Opinion is intended to facilitate communication between reader and author and reader and reader. Comments, viewpoints or suggestions arising from published papers are welcome. Discussion and debate about important issues in ecology, e.g. theory or terminology, may also be included. Contributions should be as precise as possible and references should be kept to a minimum. A summary is not required.

Population regulation: a synthetic view

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Population ecologists continue to debate population regulation. If anything, the controversy intensified during the last decade. Does it mean that our field has not progressed very far since the days of the "great debate" between Nicholson and Andrewartha? Three years ago I suggested that in actuality the broad outlines of a consensus were emerging (Turchin 1995). What I have read in the literature since then has only confirmed me in the opinion that most population ecologists are in agreement on the major, strategic issues in population regulation, while the ongoing debate increasingly focuses on narrow tactical questions. This does not mean, however, that the intensity of the debate, as well as the vituperance, has diminished (for some quotes see Turchin 1995).

What are these broad issues of agreement? Here is my attempt to formulate them:

1. The central quantity of interest in the analyses of population regulation is *the realized per capita rate of population change*, defined as $r_t = \ln N_t/N_{t-1} = \ln N_t - \ln N_{t-1}$, where $\ln N_t$ is the natural logarithm of population density at time t.
2. The realized per capita rate of change, r_t, is affected by both exogenous and endogenous factors. (*Exogenous* factors are those that affect population change, but are not themselves affected by population numbers. In other words, there is no dynamic feedback between an exogenous factor and population density. By contrast, *endogenous* factors represent dynamical feedbacks affecting population numbers, possibly involving time lags.) Exogenous factors are not "noise" to be tuned out. They represent important biological processes affecting population change, and are a legitimate and interesting subject for study.
3. Some negative feedback between r_t and population density (that is, *density dependence*) is a necessary (but not sufficient) condition for population regulation.
4. Population dynamics are inherently nonlinear. A wide variety of functional relationships between the *expected* (or average) r_t and population density is possible, both in theory and in practice (see Fig. 1). The relationship is often monotonic – either convex or concave – but not necessarily so. There may be an Allee effect (in which case the rate of change first increases and then decreases with density), or even more complex relationships leading to metastable dynamics. For some ranges of density the expected per capita rate of change may be flat, with population dynamics dominated by exogenous factors (the so-called "density-vagueness").
5. Rate of population change may be affected not only by the current population density, but also by lagged density. In some cases the lag structure of population regulation is quite complex, with more than one time delays involved in regulation (Fig. 1d).
6. Finally, a focus on testing null hypotheses against unspecified alternatives has proved to be unproductive in investigations of population regulation. In other words, the interesting question is not whether we can reject the hypothesis that a population is "unregulated". A much more fruitful approach, for example, in the analysis of time-series data is to *estimate* the relative strengths of exogenous versus endogenous contributions to population change, the lag structure of regulation, and the shapes that characterize the functional relationship between r_t and lagged population densities. Ultimately, we need to determine which of the alternative ecological mechanisms explains population regulation in any particular case study, or perhaps what are the relative contributions of several ecological factors to regulation.

To summarize, unlike the controversies in the past, the modern view of population dynamics is synthetic and

quantitative. As a consequence, few ecologists find it profitable anymore to argue whether all populations are regulated by density-dependent factors or not. Instead, we emphasize that both exogenous and endogenous factors affect population change, and that their relative strength will vary between different population systems.

I think that the above summary is a fair statement of the shared ground in the current views espoused by theoretical, statistical, and empirical ecologists (e.g., Royama 1992, Woiwod and Hanski 1992, Dennis and Taper 1994, Murdoch 1994, Harrison and Cappuccino 1995, Turchin 1995, Wolda 1995, Sinclair 1996, Huffaker et al. 1998, Berryman unpubl., Hassell et al. unpubl.). Remarkably, even such persistent critics of "the regulation paradigm" as den Boer and Reddingius appear to be in agreement with the six issues I listed above. The interested reader can check this statement by reading the following pages in their 1996 book: pp. 263–264 (issue 1), pp. 48–54 (issue 2), pp. 265 and 268 (issue 3), p. 54 (issue 4), p. 261 (issue 5), and pp. 270–271 (issue 6).

