 RESEARCH ARTICLE

Relative Frequency Controversies and the Growth of Biological Knowledge

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Relative frequency controversies, so common in the biological sciences, pose something of a puzzle. Why do biologists routinely engage in disputes that (a) are rarely settled and (b) arguably wouldn't yield interesting knowledge even if they were? Recent work suggests that relative frequency controversies can lead biologists to increase their understanding of the modal profile of the processes under dispute. Here, we consider some further consequences of this view. We contend that relative frequency controversies can generate recurrent, transient underdetermination about which causes are responsible for producing particular effects. As a result, the increases in understanding these controversies provide can come with decreases in biologists' ability to offer warranted explanations. We argue that this fits with a toolkit view of biological theory, and suggest some implications for the scientific realism debate as it pertains to biological science.

Keywords

biological theory • underdetermination • scientific controversies • relative frequency • realism

> (With things,) there is their being so and there is how they come to be so. In their being so they may be the same, but how they come to be so is not necessarily the same.
>
> –Mozi (Johnston 2013, chap. 45.3)

1 Introduction

Many controversies in biology revolve around how frequently different processes—fixation of mutations by selection vs. fixation by drift, genetic inheritance vs. extra-genetic inheritance, etc.—occur in nature (Beatty 1997). Call these "relative frequency controversies." Such disputes are puzzling: they are rarely definitively resolved (Dietrich 2006) and, even if they were resolved,

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knowledge of the relative frequency with which particular processes operate is not especially interesting. They are, however, still epistemically valuable, because they can generate modal knowledge about the conditions under which particular processes occur (Kovaka 2021). For example, controversy over how often speciation is sympatric (i.e., doesn't depend on geographic isolation) has led to a more complete understanding of the particular features of populations and environments that favor different speciation mechanisms (Via 2001). Relative frequency controversies thus contribute to the growth of knowledge even when the question ostensibly at stake—how frequently various processes operate in nature—remains unsettled. They can be knowledge-promoting even when the hypotheses at stake are not (known to be) knowledge-tracking.

Our aim here is to show that relative frequency controversies not only can, and do, produce knowledge, but also shape the dynamics of knowledge-generation in biology. We argue that such controversies generate a characteristic pattern of knowledge-change, involving *both* the growth of knowledge and the loss of it, and that this pattern is rooted in fundamental features of such controversies. By "loss of knowledge," we mean that claims which are both *true* and *warranted* at one time come to lose their warrant at a later time (as explained further below, §3.3). Moreover, growth and loss are intertwined: new knowledge changes the relevant evidential standards, and this results in the loss of warrant for other claims.

Our plan of approach is as follows. First (§3), we show that relative frequency controversies serve as generators of recurrent, transient underdetermination—a state in which multiple theories are equally well supported by available evidence. Kyle Stanford (2006) has argued, based on a historical induction, that our best current theories in biology are likely subject to such underdetermination. Our arguments to this effect rest on general features of biological science, rather than on a historical induction, and thus may be able to escape some of the challenges to Stanford's new induction. Second (§4), a consequence of this underdetermination is that relative frequency controversies can produce understanding even as they decrease scientists' ability to offer explanations of particular events. This is because increasing knowledge of the conditions under which processes of interest can occur can undermine knowledge of which particular process was operative in a particular case. Third (§5), we show how this view of relative frequency controversies fits with a view of biological theory as a toolkit of explanatory resources, and consider the implications this has for scientific realism debates in the context of biology.

2 Relative Frequency Controversies

A relative frequency controversy is a dispute about the (non-zero, non-total) proportion of the instances of a phenomenon that a theory explains (Beatty 1997). These controversies arise in cases where scientists know that a phenomenon of interest *can* be produced by a number of different processes, but do not know how often or in which particular cases these different processes *in fact* produce the phenomenon of interest. Different processes may produce instances of the phenomenon of interest *exclusively* (i.e., there is a one-to-one mapping from a specific process to a specific instance), or *integratively* (i.e., there is a many-to-one mapping from multiple processes to a specific instance). When multiple different processes can produce the same phenomenon, such that not every instance of the phenomenon can be explained in the same way, we shall refer to the phenomenon as being "multiply caused."

Evolutionary biology has a vast stock of such controversies. For example, in many species, members of one sex have evolved extravagant sexual displays that they use to attract mates. Biologists have uncovered numerous sexual selection mechanisms that can account for the evolution of these displays. Here are two. Extravagant sexual displays can indicate that their bearers have

"good genes," which is a desirable quality in a mate. Or, extravagant displays can evolve in response to arbitrary preferences of potential mates, in which case they carry no information about genetic quality. Arguments about the relative frequency of these (and other) mechanisms for producing sexual displays have dominated much of the history of sexual selection research. Further examples of relative frequency controversies abound (Fehr 2001; Skipper 2002; 2009; Plutynski 2005; Meirmans and Strand 2010; Craig 2014; Powell and Mariscal 2014; Wong 2019; Beatty 2022). New species can form due to geographical isolation, or due to non-geographical barriers to gene flow (Via 2001). An allele may become fixed in a population due to selection or due to drift (Dietrich 2006). As both philosophers and scientists have observed, many controversies in the biological sciences are about relative frequency (Lloyd and Gould 1993; Beatty 1997). Though we shall focus on biology, such controversies can arise wherever multiple causes produce similar effects.

