

# Presidential Address, PSA 2016: An Epistemology of Scientific Practice

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Philosophers' traditional emphasis on theories, theoretical modeling, and explanation mis-guides research in philosophy of science. Articulating and applying core theories is part of scientific practice, but it is not the essence of scientific practice. Insofar as science has an essence, it is to systematically investigate and learn about what is not yet understood. This lecture analyzes genetics to articulate a broad-practice-centered approach to philosophy of science. It concludes by arguing that this approach can lead to richer, deeper, and more useful philosophies of science, philosophies that can better inform science policy and the public's understanding of science.

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**1. Introduction.** I begin today's lecture with my big view about science and the world. I will not argue for the metaphysical part of the view today, but I present my overall view up front to let you know where I am coming from.<sup>1</sup> I believe the world is enormously complex. While aspects of parts of the world

Received June 2019; revised June 2019.

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†This article is my presidential address to PSA 2016. I have presented this work many times and in many places. So many people have provided helpful feedback that it is impractical to acknowledge them individually. So instead I thank them all. I would like to acknowledge Ronald Giere, who has been my chief mentor since graduate school. I have discussed the ideas in this article as they developed with Helen Longino for the past 2 decades and with Alan Love for the past decade. I would also like to recognize Jessica Pfeifer, the first executive director of PSA. Jessica is serving PSA incredibly well and made my time as president rewarding and effective. This research was supported in part by grant 50191 from the John Templeton Foundation: From Biological Practice to Scientific Metaphysics, <http://biological-practice-to-metaphysics.org>.

1. I argue for the metaphysical part of this view in Waters (2017).

*Correction:* "Presidential Address, PSA 2016:" was added to the title of this article on October 30, 2019.

Philosophy of Science, 86 (October 2019) pp. 585–611. 0031-8248/2019/8604-0001\$10.00

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are neatly organized, overall the world lacks a tidy, all-encompassing structure that spans scales. Nevertheless, scientists have learned how to systematically investigate this complexity. Their knowledge enables them to influence and control phenomena without having overall or “fundamental” understandings of the domains they are investigating. Their knowledge also enables them to explain and predict, again, without having an overall understanding. Scientific knowledge is powerful; it is also partial, piecemeal, and perspectival.

To express my point bluntly: the world is a mess. There are no metaphysical fundamentals. This means we cannot have an integrated or unified understanding of it; we cannot even have an integrated or unified understanding of the parts of the world that are most important to us. But science nevertheless succeeds. This view raises two questions that should be at the center of philosophy of science. First, how does science succeed? Second, what is the nature of its success?

A different philosophical view, embedded in a different metaphysics, is commonplace among scientists, science writers, and philosophers. Consider the opening to Herbert Simon’s *The Sciences of the Artificial*, a book that has rightly influenced important philosophers of science, including Ronald Giere (e.g., 1988) and William Wimsatt (e.g., 2007), whose writings have informed my own. Simon says:

A natural science is a body of knowledge about some class of things, objects or phenomena in the world: about the characteristics and properties that they have; about how they behave and interact with each other. The central task of a natural science is to make the wonderful commonplace: to show that complexity, correctly viewed, is only a mask for simplicity; to find pattern hidden in apparent chaos. (1996, 1)

I would like to point out two assumptions in Simon’s conception of science. First, he assumes that science is the outcome of what is produced by the activities of scientists rather than the activities themselves. As he puts it, “A natural science is a body of knowledge.” His second assumption is that complexity is an illusion. “Correctly viewed,” he claims, “complexity . . . is only a mask for simplicity.” The implication is that science reveals the world to be simple. These two assumptions frame most work in contemporary philosophy of science, but I believe both are mistaken. If you try to place today’s lecture in this framework, you will be confused. So please keep in mind my big view: the world has lots of structure but no fundamental, all-encompassing structure. This, and the fact that scientists have many different purposes, means that scientific knowledge is pluralistic as well as partial, piecemeal, and perspectival.

The view of scientific knowledge that I received as a graduate student studying the history and philosophy of science in the 1980s went something

like this. There is an independent world. The fundamental aim of science is to describe and explain this world. Science is organized into disciplines, each of which has a domain of inquiry, a part of the world it seeks to describe and explain. For example, the fundamental aim of Newtonian physics was to explain motion in physical systems. The fundamental aim of Darwinian biology was to explain the diversity of life. Explanations in a mature discipline, I was taught (by scientists as well as historians and philosophers), emanate from a core theory, such as Newton's theory of motion or Darwin's theory of evolution by means of natural selection. The potential explanatory range of a core theory extends across the entire domain of its discipline. Science in a mature discipline succeeds by filling out the explanatory range of its core theory. When Newton first proposed his theory of motion, he demonstrated how it could explain the orbit of Mars, but he believed it could potentially explain the motion of all planets. Investigators started fulfilling the promise of Newton's science by using the core theory to explain the motion of additional planets, moons, and even the appearance of mysterious comets (as well as terrestrial motions). The ambition was great, and investigators sought to use Newton's theory to explain light and to account for motions associated with magnetism. Newtonian science provided a unified understanding of the world because its explanations were all structured by its core theory. According to this view, a discipline advances as its core theory is refined and developed in ways that empower scientists to explain more and more phenomena within the discipline's domain.

Philosophers of science focused their attention on the central theories of science (their fundamental concepts and theoretical explanations) and the way these theories were justified. Some philosophers concentrated on "general issues" seeking answers to metalevel questions such as "what is a scientific theory?" and "what is scientific explanation?" Others focused on mature disciplines (i.e., fundamental physics) and sought to answer questions about particular parts of scientific knowledge, such as "how should we understand the special status of measurement in quantum mechanics?" Philosophy of biology was emerging as a possibly respectable field of study and was largely dominated by concerns about reductionism, which were cast in terms of theoretical reduction and the theory of population genetics, which was taken to be the core theory of contemporary evolutionary biology. Generalists engaged in the realism debate, which concerned the epistemic status of core theories and theoretical claims. Experimentation and field work were taken to be relevant insofar as they provided crucial evidence for theories and theoretical explanations, but they were often viewed as relatively uninteresting. Of course, it would be easy to identify philosophical outliers, but generally, I received a theory-focused view of scientific knowledge, according to which mature disciplines succeed by actualizing the explanatory potential of the disciplines' core theories, which were usually taken to consist

