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QUANTITATIVE METHODOLOGY

AN INTRODUCTION

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Preface

The principal goal of this class is to impart the fundamentals of quantitative research. Our focus will be on **drawing causal conclusions** from data—when are causal conclusions licensed, what are typical pitfalls when drawing causal conclusions, and how can you design and optimise studies that avoid these pitfalls? Throughout, it is imperative that you not only understand the recommendations given in this booklet but also the **logic** behind them. There are two reasons for this.

First, you need to know when the recommendations apply and when they don't. You might get away with just memorising the recommendations and their scope for now. But in a couple of months, you're bound to apply them where they don't make any sense. Understand the logic behind the recommendations, and you'll be better able to weigh your options.

Second, many researchers in the social sciences—even seasoned ones—operate on rules of thumb, so *they* inevitably end up applying recommendations they've picked up somewhere in situations where they don't apply. You need to be able to make an informed judgement about the research carried out by others and cogently argue for this judgement. A simple *I took this class that taught me that you shouldn't control for colliders* won't do. (You'll learn about colliders and why you shouldn't control for them soon enough.)

In this class, we'll cover the contents of this lecture script. In addition, we'll spend two sessions on questionnaire design. Furthermore, in the homework assignments, you'll learn how to visualise data using state-of-the-art software. These graphing assignments are available from `https://janhove.github.io/graphs`.

I occasionally refer to some blog entries I wrote. These links are clickable in the PDF version of this booklet, but in case you're reading this from paper, all blog entries can be found at `https://janhove.github.io`.

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Association and causality

1.1 Two examples

Below are two examples of empirical findings along with conclusions that someone could draw from them. Answer the following questions for both of these examples.

1. Does the conclusion follow logically from the finding? If not, what are some plausible alternative explanations for the finding?
2. Which additional finding would strengthen the conclusions?
3. Which additional finding would call the conclusion into question?

Example 1.1 (Receptive multilingualism in Scandinavia)**.** When talking their respective native languages, Danes understand Swedes better than the other way around. Furthermore, Danes like Swedish better than Swedes do Danish (e.g., Delsing & Lundin Åkesson, 2005).

Conclusion: Danes understand Swedes better than the other way around because they like the language better. ◇

The diamond symbol signals where an example, exercise, remark, definition and such like ends.

Example 1.2 (Content and language integrated learning)**.** Pupils in *Content and Language Integrated Learning* (CLIL) programmes in Andalusia perform better on English proficiency tests than other Andalusian pupils (Lorenzo et al., 2010).

Conclusion: Taking CLIL classes improves pupils' English proficiency. ◇

In both examples, an **association** of some sort is found in the data, and a **causal explanation** for this association is put forward: Not only do Danes both understand and like Swedish better than Swedes do Danish (association), it's suggested that one reason why they understand the other language better is that they like it better (explanation). Similarly, not only do CLIL pupils in Andalusia outperform non-CLIL pupils (association), it's suggested that they outperform them *because* of the CLIL programme (explanation).

Uncovering associations and drawing causal conclusions from them is a key goal in empirical research. But it's also fraught with difficulty: after a moment's thought, you'll often be able to come up with alternative explanations for the findings. To the extent that there exist more, and more plausible, alternative explanations, the causal explanation proferred becomes more tenuous: The causal claim may still be correct, but in the presence of competing explanations, it can't be *shown* to be correct—that is, there isn't much **evidence** for the claim. A key goal when designing an empirical study is to reduce the number and the plausibility of such alternative explanations.

1.2 Definitions

Definition 1.3 (Association)**.** Two factors (or variables)[1] are **associated** if knowing the value of one factor can help you hazard a more educated guess about the value of the other factor.[2] ♢

That's a mouthful, but convince yourself that the following are examples of associations:

- a person's size in centimetres and their size in inches;
- the time of day and the temperature outside;
- a person's height and their weight;
- a person's shoesize and the size of their vocabulary in their native language;
- a person's age and their father's age;
- a person's nationality and the colour of their eyes.

Five remarks are in order:

- Associations work in both directions: knowing the time of day allows you to venture a more educated guess about the temperature outside than not knowing it, but also vice versa.
- Associations needn't be linear (e.g., the relation between weight and height levels off after a certain weight).
- Associations needn't be monotonic, e.g., the relationship between the two variables can go up and then down again (as in the time of day/temperature example).
- Associations needn't be perfect (e.g., there's a lot of variation

[1] I use these terms interchangeably. Sometimes, factors are constant rather than variable in the context of a study, but let's save our pedantic inclinations for other things.

[2] The more rigorous mathematical definition is that we call two random variables X, Y associated if there exist sets A, B such that $\mathbb{P}(X \in A, Y \in B) \neq \mathbb{P}(X \in A)\mathbb{P}(Y \in B)$. For the purposes of this class, though, a conceptual understanding along the lines of the more informal definition in the previous paragraph is sufficient.

about the general trend for taller people to be heavier).

- Associations can be found between variables that aren't typically expressed numerically (e.g., eye colour and nationality).

Typical examples of associations in research are mean differences between groups and correlations.[3]

As for **causality**, a common-sense understanding will be sufficient for our purposes. But when in doubt, you can turn to the following broad definition:

Definition 1.4 (Causality)**.** "We say that there is a *causal relationship* between [two variables] D and Y in a population if and only if there is at least one unit in that population for which intervening in the world to change D will change Y . . . ." (Keele et al., 2019, p. 3) ♢

Three remarks are in order:

- Saying that D causally influences Y doesn't imply that D *alone* causally influences Y. (You can get lung cancer from smoking, but also from exposure to radon, air polution or just genetic bad luck.)
- Saying that D causally influences Y doesn't mean that changing D will result in a change in Y for *all* members of the population. (Some non-smokers get lung cancer, and not all smokers get it.)
- Saying that D causally influences Y doesn't imply that changing D will result in a change in Y in all situations. (Smoldering cigarette stubs cause forest fires, but only during droughts. By the same token, droughts cause forest fires, but these need a spark to get started.)

1.3 *Visualising causality: directed acyclic graphs (DAGS)*

1.3.1 *Why?*

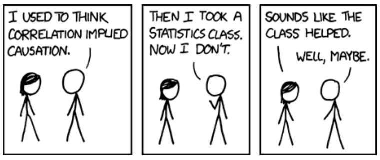

I USED TO THINK CORRELATION IMPLIED CAUSATION.
THEN I TOOK A STATISTICS CLASS. NOW I DON'T.
SOUNDS LIKE THE CLASS HELPED.
WELL, MAYBE.

Figure 1.1: Source: https://xkcd.com/552.

Research would be pretty easy if you could safely conclude that a causal link existed between two variables any time you observed an association between them. Fortunately for teachers of methodology courses who'd be on the dole otherwise, this isn't the case. But simply parrotting back *Correlation is not causation* isn't too helpful. To help us figure out how associations between two variables can arise in the absence of a causal link between them, we turn to **directed acylic graphs** (DAGS).

Definition 1.5 (Graphs)**. Graphs** are mathematical objects in which nodes (also called vertices) can be connected by edges. In **directed** graphs, these edges point from one node to another, i.e., they're arrows. If, in a directed graph, it is impossible to start from some node and end up in the same node by following edges, then that graph is also **acyclic**.

[3] You'll also often see the words 'association' and 'correlation' used interchangeably. I prefer to use 'association' as the hypernym and reserve 'correlation' for a specific type of association. See Chapter 8.

A **path** is a sequence of distinct nodes that are linked by edges; the direction of the edges does not matter. A **directed path** from a node X to a node Y is a path in which you start at X and end at Y by following edges in the direction they are pointing in (i.e., $X \to W \to \cdots \to Z \to Y$).

If, in a directed graph, there exists an edge from a node X to a node Y (i.e., $X \to Y$), then we call X a **parent** of Y and Y a **child** of X. If there exists a directed path from a node X to a node Y, then we call X an **ancestor** of Y and Y a **descendant** of X. In particular, parents are ancestors, and children are descendants. ♢

Graphs are studied in mathematics and computer science for sundry purposes; here, we will use DAGs as a tool for visually representing the causal links between the factors at play in a study.

As we'll see below, when DAGs are used to represent causal links, they are subject to a number of rules that may appear cumbersome at first. However, when DAGs are properly specified, they allow researchers to figure out which factors they *should* control for, which factors they *can but needn't* control for and which factors they *must not* control for. Moreover, DAGs are useful for learning how associations in empirical data can occur both in the presence and in the absence of causal links between the variables of interest.

1.3.2 *Some examples*

Before spelling out the rules for drawing DAGs, let's look at a couple of possible DAGs for the Andalusian CLIL study.

Figure 1.2 is the simplest of DAGs. It represents the assumption that there is a direct causal influence from the **treatment** variable (CLIL) on the **outcome** variable. These variables are represented by nodes. There exists a directed edge (i.e., an arrow) between them that shows the assumed direction of the causal link.

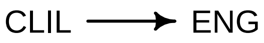


Figure 1.2: DAG representing a causal influence of CLIL on English proficiency (ENG).

The pupils' English proficiency won't be affected by their taking CLIL classes or not *alone* but by a host of other unobserved factors as well. In Figure 1.3, the unobserved factors are conveniently bundled and represented as 'U'. The U is circled to make it clear that these factors were not observed or measured. While this convention isn't universal, it's useful and we'll adopt it here.

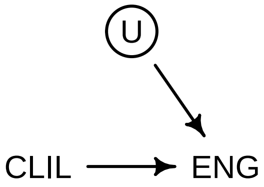


Figure 1.3: DAG representing a causal influence of CLIL and of unobserved factors on English proficiency.

Important: If we don't draw an arrow between U and CLIL, this means that we assume that there is *no* direct causal relationship between these two factors. But presumably, some unobserved factors will also account for why some pupils are enrolled in CLIL classes and others aren't; see Figure 1.4. As we'll discuss later, these unobserved factors, some of which may affect both the 'treatment' (CLIL) and the 'outcome' (English proficiency), **confound** the causal link of interest.

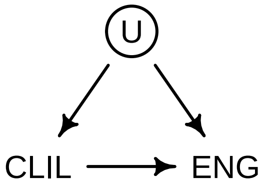


Figure 1.4: Unobserved factors as confounders (1).

Figure 1.5 on the facing page also features the unobserved factors as possible confounders, but this time there is no edge between

CLIL and ENG. Such a DAG can be useful for playing devil's advocate: We assume that there is no causal link between CLIL and English proficiency and subsequently use the DAG to deduce if it is possible that CLIL and English proficiency are nonetheless associated. If so, the mere presence of an association between CLIL and English proficiency does not imply that there is some causal link between them.

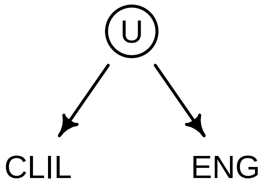


Figure 1.5: Unobserved factors as confounders (2).

1.3.3 *Rules for drawing* DAGs

1. The direction of the arrows shows the direction of the assumed causality (hence *directed*).
2. Bidirectional arrows are forbidden, i.e., no $A \leftrightarrow B$.
3. You're not allowed to draw graphs where you can end up at the same place where you started by just following arrows (hence *acyclic*). For instance, you're not allowed to draw a DAG like Figure 1.6. Mutually influencing factors can be represented in a DAG, however, but you need to break down the temporal structure. Figure 1.7 shows how you can break down the temporal structure implicit in Figure 1.6 to produce a legal DAG.

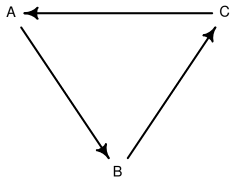


Figure 1.6: Not a DAG: A, B and C are allowed to influence themselves, making the graph cyclic rather than acyclic.

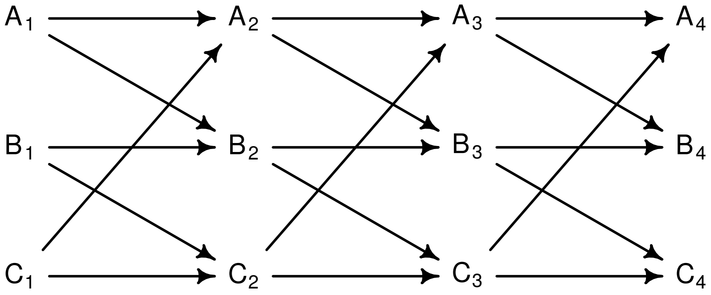


Figure 1.7: Reciprocal influences can be represented legally in a DAG if you break down the temporal structure. A_1, A_2, A_3 and A_4 represent the same variable measured at four points in time. The value of this variable at a given point in time is determined in part by its value at the previous point in time (e.g., A_3 is influenced by A_2) as well as by the value of another variable at the previous point in time (e.g., C_2 influences A_3).

4. Unobserved factors can, and often should, be drawn. By convention, we draw a circle around them to make it clear that they are not directly observed.
5. "DAGs insistently redirect the analyst's attention to justifying what arrows do not exist. Present arrows represent the analyst's ignorance. Missing arrows, by contrast, represent definitive claims of knowledge." (Elwert, 2013, p. 248)
6. A factor that isn't of interest and that only affects one factor already in the DAG and/or is affected by only one factor already in the DAG doesn't have to be drawn for you to be able to derive correct conclusions from the DAG. For instance, the U in Figure 1.3 doesn't have to be drawn (it only affects one factor that was already in the DAG). However, the U in Figure 1.4 *does* have to be drawn since it affects *two* factors that were already in the DAG. That said, it can be difficult to decide if a variable should be included in a DAG or not, and we shouldn't let perfect be the enemy of good. The purpose of DAGs is less to perfectly capture

the true causal structure than making explicit one's imperfect assumptions.

Note that DAGs don't specify *how* one variable causally influences another. For instance, if we draw $X \to Y$, this could mean that Y increases if we increase X, but it could also mean that Y decreases if we increase X. It's even possible that Y first increases and then decreases, or that the pattern is another altogether.

Exercise 1.6. Draw a DAG that represents the belief that Danes' attitudes towards Swedish and their understanding of Swedish causally affect each other (i.e., the more the like it, the better they understand it, which leads to their liking it even better). ♢

Exercise 1.7. Draw a DAG that represents the belief that Danes who like Swedish seek out more contact with Swedish (e.g., by watching Swedish television), which leads to their understanding it better, which in turn leads to their seeking out even more contact with Swedish, and so on. ♢

1.3.4 Chains, forks and inverted forks

A DAG that is drawn by following the rules specified above is always built up out of at most three types of building blocks: chains, forks, and inverted forks.

Chains A chain is a sequence of causal links; in a DAG, chains show up as directed paths. In Figure 1.8, $A \to B \to C \to D$ forms a causal chain. Note that causality doesn't flow 'upstream' against the direction of the arrows, so there is no causal chain from D back to A.

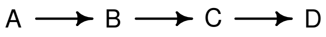


Figure 1.8: A chain.

Chains may transmit genuine causal influences, that is, altering the values of (say) A may bring about a change in some values in (say) D. In other words, A may causally affect D, albeit indirectly through B and C. Since the causality is directional, altering the values of D won't bring about any changes in the values of A, B or C.

Moreover, **chains may induce associations between the variables involved**. Based on the DAG in Figure 1.8, we wouldn't be surprised to find some association between the values of A, B, C and D. The DAG doesn't tell us what this association will look like, but we'll encounter some common forms of association in the weeks to come.

Note that it is possible that changes in A are not reflected in changes in D downstream. For instance, the effect that A has on B may be quite small, and perhaps only large changes in B affect C. This is why I wrote that changes in A *may* (rather than *will*) bring about changes in D.

If, for whatever reason, you want to prevent a chain from transmitting associations between two variables, the path between these variables has to be **blocked** somewhere. This is achieved by **controlling** for one (or several) of the variables along the path. A conceptually easy (if often practically arduous) way is to ensure that only people, words, etc. with the same value on that variable are included in the study. For instance, if for some reason you need to control for eye colour, you could include only green-eyed people in your study.

Forks When a single factor causally affects two or more other factors, a fork is formed; see Figure 1.9. In this example, A causally influences both B and C.

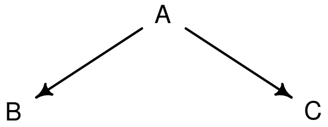


Figure 1.9: A fork.

Forks themselves don't transmit causal influences between the prongs, that is, altering the values of B won't change the values of C and vice versa: Causality doesn't travel upstream. If you want to represent a causal link between B and C, you have to add it to the DAG.

Importantly, **forks may induce associations between the factors at the prongs**: Based on the DAG in Figure 1.9, we wouldn't be surprised to find some association between the values of B and C. This is not because of a causal link between them but because A influences both of them. A is also referred to as a **confounding variable** or **confounder**.

To better appreciate the fact that causal forks can give rise to associations between the variables at the prongs, consider the fictitious example in Table 1.1. Here, A causally influences both B and C, and both B and C are additionally influenced by separate factors (U_B and U_C). The causal factors A, U_B and U_C can each take on two values (0, 1), and the outcomes of B and C are determined by simple equations. We assume that the probability of observing the values in a particular row is the same for all rows, i.e., that it is 1/8.

A	U_B	U_C	$B := A + U_B$	$C := A + U_C$
0	0	0	0	0
0	0	1	0	1
0	1	0	1	0
0	1	1	1	1
1	0	0	1	1
1	0	1	1	2
1	1	0	2	1
1	1	1	2	2

Table 1.1: Illustration of how a causal fork can give rise to associations between the variables at the prongs.

Taking a closer look at this table, we see that B and C are associated: The overall probability that B is at least equal to 1 is $\mathbb{P}(B \geq 1) = 6/8 = 75\%$. But if you already know that an observation's value for C is 2, then you can be absolutely confident that its B value is at least 1: $\mathbb{P}(B \geq 1|C = 2) = 2/2 = 100\%$.[4] By Definition 1.3, B and C are associated. By the same token, if you know that its C value is 0, you'd be less confident about this guess:

[4] $\mathbb{P}(B \geq 1|C = 2)$ reads as 'the probability that B will be at least 1 when C equals 2.' This kind of probability is referred to as a conditional probability. If the variables B, C are not associated, then $\mathbb{P}(B \geq 1|C = 2) = \mathbb{P}(B \geq 1)$. Hence, if $\mathbb{P}(B \geq 1|C = 2) \neq \mathbb{P}(B \geq 1)$, as is the case here, B, C are associated.

- $\mathbb{P}(B \geq 1|C = 0) = 1/2$.
- $\mathbb{P}(B \geq 1|C = 1) = 3/4$.
- $\mathbb{P}(B \geq 1|C = 2) = 2/2$.

If you want to prevent a fork from transmitting an association between the variables at the prongs, you can control for the confounder or otherwise block the path on which the confounder lies. To appreciate this fact, again consider Table 1.1. We've already established that $\mathbb{P}(B \geq 1|C = 0) \neq \mathbb{P}(B \geq 1|C = 1)$. But once we 'control for' A by fixing it at a specific value (e.g., $A = 0$), we find that the probability of observing $B \geq 1$ doesn't depend on C any more.

- $\mathbb{P}(B \geq 1|C = 0, A = 0) = 1/2$.
- $\mathbb{P}(B \geq 1|C = 1, A = 0) = 1/2$.

Similarly, we could fix A at 1 and vary C and observe the same phenomenon:[5]

- $\mathbb{P}(B \geq 2|C = 1, A = 1) = 1/2$.
- $\mathbb{P}(B \geq 2|C = 2, A = 1) = 1/2$.

Exercise 1.8. While I don't have the numbers handy, I'm confident that there is some positive association between the number of drownings in the Aare and the daily revenue of Bernese ice-cream vendors. Why? ♢

Inverted forks Figure 1.10 shows an inverted fork where two variables both influence a third one. The 'handle' of an inverted fork is called a **collider** since the two causal arrows clash into each other in A.

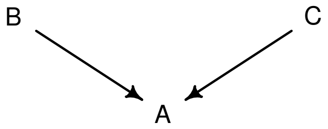


Figure 1.10: An inverted fork.

Inverted forks don't transmit causal influences between the variables at the prongs, that is, there is no causal link between B and C (causality doesn't travel upstream). The intriguing thing about inverted forks is this, though: When the collider (i.e., A) is *not* controlled for, the variables at the prongs remain unassociated. However, **controlling for the collider may induce an association between the variables at the prongs even in the absence of a causal link between them**. Controlling for a descendant of a collider may likewise induce an association between the variables at the prongs.

The effects of controlling for a collider are not intuitive, so let's consider an example.

Example 1.9. University teachers can testify that there is some negative association between their students' intelligence and their diligence. This doesn't mean that the most intelligent students are *all* lazy and none of the most diligent students are particularly clever—just that there is some tendency for the most intelligent students to be less hard-working than the less clever ones. There is a simple and plausible causal explanation for this association: The

[5] We can't fix A at 1 and evaluate this probability at $C = 0$ for the simple reason that there's no row in the table with $A = 1$ and $C = 0$.

most intelligent students quickly figure out that they don't need to work as hard in order to obtain their degree, so they shift down a gear.