One of the more interesting features of relative frequency controversies is that they are rarely resolved in any traditional sense. Instead, scientists typically lose interest in answering the original animating question (e.g., How often is the evolution of extravagant sexual displays driven by mate preferences for good genes versus arbitrary mate preferences?) and abandon the controversy, often after decades of intense disagreement (Hey 1999; Dietrich 2006). Loss of interest may be a *kind* of resolution, but it does not involve settling, by reasoning and argument, the original question motivating the controversy.

To date, the literature on relative frequency controversies has focused on the fact that these controversies resist resolution and, in light of this, on whether they have epistemic value. Why do relative frequency controversies "depolarize," that is, fizzle out over time (Dietrich 2006)? What alternative model of controversy resolution can account for their dynamics (Skipper 2002; 2009)? How, if at all, do these controversies further the epistemic aims of science (Beatty 1997)? Lurking behind this last question is the nagging sense that the question explicitly at stake in these controversies is just not that interesting (Beatty 1997, sec. 4). The pessimist may conclude that relative frequency controversies are epistemically worthless, whatever their value for ends such as securing funding.

While the possibility that biologists routinely exhaust themselves in pointless disputes cannot be dismissed out of hand, our focus here is on a different question: How do relative frequency controversies affect the dynamics of theory change and growth in biology? We build on Kovaka's (2021) suggestion that relative frequency controversies have epistemic value because they generate modal knowledge concerning the conditions under which particular processes operate. When scientists disagree about the relative frequency of different processes, they try to resolve their disagreement by investigating the *scope* of these processes, e.g., What is required for a particular kind of sexual selection to occur? In asking about the conditions that favor one process over another, they produce knowledge about how these processes are related to the larger causal structure of the world. That relative frequency controversies generate modal knowledge is central to the three claims we defend in the rest of the paper, including the idea that these controversies are both generative and destructive of scientific knowledge claims.

3 Relative Frequency Controversies Generate Recurrent, Transient Underdetermination

We are primarily interested in how relative frequency controversies change biology's theoretical landscape. In this section, we argue that relative frequency controversies tend to produce recurrent, transient underdetermination. This underdetermination can affect both claims about what explains specific instances of a phenomenon, as well as claims about what overall proportion of

instances of that phenomenon a particular explanation accounts for. At any given time, two or more claims might be *underdetermined* relative to one another, that is, they are equally well-supported by the available evidence. When we expect our evidential situation to change in the future, this underdetermination is likely *transient*, because as new evidence becomes available, it will break the tie between the underdetermined theories. The same scientific areas of inquiry may be subject to transient underdetermination over and over again, in which case the underdetermination is also *recurrent*. We owe the notion of recurrent, transient underdetermination to Kyle Stanford (2001; 2006; see also Turner 2005; 2007); we address differences between his account and ours below (§3.3).

3.1 Case study: speciation

As noted, relative frequency controversies arise when scientists are studying natural phenomena that are multiply caused. This opens up the space for debate about the relative frequency with which these different causes operate. We think it is also one of the features of scientific areas of inquiry that make recurrent, transient underdetermination likely. We explain this latter point below, beginning with a case study, then identifying the more general pattern this case exemplifies.

Our case is the twentieth century debate about allopatric versus sympatric speciation. Prior to the Modern Synthesis, biological consensus allowed that there may be multiple speciation mechanisms, including both allopatric and sympatric. Between the 1940s and 1960s, however, Ernst Mayr (1942; 1963) defended the idea that new species almost always form due to geographical separation. As long as geography allowed gene flow within a population, he argued, it would be just about impossible for genetically distinct subpopulations to emerge. While Mayr stopped short of declaring that sympatric speciation was impossible, he denied that purported cases of sympatric speciation really did occur without geographical separation (Mayr 1963, 458) and attacked theoretical models of the process (Mayr 1963, 468). The chapter on geographic (allopatric) speciation in his 1963 book *Animal Species and Evolution* begins: "That geographic speciation is the almost exclusive mode of speciation among animals, and most likely the prevailing mode even in plants, is now quite generally accepted" (Mayr 1963, 481).

Mayr was correct that, by 1963, most biologists agreed allopatric speciation was the only speciation mechanism that mattered. The quest to explain how new species form, it seemed, was over. Also during the 1960s, however, two theoretical papers helped keep the idea of sympatric speciation alive (reviewed in Via 2001). Maynard Smith (1966) and Thoday and Gibson (1962) showed that disruptive selection, which favors multiple extreme trait values, can produce stable polymorphisms, followed by assortative mating, and finally speciation. This work helped establish the possibility of sympatric speciation against Mayr's critiques.

Still, in these models, sympatric speciation can only occur in a very narrow range of conditions, and empirical research purporting to identify cases of sympatric speciation in nature (e.g., Bush 1969) was hotly contested. The field conceded that sympatric speciation was possible, but most biologists still did not believe it was an important process in nature. This changed somewhat in the 1980s when theoretical work by Rice (e.g., 1984), Diehl and Bush (1989) and Kondrashov (e.g., 1983a; 1983b; 1986) revealed that the conditions under which disruptive selection can lead to sympatric speciation are not as restrictive as had been supposed (reviewed in Via 2001).