Once again, I want to emphasize that despite agreement on some broad issues arguing about density dependence continues to be a favorite occupation among population ecologists. Some of these controversial issues are technical, such as what are the best statistical methods for characterizing nonlinearities, for detecting delayed density dependence, or for dealing with measurement noise. More fundamental issues include disagreements about how to define *regulation* (see Turchin 1995 for a discussion) and how prevalent regulation is in nature. (Note that the question of what are relative contributions of endogenous versus exogenous factors to population change is a separate issue from what factors contribute to population regulation; see Hassell et al. unpubl. for a very clear statement of this difference.) Perhaps the key issue in this disagreement is how to relate statistically detected patterns in time-series data to possible ecological mechanisms responsible for population fluctuations. For example, den Boer and Reddingius (1996), while admitting that "perhaps

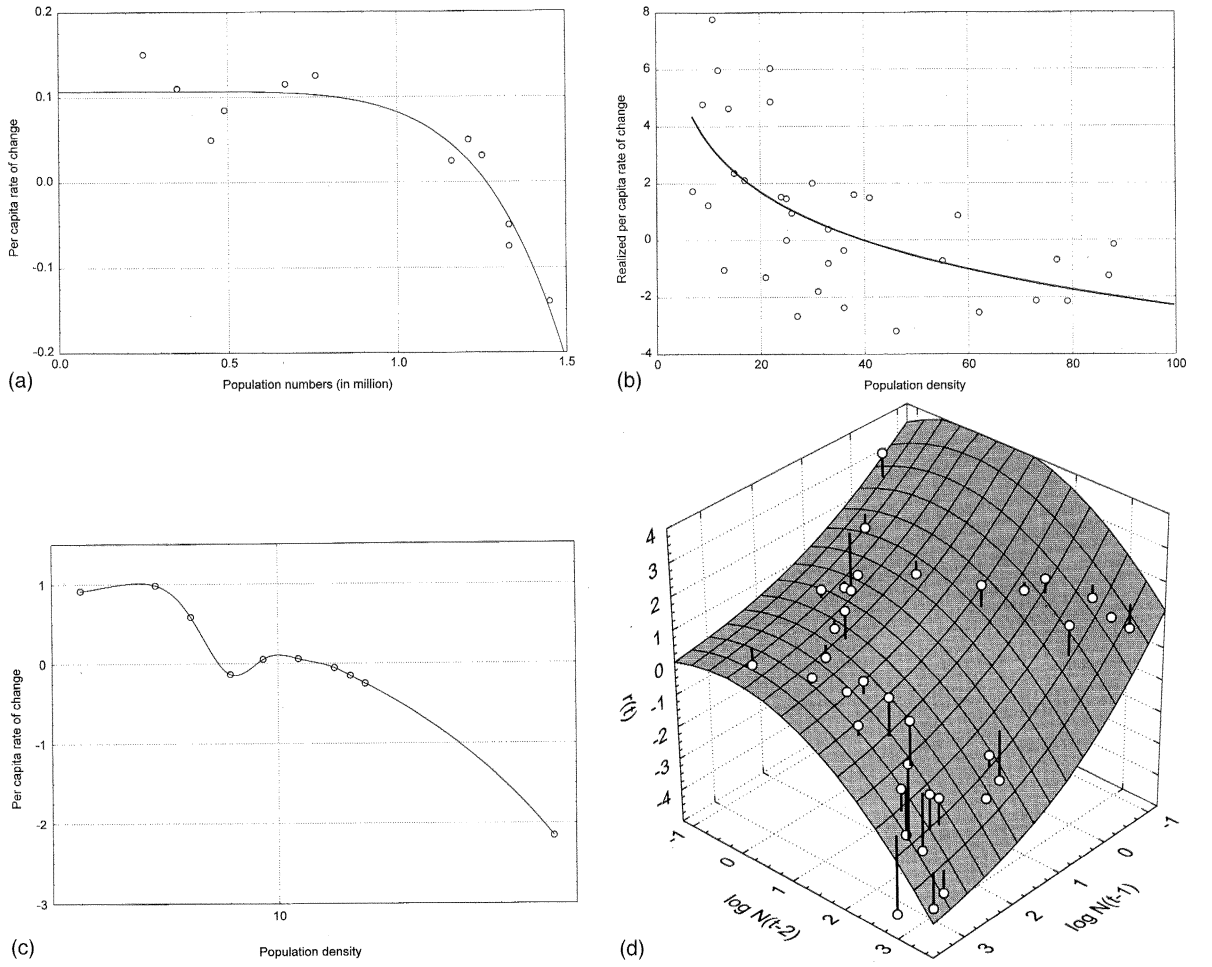

Fig. 1. Some examples of structures of density dependence in real populations. (a) Wildebeest (data from Sinclair 1996: Fig. 1). (b) Vole *Microtus pennsylvanicus* (data from Turchin and Ostfeld 1997). (c) Rangeland grasshopper *Melanoplus sanguinipes* (data from Belovsky and Joern 1995: Fig. 8a). (d) Vole *Clethrionomys rufocanus* (data from Henttonen and Hanski 1999).

statistically significant density dependence occurs more often in insect populations than we thought beforehand", nevertheless argue that "this statistical testing ... did not contribute very much to our understanding of regulation and stabilization in animal populations. We were not sure population *regulation* very often occurs in nature, and we still are not" (page 268). A more technical criticism was advanced by Williams and Liebhold (1995) who argued that a statistical pattern suggestive of second-order feedbacks (delayed density dependence) may arise spuriously as a result of serial correlations in some exogenous variable (see also the response by Berryman and Turchin 1997). A very important general issue that remains to be resolved is how much we can infer about population processes by combining mathematical models with time-series analysis (Kendall et al. unpubl.). Finally, I have not even touched on the spatial aspects of population regulation (and do not intend to do so, as it would require a whole other article, if not a book).

