for, among other things, the decay of a radioactive isotope.

Interpreting the coefficient in an exponential model. To interpret the coefficient in an exponential growth model, we will use it to calculate the doubling time—that is, how many time steps it takes for the response variable (here, Ebola cases) to double.

In terms of our estimated model, the number of cases doubles between days t_1 and t_2 whenever

$$\frac{\alpha e^{\beta_1 t_2}}{\alpha e^{\beta_1 t_1}} = 2,$$

so that the number of cases on day t_2 (in the numerator) is precisely twice the number of cases on day t_1 , in the denominator. If we simplify this equation using the basic [rules of algebra for exponentials](#), we find that the number of days that have elapsed between t_1 and t_2 is

$$t_2 - t_1 = \frac{\log 2}{\beta_1}.$$

This is our doubling time. For Ebola in West Africa, the number of cases doubled roughly every

$$\frac{\log 2}{0.021} \approx 32$$

days during the spring and early summer of 2014.

In an exponential decay model (where $\beta_1 < 0$), a similar calculation would tell you the [half life](#), not the doubling time.⁶

Double log transformations

In some cases, it may be best to take the log of both the predictor and the response, and to work on this doubly transformed scale. For example, in the upper left panel of Figure 2.12, we see a scatter plot of brain weight (in grams) versus body weight (in kilos) for 62 different mammalian species, ranging from the lesser short-tailed shrew (weight: 10 grams) to the African elephant (weight:

⁶ Instead, solve the equation

$$\frac{\alpha e^{\beta_1 t_2}}{\alpha e^{\beta_1 t_1}} = 1/2$$

for the difference $t_2 - t_1$.

6000+ kilos). You can see that most species are scrunched up in a small box at the lower left of the plot. This happens because the observations span many orders of magnitude, and most are small in absolute terms.

But if we take the log of both body weight and brain weight, as in the top-right panel of Figure 2.12, the picture changes considerably. Notice that, in each of the top two panels, the red box encloses the same set of points. On the right, however, the double log transformation has stretched the box out in both dimensions, allowing us to see the large number of data points that, on the left, were all trying to occupy the same space. Meanwhile, the two points outside the box (the African and Asian elephants) have been forced to cede some real estate to the rest of Mammalia.

This emphasizes the taking the log is an “unsquishing” operator. To see this explicitly, look at the histograms in the second and third row of panels in Figure 2.12. Whenever the histogram of a variable looks highly skewed right, as on the left, a log transformation is worth considering. It will yield a much more nicely spread-out distribution of points, as on the right.

Power laws. It turns out that when we take the log of both variables, we are actually fitting a *power law* for the relationship between y and x . The equation of a power law is

$$y = \alpha \cdot x^{\beta_1}$$

for some choices of α and β . This is a very common model for data sets that span many orders of magnitude (like the body/brain weight data). To see the connection with the double log transformation, simply take the logarithm of both sides of the power law:

$$\begin{aligned} \log y &= \log (\alpha \cdot x^{\beta_1}) \\ &= \log \alpha + \log x^{\beta_1} \\ &= \log \alpha + \beta_1 \log x. \end{aligned}$$

Therefore, if y and x follow a power law, then $\log y$ and $\log x$ follow a linear relationship with intercept $\log \alpha$ and slope β_1 . This implies that we can fit the parameters of the power law by applying the double log transformation and using ordinary least squares. For our mammalian brain weight data, applying this recipe yields the fitted equation

$$\log \text{brain} = 2.13 + 0.75 \cdot \log \text{body},$$

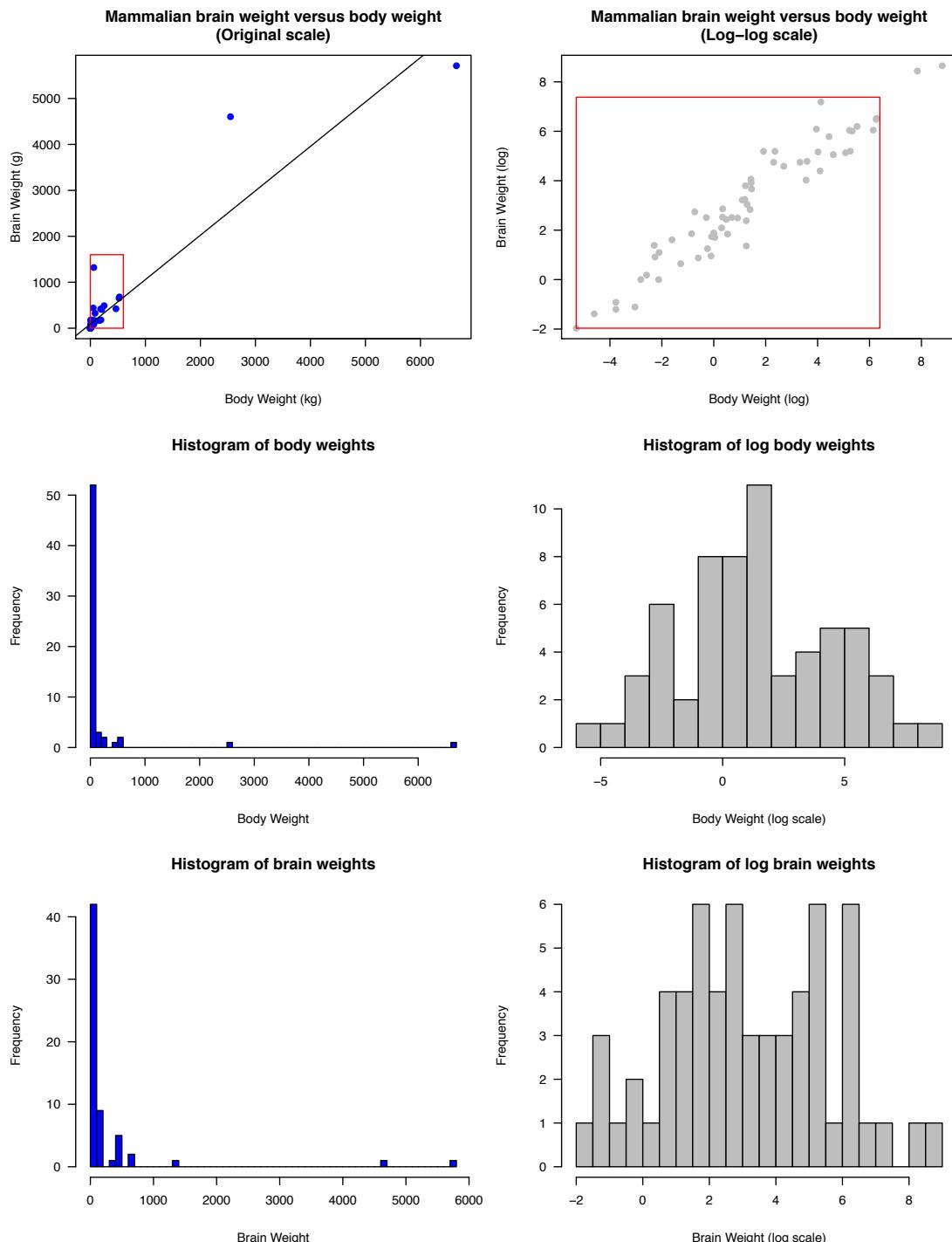
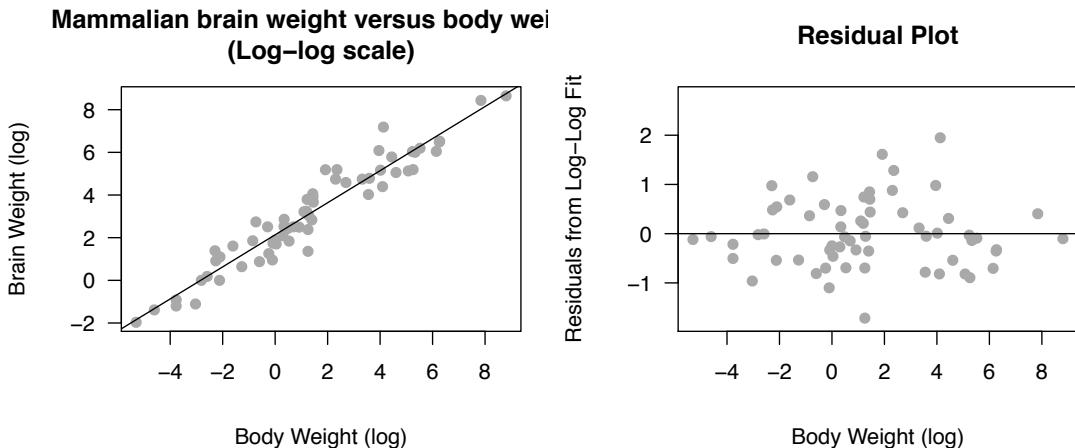


Figure 2.12: Brain weight versus body weight for 62 mammalian species, both on the original scale and the log scale. Notice how the log transformation “unsquishes” the points.



or expressed as a power law on the original scale,

$$\text{brain} = 8.4 \cdot \text{body}^{0.75}.$$

The residuals in a power-law model. As we've just seen, we can fit power laws using ordinary least squares after a log transformation of both the predictor and response. In introducing this idea, we ignored the residuals and focused only on the part of the model that describes the systematic relationship between y and x . If we keep track of these residuals a bit more carefully, we see that the model we're fitting for the i th response variable is this:

$$\log y_i = \log \alpha + \beta_1 \log x_i + e_i, \quad (2.7)$$

where e_i is the amount by which the fitted line misses $\log y_i$. We suppressed these residuals before the lighten the algebra, but now we'll pay them a bit more attention.

Equation 2.7 says that the residuals affect the model in an additive way on the log scale. But if we exponentiate both sides, we find that they affect the model in a multiplicative way on the original scale:

$$\begin{aligned} \exp(\log y_i) &= \exp(\log \alpha) \cdot \exp(\beta_1 \log x) \exp(e_i) \\ y_i &= \alpha x^{\beta_1} \exp(e_i). \end{aligned}$$

Therefore, in a power low, the exponentiated residuals describe the percentage error made by the model on the original scale. Let's work through the calculations for two examples:

Figure 2.13: A straight-line fit to the mammalian brain weight data after a double log transformation.

- If $e_i = 0.2$ on the log–log scale, then the actual response is $\exp(0.2) \approx 1.22$ times the value predicted by the model. That is, our model underestimates this particular y_i by 22%.
- If $e_i = -0.1$ on the log–log scale, then the actual response is $\exp(-0.1) \approx 0.9$ times the value predicted by the model. That is, our model overestimates this particular y_i by 10%.

The key thing to realize here is that the *absolute* magnitude of the error will therefore depend on whether the y variable itself is large or small. This kind of multiplicative error structure makes perfect sense for our body–brain weight data: a 10% error for a lesser short-tailed shrew will have us off by a gram or two, while a 10% error for an elephant will have us off by 60 kilos or more. Bigger critters mean bigger errors—but only in an absolute sense, and not if we measure error relative to body weight.

Interpreting the slope under a double log transformation. To correctly interpret the slope β_1 under a double log transformation, we need a little bit of calculus. The power law that we want to fit is of the form $y = \alpha x^{\beta_1}$. If we take the derivative of this expression, we get

$$\frac{dy}{dx} = \beta_1 \alpha x^{\beta_1 - 1}.$$

We can rewrite this as

$$\begin{aligned}\frac{dy}{dx} &= \frac{\beta_1 \alpha x^{\beta_1}}{x} \\ &= \beta_1 \frac{y}{x}.\end{aligned}$$

If we solve this expression for β_1 , we get

$$\beta_1 = \frac{dy/y}{dx/x}. \quad (2.8)$$

Since the dy in the derivative means “change in y ”, the numerator is the rate at which the y variable changes, as a fraction of its value. Similarly, since dx means “change in x ”, the denominator is the rate at which the x variable changes, as a fraction of its value.

Putting this all together, we find that β_1 measures the ratio of percentage change in y to percentage change in x . In our the mammalian brain-weight data, the least-squares estimate of the slope on a log-log scale was $\hat{\beta}_1 = 0.75$. This means that, among mammals, a 100% change (i.e. a doubling) in body weight is associated with a 75% expected change in brain weight. The bigger you are, it

would seem, the smaller your brain gets—at least relatively speaking.

The coefficient β_1 in a power law is often called an *elasticity* parameter, especially in economics, where it is used to quantify the responsiveness of consumer demand to changes in the price of a good or service. The underlying model for consumer behavior that's often postulated is that

$$Q = \alpha P^{\beta_1},$$

where Q is the quantity demanded by consumers, P is the price, and $\beta_1 < 0$. Economists would call β_1 the **price elasticity of demand**,⁷ which may be a familiar concept from a microeconomics course.

⁷ They actually define elasticity as the ratio in Equation 2.8, but as we've seen, this is mathematically equivalent to the regression coefficient you get when you fit the x - y relationship using a power law.

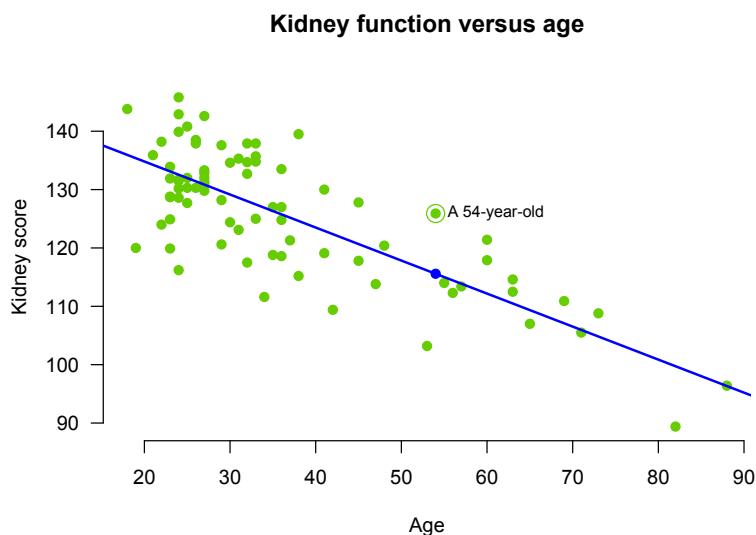
3

Predictable and unpredictable variation

Quantifying uncertainty in a prediction

THERE are many things we can look forward to as we age—for example, richer relationships, improved confidence, better self-knowledge, and the right to go to bed early without being judged.

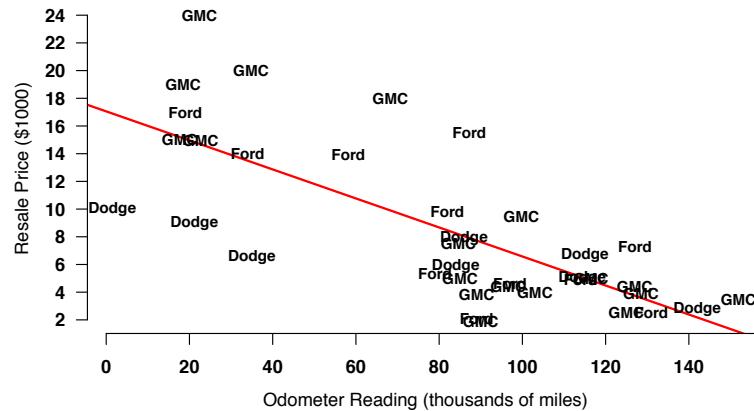
Unfortunately, improved kidney function isn't one of them. The following plot shows a sample of 78 patients from an ordinary doctor's office. The x -axis shows the patient's age, while the y -axis shows the patient's creatinine-clearance rate in mL/min, which is a common measure of kidney function (higher is better):¹



¹ According to the National Institutes of Health, “The creatinine clearance test helps provide information about how well the kidneys are working. The test compares the creatinine level in urine with the creatinine level in blood. . . . Creatinine is removed, or cleared, from the body entirely by the kidneys. If kidney function is abnormal, creatinine level increases in the blood because less creatinine is released through the urine.”

Suppose you're the doctor running this clinic, and a 54-year old man walks through the door. He tests at 126 mL/min, which is 10 points above the prediction of the regression line (blue dot on the line). Is the man's score too high, or is it within the range of normal variation from the line?

This question is fundamentally about *prediction uncertainty*. Anytime we use a statistical model to make a prediction, some version of this question comes up. For example, among pickup trucks for sale on Craigslist, those with higher odometer readings tend to have lower asking prices:



Now imagine you have your eye on a pickup truck with 80,000 miles on it. The least squares fit says such that the expected price for such a truck is about \$8,700. If the owner is asking \$11,000, is this reasonable, or drastically out of line with the market?

Here's another example. Mammals more keenly in danger of predation tend to dream fewer hours.

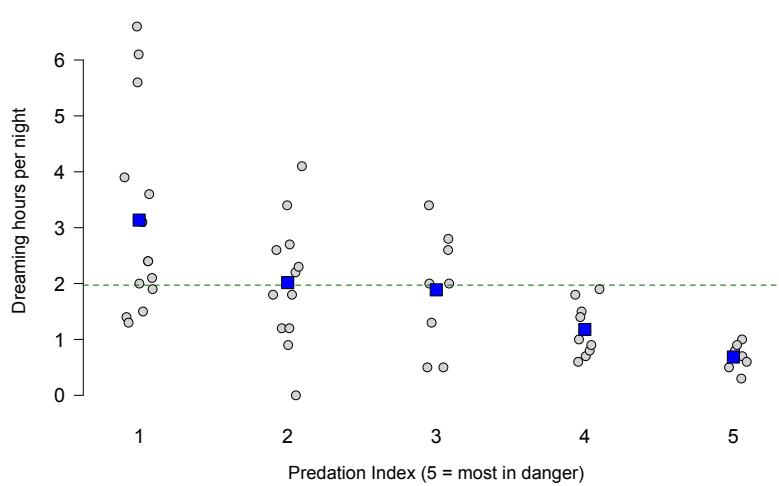


Figure 3.1: Dreaming hours per night versus danger of predation for 50 mammalian species. In this and in Figure 3.2, the blue squares show the group-wise means, while the dotted green line shows the grand mean for the entire data set.

But there is still residual variation that practically begs for a Zen

proverb. Why does the water rat dream at length? Why does the wolverine not?

Finally, the people of Raleigh, NC tend to use less electricity in the milder months of autumn and spring than in the height of winter or summer—but not uniformly. Many spring days see more power usage than average; many summer days see less. What is the normal range of electricity consumption for a day in August, the hottest month of the year?

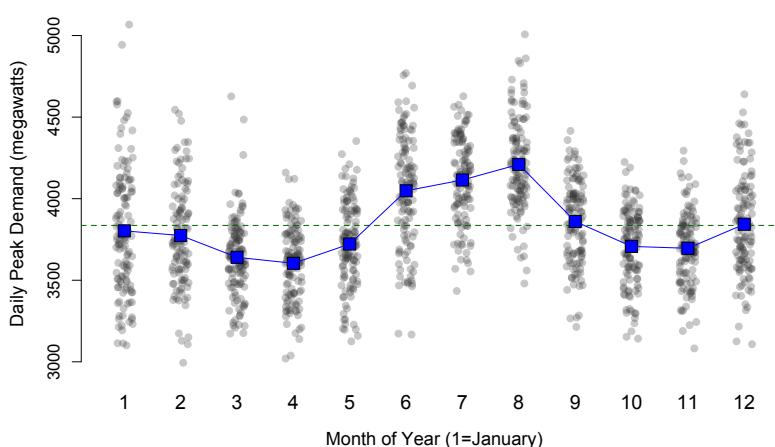


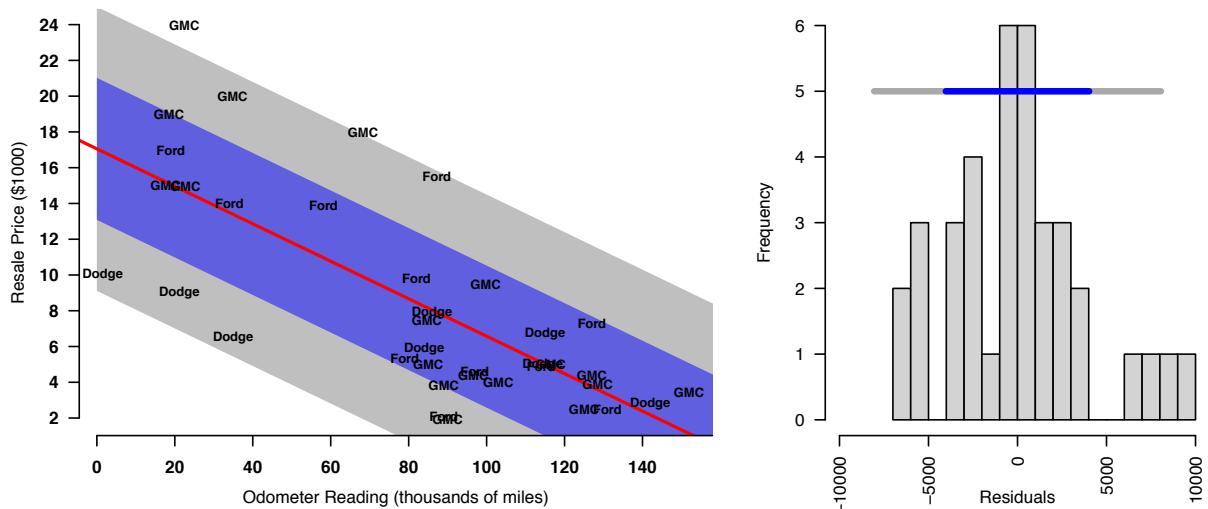
Figure 3.2: Daily peak demand for electricity versus month of the year in Raleigh, NC from 2006–2009.

In all of these cases, one must remember that the fitted values from a statistical model are generalizations about a typical case, given the information in the predictor. But no generalization holds for all cases. This is why we explicitly write models as

$$\text{Observed } y \text{ value} = \text{Fitted value} + \text{Residual}.$$

It is common to view a statistical model as nothing more than a recipe for calculating the fitted values, and to think that the residuals are just the errors made by this model. But we'll have a richer picture if instead we view the residuals as *part of* the model. If you've ignored the variation in the residuals, then you really haven't specified a complete forecast.

An important distinction here is that of a *point estimate*, or single best guess, versus an *interval estimate*, or a range of likely values. Fitted values are point estimates. Point estimates are useful, but interval estimates are much better. After all, variation from the average, far from being an “error,” is a normal part of life.



Prediction intervals

THE key question we must answer to quantify our prediction uncertainty is: “How much does a typical case vary from the prediction of the regression model?” We have a lot of ways to answer this question (box plots, histograms, dot plots, and so forth). The most common way is to calculate the *residual standard deviation*:

$$s_e = \sqrt{\frac{1}{n-p} \sum_{i=1}^n (y_i - \hat{y}_i)^2},$$

where p is the number of free parameters in the model (e.g. two for a straight-line fit: the slope and the intercept). This quantity describes how much a typical case deviates from the fitted line, just like the ordinary standard deviation tells us how much a typical case deviates from sample mean \bar{y} (page 15).

To see how the residual standard deviation s_e can be used to quantify prediction uncertainty, let’s take another look at the data set of pickup trucks advertised on Craigslist. In Figure 3.3, the red line is the least-squares fit: $Y = 17054 - 0.105 x$. The residual standard deviation is \$3,971, compared to the original standard deviation of \$5,584. That is, a typical truck deviates from the sample mean \bar{y} by about \$5,584, and from the least-squares line by about \$3,971. Knowledge of the truck’s mileage has improved our

Figure 3.3: Prediction intervals for pickup-truck prices on Craigslist. Shaded grey area = 1 residual standard deviation to either side of the fitted line (red); shaded blue = 2 standard deviations. The right panel shows a histogram of the residuals, along with the width of the $1s_e$ (blue) and $2s_e$ (grey) envelopes.

The residual standard deviation is also called the *residual standard error*. The formula for the residual standard deviation is *almost* identical to the formula for the sample standard deviation of the residuals. The minor difference is the divisor: $n - p$ instead of $n - 1$. The reason is that the sample standard deviation centers y_i by the sample mean, which involves computing 1 extra number (\bar{y}) from the data. The residual standard deviation centers y_i by the OLS fitted values, which involves computing p extra numbers ($\hat{\beta}_0$ and $\hat{\beta}_1$ in the case of a straight-line fit).

predictive accuracy by about $\$5584 - \$3971 = \$1613$, but there is still a lot of uncertainty. The two shaded strips in Figure 3.3 depict this uncertainty visually. The blue extends to 1 residual standard deviation (line $\pm \$3,971$) on either side of the line, while the grey strip extends to 2 residual standard deviations (line $\pm \$7,942$).

The key idea of a prediction interval is that these grey strips can be used to provide an interval estimate for forecasting the price of a future truck—that is, one not in our original data set. For our hypothetical pickup truck with 80,000 miles, the point estimate for the expected price (from the least-squares line) is \$8,672. But if we go out one residual standard deviation, the *interval estimate* is $\$8,672 \pm \$3,971$, or (4701, 12643). You can see where the wide of these one- and two-standard-deviation envelopes comes from, in the histogram in the right panel of Figure 3.3.

How accurate is the interval estimate? A simple way to quantify this is just to count the number of cases that fall within the one-standard deviation band to either side of the line, as a fraction of the total number of cases. Since the medium grey strip,

$$y \in 17054 - 0.105 \cdot x \pm 3971,$$

captures 27 out of 37 total cases, it therefore constitutes a family of *prediction intervals* at a *coverage level* of 73% (27/37). We call it a family of intervals, because there is actually one such prediction interval for every possible value of x . At $x = 80000$, the interval is (4701, 12643); at $x = 40,000$, the interval is (8892, 16834).

To summarize, forming a prediction interval requires two steps: constructing the interval, and quantifying its accuracy. In a simple linear regression model, the interval itself takes the form

$$y \in \hat{\beta}_0 + \hat{\beta}_1 x \pm k \cdot s_e,$$

or more concisely, $y \in \hat{y} \pm k \cdot s_e$. Here s_e is the residual standard deviation, and k is a chosen multiple that characterizes the width of the intervals. There is a clear trade-off here: larger choices of k mean wider intervals, which mean more uncertainty, but greater coverage. Typical values for k are 1 or 2. To quantify the accuracy of the interval, we look at its coverage: that is, what fraction of examples in our original data set are contained within their corresponding interval.

Most good statistical software makes it easy to calculate prediction intervals. In R, for example, the `predict` function allows you to specify a given coverage level (e.g. 95%) and will output the

Here the notation $y \in c \pm h$ means that y (the response) is in the interval centered at c that extends h units to either side. Thus h is the half-width of the interval. The sign \in is concise mathematical notation for “is in.”

lower and upper bounds of a prediction interval at that specified coverage.

Standardized residuals. Return to the question we posed on the beginning of the chapter. You're the doctor at a clinic, and a 54-year old man has score of 126 mL/min for his creatinine clearance test. Is the man's score too high, or is it within the range of normal variation from the line?

We can answer this question by calculating a *standardized residual*, which is just a z -score based on dividing the residual by the residual standard deviation:

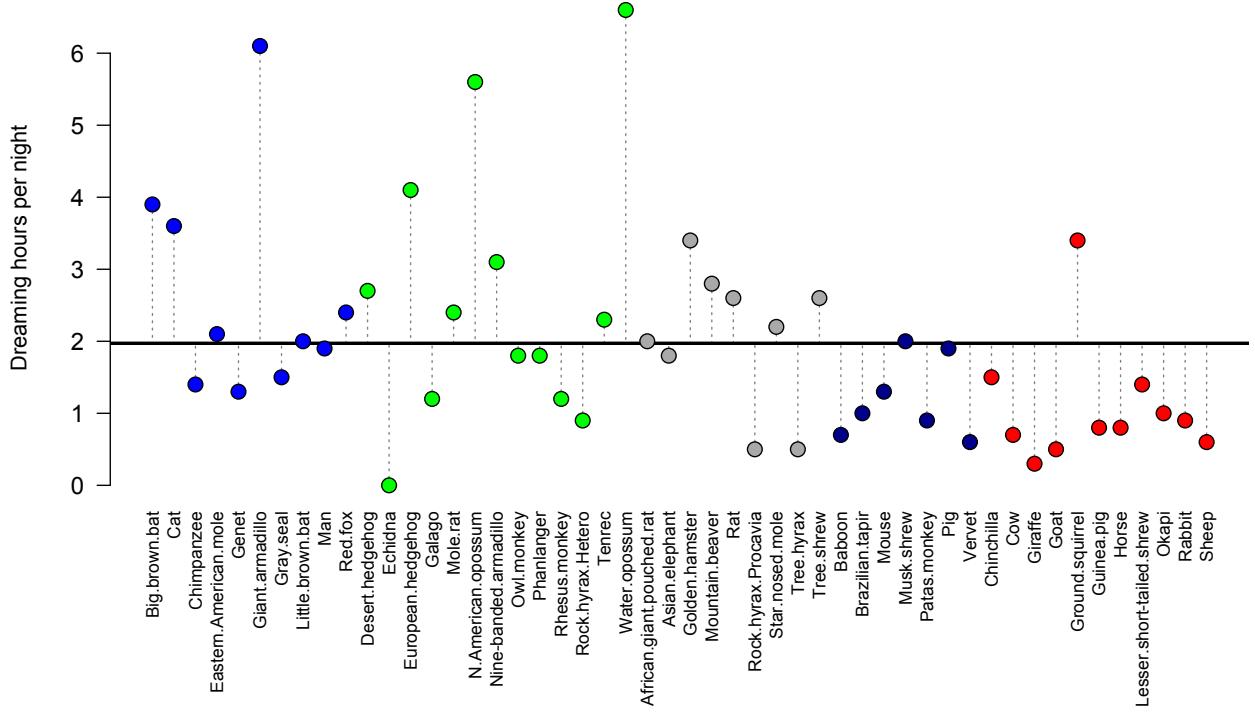
$$z = \frac{y_i - \hat{y}_i}{s_e} = \frac{e_i}{s_e}.$$

In this example, $y_i = 126$, $\hat{y}_i = 116$, and the residual standard deviation is $s_e = 7.2$. Therefore the man's z -score is $(126 - 116)/7.2$, or about 1.4 standard deviations above normal. This is on the higher side, but within the range of typical values seen in the clinic.

A caveat. The technique we've learned for forming prediction intervals is pretty useful, but it's not perfect. That's because it ignores uncertainty about the parameters of the model itself, and only accounts for uncertainty about residuals, assuming that the fitted model is true. (That is, we're ignoring the fact that we might have been a bit off in our estimates of the slope and intercept, due to sampling variability.) As a result, these prediction intervals actually underestimate the total amount of uncertainty that we'd like to incorporate into our interval estimate. We'll soon learn how to quantify these additional forms of uncertainty. But imperfections aside, even these slightly naïve prediction intervals that don't account for parameter uncertainty are much better than a point estimate.

Partitioning sums of squares

WHEN we introduced the concept of the sample standard deviation, we asked the question: what's so great about sums of squares for measuring variation? The answer is: because linear statistical models *partition the total sum of squares* into predictable and unpredictable components. This isn't true of any other simple measure of variation. Sums of squares are special.



Let's return to those grand and group means for the mammalian sleeping-pattern data. We will use sums of squares to measure three quantities: the total variation in dreaming hours; the variation that can be predicted using the predation index; and unpredictable variation that remains "in the wild."

In Figure 3.4, we see the observed y value (dreaming hours per night) plotted for every species in the data set. The horizontal black line shows the grand mean, $\bar{y} = 1.97$ hours. The dotted vertical lines show the deviations between the grand mean and the actual y values, $y_i - \bar{y}$.

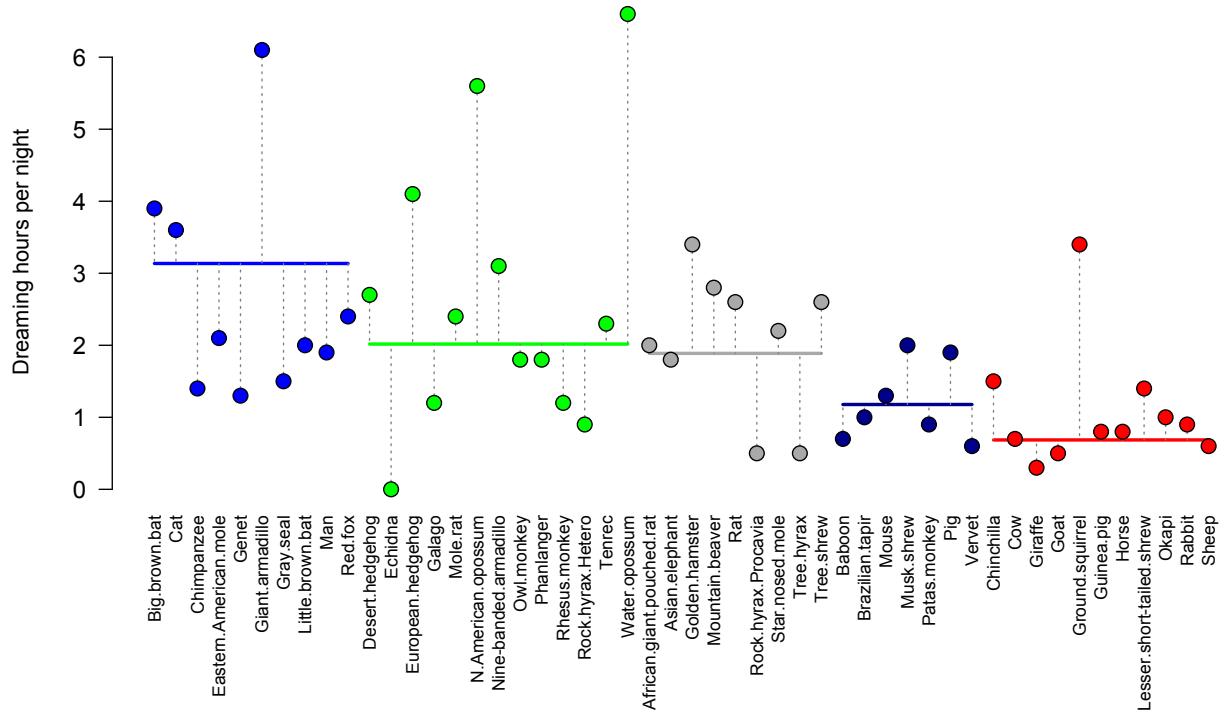
To account for the information in the predictor, we fit the model "dreaming hours \sim predation index," computing a different mean for each group:

$$\underbrace{y_i}_{\text{Observed value}} = \underbrace{\hat{y}_i}_{\text{Group mean}} + \underbrace{e_i}_{\text{Residual}}.$$

There are three quantities to keep track of here:

- The observed values, y_i .

Figure 3.4: Dreaming hours by species, along with the grand mean. For reference, the colors denote the predation index, ordered from left to right in increasing order of danger (1–5). The vertical dotted lines show the deviations from the grand mean: $y_i - \bar{y}$.



- The grand mean, \bar{y} .
- The fitted values, \hat{y}_i , which are just the group means corresponding to each observation. These are shown by the colored horizontal lines in Figure 3.5 and again as diamonds in Figure 3.6. For example, cats and foxes in group 1 (least danger, at the left in dark blue) both have fitted values of 3.14; goats and ground squirrels in group 5 (most danger, at the right in bright red) both have fitted values of 0.68. Notice that the fitted values also have a sample mean of \bar{y} : the average fitted value is the average observation.

There are also three important relationships among y_i , \hat{y}_i , and \bar{y} to keep track of. We said we'd measure variation using sums of squares, so let's plunge ahead.

- The total variation, or the sum of squared deviations from the mean \bar{y} . This measures the variability in the original data:

$$TV = \sum_{i=1}^n (y_i - \bar{y})^2 = 102.1.$$

Figure 3.5: Dreaming hours by species, along with the group means stratified by predation index. The vertical dotted lines show the residuals from the group-wise model “Dreaming hours \sim predation index.”

This equation says that the number 102.1 comes from summing all the squared deviations in the data set—that is, $(3.9 - \bar{y})^2 + (3.6 - \bar{y})^2 + \dots + (0.6 - \bar{y})^2 = 102.1$.

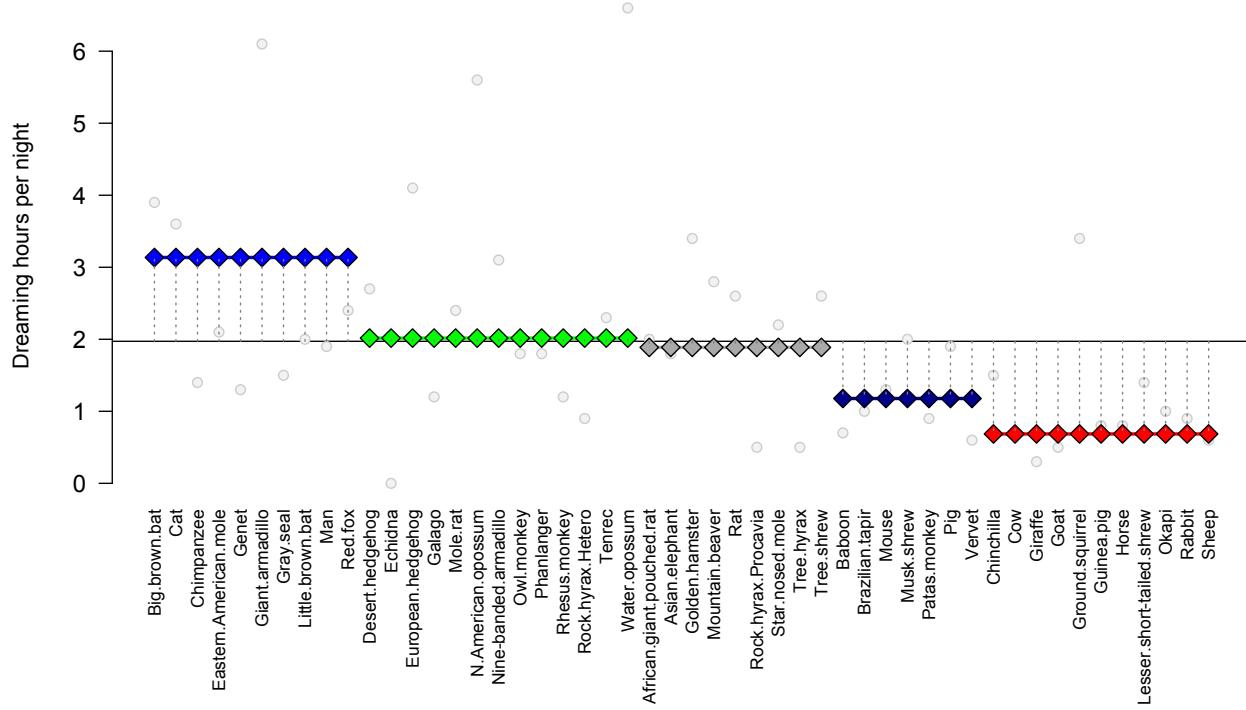


Figure 3.6: Dreaming hours by species (in grey), along with the fitted values (colored diamonds) from the group-wise model using predation index as a predictor. The vertical lines depict the differences $\hat{y}_i - \bar{y}$.

- The predictable variation, or the sum squared differences between the fitted values and the grand mean. This measures the variability described by the model:

$$PV = \sum_{i=1}^n (\hat{y}_i - \bar{y})^2 = 36.4.$$

- The unpredictable variation, or the sum of squared residuals from the group-wise model. This is the variation left over in the observed values after accounting for group membership:

$$UV = \sum_{i=1}^n (y_i - \hat{y}_i)^2 = \sum_{i=1}^n e_i^2 = 65.7.$$

What's special about these numbers? Well, notice that

$$102.1 = 36.4 + 65.7,$$

so that $TV = PV + UV$. The model has cleanly partitioned the original sum of squares in two components: one predicted by the model, and one not.

What if we measured variation using sums of absolute values instead? Let's try it and see:

$$\begin{aligned}\sum_{i=1}^n |y_i - \bar{y}| &= 53.0 \\ \sum_{i=1}^n |\hat{y}_i - \bar{y}| &= 33.7 \\ \sum_{i=1}^n |y_i - \hat{y}_i| &= 42.5.\end{aligned}$$

Clearly $53.0 \neq 33.7 + 42.5$. If this had been how we'd defined TV, PV, and UV, we wouldn't have such a clean "partitioning effect" like the kind we found for sums of squares.

Is this partition effect a coincidence, or a meaningful generalization? To get further insight, let's try the same calculations on the peak-demand data set from Figure 3.2, seen again at right. First, we sum up the squared deviations $y_i - \bar{y}$ to get the total variation:

$$TV = \sum_{i=1}^n (y_i - \bar{y})^2 = 166,513,967.$$

Next, we sum up the squared deviations of the fitted values. For each observation, the fitted value is just the group-wise mean for the corresponding month, given by the blue dots at right:

$$PV = \sum_{i=1}^n (\hat{y}_i - \bar{y})^2 = 50,262,962.$$

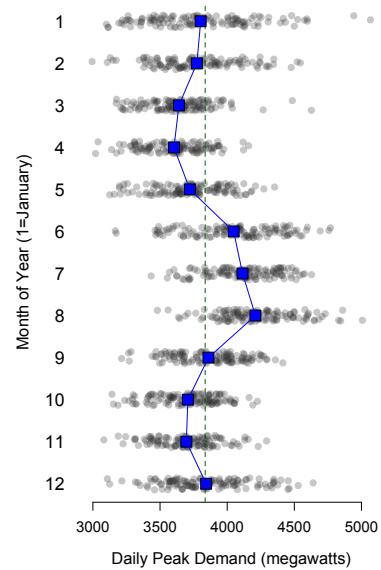
Finally, we sum up the squared residuals from the model:

$$UV = \sum_{i=1}^n (y_i - \hat{y}_i)^2 = 116,251,005.$$

Sure enough: $166,513,967 = 50,262,962 + 116,251,005$. The same "TV = PV + UV" statement holds when using sums of squares, just as for the previous data set.

And if we try sums of absolute values?

$$\begin{aligned}\sum_{i=1}^n |y_i - \bar{y}| &= 397,887.7 \\ \sum_{i=1}^n |\hat{y}_i - \bar{y}| &= 220,382.1 \\ \sum_{i=1}^n |y_i - \hat{y}_i| &= 325,409.0.\end{aligned}$$



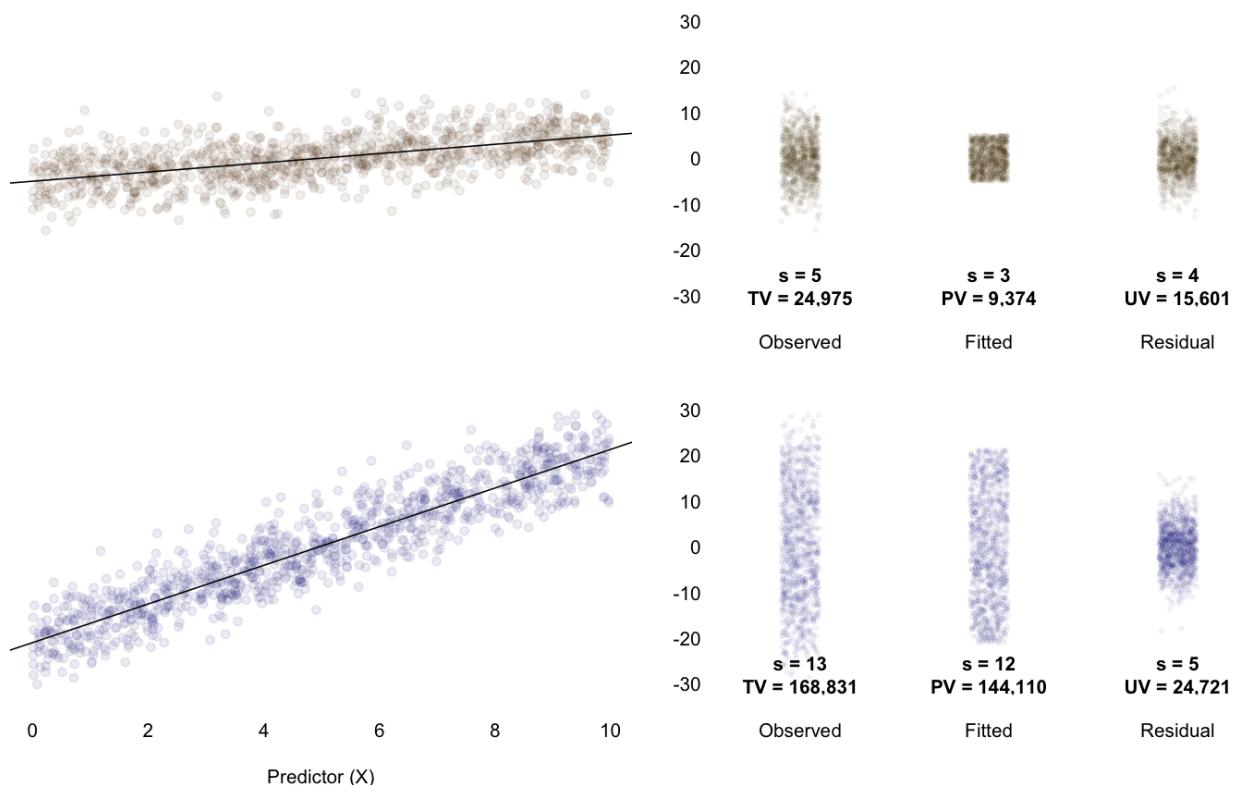


Figure 3.7: Two imaginary data sets, along with their least squares lines.

Clearly, $397,887.7 \neq 220,382.1 + 325,409.0$. Just like the mammalian sleep-pattern data, the peak-demand data exhibits no partitioning-of-variation effect using sums of absolute deviations.

The same decomposition also holds for linear regression models. In Figure 3.7 we see two scatter plots of two simulated data sets, both measured on the same X and Y scales. Next to each are dot plots of the original Y variable, the fitted values, and the residuals. In each case, $TV = PV + UV$, and therefore the three standard deviations form Pythagorean triples.

The analysis of variance: a first look

MEASURING variation using sums of squares is not at all an obvious thing to start out doing. But obvious or not, we do it for a very good reason: sums of squares follow the lovely, clean decomposition that we happened upon in the previous section:

$$\sum_{i=1}^n (y_i - \bar{y})^2 = \sum_{i=1}^n (\hat{y}_i - \bar{y})^2 + \sum_{i=1}^n (y_i - \hat{y}_i)^2$$

$$TV = PV + UV. \quad (3.1)$$

This is true both for group-wise models and for linear models. TV and UV tell us much variation we started with, and how much we have left over after fitting the model, respectively. PV tells us where the missing variation went—into the fitted values!

As we've repeatedly mentioned, it would be perfectly sensible to measure variation using sums of absolute values $|y_i - \hat{y}_i|$ instead, or even something else entirely. But if we were to do this, the analogous "TV = PV + UV" decomposition would not hold as a general rule:

$$\sum_{i=1}^n |y_i - \bar{y}| \neq \sum_{i=1}^n |\hat{y}_i - \bar{y}| + \sum_{i=1}^n |y_i - \hat{y}_i|.$$

In fact, a stronger statement is true: there is literally no power other than 2 that we could have chosen that would have led to a decomposition like Equation 3.1. Sums of squares are special because they, and they alone, can be partitioned cleanly into predictable and unpredictable components.

This partitioning effect is something of a mystery—most things in everyday life simply don't work this way. For example, imagine that you and your sibling are trying to divide up a group of 100

stuffed animals that you own in common. It makes no sense to say: "Well, there are 10,000 (100^2) squared-stuffed-animalss in total, so I'll take 3,600 (60^2) squared stuffed animals, and you take the remaining 1,600 (40^2)."¹ Not only is the statement itself barely interpretable—what the heck is a squared stuffed animal?—but the math doesn't even work out ($100^2 \neq 60^2 + 40^2$).

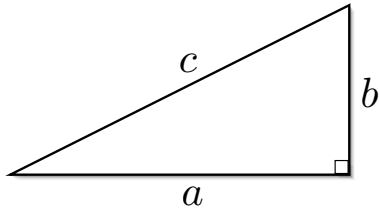
Is there a deeper reason why this partitioning effect occurs for sums of squares in statistical models, and not for some other measure of variation? The figure at right should jog your memory, for this isn't the first time you've seen a similar result before. Pythagoras' famous theorem says that $c^2 = a^2 + b^2$, where c is the hypotenuse of a right triangle, and a and b are the legs. Notice that Pythagoras *doesn't* have anything interesting to say about the actual numbers: $c \neq a + b$. It's the squares of the numbers that matter.

This way of partitioning a whole into parts makes no sense for DVDs, but it does occur in real life—namely, every time you traverse a city or campus laid out on a grid. In Figure 3.8, for example, you see part of a 1930 map of the University of Texas. Both then and now, any student who wanted to make her way from the University Methodist Church (upper left star) to the football stadium (lower right star) would need to travel about 870 meters as the crow flies. She would probably do so in two stages: first by going 440 meters south on Guadalupe, and then by going 750 meters east on 21st Street.

Notice how the total distance gets partitioned: $870 \neq 440 + 750$, but $870^2 = 440^2 + 750^2$. North–south and east–west are perpendicular directions, and if you stay along these axes, total distances will add in the Pythagorean way, rather than in the usual way of everyday arithmetic.

So it is with a statistical model. You can think of the fitted values \hat{y}_i and the residuals e_i as pointing in two different directions that are, mathematically speaking, perpendicular to one another: one direction that can be predicted by the model, and one direction that can't. The total variation is then like the hypotenuse of the right triangle so formed:

This business of partitioning sums of squares into components is called the *analysis of variance*, or ANOVA. (Analysis, as in splitting apart.) So far we've only split TV into two components, PV and UV. Later on, we'll learn that the same partitioning effect still holds even when we have more than one X variable, and that we



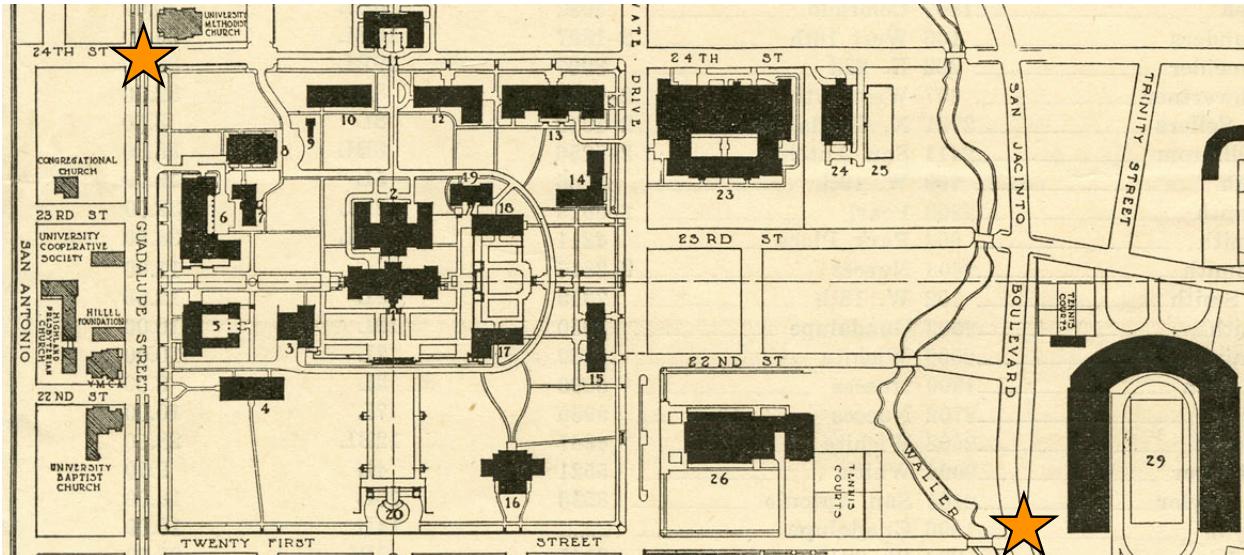
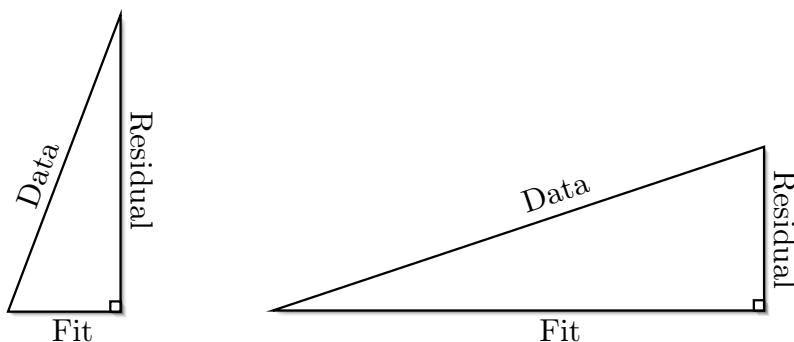


Figure 3.8: A map of the University of Texas in 1930, with two houses of worship highlighted: the University Methodist Church (upper left) and the football stadium (lower right).



can actually sub-partition PV into different components corresponding to the different predictors.

One final note on sums of squares: I've been vague about one crucial point. It turns out that this story about the fitted values and residuals pointing in perpendicular directions isn't a metaphor. It's a genuine mathematical reality—a deep consequence, in fact, of the geometry of vectors in high-dimensional Euclidean space. We'll leave it at the metaphorical level for now, though; it's not that the math is all that hard, but it does require some extra notation that is best deferred to a more advanced treatment of regression. Just be aware that the standard deviations of the three main quantities—the residuals, the fitted values, and the y values—will always form a Pythagorean triple.

The coefficient of determination: R^2

BY THEMSELVES, sums of squares are hard to interpret, because they are measured in squared units of the Y variable. But their ratios are highly meaningful. In fact, the ratio of PV to TV—or what fraction of the total variation has been predicted by the model—is one of the most frequently quoted summary measures in all of statistical modeling. This ratio is called the *coefficient of determination*, and is usually denoted by the symbol R^2 :

$$R^2 = \frac{PV}{TV} = 1 - \frac{UV}{TV}.$$

Dividing by TV simultaneously cancels the units of PV and standardizes it by the original scale of the data.

The value of R^2 is a property of a model and a data set considered jointly, and not of either one considered on its own. In analyzing the mammalian sleep-pattern data, for example, we started out with $TV = 102.1$ squared hours in total variation, and were left with $UV = 65.7$ squared hours in unpredictable variation after fitting the group-wise model based on the predation index. Therefore $R^2 = PV/TV \approx 0.36$, meaning that the model predicts 36% of the total variation in dreaming hours.

The correct interpretation of R^2 sometimes trips people up, and is therefore worth repeating: it is the proportion of variance in the data that can be predicted using the statistical model in question. Here are three common mistakes of interpretation to look out for, both in your own work and in that of others.

Mistake 1: Confusing R^2 with the slope of a regression line. We've now encountered three ways of summarizing the dependence between a predictor X and response Y :

r , the sample correlation coefficient between Y and X .

$\hat{\beta}_1$, the slope from the least-squares fit of Y on X . This describes the average rate of change of the Y variable as the X variable changes.

R^2 , the coefficient of determination from the least-squares fit of Y on X . This measures how much of the variation in Y can be predicted using the least-squares regression line of Y on X :

$$R^2 = 1 - \frac{UV}{TV} = \frac{PV}{TV},$$

An interesting fact is that, for a linear regression model, $R^2 = r^2$. That is, the coefficient of determination is precisely equal to the square of the sample correlation coefficient between X and Y . This is yet another reason to use correlation only for measuring linear relationships.

or predictable variation divided by total variation.

These are different quantities: the slope β_1 quantifies the trend in Y as a function of X , while both r and R^2 quantify the amount of variability in the data that is predictable using the trend.

Another difference is that both r and R^2 are unit-free quantities, while β_1 is not. No matter how Y is measured, its units cancel out when you churn through the formulas for r and R^2 —you should try the algebra yourself. This is as it should be: r and R^2 are meant to provide a measure of dependence that can be compared across different data sets. They must not, therefore, be contingent upon the units of measure for a particular problem.

On the other hand, β_1 is measured as a ratio of the units of Y to units of X , and is inescapably problem-specific. The slope, after all, is a rate of change:

- If X is years of higher education and Y is future salary in dollars, then β_1 is dollars per year of education.
- If X is seconds and Y is meters, then β_1 is meters per second.
- If X is bits and Y is druthers, then β_1 is druthers per bit.

And so forth.

These quantities are also related to each other. We already know that R^2 is also the square of the sample correlation between X and Y . What may come as more of a surprise is that R^2 is *also* the square of the correlation coefficient between y_i and \hat{y}_i , the fitted values from the regression line.² Intuitively, this is because the least-squares line absorbs all the correlation between X and Y into the fitted values \hat{y} , leaving us with $r(\hat{y}, x) = r(y, x)$ and $r(e, x) = 0$. Remember: $TV = PV + UV$, and the PV is precisely the variation we can explain by taking the “ X -ness” out of Y .

The upshot is that all three of our summary quantities— r , $\hat{\beta}_1$, and R^2 —can be related to each other in a single line of equations:

$$\{r(y, x)\}^2 = \{r(y, \hat{y})\}^2 = R^2.$$

That is: the squared correlation between y and x equals the squared correlation between y and the fitted values of the model (\hat{y}), which also equals the R^2 of the model.

Mistake 2: Quoting R^2 while ignoring the story in the residuals. We have seen that the residuals from the least-squares line are uncorrelated with the predictor X . Uncorrelated, yes—but not necessarily independent. Take the four plots from Figure 1.9, shown

² To see this algebraically, note that

$$r = \frac{\sum_{i=1}^n (y_i - \bar{y})(\hat{y}_i - \bar{\hat{y}})}{(n-1)s_y s_{\hat{y}}}.$$

Plug in the fitted values $\hat{y}_i = \hat{\beta}_0 + x_i \hat{\beta}_1$, and by churning through the algebra you will be able to recover $r(y, x)$ at the end.

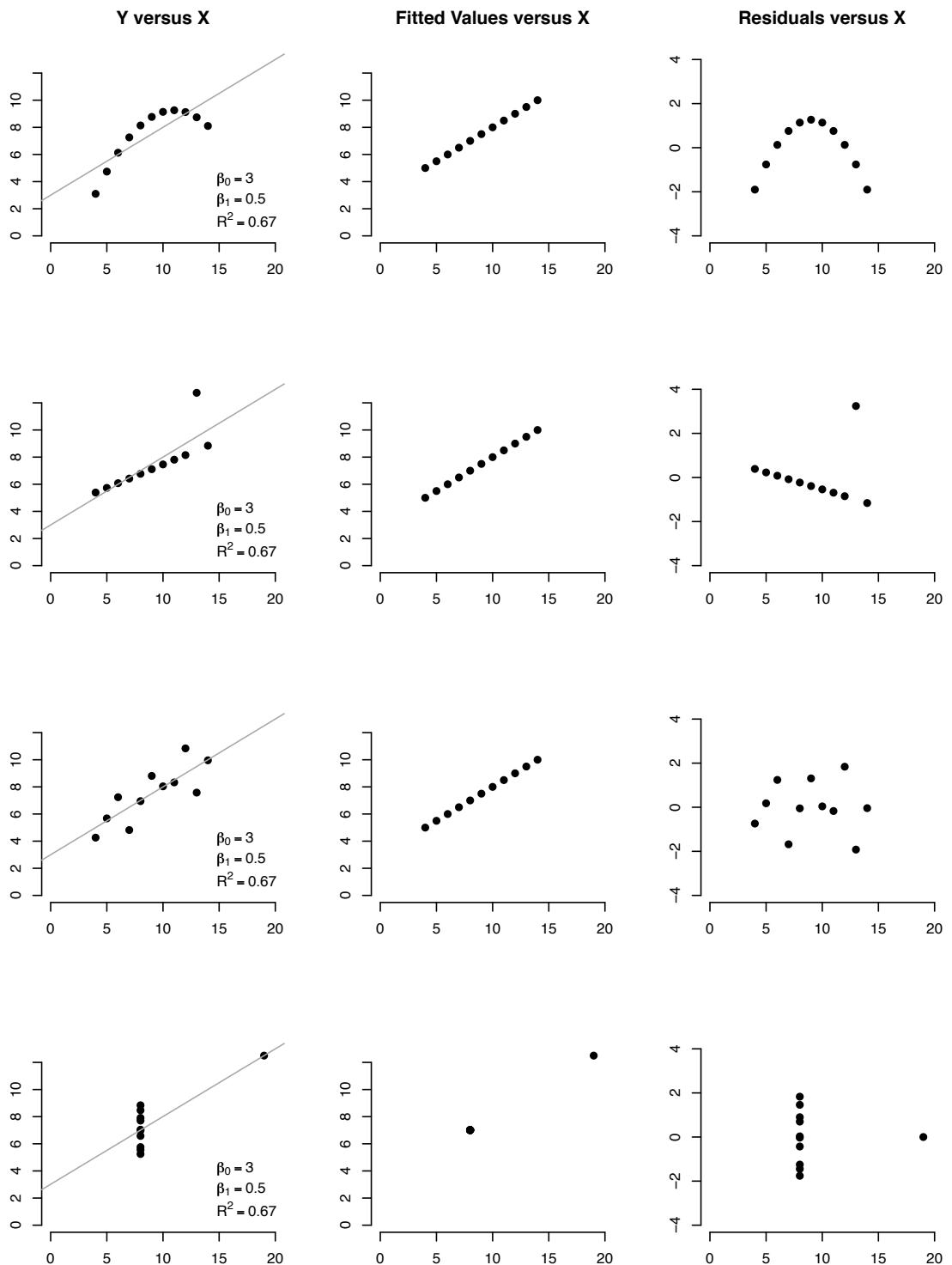


Figure 3.9: These four data sets have the same least-squares line.

again on page 73. These four data sets have the same correlation coefficient, $r = 0.816$, despite having very different patterns of dependence between the X and Y variable.

The disturbing similarity runs even deeper: the four data sets all have the same least-squares line and the same value of R^2 , too. In Figure 3.9 we see the same set of three plots for each data set: the data plus the least-squares line; the fitted values versus X ; and the residuals versus X . Note that in each case, despite appearances, the residuals and the predictor variable have zero sample correlation; this is an inescapable property of least squares.

Despite being equivalent according to just about every standard numerical summary, these data sets are obviously very different from one another. In particular, only in the third case do the residuals seem truly *independent* of X . In the other three cases, there is clearly still some X -ness left in Y that we can see in the residuals. Said another way, there is still information in X left on the table that we can use for predicting Y , even if that information cannot be measured using the crude tool of sample correlation. It will necessarily be true that $r(e, x) = 0$. But sometimes this will be a truth that lies, and if you plot your data, your eyes will pick up the lie immediately.

The moral of the story is: like the correlation coefficient, R^2 is just a single number, and can only tell you so much. Therefore when you fit a regression, always plot the residuals versus X . Ideally you will see a random cloud, and no X -ness left in Y . But you should watch out for systematic nonlinear trends—for example, groups of nearby points that are all above or below zero together. This certainly describes the first data set, where the real regression function looks to be a parabola, and where we can see a clear trend left over in the residuals. You should also be on the lookout for obvious outliers, with the second and fourth data sets providing good examples. These outliers can be very influential in a standard least-squares fit.

We will soon turn to the question of how to remedy these problems. For now, though, it's important to be able to diagnose them in the residuals.

Mistake 3: Confusing statistical explanations with real explanations.

You will often hear R^2 described as the proportion of variance in Y “explained” by the statistical model. Do not confuse this usage of the word “explain” with the ordinary English usage of the word,

which inevitably has something to do with causality. This is an insidious ambiguity. As Edward Tufte writes:

A big R^2 means that X is relatively successful in predicting the value of Y —not necessarily that X causes Y or even that X is a meaningful explanation of Y . As you might imagine, some researchers, in presenting their results, tend to play on the ambiguity of the word “explain” in this context to avoid the risk of making an out-and-out assertion of causality while creating the appearance that something really was explained substantively as well as statistically.³

You’ll notice that, for precisely this reason, we’ve avoided describing R^2 in terms of “explanation” at all, and have instead referred to it as the “ratio of predictable variation to total variation.”

We know that correlation and causality are not the same thing, and R^2 quantifies the former, not the latter. Consider the data set in the table at right. Regressing the number of patent applications on the number of letters in the vice president’s first name yields $\hat{\beta}_1 = -26,920$ applications per letter, suggesting a negative trend. Moreover, the regression produces an impressive-looking R^2 of 0.71, meaning that over two-thirds of the variability in patent applications can be predicted using the length of the vice president’s first name alone.

Nothing has been “explained” here at all, the high R^2 notwithstanding. The least-squares fit is capable of answering the question: *if X has a causal linear effect on Y, then what is the best estimate of this effect, and how much variation does this effect account for?* This question assumes a causal hypothesis, and therefore patently cannot be used to test this hypothesis. In particular, calling one variable the “predictor” and the other variable the “response” simply does not decide the issue of causation.

³ *Data Analysis for Politics and Policy*, p. 72.

| Year | Letters in first name of U.S. vice president | Number of U.S. patent applications |
|------|--|------------------------------------|
| 2000 | 2 | 315,015 |
| 1999 | 2 | 288,811 |
| 1998 | 2 | 260,889 |
| 1997 | 2 | 232,424 |
| 1996 | 2 | 211,013 |
| 1995 | 2 | 228,238 |
| 1994 | 2 | 206,090 |
| 1993 | 2 | 188,739 |
| 1992 | 3 | 186,507 |
| 1991 | 3 | 177,830 |
| 1990 | 3 | 176,264 |
| 1989 | 3 | 165,748 |
| 1988 | 6 | 151,491 |
| 1987 | 6 | 139,455 |
| 1986 | 6 | 132,665 |
| 1985 | 6 | 126,788 |
| 1984 | 6 | 120,276 |
| 1983 | 6 | 112,040 |
| 1982 | 6 | 117,987 |
| 1981 | 6 | 113,966 |

Table 3.1: Patent-application data available from the United States Patent and Trademark Office, Electronic Information Products Division.

4

Grouping variables in regression

Grouping variables and aggregation paradoxes

THE previous chapters have taught us to fit equations to data involving a numerical response and a numerical predictor. In this chapter, we'll generalize these ideas to incorporate grouping variables as predictors, too.

It's very common in real-world systems for one variable to modulate the effect of another. For example, a person's overall size and weight modulate the relationship between alcohol and cognitive impairment. A single glass of wine might make a small person feel drunk, but have a negligible effect on a big person.

This phenomenon is easiest to visualize in data when the variable that does the modulating is categorical. To see an example of this, we'll revisit the data set on college GPA versus high-school SAT scores. You'll recall that this data set catalogues all 5,191 students at the University of Texas who matriculated in the fall semester of 2000, and who went on to graduate within five years. In Figure 4.2, we notice the expected positive relationship between combined SAT score and final GPA. We also notice the fact that SAT scores and graduating GPAs tend to differ substantially from one college to the next. Figure 4.1 shows boxplots of SAT and GPA stratified by the ten undergraduate colleges at UT.

What we see in Figures 4.2 and 4.1 is an example of an *aggregation paradox*, where the same trend that holds for individuals does not hold for groupings of individuals. Why is this a paradox? Look carefully at the data: Figure 4.1 says that students with higher SAT scores tend to have higher GPAs. Yet this trend does not hold at the college level, even broadly. For example, Engineering students (as a group) have among the highest average SAT scores, and among the lowest average GPAs. Thus we have a paradox: it looks as though high SAT scores predict high GPAs, but being in a college with high SAT scores does not predict being in a

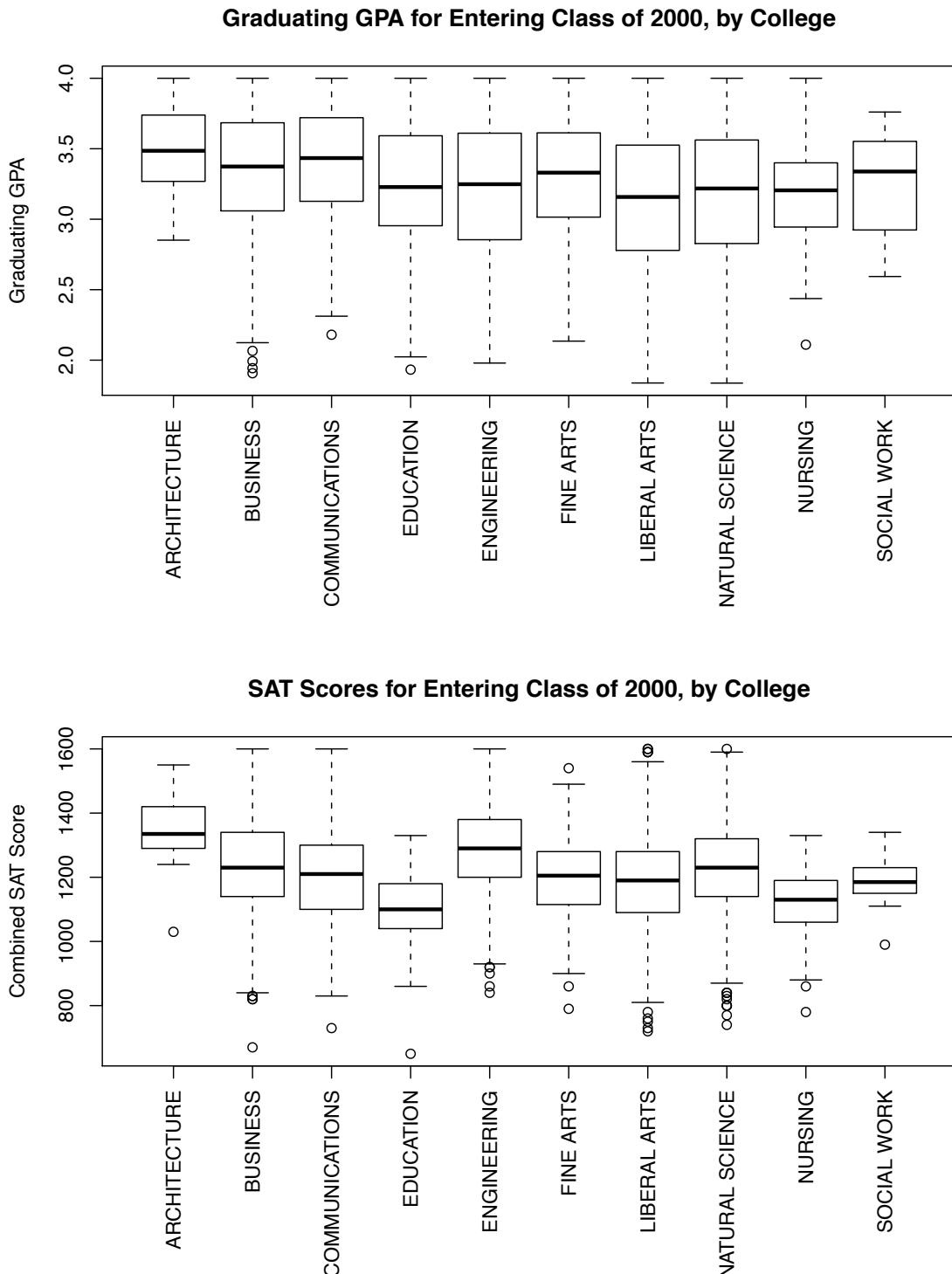


Figure 4.1: GPA and SAT scores stratified by the ten undergraduate colleges at UT.

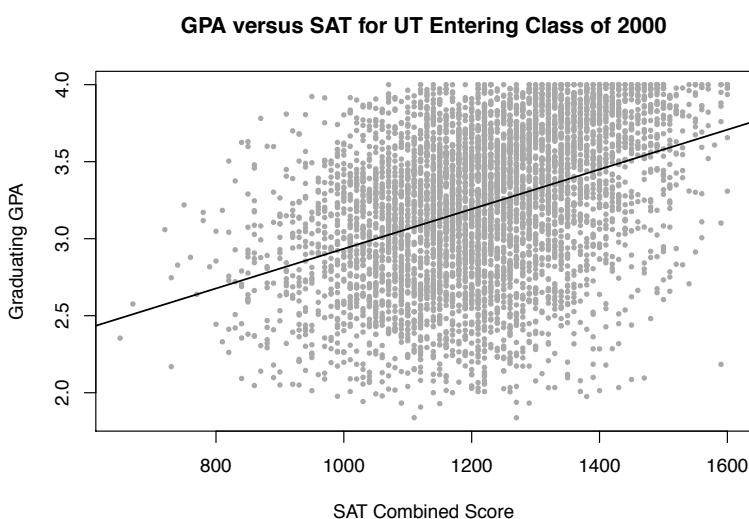


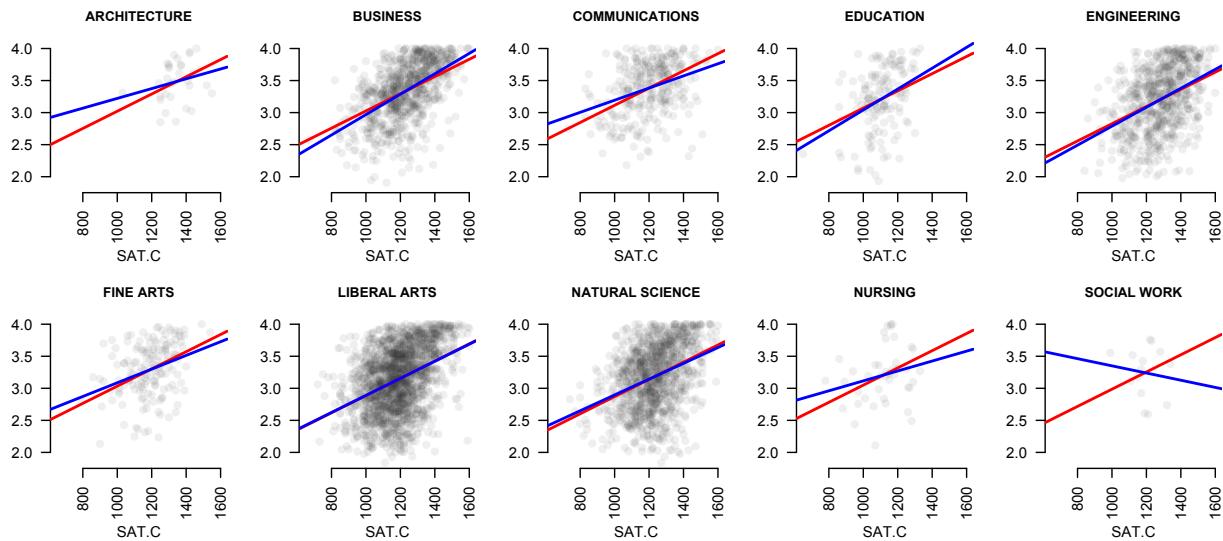
Figure 4.2: Combined SAT scores versus graduating GPA for the entering fall class of 2000 at the University of Texas.

college with high GPAs.

The paradox disappears when we realize the the “College” variable modulates the relationship between SAT score and GPA. A student’s college is systematically associated with both SAT and GPA: some degrees are harder than others, and these degrees tend to enroll students with higher test scores.

The right way to proceed here is to disaggregate the data and fit a different regression line within each of the ten colleges, to account for the effect of the modulating variable. There are two different ways to do this:

1. We could fit ten different lines, each with a different intercept ($\beta_0^{(k)}$), but all with the same slope (β_1). This would make sense if we thought that the same SAT–GPA relationship ought to hold within each college, but that each college had a systematically higher or lower intercept (average GPA). These are the red lines in Figure 4.3. You can see the differences among the red lines if you look carefully at where they hit the y axis in relation to a GPA of 2.5—for example, compare Communications and Engineering.
2. We could fit ten different lines, allowing both the slope and the intercept to differ for each college. We would do this if we thought that the SAT–GPA relationship differed fun-



damentally across the colleges. These are the blue lines in Figure 4.3.

But which strategy should we take? And how would we even accomplish strategy 1 using ordinary least squares?

Things get even more complex in the presence of more than one grouping variable. For example, we might want to look at these relationships separately for different years, for men versus women, and for in-state and out-of-state students. To be able to model the effect of all these variables on GPA simultaneously, we will need to introduce some new notation and a few new concepts.

Models for a single grouping variable

Dummy variables

LET's return to a simple scenario where we have numerical data that falls into two groups, and we want to compare the variation between the groups. The dotplot in Figure 4.4 shows the weekly sales volume of package sliced cheese over 61 weeks at a Dallas-area Kroger's grocery store. In 38 of these weeks, the store set up a prominent display near the entrance, calling shoppers' attention to the various culinary adventures they might undertake with the

Figure 4.3: Separate regression models fit for GPA versus SAT within each college. The red lines all have the same slope, but a different intercept for each college. The blue lines all have different intercepts and different slopes.

cheese. The data show that, in these 38 weeks, sales were higher overall than when no display was present.

How much higher? The average sales volume in display weeks was 5,577 units (the blue dotted line in Figure 4.4), versus an average of 2341 units in non-display weeks (the red dotted line). Thus sales were 3236 units higher in the display weeks. This difference is depicted in Figure 4.4 as the difference or offset between the dotted lines.

This example emphasizes that in many data sets, we care less about the absolute magnitude of a response under different conditions, and more about the differences between those conditions. We therefore often build our model in such a way that these differences are estimated directly, rather than indirectly (i.e. by calculating means and then subtracting them).

We do this using *indicator* or *dummy* variables. To understand this idea, take the simple case of a single grouping variable x with two levels: “on” ($x = 1$) and “off” ($x = 0$). We can write this model in “baseline/offset” form:

$$y_i = \beta_0 + \beta_1 \mathbf{1}_{\{x_i=1\}} + e_i .$$

The quantity $\mathbf{1}_{\{x_i=1\}}$ is called a dummy variable; it takes the value 1 when $x_i = 1$, and the value 0 otherwise. Just as in an ordinary linear model, we call β_0 and β_1 the *coefficients* of the model. This way of expressing the model implies the following.

$$\begin{aligned} \text{Group mean for case where } x \text{ is off} &= \beta_0 \\ \text{Group mean for case where } x \text{ is on} &= \beta_0 + \beta_1 . \end{aligned}$$

Therefore, we can think of β_0 as the baseline (or *intercept*), and β_1 as the offset. To see this in action, consult Figure 4.4 again. Here the dummy variable encodes the presence of an in-store display. The red dot at 2341, in the non-display weeks, is β_0 . This is the baseline case, when the dummy variable x is “off.” The coefficient for the dummy variable, $\beta_1 = 3236$, is the vertical distance between the two means. Thus if we wanted to reconstruct the mean for the with-display weeks, we would just add the baseline and the offset, to arrive at $2341 + 3236 = 5577$, where the blue dot sits.

As before, we estimate the values of β_0 and β_1 using the least-squares criterion: that is, make the sum of squared errors, $\sum_{i=1}^n e_i^2$, as small as possible. This is mathematically equivalent to computing the group-wise means separately, and then calculating the difference between the means.