Once it became clear that sympatric speciation need not be vanishingly rare, the debate shifted to a focus on relative frequency (Bolnick and Fitzpatrick 2007, 460; Bird et al. 2012, 175). Molecular phylogenetic techniques have allowed researchers to identify actual cases of sympatric

speciation (e.g., Krug 2011), so with the possibility and actuality of the process established, questions of frequency came to the fore.

Most recently, scientists have expressed both a belief that the controversy is waning, and support for moving on from it. Bird et al. (2012, 176) write that, "The popularity of sympatric speciation is at its peak and if the past is any indicator of the future, it will become less popular …." Some researchers question the very distinction between sympatric and allopatric speciation (Via 2001; Kirkpatrick and Ravigné 2002), while others have urged biologists to move on from trying to classify particular speciation events as allopatric or sympatric at all (Jiggins 2006; Butlin et al. 2012). One consequence of this decades-long debate has been a proliferation of speciation mechanisms, and a number of competing classifications of these mechanisms. The controversy is no longer primarily between allopatric and sympatric speciation. At a minimum, peripatric speciation (involving isolated peripheral populations) and parapatric speciation (involving distinct subpopulations that are exchanging genetic material) are on the scene as well. For these reasons, and because estimating the frequency of sympatric speciation is difficult and frustrating work, the fascination with frequency may decrease in favor of other questions about speciation, such as the extent to which it is possible to generalize the conditions under which sympatric and other kinds of speciation occur (Via 2001).

3.2 The general pattern

The case of sympatric speciation exemplifies a pattern that we think theory-building about multiply caused phenomena often follows: investigators look for *a* theory that can explain a phenomenon. They find one, and this theory is taken by many in the research community to be *the* explanation for all instances of the target phenomenon. When additional explanations for the target phenomenon are recognized, they are initially cast as marginal. Scientists who want to explore these additional explanations face an uphill battle to convince their research community, first that these additional explanations occur in nature at all, and second that they occur often enough to be biologically important. One often sees a shift from debates about whether a mechanism for producing a phenomenon is possible, to debates about whether the mechanism actually occurs in nature, to debates about the relative frequency of the mechanism, including debates about whether the mechanism operates exclusively or integratively.

Underdetermination is another typical consequence of research into multiply caused phenomena. For some time after the development of additional explanations for a target phenomenon, investigators typically cannot identify which instances of the target phenomenon are explained by which theory. Where once scientists believed they had an explanation for all or nearly all instances of a target phenomenon, there turn out to be several possible explanations, and no immediate way of determining which one is correct. Hence, underdetermination.

Relative frequency controversies are both effects and causes of underdetermination. They can emerge in response to the underdetermination generated by research into multiply caused phenomena, and research that expands our knowledge of one or more causes of a phenomenon can in turn generate further underdetermination. This underdetermination is transient, happily, and relative frequency controversies can thus make progress toward resolving it. Even in the best cases, however, residual uncertainty remains about which of the possible mechanisms is the correct explanation for particular cases. In fact, underdetermination goes hand in hand with the fact that relative frequency controversies are difficult to resolve and, indeed, rarely are resolved. If scientists could estimate relative frequency, underdetermination would be less acute. And if there were no underdetermination, a relative frequency controversy would be easy to resolve.

This case, and the pattern it exemplifies, suggest three claims relevant to recurrent, transient underdetermination. First, in sciences such as biology, where many phenomena are multiply caused, we should expect that many areas of investigation will shift from pursuing one mechanism that explains the target phenomenon to pursuing multiple mechanisms that explain the target phenomenon. This generates transient underdetermination. Second, areas of investigation where there are multiple mechanisms that can explain the target phenomenon don't typically resolve into precise knowledge about which of these mechanisms explains each instance of the target phenomenon. Information about the conditions under which different mechanisms apply does increase over time, so underdetermination is partially, but not entirely, relieved. Third, over time researchers keep identifying new mechanisms that can explain the target phenomenon. To take just one example, Mary Jane West-Eberhard did not propose the sensory bias model for sexual selection (in which mating preferences evolve from pre-existing sensory preferences) until 1984, years into the ongoing debate about good genes versus arbitrary choice (West-Eberhard 1984; Fuller, Houle, and Travis 2005). This sets the stage for multiple rounds (recurrence) of transient underdetermination.

3.3 Differences from Stanford's account

Thus, we think there are reasons to expect recurrent, transient underdetermination and unconceived alternatives in some areas of science. This is also what Stanford argues, though our view differs from his in three important respects.

First, we do not expect recurrent, transient underdetermination to persist indefinitely. There are a limited number of mechanisms that can produce a given phenomenon, and once scientists have identified all of them, the potential for unconceived alternatives no longer exists, though underdetermination among conceived alternatives can still persist.