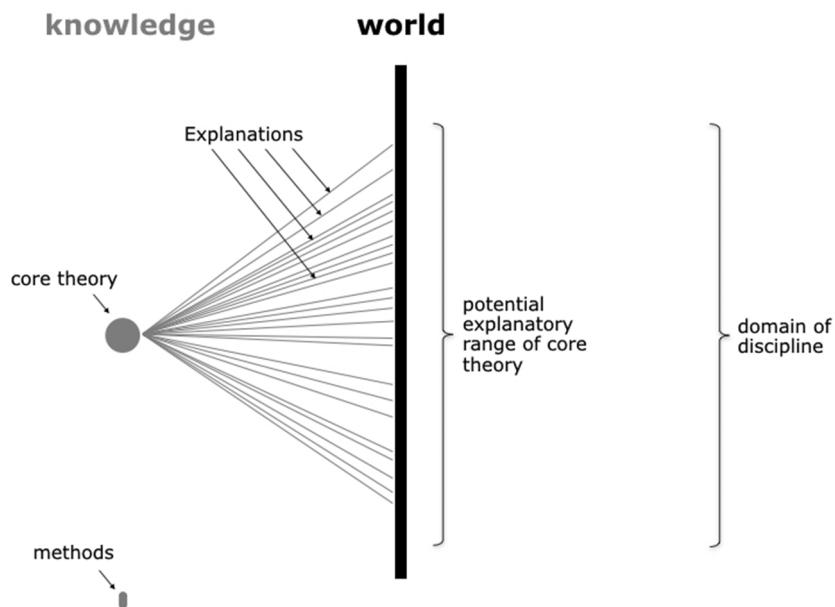


Figure 1. Received view of knowledge in a mature scientific discipline. The long vertical bar represents a part of the world. Knowledge is represented to the left of the bar. Bracket on the far right designates the domain of the discipline (the portion of the world that the discipline potentially covers). The bracket closer to the vertical bar designates the potential explanatory scope of the core theory. Thin gray lines to the left of the vertical bar represent actual explanations. These lines indicate that explanations connect different phenomena in the world to the core theory.

of scientific laws. This view is represented in figure 1. This picture still backgrounds current work in philosophy, even in philosophy of science. I will not have time to substantiate this point today, but I will mention that we could update this view by reformulating it in terms of model types and models.

My ultimate aim today is to argue in favor of a different approach for philosophy of science, one that is already taking root in our profession but one that many in philosophy take to be a weed that is drawing attention away from the ‘real’ philosophical issues. Chang (2014) describes this different kind of approach in terms of analyzing actions rather than products (also see Waters 2014). Woody (2014) offers a multifaceted description that includes moves to treat theories as artifacts and rationality as a social phenomenon. Regardless of its description, the shift in approach is marked by philosophers broadening their attention, for example, toward exploratory experimentation that is not guided by an encompassing theory (Rheinberger 1997; Steinle 2016) and toward data practices (Suppes 2001; Leonelli 2016). It has also been marked by philosophers foregrounding social facets of science that

have been relegated to the background in traditional accounts (Star and Griesemer 1989; Longino 1990, 2002; Solomon 2001). Nevertheless, much philosophical work on scientific practice focuses on practices structured by modeling, explaining, and justifying models and explanations (Giere 1988, 2006; van Fraassen 2008; Weisberg 2013; Peschard and van Fraassen 2018).

Today, I will argue that the traditional philosophical emphasis on theories and theorizing, and the metaphysics on which it rests, misguides research in philosophy of science. I will propose that we analyze science as an investigative enterprise rather than an explanatory one, an enterprise that functions in a very messy world. I believe this approach can lead to richer, deeper, and more useful philosophies of science, philosophies that can better inform science policy and the public's understanding of science.

To make my case, I will discuss my philosophical work on genetics and allied sciences. In the first part of this lecture, I analyze contemporary genetics from a theory-focused perspective. I will identify its core theory and show how its gene-based explanations work. This analysis reveals that the core theory cannot explain much. One might conclude that genetics and allied sciences are not mature or truly successful. Some have found this conclusion appealing, perhaps because it undermines genetic determinism. But I find it implausible. As far as scientific enterprises go, genetics and allied sciences are incredibly successful. They lead to explanations across a wide diversity of phenomena. But more importantly, they provide the means to manipulate and control a wide variety of biological processes. And this makes genetics an incredibly useful tool for investigators. The best way to undermine the ideology of genetic determinism is not to deny the success or maturity of genetics but to show why its success does not rest upon or support genetic determinism.

In the second part of this lecture, I broaden my attention from theory and explanation to investigation. I develop an epistemology of investigation by describing an example of an investigation from cell biology. My analysis of this example will reveal the usefulness of genetics and motivate a broad-practice-centered approach for understanding and analyzing science. I use the cumbersome term 'broad-practice-centered' to distinguish this approach from approaches that focus on practices of theorizing (or modeling or explaining) without analyzing how these practices function within larger contexts of scientific practice. Practice, broadly conceived, includes theorizing and explaining, but it also includes much more: collecting data, manipulating entities and processes, and investigating phenomena for which scientists have no covering theory. I conclude by suggesting that broad-practice-centered epistemology is important for philosophers study of scientific knowledge and also for the public understanding of science.

Before starting, I need to add a caveat. The power of genetics also has been exploited to advance harmful social and political agendas. In this

lecture, I analyze the practice of genetics from a relatively narrow perspective. I will view its success from this perspective. I will not be addressing important questions about whether genetics and allied sciences improve lives or societies. In fact, what I say about the nature of the success of these sciences undermines many of the arguments that have been used to motivate the broad application of them in medicine and agriculture. Placing the investigative practices of these sciences into social and political contexts is of great importance, but it goes beyond the scope of today's lecture. Science, like the world, is complex, and the account I provide today will be partial, piecemeal, and perspectival.

## 2. The Core Theory of Contemporary Genetics.

The “philosopher” is a man with a system which he thinks embodies all that is best worth knowing. (Peirce 1931, 44)

The core theory of contemporary genetics begins with the slogan that DNA is transcribed into RNA, RNA is translated into proteins, and proteins direct the processes in cells and organisms. It is often said that genes direct the maintenance and development of organisms via the production of proteins. Critics have long disputed this idea, sometimes by arguing that there are no such things as genes and sometimes by arguing against the idea that genes have causal efficacy (Oyama 1985; Keller 2000). My aim in this section is twofold. First, I intend to defend the idea that DNA segments called genes have causal efficacy and that gene-based explanations are genuinely explanatory. Second, I wish to show that gene-based explanations have very little explanatory power. Establishing these two points will motivate broadening our conception of practice beyond theories and explanations.

Given that philosophers have argued that gene-based explanations are hopelessly flawed and that scientists and science writers often presume that gene-based explanations have tremendous explanatory power, establishing my two points about the core theory of genetics will require going into technical details. I emerge from the technical details in section 3, to argue that philosophical attention should be broadened; we should analyze investigative strategies to see how they draw on procedural knowledge and data, as well as core theories, in order to advance scientific inquiry.

*2.1. From DNA to RNA to Proteins.* I begin by examining the relationship between DNA and proteins. Many biologists, philosophers, and science writers assume that the relationships between DNA, RNA, and proteins are informational. This is implied by the terms used to describe the processes from DNA to RNA and from RNA to protein: ‘transcription’ and ‘translation’. But biochemists and geneticists have investigated the processes in terms of

chemical syntheses. Instead of saying RNA is transcribed from DNA, I will say RNA is synthesized. I avoid the usual informational language because it is inflationary. I use the language of causation because recent developments in the philosophy of causation have made it possible to clarify the key theoretical ideas in ways that capture the practical consequences of these ideas without adding elements that obscure the basic reasoning. Here, I follow Peirce's (1878) guidelines for clarifying ideas.

It is sometimes said that DNA "produces" RNA and RNA "produces" proteins. But biochemists and cell biologists have shown this to be false. The synthesis of RNA depends on many molecules in addition to DNA. Likewise, the synthesis of proteins depends on many molecules in addition to RNA. That is, the core theory should not be understood as implying that DNA produces RNA on its own or that RNA produces proteins on its own. But the theory does imply that DNA plays a distinctive causal role in the synthesis of RNA and that RNA plays a distinctive causal role in the synthesis of proteins. How should we explicate these distinctive roles?