But there is an equally plausible if less simple explanation: by only looking at university students, we've controlled for a collider without realising it; see Figure 1.11. Even if diligence and intelligence were completely unassociated in the human population, they are bound to be associated if we only look at university students. Figure 1.12 illustrates why: If we consider the population as a whole, it's possible that there is no (or hardly any) association between diligence and intelligence (left panel): If we know a person's degree of diligence, we can't make a more educated guess as to their intelligence than if we don't. But if we only consider university students (filled circles in the right panel), we're bound to find a negative association between diligence and intelligence: If we know that a university student is pretty lazy, we also know that they need to be pretty intelligent—otherwise they couldn't have made it into university.

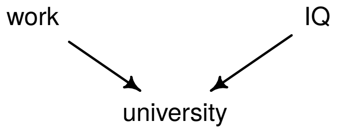


Figure 1.11: To the extent that diligence (work) and intelligence (IQ) both determine if someone gets into university, some association between these two factors will be found if we only look at university students.

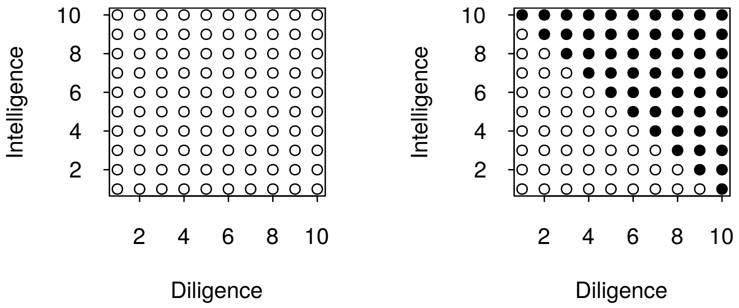


Figure 1.12: Collider bias in action: If you only look at the filled (or at the unfilled) circles, you'll discover an association between diligence and intelligence, even though there is no causal link between them.

By only looking at university students, we've unwittingly controlled for a collider, which by itself can explain the negative association between diligence and intelligence observed among university students. This doesn't mean that our first causal explanation is necessarily wrong. But it does illustrate that there is a non-obvious but plausible additional explanation that we need to reckon with. It's also possible that both explanations are simultaneously be correct: There may be some (negative) causal influence of intelligence on diligence, but by only looking at university students, we would then end up overstating the strength of this causal effect.

In Figure 1.12, we've assumed—for ease of exposition—that there is a perfect deterministic relationship between diligence and intelligence on the one hand and university enrolment on the other hand (viz., if the sum of both scores is above 10, enrolment is granted). In reality, this relationship won't be perfect (some highly intelligent and highly diligent people don't go to university), but even so, controlling for (or 'conditioning on') a collider can produce associations between two factors in the absence of a causal link between them. ♢

Other examples of this collider bias are only superficially different from the example we've just considered. The difficulty lies in figuring out what collider was unwittingly controlled for.

Exercise 1.10. There is a negative association between how easily accessible a restaurant is from a tourist resort and how good the food is. Come up with an explanation that does not assume any direct or indirect causal influence of food quality on location or vice versa. ◊

Exercise 1.11. People with a highly active dating life sometimes complain that their hottest dates tend to be comparatively boring. Come up with an explanation that does not assume any direct or indirect causal influence of attractiveness on interestingness. ◊

> In sum, unbroken chains both transmit causality and induce associations; forks induce associations without causality unless measures are taken (e.g., controlling for the confounder); and inverted forks induce associations without causality if the collider (or one of its descendants) is controlled for.

Let's now turn to some DAGs that are made up of several of the building blocks we've discussed.

Exercise 1.12. Consider the DAG in Figure 1.13.

(a) Can A causally affect F? In other words, does there exist a directed path (chain) from A to F on which no variable has been controlled for?

(b) Can C causally affect D?

(c) Can there be an association between C and D if no factors are controlled for? Why (not)?[6]

(d) Can there be an association between C and D if E is controlled for? Why (not)?

(e) Can there be an association between C and D if F is controlled for? Why (not)?

(f) Can there be an association between C and D if A is controlled for? Why (not)?

(g) Can there be an association between C and D if B is controlled for? Why (not)? ◊

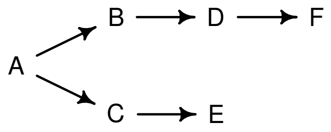


Figure 1.13: DAG for Exercise 1.12.

Exercise 1.13. Consider the DAG in Figure 1.14.

(a) Can A causally affect F?

(b) Can A causally affect E?

(c) Can there be an association between A and E? Why (not)?

(d) Can there be an association between A and E if F is controlled for? Why (not)?

(e) Can there be an association between B and D if no factors are

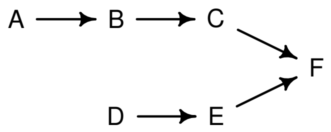


Figure 1.14: DAG for Exercise 1.13.

[6] If the answer is 'yes', it suffices to list a path via which such an association could be transmitted. If the answer is 'no', you need to check for each path between C and D that this path cannot transmit associations.

controlled for? Why (not)?

(f) Can there be an association between B and D if A is controlled for? Why (not)?

(g) Can there be an association between B and D if F is controlled for? Why (not)?

(h) Can there be an association between B and D if C and F are controlled for? Why (not)?

(i) Can there be an association between B and D if E and F are controlled for? Why (not)? ♢

Exercise 1.14. Consider the DAG in Figure 1.15.

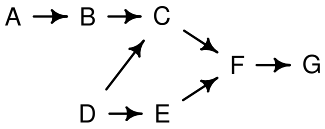


Figure 1.15: DAG for Exercise 1.14.

(a) Can A causally affect D?

(b) Can there be an association between A and D if no factor is controlled for? If so, via which path?

(c) Can there be an association between A and D if C is controlled for? If so, via which path?

(d) Can there be an association between A and D if F is controlled for? If so, via which paths (plural!)?

(e) Can there be an association between A and D if G is controlled for? If so, via which paths (plural!)?

(f) Can there be an association between A and D if B and C are controlled for? If so, via which path?

(g) Can C causally affect E?

(h) Can there be an association between C and E if no factor is controlled for? If so, via which path?

(i) Can there be an association between B and E if no factor is controlled for? If so, via which path?

(j) Can there be an association between B and E if C is controlled for? If so, via which path?

(k) Can there be an association between B and E if D is controlled for? If so, via which path?

(l) Can there be an association between B and E if D and F are controlled for? If so, via which path?

(m) Can there be an association between B and E if D and G are controlled for? If so, via which path? ♢

Definition 1.15 (Bias)**.** The term **bias** refers to a systematic distortion of the results, e.g., due to confounding variables. A single **unbiased** study isn't guaranteed to estimate the size of the causal effect correctly, but roughly speaking, if we were to run the same study lots of times, the under- and overestimates would cancel each other out. If the under- and overestimates don't cancel each other out, then the study is **biased**.[7] ♢

Exercise 1.16. If we want to obtain an unbiased estimate of the total

[7] The more formal mathematical definition is that an estimate is unbiased if its expected value equals the parameter value it attempts to estimate, and biased otherwise.

causal influence of a variable X on another variable Y, we need to ensure

- that all directed paths from X to Y are open (i.e., no intermediate variable is controlled for);
- and that X and Y cannot be associated via any other paths.

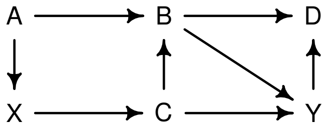


Figure 1.16: DAG for Exercise 1.16.

Now consider the DAG in Figure 1.16 and answer the following questions:

(a) List all paths via which X may causally affect Y if no variables are controlled for.

(b) List all paths via which X and Y may be associated if no variables are controlled for.

(c) Which variable(s) do you *need* to control for if you want to obtain an unbiased estimate of the causal effect of X on Y? Also quickly check if controlling for these variable(s) doesn't open up any new paths via which X and Y may be associated![8] ♢

1.4 Some further terms

Exercise 1.17 (Descriptive statistics)**.** Read Johnson (2013) with an eye towards explaining the following terms and concepts in a manner that you find intelligible by providing your own definition, clarifying example or illustration.

(a) Continuous vs. categorical variables (pp. 289–290 in Johnson, 2013)

(b) Histogram (pp. 292–293)

(c) Bimodal distribution (p. 292)

(d) Outliers (p. 292)

(e) Normal distribution (pp. 293–294)

(f) Arithmetic mean vs. median vs. mode (pp. 295–296)

(g) The effect of outliers on the mean and median (p. 297)

(h) Quantile, percentile, and quartile (p. 298)

(i) Standard deviation and variance (p. 299)

(j) Left- and right-skewed distributions (p. 301)

(k) Ordinal vs. nominal variables (p. 307)

(l) Bar chart (pp. 307–308)

(m) Contingency table (p. 311) ♢

1.5 *Further reading

Sections marked with an asterisk are optional.

Rohrer (2018) is an accessible introduction to DAGs. I don't recommended you read it right away, though, but save it in case you

[8] Sometimes, different sets of variables can be controlled for to this end. But in this case, there is just a single correct solution.

need a refresher from a different source in a couple of months or years.

Discussions about biased estimates are easier to follow if you have some experience with analysing quantitative data. The relevant concepts are discussed in the lecture notes *Einführung in die quantitative Datenanalyse*. An abridged English-language version is available in the form of the lecture notes *Introduction to the general linear model*.

2

Constructing a control group

2.1 A made-up example

Imagine that a new self-learning method for fostering Danish reading skills in speakers of German has been developed. You're tasked with finding out if this new method works better than the old one.

First attempt You find four students of German philology who want to learn Danish. You ask them to work autonomously with the new learning method half an hour a day for three weeks. After three weeks, you give them an article from a Danish newspaper, which they are to summarise orally in German. Two raters judge these summaries at their own discretion (20-point scale); the mean of the two ratings per learner counts as their reading comprehension score. The average group score is 11/20.
What can you conclude from this study?

One of several problems with this study is that there is no baseline against which to compare the participants' average result: We don't know whether 11/20 indicates that the new learning method works better or worse than the old one, or whether the old and new learning method are roughly equally effective. So we need a comparison or **control group** of people that didn't take part in the intervention.

Second attempt You convince four law students to also take part in the study. They're asked to work with the old learning method half an hour a day for three weeks. Then they take the same test as the German philology students. Their group mean is 8/20.
What can you conclude from this study?

2.2 Critical questions

The second attempt outlined above also falls short on a number of criteria. There are a couple of critical questions we can ask, and slightly modified versions of these questions can be asked about studies in general.

Internal validity Can the difference in test scores between the two groups be ascribed to the difference in learning methods, or do alternative explanations for it present themselves?

External validity Does the finding apply only to the present **sample** or can it be generalised to a larger **population**? To what population, exactly?

Ecological validity (Especially for applied research.) To what extent are the findings applicable to the world outside of the lab (e.g., teaching, policy)?

Reproducibility Would different observers agree on the measurements? Would the *same* observers agree on the measurements if they had to redo them? (The raw data may leave room for interpretation.)[1]

Interpretative consensus Confronted with the same data, would other researchers draw similar conclusions? (The overall results may leave room for interpretation.)[2]

Replicability Can the results of this study be confirmed in an independent **replication**, that is, in a new but otherwise similar study?

The labels above aren't important; the questions behind them are. Further, the questions asked above can rarely be answered in absolute terms. But our second attempt outlined above is deficient in several of these respects. Discuss a few problems.

Some relevant terminology:

Confounding variable See Chapter 1.

Inter-rater reliability The extent to which different raters would score the observations similarly.

Intra-rater reliability The extent to which the same raters would score the observations similarly on a different occasion.

> Anticipate and resolve problems related to lacking validity and reproducibility/reliability *before* collecting the data. This often involves making compromises or coming to the realisation that you can't satisfactorily answer all your questions in a single study. Do *not* assume that some statistical method will solve your problems.

Depending on your goals, some types of validity or reliability may not be as important as others. For instance, for most studies in

[1] My use of the term 'reproducibility' is definitely non-standard. The term is usually used to mean 'computational reproducibility': Given the study's data, can its results (e.g., the statistical analyses) be reproduced?

[2] For instance, a study with considerable internal, external and ecological validity could show that beginning L2 French learners vastly prefer subject-verb inversion over other strategies for formulating questions, but that more seasoned learners show a more balanced use of the different strategies. Researchers may agree on these results, but are bound to disagree on which implications, if any, they ought to have for L2 French teaching.

psycholinguistics, university students are recruited as participants, and their results don't necessarily generalise to the population at large. But the purpose of these studies is often to demonstrate that some experimental manipulation *can* affect language use and processing, not that it will yield the exact same effect for everyone. From this perspective, these studies' lack of external validity isn't too damning (Mook, 1983).

2.3 *Increasing internal validity through randomisation*

Of the three types of validity considered, internal validity is the most pressing one: If internal validity is low, external and ecological validity are both essentially irrelevant. Hence, our first priority is to maximise the study's internal validity, that is, we want to maximise the chances that any association we find the data is due to the factor of interest. Confounding in particular represents a substantial threat to internal validity: As we've seen in Chapter 1, confounding variables induce associations between the variables of interest even in the absence of a causal link between them. Moreover, even if a causal link does exist between the variables of interest, confounding variables can bias the association between them: The association may systematically under- or overestimate the strength of the causal link. Keeping confounding in check is therefore key.

Your first inclination may be to try to ensure that the intervention and control groups are identical in all respects save for the treatment itself. That way, any differences in the outcome variable can't be explained by confounding due to pre-existing differences between the groups. However, it is practically impossible to assign a fixed number of participants to two groups in such a way that these groups are identical in all respects even in the utterly unrealistic case where all the relevant information is available beforehand. Clearly, it's entirely impossible to do so when not all of the relevant information is available beforehand.

The solution is to assign the participants to the study's conditions **at random**, i.e., to deliberately leave the allocation up to chance and chance alone. The DAGs in Figure 2.1 on the next page show what such randomisation achieves. When the participants themselves (or their parents, or their circumstances, etc.) decide or otherwise affect which condition they end up in (X), confounding is a genuine concern (left). However, when we assign the participants to the conditions at random, we *know* that there is no systematic link between pre-existing characteristics (U) and X, let alone a causal one. That is, randomisation prevents any causal arrows from entering X (right)! The result of this is that the non-causal path between X and Y (via U) is broken and that the X-Y relationship is no longer confounded by U. To highlight that the values of X were specified at random, we use a subscript $_r$.

Studies in which the participants are randomly assigned are

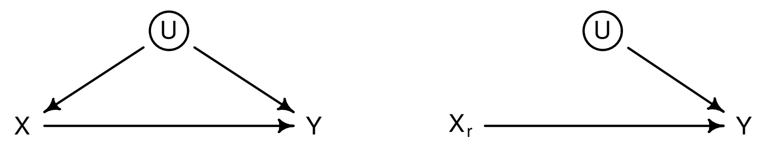


Figure 2.1: *Left:* The X-Y relationship is confounded by U: there are two paths from X to Y, but only one causal one. *Right:* Randomising the values of X prevents arrows from U entering X, which effectively closes the non-causal path via the confounder.

called **true experiments**. Random allocation by itself doesn't guarantee that the results of the experiment can be trusted or interpreted at face value, but it does eliminate confounding as a threat to the study's internal validity.

> Randomise wherever possible – unless you have a *very* good reason not to!

2.3.1 *Why experiments?*

1. "Experiments allow us to set up a **direct comparison** between the treatments of interest.
2. "We can design experiments to **minimize any bias** in the comparison. [especially randomisation]
3. "We can design experiments so that the **error** in the comparison is **small**. [see later chapters]
4. "Most important, we are **in control** of experiments, and having that control allows us to make stronger inferences about the nature of differences that we see in the experiment. Specifically, we may make **inferences about causation**." (Oehlert, 2010, p. 2, my emphasis)

What's meant by the first point is that we don't need to piece together snippets of evidence from different and often difficult-to-reconcile sources (e.g., in a literature review) to figure out which treatment (e.g., which learning method) works best in some situation. Instead, we set up an experiment that directly evaluates the efficacy of several treatments in the context we're interested in.

The second point alludes to techniques such as randomisation; the third to techniques that we will encounter in the chapters to come.

2.3.2 *What does randomisation do?*

1. "Randomization balances the population on average."
2. "The beauty of randomization is that it helps prevent confounding, *even for factors that we do not know are important*." (Oehlert, 2010, p. 15, my emphasis)

We've already discussed the second point, but the first point warrants some explanation. Some fundamental results from combinatorics are useful at this stage.

Lemma/Definition 2.1 (Factorial)**.** If you have $n \geq 0$ distinct objects, there are

$$n! := \begin{cases} n(n-1)\cdots 2\cdot 1, & \text{if } n \geq 1, \\ 1, & \text{if } n = 0. \end{cases}$$

different ways to sort them ('permutations').

We say 'n factorial' for 'n!'. ♢

Proof. If $n = 0$, there is only one way to sort the objects: do nothing.

If $n \geq 1$: There are n candidates to fill the first slot. Once a candidate for the first slot has been picked, $n - 1$ candidates remain for the second slot, and so on, till there is only a single candidate left for the final slot. □

Incidentally, for $n \geq 1$, $n!$ can also be written recursively as $n! = n \cdot (n-1)!$. The factorial function increases spectacularly quickly—much more quickly than the exponential functions of Covid 19 fame.

Lemma/Definition 2.2 (Binomial coefficient)**.** There are

$$\binom{n}{k} := \frac{n!}{k!(n-k)!}$$

different ways to choose $k \geq 0$ objects from among $n \geq k$ distinct objects.

A term of the form $\binom{\cdot}{\cdot}$ is called a **binomial coefficient**[3]; we say 'n choose k' for '$\binom{n}{k}$'. ♢

Proof. With the previous lemma, there are $n!$ ways to arrange the $n \geq 0$ distinct objects. For each possible rearrangement, pick the first k objects. This covers all possible choices of k from n objects. However, several rearrangements result in the same selection. Specifically, if we rearrange the first k elements and the last $n - k$ elements separately from each other, we end up with the same selection: the order among the first k and among the last $n - k$ doesn't matter. Hence, again with the previous lemma, there are, for each of the $n!$ permutations, $k!(n-k)!$ rearrangements that result in the same selection. So there are $\frac{n!}{k!(n-k)!}$ different possible selections. □

Let's say that we have ten participants and we know both their sex and their IQ (Figure 2.2). We can pick five among them at random and assign them to the first condition; the others are assigned to the second condition. By the previous lemma, there are

$$\binom{10}{5} = \frac{10!}{5!(10-5)!} = \frac{3628800}{(120)^2} = 252$$

different choices for the participants in the first group; each of these choices has an equal probability of being our allocation. Six out of the 252 possible allocations are shown in Figure 2.3. Note that in none of them, the intervention and control groups are perfectly balanced with respect to both IQ and sex. So randomisation

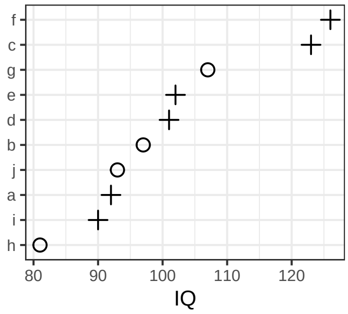


Figure 2.2: Ten participants sign up for a study. You measure their IQ and you also know their sex (represented here using circles and crosses).

[3] Called so because these terms appear when expanding binomial expressions such as $(a+b)^n$.

clearly does not generate balanced groups in any particular study. However, each participant is as likely to end up in the intervention group as they are to end up in the control group, so *on average*—across all 252 possible allocations—sex, IQ, as well as all unmeasured variables, are balanced between the two groups. For our present purposes, this means that randomisation is an equaliser: the result may not be two perfectly equal groups, but at least one group isn't systematically given an advantage relative to the other. As we'll see in Chapter 4, randomisation also justifies the use of some common statistical procedures.

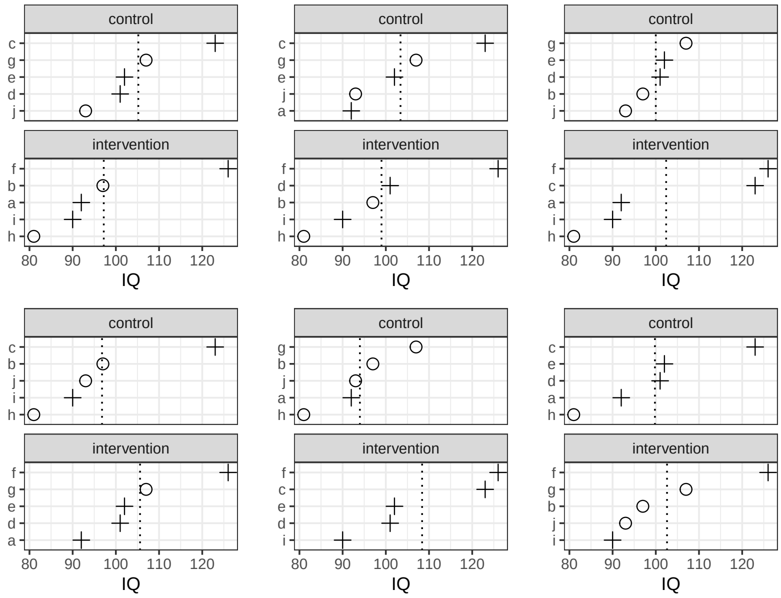


Figure 2.3: Six possible random assignments (out of 252) of the ten participants from Figure 2.2. The dotted vertical lines show the mean IQ in each group.