There is a further complication here. Scientists may not know whether or not they have identified all the processes that produce a target phenomenon. Above, we discussed cases where most of the scientific community believed they had identified all of the relevant processes when in fact they had not. The mistake can also run in the other direction. Even if scientists have identified all of the processes that can produce a target phenomenon, they may not know that they have reached this point. Recurrent, transient underdetermination thus emerges at two levels. There is underdetermination generated by continued discoveries of new mechanisms which can explain the target phenomenon *as well as* underdetermination generated by second-order uncertainty about when all of the relevant mechanisms have been identified. For now, we are focused on the first level; the second will become more relevant when we turn to discussions of the implications for realism about the biological sciences (§5).

Second, the generator of recurrent transient underdetermination is different on our account than on Stanford's. For Stanford, recurrent, transient underdetermination arises due to human cognitive limitations: we simply are not very good at exhaustively searching hypothesis space concerning remote aspects of nature. This leaves our theories vulnerable to the problem of unconceived alternatives. Stanford justifies this via a historical induction. His critics reply by citing distinctive features of the institutional or social structure of science in the twenty-first century that may block the historical induction (Godfrey-Smith 2008; Ruhmkorff 2011; Dellsén 2017; see Stanford 2019 for a reply). A second worry for Stanford's account is that the remoteness of some aspect of nature is not fixed, but shifts with technical and instrumental advances that render those aspects more accessible to scrutiny (Laudan and Leplin 1991; Egg 2016; Novick and Scholl 2020).

On our account, by contrast, an important source of recurrent, transient underdetermination is the epistemic structure of relative frequency controversies, which is in turn dependent on features of the target system under study: such controversies may arise wherever multiple distinct causes produce similar effects. In these cases, underdetermination is generated primarily by *conceived* alternatives (at the first level; the second order underdetermination referenced above turns on the possibility of unconceived alternatives).

Nor do we need to rely on a historical induction to make our case. Our claim is that *wherever* relative frequency controversies occur, the potential for recurrent, transient underdetermination lurks, because of what relative frequency controversies *are*. For this reason, we should expect recurrent, transient underdetermination to arise even in contemporary contexts. There may be sociological interventions that improve the ability of scientific communities to explore hypothesis space (Stanford 2019), but there is no comparable sociological intervention to make relative frequency controversies stop generating recurrent, transient underdetermination. While it may be possible to change the incentives surrounding how scientists argue about multiply caused phenomena (and so eliminate relative frequency *controversies*), it is multiple causation itself that generates underdetermination.

Likewise, remoteness plays little role in our account. Relative frequency controversies may arise concerning both remote and non-remote phenomena. Perhaps transient underdetermination persists longer when studying remote phenomena, and undoubtedly technical advances can help break underdetermination, but the underlying logic of inquiry is the same in both cases.

A third difference from Stanford's account concerns which types of claims are affected by recurrent, transient underdetermination. Stanford focuses on general theories proposed as *the* correct theory in a given problem area, e.g., Darwin's gemmule theory as the correct explanation of heredity and development. The problem of unconceived alternatives, combined with Stanford's new historical induction, is meant to show that we have good reason to expect our best such general theories to be no better off, relative to currently available evidence, than at least some theories located in hitherto unexplored regions of hypothesis space.

Where phenomena are multiply caused, however, the conclusion that our existing theories are no better off than unconceived alternatives cannot be the correct picture. In this context, theorizing must be considered at three levels. One level concerns the *existence* of causes; another, *competence*, concerns those causes' ability to produce certain effects; and a third level concerns a given cause's *responsibility* for producing particular instances of an effect (Hodge 1977; 1992; 2013; Stanford 2011; Novick and Scholl 2020). In the cases we are considering, recurrent, transient underdetermination affects responsibility claims: e.g., that *this* case of speciation occurred in allopatry, which in turn affects more general claims about the responsibility of allopatry for speciation qua phenomenon. When biologists discover that sympatric processes are competent to cause speciation under a wider variety of conditions than previously thought, this undermines their justification for believing that allopatric processes were *responsible* for particular cases of speciation, but does not undermine their warrant for believing that allopatric processes *exist* or are *competent* to produce those cases.

Perhaps counterintuitively, then, as biologists learn more about the competence of particular causes to produce a given effect, they may (at least temporarily) have less warrant for claims that particular causes were indeed responsible for producing particular effects. Growth of knowledge about causal competence may undermine knowledge of causal responsibility. In cases where the claims that have lost their warrant in this way are true, there is a sense in which *genuine scientific knowledge has been lost, and this loss is in fact a sign of epistemic progress.*

This may seem a striking and implausible claim: one might worry (as one of our reviewers did) that the claims in question were never really warranted at all. Rather the claims merely

seemed warranted, and they seemed warranted only because scientists had been "neglecting or dismissing other serious possible causes." We think, however, that we are working with fairly standard and appropriate notions both of scientific knowledge and of warrant.

By "scientific knowledge," we refer to beliefs, endorsed by some relevant community of inquirers, that are both *warranted* and *true*. We take truth to be eternal and warrant to be contextual. To say a claim is warranted at a given time is to say that it satisfies the evidential standards (for claims of its type) at that time. Warrant for a claim may both increase and decrease over time, both as new evidence is gathered and as evidential standards change. Thus, claims that are warranted at t_1 may cease to be warranted at t_2. Moreover, this may happen *even if the claim is true*. Thus, at t_1, the claim counts as knowledge (it is true and warranted), whereas at t_2, it does not (it is true but unwarranted). This is what we mean when we speak of the "destruction" or "loss" of knowledge.