I have argued that the distinctive roles played by DNA and RNA can be explicated in terms of causation (Waters 2007). First, some background: DNA consists of two long molecules twisted around each other in the form of a double helix. Each strand in a double helix consists of a long string of molecules called nucleotides. There are four kinds of nucleotides in DNA, often symbolized by the capital letters A, T, G, and C. Each strand of DNA can be specified by its linear sequence of nucleotides, for example, a strand might contain the linear sequence ATTGCAG. The two strands of nucleotides in a DNA molecule "complement" one another in the sense that every A in one strand is located across from a T in the adjacent strand, and every G is always located across from a C. So, the linear sequence ATTGCAG in one strand is paired with a sequence TAACGTC in the adjacent strand. An RNA molecule consists of a single strand. These strands are also made up of four kinds of nucleotides, although a nucleotide designated as 'U' takes the place of the nucleotide designated as 'T' (U complements A). Proteins also consist of linear sequences of molecules, but the molecules in proteins are amino acids, not nucleotides. I will use the term 'polypeptide' to designate the linear chains of amino acids that are first synthesized because biologists often use the term 'protein' to designate complexes that include several polypeptides.<sup>2</sup>

The linear sequence of nucleotides in RNA molecules complements the linear sequence of nucleotides in the DNA segments that participate in their syntheses. For example, when the linear sequence of an RNA molecule being synthesized includes the sequence UAACGUC, then the DNA strand

2. I am simplifying. Proteins can take on complex structures that include branching polypeptides as well other atoms and molecules. But the complications I am ignoring here would only strengthen the argument I am making.

participating in the synthesis includes the sequence ATTGCAG. A correspondence also exists between the linear sequence of amino acids in polypeptides and the linear sequence of triplets of nucleotides in the mRNA molecules that participate in their syntheses (this correspondence is often described as the “genetic code”).

What is distinctive about the role of DNA in RNA synthesis? What is distinctive about the role of RNA in polypeptide synthesis? Lots of molecules play critical roles in these syntheses, and this has led some skeptics to conclude that DNA and RNA do not have distinctive roles. They are just one kind of molecule among many. Some critics are interpreted as supporting this view because they have argued against the idea that DNA and RNA molecules provide “the information” in proteins and that proteins provide “the information” for the development and functioning of cells and multicellular organisms. I am sympathetic with some of these latter arguments (e.g., Oyama 1985; Gray, Griffiths, and Oyama 2003). But there is another way to understand the distinctive role of DNA and RNA, namely, that DNA and RNA are causally distinctive. DNA is causally distinctive because it is a maker of actual differences among RNA molecules, and RNA is causally distinctive because it is a maker of actual differences among polypeptides.

I have analyzed this sense of causal distinctiveness by introducing the concept of an “actual-difference maker” (Waters 2007). This analysis conceives of causation as a counterfactual relationship between variables along the lines developed by James Woodward (2003). According to Woodward’s interventionist theory, there is a causal relationship between two variables just in case an appropriate intervention in the value of one variable would lead to a change in value of the other variable.<sup>3</sup> On this view, an effect variable may stand in causal relations to many different causal variables (and vice versa). So, for example, let us take car speed to be an effect variable. The value of this variable for a particular car might be caused by the position of the accelerator pedal (one causal variable), the engagement of gears (a second causal variable), and the incline of the road on which it is traveling (a third causal variable). This means that an appropriate intervention on any of these variables would lead to a change in the speed of a car. What determines the speed at some particular time of a particular car? The answer is lots of variables including the three I have identified. Trying to allocate causal responsibility among the variables in a singular case is a vexing philosophical problem.

3. I am oversimplifying here. There is a causal relationship between two variables if there is at least one combination of values of background variables under which an appropriate intervention on one variable results in a change in value of the other variable. For example, there is a causal relationship between the position of the accelerator pedal and the speed of a car even though an intervention of the position of the accelerator pedal would not result in a change of speed when a car’s engine is turned off.

But the problem situation changes if we are considering causes of differences in actual populations. Instead of focusing on the causes responsible for the speed of a single actual car, let us consider a difference in speeds among three actual cars. I am including the term ‘actual’ to emphasize that this analysis applies to actual differences in the value of an effect variable among the entities of a real population (not potential differences that could counterfactually obtain in this population or actual differences that might obtain in different populations). Suppose the population consists of three nearly identical Toyota Corollas traveling at different speeds along a smooth highway in Saskatchewan. The incline of the road is a causal variable for the speed of the cars, but since the value of this variable is zero for each of them (they are traveling on a prairie road, after all), a difference in incline is not responsible for the difference in speeds. Suppose we consider a number of causal variables and learn they take matching values in the three cars as well: the cars are in the same gear, their emergency brakes are not engaged, and so on. Then, these causes are not responsible for the actual difference in the speeds of these three cars either. But suppose the accelerator pedal is depressed a bit in one Corolla, halfway down in the second, and all the way to the floor in the third. Then, this causal variable is distinctive with respect to the difference in speeds in this population of cars. It is an actual-difference maker.

A causal variable is an *actual-difference maker* of an effect variable in a population of entities when actual variation in the value of the effect variable in the population is determined (at least in part) by actual variation in the value of the causal variable in the population. I will illustrate how this concept can be employed to reveal the distinctive causal roles of DNA and RNA by considering their roles in the syntheses of RNA and polypeptides in a bacterial cell. First, we have to identify an actual difference in some population of entities in a bacterial cell. Let us begin by considering the population of RNA molecules in a bacterial cell. An important difference among these molecules is the difference in the linear sequences of nucleotides within them. Call this the effect variable. What is the causal variable that takes on different values such that these different values cause differences in linear sequences in RNA molecules? The answer is the variable of linear sequences in relevant portions of DNA. The relevant causal variable is the linear sequence of nucleotides in different DNA segments that are participating in the synthesis of the different kinds of RNA molecules.

Lots of molecules play essential causal roles in the syntheses of RNA molecules in a bacterial cell (e.g., RNA polymerase). But RNA polymerase molecules do not take on different values that are responsible for the actual differences among RNA sequences. The same kinds of polymerase molecules play their roles in the syntheses of different kinds of RNA molecules in a bacterial cell. (RNA polymerase molecules are like the parking brakes

that are in the same position in the three Corollas.) But different DNA segments participate in the syntheses of different kinds of RNA. The DNA segments differ from one another because they are made up of different nucleotide sequences. Actual differences in the linear sequences of participating DNA segments determine actual differences in the linear sequences of RNA molecules. So, differences in the linear sequences in DNA molecules are actual-difference makers of linear sequences in RNA molecules in a bacterial cell. The same kind of analysis can reveal the distinctive causal role of RNA in protein synthesis. Differences in the linear sequences among mRNA molecules are actual-difference makers of differences in the linear sequences in proteins in a bacterial cell. (For a fuller account, see Waters [2007].) There are lots of causes in the syntheses of RNA and polypeptides. But DNA is a distinctive cause in the synthesis of RNA because differences in linear sequences in DNA segments are makers of actual differences in linear sequences in RNA molecules. RNA is a distinctive cause in the synthesis of polypeptides because differences in the linear sequences in RNA molecules are makers of actual differences in amino acid sequences in polypeptides.