Even more fundamentally, some critics (see the preceding paper by Murray) continue to question the basic tenets of the current research program into population regulation. I believe that such criticisms should not be left unanswered. However, instead of the usual strategy in debates on population regulation, pointing out where the opponent is wrong, I follow a somewhat different tack. Essentially, my argument will be that an alternative philosophical stance than the one championed by Murray leads to a more fruitful research program. I want to make clear that this is the point where I shift gears and, instead of trying to discern the consensus, I present my personal view. This is not to say that I claim any originality; in fact, the view I present is a kind of a Frankenstein monster put together from the pieces I liked most in all the papers I cite above plus the pioneering work by Royama 1977, Berryman 1978, Chesson 1982, Ellner 1989 and many others I am probably not even aware of. The point is that all these authors, while recognizing their piece of Frankenstein, may not agree with how these pieces were fitted together.

The main issue that Murray raises is, what is the "density-dependent regulation hypothesis"? This question reflects the philosophical gulf between our approaches to doing science. Like many other authors (see item 6 above), I find the narrow emphasis on hypothesis testing counterproductive. The problem is that a consistent application of the strict (naïve?) Popperianism does not seem to lead anywhere in population ecology. Let us consider Murray's alternative to "density-dependent regulation hypothesis". Recasting his Fig. 2 in terms of $r = b - d$ where b and d are the per capita birth and death rates, we have the relationship depicted in Fig. 2a. (Note that there is nothing particularly remarkable about this form – given the variety of density-dependence patterns exhibited by real