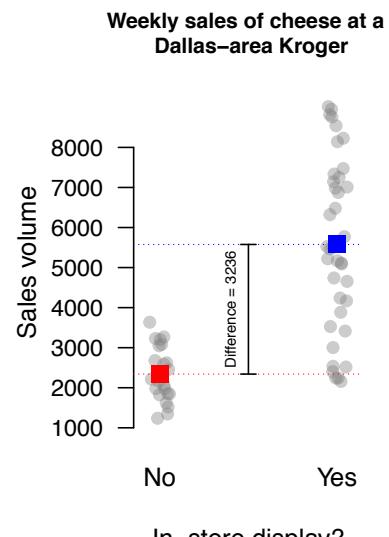
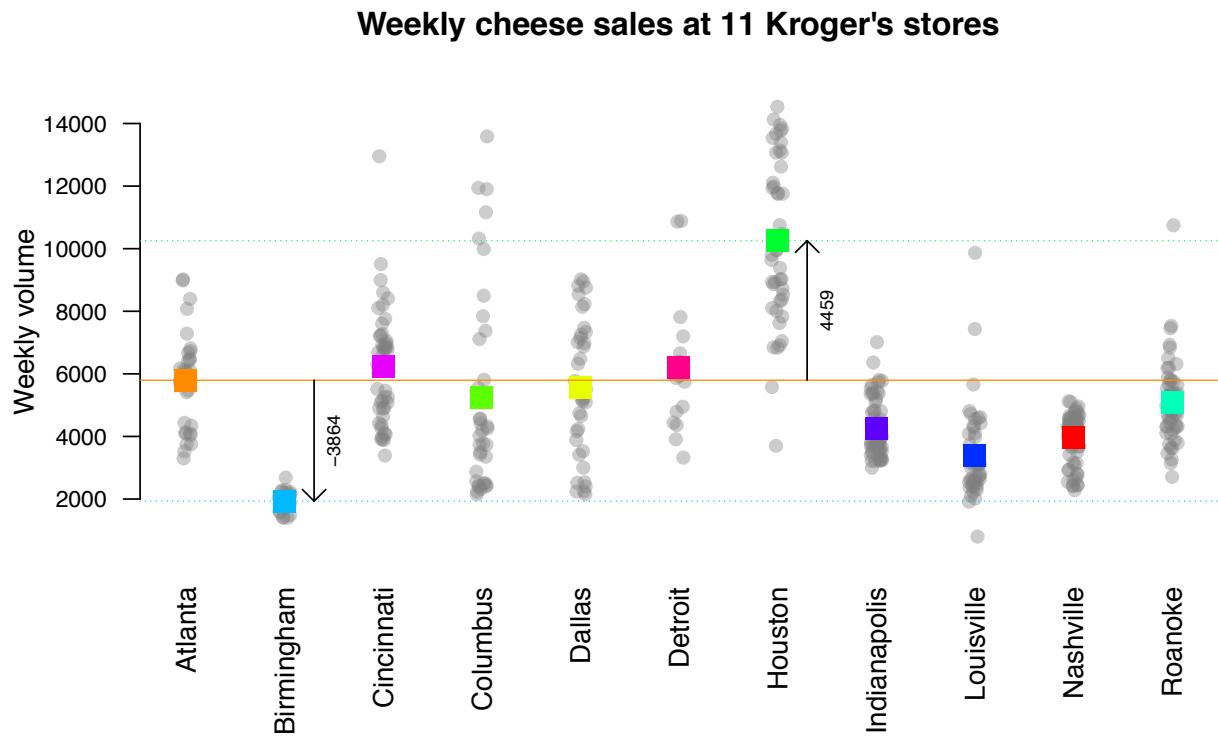


Figure 4.4: Weekly sales of packaged cheese slices at a Dallas-area Kroger's grocery store, both with and without the presence of an in-store display ad for the cheese. The red dot shows the mean of the no-display weeks, and the blue dot shows the mean of the with-display weeks. The estimated coefficient for the dummy variable that encodes the presence of a display ad is 3236, which is the vertical distance between the two dots.



More than two levels

If the categorical predictor x has more than two levels, we represent it in terms of more than one dummy variable. Suppose that x can take three levels, labeled arbitrarily as 0 through 2. Then our model is

$$y_i = \beta_0 + \beta_1^{(1)} \mathbf{1}_{\{x_i=1\}} + \beta_1^{(2)} \mathbf{1}_{\{x_i=2\}} + e_i.$$

The dummy variables $\mathbf{1}_{\{x_i=1\}}$ and $\mathbf{1}_{\{x_i=2\}}$ tell you which of the levels is active for the i th case in the data set.¹

More generally, suppose we have a grouping variable with K levels. Then $\beta_1^{(k)}$ is the coefficient associated with the k th level of the grouping variable, and we write the full model as a sum of $K - 1$ dummy-variable effects, like this:

$$y_i = \beta_0 + \sum_{k=1}^{K-1} \beta_1^{(k)} \mathbf{1}_{\{x_i=k\}} + e_i \quad (4.1)$$

Figure 4.5: Weekly sales of packaged cheese slices during weeks with an advertising display at 11 Kroger's grocery stores across the country.

¹ Normal people count starting at 1. Therefore you might find it strange that we start counting levels of a categorical variable at 0. The rationale here is that this makes the notation for group-wise models a lot cleaner compared to starting at 1.

We call this a *group-wise model*. Notice that there is no dummy variable for the case $x = 0$. This is the baseline level, whose group mean is the intercept β_0 . In general, for a categorical variable with K levels, we will need $K - 1$ dummy variables, and at most one of these $K - 1$ dummy variables is ever active for a single observation. The coefficient on each dummy variable ($\beta_1^{(k)}$) is the differences between the baseline and the mean of group k :

$$\begin{aligned}\text{Group mean for case where } (x_i = 0) &= \beta_0 \\ \text{Group mean for case where } (x_i = k) &= \beta_0 + \beta_1^{(k)}.\end{aligned}$$

In Figure 4.5, we see an example of a single categorical variable with more than two levels. The figure shows weekly cheese sales (during display-present weeks only) at 11 different Kroger stores in 11 different markets across the country. The grouping variable here is the market: Atlanta, Birmingham, Cincinnati, and so forth. If we fit a model like Equation 4.1 to the data in this figure, choosing Atlanta to be the baseline, we get the set of estimated coefficients in the second column (“Coefficient”) of the table below:

| Variable | Coefficient | Group mean |
|--------------|-------------|------------|
| Intercept | 5796 | — |
| Birmingham | -3864 | 1932 |
| Cincinnati | 427 | 6223 |
| Columbus | -543 | 5253 |
| Dallas | -219 | 5577 |
| Detroit | 400 | 6196 |
| Houston | 4459 | 10255 |
| Indianapolis | -1542 | 4254 |
| Louisville | -2409 | 3387 |
| Nashville | -1838 | 3958 |
| Roanoke | -717 | 5079 |

Atlanta is the baseline, and so the intercept is the group mean for Atlanta: 5796 packages of crappy cheese. To get the group mean for an individual market, we add that market’s offset to the baseline. For example, the mean weekly sales volume in Houston is $5796 + 4459 = 10255$ units. Group mean = baseline + offset.

The figure also shows you two of the offsets as arrows, to give you a visual sense of what these numbers in the above table represent. The coefficient for Houston is $\beta_6^{(1)} = 4459$, because the group

mean for Houston (10255) is 4459 units *higher* than the baseline group mean for Atlanta (a positive offset). Similarly, the coefficient for Birmingham is $\beta_1^{(1)} = -3864$, because the group mean for Birmingham (1932) is 3864 units *lower* than the baseline group mean for Atlanta (a negative offset).

The choice of baseline. In the above analysis, we chose Atlanta as the baseline level of the grouping variable. This was arbitrary.

We could have chosen any city as a baseline, measuring the other cities as offsets from there instead.

A natural question is: does the model change depending on what level of the grouping variable we choose to call the baseline? The answer is: yes and no. Yes, the estimated model coefficients will change when a different baseline is used; but no, the underlying group means do not change. To see this, consider what happens when we fit another model like Equation 4.1 to the Kroger cheese-sales data, now choosing the Dallas store to be the baseline:

| Variable | Coefficient | Group mean |
|--------------|-------------|------------|
| Intercept | 5577 | — |
| Atlanta | 219 | 5796 |
| Birmingham | -3644 | 1932 |
| Cincinnati | 646 | 6223 |
| Columbus | -324 | 5253 |
| Detroit | 619 | 6196 |
| Houston | 4678 | 10255 |
| Indianapolis | -1323 | 4254 |
| Louisville | -2190 | 3387 |
| Nashville | -1619 | 3958 |
| Roanoke | -498 | 5079 |

The intercept is the Dallas group mean of 5577, and the other market-level coefficients have changed from the previous table, since these now represent offsets compared to a different baseline. But the group means themselves do not change. The moral of the story is that the coefficients in a model involving dummy variables *do* depend upon the choice of baseline, but that the information these coefficients encode—the means of the underlying groups—does not. Different choices of the baseline just lead to different ways of expressing this information.

Multiple grouping variables

WE BEGAN our discussion of dummy variables by looking at a simple group-wise model with a binary predictor, meaning that x_i is either 0 or 1. Such a model takes the form

$$y_i = \beta_0 + \beta_1 \mathbf{1}_{\{x_i=1\}} + e_i.$$

We learned something important about this model: that the coefficient β_1 can be interpreted as the differential effect of being in group 1, as opposed to the baseline (group 0).² That's a nice feature of using dummy variables: if we care primarily about the difference in the average response between conditions, we get an estimate of that difference ($\hat{\beta}_1$) directly from the fitted model.

This approach of using dummy variables to encode the grouping structure of our data really comes into its own when we encounter data sets with more than one grouping variable. To see why, we'll spend some time with the data in Figure 4.6.

² Remember, we start counting groups/levels at 0.

Main effects

Making a best-selling video game is hard. Not only do you need a lot of cash, a good story, and a deep roster of artists, but you also need to make the game fun to play. Take Mario Kart for the Super Nintendo, my favorite video game from childhood. In Mario Kart, you had to react quickly to dodge banana peels and Koopa shells launched by your opponents as you all raced virtual go-karts around a track. The game was calibrated just right. If the required reaction time had been just a little slower, the game would have been too easy, and therefore boring. But if the required reaction time had been a little bit faster, the game would have been too hard, and therefore also boring.

Human reaction time to visual stimuli is a big deal to video game makers. They spend a lot of time studying it and adjusting their games according to what they find. Figure 4.6 shows the results of one such study. Participants were presented with a natural scene on a computer monitor, and asked to react (by pressing a button) when they saw an animated figure appear in the scene.³

The experimenters varied the conditions of the natural scene: some were cluttered, while others were relatively open; in some, the figure appeared far away in the scene, while in others it appeared close up. They presented all combinations of these conditions to each participant many times over. The top two panels of

³ Essentially the company was measuring how quickly people could react to a bad guy popping up on the screen in a video game.

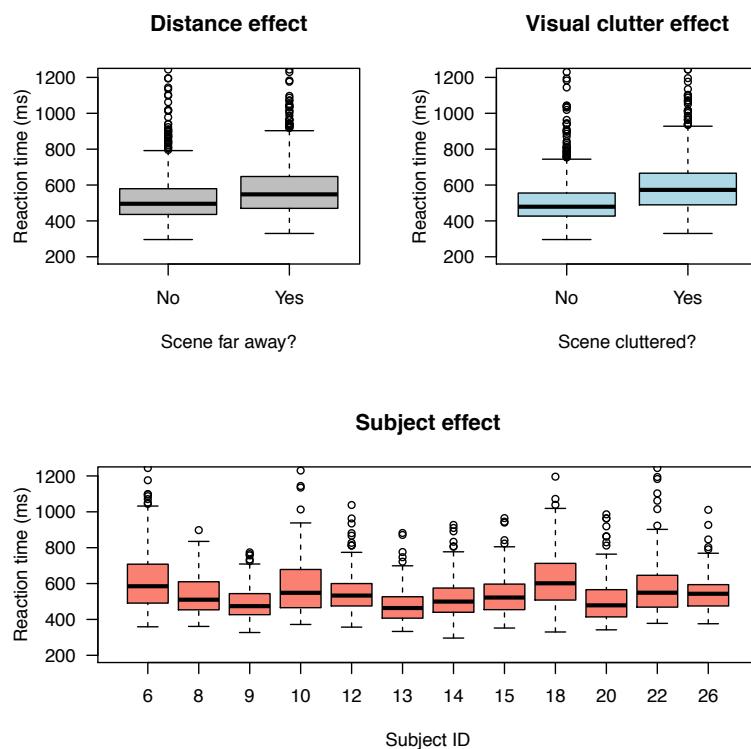


Figure 4.6: Reaction time to visual stimuli in a controlled experiment run by a major video-game maker. Top-left: participants reacted more slowly, on average, when the stimulus was far away within the scene. Top-right: participants reacted more slowly, on average, in a scene with significant visual clutter. Bottom: systematic differences in reaction time across participants in the trial.

Figure 4.6 show boxplots of all participants' reaction times across all trials under these varying conditions. On average, participants reacted more slowly to scenes that were far away (top left panel) and that were cluttered (top right panel).

We'll return to the bottom panel of Figure 4.6 shortly. For now, let's focus on the "distance effect" and the "clutter effect" in the top two panels. This presents us with the case of two grouping variables, x_1 and x_2 , each of which affects the response variable, and each of which can take the value 0 ("off") or 1 ("on"). To account for this, we need to build a model that is capable of describing the joint effect of both variables at once.

Strategy 1: slice and dice. One approach to modeling the joint effect of x_1 and x_2 on the response y is to slice and dice the data. In other words: take subsets of the data for each of the four combinations of x_1 and x_2 , and compute the mean within each subset. For our video-game data, we get the result in Table 4.1. Clearly the

Table 4.1: Mean reaction time across all trials and participants for the four combinations of the two experimental factors in the video game data.

| Cluttered | Far away | Time (ms) |
|-----------|----------|-----------|
| No | No | 491 |
| | Yes | 522 |
| Yes | No | 559 |
| | Yes | 629 |

“cluttered + far away” scenes are the hardest, on average.

This slice-and-dice approach is intuitively reasonable, but combinatorially explosive. With only two binary grouping variables, we have four possible combinations—not a big deal. But suppose we had 10 binary grouping variables instead. Then there would be $2^{10} = 1024$ possible subsets of the data, and thus 1024 group-wise means to estimate. For a scenario like this, if you were to take the slice-and-dice approach, you would need a lot of data—and not merely a lot of data overall, but a lot of data for each combination separately.

Strategy 2: use dummy variables. A second strategy is to estimate the effect of x_1 and x_2 by building a model that uses dummy variables. Intuitively, the model we'll fit assumes that the response can be expressed as:

$$y_i = \hat{y}_i + e_i = \text{Baseline} + (\text{Effect if } x_{i1} \text{ on}) + (\text{Effect if } x_{i2} \text{ on}) + \text{Residual}.$$

Notice that we need two subscripts on the predictors x_{i1} and x_{i2} : i , to index which case in the data set is being referred to; and 1 or 2, to indicate which categorical predictor is being referred to (e.g. far away versus cluttered).

This notation gets cumbersome quickly. We can write it more concisely in terms of dummy variables, just as we learned to do in the case of a single grouping variable:

$$y_i = \beta_0 + \beta_1 \mathbf{1}_{\{x_{i1}=1\}} + \beta_2 \mathbf{1}_{\{x_{i2}=1\}} + e_i.$$

Notice how the dummy variables affect the expected value of y_i by being either present or absent, depending on the case. For example, if $x_{i2} = 0$, then the $\beta_2 \mathbf{1}_{\{x_{i2}\}}$ term falls away, and we're left with the baseline, plus the effect of x_1 being on, plus the residual. We refer to β_1 and β_2 as the *main effects* of the model, for reasons that will become clear in a moment.

If we fit this model to the video-game data in Figure 4.1, we get the equation

$$\text{Reaction} = 482 + 87 \cdot \mathbf{1}_{\{x_{i1}=1\}} + 50 \cdot \mathbf{1}_{\{x_{i2}=1\}} + \text{Residual}, \quad (4.2)$$

where $x_{i1} = 1$ means that the scene was cluttered, and $x_{i2} = 1$ means that the scene was far away. This equation says that if the scene was cluttered, the average reaction time became 87 milliseconds slower; while if the scene was far away, the average reaction time became 50 milliseconds slower.

Interactions

A key assumption of the model in Equation 4.2 is that the effects of clutter and distance on reaction time are separable. That is, if we want to compute the joint effect of both conditions, we simply add the individual effects together.

But what if the effects of x_1 and x_2 aren't separable? We might instead believe a model like this:

$$y_i = \text{Baseline} + (\text{Effect if } x_1 \text{ on}) + (\text{Effect if } x_2 \text{ on}) + (\text{Extra effect if both } x_1 \text{ and } x_2 \text{ on}) + \text{Residual}.$$

In the context of our video-games data, this would imply that there's something different about scenes that are both cluttered *and* far away that cannot be described by just summing the two individual effects.

The world is full of situations like this, where the whole is different than the sum of the parts. The ancient Greeks referred to this idea as *συνέργεια*, or synergia. This roughly means "working together," and it's the origin of the English word "synergy." Synergies abound:

- Neither an actor nor a cameraman can do much individually, but together they can make a film.
- Two hydrogens and an oxygen make water, something completely unlike either of its constituent parts.
- Biking up a hill is hard. Biking in a big gear is hard. Biking up a hill in a big gear is impossible, unless you take drugs.

Examples of the whole being worse than the sum of the parts also abound—groupthink on committees, ill-conceived corporate mergers, Tylenol and alcohol, and so forth.⁴

In statistics, we operationalize the idea of synergy using *interactions among variables*. An interaction is what we get when we multiply two variables together. In the case of two binary categorical predictors, a model with an interaction looks like this:

$$y_i = \beta_0 + \beta_1 \mathbf{1}_{\{x_1=1\}} + \beta_2 \mathbf{1}_{\{x_2=1\}} + \beta_{12} \mathbf{1}_{\{x_1=1\}} \mathbf{1}_{\{x_2=1\}} + e_i.$$

We call β_{12} an *interaction term*; this term disappears from the model unless x_1 and x_2 are both equal to 1. Fitting this model to the video-games data gives the following estimates:

$$\text{Reaction} = 491 + 68 \cdot \mathbf{1}_{\{x_{i1}=1\}} + 31 \cdot \mathbf{1}_{\{x_{i2}=1\}} + 39 \cdot \mathbf{1}_{\{x_{i1}=1\}} \mathbf{1}_{\{x_{i2}=1\}} + \text{Residual},$$

We interpret this model as follows:

- The baseline reaction time for scenes that are neither cluttered nor far away is 491 milliseconds (ms).

⁴ Don't take Tylenol and alcohol together or you'll risk liver damage.

- The main effect for the “cluttered” variable is 68 ms.
 - The main effect for the “far away” variable is 31 ms.
 - The interaction effect for “cluttered” and “far away” is 39 ms.
- In other words, scenes that are both cluttered and far away yield average reaction times that are 39 milliseconds slower than what you would expect from summing the individual effects of the two variables.

From these main effects and the interaction we can use the model to summarize the expected reaction time under any combination of experimental variables:

- $(x_1 = 0, x_2 = 0)$: $\hat{y} = 491$ (neither cluttered nor far).
- $(x_1 = 1, x_2 = 0)$: $\hat{y} = 491 + 68 = 559$ (cluttered, near).
- $(x_1 = 0, x_2 = 1)$: $\hat{y} = 491 + 31 = 522$ (not cluttered, far).
- $(x_1 = 1, x_2 = 1)$: $\hat{y} = 491 + 68 + 31 + 39 = 629$ (cluttered, far).

A key point regarding the fourth case in the list is that, when a scene is both cluttered and far away, both the main effects *and* the interaction term enter the prediction. You should also notice that these predictions exactly match up with the group means in Table 4.1 on page 86.

Incorporating still more categorical predictors

Once you understand the basic recipe for incorporating two categorical predictors, you can easily extend that recipe to build a model involving more than two. For example, let’s return one last time to the video-game data in Figure 4.6 on page 86. So far, we’ve been ignoring the bottom panel, which shows systematic differences in reaction times across different subjects in the study. But we can also incorporate subject-level dummy variables to account for these differences. The actual model equation starts to get ugly with this many dummy variables, so we often use a shorthand that describes our model intuitively rather than mathematically:

$$\begin{aligned} \text{Time} \sim & \text{ Clutter effect} + (\text{Distance effect}) \\ & + (\text{Interaction of distance/clutter}) + (\text{Subject effects}). \end{aligned} \quad (4.3)$$

Here the \sim symbol means “is modeled by” or “is predicted by.”

There are 12 subjects in the data set. Thus to model the subject-level effects, we introduce 11 dummy variables, in a manner similar to what was done in Equation 4.1. The estimated coefficients for this model are in Table 4.2.

Table 4.2: Fitted coefficients for the model incorporating subject-level dummy variables into the video-game data. Remember, K levels of a factor require $K - 1$ dummy variables, because one level—in this case, the subject labeled “Subject 6” in Figure 4.6—is the baseline.

| Variable | $\hat{\beta}$ |
|-------------------|---------------|
| Intercept | 570 |
| Cluttered | 68 |
| FarAway | 31 |
| Subject 8 | -90 |
| Subject 9 | -136 |
| Subject 10 | -44 |
| Subject 12 | -76 |
| Subject 13 | -147 |
| Subject 14 | -112 |
| Subject 15 | -93 |
| Subject 18 | -8 |
| Subject 20 | -118 |
| Subject 22 | -34 |
| Subject 26 | -79 |
| Cluttered:FarAway | 39 |

When to include interactions. In the model above, we're assuming that clutter and distance affect all subjects in the same way. Thus we have 15 parameters to estimate: an intercept/baseline, two main effects for Littered and FarAway, one interaction term, and 11 subject-level dummy variables. If instead we were to compute the groupwise means for all possible combinations of subject, clutter, and distance, we'd have 48 parameters to estimate: the group mean for each combination of 12 subjects and 4 experimental conditions. Moreover, we'd be implicitly assuming an interaction between the experimental conditions and the subject, allowing clutter and distance to affect each person's average reaction time in a different way, rather than all people in the same way.

This example should convey the power of using dummy variables and interactions to express how a response variable changes as a function of several grouping variables. This framework forces us to be explicit about our assumptions, but it also allows us to be selective about the complexity of our models. Compare estimating 15 parameters versus estimating 48 parameters in the video-games example—that's a big difference in what we're asking of our data.

The essence of the choice is this:

- If a variable affects the response in a similar way under a broad range of conditions, regardless of what the other variables are doing, then that variable warrants only a main effect in our model.
- But if a variable's effect is modulated by some other variable, we should describe that using an interaction between those two variables.

The choice of which variables interact with which other ones should ideally be guided by knowledge of the problem at hand. For example, in a rowing race, a strong headwind makes all crews slower. But wind affects lighter crews more than heavier crews: weight modulates the effect of wind. Thus if we want to build a model to predict the winner of an important race, like [the one between Oxford and Cambridge every spring](#) on the Thames, we should strongly consider including an interaction between wind speed and crew weight. This is something that anyone with knowledge of rowing could suggest, even before seeing any data. But the choice of whether to include an interaction term in a model can also be guided by the data itself. We will now learn about a process called the analysis of variance that can help us

address this important modeling question.

Before we get there, however, here's one final generic guideline about interactions: it is highly unusual to include an interaction in a regression model without also including both corresponding main effects. There are various technical math reasons why most textbooks warn you about this, and why I'm doing so now. But the most important concern is that it is very difficult to interpret a model having interaction terms but no main effects. You should fit such a model only if you have a very good reason.

ANOVA: the analysis of variance

THE model in Equation 4 postulates four effects on the reaction time for the video-game data: (1) an effect due to visual clutter; (2) an effect due to distance of the stimulus in the scene; (3) an interaction effect (synergy) of distance and clutter; and (4) effects due to differences among experimental subjects. The R^2 for this model is about 0.23, and the residual standard deviation is about 126 milliseconds. This tells us something about the overall predictive abilities of the model. But can we say something about the predictive abilities of the individual variables within this model?

Yes, we can, by conducting an analysis of variance (ANOVA). An analysis of variance is just a simple book-keeping exercise aimed at attributing credit to individual variables in a model. To run an ANOVA, we build a model one step at time, adding one new variable (or one new interaction among variables) at each step. Every time we do this, we ask two questions:

- (1) How many parameters did we have to add to the model to account for the effects of this variable?⁵ This is usually called the *degrees of freedom* associated with that parameter.
- (2) By how much did we improve the predictive power of the model when we added this variable? There are a couple of ways to measure this. First, remember the variance decomposition:

$$\sum_{i=1}^n (y_i - \bar{y})^2 = \sum_{i=1}^n (\hat{y}_i - \bar{y})^2 + \sum_{i=1}^n (y_i - \hat{y}_i)^2$$

$$\text{TV} = \text{PV} + \text{UV}.$$

Every time we add a new variable to a model, the total variation in the response variable (TV) stays the same, but we move

⁵ For example, we needed to add 11 parameters to account for the "Subject" variable in the video-games data, because we needed to represent this information in terms of 11 dummy variables.

some of this variation out of the “unpredictable” column (UV) and into the “predictable” (PV) column. As a result, R^2 will always go up as a result of adding a variable to a model. In ANOVA, we keep track of the precise numerical value of this change in R^2 .

We could also measure the improvement in the model’s predictive power using the residual standard deviation, which we recall is calculated using the formula

$$s_e = \sqrt{\frac{1}{n-p} \sum_{i=1}^n (y_i - \hat{y}_i)^2}.$$

There’s an important difference with R^2 here, in that s_e can actually get worse (i.e. go up) when we add a variable to a model. If this happens, it is generally a good indication of overfitting.

The final result of an analysis of variance is a table—called the ANOVA table—that shows the answers to these two questions at each model-building step.

Let’s take the specific example of our model for the video-games data, for which $TV = 39,190,224$. We’ll add one variable at a time and track how TV is partitioned among PV and UV.⁶

Step 1. First, we add an effect due to visual clutter ($Time \sim Clutter$). The variance decomposition for this model is

$$\begin{array}{rcl} 39,190,224 & = & 3,671,938 + 35,518,285 \\ TV & & PV \\ & & UV \end{array}$$

Thus the clutter effect gets credit for predicting 3,671,938 (out of a possible 39,190,224) units of total variation, at the cost of adding one parameter to the model.

Step 2. Next, we add the distance effect to the model already containing the clutter variable ($Time \sim Clutter + Distance$). The new variance decomposition is:

$$\begin{array}{rcl} 39,190,224 & = & 4,878,397 + 34,311,827 \\ TV & & PV \\ & & UV \end{array}$$

The previous PV was 3,671,938, and the new one is 4,878,397. Thus the distance effect gets credit for $4,878,397 - 3,671,938 = 1,206,459$ units of total variation.

⁶ The quantity $TV = 39,190,224$ highlights one feature that makes ANOVA tricky at first: the units are non-intuitive, since we measure improvement using sums of squares. Here the units are squared milliseconds; when you square a quantity like 1000 ms (1 second), you get 1,000,000 ms², which is why we’re seeing numbers in the millions here.

| Variable added | # Pars (DF) | ΔPV | R^2 | ΔR^2 | s_e | Δs_e |
|-------------------------|-------------|-------------|-------|--------------|-------|--------------|
| Intercept only | 1 | | 0.000 | | 142.9 | |
| Clutter | 1 | 3671938 | 0.094 | 0.094 | 136.1 | 6.8 |
| Distance | 1 | 1206459 | 0.125 | 0.031 | 133.8 | 2.3 |
| Clutter:Distance | 1 | 183633 | 0.129 | 0.005 | 133.5 | 0.3 |
| Subject | 11 | 4060822 | 0.223 | 0.104 | 125.6 | 7.9 |
| Predictable Variation | | 9122852 | | | | |
| Unpredictable Variation | | 30067371 | | | | |
| Total Variation | | 39190224 | | | | |

Step 3. Third, we add the interaction of distance and clutter to the previous model ($\text{Time} \sim \text{Clutter} + \text{Distance} + \text{Clutter:Distance}$). The new variance decomposition is:

$$39,190,224 = 5,062,030 + 34,128,194. \quad \begin{matrix} TV \\ PV \\ UV \end{matrix}$$

The previous PV was 4,878,397, and the new one is only slightly better at 5,062,030. Thus the interaction effect gets credit for a measly $5,062,030 - 4,878,397 = 183,633$ units of total variation.

Step 4. Finally—almost done here—we add the 11 subject-level dummy variables to the previous model ($\text{Time} \sim \text{Clutter} + \text{Distance} + \text{Clutter:Distance} + \text{Subject}$). The new variance decomposition reveals a big bump in PV:

$$39,190,224 = 9,122,852 + 30,067,371. \quad \begin{matrix} TV \\ PV \\ UV \end{matrix}$$

The previous PV was 5,062,030, and the new one is better at 9,122,852. Thus the subject effects get credit for $9,122,852 - 5,062,030 = 4,060,822$ units of total variation.

Interpreting the ANOVA table. As you've now seen, the analysis of variance really is just bookkeeping! The ANOVA table for the final model ($\text{Time} \sim \text{Clutter} + \text{Distance} + \text{Clutter:Distance} + \text{Subject}$) is shown in Table 4.3. The change in predictable variation at each stage gives us a more nuanced picture of the model, compared with simply quoting R^2 , because it allows us to partition credit among the individual predictor variables in the model.

The most intuitive way to summarize this information is to track the change in R^2 and residual standard deviation (s_e) at

Table 4.3: The analysis of variance (ANOVA) table for the model incorporating effects due to clutter, distance, and subject, along with an interaction between clutter and distance. In an ANOVA table, we add each variable in stages, one at a time. “# Pars” refers to the number of new parameters added to the model at each stage. ΔPV refers to the change in predictable variation at each stage. R^2 is the coefficient of determination for the model at each stage, and s_e is the residual standard deviation. Remember that R^2 always goes up when we add a variable, while s_e usually (but not always) goes down.

each step. For example, in Table 4.3, it's clear that accounting for subject-level variation improves our predictions the most, followed by clutter and then distance. The distance–clutter interaction contributes a small amount to the predictive ability of the model, relatively speaking: it improves R^2 by only half a percentage point. In fact, the distance/clutter interaction looks so negligible that we might even consider removing this effect from the model, just to simplify. We'll revisit this question later in the book, when we learn some more advanced tools for statistical hypothesis testing and predictive model building.

Finally, always remember that the construction of an ANOVA table is inherently sequential. For example, first we add the clutter variable, which remains in the model at every subsequent step; then we add the distance variable, which remains in the model at every subsequent step; and so forth. Thus the actual question being answered at each stage of an analysis of variance is: how much variation in the response can this new variable predict, in the context of what has already been predicted by other variables in the model? This point—the importance of context in interpreting an ANOVA table—is subtle, but important. We'll revisit it soon, when we discuss the issues posed by correlation among the predictor variables in a regression model.

Numerical and grouping variables together

Now we are ready to add a continuous predictor into the mix. Start with the simplest case of two predictors for each observation: a grouping variable $x_{i,1}$ that can take levels 0 to K , and a numerical predictor $x_{i,2}$. We start with the regression equation involving a set of K dummy variables, and add the effect of the continuous predictor onto the right-hand side of the regression equation:

$$y_i = \beta_0 + \beta_1^{(1)} \mathbf{1}_{\{x_{i1}=1\}} + \beta_1^{(2)} \mathbf{1}_{\{x_{i1}=2\}} + \cdots + \beta_1^{(K)} \mathbf{1}_{\{x_{i1}=K\}} + \beta_2 x_{i2} + e_i .$$

Now each group has its own regression equation:

$$\text{Regression equation for case where } (x_i = 0): \quad y_i = \beta_0 + \beta_2 x_{i2} + e_i$$

$$\text{Regression equation for case where } (x_i = k): \quad y_i = (\beta_0 + \beta_1^{(k)}) + \beta_2 x_{i2} + e_i .$$

Each line has a different intercept, but they all have the same slope. These are the red lines in Figure 4.3 back on page 80.

The coefficients $\beta_1^{(k)}$ are associated with the dummy variables that encode which college a student is in. Notice that only one of these dummy variables will be 1 for each person, and the rest will be zero, since a person is only in one college. Here's the regression output when we ask for a model of $\text{GPA} \sim \text{SAT.C} + \text{School}$:

Coefficients:

| | Estimate | Std. Error | t value | Pr(> t) |
|-----------------------|-----------|------------|---------|------------|
| (Intercept) | 1.678365 | 0.096062 | 17.472 | <2e-16 *** |
| SAT.C | 0.001343 | 0.000043 | 31.235 | <2e-16 *** |
| SchoolBUSINESS | 0.004676 | 0.078285 | 0.060 | 0.9524 |
| SchoolCOMMUNICATIONS | 0.092682 | 0.080817 | 1.147 | 0.2515 |
| SchooledUCATION | 0.048688 | 0.085520 | 0.569 | 0.5692 |
| SchoolENGINEERING | -0.195433 | 0.078460 | -2.491 | 0.0128 * |
| SchoolFINE ARTS | 0.012366 | 0.084427 | 0.146 | 0.8836 |
| SchoolLIBERAL ARTS | -0.134092 | 0.077629 | -1.727 | 0.0842 . |
| SchoolNATURAL SCIENCE | -0.150631 | 0.077908 | -1.933 | 0.0532 . |
| SchoolNURSING | 0.028273 | 0.102243 | 0.277 | 0.7822 |
| SchoolSOCIAL WORK | -0.035320 | 0.139128 | -0.254 | 0.7996 |

There is no dummy variable associated with Architecture, because it is the baseline case, against which the other colleges are compared. The regression coefficients associated with the "School" dummy variables then shift the line systematically up or down relative to the global intercept, but they do not change the slope of the line. As the math above shows, we are fitting a model where all colleges share a common slope, but have unique intercepts (11 parameters total). This is clearly a compromise solution between two extremes: fitting a single model, with one slope and one intercept common to all colleges (2 parameters); versus fitting ten distinct models for the ten individual colleges, each with their slope and intercept (20 parameters).

Interactions between grouping and numerical variables

We can also have modulating effects between numerical and grouping predictors. For example, we might expect that, for students in Liberal Arts, GPA's will vary more sharply with SAT Verbal scores, and less sharply with Math scores, than for students in Engineering. Mathematically, this means that College modulates the slope of the linear relationship between GPA and SAT scores.

If this is the case, then we should include an interaction term in the model. Remember, in statistical models, interactions are

formed by multiplying two predictors together—in this case, a numerical predictor and a dummy (0–1) variable. When the dummy variable is 0, the interaction term disappears. But when the dummy is 1, the interaction is equal to the original quantitative predictor, whose effective partial slope then changes.

Let's take a simple example involving baseball salaries, plotted in Figure 4.7 on page 97. On the y -axis are the log salaries of 142 baseball players. On the x -axis are their corresponding batting averages. The kind of mark indicates whether the player is in the Major League, AAA (the highest minor league), or AA (the next-highest minor league). The straight lines reflect the least-squares fit of a model that regresses log salary upon batting average and dummy variables for a player's league. The corresponding model equation looks like this:

$$\hat{y}_i = \beta_0 + \underbrace{\beta_1^{(AAA)} \cdot 1_{AAA} + \beta_1^{(MLB)} \cdot 1_{MLB}}_{\text{Dummy variables}} + \beta_1 \cdot AVG$$

The three lines are parallel: the coefficients on the dummy variables shift the line up or down as a function of a player's league.

But if we want the slope to change with league as well—that is, if we want league to modulate the relationship between salary and batting average—then we must fit a model like this:

$$\hat{y}_i = \beta_0 + \underbrace{\beta_1^{(AAA)} \cdot 1_{AAA} + \beta_1^{(MLB)} \cdot 1_{MLB}}_{\text{Dummy variables}} + \beta_2 \cdot AVG + \underbrace{\beta_3^{(AAA)} \cdot AVG \cdot 1_{AAA} + \beta_3^{(MLB)} \cdot AVG \cdot 1_{MLB}}_{\text{Interaction terms}}$$

The y variable depends on β_0 and β_2 for all players, regardless of league. But when a player is in AAA, the corresponding dummy variable (1_{AAA}) fires. Before, when a dummy variable fired, the entire line was merely shifted up for down (as in Figure 4.7). Now, an offset to the intercept ($\beta_1^{(AAA)}$) and an offset to slope ($\beta_3^{(AAA)}$) are activated. Ditto for players in the Major League: then the MLB dummy variable (1_{MLB}) fires, and both an offset to the intercept ($\beta_1^{(MLB)}$) and an offset to the slope ($\beta_3^{(MLB)}$) are activated:

$$\text{Regression equation for AA: } y_i = (\beta_0) + (\beta_2) \cdot AVG + e_i$$

$$\text{Regression equation for AAA: } y_i = (\beta_0 + \beta_1^{(AAA)}) + (\beta_2 + \beta_3^{(AAA)}) \cdot AVG + e_i$$

$$\text{Regression equation for MLB: } y_i = (\beta_0 + \beta_1^{(MLB)}) + (\beta_2 + \beta_3^{(MLB)}) \cdot AVG + e_i.$$

Fitting such model produces a picture like the one in Figure 4.8.

Without any interaction terms, the fitted model is:

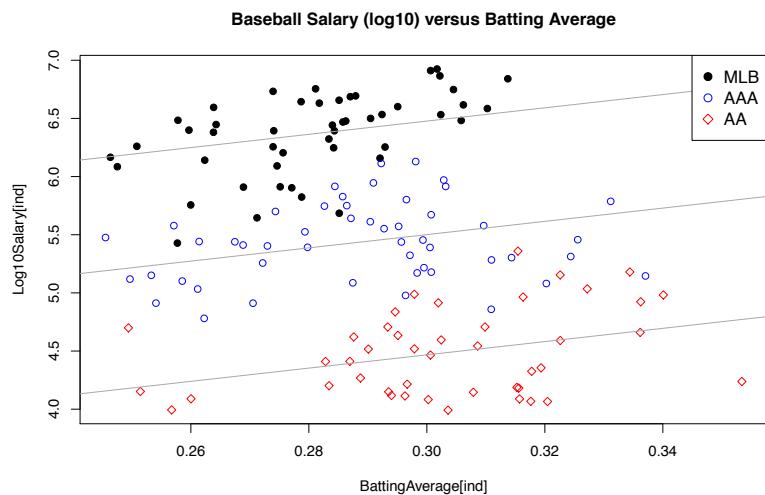


Figure 4.7: Baseball salaries versus batting average for Major League, AAA, and AA players.

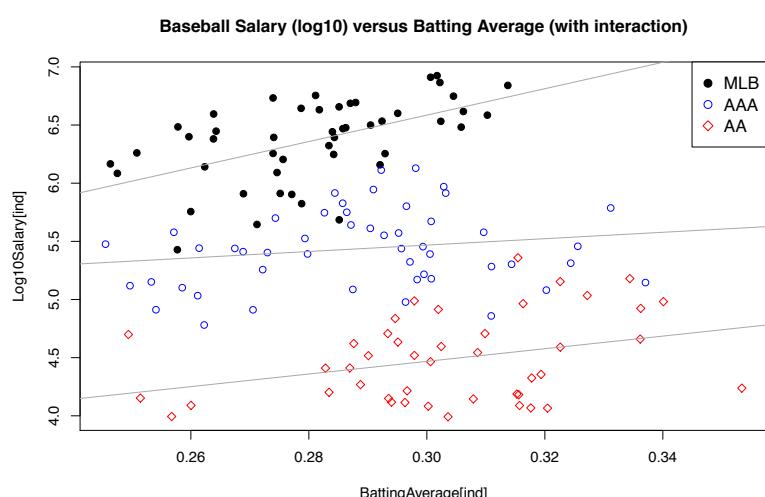


Figure 4.8: Baseball salaries versus batting average for Major League, AAA, and AA players. The fitted lines show the model with an interaction term between batting average and league.

```
          Estimate Std. Error t value Pr(>|t|)  
(Intercept) 2.75795  0.41893  6.583 8.88e-10 ***  
BattingAverage 5.69745  1.37000  4.159 5.59e-05 ***  
ClassAAA     1.03370  0.07166 14.426 < 2e-16 ***  
ClassMLB      2.00990  0.07603 26.436 < 2e-16 ***  
---  
Multiple R-squared: 0.845, Adjusted R-squared: 0.8416
```

With the interaction terms, we get:

```
          Estimate Std. Error t value Pr(>|t|)  
(Intercept) 2.8392   0.6718   4.227 4.33e-05 ***  
BattingAverage 5.4297   2.2067   2.461  0.0151 *  
ClassAAA      1.8024   0.9135   1.973  0.0505 .  
ClassMLB       0.3393   1.0450   0.325  0.7459  
BattingAverage:ClassAAA -2.6758   3.0724  -0.871  0.3853  
BattingAverage:ClassMLB  5.9258   3.6005   1.646  0.1021  
---  
Multiple R-squared: 0.8514, Adjusted R-squared: 0.846
```

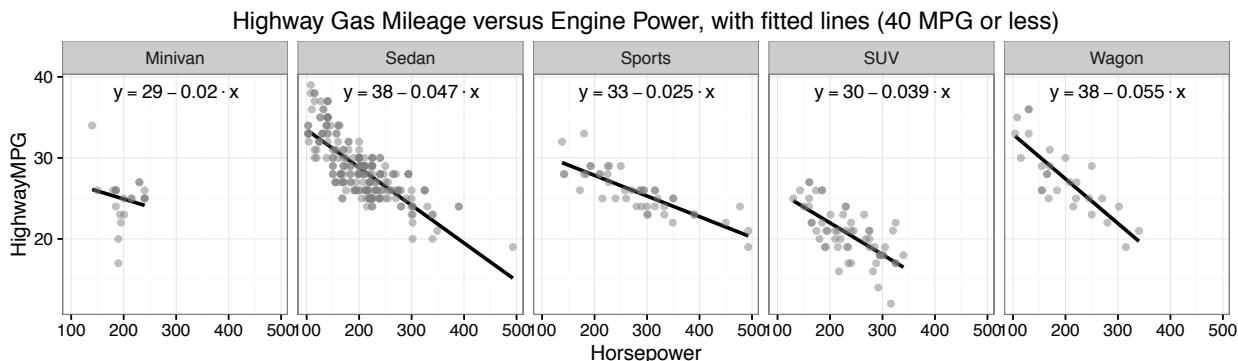


Figure 4.9: A model for the car-mileage data involving an interaction between class and horsepower. Here we've focused only on cars whose gas mileage is less than 40 miles per gallon. For this subset of the data, linearity looks like a reasonable, if imperfect, assumption.

Dependence among predictors

IN THIS section, we'll discuss the issue of how to interpret an analysis of variance for a model where the predictors themselves are correlated with each other. (Another term for correlation among predictors is *collinearity*.) This discussion will expand upon a point raised before—but only briefly—about the importance of context in the sequential construction of an ANOVA table.

Let's briefly review the analysis of variance (ANOVA). You'll recall that, in our look at the data on reaction time in video games, we ran an ANOVA (Table 4.3) of a regression model that predicted variation in human reaction time in terms of distance, visual clutter, subject-level variation, and a distance/clutter interaction. Our goal was to apportion credit among the individual parts of the model, where “credit” was measured by each variable’s contribution to the predictable variation in the model’s variance decomposition ($TV = PV + UV$). This led us, for example, to the conclusions that subject-level variation was large relative to the other effects, and that the distance/clutter interaction contributed only a modest amount to the predictive abilities of the model.

We can also run an analysis of variance on models containing numerical predictors. To see this in action, let's revisit the data on the gas mileage of cars from Figure 1.11, back on page 27. Recall that this data set involved 387 cars and three variables: gas mileage, engine horsepower, and vehicle class (minivan, sedan, sports car, SUV, or wagon). We can see this data once more in Figure 4.9, which shows a lattice plot of mileage versus horsepower, stratified by vehicle class.

In our earlier discussion of this data, we noted two facts:

- (1) The classes exhibit systematic differences in their typical mileages. For example, sedans have better gas mileage, on average, than SUVs or minivans.
- (2) Vehicle class seems to modulate the relationship between MPG and engine power. As engine power increases, mileage gets worse on average, regardless of vehicle class. But this drop-off is steeper for wagons than for sports cars.

Previously, we described these facts only informally. But we now have the right tools—dummy variables and interactions—that allow us to quantify them in the context of a regression model. Specifically: point (1) suggests that we need class-level dummy variables, to move the intercepts up and down as appropriate for each class; while point (2) suggests that we need an interaction between class and horsepower, to make the slope of the regression line get steeper or shallower as appropriate for each class. Using our informal notation from earlier, our regression model should look like this:

$$\text{MPG} \sim \text{Horsepower} + \text{Class} + \text{Class: Horsepower}.$$

Upon fitting this model by least squares, we get the coefficients in Table 4.4, at right. The corresponding fitted lines within each class are also shown in Figure 4.9. The parameters of this fitted model confirm our earlier informal observations based on the lattice plot: that both the average mileage and the steepness of the mileage/horsepower relationship are affected by vehicle class.

An analysis of variance table for this model looks like this.

| Variable added | # Pars | R^2 | ΔR^2 | s_e | Δs_e |
|------------------|--------|-------|--------------|-------|--------------|
| Intercept only | 1 | 0 | | 4.59 | |
| Horsepower | 1 | 0.426 | 0.426 | 3.48 | 1.11 |
| Class | 4 | 0.725 | 0.299 | 2.42 | 1.06 |
| Horsepower:Class | 4 | 0.743 | 0.018 | 2.36 | 0.07 |

According to this table, we can attribute most of the credit for predicting fuel economy to the horsepower variable ($\Delta R^2 = 0.426$). Most of the remaining credit goes to vehicle class ($\Delta R^2 = 0.299$). The interaction produces a modest change in R^2 ; this bears out the

Table 4.4: Fitted coefficients (rounded to the nearest hundredth) for the model that predicts car gas mileage in terms of engine horsepower, vehicle class, and a class/horsepower interaction.

| Variable | $\hat{\beta}$ |
|-------------------|---------------|
| Intercept | 28.86 |
| Horsepower | -0.02 |
| Sedan | 9.28 |
| Sports | 4.08 |
| SUV | 0.94 |
| Wagon | 9.55 |
| Horsepower:Sedan | -0.03 |
| Horsepower:Sports | -0.01 |
| Horsepower:SUV | -0.02 |
| Horsepower:Wagon | -0.04 |

Table 4.5: An analysis of variance (ANOVA) table for the model that predicts highway gas mileage in terms of a car's engine power and vehicle class, including both main effects and an interaction term. In this ANOVA table, the horsepower variable has been added first, followed by vehicle class.

visual impression conveyed by Figure 4.9, in which the slopes in each panel are clearly different, but not dramatically so.

But this conclusion about the relative importance of horsepower and vehicle class involves a major, even deal-breaking, caveat. Remember that an analysis of variance is inherently sequential: first we add the horsepower variable, then we add vehicle class, and then we add the interaction, tracking the variance decomposition at each stage. What happens if we build an ANOVA table by adding vehicle class before we add horsepower?

| Variable added | # Pars | R^2 | ΔR^2 | s_e | Δs_e |
|------------------|--------|-------|--------------|-------|--------------|
| Intercept only | 1 | 0 | | 4.59 | |
| Class | 4 | 0.397 | 0.397 | 3.58 | 1.01 |
| Horsepower | 1 | 0.725 | 0.328 | 2.42 | 1.16 |
| Class:Horsepower | 4 | 0.743 | 0.018 | 2.36 | 0.07 |

Now we reach the opposite conclusion: that vehicle class contributes more ($\Delta R^2 = .397$) to the predictable variation than does horsepower ($\Delta R^2 = .328$). Why does this happen? How could our conclusion about the relative importance of the variables depend upon something so arbitrary as the order in which we decide to add them?

Shared versus unique information

Figure 4.10 provides some intuition why this is so. In our data on gas mileage, the two predictors (horsepower and vehicle class) are correlated with each other: vehicles in certain classes, like SUVs and sports cars, have more powerful engines on average than sedans, wagons, and minivans.

To understand why this correlation between predictors would matter so much in an analysis of variance, let's consider the information provided by each variable. First, a vehicle's class tells us at least two important things relevant for predicting gas mileage.

- 1) *Weight*: for example, SUVs tend to be heavier than sedans, and heavier vehicles will get poorer gas mileage.
- 2) *Aerodynamics*: for example, minivans tend to be boxier than sports cars, and boxier cars will get poorer gas mileage due to increased drag at highway speeds.

Similarly, the horsepower of a vehicle's engine also tells us at

Table 4.6: A second analysis of variance (ANOVA) table for the model that predicts highway gas mileage in terms of a car's engine power and vehicle class, including both main effects and an interaction term. In this ANOVA table, vehicle class has been added first, followed by horsepower.

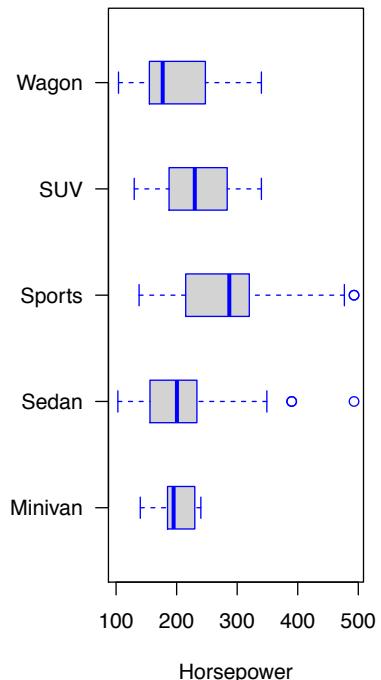


Figure 4.10: Correlation between vehicle class and horsepower.

least two important things relevant for predicting gas mileage.

- 1) *Weight*: more powerful engines are themselves heavier, and tend to come in cars that are heavier in other ways, too.
- 2) *Fuel consumption*: a smaller engine consumes less fuel and typically has better mileage than a bigger engine.

Notice that both variables provide information about a vehicle's weight; let's call this the shared information. But each also provides information on something else specific to that variable; let's call this the unique information. The shared information between the predictors manifests itself as correlation: bigger cars tend to have both bigger engines, and they also tend to be in certain classes. We can use a Venn diagram to represent both the shared and the unique information provided by the predictors in a stylized (i.e. non-mathematical) way:

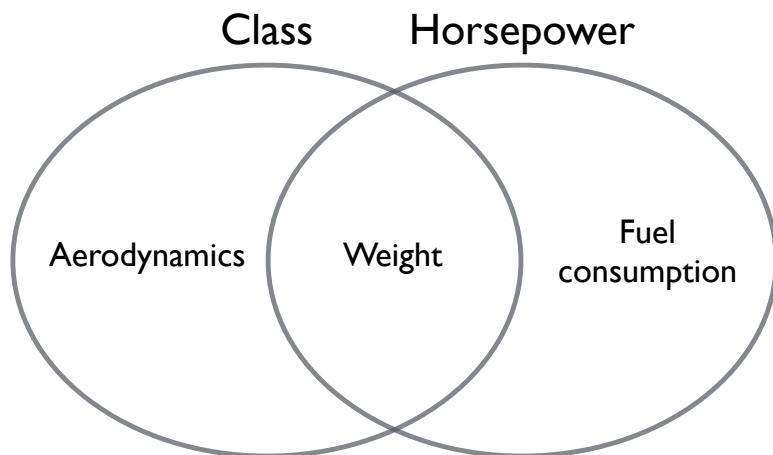


Figure 4.11: The two predictors in the gas-mileage data set provide some information content that is shared between them, in addition to some information that is unique to each one.

In the first analysis of variance (Table 4.5), we added horsepower first. When we did so, the regression model greedily used all the information it could from this predictor, including both the “shared” and “unique” information. As a result, when we added the class variable second, the shared information is redundant—it was already accounted for by the model. We therefore end up giving the class variable credit only for its unique information content; all the information content it shares with horsepower was already counted in step 1. This is illustrated in Figure 4.12.

But when we flip things around and add vehicle class to the

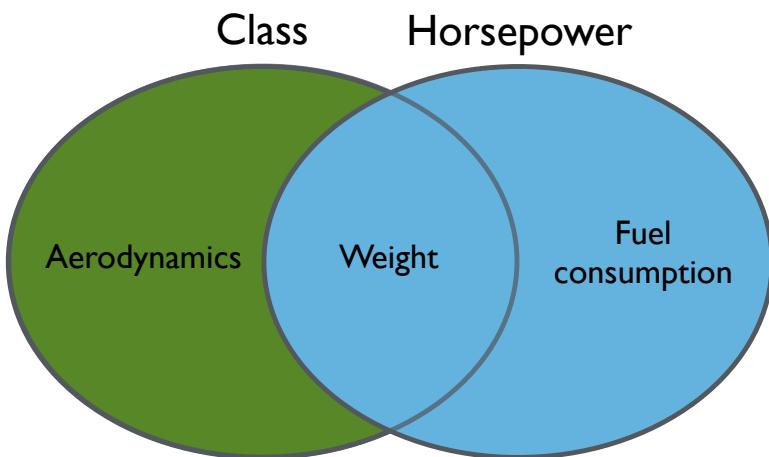


Figure 4.12: Our model for gas mileage includes two variables: engine horsepower and vehicle class. These variables both convey information about a vehicle's size, in addition to some unique information (e.g. class tells us about aerodynamics, while horsepower tells us about fuel consumption). When we add the Horsepower variable first in an analysis of variance (Table 4.5), we attribute all of the shared information content to Horsepower, and none to Vehicle class, in our ANOVA table.

model first (Table 4.6), this picture changes. We end up giving the class variable credit both for its unique information content *and* for the information it shares with Horsepower. This leaves less overall credit for Horsepower when we add it in step 2 of the ANOVA. This is illustrated in Figure 4.13.

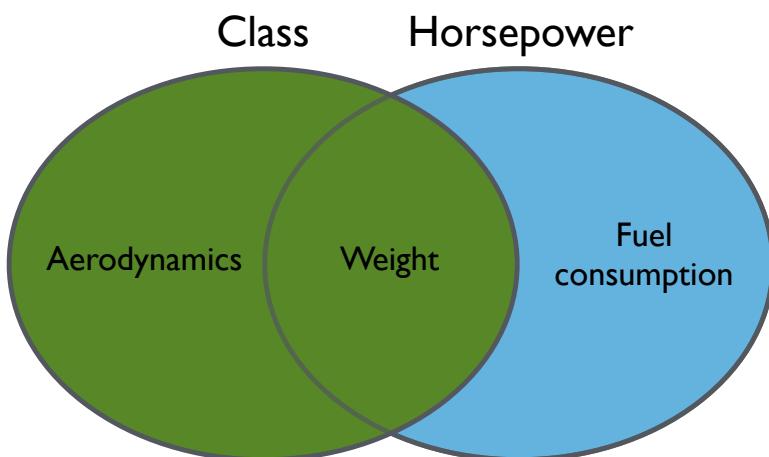


Figure 4.13: (Continued from Figure 4.12.) But when we add the Class variable first in an analysis of variance (Table 4.5), we attribute all of the shared information content to Class, and none to Horsepower, in our ANOVA table.

This example highlights an unsatisfying but true feature of the analysis of variance: when the variables are correlated, *their ordering matters* when you build the ANOVA table.

This feature of an ANOVA table at first seems counterintuitive, even disturbing. Yet similar phenomena occur all the time in everyday life. A good analogy here is the dessert buffet at Thanksgiving dinner. Imagine two different versions of dessert.

Version 1: After dinner, your aunt offers you apple pie, and you eat your fill. The apple pie is delicious—you were really looking forward to something sweet after a big Thanksgiving meal. It makes you very happy.

Next, after you've eaten your fill of apple pie, your aunt offers you pumpkin pie. Pumpkin pie is also delicious—you love it just as much as apple. But your dessert tummy is pretty full already. You eat a few bites, and you enjoy it; that spicy pumpkin flavor is a little different to what you get from an apple pie. But of course, pumpkin pie is still a dessert, and you don't enjoy it as much as you might have if you hadn't eaten so much apple pie first.

Version 2: After dinner, your aunt offers you pumpkin pie, and you eat your fill. The pumpkin pie is delicious—all that whipped cream on top goes so well with the nutmeg and earthy pumpkin flavor. It makes you very happy.

Next, after you've eaten your fill of pumpkin pie, your aunt offers you apple pie. Apple pie is also delicious—you love it just as much as pumpkin. But your dessert tummy is pretty full already. You eat a few bites, and you enjoy it; those tart apples with all the cloves and cinnamon give a little different flavor to what you get from a pumpkin pie. But apple pie is still a dessert, and you don't enjoy it as much as you might have if you hadn't eaten so much pumpkin pie first.

That evening, which pie are you going to remember? In version 1, you'll attribute most of your Thanksgiving dessert afterglow to the apple pie; while in version 2, you'll attribute most of it to pumpkin pie. *Context matters*, even if in the abstract you like both pies the same amount.

An analysis of variance is like the one-at-a-time dessert eater at Thanksgiving. Whatever variable we add to the model first, the model greedily eats its fill of that, before turning to the second variable. This affects how credit gets attributed. In our ANOVA tables for the gas mileage data, our two variables (horsepower and vehicle class) are like apple and pumpkin pie. Yes, they each offer

something unique, but they also share a lot of their information content (just like the pies are both desserts). Because of this, the order in which they are added to the ANOVA table—or equivalently, the context in which each variable's marginal contribution to the model is evaluated—matters a lot.

The moral of the story is that it rarely makes sense to speak of “the” ANOVA table for a model—only “an” ANOVA table. Thus there is no unique way to partition credit among multiple variables for their shared information content in a regression model. We must make an arbitrary choice, and in an ANOVA table, that choice is “winner take all” to the first variable added to the model.

Final thoughts on ANOVA. There are two further points to bear in mind about the analysis of variance. First, the ANOVA table is not the model itself, only an attempt to partition credit for predicting the outcome among the variables in the model by adding those variables one at a time. And while the ANOVA table is order-dependent, the model itself isn't. Regardless of the order in which you add variables, you will always get the same model coefficients, fitted values, and residuals at the end.

Second, we've discussed the subtleties of interpreting an ANOVA table in the presence of correlation among the predictors. However, if the variables in the model are independent of one another, then they have no shared information content, and the ANOVA table does not depend upon the ordering of the variables.

This is why we ignored the issue of variable ordering when building an ANOVA table for our model of reaction time in video games versus distance, clutter, and subject-level effects. For that data set, the predictor variables were independent with each other: the experimental design was perfectly balanced, with each subject sitting for exactly 40 trials for each pairwise combination of the cluttered and distance variables. Regardless of the order in which we add the variables, we will always get the same ΔPV for each one. Thus in the absence of dependence among the predictors, we can uniquely assign credit for predicting the outcome to each one.⁷

Regression models, just like Thanksgiving guests, thrive on variety—that is, on multiple independent sources of information.

⁷ For this reason, ANOVA is a commonly used tool in the analysis of designed experiments, when we can ensure that the predictors are independent of one another. It is less common in the analysis of observational studies, where the inevitable presence of collinearity significantly weakens the conclusions that we can draw from an ANOVA.

5

Quantifying uncertainty using the bootstrap

Quantifying parameter uncertainty

IN COMING this far through the book, you've already learned many valuable skills: how to summarize evidence both graphically and numerically; how to fit basic group-wise and linear statistical models to data; how to combine grouping and numerical variables; and how to use these models to explore trends and predict new outcomes.

But we're missing a crucial piece of the puzzle. Earlier we defined statistical modeling as the structured quantification of uncertainty. We've focused a lot so far on the "structure" part; now we'll begin to focus on the "uncertainty" part.

A question that almost always arises in statistical modeling is: how confident are we in our estimate of an effect size? Take the following study of a new therapeutic regime for esophageal cancer, from the New England Journal of Medicine in 2006:

We randomly assigned patients with resectable adenocarcinoma of the stomach, esophagogastric junction, or lower esophagus to either perioperative chemotherapy and surgery (250 patients) or surgery alone (253 patients). . . . With a median follow-up of four years, 149 patients in the perioperative-chemotherapy group and 170 in the surgery group had died. As compared with the surgery group, the perioperative-chemotherapy group had a higher likelihood of overall survival (five-year survival rate, 36 percent vs. 23 percent).¹

Thus the chemotherapy regime appears to save lives: the relative risk of survival under chemo is 36/23, or about 1.6. But 1.6 plus-or-minus what? What if the physicians running the trial had enrolled a different sample of patients? Might the relative risk have looked more like 1.3 (a smaller effect) or even 1.0 (no effect)? Chemotherapy has nasty side effects and is very expensive. If you're a cancer patient or a Medicare administrator, uncertainty about the effect size matters.

¹ Cunningham, et. al. "Perioperative chemotherapy versus surgery alone for resectable gastroesophageal cancer." *New England Journal of Medicine*, 2006 July 6; 355(1):11-20.

We use the phrase *statistical inference* to describe the framework and procedures we use to address uncertainty in statistical models. In this chapter, we'll approach statistical inference using a technique called the bootstrap.

Sampling distributions, estimators, and alternate universes

In fitting statistical models, we typically equate the trustworthiness of a procedure with its stability under the influence of luck, and we seek to measure the degree to which that procedure might have given a different answer if the forces of randomness had made the world look a bit different. Specifically, the question we seek to answer is: “if our data set had been different merely due to chance, would our answer have been different, too?”

$$\begin{array}{ccc} \text{Confidence in} & \iff & \text{Stability of those estimates} \\ \text{your estimates} & & \text{under the influence of chance} \end{array}$$

You can see why it makes sense to equate stability with trustworthiness if you imagine a suspect who gives the police three different answers to the question, “Where were you last Tuesday night?” If the story keeps changing, there is little basis for trust.

Sources of instability. One obvious source of instability in our estimates is when our observations are subject to random forces. For example, suppose we wish to characterize the relationship between SAT score and graduating GPA for the entering class of 2000 at the University of Texas. Figure 5.1 shows the entire relevant population, yet there is still randomness to worry about—for, as the teacher in Ecclesiastes puts it, “time and chance happeneth to them all.” If any of these 5,191 students had taken the SAT on a different day, or eaten a healthier breakfast on the day of their chemistry finals, we would be looking at a slightly different data set, and thus a slightly different least-squares line—even if the underlying SAT–GPA relationship had stayed the same.

Another source of instability is the effect of sampling variability, which arises when we're unable to study the entire population of interest. The key insight here is that a different sample would have led to different estimates of the model parameters. Consider the example above, about the study of a new chemotherapy regime for esophageal cancer. If doctors had taken a different sample of

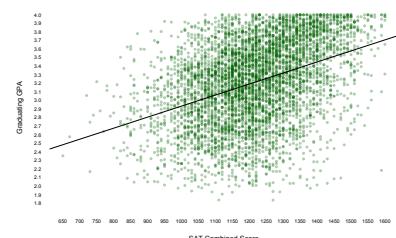
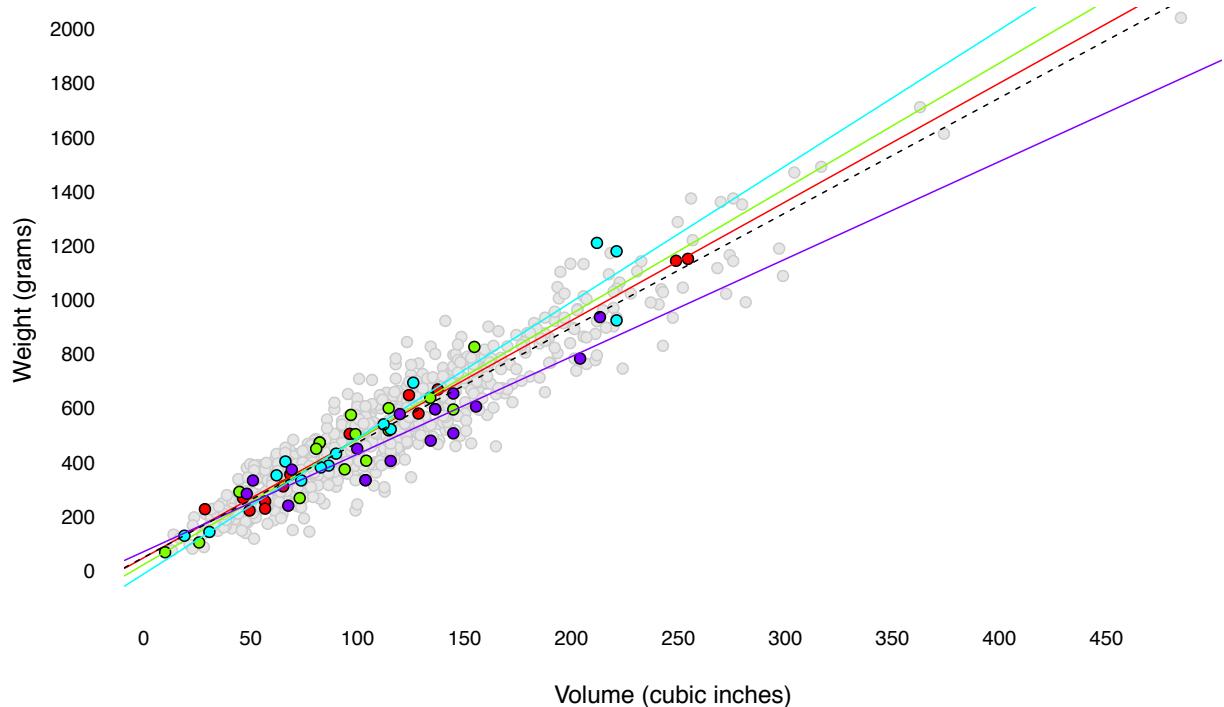


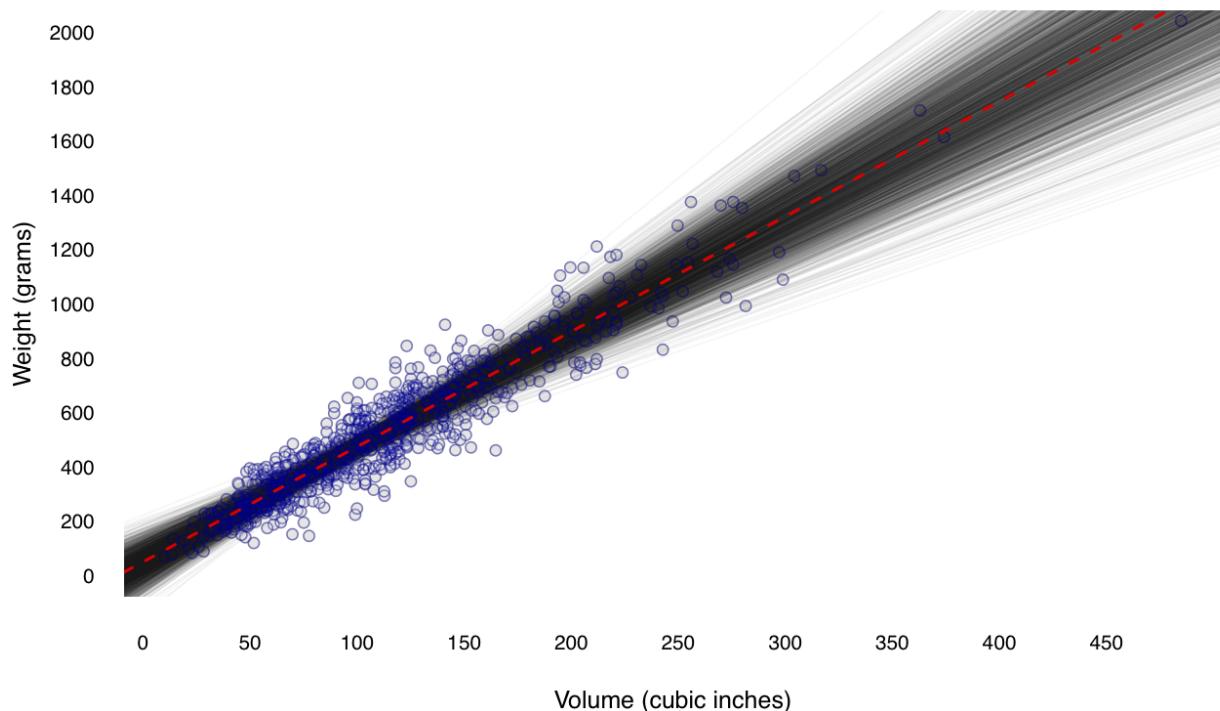
Figure 5.1: Graduating GPA versus high-school SAT score for all students who entered UT–Austin in the fall of 2000 and went on to earn a bachelor's degree within 6 years. The black line shows the least-squares fit.



503 cancer patients and gotten a drastically different estimate of the new treatment's effect, then the original estimate isn't very trustworthy. If, on the other hand, pretty much any sample of 503 patients would have led to the same estimates, then their answer for *this particular* subset of 503 is likely to be accurate.

An example: simulating a sampling distribution by Monte Carlo. To get some intuition for this way of thinking, imagine that you go on a four-day fishing trip to a lovely small lake out the woods. The lake is home to a population of 800 fish of varying size and weight, depicted in Figure 5.2. On each day, you take a random sample from this population—that is, you catch (and subsequently release) 15 fish, recording the weight of each one, along with its length, height, and width (which multiply together to give a rough estimate of volume). You then use the day's catch to compute a different estimate of the volume–weight relationship for the entire population of fish in the lake. These four different days—and the four different least-squares fits—show up in different colors in Figure 5.2.

Figure 5.2: Four different days of fishing, coded by color, on an imaginary lake home to a population of 800 fish. On each day's fishing trip, you catch 15 fish, and end up estimating a slightly different weight–volume relationship. The dashed black line is the true relationship for the entire population.

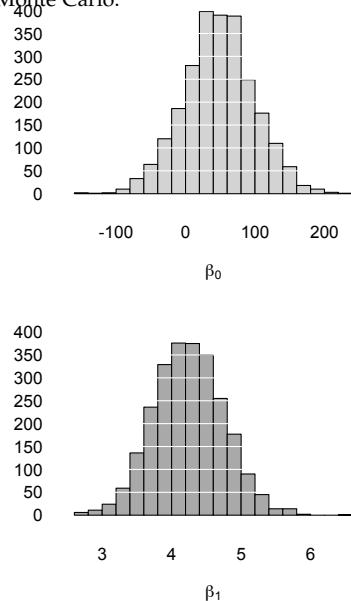


Four days of fishing give us some idea of how the estimates for β_0 and β_1 vary from sample to sample. But 2500 days of fishing, simulated by computer, give us a better idea. Figure 5.3 shows just this: 2500 different samples of size 15 from the population, together with 2500 different least-squares estimates of the weight-volume relationship. This is an example of a *Monte Carlo simulation*, in which we run a computer program to repeatedly simulate a random process (in this case, sampling from a population).

These pictures show the *sampling distribution* of the least-squares line—that is, how the estimates for β_0 and β_1 change from sample to sample, shown in histograms in the right margin. In theory, to know the sampling distributions exactly, we'd need to take an infinite number of samples, but 2500 gives us a rough idea.

The sampling distribution. To understand the concept of a sampling distribution, it helps to distinguish between an *estimator* and an *estimate*. A good analogy here is that an estimator is to a court trial as an estimate is to a verdict. Just like a trial is a procedure for reaching a verdict about guilt or innocence, an estimator is

Figure 5.3: 2500 days of fishing, together with the 2500 different estimates of β_0 and β_1 (below), simulated by Monte Carlo.



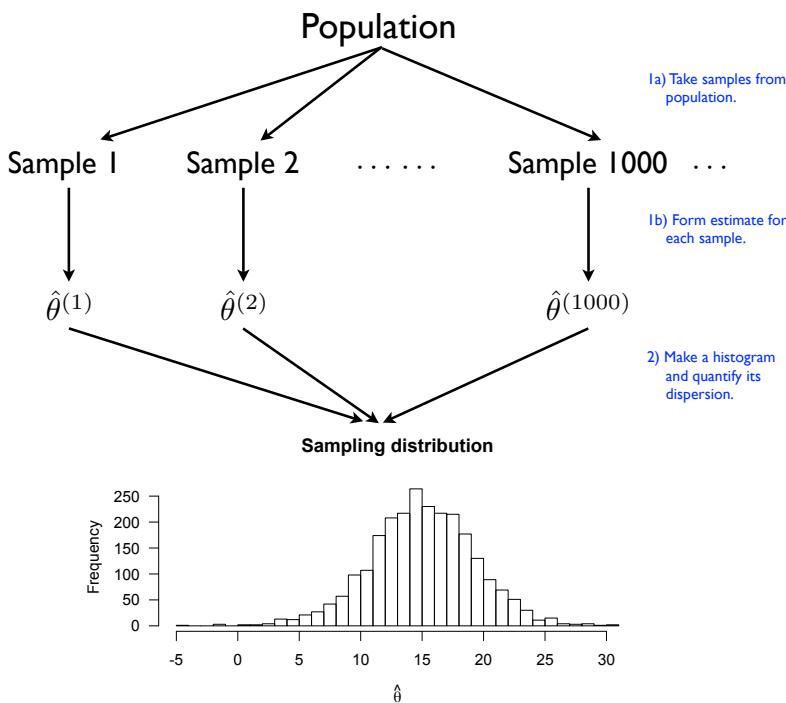


Figure 5.4: A stylized depiction of a sampling distribution of an estimator $\hat{\theta}$. To construct this distribution, we must imagine the following thought experiment. We repeatedly take many samples (say, 1000) from the population (step 1a). For each sample, we apply our estimator to compute the estimate $\hat{\theta}^{(r)}$ (step 1b). At the end, we combine all the estimates $\hat{\theta}^{(1)}, \dots, \hat{\theta}^{(1000)}$ into a histogram, and we summarize the dispersion of that histogram (step 2). Technically, the sampling distribution is the distribution of estimates we'd get with an infinite number of samples, and the histogram is an approximation of this distribution. The difference between the true distribution and the approximation generated by Monte Carlo is called *Monte Carlo error*.

a procedure for reaching an estimate of some population-level quantity on the basis of a sample. The least-squares procedure is a specific set of steps (i.e. equations) that one applies to a data set. The procedure yields estimators $\hat{\beta}_0$ and $\hat{\beta}_1$ for the slope and intercept of a population-wide linear trend; while the values of $\hat{\beta}_0$ and $\hat{\beta}_1$ you get for a specific data set are the estimates. An estimator's sampling distribution is the distribution of results (that is, the estimates) that one obtains from that estimator under repeated sampling from a population. Figure 5.4 shows graphically how, in principle, this distribution is constructed. Concrete examples of an estimator include the sample mean, the least squares procedure, and the residual standard deviation.

Good estimators are those that usually yield estimates close to the truth, with minimal variation. Therefore, we typically summarize a sampling distribution using its standard deviation, which we refer to as the *standard error*.² In quoting the standard error of an estimator's sampling distribution, you are saying: "If I were to take repeated samples from the population and use this estimator for every sample, my estimate is typically off from the truth by

² We are also sometimes interested in the mean of a sampling distribution. If the mean of an estimator's sampling distribution is equal to the true population value, we say that the estimator is *unbiased*. This term has a precise mathematical meaning, but also an unwarranted connotation of universal desirability that many statisticians find problematic. Alas, for historical reasons, we're basically stuck with the term. It turns out that unbiasedness is not always a good property of an estimator. There can be very good reasons to use estimators that we know to be biased. But that's for another book.

about this much." Notice again that this is a claim about a procedure, not a particular estimate. The bigger the standard error, the less stable the estimator across different samples, and the less you can trust the estimate for any particular sample. To give a specific example, for the 2500 samples in Figure 5.3, the standard error of $\hat{\beta}_0$ is about 50, while the standard error of $\hat{\beta}_1$ is about 0.5.

Of course, if you really could take repeated samples from the population, life would be easy. You could simply peer into all of those alternate universes, tap each version of yourself on the shoulder, and ask, "What slope and intercept did you get for *your* sample?" By tallying up these estimates and seeing how much they differed from one another, you could discover precisely how much confidence you should place in your own estimates of β_0 and β_1 , and report appropriate error bars based on the standard error of your estimator.³

Most of the time, however, we're stuck with one sample, and one version of reality. We cannot know the actual sampling distribution of our estimator, for the same reason that we cannot peer into all those other lives we might have lived, but didn't:

Two roads diverged in a yellow wood,
And sorry I could not travel both
And be one traveler, long I stood
And looked down one as far as I could
To where it bent in the undergrowth. . . .⁴

³ Let's ignore the obvious fact that, if you had access to all those alternate universes, you'd also have more data. The presence of sample-to-sample variability is the important thing to focus on here.

⁴ Robert Frost, *The Road Not Taken*, 1916.

Quantifying our uncertainty would seem to require knowing all the roads not taken—an impossible task.

Surprisingly, we can come close to performing the impossible. There are two ways of feasibly constructing something like the histogram in Figure 5.4, thereby approximating an estimator's sampling distribution without ever taking repeated samples from the population.

- 1) *Resampling*: that is, by pretending that the sample itself is the population, which allows one to approximate the effect of sampling variability by resampling from the sample.
- 2) *Parametric probability modeling*: that is, by assuming that the forces of randomness obey certain mathematical regularities, and by drawing conclusions about these regularities using probability theory.

In this chapter, we'll discuss the resampling approach, deferring the probability-modeling approach to a later chapter.

Bootstrapping: standard errors through resampling

AT THE core of the resampling approach to statistical inference lies a simple idea. Most of the time, we can't feasibly take repeated samples of size n from the population, to see how our estimate changes from one sample to the next. But we can repeatedly take samples of size n from the sample itself, and apply our estimator afresh to each notional sample. The idea is that the variability of the estimates across all these samples can be used to approximate our estimator's true sampling distribution.

This process—pretending that our sample is the whole population, and taking repeated samples of size n with replacement from our original sample of size n —is called *bootstrap resampling*, or just *bootstrapping*.⁵ Each block of n resampled data points is called a bootstrapped sample. To bootstrap, we write a computer program that repeatedly resamples our original sample and recomputes our estimate for each bootstrapped sample. Modern software makes a non-issue of the calculational tedium involved.

You may be puzzled by something here. There are n data points in the original sample. If we repeatedly resample n data points from our “pseudo-population” of size n , won’t each bootstrapped sample be identical to the original sample? If so, and every bootstrapped sample looks the same, then how can this process be used to simulate sampling variability?

This fact highlights a key requirement of bootstrapping: the resampling must be done *with replacement* from the original sample, so that each bootstrapped sample contains duplicates and omissions from the original sample.⁶ These duplicates and omissions induce variation from one bootstrapped sample to the next, mimicking the variation you’d expect to see across the real repeated samples that you can’t take.

To summarize, let’s say we have a data set D , consisting of n cases. We want to understand how our estimator $\hat{\theta}$ might have behaved differently with a different sample of size n . To answer this question using bootstrapping, we follow two main steps.

(1) Repeat the following substeps many times (e.g. 1000 or more):

- a. Generate a new bootstrapped sample $D^{(r)}$ by taking n samples with replacement from D .
- b. Apply the estimator $\hat{\theta}$ to the bootstrapped sample $D^{(r)}$ and save the resulting estimate, $\hat{\theta}^{(r)}$.

⁵ The term “bootstrapping” is a metaphor. It is an old-fashioned phrase that means performing a complex task starting from very limited resources. Imagine trying to climb over a tall fence. If you don’t have a rope, just “pull yourself up by your own bootstraps.”

⁶ Imagine a lottery drawing, where there’s a big urn with 60 numbered balls in it. We want to choose a random sample of 6 numbers from the urn. After we choose a ball, we could do one of two things: 1) put the ball to the side, or 2) record the number on the ball and then throw it back into the urn. If you set the ball aside, it can be selected only once; this is sampling without replacement, and it’s what happens in a real lottery. But if instead you put the ball back into the urn, it has a chance of being selected more than once in the final sample; this is sampling with replacement, and it’s what we do when we bootstrap.

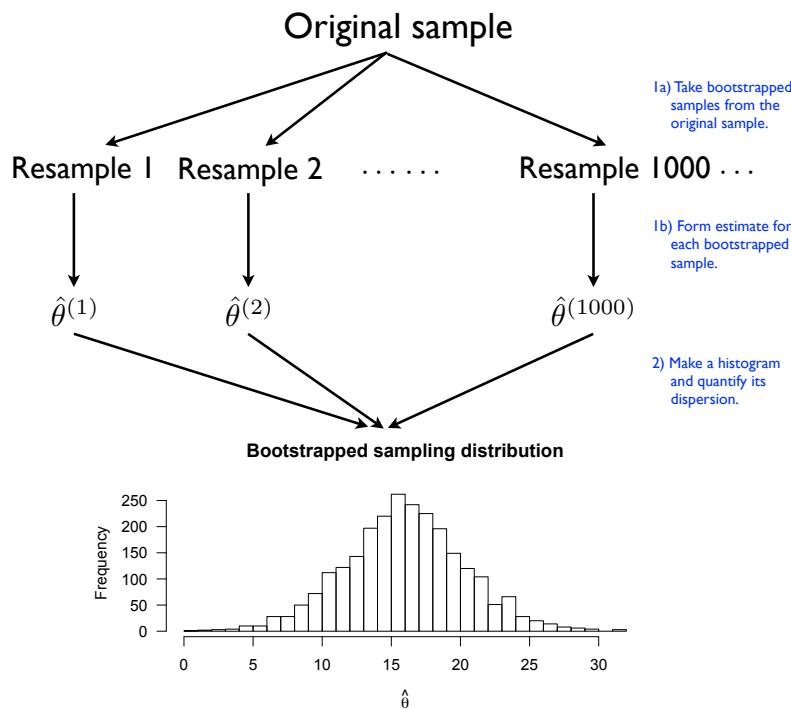


Figure 5.5: A stylized depiction of a bootstrapped sampling distribution of an estimator $\hat{\theta}$. We have a single original sample. We repeatedly take many bootstrapped samples (say, 1000) from the original sample (step 1a). For each resample, we compute the estimator $\hat{\theta}$ (step 1b). At the end, we combine all the estimates $\hat{\theta}^{(1)}, \dots, \hat{\theta}^{(1000)}$ into a histogram of the bootstrapped sampling distribution, and we summarize the dispersion of that histogram (step 2). Compare with Figure 5.4.