*2.2. Genes.* Some skeptics argue that there are no such things as genes or molecular genes (e.g., Kitcher 1984, 1992, 2012); other skeptics do not necessarily contest the existence of genes but argue that explanations based on genes are fundamentally flawed (e.g., Keller 2000). In this section, I draw on the analysis in section 2.1 to present two concepts of the gene employed in contemporary biology. In the next section (2.3), I show how these concepts can be used in legitimate explanations, but I also argue that the power of these explanations is very limited.

There are multiple gene concepts used in contemporary genetics. One of them is a blunt concept inherited from classical genetics that takes a gene to be a segment of DNA that can differ in ways that will cause outward (“phenotypic”) differences in an organism. A gene conceived this way is not a gene for anything generally. It is just a gene for an open-ended number of differences. Classically conceived genes are associated with differences, but causing a particular difference is not their function. Thomas Hunt Morgan and his collaborators knew a difference in the “purple gene” caused uniform differences in eye color, but they knew it causes additional differences as well. They knew the chromosomal location of the gene and knew that a difference in the gene caused a number of phenotypic differences. But they did not know what genes were made of, what their functions were, or even how they made their differences (Waters 2004). Today, biologists know genes consist of segments of replicating chains of nucleotides (usually in DNA), and they have a molecular understanding of the proximate function of genes. But it is still useful to think of genes as segments that cause phenotypic differences

in certain investigative and explanatory contexts. When biologists do so, they are usually conceiving of genes in a classical way (Waters 1994). But now there are molecular ways to conceive of genes as well. I have articulated one that I call (perhaps too categorically) the “molecular conception of the gene.” Unlike the classical concept, the molecular concept is a gene for concept.

The molecular concept of the gene is indexical. A segment of DNA is not simply a gene; it is a gene *for a linear sequence in a product synthesized in some cellular context*. This concept can be represented as a four-tuple

$$\langle n, l, p, c \rangle,$$

where  $n$  designates a segment in a potentially replicating linear sequence of nucleotides, usually in DNA (but in the case of retroviruses,  $n$  is a sequence in RNA);  $l$  is a linear sequence in a product along the biochemical pathway from DNA to polypeptide;  $p$  is the product along this pathway— $p$  might refer to RNA (e.g., mRNA, processed mRNA, tRNA, rRNA), or  $p$  might refer to a polypeptide—and  $c$  designates a cellular context, which can include developmental stage as well as cellular constituents. In multicellular organisms,  $c$  might refer to a context that includes tissue location and more. The intuitive idea is that the linear sequence  $n$  determines the linear sequence  $l$  in product  $p$  in cellular context  $c$ .

Gene skeptics reached their conclusion that there are no such things as genes at the molecular level for several reasons, starting with the assumption that the gene was a structural rather than functional or mixed-functional concept. In addition, many assumed that any adequate concept of the gene would need to provide an unequivocal answer to questions about whether a certain segment is a gene or is not a gene. Finally, it was often assumed that if there is a coherent molecular conception, it must provide a single parsing of DNA into genes (or perhaps into genes and other units). All these assumptions are mistaken.

The molecular gene concept is not structural. It includes a structural element  $n$ , but it also includes the proximate function of determining a linear sequence in a particular product in a cellular context. The sense of ‘determining a linear sequence’ has been described in section 2.2. Hence, the concept is mixed functional.

The second and third assumptions behind the skeptics’ critique are mistaken because the molecular gene concept can be applied to pick out different, overlapping DNA segments that determine different sequences along the biochemical pathway from a primary RNA to polypeptide. In bacteria, this pathway is relatively simple. But in eukaryotes, the pathway can be complex because of RNA editing. The sequence of primary RNA (the molecule being synthesized) is often spliced (even as its being synthesized). Certain portions of the primary RNA molecule are removed (the “introns”),

and the remaining segments (“exons”) are spliced together. The processed RNA molecules resulting from splicing play the functional role of determining the linear sequence of amino acids in polypeptides. There is a large literature arguing about whether the parts of DNA that determine the sequence of nucleotides in introns are part of the gene. Some philosophers have thought that the apparent fruitlessness of this debate indicates that the gene concept is hopelessly ambiguous.

The critics’ argument can be understood as follows. I use informational language for the sake of argument here. There is a segment of DNA that codes for the primary RNA, both introns and exons. But there is a collection of split segments that code for just the exons that are spliced together. Biologists sometimes talk as if the gene is the long sequence that codes for both exons and introns. But at other times they talk as if the gene is the split sequences that code for the exons alone. The situation becomes even more complicated because of “alternative splicing.” Some RNA molecules are spliced in different ways. A portion of the RNA molecule that is spliced in the molecule in some contexts is spliced out in others. Sometimes it is an exon and considered part of the gene; other times it is an intron and considered not to be part of the gene. The situation (or one might say our conceptualization of it) seems hopelessly ambiguous, especially when one realizes that some RNA molecules are spliced in hundreds, even thousands, of different ways. Some philosophers have concluded that the gene concept is inherently imprecise and ambiguous. They suggest that the situation would be clearer if biologists dropped the term ‘gene’ altogether (e.g., Keller 2000). But, biologists continue to use the term, and as a philosopher of pragmatist persuasion, I would like to understand why. Is it because biologists are trapped in an ideology of genetic determinism, as some critics suggest, or is there a molecular level conceptualization of genes that is useful?

Let us return to the molecular concept of the gene presented above and consider situations in which an RNA molecule is spliced in one way. Does the molecular gene concept apply to the DNA segment that determines the linear sequence of the primary RNA product (introns and exons) or to the linear sequence of the processed RNA product (just the exons)? The answer is that it applies to both. But the concept is not ambiguous. We simply need to specify  $l$ ,  $p$ , and  $c$ . The gene for the linear sequence in primary RNA molecules includes the DNA portions that determine the sequences within introns as well as exons. The gene for the linear sequence in processed RNA molecules includes only the DNA segments that determine the sequences within exons. The same conceptual practice can be used in cases of alternative splicing. RNA molecules are spliced in different ways in different contexts (the  $c$  in the four-tuple). Provided we specify the value of  $l$ ,  $p$ , and  $c$ , the molecular gene concept precisely picks out the split segment of DNA that determines the linear sequence within a functional product.

The molecular gene concept is not ambiguous; rather, it is flexible and precise. The flexibility is useful because it enables biologists to slip and slide through tremendous complexities to pick out portions of DNA relevant to particular causal pathways. The portions relevant to causal pathways in some tissues or stages of development overlap with portions relevant to different causal pathways in other tissues or stages of development. This means that the gene concept does not parse DNA in a single way. It parses DNA in a multiplicity of ways. But this multiplicity does not represent a conceptual weakness. It represents the virtue of being able to identify hundreds or even thousands of overlapping segments that are relevant in hundreds or even thousands of different contexts in the immensely complicated worlds within eukaryotic organisms.