Exercise 2.3. For each description, decide whether the participants were randomly assigned to the experiment's conditions and, if not, explain how the lack of randomisation could result in confounding.

(a) 60 participants trickle into the lab. The first 30 are assigned to the experimental condition, the final 30 are assigned to the control group.

(b) Experiment with a school class: Pupils whose last name starts with a letter between A and K are assigned to the control group, the others to the experimental group.

(c) Participants come to the lab one by one. For each participant, the researcher throws a dice. If the dice comes up 1, 2, or 3, the participant is assigned to the experimental condition; if it comes up 4, 5, or 6, the participant is assigned to the control condition. After four weeks, no more participants sign up. The control group consists of 17 participants; the experimental group of 12.

(d) To investigate the effects of bilingualism on children's cognitive development, 20 bilingual 4-year-olds (10 girls, 10 boys) are recruited. 20 monolingual 4-year-olds (10 girls, 10 boys) serve as the control group.

(e) 32 participants sign up for an experiment. The researcher enters their names into `http://www.random.org/lists/`, clicks `Randomize` and assigns the first 16 to the control group and the others to the experimental group. ♢

> 'Random' does not mean 'haphazard', 'arbitrary' or 'at the researcher's whim'.

Remark 2.4 (Randomisation and estimation)**.** When participants are assigned randomly to the experiment's conditions, the results of the experiment are also subject to randomness. To appreciate this, consider an experiment for which sixteen participants sign up. The left-side panel in Figure 2.4 shows the score that each participant would have obtained in both conditions. The difference between both of these scores varies somewhat between participants, but on average, it amounts to one point. This is the true average intervention (or treatment) effect that is the target of the experiment.

Depending on the condition they are assigned to, we would only observe one of the scores for each participant, however. If we randomly assign eight of the sixteen participants to the intervention condition and the others to the control condition, we end up with one out of $\binom{16}{8} = 12870$ possible allocations. To estimate the true average intervention effect, we can compute the mean difference between the scores of the participants assigned to the intervention condition and those assigned to the control condition. The estimated intervention effect would then be one of the 12,870 values summarised in the histogram on the right-hand side. While the *average* estimated intervention effect coincides with the true intervention effect (i.e., the estimation is unbiased), depending on the actual allocation, the estimated intervention effect may differ considerably from the true one. In Chapter 5, we'll discuss techniques for increasing the precision of the estimation. ♢

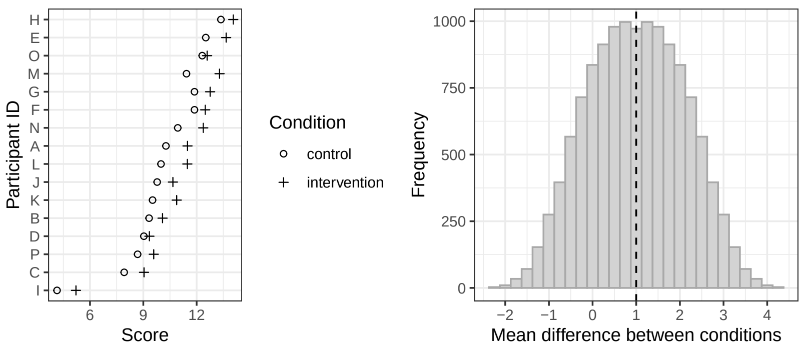


Figure 2.4: *Left:* The scores that each of the sixteen participants would have obtained if they had been assigned to the control group (circles) or to the intervention group (crosses). The average difference between the intervention and the control scores, i.e., the average intervention effect, is one point. *Right:* The histogram shows the distribution of the mean difference between the intervention and control scores for each of the $\binom{16}{8} = 12870$ possible allocations of participants to the two conditions. Averaged over all 12,870 allocations, the mean difference is also one point. But for many individual allocations, the mean difference may differ considerably from the true average intervention effect of one point.

2.3.3 How to randomise?

When collecting data using computers Have the computer randomly assign the participants to the conditions without your involvement. Frameworks for running experiments such as OpenSesame (`https://osdoc.cogsci.nl/`), PsychoPy (`https://www.psychopy.org/`) or jsPsych (`https://www.jspsych.org/`) all contain functions for allocating participants randomly.

When the data collection does not take place at the computer and you know who'll be participanting beforehand Randomise the list of participants using `https://www.random.org/`. Assign the first half of the list to the experimental condition and the second half to the control condition. If the experiment features $k \geq 3$ conditions, similarly split up the list into k parts.

This procedure is known as **complete randomisation**. It guarantees that the number of experimental units is the same in each condition (or at most one off if the number of units isn't divisible by the number of conditions).

When the data collection does not take place at the computer and you don't know who'll be participating beforehand Randomly assign each participant individually and with the same probability to a condition as they sign up. This procedure is known as **simple randomisation**. In contrast to complete randomisation, you're not guaranteed to end up with an equal number of units in each condition. This is usually of little concern, and in fact, simple randomisation arguably reduces the potential for the researchers' biases to affect the study's results (Kahan et al., 2015). Importantly, **there is nothing wrong with having unequal sample sizes.**[4]

> Humans make for poor randomisation devices. Always randomise mechanically (preferably with a computer).

More complex allocation procedures are possible. Some of these are common in medical research (Rosenberger & Lachin, 2016),[5] but they are rarely applied in the social sciences.

Exercise 2.5. For each description, decide if the study is a true experiment.

(a) Eight Swiss speakers of German indicate how beautiful they find the French language on a 7-point scale. Additionally, they all record a text in French. In a 'perception experiment', 20 native speakers rate all recordings on a 5-point scale from 'very strong foreign accent' till 'no foreign accent whatsoever'. The question is whether the speakers' attitudes are related to the strength of their accent in French (Kolly, 2011).

(b) "This study presents the first experimental evidence that singing can facilitate short-term paired-associate phrase learn-

[4] See blog entry *Causes and consequences of unequal sample sizes*.

[5] Incidentally, medical researchers refer to simple randomisation as 'complete randomisation'... It's probably best to just spell out how the randomisation was carried out rather than to rely on short-hand terminology.

ing in an unfamiliar language (Hungarian). Sixty adult participants were randomly assigned to one of three ”listen-and-repeat” learning conditions: speaking, rhythmic speaking, or singing.” After 15 minutes of learning, the learners’ Hungarian skills are tested and compared between the three conditions (Ludke et al., 2014).

(c) ”The possible advantage of bilingual children over monolinguals in analyzing word meaning from verbal context was examined. The subjects were 40 third-grade children (20 bilingual and 20 monolingual) . . . The two groups of participants were compared on their performance on a standardized test of receptive vocabulary and an experimental measure of word meanings, the Word–Context Test.” (Marinova-Todd, 2011) ◊

The word ’experiment’ can be used in a stricter or in a looser sense. The mere fact that a study is referred to as an ’experiment’ does *not* mean that it’s a *true experiment* (control group + randomisation): the use of the label doesn’t automatically imply that confounding has been taken care of.

Many quantitative studies in our field aren’t experiments in the strict sense.

3
Alternative explanations

3.1 The roles of variables in research

Some common terminology:

Dependent variable or *outcome variable*.

Independent variable or *predictor variable*. In experiments, such variables are 'manipulated' by the researchers. The goal is then to find out the effects of such manipulation of the independent (or predictor) variables on the dependent (or outcome) variables.

Control variable. Additional variable that was collected as it may be related to the *outcome*. We'll discuss the usefulness of control variables later.

Exercise 3.1 (Reading assignment)**.** Read Ludke et al. (2014) in light of the questions below and briefly answer them. The answers to these questions can be found in the 'Method' section (pp. 43–47), so you don't have to read the rest.

(a) What is or what are the independent variable(s) in Ludke et al.'s study?

(b) What is or what are the dependent variable(s) in Ludke et al.'s study?

(c) How did the researchers assign the participants to the conditions? ♢

3.2 Alternative explanations for results

In the study by Ludke et al. (2014) (see Exercise 3.1), the question about internal validity boils down to this: Can the differences in the outcomes between the conditions actually be ascribed to the difference between the conditions (control vs. intervention; singing vs. rhythmic speaking vs. speaking)? Or are there other explanations for it?

In Chapter 2, we focused on the threat that confounding poses to a study's internal validity and how this threat can be neutralised using randomisation. We saw that randomised ('true') experiments (probabilistically) negate the influence of confounding variables on the results: one group isn't systematically given an advantage compared to the other (e.g., higher motivation, greater affinity with a topic etc.). This increases the study's internal validity, but:

> Even if confounding variables are taken into account, other systematic factors may give rise to a spurious difference between the experiment's conditions or may mask an existing effect of the conditions.

3.3 Explanation 1: Expectancy effects

Perhaps the researchers or their assistants (subconsciously) nudged the data in the hypothesised direction. This can happen even when the measurements seem perfectly objective. For instance, when you're counting the number of syllables in a snippet of speech, there are bound to be a number of close decisions (Does German *haben* [ha(b)m] have one or two syllables?). This isn't too big a problem in itself. But it does become a cause for concern if you tend to reach different decisions depending on which condition the participant was assigned to.

Relatedly, it's possible that the participants want to help (or thwart) the researchers achieve what they think are the researchers' goals. In this case, differences in the outcome variable between the conditions may arise not because of the intervention itself but because of unwanted changes in the participants' behaviour. Such changes in behaviour needn't come about consciously.

Expectancy effects Both on the part of the participants (e.g., *placebo* effect) or on the part of the researchers.

Single-blind experiment Typically used to describe that the participants don't know which condition they're assigned to.

Double-blind experiment If neither the participants nor the researchers themselves (at the time of collecting and preparing the data) know which condition the participants were assigned to.

Blinding isn't always possible, and it may be immediately obvious to the participants what the intervention entails. But in studies with raters, it's usually easy to prevent them from knowing which condition the participants were assigned to.

3.4 Explanation 2: Failed manipulation

A second class of alternative explanations is that the experiment didn't run quite as the researchers expected it to. For instance, the participants may have misunderstood, or failed to act on, the instructions, or the script used to run the experiment could contain a crucial coding error.

Manipulation checks Example 1: Ludke et al. (2014) (Exercise 3.1) wanted to find out if foreign-language phrases are more easily learnt if the learners practice them while singing or speaking rhythmically. Their experimental manipulation involved asking the participants to practice Hungarian phrases while singing or speaking rhythmically. In order to verify whether they indeed did as they were asked, they recorded their participants.

Example 2: Lardiere (2006) had her participant judge L2 sentences for their grammaticality. To ensure that the participant rejected sentences for the (syntactic) reason intended by the researcher, she was also asked to correct any sentences she rejected. The researcher found out that the participant rejected a fair number of syntactically correct sentences, but that she did so for stylistic (rather than strictly syntactic) reasons. The researcher then (correctly) didn't draw the conclusion that the participant's syntactic knowledge was incomplete.

Satisficing Sometimes, participants don't really pay any attention to the stimuli or to the instructions. For instance, questionnaire respondents may answer in a specific pattern (e.g., ABCDEDCBA. . .) rather than give their mind to each question. Figure 3.1 provides another example of satisficing.

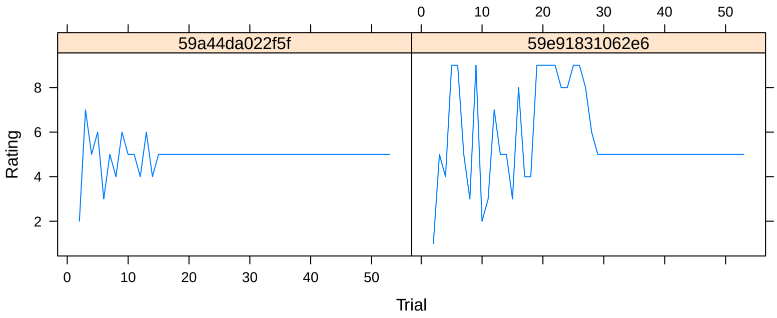


Figure 3.1: A large number of raters were asked to each rate about 50 short texts on a 9-point scale. These two clearly lost interest at some point (Vanhove, 2017).

If you want to run a study online or at the computer, check out Oppenheimer et al. (2009) for a neat and unintrusive way to find out if your participants read the instructions.

Positive control Does the intervention yield an effect in cases where it *should* (with near-certainty) yield an effect? If not, then the experiment may have been carried out suboptimally.

Example 1: Let's say a researcher wants to find out if knowledge of L2 verb morphology contributes to L2 French speakers' comprehension of temporally complex texts. To this end, she proposes an experiment that involves the intensive teaching of French verb morphology. In this case, it would make sense for the researcher to check if the intervention does in fact succeed in improving the participants' command of French verb morphology. If it doesn't, then the results of the study can't speak to the link between knowledge of verb morphology and text comprehension.

Example 2: In L2 research, the task given to the L2 speakers is often also given to a group of L1 speakers. This helps identify issues in the materials used. For example, if sentences meant to be grammatically correct are frequently rejected by L1 speakers and L2 speakers alike (perhaps due to awkward style), it suggests the problem lies with the stimuli, not necessarily with the L2 speakers' abilities.

The term **negative control** refers to traditional control groups (of which we know that they shouldn't show an effect of the intervention).

Pilot study A pilot study is a small-scale study conducted before the main study. Typical goals of a pilot study are

(a) ensuring that the participants understand, and act on, the instructions;

(b) identifying any remaining glitches in the experimental software or in the analysis pipeline;

(c) if relevant, checking whether the responses obtained can be coded satisfactorily;

(d) estimating how much time participants need to complete the study. This is useful for recruiting participants and scheduling;

(e) detecting any **floor** or **ceiling effects**. A floor effect occurs when a task is so difficult that many participants score near the bottom. Even if participants differ in ability, the task fails to capture it. Ceiling effects occur if a task is too easy.

3.5 Explanation 3: Chance

A third important possible non-causal explanation for one's results is that they're due to chance. The entire next chapter is devoted to an attempt to get a handle on this explanation.

4
Inferential statistics 101

If you've ever read a quantitative research articles, you've probably encountered p-values. This chapter explains the basic logic behind p-value-based inferential statistics. It does so by explicitly linking the computation of p-values to the random assignment of participants to conditions in experimental research. If you have ever taken an introductory statistics class, chances are p-values were explained to you in a different fashion, presumably by making assumptions about how the observations in the sample were sampled from a larger population and by making reference to the Central Limit Theorem. For the explanation in this chapter, however, we're going to take a different tack and we will ignore the sampling method and the larger population. Instead, we're going to leverage what we know about how the observations, once sampled, were *assigned* to the different conditions of an experiment. The advantages of this approach are that it connects the design of a study more explicitly to the analysis of its data and that it is less math-intensive while permitting one to illustrate several key concepts about inferential statistics.

The goal of this chapter is for you to *understand conceptually* what statistical tests attempt to achieve, not for you to be able to use them yourself. As a matter of personal opinion, statistical tests are overused (Vanhove, 2021). I think that, in your own research, your focus should be on describing your data (e.g., by means of appropriate graphs) rather than running umpteen significance tests. Analysing data and running statistical tests are not synonymous.

4.1 An example: Does alcohol intake affect fluency in a second language?

Research question Does moderate alcohol consumption affect verbal fluency in an L2?

Method Ten students (L1 German, L2 English)[1] are randomly assigned to either the control or the experimental condition (five each); they don't know which condition they're assigned to. Participants in the experimental condition drink one pint of ordinary beer;

[1] Ten participants is obviously a very low number of participants, but it keeps things more tractable here.

those in the control condition drink one pint of alcohol-free beer.

Afterwards, they watch a video clip and relate what happens in it in English. This description is taped, and two independent raters who don't know which condition the participants were assigned to count the number of syllables uttered by the participants during the first minute. The mean of these two counts serves as the verbal fluency/speech rate variable.

Results The measured speech rates are shown in Figure 4.1. On average (mean), the participants in the *with alcohol* condition uttered 4.3 syllables/second, compared to 3.7 syllables/second in the *without alcohol* condition.

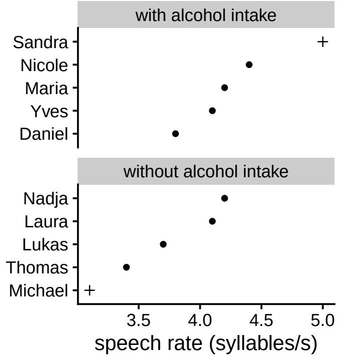


Figure 4.1: Individual results of a randomised experiment.

4.2 *The basic question in inferential statistics*

We have dealt with major threats to internal validity, viz., confounders (neutralised using randomisation) and expectancy effects (neutralised using double blinding). But there is another threat to internal validity that we need to keep in check: While we found a mean difference between the two conditions (4.3 vs. 3.7), this difference could have come about through *chance*. We are, then, faced with two types of account for this mean difference:

- The **null hypothesis** (or H_0): The difference between the means is due *only* to chance.
- The **alternative hypothesis** (or H_A): The difference between the means is due to chance *and* systematic factors.

Assuming the H_0 is correct, the participants' results aren't affected by the condition (alcohol vs. no alcohol) they were assigned to. For instance, Sandra was assigned to the *with alcohol* condition and her speech rate was measured to be 5.0. But had she been assigned to the *without alcohol* condition, her speech rate would also have been 5.0. Assuming the H_0 is correct, then, the difference in speech rate between the two conditions must be due solely to the random assignment of participants to conditions, due to which more fluent talkers ended up in the *with alcohol* condition. Another roll of the dice could have assigned Sandra to the control condition instead of Michael, and since under the H_0, the speech rate of neither is influenced by the condition, this would have produced a slower speech rate in the *with alcohol* condition than in the *without alcohol* one (3.9 vs. 4.1; see Figure 4.2).

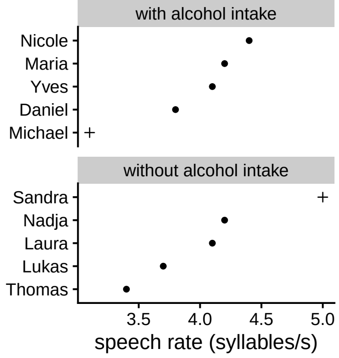


Figure 4.2: If only chance were at play, Michael's (3.1) and Sandra's (5.0) results would be unaffected by the experimental condition and the outcome might equally well have looked like this (swapping Michael and Sandra).

Frequentist inferential statistics seeks to quantify how surprising the results are if we assume that only chance is at play. To do so, it attempts to answer the following key question: **How likely is it that a difference at least this large would've come about if chance alone were at play?**

If it's pretty unlikely that chance alone would give rise to at least the difference observed, then this can lead one to revisit the assumption that the results are due only to chance—perhaps some systematic factors are at play after all. By tradition, the threshold between 'pretty unlikely' and 'still too likely', usually written as α, is 5%, but there is nothing special about this number. If the result falls below this 5% threshold, the difference is said to be 'statistically significant'. This is just a phrase, however, and arguably a poorly chosen one: statistical 'significance' doesn't tell you anything about a result's practical or theoretical import.[2] Before discussing these and other misunderstandings about significance tests, let's see how you can compute how often you would observe a mean difference of at least $4.3 - 3.7 = 0.6$ if chance alone were at play.

4.3 *Testing the null hypothesis by exhausitive rerandomisation*

With 10 participants in two equal-sized groups, there were $\binom{10}{5} = 252$ possible assignments of participants to conditions, each of which was equally likely to occur. To see how easily a difference as least as large as the one observed (4.3 vs. 3.7) could occur due to random assignment alone, we can re-arrange the participants' speech rates into each of these 252 combinations and see for each combination what the difference between the *with* and *without alcohol* condition means is (Figure 4.3).

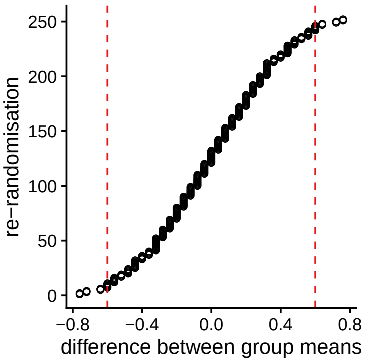


Figure 4.3: There are 252 different ways in which the 10 participants could have been split up into two groups. These are the differences between the means for all 252 possibilities. The mean difference we actually observed (0.6) is represented by the dashed red vertical line on the right-hand side; the dashed red vertical line on the left-hand side is the opposite of this observed mean difference (−0.6).