- (2) Take all of the $\hat{\theta}^{(r)}$'s you've generated and make a histogram.
This is your estimate of the sampling distribution.

See Figure 5.5, and compare with Figure 5.4.

Resampling won't yield the true sampling distribution of an estimator, but it is often good enough for approximating the standard error (which you'll remember is just the standard deviation of the sampling distribution). We use the term *bootstrapped standard error* for the standard deviation of the bootstrapped sampling distribution. The bootstrapped standard error is an estimate of the true standard error.

The quality of this estimate depends almost entirely on one thing: how closely the original sample resembles the wider population. This is a question of judgment best answered by someone with subject-area expertise relevant to the data set at hand. As a data analyst this often isn't under your control, and therefore it's almost worth remembering that the bootstrap is not entirely free of assumptions. You can't magic your way to sensible estimates of the true sampling distribution by bootstrapping a biased, woefully small, or otherwise poor sample.

The quality of the Monte Carlo approximation also depends to a lesser extent on how many bootstrapped samples you take from the original sample. Simulating more bootstrapped samples help to reduce the variability inherent in any Monte Carlo simulation—up to a point. But taking more bootstrapped samples is never a substitute for having more actual samples in the real data set. Fundamentally, it is the size of your original sample that governs the precision of your estimates.

A natural question is: how well does bootstrapping work in practice? To see the procedure in action, let's reconsider the least-squares estimator of the slope (β_1) for the weight–volume line describing the fish in our hypothetical lake. The top row of Figure 5.6 shows three actual sampling distributions, corresponding to samples of size $n = 15$, $n = 50$, and $n = 100$ from the entire population. These were constructed using the Monte Carlo method described several pages ago, as depicted in Figures 5.3 and 5.4. For example, the top left panel (for $n = 15$) was constructed by taking 2,500 Monte Carlo samples from the true population in Figure 5.3, and computing the least-squares estimate of the slope for each sample as in Figure 5.4.

Below each true sampling distribution, we have focused on four of these 2500 samples. For each of these real samples, we ran the bootstrapping procedure by 2500 bootstrapped samples from the original sample of size n , treating it as a pseudo-population. For each bootstrapped sample, we compute the least-squares line for weight versus volume. These 2500 estimates of β_1 are what you see in each grey-colored panel of Figure 5.6. For example, the first grey panel in column 1 corresponds to the bootstrapped sampling distribution from the first sample of size 15; the second grey panel corresponds to the bootstrapped sampling distribution from the second sample of size 15; and so on for the rest of the grey panels.

If bootstrapping were perfect, each grey panel would look exactly like the corresponding orange panel above, regardless of the same size. But of course, bootstrapping isn't perfect. If you study these pictures closely, you'll notice a few things.

- (1) The bootstrapped sampling distribution can differ substantially from one original sample to the next (top to bottom). The sample-to-sample differences are larger when the original sample size is small.
- (2) The bootstrapped sampling distribution gets both closer to the truth, and less variable from one original sample

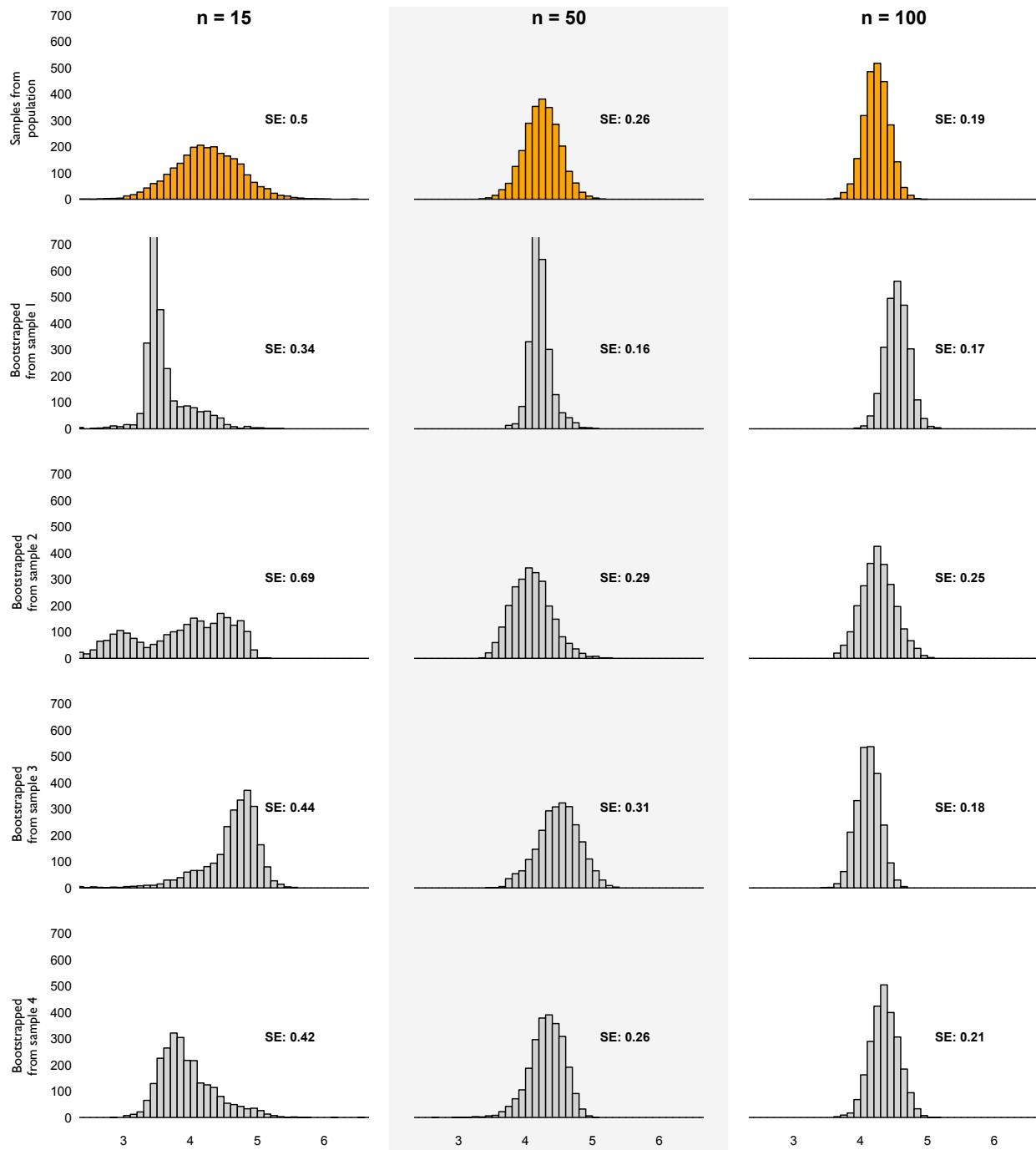


Figure 5.6: Actual (top, in orange) and bootstrapped sampling distributions (four replications) for the least-squares estimator of β_1 from Figure 5.2.

- to the next, as the original sample size gets larger.
- (3) The bootstrapped standard errors (printed next to each histogram) are often closer to the true standard error than you might naïvely expect, based on the visual correspondence of the bootstrapped sampling distribution to the true one.

Confidence intervals and coverage

Now that we've learned to approximate an estimator's sampling distribution via bootstrapping, what do we do with this information? The answer is: we quantify the uncertainty of our estimate via a *confidence interval*: a range of plausible values for the true value of a parameter, together with an associated *confidence level* between 0% and 100%. The width of a confidence interval conveys the precision with which the data have allowed you to estimate the underlying population parameter. If your interval actually contains the true population value, we say that the interval *covers* the truth. If it doesn't, the interval *fails to cover* the truth. In real life, you won't know whether your interval covers. The confidence level expresses how confident you are that it actually does.

There are many ways of generating confidence intervals from bootstrapped sampling distributions, ranging from the simple to the highly sophisticated (and mathematically daunting). We'll focus on two simple ways here, with the understanding that the more technical ways we don't discuss are a bit more accurate.⁷

First, there's the basic standard-error method. Here, you quote a symmetric error bar centered on the estimate from the original sample, plus-or-minus some multiple k of the bootstrapped standard error. To be precise, let's say that θ is some population parameter you're trying to estimate; that $\hat{\theta}$ is the estimate of θ generated by your actual sample; and that you've run the bootstrapping procedure on your sample and found that the bootstrapped standard error is $\hat{\sigma}$. Your confidence interval would then be

$$\theta \in \hat{\theta} \pm t^* \hat{\sigma},$$

where t^* is a chosen multiple. This number t^* is called the *critical value*. It is the number of standard errors you must go out from the center to capture a certain percentage of the sampling distribution. Typical values are $t^* = 1$ (for an approximate 68% confidence interval) and $t^* = 2$ (for an approximate 95% confidence interval).

⁷ If you want to get an introduction to the more technical ways of getting confidence intervals from the bootstrap, see the following article: "Bootstrap confidence intervals: when, which, what? A practical guide for medical statisticians." James Carpenter and John Bithell. *Statistics in Medicine* 2000; 19:1141–64.

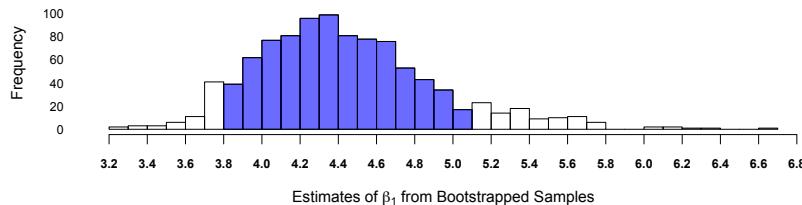


Figure 5.7: The estimated sampling distribution of $\hat{\beta}_1$ that arises from bootstrapping one sample of size 30 from the full fish population. The blue area reflects an 80% confidence interval generated by the coverage method, with symmetric tail areas of 10% above and 10% below the blue area.

The answer to the question of *why* $t^* = 1$ corresponds to 68% and $t^* = 2$ to 95% is beyond the scope of this chapter. It has to do with the normal distribution and something called the central limit theorem. For now, it is fine if you accept this is an empirical rule of thumb that statisticians have found gives a good approximation in situations where your bootstrapped sampling distribution looks approximately bell-shaped. Some of the more sophisticated bootstrap techniques, mentioned in Footnote 7, are focused on improving the choice of t^* given by these simple guidelines.

Second, there's the coverage-interval method, in which you simply calculate a coverage interval using the quantiles of your bootstrapped sampling distribution. For example, Figure 5.7 shows the bootstrapped sampling distribution for the slope of the weight–volume relationship arising from a single sample of 30 fish from the same lake as before. If you wanted to compute an 80% confidence interval based on this data, you would calculate the 10th and 90th percentiles of this histogram, giving you an interval that contains 80% of the bootstrapped estimates of the slope. In Figure 5.7, this interval is (3.8, 5.1), shown in blue. This example highlights that, unlike the intervals generated by the standard-error method, the intervals generated by the coverage method need not be symmetric about the estimate $\hat{\theta}$ derived from your actual sample.

Is one of these two methods better? Not as a general rule. The coverage-interval approach is more common in practice, and it's a fine default option. The most conservative thing to do, assuming you don't want to go the very technical⁸ route, is to compute both and report the wider interval.

What does “confidence” mean?

The word “confidence,” as it is used in the phrase “confidence interval,” has a notoriously tricky interpretation. To put it con-

⁸ See Footnote 7.

cisely but opaqely, confidence intervals are intervals generated by a method that satisfies the frequentist coverage principle.

The frequentist coverage principle: If you were to analyze one data set after another for the rest of your life, and you were to quote X% confidence intervals for every estimate you made, those intervals should cover their corresponding true values at least X% of the time. Here X can be any number between 0 and 100.

Let's unpack this a bit. Imagine that your interval was generated with a procedure that, under repeated use on one sample after the next, tends to yield intervals that cover the true value with a relative frequency of at least 80%. Then, and only then, may you claim a bona fide 80% confidence level for your specific interval. (You may, of course, aim for whatever coverage level you wish in lieu of 80%. Many people seem stuck on 95%, but it's entirely your choice.) Thus confidence intervals involve something of a bait-and-switch: they purport to answer a question about an individual interval, but instead give you information about some hypothetical assembly line that could be used to generate a whole batch of intervals. Nonetheless, there is an appealing "truth in advertising" property at play here: that if you're going to claim 80% confidence, you should be right 80% of the time over the long run.

An obvious question is: do bootstrapped confidence intervals satisfy the frequentist coverage property? If your sample is fairly representative of the population, then the answer is a qualified yes. That is, the bootstrapping procedure yields nominal X% intervals that cover the true value "approximately" X% of the time. Moreover, as the size of the original sample gets bigger, the quality of the approximation gets better. Alas, it is necessary to appeal to some very advanced probability theory to put both of these claims on firm footing. (This is best deferred to another, much more advanced book. For those that like fancy math, the relevant branch of probability theory is called empirical-process theory, which part of a wider area called stochastic processes.)

For our purposes, it is better to show the procedure in action. Figure 5.8, for example, depicts the results of running 100,000 regressions—1,000 bootstrapped samples for each of 100 different real samples from the population in Figure 5.2. The vertical black line shows the true population value of the weight–volume slope ($\beta_1 = 4.24$) for our population of fish. Each row corresponds to a different actual sample of size $n = 30$ from the population. Dots

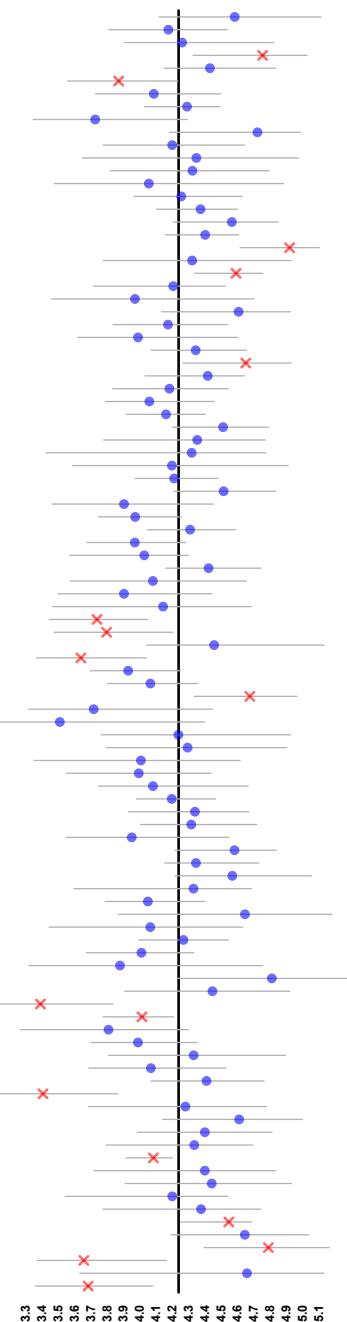


Figure 5.8: 100 different samples of size 30 from the population in Figure 5.2, along with each least-squares estimate of the weight–volume slope, and an 80% bootstrapped confidence interval, just like that at the top left. Blue dots show confidence intervals that cover; red crosses show those that don't.

and crosses indicate the least-squares estimate of the slope arising from that sample, while the grey bars show the corresponding 80% bootstrapped confidence intervals generated by the coverage method (just like the blue region in Figure 5.7).

The nominal confidence level of 80% for each individual interval must be construed as a claim about the *whole ensemble* of 100 intervals: 80% should cover, 20% shouldn't. In fact, 83 of these intervals cover and 17 don't, so the claim is approximately correct.

Gaussian versus bootstrapped confidence intervals

Most statistical software packages have built-in routines for calculating standard errors and confidence intervals, and will show them as part of a routine summary output for a regression model. For example, in R, the `summary` and `confint` functions do just this.

Chances are, however, that the package you use is *not* using the bootstrap to calculate these confidence intervals. So what is it doing instead? The full answer to this question turns out to be rather long and drawn-out, and we'll return to it in a later chapter. But we can give a quick summary here.

The short answer is that your statistical software is calculating *Gaussian* standard errors and confidence intervals, which are based on the assumption that the residuals in the regression model follow a Gaussian, or normal, distribution:

$$\begin{aligned} y_i &= \beta_0 + \beta_1 x_i + e_i \\ e_i &\sim N(0, \sigma^2). \end{aligned} \quad (5.1)$$

The first equation is familiar: observation = fitted value + residual. But the second equation is new. It invokes an assumption that we never needed to make before: that the residuals e_i arise from a normal distribution with mean 0 and variance σ^2 . In fact, this assumption long predicated the use of the bootstrap to calculate confidence intervals in regression modeling, and it is embedded in most statistical software today. Gaussian standard errors are sometimes numerically similar to bootstrapped standard errors, but they are not calculated in the same way.

There are three obvious questions that arise in conjunction with this assumption.

- (1) Huh? How does the assumption of normally distributed residuals let us calculate standard errors and confidence intervals?

- (2) This seems useless and kind of goofy. Why bother with this assumption? That is, under what circumstances would we use this assumption to calculate confidence intervals and standard errors, as opposed to the bootstrapping technique that we've already learned?
- (3) OK, fine. But how do we check whether the assumption of normally distributed residuals is satisfied for some particular data set?

Here are some very brief answers to these three questions.

- (1) *How does this even work?* Using probability theory, it is possible to mathematically derive formulas for standard errors and confidence intervals, based on the assumption of normally distributed residuals. The math, which exploits the nice properties of the normal distribution, isn't actually hard. But you do have to know a bit of probability theory to understand it. Moreover, the math is tedious, with lots of algebra; and it's just not that important, in the sense that it will add little to your conceptual understanding of regression. So we'll skip the math for now, and trust that our software has implemented it correctly. If you're really interested, turn to the chapter on the normal linear regression model, later in the book.
- (2) *Why bother with this assumption?* There are several possible answers here. The simplest one, and the one we'll go with for now, is that the Gaussian standard errors are often a good approximation to the bootstrapped standard errors—assuming the normality assumption is met (see point 2, above). Moreover, the Gaussian standard errors take our software a lot less time to calculate, because they don't require us to resample the data set and refit the model thousands of times. So if your data set is very large and bootstrapping would take a prohibitively long time—or even if bootstrapping is just giving you strange software bugs—then the Gaussian standard errors and confidence intervals might be your next-best option.
- (3) *How can we check the normality assumption?* Just make a histogram of your residuals. If they look like a normal distribution, then the normality assumption is probably reasonable. If they don't, then you should stick with bootstrapped standard errors if you can. For example, Figure 5.9 shows three examples of regression models, together with a histogram of the

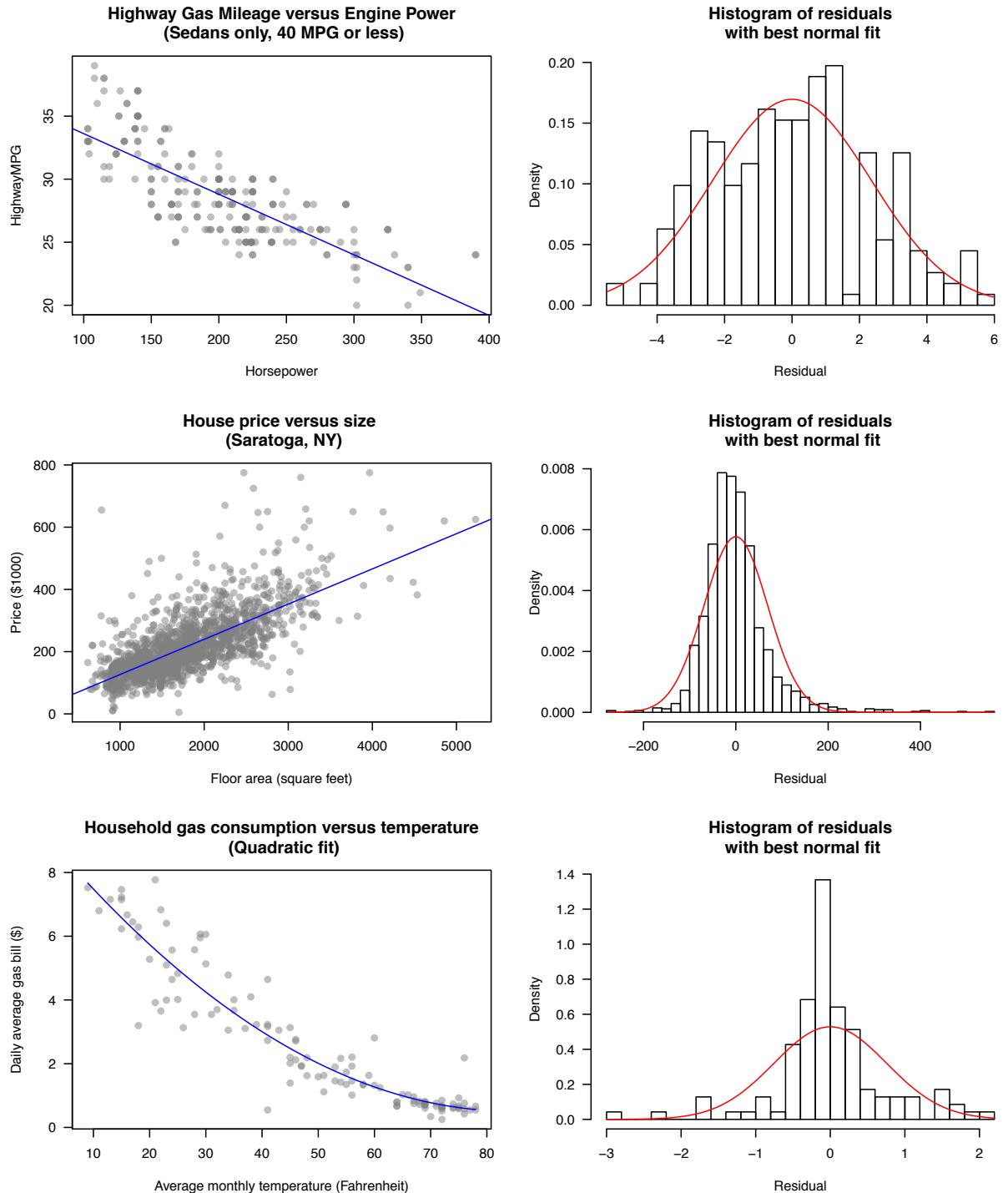


Figure 5.9: Three examples of regression models (left column), together with the best-fitting normal approximation to the histogram of each model's residuals (right column).

residuals. The top panel looks approximately normal, while the middle and bottom panels obviously don't. As a result, for the data sets in the middle and bottom panels, we can't necessarily trust the Gaussian confidence intervals; they may be a case of "garbage in, garbage out."⁹

We'll elaborate on these much more in a later chapter. For the time being, it's fine to think of the confidence intervals returned by regression software as just an approximation to the bootstrapped confidence intervals you've become familiar with.

⁹ This is an oversimplification. Even if the residuals don't look Gaussian, the Gaussian confidence intervals can still be approximately correct, because of something called the central limit theorem. But this topic is for a much more advanced treatment of regression analysis.

Bootstrapped prediction intervals (advanced topic)

Recall the problem of forecasting a future y^* corresponding to some predictor x^* , using past data as a guide. (For example, how much should a used truck with 80,000 miles cost? How much can an Austin restaurant with a food rating of 7.5 charge for a meal?) Previously, we were content to quote a prediction interval of the form

$$\hat{y}^* \in \hat{\beta}_0 + \hat{\beta}_1 x^* \pm s_e,$$

or the best guess, plus-or-minus one residual standard deviation. (We could, if we wish, also go out two residual standard deviations to get a wider interval that covered more of the data.)

These prediction intervals are good enough for most purposes. However, when we introduced them, we point that they were a bit naïve, because of how they ignore uncertainty in our estimates for β_0 and β_1 . For example, imagine that you work for a major metropolitan newspaper with a daily (Monday–Friday) circulation of 200,000 newspapers, and that your employer is contemplating a new weekend edition. You could certainly use the data in Figure 5.10, which correlates Sunday circulation with daily circulation for 34 major metropolitan newspapers, to inform your guess about the new Sunday edition's likely circulation. But the available data don't pin down β_0 and β_1 for sure; we have some uncertainty about the true values for these parameters. The kind of basic or naïve prediction interval that we've constructed until now will mask these sources of uncertainty, which may be large.

Luckily, now that we understand the logic of the bootstrap, we can try to account for this extra uncertainty. Suppose we have some value of the predictor x^* , and we want to form a prediction interval for the corresponding value of the response, y^* . The

idea is to break down our uncertainty about y^* into its constituent parts—uncertainty due to lack of perfect knowledge about the parameters, and uncertainty about the residual. The key equation is that $y^* = \hat{y}^* + e^*$, or future data point = point estimate + residual. We will use bootstrapping to approximate the uncertainty in each of these two terms individually.

To do so, we repeat the following steps a few thousand times.

- (1) Take a single bootstrapped sample from the original sample, and compute the least-squares estimates $\hat{\beta}_0^{(r)}$ and $\hat{\beta}_1^{(r)}$. This gives you your best guess for the future y^* , given the information in the bootstrapped sample:

$$\hat{y}^{(r)} = \hat{\beta}_0^{(r)} + \hat{\beta}_1^{(r)} x^*.$$

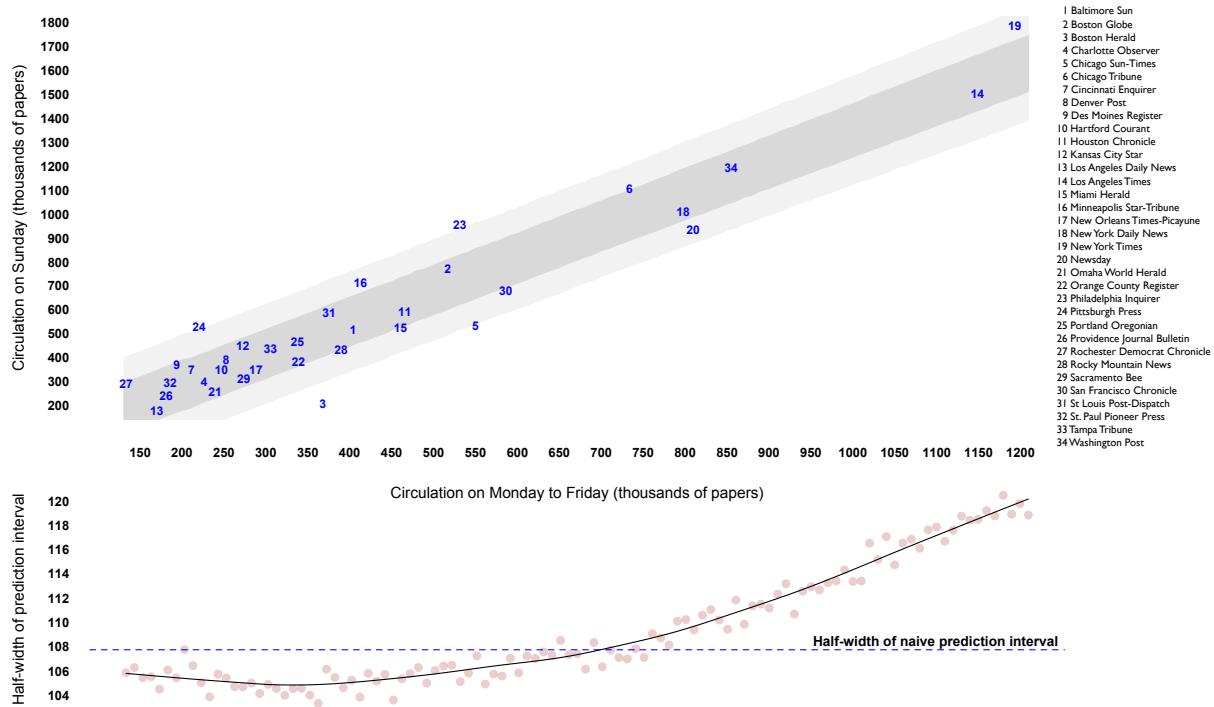
Here the superscript r denotes the r^{th} bootstrap sample.

- (2) Sample a residual $e^{(r)}$ at random from the bootstrapped least-squares fit, to mimic the unpredictable variation in the model.
- (3) Set $y^{(r)} = \hat{y}^{(r)} + e^{(r)}$. This is your notional “future y ” for the r^{th} bootstrapped sample.

In step 1, we simulate the uncertainty in \hat{y} by using different parameter estimates $\hat{\beta}_0^{(r)}$ and $\hat{\beta}_1^{(r)}$ each time through the three-step loop. In step 2, we simulate the uncertainty in e , the future residual, by resampling the residuals from the model fit in step 1. Finally, in step 3, we combine these two sources of uncertainty to form the notional future data point, $y^{(r)} = \hat{y}^{(r)} + e^{(r)}$.

By repeating this process many thousands of times, we can build up a distribution of values for y^* . If you take the standard deviation of all those $y^{(r)}$'s, you can directly quantify the uncertainty in your prediction corresponding to x^* —for example, by quoting the dark- and light-grey prediction intervals in Figure 5.10, which stretch to one and two standard deviations (respectively) on either side of the least-squares line.

One noticeable feature of the bootstrapped prediction intervals is the way they bend outwards as they get further away from the center of the sample. This is a bit hard to see in the top panel of Figure 5.10. To show this effect more clearly, the bottom panel explicitly plots the half-width of the dark grey bootstrapped prediction intervals at 109 different hypothetical X points: every increment of 10,000 newspapers across the entire range of daily circulation, from 130,000 to 1.2 million.



The black curve shows an unmistakeable trend. Prediction uncertainty increases when you move away from the mean of X . Figure 5.3, several pages earlier, will give you some intuition for why this is so: small differences in the slope get magnified when you move further away from the middle of the sample. The naïve prediction interval fails to capture this effect entirely. On this problem, for example, the naïve interval understates prediction uncertainty by 10,000 newspapers or more for large values of X .

A final point worth noting: all of the previous warnings about bootstrapped standard errors also apply to bootstrapped prediction intervals. If the observed data is unrepresentative of the population, bootstrapping will mislead rather than inform.

Figure 5.10: Sunday circulation versus daily circulation for 34 major metropolitan newspapers, together with one- and two-standard-deviation bootstrapped prediction intervals across the range of the X variable (top panel). Also shown is the half-width of the darker-grey prediction interval across the range of X (bottom panel), versus the half-width of the naïve prediction interval, shown by the dotted blue line.

You'll notice that the pink dots marking the half-width of each bootstrapped prediction interval wiggle up and down a bit from the black curve. This happens because we only took 2,500 bootstrap samples, which produces a bit of unwanted noise. Taking more bootstrapped samples would make the pink points fall closer to the black curve, but it wouldn't shift the black curve up or down.

6

Multiple regression: the basics

From lines to planes

LINEAR regression, as we've learned, is a powerful tool for finding patterns in data. So far, we've only considered models that involve a single numerical predictor, together with as many grouping variables as we want. These grouping variables were allowed to modulate the intercept, or both the slope and intercept, of the underlying relationship between the numerical predictor (like SAT score) and the response (like GPA). This allowed us to fit different lines to different groups, all within the context of a single regression equation.

In this chapter, we learn how to build more complex models that incorporate two or more numerical predictors. For example, consider the data in Figure 6.1 on page 128, which shows the highway gas mileage versus engine displacement (in liters) and weight (in pounds) for 59 different sport-utility vehicles.¹ The data points in the first panel are arranged in a three-dimensional point cloud, where the three coordinates (x_{i1}, x_{i2}, y_i) for vehicle i are:

- x_{i1} , engine displacement, increasing from left to right.
- x_{i2} , weight, increasing from foreground to background.
- y_i , highway gas mileage, increasing from bottom to top.

Since it can be hard to show a 3D cloud of points on a 2D page, a color scale has been added to encode the height of each point in the y direction.

Fitting a linear equation for y versus x_1 and x_2 results in a regression model of the following form:

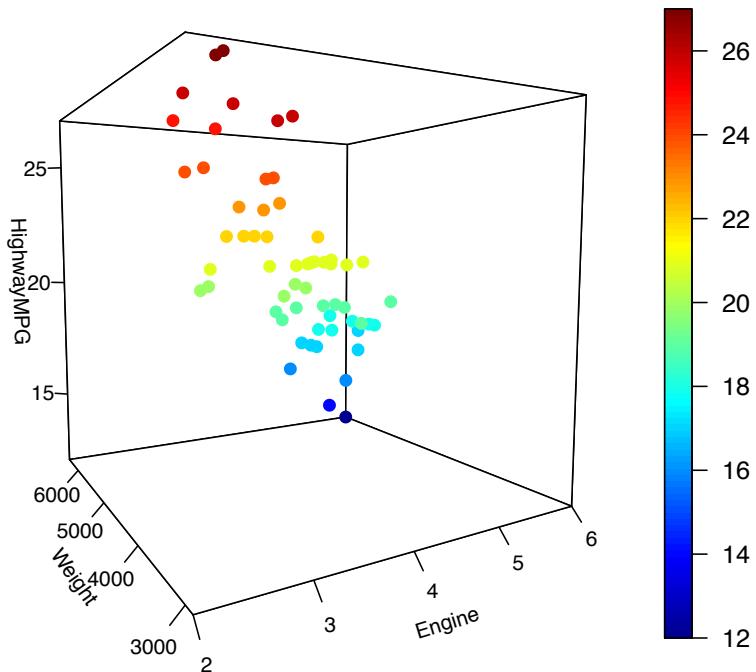
$$y_i = \beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} + e_i .$$

Just as before, we call the β 's the coefficients of the model and the e_i 's the residuals. In Figure 6.1, this fitted equation is

$$\text{MPG} = 33 - 1.35 \cdot \text{Displacement} - 0.00164 \cdot \text{Weight} + \text{Residual} .$$

¹ These are the same SUVs shown in the second-from-right panel in Figure 4.9, when we discussed ANOVA for models involving correlated predictors.

Mileage versus weight and engine power



With fitted plane

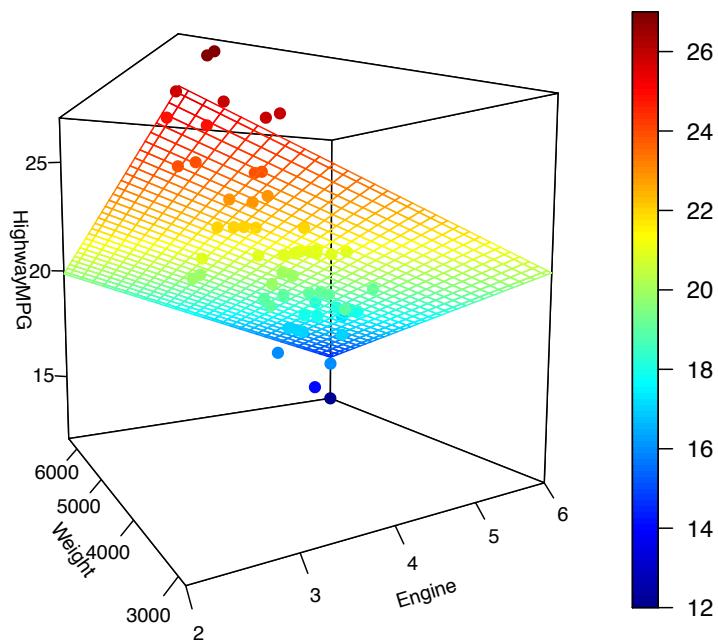


Figure 6.1: Highway gas mileage versus weight and engine displacement for 59 SUVs, with the least-squares fit shown in the bottom panel.

Both coefficients are negative, showing that gas mileage gets worse with increasing weight and engine displacement.

This equation is called a *multiple regression model*. In geometric terms, it describes a plane passing through a three-dimensional cloud of points, which we can see slicing roughly through the middle of the points in the bottom panel in Figure 6.1. This plane has a similar interpretation as the line did in a simple one-dimensional linear regression. If you read off the height of the plane along the y axis, then you know where the response variable is expected to be, on average, for a particular pair of values (x_1, x_2) .

In more than two dimensions. In principle, there's no reason to stop at two predictors. We can easily generalize this idea to fit regression equations using p different predictors $\mathbf{x}_i = (x_{i,1}, x_{i,2}, \dots, x_{i,p})$:

$$\hat{y}_i = \beta_0 + \beta_1 x_{i,1} + \beta_2 x_{i,2} + \cdots + \beta_p x_{i,p} = \beta_0 + \sum_{k=1}^p \beta_k x_{i,k}.$$

This is the equation of a p -dimensional plane embedded in $(p+1)$ -dimensional space. This plane is nearly impossible to visualize beyond $p = 2$, but straightforward to describe mathematically.

From simple to multiple regression: what stays the same. In this jump from the familiar (straight lines in two dimensions) to the foreign (planes in arbitrary dimensions), it helps to start out by cataloguing several important features that don't change.

First, we still fit parameters of the model using the principle of least squares. As before, we will denote our estimates by $\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2$, and so on. For a given choice of these coefficients, and a given point in predictor space, the fitted value of y is

$$\hat{y}_i = \hat{\beta}_0 + \hat{\beta}_1 x_{i,1} + \hat{\beta}_2 x_{i,2} + \cdots + \hat{\beta}_p x_{i,p}.$$

This is a scalar quantity, even though the regression parameters describe a p -dimensional hyperplane. Therefore, we can define the residual sum of squares in the same way as before, as the sum of squared differences between fitted and observed values:

$$\sum_{i=1}^n e_i^2 = \sum_{i=1}^n (y_i - \hat{y}_i)^2 = \sum_{i=1}^n \left\{ y_i - (\hat{\beta}_0 + \hat{\beta}_1 x_{i,1} + \hat{\beta}_2 x_{i,2} + \cdots + \hat{\beta}_p x_{i,p}) \right\}^2.$$

The principle of least squares prescribes that we should choose the estimates so as to make the residual sum of squares as small as possible, thereby distributing the “misses” among the observations

We use a bolded \mathbf{x}_i as shorthand to denote the whole vector of predictor values for observation i . That way we don't have to write out $(x_{i,1}, x_{i,2}, \dots, x_{i,p})$ every time. When writing things out by hand, a little arrow can be used instead, since you obviously can't write things in bold: $\vec{x}_i = (x_{i,1}, x_{i,2}, \dots, x_{i,p})$. By the same logic, we also write $\vec{\beta}$ for the vector $(\beta_0, \beta_1, \dots, \beta_p)$.

in a roughly equal fashion. Just as before, the little e_i is the amount by which the fitted plane misses the actual observation y_i .

Second, these residuals still have the same interpretation as before: as the part of y that is unexplained by the predictors. For a least-squares fit, the residuals will be uncorrelated with each of the original predictors. Thus we can interpret $e_i = y_i - \hat{y}_i$ as a statistically adjusted quantity: the y variable, adjusted for the systematic relationship between y and all of the x 's in the regression equation. Here, as before, statistical adjustment just means subtraction.

Third, we still summarize preciseness of fit using R^2 , which has the same definition as before:

$$R^2 = 1 - \frac{\sum_{i=1}^n (y_i - \hat{y}_i)^2}{\sum_{i=1}^n (y_i - \bar{y})^2} = 1 - \frac{UV}{TV} = \frac{PV}{TV}.$$

The only difference is that \hat{y}_i is now a function of more than just an intercept and a single slope. Also, just as before, it will still be the case R^2 is the square of the correlation coefficient between y_i and \hat{y}_i . It will not, however, be expressible as the correlation between y and any of the original predictors, since we now have more than one predictor to account for. (Indeed, R^2 is a natural generalization of Pearson's r for measuring correlation between one response and a whole basket of predictors.)

Finally, we still estimate the residual standard deviation using the same formula as before:

$$s_e = \sqrt{\frac{1}{n-p} \sum_{i=1}^n (y_i - \hat{y}_i)^2}.$$

One slightly tricky thing to keep in mind is that p refers to the number of free parameters in the model. So in the model for mileage versus engine size and weight, we have $p = 3$: an intercept (β_0), an engine-size coefficient (β_1) and a weight coefficient (β_2). Your regression software should keep track of this for you.

Multiple regression and partial relationships

NOT everything about our inferential process stays the same when we move from lines to planes. We will focus more on some of the differences later, but for now, we'll mention a major one: the interpretation of each β coefficient is no longer quite so simple as the interpretation of the slope in one-variable linear regression.

The best way to think of $\hat{\beta}_k$ is as an estimated *partial slope*: that is, the change in y associated with a one-unit change in x_k , holding all other variables constant. This is a subtle interpretation that is worth considering at length. To understand it, it helps to isolate the contribution of x_k on the right-hand side of the regression equation. For example, suppose we have two numerical predictors, and we want to interpret the coefficient associated with x_2 . Our equation is

$$\underbrace{y_i}_{\text{Response}} = \beta_0 + \underbrace{\beta_1 x_{i1}}_{\text{Effect of } x_1} + \underbrace{\beta_2 x_{i2}}_{\text{Effect of } x_2} + \underbrace{e_i}_{\text{Residual}}.$$

To interpret the effect of the x_2 variable, we isolate that part of the equation on the right-hand side, by subtracting the contribution of x_1 from both sides:

$$\underbrace{y_i - \beta_1 x_{i1}}_{\text{Response, adjusted for } x_1} = \underbrace{\beta_0 + \beta_2 x_{i2}}_{\text{Regression on } x_2} + \underbrace{e_i}_{\text{Residual}}.$$

On the left-hand side, we have something familiar from one-variable linear regression: the y variable, adjusted for the effect of x_1 . If it weren't for the x_2 variable, this would just be the residual in a one-variable regression model. Thus we might call this term a *partial residual*.

On the right-hand side we also have something familiar: an ordinary one-dimensional regression equation with x_2 as a predictor. We know how to interpret this as well: the slope of a linear regression quantifies the change of the left-hand side that we expect to see with a one-unit change in the predictor (here, x_2). But here the left-hand side isn't y ; it is y , adjusted for x_1 . We therefore conclude that β_2 is the change in y , *once we adjust for the changes in y due to x_1* , that we expect to see with a one-unit change in the x_2 variable.

This same line of reasoning can allow us to interpret β_1 as well:

$$\underbrace{y_i - \beta_2 x_{i2}}_{\text{Response, adjusted for } x_2} = \underbrace{\beta_0 + \beta_1 x_{i1}}_{\text{Regression on } x_1} + \underbrace{e_i}_{\text{Residual}}.$$

Thus β_1 is the change in y , *once we adjust for the changes in y due to x_2* , that we expect to see with a one-unit change in the x_1 variable.

We can make the same argument in any multiple regression model involving two or more predictors, which we recall takes the form

$$y_i = \beta_0 + \sum_{k=1}^p \beta_k x_{i,k} + e_i.$$

To interpret the coefficient on the j th predictor, we isolate it on the right-hand side:

$$\underbrace{y_i - \sum_{k \neq j} \beta_k x_{i,k}}_{\text{Response adjusted for all other } x\text{'s}} = \underbrace{\beta_0 + \beta_j x_{ij}}_{\text{Regression on } x_j} + \underbrace{e_i}_{\text{Residual}}.$$

Thus β_j represents the rate of change in y associated with one-unit change in x_j , after adjusting for all the changes in y that can be predicted by the other predictor variables.

Partial versus overall relationships. A multiple regression equation isolates a set of *partial relationships* between y and each of the predictor variables. By a partial relationship, we mean the relationship between y and a single variable x , holding other variables constant. The partial relationship between y and x is very different than the *overall relationship* between y and x , because the latter ignores the effects of the other variables. When the two predictor variables are correlated, this difference matters a great deal.

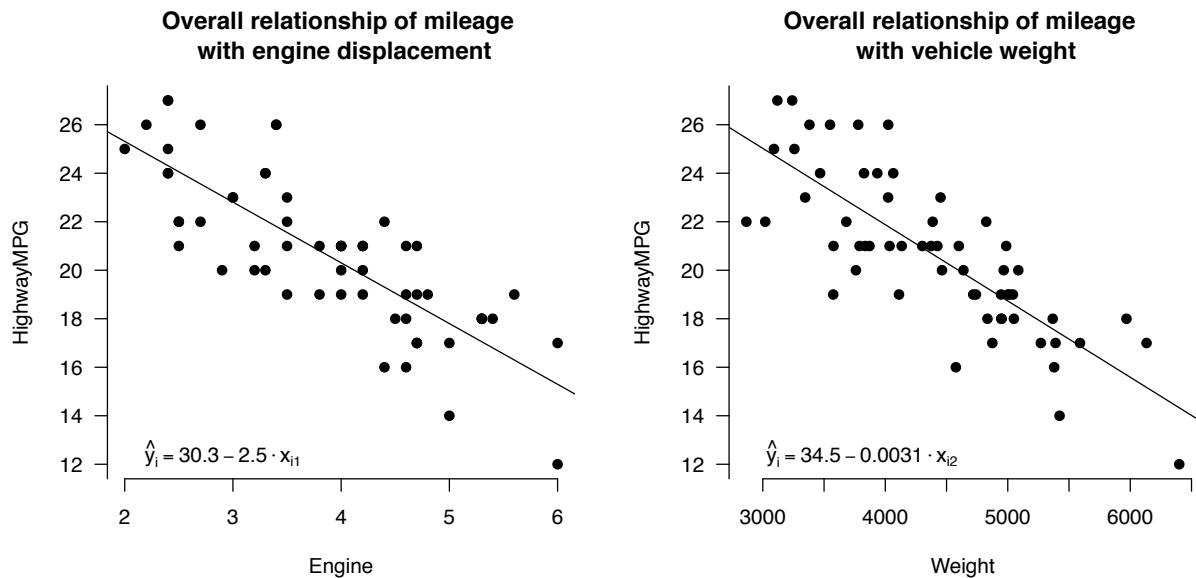
To compare these two types of relationships, let's take the multiple regression model we fit to the data on SUVs in Figure 6.1:

$$\text{MPG} = 33 - 1.35 \cdot \text{Displacement} - 0.00164 \cdot \text{Weight} + \text{Residual}.$$

This model isolates two partial relationships:

- We expect highway gas mileage to decrease by 1.35 MPG for every 1-liter increase in engine displacement, after adjusting for the simultaneous effect of vehicle weight on mileage. That is, if we held weight constant and increased the engine size by 1 liter, we'd expect mileage to go down by 1.35 MPG.
- We expect highway gas mileage to decrease by 1.64 MPG for every additional 1,000 pounds of vehicle weight, after adjusting for the simultaneous effect of engine displacement on gas mileage. That is, if we held engine displacement constant and added 1,000 pounds of weight to an SUV, we'd expect mileage to go down by 1.64 MPG.

Let's compare these partial relationships with the overall relationships depicted in Figure 6.2. Here we've fit two separate one-variable regression models: mileage versus engine displacement on the left, and mileage versus vehicle weight on the right.



Focus on the left panel of Figure 6.2 first. The least-squares fit to the data is

$$\text{MPG} = 30.3 - 2.5 \cdot \text{Displacement} + \text{Residual}.$$

Thus when displacement goes up by 1 liter, we expect mileage to go down by 2.5 MPG. This overall slope is quite different from the partial slope of -1.35 isolated by the multiple regression equation. That's because this model doesn't attempt to adjust for the effects of vehicle weight. Because weight is correlated with engine displacement, we get a steeper estimate for the overall relationship than for the partial relationship: for cars where engine displacement is larger, weight also tends to be larger, and the corresponding effect on the y variable isn't controlled for in the left panel.

Similarly, the overall relationship between mileage and weight is

$$\text{MPG} = 34.5 - 0.0031 \cdot \text{Weight} + \text{Residual}.$$

The overall slope of -0.0031 is nearly twice as steep as the partial slope of -0.00164 . The one-variable regression model hasn't successfully isolated the marginal effect of increased weight from that of increased engine displacement. But the multiple regression model has—and once we hold engine displacement constant, the marginal effect of increased weight on mileage looks smaller.

Figure 6.2: Overall relationships for highway gas mileage versus weight and engine displacement individually.

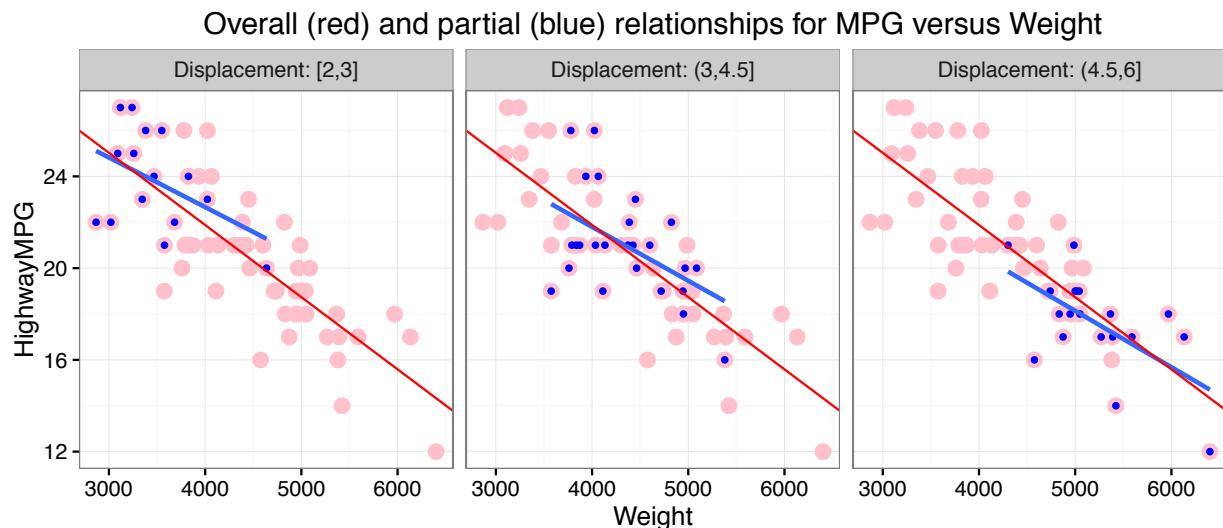


Figure 6.3 provides some intuition here about the difference between an overall and a partial relationship. The figure shows a lattice plot where the panels correspond to different strata of engine displacement: 2–3 liters, 3–4.5 liters, and 4.5–6 liters. Within each stratum, engine displacement doesn't vary by much—that is, it is approximately held constant. Each panel in the figure shows a straight line fit that is specific to the SUVs in each stratum (blue dots and line), together with the overall linear fit to the whole data set (red dots and line).

The two important things to notice here are the following.

- (1) The SUVs within each stratum of engine displacement are in systematically different parts of the x - y plane. For the most part, the smaller engines are in the upper left, the middle-size engines are in the middle, and the bigger engines are in the bottom right. When weight varies, displacement also varies, and each of these variables have an effect on mileage. Another way of saying this is that engine displacement is a *confounding variable* for the relationship between mileage and weight. A confounder is something that is correlated with both the predictor and response.
- (2) In each panel, the blue line has a shallower slope than the red line. That is, when we compare SUVs that are similar in engine displacement, the mileage–weight relationship is not as steep

Figure 6.3: A lattice plot of mileage versus weight, stratified by engine displacement. The blue points within each panel show only the SUVs within a specific range of engine displacements: ≤ 3 liters on the left, 3–4.5 liters in the middle, and > 4.5 liters on the right. The blue line shows the least-squares fit to the blue points alone within each panel. For reference, the entire data set is also shown in each panel (pink dots), together with the overall fit (red line) from the right-hand side of Figure 6.2. The blue lines are shallower than the red line, suggesting that once we hold engine displacement approximately (thought not perfectly) constant, we estimate a different (less steep) relationship between mileage and weight.

as it is when we compare SUVs with very different engine displacements.

This second point—that when we hold displacement roughly constant, we get a shallower slope for mileage versus weight—explains why the partial relationship estimated by the multiple regression model is different than the overall relationship from the left panel of Figure 6.2.² The slope of -1.64×10^{-3} MPG per pound from the multiple regression model addresses the question: how fast should we expect mileage to change when we compare SUVs with different weights, but with the same engine displacement? This is similar to the question answered by the blue lines in Figure 6.3, but different than the question answer by the red line.

It is important to keep in mind that this “isolation” or “adjustment” is statistical in nature, rather than experimental. Most real-world systems simply don’t have isolated variables. Confounding tends to be the rule, rather than the exception. The only real way to isolate a single factor is to run an experiment that actively manipulates the value of one predictor, holding the others constant, and to see how these changes affect y . Still, using a multiple-regression model to perform a statistical adjustment is often the best we can do when facing questions about partial relationships that, for whatever reason, aren’t amenable to experimentation.

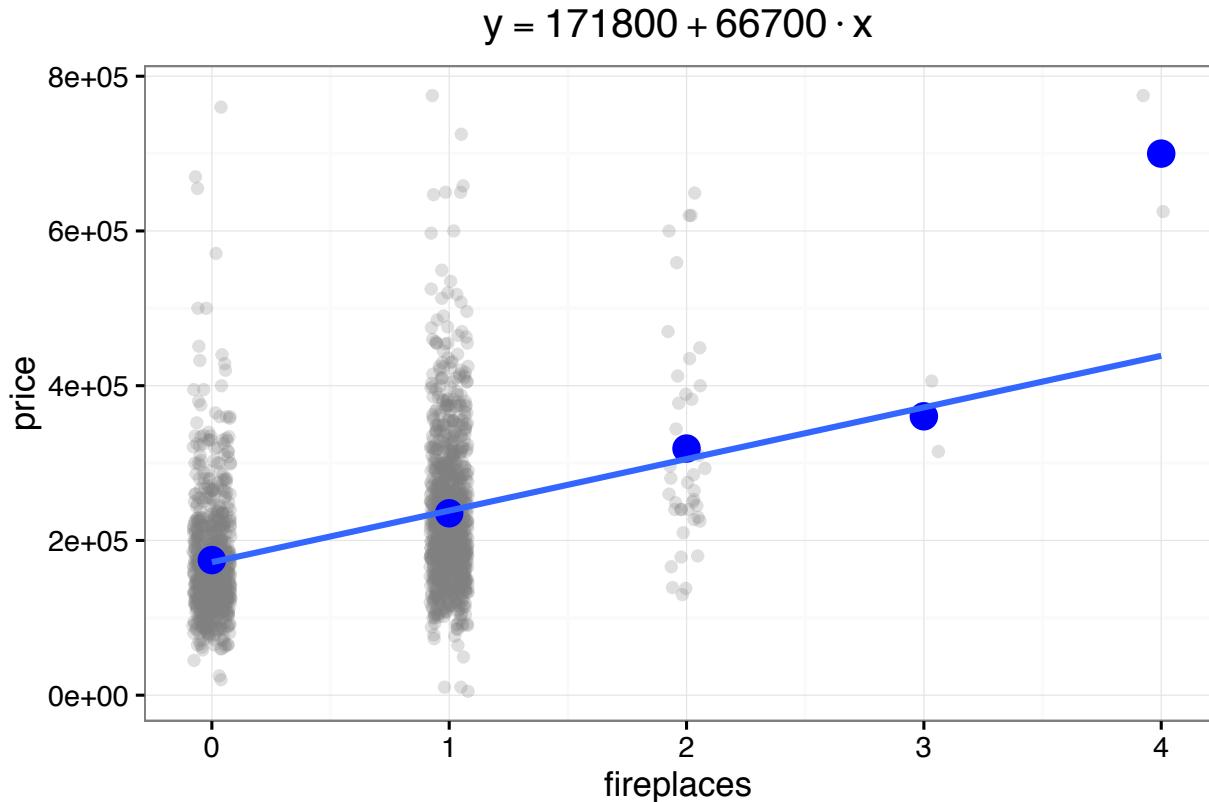
² This is a very general property of regression: if x_1 and x_2 are two correlated (collinear) predictors, then adding x_2 to the model will change the coefficient on x_1 , compared to a model with x_1 alone.

Using multiple regression to address real-world questions

While there are many possible uses of multiple regression, most applications will fall into one of two categories:

- (1) Isolating a partial relationship between the response and a predictor of interest, adjusting for possible confounders.
- (2) Building a predictive model for forecasting the response, using all available sources of information.

In the rest of this chapter, we’ll see examples in each category. As a case study, we’ll use a running example on house prices from Saratoga County, New York, distributed as part of the `mosaic` R package. We’ll show how, together with multiple regression, this data set can be used to address a few interesting questions of the kind that might be relevant to anyone buying, selling, or assessing the taxable value of a house.



How much is a fireplace worth?

Our first question is: how much does a fireplace improve the value of a house for sale? Figure 6.4 would seem to say: by about \$66,700 per fireplace. This dot plot shows the sale price of houses in Saratoga County, NY that were on the market in 2006.³ We also see a linear regression model for house price versus number of fireplaces, leading to the equation

$$\text{Price} = \$171800 + 66,700 \cdot \text{Fireplaces} + \text{Residual},$$

This fitted equation is shown as a blue line in Figure 6.4. The means of the individual groups (1 fireplace, 2 fireplaces, etc) are also shown as blue dots. This helps us to verify that the assumption of linearity is reasonable here: the line passes almost right through the group means, except the one for houses with four fireplaces (which corresponds to just two houses).

But before you go knocking a hole in your ceiling and hiring a

Figure 6.4: The relationship between the price of a house and the number of fireplaces it has.

³ Data from “House Price Capitalization of Education by Part Year Residents,” by Candice Corvetti. Williams College honors thesis, 2007, [available here](#), and in the mosaic R package.

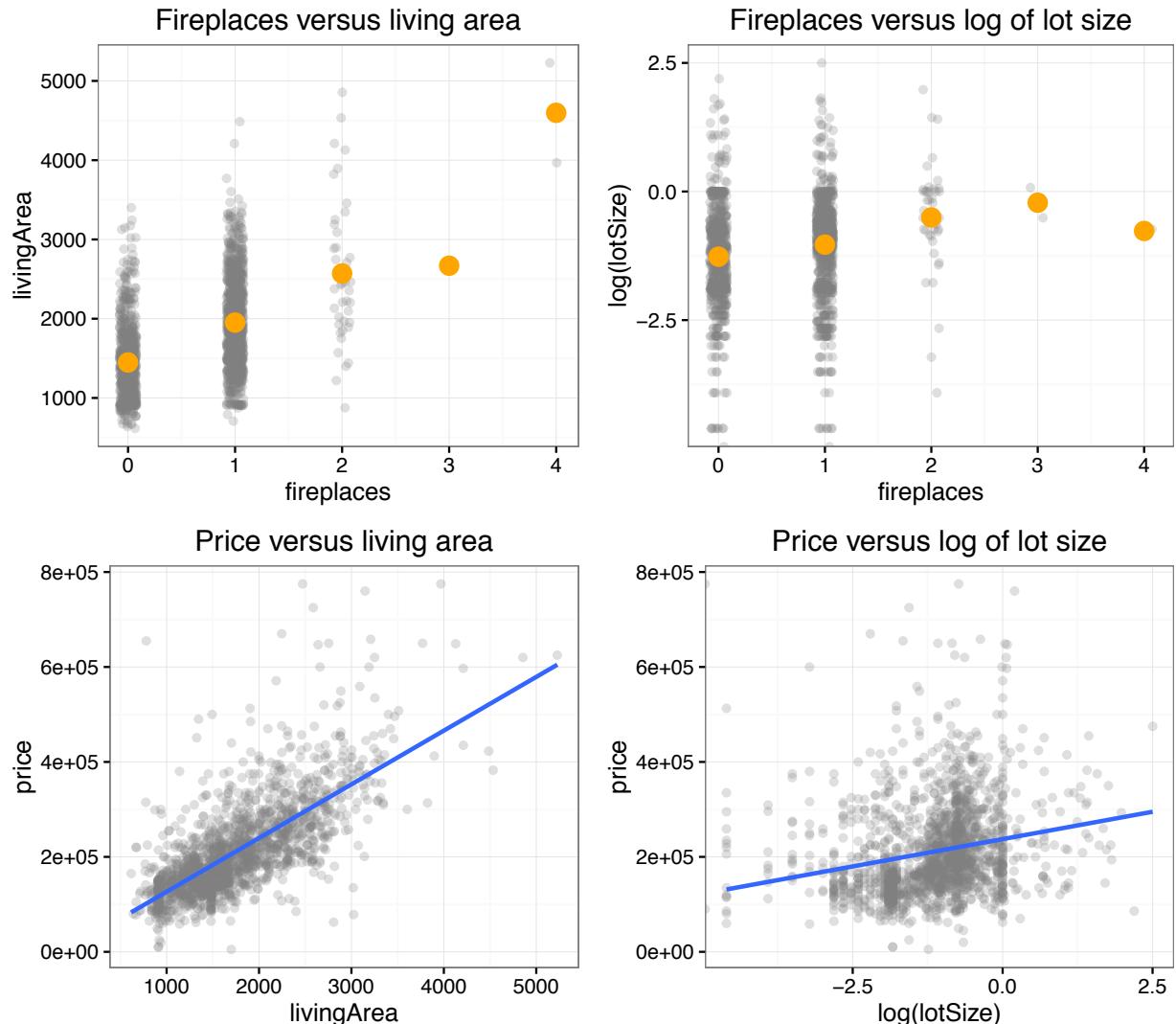


Figure 6.5: The relationship of house price with living area (bottom left) and with the logarithm of lot size in acres (bottom right). Both of these variables are potential confounders for the relationship between fireplaces and price, because they are also correlated with the number of fireplaces (top row).

bricklayer so that you might cash in on your new fireplace, consult Figure 6.5 on page 137. This figure shows that we should be careful in interpreting the figure of \$66,700 per fireplace arising from the simple one-variable model. Specifically, it shows that houses with more fireplaces also tend to be bigger (top left panel) and to sit on lots that have more land area (top right). These factors are also correlated with the price of a house.

Thus we have two possible explanations for the relationship we see in Figure 6.4. This correlation may happen because fireplaces are so valuable. On the other hand, it may instead (or also) happen because fireplaces happen to occur more frequently in houses that are desirable for other reasons (i.e. they are bigger). This is confounding again: when some third variable is correlated with both the response and the predictor of interest.

Disentangling these two possibilities requires estimating the partial relationship between fireplaces and prices, rather than the overall relationship shown in Figure 6.4. After all, when someone like a realtor or the county tax assessor asks how much a fireplace is worth, what they really want to know is: how much is a fireplace worth, holding other relevant features of the house constant?

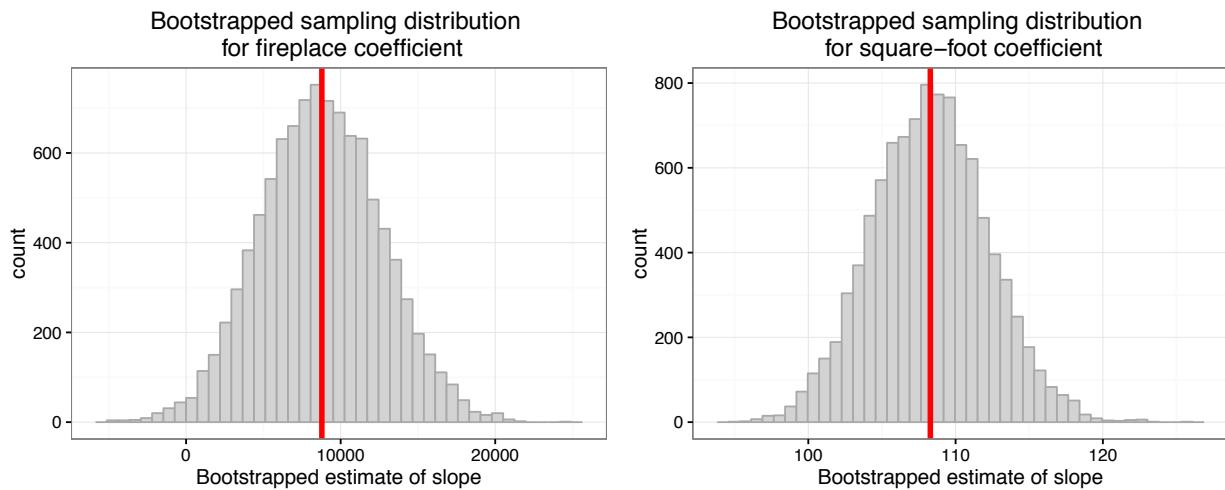
To address this question, we can fit a multiple regression model for price versus living area, lot size, and number of fireplaces. This will allow us to estimate the partial relationship between fireplaces and price, holding square footage and lot size constant. Such a model can tell us how much more we should expect a house with a fireplace to be worth, compared to a house that is identical in size and acreage but without a fireplace.

Fitting such a model to the data from Saratoga County yields the following equation:

$$\text{Price} = \$17787 + 108.3 \cdot \text{SqFt} + 1257 \cdot \log(\text{Acres}) + 8783 \cdot \text{Fireplaces} + \text{Residual}. \quad (6.1)$$

According to this model, the value of one extra fireplace is about \$8,783, holding square footage and lot size constant. This is a much lower figure than the \$66,700 fireplace premium that we would naively estimate from the overall relationship in Figure 6.4.

The example emphasizes the use of multiple regression to adjust statistically for the effect of confounders, by estimating a partial relationship between the response and the predictor of interest. This is one of the most useful real-world applications of regression modeling, and we'll see many similar examples. In general, the advice is: if you want to estimate a partial relationship, make sure



you include the potential confounders in the model.

Uncertainty quantification

We can use bootstrapping to get confidence intervals for partial relationships in a multiple regression model, just as we do in a one-variable regression model.

The left panel of Figure 6.6 shows the bootstrapped estimate of the sampling distribution for the fireplace coefficient in our multiple regression model. The 95% confidence interval here is (1095, 16380). Thus while we do have some uncertainty we have about the value of a fireplace, we can definitively rule out the number estimated using the overall relationship from Figure 6.4. If the county tax assessor wanted to value your new fireplace at \$66,700 for property-tax purposes, Figure 6.6 would make a good argument in your appeal.⁴

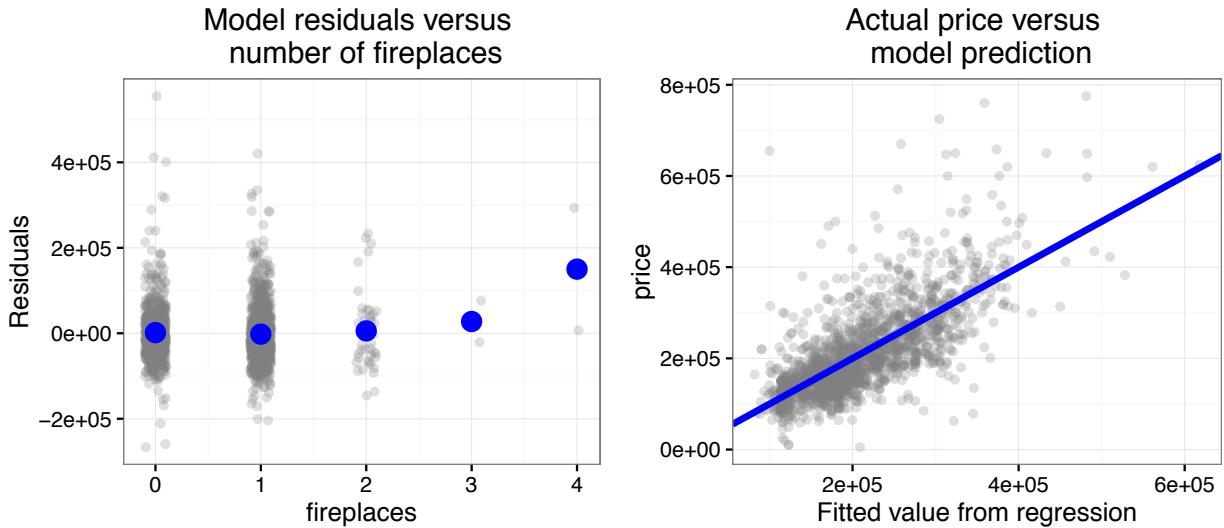
The right-hand side of Figure 6.6 shows the bootstrapped sampling distribution for the square-foot coefficient. While this wasn't the focus of our analysis here, it's interesting to know that an additional square foot improves the value of a property by about \$108, plus or minus about \$8.

Model checking

However, before we put too much faith in the conclusions of your fitted model, it's important to check whether the assumption of a

Figure 6.6: Bootstrapped estimates for the sampling distributions of the partial slopes for number of fireplaces (left) and square footage (right) from the model in Equation 6.1 on page 138. The least-squares estimates are shown as vertical red lines.

⁴ At a 2% property tax rate, this might save you over \$1000 a year in taxes.



linear regression model is appropriate in the first place. We call this step *model checking*. We'll learn a lot more about model checking later, but for now we'll cover the most basic step: validating that the response varies linearly with the predictors.

In one-variable regression models, we addressed this question using a plot of the residuals e_i versus the original predictor x_i . This allowed us to check whether there was still a pattern in the residuals that suggested a nonlinear relationship between the predictor and response. There are two ways to extend the idea of a residual plot to multiple regression models:

- plotting the residuals versus each of the predictors x_{ij} individually. This allows us to check whether the response changes linearly as a function of the j th predictor.
- plotting the actual values y_i versus the fitted values \hat{y}_i and looking for nonlinearities. This allows us to check whether the responses depart in a systematically nonlinear way from the model predictions.

Figure 6.7 shows an example of each plot. The left panel shows each the residual for each house versus the number of fireplaces it contains. Overall, this plot looks healthy: there are no obvious departures from linearity. The one caveat is that the predictions for houses with four fireplaces may be too low, which we can see from the fact that the mean residual for four-fireplace houses is positive. Then again, there are only two such houses, making it difficult to

Figure 6.7: Left: model residuals versus number of fireplaces. Right: observed house prices versus fitted house prices from the multiple regression model.

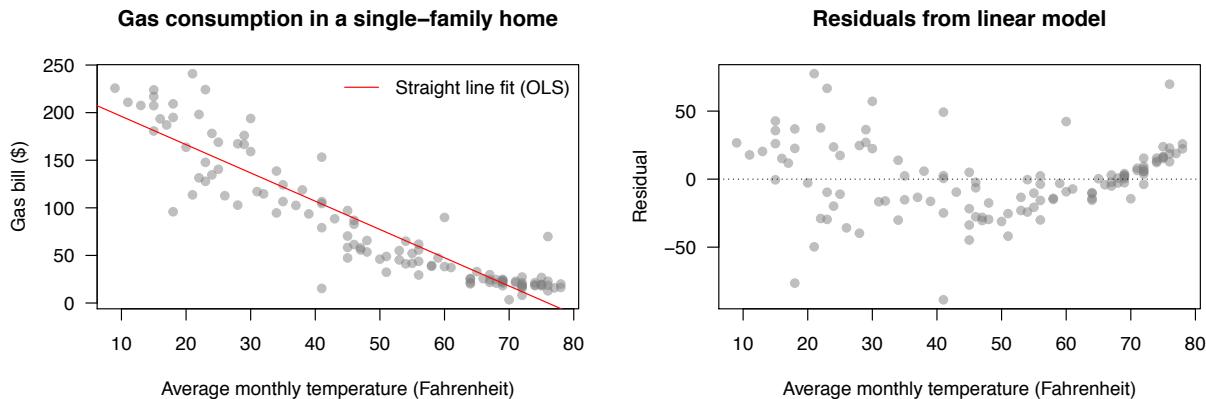


Figure 6.8: A copy of Figure 2.7 from the unit on nonlinear curve-fitting.

draw a firm conclusion here. We probably shouldn't change our model just to chase a better fit for two (very unusual) houses out of 1,726. But we should also recognize that our model might not be great at predicting the price for a house with four fireplaces, simply because it would involve extrapolation: we don't have a lot of data that can inform us about these houses.

The right panel of Figure 6.7 shows a plot of y_i versus \hat{y}_i . This also looks like a nice linear relationship, giving us further confidence that our model isn't severely distorting the true relationship between predictors and response. In a large multiple regression model with many predictors, it may be tedious to look at e_i versus each of those predictors individually. In such cases, a plot of y_i versus \hat{y}_i should be the first thing you examine to check for nonlinearities in the overall fit.

What would an unhealthy residual plot look like? To see an example, recall Figure 2.7 from the data set on gas consumption versus temperature, on page 44 (reproduced in Figure 6.8). Notice the pattern in the residual plot in the right panel:

- Below 20 degrees, the residuals are systematically above zero.
- Between 40 and 60 degrees, the residuals are below zero.
- Above 65 degrees, the residuals are again above zero.

The residuals *should* look like a random cloud centered around zero, and the fact that they don't suggests nonlinearity in the data.

In the case of the house-price model, imagine that we saw that the residuals for houses with no fireplace were systematically above zero, while the residuals for houses with one fireplace were

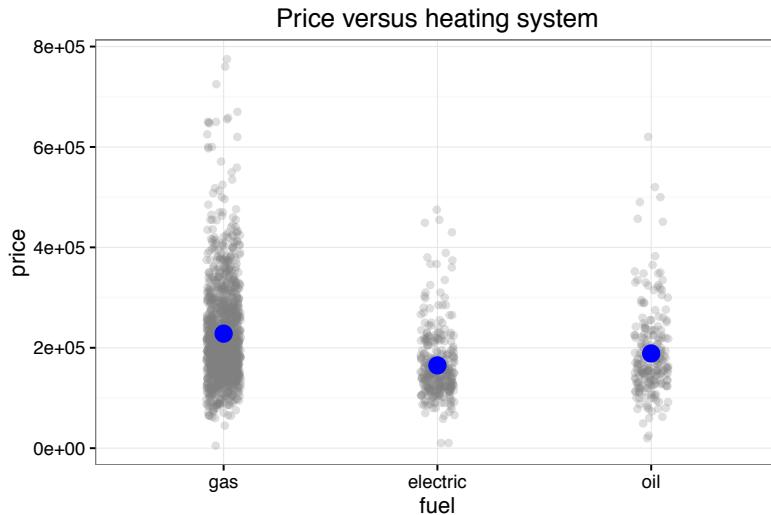


Figure 6.9: Prices of houses with gas, electric and fuel-oil heating systems.

systematically below zero. This would suggest a nonlinear effect that our model hasn't captured. Of course, we don't see these things, which gives credence to the linear model.

How much is gas heating worth? Grouping variables in multiple regression

Saratoga, NY is cold in the winter: the average January day has a low of 13° F and a high of 31° F. As you might imagine, residents spend a fair amount of money heating their homes, and are sensitive to the cost differences between gas, electric, and fuel-oil heaters. Figure 6.9 suggests that the Saratoga real-estate market puts a big premium for houses with gas heaters (mean price of \$228,000) versus those with electric or fuel-oil heaters (mean prices of \$165,000 and \$189,000, respectively). One possible reason is that gas heaters are cheaper to run and maintain.

But this figure shows an overall relationship. What does the story look like when we adjust for the effect of living area, lot size, and the number of fireplaces? There could be a confounding effect here. For example, maybe the bigger houses tend to have gas heaters more frequently than the small houses, or maybe fireplaces are used more in homes with expensive-to-use heating systems.

Remember: if you want to isolate a partial relationship, include potential confounders in the model. We'll do this here by including two sets of terms: (1) dummy variables for heating-system

| Variable | Estimate | Std. Error | 2.5% | 97.5% |
|---------------|----------|------------|--------|-------|
| Intercept | 29868 | 6743 | 16644 | 43093 |
| livingArea | 105 | 3 | 99 | 112 |
| log(lotSize) | 2705 | 1913 | -1047 | 6457 |
| fireplaces | 7547 | 3348 | 980 | 14113 |
| fuel=electric | -14010 | 4471 | -22778 | -5242 |
| fuel=oil | -15879 | 5295 | -26265 | -5494 |

Table 6.1: Coefficients, standard errors, and 95% confidence intervals for the multiple regression model for house price (y) versus living area, log of lot size, number of fireplaces, and heating system type.

type, to model the partial relationship of interest; and (2) all the possible confounding variables that we had in our previous regression equation (on page 138), which includes living area, lot size, and number of fireplaces. Fitting this model by least squares yields the following equation:

$$\text{Price} = \$29868 + 105.3 \cdot \text{SqFt} + 2705 \cdot \log(\text{lotSize}) + 7546 \cdot \text{Fireplaces} \\ - 14010 \cdot \mathbf{1}_{\{\text{fuel} = \text{electric}\}} - 15879 \cdot \mathbf{1}_{\{\text{fuel} = \text{oil}\}} + \text{Residual}.$$

The full table of coefficients, standard errors, and 95% confidence intervals is in Table 6.1. The baseline here is gas heating, since it has no dummy variable.

Notice how the coefficients on the dummy variables for the other two types of heating systems shift the entire regression equation up or down. This model estimates the premium associated with gas heating to be about $\$14,000 \pm 4500$ over electric heating (estimate, plus-or-minus one standard error), and about $\$16,000 \pm 5300$ over fuel-oil heating. Because these are terms in a multiple regression model, these numbers represent partial relationships, adjusting for size, lot acreage, and number of fireplaces.

Assessing statistical significance

A question that often comes up in multiple regression is whether a particular term in the model is “statistically significant” at some specified level (e.g. 5%). All this means is whether zero is a plausible value for that partial slope in the model. Remember, a coefficient of zero means that there is no partial relationship between the response and the corresponding predictor, adjusting for the other terms in the model. So when we say that a predictor is statistically significant, all we mean is that it we think it has a nonzero (partial) relationship with the response.

We'll take up the question of assessing statistical significance

in much more detail in the chapters to come. But here are a few quick observations and guidelines.

First, by convention, people express the statistical significance level as the opposite of the confidence level. So a confidence level of 95% means a significance level of 5%; a confidence level of 99% means a significance level of 1%; and so forth. This is confusing at first, but you'll get used to it. Just remember: the *lower* the significance level, the stronger the evidence that some variable has a nonzero relationship with the response.

Second, in regression models we can often⁵ assess statistical significance just by looking at whether zero is included in the confidence interval. That's because "statistically significant" just means "zero is not a plausible value," and a confidence interval gives us a range of plausible values. For example, let's take the 95% confidence intervals for two terms in Table 6.1:

- The 95% confidence interval for the partial slope on fireplaces is (980, 14113). We can rule out zero as a plausible value at a 95% confidence level, and so we can say that the lot size variable is statistically significant at the 5% level.
- The 95% confidence interval for the partial slope on lot size is (-1047, 6457). We cannot rule out zero as a plausible value with 95% confidence, and so the lot size variable is not statistically significant at the 5% level.

Third, the fact that some variable is "statistically significant" does not mean that this variable is important in practical terms. A "significant" variable does not necessarily have a large effect on the response, nor is it automatically important for generating good predictions. Statistical significance means that we think the corresponding coefficient isn't zero. But it could still be very small. This is why, in most cases, it is better to focus on a variable's confidence interval, rather than on whether a variable is significant. The confidence interval carries a lot more information than a simplistic distinction between "significant" and "insignificant," because it gives you a range of plausible values for the coefficient.

Finally, the fact that some variable is *not* statistically significant does not imply that this variable has no relationship with the response, or that it should automatically be dropped from the model. A lack of statistical significance could just mean a big standard error—in other words, that we have a lot of uncertainty about the numerical magnitude of some variable's partial relationship

⁵ But not always; see the next chapter.

with the response. There's an important but subtle distinction here: an insignificant coefficient means that we have an *absence of compelling evidence* for a nonzero effect. It does not mean that we have found *compelling evidence that the effect is absent*.