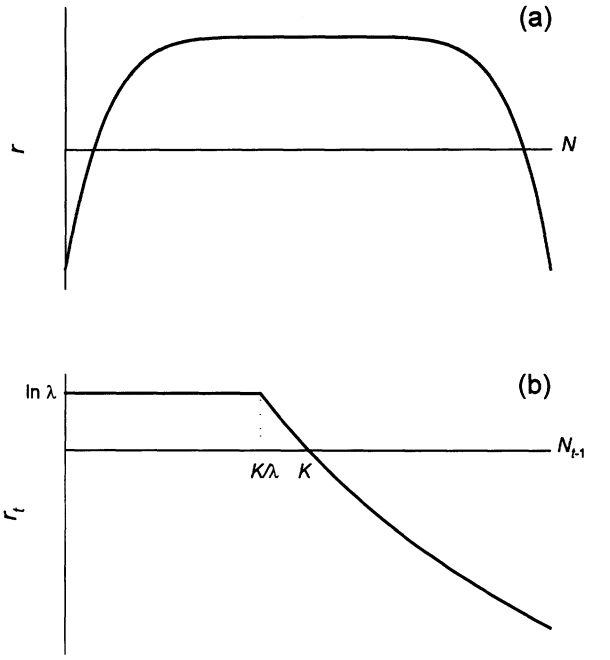

Fig. 2. (a) The relationship between $r = b - d$ and N implied by Murray (1998: Fig. 2). (b) The relationship between r_t and N_{t-1} implied by the numerical example in Murray (1998). This formula is derived as follows. Let K be the maximum density of individuals that can be supported by the environment, and λ be the average number of offspring left by individuals who obtain all the resources they can consume. For $N_t < K$, then, we have the relationship $N_t = \lambda N_{t-1}$. Translating the condition in terms of N_{t-1} we have: $N_t = \lambda N_{t-1}$ if $N_{t-1} < K/\lambda$. If N_t "tries" to get above K, then it is immediately reduced to K. That is, $N_t = K$ if $N_{t-1} \geq K/\lambda$. Now translating this model in terms of $r_t = \ln N_t/N_{t-1}$ we obtain:

$$r_t = f(N_{t-1}) = \begin{cases} \ln \lambda & \text{if } N_{t-1} < K/\lambda \\ \ln K/N_{t-1} & \text{if } N_{t-1} \geq K/\lambda \end{cases}.$$

populations, of which Fig. 1 gives a few examples, I have no trouble believing that some populations are characterized by precisely this kind of density dependence.) Is this "population limitation hypothesis" universally applicable and falsifiable? It is certainly falsifiable, since it predicts, among other things, that at high population densities the relationship between r and N should be characterized by a downward curvature (this is a mathematical consequence of the fact that the slope is zero for intermediate densities and negative for high densities). Such a curvature can be detected by fitting a quadratic polynomial to r as a function of N. However, fitting a quadratic function to the data in Fig. 1b (voles), we find that the quadratic coefficient is *positive* and statistically significant at $P < 0.005$ level. In other words, the population limitation hypothesis is soundly rejected for this vole population, and therefore it is also rejected as a universally applicable general hypothesis. We have now followed the steps prescribed

by Murray: formulated a falsifiable hypothesis, tested it with real data, and rejected it. Have we made any progress towards an understanding of the dynamics of this vole population, or towards population dynamics in general? I don't think so.

Let me now present a positive alternative to the approach advocated by Murray. The main question motivating my research on population dynamics has been succinctly stated by Royama (1992:1), "why do populations behave as they do?" Specifically, we ask the following series of questions about any particular population. First, are dynamics of this population characterized by a stationary distribution of densities? If yes, there is some characteristic mean level around which the population fluctuates, and fluctuations are characterized by a certain (finite) variance. What ecological mechanisms are responsible for setting this mean level? Why does not this population fluctuate more, or less? Furthermore, what is the process order – is population change affected only by immediate density dependence, or are there also delayed density effects? Are there detectable periodicities? If so, what ecological mechanisms are responsible for the *oscillations* in density?

Perhaps I should clarify what I mean by *mechanisms*. The mechanistic basis for population ecology is provided by the properties of entities one hierarchical level lower than populations, that is, by the behavior and physiology of individual organisms. Thus, ultimate explanations of population dynamics should be built on the basis of individual consumption, growth, and reproduction rates; the probabilities of being killed by a predator or succumbing to a pathogen; characteristics of individual movement; and so on. In practice, however, it is not always possible or desirable to follow such a reductionist program to the logical extreme, and we find that some level of abstraction can be quite productive. For example, when studying a predator-prey interaction, we need not follow each individual predator while keeping track of its size, sex, hunger level, spatial position, and so on. We might instead summarize this wealth of information with just a few numbers – e.g., the number of predators in each size class at any given time, or the density of predators in each patch, or even, most simply, the density of all predators. The mapping here is "many to one", because many potential descriptions in individual terms will map to a single number or set of numbers at the population level. Thus, an understanding of predator-prey dynamics may be approached in two steps: in the first step the investigator performs a careful study of the individual predation process and attempts to summarize it with simple relationships, such as the functional response curve. In the second step, functions summarizing behavior and physiology of individuals serve as building blocks in a population dynamics model. In sum, *ecological mechanisms* can refer both to detailed descriptions of what individuals do, and to functions summarizing salient features of individual behaviors. It is perhaps more satisfying to build fully mechanistic explanations of population dynamics that are firmly based on what individuals do, but it is not necessary to do it in one step. History of population ecology shows that such concepts as "population density", "functional response", and "density-dependent population growth rate" turn out to be very useful in connecting population dynamics to individual-based explanations.