In eleven out of 252 cases, the re-arrangement of participants to conditions produces a mean difference in speech rate in favour of the *with alcohol* group that is a least as large as the one we actually observed (0.6). In another eleven out of 252 cases, the re-arrangement produced a difference that went in the opposite direction (i.e., in favour of the *without alcohol* condition) but was also at least as large in magnitude as the difference actually observed. In other words, the probability with which we would observe a difference of at least 0.6 between the conditions (in either direction) if chance alone (random assignment) is at play is $\frac{22}{252} = 0.087$ (8.7%). This is the infamous **p-value**.

Using the threshold $\alpha = 0.05$, we would find that $p = 0.087 > \alpha$ and conclude that a difference of 0.6 or more is still likely enough to occur under the null hypothesis of chance alone. Hence, we would see little need to revisit the assumption that the results may be due to chance alone. **Crucially, this doesn't mean that we have shown H_0 to be true.** It's just that H_0 would account reasonably well for these data.

Remark 4.1 (One- and two-sided p-values)**.** In the example above, we computed a two-sided p-value: we counted the rerandomisations that resulted in differences as least as extreme as the one observed in either direction. If we had hypothesised in advance that we would observe a difference in favour of the *with alcohol* condition, we could have computed a one-sided (right-sided) p-value and only have counted the eleven rerandomisations resulting in a mean

[2] From now on, avoid using the words 'significance' and 'significant' in their non-technical sense when writing about quantitative research.

difference as least as large as 0.6. The resulting p-value would have been $p_r = \frac{11}{252} = 0.044$.

Conversely, if we had hypothesised in advance that we would observe a difference in favour of the *without alcohol* condition, we could have computed a *left*-sided p-value and only have counted the rerandomisations resulting a mean difference at *most* as large as 0.6. The resulting p-value would have been $p_\ell = \frac{245}{252} = 0.972$.

Since we didn't specify the direction of the expected difference in advance, we need to consider both possibilities simultaneously and compute a two-sided p-value. One way to compute two-sided p-values is to count the rerandomisations resulting in a mean difference at least as large in absolute value as the difference actually observed (in our case: $p = \frac{22}{252}$). Alternatively, a two-sided p-value can be computed as

$$p = 2\min(p_\ell, p_r).$$

Both of these valid ways coincide in 'nice' cases (e.g., exhaustive rerandomisation tests with an equal number of participants in both conditions), but they may yield somewhat different results in other cases. ♢

Exercise 4.2 (Other test statistics)**.** In your own words, outline how you can compute a (two-sided) p-value for the difference between the *medians* in the example above. ♢

Exercise 4.3 (More than two conditions)**.** Let's say we want to conduct an experiment with twelve participants who are randomly allocated to three conditions with four participants each. (Again, the numbers are kept low to keep exhaustive rerandomisation feasible.) With three conditions, we could compute the three pairwise differences between the condition means. But if we want to capture the differences between these in a single number, we could instead compute, for instance, the standard deviation of the condition means (see Exercise 1.17 on page 18). Standard deviations can't be negative. Under H_0, you'd expect the condition means to be close to one another, so this standard deviation would be close to 0; under H_A, you'd expect it to be higher.

(a) Would you compute a left-sided, a right-sided or a two-sided p-value in this case?

(b) How many possible ways are there to split up twelve participants into three groups of four participants each?

(c) Explain how you can compute an appropriate p-value using exhausitive rerandomisation in this setting.

(d) Now assume that we're interested in the group medians rather than in the group means. What would you need to change in your answer to (c)? ♢

4.4 On 'rejecting' null hypotheses

Researchers will often say that they 'reject' the null hypothesis in favour of the alternative hypothesis if $p \leq \alpha$, where typically $\alpha = 0.05$. This practice is subject to often heated debate (see McShane et al., 2019), and it's important to realise that $p \leq \alpha$ is possible even if the null hypothesis is true. In theory, significance tests guarantee that $p \leq \alpha$ in at most $100\alpha\%$ of the studies in which H_0 actually is true (for all $\alpha \in (0,1)$). This is true in particular for p-values computed using exhaustive rerandomisation.

Lemma 4.4. Under the null hypothesis, exhaustive rerandomisation results in a p-value such that $\mathbb{P}(p \leq \alpha) \leq \alpha$ for each $\alpha \in (0,1)$. ♢

Proof. We'll first consider left-sided p-values. Assume there are M possible rerandomisations. Sort these ascendingly by the value of the test statistic they result in (e.g., the mean difference in our example), breaking ties randomly if necessary. Note that, after this sorting, the left-sided p-value that the i-th rerandomisation would have resulted in (call it p_i) is at least $i/M, i = 1, \dots, M$: there are at least i out of M rerandomisations with test statistics that are at least as extreme as the one in the i-th rerandomisation; an even higher value than i/M is possible if several rerandomisations give rise to the same test statistic value.

Now, for $\alpha \in (0,1)$, compute $\kappa := \lfloor \alpha M \rfloor$. ($\lfloor \cdot \rfloor$ rounds down the number to the nearest integer.) Under the null hypothesis, we were equally likely to have generated each of the M possible randomisations. Hence, the probability that we generated a randomisation resulting in a p-value no larger than α is

$$\begin{aligned}\mathbb{P}(p \leq \alpha) &= \frac{\text{\# rerandomisations with } p_i \leq \alpha}{M} \\ &\leq \frac{\text{\# rerandomisations with } i/M \leq \alpha}{M} \\ &= \frac{\text{\# rerandomisations with } i \leq \kappa}{M} \\ &= \frac{\kappa}{M} \\ &\leq \alpha.\end{aligned}$$

For right-sided p-values, sort the rerandomisations descendingly by the test statistic value and proceed analogously. For two-sided p-values, sort the rerandomisations by the absolute value of the test statistic and again proceed analogously.[3] □

In principle, then, p-values can be used to bound the probability that we claim that something is going on in the data if in fact nothing is going on: We pick some value for α (typically 0.05). If the H_0 is in fact true, then we'd only observe p-values lower than α with a probability of at most α. In practice, however, things aren't so simple. We'll return to this when discussing questionable research practices; but already see Exercise 4.7.

[3] When using the alternative definition of two-sided p-values, consider that $\mathbb{P}(p_\ell \leq \alpha/2) \leq \alpha/2$ and $\mathbb{P}(p_r \leq \alpha/2) \leq \alpha/2$. Hence $\mathbb{P}(\min(p_\ell, p_r) \leq \alpha/2) \leq \alpha/2 + \alpha/2 = \alpha$.

Furthermore, $p > \alpha$ can occur even if the alternative hypothesis is true. Consequently, researchers who are in the business of 'rejecting' null hypotheses can make two types of errors, depending on whether H_0 or H_A is actually true; see Table 4.1. Without additional information (e.g., in the form of converging evidence from other studies or logical reasoning), we can't really know whether '$p \leq 0.05$' represents an error or a true finding. **(!)**

	H_0 is actually correct	H_A is actually correct
$p > \alpha$	Fine—we didn't reject H_0	Wrong conclusion
$p \leq \alpha$	Wrong conclusion	Fine—we rejected H_0

Table 4.1: If you're in the business of rejecting null hypotheses, there are two types of errors you can make. Incorrectly rejecting the H_0 is commonly referred to as a **Type-I error**; incorrectly not rejecting the H_0 is referred to as a **Type-II error**.

Note, furthermore, that the H_A stipulates that the results are due to a combination of chance and systematic factors. It doesn't stipulate *which* systematic factors, though. What we would like to conclude is that the systematic factor at play is our experimental manipulation, but expectancy effects, failed manipulations, confounding and collider bias are also systematic factors. What is more, the experimental manipulation may exert a systematic effect on the results, but for different reasons from what we think it does. For instance, a systematic difference between the *with* and *without alcohol* conditions needn't be due to alcohol intake per se but may be related to the taste of the beers in question instead. Or maybe alcohol increases speech rate—not because the participants become more fluent per se, but because they use simpler syntactic constructions that they can produce more quickly. In other studies, different theoretical explanations may account for any given finding—in addition to more mundane reasons such as confounding, expectancy effects and the like.

Exercise 4.5. Assume that 10,000 experiments are carried out and analysed appropriately. Further assume that the null hypothesis is correct in all of these experiments. Which of the four histograms shown in Figure 4.4 could plausibly represent the distribution of the 10,000 p-values? What if the alternative hypothesis were correct in all of the 10,000 experiments? ♢

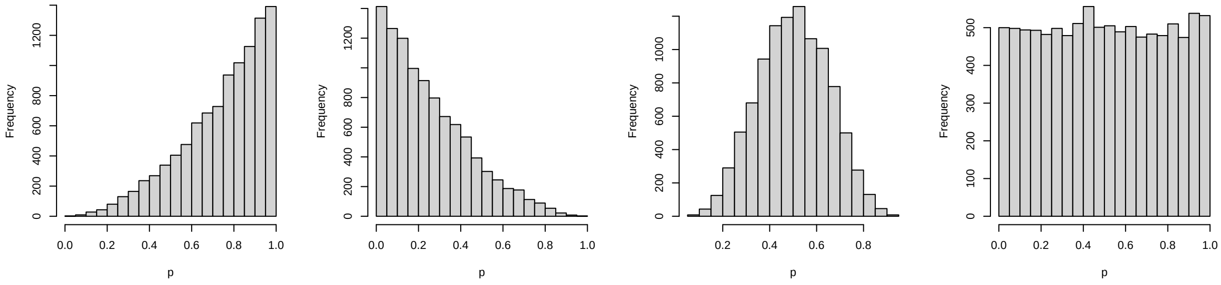


Figure 4.4: What could the distribution of 10,000 p-values look like under the null hypothesis?

Exercise 4.6 (False discovery rate)**.** Imagine that in a given academic field, 1,000 experiments are conducted over the course of a year. Assume that in 80% of these experiments, the null hypothesis is correct, whereas in 20%, the alternative hypothesis is correct. Further assume that if the null hypothesis is correct, there is a 5% chance that a statistically significant difference will be observed between the experiment's conditions; if the alternative hypothesis is correct, there is a 60% chance of observing a statistically significant difference.

For the following questions, you may use the following probabilistic fact: If n attempts each succeed with probability p, then the expected number of successful attempts out of the n attempts is np.

(a) In how many of the 1,000 experiments can we expect that the null hypothesis will be incorrectly rejected?

(b) In how many of the 1,000 experiments can we expect that the null hypothesis will be correctly rejected?

(c) Now consider the total number of experiments in which we expect to reject the null hypothesis. In what percentage of these will the null hypothesis actually be correct? ♢

Exercise 4.7 (Multiple testing)**.** A research team is conducting an experiment in 30 school classes simultaneously. In each school class, the children are assigned to the control or experimental group using complete randomisation. That is, the researchers are in effect conducting 30 parallel and independent experiments.

Assume that in all of these classes, the null hypothesis is true. Further assume that, if the null hypothesis is true, there is exactly a 5% chance of observing a statistically significant difference between the control and experimental conditions.

What is the probability of obtaining a statistically significant difference in at least one of the 30 experiments? ♢

4.5 *Monte Carlo rerandomisation p-values*

Exhausitive rerandomisation is computationally impossible for larger samples[4] and may be infeasible for more complex research designs. One alternative is to use a Monte Carlo simulation: Instead of generating all possible rerandomisations, we only generate, say, 20,000 of them. Then, we compute the proportion of the rerandomisations considered that resulted in test statistic values at least as extreme as the one we actually observed; this proportion serves as the Monte Carlo p-value. The guarantee encapsulated in Lemma 4.4 also holds for such Monte Carlo p-values. However, if you run the same analysis multiple times, the resulting p-value is bound to vary somewhat from run to run due to the inherent randomness in the simulation.

[4] How many ways are there to split up 40 participants into two equal-sized groups?

4.6 *Analytical shortcuts*

Statistical inference predates computers, and analytical shortcuts were developed to circumvent the tedious computations required for rerandomisation tests. These shortcuts include the t-test, the χ^2-test, and ANOVA, as well as their generalisations. They typically produce similar results to those produced by rerandomisation tests and are usually used instead. In the context of experiments with random assignment, the p-values that these procedures return have the essentially same interpretation and are subject to the same caveats as those above. That said, they are based on a different set of assumptions from rerandomisation tests. Typically, the guarantee from Lemma 4.4 only holds approximately when using these shortcuts (i.e., $\mathbb{P}(p \leq \alpha) \approx \alpha$ under H_0).

4.7 *Statistical power*

A study's statistical power is the probability with which its significance test will yield $p \leq \alpha$, for some predetermined $\alpha \in (0, 1)$. In studies in which one group is compared to a different group, this probability depends on three factors apart from α itself (see Figure 4.5):

1. The size of the difference in the outcome between the groups that the systematic factors cause. Even if they don't cause any difference, it is possible to obtain a statistically significant difference due to chance (see table above).
2. The number of observations.
3. The variability in the outcome variable within each group.

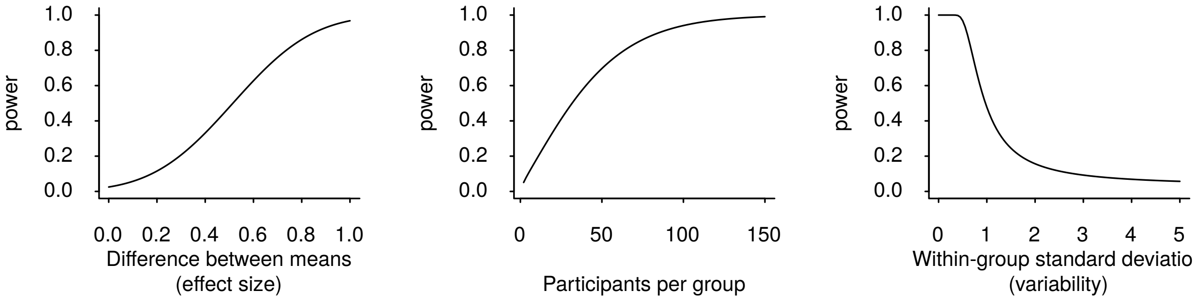


Figure 4.5: These three graphs show how the statistical power of a study varies with the effect size (left), the number of observations per group (middle) and the variability in the outcome variable within each group (right).

The precise numbers along the y-axis in Figure 4.5 aren't important; what's relevant is the direction and the shape of the curves.

Exercise 4.8. Take a look at Figure 4.5 and answer the following questions:

(a) How do the effect size, the number of observations and the within-group variability in the outcome affect the probability that a study will yield a statistically significant result?

(b) Other things equal, what yields a greater improvement in a study's power: 10 additional participants per group when each group already consists of 10 participants, or 20 additional participants per group, when each group already consists of 50 participants?

(c) How could researchers reduce the within-group variability in the outcome variable in order to increase their statistical power? ♢

Some further exercises to wrap up this chapter.

Exercise 4.9. *p*-values are commonly misinterpreted. By way of preparation for the next exercise, answer the following questions.

(a) What, roughly, is the probability that, when you'll die, it'll be because a shark bit your head clean off?[5]

(b) What, roughly, is the probability that, when a shark bites your head clean off, you'll die?[6]

(c) Is 0.087 the probability that the null hypothesis in the alcohol example is correct? If not, then which probability exactly does this *p*-value of 0.087 refer to? ♢

Exercise 4.10. Consider the following vignette and some possible interpretations of the results reported in them. Decide for each interpretation if it follows logically from the vignette and the correct definition of the *p*-value. Explain your reasoning.

Vignette: In an experiment that was carried out and analysed rigorously, we find that the mean difference between the control and intervention groups amounts to 5 points on a 100-point scale. This difference is "statistically significant", with a *p*-value of 0.02.

(a) It's unlikely that we would have found a difference of 5 points or larger between both groups if the null hypothesis were indeed true. More precisely, this probability would have only been 2%.

(b) The null hypothesis is incorrect; the alternative hypothesis is correct.

(c) It's unlikely that the null hypothesis is indeed correct. More precisely, the probability that it is correct is only 2%.

(d) It's highly likely that the alternative hypothesis is correct. More precisely, the probability that it is correct is 98%.

(e) A new but similar study would likely yield a low *p*-value as well. More precisely, there is a 98% probability that such a study would yield a significant *p*-value (i.e., $p \leq 0.05$).

(f) If we concluded that the alternative hypothesis is correct, we would be wrong at most 2% of the time.

[5] In the notation of probability theory, you'd write this as $\mathbb{P}(\text{head bitten off by shark} \mid \text{dead})$.

[6] I.e., $\mathbb{P}(\text{dead} \mid \text{head bitten off by shark})$.

(g) If we concluded that the alternative hypothesis is correct, we would be wrong at most 5% of the time. ♢

*4.8 *Examples in R*

See the file `rerandomisation_tests.html` in the `tutorials` directory on `https://github.com/janhove/QuantitativeMethodology`.

*4.9 *Further reading*

The blog entries *Explaining key concepts using permutation tests* and *A purely graphical explanation of p-values* may be of some use. For an explanation in German, see Chapter 13 of my statistics booklet (available from `https://janhove.github.io`). Goodman (2008) discusses some common misinterpretations of *p*-values; his list is far from exhaustive.

Analysing quantitative data and running significance tests aren't synonymous; see my booklet as well as Winter (2019) for introductions to statistics for linguists that don't emphasise signficance testing. Nonetheless, quantitative research in the social sciences, including in applied linguistics, has developed something of a significance fetish, with authors inundating their readers with significance tests and *p*-values that they themselves don't seem to really understand while giving them little insight into what the data actually look like. For further lamentations and some suggestions, see Vanhove (2021).

5
Increasing precision

5.1 Precision

Up till now, our chief goal has been to increase the study's internal validity:

- Bias introduced by confounding can be countered by randomly assigning the participants (or whatever is being investigated) to the conditions. This won't always be possible, but randomisation remains the ideal.
- Bias introduced by expectancy effects, especially on the part of the researchers, can be reduced by blinding—for instance, by preventing raters from knowing which experimental condition the participant was assigned to.
- To decrease the chances that the results are affected by technical glitches or misunderstandings, the experiment should be piloted, and checks for comprehension and satisficing can be incorporated.

These three points concern bias—we want to prevent our study from systematically under- or overestimating the answer to the question we're interested in, which is often a causal question. But as we saw when discussing statistical tests, there is a random element to the results of any given study (see Remark 2.4 on page 27). In a study with random assignment, the luck of the draw may produce an estimated effect that is larger or smaller than the actual effect—it's just that randomisation helps to prevent this estimate from being *systematically* too large or too small. Roughly speaking, randomisation is equally like to yield overestimates as it is to yield underestimates.

But *an estimate obtained from an unbiased study can be completely off-target.* We've demonstrated this in Remark 2.4, but to better appreciate this, consider a six-sided dice. The average number of pips on a six-sided dice is $\frac{1+2+3+4+5+6}{6} = 3.5$. Let's pretend we didn't know this and we wanted to estimate this number (i.e., 3.5) by throwing the dice and jotting down the number of pips showing face-up.

- If we do this just once, we'll just obtain an integer between 1 and 6 with equal probability (six possibilities). If we obtain a 1, iyr estimate will be off by 2.5 pips; if we obtain a 2, we'll be off by 1.5 pips; ...; if we obtain a 6, we'll be off by 2.5 pips. Taking into account all six possibilities, our average estimation error is 1.5 pips. Note that the estimation procedure itself is unbiased: underestimates and overestimates are equally likely to occur, and they're of the same size, so they will cancel each other out.
- If we throw the dice twice, we'll now observe one of $6^2 = 36$ possible outcomes. When we average the number of pips on both throws, we can still obtain an estimate of 1 (when you throw two 1s), but there's just a 1-in-36 probability of that happening. Six of the possible outcomes will be right on the mark (1+6, 6+1, 2+5, 5+2, 3+4, 4+3). Taking into account all 36 possibilities, our average estimation error is 0.97 pips. Again, this estimation procedure is unbiased.
- If we throw the dice five times, we'll observe one of $6^5 = 7776$ possible outcomes. When we average the number of pips on the five throws, there's just a 1-in-7,776 probability that we'll end up estimating the average number of pips on the dice as 1. Taking into account all 7,776 possibilities, our average estimation error is 0.62 pips. Again, this estimation procedure is unbiased.

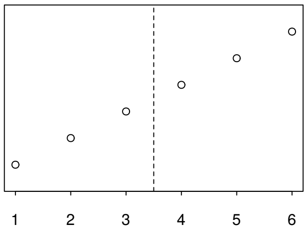


Figure 5.1: If we throw a six-sided dice once, we'll observe one of these 6 outcomes. The dashed vertical line highlights the true mean number of pips.