For example, the confidence interval for the log(acres) term in Table 6.1 is $(-1047, 6457)$. We therefore cannot rule out zero as a plausible value. But there are lots of large values, like 5000 or 6000, that we cannot rule out, either! There's a lot of uncertainty here. One symptom of this is a big standard error; another symptom is a lack of statistical significance at the 5% level. But it does not follow that lot size is irrelevant for predicting house price.⁶

Prediction intervals from multiple regression models

Suppose you have a house in Saratoga, NY that you're about to put up for sale. It's a 1900 square-foot house on a 0.7-acre lot. It has 3 bedrooms, 2.5 bathrooms,⁷ 1 fireplace, gas heating, and central air conditioning. The house was built 16 years ago. (If you're counting, that's 8 possible predictors.) How much would you expect it to sell for?

A great way to assess the value of the house is to use the available data to fit a multiple regression model for its price, given its features. Building regression models for prediction is a rich, important topic that we'll consider in more detail later. For now, let's suppose we choose to fit a model for price versus all 8 variables mentioned above: bedrooms, bathrooms, living area, lot size, fireplaces, fuel system type, presence of central air conditioning, and the age of the home. Table 6.2 gives the coefficients, standard er-

⁶ In this the large standard error is almost surely due to collinearity between lot size and other predictors, which we will discuss further in a later chapter.

⁷ A half-bathroom has a toilet but no bath or shower.

| Variable | Estimate | Std. Error | 2.5% | 97.5% |
|---------------|----------|------------|--------|--------|
| Intercept | 48549 | 8190 | 32486 | 64611 |
| bedrooms | -12263 | 2653 | -17467 | -7059 |
| bathrooms | 21330 | 3756 | 13965 | 28696 |
| livingArea | 98 | 5 | 89 | 107 |
| lotSize | 9514 | 2387 | 4832 | 14195 |
| fireplaces | 1017 | 3304 | -5464 | 7498 |
| fuel=electric | -14318 | 4467 | -23080 | -5556 |
| fue=oil | -10465 | 5290 | -20841 | -89 |
| centralAir=No | -19964 | 3665 | -27151 | -12776 |
| age | 28 | 63 | -95 | 151 |

Table 6.2: Coefficients, standard errors, and 95% confidence intervals for our basic predictive model of house price.

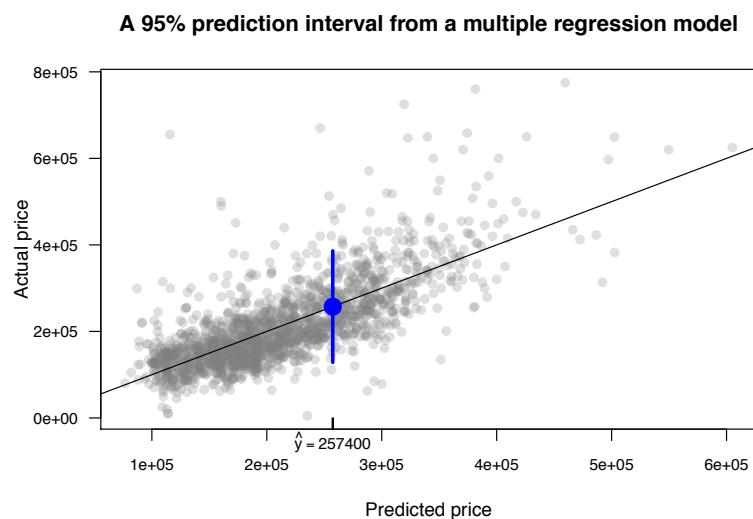


Figure 6.10: A 95% prediction interval (vertical blue line) for a house whose forecasted price is $\hat{y} = \$257,400$, using the model shown in Table 6.2.

rors, and 95% confidence intervals for this model. These, in turn, can be used to form a prediction interval for a “future” house with predictors (x_1^*, \dots, x_p^*) , just as we did back in the chapter on one-variable linear regression:

$$y^* \in \underbrace{\hat{\beta}_0 + \sum_{j=1}^p \hat{\beta}_j x_j^*}_{\text{Best guess, } \hat{y}^*} \pm \underbrace{k \cdot s_e}_{\text{Uncertainty}} ,$$

where k is a chosen multiple, and where s_e is the standard deviation of the model residuals.⁸

We’re still using multiple regression here, but the goal here is slightly different than in the previous examples. Here, we don’t care so much about isolating and interpreting one partial relationship (like that between fireplaces and price). Instead, we just want to include any variables that will help us improve our predictions.

The model in Table 6.2 tells us that, for your 1900-square-foot house in Saratoga on 0.7 beautiful acres, the expected price is $\hat{y} = 257400$; that the residual standard deviation is $s_e = 65600$; and that the 95% prediction interval is $(128600, 386200)$. See Figure 6.10; that’s a pretty wide range, reflecting the considerable variation in the price of different houses—even houses that look pretty similar on the page.

⁸ As before, this is a slightly oversimplified formula, in that it ignores uncertainty due to lack of perfect knowledge about the parameters. Most regression software will use the correct (but much more complicated) formulas to calculate prediction intervals, e.g. the “predict” function in R.

7

Testing hypotheses

Assessing the evidence for a hypothesis

AMONG professional football fans, the New England Patriots are a polarizing team. Their fan base is hugely devoted, probably due to their long run of success over more than a decade. Many others, however, dislike the Patriots for their highly publicized cheating episodes, whether for deflating footballs or clandestinely filming the practice sessions of their opponents. This feeling is so common among football fans that sports websites often run images like the one at right (of the Patriots' be-hoodied head coach, Bill Belichick), or articles with titles like “[11 reasons why people hate the Patriots.](#)” Despite—or perhaps because of—their success, the Patriots always seem to be dogged by scandal and ill will.

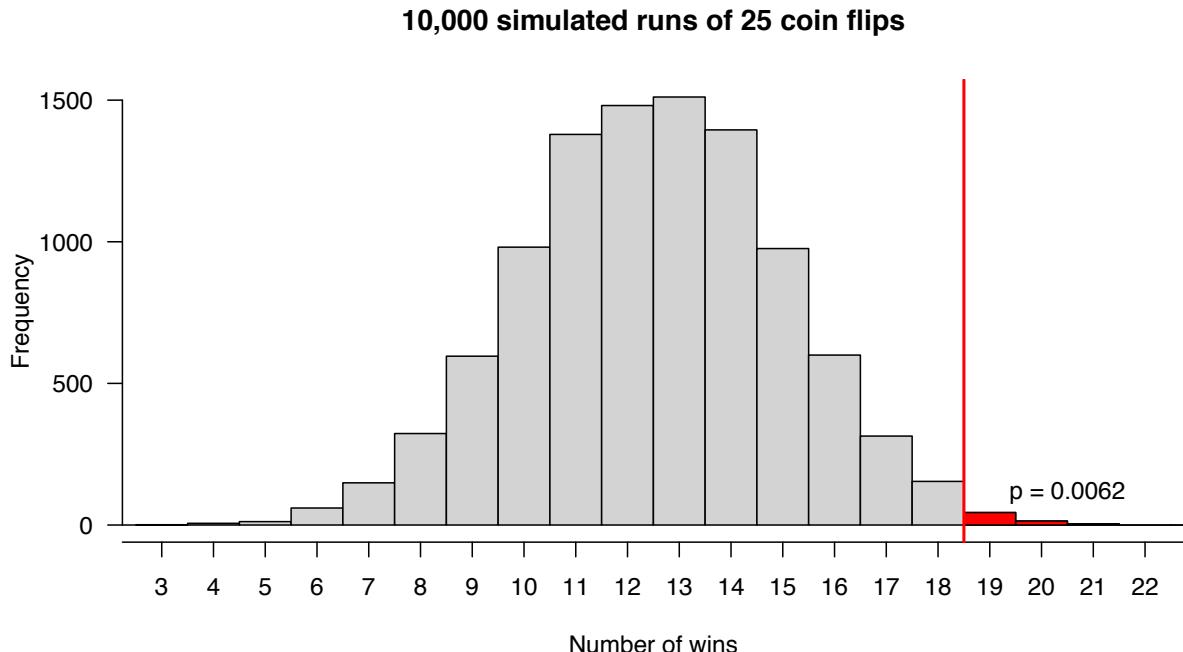
But could even the Patriots cheat at the pre-game *coin toss*?

Believe it or not, many people think so! That’s because, for a stretch of 25 games spanning the 2014–15 NFL seasons, the Patriots won 19 out of 25 coin tosses—that’s a 76% winning percentage. Needless to say, the Patriots’ detractors found this infuriating. As one TV commentator remarked when this unusual fact was brought to his attention: “This just proves that either God or the devil is a Patriots fan, and it sure can’t be God.”

But before turning to religion, let’s take a closer look at the evidence. Just how likely is it that one team could win the pre-game coin toss at least 19 out of 25 times, assuming that there’s no cheating going on?

This question is easy to answer using probability theory—specifically, something called the binomial distribution. But it’s also very easy to answer using the Monte Carlo method, in which we write a computer program that simulates a random process. In Figure 7.1, we see the results of a Monte Carlo simulation for pre-game NFL coin tosses, where the Patriots ought to have a 50% chance of winning each toss. Specifically, we have repeated the





following simple process 10,000 times:

1. Simulate 25 coin tosses in which the Patriots have a 50% chance of winning each toss.
2. Count how many times out of 25 that the Patriots won the toss.

If you're counting, that's 250,000 coin tosses: 10,000 simulations of 25 tosses each.

Figure 7.1 shows a histogram of the number of coin tosses won by the Patriots across 10,000 simulations. Clearly 19 wins is an unusual, although not impossible, number under this distribution: in our simulation, the Patriots won at least 19 tosses only 62 of 10,000 times ($p = 0.0062$), shown as the red area in Figure 7.1.

So did the Patriots win 19 out of 25 coin tosses by chance? Well, nobody knows for sure—I report, you decide.¹ But unless you're a hard-core NFL conspiracy theorist, let me encourage you to forget the Patriots for a moment and focus instead on the process we've just gone through. This simple example has all the major elements of *hypothesis testing*, which is the subject of this chapter:

Figure 7.1: This histogram shows the results of a Monte Carlo simulation, in which we count the number of wins in 25 simulated coin flips over 10,000 different simulations. The red area (which has cumulative probability of 0.0062) approximates the probability of winning 19 or more flips, out of 25.

¹ Despite the small probability of such an extreme result, it's hard to believe that the Patriots cheated on the coin toss, for a few reasons. First, how could they? The coin toss would be extremely hard to manipulate, even if you were inclined to do so. Moreover, the Patriots are just one team, and this is just one 25-game stretch. There are 32 NFL teams, so the probability that *one* of them would go on an unusual coin-toss winning streak over *some* 25-game stretch over a long time period is a lot larger than the number we've calculated. Finally, after this 25-game stretch, the Patriots reverted back to a more typical coin-toss winning percentage, closer to 50%. The 25-game stretch was probably just luck.

- (1) We have a *null hypothesis*, that the pre-game coin toss in the Patriots' games was truly random.
- (2) We use a *test statistic*, number of Patriots' coin-toss wins, to measure the evidence against the null hypothesis.
- (3) There is a way of calculating the probability distribution of the test statistic, assuming that the null hypothesis is true. Here, we just ran a Monte Carlo simulation of coin flips, assuming an unbiased coin.
- (4) Finally, we used this probability distribution to assess whether the null hypothesis looked believable in light of the data.

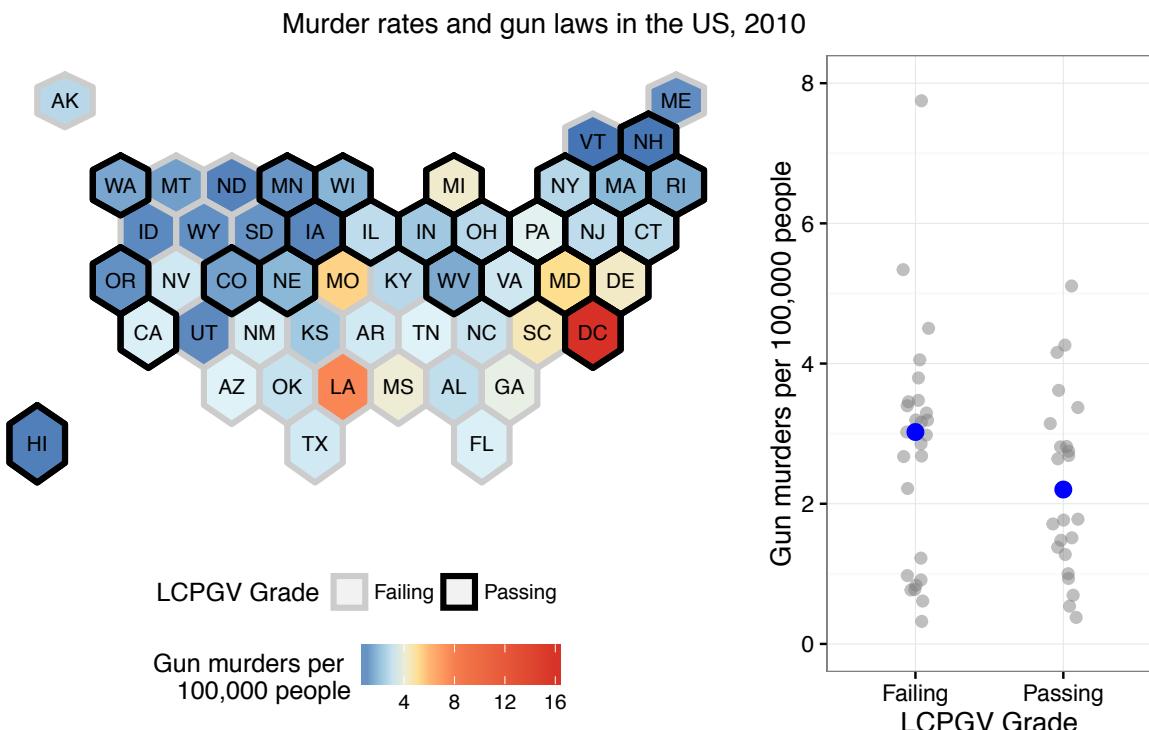
All hypothesis testing problems have these same four elements. Usually the difficult part is Step 3: calculating the probability distribution of the test statistic, assuming that the null hypothesis is true. The essence of the problem is that, in most cases, we can't just run a simple simulation of coin flips. Luckily, there is a very general way of proceeding here, called the permutation test, which we will now learn about.

Permutation tests

Is gun violence correlated with gun policy?

GUN policy is an important and emotionally charged topic in 21st-century America, where gun violence occurs with far higher frequency than it does in other rich countries. Many people feel strongly that certain types of guns, like military-style assault weapons, should be banned, and that all gun purchases should be subject to stronger background checks. Others view gun ownership as both an important part of their cultural heritage and a basic right protected by the U.S. Constitution. Like with many issues, there seems to be little prospect of a national consensus.

Both gun laws, and the likelihood of dying violently as a result of gun crime, vary significantly from state to state. Figure 7.2 shows some of this variation in a *chloropleth map*, where discrete areas on the map are shaded according to the value of some numerical variable. Notice that the states are shown as a gridded tile of equal-sized hexagons, rather than as an actual map of the United States. This is common technique used to avoid the visual imbalances due to large differences in the states' total area.



In the chloropleth map in Figure 7.2, the fill color indicates each state's gun-murder rate in 2010: blue is lower, red is higher. The outline color indicates whether a state's gun-control laws received a passing or failing grade from the Law Center to Prevent Gun Violence (LCPGV). The center graded each state's gun laws on an A–F letter-grade scale; here “failing” means a grade of F. In the figure, a black outline means a passing grade, while a grey outline means a failing grade.

The right panel of Figure 7.2 summarizes the relationship between gun laws and gun violence via a dot plot, together with the median for each group in blue. We use the median rather than the mean to estimate the center of each group, because the median is more robust to outliers; a clear example of an outlier here is Washington (D.C.), which at 16.2 gun murders per 100,000 people has a drastically higher rate than everywhere else in the country.

This dotplot shows that the median murder rate of states with a failing gun-laws grade is 3 murders per 100,000 people, while the median murder rate of states with a passing grade is 2.2 per

Figure 7.2: Left panel: a chloropleth map of murder rates versus gun laws across the U.S. states. The shaded color shows the state's gun-murder rate; blue is lower, and red is higher. The outline indicates whether a state's gun-control laws received a passing or a failing grade from the Law Center to Prevent Gun Violence (black for passing, grey for failing). The right panel shows a dot plot of the gun-murder rates across the two groups, together with the median for each group in blue. Washington (D.C.), at 16.2 gun murders per 100,000 people, is far off the top of the plot, but is still included in all calculations. According to its website, <http://smartgunlaws.org>, the LCPGV is “a national law center focused on providing comprehensive legal expertise in support of gun violence prevention and the promotion of smart gun laws that save lives.” You can read a full description of the methodology used to grade states at [this link](#).

100,000. On the face of it, it would seem as the states with stricter gun laws have lower murder rates.

Let's aside for a moment the fact that correlation does not establish causality. We will instead address the question: could this association have arisen due to chance? To make this idea more specific, imagine we took all 50 states and randomly divided them into two groups, arbitrarily labeled the "passing" states and the "failing" states. We would expect that the median murder rate would differ a little bit between the two groups, simply due to random variation (for the same reason that hands in a card game vary from deal to deal). But how big of a difference between these two groups could be explained by chance?

Null and alternative hypotheses

Thus there are two hypotheses that can explain Figure 7.2:

- (1) There is no systematic relationship between murder rates and gun laws; the observed relationship between murder rates and gun laws is consistent with other unrelated sources of random variation.
- (2) The observed relationship between murder rates and gun laws is too large to be consistent with random variation.

We call hypothesis 1 the *null hypothesis*, often denoted H_0 . Loosely, it states that nothing special is going on in our data, and that any relationship we thought might have existed isn't really there at all.² Meanwhile, hypothesis 2 is *alternative hypothesis*. In some cases the alternative hypothesis may just be the logical negation of the null hypothesis, but it can also be more specific.

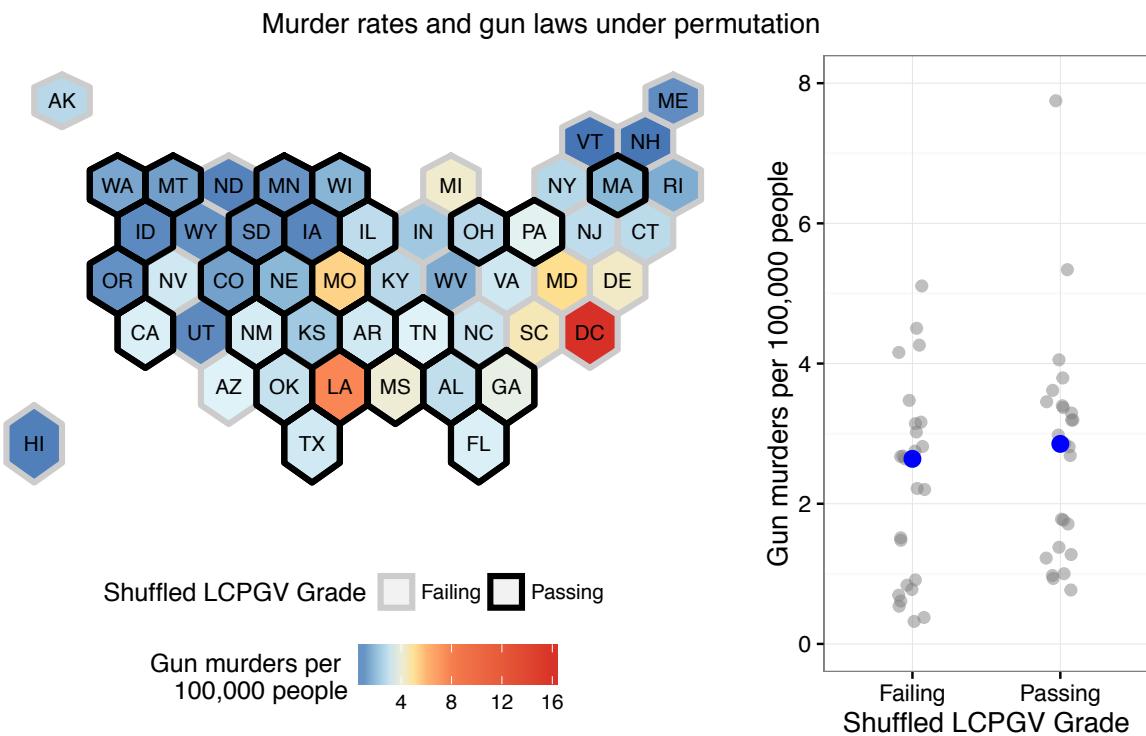
In the approach to hypothesis testing that we'll learn here, we don't focus a whole lot on the alternative hypothesis.³ Instead, we set out to check whether the null hypothesis looks plausible in light of the data—just as we did when we tried to check whether randomness could explain the Patriots' impressive run of 19 out of 25 coin flips won.

A permutation test: shuffling the cards

In the Patriots' coin-flipping example, we could easily simulate data under the null hypothesis, by programming a computer to repeatedly flip a virtual coin and keep track of the winner. But of course, most real-life hypothesis-testing situations don't involve

² "Null hypothesis" is a term coined in the early twentieth century, back when "null" was a common synonym for "zero" or "lacking in distinctive qualities." So if the term sounds dated, that's because it is.

³ Specifically, this approach is called the *Fisherian* approach, named after the English statistician Ronald Fisher. There are more nuanced approaches to hypothesis testing in which the alternative hypothesis plays a major role. These include the Neyman–Pearson framework and the Bayesian framework, both of which are widely used in the real world, but which are a lot more complicated to understand.



actual coin flips, which makes the virtual coin-flipping approach somewhat unhelpful as a general strategy.

It turns out, however, that in most situations, we can still harness the power of Monte Carlo simulation to understand what our data would look like if the null hypothesis were true. Rather than flipping virtual coins, we run something called a *permutation test*, which involves repeatedly permuting (or shuffling) the predictor variable and recalculating the statistic of interest.

To understand how this works, let's see an example. Figure 7.3 shows a map and dotplot very similar to those in Figure 7.2, with one crucial difference: in Figure 7.3, the identities of the states with notionally “passing” and “failing” gun laws have been randomly permuted. These grades bear no correspondence to reality. It's as though we took a deck of 51 cards, each card having some state's grade on it (treating D.C. as a state); shuffled the deck; and then dealt one card randomly to each state. The mathematical term for this is a *permutation* of the grades.

As expected, the median gun-murder rates of these two ran-

Figure 7.3: This map is almost identical to Figure 7.2, with one crucial difference: the identities of the states with passing and failing grades have been randomly permuted. There is still a small difference in the medians of the notionally passing and failing groups, due to random variation in the permutation process.

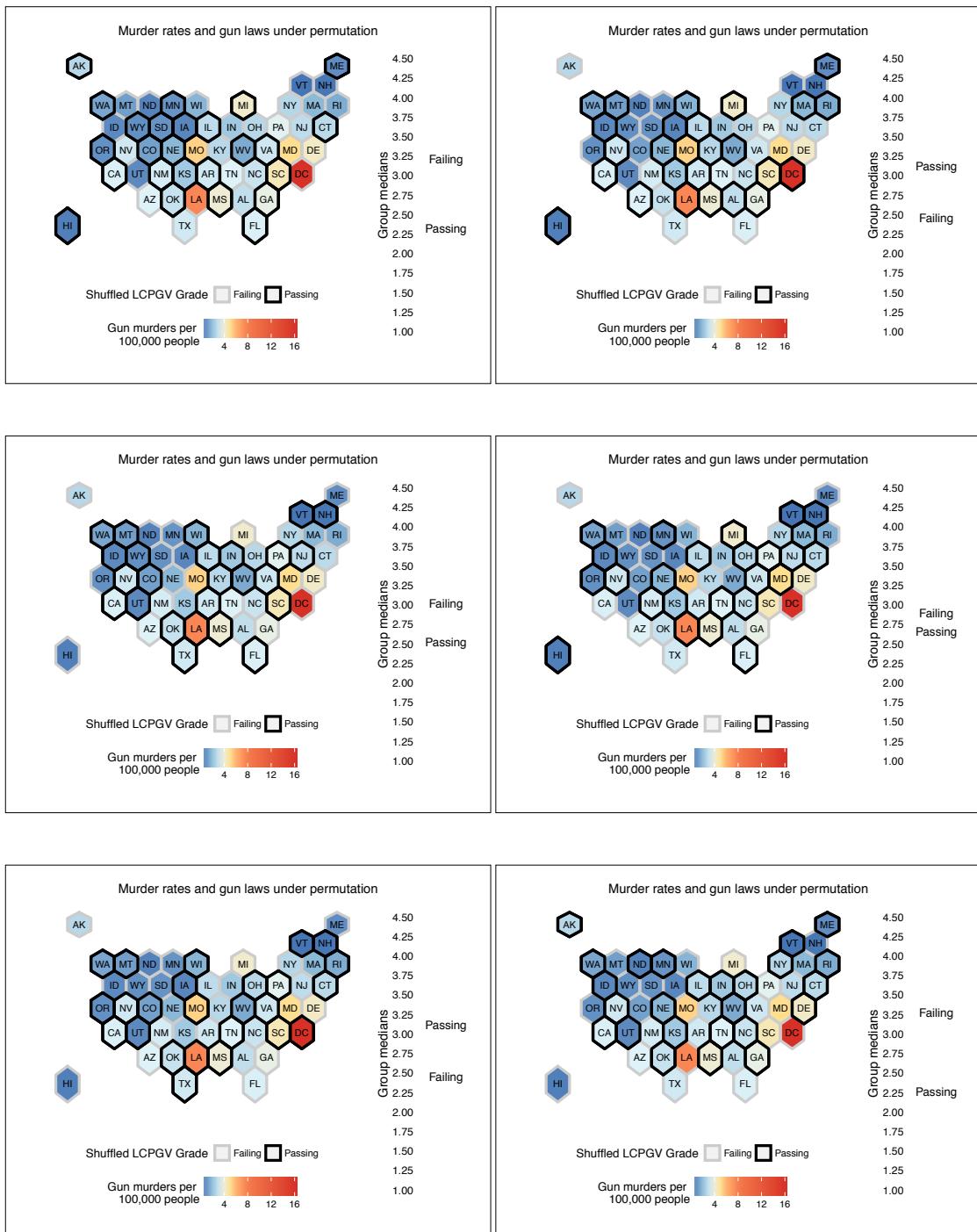


Figure 7.4: Six maps with permuted gun-law grades, with the medians for the passing and failing groups.

dom chosen “passing” and “failing” groups aren’t identical (right panel). The randomly chosen “failing” states have a median of 2.6, while the randomly chosen “passing” states have a slightly larger median of 2.8. Clearly we can get a difference in medians of at least 0.2 quite easily, just by random chance—that is, when the null hypothesis is true by design.

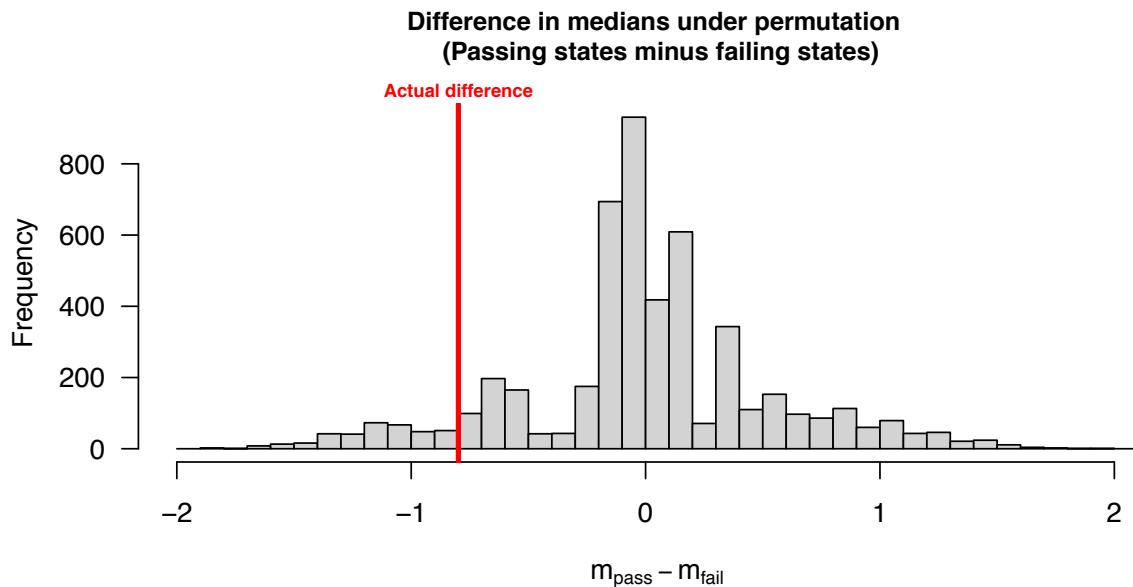
But Figure 7.3 shows the difference in medians for only a single permutation of the states’ gun-law grades. This permutation is random, and a different permutation would have given as a slightly different answer. Therefore, to assess whether could we get a difference in group medians as large as 0.8 just by random chance, we need to try several more permutations.

Figure 7.4 shows 6 more maps generated using the same permutation procedure. For each map, we shuffle the grade variables for all the states and recompute the median murder rates for the notionally “passing” and “failing” groups. Each map leads to its own difference in medians. In some maps, the difference is positive (“passing” states are higher), while in others it is negative (“failing” states are higher). In at least one of the 6 maps—the bottom right one—the median for the “failing” states exceeds the median for the “passing” states by more than 1 murder per 100,000 people, just by chance. This is a larger difference than we see for the real map, in Figure 7.2.

Six permutations give us some idea of how much a difference in the medians we could expect to see if the null hypothesis were true. But ideally we’d have many more than 6. Figure 7.5 addresses this need, showing the result of a much larger Monte Carlo simulation in which we generated 5,000 random maps, each one with its own random permutation of the states’ gun-law grades. For each of these 5,000 maps, we computed the difference in medians between the notionally passing and failing groups. These 5,000 differences in group medians across the 5,000 maps are shown as a histogram in Figure 7.5.

Hypothesis testing: a four-step process

Let’s review the vocabulary that describes what we’ve done here. First, we specified a null hypothesis: that the correlation between rates of gun violence and state-level gun policies could be explained by other unrelated sources of random variation. We decided to measure this correlation using a specific statistic: the difference in medians between the states with passing grades and



those with failing grades. (Remember that a statistic is just some numerical summary of a data set.) To give this statistic a name, let's call it Δ (for difference in medians). It's intuitively clear that the larger Δ is, the less plausible the null hypothesis seems.

Figure 7.5 quantifies this intuition by giving us an idea of how much variation we can expect in the sampling distribution of our Δ statistic under the hypothesis that there is no systematic relationship between gun laws and rates of gun violence. As before, the sampling distribution is simply the probability distribution of the statistic under repeated sampling from the population—in this case, assuming that the null hypothesis is true.

There are two possibilities here, corresponding to the null and alternative hypotheses. First, suppose that we frequently get at least as extreme a value of Δ for a random map, like those in Figure 7.4, as we do in the real map from Figure 7.2. Then there's no reason to be especially impressed by the actual value of $\delta = -0.8$ we calculated from the real map.⁴ It could have easily happened by chance. Hence we will be unable to reject the null hypothesis; it could have explained the data after all. (An important thing to remember is that *failing to reject* the null hypothesis is not the

Figure 7.5: The histogram shows the difference in group medians for 5,000 simulated maps generated by the same permutation procedure as the 6 maps in Figure 7.4. Negative values indicate that the “failing” states had higher rates of gun violence than the “passing” states. The actual difference in medians for the real map in Figure 7.2 is shown as a vertical red line. This difference seems to be consistent with (although does not prove) the null hypothesis that other sources of random variation, and not necessarily state-level gun policy, explains the observed difference in murder rates.

⁴ We use the lower-case δ to denote the value of the test statistic for your specific sample, to distinguish it from the Δ 's simulated under permutation.

same thing as *accepting* the null hypothesis as truth. To use a relationship metaphor: failing to reject the null hypothesis is not like getting married. It's more like agreeing not to break up this time.)

On the other hand, suppose that we almost always get a smaller value of Δ in a random map than we do in the real map. Then we will probably find it difficult to believe that the correlation in the real map arose due to chance. We will instead be forced to reject the null hypothesis and conclude that it provides a poor description of the observable data.

Which of these two possibilities seems to apply in Figure 7.5? Here, the actual difference of -0.8 for the real map in Figure 7.2 is shown as a vertical red line. Its position on the histogram suggests possibility (1) here: $\delta = -0.8$ is consistent with (although does not prove) the null hypothesis that other sources of random variation unrelated to state-level gun policy can explain the observed difference in murder rates between the passing-grade and the failing-grade states.

To summarize, the four steps we followed above were:

- (1) Choose a null hypothesis H_0 , the hypothesis that there is no systematic relationship between the predictor and response variables.
- (2) Choose a test statistic Δ that is sensitive to departures from the null hypothesis.
- (3) Approximate $P(\Delta | H_0)$, the sampling distribution of the test statistic T under the assumption that H_0 is true.
- (4) Assess whether the observed test statistic for your data, δ , is consistent with $P(\Delta | H_0)$.

For the gun-laws example, our test statistic in step (2) was the difference in medians between the “passing” states and the “failing” states. We then accomplished step (3) by randomly permuting the values of the predictor (gun laws) and recomputing the test statistic for the permuted data set. This shuffling procedure is called a permutation test when it’s done in the context of this broader four-step process. There are other ways of accomplishing step (3)—for example, by appealing to probability theory and doing some math. But the permutation test is nice because it works for any test statistic (like the difference of medians in the previous example), and it doesn’t require any strong assumptions.

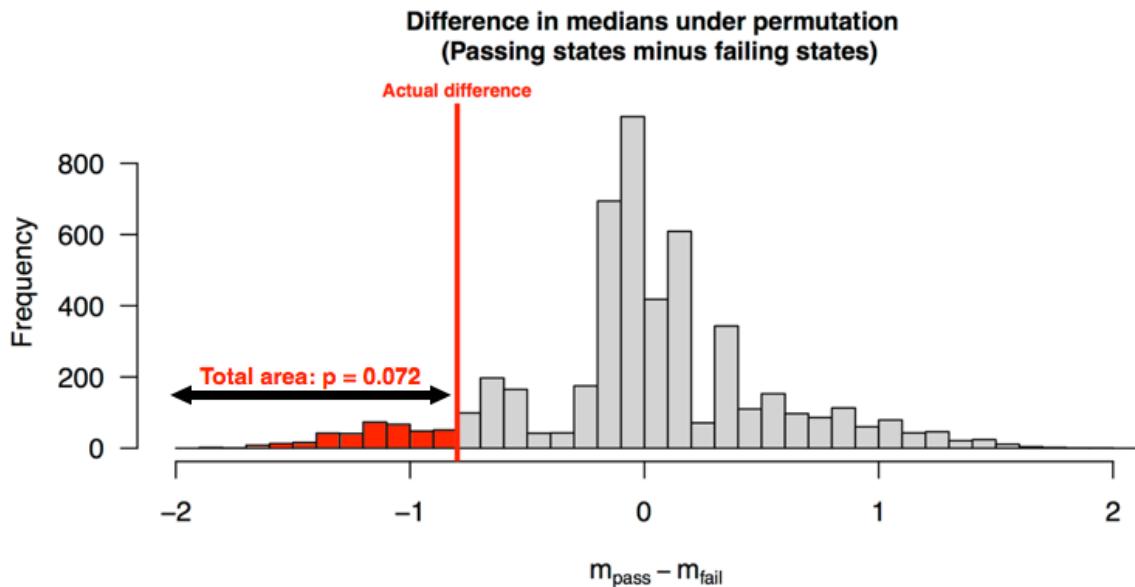


Figure 7.6: Assuming that the null hypothesis is true, the probability of observing a difference in medians at least as extreme as $\delta = -0.8$ is $p = 0.072$. This tail area to the left of $\delta = -0.8$ is the p -value of the test.

Using and interpreting p -values

There's one final question we haven't answered. How do we accomplish step (4) in the hypothesis test? That is, how can we measure whether the observed statistic for your data is consistent with the null hypothesis?

The typical approach here is to compute something called a *p-value*. Although we didn't call it by the name "*p-value*," this is exactly what we did for the Patriots' coin-flipping example at the beginning of the chapter.

Let's begin with a concise definition of a *p-value*, before we slowly unpack the definition (which is dense and non-intuitive).

A p-value is the probability of observing a test statistic as extreme as, or more extreme than, the test statistic actually observed, given that the null hypothesis is true. The way to compute the *p-value* is to calculate a *tail area* indicating what proportion of the sampling distribution, $P(\Delta | H_0)$, lies beyond the observed test statistic δ .

This all sounds a bit abstract, but is much easier to understand by example. Let's go back to the gun-laws hypothesis test, where we observed a difference in the medians of $\delta = -0.8$. If the null hypothesis were true, the probability of getting $\delta = -0.8$ (or

something more extreme in the negative direction) would be $p = 0.072$. We calculate this by taking the tail area under the sampling distribution that to the left of our observed δ of -0.8 . Figure 7.6 highlights this area in the left tail of the sampling distribution $P(\Delta | H_0)$. This is the p -value.

Using p -values has both advantages and disadvantages. The main advantage is that the p -value gives us a continuous measure of evidence against the null hypothesis. The smaller the p -value, the more unlikely it is that we would have seen our data under the null hypothesis, and therefore the greater the evidence the data provide that H_0 is false.

The main disadvantage is that the p -value is hard to interpret correctly. Just look at the definition—it's pretty counterintuitive! To avoid having to think too hard about what a p -value actually means, people often take $p \leq 0.05$ as a very important threshold that demarcates “significant” ($p \leq 0.05$) from “insignificant” ($p > 0.05$) results. While there are some legitimate reasons⁵ for thinking in these terms, in practice, the $p \leq 0.05$ criterion can feel pretty silly. After all, there isn't some magical threshold at which a result becomes important: in all practical terms, $p = .049$ and $p = .051$ are nearly identical in terms of the amount of evidence they provide against a null hypothesis.

Because of how counterintuitive p -values are, people make mistakes with them all the time, even (perhaps especially) people with Ph.D.'s quoting p -values in original research papers. Here is some advice about a few common misinterpretations:

- The p -value is *not* the probability that the null hypothesis is true, given that we have observed our statistic.
- The p -value is *not* the probability of having observed our statistic, given that the null hypothesis is true. Rather, it is the probability of having observed our statistic, *or any more extreme statistic*, given that the null hypothesis is true.
- The p -value is *not* the probability that your procedure will falsely reject the null hypothesis, given that the null hypothesis is true.⁶

The moral of the story is: always be careful when quoting or interpreting p -values. In many circumstances, a better question to ask than “what is the p -value?” is “what is a plausible range for the size of the effect?” This question can be answered with a confidence interval.⁷

⁵ If you are interested in these reasons, you should read up on the Neyman–Pearson school of hypothesis testing.

⁶ To get a guarantee of this sort, you have to set up a pre-specified rejection region for your p -value (like 0.05), in which case the size of that rejection region—and not the observed p -value itself—can be interpreted as the probability that your procedure will reject the null hypothesis, given that the null hypothesis is true. As above: if you're interested, read about the Neyman–Pearson approach to testing.

⁷ In this case, you could get a confidence interval by bootstrapping the difference in medians between the two groups of states.

Hypothesis testing in regression

To finish off this chapter, we will show how the permutation-testing framework can be used to answer questions about partial relationships in multiple regression modeling.

In a previous chapter, we asked the following question about houses in Saratoga, NY: what is the partial relationship between heating system type (gas, electric, or fuel oil) and sale price, once we adjust for the effect of living area, lot size, and the number of fireplaces? We fit a multiple regression model with these four predictors, which led to the following equation:

$$\begin{aligned} \text{Price} = & \$29868 + 105.3 \cdot \text{SqFt} + 2705 \cdot \log(\text{Acres}) + 7546 \cdot \text{Fireplaces} \\ & - 14010 \cdot \mathbf{1}_{\{\text{fuel} = \text{electric}\}} - 15879 \cdot \mathbf{1}_{\{\text{fuel} = \text{oil}\}} + \text{Residual}. \end{aligned}$$

Remember that the baseline case here is gas heating, since it has no dummy variable. Our model estimated the premium associated with gas heating to be about \$14,000 over electric heating, and about \$16,000 over fuel-oil heating.

But are these differences due to heating-system type statistically significant, or could they be explained due to chance?

To answer this question, you could look at the confidence intervals for every coefficient associated with the heating-system variable, just as we learned to do in the chapter on multiple regression. The main difference is that before, we had one coefficient to look at, whereas now we have two: one dummy variable for fuel = electric, and one for fuel = oil. Two coefficients means two confidence intervals to look at.

Sometimes this strategy—that is, looking at the confidence intervals for all coefficients associated with a single variable—works just fine. For example, when the confidence intervals for all coefficients associated with a single variable are very far from zero, it's pretty obvious that the categorical variable in question is statistically significant.

But at other times, this strategy can lead to ambiguous results. In the context of the heating-system type variable, what if the 95% confidence interval for one dummy-variable coefficient contains zero, but the other doesn't? Or what if both confidence intervals contain zero, but just barely? Should we say that heating-system type is significant or not? This potential for ambiguous confidence intervals gets even worse when your categorical variable has more than just a few levels, because then there will be many more confi-

dence intervals to look at.

The core of the difficulty here is that we want to assess the significance of the heating-system variable itself, not the significance of any individual *level* of that variable. To assess the significance of the whole variable, with all of its levels, we'll use a permutation test. Specifically, we will compare two models:

- The *full model*, which contains variables for square footage, lot size, number of fireplaces, and heating system.
- The *reduced model*, which contains variables for square footage, lot size, and number of fireplaces, but not for heating system. We say that the reduced model is *nested* within the full model, since it contains a subset of the variables in the full model, but no additional variables.

As always, we must start by specifying H_0 . Loosely speaking, our null hypothesis is that the reduced model provides an adequate description of house prices, and that the full model is needlessly complex. To be a bit more precise: the null hypothesis is that *there is no partial relationship* between heating system and house prices, once we adjust for square footage, lot size, and number of fireplaces. This implies that all of the *true* dummy variable coefficients for heating-system type are zero.

Next, we must pick a test statistic. A natural way to assess the evidence against the null hypothesis is to use improvement in R^2 under the full model, compared to the reduced model. This is the same quantity we look at when assessing the importance of a variable in an ANOVA table. The idea is simple: if we see a big jump in R^2 when moving from the reduced to the full model, then the variable we added (here, heating system) is important for predicting the outcome, and the null hypothesis of no partial relationship is probably wrong.

You might wonder here: why not use the coefficients on the dummy variables for heating-system type as test statistics? The reason is that there are two such coefficients (or in general, $K - 1$ coefficients for a categorical variable with K levels). But we need a single number to use as our test statistic in a permutation test. Therefore we use R^2 : it is a single number that summarizes the predictive improvement of the full model over the reduced model.

Of course, even if we were to add a useless predictor to the reduced model, we would expect R^2 to go up, at least by a little bit, since the model would have more degrees of freedom (i.e. param-

Remember the four basic steps in a permutation test:

- (1) Choose a null hypothesis H_0 .
- (2) Choose a test statistic Δ that is sensitive to departures from the null hypothesis.
- (3) Repeatedly shuffle the predictor of interest and recalculate the test statistic after each shuffle, to approximate $P(\Delta \mid H_0)$, the sampling distribution of the test statistic T under the assumption that H_0 is true.
- (4) Check whether the observed test statistic for your data, δ , is consistent with $P(\Delta \mid H_0)$.

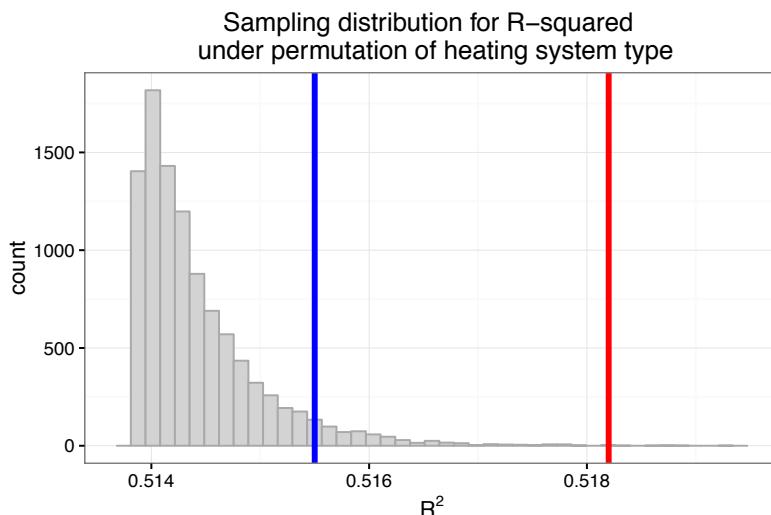


Figure 7.7: Sampling distribution of R^2 under the null hypothesis that there is no partial relationship between heating system and price after adjusting for effects due to square footage, lot size, and number of fireplaces. The blue vertical line marks the 95th percentile of the sampling distribution (and so corresponds to a rejection region at the 5% level). The red line marks the actual value of $R^2 = 0.518$ when we fit the full model by adding heating system to a model already containing the other three variables.

eters) that it can use to predict the observed outcome. Therefore, a more precise way of stating our null hypothesis is that, when we add heating system to a model already containing variables for square footage, lot size, and number of fireplaces, the improvement we see in R^2 could plausibly be explained by chance, even if this variable had no partial relationship with price.

To carry out a hypothesis test, we need to approximate the sampling distribution of R^2 under the null hypothesis. We will do so by repeatedly shuffling the heating system for every house (keeping all other variables the same), and re-fitting our model to each permuted data set. This breaks any partial relationship between heating system and price that may be present in our data. It tells us how big an improvement in R^2 we'd expect to see when fitting the full model, even if the null hypothesis were true.

This sampling distribution is shown in Figure 7.7, which was generated by fitting the model to 10,000 data sets in which the heating-system variable had been randomly shuffled, but where the response and the variables in the reduced model have been left alone. As expected, R^2 of the full model under permutation is always bigger than the value of $R^2 = 0.513$ from the reduced model—but rarely by much. The blue line at $R^2 = 0.5155$ shows the 95th percentile of the sampling distribution (i.e. the critical value for a rejection region at the 5% level). The red line shows the actual value of $R^2 = 0.518$ from the full model fit the original

data set (i.e. with no shuffling). This test statistic falls far beyond the 5% rejection region. We therefore reject the null hypothesis and conclude that there is statistically significant evidence for an effect on price due to heating-system type.

One key point here is that we shuffled *only* heating-system type—or in general, whatever variable is being tested. We don’t shuffle the response or any of the other variables. That’s because we are interested in a partial relationship between heating-system type and price. Partial relationships are always defined with respect to a specific context of other control variables, and we have to leave these control variables as they are in order to provide the correct context for that partial relationship to be measured.

To summarize: we can compare any two nested models using a permutation test based on R^2 , regardless of whether the variable in question is categorical or numerical. To do so, we repeatedly shuffle the extra variable in the full model—without shuffling either the response or the control variables (i.e. those that also appear in the reduced model). We fit the full model to each shuffled data set, and we track the sampling distribution of R^2 . We then compare this distribution with the R^2 we get when fitting the full model to the *actual* data set. If the actual R^2 is a lot bigger than what we’d expect under the sampling distribution for R^2 that we get under the permutation test, then we conclude that the extra variable in the full model is statistically significant.

F tests and the normal linear regression model. Most statistical software will produce an ANOVA table with an associated p -value for all variables. These p -values are approximations to the p -values that you’d get if you ran sequential permutation tests, adding and testing one variable at a time as you construct the ANOVA table. To be a bit more specific, they correspond to something called an F test under the normal linear regression model that we met awhile back:

$$y_i = \beta_0 + \sum_{j=1}^p \beta_j x_{ij} + e_i, \quad e_i \sim N(0, \sigma^2).$$

You might want to revisit the discussion of the normal linear regression model starting on page 120. But the upshot is that an F test is conceptually similar to a permutation test based on R^2 —and if you’re happy with the assumption of normally distributed residuals, you can treat the p -values from these two tests as virtually interchangeable.⁸

⁸ If you’re not happy with this assumption, then you’re better off with the permutation test.

Building predictive models

Building predictive models

Suppose you have a house in Saratoga, NY that you’re about to put up for sale. It’s a 1900 square-foot house on a 0.7-acre lot.

It has 3 bedrooms, 2.5 bathrooms,¹ 1 fireplace, gas heating, and central air conditioning. The house was built 16 years ago. How much would you expect it to sell for?

Although we’ve been focusing on only a few variables of interest so far, our house-price data set actually has information on all these variables, and a few more besides. A great way to assess the value of the house is to use the available data to fit a multiple regression model for its price, given its features. We can then use this model to make a best guess for the price of a house with some particular combination of features—and, optionally, to form a prediction interval that quantifies the uncertainty of our guess.

We refer to this as the process of *building a predictive model*. Although we will still use multiple regression, the goal here is slightly different than in the previous examples. Here, we don’t care so much about isolating and interpreting one particular partial relationship (like that between fireplaces and price). Instead, we just want the most accurate predictions possible.

The key principle in building predictive models is *Occam’s razor*, which is the broader philosophical idea that models should be only as complex as they need to be in order to explain reality well. The principle is named after a medieval English theologian called William of Occam. Since he wrote in Latin, he put it like this: *Frustra fit per plura quod potest fieri per pauciora* (“It is futile to do with more things than which can be done with fewer.”) A more modern formulation of Occam’s razor might be the **KISS rule**: keep it simple, stupid.

In regression modeling, this principle is especially relevant for *variable selection*—that is, deciding which possible predictor variables to add to a model, and which to leave out. In this context,

¹ A half-bathroom has a toilet but no bath or shower.

Occam's razor is about finding the right set of variables to include so that we fit the data, without overfitting the data. Another way of saying this is that we want to find the patterns in the data, without memorizing the noise.

In this chapter, we'll consider two main questions:

- (1) How can we measure the predictive power of a model?
- (2) How can we find a model with good predictive power?

Measuring generalization error

To understand how we measure the predictive power of a regression model, we first need a bit of notation. Specifically, let's say that we have estimated a multiple regression model with p predictors (x_1, x_2, \dots, x_p) to some data, giving us coefficients $(\hat{\beta}_0, \hat{\beta}_1, \dots, \hat{\beta}_p)$. Now we encounter a new case, not in our original data set. We'll let $x^* = (x_1^*, x_2^*, \dots, x_p^*)$ be the predictor variables for this new case, and y^* denote the corresponding response. We will use the fitted regression model, together with x^* , to make a prediction for y^* :

$$\hat{y}^* = \hat{\beta}_0 + \sum_{j=1}^p \hat{\beta}_j x_j^*.$$

Our goal is to make the *generalization error*—that is, the difference between y^* and \hat{y}^* —as small as possible, on average.

A natural way to measure the generalization error of a regression model is using a quantity called the *mean-squared predictive error*, or MSPE. The mean-squared predictive error is a property of a fitted model, not an individual data point. It summarizes the magnitude of the errors we typically make when we use the model to make predictions \hat{y}^* on new data:

MSPE = Average value of $(y^* - \hat{y}^*)^2$ when sampling new data points.

Here a “new” data point means one that hasn't been used to fit the model. You'll notice that, in calculating MSPE, we square the prediction error $y^* - \hat{y}^*$ so that both positive and negative errors count equally.

Low mean-squared predictive error means that $y^* - \hat{y}^*$ tends to be close to zero when we sample new data points. This gives us a simple principle for building a predictive model: find the model (i.e. the set of variables to include) with the lowest mean-squared predictive error.

Estimating the mean-squared predictive error

Conceptually, the simplest way to estimate the mean-squared predictive error of a regression model is to actually collect new data and calculate the average predictive error made by our model. Specifically, suppose that, after having fit our model in the first place, we go out there and collect n^* brand new data points, with responses y_i^* and predictors $(x_{i1}^*, \dots, x_{ip}^*)$. We can then estimate the mean-squared predictive error of our model in two simple steps:

1. Form the prediction for each new data point:

$$\hat{y}_i^* = \hat{\beta}_0 + \sum_{j=1}^p \hat{\beta}_j x_{ij}^*.$$

2. Calculate the average squared error of your predictions:

$$\widehat{\text{MSPE}}_{\text{out}} = \frac{1}{n^*} \sum_{i=1}^{n^*} (\hat{y}_i^* - y_i^*)^2.$$

Notice that we put a hat on MSPE, because the expression on the right-hand side is merely an *estimate* of the true mean-squared predictive error, calculated using a specific sample of new data points. (Calculating the *true* MSPE would require us, in principle, to average over all possible samples of new data points, which is obviously impractical.) We also use the subscript “out” to indicate that it is an *out-of-sample* measure—that is, calculated on new data, that falls outside of our original sample.

Conventionally, we report the square root of $\widehat{\text{MSPE}}_{\text{out}}$ (which is called *root mean-squared predictive error*, or RMSPE), because this has the same units as the original y variable. You can think of the RMSPE as the standard deviation of future forecasting errors made by your model.

Assuming your new sample size n^* isn’t too small, these two steps are a nearly foolproof way to estimate the mean-squared predictive error of your model. The drawback, however, is obvious: you need a brand new data set, above and beyond the original data set that you used to fit the model in the first place. This new data set might be expensive or impractical to collect.

Thus we’re usually left in the position of needing to estimate the mean-squared predictive error of a model, without having access to a “new” data set. For this reason, the usual practice is

make a *train/test split* of your data: that is, to randomly split your original data set into two subsets, called the *training* and *testing* sets.

- The training set is used only to fit (“train”) the model—that is, to estimate the coefficients $(\hat{\beta}_0, \hat{\beta}_1, \dots, \hat{\beta}_p)$.
- The testing set is used only to estimate the mean-squared predictive error of the model. It is not used at all to fit the model. For this reason, the testing set is sometimes referred to as the “hold-out set,” since it is held out of the model-fitting process.

From this description, it should be clear that the training set plays the role of the “old” data, while the testing set plays the role of the “new” data.

This gives us a simple three-step procedure for choosing between several candidate models (i.e. different possible sets of variables to include).

- (1) Split your data into training and testing sets.
- (2) For each candidate model:
 - A. Fit the model using the training set.
 - B. Calculate \widehat{MSPE}_{out} for that model using the testing set.
- (3) Choose the model with the lowest value of \widehat{MSPE}_{out} .

Choosing the training and testing sets. A key principle here is that you must *randomly* split your data into a training set and testing set. Splitting your data nonrandomly—for example, taking the first 800 rows of your data as a training set, and the last 200 rows as a testing set—may mean that your training and testing sets are systematically different from one another. If this happens, your estimate of the mean-squared prediction error can be way off.

How much of the data should you reserve for the testing set? There are no hard-and-fast rules here. A common rule of thumb is to use about 75% of the data to train the model, and 25% to test it. Thus, for example, if you had 100 data points, you would randomly sample 75 of them to use for model training, and the remaining 25 to estimate the mean-squared predictive error. But other ratios (like 50% training, or 90% training) are common, too.

My general guideline is that the more data I have, the larger the fraction of that data I will use for training the predictive model.

Thus with only 100 data points, I might use a 75/25 split between training and testing; but with 10,000 data points, I might use more like a 90/10 split between training and testing. That's because estimating the model itself is generally harder than estimating the mean-squared predictive error.² Therefore, as more data accumulates, I like to preferentially allocate more of that data towards the intrinsically harder task of model estimation, rather than MSPE estimation.

² By "harder" here, I mean "subject to more sources of statistical error," as opposed to computationally more difficult.

Averaging over different test sets. It's a good idea to average your estimate of the mean-squared predictive error over several different train/test splits of the data set. This reduces the dependence of $\widehat{\text{MSPE}}_{\text{out}}$ on the particular random split into training and testing sets that you happened to choose. One simple way to do this is average your estimate of MSPE over many different random splits of the data set into training and testing sets. Somewhere between 5 and 100 splits is typical, depending on the computational resources available (more is better, to reduce Monte Carlo variability).

Another classic way to estimate MSPE it is to divide your data set into K non-overlapping chunks, called *folds*. You then average your estimate of MPSE over K different testing sets, one corresponding to each fold of the data. This technique is called *cross validation*. A typical choice of K is five, which gives us five-fold cross validation. So when testing on the first fold, you use folds 2-5 to train the model; when testing on fold 2, you use folds 1 and 3-5 to train the model; and so forth.

Can we use the original data to estimate the MSPE?

A reasonable question is: why do even we need a new data set to estimate the mean-squared prediction error? After all, our fitted model has residuals, $e_i = y_i - \hat{y}_i$, which tell us how much our model has "missed" each data point in our sample. Why can't we just use the residual variance, s_e^2 , to estimate the MSPE? This approach sounds great on the surface, in that we'd expect the past errors to provide a good guide to the likely magnitude of future errors. Thus you might be tempted to use the *in-sample* estimate of MSPE, denoted

$$\widehat{\text{MSPE}}_{\text{in}} = s_e^2 = \frac{1}{n-p} \sum_{i=1}^n (y_i - \hat{y}_i)^2,$$

where we recall that p is the number of parameters in the model.

Using $\widehat{\text{MSPE}}_{\text{in}}$ certainly removes the need to collect a new data set. This turns out, however, to be a false economy: $\widehat{\text{MSPE}}_{\text{in}}$ is usually too optimistic as an estimate of a model's generalization error. Practically speaking, this means the following. When we use $\widehat{\text{MSPE}}_{\text{in}}$ to quantify the *in-sample* error of a model, and then we actually go out and take new data to calculate the *out-of-sample* generalization error $\widehat{\text{MSPE}}_{\text{out}}$, we tend to discover that the out-of-sample error is larger—sometimes much larger! This is called overfitting, and it is especially likely to happen when the size of the data set is small, or when the model we're fitting is very complex (i.e. has lots of parameters).

An example

Let's see these ideas in practice, by comparing three predictive models for house prices in Saratoga, New York. Our models will draw from the following set of variables:

- lot size, in acres
- age of house, in years
- living area of house, in square feet
- percentage of residents in neighborhood with college degree
- number of bedrooms
- number of bathrooms
- number of total rooms
- number of fireplaces
- heating system type (hot air, hot water, electric)
- fuel system type (gas, fuel oil, electric)
- central air conditioning (yes or no)

We'll consider three possible models for price constructed from these 11 predictors.

Small model: price versus lot size, bedrooms, and bathrooms (4 total parameters, including the intercept).

Medium model: price versus all variables above, main effects only (14 total parameters, including the dummy variables).

Big model: price versus all variables listed above, together with all pairwise interactions between these variables (90 total parameters, include dummy variables and interactions).

Table 8.1 shows both $\widehat{\text{MSPE}}_{\text{in}}$ and $\widehat{\text{MSPE}}_{\text{out}}$ for these three models. To calculate $\widehat{\text{MSPE}}_{\text{out}}$, we used 80% of the data as a training

| | In-sample RMSPE | Out-of-sample RMSPE | Difference |
|------------------------|-----------------|---------------------|------------|
| Small model: underfit | \$76,144 | \$76,229 | \$85 |
| Medium model: good fit | \$65,315 | \$65,719 | \$403 |
| Big model: overfit | \$61,817 | \$71,426 | \$9,609 |

set, and the remaining 20% as a test set, and we averaged over 100 different random train/test splits of the data. The final column, labeled “difference,” shows the difference between the in-sample and out-of-sample estimates of prediction error.

There are a few observations to take away from Table 8.1. The first is that the big model (with all the main effects and interactions) has the lowest in-sample error. With a residual standard deviation of \$61,817, it seems nearly \$3,500 more accurate than the medium model, which is next best. This is a special case of a very general phenomenon: a more complex model will always fit the data better, because it has more degrees of freedom to play with.

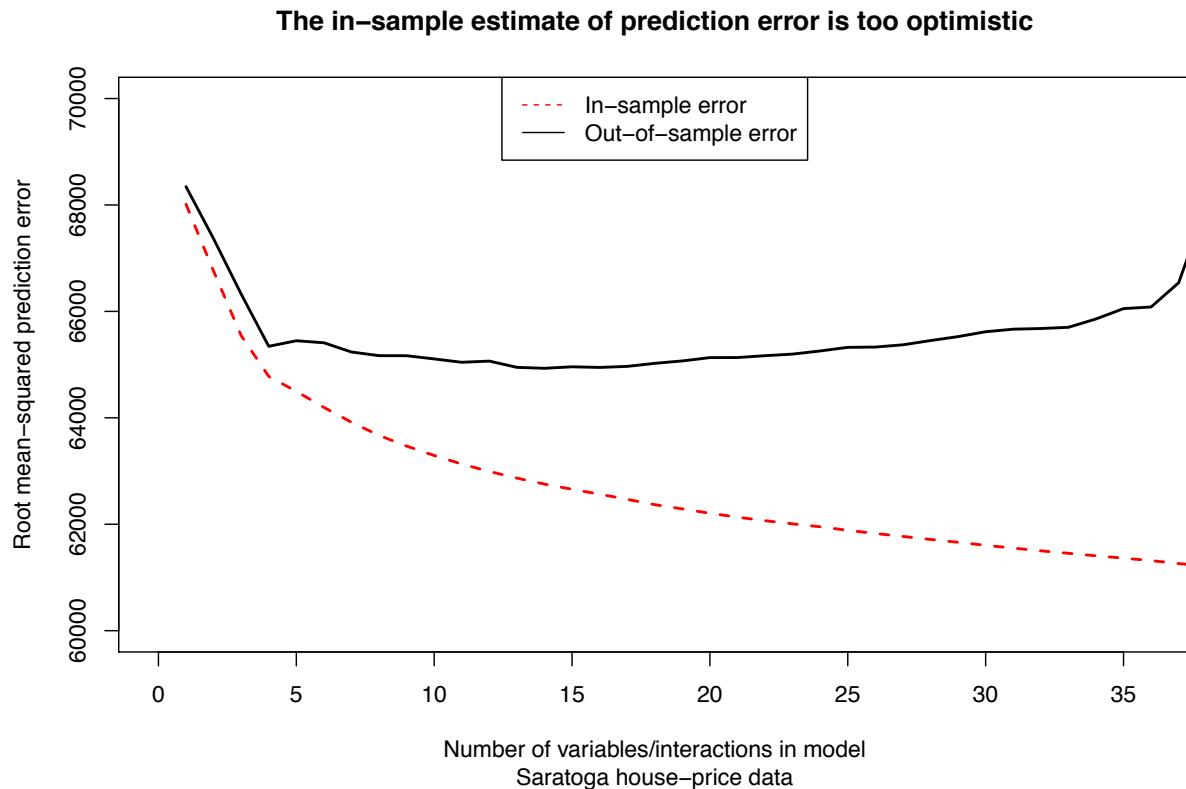
However, the *out-of-sample* measure of predictive error tells a different story. Here, the medium-sized model is clearly the winner. Its predictions on new data are off by about \$65,719, on average, which is nearly \$6,000 better than the big model.

Finally, notice how severely degraded the predictions of the big model become when moving from old (in-sample) data to new (out-of-sample) data: about \$9,600 worse, on average. This kind of degradation is a telltale sign of overfitting. The medium model suffers only a mild degradation in performance on new data, while the small model suffers hardly any degradation at all—although it’s still not competitive on the out-of-sample measure, because it wasn’t that good to begin with. This is also a special case of a more general phenomenon: *some* degradation in predictive performance on out-of-sample versus in-sample data is inevitable, but simpler models tend to degrade a lot less.

Figure 8.1 demonstrates this point visually. Starting from a very simple model of price (using only lot size as a predictor), we’ve added one variable or interaction at a time³ from the list on page 168. For each new variable or interaction, we recalculated both the in-sample (\widehat{MSPE}_{in}) and out-of-sample (\widehat{MSPE}_{out}) estimates of the generalization error. As we add variables, the out-of-sample error initially gets smaller, reflecting a better fitting model that still generalizes well to new data. But after 15 or 20 variables,

Table 8.1: In-sample versus out-of-sample estimates of the root mean-squared predictive error for three models of house prices in Saratoga, NY. The “difference” column shows the difference between the in-sample and out-of-sample estimates. The big model has a very large difference (over \$9,000), indicating that the in-sample estimate is way too optimistic, and that the model is probably overfit to the data.

³ To be specific here, at each stage we added the single variable or interaction that most improved the fit of the model. See the next section on stepwise selection.



eventually the out-of-sample error starts creeping back up, due to overfitting. The in-sample estimate of error, however, keeps going down, falling even further out of line with the real out-of-sample error as we add more variables to the model.

In summary, you should remember the basic mantra of predictive model building: out-of-sample error is larger than in-sample error, especially for bigger models. If you care about minimizing out-of-sample error, you should always use an out-of-sample estimate of a model's MSPE, to make sure that you're not overfitting the original data. Our goal here should be obvious: to find the “turning point” in Figure 8.1, and to stop adding variables before we start overfitting.

Figure 8.1: Starting from a small pricing model with just lot size as a predictor, we've added one variable or interaction at a time from the list on page 168. The red line shows the in-sample estimate of error, while the black line shows the out-of-sample estimate. After we add about 15 variables and interactions, the out-of-sample error starts to creep back up. Clearly the in-sample estimate is too optimistic, especially as the model gets more complex.

Iterative model building via stepwise selection

Now that we know how to measure generalization error of a model, we're ready to introduce the overall steps in the process of building and using a predictive model from a set of candidate variables x_1, x_2 , etc. We sometimes use the term *scope* to refer to this set of candidate variables.

The seemingly obvious approach is to fit all possible models under consideration to a training set, and to measure the generalization error of each one on a testing set. If you have only a few variables, this will work fine. For example, with only 2 variables, there are only $2^2 = 4$ possible models to consider: the first variable in, the second variable in, both variables in, or both variables out. You can fit and test those four models in no time. This is called *exhaustive enumeration*.

However, if there are lots of variables, exhaustive enumeration of all the models becomes a lot harder to do, for the simple fact that it's too exhausting—there are too many models to consider. For example, suppose we have 10 possible variables, each of which we could put in or leave out of the model. Then there are $2^{10} = 1024$ possible models to consider, since each variable could be in or out in any combination. That's painful enough. But if there are 100 possible variables, there are 2^{100} possible models to consider. That's 1 *nonillion* models—about 10^{30} , or a thousand billion billion billion. This number is larger than the number of atoms in a human body.

You will quite obviously never be able to fit all these countless billions of models, much less compare their generalization errors on a testing set, even with the most powerful computer on earth. Moreover, that's for just 100 candidate variables *with main effects only*. Ideally, we'd like the capacity to build a model using many more candidate variables than that, or to include the possibility of interactions among the variables.

Thus a more practical approach to model-building is *iterative*: that is, to start somewhere reasonable, and to make small changes to the model, one variable at a time. Model-building in this iterative way is really a three-step process:

- (1) Choose a baseline model, consisting of initial set of predictor variables to include in the model, including appropriate transformations, polynomial terms and interactions. Exploratory

data analysis (i.e. plotting your data) will generally help you get started here, in that it will reveal obvious relationships in the data. Then fit the model for y versus these initial predictors.

- (2) Check the model. If necessary, change what variables are included, what transformations are used, etc.:
 - (a) Are the assumptions of the model met? This is generally addressed using residual plots, of the kind shown in Figures 6.7 and 6.8. This allows you to assess whether the response varies linearly with the predictors, whether there are any drastic outliers, etc.
 - (b) Are we missing any important variables or interactions? This is generally addressed by *adding* candidate variables or interactions to the model from step (1), to see how much each one improves the generalization error (MSPE).
 - (c) Are there signs that the model might be overfitting the data? This is generally addressed by *deleting* variables or interactions that are already in the model, to see if doing so actually improves the model's generalization error.

You may need to iterate these three questions a few times, going through many rounds of adding or deleting variables, before you're satisfied with your final model. Remember that the best way to measure generalization error is using an out-of-sample measure, like $\widehat{\text{MSPE}}_{\text{out}}$ derived from a train/test split of the data.

Once you're happy with the model itself, then you can. . . .

- (3) Use your fitted model to form predictions (and optionally, prediction intervals) for your new data points.

Can this process be automated?

In this three-step process, step 1 (start somewhere reasonable) and step 3 (use the final model) are usually pretty easy. The part where you'll spend the vast majority of your time and effort is step 2, when you consider many different possible variables to add or delete to the current model, and check how much they improve or degrade the generalization error of that model.

This is a lot easier than considering all possible combinations of variables in or out. But with lots of candidate variables, even this

iterative process can get super tedious. A natural question is, can it be automated?

The answer is: sort of. We can easily write a computer program that will automatically check for iterative improvements to some baseline (“working”) model, using an algorithm called *stepwise selection*:

- (1) From among a candidate set of variables (the scope), check all possible one-variable additions or deletions from the working model;
- (2) Choose the single addition or deletion that yields the best improvement to the model’s generalization error. This becomes the new “working model.”
- (3) Iteratively repeat steps (1) and (2) until no further improvement to the model is possible.

The algorithm terminates when it cannot find any one-variable additions or deletions that will improve the generalization error of the working model.

Some caveats. Stepwise selection tends to work tolerably well in practice. But it’s far from perfect, and there are some important caveats. Here are three; the first one is minor, but the second two are pretty major.

First, if you run stepwise selection from two different baseline models, you will probably end up with two different final models. This tends not to be a huge deal in practice, however, because the two final models usually have similar mean-squared predictive errors. Remember, when we’re using stepwise selection, we don’t care too much about *which* combinations of variables we pick, as long as we get good generalization error. Especially if the predictors are correlated with each other, one set of variables might be just as good as another set of similar (correlated) variables.

Second, stepwise selection usually involves some approximation. Specifically, at each step of stepwise selection, we have to compare the generalization errors of many possible models. Most statistical software will perform this comparison *not* by actually calculating $\widehat{\text{MSPE}}_{\text{out}}$ on some test data, but rather using one of several possible heuristic approximations for MSPE. The most common one is called the AIC approximation:⁴

$$\widehat{\text{MSPE}}_{\text{AIC}} = \widehat{\text{MSPE}}_{\text{in}} \left(1 + \frac{p}{n} \right) = s_e^2 \left(1 + \frac{p}{n} \right),$$

⁴ In case you’re curious, AIC stands for “Akaike information criterion.” If you find yourself reading about AIC on Wikipedia or somewhere similar, it will look absolutely nothing like the equation I’ve written here. The connection is via a related idea called “Mallows’ C_p statistic,” which you can [read about here](#).

where n is the sample size and p is the number of parameters in the model.

The AIC estimate of mean-squared predictive error is not a true out-of-sample estimate, like $\widehat{\text{MSPE}}_{\text{out}}$. Rather, it is like an “inflated” or “penalized” version of the in-sample estimate, $\widehat{\text{MSPE}}_{\text{in}} = s_e^2$, which we know is too optimistic. The inflation factor of $(1 + p/n)$ is always larger than 1, and so $\widehat{\text{MSPE}}_{\text{AIC}}$ is always larger than $\widehat{\text{MSPE}}_{\text{in}}$. But the more parameters p you have relative to data points n , the larger the inflation factor gets. It’s important to emphasize that $\widehat{\text{MSPE}}_{\text{AIC}}$ is just an approximation to $\widehat{\text{MSPE}}_{\text{out}}$. It’s a better approximation than $\widehat{\text{MSPE}}_{\text{in}}$, but it still relies upon some pretty specific mathematical assumptions that can easily be wrong in practice.

The third and most important caveat is that, when using any kind of automatic variable-selection procedure like stepwise selection, we lose the ability to use our eyes and our brains each step of the way. We can’t plot the residuals to check for outliers or violations of the model assumptions, and we can’t ensure that the combination of variables visited by the algorithm make any sense, substantively speaking. It’s worth keeping in mind that your eyes, your brain, and your computer are your three most powerful tools for statistical reasoning. In stepwise selection, you’re taking two of these tools out of the process, for the sake of doing a lot of brute-force calculations very quickly.

None of these caveats are meant to imply that you *shouldn’t* use stepwise selection—merely that you shouldn’t view the algorithm as having God-like powers for discerning the single best model, or treat it as an excuse to be careless. You should instead proceed cautiously. Always verify that the stepwise-selected model makes sense and doesn’t violate any crucial assumptions. It’s also a good idea to perform a quick train/test split of your data and compute $\widehat{\text{MSPE}}_{\text{out}}$ for your final model, just as a sanity check, to make sure that you’re actually improving the generalization error versus your baseline model.

9

Understanding cause and effect

Statistical questions versus causal questions

WHY have some nations become rich while others have remained poor? Do small class sizes improve student achievement? Does following a Mediterranean diet rich in vegetables and olive oil reduce your risk of a heart attack? Does a “green” certification (like LEED, for [Leadership in Energy and Environmental Design](#)) improve the value of a commercial property?

Questions of cause and effect like these are, fundamentally, questions about *counterfactual statements*. A counterfactual is an if–then statement about something that has not actually occurred. For example: “If Colt McCoy had not been injured early in the [2010 National Championship football game](#), then the Texas Longhorns would have beaten Alabama.” If you judge this counterfactual statement to be true—and who but the most hopelessly blinkered Crimson Tide fan doesn’t?—then you might say that Colt McCoy’s injury caused the Longhorns’ defeat.

Statistical questions, on the other hand, are about correlations. This makes them fundamentally different from causal questions.

- Causal: “If we invested more money in our school system, how much faster would our economy grow?” Statistical: “In looking at data on a lot of countries, how are education spending and economic growth related?”
- Causal: “If I ate more vegetables than I do now, how much longer would I live?” Statistical: “Do people who eat a lot of vegetables live longer, on average, than people who don’t?”
- Causal: “If we hire extra teachers at our school and reduce our class sizes, will our students’ test scores improve?” Statistical: “Do students in smaller classes tend to have higher test scores?”

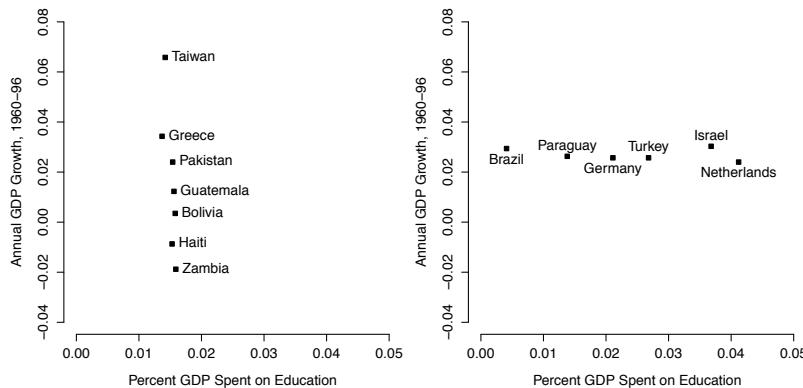


Figure 9.1: Two egregious examples of selective reporting.

Causal questions all invoke some kind of hypothetical intervention, where one thing is changed and everything else is held equal. In such a hypothetical intervention, there is no competing explanation for what might be causing the change we expect to see—in our economy, our lifespan, our students’ test scores, a football game, or whatever outcome we’re interested in.

Statistical questions, on the other hand, are about the patterns we observe in the real world. And the real world is rarely so simple as the hypothetical interventions we imagine. For example, people who eat more vegetables live longer—that’s a clear pattern. But those same people also tend to exercise more, live in better housing, and have higher-status jobs. These other factors are *confounders*. A confounder is a competing explanation—some other factor correlated with both the “treatment” assignment (whether someone eats vegetables) and the response (lifespan). So in light of these confounders, how do we know it’s the vegetables, rather than all that other stuff, that’s making veggie-eaters live longer?

This is just a specific version of the general question we’ll address in this chapter: under what circumstances can causal questions be answered using statistics?

Good evidence . . . and bad

Most of the cause-and-effect reasoning that you’ll see out there in the real world is of depressingly poor quality. A common flaw is *cherry picking*: that is, pointing to data that seems to confirm some argument, while ignoring contradictory data.

Here’s an example. In the left panel in Figure 9.1 we see a

group of seven countries that all spend around 1.5% of their GDP on education, but with very different rates of economic growth for the 37 years spanning 1960 to 1996. In the right panel, we see another group of six countries with very different levels of spending on education, but similar growth rates of 2–3%.

Both highly selective samples make it seem as though education and economic growth are barely related. If presented with the left panel alone, you'd be apt to conclude that the differences in growth rates must have been caused by something other than differences in education spending (of which there are none). Likewise, if presented with the right panel alone, you'd be apt to conclude that the large observed differences in education spending don't seem to have produced any difference in growth rates. The problem here isn't with the data—it's with the biased, highly selective *use* of that data.

This point seems almost obvious. Yet how tempting it is just to cherry pick and ignore the messy reality. Perhaps without even realizing it, we're all accustomed to seeing news stories that marshal highly selective evidence—usually even worse than that of Figure 9.1—on behalf of some plausible because-I-said-so story:

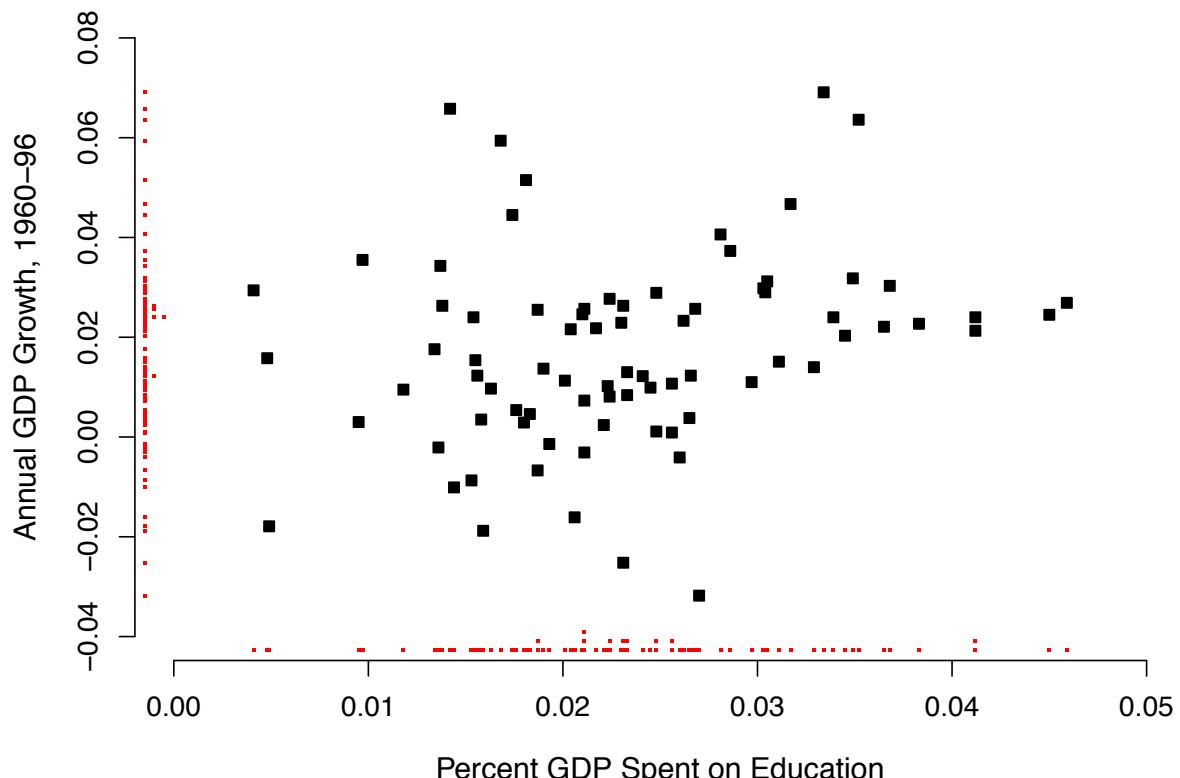
[H]igher levels of education are critical to economic growth. . . . Boston, where there is a high proportion of college graduates, is the perfect example. Well-educated people can react more quickly to technological changes and learn new skills more readily. Even without the climate advantages of a city like San Jose, California, Boston evolved into what we now think of as an “information city.” By comparison, Detroit, with lower levels of education, languished.¹

And this from a reporter who presumably has no hidden agenda. Notice how the selective reporting of evidence—one causal hypothesis, two data points—lends an air of such graceful inevitability to what is a startlingly superficial analysis of the diverging economic fates of Boston and Detroit over the last half century.