*2.3. Gene-Based Explanations and Their Limitations.* I have articulated two gene concepts in contemporary biology, the classical gene concept, which is based on the concept from classical genetics, and the molecular gene concept, which is grounded in molecular biology. In this section, I analyze explanations based on these concepts to establish the legitimacy of gene-based explanations and to show that these explanations do not really explain very much.<sup>4</sup>

The molecular gene concept can be used to pick out functional segments of DNA that determine linear sequences in RNA and polypeptide molecules in cellular contexts. Hence, linear sequences within these molecules can be explained, at least in part, by identifying the gene that determines the sequence. A gene-based explanation does not necessarily explain the synthesis, but it does explain why a molecule being synthesized has the particular sequence it has. In cases involving splicing, identifying the gene participating in the synthesis explains the linear sequence in the primitive, preprocessed RNA molecule. The linear sequence in the subsequently processed RNA molecule is explained by identifying the gene that determined the sequence in the primitive RNA and the splicing agent(s) that processed the primitive RNA molecule. If the processed RNA molecule is an mRNA that participates in the synthesis of a polypeptide, then the gene also partly explains the linear sequence of amino acids in the polypeptide. In simpler cases not involving splicing, identifying the gene can explain the linear sequence in the RNA molecule and the linear sequence in a polypeptide (in situations when the RNA molecule is an mRNA that subsequently participates in

4. Griffiths and Stotz (2013) describe limitations of gene-based explanations drawing on more details of the biological processes than I do here. We interpret the limitations differently. We also have different interpretations of my writings. That said, I agree with much of what they say about the basic biological phenomena and limitations of genetic explanations.

the synthesis of a polypeptide). These can be understood as straightforward causal explanations. Thus, gene-based explanations can be legitimate.

While my concern thus far has been gene skepticism, I now turn my attention to gene fundamentalism. According to this view, often presupposed in public discussions of genetics and allied sciences, genes are the fundamental entities of life, the entities that direct the development and functioning of cells and organisms. This view might seem plausible because so much research across the biological sciences focuses attention on genes. Nevertheless, this view is seriously mistaken.

Consider a relatively simple case in which a molecular gene participates in the synthesis of an mRNA molecule that subsequently participates in the synthesis of a polypeptide without the intervention of splicing or other RNA processing. In such cases, identifying the gene can explain the linear sequence of nucleotides in the mRNA molecule and the linear sequence of amino acids in the polypeptide. But it cannot explain why the mRNA molecule and polypeptide molecules are synthesized at the time they are synthesized. Very few genes are actively participating in the synthesis of RNA molecules at any given time. What determines which few genes are active at any particular time? It is not the nucleotide sequence within the gene itself. Gene regulation is immensely complicated and involves regions (e.g., “promoters”) on the DNA molecule separate from the gene segment as well as lots of molecules (e.g., proteins that bind to promoters) that are themselves involved in complex causal networks. The mRNA molecules have relatively short life spans that are influenced by other cellular constituents, and the activity of mRNA molecules is regulated via molecules such as RNAi. Hence, molecular genes cannot explain the timing of RNA and polypeptide synthesis.

One might say gene-based explanations are partial in at least two senses. First, they are partial in cases involving RNA synthesis because genes are not the only causally specific actual-difference makers. Splicing agents can be actual-difference makers too (Waters 2007). So, gene-based explanations are partial in the sense that one needs to identify additional causes to explain why a molecule has the linear sequence it has. Gene-based explanations are also partial in the sense that Hempel (1965) said all explanations are partial. On Hempel’s view, an explanation accounts only for an aspect of a phenomenon, not all aspects of a phenomenon. Gene-based explanations do not explain all aspects of the synthesis of an RNA molecule or polypeptide. For example, they do not explain when (e.g., developmental stage) or where (e.g., in what tissues) or under what circumstances (e.g., environmental stress) these molecules are synthesized, but they do explain the linear sequences within these molecules when they are synthesized.

Furthermore, gene-based explanations are limited in a sense related to Lewis’s (1986) point that the causal explanations we provide are partial because they convey incomplete information about the causal history of an

event. This point can be understood separately from Lewis's metaphysics and his account of explanation. One might say causal explanations identify causal relationships between values of variables at one time with values of the same or different variables at a later time. We could explain the property of a window being broken by the momentum of a ball moving toward the window at a time before the window was in a broken state. But we could also go further back in the causal history to identify the relationship between the swinging state of a leg at a time when the ball was motionless on the ground. We could go back further in time and identify the physiological processes that first put the leg into motion or the training of the child who learned how to kick the ball. All of these are a part of the causal history of the window breaking. As Lewis said, explanations only identify a chunk of this explanatory information.

Causal explanation is *historically indexical*.<sup>5</sup> From the perspective of the period of time from  $t_2$  to  $t_1$ , the dependency between the ball's momentum and the window breaking is the relevant causal relation. But from the perspective of an extension of time that goes further back in the causal history, the identification of the ball's momentum, while still important, offers a very incomplete explanation of the broken window. The explanatory significance of being an actual-difference maker is also historically indexed.<sup>6</sup>

Historical indexicality of gene-based explanations provides a check on gene fundamentalism. Gene-based explanations are indexed to particular stages of incredibly complex causal histories in the cell and beyond. A gene-based explanation might offer a relatively complete explanation why an RNA molecule has the sequence it has with respect to a historical period that begins at a time when certain genes are active. But, with respect to an interval that extends before the relevant gene is activated and from a space that extends beyond the cell, tissue, or organism, the gene-based explanation, even of linear sequences, is very partial. Identifying the gene would play a role in the explanation, but depending on how far back our perspective extends, the gene would play a very important explanatory role or a relatively minor one in accounting for the molecule's linear structure.<sup>7</sup> This is a very significant limitation of gene-based explanations.

The limitations discussed thus far mostly concern processes before and during the synthesis of RNA and polypeptides, but the significance of gene-based explanations is also severely limited because of the complexities of

5. I thank Brian Hanley for helping me clarify my account of historic indexicality.

6. This is a point overlooked in my analysis of actual-difference making concepts in Waters (2007).

7. This point applies to the scales of cell biology; one need not appeal to evolutionary timescales.

what happens afterward. Gene fundamentalism holds that genes are fundamental because they produce polypeptides (or proteins), and proteins direct everything that goes on within the cell and organism. The basic idea is that genes produce proteins with particular linear sequences of amino acids, and the linear sequence of amino acids determines the functional structure of proteins. Hence, genes determine how proteins direct the processes of life. There are many problems with this account, starting with the idea that genes produce polypeptides on their own. But I have shown that (in a technical sense) genes determine (or partially determine) the linear sequences of amino acids in polypeptides. A gene fundamentalist might think this is all that is needed to claim that genes direct the development and functioning of cells and organisms. On this view, (1) genes determine linear sequences of amino acids in proteins, (2) the linear sequences of amino acids in proteins determine their functional properties, and (3) the functional properties of proteins regulate the development and functioning of cells and organisms. All three of these ideas are problematic in the context of gene fundamentalism.

First, although genes determine linear sequences in an important technical sense (sec. 2.1) it would be misleading to say they are the fundamental determinants of these sequences. They are significant determinants over a short temporal period and biological space, but they are less significant over longer temporal periods and biological space because their significance is historically indexed. They are not fundamental determinants.

Second, it is unclear to what extent the linear sequence of amino acids in polypeptides determines the functional properties of proteins. Proteins are often made up of multiple polypeptides and sometimes other kinds of elements as well. The causal situation is complicated by processes that cleave and branch polypeptides and that attach nonpolypeptide elements as complex proteins are being formed from simple polypeptides. The functional properties of protein complexes depend on their distinctive three-dimensional structures. The three-dimensional structure of complex proteins depends on these processes as well as the linear structure(s) of the polypeptide(s) comprising them. It is even doubtful that the linear sequence of simple proteins consisting of a single nonbranching linear polypeptide depends solely on its linear sequence of amino acids. Polypeptides fold into three-dimensional forms with the aid of chaperone molecules. These molecules guide polypeptides into energetically stable three-dimensional states but not necessarily into their energetically lowest possible stable states. Hence, chaperone molecules might play an important role in determining the outcome of a folding process. These complications undermine the idea that a gene determines the functionality of a protein.