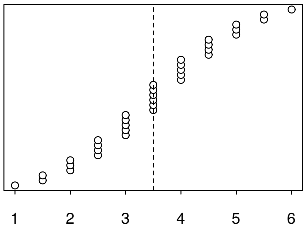


Figure 5.2: If we throw a six-sided dice twice and take the mean number of pips observed, we'll obtain one of these 36 outcomes.

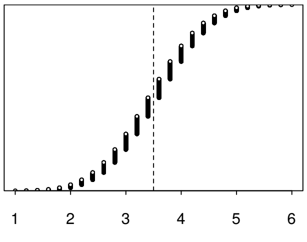


Figure 5.3: If we throw a six-sided dice five times and take the mean number of pips observed, we'll obtain one of these 7,776 outcomes.

So as we increase the number of throws (i.e., as we increase the sample size), the average observation tends to correspond more closely to the true average. Put differently, our estimate tends to become more **precise**. It's still *possible* to be completely off mark, but it's less *probable*. Clearly, the third 'design' (throwing the dice 5 times) is preferable to the first and second design—not because it's unbiased (all three attempts are unbiased), but because the estimate it yields is expected to be closer to the truth.

In a similar vein, even unbiased studies can often be improved upon by taking steps that increase their precision. The precision of an estimate obtained in a study can itself be estimated and is typically expressed by means of **standard errors**, **confidence intervals**, or **credible intervals**. We won't concern ourselves here with how these statistics are to be calculated and interpreted; a rough appreciation of precision along the lines of the dice example suffices.

5.2 *Factors affecting precision*

Precision is affected mainly by the following two factors:

- the number of data points. Other things equal,[1] larger studies yield more precise estimates. As the dice example illustrates, the effect of increasing the sample size yields diminishing returns: the same number of added observations results in a greater increase in precision if the original sample is small compared to when it is large.

[1] This phrase is crucial. There is often a trade-off between the sample size and the quality of the data.

- the variability of the data within each group. The more variable the data within the groups are, the less precise the estimates will be. (In the dice example, the estimation error would be lower if our dice didn't have the values 1 and 6.) For the exercises in Section 4.7, you may have already identified a couple of ways to reduce the variability of the data within the groups (e.g., restricting the study to a more homogeneous group; using more precise measurements). But we can also reduce this variability through a combination of experimental design and statistics, see Sections 5.3 and 5.4.

Note that both of these factors also affect statistical power in the same way (see Figure 4.5 on page 42). Indeed, high power usually translates into high precision.

5.3 *Matching and blocking*

Matching Matching is a procedure in which researchers manually assign the participants (or whatever is being investigated) to the different conditions in such a way that both conditions are comparable on one or a number of background variables.

- Actual meaning: For each participant in condition A, find a similar participant (e.g., same age, sex and L2 skills) and assign this participant to condition B. This way, each participant has a counterpart in the other condition.
- What is often meant: Assign participants to conditions A and B in such a way that the *average* age (etc.) is similar or the same in both conditions. The individual participants themselves don't need to have any particular counterpart in the other group.

The rationale behind matching is that, by equating the conditions on one or a number of background variables, these variables can't act as confounding variables. However, matching is **not recommended**: It's possible that the researchers, while matching the participants on one background variable, inadvertently introduce a bias with respect to another background variable. Moreover, (pure) matching only allows you to equate those confounding variables that you matched for (see Figure 5.4). Randomisation also equates *other* (and indeed unknown) confounding variables and is superior to matching.

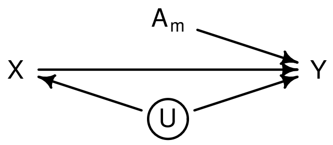


Figure 5.4: Matching the conditions (X) on A doesn't prevent confounding by other (perhaps unobserved) variables (U).

Blocking This is similar to matching, but unlike (pure) matching, it is used *in combination with* (rather than as an alternative to) randomisation:

> "[M]atching is no real help when used to overcome initial group differences. This is not to rule out matching *as an adjunct to randomization*, as when one *gains statistical precision* by assigning students to matched pairs, and then randomly assigning one member of each pair to the experimental group, the other to the control

> group. In the statistical literature this is known as 'blocking."' (Campbell & Stanley, 1963, p. 15; my emphasis)

- Example 1: Based on a pool of participants, we build pairs of participants of the same sex and age and with a similar IQ. From each pair of participants, we *randomly* (rather than arbitrarily) assign one participant to condition A and one to condition B. In doing so, we both equate the two groups in terms of age, sex and IQ, but, due to the randomised assignment, we also prevent confounding by unobserved factors.[2]
- Example 2 (see Ludke et al., 2014): We randomly assign half of the female participants to condition A and half to condition B; same for the male participants. Again, randomised assignment helps to prevent confounding variables from biasing the results, and we have the added benefit that the two conditions will be perfectly balanced in terms of sex.

Blocking can increase a study's statistical precision—provided it is taken into account during the analysis. The stronger the outcome is related to the blocking factors, the more powerful blocking is.

Note that blocking takes place *before* the random assignment. You can't block after the fact.

Remark 5.1 (Blocking and rerandomisation testing)**.** If you incorporate blocking in the design of your study, but you don't take this into account when analysing the data, you're not reaping its full benefits.

If you're testing some association using rerandomisation as discussed in the previous chapter, the idea is that you only generate randomisations that you could actually have obtained in your study. For instance, if you block your participants into k pairs based on sex, age, and IQ as in the first example above, and then randomly allocate the participants to the conditions within each pair, there are 2^k possible randomisations instead of $\binom{2k}{k}$ ones. You should then only generate randomisations in which participants belonging to the same block are assigned to different conditions.

Similarly, in the second example, only randomisations that split the participants of each sex equally among the conditions should be considered.

Further see Vanhove (2015) for some other options that don't involve rerandomisation testing. ♢

How does blocking increase precision? Let's say you're comparing the efficacy of two methods for learning Dutch as a foreign language. Six German-speaking and eight French-speaking learners sign up for your study, they work with one of the two learning methods for a while, and then take the same test at the end.[3]

- The speakers of German can be expected to have an advantage

[2] This technique is rarely used in our line of research, possibly because the pool of participants is rarely known at the start of the experiment.

[3] Again, the number of participants in this fictitious example is low to keep things tractable.

because of the similarity between Dutch and German. Let's say this advantage corresponds to 3 points on a 20-point test scale. (Realistically, you wouldn't be able to peg this number down so precisely.)

- Within each language group, learners still vary in their ability to learn Dutch.
- Let's say that, unbeknownst to you, learning method *B* boosts test performance by two points relative to learning method *A*. Figure 5.5 shows what each learner's scores would have been if they had been tested on both methods.

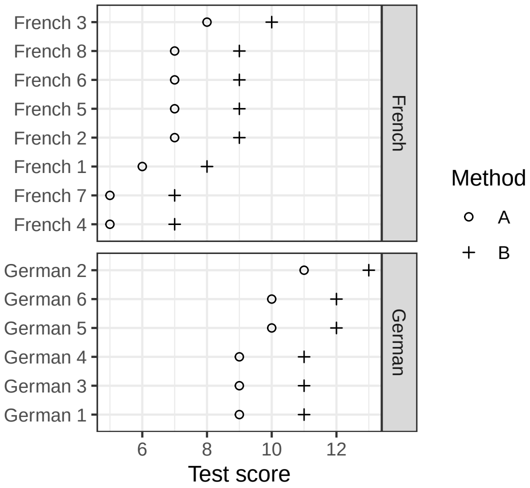


Figure 5.5: The test scores that each learner would obtain for each learning method. But we can't test all learners on one and the same method, so for half of the learners, we'll observe their scores for Method A, and for the other half, we'll observe their scores on Method B.

There are $\binom{14}{7} = 3432$ ways to randomly assign these 14 participants to two conditions (*A* and *B*) with 7 participants each. For the learners in Condition *A*, we'd observe the scores shown as circles in Figure 5.5; for the learners in Condition *B*, we'd observe the scores shown as crosses. If we then took the mean difference between these groups, we'd end up observing one of the 3,432 values shown in Figure 5.6 on the next page. Complete random assignment would yield a mean estimation error of about 0.80 points.

However, we've identified a likely important source of variability in the data: language group. It makes sense to block on this factor, i.e., restrict the random assignment in such a way that half of the learners in each language group are assigned to one learning method and the other half to the other learning method. Of the 3,432 total possible random allocations, only 1,400 feature four French speakers in one condition and four in the other, as well as three German speakers in one condition and three in the other.[4] The average estimated difference between the two learning methods among these 1,400 allocations is still 2, but the average estimation error is now only 0.63—an increase in efficiency of about 21%; see Figure 5.7. What has happened is that, by restricting the randomisation in this fashion, we limited both the number of allocations that would have yielded large overestimates (when the Method B condition would've consisted mainly of advantaged German speakers)

[4] There are $\binom{8}{4} = 70$ ways to split up the eight French speakers into two groups of four, and $\binom{6}{3} = 20$ ways of splitting up the German speakers into two groups of three. Combining these yields $70 \cdot 20 = 1400$ possibilities.

and those that would have yielded large underestimates (when the Method B condition would've consisted mainly of disadvantaged French speakers). In our example, complete randomisation resulted in 412 out of 3,432 (12%) allocations with an absolute estimation error of more than 1.5 points; blocked randomisation resulted in only 60 out of 1,400 (4%) such allocations. Also see Table 5.1 on the facing page.

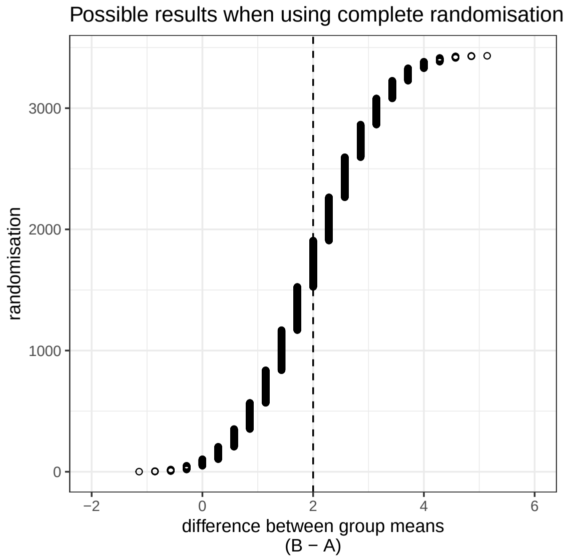


Figure 5.6: Across all 3,432 possible random assignments, the mean estimation error of the difference between the two learning methods is about 0.80 points. The vertical line highlights the average estimate across all 3,432 possible random assignments.

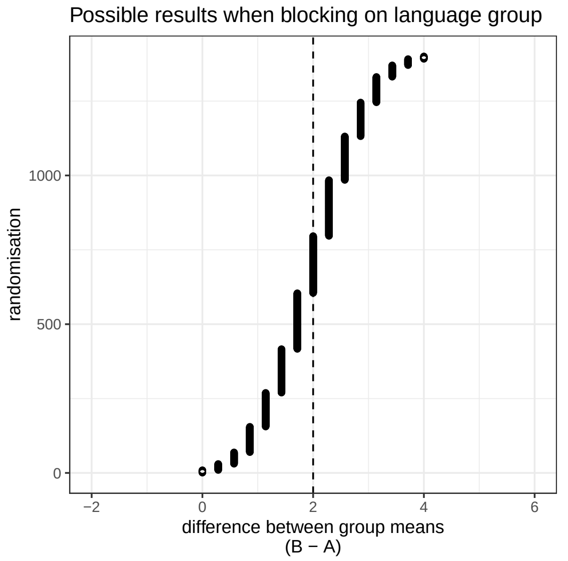


Figure 5.7: Of the 3,432 possible random assignments, only 1,400 have an equal number of French speakers assigned to each learning method, as well as an equal number of German speakers assigned to each learning method. Across these 1,400 assignments, the mean estimation error is just 0.63. The average estimate, highlighted by the vertical line, doesn't change.

So blocking on influential factors prevents the randomisation from generating some of the 'unlucky' allocations, thereby reducing the study's average estimation error, i.e., increasing its precision.

The higher precision obtained by blocking is reflected in an increase in statistical power. In this particular example, the rerandomisation-based significance test presented in the previous chapter will return a p-value of 0.05 or less in 1,165 out of the 3,432 possible allocations when using complete randomisation, that is, it has 34% power. But

Interval	Complete randomisation	Blocked randomisation
(-1.5,-0.5]	0.52	0.00
(-0.5,0.5]	5.48	2.14
(0.5,1.5]	28.03	27.57
(1.5,2.5]	31.93	40.57
(2.5,3.5]	28.03	27.57
(3.5,4.5]	5.48	2.14
(4.5,5.5]	0.52	0.00

Table 5.1: In what percentage of randomisations did the mean difference end up in each interval? Note that for blocked randomisation, a greater percentage of randomisations yield a mean difference close to the true difference between the learning methods (i.e., 2) than for complete randomisation.

when using blocking, such a significance test will return a p-value of 0.05 or less in 642 out of the 1,400 possible allocation, that is, it has 46% power.

5.4 *Leveraging control variables*

Control variable Additionally collected variable that isn't of actual interest but that may account for differences between participants in terms of the outcome.

Example: the 'language aptitude test' in Ludke et al. (2014).

In randomised experiments, the added value of control variables is mostly statistical: If control variables can account for differences in the outcome between participants, they can be used to statistically reduce the variability within the groups. Similarly to blocking, this yields greater power and precision.[5] Note that the use of a control variable to this end is planned before the data are collected. Don't try out a bunch of 'control variables' during your analysis to see which works best!

Pretest Often, the most potent indicator of a participant's performance at the end of the experiment is their performance at the start of the experiment. A pre-intervention measure of their performance—be it in the form of a score on a full-fledged pretest or some rougher proxy—is therefore a useful control variable.[6]

It's also possible to 'block' on pretest scores. To this end, sort the participants according to their pretest score and divide them up into pairs like so: (12)(34)(56)(78).... Within each pair, randomly assign one participant to the control group and one to the intervention group. You can similarly block on other continuous variables.

Pre- and post-tests don't have to look identically. Any measure of pre-experiment performance is better than no measure at all.

Of course, if the pre- and posttests aren't similar and can't be scored on the same scale, you won't be able to make any claims

[5] Contrary to common belief, including powerful control variables in the analysis is useful *even if* the groups are balanced with respect to these control variables. In fact, they're even more useful than when the groups aren't balanced.

[6] On taking into account pretest results, see Vanhove (2015).

about how much the participants progressed in each condition. *But that's not important!* What's important in a pretest/posttest design is the comparison between the conditions on the posttest scores. The purpose of the pretest scores is to increase the precision of this comparison. This can be achieved by using them as you would any other blocking or control variable, so they don't have to be expressed on the same scale as or be otherwise comparable with the posttest.

In fairly small studies, blocking tends to increase precision a bit more than merely using control variables in the analysis, but in the vast majority of cases, either is a good idea compared to the alternative of not leveraging any prior information![7]

Even if you're conducting a randomised experiment, it pays to think about which factors are likely to strongly affect the outcome so that, if feasible, you can take these factors into account using blocking or by means of control variables.

Don't go overboard with this, though. One or two strong blocking or control variables are likely to be helpful; umpteen variables that *might conceivably* bear some relation to the outcome aren't. Using several highly intercorrelated control variables doesn't hurt but it isn't too useful either: they will all tend to do the same work, which makes them mutually superfluous.

Exercise 5.2. Having taking this class, you've become the go-to expert on experimental methodology among your friends. So unavoidably, a friend of yours sollicts your input on an experiment she wants to run with one experimental condition and one control condition in a Zurich-based school class with 24 pupils. For each pupil, your friend knows if they have Swiss-German as their main language and whether they receive special educational measures. She also knows the pupils' average school grades. These data are shown in Table 5.2 on the next page.

After some discussion with your friend, you realise that, ideally, she should block on language background, whether the pupils receive special educational measures as well as average grade when randomly allocating the pupils to the experiment's condition. An equal number of pupils is to be assigned to both conditions.

(a) Explain, as you would to your friend, how to generate a suitable allocation of pupils to conditions. Also generate such a suitable allocation in order to illustrate the steps involved. List the pupil IDs of the pupils that would be assigned to the control condition under the allocation you generated. Feel free to use your spreadsheet software or R for this part.

(b) How many different allocations could your scheme generate? How many different allocations could complete randomisation without blocking have generated? Provide both the computa-

[7] We don't need to concern ourselves with the freak cases where blocking or using control variables reduces precision (viz., tiny studies in which the blocking or control variables are uninformative with respect to the outcome; Imai et al., 2008.)

Pupil ID	Main language	Special measures?	Average grade
S01	Swiss-German	no	4.9
S02	Swiss-German	no	5.4
S03	Swiss-German	yes	4.2
S04	other	yes	3.5
S05	Swiss-German	no	4.0
S06	Swiss-German	no	4.6
S07	Swiss-German	no	6.0
S08	Swiss-German	no	3.9
S09	Swiss-German	no	4.7
S10	other	yes	5.0
S11	Swiss-German	yes	5.1
S12	other	no	4.2
S13	other	yes	3.9
S14	Swiss-German	no	4.5
S15	other	yes	4.2
S16	Swiss-German	yes	4.5
S17	other	yes	3.0
S18	other	no	4.2
S19	other	no	5.3
S20	other	yes	2.7
S21	other	no	4.6
S22	Swiss-German	no	5.3
S23	Swiss-German	yes	4.9
S24	Swiss-German	no	4.7

Table 5.2: Fictional pupil characteristics.

tions as well as the numeric results. ◇

Don't control for post-treatment variables! A fairly common error researchers make is controlling for variables that are themselves (directly or indirectly) affected by the treatment. The reason is that controlling for a descendant of a variable (when drawn in a DAG) is like controlling for the variable itself, only less strongly.

- If you 'controlled' for the treatment variable (e.g., throwing away data in order to keep it constant), you wouldn't be able to compare the outcome variable according to different values of the treatment variable (since there aren't any). Controlling for a descendant of the treatment variable (even by statistical means rather than by selecting observations) similarly amounts to throwing away data, just to a lesser extent. Rather than increase power and precision, you'll lose some.

- If you 'controlled' for the outcome variable, you wouldn't be able to find any differences between the treatment groups even if the treatment produced some differences (since you fixed all outcome observations to the same value). Similarly, controlling for a descendant of the outcome variable (even by statistical means) typically amounts to artificially pulling the differences between the treatment groups towards zero.

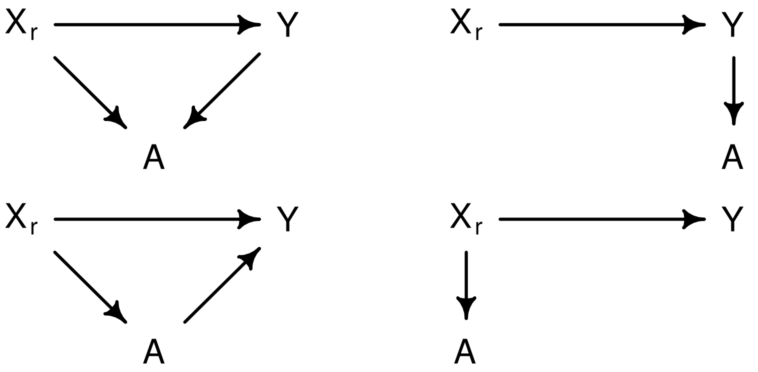


Figure 5.8: In all four DAGs, A is causally affected by X_r.

Top left: A is a collider. Controlling for it opens up a non-causal path between X_r and Y.

Bottom left: Controlling for A closes a causal path from X_r to Y. You'd be answering the question *What's the causal effect of X_r on Y other than that mediated by A?*. This may be interesting in its own right, but you have to be aware that this question differs from the question *What's the causal effect of X_r on Y?*

Top right: Controlling for A is like controlling for Y, just less strongly. If you want to estimate the causal effect of X_r on Y, controlling for Y would be a dreadful idea, so controlling for A would only be a slightly less dreadful idea.

Bottom right: Controlling for A isn't as terrible here as it is in the top right panel. But it's unnecessary, and you'll lose some precision.

See the DAGs in Figure 5.8 for these and two other cases.

Example 5.3. Say you want to find out if a pedagogical intervention boosts learners' conversational French skills. It may be a good idea to control for the learners' vocabulary knowledge. But if you collect the measure of vocabulary knowledge *after* the intervention, it's possible that this measure is also affected by the intervention. If you control for it, you could find yourself in one the situations depicted in Figure 5.8: learners could conceivably pick up some vocabulary as they're working on their conversational skills. ♢

A poorly chosen pretreatment control variable won't be too helpful, but it won't hurt your study either—other than in terms of time spent and money wasted. But controlling for a posttreatment variable can bias your results or decrease their precision. Luckily, in true experiments, there's a simple solution:

> Collect control variables at the outset of the study (before the intervention) so that you're sure that the control variables aren't themselves influenced by the intervention.

Exercise 5.4. Consider the DAG in Figure 5.9.