Of course, most bad arguments are harder to detect than this howler from the New York Times. After all, using data to understand cause-and-effect relationships is hard. For example, consider the following summary of a recent neuroscience study:

A study presented at the Society for Neuroscience meeting, in San Diego last week, shows people who start using marijuana at a young age have more cognitive shortfalls. Also, the more marijuana a person used in adolescence, the more trouble they had with focus and attention. “Early onset smokers

¹ “Economic Scene.” *New York Times* (Business section); August 5, 2004



have a different pattern of brain activity, plus got far fewer correct answers in a row and made way more errors on certain cognitive tests," says study author Staci Gruber.²

Did the marijuana smokers get less smart, or were the less-smart kids more likely to pick up a marijuana habit in the first place? It's an important question to consider in making drug policy, especially for states and countries where marijuana is legal. But can we know the answer on the basis of a study like this?

For another example, consider the bigger sample of countries in Figure 9.2, which provides a much more representative body of evidence on the GDP-versus-education story. This evidence takes the form of a scatter plot of GDP growth versus education spending for a sample of 79 countries worldwide. Notice the following two facts:

- (1) Of the 29 countries that spent less than 2% of GDP on education, 18 fall below the median growth rate (1.58%).

Figure 9.2: A scatter plot of GDP growth versus education spending for 79 countries. The tiny red dots clustered near the x and y axes are called *rug plots*. They are miniature histograms aligned with the axes of the predictor and the response.

² www.usatoday.com/yourlife/health/medical/pediatrics/2010-11-20-teendrugs22_ST_N.htm

- (2) Of the 18 countries that spent more than 3% of GDP on education, 16 fall above the median growth rate.

These two facts, together with the upward trend in the scatter plot, suggest that economic growth and education spending are correlated. But this does not settle the causal question. For example, it might be that countries spend a lot on education because they are rich, rather the other way around.

The generic difficulty is that there are many different ways that two variables X and Y can appear correlated.

- (1) *One-way causality*: the first domino falls, then the second; the rain falls, and the grass gets wet. (X causes Y directly.)

- (2) *Two-way causality*: flowers and honey bees prosper together.
(Both X and Y play a role in causing each other.)

- (3) *Common cause*: People who go to college tend to get higher-paying jobs than those who don't. Does education directly lead to better economic outcomes? Or are a good education and a good job both just markers of a person's underlying qualities? (The role of X in causing Y is hard to distinguish from the role of C , which we may not have observed.)

- (4) *Common effect*: either musical talent (X) or athletic talent (Y) will help you get into Harvard (Z). Among a population of Harvard freshmen, musical and athletic talent will thus appear negatively correlated, even if they are independent in the wider population. (X and Y both contribute to some common outcome C , inducing a correlation among a subset of the population defined by Z . This is often called Berkson's paradox; it is subtle, and we'll encounter it again.)

- (5) *Luck*: the observed correlation is a coincidence.

This is the point where most books remind you that “correlation does not imply causation.” Obviously. But if not to illuminate causes, what is the point of looking for correlations? Of course correlation does not imply causality, or else playing professional basketball would make you tall. But that hasn't stopped humans from learning that smoking causes cancer, or that lightning causes thunder, on the basis of observed correlations. The important question is: what distinguishes the good evidence-based arguments from the bad?

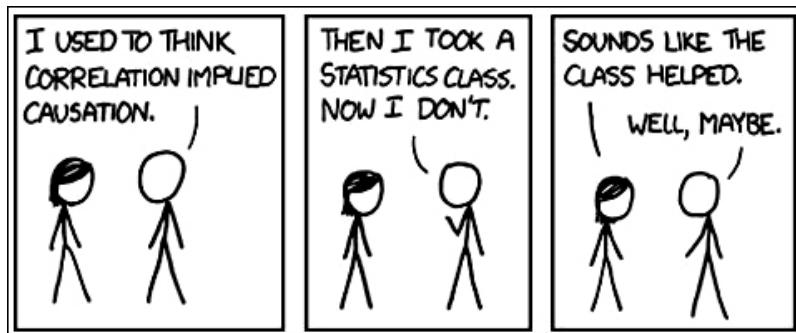


Figure 9.3: Originally published online at xkcd: <http://xkcd.com/552/>

Four common identification strategies

The key principle in using evidence to draw causal conclusions is that of a *balanced comparison*. To make things simple, we'll imagine that our predictor X is binary (i.e. has two groups), and we'll borrow the lingo of a clinical trial by referring to the two groups as the "treatment" and "control." To reach the conclusion that X causes Y , you must do two things: (1) *compare cases* in the treatment and control groups, to see how their Y values differ; and (2) *ensure balance*, by removing all other systematic differences between the cases in the treatment and control groups. Balance is crucial; it's what allows us to conclude that the differences in X (and not something else) cause the differences we observe in Y .

In general, there are four common ways to make a balanced comparison. These are often called *identification strategies*, in the sense that they are strategies for identifying a causal effect.

- (1) *Run a real experiment*, randomizing subjects to the treatment and control groups. The randomization will ensure that, on average, there are no systematic differences between the two groups, other than the treatment.
- (2) *Find a natural experiment*: that is, find a situation where the way that cases fall naturally into the treatment and control groups plausibly resembles a random assignment.
- (3) *Matching*: artificially construct a balanced data set from an unbalanced data set, by explicitly matching treated cases with similar control cases, and discarding the cases without a good match. This will correct for lack of balance between control and treatment groups.

(4) *Modeling*: use multiple regression modeling to adjust for confounders and isolate a partial relationship between the response and the treatment of interest.

We'll take each of these four ideas in turn.

The power of experiment

THE idea of an experiment is simple. If you want to know what would happen if you intervened in some system, then you should intervene, and measure what happens. There is simply no better way to establish that one thing causes another.

Indeed, one kind of experiment—the randomized, controlled clinical trial—is one of the most important medical innovations in history. Suppose we want to establish whether a brand new cholesterol drug—we'll call it Zapaclot—works better than the old drug. Also suppose that we've successfully recruited a large cohort of patients with high cholesterol. We know that diet and genes play a role here, but that drugs can help, too. We express this as

$$\text{Cholesterol} \sim \text{Diet} + \text{Genes} + \text{Drugs}.$$

Interpret the plus sign as the word “and,” not like formal addition: we're assuming that cholesterol depends upon diet, genes, and drugs, although we haven't said how. Of course, it's that third predictor in the model we care about; the first two, in addition to some others that we haven't listed, are potential confounders.

First, what not to do: don't proceed by giving Zapaclot to all the men and the old drug to all the women, or Zapaclot to all the marathon runners and the old drug to the couch potatoes. These highly non-random assignments would obviously bias any judgment about the relative effect of the new drug compared to the old one. We refer to this sort of thing as *selection bias*: that is, any bias in the selection of cases that receive the treatment. Moreover, you shouldn't just give the new drug to whomever wants it, or can afford it. The people with more engagement, more knowledge, more money, or more trust in the medical system would probably sign up in greater numbers—and if those people have systematic differences in diet or genes from the people who don't sign up, then you've just created a hidden selection bias.

Instead, you should two simple steps.

Randomize: randomly split the cohort into two groups, denoted the treatment group and the control group.

Intervene: allocate everyone in the treatment group to take the treatment (e.g. Zapaclot, the new drug), and everyone in the control group to take something else (e.g. the old drug or a placebo).³

Randomize and intervene: a simple prescription, but the surest way to establish causality. The intervention allows you to pick up a difference between the new and old drug, if there's one to be found. The randomization ensures that other factors—even unknown factors, in addition to known ones like diet and lifestyle—do not lead us astray in our causal reasoning. The Latin phrase *ceteris paribus*, which translates roughly as “everything else being equal,” is often used to describe such a situation. By randomizing and intervening, we have ensured that the only *systematic* difference between the groups is the treatment itself. The randomization gives us a balanced comparison.

This last point is crucial. It's not that diet, genes, and other lifestyle factors somehow stop affecting a patient's cholesterol level when we randomize and intervene. It's just that diet, genes, and lifestyle factors aren't correlated with the treatment assignment, and so they're balanced between the two groups, on average.

The need to avoid selection bias sounds obvious. But if selection bias in medical trials were not rigorously policed, then it would be easy for doctors to cherry pick healthy patients for newly proposed treatments. After all, a doctor who invents a new, seemingly effective form of treatment will almost surely become both rich and famous. As one physician reminisces:

One day when I was a junior medical student, a very important Boston surgeon visited the school and delivered a great treatise on a large number of patients who had undergone successful operations for vascular reconstruction. At the end of the lecture, a young student at the back of the room timidly asked, “Do you have any controls?” Well, the great surgeon drew himself up to his full height, hit the desk, and said, “Do you mean did I not operate on half of the patients?” The hall grew very quiet then. The voice at the back of the room very hesitantly replied, “Yes, that's what I had in mind.” Then the visitor's fist really came down as he thundered, “Of course not. That would have doomed half of them to their death.” God, it was quiet then, and one could scarcely hear the small voice ask, “Which half?”⁴

³ Everyone in the control group should be taking the *same* something else, whether it's the old drug or a placebo.

⁴ Dr. E. Peacock, University of Arizona. Originally quoted in *Medical World News* (September 1, 1972). Reprinted pg. 144 of *Beautiful Evidence*, Edward Tufte (Graphics Press, 2006).

These last two words—"Which half?"—should echo in your mind whenever you are asked to judge the quality of evidence offered in support of a causal hypothesis. There is simply no substitute for a controlled experiment: not a booming authoritative voice, not even fancy statistics.

In fact, government regulators are so fastidious in their attention to possible selection biases that, in most real clinical trials, neither the doctors nor the patients are allowed to know which drug each person receives. Such a "double-blind" experiment avoids the possibility that patients might simply imagine that the latest miracle drug has made them feel better, in a feat of unconscious self-deception called the placebo effect.

A placebo, from the Latin *placere* ("to please"), is a fake treatment designed to simulate the real one.

Some history

The notion of a controlled experiment was certainly around in pre-Christian times. The first chapter of the book of Daniel relates the tale of one such experiment. Daniel and his three friends Hananiah, Mishael, and Azariah arrive in the court of Nebuchadnezzar, the King of Babylon. They enroll in a Babylonian school, and are offered a traditional Babylonian diet. But Daniel wishes not to "defile himself with the portion of the king's meat, nor with the wine which he drank." He goes to Melzar, the prince of the eunuchs, who is in charge of the school. Daniel asks not to be made to eat the meat or drink the wine. But Melzar responds that he fears for Daniel's health if he were to let them follow some crank new-age diet. More to the point, Melzar observes, if the new students were to fall ill, "then shall ye make me endanger my head to the king."

So Daniel proposes a trial straight out of a statistics textbook:

Prove thy servants, I beseech thee, ten days; and let them give us pulse to eat, and water to drink.

Then let our countenances be looked upon before thee, and the countenance of the children that eat of the portion of the king's meat: and as thou seest, deal with thy servants.⁵

⁵ King James Bible, Daniel 1:12–13.

The King agreed. When Daniel and his friends were inspected ten days later, "their countenances appeared fairer and fatter in flesh" than all those who had eaten meat and drank wine. Suitably impressed, Nebuchadnezzar brings Daniel and his friends in for an audience, and he finds that "in all matters of wisdom and understanding," they were "ten times better than all the magicians and astrologers that were in all his realm."

As for a placebo-controlled trial, in which some of the patients are intentionally given a useless treatment (the “placebo”): that came much later.⁶ The first such trial seems to have taken place in 1784. It was directed by none other than Benjamin Franklin, the American ambassador to the court of King Louis XVI of France. A German doctor by the name of Franz Mesmer had gained some degree of notoriety in Europe for his claim to have discovered a new force of nature that he called “magnétisme animal,” and which was said to have magical healing powers. The demand for Dr. Mesmer’s services soon took off among the ladies of Parisian high society, whom he would “Mesmerize” using a wild contraption involving ropes and magnetized iron rods.

Much to the king’s dismay, his own wife, Marie Antoinette, was one of Mesmer’s keenest followers. The king found the whole Mesmerizing thing frankly a bit dubious, and presumably wished for his wife to have nothing to do with the Herr Doctor’s magnétisme animal. So he convened several members of the French Academy of Sciences to investigate whether Dr. Mesmer had indeed discovered a new force of nature. The panel included Antoine Lavoisier, the father of modern chemistry, along with Joseph Guillotin, whose own wild contraption was soon to put the King’s difficulties with Mesmer into perspective. Under Ben Franklin’s supervision, the scientists set up an experiment to replicate some of Dr. Mesmer’s prescribed treatments, substituting non-magnetic materials—history’s first placebo—for half of the patients. In many cases, even the patients in the control group would flail about and start talking in tongues anyway. The panel concluded that the doctor’s method produced no effect other than in the patients’ own minds. Mesmer was denounced as a charlatan, although he continues to exact his revenge via the dictionary.

A more recent and especially striking example of a placebo comes from Thomas Freeman, director of the neural reconstruction unit at Tampa General Hospital in Florida. Dr. Freeman performs placebo brain surgery. (You read that correctly.) According to the British Medical Journal,

In the placebo surgery that he performs, Dr Freeman bores into a patient’s skull, but does not implant any of the fetal nerve cells being studied as a treatment for Parkinson’s disease. The theory is that such cells can regenerate brain cells in patients with the disease. Some colleagues decry the experimental method, however, saying that it is too risky and unethical, even though patients are told before the operation

⁶ See “The Power of Nothing” in the December 12, 2011 edition of *The New Yorker* (pp. 30–6).

that they may or may not receive the actual treatment.⁷

⁷ BMJ. 1999 October 9; 319(7215): 942

"There has been a virtual taboo of putting a patient through an imitation surgery," Dr. Freeman said. (Imagine that.) "This is the way to start the discussion." Freeman has performed 106 real and placebo cell transplant operations since 1992. Dr. Freeman argues that the medical history is littered with examples of unsafe and ineffective surgical procedures—think of that small voice at the back of the room, asking "which half?"—that were not tested against a placebo and resulted in needless deaths, year after year, before doctors abandoned them.

Experimental evidence is the best kind of evidence

Let's practice here, by comparing two causal hypotheses arising from two different data sets. The first comes from a clinical trial in the 1980's on a then-new form of adjuvant chemotherapy for treating colorectal cancer, a dreadful disease that, as of 2015, has a five-year survival rate of only 60-70% in the developed world.

The trial followed a simple protocol. After surgical removal of their tumors, patients were randomly assigned to different treatment regimes. Some patients were treated with fluorouracil (the chemotherapy drug, also called 5-FU), while others received no follow-up therapy. The researchers followed the patients for many years afterwards and tracked which ones suffered from a recurrence of colorectal cancer.

The outcome of the trial are in Table 9.1, below. Among the patients who received chemotherapy, 39% (119/304) had relapsed by the end of the study period, compared with 57% of patients (177/315) in the group who received no therapy:

| Chemotherapy? | Yes | No |
|---------------|-----|---------|
| Recurrence? | Yes | 119 177 |
| | No | 185 138 |

The evidence strongly suggests that the chemotherapy reduced the risk of recurrence by a substantial amount: the relative risk of a relapse under the treatment group is 0.7, with a 95% confidence interval of (0.59, 0.83).

We can be confident that this evidence reflects causality, and not merely correlation, because patients were randomly assigned

Table 9.1: Data from: J. A. Laurie et. al. Surgical adjuvant therapy of large-bowel carcinoma: An evaluation of levamisole and the combination of levamisole and fluorouracil. J. Clinical Oncology, 7:1447–56, 1989. There was also a third treatment arm of the study in which patient received a drug called levamisole, which isn't discussed here. Survival statistics on colorectal cancer from Cunningham et. al (2010). "Colorectal cancer." Lancet 375 (9719): 1030–47.

to the treatment and control groups. Randomization ensures *balance*: that is, it ensures that there are no systematic differences between the two groups with respect to any confounding factors that might be correlated with the patients' survival chances. This would obviously not be true if we had non-randomly assigned all the healthiest patients to the treatment group, and all the sickest patients to the control group.

It's worth emphasizing a key fact here. Randomization ensures balance both for the possible confounders that we can measure (like a patient's age or baseline health status), as well as for the ones we might *not* be able to measure (like a patient's will to live). This is what makes randomization so powerful, and randomized experiments so compelling. We don't even have to know what the possible confounding variables are in order for the experiment to give us reliable information about the causal effect of the treatment. *Randomization balances everything*, at least on average.

Next, let's examine data from a study from the 1990's conducted in sub-Saharan Africa about HIV, another dreadful disease which, at the time, was spreading across the continent with alarming speed. Several studies in Kenya had found that men who were uncircumcised seemed to contract HIV in greater numbers. This set off a debate among medical experts about the extent to which this apparent association had a plausible biological explanation.

| Circumcised? | Yes | No |
|---------------|-----|-----|
| HIV positive? | Yes | 105 |
| | No | 527 |
| | | 85 |
| | | 93 |

Table 9.2, above, shows some data from one of these studies, which found that among those recruited for the survey, 48% of uncircumcised men were HIV-positive, versus only 17% of circumcised men. The evidence seems to suggest that circumcision reduced a Kenyan man's chance of contracting HIV by a factor of 3.

Table 9.2: Data from Tyndall et. al. Increased risk of infection with human immunodeficiency virus type 1 among uncircumcised men presenting with genital ulcer disease in Kenya. Clin. Infect. Dis. 1996 Sep; 23(3):449–53.

Evaluating the evidence. If you suffer from colon cancer, should you get chemotherapy? Almost certainly: the researchers in the first study randomized and intervened, giving chemotherapy only to a random subset of patients. Unless you believe that the chemotherapy patients in this trial just happened to be much luckier than their peers, this result establishes that the reduction in recurrence must have been caused by the treatment.

But should all Kenyan men head straight to a surgeon? In this case we can't really be sure. The researchers in the second study neither randomized nor performed any snipping themselves. They merely asked whether each man was circumcised. It is therefore possible that they've been fooled by a confounder. To give one plausible example, a man's religious affiliation might affect both the likelihood that he is circumcised and the chances that he contracts HIV from unprotected sex. If that were true, the observed correlation between circumcisions and HIV rates might be simply a byproduct of an imbalanced, unfair comparison, rather than a causal relationship.⁸

Natural experiments

A randomized, controlled experiment is the gold standard of evidence for a causal hypothesis. Yet many times an experiment is impossible, impractical, unethical, or too expensive in time or money. In these situations, it often pays to look for something called a *natural experiment*, also called a *quasi-experiment*. A natural experiment is not something that you, as the investigator, design. Rather, it is an "experiment" where nature seems to have done the randomization and intervention for you, thereby giving you the same type of balance between treatment and control groups that you'd expect to get out of a real experiment.

This idea is best understood by example. Suppose you want to study the effect of class size on student achievement. You reason that, in smaller classes, students can get more individual attention from the instructor, and that instructors will feel a greater sense of personal connection to their students. All else being equal, you believe that smaller class sizes will help students learn better.

A cheap, naïve way to study this question would be to compare the test scores of students in small classes to those of students in larger classes. Any of these confounders, however, might render such a comparison highly unbalanced, and therefore dubious: (1) students in need of remediation are sometimes put in very small classes; (2) highly gifted students are also sometimes put in very small classes; (3) richer school districts can afford both smaller classes and many other potential sources of instructional advantage; or (4) better teachers successfully convince their bosses to let them teach the smaller classes themselves.

An expensive, intelligent way to study this question would

⁸ The authors of the study were obviously aware of these possible confounders. They used a technique called logistic regression to attempt to account for some them and isolate the putative effect of circumcision on HIV infection. This is like our fourth method for making balanced comparisons: use a model to adjust for confounders statistically. See the original paper for details.

| Question | Problem | Natural experiment | Lingering issues |
|---|--|---|--|
| Does being rich make people happy? | Even if richer people are happier on average, maybe happiness and success are the common effect of a third factor. Or maybe the rich grade on a different curve than the rest of us. | Compare a group of lottery winners with a similar group of people who played the lottery but didn't win. | Lottery winners may play the lottery far more often than people who played the lottery but didn't win, which might correlate with other important differences. |
| Does smoking increase a person's risk for Type-II diabetes? | People who smoke may also engage in other unhealthy behaviors at systematically different rates than non-smokers. | Compare before-and-after rates of diabetes in cities that recently enacted bans on smoking in public places. | Maybe the incidence of diabetes would have changed anyway. |
| Do bans on mobile phone use by drivers in school zones reduce the rate of traffic collisions? | Groups of citizens that enact such bans may differ systematically in their attitudes toward risk and behavior on the road. | Go to Texarkana, split by State Line Avenue. Observe what happens when Texas passes a ban and Arkansas doesn't. | There may still be systematic differences between the two halves of the city. |

Table 9.3: Three hypothetical examples of natural experiments.

be to design an experiment, in conjunction with a scientifically inclined school district, that randomly assigned both teachers and students to classes of varying size. In fact, a few school systems have done exactly this. A notable experiment is Project STAR in Tennessee—an expensive, lengthy experiment that studied the effect of primary-school class sizes on high-school achievement, and showed that reduced class sizes have a long-term positive impact both on test scores and drop-out rates.⁹

But suppose you are neither naïve nor rich, and yet still want to study the question of whether small class sizes improve test scores. If you're in search of a third way—one that's better than merely looking at correlations, yet cheaper than a full-fledged experiment—you might be interested to know the following fact about the Israeli school system.

[I]n Israel, class size is capped at 40. Therefore, a child in a fifth grade cohort of 40 students ends up in a class of 40 while a child in a fifth grade cohort of 41 students ends up in a class only half as large because the cohort is split. Since students in cohorts of size 40 and 41 are likely to be similar on other dimensions, such as ability and family background, we can think of the difference between 40 and 41 students enrolled as being "as good as randomly assigned."¹⁰

This is a lovely example of a natural experiment—something you didn't design yourself, but that is almost as good as if you

⁹ The original study is described in Finn and Achilles (1990). "Answers and Questions about Class Size: a Statewide Experiment." *American Educational Research Journal* 28, pp. 557–77

¹⁰ Angrist and Pischke (2009). *Mostly Harmless Econometrics*, Princeton University Press, p. 21

had. The researchers in this study compared the students in a group of 40 (“control group,” in one large class) versus the students in a group of 41 (“treatment group,” split into two smaller classes). This is a plausibly random assignment: the “randomization mechanism” is whether a student fell into a peer group of 40 versus a peer group of 41, and we would not expect this difference to be confounded by anything else that might predict test scores. Therefore, if we see a big difference in performance between the two groups, the most likely explanation is that class size caused the difference.

Some natural experiments, of course, are better than others. Consider the examples in Table 9.3, on page 188. For each one, ask yourself two questions. (1) What are the “treatment” and “control” groups? (2) How balanced are these two groups? (Said another way: how good is the quasi-randomization of cases to these groups?) Think carefully about each one, and you may begin to see “experiment” versus “non-experiment” as the black and white ends of a spectrum, with many shades of grey in between.

Matching

To estimate a causal effect by matching, we artificially construct a balanced data set out of an unbalanced one, by explicitly matching treated cases with similar control cases. We then compare the outcomes in treatment versus control groups, using only the balanced data set. This is most easily seen by example.

An example: the value of going green

For many years now, both investors and the general public have paid increasingly close attention to the benefits of environmentally conscious (“green”) buildings. There are both ethical and economic forces at work here. To quote a recent report by Mercer, an investment-consulting firm, entitled “Energy efficiency and real estate: Opportunities for investors”:

Investing in energy efficiency has two intertwined virtues that make it particularly attractive in a world with a changing climate and a destabilized economy: It cuts global-warming greenhouse gas emissions and saves money by reducing energy consumption. Given that the built environment accounts for 39 percent of total energy use in the US and 38 percent of total indirect CO₂ emissions, real estate investment represents

one of the most effective avenues for implementing energy efficiency.

This only scratches the surface. In commercial real estate, issues of eco-friendliness are intimately tied up with ordinary decisions about how to allocate capital. Every new project involves negotiating a trade-off between costs incurred and benefits realized over the lifetime of the building. In this context, the decision to invest in an eco-friendly building could pay off in at least four ways.

- (1) Every building has the obvious list of recurring costs: water, climate control, lighting, waste disposal, and so forth. Almost by definition, these costs are lower in green buildings.
- (2) Green buildings are often associated with indoor environments that are full of sunlight, natural materials, and various other humane touches. Such environments, in turn, might result in higher employee productivity and lower absenteeism, and might therefore be more coveted by potential tenants. The financial impact of this factor, however, is rather hard to quantify *ex ante*; you cannot simply ask an engineer in the same way that you could ask a question such as, “How much are these solar panels likely to save on the power bill?”
- (3) Green buildings make for good PR. They send a signal about social responsibility and ecological awareness, and might therefore command a premium from potential tenants who want their customers to associate them with these values. It is widely believed that a good corporate image may enable a firm to charge premium prices, to hire better talent, and to attract socially conscious investors.
- (4) Finally, sustainable buildings might have longer economically valuable lives. For one thing, they are expected to last longer, in a direct physical sense. (One of the core concepts of the green-building movement is “life-cycle analysis,” which accounts for the high front-end environmental impact of acquiring materials and constructing a new building in the first place.) Moreover, green buildings may also be less susceptible to market risk—in particular, the risk that energy prices will spike, driving away tenants into the arms of bolder, greener investors.

Of course, much of this is mere conjecture. At the end of the day, tenants may or may not be willing to pay a premium for

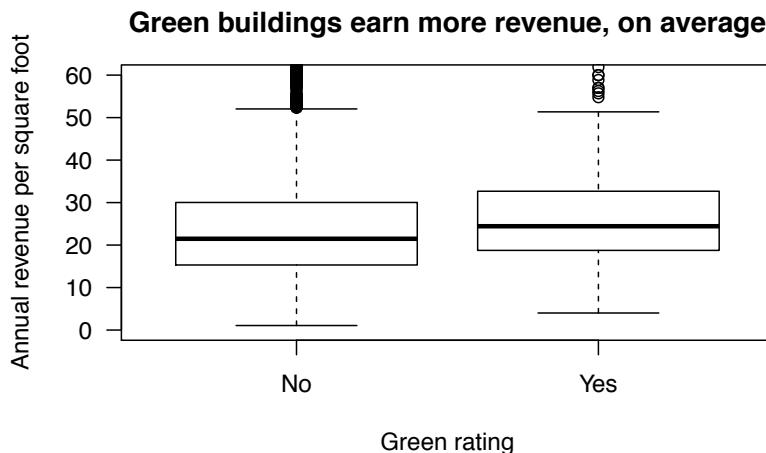


Figure 9.4: Green buildings seem to earn more revenue per square foot, on average, than non-green buildings.

rental space in green buildings. We can only find out by carefully examining data on the commercial real-estate market and comparing “green” versus “non-green” buildings. By “green,” we mean that a commercial property has received some official certification, because its energy efficiency, carbon footprint, site selection, and building materials meet certain environmental benchmarks, as certified by outside engineers.¹¹

Let’s look at some data on 678 green-certified buildings in the United States, together with 6,298 non-green buildings in similar geographic areas. The boxplot above shows that, when we measure revenue by a building’s rental rate per square foot per year, green buildings tend to earn noticeably higher revenue (mean = 26.97) than non-green buildings (mean = 24.51). That’s a difference of \$3.46 per square foot, or nearly a 15% market premium.

¹¹ The two most common certifications are LEED and EnergyStar; you can easily find out more about these rating systems on the web, e.g. at www.usgbc.org.

| Original data | | |
|---------------------|---------------------|-----------------|
| | Non-green buildings | Green buildings |
| Sample size | 6928 | 678 |
| Mean revenue/sq ft. | 24.51 | 26.97 |
| Mean age | 49.2 | 23.9 |
| Class A | 37% | 80% |
| Class B | 48% | 19% |
| Class C | 15% | 1% |

Table 9.4: Covariate balance for the original data. Class A, B, and C are relative classifications within a specific real-estate market. Class A buildings are generally the highest-quality properties in a given market. Class B buildings are a notch down, but still of reasonable quality. Class C buildings are the least desirable properties in a given market.

However, there's a problem with this comparison. As Table 9.4 shows, the green buildings tend to be newer than the non-green buildings, and are more likely to be "Class A" buildings.

So the important question is: do green buildings command a market premium *because* they are green, or simply because they are newer, better buildings in the first place? We can't tell by simply computing the average revenue in each group, because the green ("treatment") and non-green ("control") groups are highly unbalanced with respect to some important confounders.

This is where matching comes in. Matching means constructing a balanced data set from an unbalanced one. It involves three steps:

- (1) For each case in the treatment group, find the case in the control group that is the closest match in terms of confounding variables, and pair them up. Put these matched pairs into a new matched data set, and discard the cases in the original data set for which there are no close matches.
- (2) Verify covariate balance for the matched data set, by checking that the confounders are well balanced between the treatment and control groups.
- (3) Assuming that the confounders are approximately balanced, then compare the treatment-group outcomes with the control-group outcomes, using *only* the matched pairs.

Matching relies on a simple principle: compare like with like. In this example, that means if we have a 25-year-old, Class A building with a green rating, we try to find another 25-year old, Class A building without a green rating to compare it to.

In this particular example, once we've constructed the data set of matched pairs, the confounder variables are much more closely

| | | Matched data | |
|---------------------|---------------------|-----------------|--|
| | Non-green buildings | Green buildings | |
| Sample size | 678 | 678 | |
| Mean revenue/sq ft. | 25.94 | 26.97 | |
| Mean age | 23.9 | 23.9 | |
| Class A | 80% | 80% | |
| Class B | 19% | 19% | |
| Class C | 1% | 1% | |

Table 9.5: Covariate balance for the matched data.

balanced between the treatment and control groups (see Table 9.5). A comparison of revenue rates for this matched data set makes the premium for green buildings look a lot smaller: \$26.97 versus \$25.94, or about a 4% premium. Compare that with the 15% green premium we estimated from the original, unmatched data.

How do we actually find matches? The nitty-gritty algorithmic details of actually finding good matched pairs of cases are best left to the experts who write the software for these things. The two most common types of matching are called *nearest-neighbor search* and *propensity-score matching*; follow the links if you'd like to know more. In R, the package MatchIt uses propensity-score matching as a default; this is a very commonly used algorithm in real-world data analysis. In addition, [the paper linked here¹²](#) has a much more detailed overview of different matching methods.

Matching isn't a silver bullet: a bigger example

If you've ever been admitted to the intensive-care unit at a hospital, you may have undergone a diagnostic procedure called *right heart catheterization*, or RHC. RHC is used to see how well a patient's heart is pumping, and to measure the pressures in that patient's heart and lungs. RHC is widely believed to be helpful, since it allows the doctor to directly measure what's going on inside a patient's heart. But it is an invasive procedure, since it involves inserting a small tube (the catheter) into the right side of your heart, and then passing that tube through into your pulmonary artery. It therefore poses some risks—for example, excessive bleeding, partial collapse of a lung, or infection.

A natural question is: do the diagnostic benefits of RHC outweigh the possible risks? But this turns out to be tricky to answer. The reason is that doctors would not consider it ethical to run a randomized, controlled trial to see if RHC improves patient outcomes. As the authors of one famous study from the 1990s pointed out:¹³

Many cardiologists and critical care physicians believe that the direct measurement of cardiac function provided by right heart catheterization (RHC) . . . is necessary to guide therapy for certain critically ill patients, and that such management leads to better patient outcomes. While the benefit of RHC has not been demonstrated in a randomized controlled trial (RCT), the popularity of this procedure, and the widespread

¹² "Matching Methods for Causal Inference: A Review and a Look Forward." Elizabeth A. Stuart, *Statistical Science*, 2010.

¹³ "The effectiveness of right heart catheterization in the initial care of critically ill patients." Connors et. al. *Journal of the American Medical Association*. 1996 Sep 18; 276(11):889-97.

| | Original data | | Matched data | |
|--------------------------|---------------|--------|--------------|--------|
| | No RHC | RHC | No RHC | RHC |
| Sample size | 3551 | 2184 | 2184 | 2184 |
| 180-day survival rate | 0.370 | 0.320 | 0.354 | 0.320 |
| mean APACHE score | 50.934 | 60.739 | 57.643 | 60.739 |
| Trauma | 0.005 | 0.016 | 0.008 | 0.016 |
| Heart attack | 0.030 | 0.043 | 0.036 | 0.043 |
| Congestive heart failure | 0.168 | 0.195 | 0.209 | 0.195 |
| Sepsis | 0.148 | 0.321 | 0.24 | 0.321 |

belief that it is beneficial, make the performance of an RCT difficult. Physicians cannot ethically participate in such a trial or encourage a patient to participate if convinced the procedure is truly beneficial.

We're therefore left with only observational data on the effectiveness of RHC—which, on the surface, doesn't look good! Here's the data from the study quoted above, showing that critically ill patients undergoing RHC actually have a *worse* 180-day survival rate (698/2184, or 32%) than patients not undergoing RHC (1315/3551, or 37%):

| | No RHC | RHC |
|----------------------|--------|------|
| Survived 180 days | 1315 | 698 |
| Died within 180 days | 2236 | 1486 |

What's going on here? Should we conclude that right heart catheterization is actually killing people, and that the doctors are all just plain wrong about its putative benefits?

Not so fast. The problem with this conclusion is that the treatment (RHC) and control (no RHC) groups are heavily unbalanced with respect to baseline measures of health. Put simply, the patients who received RHC were a lot sicker to begin with, so it's no surprise that they have a lower 6-month survival rate. To cite a few examples: the RHC patients were three times more likely to have suffered acute trauma, 50% more likely to have had a heart attack, and 16% more likely to be suffering from congestive heart failure. The RHC patients also had an average **APACHE score** that was 10 points higher than the non-RHC patients.¹⁴ The left half of Table

Table 9.6: A before-and-after table of summary statistics showing covariate balance for the observational study on right-heart catheterization. The entries for trauma, heart attack, etc. show rates of these complications in the two groups. The left half of the table shows the original data set, while the right half shows the matched data set.

¹⁴ The APACHE score is a composite severity-of-disease score used by hospital ICUs to estimate which patients have a higher risk of death. Patients with higher numbers have a higher risk of death.

[9.6](#) shows these rates of various complications for the two groups in the original data set. They're quite different, implying that the survival rates of these two groups cannot be fairly compared.

And what about after matching? Unfortunately, Table [9.6](#) shows that, even after matching treatment cases with controls having similar complications, the RHC group still seems to have a lower survival rate. The gap looks smaller than it did before, on the unmatched data—a 32% survival rate for RHC patients, versus a 35.4% survival rate for non-RHC patients—but it's still there.

Again we find ourselves asking: what's going on? Is the RHC procedure actually killing patients? Well, it might be, at least indirectly! The authors of the study speculate that one possible explanation for this finding is “that RHC is a marker for an aggressive or invasive style of care that may be responsible for a higher mortality rate.” Given the prevalence of [overtreatment](#) within the American health-care system, this is certainly plausible.

But we can't immediately jump to that conclusion on the basis of the matched data. In fact, this example points to a couple of basic difficulties with using matching to estimate a causal effect.

The first (and most important) difficulty is that *we can't match on what we haven't measured*. If there is some confounder that we don't know about, then we'll never be able to make sure that it's balanced between the treatment and control groups within the matched data. This is why experiments are so much more persuasive: because they also ensure balance for unmeasured confounders. The authors of the study acknowledge as much, writing:

A possible explanation is that RHC is actually beneficial and that we missed this relationship because we did not adequately adjust for some confounding variable that increased both the likelihood of RHC and the likelihood of death. As we found in this study, RHC is more likely to be used in sicker patients who are also more likely to die.

Another possible explanation is that we simply haven't been able to match treatment cases with control cases very effectively. The right half of Table [9.6](#) shows that covariate balance for the matched data is noticeably better than for the unmatched data, but it's not perfect. We still see some small differences in complication rates and APACHE scores between the treatment and control group. There are two main reasons for this.

- (1) First, and most importantly, although finding a match on one or two variables is relatively easy, finding a match on several

variables is pretty hard. Think of this in terms of your own life experience—for example, in seeking a spouse or partner. It probably isn't too hard to find someone who's a good match for you in terms of your interests and your sense of humor. But if you require that this person *also* match you in terms of age, career, education, home town, height, weight, looks, and favorite sport, then you're a lot less likely to find a match. *Picky people are less likely to find a satisfying match in life.* For this same reason, it's unlikely that we'll be able to find an exact match for each treatment case in a matching problem, especially with lots of possible confounders.

- (2) Second, finding matches for cases with rare confounders is especially hard—by definition, since the confounder is rare!

These two points underline a basic difficulty with matching: perfect matches usually don't exist, and we have no choice but to accept approximate matches. In practice, therefore, we give up on the requirement that every single pair of matched observations is similar in terms of all possible confounders, and settle for having matched groups that are similar in their confounders, *on average*. That's why it's so important to check the covariate balance after finding matched pairs, to make sure that there's nothing radically different between the two groups.

Model-based statistical adjustment

A fourth identification strategy for estimating a causal effect is to build a regression model. If some important (and quite strong) assumptions are met, then such a model is capable of isolating a causal relationship between predictor and response, by adjusting for the effects of confounders *statistically*, rather than experimentally. You may have heard this process described as “statistical control” or “statistical correction,” both in the popular media and in scientific publications:

- “Schatz’s numbers are unique in that they evaluate each play against the league average for plays of its type, adjust for the strength of the opponents’ defense, and even try to divide credit for a given play among teammates.”¹⁵
- “The committee concluded that a statistical adjustment of the 1990 census leads to an improvement of the counts.”¹⁶

¹⁵ “Pigskin Pythagoras: A guy from Framingham tries to remake the muddy field of football statistics.” *Boston Globe*, February 1, 2004

¹⁶ “Judge must decide on census adjustment.” *Chicago Tribune*, 6/8/1992

- “Further adjustment for weight change and leukocyte count attenuated these risks substantially.”¹⁷

Estimating a causal effect using a regression model is, in principle, no different than estimating a partial relationship, which we’ve already learned how to do:

- (1) Build a multiple regression model for the outcome (y) versus the predictor of interest (x) and other possible confounders;
- (2) Interpret the coefficient on the x variable of interest as the partial linear relationship between y and x , holding confounders constant.

The key question is: under what circumstances can we interpret the partial relationship in a multiple regression model as the *causal* effect of x on y ? By *causal effect*, you should think in terms of the counterfactuals we entertained at the beginning of the chapter: *if* I were to intervene and change x by one unit, holding all other variables constant, *then* how much would y change on average?

There are three important assumptions that must be met in order to give a causal interpretation to a regression coefficient. First, your model must include all confounding variables (that is, variables that have a causal effect on both the treatment assignment and the outcome). Second, the model must be correct. In this context, “correct” means that you have included the right interactions among confounding variables, and that you have specified the right functional form of the model (linear, polynomial, power law, etc.). Finally, you must *not* include any post-treatment effects as covariates in the model. A post-treatment effect is something causally “downstream” from the treatment variable, and that becomes known only as a result of receiving or not receiving the treatment. This is a subtle point, and we won’t discuss it in detail. But the important thing is: include those confounders, and *only* those confounders, that affect the allocation of cases to the treatment and control groups.

If, and only if, these three assumptions about your model are true, then the regression coefficient of y on x has a causal interpretation. If, on the other hand, there are any unmeasured confounders affecting your x and y variable, then the coefficient of y on x measures association, not causation. This is called *omitted-variable bias*.¹⁸

Another way of saying this is that *if* the possible confounders are all observed, then accurately estimating the causal effect of

¹⁷ “Smoking, Smoking Cessation, and Risk for Type 2 Diabetes Mellitus: A Cohort Study.” *Annals of Internal Medicine*, January 4, 2010

¹⁸ Or *lurking-variable bias*.

x on y really just boils down to modeling the data well, and not using that model to extrapolate beyond the range of available data. However, the assumption that we've observed all relevant confounders, and can therefore adjust for them appropriately, is very strong. It's also unverifiable using the data; as with matching, you have to believe this assumption, and convince people of it, on extrinsic grounds.

Using regression analysis to estimate causal effects is a big, serious topic. Here are two full books about it:

- *Causality*, by Judea Pearl
- *Observational Studies*, by Paul Rosenbaum

For some additional, more easily digestible advice on choosing which covariates to include in a causal model, see [Chapter 17](#) of Daniel Kaplan's book on statistical modeling.¹⁹

Matching versus regression, or matching and regression?

We've seen that it's easiest to infer causality if the cases in the treatment group are comparable to those in the control group. One way to do this is via matching: explicitly constructing a balanced data set from an unbalanced one. Another way to do this is via regression: adjust for confounders using a statistical model, so that we can evaluate the partial relationship between treatment and response, holding confounders constant.

This makes it sound as though regression and matching are competing identification strategies for causal inference. Sociologically speaking, there is certainly some truth to this, in that some people tend to use matching more often, and others tend to use regression more often. So which one should *you* use?

In the real world, if you're going to use only one strategy or the other, my advice is to use matching, mainly for three reasons:

- (1) Matching is a lot easier for non-experts to understand, since you can point to the matched treatment and control groups and show that they are visibly balanced with respect to observed confounders. In other words, the nature of the "balanced comparison" being made via matching is much more transparent than the idea of a partial slope in a regression model. This will make it easier for you to convince others of your conclusions.

¹⁹ Kaplan also has a good explanation for why it's not a good idea to include post-treatment effects (i.e. variables causally downstream of the treatment) as covariates in a regression model.

- (2) Matching is a bit more robust than regression, at least in their “off the shelf” versions. The regression-based approach to causal inference relies on a whole bunch of hard-to-verify assumptions: linearity, all necessary interactions included, and so forth. By comparison, it’s a lot easier to verify covariate balance using before-and-after tables of summary statistics. (Of course, neither method is robust to unmeasured confounders—only an experiment can fix that problem.)
- (3) Unwarranted extrapolations are more apparent when matching than when using regression. Suppose that the treatment and control groups have highly nonoverlapping distributions of confounders—for example, that most the men are in the treatment group and most of the women in the control group. In such cases, the data are inherently limited in what they can tell us about the treatment-response relationship in this region of nonoverlap (i.e. how the treatment will work for women). This lack of overlap will be obvious if you use matching, because you’ll still have drastic post-match covariate imbalances that will stick out like a sore thumb. But the lack of overlap will be less obvious if you throw all the confounders into a multiple regression model without plotting your data.

In summary, it’s easier to convince others with matching, and easier to fool yourself with regression. These aren’t intrinsic *statistical* advantages to matching; they are merely *practical* advantages worth keeping in mind.

It turns out, however, that there’s no need to choose between matching and regression. Better still is to use both matching *and* regression, to get better estimates of causal effects than either technique is capable of getting on its own. In other words: first run matching to get an approximately balanced data set. Then run a regression model for the response versus the treatment variable and the confounders, to correct for minor imbalances in the matched data set. Under this approach, the primary role of matching is to correct for major covariate imbalances between the groups, while the primary role of regression is to model the treatment-response relationship in a way that adjusts for any minor confounding that remains in the matched data set.

There’s one other major advantage of using matching and regression together. By fitting a regression model to a matched data set, you are able to search for interactions *between* the treatment

variable and possible confounders. For example, what if the treatment effect is different for men than for women? You can discover this kind of modulating effect much more easily using a regression model than you can with matching alone.

In summary, matching and regression make for an excellent pair. There's rarely a good reason to use just one or other!

Expected value and probability

Risky business

FOR most of us, life is full of worry. Some people worry about tornados or earthquakes; other people won't get on an airplane. Some people worry more about lightning; others, about terrorists. And then there are the everyday worries: about love, money, career, status, conflict, kids, and so on.

Jared Diamond worries a lot, too—about slipping in the shower.

Dr. Diamond is one of the most respected scientists in the world. Though he originally trained in physiology, Diamond left his most lasting mark on the popular imagination as the author of *Guns, Germs, and Steel: The Fates of Human Societies*. This Pulitzer-prize-winning book draws on ecology, anthropology, and geography to explain the major trends of human migration, conquest, and displacement over the last few thousand years.

Strangely enough, Diamond began to worry about slipping in the shower while conducting anthropological field research in the forests of New Guinea, 7,000 miles away from home, and a long day's walk from any shower. The seed of this worry was planted one day while he was out hiking in the wilds with some New Guineans. As night fell, Diamond suggested that they all make camp under the broad canopy of a nearby tree. But his companions reacted in horror, and refused. As Diamond tells it,

They explained that the tree was dead and might fall on us.
Yes, I had to agree, it was indeed dead. But I objected that it was so solid that it would be standing for many years. The New Guineans were unswayed, opting instead to sleep in the open without a tent.¹

The New Guineans' fear initially struck Diamond as overblown. How likely could it possibly be that the tree would fall on them in the night? Surely they were being paranoid. For a famous professor like Diamond to get crushed by a tree while sleeping in the

¹ Jared Diamond, "That Daily Shower Can Be a Killer." *New York Times*, January 29, 2013, page D1.

forest would be the kind of freakish thing that made the newspaper, like getting struck by lightning at your own wedding, or being killed by a falling vending machine.

But in the months and years after this incident, it began to dawn on Diamond that the New Guineans' "paranoia" was well founded. A dead tree might stay standing for somewhere between 3 and 30 years, so that the daily risk of a toppling was somewhere between 1 in 1,000 and 1 in 10,000. This is small, but far from negligible. Here's Diamond again:

[W]hen I did a frequency/risk calculation, I understood their point of view. Consider: If you're a New Guinean living in the forest, and if you adopt the bad habit of sleeping under dead trees whose odds of falling on you that particular night are only 1 in 1,000, you'll be dead within a few years.²

² *ibid.*

Having absorbed this attitude about the importance of everyday habits, Diamond began to apply it to his own life. He refers to it as a "hypervigilant attitude towards repeated low risks," or more memorably, "constructive paranoia."

Take the simple act of showering. If you're 75 years old, as Diamond was when he recounted this story, you can expect to live another 15 years. That's $15 \times 365 = 5,475$ more daily showers. So if your risk of a bad slip is "only" one in a thousand, you should expect to break your hip, or worse, about five times over that period. The implication is that, if you want a good chance of being around to blow out 90 candles, you must ensure that, by your own careful behavior, you reduce the risk of slipping in the shower to something much, much lower than one in a thousand.

And the same goes for all those other small risks we face day in, day out. Think about crossing a busy street, driving at night, touching the handle of a public toilet, or venturing out with the mad dogs and Englishmen into the mid-day sun. Each time the chance of a disaster is low. But most of us perform these actions again and again—and if we're slapdash about it, the expected number of disasters over several years can be alarmingly high. Diamond's conclusion? He needed to ensure that, for each repeated exposure to one of these risks, the chance of a disaster wasn't just low, but extremely low.

Expected value and the NP rule

Jared Diamond's philosophy of constructive paranoia arises from an understanding of *expected value*. This concept has a formal

mathematical definition, but the basic idea is simple. Think about risks like slipping in the shower, or having a dead tree fall on you in the night. These kinds of risks involve many repeated exposures to the same chance event. In the long run, the expected number of events is the frequency of encounters (N), times the probability of the event in a single encounter (P).

This is such a common scenario that we like to give it a name: the NP rule, where expected value = frequency times risk, or $N \times P$. For example, let's say that the risk of a dead tree falling down in the night is one chance in a thousand (so $P = 0.001$), and that you and 99 friends each sleep under your own dead tree every night for a year (so $N = 365 \times 100 = 36,500$ person-nights of exposure). In your cohort of 100, how many would you expect to get crushed by a tree? The math of the NP rule doesn't look good; you can expect about 36 of you to be crushed.

$$\text{Expected crushings} = (\text{Risk of dead tree falling}) \times (\text{Number of exposures})$$

$$= \frac{1}{1000} \times (365 \times 100) \\ = 36.5.$$

What about some more familiar risks?

Of course, you probably don't live in a forest in New Guinea. How does the NP rule play out in thinking about risks for a typical 21st-century citizen of a western democracy?

To get specific, let's look at some expected values for an imaginary cohort of 100,000 Americans—about the size of a small city, like Boulder or Green Bay. Table 10.1 shows how many of these 100,000 people we would expect to die in any given year due to various causes.³ This is exactly the kind of table that a public-health organization like the Gates Foundation might look at it in order to decide what kinds of initiatives would have the biggest return on investment, or that a life-insurance company would look at to set your premiums.

There are two take-away lessons from Table 10.1. First, the expected number of deaths due to the headline-grabbing causes in the bottom half of the table—from tornadoes to shark attacks to mass shootings—is tiny. Of course, tornadoes, sharks, and crazed gunmen are still very dangerous (P is high). But they're also rare (N is small). Remember: expected value = $N \times P$.

³ Centers for Disease Control, <http://www.cdc.gov/nchs/fastats/>.

| Cause | Expected deaths |
|-------------------------|-----------------|
| Heart disease | 203 |
| Cancer | 195 |
| Respiratory disease | 50 |
| Stroke | 42 |
| Alzheimer's | 28 |
| Diabetes | 25 |
| Accidental poisoning | 12 |
| Car accident | 11 |
| Slips/falls | 10 |
| Homicide | 5 |
| Eating raw meat | 2 |
| Choking | 1.5 |
| Pregnancy | 0.2 |
| Dog bite | 0.01 |
| Falling vending machine | 0.001 |
| | |
| Hurricane | 0.03 |
| Tornado | 0.02 |
| Mass shooting | 0.01 |
| Lightning strike | 0.01 |
| Shark attack | 0.0003 |
| Plane crash | 0.0001 |

(per 100,000)

Table 10.1: Expected deaths due to various causes over one year in an imaginary cohort of 100,000 Americans, of whom 99,200 are expected to survive.

Second, in light of these numbers, it might be wise to heed Jared Diamond's advice. While most people die of disease, cancer, or the depredations of age, a shockingly high number die in preventable accidents. Even unusual kinds of accidents are still far more common than the six sensational causes of death in the bottom half of the table. In fact, we'd expect ten times as many people to die from a falling vending machine as from a falling plane, and 20 times as many to die from choking as from all the bottom six causes put together. Of course, eating lunch or buying a granola bar are usually safe, so P is small. But people do these things every day, so N is huge.

Studies, however, repeatedly find that our concept of danger is woefully incomplete: we think only about P , and rarely about N . As a result, we overestimate the chance of dying in some dramatic event like those in the bottom half of Table 10.1, while simulta-

neously underestimating the chance of dying from one of the familiar causes in the top half.

To be fair, this has a lot to do with living in a world of mass media and near-instant communication. Thousands of people anonymously choke to death every year. But if someone gets attacked by a shark or blows himself up in a train station anywhere in the world, you will hear about it, no matter how unlikely the real risk. As folks in the statistics business put it: newspapers love numerators. While this brings a website plenty of clicks, it also short-circuits our natural cues for reasoning intuitively about risk.

But dwelling on the spectacular numerators isn't a smart way to stay alive. Many of life's mundane risks, from car accidents to skin cancer, do not strike out of the blue. Rather, they are direct results of our own day-to-day behavior. So follow your mother's advice. Look both ways, don't drive while tired, wear sunscreen, wash your hands—and don't sleep under dead trees.

The NP rule in health care and social policy

THE concept of expected value is central to any cost/benefit analysis. For example, the same idea behind the NP rule is used routinely to evaluate medical procedures.

In a medical context, an expected-value calculation is usually phrased in terms of a number called the NNT: the number needed to treat. Here's the idea. Suppose you invent a perfect drug for some intractable disease. Anyone who takes the drug is cured, and it's the only cure. Here, we'd say that your drug has a "number needed to treat" of one: if you treat one person, you cure one person. You can't do any better than this.

Now let's say that the drug has only a 50% chance of curing someone ($P = 0.5$). In that case, if you treated $N = 2$ people, you would expect to cure one patient: $N \times P = 1$. Here, we'd say that the NNT is two: treat two, cure one.

An NNT of two is really good. But if you needed to treat 100 or 1000 people to cure just a single person, you might view the drug a bit more skeptically. More generally, suppose that a medical procedure has probability P of offering some specific health benefit to any one person—like curing a disease, or offering one extra year of life. If we treat N people, we would expect that $N \times P$ people would get the benefit. How many people do we need to treat so

| Treatment | Benefit | NNT |
|---|---------------------------------|------|
| Defibrillation for cardiac arrest | Prevents death | 2.5 |
| Corticosteroid injection for tennis elbow | Reduces pain | 4 |
| Zinc for the common cold | Reduces symptoms | 5 |
| Antibiotics for conjunctivitis | Full recovery within 5 days | 7 |
| Bone-marrow transplant after chemo for leukemia | Prevents relapse | 9 |
| Strength and balance programs for the elderly | Prevents falls | 11 |
| Warfarin for atrial fibrillation | Prevents stroke | 25 |
| Aspirin, for patients with known heart disease | Prevents heart attack or stroke | 50 |
| a Mediterranean diet | Prevents heart disease | 61 |
| Magnesium sulfate for preeclampsia in pregnancy | Prevents seizures | 90 |
| Statins, for patients with no known heart disease | Prevents heart attack | 104 |
| CT scans of long-term smokers | Detects lung cancer | 217 |
| Aspirin, for patients with no known heart disease | Prevents heart attack or stroke | 1667 |

Table 10.2: Numbers sourced from Cochrane reviews, as summarized by the NNT website: <http://www.thennt.com>.

⁴ This is a slight simplification. The NNT is more typically defined to be the number needed to treat in order to offer some benefit to one *additional* patient versus some baseline, like a placebo or the next-best drug.

that the expected number helped, $N \times P$, is one? That number is called the procedure's number needed to treat, or NNT.⁴ Similarly, for a medical test like a mammogram or a prostate exam, there's the NNS: the "number needed to screen."

Table 10.2 has some estimates of the number needed to treat for some common medical interventions.

Weighing medical harms and benefits

The number needed to treat is a big deal to doctors, health insurers, and governments that run national health services. A high NNT means a low expected value for the number of patients helped. Essentially, it's a measure of waste: if a treatment has an NNT of 100, then on average, it will fail to yield the stated benefit for 99 out of 100 patients.

Of course, we don't know who those 99 will be ahead of time. And if the treatment is cheap and mostly harmless, or if the possible benefit is extremely important, then a high NNT might be acceptable. For example, the use of aspirin to prevent a first heart attack has an NNT of over 1000, but plenty of doctors recommend it routinely, despite its side effects.⁵

But as you may have heard, modern health care is expensive. It already strains the budgets of most households and governments. Paying for one thing usually means not paying for something else, and knowing the NNT helps us to be clear-eyed about these

⁵ Antithrombotic Trialists Collaboration. "Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials." *Lancet.* 2009; 373(9678); 1849-60.

opportunity costs.

Moreover, a lot of procedures present at least some probability Q of unwanted side effects—for example, the risk that a mammogram will lead to a false-positive finding. That means a medical cost/benefit analysis really has two expected values to contend with: the expected number of people helped, $N \times P$; and the expected number harmed, $N \times Q$. In this context, we speak of the “number needed to harm,” or NNH: the number of people we’d need to treat in order to harm a single person in some specific way.

For these reasons, a high-NNT medical procedure usually provokes two questions.

For governments and insurers: Could we produce a greater good for a greater number of people by redirecting our limited resources to some other treatment?

For everyone: How bad are the side effects, and what’s the number needed to harm (NNH)? Imagine a treatment that produces nasty side effects in every fifth patient (NNH = 5), but only cures every hundredth (NNT = 100). Depending on how bad the side effects are compared with the original condition, you might prefer no treatment at all.

Expected value and mammograms. Indeed, it was exactly this second question that spurred the American Cancer Society to recently revise its guidelines on screening mammograms for women with no family history of breast cancer. The *New York Times* devoted a front-page story to the announcement:⁶

The American Cancer Society, which has for years taken the most aggressive approach to screening, issued new guidelines on Tuesday, recommending that women with an average risk of breast cancer start having mammograms at 45 and continue once a year until 54, then every other year for as long as they are healthy and likely to live another 10 years. The organization also said it no longer recommended clinical breast exams, in which doctors or nurses feel for lumps, for women of any age who have had no symptoms of abnormality in the breasts. Previously, the society recommended mammograms and clinical breast exams every year, starting at 40.

The key changes here were for women under 45 or over 54, for whom biennial scans were now recommended; for women aged 45-54, annual scans remained the recommendation.⁷

⁶ “American Cancer Society, in a Shift, Recommends Fewer Mammograms.” Denise Grady, *New York Times* front page, 20 October 2015. Available at <http://www.nytimes.com/2015/10/21/health/breast-cancer-screening-guidelines.html>.

⁷ It’s important to point out here (as the *New York Times* did, responsibly) that the revised guidelines applied only to women with an “average” risk of breast cancer. Women with a personal or family history of breast cancer, or any other major risk factor, were still encouraged to get annual screenings from an early age.

The Society's previous recommendation for women with no family history was to get a mammogram every year starting at age 40. This approach benefitted some people, and harmed others. Specifically, a systematic review of many earlier studies estimated that, under this approach, we'd need to regularly screen about 2,500 women aged 40-49 in order to save one life ($NNS \approx 2500$). Of these 2,500 women, about 175 would end up experiencing a false-positive biopsy result. This implies an NNH of about 14: for every 14 women screened, someone got hurt.⁸

However, if we were to apply the Society's new screening recommendations to these same 2,500 women, we'd still expect to save that one life, on average. But now we would expect only 120 false positives. That's 55 women out of every 2,500 who are spared from needless stress and medical intervention, with no detectable increase in the risk of someone dying. These expected-value calculations were a big part of the reasoning behind the American Cancer Society's new recommendation: that women with no family history should get screened every year from 45-54, and every two years after that.

Expected value and PSA screening. Screening mammograms are not the only medical procedure that requires careful thinking about expected value. A common test for prostate cancer, called the prostate-specific antigen (PSA) test, has been at the center of a similar controversy for years. Prostate cancer kills over 300,000 men per year worldwide. However, it's also incredibly common for a prostate tumor to come late in life and grow slowly. In fact, autopsy records show that something like 2/3 of all elderly men die with asymptomatic tumors in their prostates.

Here's why the PSA test is controversial. The test detects elevated levels of prostate-specific antigen in the blood, which is a potential indicator of a prostate tumor. If a man's PSA levels are high enough, he's referred for a prostate biopsy to get a tissue sample. This has some small probability P of detecting a deadly tumor. But because asymptomatic prostate cancer is so common, the test also has some other probability Q of leading to unnecessarily aggressive courses of treatment for a tumor that never would have done much harm. Some of the men who undergo these treatments end up incontinent, impotent, or dead.

Is the life-saving potential of PSA screening for prostate cancer worth these harms? The U.S. Preventive Services Task Force says

⁸ Myers et. al. "Benefits and Harms of Breast Cancer Screening: A Systematic Review." *Journal of the American Medical Association* 2015; 314(15):1615-34.

no: P is tiny and Q is large. Here's how their report describes PSA tests:

The reduction in prostate cancer mortality after 10 to 14 years is, at most, very small, even for men in what seems to be the optimal age range of 55 to 69 years. There is no apparent reduction in all-cause mortality. In contrast, the harms associated with the diagnosis and treatment of screen-detected cancer are common, occur early, often persist, and include a small but real risk for premature death. Many more men in a screened population will experience the harms of screening and treatment of screen-detected disease than will experience the benefit. The inevitability of overdiagnosis and overtreatment of prostate cancer as a result of screening means that many men will experience the adverse effects of diagnosis and treatment of a disease that would have remained asymptomatic throughout their lives.

The Task Force concludes simply that "the benefits of PSA-based screening for prostate cancer do not outweigh the harms."⁹

Postscript

Now would be a good time to issue an important disclaimer: we are not qualified to endorse or dispute the American Cancer Society's guidelines on mammograms, or the USPSTF's guidelines on PSA screening. We're merely trying to highlight the role of expected value in their thinking, and to emphasize two broader lessons to be found in these debates.

First, we appreciate that, if you're a patient thinking through your treatment options, what matters most are your own circumstances and preferences. While population-level quantities like an expected value or an NNT can guide your thinking, it's your own situation-specific, *conditional* probabilities that really ought to be decisive. However, those in the business of setting health policy—whether for a government, insurance company, or professional society—simply cannot avoid the principle of expected value. We ask these people to act like responsible utilitarians on behalf of a wider population. To do this, they must think about both N and P .

The second lesson is that cause and effect are both complicated and probabilistic. Most interventions produce the intended effect in any individual case only with some probability P . For many policies, P is very small, and the risk Q of unwanted side effects may be much higher. We should weigh the policy's costs and

⁹ Moyer et. al. "Screening for Prostate Cancer: U.S. Preventive Services Task Force Recommendation Statement." *Annals of Internal Medicine* 157(2), 2012.

benefits in light of the expected values for both the good and the bad outcomes.

But it's all too easy to let ourselves fall into some counterfactual dream state, especially if can't shake the impression left by that one awesome example where the policy really *did* work. "If things turned out like that every time," we think to ourselves, "imagine how many lives/dollars/hours/puppies we could save." But that's a big "if." Controversial medical tests are great examples of this phenomenon. If you read up on the debates surrounding mammograms or PSA screening, you'll notice a striking rhetorical pattern. The medical societies and task forces recommending fewer screens always cite expected values based on peer-reviewed medical research. The doctors and patients who cry out in opposition often cite anecdotes or "clinical experience."

There are many other examples outside medicine. For example, in the 1990s, California passed its infamous "three-strikes" law, where someone with a third felony conviction automatically received at least a 25-year prison sentence. These once-fashionable laws have now fallen out of favor, but it's easy to understand how they could have been passed in the first place. All it takes is for one judge to be a bit too lenient, and for a thrice-convicted felon to go on a headline-grabbing rampage after getting out of prison, for that single canonical example to become frozen in the public's mind. From there, the "obvious" policy solution is hardly a big leap: three-time felons must spend the rest of their lives in jail.

As it happens, while California's three-strikes laws may have prevented some crimes, many scholars have concluded that it was largely ineffective.¹⁰ One thing the law did do, however, was create a sharp incentive for criminals to avoid that third arrest. As a result, the law may have caused more felonies than it prevented, by increasing the chance Q that a suspect with two strikes will assault or murder a police officer who's about to arrest them.¹¹ It also cost taxpayers a huge amount of money to prosecute, secure, feed, and clothe all those dangerous felons whose third strike consisted of an illegal left turn with three dimebags of marijuana in the passenger seat.

So if you ever get to make any kind of policy, keep expected value at the front of your thoughts, and mind your P and Q .

¹⁰ Males et. al. "Striking Out: The Failure of California's 'Three Strikes and You're Out' Law." *Stanford Law and Policy Review*, Fall 1999.

¹¹ Johnson and Saint-Germain. "Officer Down: Implications of Three Strikes for Public Safety." *Criminal Justice Policy Review*, 16(4), 2005.

Probability: a language for uncertainty

ALL of these examples illustrate the concept of a *random variable*, which is a generic term for any uncertain outcome. For example:

- the number of trees that fall over tonight in a particular patch of New Guinean forest.
- the number of women aged 50-70, out of a group of 200, who will get breast cancer.
- how many users will click on a particular Google ad in the next hour.

These random variables all fall within the NP rule, where the expected value is found by multiplying the risk times the exposure.

But here's where we run up against the limitations of thinking about randomness purely in terms of a simple risk/exposure calculation. One problem is a lack of generality. For example, it's not at all clear how we could use this approach to calculate an expected value for *these* uncertain outcomes:

- the rate of U.S. unemployment in 18 months.
- the value of your retirement portfolio in 30 years.
- your extra lifetime earnings from going to graduate school.

What does a risk/exposure calculation even look like here? And what about these uncertainties?

- the winner of next year's Tour de France.
- whether a defendant is guilty or innocent.
- whether you'll like the next person you're matched up with through a dating app.

Here the possible outcomes aren't even numbers.

A second, even bigger problem is that an expected value conveys nothing about *uncertainty*. We may expect that 11 people in Green Bay, Wisconsin (pop. 100,000) will die this year in a car accident. But it could be 5, or 20. It's a random variable; no one knows for sure.

To really understand risk deeply, we need a better language for helping us to communicate clearly about uncertainty. That language is probability.

Probability

Probability is a rich language for communicating about uncertainty. Up to now we've spoken in fairly loose terms about this concept. And while most of us have an intuitive notion of what it means, it pays to be a bit more specific.

A probability is just a number that measures how likely it is that some event, like rain, will occur. If A is an event, $P(A)$ is its probability: $P(\text{coin lands heads}) = 0.5$, $P(\text{rainy day in Ireland}) = 0.85$, $P(\text{cold day in Hell}) = 0.0000001$, and so forth.

Some probabilities are derived from data, like the knowledge that a coin comes up heads about 50% of the time in the long run, or that 11 people out of 100,000 die in a car accident. But it's also perfectly normal for a probability to reflect your subjective assessment or belief about something. Here, you should imagine a stock-market investor who has to decide whether to buy a stock or sell it. The performance of a stock over the coming months and years involves a bunch of one-off events that have never happened before, and will never be repeated. But that's OK. We can still talk about a probability like $P(\text{Apple stock goes up next month})$. We just have to recognize that this probability reflects someone's subjective judgment, rather than a long-run frequency from some hypothetical coin-flipping experiment.

Probability and betting markets. If you don't have any data, a great way to estimate the probability of some event is to get people to make bets on it. Let's take the example of the 2014 mens' final at Wimbledon, between Novak Djokovic and Roger Federer. This was one of the most anticipated tennis matches in years. Djokovic, at 27 years old, was the top-ranked player in the world and at the pinnacle of the sport. And Federer was—well, Federer! Even at 32 years old and a bit past his prime, he was ranked #3 in the world, and had been in vintage form leading up to the final.

How could you synthesize all this information to estimate a probability like $P(\text{Federer wins})$? Well, if you walked into any betting shop in Britain just before the match started, you would have been quoted odds of 20/13 on a Federer victory.¹² To interpret odds in sports betting, think "losses over wins." That is, if Federer and Djokovic played 33 matches, Federer would be expected to win 13 of them and lose 20, meaning that

$$P(\text{Federer wins match}) = \frac{13}{13 + 20} \approx 0.4.$$

¹² There are approximately 9,000 betting shops in the United Kingdom. In fact, it is estimated that approximately 4% of all retail storefronts in England are betting shops.

The markets had synthesized all the available information for you, and concluded that the pre-match probability of a Federer victory was just shy of 40%. (Djokovic ended up winning in five sets.)

Conditional probability

Another very important concept is that of a *conditional probability*. A conditional probability is the chance that some event A happens, given that another event B happens. We write this as $P(A | B)$ for short, where the bar ($|$) means “given” or “conditional upon.”

We’re all accustomed to thinking about conditional probabilities in our everyday lives, even if we don’t do so quantitatively. For example:

- $P(\text{rainy afternoon} | \text{cloudy morning}),$
- $P(\text{rough morning} | \text{out late last night}),$
- $P(\text{rough morning} | \text{out late last night, drank extra water}),$

and so forth. As the last example illustrates, it’s perfectly valid to condition on more than one event.

A key fact about conditional probabilities is that they are not symmetric: $P(A | B) \neq P(B | A)$. In fact, these two numbers are sometimes very different. For example, just about everybody who plays professional basketball in the NBA practices very hard:

$$P(\text{practices hard} | \text{plays in NBA}) \approx 1.$$

But sadly, most people who practice hard with a dream of playing in the NBA will fall short:

$$P(\text{plays in NBA} | \text{practices hard}) \approx 0.$$

We’ll see a few examples later where people get this wrong, and act as if $P(A | B)$ and $P(B | A)$ are the same. Don’t do this.

Conditional probabilities are used to make statements about uncertain events in a way that reflects our assumptions and our partial knowledge of a situation. They satisfy all the same rules as ordinary probabilities, and we can compare them as such. For example, we all know that

$$P(\text{rainy afternoon} | \text{clouds}) > P(\text{rainy afternoon} | \text{sun}),$$

$$P(\text{shark attack} | \text{swimming in ocean}) > P(\text{shark attack} | \text{watching TV}),$$

$$P(\text{heart disease} | \text{swimmer}) < P(\text{heart disease} | \text{couch potato}),$$

and so forth, even if we don’t know the exact numbers.

The rules of probability

Probability is an immensely useful language, and there are only a few basic rules. These are sometimes called Kolmogorov's rules, after a Russian mathematician. (Like chess and gymnastics, probability is a very Russian pursuit.)

- (1) All probabilities are numbers between 0 and 1, with 0 meaning impossible and 1 meaning certain.
- (2) Either an event occurs (A), or it doesn't (not A):

$$P(\text{not } A) = 1 - P(A).$$

- (3) If two events are mutually exclusive (i.e. they cannot both occur), then

$$P(A \text{ or } B) = P(A) + P(B).$$

These are usually called Kolmogorov's rules. There's also a fourth, slightly more advanced rule for conditional probabilities:

- (4) Let $P(A, B)$ be the *joint probability* that both A and B happen. Then the conditional probability $P(A | B)$ is:

$$P(A | B) = \frac{P(A, B)}{P(B)}. \quad (10.1)$$

An equivalent way of expressing Rule 4 is to multiply both sides of the equation by $P(B)$, to yield

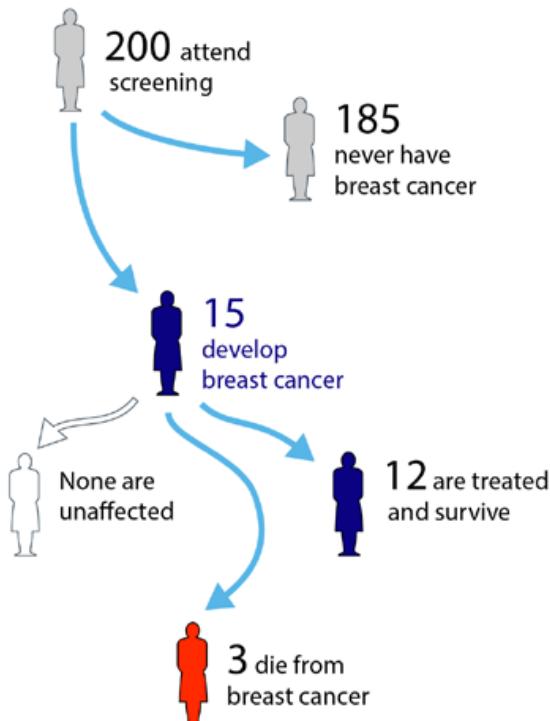
$$P(A, B) = P(A | B) \cdot P(B).$$

We can use these two versions interchangeably.

To illustrate these rules, we'll turn to Figure 10.1, which is the brainchild of David Spiegelhalter and Jenny Gage of the University of Cambridge. These researchers asked themselves the question: how can we present the evidence on the benefits and risks of screening in a way that doesn't make an explicit recommendation, but that helps people reach their own conclusion? The result of their efforts was a series of *probability trees* like Figure 10.1, each one depicting the likely experiences of women with and without screening.

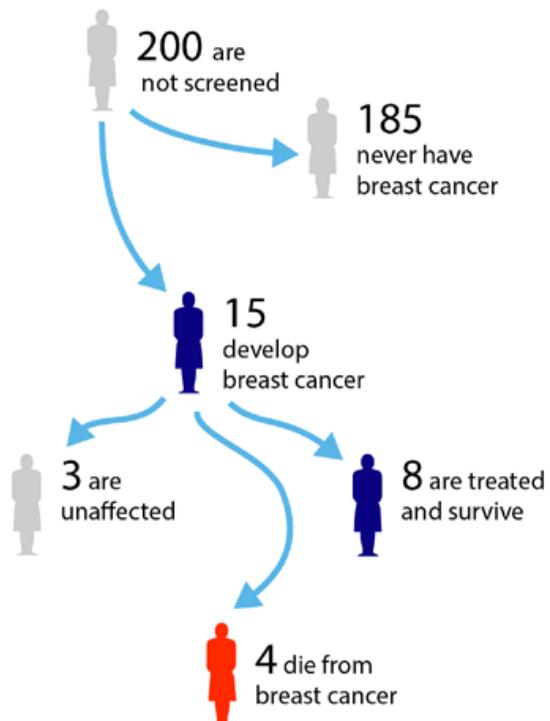
This particular figure tracks what we'd expect to happen to two hypothetical cohorts of 200 women, aged 50 to 70. In the cohort of 200 on the left, all women are screened; while in the cohort of

200 women between 50 and 70 who attend screening



3 more treatments, 1 fewer death

200 women between 50 and 70 who are not screened



3 fewer treatments, 1 extra death

200 on the right, none are screened. The expected results for each cohort are slightly different: on the right, we expect 1 fewer death, and 3 extra unnecessary screenings, versus the left.

Just about every major concept in probability is represented in this picture.

Expected value. In a group of 200 women, how many would we expect to get breast cancer? Our best guess, or expected value, is about 15, regardless of whether they get screened or not.

Probability. How likely is breast cancer for a typical woman? Fifteen cases of cancer in a cohort of 200 women means that an average woman aged 50-70 has a 7.5% chance of getting breast

Figure 10.1: Two hypothetical cohorts of 200 women, ages 50-70. The 200 women on the left all go in for mammograms; the 200 on the right do not. The branches of the tree show how many women we would expect to experience various different outcomes. Figure from: "What can education learn from real-world communication of risk and uncertainty?" David Spiegelhalter and Jenny Gage, University of Cambridge. *Proceedings of the Ninth International Conference on Teaching Statistics* (ICOTS9, July, 2014). We're not the only fans of the picture: it won an award for excellence in scientific communication in 2014 from the UK Association of Medical Research Charities.

cancer ($15/200 = 0.075$). This is like the NP rule in reverse: if E is the expected value (here 15), then the probability is $P = E/N$.

Joint probability. Suppose that a typical woman does not go for a screening mammogram. How likely is she to get breast cancer and to die from it? In the cohort of 200 unscreened women on the right, 4 are expected to get breast cancer and die from it. Thus the risk for a typical woman is about $4/200 = 0.02$, or 2%.

Conditional probability. Suppose that a woman decides to forego screening. If she then goes on to develop breast cancer, how likely is she to die from that cancer? In the unscreened cohort, 15 women are expected to get breast cancer. Of these 15 women, 4 are expected to die from their cancer. Thus for an unscreened 50-70 year-old woman, the risk of dying from breast cancer, given that she develops breast cancer in the first place, is about $4/15$, or about 27%. (Among screened women, this figure is $3/15$, or 20%).

Let's explicitly calculate this using the rule conditional probability (Equation 10.1) instead. The rule says

$$P(\text{survives} \mid \text{gets cancer}) = \frac{P(\text{gets cancer and survives})}{P(\text{gets cancer})}.$$

We'll take this equation piece by piece.

- Out of 200 women, we expect that 15 will develop cancer.
This is the denominator in our equation:

$$P(\text{gets cancer}) = \frac{15}{200}.$$

- Out of 200 women, we expect that 11 will develop cancer and survive. This is the numerator in our equation:

$$P(\text{gets cancer and survives}) = \frac{11}{200}.$$

- Therefore, using the rule for conditional probability,

$$P(\text{survives} \mid \text{cancer}) = \frac{11/200}{15/200} = 11/15.$$

Probability distributions

Now that we've learned the basic rules of probability, it's time to introduce the idea of a probability distribution.

Recall that a random variable is just a term for any uncertain outcome. We use the term *sample space* to mean the set of possible outcomes for a random variable. There are three common types of random variables, corresponding to three different types of sample spaces.

Categorical: the outcome will be one of many categories. For example, which party will win the next U.S. presidential election: Democrats, Republicans, or Other? Will your next interaction with customer service be Good, Fair, or Unrepeatable?

Discrete: the possible outcomes are whole numbers (1, 2, 3, etc.).

Most of the examples we saw in our discussion of everyday risks—numbers of shark attacks, falls in the shower, and so forth—were discrete random variables.

Continuous: the random variable could be anything within a continuous range of numbers, like the price of Apple stock tomorrow, or the size of subsurface oil reservoir.

Discrete and continuous random variables are sometimes grouped together and called *numerical* random variables, since the possible outcomes are all numbers.

A *probability distribution* is just a list of probabilities for all the outcomes in the sample space of a random variable. Here's a silly example that will get the idea across. Imagine that you've just pulled up to your new house after a long cross-country drive, only to discover that the movers have buggered off and left all your furniture and boxes sitting in the front yard.¹³ What a mess! You decide to ask your new neighbors for some help getting your stuff indoors. Assuming your neighbors are the kindly type, how many pairs of hands might come to your aid? Let's use the letter X to denote the (unknown) size of the household next door. The table at right shows a probability distribution for X , taken from U.S. census data in 2015; you might find this easier to visualize using the barplot in Figure 10.2.

This probability distribution provides a complete representation of your uncertainty in this situation. It has all the key features of any probability distribution:

1. There is a random variable, or uncertain quantity—here, the size of the household next door (X).
2. There is a *sample space*, or set of possible outcomes for the random variable—here, the numbers 1 through 8.

¹³ This actually happened to a friend of mine. -JS

| Size of household, x | Probability, $P(X = x)$ |
|------------------------|-------------------------|
| 1 | 0.280 |
| 2 | 0.336 |
| 3 | 0.155 |
| 4 | 0.132 |
| 5 | 0.060 |
| 6 | 0.023 |
| 7 | 0.011 |
| 8 | 0.003 |

Table 10.3: Probability distribution for household size in the U.S. in 2015. There is a vanishingly small probability for a household of size 9 or higher, which is just rounded off to zero here.

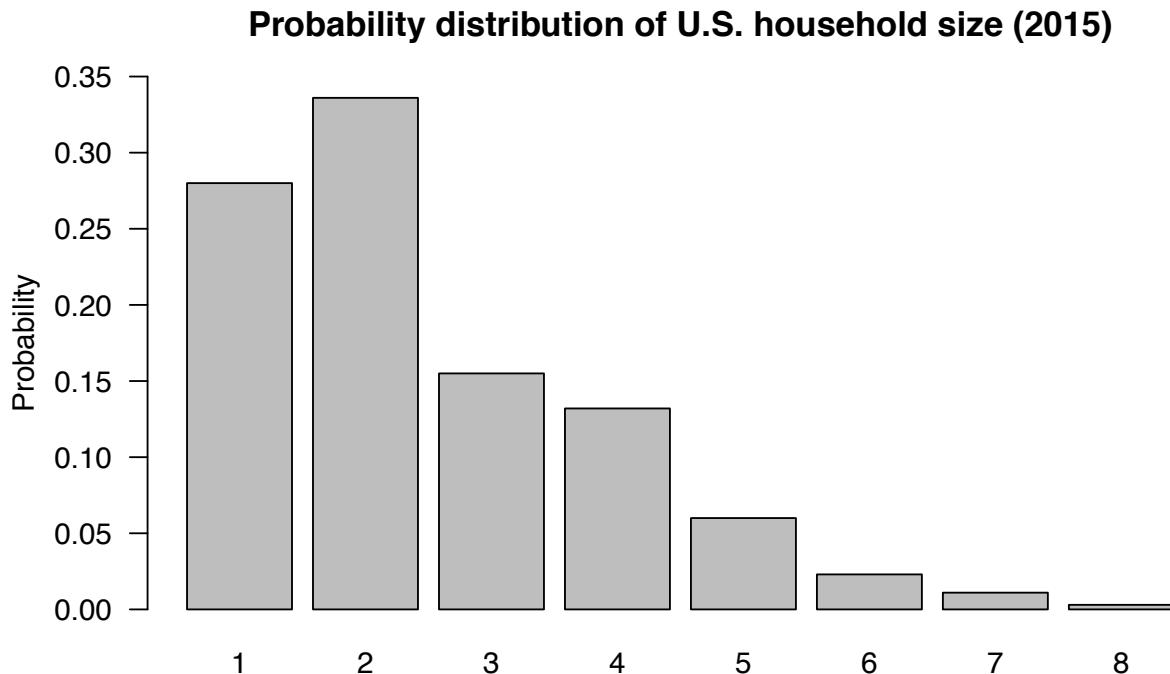


Figure 10.2: Probability distribution for the size of a random U.S. household in 2015. The elements of the sample space (the numbers $x = 1$ through $x = 8$) are shown along the horizontal axis. The probabilities $P(X = x)$ are shown on the vertical axis.