How do we build mechanistic explanations for "why populations behave as they do"? There are three general approaches: statistical analysis of observational (e.g., time-series) data, mathematical modeling of mechanisms, and experiments. Until recently, ecologists (at least, in North America) have tended to emphasize manipulative experiments as *the* way to address ecological questions. The narrow focus on experiments at the expense of attempts to synthesize all three approaches is counterproductive, in my opinion. In fact, the experimental approach is most powerful during the later stages of an investigation into dynamics of any particular population, after time-series analyses have reduced the number of viable hypotheses (see below), and the potential mechanisms have been modeled mathematically to obtain quantitative predictions that can now be tested experimentally. This observation makes it clear that I advocate a synthetic approach, in which time-series analysis, models, and experiments are all linked together. Although in this paper I focus on time-series analysis (as the approach that is most relevant to the issue in hand, being in fact an outgrowth of population regulation controversies), I want to stress that time-series analysis is not the only way to investigate population dynamics and regulation. In fact, direct manipulative experiments (admirably summarized by Harrison and Cappuccino 1995 and Cappuccino and Harrison 1996) are a more powerful way of determining whether any particular population is characterized by a return tendency.

Time-series analysis is a particularly useful approach during the initial stages of investigation (assuming time-series data are available, which they often are), when little is known about mechanisms underlying the pattern of population change. One goal of exploratory time-series analysis is description of the fluctuation pattern (e.g., the amplitude, periodicity, Lyapunov stability, and the estimated ratio of endogenous versus exogenous influences). The second goal is to characterize the structure of density dependence, by which I mean two aspects of dynamics, in particular: the order of the process (that is, the number of lagged densities that influence r_t), and the shape of the functional relationship between r_t and lagged population densities. How can results of such analyses be useful, beyond the purely descriptive function? When starting an investiga-

tion into dynamics of any particular ecological system we will typically have a list of candidate ecological processes that may explain population fluctuations. Time-series analysis can help with reducing this list of possibilities. This idea is best illustrated with an example. When I started an investigation into population dynamics of southern pine beetles (SPB) in the late 1980s, the most popular explanation among forest entomologists was that SPB fluctuations are driven by variable climate (see Turchin et al. 1991 for a review). Yet analysis of a 30-year data set indicated that the combined effects of N_{t-1} and N_{t-2} accounted for more than 70% of variation in r_t, and we could not detect any effects of several climatic variables we investigated. Moreover, more than half of variance in r_t was explained by a simple linear regression on N_{t-2}. Thus, our analysis suggested that exogenous factors play only a secondary role in explaining SPB fluctuations, and that we need to focus on those ecological mechanisms that act in a delayed density-dependent fashion (Turchin et al. 1991). The class of such mechanisms is, of course, quite broad: maternal effects, interaction with host trees, interaction with natural enemies, and so on. Nevertheless, reducing the list of potential explanations to this class was already progress. We then argued that further progress could be achieved by field experiments designed to test specific mechanisms, and published our predictions of various outcomes that could be expected in a predator-exclusion manipulation (Turchin et al. 1991). Subsequently, a long-term predator-exclusion experiment provided strong evidence for the natural enemies hypothesis (Turchin P, Taylor AD, Reeve JD unpubl.). In sum, this example illustrates both the value of time-series analysis, and the synergism between time-series analysis and manipulative experiments.

Time-series analysis alone cannot prove that a specific mechanism is important in explaining population fluctuations because many ecological mechanisms can produce any given pattern of density dependence. For example, an apparent Allee effect, a positive relationship between r_t and N_{t-1} at low N_{t-1}, could be a result of either a direct cooperation between organisms, or a side effect of Type II functional response by natural enemies, to name just two mechanisms. Similarly, delayed density dependence can arise as a result of a variety of mechanisms, as was discussed in the previous paragraph. This observation suggests that there is a sequence of model types used in population ecology, ranging from most mechanistic (individual-based models) to less mechanistic (models employing functions summarizing features of individuals) to most phenomenological (models of density dependence). It is important not to oversell the value of time-series approaches, since characterizing the structure of density dependence can, at best, get us only a part of the way to an understanding of mechanisms. Yet, it is equally important not to neglect the usefulness of this approach.