(a) If the goal is to obtain an unbiased estimate of the causal influence of X on Y, regardless of precision, which variable or which variables do you *need* to control for?

(a) Assume that all variables shown in the DAG have already been collected (i.e., there are no further costs involved in collecting them). If the goal is to obtain an unbiased and maximally precise estimate of the causal influence of X on Y, which variable or which variables would you *need* to control for? ♢

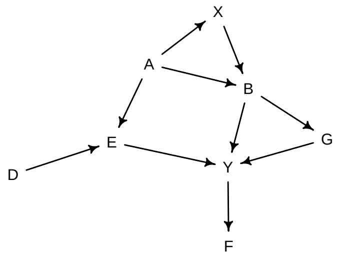


Figure 5.9: DAG for Exercise 5.4.

5.5 *Examples in R

See the file `blocking.html` in the `tutorials` directory on `https://github.com/janhove/QuantitativeMethodology`.

6 Pedagogical interventions

The following remarks are especially relevant to pedagogical interventions, but they apply to other studies, too.

6.1 Sample mortality

Sample mortality refers to research participants who become unavailable in the course of a study. In a school-based study, examples include children who can't participate in a follow-up test because of a dental appointment, because they switched schools, or quite simply, because they don't want to. Less lurid and more descriptive terms for sample mortality are *drop-outs*, *outmovers*, and *panel attrition*.

In addition to lowering the sample size, drop-outs may bias the results of the study. As Figure 6.1 shows, being a drop-out or not can be a post-treatment factor, but one that has insidiously already been controlled for: the participants whose data goes into the analysis all have the same value on this variable.

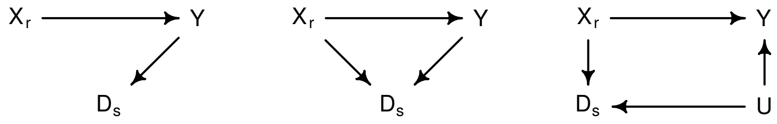


Figure 6.1: The fact that some participants stayed in the study and others can be treated as a factor in its own right (D_s; D for 'drop-out', $_s$ for making clear that selection took place).

Ideally, mortality doesn't depend on the condition (X), the participants' prior knowledge or other characteristics (U), or their progress (Y). When mortality does vary by condition, prior knowledge or learning progress, you have take into account the selection effect when interpreting the results.[1]

Figure 6.2 on the following page illustrates how mortality can bias the study's estimates. It is in fact possible that an observed 'treatment' effect is little more than a selection effect: If only gifted or highly motivated learners remain part of the treatment condition but the control condition isn't as selective, it's hardly surprising that at the end of the study, you'll find better scores in the treatment condition than in the control condition.

[1] In a study with two measurement times, assessing learning progress for the drop-outs is impossible. But if you have several measurement times, you can check if those who progressed little thus far were more likely to drop out of the study.

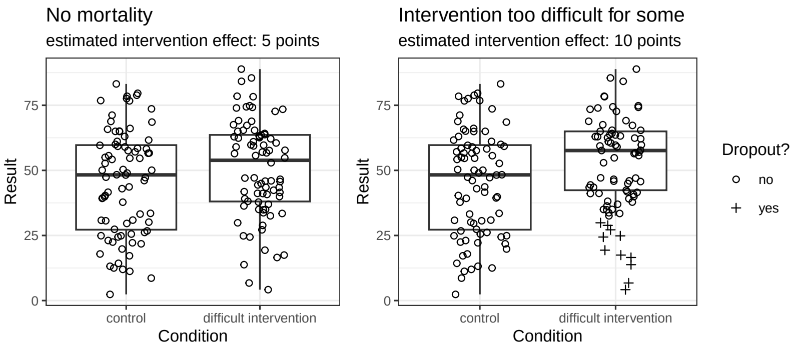
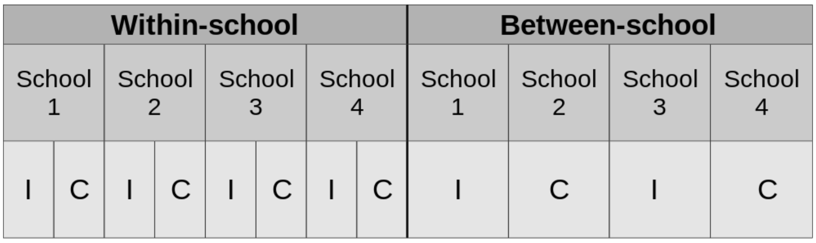


Figure 6.2: Example of the biasing effect of sample mortality. *Left:* Had it been possible to test all participants, we'd have found a mean difference of 5 points. *Right:* If the drop-out likelihood is itself affected by the intervention, we could end with a biased estimate.

6.2 *Clustering*

In typical pedagogical settings, the participants can't be randomly assigned to the experiment's conditions on an individual basis. Instead, entire intact groups of participants (e.g., entire classes, entire schools, entire school districts etc.) are assigned to the same condition. This induces **clustering**: Due to teacher/class/school etc. effects, participants belonging to the same cluster (class, school, etc.) tend to be somewhat more alike in their performance than participants belonging to different clusters.

Within- vs between-school design See Figure 6.3. In a within-school (or similarly, within-class etc.) design, the school effect is neutralised using blocking, whereas the remaining possible confounding variables are ideally taken care of using randomisation. This increases precision relative to a between-school design. One possible drawback of a within-school design is that the pupils in the control and treatment classes in the same school influence each other (e.g., by comparing notes or helping each other make sense of what's being taught), which may wash out an existing treatment effect.



Within-school								Between-school			
School 1		School 2		School 3		School 4		School 1	School 2	School 3	School 4
I	C	I	C	I	C	I	C	I	C	I	C

Figure 6.3: Within- vs between-school designs. (*I* for intervention, *C* for control.)

Cluster-randomised design When clusters are assigned in their entiry to the same condition (e.g., all pupils in the same class are assigned to the same learning condition rather than each on an individual basis).

In a cluster-randomised design, you need to take the cluster-randomisation into account when analysing the data.

To appreciate the need for taking clustering into account during the analysis, consider an experiment with 6 classes of 10 pupils each. There are over one-hundred *quadrillion* ways to split up 60 pupils into two groups of 30 ($\binom{60}{30} \approx 1.2 \times 10^{17}$). But there are only 20 ways to split up six classes into two groups of three classes each. The analysis needs to be based on the assumption that the allocation obtained is one of 20 possible ones, not one of a gazillion ones.

Remark 6.1 (Analysis of cluster-randomised designs)**.** One simple and valid approach for analysing data obtained with a cluster-randomised design is to compute the mean (or some other kind of average) of each cluster and then analyse these cluster-level summaries using one of the methods covered in Chapter 4. That said, lots of alternative approaches exist; see Vanhove (2015), Vanhove (2020), and references therein. ♢

What you also need to know about cluster-randomisation is this:

> Studies with one intact group as the experimental group and another intact group as the control group are useless.

Conceptually, the reason is that class, school and teacher effects can't be separated from the effect of the intervention if you just have one intact group per condition. If you use a rerandomisation test as per Remark 6.1 but you only have a single cluster per group, then there are only two possible allocations. As a result, the left- and right-sided p-value will both be at least 0.50, and the two-sided p-value will always be 1.

Exercise 6.2. Let's say you want to run a pedagogical experiment (e.g., to compare two learning methods for French as a foreign language) in which randomisation has to take place at the class level rather than at the individual level. Other things equal (e.g., number of classes, number of pupils), what are the advantages and drawbacks of the following designs? What's the worst option, and why? What's the best, and why?

(a) All classes are taught by the same teacher.

(b) Each class is taught by a different teacher.

(c) One teacher teaches all classes in the control condition, and another teacher teaches all classes in the intervention condition.

(d) Each teacher teaches two classes: one in the control condition, and one in the intervention condition. ♢

Exercise 6.3 (Reading assignment)**.** The article by Slavin et al. (2011) makes for pretty challenging reading, especially in terms of the analysis and the way the results are presented. But the introductory and methodological sections discuss some concepts that we've already discussed (especially p. 49).

First try to read the text in full, but skip the parts you find unintelligible. Then answer the following questions:

(a) What is the independent variable in this study? What are the dependent variables?

(b) How were the pupils assigned to the different groups?

(c) Slavin et al. discuss at length how many pupils in each group (TBE vs SEI) couldn't be tested (Table 2) and whether the characteristics of these pupils differed between the conditions (Table 3). Why do you think they discuss this at all?

(d) "Children were pretested ... on the English Peabody Picture Vocabulary Test (PPVT) and its Spanish equivalent, the Test de Vocabulario en Imagenes Peabody (TVIP)." (p. 51, right) Why did the researchers go to the bother of conducting such pretests? Try to find at least two reasons.

(e) Comparing Slavin et al.'s Tables 4 and 6, we observe that the cohorts' average TVIP (*Test de Vocabulario en Imagenes Peabody*) scores dropped from 99.85 and 90.19 to 92.86 and 85.64, respectively, over the course of two years.

Come up with the most plausible explanation for this result.[2]

♢

6.3 *Examples in R

See the file `clusters.html` in the `tutorials` directory on `https://github.com/janhove/QuantitativeMethodology`.

[2] Hint: The children's Spanish vocabulary knowledge probably did not decline on average as they got older.

7
Within-subjects experiments

7.1 Advantages and drawbacks

Blocking increases power and precision by pairing up similar participants and randomly assigning one of each pair to each condition. In within-subjects designs, this idea is taken to an extreme: the *same* participants are tested in the different conditions.

In a within-subjects experiment, every participant serves as their own control.

Advantage 1: Easier to explore interindividual differences With a between-subjects experiment, you can only estimate the average effect of an intervention. With a within-subjects experiment, you could additionally gauge which participants gain more from an intervention than others.

Advantage 2: Statistical precision A study's statistical precision depends on (a) the amount of data and (b) the variability in the data. The *main* (!) advantage of a within-subjects design is that it easily accounts for an important source of variability: interindividual differences.

How much more precise a within-subjects experiment is than a between-subjects experiment varies from case to case.[1]

Possible drawback 1: Lack of ecological validity In applied settings, you typically want the study to mimic the context in which its findings are to be implemented. But in such a context, people (e.g., pupils) won't be exposed to several conditions (e.g., learning methods) but rather to just one.

Possible drawback 2: Order and carry-over effects When participants are tested in several conditions, it's possible that they learn something in one condition that affects their performance in the other condition (**carry-over effect**). It's also possible that their performance in the last condition differs from their performance in the first condition because they've grown accustomed to the setting or because they've grown tired of being tested (**order**

[1] Quené (2010) estimates that within-subjects designs have the statistical precision of between-subjects designs with four times as many participants. The precise factor depends on the extent to which the participants' performance in one condition correlates with their performance in the other condition: The stronger this correlation, the greater the added value of a within-subjects experiment. But even if you can't quantify this added value: Within-subjects designs offer more statistical precision.

effects).

7.2 *Minimising order effects*

Complete counterbalancing To prevent learning or fatigue effects from exerting a systematic effect on the results, you can vary the order of the conditions between the participants. In complete counterbalancing, *all* possible orders are taken into account. If you have two within-subjects conditions, half of the participants first complete condition *A* and then *B*, and the other half first complete condition *B* and then *A*. If you have three conditions, there are $3! = 6$ possible orders; you can then randomly assign one sixth of the participants to first complete A, then B, then C; one sixth completes A, then C, then B, etc.:

A	A	B	B	C	C
B	C	A	C	A	B
C	B	C	A	B	A

Table 7.1: Complete counterbalancing for a within-subjects experiment with three conditions.

Latin squares If a within-subjects experiment has lots of conditions, complete counterbalancing is impractical. For four conditions, for instance, there are already $4! = 24$ possible orders—we may not even have that many participants! The Latin square lends itself to such cases.[2] Latin squares are arrangements of symbols in a grid in which each of the symbols used occurs exactly once in each row and exactly once in each column. The grid below is a Latin square of size 4—one of the 576 possible arrangements of the symbols A, B, C, and D that form a Latin square. (Can you come up with a couple of the other 575 ones?)

A	B	C	D
B	C	D	A
C	D	A	B
D	A	B	C

Table 7.2: A Latin square for a within-subjects experiment with four conditions.

Let's say you picked the Latin square above for your study. You'd then randomly assign one quarter of the participants to the condition (or stimulus set) order ABCD (first row), one quarter to BCDA (second row), one quarter to CDAB (third row) and one quarter to DABC (fourth row). The conditions (or stimulus sets) are randomly assigned to one of the letters, too.

Other possibilities In which order should we show our participants 50 pictures that they are to describe if we want to prevent order effects from biasing the results?[3] $50! = 3 \cdot 10^{64}$ is an astronomical number, and even just 50 different Latin square orders seem impractical. One of several possible solutions is to present the images in a new random order for each participant. The drawback of doing this is that perhaps image 3 occurs much more often at the start than at the end of the data collection.

[2] 'Latin' because the symbols used are typically letters of the Latin alphabet.

[3] Of course, it's possible that we just accept such 'bias' if we aren't interested in differences between the images, but just in differences between the participants. If that's the case, these steps may be superfluous.

In many psycholinguistic studies, participants need to react to several stimuli per condition (e.g., 12 stimuli per condition). The order of the stimuli in these studies are often randomised so that the conditions are mixed up (e.g., $ABAABBBAB$ etc.).

Counterbalancing and Latin squares don't negate carry-over effects. Whether carry-over effects represent an acute danger to the study's validity needs to be judged on a case-by-case basis. That said, the possible danger of carry-over effects quite often isn't large enough to offset the certain gain in statistical precision.

If you have a genuine choice between a between-subjects and a within-subjects design for your own research, pick the within-subjects design. (Unless, of course, you have an excellent reason not to do so.)

Example 7.1. In Vanhove (2019), I examined the influence of metalinguistic knowledge about some structure in the L1 on the participants' intuitions about the corresponding structure in a foreign language. Metalinguistic knowledge was manipulated experimentally. In the control condition (call it A), the participants were given correct but irrelevant information about the L1's morphosyntax. In the second condition (B), they were given correct, relevant information. In the third condition (C), they were given correct, relevant information and they were additionally taught an algorithm for identifying the structure in question.

It's quite clear that this study couldn't be realised in a within-subjects design: Once you give the participants the relevant information (conditions B and C), you can't then expect them to suppress this information as they are tested in condition A. Similarly, once you teach the participants an algorithm for identifying a structure (condition C), you can't then expect them not to apply it in the other conditions. Consequently, I opted for a between-subjects design. ♢

Exercise 7.2. From Ludke et al. (2014):

> Participants were randomly assigned to one of three learning conditions: speaking, rhythmic speaking, and singing. The participants heard 20 paired-associate phrases in English and an unfamiliar language (Hungarian) (...). (...) The 15-min learning period was followed by a series of five different production, recall, recognition, and vocabulary tests for the English–Hungarian pairs.

Re-design this between-subjects experiment as a within-subjects experiment. What would this description look like? For the time being, ignore the *rhythmic speaking* condition.

In your design, you are constrained by the resources Ludke et al. had. This means that you cannot introduce stimuli and tests that were not already used by Ludke et al., and that you have to work with at most 30 men and 30 women as participants. ♢

Exercise 7.3. As in the previous exercise, but with all three conditions. ♢

Exercise 7.4 (Reading assignment)**.** The study by Kang et al. (2013) serves as an example of a within-subjects design. Additionally, it uses some turns of phrase commonly found in research reports:

(a) "The Hebrew nouns were learned in one of two training conditions – retrieval practice or imitation – that were manipulated within subjects across separate blocks and semantic categories." (p. 1261)

  i. What does "manipulated within subjects across separate blocks" mean?

  ii. What does "manipulated within subjects across semantic categories" mean?

(b) "The order of items in each test was randomized for each learner." (p. 1262) Why? Would it have made sense to use the same fixed order of items for all learners?

(c) "In Experiment 2, the order of both tests was counterbalanced across learners..." (p. 1262). This merely means that half of the learners first took Test A and then Test B, whereas the other half first took Test B and then Test A. Would it have made sense to administer the test in the same order to all learners? ♢

7.3 *Analysing data from within-subjects designs with two conditions

7.3.1 AB/BA cross-over design

Consider a within-subjects experiment with two conditions, creatively labelled A and B, in which participants are randomly assigned to the two possible orders AB and BA. This design is called a **AB/BA cross-over design**. We will consider condition A to be the intervention (or treatment) condition and B the control condition, but nothing hinges on these labels. Let's break down the systematic factors that contribute to the first and second measurements in both orders, see Table 7.3.

Order	First measurement	Second measurement
AB	$\beta + \tau$	$\beta + \omega + \kappa$
BA	β	$\beta + \tau + \omega$

Table 7.3: Systematic factors contributing to the measurements in a within-subjects experiment with two conditions. The meaning of the Greek symbols is explained in the running text.

- We assume that there is some baseline β common to all measurements. This common baseline is of no further interest.
- There may be treatment effect τ that contributes to the measurements in condition A (i.e., the first measurement for order AB and the second for order BA), but not to those in condition B.[4]
- There may be an order effect ω that contributes to the second measurements but not to the first measurements.[5]

[4] If there is a treatment effect τ' that contributes to the measurements in condition B, this is equivalent to there being a treatment effect $\tau = -\tau'$ that contributes to the measurements in condition A.

[5] Similarly, if an order effect ω' contributes to the first measurements instead, write $\omega = -\omega'$.

- There may be a carryover effect κ that affects the measurements in condition B but only if B follows A.[6]

In any analysis of these data, we need to assume that there are no carryover effects, that is, $\kappa = 0$. If $\kappa \neq 0$, a within-subjects design was the wrong choice. Under the assumption that $\kappa = 0$, we may estimate the value of τ by first computing, for each participant, the period difference, that is, the difference between their first and their second measurement. For the participants in the AB order, and ignoring any non-systematic effects, we obtain

$$d_{AB} = (\beta + \tau) - (\beta + \omega + \kappa) = \tau - \omega - \kappa = \tau - \omega,$$

since $\kappa = 0$ by assumption. For the participants in the BA order, we similarly obtain

$$d_{BA} = \beta - (\beta + \tau + \omega) = -\tau - \omega.$$

Observe that

$$\frac{d_{AB} - d_{BA}}{2} = \frac{(\tau - \omega) - (-\tau - \omega)}{2} = \tau.$$

The consequence of this is that, under the assumption of no carryover effects, the treatment effect τ can be estimated as half the mean difference in the period differences between the two orders. A rerandomisation test or some analytical approximation thereof can be used to obtain a p-value for the null hypothesis that $\tau = 0$.[7]

7.3.2 *Interleaved conditions*

Consider again a within-subjects experiment with two conditions, A and B, in which n participants are shown a fixed list of words $w_1, \dots, w_m$. For each participant, half of the words are shown in condition A, and half are shown in condition B; the presentation software assigns the words to the conditions randomly and separately for each participant. The participants react to each word, and their response is expressed numerically in some fashion (e.g., accuracy, speed, …).

For a design like this, there is no real sense in which one condition follows another condition for each participant. A sensible way to analyse these data is to compute, for each participant, the difference score between their average response to condition A and their average response to condition B, resulting in difference scores $d_1, \dots, d_n$. We then compute the average of these differences, $\overline{d}$. Under the null hypothesis of no difference, each d_i value could just as likely have had the opposite sign. To generate the distribution of $\overline{d}$ under the null hypothesis, we can randomly flip the signs of the observed d_i values and recompute their average. Exhaustive sign-flipping would generate the full distribution of $\overline{d}$ under the null hypothesis, but it involves generating 2^n $\overline{d}$ values. A Monte Carlo version of this procedure would also work. Once the distribution

[6] Similarly, if a carryover κ' affects A when following B, write $\kappa = -\kappa'$.

[7] In practice, you'll encounter different approaches to analysing within-subjects designs when reading social science studies. Not all of these are grounded in sound statistical theory.

of $\overline{d}$ has been generated or approximated using this sign-flipping, one- or two-sided p-values can be computed as usual. An analytical short-cut to this procedure is the paired t-test on the condition averages per participant.

Remark 7.5 (Within-school/class designs)**.** In within-school or within-class designs (see Figure 6.3 on page 56), the same procedure can be applied: For each school (or class), compute the average score obtained by the pupils in the intervention and the average score obtained by the pupils in the control condition. Then analyse these averages as described above. ♢

7.4 **Further reading*

Latin-square designs are also used in studies other than within-subject experiments, for instance as a technique for blocking. See Richardson (2018) for an overview and some finer points that weren't discussed here; his article is geared towards educational researchers.

7.5 **Examples in R*

See the file `within.html` in the `tutorials` directory on `https://github.com/janhove/QuantitativeMethodology`.

8 Quasi-experiments and correlational studies

Up till now, we've discussed designs that eliminate pre-existing factors as confounders by means of random allocation or by testing all participants in all conditions. We now turn to between-subjects studies without random allocation: quasi-experiments and correlational studies.