- Finally, there are probabilities for each outcome in the sample space—here provided via a simple look-up table. Notice that the table uses big X to denote the random variable itself, and little x to denote the elements of the sample space.

Most probability distributions won't be this simple, but they will all require specifying these three features.

Expected value: the mathematical definition

When you knock on your neighbors' door in the hopes of getting some help with your moving fiasco, how many people should you “expect” to be living there?

The *expected value* of a probability distribution for a numerical random variable is just an average of the items in the sample space—but a weighted average, rather than an ordinary average. If you take the 8 numbers in the sample space of Figure 10.2 and form their ordinary average, you get

$$\text{Ordinary average} = \frac{1}{8} \cdot 1 + \frac{1}{8} \cdot 2 + \cdots + \frac{1}{8} \cdot 7 + \frac{1}{8} \cdot 8 = 4.5.$$

Here, the weight on each number in the sample space is $1/8 = 0.125$, since there are 8 numbers. This is *not* the expected value; it give each number in the sample space an equal weight, ignoring the fact that these numbers have different probabilities.

To calculate an expected value, we instead form an average using *unequal* weights, given by the probabilities of each item in the sample space:

$$\text{Expected value} = (0.280) \cdot 1 + (0.336) \cdot 2 + \dots + (0.011) \cdot 7 + (0.003) \cdot 8 \approx 2.5.$$

The more likely numbers (e.g. 1 and 2) get higher weights than $1/8$, while the unlikely numbers (e.g. 7 and 8) get lower weights.

This example conveys something important about expected values. Even if the world is black and white, an expected value is often grey. For example, the expected American household size is 2.5 people, a baseball player expects to get 0.25 hits per at bat, and so forth.

As a general rule, suppose that the possible outcomes for a random variable X are the numbers x_1, \dots, x_N . The formal definition for the expected value of X is

$$E(X) = \sum_{i=1}^N P(X = x_i) \cdot x_i. \quad (10.2)$$

This measures the “center” or mean of the probability distribution. Later, we’ll learn how this more formal definition of expected value can be reconciled with the NP rule—that is, with our previous understanding of expected value as a risk/frequency calculation.

A related concept is the *variance*, which measures the dispersion or spread of a probability distribution. It is the expected (squared) deviation from the mean, or

$$\text{var}(X) = E(\{X - E(X)\}^2).$$

The standard deviation of a probability distribution is $\sigma = \text{sd}(X) = \sqrt{\text{var}(X)}$. The standard deviation is more interpretable than the variance, because it has the same units (dollars, miles, etc.) as the random variable itself.

11

Conditional probability

IN probability, as with many things in life, the real skill is in learning to ask the right question in the first place. As we'll discover, "asking the right question" usually means focusing on the right conditional probability.

Conditional probability: the art of asking the right question

DURING World War II, the size of the Allied air campaign over Europe was truly staggering. Every morning, huge squadrons of B-17 Flying Fortress bombers, each with a crew of 10 men, would take off from their air bases in the south of England, to make their way across the Channel and onwards to their targets in Germany. By 1943, they were dropping nearly 1 million pounds of bombs per week. At its peak strength, in 1944, the U.S. Army Air Forces (AAF) had 80,000 aircraft and 2.6 million people—4% of the U.S. male population—in service.

As the air campaign escalated, so too did the losses. In 1942, the AAF lost 1,727 planes; in 1943, 6,619; and in 1944, 20,394. And the bad days were very bad. In a single mission over Germany in August of 1943, 376 B-17 bombers were dispatched from 16 different air bases in the south of England, in a joint bombing raid on factories in Schweinfurt and Regensburg. Only 316 planes came back—a daily loss rate of 16%. Some units were devastated; the 381st Bomb Group, flying out of RAF Ridgewell, lost 9 of its 20 bombers that day.¹

Like Yossarian in *Catch-22*, World War II airmen were painfully aware that each combat mission was a roll of the dice. What's more, they had to complete 25 missions to be sent home. With such poor chances of returning from a *single* mission, they could be forgiven for thinking that they'd been sent to England to die.

But in the face of these bleak odds, the crews of the B-17s had at

¹ Numbers taken from *Statistical Abstract of the United States*, U.S. Census Bureau, (1944, 1947, 1950); and the Army Air Forces Statistical Digest (World War II), available at archive.org.

least three major defenses.

1. Their own tail and turret gunners, to defend the plane below and from the rear.
2. Their fighter escorts: the squadrons of P-47 Thunderbolts, RAF Spitfires, and P-51 Mustangs sent along to protect the bombers from the Luftwaffe.
3. A Hungarian-American statistician named Abraham Wald.

Abraham Wald never shot down a Messerschmitt or even saw the inside of a combat aircraft. Nonetheless, he made an out-sized contribution to the Allied war effort, and no doubt saved the lives of many American bomber crews, using an equally potent weapon: conditional probability.

Where should the military reinforce its planes?

Abraham Wald was born in 1902 in Austria-Hungary, where he went on to earn a Ph.D. in mathematics from the University of Vienna. Wald was Jewish, and when the Nazis invaded in 1938, he—like so many brilliant European mathematicians and scientists of that era—fled to America.

Wald soon went to work as part of the Applied Mathematics Panel, which had been convened by order of President Roosevelt to function as something of a mathematical tech-support hotline for the U.S. military. It was during these years of service to his adopted country that Wald prevented the military brass from making a major blunder, thereby saving many lives.

Here's the problem Wald analyzed.² While some airplanes came back from bombing missions in Germany unscathed, many others had visibly taken hits from enemy fire. In fact, someone examining the planes just after they landed would likely have found bullet holes and flak damage everywhere: on the fuselage, across the wings, on the engine block, and sometimes even near the cockpit.

At some point, a clever person, whose identity is lost to history, had the idea of analyzing the distribution of these hits over the surface of the returning planes. The thinking was that, if you could find patterns in where the B-17s were taking enemy fire, you could figure out where to reinforce them with extra armor, to improve survivability. (You couldn't reinforce them everywhere, or they would be too heavy to fly.)



Figure 11.1: Abraham Wald.

² Distilled from: Mangel and Samaniego, "Abraham Wald's work on aircraft survivability." *Journal of the American Statistical Association* 79 (386): 259-67.

Researchers at the Center for Naval Analyses took this idea and ran with it. They examined data on hundreds of damaged airplanes that had returned from bombing runs in Germany. They found a very striking pattern³ in where the planes had taken enemy fire. It looked something like this:

| Location | Number of planes |
|-----------------------|------------------|
| Engine | 53 |
| Cockpit area | 65 |
| Fuel system | 96 |
| Wings, fuselage, etc. | 434 |

If you turn those frequencies into probabilities, so that the numbers sum to 1, you get the following. (You should recall that a table of probabilities for the different possible outcomes of a random event, like this one, is called a probability distribution.)

| Location | Probability of hit |
|-----------------------|--------------------|
| Engine | 0.08 |
| Cockpit area | 0.10 |
| Fuel system | 0.15 |
| Wings, fuselage, etc. | 0.67 |

Thus of all the planes that took hits and made it back to base, 67% of them had taken those hits on the wings and fuselage. In the aggregate, no other part of the plane had taken nearly as much damage. The Navy researchers reached a simple conclusion: put more armor on the wings and fuselage.

The tale of the missing bombers

But Wald pointed out that this recommendation suffered from a crucial flaw: *the Navy researchers only had data on the survivors*. The planes that had been shot down were missing from the analysis—and only the pattern of bullet holes on those missing planes could definitively tell the story of a B-17's vulnerabilities. In fact, Wald's thinking was nearly the opposite of the Navy researchers. If all those planes had safely made it home with damage to the wings and fuselage, then these areas probably weren't all that vulnerable!

Wald's argument was, essentially, that the Navy researchers were focusing on the wrong conditional probability. They had

³ Alas, the actual data used in the original analyses cannot be located. But Wald wrote a report for the Navy on his methods, and we have attempted to simulate a data set that hews as closely as possible to the assumptions and (patchy) information that he provides in that report ("A Method of Estimating Plane Vulnerability Based on Damage of Survivors", from 1943). These and subsequent numbers are for hypothetical cohort of 800 airplanes, all taking damage.

looked at the data and concluded that the wings and fuselage were vulnerable, on the basis of the fact that

$$P(\text{hit on wings or fuselage} \mid \text{returns safely}) \approx 0.67.$$

But that's the right answer to the wrong question. Instead, the Navy researchers should have tried to calculate the *inverse* probability, namely

$$P(\text{returns safely} \mid \text{hit on wings or fuselage}) = ?$$

This might be a very different number. Remember: $P(\text{practices hard} \mid \text{plays in NBA}) \approx 1$, while $P(\text{plays in NBA} \mid \text{practices hard}) \approx 0$.

Of course, Wald had no data on the planes that had been shot down. Therefore, to actually calculate the probability $P(\text{returns safely} \mid \text{hit on wings or fuselage})$ required that Wald approach the data set like a forensic scientist. Essentially, he had to reconstruct the typical encounter of a B-17 with an enemy fighter, using only the mute testimony of the bullet holes on the planes that had made it back, coupled with some educated guessing.

So Wald went to work. He analyzed the likely attack angle of enemy fighters. He chatted with engineers. He studied the properties of a shrapnel cloud from a flak gun. He suggested to the army that they fire thousands of dummy bullets at a plane sitting on the tarmac. And yes, he did a lot of math.⁴

Remarkably, when all was said and done, Wald was able to reconstruct an estimate for the *joint probabilities* for the two distinct types of events that each airplane experienced: where it took a hit, and whether it returned home safely. In other words, although Wald couldn't bring the missing planes back into the air, he could bring their statistical signature back into the data set. For our hypothetical cohort of 800 bombers that took damage, Wald's best guess would have looked something like this:

| | Returned | Shot down |
|-----------------------|----------|-----------|
| Engine | 53 | 57 |
| Cockpit area | 65 | 46 |
| Fuel system | 96 | 16 |
| Wings, fuselage, etc. | 434 | 33 |

For example, Wald's method would have estimated that 53 of the 800 planes, or 6.6% overall, experienced the joint event (hit type

⁴ We don't go into detail on Wald's methods here, which were very complex. But later statisticians have taken a second look at those methods, with the hindsight provided by subsequent advances in the field. They have concluded, very simply: "Wald's treatment of these problems was definitive." (Mangel and Samaniego, *ibid.*)

Table 11.1: An example of how Abraham Wald could have reconstructed the joint frequency distribution over hit type and outcome for our hypothetical cohort of 800 planes taking enemy fire.

= engine, outcome = returned home safely). You'll notice that the numbers in the left column correspond exactly to the table given earlier: the pattern of hits to airplanes that made it back home.

What's new is the right column: Wald's forensic reconstruction of the pattern of hits to planes that had been shot down.

This estimate for the joint frequencies for two random outcomes, hit type and outcome, now allowed Wald to answer the right question. Of the 467 planes that had taken hits to wings and fuselage, 434 of them had returned home, while 33 of them had not. Thus Wald estimated that the conditional probability of survival, given a hit to the wings and fuselage, was

$$P(\text{returns safely} \mid \text{hit on wings or fuselage}) = \frac{434}{434 + 33} \approx 0.93.$$

It turns out that B-17s were pretty robust to taking hits on the wings or fuselage.

On the other hand, of the 110 planes that had taken damage to the engine, only 53 only returned safely. Therefore

$$P(\text{returns safely} \mid \text{hit on engine}) = \frac{53}{53 + 57} \approx 0.48.$$

Similarly,

$$P(\text{returns safely} \mid \text{hit on cockpit area}) = \frac{65}{65 + 46} \approx 0.59.$$

The bombers were much more likely to get shot down if they took a hit to the engine or cockpit area.

Thus Wald's final, life-saving advice ran exactly counter to that of the experts at the Center for Naval Analyses: *put the armor where you don't see the bullet holes.*

Postscript. In the story of Abraham Wald and the missing B-17s, the path of counterintuitive facts eventually turns a full 360 degrees. Imagine asking any random person off the street: "Where should we put extra armor on airplanes to help them survive enemy fire?" We haven't done this survey, but we strongly suspect that most thoughtful people would answer: where the pilot and the engines are! But the data initially seem to suggest otherwise. This implies that we should turn 180 degrees away from our intuition: if the planes are taking damage on the wings and the fuselage, then let's put the armor there instead. It turns out that only a very careful analysis like Wald's can rehabilitate the initial, intuitive guess over the spurious attempt at data-based reasoning.

The moral of the story is that data alone isn't enough. You have to know enough about conditional probability to be able to pose the right question in the first place.

How Netflix knows your taste in movies so well

THE same math that Abraham Wald used to analyze bullet holes on B-17s also underpins the modern digital economy of films, television, music, and social media. To give one example: Netflix, Hulu, and other video-streaming services all use this same math to examine what shows their users are watching, and apply the results of their number-crunching to recommend new shows.

To see how this works, suppose that you're designing the movie-recommendation algorithm for Netflix, and you have access to the entire Netflix database, showing which customers have liked which films—for example, by assigning a film a five-star rating. Your goal is to leverage this vast data resource to make automated, personalized movie recommendations. The better these recommendations are, the more likely your customers are to keep their accounts on auto-pay.

You decide to start with an easy case: assessing how probable it is that a user will like the film *Saving Private Ryan* (event *A*), given that the same user has liked the HBO series *Band of Brothers* (event *B*). This is almost certainly a good bet: both are epic dramas about the Normandy invasion and its aftermath. Therefore, you might think: job done! Recommend away.

For this particular pair of shows, fine. But keep in mind that you want to be able to do this kind of thing automatically. It would not be cost effective to put a human in the loop here, laboriously tagging all possible pairs of movies for similar themes or content—to say nothing of all of the other stuff that might make two different films appeal to the same person.

As with Abraham Wald and the missing bombers, it's all about asking the right question. The key insight here is to frame the problem in terms of conditional probability. Suppose that, for some pair of films *A* and *B*, the probability $P(\text{random user likes } A \mid \text{random user likes } B)$ is high—say, 80%. Now we learn that Linda liked film *B*, but hasn't yet seen film *A*. Wouldn't *A* be a good recommendation? Based on her liking of *A*, there's an 80% chance she'll like it.

But how can we learn $P(\text{likes } A \mid \text{likes } B)$? This is where your database, coupled with the rule for conditional probability, comes in handy. Suppose that there are 5 million people in your database who have seen both *Saving Private Ryan* and *Band of Brothers*, and that the ratings data on these 5 million users looks like this:

| | Liked <i>Band of Brothers</i> | Didn't like |
|----------------------------------|-------------------------------|-------------|
| Liked <i>Saving Private Ryan</i> | 2.8 million | 0.3 million |
| Didn't like | 0.7 million | 1.2 million |

Once again, we have a joint frequency distribution for two random outcomes: A = whether a user liked *Saving Private Ryan*, and B = whether the user liked *Band of Brothers*. From this joint distribution, we can easily work out the conditional probability that we need. Of the 5 million users in the database who have watched both programs, $2.8 + 0.7 = 3.5$ million of them liked *Band of Brothers*. Of these 3.5 million people, 2.8 million (or 80%) also liked *Saving Private Ryan*. Therefore,

$$P(\text{liked } \textit{Saving Private Ryan} \mid \text{liked } \textit{Band of Brothers}) = \frac{2.8 \text{ million}}{3.5 \text{ million}} = 0.8.$$

Note that you could also jump straight to the math, and use the rule for conditional probabilities (Equation 10.1, on page 214), like this:

$$P(A \mid B) = \frac{P(A, B)}{P(B)} = \frac{2.8/5}{(2.8 + 0.7)/5} = 0.8.$$

You'd get the same answer in the end.

The key thing that makes this approach work so well is that it's automatic. Computers aren't very good (yet) at automatically scanning films for thematic content. But they're brilliant at calculating conditional probabilities from a vast database of users' movie-watching histories.

The same trick works for books, too. Suppose you examine the online book-purchase histories of two friends Albert and Pablo, and discovered the following items.

Albert: (1) Proof and Consequences. (2) A Body in Motion: Newton's Guide to Productivity. (3) Obscure Theorems of the 14th Century.

Pablo: (1) Your Face is Offside: Dora Maar at the Cubist Soccer

Match. (2) A Short History of Non-representational Art. (3) Achtung, Maybe? Dali, Danger, and the Surreal.

What sorts of books are you likely to recommend to these friends for their birthdays? Amazon learned to use conditional probability to automate this process long ago, to the chagrin of independent bookstores everywhere. Similar math also underpins recommender systems for music (Spotify), ads (Google), and even friends (Facebook).

The digital economy truly is ruled by conditional probability.

The math of conditional probability

To understand the basic math behind joint, conditional, and marginal probabilities, we'll return to the story of Abraham Wald and the B-17s.

Joint probabilities

We start by turning Table 11.1, which contains counts of different joint event types for a cohort of 800 airplanes, into a table of probabilities:

| | Returned | Shot down |
|-----------------------|----------|-----------|
| Engine | 0.066 | 0.071 |
| Cockpit area | 0.081 | 0.058 |
| Fuel system | 0.120 | 0.020 |
| Wings, fuselage, etc. | 0.542 | 0.042 |

This table gives summarizes the probabilities for two random outcomes: X = hit type, along the rows; and Y = outcome, along the columns. The entries in the table give the joint probabilities $P(X = x, Y = y)$. For example, 2% of all planes both took a hit in the fuel system and got shot down: $P(X = \text{fuel system}, Y = \text{shot down}) = 0.02$. Up to round-off error, these 8 probabilities all sum to 1.

We call a table like this a *joint probability distribution*: that is, a list of all possible joint events for multiple random variables, together with their joint probabilities.

Marginal probabilities

Next, we add an additional row and column of *marginal* (or overall) probabilities of the different event types and outcomes, like in the table below. These are called the marginal probabilities because we calculate them by summing across the relevant margin (i.e. row or column) of the table.

| | Returned | Shot down | Marginal |
|-----------------------|----------|-----------|----------|
| Engine | 0.066 | 0.071 | 0.137 |
| Cockpit area | 0.081 | 0.058 | 0.139 |
| Fuel system | 0.120 | 0.020 | 0.140 |
| Wings, fuselage, etc. | 0.542 | 0.042 | 0.584 |
| Marginal | 0.809 | 0.191 | 1 |

The marginal probabilities we've calculated just reflect the fact that the probability of some event (like returning safely) is the sum of the probabilities for all the distinct ways that event can happen. For example, an airplane that takes a hit to the engine can do so in two ways: it can take the hit and return, or it can take the hit and not return. Therefore,

$$\begin{aligned} P(\text{hit to engine}) &= P(\text{returned, hit to engine}) + P(\text{shot down, hit to engine}) \\ &= 0.066 + 0.071 = 0.137. \end{aligned}$$

The rest of the marginal probabilities are calculated similarly, e.g.

$$\begin{aligned} P(\text{returned}) &= 0.066 + 0.081 + 0.120 + 0.542 \\ &= 0.809. \end{aligned}$$

Conditional probabilities

Finally, we are ready to understand the rule for conditional probabilities. You'll recall that this was the fourth of the basic rules of probability quoted earlier. It goes like this:

$$P(A | B) = \frac{P(A, B)}{P(B)}.$$

Remember how we used Table 11.1 to calculate $P(\text{returns} | \text{hit to engine})$? We looked at the total number of planes that had taken a hit to the engine. We then asked: of these planes, how

many also returned home safely? As an equation, this gives us

$$\begin{aligned} P(\text{return} | \text{engine hit}) &= \frac{\text{Number taking engine hit and returned safely}}{\text{Number taking engine hit}} \\ &= \frac{53}{110} \approx 0.48. \end{aligned}$$

You'll notice we get the exact same answer if we use the rule for conditional probabilities: $P(A | B) = P(A, B) / P(B)$. These probabilities are estimated using the relevant fractions from the data set:

$$\begin{aligned} P(\text{return} | \text{engine hit}) &= \frac{\text{Fraction taking engine hit and returning safely}}{\text{Fraction taking engine hit}} \\ &= \frac{53/800}{110/800} \\ &= \frac{0.066}{0.137} \approx 0.48. \end{aligned}$$

While the rule for conditional probabilities may look a bit intimidating, it just codifies exactly the same intuition we used to calculate $P(\text{return} | \text{engine hit})$ from the table of counts.

The rule of total probability

CONSIDER the following data on obstetricians delivering babies at a hospital in England. The table below shows the complication rates for both junior and senior doctors on the delivery ward, grouped by delivery type:

| | Easier deliveries | Harder deliveries | Overall |
|----------------|-------------------|-------------------|--------------|
| Senior doctors | 0.052 (213) | 0.127 (102) | 0.076 (315) |
| Junior doctors | 0.067 (3169) | 0.155 (206) | 0.072 (3375) |

The numbers in parentheses are the total deliveries of each type.

This table exhibits an aggregation paradox.⁵ No matter what kind of delivery you have, whether easy or hard, you'd prefer to have a senior doctor. They have lower complication rates than junior doctors in both cases. Yet counterintuitively, the senior doctors have a higher overall complication rate: 7.6% versus 7.2%. Why? Because of a lurking variable: most of the deliveries performed by junior doctors are easier cases, where complication rates

⁵ Also called *Simpson's paradox*.

are lower overall. The senior doctors, meanwhile, work a much higher fraction of the harder cases. Their overall complication rate reflects this burden.

Here's another example. Jacoby Ellsbury and Mike Lowell were two baseball players for the Boston Red Sox during the 2007 and 2008 seasons. The table below shows their batting averages for those two seasons, with their number of at-bats in parentheses. We see that Ellsbury had a higher batting average when he was a rookie, in 2007; a higher batting average a year later, in 2008; but a lower batting average overall!

| | 2007 | 2008 | Overall |
|----------|------------|------------|-------------|
| Lowell | .324 (589) | .274 (419) | .304 (1008) |
| Ellsbury | .353 (116) | .280 (554) | .293 (670) |

Again we have an aggregation paradox, and again it is resolved by pointing to a lurking variable: in 2007, when both players had higher averages, Ellsbury had many fewer at-bats than Lowell.

It turns out the math of these aggregation paradoxes can be understood a lot more deeply in terms of something called the *rule of total probability*, or the *mixture rule*. This rule sounds impressive, but is actually quite simple. It says: the probability of any event is the sum of the probabilities for all the different ways in which the event can happen. In that sense, the law of total probability is really just Kolmogorov's third rule in disguise. The distinct ways in which some event A can happen are mutually exclusive. Therefore we just sum all their probabilities together to get $P(A)$.

Let's return to the example on obstetric complication rates on junior doctors at a hospital in England. In the table, there are two ways of having a complication: with an easy case, or with a hard case. Therefore, the total probability is the sum of two joint probabilities:

$$P(\text{complication}) = P(\text{easy and complication}) + P(\text{hard and complication}).$$

If we now apply the rule for conditional probabilities (Equation 10.1) to each of the two joint probabilities on the right-hand side of this equation, we have this:

$$P(\text{complication}) = P(\text{easy}) \cdot P(\text{complication} \mid \text{easy}) + P(\text{hard}) \cdot P(\text{complication} \mid \text{hard})$$

Thus the rule of total probability says that overall probability is a weighted average—a mixture—of the two conditional probabilities.

For senior doctors we get

$$P(\text{complication}) = \frac{213}{315} \cdot 0.052 + \frac{102}{315} \cdot 0.127 = 0.076.$$

And for junior doctors, we get

$$P(\text{complication}) = \frac{3169}{3375} \cdot 0.067 + \frac{206}{3375} \cdot 0.155 = 0.072.$$

This is a lower *overall* probability of a complication, despite the fact the junior doctors have higher conditional probabilities of a complication in all scenarios.

So which probabilities should we report: the conditional probabilities, or the overall (total) probabilities? There's no one right answer; it depends on your conditioning variable, and your goals. In the obstetric data, the overall complication rates are clearly misleading. The distinction between easier and harder cases matters a lot. Senior doctors work harder cases, on average, and therefore have higher overall complication rates. But what matters to the patient, and to anyone who assesses the doctors' performance, are the *conditional* rates. You have to account for the lurking variable.

The baseball data is different. Here the *conditional* probabilities for 2007 and 2008 are probably misleading. The distinction between 2007 and 2008 is nothing more than an arbitrary cutoff on the calendar. It's barely relevant from the standpoint of assessing baseball skill, and it needlessly splits one big sample of each player's history into two smaller, more variable samples. So in this case we'd probably go with the overall averages if we wanted to say which player was performing better.

A formal statement of the rule of total probability. Suppose that events B_1, B_2, \dots, B_N constitute an exhaustive partition of all possibilities in some situation. That is, the events themselves are mutually exclusive, but one of them must happen. This can be expressed mathematically as

$$P(B_i, B_j) = 0 \text{ for any } i \neq j, \quad \text{and} \quad \sum_{i=1}^N P(B_i) = 1. \quad (11.1)$$

Now consider any event A . If Equation 11.1 holds, then

$$P(A) = \sum_{i=1}^N P(A, B_i) = \sum_{i=1}^N P(B_i) \cdot P(A | B_i). \quad (11.2)$$

Equation 11.2 is what is usually called the rule of total probability.

Surveys and the rule of total probability

ONE of the least surprising headlines of 2010 must surely have been the following, from the ABC News website:

Teens not always honest about drug use.⁶

In other news, dog bites man.

To be fair, the story itself was a bit more surprising than the headline. Yes, it's hardly news that teenagers would lie to their parents, teachers, coaches, and priests about drug use. But the ABC News story was actually reporting on a study showing that teenagers also lie to researchers who conduct anonymous surveys about drug use—even when those teenagers know that their answers will be verified using a drug test.

Here's the gist of the study. Virginia Delaney-Black and her colleagues at Wayne State University, in Detroit, gave an anonymous survey to 432 teenagers, asking whether they had used various illegal drugs.⁷ Of these 432 teens, 211 of them also agreed to give a hair sample. Therefore, for these 211 respondents, the researchers could compare people's answers with an actual drug test.

The two sets of results were strikingly different. For example, of the 211 teens who provided a hair sample, only a tiny fraction of them (0.7%) admitted to having used cocaine. However, when the hair samples were analyzed in the lab, 69 of them (33.7%) came back positive for cocaine use.

And it wasn't just the teens who lied. The survey researchers also asked the *parents* of the teens whether they themselves had used cocaine. Only 6.1% said yes, but 28.3% of the hair samples came back positive.

Let's emphasize again that we're talking about a group of people who were guaranteed anonymity, who wouldn't be arrested or fired for saying yes, and who willingly agreed to provide a hair sample that they knew could be used to verify their survey answers. Yet a big fraction lied about their drug use anyway.

Surveys and lies

Drug abuse—whether it's crack cocaine in Detroit, or bathtub speed in rural Nebraska—is a huge social problem. It fills our jails, drains public finances, and perpetuates a trans-generational cycle of poverty. Getting good data on this problem is important.

⁶ Kim Carollo, ABC News, Oct. 25, 2010. [Link here.](#)

⁷ V. Delaney-Black et. al. "Just Say 'I Don't': Lack of Concordance Between Teen Report and Biological Measures of Drug Use." *Pediatrics* 165:5, pp. 887-93 (2010).

As it stands, pediatricians, schools, and governments all rely on self-reported measures of drug use to guide their thinking on this issue. Yet distressingly, the proportion of self-reported cocaine use in the Detroit study, 0.7%, was broadly similar to the findings in large, highly regarded surveys—for example, the federally funded [National Survey on Drug Use and Health](#). The work of Dr. Delaney-Black and her colleagues would seem to imply that all of these self-reported figures might be way off the mark.

Moreover, theirs hasn't been the only study to uncover evidence that surveys cannot necessarily be taken at face value. Here are some other things that, according to research *on* surveys, people lie about *in* surveys.

- Churchgoers overstate the amount of money they give when the hat gets passed around during the service.
- Gang members embellish the number of violent encounters they have been in.
- Men exaggerate their salary, among other things.
- Ravers will “confess” to having gotten high on drugs that do not actually exist.

How to ask an embarrassing question: probability as an invisibility cloak

But there's actually some good news to be found here. It's this: when people lie in surveys, they tend to do so for predictable reasons (to impress someone or avoid embarrassment), and in predictable ways (higher salary, fewer warts). This opens the door for survey designers to use a bit of probability, and a bit of psychology, to get at the truth—even in a world of liars.

Let's go back to the example of drug-use surveys so that we can see this idea play out. Suppose that you want to learn about the prevalence of drug use among college students. You decide to conduct a survey at a large state university to find out how many of the students there have smoked marijuana in the last year. But as you now appreciate, if you ask people direct questions about drugs, you can't always trust their answers.

Here's a cute trick for alleviating this problem, in a way that uses probability theory to mitigate someone's psychological incentive to lie. Suppose that, instead of asking people point-blank about marijuana, you give them these instructions.

1. Flip a coin. Look at the result, but keep it private.
2. If the coin comes up heads, please use the space provided to write an answer to question Q1: “Is the last digit of your Social Security number odd?”
3. If the coin comes up tails, please use the space provided to write an answer to question Q2: “Have you smoked marijuana in the last year?”

The key fact here is that only the respondent knows which question he or she is answering. This gives people plausible deniability. Someone answering “yes” might have easily flipped heads and answered the first, innocuous question rather than the second, embarrassing one, and the designer of the survey would never know the difference. This reduces the incentive to lie.

Moreover, despite the partial invisibility cloak we’ve provided to the marijuana users in our sample, we can still use the results of the survey to answer the question we care about: what fraction of students have used marijuana in the past year? We’ll use the following notation:

- Let Y be the event “a randomly chosen student answers yes.”
- Let Q_1 be the event “the student provided an answer to question 1, about their Social Security number.”
- Let Q_2 be the event “the student provided an answer to question 2, about their marijuana use.”

From the survey, we have an estimate of $P(Y)$, which is the overall fraction of survey respondents providing a “yes” answer. We really want to know $P(Y | Q_2)$, the probability that a randomly chosen student will answer “yes”, given that he or she was answering the marijuana question. The problem is that we don’t know which students were answering the marijuana question.

To understand the rule of total probability, let’s return to our hypothetical survey in which we want to know the answer to the question: what fraction of students have used marijuana in the past year? Then we have each survey respondent privately flip a coin to determine whether they answer an innocuous question (Q_1) or the question about marijuana use (Q_2). We used the following notation:

- Let Y be the event “a randomly chosen student answers yes.”

- Let Q_1 be the event “the student provided an answer to question 1, about their Social Security number.”
- Let Q_2 be the event “the student provided an answer to question 2, about their marijuana use.”

To solve this problem, we’ll use rule of total probability. In the case of our drug-use survey, this means that

$$P(Y) = P(Y, Q_1) + P(Y, Q_2). \quad (11.3)$$

In words, this equation says that there are two ways to get a yes answer: from someone answering the social-security-number question, and from someone answering the drugs question. The total number of yes answers will be the sum of the yes answers from both types in this mixture.

Now let’s re-write Equation 11.3 slightly, by applying the rule for conditional probabilities to each of the two joint probabilities on the right-hand side of this equation:

$$P(Y) = P(Q_1) \cdot P(Y | Q_1) + P(Q_2) \cdot P(Y | Q_2). \quad (11.4)$$

This equation now says that the overall probability $P(Y)$ is a weighted average of two conditional probabilities:

- $P(Y | Q_1)$, the probability that a randomly chosen student will answer “yes”, given that he or she was answering the social-security-number question.
- $P(Y | Q_2)$, the probability that a randomly chosen student will answer “yes”, given that he or she was answering the marijuana question.

The weights in this average are the probabilities for each question: $P(Q_1)$ and $P(Q_2)$, respectively.

Now we’re ready to use Equation 11.4 to calculate the probability that we care about: $P(Y | Q_2)$. We know that $P(Q_1) = P(Q_2) = 0.5$, since a coin flip was used to determine whether Q_1 or Q_2 was answered. Moreover, we also know that $P(Y | Q_1) = 0.5$, since it is equally likely that someone’s Social Security number will end in an even or odd digit.⁸

We can use this information to simplify the equation above:

$$P(Y) = 0.5 \cdot 0.5 + 0.5 \cdot P(Y | Q_2),$$

or equivalently,

$$P(Y | Q_2) = 2 \cdot \{P(Y) - 0.25\}.$$

⁸ This survey design relies upon the fact that the survey designer doesn’t know anyone’s Social Security number. If you were running this survey in a large company, where people’s SSNs were actually on file, you’d need to come up with some other innocuous question whose answer was unknown to the employer, but for which $P(Y | Q_1)$ was known.

Suppose, for example, that 35% of survey respondents answer yes, so that $P(Y) = 0.35$. This implies that

$$P(Y | Q_2) = 2 \cdot (0.35 - 0.25) = 0.2.$$

We would therefore estimate that about 20% of students have smoked marijuana in the last year.

12

Independence and compounding

LONG unbroken runs of outstanding performance, whether in sports or life, hold a special fascination. They can teach us a lot about probability—specifically, something called the compounding rule—and about the idea of a lurking variable.

Joltin' Joe and the compounding rule

JOE DiMaggio, widely regarded as one of the greatest baseball players in history, was a mid-century American icon. Born in 1914 to a poor family of Italian immigrants in California, "Joltin' Joe" would eventually reach a level of fame that transcended sport. Ordinary people regarded him as a folk hero. Marilyn Monroe eloped with him. Hundreds of writers and artists—from Hemingway to Madonna, Rodgers and Hammerstein to Simon and Garfunkel—mentioned him in their most enduring works.

Why was DiMaggio so famous? Why, in 1999, during the last days of his final battle with lung cancer, did the *New York Times* describe the scene as a "national vigil"?

Part of it was DiMaggio's courtly manner. His teammate Phil Rizzuto said of him:

There was an aura about him. He walked like no one else walked. He did things so easily. He was immaculate in everything he did. Kings of State wanted to meet him and be with him. He could fit in any place in the world.¹

When DiMaggio died in 1999, he was buried in a grave bearing a simple inscription: "Dignity, grace, and elegance personified."

Of course, the larger part of DiMaggio's fame derived from his accomplishments on the baseball diamond. His most impressive feat there undoubtedly came over the summer of 1941, when he successfully got a hit in 56 straight games. This singular, record-smashing performance put DiMaggio squarely in the national

¹ Baseball Hall of Fame biography of Joe DiMaggio, <http://baseballhall.org/hof/dimaggio-joe>.

spotlight for the rest of his life. As of 2016, his 56-game hitting streak is still the longest ever; most baseball fans consider it unbeatable. In fact, Stephen Jay Gould, the eminent biologist and baseball fan, once called DiMaggio's hitting streak "the most extraordinary thing that ever happened in American sports."²

So if you want to know why Joe DiMaggio was such a cultural icon, it helps to know why that hitting streak in the summer of 1941 was so extraordinary. Here's one reason: most sporting records are only incrementally better than the ones they supercede. Not so here. DiMaggio's 56-game record towers over the second- and third-place hitting streaks in Major League history: 45 games, by Willie Keeler, in 1897; and 44 games, by Pete Rose, in 1978.

But the deeper reason has to do with probability. As Gould put it: not only did DiMaggio successfully beat 56 Major League pitchers in a row, but "he beat the hardest taskmaster of all . . . Lady Luck."³ As we'll now see, that 56-game hitting streak was so wildly improbable that it really never should have happened in the first place—even for a player as good as Joe DiMaggio.

² Stephen Jay Gould, "The Streak of Streaks." *New York Review of Books*, August 18, 1988.

³ *idid.*

Winning streaks and the compounding rule

Winning streaks in sports, like Joe DiMaggio's, provide a handy metaphor for other, more familiar runs of luck:

- A mutual-fund manager outperforms the stock market for 15 years straight.
- A World-War II airman completes 25 combat missions, and gets to go home.
- An ordinary person successfully takes a shower for 5000 days in a row without slipping.
- A child goes three straight years without catching a cold from other kids at school.

Each of these is, in its own way, a winning streak—although, as any parent of young kids will tell you, some winning streaks are more miraculous than others.

So in the spirit of understanding the mathematics behind any long unbroken run of good (or bad) luck, let's take up the following question. What probability might we reasonably associate with Joe DiMaggio's all-time record hitting streak of 56 games?

This question brings us to another very useful rule in probability theory, called the *compounding rule*. The essence of the compounding rule is that probabilities for independent events can be multiplied together to calculate their joint probability. To state this as an equation, let's suppose that A and B are independent events—that is, they convey no information about each other. Then the joint probability of A and B can be calculated as $P(A \text{ and } B) = P(A) \cdot P(B)$.

The obvious example is when flipping a coin. Since each flip is independent, the probability of getting heads on two successive flips is

$$\begin{aligned} P(\text{Two heads in a row}) &= P(\text{H on flip 1}) \cdot P(\text{H on flip 2}) \\ &= 0.5 \cdot 0.5 = 0.25. \end{aligned}$$

The same line of reasoning works for any number of coin flips. For example,

$$\begin{aligned} P(\text{Three heads in a row}) &= P(\text{H on flip 1}) \cdot P(\text{H on flip 2}) \cdot P(\text{H on flip 3}) \\ &= 0.5 \cdot 0.5 \cdot 0.5 \\ &= 0.5^3 = 0.125; \end{aligned}$$

and so on for four, five, or a hundred flips.

As a general rule, suppose that we have N independent encounters in a row with a random outcome—like a coin flip or a baseball game. On each encounter, there is some probability P that an event of interest will happen—like the coin coming up heads, or Joe DiMaggio getting a hit. The compounding rule tells us the probability that we'll experience a “winning streak” of N events in a row.

$$\begin{aligned} P(N \text{ events in } N \text{ encounters}) &= \underbrace{P \cdot P \cdot \dots \cdot P}_{N \text{ times}} \\ &= P^N. \end{aligned}$$

Of course, if the event itself is a bad one, we'd think of this as a losing streak instead, but the math is the same.

You may recall that this setting—a run of N independent encounters with a random outcome, each of which has some chance P of yielding an event—is exactly where we've used the NP rule to calculate an expected value. In this sense, the compounding rule is a close cousin of the NP rule: they have the same assumptions, but they answer different questions. The NP rule tells us that the expected number of events is $N \times P$ —remember, frequency times

risk—while the compounding rule tells us that the probability of experiencing the event on every single encounter is P^N .

The probability of Joe DiMaggio's hitting streak

The compounding rule now gives us a handy tool to estimate the probability of Joe DiMaggio's hitting streak. Let's make a key simplifying assumption: each baseball game is like the flip of a coin, where "heads" means that DiMaggio gets a hit in that game. We'll use the symbol P_{hit} to denote the probability that DiMaggio gets a hit in a single game; unlike for a real coin flip, P_{hit} is not necessarily 50%.

Under this coin-flipping model, each game is independent: the current game doesn't affect the next one. So the probability that Joe DiMaggio gets a hit for two games in a row is

$$P(\text{hit in two games in a row}) = P_{\text{hit}} \cdot P_{\text{hit}}.$$

And the probability that he gets a hit for N games in a row is

$$P(\text{hit in } N \text{ games in a row}) = (P_{\text{hit}})^N,$$

using the compounding rule.

Calculating P_{hit} . To calculate P_{hit} , the probability that DiMaggio gets a hit in a single game, we'll make three basic assumptions.

1. DiMaggio bats 4 times per game.
2. In 1941 DiMaggio was a .321 hitter, so we'll say that his average chance of getting a hit in any single at-bat was 32.1%.
3. Each at-bat is independent: the current at-bat doesn't affect the next one.

Now, either DiMaggio gets at least one hit in a game, or he doesn't. Therefore, for a single game,

$$P(\text{DiMaggio gets a hit}) = 1 - P(\text{DiMaggio gets 0 hits in 4 tries}).$$

Why write it this way? Because the probability that DiMaggio gets 0 hits in 4 tries is easy to calculate directly: it's a losing streak of 4, and so we can use the compounding rule! Remember, DiMaggio's batting average was 0.321, meaning that he had a 67.9% chance of

failing to hit in a single at-bat. So for a single game of four at-bats in a row,

$$P(\text{DiMaggio gets 0 hits in 4 tries}) = 0.679^4 \approx 0.213.$$

meaning that

$$P(\text{DiMaggio gets a hit}) = 1 - 0.213 = 0.787.$$

Putting it all together. Finally, we're ready to calculate the probability of DiMaggio's 56-game hitting streak. Using the compounding rule, we find that

$$\begin{aligned} P(\text{DiMaggio gets a hit 56 games in a row}) &= (0.787)^{56} \\ &\approx \frac{1}{1.5 \text{ million}}. \end{aligned}$$

So Joe DiMaggio had less than a million-to-one shot at hitting safely every game in a row for any given 56-game stretch in his career. Yet he did anyway. The daunting improbability of this feat helps to explain why his record has never been broken.

Luck, or skill?

Both Western and Eastern philosophical traditions have, for thousands of years, emphasized the role of luck in our lives. As King Solomon said in the book of Ecclesiastes:

I returned, and saw under the sun, that the race is not to the swift, nor the battle to the strong, neither yet bread to the wise, nor yet riches to men of understanding, nor yet favour to men of skill; but time and chance happeneth to them all.

Joe DiMaggio's 56-game hitting streak can serve as a metaphor for us all, as we face those inevitable curve balls and unlucky bounces in our own lives. Remember: Joltin' Joe had to be very lucky to achieve what he did, and even he had a game without a hit every now and again.

But does that mean that DiMaggio's streak was all down to chance? Not even close! His incredible skill at baseball had everything to do with it.

To see why, let's run through the same calculation, except with a different player's statistics—those of Pete Rose, another of the greatest hitters in the history of baseball. In 1978, when he went on his own hitting streak of 44 games, Rose was a .300 hitter, meaning

that his chance of getting a hit in any one at-bat were 30%. How likely is it that someone as good as Rose could challenge DiMaggio's 56-game record? A similar calculation to the one we did for DiMaggio shows that, for a single game,

$$P(\text{Rose gets a hit}) = 1 - 0.7^4 = 0.760.$$

This is only 2.7% lower than DiMaggio's per-game hit probability of 78.7%. Yet when we use the compounding rule, we find that

$$\begin{aligned} P(\text{Rose gets a hit 56 games in a row}) &= (0.760)^{56} \\ &\approx \frac{1}{21 \text{ million}}. \end{aligned}$$

Compare this with DiMaggio's figure of 1 in 1.5 million. The compounding rule has magnified a tiny 2.7% one-game difference between DiMaggio and Rose into an enormous gulf of probability.

And Rose himself was an extraordinary player! What about for an average Major League player, who hits .250? Here, we find that

$$\begin{aligned} P(\text{Average player gets a hit 56 games in a row}) &= (0.684)^{56} \\ &\approx \frac{1}{58 \text{ billion}}. \end{aligned}$$

A 56-game streak by an average player will simply never happen.

The lesson here is that, whether it's in baseball or stock-picking, extraordinary streaks tend to happen to extraordinary performers precisely because those performers are so skillful. They have a higher P , a higher probability of success in a single encounter, than the rest of us do. It is certainly true that DiMaggio needed a lot of luck—some pitching mistakes here, some friendly bounces there—to hit safely for 56 games in a row. But he also needed to be very skillful in the first place, so that the odds he needed to overcome were "only" a million to one.

What Solomon said is true: chance happeneth to them all. But it happeneth a lot more often to those who are better prepared.

Everyday risks and the compounding rule

THE same math behind Joe DiMaggio's hitting streak can help us analyze the kind of repeated, everyday risks that Jared Diamond warned us about. To take a specific example, let's revisit the following question: what is your probability of dying from an

accidental fall at some point over the next 30 years? And how can small differences in your own behavior affect this number?

Let's first observe, from Table 10.1 on page 204, that the yearly death rate due to an accidental fall is about 10 per 100,000 people: $P(\text{deadly fall this year}) = 0.0001$. Now, as a guide to thinking about what is likely to happen to any one person, a population average can be misleading. After all, the average person has one testicle; averages obscure a lot variation. In the case at hand, some people will have a much lower-than-average risk of deadly fall, and others will have a higher risk.

Still, we can work through a thought experiment involving some imaginary Homo Mediocritus, whose individual risk of a daily fall is equal to the population average—just like we sometimes talk about the average Major League hitter as if he were a real person. But we should keep in mind that it's just a thought experiment, and not a prediction about the future. (In fact, soon you'll see an example of how forgetting this point can lead you badly astray.)

With that caveat issued, let's say that our "average person" has a yearly risk of a deadly fall equal to 0.0001. What about the daily risk? We know that surviving the year without a deadly fall means going on a 365-day winning streak, which has probability

$$P(365\text{-day streak without a deadly fall}) = 0.9999.$$

If we assume that each day is independent of the last, then the compounding rule allows us to back out what the daily risk of a deadly fall, must be. How? Well, the compounding rule says that

$$P(365\text{-day streak without a deadly fall}) = 0.9999 = (\text{something})^{365},$$

where "something" is your daily survival probability. From this equation, we can deduce that this number is pretty high: 99.99997%. This means that your chance of a deadly fall on any given day is roughly the same chance that, if you threw 22 quarters into the air right now, all of them would land heads.

What about surviving for 30 years with no deadly fall? To calculate this, we compound the daily survival probability over a much longer period:

$$P(30\text{-year streak without a deadly fall}) = (0.9999997)^{365 \times 30} \approx 0.997.$$

So if you have an average daily risk, then you have a 0.3% chance of dying in a fall at some point over the next 30 years—hardly negligible, but still small.

The role of behavior. Now let's change the numbers just a tiny bit. What if your daily survivorship probability was a bit smaller than that of our hypothetical average person, because of some choice you made regularly—like not putting a towel down on the bathroom floor after a shower, or not holding the handrail as you walk down the stairs? To invoke the DiMaggio/Rose example: what if you became only slightly less skillful at not falling?

For some specific numbers, we'll make an analogy with losing weight. Imagine that your daily habit is to have a single mid-morning Tic-Tac, which has 2 calories. One day, you decide that this indulgence is incompatible with the healthy lifestyle you aspire to. You resolve to cut back. But you know that crash diets rarely work, so you decide to go slowly: you'll forego that Tic-Tac only once every 10 days.

You've just reduced your average daily calorie consumption by about 1/100th of a percent. Will you lose weight over the long run? Alas, no: even the most dubiously optimistic of online calorie calculators would report that, over 30 years, you will shed about half a pound of body fat. For reasons not worth going into, you'd probably lose a lot less.

But what if you made choices that reduced your daily fall-survivorship probability by the same tiny amount of 1/100 of a percent? We're not talking here about the kind of lifestyle change that has you making daily, feckless attempts at Simone Biles-level gymnastics on a wet bathroom floor. This is more like "walking slightly too fast with scissors" territory—something modestly inadvisable that would reduce your daily survival probability from 99.99997% to "merely" 99.99%. Nonetheless, while this change may seem harmless, the 30-year math looks forbidding:

$$P(\text{30-year streak without a deadly fall}) = (0.9999)^{365 \times 30} \approx 0.33.$$

Reducing your daily calorie consumption by one-tenth of a Tic-Tac will not make you any thinner. But reducing your daily fall-survivorship probability by the same amount has about a 67% chance of killing you.⁴

Post script. There are two lessons here. First, population-average probabilities, like $P(\text{deadly fall}) = 0.0001$, can be misleading. When reasoning about risks over the long term, what really matters is your own conditional probability, $P(\text{deadly fall} | \text{behavior})$. That's what gets compounded to calculate the probability of a long

⁴ One caveat here: viewed from another angle, reducing your daily survivorship probability to 99.99% is actually pretty extreme. It means that your daily risk of a deadly fall has become one in 10,000, which is 300 times higher than average. So while the absolute difference in risk is tiny, and the relative difference in survivorship probability is also tiny, the relative difference in fall risk is large—but of course, it's the survivorship probability that gets compounded up.

winning streak. And it's the people with the highest conditional probabilities that contribute disproportionately to the overall figures in Table 10.1. If you don't want to end up as a statistic, keep your conditional probability of a disaster low!

Second, always remember that probability compounds multiplicatively, like interest on your credit cards, and not additively, like calories. Small differences in probability can have a dramatic effect over the long term.

The hot hand: fact or fiction?

THE last few examples have taught us to calculate joint probabilities under the assumption of independence, using the compounding rule. Ideally, of course, we should never just assume that two events A and B are independent. Rather, we should use data to check whether they are!

Remember the definition of independence here: two events are independent if they convey no information about each other. Mathematically, we can express this idea in terms of conditional probabilities: events A and B are independent if $P(A) = P(A | B) = P(A | \text{not } B)$. Therefore, if we want to check whether A and B are really independent, we can carefully observe how often these two events occur together, and verify whether this equation is true.

A good example here is related to the “hot-hand” phenomenon in sports. Basketball fans in particular—and even coaches, players, and broadcasters—tend to believe in the hot hand: that if a player makes one shot, then he or she is more likely to make the next shot.⁵ To express this idea in an equation, believers in the hot hand would assert that

$$P(\text{makes 2nd shot} | \text{makes 1st}) > P(\text{makes 2nd shot} | \text{misses 1st}).$$

In other words, two successive shots are not independent. You can imagine analogous formulations of this idea in walks of life other than sports, from picking stocks to playing poker to creating hit songs or viral videos.

So is the hot hand actually real? This turns out to be a surprisingly tricky question to answer. In a famous study from 1985, three economists looked at data from both the NBA and college basketball and concluded as follows:

Detailed analyses of the shooting records of the Philadelphia 76ers provided no evidence for a positive correlation

⁵ A popular video game from the 1990s, NBA Jam, immortalized this idea for anyone of that era. If you made three shots in a row, a game announcer would bellow “He’s on fire!!” Your basketball avatar would temporarily be granted otherworldly speed, hops, and accuracy—and yes, the ball would actually be on fire whenever you touched it.

| Player | Frequency of made shots after . . . | | | | | | |
|------------------|-------------------------------------|----------|--------|---------|-------|--------|--------|
| | 3 misses | 2 misses | 1 miss | overall | 1 hit | 2 hits | 3 hits |
| Clint Richardson | 0.5 | 0.47 | 0.56 | 0.5 | 0.5 | 0.49 | 0.48 |
| Julius Erving | 0.52 | 0.51 | 0.51 | 0.52 | 0.52 | 0.53 | 0.48 |
| Lionel Hollins | 0.5 | 0.49 | 0.46 | 0.46 | 0.46 | 0.46 | 0.32 |
| Maurice Cheeks | 0.77 | 0.6 | 0.6 | 0.54 | 0.56 | 0.55 | 0.59 |
| Caldwell Jones | 0.5 | 0.48 | 0.47 | 0.43 | 0.47 | 0.45 | 0.27 |
| Andrew Toney | 0.52 | 0.53 | 0.51 | 0.4 | 0.46 | 0.43 | 0.34 |
| Bobby Jones | 0.61 | 0.58 | 0.58 | 0.47 | 0.54 | 0.53 | 0.53 |
| Steve Mix | 0.7 | 0.56 | 0.52 | 0.48 | 0.52 | 0.51 | 0.36 |
| Daryl Dawkins | 0.88 | 0.73 | 0.71 | 0.58 | 0.62 | 0.57 | 0.51 |

between the outcomes of successive shots. The same conclusions emerged from free-throw records of the Boston Celtics, and from a controlled shooting experiment with the men and women of Cornell's varsity teams.⁶

For example, Table 12.1 shows the authors' data for the 9 players on the 1980–81 Philadelphia 76ers. The table shows how frequently players made shots after streaks of different lengths (e.g. after 2 hits in a row, or after 1 miss). For example, Julius Erving made 52% of his shots overall, 52% of his shots after 1 made basket, and 48% of his shots after 3 made baskets—no “hot hand” at all. If you examine the table closely, you'll find that there's not much evidence for the hot-hand hypothesis for any of the players.⁷ If anything, the evidence seems to go the other way: that most players on the 76ers were less likely to make a shot after 2 or 3 made baskets. Ironically, this might reflect the fact that the players themselves believed in the hot-hand phenomenon: if a player who fancies himself “hot” starts to take riskier shots, his shooting percentage will predictably drop.

However, some recent studies have questioned both the methods and the conclusions of the original 1985 study. For example, two researchers at Stanford analyzed data from Major League Baseball and claimed to have found robust evidence for a hot hand across many different statistical categories.⁸ Their basic argument is that, in most sports, it would be really hard to find evidence for the hot-hand phenomenon, even if it really existed. The reason: defenses adapt. For example, as they point out, “a hot shooter in basketball should be defended more intensely. . . which will lower his shooting percentage.” This happens a lot less in baseball,

Table 12.1: Data on the “hot hand” phenomenon for the 1980–81 Philadelphia 76ers. Keep in mind that some of the sample sizes used to calculate these frequencies are quite small, and thus potentially non-representative.

⁶ Gilovich, Thomas; Tversky, A.; Vallone, R. (1985). “The Hot Hand in Basketball: On the Misperception of Random Sequences.” *Cognitive Psychology* 3 (17): 295–314.

⁷ The authors of the 1985 study verified this using formal statistical hypothesis tests.

⁸ The Hot-Hand Fallacy: Cognitive Mistakes or Equilibrium Adjustments? Evidence from Major League Baseball. Brett Green and Jeffrey Zwiebel, Stanford University 2013. As of this writing, the paper had not been peer reviewed.

where the scope for defensive adaptation is limited.

So the jury is still out on the hot-hand phenomenon in sports. However, it seems fair to say that any effects that might be found in the future are likely to be small, given that nobody else has found them yet despite looking pretty intensely.

The fallacy of mistaken compounding

THE compounding rule is very useful for understanding long unbroken runs of luck. However, it's easy to take this rule too far, by applying it to situations where it isn't appropriate.

Recall the key assumption of the compounding rule: N encounters in a row with a random outcome, and each encounter is independent of the previous one. The assumption of independence—that no single event conveys any information about any other event—is crucial here. Without this assumption, we cannot calculate a joint probability by naïvely multiplying together individual probabilities.⁹

It turns out that true independence is rarer than you might think! Yet despite this, it's common for people to assume that two events are independent, and to plunge ahead with some probability calculation, without thinking too hard about whether independence is even approximately correct. This *fallacy of mistaken compounding* occurs so frequently that we'll pause to consider three examples: two of them silly, one very serious indeed.

Lurking variables and mistaken compounding

Ken Cho, a tech entrepreneur in Austin, Texas, has been nicknamed the “Forrest Gump of financial disasters.” You might recall that, in the film, Forrest Gump ends up witnessing some of the most important historical events of the 20th century. Similarly, Ken Cho had a ringside seat for two of the biggest and most startling bankruptcies in history. Before starting his own successful company, called Spredfast, Mr. Cho held jobs in finance at both [Enron](#) and [Lehman Brothers](#), before each went kaput. Of course, Mr. Cho himself had nothing to do with any accounting shenanigans. “It’s just a coincidence,” he says ruefully—a coincidence that makes for a strikingly unlucky CV.

But of course, it also makes for some interesting conversations. In fact, Cho admits that he's come to embrace the nickname.

⁹ In fact, we'd need a different rule: if A and B are not independent, then $P(A \text{ and } B) = P(A) \cdot P(B | A)$. See our “Math Notes” section.

"Whenever I'm chatting with someone next to me on an airplane, and they find out about Lehman and Enron, they always laugh," Cho recounts. "But sometimes they also give me a funny look, like they're a little embarrassed to ask me where I'm working now. I get the impression that they might want to go sell some stock."

Naturally, as statisticians, we found ourselves wondering: just how improbable is a CV like Mr. Cho's?

Reasoning about coincidence

Reasoning correctly about coincidences of this kind often boils down to understanding the concept of independence. Remember, two events A and B are independent of each other if $P(A) = P(A | B) = P(A | \text{not } B)$. That is, knowing whether B occurs doesn't change your assessment of how likely A is to occur.

When we're doing probability calculations, we sometimes *assume* that two events are independent of each other, especially when the causes of the events are thought to be unrelated. If you're flipping coins or rolling dice, this seems obvious. In other cases, we might question whether two events are *really* independent, but assume so anyway. Usually we do this in the belief that the events are *approximately* independent, and that the stakes are small enough to tolerate the approximation. A good example of this came when we assumed independence for two of Joe DiMaggio's at-bats, in our discussion of winning streaks. While might not be exactly true, most of the research on the "hot hand" in sports suggests that it's at least approximately true.

But in other cases, you can get badly tripped up by naively assuming independence. Unexpected correlations, especially in the form of *lurking variables*, come up everywhere.

A lurking variable is some third variable that is correlated with each of the two variables you're interested in. Lurking variables can produce some surprising correlations. In 2012, for example, data scientists at the predictive-analytics firm Kaggle claimed that, based on an analysis of the used-car market, orange used cars were more dependable than used cars in tamer colors. In other words:

$$P(\text{dependable} | \text{orange}) > P(\text{dependable} | \text{not orange}).$$

Why would this be? To most people, paint color and dependability seem like they ought to be independent. To explain why they weren't, Kaggle invoked a possible lurking variable: maybe

owners of orange cars tend to be more devoted to their cars than the average person, and this difference shows up in the reliability statistics.¹⁰ Another possible lurking variable here might involve the rental-car market. Former rental cars have often been driven hard, and are not known for their reliability; two minutes of casual web surfing will reveal that the tag “drive it like a rental” shows up repeatedly on viral videos of dangerous automotive stunts. And since rental cars are almost never orange—few would want to rent them—the used-car market is effectively missing a cohort of unreliable orange cars.

Just how many financial Forrest Gumps are there?

The point is simple: life is full of lurking variables. Once we properly account for them, many things that seem like bizarre coincidences turn out to be much more prosaic. Let’s take the example of Ken Cho’s unlucky CV. How small is $P(E, L)$, the joint probability that a randomly chosen American worked at both Enron and Lehman Brothers?

You might naïvely calculate it as follows. There were about 200 million working-age Americans throughout the period in question (2001–7). At the time of their implosions, Enron and Lehman Brothers had about 20,000 and 26,000 employees, respectively. Therefore, if we assume that these events are independent, we might estimate $P(E, L)$ as

$$\begin{aligned} P(E, L) &= P(E) \cdot P(L) \\ &\approx \frac{20,000}{200,000,000} \cdot \frac{26,000}{200,000,000} \approx 1.3 \times 10^{-8}, \end{aligned}$$

or about 1 in 100 million. This looks pretty unusual! Remember the NP rule: if this back-of-the-envelope reckoning is right, we would only expect that there are two such financial Forrest Gumps in the entire country. (That’s 200 million adults, times a probability of 1 in 100 million.)

But this is an example of the fallacy of mistaken compounding: working at Enron (E) and working at Lehman Brothers (L) are far from independent events. In fact, once we condition on event E , we become aware of an obvious lurking variable: we know that Mr. Cho worked in the finance industry, making it much more likely that he would also have held a job at Lehman.

The correct calculation properly accounts for this fact. For non-independent events, one of our four basic rules of probability

¹⁰ “Big Data Uncovers Some Weird Correlations.” Deborah Gage, *Wall Street Journal* online edition, March 23, 2014.

(Equation 10.1 on page 214) says that

$$P(E, L) = P(E) \cdot P(L | E).$$

In other words, we can't just multiply $P(E)$ and $P(L)$ together to get $P(E, L)$. In lieu of $P(L)$, we should be using the $P(L | E)$: the conditional probability that someone worked for Lehman Brothers (L), given that they also worked in finance for Enron (E).

Let's calculate a very rough estimate for $P(L | E)$. There were about 2 million professionals working in this sector of the finance industry at the time, meaning that the correct denominator in $P(L | E)$ is more like 2 million, not 200 million.¹¹ Therefore, a better estimate for $P(E, L)$ would be

$$\begin{aligned} P(E, L) &= P(E) \cdot P(L | E) \\ &\approx \frac{20,000}{200,000,000} \cdot \frac{26,000}{2,000,000} \approx 1.3 \times 10^{-6}, \end{aligned}$$

or more like one in a million. In the context of a country as large as the U.S., this no longer looks unusual. In fact, since there are 200 million working-age adults, we would actually expect that there are about 200 such Forrest Gumps out there who held jobs at both Lehman and Enron.

Lock up the Christmas sweaters

A very common source of lurking variables can be found in our genes. Consider the example of colorblindness, which runs strongly in families. For example, there is at least one family out there in which there are seven male cousins from one set of grandparents—four Monroe brothers, two Wappler brothers, and one Scott—all of whom are red-green colorblind. Christmas with this family involves some notably poor choices of sweaters, chromatically speaking.

How big of a coincidence is it that all seven male cousins in an extended family will be colorblind? Working this out exactly gets a bit tedious. So instead, we'll use the rule for joint probabilities to calculate a related but simpler probability: the chance that a randomly selected pair of brothers from the U.S. population will be red-green colorblind. Let A indicate that the first brother is colorblind, and B that the second brother is colorblind. We want the joint probability $P(A, B)$.

It's known that about 8% of men are red-green colorblind, meaning that, without any additional information, $P(A) = P(B) =$

¹¹ The real denominator is probably even smaller than 2 million, because these were considered *excellent* jobs in the finance industry, and candidates for them would have been drawn from a smaller pool. But we're just going for a ballpark figure here.

0.08. Therefore, the naïve (and wrong) estimate for $P(A, B)$ would be $0.08^2 = 0.0064$. This would imply that, of all pairs of brothers, roughly half a percent of these pairs are both colorblind.

But again, this is an example of the fallacy of mistaken compounding. To calculate $P(A, B)$ correctly, we need to properly account for non-independence, meaning that we need to know both $P(A)$ and $P(B | A)$. Remember, we are conditioning on the knowledge that the first brother is colorblind. Since colorblindness is genetic, $P(B | A)$ will be larger than 0.08.

Specifically, Mom's genes are the lurking variable here: a colorblind male must have inherited an X chromosome with the colorblindness gene from his mother.¹² To make things simple, let's assume that the brothers' mother has normal color vision, which is true of 99.5% of women. Thus the only way the first brother could be colorblind is if mom has one normal X chromosome, and one X chromosome with the colorblindness gene. The second brother inherits one of these two X chromosomes; either one is equally likely. From this, we can deduce that $P(B | A) = 0.5$.

Putting these facts together, we find that

$$\begin{aligned} P(\text{both brothers colorblind}) &= P(\text{1st brother colorblind}) \\ &\quad \times P(\text{2nd brother colorblind} | \text{1st brother colorblind}) \\ &= 0.08 \times 0.5 \\ &= 0.04. \end{aligned}$$

So about 4% of all pairs of brothers will be colorblind. For a randomly selected family of four boys, the probability that they will all be colorblind drops only by a factor of four, to $0.08 \cdot 0.5^3$, or 1%. Such families are rare, but not exceedingly rare.

Postscript. We've now seen two simple examples of the fallacy of mistaken compounding, where the assumption of independence made our naïve calculations diverge badly from the truth. We will now turn to a third, much more serious example of mistaken compounding, in which a major newspaper made exactly this error, and ended up needlessly frightening millions of people.

¹² This is why colorblindness is so much rarer in women than in men. Men have only one X chromosome, and so they need only one copy of the gene to end up colorblind. But females need two copies of the gene, one on each X chromosome, to end up colorblind. This is much less likely.

How effective is your method?

THE Mesopotamians used acacia leaves and lint. The Egyptians of the Lower Kingdom used a mixture of honey and sodium carbonate. The ancient Persians used elephant dung, cabbages, and plant resin; the Chinese of the 7th century BCE, oil and quicksilver; the Renaissance Europeans, lily root and silkworm gut.

Modern Americans have it a bit easier. Most people choose condoms or the pill, or they get voluntarily and painlessly sterilized.

Birth control is at least as old as civilization; the big difference from ancient times is that our methods actually work well. Since the 1960s, when cheap and effective contraception became widely available, birthrates across the industrialized world have plummeted. Today, some experience with contraception is nearly universal among sexually active adults in rich countries. In 2013, more than 99% of American women aged 15–44 who had ever had sex reported using at least one method of contraception.¹³ The numbers in Europe and Australia look similar.

However, when it comes to choosing a contraceptive method, the modern world offers a dizzying variety of options, each with different trade-offs. Condoms are cheap and ubiquitous—but they suffer from a stigma as a mood killer, and must be used correctly every single time. The pill or the patch are more effective, but also more expensive, and with the potential for hormonal side effects. Vasectomy is extremely effective, but requires surgery to reverse, and gives some men the heebie-jeebies.¹⁴ And so on and so forth, for each of the dozens of other methods studied by experts.¹⁵

Quantifying contraceptive effectiveness

We recognize that, for many people, the choice of when to use contraception, and what method to use, may be personally or culturally fraught, and cannot be reduced to a single variable. However, one question that occurs to everyone in thinking about contraception surely must be: how likely is a pregnancy if I use this thing?

It was with this question in mind that, in 2014, the *New York Times* published an article, and an accompanying data visualization, entitled “How Likely Is It That Birth Control Could Let You Down?”¹⁶ The authors of the article began from a simple premise: the more times you use any method of birth control, the more

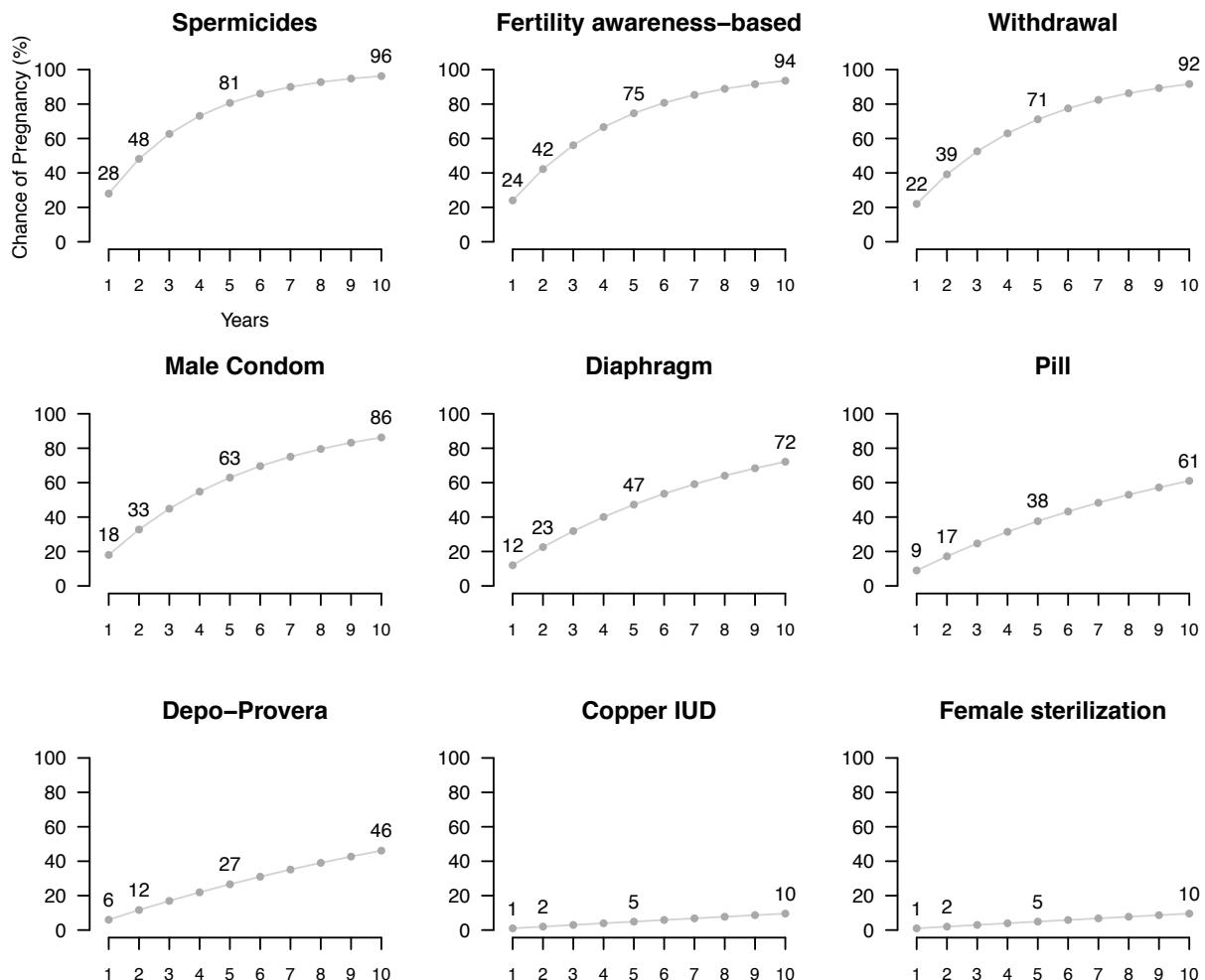
¹³ Daniels K, Mosher and Jones, “Contraceptive methods women have ever used: United States, 1982–2010,” National Health Statistics Reports, 2013, No. 62, <http://www.cdc.gov/nchs/data/nhsr/nhsr062.pdf>.

¹⁴ “Vasectomy: the other (better) form of sterilization.” Grace Shiha, David Turok, Willie Parker. *Contraception* 83: 4, April 2011, pp. 310–15

¹⁵ See, e.g. <https://www.guttmacher.org/fact-sheet/contraceptive-use-united-states>.

¹⁶ “How Likely Is It That Birth Control Could Let You Down?” Gregor Aisch and Bill Marsh. Sunday Review section, September 13, 2014.

The New York Times' incorrect calculation of 10-year failure rates for 9 contraceptive methods



opportunities there are for it to fail.

To try to put some numbers behind this idea, the authors of the article looked at published data on the efficacy of fifteen popular contraceptive methods. They used this data—together with their own calculations, which we'll describe below—to create a [slick interactive chart](#) that purported to show each method's failure rate over the long term, going to ten years.

We have used the same published data¹⁷ to replicate the *Times'*

Figure 12.1: Spurious ten-year failure rates under "typical use" of 9 contraceptive methods, calculated according to the incorrect methodology from the New York Times' article of 9/13/2014.

¹⁷ James Trussell, "Contraceptive failure in the United States." *Contraception* 2011 May ; 83(5): 397-404

chart, using the same methodology employed by the authors of the article, for a subset of nine of these methods. Our numbers, which you can see in Figure 12.1 on page 255, agree with theirs.

If the numbers in Figure 12.1 surprise you, you are not alone: the *Times'* article shocked a lot of people. For example, the article stated that the one-year failure rate for typical pill users was 9%, but that the five-year failure rate was 38%, and the ten-year failure rate was 61%.¹⁸ This implies that a woman who uses the pill as her only contraceptive method over the course of a decade has a better-than-even chance of getting pregnant at least once. The numbers for the condom came off looking even worse: its one-year failure rate was shown at 18%, and its ten-year failure rate at 86%. See Figure 12.1 for similar numbers on all nine methods.

To many people, these numbers seemed astonishingly high, and implied a far greater long-term risk of unplanned pregnancy than they were prepared for. Unsurprisingly, the article quickly went viral on social media. Judging by the reaction there, while the article may not have sparked a mass rush to join a convent, it did cause a lot of anxiety among ordinary *Times* readers, many of whom had presumably believed their own contraceptive methods to be more reliable. Even gynecologists, who should know the research just about as well as anyone, went straight online to share the link and to express their alarm.¹⁹

Mistaken compounding strikes again

But there was a major problem with the *New York Times'* article: its putative long-term failure rates are almost surely wrong. In fact, very wrong.

It turns out that nobody—not even the experts who study this stuff—knows what the ten-year failure rates are for any of the contraceptive methods described in the article.²⁰ The question just hasn't been studied. However, despite this lack of evidence, there are strong reasons to believe that the *Times'* article drastically overstated the chance of pregnancy under long-term typical use of most contraceptive methods.²¹

How did such a highly regarded newspaper make such a terrible error, and end up needlessly scaring so many people? The answer to this question reveals, once again, a spurious assumption of independence and a mistaken application of the compounding rule for calculating joint probabilities.

Here's how the *Times* calculated each method's purported long-

¹⁸ Remember, these failure rates are for “typical” users, according to the Food and Drug Administration’s definition. This standard can actually encompass a surprisingly high degree of variation. The one-year failure rate of the pill under perfect use is much, much lower: less than 1 chance in 100.

¹⁹ For example, @hricciot: “Shocking - even to a gynecologist like me! #LARC-CisBest.” LARC stands for long-acting reversible contraception—for example, IUDs.

²⁰ The sole exception is female sterilization, for which long-term data does exist.

²¹ By “long term” we mean more than one year.

term failure rate. First, they took the one-year “typical-use” failure rate from the published literature—for example, 9% for the pill. These one-year efficacy numbers had been calculated using data from randomized clinical trials regulated by the Food and Drug Administration (FDA), or from nationally representative surveys, and therefore reflected the best estimates available.

Next, the authors assumed that a contraceptive failure in one year was independent of a contraceptive failure in the previous year. This assumption made it easy to use the compounding rule to calculate the probability of a “no-pregnancy streak”—that is, for a method to remain successful at preventing pregnancy for several years in a row. Effectively, the *Times* journalists were treating a multi-year stretch of contraceptive use without a pregnancy exactly as if it were a Joe DiMaggio hitting streak.

Let’s take an example. For typical pill users, the probability of no pregnancy in year 1 was 0.91. The article used this fact, together with the compounding rule, to calculate the following probabilities:

$$P(\text{no pregnancy through 1 year}) = 0.91$$

$$P(\text{no pregnancy through 2 years}) = 0.91^2 = 0.821$$

⋮

$$P(\text{no pregnancy through 5 years}) = 0.91^5 = 0.624,$$

and so forth. By the time you get out to 10 years, the probability of a long winning streak starts to look pretty small: about 39%. Since you either get pregnant or you don’t, this implies a 61% chance of at least one pregnancy over a ten-year period of typical pill use.

But this calculation has one major, deal-breaking flaw: it’s the fallacy of mistaken compounding once again. It turns out that, under the established definition of “typical use,” avoiding pregnancy in two successive years cannot be treated as two independent events with a common probability. Therefore, we can’t just keep raising 0.91 to higher and higher powers to calculate the probability of a winning streak over longer and longer periods. Instead, we would need to use joint probabilities calculated using the rule for conditional probability, like this:

$$P(\text{NP 1}, \text{NP 2}) = P(\text{NP 1}) \cdot P(\text{NP 2} | \text{NP 1}),$$

and so on for longer periods. (Here “NP 1” means no pregnancy in year 1.)

Unfortunately, this conditional probability $P(\text{NP 2} \mid \text{NP 1})$ is not known precisely for the pill, or indeed for any method. However, for reasons explained below, the number is almost certainly higher than the published one-year success rate of 0.91. This means that the two-year success rate will not be $0.91 \times 0.91 \approx 0.82$, but rather something more favorable. Long winning streaks are actually *more* likely than the assumption of independence would suggest, implying that the *Times'* long-term failure rates—just like the ones you see in Figure 12.1—are too high.

Lurking variables in measuring contraceptive effectiveness

Here's a really important question that we haven't yet answered. Why are contraceptive failures in successive years non-independent?

On this question, you shouldn't just take our word for it. Instead, you should listen to James Trussell, a professor at Princeton University, and the world's leading expert on measuring the effectiveness of contraceptive methods. In fact, it was Dr. Trussell's research that the *Times* article had been relying upon for one-year contraceptive failure rates in the first place.²² We contacted him for his assessment of the article, and he graciously shared his thoughts with us. To summarize: he was highly critical of the *Times* for publishing numbers that lacked any firm scientific basis, and that stood a good chance of misleading people.

Now for the longer version. In our e-mail exchange, Trussell pointed out several lurking variables that invalidated the article's assumption of independence—and therefore, all of its typical-use calculations that used the compounding rule, including those scarily high five- and ten-year failure rates.

Here's one possible lurking variable that would make failure rates decline over time: experience. With some contraceptive methods, people get better with practice. If you have regularly used a method for several years—whether it's a pill you have to remember to take every day, or a condom you have to put on correctly every time—you may have learned to use it more effectively than you did in your younger, greener days. Therefore, your own conditional probability of a pregnancy in year five or ten, given that you're already a regular user of a method, could be lower than the year-one failure rate estimated from a trial or national survey, which encompasses both experienced and novice users.

²² Ibid. (See footnote 17 on page 255.)

What does “typical use” actually mean?

However, in Trussell’s assessment, there is a different, overwhelmingly important lurking variable that explains why measurable contraceptive failure rates decline over time: “The most important factor is adherence. For methods requiring adherence, inconsistent users are the first to fail, leaving behind a pool increasingly composed of consistent users.”²³

Let’s back up here. The effect highlighted by Dr. Trussell is subtle but important, and merits some further explanation.

Clinical trials for contraceptive methods actually estimate two failure rates: one rate assuming “perfect use,” and a second rate assuming “typical use.” Perfect use is basically just what it says. It’s calculated based on data from only those women in a trial who reported correctly using the method every time they had sex. Therefore, perfect use can easily be defined in terms of a specific behavior: using a method correctly, each and every time.

But typical use has nothing to do with a specific behavior. Rather, it is a vague standard that encompasses a range of behaviors, some of them more likely to lead to unplanned pregnancy than others. In fact, according to the definition of this term long agreed-upon in the scientific literature, the only requirement for someone to be using a method in a “typical” fashion is that she believes herself to be using that method—however infrequently or incorrectly she is actually using it.²⁴ As a result, the *subtype of typical user that you are*, whether consistent or erratic, is a very important lurking variable for reasoning correctly about long-term contraceptive failure rates.

You might imagine that there exists some particular user, whether actual or archetypal, whom we can point to and say, “See! She’s using this method typically.” If that were true, then you could imagine measuring the failure rate of people like this typical user, and compounding this probability over many years to estimate a long-term failure rate, just like the *Times* did.²⁵

The problem is: no such user exists. For one thing, this would be a wildly impractical standard. Just imagine the bedroom snooping that would be required to identify what this “typical” behavior actually looks like. More to the point, even if you *could* find this typical user, no scientist actually has. Contraceptive research isn’t like some voyeuristic game of baseball, where scientists search high and low for the average Major League player, who hits .250. In fact, “typical use” isn’t a claim about a person at all, but about a

²³ James Trussell, personal communication, 31 July 2016.

²⁴ According to Trussell, this standard has been widely adopted in the literature since it was proposed by him and Kost in the 1980s in the following paper: Trussell J, Kost K. “Contraceptive failure in the United States: a critical review of the literature.” *Stud. Fam. Plann.* 1987 Sep-Oct;18(5):237-83.

²⁵ Actually this would still be wrong, merely less wrong. Dr. Trussell also cited two other other lurking variables that would make failure rates decline over time: “underlying fecundity (biological capacity to conceive) and coital frequency of the couple. More fecund couples would be prone to fail earlier, even during perfect use.” This also explains why the *Times*’s numbers for perfect use are less objectionable—because perfect use *can* be linked to a specific set of behaviors—but still incorrect.

group of people—a group with lots of variation in it.

A thought experiment

No, the only right way to measure a long-term contraceptive failure rate under “typical use” is to follow a group of people who believe themselves to be using the method, and to check how many of them get pregnant over time.

Needless to say, the *Times* didn’t actually do this. But let’s imagine that they had. To illustrate what happens, let’s first divide the typical-use population into two subtypes: consistent users (subtype C) and erratic users (subtype E). For example, typical use of a condom can mean using one every time you have sex (subtype C), or only some of the time (E). Typical use of the pill can mean taking a pill every day (C), or only on some days (E). And so on. Real life is obviously more complicated than this cartoon, but the cartoon will do for our purposes here.

Now imagine following a cohort of 1000 typical pill users over several years, as we might do in a clinical trial. In the first year, if previous trials are a good indication, we would expect about 90 of these women to become pregnant. Now pause to ask yourself a simple question: who were they?

The question almost answers itself. You can bet that a disproportionate number of the 90 women who got pregnant in year one were erratic users of the pill—that is, subtype E of typical users. Therefore, what remains at the start of year two is a cohort of 910 women biased a bit more towards the subtype-C users, compared to the original cohort.

Now let’s track this remaining cohort of 910 women through the second year of our study. Here’s a follow-up question: would you expect that 9% of these 910 women would get pregnant?