The third idea behind genetic fundamentalism, namely, that the functional properties of proteins regulate the development and functioning of cells and organisms, is also wrong. The functioning of proteins is immensely complicated.

A protein typically has many different functions. The functions differ in different processes occurring at different stages of development, in different parts of the cell and organism, and in response to different environmental and internal states. Being in the right place at the right time is not determined by the linear sequence(s) of the amino acids in the protein (or in the linear sequence[s] in DNA that determine[s] the sequence[s] in the protein). Proteins are produced at the right time because of regulatory processes (discussed earlier), including gene activation and mRNA management, not because of the linear sequence of amino acids in the protein (and not because of the linear sequence of nucleotides in the gene). Proteins are in the right place partly because of transportation processes that involve lots of molecules including nonpolypeptides such as lipids. To say the overall functioning of cells is incredibly complex would be an understatement. To say that genes direct this complexity by determining linear sequences in polypeptides would be a gross exaggeration of the relative significance of genes.

So far, I have analyzed explanations based on the molecular concept of the gene and their inherent limitations. Unfortunately, time limitations prevent me from going into similar details with respect to explanations based on the classical concept of the gene. These explanations only address differences in outward (phenotypic) appearances in particular populations. Morgan and his collaborators could use their version of this concept to explain ratios of flies with red eyes to flies with purple eyes in a particular offspring generation. But they could not explain why a fly with the “red gene” developed red eyes. The explanatory scope of their gene-based explanations was extremely narrow (Waters 2004). The same is true of the contemporary use of this concept. The contemporary version of this concept is still useful in contexts in which, despite the complexities, a distinctive difference in a pathway leads to a distinctive difference in an important outcome (e.g., in cases of simple genetic diseases). But these patterns (exhibited within immensely complex systems) do not support the fundamentalist idea that genes are directing the development and function of organisms. They just point to a difference principle which states that distinctive differences in an element can give rise to distinctive differences in outcome (in particular contexts).

### **3. From a Narrow Focus on Theories to a Broad View of Practices.**

It is not knowing, but the love of learning, that characterizes the scientific man. (Peirce 1931, 44)

Philosophers of science often analyze knowledge as a system that is organized for understanding the world. This approach acknowledges that science involves investigation and manipulation, but it takes justified understanding to be the fundamental aim of science. It assumes that philosophers

should focus their attention on the achievement of this aim by analyzing the core theories and explanations of scientific disciplines and showing how these theories and explanations are justified. The first part of this lecture focused on theorizing and explanation. But it yielded a result (represented in fig. 2) that calls for a broader outlook.

Section 2 raises an important question: if gene-based explanations are partial and perspectival, and if their potential explanatory scope is extremely narrow, then why are genes at the center of so many biological investigations? There is no denying that genes are at the center of attention. Open practically any research journal in a biological or biomedical science, and you will see that genes are typically front and center. The collective domain of these sciences is extremely broad, yet the potential explanatory power of gene-based explanations is very narrow (see fig. 2). Why?

One possibility is that researchers are paying far too much attention to genes because they are driven by an ideology of genetic reductionism. They are determined to show that all the phenomena they are investigating can be explained in terms of genes. But there is another possibility. Perhaps genes are at the center of biologists' research because genes are useful tools. To

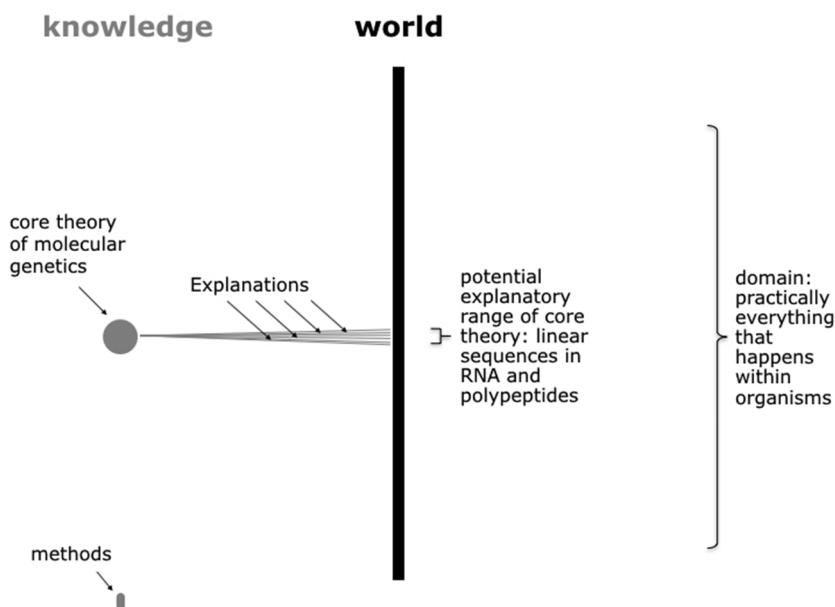


Figure 2. View of the knowledge of genetics and allied sciences from a theory-focused perspective. Rigorous analysis of the core theory and explanations of contemporary genetics and allied sciences reveals a different picture (represented here) than the one represented in figure 1.

explore this possibility, I broaden my attention from a narrow focus on the core theory of genetics to investigative practices of geneticists and allied scientists.

The approach I advance in this part of my lecture assumes that scientists have several basic aims and that none of them constitutes *the* fundamental aim. In particular, I will not assume that the aim of justified understanding is more fundamental to a mature scientific discipline than the aims of fruitful investigation and effective manipulation. A broad-practice-centered approach is open to the idea that knowledge in some scientific disciplines might be structured by practices of theorizing about their domains, but it is also open to the idea that knowledge in other disciplines is structured by an integration of practices aimed at investigating their domains.<sup>8</sup> Such practices are informed by theories and explanations, and they may include theorizing and explaining, but they can also include employing strategies for revealing important information; using material entities; following experimental procedures; and collecting, organizing, and drawing on data. Analyzing the integration of these activities in practice is what I call a “broad-practice-centered” philosophy of science.

I illustrate this philosophical approach by considering an example of investigation typical of cell biology, a discipline that is closely allied with genetics. The investigators whose research I describe draw on the core theory of contemporary genetics. Drawing on the core theory of genetics and gene-based explanations (described in sec. 2) is part of their investigative practice. But it does not structure their practice. Their investigative practice is structured by an integration of different practices that is aimed at producing new knowledge that falls outside the potential scope of the core theory of genetics. The core theory informs their research, but it does not drive it.