Whether a study counts as a quasi-experiment or a correlational study depends on whom you ask. Some researchers confusingly use the term *quasi-experiment* to refer to cluster-randomised experiments, whereas others use the term *pre-experiment* to refer to group comparisons without randomisation. Similarly, different researchers draw the border between quasi-experiments and correlational studies at different places. As far as I'm concerned, the communalities between the two outnumber the differences. For what it's worth, I use the term **quasi-experiment** for group comparisons and **correlational study** when the predictor is continuous. What's important is that, because no random assignment was used, we can't assume that the treatment variable is independent of pre-treatment variables—confounding is a real threat.

Quasi-experiment Group comparison, but the groups weren't constructed using random assignment.

- Example 1: Comparison of pupils with and without an immigration background.
- Example 2: Comparison of children that take heritage language classes and children that don't.

It doesn't matter whether the groups *could* have been constructed using random assignment, just whether they were.

Correlational study No group comparison. Instead, one assesses to what extent variation in an outcome (dependent) variable can be accounted for by differences in one or more continuous predictors (independent variables). The values of these predictors weren't assigned to the units of observation randomly.

- Example 1: Researchers collect IQ and L2 proficiency data in a group of learners and assess how strongly both types of data covary.

- Example 2: Using archival data, researchers gauge how well they can account for whether children will pass their A-levels based on the results of a vocabulary test when the children were 12 years old.

Why carry out quasi-experiments and correlational studies?

> "But just because full experimental control *is* lacking, it becomes imperative that the researcher **be thoroughly aware of which specific variables his particular design fails to control**.
>
> "The average student or potential researcher reading the previous section of this chapter probably ends up with more things to worry about in designing an experiment that he had in mind to begin with. This is all to the good if it leads to the **design and execution of better experiments and to more circumspection in drawing inferences from the results**. It is, however, an unwanted side effect if it creates a feeling of hopelessness with regard to achieving experimental control and leads to the abandonment of such efforts in favor of even more informal methods of investigation.
>
> "[W]e shall ... survey the strenghts and weaknesses of a heterogeneous collection of quasi-experimental designs, each deemed worthy of use *where better designs are not feasible*." (Campbell & Stanley, 1963, p. 34; their emphasis in italics, mine in bold-face)

The goal of quasi-experiments and correlational studies is often to draw causal conclusions, but the findings—for better or for worse—tend to be couched in non-causal language (Grosz et al., 2020).

Exercise 8.1 (Controlling for confounds is difficult)**.** Consider the following description:

> "There were 40 participants who composed two language groups and two age groups. Twenty of the participants were younger adults ranging in age from 30 to 54 years (mean age = 43.0 years), and 20 were older adults ranging in age from 60 to 88 years (mean age = 71.9 years). In each age group, half the participants were monolingual English speakers living in Canada, and the other half were Tamil–English bilinguals living in India. (...) All the participants in both groups had bachelor's degrees ..." (Bialystok et al., 2004, p. 44)

While the authors didn't explicitly claim to have done so, you might end up thinking that level of education was controlled for in this study. A couple of minutes' thought should reveal that this wasn't the case. (Does having a minimum requirement of having Bachelor's degrees equate both groups with respect to level of education?) But more interestingly, by introducing this minimum requirement, the authors may have introduced *additional* bias. How so?[1] ◇

8.1 Correlation coefficients

Pearson correlation coefficients, typically just called correlation coefficients and abbreviated as r, express how closely the (X,Y) data

[1] If you're stuck, consult https://gpseducation.oecd.org/CountryProfile?primaryCountry=CAN&treshold=5&topic=EO and look up similar data for India.

points fall on a straight line.

- $r = 1$: All points fall exactly on an increasing line.
- $r = -1$: All points fall exactly on a decreasing line. Correlation coefficients of 1 or -1 (or close to it, e.g., $r = 0.99$) tend not to be too interesting: They typically indicate that the two variables express the same thing (e.g., body length in centimetres and in inches).
- $r = 0$: There's no linear relation between the two variables whatsoever.

Correlation coefficients work in both directions: $r_{XY} = r_{YX}$.

Figure 8.1 shows eight examples of scatterplots and the correlation coefficients for the data presented in them. Note that a correlation coefficient close to zero doesn't imply that there is no relation between them; correlation coefficients different from 1 or -1 don't imply that the relation between two variables is imperfect; and it's possible for a positive correlation coefficient to reflect a relationship that's largely negative, and vice versa. Do these examples contradict the rough definition of correlation coefficients given above?

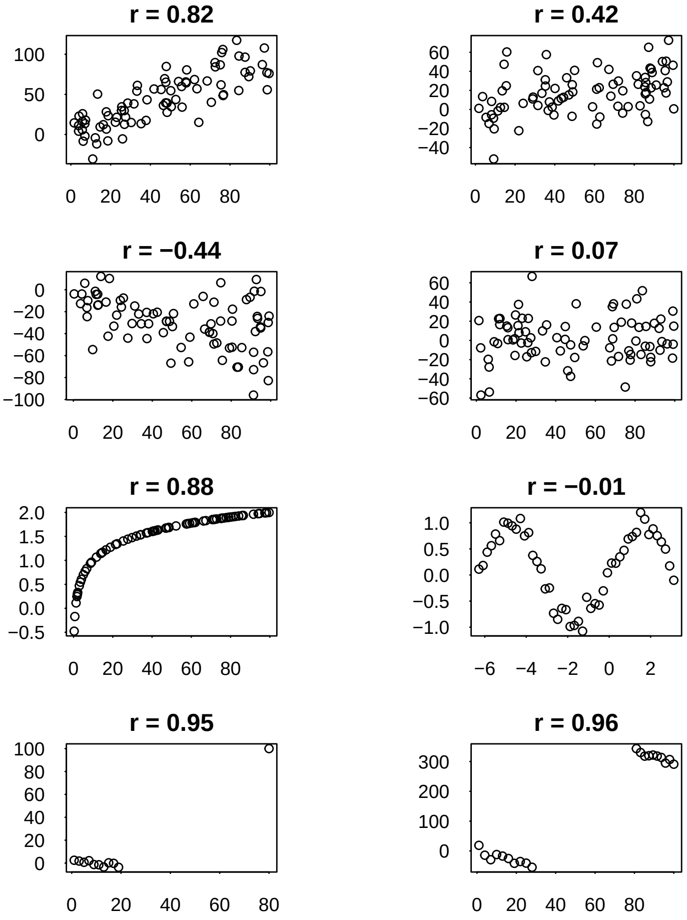


Figure 8.1: Examples of scatterplots and their associated correlation coefficients.

> The same correlation coefficient can correspond to a multitude of relationships between two variables. Never *ever* compute a correlation coefficient without drawing a scatterplot first.

By the same token, don't put stock in conclusions that hinge crucially on correlation coefficients for which no scatterplots are provided.

Exercise 8.2 (Honing intuitions about correlation coefficients)**.** To hone your intuitions about correlation coefficients, you can use the `plot_r()` function from the `cannonball` package for R.[2]

```r
# Install the package
install.packages("devtools")
devtools::install_github("janhove/cannonball")

# Load the functions
library(cannonball)

# Draw 16 plots with 20 data points each and r = 0.6
plot_r(n = 20, r = 0.6)

# With 50 data points each and r = 0.0
plot_r(n = 50, r = 0.0)

# With 40 data points and r = -0.9
plot_r(n = 40, r = -0.9)
```

Type `?plot_r` at the R prompt to access the function's help page and read the text under 'Details'. ◇

8.2 *Statistical control using hierarchical regression*

Exercise 8.3 (Reading assignment)**.** The next reading assignment concerns the study by Slevc & Miyake (2006). The "'zero-order correlations" mentioned are correlation coefficients expressing the relationship between two measured variables. ("First-order correlations" would be correlation coefficients expressing the relationship between two variables from which the influence of a third variable was statistically 'partialled out'.)

To help you make sense of Table 3:

- R^2: The proportion of the variation ('variance') in the outcome variable that can be described using the predictor variables included in the regression model.
- ΔR^2: The increase in R^2 compared to the previous *step* (i.e., the improvement in R^2 attributable to the current predictor).
- *df*, *F*: You can ignore this for this class.

[2] If you run into the error message 'Failed to install 'unknown package' from GitHub', try running the command `Sys.unsetenv("GITHUB_PAT")` first.

- *Final β*: Expresses the form of the relationship between the predictor in question and the outcome.

Now to the questions:

(a) What was the most important goal that Slevc & Miyake (2006) set themselves?

(b) Why did they have this aim?

(c) Why did they collected the variables *age of arrival*, *length of residence*, *language use and exposure* and *phonological short-term memory*?

(d) Which conclusions do they draw on the basis of their results?

(e) Regardless of whether you agree with these conclusions: Try to find one or two alternative explanations for their results that call into question the claim that "musical skills may facilitate the acquisition of L2 sound structure" (abstract). ♢

In correlational studies, control variables are often used to adjust statistically for known confounders. One technique used to accomplish this is hierarchical regression; see Table 3 in Slevc & Miyake (2006) for an example. We will discuss this technique mainly so that you are better able to appreciate the shortcomings of this technique and ones similar to it.

Example If you measure the shoe size and vocabulary knowledge of 4- to 16-year-olds, you'll observe a positive correlation between the two. This isn't surprising; see Figure 8.2. We'll use this silly example to illustrate the principle behind hierarchical regression; see Figure 8.3 on the following page.

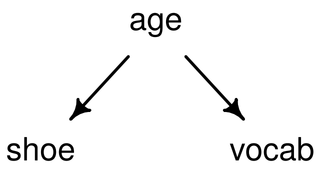


Figure 8.2: Shoe size and vocabulary knowledge are correlated since age acts as a confound.

- Top left: Shoe size and vocabulary knowledge are positively correlated.
- Top right and middle left: Age—the confound—is correlated positively with both shoe size and vocabulary knowledge.
- Middle right: This plot shows the vertical distance between the points in the middle left panel and the regression line. This shows how much the participants vary in their vocabulary test scores once the linear association between age and vocabulary knowledge has been partialled out.
- Bottom left: The association between shoe size and the vocabulary test scores with the linear association of age partialled out is much less strong. In this simulated example, the fact that the remaining association isn't exactly zero is due entirely to chance.

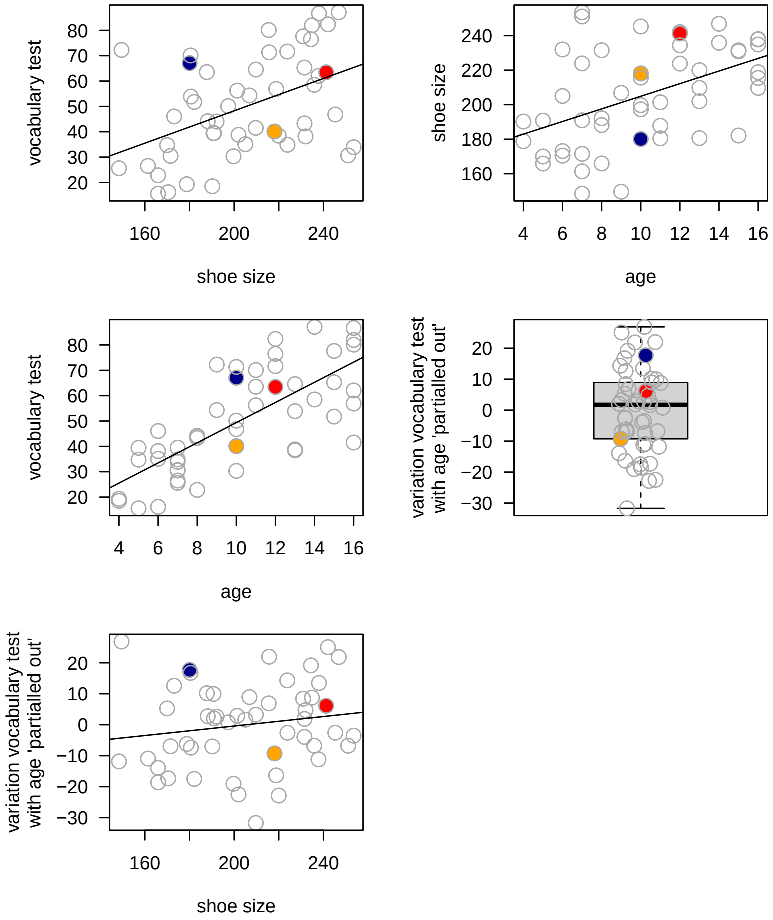


Figure 8.3: Hierarchical regression used to control for the age confound in the relationship between shoe size and vocabulary knowledge. The coloured circles in each panel show data belonging to the same three participants. The straight lines are regression lines. A regression line is the straight line that best captures the tendency in the cloud of data points, according to some definition of 'best'.

8.3 Caveats

You need to be hyper-aware of the following caveats concerning statistical control:

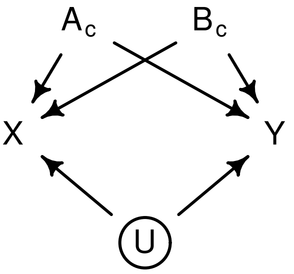


Figure 8.4: Perfectly controlling for A and B closes the non-causal paths $X \leftarrow A \rightarrow Y$ and $X \leftarrow B \rightarrow Y$. But it leaves open the non-causal path via U.

1. Controlling for a number of possible confounds doesn't rule out the possibility that there are even more confounds; Figure 8.4.
2. The methods typically used to account for confounding variables account for *linear* relationships between the confounds and the variables of interest. If these relationships aren't linear, the confound won't be fully accounted for. In DAG parlance, the path via the confound won't be fully closed.
3. The 'confound' may be a post-treatment variable. See Section 5.4 on page 51.
4. Statistical control may be imperfect because the confound was measured with some error. We'll treat this in more detail in Chapter 9.

The following excerpt makes the same points:

> "When experimental designs are premature, impractical, or impossible, researchers must rely on statistical methods to adjust for potentially confounding effects. Such procedures, however, are quite fallible. We examine several errors that often follow the use of statistical adjustment.
>
> "The first is inferring a factor is causal because it predicts an outcome even after "statistical control" for other factors. This inference is fallacious when (as usual) such control involves removing the linear contribution of imperfectly measured variables, or when some confounders remain unmeasured.
>
> "The converse fallacy is inferring a factor is not causally important because its association with the outcome is attenuated or eliminated by the inclusion of covariates in the adjustment process. This attenuation may only reflect that the covariates treated as confounders are actually mediators (intermediates) and critical to the causal chain from the study factor to the study outcome.[3]
>
> "Other problems arise due to mismeasurement of the study factor or outcome, or because these study variables are only proxies for underlying constructs.
>
> "*Statistical adjustment serves a useful function, but it cannot transform observational studies into natural experiments, and involves far more subjective judgment than many users realize.*" (Christenfeld et al., 2004, abstract, my emphasis)

Large sample sizes don't solve these problems.

Also see the blog entry *Controlling for confounding variables in correlational research: Four caveats*.

Exercise 8.4. In this series of exercises, we will use R to simulate some data and perform correlation analyses on them. More concretely, we will generate $n = 200$ pairs of predictor–outcome ob-

[3] What's meant is a causal chain such as $A \rightarrow B \rightarrow C$. A is causally important, but if you control for B, you won't find any association between A and C.

servations. The predictor $\boldsymbol{x} = (x_1, \ldots, x_n)$ will be sampled from a normal distribution with mean 0 and variance 1 (i.e., $x_i \sim \mathcal{N}(0, 1)$). The outcome $\boldsymbol{y}$ is described by a simple function of $\boldsymbol{x}$ and some random error, namely,

$$y_i = 0.4 + 0.7 \cdot x_i + \varepsilon_i,$$

$i = 1, \ldots, n$, where the random error $\boldsymbol{\varepsilon} = (\varepsilon_1, \ldots, \varepsilon_n)$ is also sampled from a normal distribution with mean 0 and variance 1 (i.e., $\varepsilon_i \sim \mathcal{N}(0, 1)$), independently of both $\boldsymbol{x}$ and the other ε_i values.

Run the following commands in R to simulate data according to this scheme. Note that the `rnorm()` command takes an argument that specifies the standard deviation (`sd`) of the normal distribution from which to sample the data, not its variance. But since $\sqrt{1} = 1$, the standard deviation of the distribution from which we sample is also 1. (Like for the graphing assignments, don't enter these commands directly to the console. Use a script.)

```
n <- 200
x <- rnorm(n, mean = 0, sd = 1)
y <- 0.4 + 0.7 * x + rnorm(n, mean = 0, sd = 1)
```

The expected correlation between $\boldsymbol{x}$ and $\boldsymbol{y}$ is

$$\begin{aligned}\rho_{xy} &= \frac{0.7 \cdot \mathrm{Var}(\boldsymbol{x})}{\sqrt{\mathrm{Var}(\boldsymbol{x})(0.7^2 \cdot \mathrm{Var}(\boldsymbol{x}) + \mathrm{Var}(\boldsymbol{\varepsilon}))}} \\ &= \frac{0.7}{\sqrt{0.7^2 + 1}} \\ &\approx 0.57,\end{aligned}$$

since $\mathrm{Var}(\boldsymbol{x}) = 1 = \mathrm{Var}(\boldsymbol{\varepsilon})$.

We now put these $\boldsymbol{x}, \boldsymbol{y}$ observations into a tibble.

```
library(tidyverse)
d <- tibble(predictor = x, outcome = y)
```

1. Using `ggplot()`, draw a scatterplot of the `predictor` vs. `outcome` values in `d`.

2. Compute the sample correlation coefficient between `predictor` and `outcome` using the R function `cor()` like so:

   ```
   cor(d$outcome, d$predictor)
   ```

   Jot down this number, rounded to two decimal places. Now simulate the data again and jot down the number again; do this five times. Compare the correlation coefficients you observed to the expected correlation coefficient and summarise your findings.

3. Now imagine that instead of observing a random sample of n observations, we only observe data from those pairs of observations where $x_i > 0$. To emulate this scenario, we create a new tibble (`d2`) consisting of those rows in `d` where the predictor value is greater than 0:

```
d2 <- d |>
  filter(predictor > 0)
```

Draw a scatterplot of the `predictor` vs. `outcome` values in `d2`. Compute the sample correlation coefficient between `predictor` and `outcome` in this reduced sample. Repeat the entire simulation five times. Compare the correlation coefficients you observed to the expected correlation coefficient and summarise your findings.

4. Now imagine that instead of observing a random sample of n observations, we only observe data from those pairs of observations where $x_i > 1$ or where $x_i < -1$; equivalently, where $|x_i| > 1$. That is, we're only retaining data with fairly extreme x_i observations.

   Using `filter()`, create a new tibble (`d3`) consisting of those rows in `d` where the absolute predictor value is greater than 0. You can use the `abs()` function to compute absolute values.

   Draw a scatterplot of the `predictor` vs. `outcome` values in `d3`. Compute the sample correlation coefficient between `predictor` and `outcome` in this reduced sample. Repeat the entire simulation five times. Compare the correlation coefficients you observed to the expected correlation coefficient and summarise your findings. ◇

9
Constructs and indicators

We're faced with an inescapable fact of life:

> Most measurements are imperfect.

Saying that a study's measurements aren't perfect isn't much of a criticism. But it's crucial to appreciate the consequences of imperfect measures—pointing out that a study's findings can plausibly be accounted for by the fact that its measurements are imperfect *is* a valid criticism.

Construct or *latent variable*. Lots of characteristics can't be observed or measured directly. Instead, their existence, as well as their relative value, are inferred on the basis of other, observable variables.

Indicator or *manifest variable*. These are variables that can be measured or observed directly and from which information about the construct is inferred. Table 9.1 lists some examples.

Table 9.1: Examples of constructs and indicators.

Construct	Example indicator
Intelligence	Result on an intelligence test
Working memory capacity	Length of a sequence of digits you can repeat in reversed order
Language aptitude	Result on the LLAMA-D test
L2 reading skills	Number of correctly answered items on a reading test
Attitudes towards Danish	Answer to the question 'How beautiful do you think Danish is?'
Socio-economic status	Father's occupational category

Measurement error Even the best indicators are rarely perfect. Better indicators just have a smaller measurement error.

Even variables that don't act as proxies for some cognitive or social construct are often measured with some error. Examples include body weight (bathroom scales are imperfect, and the result is rounded), blood pressure (if you have a sphygmomanometer, check its manual), and age (invariably rounded down to the integer below when reported).

9.1 Systematic and random measurement error

Measurement error can include both a systematic and a random component.

The **systematic** component of an instrument's measurement error is the extent to which it tends to over- or underestimate what it's supposed to measure. For instance, a miscalibrated kitchen scale may overestimate weights by 10 g on average, and an overly harsh language test may tend to label learners' L2 skills one CEFR level below their actual proficiency on average.

Note that it's possible for an instrument to systematically overestimate values on one part of the scale and to underestimate them on another part.