Probably not! In fact you should expect the number to be less than 9%. The most erratic users from the original cohort have been weeded out. The ones left in the study are more likely to be consistent users, and therefore less likely to get pregnant. Therefore, the data would lead us to estimate that

$$P(\text{no pregnancy in year 2} \mid \text{no pregnancy in year 1}) > 0.91,$$

which implies that the two-year failure rate for typical pill users will be lower than 17%, as reported by the *Times*.

What’s more, the influence of the subtype-C-or-E lurking variable gets stronger over time. As we track our cohort over longer

periods, the type-E folks tend to get pregnant, and the people who remain collectively come closer to resembling our pure class-C subtype—which we can infer from the fact that they haven’t gotten pregnant! Therefore, the data would lead us to conclude that

$$P(\text{NP year 3} \mid \text{NP year 2}) > P(\text{NP year 2} \mid \text{NP year 1}),$$

$$P(\text{NP year 4} \mid \text{NP year 3}) > P(\text{NP year 3} \mid \text{NP year 2}),$$

and so on. As a result, the longer your horizon for calculating failure rates, the further your numbers will be from the truth if you mistakenly assume independence. In fact, the error is likely to compound in an accelerating way over time. So while the *Times'* two-year typical-use numbers are probably wrong, its ten-year typical-use numbers are probably *very* wrong.

Finally, we want to close the loop on one our own earlier examples: the one about an average person’s chance of surviving 30 years without a deadly fall, which we calculated by compounding a daily survivorship probability. That example suffers from the same error we’ve described here. If you were to actually follow a group of 100,000 people for 30 years, the people with a high risk of falling would tend to fall and die early, leaving behind a more careful cohort of people who are, over time, progressively less likely to die from a fall. As a result, the measured 30-year survival rate would be higher than what the compounded average would suggest. That’s why we were very careful to emphasize that our earlier example was not a prediction about the future—only a thought experiment to highlight the role of your own behavior in determining your risk of a disaster.

We wish the *Times* had emphasized the same point.

Postscript. There are two important lessons to be gleaned from this story. First, there’s a big difference between what “average” means for a Joe DiMaggio at-bat, and what “average” means in a study about the effectiveness of a contraceptive method. Joe DiMaggio’s average of 32.1% is a property of a single person. But the one-year success average of 91% for the contraceptive pill is a property of a group of people, each of whom is different. Group averages can obscure a lot of interesting variability.

Second, life is full of lurking variables. These have a way of making a fool of anyone who assumes independence without bothering to check, or to think the matter through. Therefore, always think before you compound.

13

Bayes' rule

OUR conditional probabilities always depend on what we know. When our knowledge changes, these probabilities must also change. A 250-year-old mathematical principle called Bayes' Rule tells us how.

Bayesian search: finding the USS *Scorpion*

IN February of 1968, the USS *Scorpion* set sail from the naval base in Norfolk, Virginia, under the command of Francis Slattery. The *Scorpion* was a Skipjack-class high-speed attack submarine, the fastest in the American fleet. Like other subs of her class, she played a major role in U.S. military strategy. Think *The Hunt for Red October* here: throughout the Cold War, both the Americans and the Soviets deployed large fleets of attack subs, whose mission was to locate, track, and—should the unthinkable happen—destroy the other side's ballistic-missile submarines.

On this deployment, the *Scorpion* sailed east, bound for the Mediterranean Sea, where for three months she participated in training exercises alongside the 6th Naval Fleet. Then in mid-May, the *Scorpion* was sent back west, past Gibraltar and out into the Atlantic. There she was ordered to observe Soviet naval vessels operating near the Azores—a remote island chain in the middle of the North Atlantic, about 850 miles off the coast of Portugal—and then to continue west, bound for home. The sub was due back in Norfolk at 1 PM on Monday, May 27th, 1968.

On the docks in Norfolk that day, the families of the *Scorpion*'s 99 crew members were gathered to welcome their loved ones back home. But as 1 PM came and went, the sub had not yet surfaced. Minutes stretched into hours; day gave way to night. Still the families waited. But there was no sign of the *Scorpion*.

With growing alarm, the Navy ordered a search. By 10 PM, the

operation involved 18 ships; by the next morning, 37 ships and 17 long-range patrol aircraft. But the odds of a good outcome were slim. The *Scorpion* had last made contact off the Azores, 6 days ago, and 2,670 miles away from Norfolk. She could have been anywhere along that strip of ocean between the Azores and the eastern seaboard. As the hours ticked by, the chances that the sub could be located, and that rescue gear could be deployed in time, were rapidly diminishing. At a tense news conference on May 28th, President Lyndon Johnson summarized the mood of a nation: "Nothing encouraging to report. . . . We are all quite distressed."

Day after day went by, but the search for the *Scorpion* turned up no results. Finally, after eight days, the Navy was forced to concede the obvious: the *Scorpion*'s crew of 99 men were declared lost at sea, presumed dead.

The Navy now turned to the grim task of locating the *Scorpion*'s final resting place—a tiny needle in a vast haystack stretching three-fourths of the way across the North Atlantic. Although hopes for saving the crew had been dashed, the stakes were still high, and not only for the families of those lost: the *Scorpion* had carried two nuclear-tipped torpedoes, each capable of sinking an aircraft carrier with a single hit. These dangerous warheads were now somewhere on the bottom of the sea.

John Craven, Bayesian search guru

To lead the search for the *Scorpion*, the Pentagon turned to Dr. John Craven, chief scientist in the Navy's Special Projects Office, and the leading guru on finding lost objects in deep water.

Remarkably, Dr. Craven had done this kind of thing before. Two years earlier, in 1966, an American B-52 bomber had collided in mid-air with a refueling tanker over the Spanish coast, near the seaside village of Palomares. Both planes crashed, and the B-52's four hydrogen bombs, each of them 50 times more powerful than the Hiroshima explosion, were scattered for miles. Luckily none of the warheads had detonated, and three of the bombs were found more or less immediately.¹ But the fourth bomb was missing, and was presumed to have fallen into the sea. John Craven was called upon to help find it.

Craven and his team had to ponder many unknown variables about the crash. Had the bomb remained in the plane, or had it fallen out? If the bomb had fallen out, had either or both of its parachutes deployed? If the parachutes had deployed, had the

¹ Albeit after one of them had contaminated a roughly one-square-mile area of tomato farms and woodland with radioactive plutonium. The clean-up operation in the wake of this incident continues 50 years later, with the latest negotiations between Spain and the U.S. taking place in 2015.

winds taken the bomb far out to sea? If so, in what direction, and exactly how far?

Bayesian search. To sort through this thicket of unknowns, Craven turned to his preferred strategy: *Bayesian search*. This search methodology had been pioneered during World War II, when the Allies used it to help locate German U-boats. But its origins stretched back much further, all the way to a mathematical principle called *Bayes' rule*, first worked out by an English reverend named Thomas Bayes, in the 1750s.

Bayesian search has three essential principles. First, you should combine the pre-search opinions of various experts about the plausibility of each possible scenario. In the case of the missing H-bomb, some of these experts would be familiar with mid-air crashes, some of them familiar with nuclear bombs, some with coastal winds and ocean currents, and so forth. These opinions should be synthesized to form a *prior probability* for each crash scenario—and, by extension, a prior probability that the bomb might be found in each possible search location. These probabilities are “prior,” in the sense that they represent the best guess available, before anyone has any data.

Second, you must evaluate the capability of your search instruments to establish the likelihood that, if the object were in a given sector, you'd actually be able to find it there. This likelihood is combined with the prior probability to form a single search-effectiveness probability for each location. For example, let's say that the most plausible scenario puts the lost bomb at the bottom of a very deep ocean trench. Despite its high prior probability, this trench might still be a poor candidate location to begin your search, for the simple reason that the trench is so dark and remote that, even if the bomb were there, you'd be very unlikely to find it. To draw on a familiar metaphor, a Bayesian search has you start looking for your lost keys using a precise mathematical combination of two factors: where you think you lost them, and where the streetlight is shining brightest.

Third and finally, as new data comes in during the search process, you should use that new data to update your prior probability for each search location into a *posterior probability*. This Bayesian updating process is iterative, in the sense that today's posterior becomes tomorrow's prior. Suppose you search in today's region of high posterior probability, but find nothing. Then for tomorrow,

you reduce the probability in the region you just searched, reassess your beliefs about each scenario, and bump up the probability in the other regions accordingly. You keep doing this day after day, always concentrating on that day's new region of highest probability, until you find what you're looking for.

Craven is stymied. Unfortunately, military politics, and a clash of personalities, prevented Dr. Craven and his team from actually applying these Bayesian principles to the 1966 search for the missing H-bomb off the coast of Palomares. In a classic military move, the Pentagon had asked the right hand to do one thing, and then asked the left hand to put some handcuffs on the right one, to make its job more difficult. The commanding officer on the scene, Rear Admiral William S. Guest—nickname: Bull Dog—had a notably different view of the way the search should be conducted. Bull Dog was an excellent fast thinker. He had little patience for probabilities, and even less patience for the team of twentysomething-year-old math Ph.D.'s he now found himself commanding. His initial orders to Craven's team, perhaps only half sarcastic, were for them to prove that the bomb had fallen on land rather than in the sea, so that it would be someone else's job to find the damned thing.²

As a result, the search for the Palomares H-bomb was really two searches. There was Craven's Bayesian search, with its slide rules and probability maps, and with updated probabilities constantly chattering over the teletype machine as the mathematicians fed remote calculations to a mainframe computer back in Pennsylvania. But the insights arising from the Bayesian search were largely ignored in favor of Admiral Guest's "plan of squares," which guided the *real* search, and which was pretty much exactly what it sounds like. The frustrated Craven was like a high-school stock picker who records virtual trades in a ledger and watches his paper fortune grow, but never gets to buy and sell any shares.

Eventually, the bomb was found. It turns out that a fisherman named Francisco Orts had seen the bomb fall into the water under parachute, and he was able to guide the Navy to its exact point of entry. Thus while the search was a success, the Bayesian part of it had been a failure, for the simple reason that it had never been given a chance. Nonetheless, the Palomares incident taught John Craven some valuable lessons—both about the practicalities of conducting a Bayesian search, and about how to get the

² Sharon Bertsch McGrayne, *The Theory That Would Not Die*, Yale University Press, 2011 (pg. 190).

necessary support for that search from the military brass.

And two years later, when he was called upon to find the USS *Scorpion*, Craven was ready.

The search for the Scorpion continues

When the *Scorpion* disappeared in May of 1968, Craven and his Bayesian search team—including Frank Andrews, Daniel Wagner, Tony Richardson, and many others—were quickly reconvened. At first, the task seemed vastly more daunting than the search for the Palomares H-bomb had been. Back then, they had known to confine the search to a relatively small area off the coast of southern Spain. But here, the team had to find a submarine under 2 miles of water, somewhere between Virginia and the Azores, without so much as a single clue.

Luckily, they caught a break. Starting in the early 1960s, the U.S. military had spent \$17 billion installing an enormous, highly classified network of underwater microphones throughout the North Atlantic. Essentially, they had wired the entire ocean for sound, so that they could track the movements of the Soviet navy. Highly trained technicians at secret listening posts were monitoring these microphones around the clock. The technicians could look at the output from these devices and immediately distinguish the acoustic signature of a submarine from that of a whale, an oil tanker, or hot magma under the seabed.

After sniffing around, Craven discovered that one of these secret listening posts in the Canary Islands had, one day in late May, recorded a very unusual series of 18 underwater sounds. Then he learned that two other listening posts—both of them thousands of miles away, off the coast of Newfoundland—had recorded those very same sounds around the same time. Craven's team compared these three readings and, by triangulation, worked out that the sounds must have emanated from a very deep part of the Atlantic Ocean, about 400 miles southwest of the Azores.

This location fell along the *Scorpion*'s expected route home. Moreover, the sounds themselves were highly suggestive: a muffled underwater explosion; then 91 seconds of silence; and then 17 further sonic events in rapid succession that, to Craven, sounded like the implosion of various compartments of a submarine as it sank beneath its hull-crush depth.³

This acoustic revelation dramatically narrowed the size of the search area. Still, the team had about 140 square miles of ocean

³ PBS Nova documentary, "Submarines, Secrets, and Spies." Originally broadcast January 19, 1999. <https://www.youtube.com/watch?v=NJWHiPSvzh8>

floor to cover, all of it 10,000 feet below the surface, and therefore inaccessible to all but the most advanced submersibles.

The Bayesian search now kicked into high gear. Craven's first step was to take a map of the seabed and divide it up into a grid of little rectangles, each one a possible search location. Each rectangle got an alphanumeric code—B6, H3, and so on, just like in the board game *Battleship*. Craven and his team then interviewed expert submariners, and came up with nine possible scenarios—a fire on board, a torpedo exploding in its bay, a clandestine Russian attack, and so on—for how the submarine had sunk. They weighed the prior probability of each scenario, and ran Monte Carlo simulations to understand how the *Scorpion*'s likely movements might have unfolded under each one. They assessed the capabilities of the search fleet: its cameras, its magnetic-sensing instruments, its sonars, its submersible robots. They even blew up depth charges at precise locations, in order to calibrate their original acoustical data from the listening posts in the Canary Islands and Newfoundland.

Finally, they put all this information together to form a single search-effectiveness probability for each cell on the grid. This map crystallized thousands upon thousands of hours of interviews, calculations, experiments, and careful thinking. It would have looked something like this:

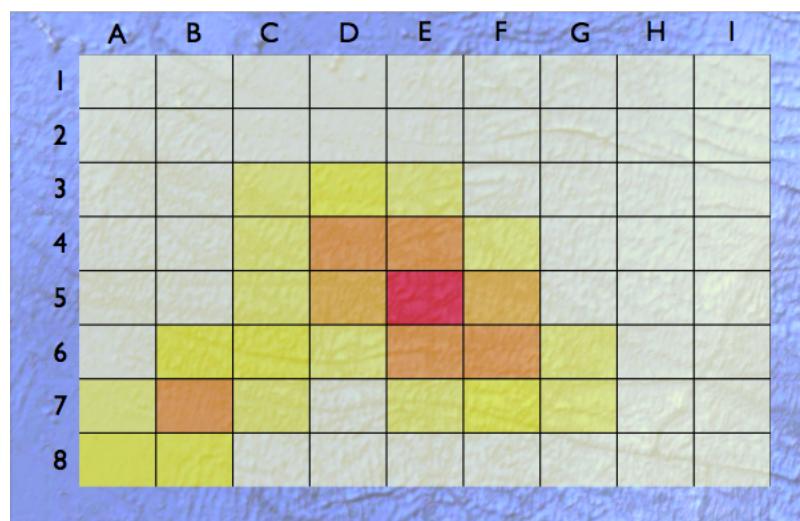


Figure 13.1: An attempted reconstruction of John Craven's probability map for the *Scorpion* search. Darker red rectangles indicate regions of relatively higher prior probability.

Mathematically speaking, this map represented the best chance for

finding the *Scorpion*.

Predictably, Craven encountered both logistical and bureaucratic difficulties in getting the Pentagon to pay attention to his map of probabilities. Summer came and went. By this point, the search for the *Scorpion* had been going on since early June, to no avail.

But eventually his cajoling paid off, and the military brass ordered that Craven's map be used to guide the now months-old search. So starting in October, when commanders leading the search aboard the USS *Mizar* finally got ahold of the map, the operation became truly Bayesian. Day by day, the team rigorously searched the region of highest probability, and crunched the numbers to update the map for tomorrow. And day by day, those numbers were slowly homing in on rectangle F6.

Found

On October 28th, Bayes finally paid off.

The *Mizar* was in the midst of its 5th cruise, and its 74th individual run over the ocean floor. All of a sudden the ship's magnetometer spiked, suggesting an anomaly on the sea floor. Cameras were hurriedly deployed to investigate—and sure enough, there she was:



Figure 13.2: A photo of the bow section of the USS *Scorpion*, taken in 1968 by the crew of the bathyscape *Trieste II*. USN photo #1136658.

Partially buried in the sand, 400 miles from the nearest landfall and two miles below the surface of a restive sea, the USS *Scorpion*

had been found at last.

To this day, nobody knows for sure what actually happened to the *Scorpion*—or if they do, they’re not talking. The Navy’s official version of events, though inconclusive, cites the accidental explosion of a torpedo or the malfunctioning of a garbage-disposal unit as two of the most likely possible causes of the tragedy. Many other explanations have been proposed over the years. And as with any famous mystery, conspiracy theories abound.

But there was at least one definitive conclusion to come out of the *Scorpion* incident: Bayesian search was a truly winning idea. As it turned out, the sub’s final resting place lay a mere 260 yards away from rectangle E5, the initial region of highest promise on Craven’s map of prior probabilities. The search team had actually passed over that location on a previous cruise, but had missed the telltale signs due to a broken sonar.⁴

Ponder that for a moment more. A lone submarine had been lost somewhere in a 2600-mile stretch of open ocean, and the Bayesian search had pinpointed her location to within 260 yards—only three lengths of the submarine itself. It was a remarkable triumph for Craven’s team, and for Bayes’ rule, the 250-year-old mathematical formula that had served as the search’s guiding principle.

Today, Bayesian search is a small industry, with at least one college textbook⁵ explaining the details, and with entire companies whose mission is to apply Bayesian principles to find what has been lost. To cite a recent example, many readers will remember the tragedy of Air France Flight 447, which crashed in the Atlantic Ocean on its way from Rio de Janeiro to Paris, in June of 2009. The search for the wreckage had been going on for two fruitless years; then in 2011, a Bayesian search firm was hired, a map of probabilities was drawn up—and the plane was found within one week of undersea search.⁶

Moreover, the broader principle behind Bayesian search, Bayes’ rule, is used almost everywhere: from courtrooms to doctor’s offices, and from spam filters to self-driving cars. So if you want to learn more about the key equation that found the *Scorpion* and that helps power the modern world, then this chapter is for you.

⁴ McGrayne, *ibid.*

⁵ The Theory of Optimal Search (Operations Research Society of America, 1975), by Lawrence D. Stone.

⁶ Stone et. al. “Search for the wreckage of Air France Flight AF 447.” *Statistical Science* 2014, Vol. 29(1), pp. 69-80.

Updating conditional probabilities

Our probabilities are always contingent upon what we know.

The probability that a patient with chest pains has suffered a heart attack:

Does the patient feel the pain radiating down his left side?

What does his ECG look like? Does his blood test reveal elevated levels of myoglobin?

The probability of rain this afternoon in Milwaukee: What are the current temperature and barometric pressure? What does the radar show? Was it raining this morning in Chicago?

The probability that a person on trial is actually guilty: Did the accused have a motive? Means? Opportunity? Were any bloody gloves left at the scene that reveal a likely DNA match?

When our knowledge changes, our probabilities must change, too. Bayes' rule, the mathematical formula at the heart of the *Scorpion* story, tells us how to change them.

Imagine the person in charge of a Toyota factory who starts with a subjective probability assessment for some proposition A , like “our engine assembly robots are functioning properly.” Just to put a number on it, let’s say $P(A) = 0.95$; we might have arrived at this judgment, for example, based on the fact that the robots have been down for 5% of the time over the previous month. In the absence of any other information, this is as good a guess as any.

Now we learn something new, like information B : the last 5 engines off the assembly line all failed inspection. Before we believed there was a 95% chance that the assembly line was working fine. What about now?

Bayes’s rule is an explicit equation that tells us how to incorporate this new information, turning our initial probability $P(A)$ into a new, updated probability:

$$P(A | B) = \frac{P(A) \cdot P(B | A)}{P(B)}. \quad (13.1)$$

Each piece of this equation has a name:

- $P(A)$ is the prior probability: how probable is A , before ever having seen data B ?
- $P(A | B)$ is the posterior probability: how probable is A , now that we’ve seen data B ?



Figure 13.3: Bayes’ rule is named after Thomas Bayes (above), an English reverend of the 18th century who first derived the result. It was published posthumously in 1763 in “An Essay towards solving a Problem in the Doctrine of Chances.”

- $P(B | A)$ is the likelihood: if A were true, how likely is it that we'd see data B ?
- $P(B)$ is the marginal probability of B : how likely is it that we'd see data B anyway, regardless of whether A is true or not? This calculation is usually the tedious part of applying Bayes' rule. Usually, as we'll see in the examples, we use the rule of total probability, which we learned in the previous chapter.

Have you found the two-headed coin?

To get a feel for what's going on here, let's see an example of Bayes' rule in action.

Imagine a jar with 1024 normal quarters. Into this jar, a friend places a single two-headed quarter (i.e. with heads on both sides). Your friend then gives the jar a good shake to mix up the coins. You draw a single coin at random from the jar, and without examining it closely, flip the coin ten times. The coin comes up heads all ten times. Are you holding the two-headed quarter, or an ordinary quarter?

Now, you might be thinking that this example sounds pretty artificial. But it's not at all. In fact, in the real world, an awful lot of time and energy is spent looking for metaphorical two-headed coins—specifically, in any industry where companies compete strenuously for talented employees. To see why, let's change the story just a little bit.

Suppose you're in charge of a large trading desk at a major Wall Street bank. You have 1025 employees under you, and each one is responsible for managing a portfolio of stocks to make money for your firm and its clients.

One day, a young trader knocks on your door and confidently asks for a big raise. You ask her to make a case for why she deserves one. She replies:

Look at my trading record. I've been with the company for ten months, and in each of those ten months, my portfolio returns have been in the top half of all the portfolios managed by my peers on the trading floor. If I were just an average trader, this would be very unlikely. In fact, the probability that an average trader would see above-average results for ten months in a row is only $(1/2)^{10}$, which is less than one chance in a thousand. Since it's unlikely I would be that lucky,

the implication is that I am a talented trader, and I should therefore get a raise.

The math of this scenario is exactly the same as the one involving the big jar of quarters. Metaphorically, the trader is claiming to be a two-headed coin (T), on the basis of some data D : that she performs above average, every single month without fail.

But from your perspective, things are not so clear. Is the trader lucky, or good? There are 1025 people in your office (i.e. 1025 coins). Now you're confronted with the data that one of them has had an above-average monthly return for ten months in a row (i.e. D = "flipped heads ten times in a row"). This is admittedly unlikely, and this person might therefore be an excellent performer, worth paying a great deal to retain. But excellent performers are probably also rare, so that the prior probability $P(T)$ is pretty small to begin with. To make an informed decision, you need to know $P(T | D)$: the posterior probability that the trader is an above-average performer, given the data.

Applying Bayes' rule. So our two-headed coin example definitely has real-world applications. Let's use it to see how a posterior probability is calculated using Bayes' rule:

$$P(T | D) = \frac{P(T) \cdot P(D | T)}{P(D)}.$$

We'll take this equation one piece at a time. First, what is $P(T)$, the prior probability that you are holding the two-headed quarter? Well, there are 1025 quarters in the jar: 1024 ordinary ones, and one two-headed quarter. Assuming that your friend mixed the coins in the jar well enough, then you are just as likely to draw one coin as another, and so $P(T)$ must be 1/1025.

Next, what about $P(D | T)$, the likelihood of flipping ten heads in a row, given that you chose the two-headed quarter? Clearly this is 1—if the quarter has two heads, there is no possibility of seeing anything else.

Finally, what about $P(D)$, the marginal probability of flipping ten heads in a row? As is almost always the case when using Bayes' rule, $P(D)$ is the hard part to calculate. We will use the law of total probability to do so:

$$P(D) = P(T) \cdot P(D | T) + P(\text{not } T) \cdot P(D | \text{not } T).$$

Taking the pieces on the right-hand one by one:

- As we saw above, the prior probability of the two-headed coin, $P(T)$, is $1/1025$.
- This means that the prior probability of an ordinary coin, $P(\text{not } T)$, must be $1024/1025$.
- Also from above, we know that $P(D | T) = 1$.
- Finally, we can calculate $P(D | \text{not } T)$ quite easily. If the coin is an ordinary quarter, then there is a 50% chance of getting heads on any one coin flip. Each flip is independent. Therefore, the probability of a 10-head winning streak is

$$\begin{aligned} P(D | \text{not } T) &= \frac{1}{2} \times \frac{1}{2} \times \cdots \times \frac{1}{2} \quad (\text{10 times}) \\ &= \left(\frac{1}{2}\right)^{10} = \frac{1}{1024}. \end{aligned}$$

We can now put all these pieces together:

$$\begin{aligned} P(T | D) &= \frac{P(T) \cdot P(D | T)}{P(T) \cdot P(D | T) + P(\text{not } T) \cdot P(D | \text{not } T)} \\ &= \frac{\frac{1}{1025} \cdot 1}{\frac{1}{1025} \cdot 1 + \frac{1024}{1025} \cdot \frac{1}{1024}} = \frac{1/1025}{2/1025} \\ &= \frac{1}{2}. \end{aligned}$$

Perhaps surprisingly, there is only a 50% chance that you are holding the two-headed coin. Yes, flipping ten heads in a row with a normal coin is very unlikely. But so is drawing the one two-headed coin from a jar of 1024 normal coins! In fact, as the math shows, both explanations for the data are equally unlikely, which is why we're left with a posterior probability of 0.5.

Two-headed coins in the wild. Let's return to the scenario of the trader knocking at your door, asking for a rise on the basis of a 10-month winning streak. In light of what you know about Bayes' rule, which of the following replies is the most sensible?

- (A) "You're right. Here's a giant raise."
- (B) "Thank you for letting me know. While I need more data to give you a raise, you've had a good ten months. I'll review your case again in 6 months and will look closely at the facts you've showed me."

The best answer depends very strongly on your beliefs about whether excellent stock traders are common or rare. For example, suppose you believe that 10% of all stock traders are truly excellent, in the sense that they can reliably finish with above-average returns, month after month; and that the other 90% just muddle through and collect their thoroughly average bonus checks. Then $P(T) = 0.1$, and

$$P(T | D) = \frac{0.1 \cdot 1}{0.1 \cdot 1 + 0.9 \cdot \frac{1}{1024}} \approx 0.991,$$

so that there is better than a 99% chance that your employee is among those 10% of excellent performers. You should give her a raise, or risk letting some other bank save you the trouble.

What if, however, you believed that excellence were much rarer, say $P(T) = 1/10000$? In that case,

$$P(T | D) = \frac{0.0001 \cdot 1}{0.0001 \cdot 1 + 0.9999 \cdot \frac{1}{1024}} \approx 0.093.$$

In this case, even though the ten-month hot streak was unusual— $P(D | \text{not } T)$ is small, at $1/1024$ —there is still more than a 90% chance that your employee got lucky.

The moral of the story is that the prior probability in Bayes' rule—in this case, the baseline rate of excellent stock traders, or two-headed coins—plays a very important role in correctly estimating conditional probabilities. Ignoring this prior probability is a big mistake, and such a common one that it gets its own name: the base-rate fallacy.⁷

So just how rare are two-headed coins? While it's very difficult to know the answer to this question in something like stock-trading, it is worth pointing out one fact: in the above example, a prior probability of 10% is almost surely too large. Remember the NP rule: if this prior probability were right, then out of your office of 1025 traders, you would expect there to be $0.1 \times 1025 \approx 100$ of them with 10-month winning streaks, all at your door at once clamoring for a raise. (Traders are not known for being shy about their winning streaks, or anything else.) Since this hasn't happened, the prior probability $P(T) = 0.1$ is too high to be consistent with all the data available, and should be revised downward.

On the flip side, we also know that two-headed coins in stock-picking do exist, or else there would be no explanation for Warren Buffett, known as the "Oracle of Omaha." Over the last 50 years, Warren Buffett has beaten the market so badly that it almost defies

⁷ en.wikipedia.org/wiki/Base_rate_fallacy

belief: between 1964 and 2013, the share price of his holding company, Berkshire Hathaway, has risen by about 1 million percent, versus only 2300% for the S&P 500 stock index.

This line of reasoning demonstrates that, while the prior probability often reflects your own knowledge about the world, it can also be informed by data. Either way, it is very influential, and should not be ignored.

Understanding Bayes' rule using trees

Let's try a second example of Bayes' rule in action. You may have encountered the following brain-teaser, which is the frequent subject of "first caller with the right answer wins a prize"-style contests on the radio. Here's how one San Francisco radio station described the puzzle:

There are two tribes in the jungle. The truth tellers always tell the truth and the liars always lie. A scientist comes to a fork in the road. He knows that the truth tellers' tribe is in one direction and the liars' tribe is in the other direction. But he does not know which direction is the truth tellers' tribe. There is a native sitting at the intersection of the roads. The scientist does not know whether this native is a truth teller or a liar. The scientist may ask the native one and only one question to determine the direction to the truth tellers' tribe. What question should he ask?

The scientist should ask "Which way to your village?" (Ponder for a minute why this is guaranteed to be informative.)

To illustrate Bayes' theorem, let's amend this example to include probabilities, and to adjust for the fact that it's really not cool to talk about lying tribes of jungle natives these days. Suppose you face the following situation:

You are driving through unfamiliar territory in East Texas in your burnt-orange car sporting a bumper sticker from the University of Texas. You reach a fork in the road. In one direction lies College Station; in another direction, Austin. The road sign pointing to Austin has been stolen, but you see a man selling watermelons out of his pickup truck. You pull over and ask him for directions.

You know that there are two kinds of people in this part of Texas, Longhorns and Aggies, with Aggies outnumbering Longhorns by a 60/40 margin. But you don't know which one this man is. If he's a Longhorn, he is sure to help you out to the best of his ability, and you judge that there is only a 5%

chance that he will get confused and point you in the wrong direction. But you believe that, if he is an Aggie and you ask him for directions, there is a 70% chance that he will see the bumper sticker on your car and send you the opposite way.

Having once won a pair of concert tickets by solving a similar brain teaser posed by a radio station, you decide to ask him “Which way is your university?” He stares for a moment at your bumper sticker, then smiles and points to the left. You go in the direction indicated, and two hours later you arrive in Austin.

Given that you ended up in Austin, what is the probability that the man you encountered was a Longhorn?

One way of solving this is to use Bayes’ rule directly:

$$\begin{aligned} P(\text{Longhorn} \mid \text{pointed to Austin}) &= \frac{P(\text{Longhorn}) \cdot P(\text{pointed to Austin} \mid \text{Longhorn})}{P(\text{pointed to Austin})} \\ &= \frac{0.4 \cdot 0.95}{0.6 \cdot 0.7 + 0.4 \cdot 0.95} \\ &= 0.475. \end{aligned}$$

There is slightly better than an even chance that you were talking to an Aggie.

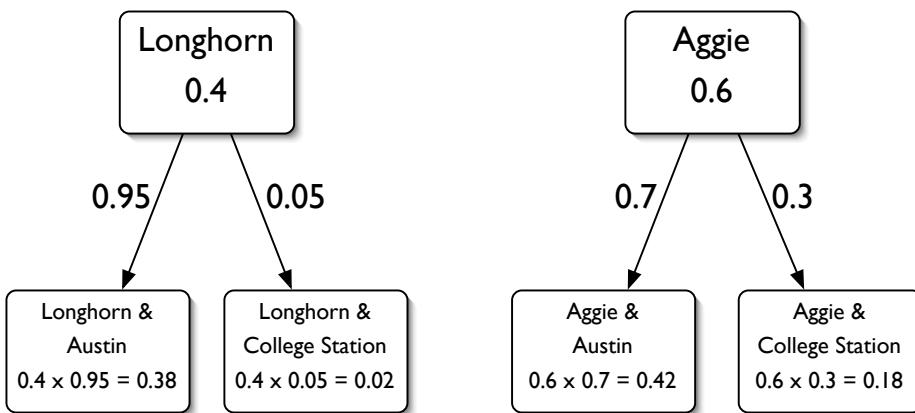
So Bayes’ rule gets us the answer with little fuss, by simply plugging in the appropriate terms to the formula. But an alternative, very intuitive way of solving this problem—and of understanding Bayes’ theorem more generally—is to use a tree.

Let’s see how this works. First, start by listing the possible states of the world, along with their probabilities. I like to put these in boxes:

Longhorn
0.4

Aggie
0.6

Next, draw arrows from each state of the world to the possible observational consequences. Along the arrows, put the conditional probabilities that you will observe each data point, given the corresponding state of the world:



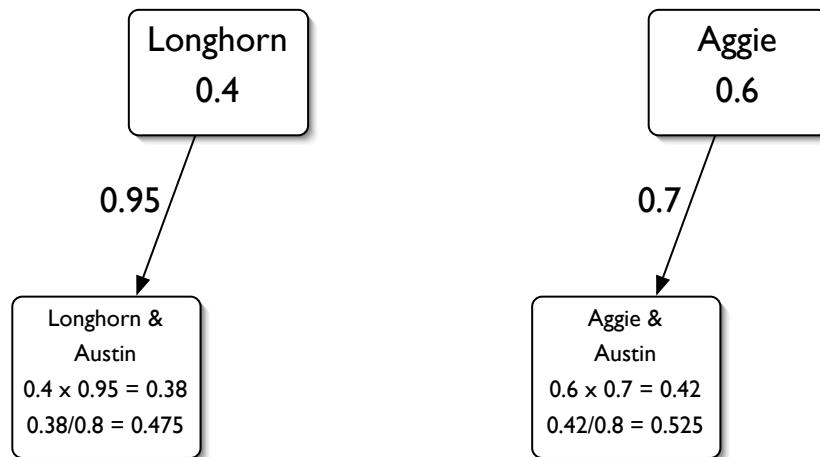
At the terminal leaves of the tree, multiply out the probabilities according to the multiplication rule: $P(A, B) = P(A) \cdot P(B | A)$. So, for example, the probability that the man is an Aggie and that he points you to Austin is $0.7 \times 0.6 = 0.42$. The sum of all the probabilities in the leaves of the tree must be 1, since they exhaust all possibilities.

But now that you've arrived back home, you know that the man was pointing to Austin. To use the tree to compute the probability that he was a Longhorn, simply cut off all the branches corresponding to data that wasn't actually observed, leaving only the actual data:



The remaining leaf probabilities are proportional to the posterior probabilities of the corresponding states of the world. These prob-

abilities, of course, do not sum to 1 anymore. But this is easily fixed: simply divide each probability by the sum of all remaining probabilities (in this case, $0.38 + 0.42 = 0.8$):



As before, we find that there is a probability of 0.475 that the man at the watermelon stand was a Longhorn. Just so you can keep things straight, the sum of 0.8, by which you divided the terminal-leaf probabilities in the final step to ensure that they summed to 1, corresponds exactly to the denominator, $P(\text{points to Austin})$, in Bayes' rule.

Bayes' rule and the law

SUPPOSE you're serving on a jury in the city of New York, with a population of roughly 10 million people. A man stands before you accused of murder, and you are asked to judge whether he is guilty (G) or not guilty ($\sim G$). In his opening remarks, the prosecutor tells you that the defendant has been arrested on the strength of a single, overwhelming piece of evidence: that his DNA matched a sample of DNA taken from the scene of the crime. Let's call denote this evidence by the letter D . To convince you of the strength of this evidence, the prosecutor calls a forensic scientist to the stand, who testifies that the probability that an innocent person's DNA would match the sample found at the crime scene is only one in a million. The prosecution then rests its case.

Would you vote to convict this man?

If you answered “yes,” you might want to reconsider! You are charged with assessing $P(G | D)$ —that is, the probability that the defendant is guilty, given the information that his DNA matched the sample taken from the scene. Bayes’ rule tells us that

$$P(G | D) = \frac{P(G) \cdot P(D | G)}{P(D)} = \frac{P(G) \cdot P(D | G)}{P(D | G) \cdot P(G) + P(D | \sim G)P(\sim G)}.$$

We know the following quantities:

- The prior probability of guilt, $P(G)$, is about one in 10 million. New York City has 10 million people, and one of them committed the crime.
- The probability of a false match, $P(D | \sim G)$, is one in a million, because the forensic scientist testified to this fact.

To use Bayes’ rule, let’s make one additional assumption: that the likelihood, $P(D | G)$, is equal to 1. This means we’re assuming that, if the accused were guilty, there is a 100% chance of seeing a positive result from the DNA test.

Let’s plug these numbers into Bayes’ rule and see what we get:

$$\begin{aligned} P(G | D) &= \frac{\frac{1}{10,000,000} \cdot 1}{1 \cdot \frac{1}{10,000,000} + \frac{1}{1,000,000} \cdot \frac{9,999,999}{10,000,000}} \\ &\approx 0.09. \end{aligned}$$

The probability of guilt looks to be only 9%! This result seems shocking in light of the forensic scientist’s claim that $P(D | \sim G)$ is so small: a “one in a million chance” of a positive match for an innocent person. Yet the prior probability of guilt is very low— $P(G)$ is a mere one in 10 million—and so even very strong evidence still only gets us up to $P(G | D) = 0.09$.

Conflating $P(\sim G | D)$ with $P(D | \sim G)$ is a serious error in probabilistic reasoning. These two numbers are typically very different from one another, because conditional probabilities aren’t symmetric. As we’ve said more than once, $P(\text{practices hard} | \text{plays in NBA}) \approx 1$, while $P(\text{plays in NBA} | \text{practices hard}) \approx 0$. Getting this wrong—that is, conflating $P(A | B)$ with $P(B | A)$ —is so common that it has its own name: the prosecutor’s fallacy.⁸

An alternate way of thinking about this result is the following. Of the 10 million innocent people in New York, ten would have DNA matches merely by chance. The one guilty person would also have a DNA match. Hence there are 11 people with a DNA match, only one of whom is guilty, and so $P(G | D) \approx 1/11$. Your intuition may mislead, but Bayes’ rule never does!

⁸ en.wikipedia.org/wiki/Prosecutor's_fallacy

Parametric probability models

Describing randomness

THE MAJOR ideas of the last few chapters all boil down to a simple idea: even random outcomes exhibit structure and obey certain rules. In this chapter, we'll learn to use these rules to build probability models, which employ the language of probability theory to provide mathematical descriptions of random phenomena. Probability models can be used to answer interesting questions about real-world systems. For example:

- American Airlines oversells a flight from Dallas to New York, issuing 140 tickets for 134 seats, because they expect at least 6 no-shows (i.e. passengers who bought a ticket but fail to show up for the flight). How likely is it that the airline will have to bump someone to the next flight?
- Arsenal scores 1.6 goals per game; Manchester United scores 1.3 goals per game. How likely is it that Arsenal beats Man U when they play each other?
- Since 1900, stocks have returned about 6.5% per year on average, net of inflation, but with a lot of variability around this mean. How does this variability affect the likely growth of your investment portfolio? How likely is it that you won't meet your retirement goals with your current investment strategy?

Building a probability model involves three steps.

- (1) Identify the *random variables* in your system. A random variable is just a term for any uncertain quantity or source of randomness. In the airline example, there is just one uncertain quantity: X = the number of no-shows on the Dallas–NYC flight. In the soccer game between Arsenal and Man U, there

are two uncertain quantities: X_1 = the number of goals scored by Arsenal, and X_2 = the number of goals scored by Man U.

- (2) Describe the possible outcomes for the random variables.

These possible outcomes are called *events*, and the set of all possible events is referred to as the *sample space* of the probability model. In the airline example, our random variable X , the number of no-shows, could be any number between 0 and 140 (the number of tickets sold). Thus the sample space is the set of integers 0 to 140.

In the soccer-game example, the sample space is a bit more complicated: it is the set of all possible scores (1-0, 2-3, 7-0, etc.) in a soccer game.

- (3) Finally, provide a rule for calculating probabilities associated with each event in the sample space. This rule is called a *probability distribution*. In the airline example, this distribution might be described using a simple lookup table based on historical data, e.g. 1% of all flights have 1 no-show, 1.2% have 2 no-shows, 1.7% have 3 no-shows, and so forth.

Parametric models for discrete outcomes

Of the three steps required to build a probability model, the third—provide a rule that can be used to calculate probabilities for each event in the sample space—is usually the hardest one. In fact, for most scenarios, if we had to build such a rule from scratch, we'd be in for an awful lot of careful, tedious work. Imagine trying to list, one by one, the probabilities for all possible outcomes of a soccer game, or all possible outcomes for the performance of a portfolio containing a mix of stocks and bonds over 40 years.

Thus instead of building probability distributions from scratch, we will rely on a simplification called a *parametric probability model*. A parametric probability model involves a probability distribution that can be completely described using a relatively small set of numbers, far smaller than the sample space itself. These numbers are called the parameters of the distribution. There are lots of commonly used parametric models—you might have heard of the normal, binomial, Poisson, and so forth—that have been invented for specific purposes. A large part of getting better at probability modeling is to learn about these existing parametric models, and to gain an appreciation for the typical kinds of real-world prob-

lems where each one is appropriate.

Before we start, we need two quick definitions. First, by a *discrete random variable*, we mean one whose sample space consists of events that you can count on your fingers and toes. Examples here include the number of no-shows on a flight, the number of goals scored by Man U in a soccer game, or the number of gamma rays emitted by a gram of radioactive uranium over the next second. (In a later section, we'll discuss continuous random variables, which can take on any value within a given range, like the price of a stock or the speed of a tennis player's serve.)

Second, suppose that the sample space for a discrete random variable X consists of events x_1, x_2 , and so forth. You'll recall that, to specify a probability model, we must provide a rule that can be used to calculate $P(X = x_k)$ for each event. When building parametric probability models, this rule takes the form of a *probability mass function*, or PMF:

$$P(X = x_k) = f(x_k | \theta).$$

In words, this equation says that the probability that X takes on the value x_k is a function of x_k . The probability mass function depends a number (or set of numbers) θ , called the parameter(s) of the model.

To specify a parametric model for a discrete random variable, we must both provide both the probability mass function f and the parameter θ . This is best illustrated by example. We'll consider two: the binomial and Poisson distributions.

The binomial distribution

ONE of the simplest parametric models in all of probability theory is called the binomial distribution, which generalizes the idea of flipping a coin many times and counting the number of heads that come up. The binomial distribution is a useful parametric model for any situation with the following properties:

- (1) We observe N different random events, each of which can be either a "yes" or a "no."
- (2) The probability of any individual event being "yes" is equal to P , a number between 0 and 1.
- (3) Each event is independent of the others.

- (4) The random variable X of interest is total number of “yes” events. Thus the sample space is the set $\{0, 1, \dots, N - 1, N\}$.

The meaning of “yes” events and “no” events will be context-dependent. For example, in the airline no-show example, we might say that a “yes” event corresponds to a single passenger failing to show up for his or her flight (which is probably not good for the passenger, but definitely a success in the eyes of an airline that’s overbooked a flight). Another example: in the PREDIMED study of the Mediterranean diet, a “yes” event might correspond to single study participant experiencing a heart attack.

If a random variable X satisfies the above four criteria, then it follows a binomial distribution, and the PMF of X is

$$P(X = k) = f(k | N, P) = \binom{N}{k} P^k (1 - P)^{N-k}, \quad (14.1)$$

where N and P are the parameters of the model. The notation $\binom{N}{k}$, which we read aloud as “ N choose k ,” is shorthand for the following expression in terms of factorials:

$$\binom{N}{k} = \frac{N!}{k!(N - k)!}.$$

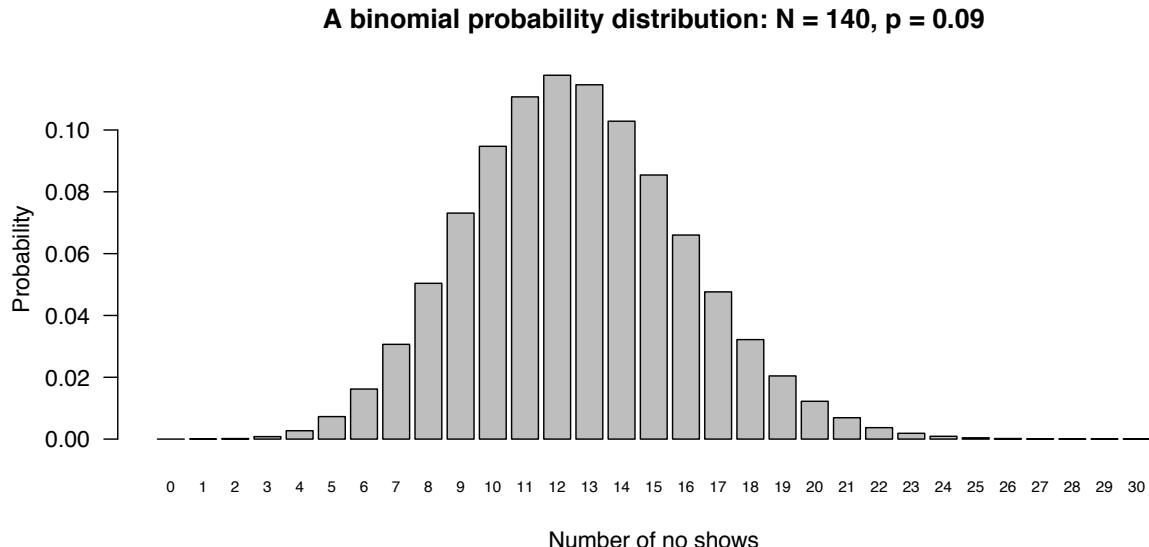
This term, called a binomial coefficient, counts the number of possible ways there are to achieve k “yes” events out of N total events. (You’ll see how this is derived in a bit.)

Example: airline no-shows Let’s use the binomial distribution as a probability model for our earlier example on airline no-shows. The airline sold tickets 140 people, each of which will either show up to fly that day (a “yes” event) or not (a “no” event). Let’s make two simplifying assumptions: (1) that each person decides to show up or not independently of the other people, and (2) that the probability of any individual person failing to show up for the flight is 9%.¹ These assumptions make it possible to apply the binomial distribution. Thus the distribution for X , the number of ticketed passengers who fail to show up for the flight, has PMF

$$P(X = k) = \binom{140}{k} (0.09)^k (1 - 0.09)^{140-k}.$$

This function of k , the number of no-shows, is plotted in Figure 14.1. The horizontal axis shows k ; the vertical axis shows $P(X = k)$ under the binomial model with parameters $N = 140, p = 0.09$.

¹ This is the industry average, quoted in “Passenger-Based Predictive Modeling of Airline No-show Rates,” by Lawrence, Hong, and Cherrier (SIGKDD 2003 August 24-27, 2003).



According to this model, the airline should expect to see around $E(X) = Np = 140 \cdot 0.09 = 12.6$ no shows, with a standard deviation of $\text{sd}(X) = \sqrt{140 \cdot 0.09 \cdot (1 - 0.09)} \approx 3.4$. But remember that the question of interest is: what is the probability of fewer than 6 no-shows? If this happens, the airline will have to compensate the passengers they bump to the next flight. We can calculate this as

$$P(X < 6) = P(X = 0) + P(X = 1) + \dots + P(X = 5) \approx 0.011,$$

i.e. by adding up the probabilities for 0 no-shows through 5 no-shows. The airline should anticipate a 1.1% chance that more people will show up than can fit on the plane.

The trade-offs of the binomial model. It's worth noting that real airlines use much more complicated models than we've just built here. These models might take into account, for example, the fact that passengers on a late connecting flight will fail to show up together non-independently, and that business travelers are more likely no-shows than families on a vacation.

The binomial model—like all parametric probability models—cannot incorporate these (very real) effects. It's just an approximation. This approximation trades away flexibility for simplicity: instead of having to specify the probability of all possible outcomes between 0 and 140, we only have to specify two numbers:

Figure 14.1: A barplot showing the probability distribution for the number of no-shows on an overbooked airline flight with 140 tickets sold, assuming a no-show rate of 9% and that individual no-shows are independent. The horizontal axis has been truncated at $k = 30$ because the probability of more than 30 no-shows is vanishingly small under the binomial model.

$N = 140$ and $p = 0.09$, the parameters of the binomial distribution. These parameters then determine the probabilities for all events in the sample space.

In light of this trade-off, any attempt to draw conclusions from a parametric probability model should also involve the answer to two important questions. First, what unrealistic simplifications have we made in building the model? Second, have these assumptions made our model *too simple*? This second answer will always be context dependent, and it's hard to provide general guidelines about what "too simple" means. Often this boils down to the question of what might go wrong if we use a simplified model, rather than invest the extra work required to build a more complicated model. This is similar to the trade-off that engineers face when they build simplified physical models of something like a suspension bridge or a new fighter jet. Like many things in statistics and probability modeling, this is a case where there is just no substitute for experience and subject-area knowledge.

The connection with the NP rule

The binomial distribution brings us back to our discussion of the NP rule, and establishes a connection between the two definitions we've seen so far of *expected value*:

The simple definition. Suppose we are in a situation with many repeated exposures (N) to the same chance event that has probability P of happening. In the long run, the expected number of events is the frequency of encounters (N), times the probability of the event in a single encounter (P). Thus expected value = $N \times P$.

The formal definition. Suppose that the possible outcomes for a random variable X are the numbers x_1, \dots, x_N . Back in Equation 10.2 on page 219, we learned that the formal definition for the expected value of X is

$$E(X) = \sum_{K=1}^N P(X = x_i) \cdot x_i .$$

Thus the expected value is the probability-weighted average of possible outcomes.

To see the connection between these two definitions, let's suppose that X is a binomial random variable: $X \sim \text{Binomial}(N, P)$.

If we apply the formal definition of expected value and churn through the math, we find that

$$\begin{aligned} E(X) &= \sum_{k=0}^{k=N} \binom{N}{k} P^k (1 - P)^{N-k} \cdot k \\ &= NP. \end{aligned}$$

We've skipped a lot of algebra steps here, but the punchline is a lot more important than the derivation: a random variable with a binomial distribution has expected value $E(X) = NP$.

This gives us a richer understanding of NP rule for expected value. The NP rule is a valid way of calculating an expected value precisely for those random events that can be described by a binomial distribution—that is, those events satisfying criteria (1)-(3) on page 283. For random events that *don't* meet these criteria, you'll need to use the formal definition from Equation 10.2 on page 219.

Note: a similar calculation shows that a random variable with a binomial distribution has standard deviation $\text{sd}(X) = \sqrt{NP(1 - P)}$.

Advanced topic: a derivation of the binomial distribution

To motivate the idea of the binomial distribution, suppose we flip a fair coin only twice.² Let our random variable X be the number of times we see “heads” in two coin flips. Thus our sample space for X has three possible outcomes—zero, one, or two. Since the coin flips are independent, all four possible sequences for the two flips (HH, HT, TH, TT) are equally likely, and the probability distribution for X is given by the following table:

| x_k | $P(X = k)$ | Cases |
|-------|------------|-------------------|
| 0 | 0.25 | 0 heads (TT) |
| 1 | 0.50 | 1 head (HT or TH) |
| 2 | 0.25 | 2 heads (HH) |

The logic of this simple two-flip case can be extended to the general case of N flips: by accounting for every possible sequence of heads and tails that could arise from N flips of a fair coin. Since successive flips are independent, every sequence of heads and tails has the same probability: $1/2^N$. Therefore,

$$P(X = k \text{ heads}) = \frac{\text{Number of sequences with } k \text{ heads}}{\text{Total number of possible sequences}}. \quad (14.2)$$

² By fair, we mean that coin is equally likely to come up heads or tails when flipped.

There are 2^N possible sequences, which gives us the denominator. To compute the numerator, we must count the number of these sequences where we see exactly k heads.

How many such sequences are there? To count them, imagine distributing the k heads among the N flips, like putting k items in N boxes, or handing out k cupcakes among N people who want one. Clearly there are N people to which we can assign the first cupcake. Once we've assigned the first, there are $N - 1$ people to which we could assign the second cupcake. Then there are $N - 2$ choices for the third, and so forth for each successive cupcake. Finally for the k th and final cupcake, there are $N - k + 1$ choices. Hence we count

$$N \times (N - 1) \times (N - 2) \times \cdots \times (N - k + 1) = \frac{N!}{(N - k)!}$$

possible sequences, where $N!$ is the factorial function. For example, if $m = 10$ and $k = 7$, this gives 604,800 sequences.

But this is far too many sequences. We have violated an important principle of counting here: don't count the same sequence more than once. The problem is that have actually counted all the ordered sequences, even though we were trying to count unordered sequences. For example, in the $N = 10, k = 7$ case, we have counted "Heads on flips {1,2,3,4,5,6,7}" and "Heads on flips {7,6,5,4,3,2,1}" as two different sequences. But they clearly both correspond to the same sequence: `HHHHHHHTTT`.

So how many times have we overcounted each unordered sequence in our tally of the ordered ones? The way to compute this is to count the number of ways we could order k objects. Given a group of k numbers which will be assigned to the "heads" category, we could have chosen from k of the objects to be first in line, from $k - 1$ of them to be second in line, from $k - 2$ of them to be third in line, and so forth. This means we have counted each unordered sequence $k!$ times. Thus the correct number of ways we could choose k objects out of N possibilities is

$$\frac{N!}{k!(N - k)!} = \binom{N}{k}.$$

For $N = 10$ and $k = 7$, this is 120 sequences—the right answer, and a far cry from the 604,800 we counted above.

Putting all these pieces together, we find that the probability of getting k heads in N flips of a fair coin is

$$P(k \text{ heads}) = \frac{N!}{k!(N - k)!} \frac{1}{2^N} = \binom{N}{k} \frac{1}{2^N}. \quad (14.3)$$

The general case. The above derivation assumes that “yes” (success) and “no” (failure) events are equally likely. Let’s now relax this assumption to see where the general definition of the binomial distribution comes from, when the probability of any individual success is not 0.5, but some rather some generic probability p .

Let’s take a sequence of N trials where we observed k successes. Each success happens with probability p , and there are k of them. Each failure happens with probability $1 - p$, and there are $m - k$ of them. Because each trial is independent, we multiply all of these probabilities together to get the probability of the whole sequence: $p^k (1 - p)^{m-k}$. Moreover, our analysis above shows that there are precisely $\binom{N}{k}$ such sequences (i.e. unique ways of getting exactly k successes and $N - k$ failures).

So if we let X denote the (random) number of successes in N trials, then for any value of k from 0 to N ,

$$P(X = k) = \binom{N}{k} p^k (1 - p)^{N-k},$$

which is the probability mass function given in Equation 14.1.

The Poisson distribution

OUR second example of a parametric probability model is the Poisson distribution, named after the French mathematician Siméon Denis Poisson.³ The Poisson distribution is used to model the number of times than some event occurs in a pre-specified interval of time. For example:

- (1) How many goals will Arsenal score in their game against Man U? (The event is a goal, and the interval is a 90-minute game.)
- (2) How many couples will arrive for dinner at a hip new restaurant between 7 and 8 PM on a Friday night? (The event is the arrival of a couple asking to sit at a table for two, and the interval is one hour).
- (3) How many irate customers will call the 1-800 number for AT&T customer service in the next minute? (The event is a phone call that must be answered by someone on staff, and the interval is one minute.)

³ The French speakers among you, or at least the fans of Disney movies, might recognize the word **poisson** from a different context. Run, Sebastian!

In each case, we identify the random variable X as the total number of events that occur in the given interval. The Poisson dis-

tribution will provide an appropriate description for this random variable if the following criteria are met:

- (1) The events occur independently; seeing one event neither increases nor decreases the probability that a subsequent event will occur.
- (2) Events occur the same average rate throughout the time interval. That is, there is no specific sub-interval where events are more likely to happen than in other sub-intervals. For example, this would mean that if the probability of Arsenal scoring a goal in a given 1-minute stretch of the game is 2%, then the probability of a goal during *any* 1-minute stretch is 2%.
- (3) The chance of an event occurring in some sub-interval is proportional to the length of that sub-interval. For example, this would mean that if the probability of Arsenal scoring a goal in any given 1-minute stretch of the game is 2%, then the probability that they score during a 2-minute stretch is 4%.

A random variable X meeting these criteria is said to follow a Poisson distribution. The sample space of a Poisson distribution is the set of non-negative integers 0, 1, 2, etc. This is one important way in which the Poisson differs from the binomial. A binomial random variable cannot exceed N , the number of trials. But there is no fixed upper bound to a Poisson random variable.

The probability mass function of Poisson distribution takes the following form:

$$P(X = k) = \frac{\lambda^k}{k!} e^{-\lambda},$$

with a single parameter λ (called the rate). This parameter governs the average number of events in the interval: $E(X) = \lambda$. It also governs the standard deviation: $sd(X) = \sqrt{\lambda}$.

Example: modeling the score in a soccer game. Let's return to our soccer game example. Across all games in the 2015-16 English Premiere League (widely considered to be the best professional soccer league in the world), Arsenal scored 1.6 goals per game, while Manchester United scored 1.3 goals per game. How likely is it that Arsenal beats Man U? How likely is a scoreless draw at 0-0? To answer these questions, let's make some simplifying assumptions.

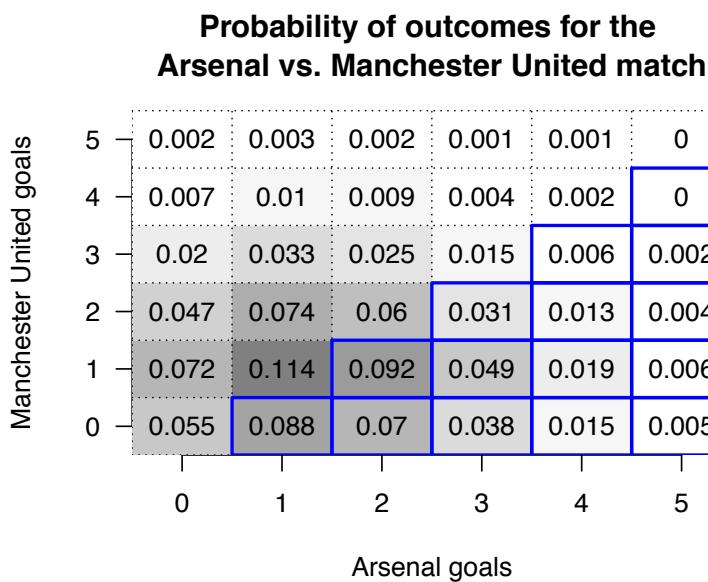


Figure 14.2: A matrix of probabilities associated with various match scores under the independent Poisson model of an Arsenal vs. Man U match, based on scoring statistics from 2015–16 Premier League season. Each entry in the matrix is the probability with the corresponding score (darker grey = higher probability). The cells outlined in blue correspond to an Arsenal win, which happens with probability 44% (versus 25% for a draw and 31% for a Manchester United win).

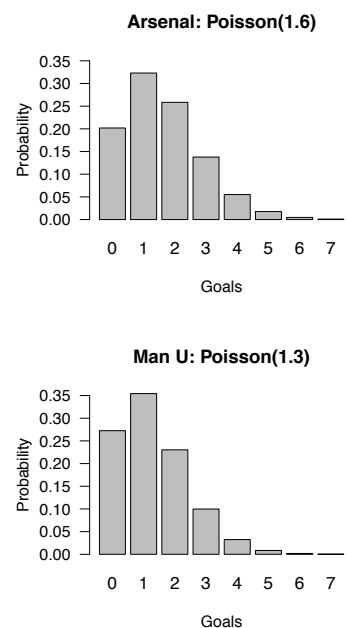
- (1) Let X_A be the number of goals scored in a game by Arsenal. We will assume that X_A can be described by a Poisson distribution with rate parameter 1.6: that is, $X_A \sim \text{Poisson}(\lambda = 1.6)$.
- (2) Let X_M be the number of goals scored in a game by Manchester United. We will assume that $X_M \sim \text{Poisson}(\lambda = 1.3)$.
- (3) Finally, we will assume that X_A and X_M are independent of one another.

Our model sets the rate parameters for each team's Poisson distribution to match their average scoring rates across the season. The corresponding PMFs are shown at right.

Under these simplifying assumptions, we can calculate the probability of any possible score—for example, Arsenal 2–0 Manchester United. Because we have assumed that X_A and X_M are independent, we can multiply together the two probabilities we get from each random variable's Poisson distribution:

$$P(X_A = 2, X_M = 0) = \left(\frac{1.6^2}{2!} e^{-1.6} \right) \cdot \left(\frac{1.3^0}{0!} e^{-1.3} \right) \approx 0.07.$$

Figure 14.2 shows a similar calculation for all scores ranging from 0–0 to 5–5 (according to the model, the chance of a score



larger than this is only 0.6%). By summing up the probabilities for the various score combinations, we find that:

- Arsenal wins with probability 44%.
- Man U wins with probability 31%.
- The game ends in a draw with probability 25%. In particular, a scintillating 0–0 draw happens with probability 5.5%.

The normal distribution

This chapter's third and final example of a parametric probability model is the normal distribution—the most famous and widely used distribution in the world.

Some history

Historically, the normal distribution arose as an approximation to the binomial distribution. In 1711, a Frenchman named Abraham de Moivre published a book called *The Doctrine of Chances*. The book was reportedly prized by gamblers of the day for its many useful calculations that arose in dice and card games. In the course of writing about these games, de Moivre found it necessary to perform computations using the binomial distribution for very large values of N , the number of independent trials in a binomial distribution. (Imagine flipping a large number of coins and making bets on the outcomes, and you too will see the necessity of this seemingly esoteric piece of mathematics.)

As you recall from the previous section, these calculations require computing binomial coefficients $\binom{N}{k}$ for very large values of N . But because these computations involve the factorial function, they were far too time-consuming without modern computers, which de Moivre didn't have. So he derived an approximation based on the number $e \approx 2.7183$, the base of the natural logarithm. He discovered that, if a random variable X has a binomial distribution with parameters N and p , which we recall is written $X \sim \text{Binomial}(N, p)$, then the approximate probability that $X = k$ is

$$P(X = k) \approx \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{(k-\mu)^2}{2\sigma^2}}, \quad (14.4)$$

where $\mu = mp$ and $\sigma^2 = Np(1-p)$ are the expected value and variance, respectively, of the binomial distribution. When consid-

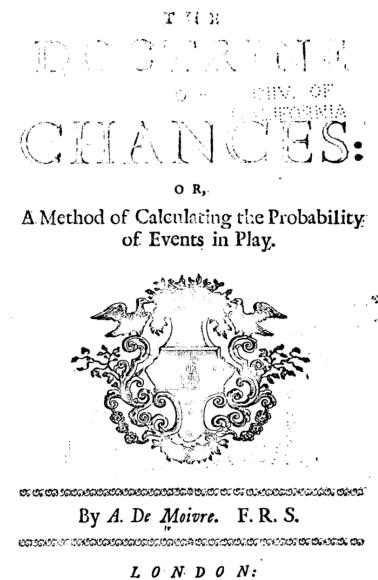


Figure 14.3: The title page of de Moivre's "The Doctrine of Chances" (1711), from an early edition owned by the University of California, Berkeley. One interesting thing about the history of statistics is the extent to which beautiful mathematical results came out of the study of seemingly trivial gambling and parlor games.

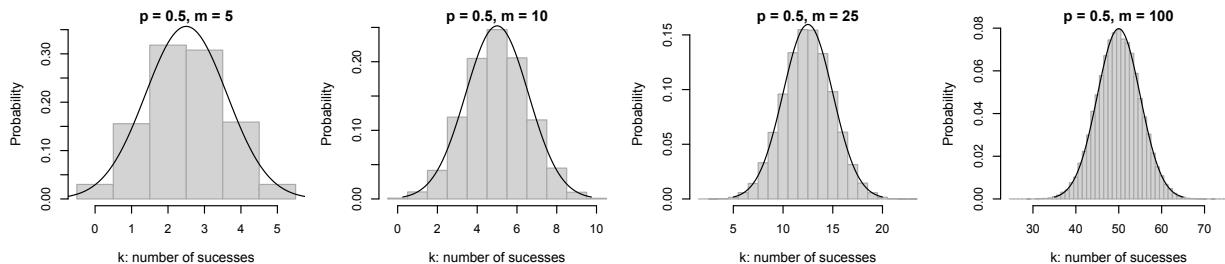


Figure 14.4: The binomial distribution for $p = 0.5$ and an increasingly large number of trials, together with de Moivre's normal approximation.

ered as a function k , this results in the familiar bell-shaped curve plotted in Figure 14.4—the famous *normal distribution*.

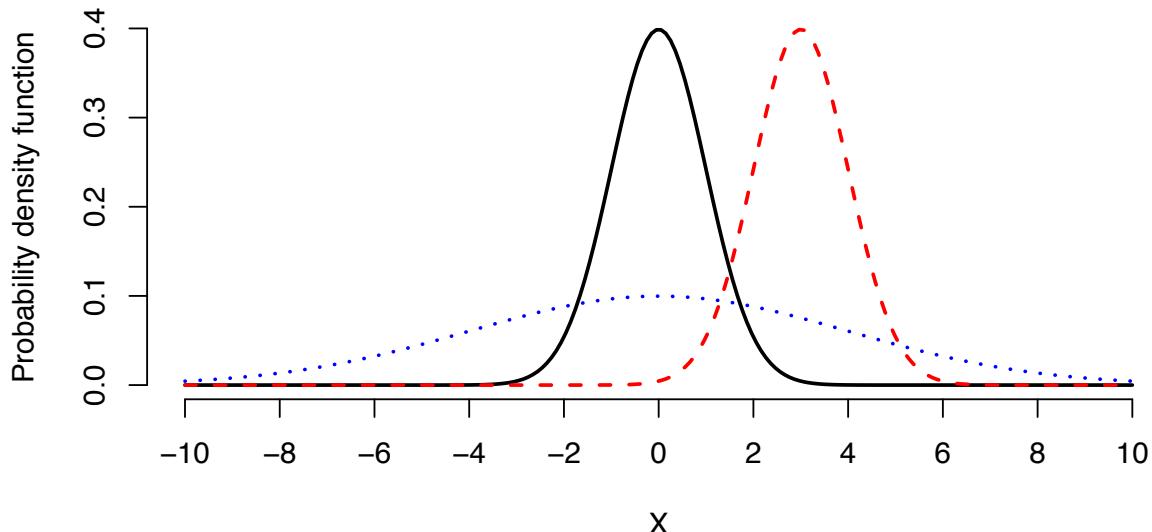
We can usually (though not always) avoid working with this expression directly, since every piece of statistical software out there can compute probabilities under the normal distribution. The important thing to notice is how the binomial samples in Figure 14.4 start to look more normal as the number of trials N gets progressively larger: first 5, then 10, 25, and finally 100. The histograms show the binomial distribution itself, while the black curves show de Moivre's approximation. Clearly he was on to something. This famous result of de Moivre's is usually thought of as the first *central limit theorem* in the history of statistics, where the word “central” should be understood to mean “fundamental.”

The normal distribution: a modern understanding

The other term for the normal distribution is the Gaussian distribution, named after the German mathematician Carl Gauss. This raises a puzzling question. If de Moivre invented the normal approximation to the binomial distribution in 1711, and Gauss (1777–1855) did his work on statistics almost a century after de Moivre, why then is the normal distribution also named after Gauss and not de Moivre? This quirk of eponymy arises because de Moivre only viewed his approximation as a narrow mathematical tool for performing calculations using the binomial distribution. He gave no indication that he saw it as a more widely applicable probability distribution for describing random phenomena. But Gauss—together with another mathematician around the same time, named Laplace—did see this, and much more.

If we want to use the normal distribution to describe our uncertainty about some random variable X , we write $X \sim N(\mu, \sigma^2)$. The numbers μ and σ^2 are parameters of the distribution. The first

Three members of the normal family



parameter, μ , describes where X tends to be centered; it also happens to be the expected value of the random variable. The second parameter, σ^2 , describes how spread out X tends to be around its expected value; it also happens to be the variance of the random variable. Together, μ and σ^2 completely describe the distribution, and therefore completely characterize our uncertainty about X .

The normal distribution is described mathematically by its probability density function, or PDF:

$$p(x) = \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left(-\frac{(x-\mu)^2}{2\sigma^2}\right). \quad (14.5)$$

If you plot this as a function of x , you get the famous bell curve (Figure 14.5). How can you interpret a “density function” like this one? If you take the area under this curve between two values z_1 and z_2 , you will get the probability that the random variable X will end up falling between z_1 and z_2 (see Figure 14.6). The height of the curve itself is a little more difficult to interpret, and we won’t worry about doing so—just focus on the “area under the curve” interpretation.

Here are two useful facts about normal random variables

Figure 14.5: Three members of the normal family: $N(0, 1^2)$, $N(0, 4^2)$, and $N(3, 1^2)$. See if you can identify which is which using the guideline that 95% of the probability will be within two standard deviations σ of the mean. Remember, the second parameter is the variance σ^2 , not the standard deviation. So $\sigma^2 = 4^2$ means a variance of 16 and a standard deviation of 4.

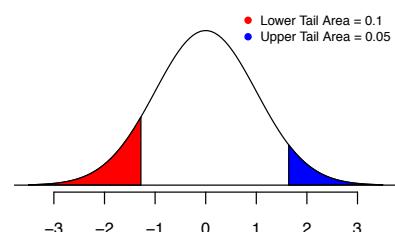


Figure 14.6: Examples of upper and lower tail areas. The lower tail area of 0.1 is at $z = -1.28$. The upper tail area of 0.05 is at $z = 1.64$.

areas—or more specifically, about the central areas under the curve, between the tails. If $X \sim N(\mu, \sigma^2)$, then the chance that X will be within 1σ of its mean is about 68%, and the chance that it will be within 2σ of its mean is about 95%. Said in equations:

$$\begin{aligned} P(\mu - 1\sigma < X < \mu + 1\sigma) &\approx 0.68 \\ P(\mu - 2\sigma < X < \mu + 2\sigma) &\approx 0.95. \end{aligned}$$

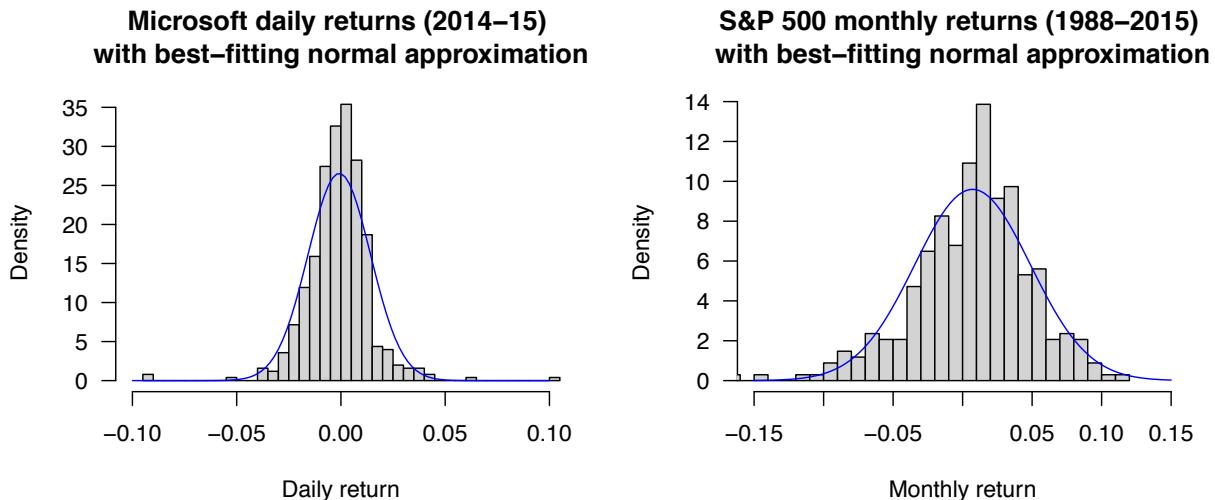
Actually, it's more like 1.96σ rather than 2σ for the second part. So if your problem requires a level of precision to an order of 0.04σ or less, then don't use this rule of thumb, and instead go with the true multiple of 1.96.

When is the normal distribution an appropriate model?

The normal distribution is now used as a probability model in situations far more diverse than de Moivre, Gauss, or Laplace ever would have envisioned. But it still bears the unmistakeable traces of its genesis as a large-sample approximation to the binomial distribution. That is, it tends to work best for describing situations where each normally distributed random variable can be thought of as the sum of many tiny, independent effects of about the same size, some positive and some negative. In cases where this description doesn't apply, the normal distribution may be a poor model of reality. Said another way: the normal distribution describes an aggregation of nudges: some up, some down, but all pretty small.

As a result, the normal distribution shares the property of the binomial distribution that huge deviations from the mean are unlikely. It has, in statistical parlance, "thin tails." Using our rule of thumb above, a normally distributed random variable has only a 5% chance of being more than two standard deviations away from the mean. It also has less than a 0.3% chance of being more than three standard deviations away from the mean. Large outliers are vanishingly rare.

For example, in the histogram of daily returns for Microsoft stock in the left panel Figure 14.7, notice the huge outliers in the lower tail. These returns would be wildly implausible if the returns really followed a normal distribution. A daily return tends to be dominated by one or two major pieces of information. It does not resemble an aggregation of many independent up-or-down nudges, and so from first principles alone, we should probably expect the normal distribution to provide a poor fit. As we



would expect, the best-fitting normal approximation (i.e. the one that matches the sample mean and standard deviation of the data) does not fit especially well.

The example of Microsoft stock recalls the earlier discussion on the trustworthiness of the simplifying assumptions that must go into building a probability model. To recap:

Have these assumptions made our model too simple? This . . . answer will always be context dependent, and it's hard to provide general guidelines about what "too simple" means.

Often this boils down to the question of what might go wrong if we use a simplified model, rather than invest the extra work required to build a more complicated model.

What might go wrong if we use a normal probability model for Microsoft returns? In light of what we've seen here, the answer is: we might be very unpleasantly surprised by monetary losses that are far more extreme than envisioned under our model. This sounds very bad, and is probably a sufficient reason not to use the normal model in the first place. To make this precise, observe that the 2 most extreme daily returns for Microsoft stock were both 6 standard deviations below the mean. According to the normal model, we should only expect to see such an extreme result once every billion trading days, since

$$P(X < \mu - 6\sigma) \approx 10^{-9}.$$

Figure 14.7: Daily stock returns for Microsoft (left) and the S&P 500 (right), together with the best-fitting normal approximations. The approximation on the right is not bad, while the approximation on the left drastically underestimates the probability of extreme results.

This is a wildly overoptimistic assessment, given that we actually saw two such results in the 503 trading days from 2014–15.

On the other hand, the normal distribution works a lot better for stock indices than it does for individual stocks, especially if we aggregate those returns over a month rather than only a day, so that the daily swings tend to average out a bit more. Take, for example, the best-fitting normal approximation for the monthly returns of the S&P 500 stock index from 1988 to 2015, in the right panel of Figure 14.7. Here the best-fitting normal distribution, though imperfect, looks a lot better than the corresponding fit for an individual stock on the left. Here, the most extreme monthly return was 4 standard deviations below the mean (which happened in October 2008, during the financial crisis of that year that augured the Great Recession). According to the normal model, we would expect such an extreme event to happen with about 2% probability in any given 27-year stretch. Thus our model looks a tad optimistic, but not wildly so.

Example: modeling a retirement portfolio

From 1900–2015, the average annual return⁴ of the S&P 500 stock index is 6.5%, with a standard deviation of 19.6%. Let's use these facts to build a probability model for the future 40-year performance of a \$10,000 investment in a diversified portfolio of U.S. stocks (i.e. an index fund). While there's no guarantee that past returns are a reliable guide to future returns, they're the only data we have. After all, as Mark Twain is reputed to have said, "History doesn't repeat itself, but it does rhyme."

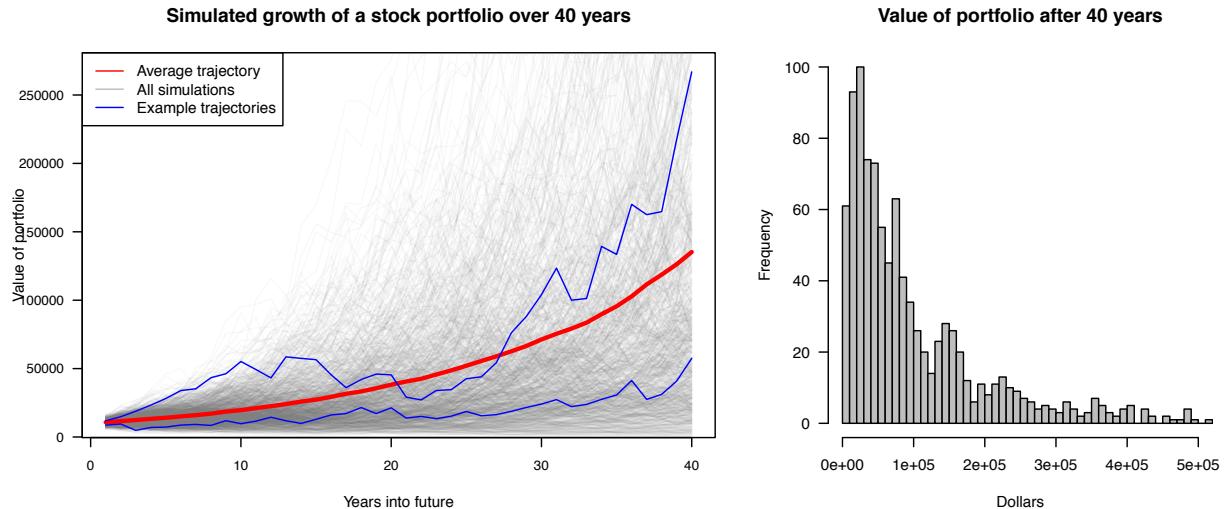
Let's say that your initial investment is $W_0 = \$10,000$, and that X_t is the return of your portfolio in year t expressed as a decimal fraction (e.g. a 10% return in year 1 would mean that $X_t = 0.1$). Here t will run from 1 to 40, since we want to track your portfolio over 40 years. If we knew the returns X_1, X_2, \dots, X_{40} all the way into the future, we could calculate your terminal wealth as

$$W_{40} = W_0 \cdot \prod_{t=1}^{40} (1 + X_t),$$

by simply compounding the interest year after year.⁵ This formula follows from the fact that W_{t+1} , your wealth in year t , is given by the simple interest formula: $W_{t+1} = W_t \cdot (1 + X_t)$. Accumulating returns year after year then gives us the above formula.

⁴ Real returns net of inflation and dividends. Remember that a return is simply the implied interest rate from holding an asset for a specified period. If you buy a stock at \$100 and sell a year later at \$110, then your return is $(110 - 100)/100 = 0.1$, or 10%. If inflation over that year was 3%, then your real return was 7%.

⁵ Here the symbol \prod means we take the running product of all the terms, from $t = 1$ to $t = 40$, just like Σ means we take a running sum.



Of course, we don't know these interest rates. But we do have a probability model for them, whose parameters have been chosen to match the historical record: $X_t \sim N(\mu = 0.065, \sigma^2 = 0.196^2)$. Thus to estimate the probability distribution of the random variable W_{40} , your terminal wealth after 40 years, we will use a Monte Carlo simulation, in which we repeat the following steps many thousands of times:

- (1) Simulate random returns from the normal probability model:

$$X_t \sim N(0.065, 0.196^2) \text{ for } t = 1, \dots, 40.$$
- (2) Starting with year $t = 1$ and ending with year $t = 40$, chain these simulated interest rates together using the simple-interest formula

$$W_{t+1} = W_t \cdot (1 + X_t)$$

to form a single simulated trajectory W_1, W_2, \dots, W_{40} of wealth.

As a byproduct of this, we get a simulated probability distribution of W_t for all values of t from 1 up to 40.

Figure 14.8 shows 1000 trajectories simulated according to this algorithm, along with the histogram of the 1000 different values of W_{40} , your wealth in 40 years. There are several interesting things to point out about the result:

- (1) The *average* trajectory in Figure 14.8 results in a final value of $W_{40} \approx \$135,000$ from your initial \$10,000 investment.⁶

Figure 14.8: Left panel: 1000 simulated trajectories for the growth of a \$10,000 stock investment over 40 years, assuming that year stock returns are normally distributed with a mean of 6.5% and a standard deviation of 19.6%. Two individual trajectories (leading to very different outcomes) are highlighted in blue; the average trajectory is shown in red. The right panel shows the simulated probability distribution for W_{40} , the final value of the portfolio after 40 years of random returns.

⁶ Remember that our assumed rates of return are adjusted for inflation, so this corresponds to the purchasing power of \$135,000 in today's money. The actual dollar value of this portfolio, as measured in the currency of the future, would be a good deal higher.