*3.1. Case Study of the Investigative Practice in Genetics and Allied Sciences.* The investigation I will discuss is typical of cell biology. Cell biologists often center their research on questions about the function of molecules in cellular processes. The Jorgensen lab at the University of Utah conducted research to learn about the function of a  $\beta$ -spectrin proteins in the development of the neurological system of the model organism *Caenorhabditis elegans* (Hammarlund, Jorgensen, and Bastiani 2007). *C. elegans* is a 1 millimeter roundworm. The neurological system of the worm largely develops in an embryonic stage. The worm has two nerve chords that run along its length, one on its upper or backside (the *dorsal* nerve chord) and the other on

8. Ananya Chattoraj has helped me keep this point in mind and is developing ideas about the variation of practices across different disciplines.

its underside (the *ventral* nerve chord). These chords are connected by *axons*, which are threadlike extensions of nerve cells. During development, the axons grow from the ventral nerve chord toward the dorsal nerve chord. During this growth period, the axons have cone-like structures on their tips that eventually reach the dorsal nerve chord. Upon reaching the dorsal chord, the cone-like structures flatten as axons send protrusions to the dorsal chord.

Investigators had already learned that  $\beta$ -spectrin functioned in the development of the neurological system because worms lacking this molecule exhibited neurological defects ( $\beta$ -spectrin has many functions in animals in addition to its function[s] in the development of neurological systems). Previous experiments showed that  $\beta$ -spectrin is located in growth cones when axons are growing from the ventral to dorsal nerve chord. Growth cones play a number of important functions in axon outgrowth, including the elongation of the axon structure as well as directional guidance.

The Jorgensen lab wanted to learn about the role of  $\beta$ -spectrin in axon outgrowth. Their strategy for doing so was to prevent the synthesis of the  $\beta$ -spectrin protein and observe what goes wrong in the development of the axon connections between nerve chords. To do so, they manipulated two genes. They manipulated the gene for the  $\beta$ -spectrin protein to prevent the synthesis of the protein, and they inserted a gene for a green fluorescent protein to illuminate the neuronal extensions for imaging.

What they learned was contrary to expectations: outgrowth in embryos lacking  $\beta$ -spectrin proceeded normally. Growth cones formed in a regular manner, elongation proceeded normally, and the axons sent protrusions that secured the axon to the dorsal chord. This result led the researchers to suspect that the function of  $\beta$ -spectrin might be in the maintenance of axon connections rather than in the initial outgrowth of the axons. To test this hypothesis, they used genes to image the process and prevent the synthesis of  $\beta$ -spectrin. But this time they observed the process over a longer period of time to observe whether the axon structures were maintained after outgrowth was complete. They observed that after normal outgrowth and connection to the dorsal chord, the axons broke, outgrowth resumed, and the new connections subsequently broke in a fashion that they had never observed. This result confirmed the idea that the function of  $\beta$ -spectrin was in the maintenance, not in the initial development, of axon connections. But it also raised the question of what function  $\beta$ -spectrin plays in the maintenance.

The Jorgensen lab considered two hypotheses about the role  $\beta$ -spectrin might play. Embryos grow throughout development, and the axons between the chords must elongate to accommodate the increase in distance between chords. The first hypothesis was that  $\beta$ -spectrin facilitates the elongation of axons after they have formed connections between the two chords. In addition to growing, embryos also move, which presumably exerts acute strains on the axons that connect the dorsal and ventral chords. The second hypothesis

was that  $\beta$ -spectrin protects axons from acute strains. To test these hypotheses, the researchers immobilized the worms. If the function was to protect axons from acute strains, then the axons should be maintained in the absence of  $\beta$ -spectrin. If the function included facilitating further elongation, then the axons should break as they are stretched.

How did the Jorgensen lab immobilize the embryos? They greatly reduced the production of myosin, a protein necessary for movement. They used genetics to do so, although this time they used RNAi to interfere with the biochemical processes (sec. 2) from mRNA to polypeptide. The researchers also used the knockout gene to prevent synthesis of  $\beta$ -spectrin and the gene for green fluorescent protein to make neurological structures visible under the microscope. What they observed was that the axons were maintained through development when the worms did not move. This confirmed the hypothesis that  $\beta$ -spectrin plays a functional role in protecting the axons against acute strains.

The researchers carried out additional experiments to rule out the possibility that their procedures for preventing movement provided independent protection against strain. They again used a different genes to reduce acute strains on the axons, and the axons were still maintained in the absence of  $\beta$ -spectrin.

The experiments described here employed a general strategy that is used throughout many biological and biomedical investigations. Investigators sometimes call it the “genetic approach,” and it was first employed by classical geneticists (Waters 2004). The strategy is to use genes to interfere with the processes under investigation. The general strategy of learning about processes by interfering with them has a vaulted history in physiology. William Harvey used this strategy to investigate the functioning of the circulatory system. Harvey used scalpels and tourniquets to interfere with the functioning of the circulatory system. His resulting knowledge was not primarily about tourniquets and scalpels; it was about the entities and processes of circulation. Physiologists followed Harvey’s approach to investigate the function of physiological systems quite generally. Contemporary biologists also follow this approach, but they use genes to interfere with processes. Their resulting knowledge is often not about genes or processes from genes to proteins. Their resulting knowledge is about entities and processes that are beyond those described and explained by the core theory of genetics. The conclusions the Jorgensen lab drew were about the role of a protein in the processes of neurological development and maintenance. They used genes to learn about the processes, not to explain the processes.

The core theory of genetics did not direct the investigation described here. The hypotheses the lab proposed and tested were not hypotheses generated from the core theory. They were hypotheses generated by reasoning about proteins and their possible roles in biological processes involving structural

elements not made of DNA. They used genes to test these hypotheses. They used the results of their experiments and their knowledge about neurological structures and embryonic development to generate new hypotheses. This is quite unlike the story we hear about the practice of classical mechanics, which presumably involves testing models of the core theory of Newtonian mechanics. The hypotheses tested by the Jorgensen lab were not models of the core theory of contemporary genetics.

The investigation I have described, however, was informed by the core theory of genetics. Clearly the investigators understanding of how the use of the knockout gene prevented the production of  $\beta$ -spectrin and how the use of RNAi interfered with the synthesis of myosin was based on models of the core theory, as was their understanding of how the insertion of the reporter gene led to the synthesis of the green fluorescent protein. But they also drew on lots of other kinds of knowledge to conduct their research. They drew on procedural knowledge about how to insert genes to image particular processes as well as how to do lots of mundane things. They drew on and produced data about the outcomes of experiments and the existence of genetic materials including knockout genes and RNAi. They also used background knowledge about biological processes including neurological development, axon growth, and muscle contraction. Their research drew on the strategy called the genetic approach. They learned about the role of  $\beta$ -spectrin in processes of axon development by using genes to manipulate the processes (and to make them visible). Genes were central in this research because they were the primary tools for manipulating the processes being investigated.

*3.2. An Epistemology of Scientific Practice.* An epistemology of scientific practice should be centered on practice viewed broadly, not just focused on practices of abstract theorizing, model construction, and testing. It should include practices of investigating as well as practices of explaining. I will offer a view of scientific practice that emerges from my analysis of the practice of genetics and allied sciences.