But why think, in such cases, that the claim really was warranted at t_1? In the cases we are considering, this loss of warrant predominantly occurs by the discovery of new ways in which a particular phenomenon can be produced. This introduces new alternatives (or changes our understanding of the plausibility of past alternatives), changing the relevant evidential standards for ascribing a given instance of the phenomenon to any one of these alternatives. Can we not then ascribe the *false appearance* of warrant at t_1 to the neglect or improper consideration of these serious alternatives?

However, what counts as a serious alternative is itself contextually determined. If a possible cause (e.g., sympatric speciation) has been conceived, but the best available evidence provides good reason to think that it cannot explain the phenomenon in question, it is reasonable to rule it out as a live possibility. Likewise, before a cause has been conceived, it is reasonable not to consider it. This can be nuanced: it is unreasonable to find one possible hypothesis and stop *looking* for alternatives; *some* deliberate exploration of hypothesis space is required. The standards for this will again be contextually determined; if they have been met, a claim can be warranted even if it fails to consider certain unconceived alternatives.

We think this is the appropriate conception of knowledge to apply to science. As our knowledge of possible causes and their potential explanatory power grows, evidential standards will change. We cannot know in advance how these standards will change; the best we can do *now* is to use the best available standards. This is precisely why it is important to distinguish warrant from truth: truth is inaccessible except through warrant; to require that warrant, once acquired, cannot be lost, renders warrant equally inaccessible. This opens up the possibility that genuine warrant for true claims will in some cases be lost.

Our claim is that such loss of warrant not only *can* happen, but that the nature of relative frequency controversies means that it in fact *does* happen, not just sometimes, but in predictable ways. The next two sections explore the consequences of this idea, both for the interplay of understanding and explanation in the biological sciences (§4), and for our models of changes in biological theory over time (§5).

4 Gaining Understanding While Losing Explanatory Power

We've seen above that relative frequency controversies generate recurrent, transient underdetermination within particular areas of inquiry. In this sense, they are often destructive of knowledge. At the same time, they contribute to the growth of knowledge by expanding scientists' understanding of when particular processes are competent to generate certain effects. In a sense, relative frequency controversies allow scientists to *understand* more about competence even as

they are able to *explain* less about responsibility. In this section, we clarify what occurs in such cases.

4.1 The destruction of knowledge

The paradoxical flavor of saying that scientists understand more even though they can explain less is linked to the widespread view that scientific understanding requires grasping a correct explanation (de Regt 2017; Khalifa 2012; 2013; 2017; Strevens 2013; Trout 2007). How can relative frequency controversies increase understanding while diminishing scientists' ability to justifiably offer correct explanations? Because what scientists better understand in these cases is distinct from what they become less able to explain. To make this point, we draw on the framework for causal pattern explanations developed by Angela Potochnik (2017, chap. 5).

On Potochnik's view, scientific models provide a grasp of causal patterns, understood as causal regularities manifested in real-world phenomena. Potochnik (2017, 139) recognizes two explanatorily relevant features of causal patterns: "(1) they show how the phenomenon to be explained causally depends on one or more properties of the world, and (2) they indicate the *scope* of that dependence." The first concerns how the causal process operates, when it operates (e.g., how disruptive selection can generate sympatric speciation). The second concerns the range of background conditions under which it operates. Both features involve existence and competence claims associated with the causal pattern.

To this, we add a third explanatorily relevant feature, related to but distinct from a causal pattern's scope. This is the *domain* of actual instances of the phenomenon that are produced by the causal pattern in question. Scope is conditional: it concerns the conditions under which the causal pattern *would* manifest *if* those conditions were met. Domain is actual: it concerns the range of instances of the phenomenon that are *in fact* produced by the manifestation of that causal pattern—i.e., responsibility. A causal pattern with a broad scope (a very wide range of conditions under which it manifests) may nonetheless have a limited domain, so long as those conditions are only rarely met.

Using this distinction between the scope and the domain of a causal pattern, we can distinguish between explaining a phenomenon and explaining its instances. Explaining a multiply caused phenomenon involves knowledge of the two factors Potochnik identified: the causal dependencies involved in producing the phenomenon, and the scope of those dependencies. Explaining an instance of a phenomenon, by contrast, involves showing that it falls within the domain of a particular causal pattern. In what follows, we examine and qualify the notion of explaining a phenomenon, but this rough distinction will do for now.

As we saw above, relative frequency controversies can generate knowledge of the modal profile of a causal pattern. For instance, debates concerning speciation processes have increased knowledge of the conditions under which different speciation processes can operate. They have shown, among other things, that disruptive selection is a factor that can enable sympatric speciation (Maynard Smith 1966; Coyne and Orr 2004). In this sense, biologists discovered (presumably) true explanations of the phenomenon of speciation. At the same time, however, their ability to explain particular instances of speciation diminished, as the growth of modal knowledge generated underdetermination about the respective domains of the various causal patterns competent to generate speciation.