- (2) But there is tremendous variability about this average trajectory, both over time for a single trajectory, and across all trajectories. To illustrate this point, two simulated trajectories are shown in blue in Figure 14.8: one resulting in a final portfolio of about \$250,000, and another resulting in less than \$50,000.
- (3) The simulated probability distribution of final wealth (right panel of Figure 14.8) was constructed using nothing but normally distributed random variables as inputs. But this distribution is itself highly non-normal.⁷ This provides a good example of using Monte Carlo simulation to simulate a complex probability distribution by breaking down into a function of many smaller, simpler parts (in this case, the yearly returns).
- (4) The estimated probability that your \$10,000 investment will have lost money (net of inflation) after 10 years is about 19%; after 20 years, about 13%; after 40 years, about 6%.
- (5) The estimated probability that your investment will grow to \$1 million or more after 40 years is about 1%.

The moral of the story is that the stock market is probably a good way to get rich over time. But there's a nonzero chance of losing money—and the riches come only in the long run, and with a lot of uncertainty about how things will unfold along the way.

Postscript

We've now seen three examples of parametric probability models: a binomial model for airline no-shows, a Poisson model for scoring in a soccer game, and a normal model for annual returns of the stock market. In each case, we chose the parameters of the probability model from real-world data, using simple and obvious criteria (e.g. the overall no-show rate for commercial flights, or the mean return of stocks over the last century).⁸ In essence, we performed a naïve form of statistical inference for the parameters of our probability models. This intersection where probability modeling meets data is an exciting place where the big themes of the book all come together.

⁷ In particular it has a long right tail, reflecting the small probability of explosive growth in your investment.

⁸ Technically what we did here was called *moment matching*, wherein we match sample moments (e.g. mean, variance) of the data to the corresponding moments of the probability distribution.

15

Correlated random variables

Joint distributions for discrete variables

In this chapter, we study probability distributions for coupled sets of random variables. We'll first work through a simple example involving two discrete random variables. This will allow us to introduce some basic concepts before turning to more complex examples.

A simple example

The key concept in this chapter is that of a *joint distribution*. We recall that a joint distribution is a list of joint outcomes for two or more variables at once, together with the joint probabilities for each of these outcomes.

Let's look at a simple example, regarding the number of bedrooms and bathrooms for houses and condos currently up for sale in Austin, Texas. Let X_{be} be the number of bedrooms that a house has, and let X_{ba} be the number of bathrooms. The following matrix of joint probabilities specifies a joint probability distribution $P(X_{ba}, X_{be})$:

| Bedrooms | Bathrooms | | | | Marginal |
|----------|-----------|-------|-------|-------|----------|
| | 1 | 2 | 3 | 4 | |
| 1 | 0.003 | 0.001 | 0.000 | 0.000 | 0.004 |
| 2 | 0.068 | 0.113 | 0.020 | 0.000 | 0.201 |
| 3 | 0.098 | 0.249 | 0.126 | 0.004 | 0.477 |
| 4 | 0.015 | 0.068 | 0.185 | 0.015 | 0.283 |
| 5 | 0.002 | 0.005 | 0.017 | 0.006 | 0.030 |
| 6 | 0.001 | 0.001 | 0.002 | 0.001 | 0.005 |
| Marginal | 0.187 | 0.437 | 0.350 | 0.026 | |

Using the marginal probabilities alone, we can straightfor-

wardly calculate the expected value and variance for the number of bedrooms and bathrooms. We'll explicitly show the calculation for the expected number of bathrooms, and leave the rest as an exercise to be verified:

$$\begin{aligned} E(X_{ba}) &= 0.187 \cdot 1 + 0.437 \cdot 2 + 0.350 \cdot 3 + 0.026 \cdot 4 \\ &= 2.215 \end{aligned}$$

$$\text{var}(X_{ba}) = 0.595$$

$$E(X_{be}) = 3.149$$

$$\text{var}(X_{be}) = 0.643$$

Covariance

But these moments only tell us about the two variables in isolation, rather than the way they vary together. When two or more variables are in play, the mean and the variance of each one are no longer sufficient to understand what's going on. In this sense, a quantitative relationship is much like a human relationship: you can't describe one by simply listing off facts about the characters involved. You may know that Homer likes donuts, works at the Springfield Nuclear Power Plant, and is fundamentally decent despite being crude, obese, and incompetent. Likewise, you may know that Marge wears her hair in a beehive, despises the *Itchy and Scratchy Show*, and takes an active interest in the local schools. Yet these facts alone tell you little about their marriage. A quantitative relationship is the same way: if you ignore the interactions of the "characters," or individual variables involved, then you will miss the best part of the story.

To quantify the strength of association between two variables, we will calculate their *covariance*. The general definition of covariance is as follows. Suppose that there are N possible joint outcomes for X and Y . Then

$$\text{cov}(X, Y) = E\left\{ [X - E(X)][Y - E(Y)] \right\} = \sum_{i=1}^n p_i [x_i - E(X)] [y_i - E(Y)].$$

This sum is over all possible combinations of joint outcomes for X and Y . In our example about houses for sale, there are 24 terms in the sum, because there are 24 unique combinations for X_{be} and X_{ba} . In the following calculation, a handful of these terms are

shown explicitly, with most shown as ellipses:

$$\begin{aligned}\text{cov}(X_{ba}, X_{be}) &= 0.003 \cdot (1 - 2.215)(1 - 3.149) \\ &\quad + 0.068 \cdot (1 - 2.215)(2 - 3.149) \\ &\quad + \dots \\ &\quad + 0.185 \cdot (3 - 2.215)(4 - 3.149) \\ &\quad + \dots \\ &\quad + 0.005 \cdot (4 - 2.215)(6 - 3.149) \\ &\approx 0.285.\end{aligned}$$

In this summation, some of the terms are positive and sum of the terms are negative. The positive terms correspond to joint outcomes when the number of bedrooms and bathrooms are on the *same side* of their respective means—that is, both above the mean, or both below it. The negative terms, on the other hand, correspond to outcomes where the two quantities are on *opposite sides* of their respective means. In this case, the “same side” outcomes are more likely than the “opposite side” outcomes, and therefore the covariance is positive.

Correlation as standardized covariance

One difficulty that arises in interpreting covariance is that it depends upon the scale of measurement for the two sets of observations. This isn’t so objectionable in the above example (it’s hard to imagine what other units we would use). Nonetheless, it’s nice to have a unit-free measure of association—especially for a variable like distance, which we could measure in miles or millimeters.

One such scale-invariant measure is the *correlation* between two random variables, which is analogous to the concept of sample correlation between two variables in a data set. The correlation coefficient for two random variables X and Y is just their covariance, rescaled by their respective standard deviations:

$$\text{cor}(X, Y) = \frac{\text{cov}(X, Y)}{\sqrt{\text{var}(X)} \cdot \sqrt{\text{var}(Y)}}.$$

It runs from -1 (perfect negative correlation) to $+1$ (perfect positive correlation).

Let’s apply this definition to calculation the correlation between the number of bedrooms (X_{be}) and number of bathrooms (X_{ba})

under the joint distribution given earlier:

$$\text{cor}(X_{ba}, X_{be}) = \frac{0.285}{\sqrt{0.595} \cdot \sqrt{0.643}} \approx 0.745.$$

The bivariate normal distribution

Heredity and regression to the mean

THE history of statistics is intertwined with the history of how scientists came to understand heredity. How strongly do the features of one generation manifest themselves in the next generation? What governs this process, and how can we quantify it mathematically? These questions fascinated scientists of the late 19th and early 20th centuries. As they grappled with them, they also invented a lot of new statistical tools.¹

One famous study of heredity, by Francis Galton in the 1880's, resulted in the data similar to what you see in the left panel of Figure 15.1.² As part of Galton's study of heredity, he collected data on the adult height of parent-child pairs. He wanted to quantify mathematically the extent to which height was inherited from one generation to the next. In looking into this question, Galton noticed some interesting facts about his data.

- Consider the 20 tallest fathers in the data set, highlighted in blue in Figure 15.1. These 20 men had a mean height that was about 6.2 inches above their generation's average height. But the sons of these 20 men had an average height that was only 2.8 inches above their generation's average height. Thus the sons of very tall men were taller than average, but not by as much as their fathers were.
- Now consider the 20 shortest fathers in the data set, highlighted in red in Figure 15.1. These 20 men had a mean height that was about 6.9 inches below their generation's average height. But the sons of these 20 men had an average height that was only 3.3 inches below their generation's average height. Thus the sons of very short men were shorter than average, but not by as much as their fathers were.

Galton called this phenomenon "regression towards mediocrity," where "mediocre" should be understood in the sense of "average." Galton's proposed explanation for this phenomenon

¹ It's important to mention that many of these developments were pursued at least partially in the name of the eugenics movement. While the mathematical tools left to us as a result of these studies remain valuable, their history is not something to be unreservedly proud of. If you're interested in reading more about this, try the following article: "Sir Francis Galton and the birth of eugenics," by N.W. Gilham. *Annual Review of Genetics*, 2001, 35:83–101.

² This data was actually collected and analyzed by Galton's protégé, Karl Pearson. But Galton worked with very similar data, so we'll pretend for the purposes of exposition that this was Galton's data, since he was the first one to follow this line of thought.

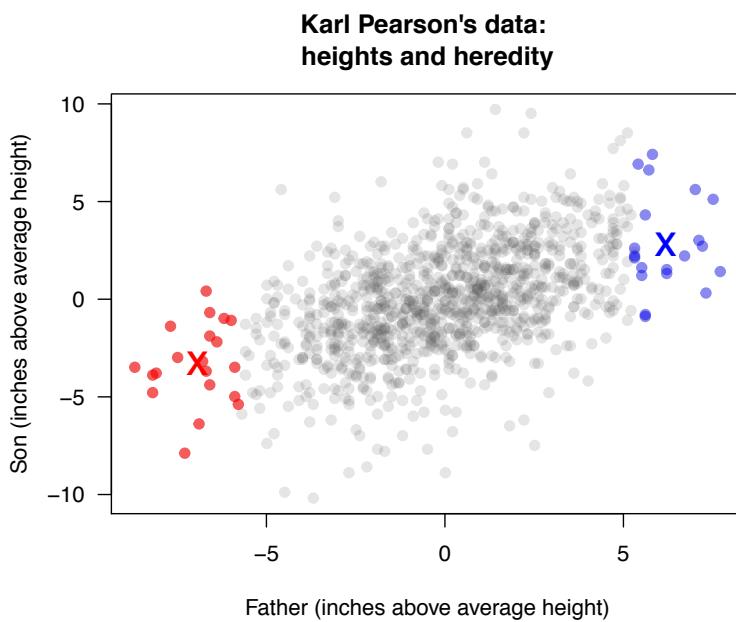


Figure 15.1: Karl Pearson's data on the height of fathers and their adult sons. The 20 tallest fathers (and their sons) are highlighted in blue, with the bivariate mean of this group shown as a blue X. Similarly, the 20 shortest fathers (and their sons) are highlighted in red, with the bivariate mean of this group shown as a red X. The points show fathers and sons only, to avoid any confounding due to sex. We've also mean-centered the data, by subtracting the average height of all fathers from each father's height, and the average height of all sons from each son's height. This doesn't change the shape of the point cloud; it merely re-centers it at $(0, 0)$. This accounts for the fact that the sons' generation, on average, was about an inch taller than the fathers' generation—possibly due to improving standards of health and nutrition.

turned out to be incorrect, but today we understand it as a product of genetics. It's hard to explain exactly why this happens without getting deep into the weeds on multifactorial inheritance, but the rough idea is the following. (We'll focus on the tallest fathers in the data set, but the same line of reasoning works for the shortest fathers, too.)

- Very tall people, like Yao Ming at right, turn out that way for a combination of two reasons: height genes and height luck. (Here "luck" is used to encompass both environmental forces as well as some details of multifactorial inheritance not worth going into here.)
- Therefore, our selected group of very tall people (the blue dots in Figure 15.1) is biased in two ways: extreme height genes *and* extreme height luck.
- These very tall people pass on their height genes to their children, but not their height luck.
- Height luck will average out in the next generation. Therefore, the children of very tall parents will still be tall (be-



Figure 15.2: Yao Ming makes J.J. Watt (6'5" tall, 290 pounds) look like a child.

cause of genes), but not as well as their parents (because they weren't as lucky, on average).

Notice that this isn't a claim about causality. It is not true that the children of very tall people are likely to have less extreme "height luck" *because* their parents had a lot of it. Rather, these children are likely to have less luck than their parents because extreme luck is, by definition, rare—and they are no more likely to experience this luck than any randomly selected group of people.

This phenomenon that we've observed about height and heredity is actually quite general. Take any pair of correlated measurements. If one measurement is extreme, then the other measurement will tend to be closer to the average. Today we call this *regression to the mean*. Just as Galton did in 1889, we can make this idea mathematically precise using a probability model called the bivariate normal distribution. This requires a short detour.

Notation for the bivariate normal

The *bivariate normal distribution* is a parametric probability model for the joint distribution of two correlated random variables X_1 and X_2 . You'll recall that the ordinary normal distribution is a distribution for one variable with two parameters: a mean and a variance. The bivariate normal distribution is for two variables (X_1 and X_2), and it has five parameters:

- The mean and variance of the first random variable: $\mu_1 = E(X_1)$ and $\sigma_1^2 = \text{var}(X_1)$.
- The mean and variance of the second random variable: $\mu_2 = E(X_2)$ and $\sigma_2^2 = \text{var}(X_2)$.
- The covariance between X_1 and X_2 , which we denote as σ_{12} .

Equivalently, we can specify the correlation instead of the covariance. We recall that the correlation is just the covariance rescaled by both standard deviations:

$$\rho = \frac{\text{cov}(X_1, X_2)}{\text{sd}(X_1) \cdot \text{sd}(X_2)} = \frac{\sigma_{12}}{\sigma_1 \cdot \sigma_2}.$$

In practice we will usually instead refer to the standard deviations σ_1 and σ_2 and correlation ρ rather than the variances and covariances, and use the shorthand $(X_1, X_2) \sim N(\mu_1, \mu_2, \sigma_1, \sigma_2, \rho)$.

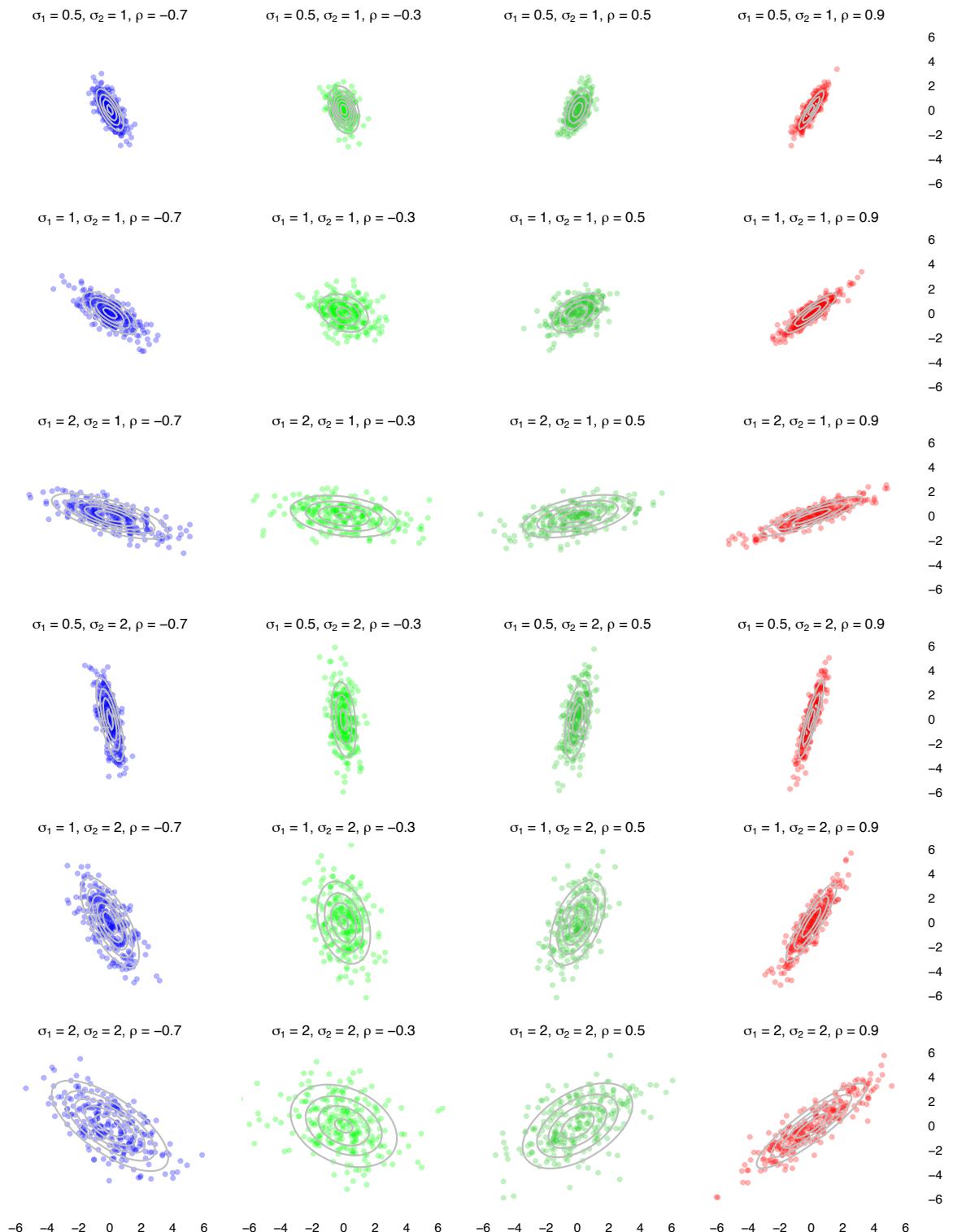


Figure 15.3: 24 examples of a bivariate normal distribution (250 samples in each plot).

We can also write a bivariate normal distribution using matrix-vector notation, to emphasize the fact that $X = (X_1, X_2)$ is a random vector:

$$\begin{pmatrix} X_1 \\ X_2 \end{pmatrix} \sim N \left(\begin{bmatrix} \mu_1 \\ \mu_2 \end{bmatrix}, \begin{bmatrix} \sigma_1^2 & \sigma_{12} \\ \sigma_{12} & \sigma_2^2 \end{bmatrix} \right),$$

or simply $X \sim N(\mu, \Sigma)$, where μ is the mean vector and Σ is called the covariance matrix.

The bivariate normal distribution has the nice property that each of its two marginal distributions are ordinary normal distributions. That is, if we ignore X_2 and look only at X_1 , we find that $X_1 \sim N(\mu_1, \sigma_1^2)$. Similarly, if we ignore X_1 and look only at X_2 , we find that $X_2 \sim N(\mu_2, \sigma_2^2)$.

Visualizing the bivariate normal distribution

Figure 15.3 provides some intuition for how the various parameters of the bivariate normal distribution affect its shape. Here we see 24 examples of a bivariate normal distribution with different combinations of standard deviations and correlations. In each panel, 250 random samples of (X_1, X_2) from the corresponding bivariate normal distribution are shown:

- Moving down the rows from top to bottom, the standard deviations of the two variables change, while the correlation remains constant within a column.
- Moving across the columns from left to right, the correlation changes from negative to positive, while the standard deviations of the two variables remain the same within a row.

The mean of both variables is 0 in all 24 panels. Changing either mean would translate the point cloud so that it was centered somewhere else, but would not change the shape of the cloud.

Each panel of Figure 15.3 also shows a *contour plot* of the probability density function for the corresponding bivariate normal distribution, overlaid in grey. We read these contours in a manner similar to how we would on an ordinary *contour map*: they tell us how high we are on the three-dimensional surface of the bivariate normal density function, like the one shown at right.

To interpret this density function, imagine specifying two intervals, one for X_1 and another for X_2 , and asking: what is the probability that both X_1 and X_2 fall in their respective intervals?

$$\sigma_1 = 1, \sigma_2 = 1.5, \rho = 0.5$$

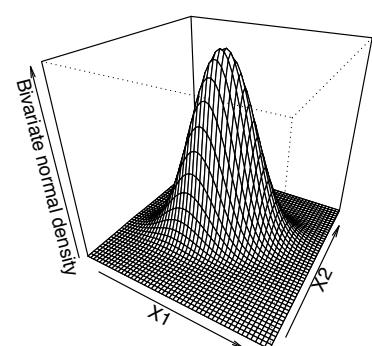


Figure 15.4: A three-dimensional wireframe plot of a bivariate normal density function.

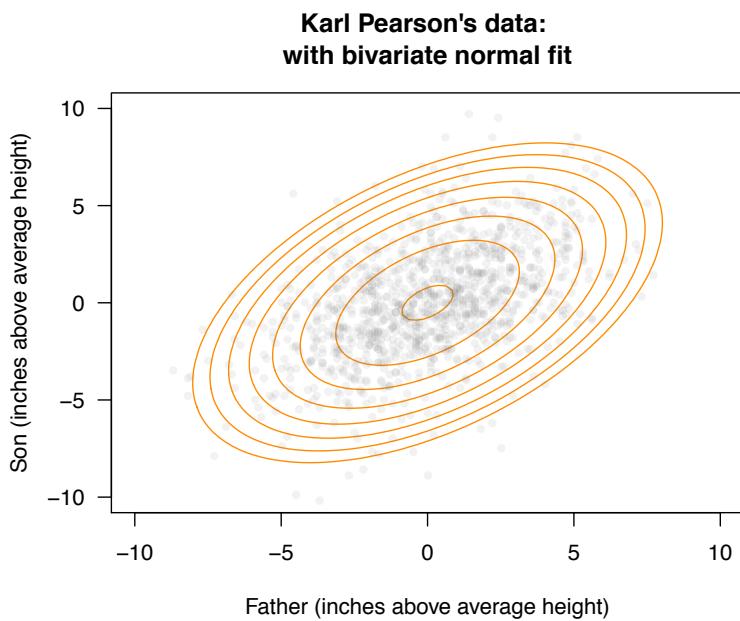


Figure 15.5: Best-fitting bivariate normal distribution for Karl Pearson's height data based on the sample standard deviations and sample correlation.

Written mathematically, we want to know the joint probability $P[X_1 \in (a, b), X_2 \in (c, d)]$. The two intervals (a, b) and (c, d) define a rectangle in the (X_1, X_2) plane (i.e. the “floor” of the 3D plot in Figure 15.4). To calculate this joint probability, we ask: what is the volume under the density function that sits above this rectangle? This generalizes the “area under the curve” interpretation of a density function for a single random variable.

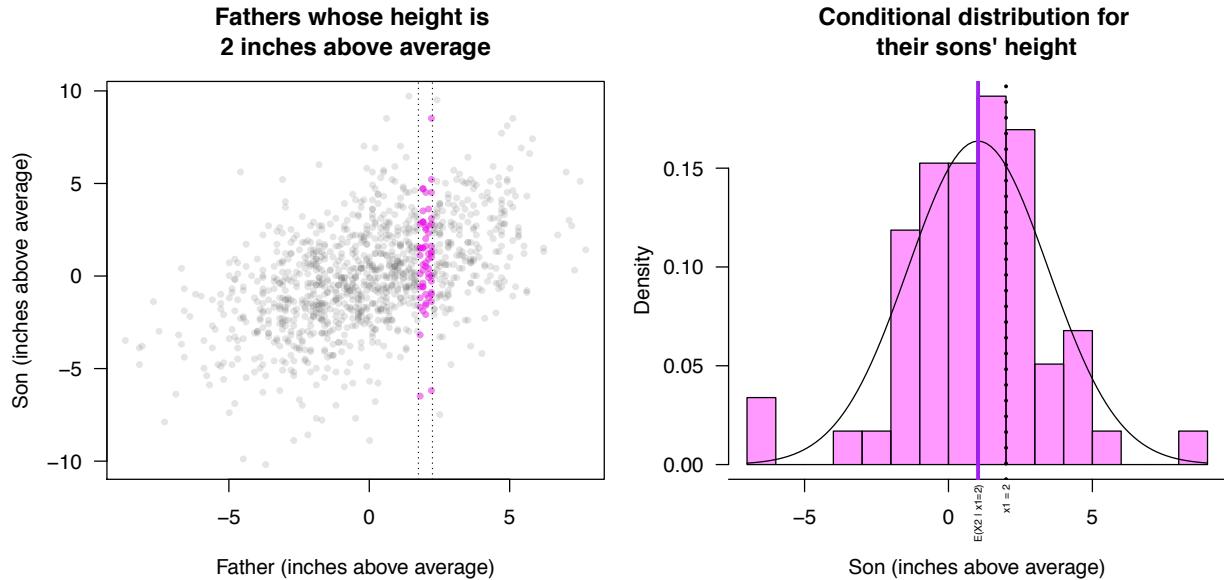
Figure 15.5 shows the best fitting bivariate normal distribution to the heights data:

$$(X_1, X_2) \sim N(\mu_1 = 0, \mu_2 = 0, \sigma_1 = 2.75, \sigma_2 = 2.82, \rho = 0.5).$$

Remember that both means are zero because we centered the data.

Conditional distributions for the bivariate normal

Take any pair of correlated random variables X_1 and X_2 . Because they are correlated, the value of one variable gives us information about the value of the second variable. To make this precise, say we fix the value of X_1 at some known value x_1 . What is the conditional probability distribution of X_2 , given that $X_1 = x_1$? In our heights example, this would be like asking: what is the dis-



tribution for the heights of sons (X_2) for fathers whose height is 2 inches above the mean ($X_1 = 2$)?

If X_1 and X_2 follow a bivariate normal distribution, i.e.

$$(X_1, X_2) \sim N(\mu_1, \mu_2, \sigma_1, \sigma_2, \rho),$$

then this question is easy to answer. It turns out that the conditional probability distribution $P(X_2 | X_1 = x_1)$ is an ordinary normal distribution, with mean and variance

$$E(X_2 | X_1 = x_1) = \mu_2 + \rho \cdot \frac{\sigma_2}{\sigma_1} \cdot (x_1 - \mu_1) \quad (15.1)$$

$$\text{var}(X_2 | X_1 = x_1) = \sigma_2^2 \cdot (1 - \rho^2), \quad (15.2)$$

where σ_1 , σ_2 , and ρ are the standard deviations of the two variables and their correlation, respectively. You'll notice that the conditional mean $E(X_2 | X_1 = x_1)$ is a linear function of x_1 , the assumed value for X_1 . Galton called this the regression line—that is, the line that describes where we should expect to find X_2 for a given value of X_1 .³

This fact brings us straight back to the concept of regression to the mean. Let's re-arrange Equation 15.1 to re-express the condi-

Figure 15.6: Left: father–son pairs where the father’s height is about 2 inches above average are highlighted in purple. Right: the histogram of the sons’ height, together with the conditional distribution $P(X_2 | X_1 = 2)$ predicted by the bivariate normal fit to the joint distribution for (X_1, X_2) . The sons’ average height, $E(X_2 | X_1 = 2)$ (purple line) is shrunk back towards 0 compared to the fathers’ height of 2 inches above average (black dotted line). This illustrates regression to the mean.

³ This use of the term “regression” is the origin of the phrase “linear regression” to describe the process of fitting lines to data. But keep in mind that linear regression (in the sense of fitting equations to data) actually predates Galton’s use of the term by almost 100 years. So while Galton’s reasoning using the bivariate normal distribution does provide the historical underpinnings for the *term* regression in the sense that we used it earlier in the book, it is not the origin for the idea of curve fitting.

tional mean in a slightly different way:

$$\frac{E(X_2 | X_1 = x_1) - \mu_2}{\sigma_2} = \rho \cdot \left(\frac{x_1 - \mu_1}{\sigma_1} \right). \quad (15.3)$$

The left-hand side asks: how many standard deviations is X_2 expected to be above (or below) its mean, given that $X_1 = x_1$? The right-hand side answers: the number of standard deviations that x_1 was above (or below) its mean, *discounted by a factor of ρ* . Because ρ can never exceed 1, we expect that X_2 will be “shrunk” a bit closer to its mean than x_1 was—and the weaker the correlation between the two variables, the stronger this shrinkage effect is.

Equation 15.3 therefore provides a formal mathematical description of regression to the mean. In the extreme case of $\rho = 1$, there is no regression to the mean at all.

Let’s return to the data on the heights of fathers and sons and use this result to measure the magnitude of the regression-to-mean effect. Specifically, let’s consider fathers whose heights are about 2 inches above average ($X_1 = 2$). Using Equation 15.1 together with the parameters of the best-fitting bivariate normal distribution from Figure 15.5, we find that:

$$E(X_2 | X_1 = 2) = \rho \cdot \frac{\sigma_2}{\sigma_1} \cdot 2 = 0.5 \cdot \frac{2.81}{2.75} \cdot 2 \approx 1.03.$$

That is, the sons should be about 1 inch taller than average for their generation (rather than 2 inches taller, as their fathers were).

Sure enough, as Figure 15.6 shows, this prediction is borne out. We have highlighted all the fathers in the data set who are approximately 2 inches above average (purple dots, left panel). On the right, we see a histogram for the height of their sons. This histogram shows us the conditional distribution $P(X_2 | X_1 = 2)$, together with the normal distribution whose mean and variance are calculated using the formulas for the conditional mean and variance in Equations 15.1 and 15.2. Given the small sample size ($n = 59$), the normal distribution looks like a good fit—in particular, it captures the regression-to-the-mean effect, correctly predicting that the conditional distribution will be centered around $X_2 = 1$.

Further applications of the bivariate normal

Example 1: regression to the mean in baseball

Regression to the mean is ubiquitous in professional sports. If you're a baseball fan, you may have heard of the "sophomore jinx":

A sophomore jinx is the popularly held belief that after a successful rookie season, a player in his second year will be jinxed and not have the same success. Most players suffer the "sophomore jinx" as scouting reports on the former rookie are now available and his weaknesses are known around the league.⁴

This idea comes up all the time in discussion among baseball players, coaches, and journalists:

Fresh off one of their best seasons in decades, the Cubs look primed to compete for a division title and more in 2016. As rookies in 2015, Kris Bryant, Addison Russell, Jorge Soler and Kyle Schwarber had significant roles in the success and next year, Cubs manager Joe Maddon is looking to help them avoid the dreaded sophomore jinx. "I think the sophomore jinx is all about the other team adjusting to you and then you don't adjust back," Maddon said Tuesday at the Winter Meetings. "So the point would be that we need to be prepared to adjust back. I think that's my definition of the sophomore jinx."⁵

The sophomore jinx—that outstanding rookies tend not to do quite as well in their second seasons—is indeed real. But it can be explained in terms of regression to the mean! Recall our definition of this phenomenon, from several pages ago: "Take any pair of correlated measurements. If one measurement is extreme, then the other measurement will tend to be closer to the average."

Let's apply this idea to baseball data. Say that X_1 is batting average of a baseball player last season, and that X_2 is that same player's batting average this season. Surely these variables are correlated, because more skillful players will have higher averages overall. But the correlation will be imperfect (less than one), because luck plays a role in a player's batting average, too.

Now focus on the players with the very best batting averages last year—that is, those where X_1 is the most extreme. Among players in this group, we should expect that X_2 will be less extreme overall than X_1 . Again, this isn't a claim about good performance last year *causing* worse performance this year. It's just that

⁴ http://www.baseball-reference.com/bullpen/Sophomore_jinx

⁵ "Focus for Joe Maddon: Avoiding 'sophomore jinx' with young Cubs." Matt Snyder, CBSsports.com, December 8, 2015.

Regression to the mean in repeated measurements: 2014 and 2015 baseball batting averages

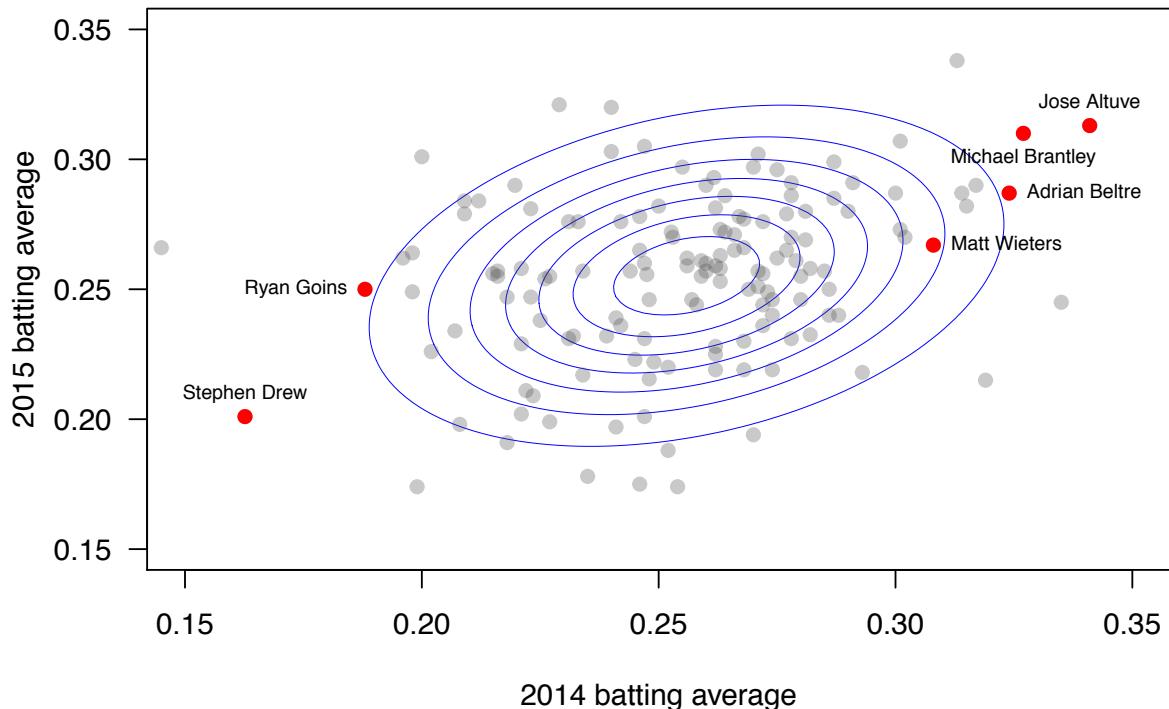
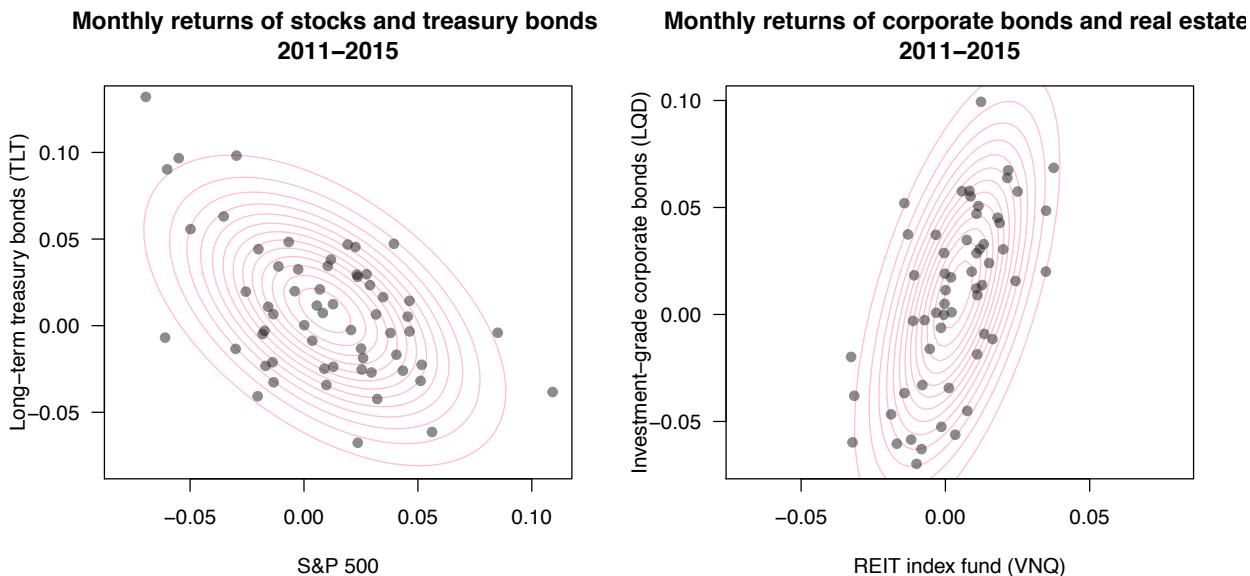


Figure 15.7: Baseball batting averages in the 2014 and 2015 seasons for all players with at least 100 at-bats in both years.

last year's very best performers were both lucky and good—and while they might still be good this year, they are no more likely to be lucky than any other group of baseball players.⁶

Figure 15.7 shows this phenomenon in action. Here we see the batting averages across the 2014 and 2015 baseball seasons for all players with at least 100 at-bats in both seasons. The figure highlights some of the very best and very worst performers in 2014. Sure enough, although 2014's best were still good in 2015, they weren't *as good* as they had been the previous year. Similarly, the very worst performers in 2014 were still not very good in 2015, but they weren't *as bad* as they'd been the previous year. This is another great example of regression to the mean.

⁶ Although it's possible Joe Maddon's theory of "not adjusting back" might be partially true, too, the mere existence of the "sophomore jinx" phenomenon certainly doesn't prove it.



Example 2: stocks and bonds.

The bivariate normal distribution is useful for more than simply describing regression to the mean. We can also use it as a building block for describing the joint probability distribution for two correlated random variables. As a final example, let's look at correlation between different pairs of financial assets.

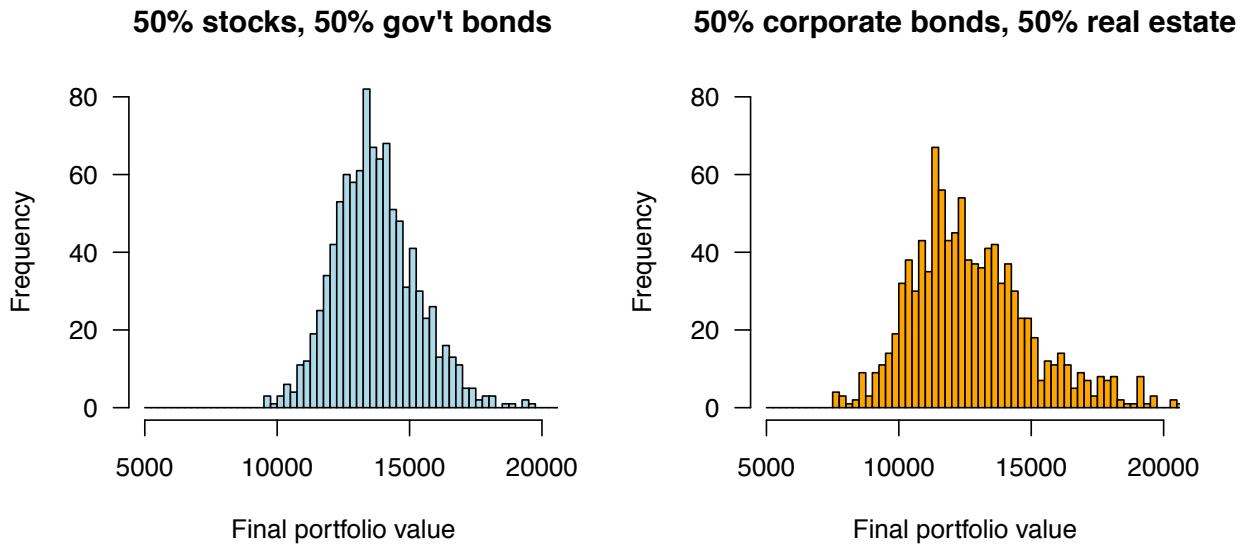
First, say that X_1 is the return on the S&P 500 index next month, while X_2 is the return on 30-year treasury bond next month.⁷ These two variables are almost sure to be correlated, although the magnitude and even the direction of this correlation has changed a lot over the last century. The conventional explanation for this is the so-called “flight to quality” effect: when stock prices plummet, investors get scared and pile their money into safer assets (like bonds), thereby driving up the price of those safer assets. This effect will typically produce a negative correlation between the returns of stocks and bonds held over a similar period.⁸ The left panel of Figure 15.8 shows the 2011–2015 monthly returns for long-term U.S. Treasury bonds versus the S&P 500 stock index, together with the best-fitting bivariate normal approximation.

Next, consider the right panel of Figure 15.8, which shows returns for real-estate investment trusts (X_1) and corporate bonds

Figure 15.8: Correlation between stocks and government bonds (left); correlation between corporate bonds and real estate (right).

⁷ Recall that a Treasury bond entailed lending money to the U.S. federal government and collecting interest in return.

⁸ This need not happen. In fact, a “flight to quality” effect can also produce a positive correlation between U.S. stocks and bonds. If you're interested in more detail, see [this short article](#) written by two economists at the Reserve Bank of Australia.



(X_2) . These assets' monthly returns were positively correlated, presumably because they both respond in similar ways to underlying macroeconomic forces.

How do these patterns of correlation affect the medium-term growth of a portfolio of mixed assets? To understand this, we'll run a Monte Carlo simulation where we chain together the results of 36 months (3 years) of investment. We'll compare two portfolios with an initial value of $W_0 = \$10,000$: a mix of stocks (X_1) and government bonds (X_2), versus a mix of real-estate (X_1) and corporate bonds (X_2). We'll let $W_{t,1}$ and $W_{t,2}$ denote the amount of money you have at step t in assets 1 and 2, respectively. Each 36-month period will be simulated as follows, starting with month $t = 1$ and ending with month $t = 36$.

- (1) Simulate a random return for month t from the bivariate normal probability model: $(X_{t1}, X_{t2}) \sim N(\mu_1, \mu_2, \sigma_1, \sigma_2, \rho)$.
- (2) Update the value of your investment to account for the period- t returns in each asset:

$$W_{t+1,i} = W_{t,i} \cdot (1 + X_{t,i})$$

for $i = 1, 2$.

Figure 15.9: Final value of 36-month investments in 50/50 mixes of: (1) stocks and government bonds (left), and (2) corporate bonds and real estate (right).

At every step, your current total wealth is $W_t = W_{t,1} + W_{t,2}$. For the sake of illustration, we'll assume that the initial allocation is a 50/50 mix, so that $W_{0,1} = W_{0,2} = \$5,000$.

Figure 15.9 shows the results of this simulation, assuming that returns following the bivariate normal distributions fit to the data in Figure 15.8. Clearly the 50/50 mix of stocks and government bonds is preferred under this scenario: it has both a higher return and a lower variance than the mix of corporate bonds and real-estate. In particular, in the second portfolio, the positive correlation between corporate bonds and real estate is especially troublesome. This results in a portfolio with far higher variance than necessary, because the ups and the downs tend to occur together.

Two major caveats here are: (1) the assumption that future returns will be statistically similar to past returns, and (2) that we can describe correlation among pairs of asset returns using a bivariate normal. Both of these assumptions can be challenged.

Therefore, it's better to think of simulations like these as a way of building scenarios under various assumptions about future performance, rather than as a firm guide to what it is likely to happen.

Functions of random variables (advanced topic)

A **VERY** important set of equations in probability theory describes what happens when you construct a new random variable as a linear combination of other random variables—that is, when

$$W = aX + bY + c$$

for some random variables X and Y and some constants a , b , and c .

The fundamental question here is: how does *joint* variation in X and Y (that is, correlation) influence the behavior of a random variable formed by adding X and Y together? To jump straight to the point, it turns out that

$$E(W) = aE(X) + bE(Y) + c \quad (15.4)$$

$$\text{var}(W) = a^2 \text{ var}(X) + b^2 \text{ var}(Y) + 2ab \text{ cov}(X, Y). \quad (15.5)$$

Why would you care about a linear combination of random variables? Consider a few examples:

- You know the distribution for X , the number of points a basketball team will score in one quarter of play. Then the

random variable describing the points the team will score in four quarters of play is $W = 4x$.

- A weather forecaster specifies a probability distribution for tomorrow's temperature in Celsius (a random variable, C). You can compute the moments of C , but you want to convert to Fahrenheit (another random variable, F). Then F is also a random variable, and is a linear combination of the one you already know: $F = (9/5)C + 32$.
- You know the joint distribution describing your uncertainty as to the future prices of two stocks X and Y . A portfolio of stocks is a linear combination of the two; if you buy 100 shares of the first and 200 of the second, then

$$W = 100X + 200Y$$

is a random variable describing the value of your portfolio.

- Your future grade on the statistics midterm is X_1 , and your future grade on the final is X_2 . You describe your uncertainty for these two random variables with some joint distribution. If the midterm counts 40% and the final 60%, then your final course grade is the random variable

$$C = 0.4X_1 + 0.6X_2,$$

a linear combination of your midterm and final grades.

- The speed of Rafael Nadal's slice serve is a random variable S_1 . The speed on his flat serve is S_2 . If Rafa hits 70% slice serves, his opponent should anticipate a random service speed equal to $0.7S_1 + 0.3S_2$.

In all five cases, it is useful to express the moments of the new random variable in terms of the moments of the original ones.

This saves you a lot of calculational headaches! We'll now go through the mathematics of deriving Equations (15.4) and (15.5).

Multiplying a random variable by a constant

Let's first examine what happens when you make a new random variable W by multiplying some other random variable X by a constant:

$$W = aX.$$

This expression means that, whenever $X = x$, we have $W = ax$. Therefore, if X takes on values x_1, \dots, x_n with probability p_1, \dots, p_n , then we know that

$$E(X) = \sum_{i=1}^n x_i p_i,$$

and so

$$E(W) = \sum_{i=1}^n ax_i p_i = a \sum_{i=1}^n x_i p_i = aE(X).$$

The constant a simply comes out in front of the original expected value. Mathematically speaking, this means that the expectation is a linear function of a random variable.

The variance of W can be calculated in the same way. By definition,

$$\text{var}(X) = \sum_{i=1}^n p_i \{x_i - E(X)\}^2.$$

Therefore,

$$\begin{aligned} \text{var}(W) &= \sum_{i=1}^n p_i \{ax_i - E(W)\}^2 \\ &= \sum_{i=1}^n p_i \{ax_i - aE(X)\}^2 \\ &= \sum_{i=1}^n p_i a^2 \{x_i - E(X)\}^2 \\ &= a^2 \sum_{i=1}^n p_i \{x_i - E(X)\}^2 \\ &= a^2 \text{var}(X) \end{aligned}$$

Now we have a factor of a^2 out front.

What if, in addition to multiplying X by a constant a , we also add another constant c to the result? This would give us

$$W = aX + c.$$

To calculate the moments of this random variable, revisit the above derivations on your own, adding in a constant term of c where appropriate. You'll soon convince yourself that

$$\begin{aligned} E(W) &= aE(X) + c \\ \text{var}(W) &= a^2 \text{var}(X). \end{aligned}$$

The constant simply gets added to the expected value, but doesn't change the variance at all.

A linear combination of two random variables

Suppose X and Y are two random variables, and we define a new random variable as $W = aX + bY$ for real numbers a and b . Then

$$\begin{aligned} E(W) &= \sum_{i=1}^n p_i \{ax_i + by_i\} \\ &= \sum_{i=1}^n p_i ax_i + \sum_{i=1}^n p_i by_i \\ &= a \sum_{i=1}^n p_i x_i + b \sum_{i=1}^n p_i y_i \\ &= aE(X) + b(E(Y)). \end{aligned}$$

Again, the expectation operator is linear.

The variance of W , however, takes a bit more algebra:

$$\begin{aligned} \text{var}(W) &= \sum_{i=1}^n p_i \left\{ [ax_i + by_i] - [aE(X) + bE(Y)] \right\}^2 \\ &= \sum_{i=1}^n p_i \left\{ [ax_i - aE(X)] + [by_i - bE(Y)] \right\}^2 \\ &= \sum_{i=1}^n p_i \left\{ [ax_i - aE(X)]^2 + [by_i - bE(Y)]^2 + 2ab[x_i - E(X)][y_i - E(Y)] \right\} \\ &= \sum_{i=1}^n p_i [ax_i - aE(X)]^2 + \sum_{i=1}^n p_i [by_i - bE(Y)]^2 + \sum_{i=1}^n p_i 2ab[x_i - E(X)][y_i - E(Y)] \\ &= \text{var}(aX) + \text{var}(bY) + 2abcov(X, Y) \\ &= a^2\text{var}(X) + b^2\text{var}(Y) + 2abcov(X, Y) \end{aligned}$$

The covariance of X and Y strongly influences the variance of their linear combination. If the covariance is positive, then the variance of the linear combination is *more than* the sum of the two individual variances. If the covariance is negative, then the variance of the linear combination is *less than* the sum of the two individual variances.

An example: portfolio choice under risk aversion

Let's revisit the portfolio-choice problem posed above. Say you plan to allocate half your money to one asset X , and the other half to some different asset Y . Look at Equations (15.4) and (15.5), which specify the expected value and variance of your portfolio in terms of the moments of the joint distribution for X and Y . If you are a risk-averse investor, would you prefer to hold two assets with a positive covariance or a negative covariance?

To make things concrete, let's imagine that the joint distribution for X and Y is given in the table at right. Each row is a possible joint outcome for X and Y : the first column lists the possible values of X ; the second, the possible values of Y ; and the third, the probabilities for each joint outcome. You should interpret the numbers in the X and Y columns as the value of \$1 at the end of the investment period—for example, after one year. If $X = 1.1$ after a year, then your holdings of that stock gained 10% in value.

Under this joint distribution, a single dollar invested in a portfolio with a 50/50 allocation between X and Y is a random variable W . This random variable has an expected value of 1.1 and variance

$$\begin{aligned}\text{var}(W) &= 0.5^2\text{var}(X) + 0.5^2\text{var}(Y) + 2 \cdot 0.5^2 \cdot \text{cov}(X, Y) \\ &= 0.5^2 \cdot 0.006 + 0.5^2 \cdot 0.006 + 2 \cdot 0.5^2 \cdot (0.002) \\ &= 0.004,\end{aligned}$$

for a standard deviation of $\sqrt{0.004}$, or about 6.3%.

What if, on the other hand, the asset returns were negatively correlated, as they are in the table at right? (Notice which entries have been switched, compared to the previous distribution.)

Under this new joint distribution, the expected value of \$1 invested in a 50/50 portfolio is still 1.1. But since the covariance between X and Y is now negative, the variance of the portfolio changes:

$$\begin{aligned}\text{var}(W) &= 0.5^2\text{var}(X) + 0.5^2\text{var}(Y) + 2 \cdot 0.5^2 \cdot \text{cov}(X, Y) \\ &= 0.5^2 \cdot 0.006 + 0.5^2 \cdot 0.006 + 2 \cdot 0.5^2 \cdot (-0.002) \\ &= 0.002,\end{aligned}$$

for a standard deviation of $\sqrt{0.002}$, or about 4.5%. Same expected return, but lower variance, and therefore more attractive to a risk-averse investor!

What's going on here? Intuitively, under the first portfolio, where X and Y are positively correlated, the bad days for X and Y tend to occur together. So do the good days. (When it rains, it pours; when it's sunny, it's 100 degrees.) But under the second portfolio, where X and Y are negatively correlated, the bad days and good days tend to cancel each other out. This results in a lower overall level of risk.

The morals of the story are:

1. Correlation creates extra variance.
2. Diversify! (Extra variance hurts your compounded rate of return.)

| x | y | $P(x, y)$ |
|-----|-----|-----------|
| 1.0 | 1.0 | 0.15 |
| 1.0 | 1.1 | 0.10 |
| 1.0 | 1.2 | 0.05 |
| 1.1 | 1.0 | 0.10 |
| 1.1 | 1.1 | 0.20 |
| 1.1 | 1.2 | 0.10 |
| 1.2 | 1.0 | 0.05 |
| 1.2 | 1.1 | 0.10 |
| 1.2 | 1.2 | 0.15 |

Table 15.1: Positive covariance.

| x | y | $P(x, y)$ |
|-----|-----|-----------|
| 1.0 | 1.0 | 0.05 |
| 1.0 | 1.1 | 0.10 |
| 1.0 | 1.2 | 0.15 |
| 1.1 | 1.0 | 0.10 |
| 1.1 | 1.1 | 0.20 |
| 1.1 | 1.2 | 0.10 |
| 1.2 | 1.0 | 0.15 |
| 1.2 | 1.1 | 0.10 |
| 1.2 | 1.2 | 0.05 |

Table 15.2: Negative covariance.

16

Generalized linear models

Binary responses

In many situations, we would like to predict the outcome of a binary event, given some relevant information:

- Given the pattern of word usage and punctuation in an e-mail, is it likely to be spam?
- Given the temperature, pressure, and cloud cover on Christmas Eve, is it likely to snow on Christmas Day?
- Given a person's credit history and income, is he or she likely to default on a mortgage loan?

In all of these cases, the y variable is the answer to a yes-or-no question. This is a bit different to the kinds of problems we've become used to seeing, where the response is a real number.

Nonetheless, we can still use regression for these problems.

Let's suppose, for simplicity's sake, that we have only one predictor x , and that we let $y_i = 1$ for a "yes" and $y_i = 0$ for a "no." One naïve way of forecasting y is simply to plunge ahead with the basic, one-variable regression equation:

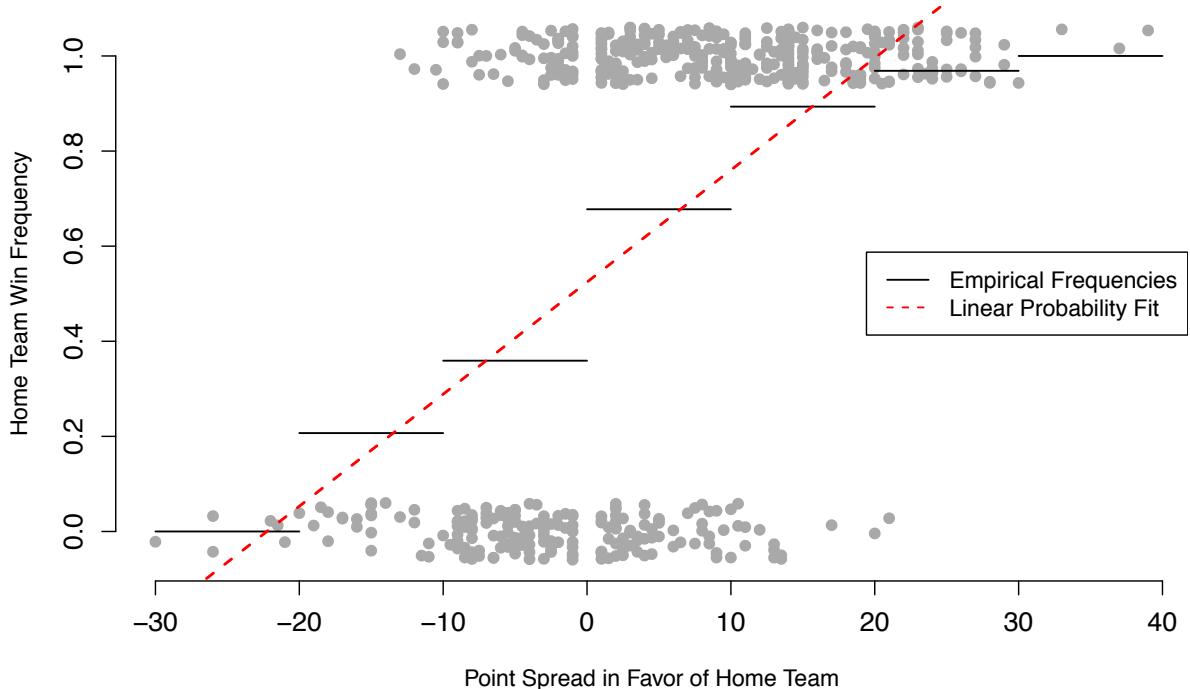
$$\hat{y}_i = E(y_i | x_i) = \beta_0 + \beta_1 x_i .$$

Since y_i can only take the values 0 or 1, the expected value of y_i is simply a weighted average of these two cases:

$$\begin{aligned} E(y_i | x_i) &= 1 \cdot P(y_i = 1 | x_i) + 0 \cdot P(y_i = 0 | x_i) \\ &= P(y_i = 1 | x_i) \end{aligned}$$

Therefore, the regression equation is just a linear model for the conditional probability that $y_i = 1$, given the predictor x_i :

$$P(y_i = 1 | x_i) = \beta_0 + \beta_1 x_i .$$



This model allows us to plug in some value of x_i and read off the forecasted probability of a “yes” answer to whatever yes-or-no question is being posed. It is often called the linear probability model, since the probability of a “yes” varies linearly with x .

Let’s try fitting it to some example data to understand how this kind of model behaves. In Table 16.1 on page 323, we see an excerpt of a data set on 553 men’s college-basketball games. Our y variable is whether the home team won ($y_i = 1$) or lost ($y_i = 0$). Our x variable is the Las Vegas “point spread” in favor of the home team. The spread indicates the betting market’s collective opinion about the home team’s expected margin of victory—or defeat, if the spread is negative. Large spreads indicate that one team is heavily favored to win. It is therefore natural to use the Vegas spread to predict the probability of a home-team victory in any particular game.

Figure 16.1 shows each of the 553 results in the data set. The

Figure 16.1: Win frequency versus point spread for 553 NCAA basketball games. Actual wins are plotted as 1’s and actual losses as zeros. Some artificial vertical jitter has been added to the 1’s and 0’s to allow the dots to be distinguished from one another.

home-team point spread is plotted on the x -axis, while the result of the game is plotted on the y -axis. A home-team win is plotted as a 1, and a loss as a 0. A bit of artificial vertical jitter has been added to the 1's and 0's, just so you can distinguish the individual dots.

The horizontal black lines indicate empirical win frequencies for point spreads in the given range. For example, home teams won about 65% of the time when they were favored by more than 0 points, but less than 10. Similarly, when home teams were 10–20 point underdogs, they won only about 20% of the time.

Finally, the dotted red line is the linear probability fit:

Coefficients:

| | Estimate | Std. Error | t value | Pr(> t) |
|--------------------------|----------|---------------------------|---------|------------|
| (Intercept) | 0.524435 | 0.019040 | 27.54 | <2e-16 *** |
| spread | 0.023566 | 0.001577 | 14.94 | <2e-16 *** |
| --- | | | | |
| Residual standard error: | 0.4038 | on 551 degrees of freedom | | |
| Multiple R-squared: | 0.2884 | | | |

This is the result of having regressed the binary y_i 's on the point spreads, simply treating the 1's and 0's as if they were real numbers. Under this model, our estimated regression equation is

$$E(y_i | x_i) = P(y_i = 1 | x_i) = 0.524 + 0.024 \cdot x_i.$$

Plug in an x , and read off the probability of a home-team victory. Here, we would expect the intercept to be 0.5, meaning that the home team should win exactly 50% of the time when the point spread is 0. Of course, because of sampling variability, the estimated intercept $\hat{\beta}_0$ isn't exactly 0.5. But it's certainly close—about 1 standard error away.

The linear probability model, however, has a serious flaw. Try plugging in $x_i = 21$ and see what happens:

$$P(y_i = 1 | x_i = 21) = 0.524 + 0.024 \cdot 21 = 1.028.$$

We get a probability larger than 1, which is clearly nonsensical. We could also get a probability less than zero by plugging in $x_1 = -23$:

$$P(y_i = 1 | x_i = -23) = 0.524 - 0.024 \cdot 23 = -.028.$$

The problem is that the straight-line fit does not respect the rule that probabilities must be numbers between 0 and 1. For many values of x_i , it gives results that aren't even mathematically legal.

| Game | Win | Spread |
|------|-----|--------|
| 1 | 0 | -7 |
| 2 | 1 | 7 |
| 3 | 1 | 17 |
| 4 | 0 | 9 |
| 5 | 1 | -2.5 |
| 6 | 0 | -9 |
| 7 | 1 | 10 |
| 8 | 1 | 18 |
| 9 | 1 | -7.5 |
| 10 | 0 | -8 |
| ⋮ | | |
| 552 | 1 | -4.5 |
| 553 | 1 | -3 |

Table 16.1: An excerpt from a data set on 553 NCAA basketball games. "Win" is coded 1 if the home team won the game, and 0 otherwise. "Spread" is the Las Vegas point spread in favor of the home team (at tipoff). Negative point spreads indicate where the visiting team was favored.

Link functions and generalized linear models

THE PROBLEM can be summarized as follows. The right-hand side of the regression equation, $\beta_0 + \beta_1 x_i$, can be any real number between $-\infty$ and ∞ . But the left-hand side, $P(y_i = 1 | x_i)$, must be between 0 and 1. Therefore, we need some transformation g that takes an unconstrained number from the right-hand side, and maps it to a constrained number on the left-hand side:

$$P(y_i | x_i) = g(\beta_0 + \beta_1 x_i).$$

Such a function g is called a *link function*; a model that incorporates such a link function is called a *generalized linear model*, or GLM. The part inside the parentheses $(\beta_0 + \beta_1 x_i)$ is called the *linear predictor*.

We use link functions and generalized linear models in most situations where we are trying to predict a number that is, for whatever reason, constrained. Here, we're dealing with probabilities, which are constrained to be no smaller than 0 and no larger than 1. Therefore, the function g must map real numbers on $(-\infty, \infty)$ to numbers on $(0, 1)$. It must therefore be shaped a bit like a flattened letter "S," approaching zero for large negative values of the linear predictor, and approaching 1 for large positive values.

Figure 16.2 contains the most common example of such a link function. This is called the *logistic link*, which gives rise to the *logistic regression model*:

$$P(y_i = 1 | x_i) = g(\beta_0 + \beta_1 x_i) = \frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}}.$$

Think of this as just one more transformation, like the logarithm or powers of some predictor x . The only difference is that, in this case, the transformation gets applied to the whole linear predictor at once. The logistic regression model is often called the logit model for short.¹

With a little bit of algebra, it is also possible to isolate the linear predictor $\beta_0 + \beta_1 x_i$ on one side of the equation. If we let p_i denote

¹ The "g" in "logit" is pronounced softly, like in "gentle" or "magic."

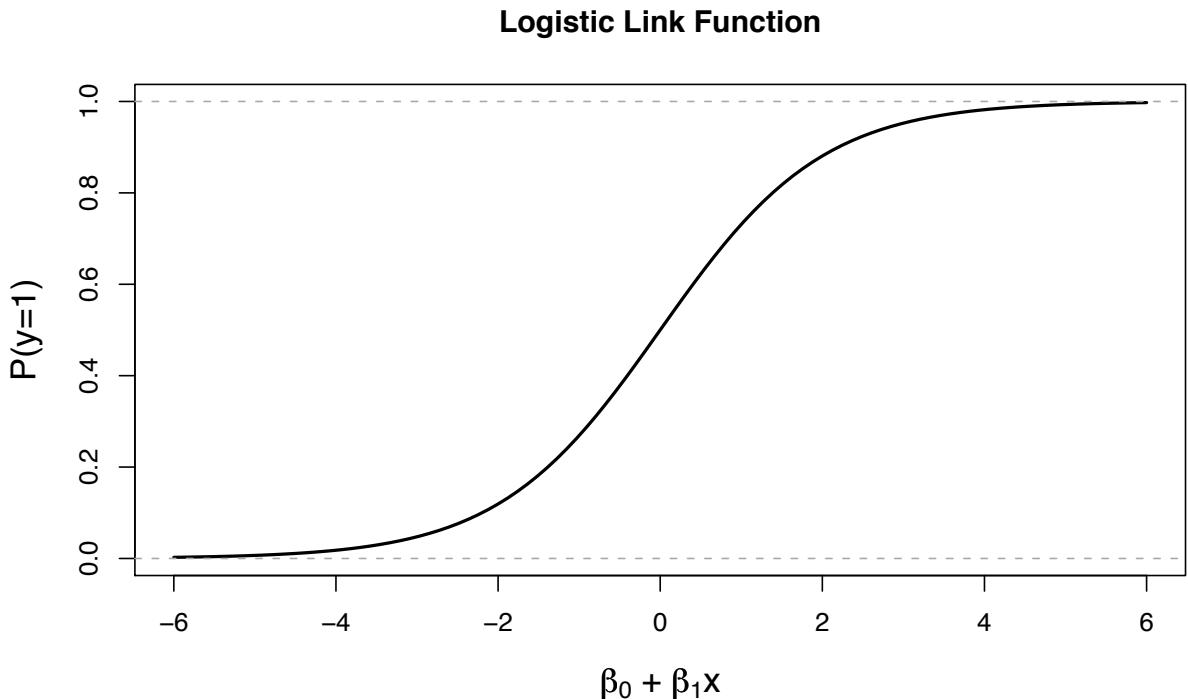
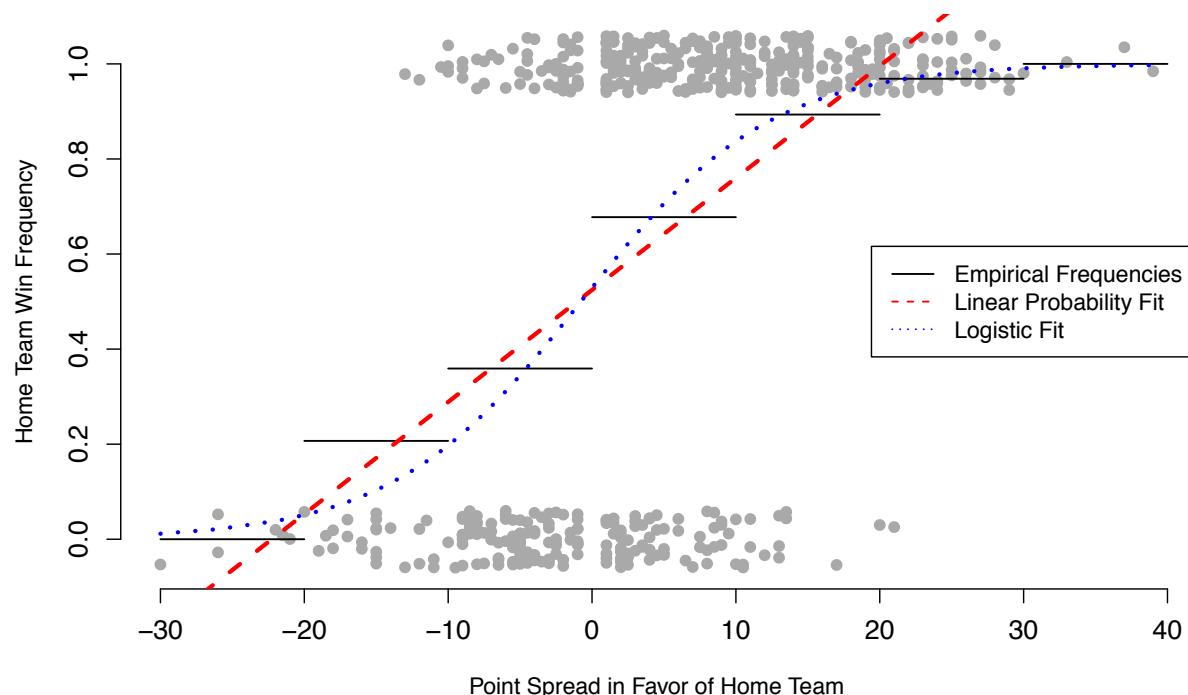


Figure 16.2: The logistic link function.

the probability that $y_i = 1$, given x_i , then

$$\begin{aligned}
 p_i &= \frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}} \\
 p_i + p_i e^{\beta_0 + \beta_1 x_i} &= e^{\beta_0 + \beta_1 x_i} \\
 p_i &= (1 - p_i) e^{\beta_0 + \beta_1 x_i} \\
 \log\left(\frac{p_i}{1 - p_i}\right) &= \beta_0 + \beta_1 x_i
 \end{aligned}$$

Since $p_i = P(y_i = 1 | x_i)$, we know that $1 - p_i = P(y_i = 0 | x_i)$. Therefore, the ratio $p_i/(1 - p_i)$ is the odds in favor of the event $y_i = 1$, given the predictor x_i . Thus the linear predictor $\beta_0 + \beta_1 x_i$ (on the right-hand side of the last equation) gives us the logarithm of the odds in favor of success ($y_i = 1$), on the left-hand side of the last equation.



The logistic regression fit for the point-spread data

Let's return briefly to the data on point spreads in NCAA basketball games. The figure above compares the logistic model to the linear-probability model. The logistic regression fit ($\hat{\beta}_0 = 0.117$, $\hat{\beta}_1 = 0.152$) eliminates the undesirable behavior of the linear model, and ensures that all forecasted probabilities are between 0 and 1. Note the clearly non-linear behavior of the dotted blue curve. Instead of fitting a straight line to the empirical success frequencies, we have fit an S-shape.

Interpreting the coefficients

Interpreting the coefficients in a logistic regression requires a bit of algebra. For the sake of simplicity, imagine a data set with only a single regressor x_i that can take the values 0 or 1 (a dummy variable). Perhaps, for example, x_i denotes whether someone received

Figure 16.3: Win frequency versus point spread for 553 NCAA basketball games. Actual wins are plotted as 1's and actual losses as zeros. Some artificial vertical jitter has been added to the 1's and 0's to allow the dots to be distinguished from one another.

the new treatment (as opposed to the control) in a clinical trial.

For this hypothetical case, let's consider the ratio of two quantities: the odds of success for person i with $x_i = 1$, versus the odds of success for person j with $x_j = 0$. Denote this ratio by R_{ij} . We can write this as

$$\begin{aligned} R_{ij} &= \frac{O_i}{O_j} \\ &= \frac{\exp\{\log(O_i)\}}{\exp\{\log(O_j)\}} \\ &= \frac{\exp\{\beta_0 + \beta_1 \cdot 1\}}{\exp\{\beta_0 + \beta_1 \cdot 0\}} \\ &= \exp\{\beta_0 + \beta_1 - \beta_0 - 0\} \\ &= \exp(\beta_1). \end{aligned}$$

Therefore, we can interpret the quantity e^{β_1} as an *odds ratio*. Since $R_{ij} = O_i/O_j$, we can also write this as:

$$O_i = e^{\beta_1} \cdot O_j.$$

In words: if we start with $x = 0$ and move to $x = 1$, our odds of success ($y = 1$) will change by a multiplicative factor of e^{β_1} .

For this reason, we usually refer to the exponentiated coefficient e^{β_j} as the odds ratio associated with predictor j .

Advanced topic: estimating the parameters of the logistic regression model

In previous chapters we learned how to estimate the parameters of a linear regression model using the least-squares criterion. This involved choosing values of the regression parameters to minimize the quantity

$$\sum_{i=1}^n (y_i - \hat{y}_i)^2,$$

where \hat{y}_i is the value for y_i predicted by the regression equation.

In logistic regression, the analogue of least-squares is Gauss's principle of maximum likelihood, which we introduced when discussing the normal linear regression model. The idea here is to choose values for β_0 and β_1 that make the observed patterns of 1's and 0's look as likely as possible.

To understand how this works, observe the following two facts:

- If $y_i = 1$, then we have observed an event that occurred with probability $P(y_i = 1 \mid x_i)$. Under the logistic-regression

model, we can write this probability as

$$P(y_i = 1 \mid x_i) = \frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}}$$

- If $y_i = 0$, then we have observed an event that occurred with probability $P(y_i = 0 \mid x_i) = 1 - P(y_i = 1 \mid x_i)$. Under the logistic regression model, we can write this probability as

$$1 - P(y_i = 1 \mid x_i) = 1 - \frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}}$$

Since all of the individual 1's and 0's are independent, given the parameters β_0 and β_1 , the joint probability of all the 1's and 0's is the product of their individual probabilities. We can write this as:

$$P(y_1, \dots, y_n) = \prod_{i:y_i=1} \left(\frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}} \right) \cdot \prod_{i:y_i=0} \left(1 - \frac{e^{\beta_0 + \beta_1 x_i}}{1 + e^{\beta_0 + \beta_1 x_i}} \right).$$

This expression is our *likelihood*: the joint probability of all our data points, given some particular choice of the model parameters.² The logic of maximum likelihood is to choose values for β_0 and β_1 such that $P(y_1, \dots, y_n)$ is as large as possible. We denote these choices by $\hat{\beta}_0$ and $\hat{\beta}_1$. These are called the *maximum-likelihood estimates* (MLE's) for the logistic regression model.

This likelihood is a difficult expression to maximize by hand (i.e. using calculus and algebra). Luckily, most major statistical software packages have built-in routines for fitting logistic-regression models, absolving you of the need to do any difficult analytical work.

The same is true when we move to multiple regression, when we have p predictors rather than just one. In this case, the logistic-regression model says that

$$P(y_i = 1 \mid x_{i1}, \dots, x_{ip}) = g(\beta_0 + \beta_1 x_i) = \frac{e^{\psi_{ij}}}{1 + e^{\psi_{ij}}}, \quad \psi_{ij} = \beta_0 + \sum_{j=1}^p \beta_j x_{ij}$$

where ψ_{ij} is the linear predictor for observation i .

² Remember that the big \prod signs mean "product," just like \sum means "sum." The first product is for the observations where y_i was a 1, and the second product is for the observations where y_i was a 0.

Extensions to the basic logit model

The ordinal logit model

We can modify the logistic regression model to handle ordinal responses. The hallmark of ordinal variables is that they are measured on a scale that can't easily be associated with a numerical

magnitude, but that does imply an ordering: employee evaluations, survey responses, bond ratings, and so forth.

There are several varieties of ordinal logit model. Here we consider the *proportional-odds* model, which is most easily understood as a family of related logistic regression models. Label the categories as $1, \dots, K$, ordered in the obvious way. Consider the probability $c_{ik} = P(y_i \leq k)$: the probability that the outcome for the i th case falls in category k or any lower category. (We call it c_{ik} because it is a cumulative probability of events at least as “low” as k .) The proportional-odds logit model assumes that the logit transform of c_{ik} is a linear function of predictors:

$$\text{logit}(c_{ik}) = \log \left(\frac{c_{ik}}{1 - c_{ik}} \right) = \eta_k + \beta_1 x_{i1} + \dots + \beta_p x_{ip}.$$

Crucially, this relationship is assumed to hold for all categories at once. Because $c_{iK} = 1$ for the highest category K , we have specified $K - 1$ separate binary logit models that all share the same predictors x_j and the same coefficients β_j . The only thing that differs among the models are the intercepts η_k ; these are commonly referred to as the *cutpoints*. Since the log odds differ only by an additive constant for different categories, the odds differ by a multiplicative factor—thus the term “proportional odds.”

To interpret the ordinal-logit model, I find it easiest to re-express individual fitted values in terms of covariate-specific category probabilities $w_{ik} = P(y_i = k)$:

$$w_{ik} = P(y_i \leq k) - P(y_i \leq k - 1) = c_{ik} - c_{i,k-1},$$

with the convention that $c_{i0} = 0$. Good software makes it fairly painless to do this.

The multinomial logit model

Another generalization of the binary logit model is the multinomial logit model. This is intended for describing *unordered* categorical responses: PC/Mac/Linux, Ford/Toyota/Chevy, plane/train/automobile, and so forth. Without a natural ordering to the categories, the quantity $P(y_i \leq k)$ ceases to be meaningful, and we must take a different approach.

Suppose there are K possible outcomes (“choices”), again labeled as $1, \dots, K$ (but without the implied ordering). As before, let $w_{ik} = P(y_i = k)$. For every observation, and for each of the K

choices, we imagine that there is a linear predictor ψ_{ik} that measures the preference of subject i for choice k . Intuitively, the higher ψ_{ik} , the more likely that $y_i = k$.

The specific mathematical relationship between the linear predictors and the probabilities w_{ik} is given the multinomial logit transform:³

$$\begin{aligned} w_{ik} &= \frac{\exp(\psi_{ik})}{\sum_{l=1}^K \exp(\psi_{il})} \\ \psi_{ik} &= \beta_0^{(k)} + \beta_1^{(k)} x_{i1} + \cdots + \beta_p^{(k)} x_{ip}. \end{aligned}$$

Each category gets its own set of coefficients, but the same set of predictors x_1 through x_p .

There is one minor issue here. With a bit of algebra, you could convince yourself that adding a constant factor to each ψ_{ik} would not change the resulting probabilities w_{ik} , as this factor would cancel from both the numerator and denominator of the above expression. To fix this indeterminacy, we choose one of the categories (usually the first or last) to be the reference category, and set its coefficients equal to zero.

³ Some people, usually computer scientists, will refer to this as the softmax function.

Models for count outcomes

The Poisson model. For modeling event-count data (photons, mortgage defaults in a ZIP code, heart attacks in a town), a useful place to start is the Poisson distribution. The key feature of counts is that they must be non-negative integers. Like the case of logistic regression, where probabilities had to live between 0 and 1, this restriction creates some challenges that take us beyond ordinary least squares.

The Poisson distribution is parametrized by a rate parameter, often written as λ . Let k denote an integer, and y_i denote the event count for subject i . In a Poisson model, we assume that

$$P(y_i = k) = \frac{\lambda_i^k}{k!} e^{-\lambda_i},$$

and we wish to model λ_i in terms of covariates. Because the rate parameter of the Poisson cannot be negative, we must employ the same device of a link function to relate λ_i to covariates. By far the most common is the (natural) log link:

$$\log \lambda_i = \beta_0 + \beta_1 x_{i1} + \cdots + \beta_p x_{ip},$$

or equivalently,

$$\lambda_i = \exp\{\beta_0 + \beta_1 x_{i1} + \cdots + \beta_p x_{ip}\}.$$

As with the case of logistic regression, the model is fit via maximum-likelihood.

Interpreting the coefficients. Because we are fitting a model on the log-rate scale, additive changes to an x variable are associated with multiplicative changes in the y variable. As before, let's consider the ratio of two quantities: the rate of events for person i with $x_1 = x^* + 1$, versus the rate of events for person j with $x_1 = x^*$. Let's further imagine that all other covariates are held constant at values x_2 to x_p , respectively. This implies that the only difference between subjects i and j is a one-unit difference in the first predictor, x_1 .

We can write their ratio of rates as

$$\begin{aligned} R_{ij} &= \frac{\lambda_i}{\lambda_j} \\ &= \frac{\exp\{\beta_0 + \beta_1 \cdot (x^* + 1) + \beta_2 x_2 + \cdots + \beta_p x_p\}}{\exp\{\beta_0 + \beta_1 \cdot x^* + \beta_2 x_2 + \cdots + \beta_p x_p\}} \\ &= \exp\{\beta_1(x^* + 1 - x^*)\} \\ &= \exp(\beta_1). \end{aligned}$$

Thus person i experiences events events e^{β_1} times as frequently as person j .

Overdispersion. For most data sets outside of particle physics, the Poisson assumption is usually one of convenience. Like the normal distribution, it is familiar and easy to work with. It also has teeth, and may bite if used improperly. One crucial feature of the Poisson is that its mean and variance are equal: that is, if $y_i \sim \text{Pois}(\lambda_i)$, then the expected value of y_i is λ_i , and the standard deviation of y_i is $\sqrt{\lambda_i}$. (Since λ_i depends on covariates, we should really be calling these the *conditional* expected value and standard deviation.)

As a practical matter, this means that if your data satisfy the Poisson assumption, then roughly 95% of observations should fall within $\pm 2\sqrt{\lambda_i}$ of their conditional mean λ_i . This is quite narrow, and many (if not most) data sets exhibit significantly more variability about their mean. If the conditional variance exceeds the

conditional mean, the data exhibits *overdispersion with respect to the Poisson*, or just *overdispersion* for short.

Overdispersion can really mess with your standard errors. In other words, if you use (i.e. let your software use) the Poisson assumption to calculate error bars, but your data are overdispersed, then you will end up overstating your confidence in the model coefficients. Sometimes the effect is dramatic, meaning that the blind use of the Poisson assumption is a recipe for trouble.

There are three common strategies for handling overdispersion:

- (1) Use a quasi-likelihood approach (“family=quasipoisson” in R’s `glm` function);
- (2) Fit a different count-data model, such as the negative binomial or Poisson-lognormal, that can accommodate overdispersion;
- (3) Fit a hierarchical model.

Alas, these topics are for a more advanced treatment of generalized linear models.