The domain of the practice of these sciences is extremely broad. It includes practically all processes occurring in organisms. But the domain of the core theory informing these practices is extremely narrow. It covers only a narrow swath of processes from DNA to proteins. And the explanatory scope of its key models, gene-based models, covers only historically indexed explanations of linear sequences in RNA and polypeptides. This is represented in figure 2. There is a mismatch between the domain of the science and the domain of the core theory. It is helpful to distinguish between three domains. First, there is the domain these sciences in some sense “cover.” Second, there is the potential explanatory range of their core theory, the molecular theory of genetics. Third, there is the investigative reach of the practice. This includes the breadth of phenomena geneticists and allied

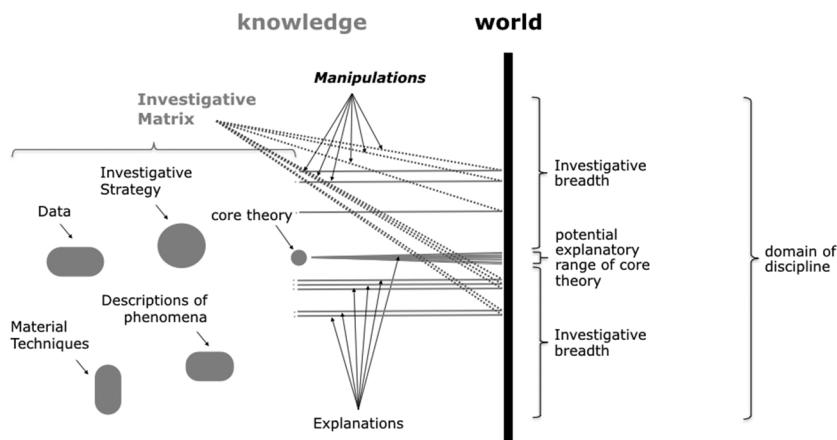


Figure 3. Broad-practice-centered view of scientific knowledge. Manipulating phenomena in the world reveals information that lead to explanations. Dashed gray lines represent manipulations of phenomena being investigated. Performing these manipulations involves drawing upon knowledge within the investigative matrix. Solid gray lines represent explanations of phenomena that were manipulated. The tiny dots to the left of solid gray lines designate models (e.g., mechanistic models) that are not part of the core theory. Investigation is informed but not directed by the core theory.

scientists are investigating that are not potentially explained by the core theory. So, the domain of the scientific practice covers the narrow potential explanatory scope of the core theory as well as the much wider breadth of processes that can be investigated by employing the genetic approach.

Biologists draw on and contribute to different kinds of knowledge when they employ the genetic approach: procedural knowledge, background knowledge about the phenomena they are investigating, and data. I introduce the term “investigative matrix” to cover the different kinds of knowledge that are integrated in the practice of genetics. The investigative matrix of contemporary genetics and allied sciences is represented in figure 3.

Figure 3 depicts investigation as piecemeal. Biologists manipulate and subsequently explain aspects of particular phenomena in a piecemeal fashion without having an overarching theoretical understanding of the phenomena they are investigating.

**4. Why Broad-Practice-Centered Philosophy of Science Is Important.** Philosophy of science should account for how science succeeds. According to the view I have advanced today, mature science succeeds by systematically investigating parts of the world. Successful investigation in science is not structured by core theories but rather by strategies that draw on a number

of different entities and practices. The entities are material as well as epistemic things. The practices include material techniques, collecting and systematizing data, developing and drawing descriptive knowledge, explaining, and theorizing. Science proceeds in piecemeal fashion and is not organized around articulating and applying its core theories. Articulating and applying core theories is part of scientific practice but not the essence of scientific practice. Insofar as it makes sense to say science has an essence, it is to systematically investigate and learn about what is not yet understood.

Philosophy of science should also reveal the nature of the success of science. According to this view, the success of science consists of providing effective means for learning about aspects of parts of the world that are important to us. The success of science does not consist in establishing a fundamental understanding of the world. Science does not provide an understanding of the fundamentals of the world. The world is way too complex for this kind of achievement. In fact, parts of the world that are most important to our lives, such as organisms and cells, have no fundamentals. But science nevertheless provides us with the means to understand and sometimes manipulate and control aspects of the world.

This view has important implications for how we practice philosophy of science. Consider a currently hot topic in philosophy of biology: biological individuality. Much of the philosophical work on this topic focuses on the ontological or metaphysical question about what it is to be a biological individual as if there is a single truth about the matter (see Waters 2018). Philosophers are conducting sophisticated analyses of various biological theories ranging from Darwinian theory to theories of immunology to delimit individuals. While there is a growing trend toward pluralism on this topic, the emphasis is still on the question, "what is an individual?" But the view I have advanced today suggests that philosophers should approach the topic by asking how do biologists conceive biological individuality? In what contexts and for what purposes might different conceptions be useful? Might purposes exist in medicine, public health, or environmental science that would be better achieved by using new concepts of individuality? Pursuing these kinds of questions could move us beyond analyzing science solely in terms of description, explanation, and prediction. It could decrease our obsession with the philosophical methodology of grounding analyses in abstract theories. Pursuing these questions could also lead to deeper and more inclusive understandings of science and a more useful philosophy of science.

This view also has important implications for the public understanding of science. Science writers and journalists often describe science as if its central aim is to get behind surface complexities and reveal the underlying fundamentals. A recent article on measures of inflation published in Toronto's newspaper the *Globe and Mail* detailed several different measures of inflation and the pros and cons of each. The author of the article, the paper's

economics reporter David Parkinson, concludes by suggesting that different measurements might be useful for different purposes. But he begins by describing the concerns of the economists at the Bank of Canada. “[The Bank of Canada will soon] report something called the Bank of Canada’s ‘core’ inflation rate, which is supposed to tell us what’s going on broadly, underneath the surface of short-term price gyrations in a handful of goods . . . the Bank of Canada has been saying that even its core measure isn’t getting at the core of inflation. A few weeks ago, it estimated that true underlying inflation in Canada is more like 1.6 to 1.8 per cent. Pick a number” (Parkinson 2015).

This passage reinforces the idea that scientists, economists in this case, should identify and measure fundamental parameters underlying complex phenomena. The sentence “pick a number” plants the idea in readers that economists might not know what they are doing. The article continues by reporting, “Indeed, the central bank has started looking increasingly at alternative ways of measuring what it calls ‘underlying inflation,’ to look through the temporary, sector-specific inflation noise and get at what is really going on with price pressures in a broader, economy-wide sense.” Then the article introduces its description of the different measure as follows. “Here’s a closer look at some of those measures, how the measures currently differ, and their pros and cons in trying to explain Canada’s prices and the overall economy” (Parkinson 2015). Newspaper readers might conclude that economists are failing because they have not succeeded in identifying what inflation really is or how to truly measure it. But then they learn that economists have different measures, none of which succeed in providing a completely satisfactory explanation of Canada’s prices and economy. The title of the article might reinforce this skepticism: “The Logic and Lunacy of Calculating the Inflation Rate.”

The epistemology of scientific practice I have advanced today would give readers a different way to understand the merits and limitations of economics. If newspaper readers viewed science this way, they would not be assuming that the aim of economics is to establish a core theory that will explain everything. They would be looking for an investigative enterprise that can generate useful knowledge. Instead of asking, “What is inflation?” and “Are economists measuring its true value?” they would be primed to ask: “How do economists measure inflation?” “What purposes do different measurements serve?” “Are economists looking to improve their measurements to better address the variety of needs that might be served?” The article in the *Globe and Mail* provides information to answer these questions. I suggest that philosophers should be developing an epistemology of science that will lead the public to ask questions like these, rather than questions that lead to significant skepticism about the usefulness of science in some members of our society and greatly inflated confidence about the power of science in others.

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