This characterization of relative frequency controversies does not challenge the dominant view that understanding requires grasping a true explanation. Understanding the modal profile of a causal pattern contributes to explaining the phenomenon that causal pattern produces. It does so even if, for most instances, it is unclear which among several potential causal patterns

produced those particular instances—even if scientists cannot explain very many instances of the phenomenon.

Rather than severing the understanding/explanation link entirely, our case suggests instead that explaining a general phenomenon such as speciation and explaining its instances are to some degree independent endeavors. Though understanding the domain of actual instances of the phenomenon ultimately requires integrating understanding of scope and explanations of instances, this integration may be possible only much later in the investigation of a phenomenon. In earlier stages of investigation, scientists may find themselves in possession of a true explanation of a phenomenon (in the sense that, in principle, it could be successfully invoked to explain many of the phenomenon's instances) without knowing, for most instances of that phenomenon, whether that explanation applies.

Our view goes further, however. It is not just that explaining a phenomenon and explaining its instances are semi-independent endeavors. Rather, the growth in modal knowledge that allows scientists to explain phenomena can come *at the expense* of their ability to explain instances of those phenomena. This occurs when relative frequency controversies generate underdetermination.

Consider an instance of speciation that appears, at t_1, to require explanation by a model of allopatric speciation, on the grounds that recombination prevents disruptive selection from producing a stable polymorphism and, consequently, the assortative mating required for sympatric speciation (à la Felsenstein 1981). Suppose, further, that the explanation is factive: this instance was in fact the result of allopatric speciation. Later, new information reveals that there is a mechanism for limiting recombination in sympatry: linkage between loci under disruptive selection and mating loci. Thus, this could be a case of sympatric speciation. This new information generates underdetermination, with the result that the allopatric explanation is no longer justified. By assumption, however, the allopatric explanation is correct, in which case biologists will have lost their warrant for asserting the explanation. Where biologists once possessed a justified, correct explanation, now they possess two candidate explanations, neither of which can be justifiably asserted until the underdetermination is resolved. In this way, improvement of explanations of phenomena can (temporarily) diminish scientists' ability to explain particular instances of those phenomena. Because this underdetermination affects both correct and incorrect explanations equally, this amounts to a genuine loss of knowledge.

4.2 What is it to explain a phenomenon?

To this point, we've written somewhat cavalierly about general explanations of phenomena, but the notion bears closer scrutiny. When scientists possess a true explanation of a phenomenon, what do they possess? If we are considering a phenomenon that is defined or individuated by its etiology, this question is easy to answer: scientists possess knowledge of the phenomenon's single type of cause. But relative frequency controversies can arise only when the phenomenon in question is multiply caused, and thus not etiologically defined. For such phenomena, it is more difficult to say what scientists possess when they possess a true explanation.

The difficulty is that, in possessing these explanations, scientists in an important sense *do not know what they possess an explanation of*. An allopatric model of speciation explains speciation, yes, but not all speciation, just some actual subset of its instances. But which instances? While biologists may be warranted in taking certain well-studied cases to be the products of allopatric speciation processes, in the majority of cases they will not, whether for lack of study or due to the underdetermination issues previously raised. Nor do biologists know the general contours of this range of instances (this is precisely what is at stake in the relative frequency controversy).

What, then, does such a model actually explain? What is its explanandum? It does not explain the phenomenon as such, only some unknown subset of its instances. We were searching, however, for some explanatory target more general than just these instances. One tempting possibility is to define sub-phenomena etiologically: there is allopatric speciation, explained by allopatric models, and sympatric speciation, explained by sympatric models. Such may be the eventual outcome of relative frequency controversies, if scientists reach a point where they can distinguish which instances of a phenomenon were produced by which cause (Allchin 1994; see also Darden 1991 on scope changes)—if they achieve explanatory adequacy in the sense of Skipper (2002). But, prior to achieving such powers of discrimination, these etiologically defined subphenomena remain epistemically inaccessible, and this possibility amounts to the empty claim that allopatric models explain what they explain (and sympatric models explain what *they* explain).

The worry, in other words, is that *there is no suitably general, epistemically accessible explanandum for which these models serve as explanans* (what does the explanatory work). What the models explain *in principle*, in the end, are particular instances, and what they explain *in practice* are the small subset of particular instances to which they can be justifiably applied. The models themselves are best conceived as explanatory resources, available to be invoked in explanations (potential or actual) of particular cases, but not amounting to explanations in themselves. And that brings us back to the relationship between understanding and explanation. While our view allows for a close connection between understanding and explanation, the growth of understanding in these cases involves improving our grasp of explanatory resources, not of explanations *per se*. For this reason, *even if we accept that understanding requires grasping an explanatory resource, growth in understanding can nevertheless be accompanied by the decline in the ability to offer explanations using that resource.*

How should we make sense of this? We suggest that it requires recognizing that sometimes scientific investigation provides us with explanatory resources—such as modal knowledge or new models of speciation—that have value even when they cannot, at least for a time, be converted into actual explanations. The modal knowledge acquired over the course of relative frequency controversies is a resource in the scientist's explanatory toolkit, with value over and above its role in explaining particular instances. Scientists develop knowledge about this resource *as a resource*, and they can do this even if the ability to apply it to particular cases remains limited.