When there's no gold standard to which the measurements can be compared, it may be impossible to assess their systematic measurement error.

The **random** component of an instrument's measurement error is the extent to which the measured values differ from the true values + systematic error. Another way of putting this is: By how much will the measurements vary if the true values are the same? For instance, an kitchen scale may, on average, measure weights accurately (no systematic error), but the individual readings may be off by up to a couple of grams in either direction (random error).

As a second example, consider a group of 365 7-year-olds, all born on different days of the year. Just one of them actually is 7 years old on the day; the reported values of the others will be off by 1 day, 2 days, ..., 364 days. The reported age, then, systematically underestimates their true age by $\frac{0+1+2+\dots+364}{365} = 182$ days. The random component is 0, though, as children born on the same day will report the same age, even though this reported age will be lower than their actual age.

As a final example, consider a poorly calibrated bathroom scale. If you put a calibrated mass of precisely 60 kg on it on five different occassions, it returns readings of 61.1, 60.4, 60.4, 60.5 and 61.2. The mean observation for the same mass is $\frac{61.1+60.4+60.4+60.5+61.2}{5} =$ 60.78, i.e., an overestimate of 0.78 kg. The mean absolute difference between the observations and their expected value (here: 60.78) is $\frac{|0.32|+|-0.38|+|-0.38|+|-0.28|+|0.72|}{5} = 0.42$.

9.2 *Consequences of measurement error*

The consequence of systematic measurement error is clear: Your data are biased. This isn't necessarily a problem: If you're comparing two groups for both of which you have data that are biased to the same extent, the difference between them won't be biased. And for variables such as age, the systematic error (roughly 182 days) tends to be small relative to the variability of the true values, in which case it's probably inconsequential.[1]

The consequences of random measurement error are much less intuitive and bear pointing out.

Less power and precision Measurement error on the *outcome* variable will increase its variability. Since power and precision are lower when there's more variability in the outcome, measurement error on the outcome lowers power and precision.

Statistical control is imperfect Measurement error on a *control* variable means that controlling for this observed variable won't fully eradicate the confounding caused by the construct itself. The DAG in Figure 9.1 illustrates this.

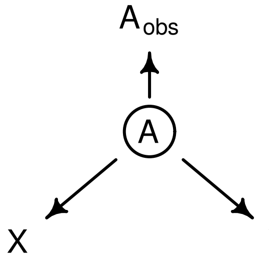


Figure 9.1: The X–Y relationship is confounded by A. A, however, can't be observed directly. A proxy (indicator) A_{obs} can be controlled for instead, but this won't fully shut the non-causal path $X \leftarrow A \rightarrow Y$.

> "[F]allibility in a covariate usually implies that there would be more adjustment if the variable were measured without error." (Huitema, 2011, p. 569)

Controlling for A_{obs} is better than not controlling for it. But researchers routinely mistake controlling for an indicator with controlling for a construct, and their causal conclusions are overconfident as a result. A discussion of this problem can be found in Westfall & Yarkoni (2016), Vanhove & Berthele (2017) and Berthele & Vanhove (2020).

Regression to the mean When observations are due partly to skill or some underlying construct and partly to chance (e.g., measurement error), a second round of observations will likely show that the extreme scores have become less extreme, i.e., that they've regressed to the mean.

- First consider an example where the observations are purely due to luck, with no skill or construct involved: playing roulette. Playing roulette is a losing proposition: For every 100 francs bet, you stand to lose about 5 francs (= the mean). But on any given night, some players will luck out and make a killing, whereas other players get extraordinarily unlucky and lose much more than the expected 5 francs. Their winnings or losses are a dreadful measure of their skill level: they all have the same skill level, which corresponds to a loss of 5 francs.

  The next night, however, the lucky players from the day before probably won't get as lucky again (their luck the day before was extraordinary), and similarly for the unlucky players—all again

[1] But see, for instance, Helsen et al. (2005) and Sprietsma (2010) on the consequences of 'relative age' (i.e., age differences within an age group, e.g., 15-year-olds) in sports and education.

stand to lose about 5 francs. Some might get lucky or unlucky twice in a row, but they're more likely to end up somewhere near the 5-franc mark, i.e., most of the lucky and unlucky players will regress to the mean.

- The same principle is at play when the observations come about in part through skill (or some other construct) and in part through chance. For instance, the most successful stock picker of the year 2032 is likely not to perform as well in the year 2033—even if the conditions on the stock market are comparable and the stock picker didn't start to rest on his laurels. The reason could simply be that he had more than his fair share of luck in 2032—you need some luck to come out on top—and wasn't as lucky in 2033. As a result, his performance in the next year is likely to be closer to the average performance (i.e., he's regressed to the mean of stock picker performance).
- If you administer a reading test to a group of learners one week and another reading test a couple of weeks later, you're likely to find that the very worst readers on the first test are still pretty poor readers on the second test (= the skill part), but their performance won't be as atrocious—it'll seem as though they've made some progress. Similarly, the best readers on the first test are likely to still be good readers on the second test, but their performance probably won't be as exceptional—it'll seem as though they've become worse.

  But this pattern can be explained in terms of measurement error: Even if none of the learners actually learnt or unlearnt something, you're likely to find such a pattern. The reason is that, if you obtained a dismal score, you're likely to be a pretty poor reader *and* to have had some bad luck—perhaps the topic of the reading test just wasn't suited for you, or you were coming down with the flu. A couple of weeks later, you might encounter a topic you know a thing or two about or you might be in better physical shape. Similarly, if you scored exceptionally well on the first test, you may have had some luck with the test's topic or with other circumstances, and these may not be as favourable next time round.

Exercise 9.1. A nationwide standardised maths test is administered to all 5th graders. It turns out that the classes with the highest mean test scores tend to be pretty small. One possible explanation is that small classes are more conducive to learning maths. Another explanation is that this finding is an artefact of measurement error. These explanations aren't mutually exclusive.

(a) Explain how measurement error can give rise to this finding.

(b) How could you check if measurement error accounts (fully or in part) for this finding?[2] ◇

[2] Hint: Which graph could you draw?

10

Questionable research practices

Exercise 10.1 (Reading assignment)**.** Read Chapter 2 in Chambers (2017). There are no guiding questions for this text; it should be intelligible enough. But by way of preparing for it, try to answer these questions.

(a) You recruit 60 participants, aged 8–88. Half of them are assigned to the experimental group; the others to the control group (random assignment). You run a significance test comparing the mean age in both groups. What is, at most, the probability that you'll obtain a significant result (i.e., $p \leq 0.05$)?

(b) Each of your participants throws a fair six-sided dice. You run another significance test to check if there's a mean difference in the number of pips obtain in the control and in the intervention groups. (Evidently, the intervention doesn't make you throw dice any better.) What is, at most, the probability that you'll obtain a significant result (i.e., $p \leq 0.05$)?

(c) What do you know about the probability of observing *either* a significant age difference between the two groups, *or* a significant difference in the mean number of pips obtained, *or* two significant differences? ♢

10.1 A paradox

Sterling et al. (1995) inspected 563 articles in psychology journals (published in 1986–1987) in which significance tests were used to answer the research question. In 538 of them (96%), the researchers reported a significant result that confirmed their own hypothesis. In medical journals, the figure was lower but still pretty high (270/316, 85%).

But at the same time, the sample sizes in psychological research are fairly small (Marszalek et al., 2011; Sedlmeier & Gigerenzer, 1989): The average study in applied psychology published in 1995 only contained 22 participants per condition. This implies that many of these studies must have had fairly low statistical power (see Section 4.7 on page 42): Even if the null hypothesis hadn't been

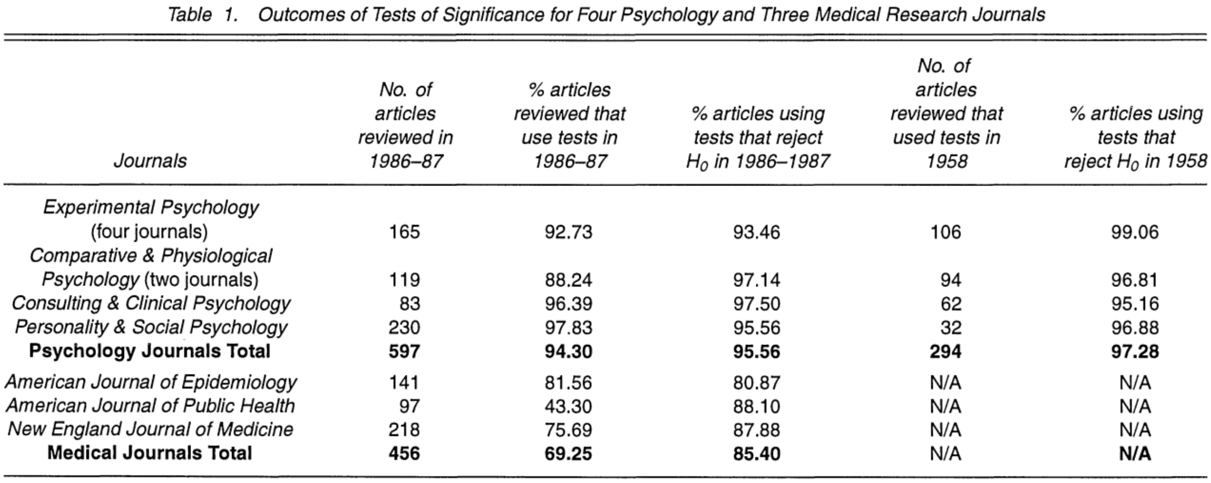


Table 1. Outcomes of Tests of Significance for Four Psychology and Three Medical Research Journals

Journals	*No. of articles reviewed in 1986–87*	*% articles reviewed that use tests in 1986–87*	*% articles using tests that reject H_0 in 1986–1987*	*No. of articles reviewed that used tests in 1958*	*% articles using tests that reject H_0 in 1958*
Experimental Psychology (four journals)	165	92.73	93.46	106	99.06
Comparative & Physiological Psychology (two journals)	119	88.24	97.14	94	96.81
Consulting & Clinical Psychology	83	96.39	97.50	62	95.16
Personality & Social Psychology	230	97.83	95.56	32	96.88
Psychology Journals Total	**597**	**94.30**	**95.56**	**294**	**97.28**
American Journal of Epidemiology	141	81.56	80.87	N/A	N/A
American Journal of Public Health	97	43.30	88.10	N/A	N/A
New England Journal of Medicine	218	75.69	87.88	N/A	N/A
Medical Journals Total	**456**	**69.25**	**85.40**	N/A	**N/A**

Figure 10.1: Table 1 from Sterling et al. (1995).

correct in any of these studies, it'd have been impossible to reject it in 96% of cases.

For a long time (see already Sterling, 1959), it was believed that the reason for this discrepancy (low power, lots of significant results) was due to **publication bias**: Researchers prefer to write up the studies in which they obtained significant results, and editors and reviewers tend to reject studies with non-significant findings. The studies that were conducted but that produced non-significant findings were believed to languish in the researchers' file-drawers.

But while some studies never make it into print, the vast majority do. So where did the non-significant findings go?

10.2 *Hidden flexibility*

More recently, scholars with an interest in meta-science (i.e., science about science) have come to realise that research projects afford a great deal of flexibility. Researchers can—consciously or subconsciously—leverage this flexibility to produce a steady stream of significant findings—*even if the data are nothing but noise.*[1] Simmons et al. (2011) call this flexibility **researcher degrees of freedom** and demonstrate how significant findings can be conjured from thin air if researchers afford themselves some leeway in analysing their data.

Sources of researcher degrees of freedom include:

- A researcher can run intermediate analyses and decide to stop or to continue collecing data based on the results. See Simmons et al. (2011) and Exercise 10.2 for the consequences of this.
- Sometimes, there are several ways in which a task or test can be scored, or how some variable can be constructed. When one way yields a significant finding and the other doesn't, it's easy to convince yourself that the one that produced significance was

[1] If the data aren't just noise, such flexibility will spuriously amplify the signal. For instance, even if A influences B, the literature as a whole will tend to overestimate the extent of this influence.

obviously the right one. Relatedly, researchers routinely collect multiple outcome variables, but it's tempting to focus on the one that 'worked' (i.e., produced significance) rather than on those that didn't. See Simmons et al. (2011), Gelman & Loken (2013), and von der Malsburg & Angele (2017). For a discussion with a focus on bilingualism research, see Poarch et al. (2019).

- **HARKing** (hypothesizing after the results are known; Kerr, 1998): A largely exploratory analysis is reported as though it were planned all along. Inevitably, the researchers will find in the data what they claim to have anticipated. (This can happen without any bad intent on the part of the researchers.)
- Convenient errors and biased debugging: Everyone makes mistakes, but you're more likely to catch your own mistakes when the results don't pan out than when they do. As a result, the mistakes that remain in the literature aren't distributed randomly but tend to favour the researchers' hypotheses.

Trying out several defensible analyses and glossing over the ones that didn't produce significance is referred to as **p-hacking**.

The practices listed above are examples of questionable research practices. Traditionally, these aren't viewed as outright fraud (which includes fabricating or manipulating data), though arguably, it will become increasingly difficult to invoke plausible deniability as professional researchers can be expected to know their consequences.

For possible solutions, see Chambers (2017).

Exercise 10.2 (False-positive psychology)**.** The consequences of researcher degrees of freedom/p-hacking are best appreciated by seeing them. Do these exercises in order.

(a) Open the app at `https://plurilinguisme.shinyapps.io/fppsy/` and carefully read the description.

(b) Click 'Simulate!', leaving all settings at their default values. Describe what the two graphs (reproduced here as Figure 10.2 for your convenience) are showing.

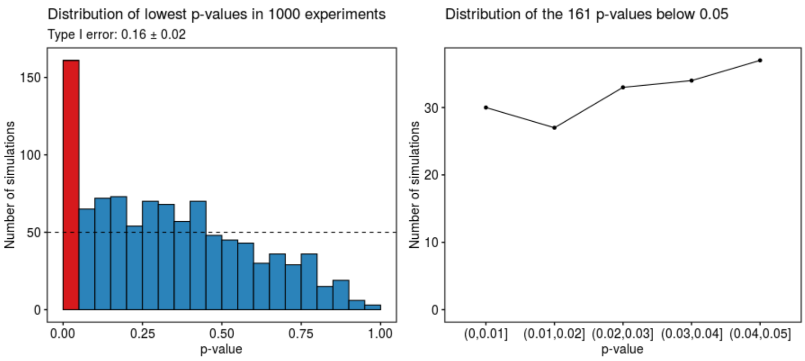


Figure 10.2: When you run the app using its default settings, you'll obtain two graphs similar to these. Your graphs won't be identical as they are based on simulations with random data.

(c) First try to answer the following questions by *thinking* about them. Once you've written down your answer, check it by running the simulation.

i. Increase the 'maximum number of additional participants in each group' to 30. Leave the other settings at their default values. How will the graphs change?

ii. Leaving all other settings as they currently are, what will happen if instead of analysing the data after 10 new participants per condition, they're analysed after 5 new participants per condition? Or after just 2 new participants per condition?

iii. What'll happen when the correlation between the two dependent variables becomes weaker (e.g., $r = 0.1$ instead of $r = 0.5$)? Why?

iv. What'll happen when the correlation between the two dependent variables becomes stronger (e.g., $r = 0.95$)? Why?

v. For which combination of the different parameters will you obtain the highest Type-I error? Think before running the simulation!

vi. For which combination of the different parameters will you find a Type-I error rate of about 5%? Are there any parameters that don't play a role? Think before running the simulation! ◇

10.3 *Further reading

Most studies referred to in this chapter are both accessible and short. If you read Simmons et al. (2011) (warmly recommended!), also read their short retrospective article (Simmons et al., 2018) lest you misinterpret the take-home message. Peterson (2016) presents an ethnographic study that gives you some insight into what questionable research practices look like in the field.

A highly accessible book-length treatment of these topics, and then some, which I cannot recommend highly enough, is Ritchie (2021). Chambers (2017) is also recommended.

A
Reading difficult results sections

Results sections in quantitative research reports can be daunting. Sometimes, the analyses are necessarily complex and require sophisticated knowledge about statistics and research design on the part of the reader. But too often, results sections are more difficult than they need to be (see Vanhove, 2021).

> Don't allow yourself to be dazzled by complicated analyses and incomprehensible results sections—the complexity may be largely superficial.

Here are some tips for muddling through difficult results sections with minimal psychological damage.[1]

1. Identify the central, genuine research questions and the corresponding hypotheses. Research papers in applied linguistics surprisingly often contain 'padding' research questions that are unrelated to the core goal of the study. When scanning the results section, you can usually leave aside the paragraphs about these uninteresting research questions. For example, in a report on a pretest/posttest experiment where participants were randomly assigned to conditions, you may find 'research' questions such as *Do participants in the treatment condition have different pretest scores from those in the control condition?* or *Do participants have higher scores on the posttest than on the pretest?* Both questions are uninteresting as they don't tell you whether the treatment actually worked.

2. **Draw a graph of the predictions.** (!) Having identified the key research questions and hypotheses, I often find it useful to sketch what the data would look like if the researchers' predictions panned out and what kind of data would, within reason, falsify their hypotheses. These graphs are usually simple hand-drawn line charts that illustrate the expected group differences. I find that they help me to better understand the logic behind the study and to focus on the important analyses in the Results section. You may find that several radically different patterns are in line with the authors' stated predictions; this tells you something

[1] Based on the blog entry *Surviving the ANOVA onslaught*.

about how specific their predictions are. (It's good to have specific as opposed to very general hypotheses!) It can also be useful to draw some simple graphs of data that would *not* be consistent with the authors' predictions. This, too, can help you work out if the predictions are fairly specific (a good thing) or if pretty much any pattern in the data would be consistent with them (a bad thing).

3. Look for a graph in the paper. Ideally, the paper will contain a graph of the main results that you can then compare with the graphs you drew yourself. Do the results seem to confirm or disconfirm the researchers' predictions? Sometimes, a good graph will allow you to carry out the easiest of significance tests yourself: the **inter-ocular trauma test**—if the conclusion hits you between the eyes, it's significant.[2] If the results are less clear cut, you'll need to scan the Results section for the more details, but by now, you should have a clearer idea of what you're looking for—and what you can ignore for now. If the paper doesn't contain a graph, you can often draw one yourself on the basis of the data provided in the tables.

4. Ignore tests unrelated to the central research questions. Results sections sometimes contain significance tests that are unrelated to the research questions the authors formulated up front (see Vanhove, 2021). Such tests include include balance tests in randomised experiments (e.g., "The control and intervention group did not differ in terms of SES ($t(36) = 0.8$, $p = 0.43$)."), tautological tests (e.g., "A one-way ANOVA confirmed that participants categorised as young, middle-aged and old differed in age ($F(2, 57) = 168.2$, $p < 0.001$).") as well as some less obvious cases. By and large, these tests tend to add little to the study. In non-randomised experiments, systematic differences on background variables between the groups may represent confounds, but these can be assessed based on the descriptive statistics and don't need to be rubber-stamped with a significance test.

Evidently, you'll get better at this with practice, and it'll be helpful to educate yourself on basic statistics, too. The latter will help you to understand better what was done, but also it will also allow you to ask more critical questions, not least of which is *Are these analyses at all relevant?*.

[2] To be clear, this isn't a formal significance test. But it's a useful heuristic!

B
Reporting research transparently

Many a research report leaves out information that is crucial for interpreting its findings correctly. And often, readers are implicitly asked to just take the authors word for it and trust that the analyses were run appropriately—even though reporting errors are common (Nuijten et al., 2016) and suboptimal or downright wrong analyses abound. Here are some tips to help ensure that your methods and findings are transparent to the readers.

1. ong reports detailing everything quickly become unreadable. My own preference is to aim for a crisp main text that doesn't inundate the reader with numbers and numbing details. Instead, I try to communicate the findings mainly through plots and refer to copious online materials for the details (Vanhove, 2021, also contains further guidance for writing quantitative research reports and some useful references). Using these online materials, interested readers should at least be able to reproduce the results occurring in the report, and so the online materials that I make available minimally comprise the data sets and the computer code necessary for reproducing the plots and numbers in the main text. Materials such as stimulus lists, questionnaires, code for running the experiment itself etc. should in my view also be shared by default. For projects involving lots of tedious steps that will be of little interest to the average reader, I also like to make available a technical report that documents every little detail (e.g., Vanhove et al., 2019).

   That said, **don't let perfect be the enemy of good**. If you're able to share your computer code but it's poorly documented, that's better than not sharing your code at all.

2. It's easier to share code, data, and materials if you made the decision to do so at the start of the project rather than a couple of weeks before handing in your report.

3. Nowadays I exclusively use `https://osf.io/` for making available online materials. See `https://osf.io/yxzfm/` for examples.

4. For further guidance, see Klein et al. (2018), Levenstein & Lyle (2018), and Soderberg (2018).

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