Returning to the original issue raised by Murray, I reiterate that "what is density-dependent regulation hypothesis?" is a wrong question to ask. To me density dependence is a research program, a theoretical framework with which to investigate the causal factors of population fluctuations, not a hypothesis to be falsified or corroborated. In any particular situation, of course, we can apply a statistical test to determine whether the population process is stationary or not. In itself, however, this is not a particularly interesting question, and has value only if it is a first step of an investigation into the causes of population fluctuations.

Although detailed criticism of Murray's logic is not the goal of this paper, it is worth addressing one of the secondary issues, the negative view Murray takes of mathematical models as exemplified by his objection to my phrase "healthy doses of mathematics" (in Turchin 1995). Perhaps the best way to demonstrate the value of mathematics is to take Murray's verbal description and his numerical example illustrating his "population limitation hypothesis" and to translate it into a mathematical model. The functional relationship implied by the numerical example is depicted in Fig. 2b (see the caption for algebra). There is nothing remarkable about this function (apart from, possibly, the sharp corner at K/λ), although it differs from the form implied by Murray's Fig. 2 (compare Fig. 2a to b). From the dynamical point of view, the important observation is that this function is characterized by a negative slope in the vicinity of the equilibrium (K), where r_t changes its sign. Thus, contrary to what Murray says, the population system in his numerical example actually exhibits "density-induced negative feedback loop". There is no need to imagine "density-independent processes [that] have a density-dependent effect". The general point here is that mathematics provides us with a rigorous, formalized language to state our hypotheses and to derive explicit and quantitative predictions from them. Controversies about how to translate biology into mathematical assumptions and how to relate model predictions to data are legitimate and productive. But arguing about what predictions are implied by the assumptions is as silly as arguing about whether $2 + 2$ is really 4. The issue of whether we need density-dependent processes for population regulation is this kind of question. Once you translate what you mean by "density dependence" and "regulation" into mathematical terms, the formal methods of mathematical proof will tell you whether the second is a logical consequence of the first or not.

At this point I need to reiterate that quantifying density dependence is not the same as determining the biological mechanisms responsible for various aspects of population dynamics, such as keeping density within certain bounds, or inducing oscillations. It is important

to stress this caveat because many controversies in the past resulted from the critics of the so-called "density-dependence paradigm" thinking that the other side does not understand this distinction. For example, Chitty (1996) stressed that an explanation based on population density is not an explanation at all, because density is a derived concept. For example, it is not the density of predators that affects the density of prey, but the probability that a prey individual would encounter, and be killed by, a predator individual. The same general point underlies, I think, the distinction between "statistically significant density dependence" and the biological mechanisms explaining why populations are regulated, which was stressed by Wolda and Dennis (1993) and den Boer and Reddingius (1996). In fact, I do not disagree with this point but qualify it by pointing out that there is a hierarchy of explanations in population ecology, ranging from completely mechanistic (individual-based) to largely phenomenological (density dependence) ones. Thus, suppose we find that some population is characterized by the relationship depicted in Fig. 2b. We are still a long way from a mechanistic understanding of regulation in this population because this pattern of density dependence is equally consistent with population regulation by territoriality or by sharing a fixed amount of food, to name just two of many possible explanations (I think this is the point that Murray is trying to make). However, we have made progress, because the observed pattern is not consistent with any conceivable mechanism of regulation (for example, a dynamical interaction with food or specialist enemies should lead to a delayed density-dependent relationship between r_t and N_{t-2}, not N_{t-1}).

To close, progress in ecology results from collecting and analyzing data using innovative techniques, developing insightful mathematical models, performing well-designed experiments, and, particularly, from interactions between the different approaches. As far as I can see, the research program advocated by Murray makes no contribution to any of these approaches. It is particularly amazing to me that Murray presents not a single real data set in this or previous papers he wrote on the subject of density dependence (Murray 1982, 1986). By contrast, papers and books by Berryman, den Boer and Reddingius, Dennis, Hanski, Hassell, Royama, Sinclair, Wolda, and others, are practically crawling with data sets. I am convinced that the convergence between the Nicholson and Andrewartha "schools" is occurring largely as a result of the actual experience of analyzing data back and forth and thinking very hard about what the results mean.

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