In this regard, relative frequency controversies are misleading: on the surface, they appear to be about problems of application. Given two (or more) potential causes of some effect, which accounts for the majority of instances? The value of such controversies does not, however, lie in answering such questions, but rather in learning more about the underlying causal pattern as such, fleshing out their modal profiles. This can be done without concomitantly increasing the ability to explain particular instances—such ability may even (at least for a time) decrease.

5 Theoretical Conflict and Theoretical Change in Biology

Thus far, we have been considering how relative frequency controversies affect the dynamics of knowledge production in the biological sciences. We have suggested that they both generate and destroy knowledge. Our aim now is to connect these contentions to a broader picture of the nature of biological theorizing, and to draw out some implications for debates over scientific realism.

Discussions of scientific realism often concern patterns of change and stability in scientific knowledge over time (e.g., Laudan 1981; Worrall 1989; Psillos 1999; Stanford 2006; Chakravartty 2007). The basic concern is that if, as anti-realists allege, we have good reason

now to expect our best current theories to be replaced, we should not take them to be approximately true. Realists, accordingly, attempt to show that, even when successful theories are replaced, core elements of them are nonetheless preserved in their successors.

Underlying this debate is a particular model of how theoretical conflicts are resolved. On this model, theories are direct, incompatible competitors. They are direct competitors in the sense that they are competing over the same domain, and incompatible in the sense that they make contradictory claims about this domain. As a consequence, when a rival emerges to a currently successful theory, the conflict can only be resolved by one of the rivals vanquishing the other. For instance, Darwinian transmutation replaced species fixism, in the process explaining why fixism was an improvement over certain non-Darwinian forms of transmutation (Amundson 2005, chap. 2).

One problem with assessing the merits of realism versus anti-realism in the context of biology is that, while theoretical conflicts in biology are sometimes resolved as the replacement model predicts, they frequently are not. Even when biologists take themselves to be engaging in disputes that should be resolved by replacement, these disputes often resolve with all rivals left standing. Examples include the conflict between molecular and systems biology (Gross, Kranke, and Meunier 2019), the controversy concerning the neutral theory of molecular evolution (Dietrich 2006), and the debate over Sewall Wright's shifting balance theory (Skipper 2002; 2009). Further, while debates over the extended evolutionary synthesis remain unresolved, there is good reason to think that they, too, will not end in replacement (Buskell 2020). Indeed, recognition that replacement models of conflict resolution do not apply is part of what motivates discussion of relative frequency controversies in the first place (Allchin 1994; Beatty 1997).

This pattern is explicable in light of the distinction between, on the one hand, claims about the nature and scope of a causal pattern (the conditions under which it is competent to produce a phenomenon) and, on the other, claims about the domain of that pattern (the instances of the phenomenon it in fact produced). Theory replacement, as we saw, occurs in cases where theories are direct competitors—that is, in cases where they make strictly incompatible claims, such that it is a matter of *logic* that both cannot be true.

In relative frequency controversies, by contrast, direct competition *between two distinct explanatory resources* occurs only concerning claims of responsibility or frequency of responsibility. If an instance of a phenomenon is produced exclusively by a single cause, multiple processes cannot be responsible. Even in cases where an instance is produced integratively (e.g., a trait that evolves due to both genetic and extra-genetic inheritance), there is still a single story about how these processes interacted. But such conflict at the level of responsibility claims need not, and often does not, produce conflict at the level of existence and competence claims. That one cause exists and is competent to produce a given instance of a phenomenon is logically compatible with the existence of any number of other equivalently competent causes—existence and competence claims are not direct competitors in the way responsibility claims are. This does not rule out direct competition about the competence of a single cause (e.g., disagreement about whether a particular mechanism could generate speciation in sympatry), but these disagreements concern *only* the competence of a single cause.

This ubiquity of conflicting responsibility claims and the comparative rarity of conflicting existence or competence claims has motivated the development of a toolkit view of biological theories (Wimsatt 2007; Booth, Mariscal, and Doolittle 2016; Novick and Doolittle 2019). On this view, the core of biological theories are explanatory resources: related, but not unified, tools for making sense of domains of inquiry (cf. Currie 2019). A model of sympatric speciation, for instance, is just such a resource. By itself, it explains nothing (it merely states how speciation might occur), but it is available to use in explaining particular cases of speciation, or for other

explanatory purposes. Because they explain nothing on their own, explanatory resources are rarely in direct conflict with one another, and indeed the contingency of life's history should lead us to expect the multiple causation of biological phenomena (Beatty 1995; McConwell 2019) and thus the need to draw on multiple distinct explanatory resources to explain all instances of a given phenomenon. Instead, explanatory resources tend to conflict when it comes to applying them to particular cases. The toolkit view thus accounts for the pattern of many conflicting responsibility claims, but few conflicting existence or competence claims.

The toolkit view suggests three layers of biological theorizing: the development of particular explanatory resources, elucidating relations and interactions between these, and applications of resources to particular cases. Alan Love (2013) provides a helpful example in the context of evolutionary-developmental biology (see Buskell 2020 for a similar analysis of the extended synthesis). Love (2013, 329) analyzes Carroll's (2008) genetic theory of morphological evolution as consisting of a variety of explanatory resources (e.g., models of pleiotropy and gene regulation; first level) that are organized into an explanatory template (second level) that can be invoked to explain particular instances of the evolution of form (especially animal form; third level).

Armed with the toolkit view's model of theoretical conflict, we can return to the question of realism. Above (§§3–4), we argued that the dynamics of relative frequency controversies often involve the growth of modal knowledge concerning the conditions under which particular processes can occur, even as they are destructive of biologists' ability to justifiably invoke these processes to explain particular cases. This suggests is that claims about responsibility are often more fragile than claims about competence (Novick and Scholl 2020; Novick et al. 2020). Discovery of a new process capable of producing some effect, or discovery that a known process can apply under a wider range of conditions than previously suspected, can generative alternative explanations of particular cases, and so undermine our warrant for invoking any particular explanation thereof. Relative frequency controversies generate such discoveries (Kovaka 2021), and so generate recurrent, transient underdetermination of responsibility claims. At the same time, such discoveries do not challenge competence claims in the same way. For instance, discoveries showing that sympatric speciation can occur under a wider range of conditions than previously realized do not challenge allopatric speciation models *as resources*, however much they challenge particular applications of those models. At the intermediate level of theorizing, the discovery of a new resource (or expansion of an old) will make possible new accounts of how various processes interact, and may or may not challenge old accounts.

In themselves, these reflections are neither realist nor anti-realist. We are recommending neither a general realism about existence and competence claims nor a general anti-realism about responsibility claims. Rather, we are offering suggestions for how to approach the question of realism at all, in disciplines where relative frequency controversies are common. In such areas, it is important to recognize distinct layers of biological theorizing. These layers are subject to distinct dynamics of change.

Rather than asking (as the traditional realism debate does) whether we should expect our current best theories to be replaced, we can ask which kinds of explanatory resources are most vulnerable to replacement. Similarly, we can shift from investigating whether core elements of old theories preserved in new ones to investigating how theory preservation works at different levels of theorizing. Such a reorientation brings the realism debate more in line with the actual dynamics of theoretical conflict in biology, and allows for a better assessment of the epistemic consequences of realism or anti-realism for the biological sciences.

6 Conclusion

Our starting point in this paper was the idea that relative frequency controversies contribute to science by producing knowledge about the conditions under which particular processes occur. We then argued that in producing this knowledge, relative frequency controversies both increase our understanding of explanatory resources and decrease our ability to explain particular cases, due to the generate recurrent, transient underdetermination they generate. Explaining how it is that relative frequency controversies can both produce and destroy knowledge led us to the conclusion that understanding theory change in the biological sciences depends on recognizing different levels (existence, competence, and responsibility of causes) of theory-building in biology.

The arguments we have provided are of a primarily logical character. Given certain characteristic features of a particular kind of scientific inquiry (the kind that gives rise to relative frequency controversies), we should expect that such inquiry also has certain other, philosophically interesting features. We have, in effect, provided a template for thinking about the growth of scientific knowledge: where you find (a) attempts to explain phenomena with multiple etiologies, expect to also find (b) recurrent, transient underdetermination regarding the explanations of particular instances of that phenomenon, and thus also (c) growth of "how possibly" (modal) knowledge at the (at least temporary) expense of knowledge of "how actually" knowledge.

We have discussed these points with continuous reference to a particular case study: the long-standing relative frequency controversy concerning mechanisms of speciation. The history of this controversy shows that, in at least one actual case, the course of inquiry has looked as our template predicts, thereby illustrating the explanatory capacity of our account (Currie 2015). Importantly, we are not making an inductive projection from a single case to relative frequency controversies more generally, which would be problematic for obvious reasons. The engine of generalization lies in the logical relations between the different features of our template. That—and not its successful application to a single case—is what gives us confidence that this template may capture a general feature of a particular kind of scientific inquiry. But, ultimately, our view is best understood as itself an explanatory resource, and its domain remains to be fully determined (we have focused primarily on its scope).

One might worry at this point that the particular kind of scientific inquiry we have chosen to focus on is in fact one we should seek to eliminate, rather than one that should guide further philosophical research into biological theory-building and scientific realism. After all, the outcomes of relative frequency controversies are predictable. Shouldn't scientists recognize this and then save themselves the trouble of engaging in these controversies in the first place? Perhaps. But we are not committed to the claim that relative frequency controversies are, all things considered, good (or bad) for biological research. The template we have developed here depends only on the nature of multiply caused phenomena, regardless of the particular shape taken by debates surrounding these phenomena.

Further study of multiply caused phenomena or of particular relative frequency controversies could, of course, reveal that our template does not often apply. But the reason for this would have to lie in some error in the reasoning by which we arrived at it: either that we overlooked the effects of some salient factor, or that we have mischaracterized some or all of the relationships between the features of relative frequency controversies that we do discuss. In either case, the failure would be a philosophically interesting one—certainly more interesting than just another lesson on the risks of enumerative induction.

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