Discovery in Medicine

Chapter for The Routledge Companion to the Philosophy of Medicine

Author: Brendan Clarke, Department of Science and Technology Studies, UCL.

b.clarke@ucl.ac.uk

Abstract

In this chapter, I discuss the discovery of McArdle disease. I then use two philosophical account of discovery – one by Thomas Kuhn, the other by Norwood Hanson – to argue against the idea that discovery is merely a psychological phenomenon.

Keywords

Discovery

Justification

McArdle disease

Thomas Kuhn

Norwood Russell Hanson

Chapter: Discovery in Medicine

Introduction

Medical textbooks often begin with a concise account of key discoveries in the field. In the first chapter of the textbook *Medical Microbiology* (Greenwood, Slack and Peutherer 2000), for example, several of the important milestone discoveries in microbiology are set out. I quote one of these below:

> "Among notable events were: the discovery by James Paget (while a first-year medical student at St Bartholomew's Hospital, London) of the larvae of *Trichinella spiralis* in muscle during an autopsy (1835)…" (Greenwood 2000: 4)

Excerpts such as these illustrate the importance of discovery to medical practice. We can find other signifiers, too, of the esteem in which discoveries (and discoverers) are held, such as the Nobel Prize in physiology or medicine. We might therefore expect discovery to be of the foremost importance for philosophers of medicine. So it might come as a surprise that, far from being a central topic, discovery has been often neglected by philosophers. In the first section of this chapter, I will discuss when and why this was the case. In order to do this, I will give a brief account of the early history of philosophy of science, concentrating in particular on the positivist roots of much contemporary philosophical work. In section two, I will then interrupt this historical story about the place of discovery in the philosophy of science to introduce an extended case-study of discovery in medicine. This is the case of the

discovery of McArdle disease during the 1950s. Finally, in section three, I will use the details of the McArdle case to introduce two substantive accounts of discovery that offer complementary understandings of discovery (and its role) in the sciences.

Section one: the (pre-) history of the philosophy of discovery

Above, I noted that the topic of discovery has been neglected by many philosophers of science. In this section, I will substantiate and clarify this claim by emphasizing that the status of discovery, as a topic of philosophical study, has ebbed and flowed across the twentieth century. I begin by suggesting that the neglect of discovery can be effectively localized to philosophical work influenced by logical positivism during the early twentieth century. I will discuss the reasons that discovery was cast out by these authors by concentrating on the distinction that they drew between discovery and other kinds of scientific work. I will then conclude this section by noting what happened once this distinction was challenged.

Much in contemporary philosophy of science is rooted in the influence of the Vienna Circle (Reisch 2005 gives an accessible introduction). While a detailed discussion is beyond the scope of this chapter, the Vienna Circle was a group of scientists and philosophers who met in Vienna in the period between the first and second world wars. Skeptical of the traditional metaphysical interests of philosophers, they sought to develop a philosophical method compatible with the rigorous and mathematical techniques used in the sciences. Drawing on Whitehead and Russell's attempt to axiomatize mathematics using symbolic logic (Whitehead and Russell 1910), the philosophers of the Vienna Circle sought to bring similar methods of analysis to bear on the structure of scientific reasoning. It was this importance placed on symbolic logic that led to the neglect of discovery as a philosophical question.

Hempel, for example, describes the way that new scientific ideas arise. New scientific ideas are first invented by "guesses" (Hempel 1966: 15), "free invention" or the "imagination" (Hempel 1966: 16) of scientists. These guesses do not come about from "any process of systematic inference" (Hempel 1966: 15). By this, Hempel means to reject the "narrow inductivist account of scientific inquiry" (Hempel 1966: 11) that might claim the existence of "generally applicable 'rules of induction' by which hypotheses or theories can be mechanically derived or inferred from empirical data" (Hempel 1966: 15). These "guesses" are then strenuously tested by a process of "critical scrutiny" via "careful observation or experiment" (Hempel 1966: 16). This two-step process – free invention of a hypothesis, and then its strict testing - Hempel terms "the method of hypothesis" (Hempel 1966: 17). According to Hempel (and other followers of the Vienna Circle) as the creation of theories is untrammelled and creative, it is the method of their acceptance that bears the onus of ensuring that they are correct. Hempel summarizes:

"…the scientist may give free rein to his imagination…yet scientific objectivity is safe-guarded by the principle that while hypotheses and theories may be freely invented and *proposed* in science, they can be *accepted* into the body of scientific knowledge only if they pass critical scrutiny, which includes in particular the checking of suitable test implications by careful observation or experiment." (16)

Hempel illustrates this process by reference to the process by which Semmelweis discovered the cause of childbed fever (Hempel 1966, chapter 2, see also discussion in Gillies 2005). Here, much more of the chapter is concerned with the manner in which various hypotheses were tested, rather than how they arose.

It is worth emphasizing at this point that the “guesses”, “free invention” or “imagination” required to invent new scientific ideas made their genesis not at all compatible with the methods of logical analysis used by followers of the Vienna circle. This difficulty was avoided by drawing a distinction between discovery, and the from the rest of science, which was amenable to logical analysis. According to Brown (1994: 1), “In this view, discoveries come from chance or intuition; what counts as science is their logic and validity - not their discovery, but their justification.”

This distinction between discovery and justification was first expressed by Hans Reichenbach (1938), but was most forcefully stated by another philosopher associated with philosophical work in Mitteleuropa during the 1930s: Karl Popper. Brown (1994: 1) suggests that Popper was responsible for the way that discovery was “banished… from the kingdom of philosophy to the netherworld of ‘empirical psychology’”

With a turn towards historically-influenced philosophy of science during the early 1960s, several accounts critical of this strict distinction between contexts were written. One such author was the philosopher Norwood Russell Hanson. His complaint was that the dominance of the context of justification, at the cost of the context of discovery, was mistaken. Hanson argued that philosophers working in the positivist tradition that arose from the Vienna Circle unfairly neglected the process of scientific discovery in favor of analyzing the logic of completed scientific work (Hanson 1958a and b; 1960a and b). Of this kind of philosophical work, he wrote that it "reads less like a Logic of Discovery than like a Logic of the Finished Research Report." (Hanson 1958a: 1073). Far from being an inexplicable creative act, fit only for the attentions of psychologists, the study of discovery in the broad sense was an essential

task for philosophy of science, and a necessary complement to work in the context of justification:

> "More philosophers must venture into these unexplored regions in which the logical issues are often hidden by the specialist work of historians, psychologists, and the scientists themselves. We must attend as much to how scientific hypotheses are caught, as to how they are cooked." (Hanson 1958a: 1089).

We can see the emphasis on "cooking" (justification) at the expense of "catching" (discovery) in Hempel's account (discussed above). Discovery, and more specifically the project of accounting for discovery in terms of inductive logic, became a key issue for historically motivated philosophers of science like Kuhn and Hanson. In terms of finding an inductive logic of discovery, capable of algorithmically arriving at novel discoveries from observation, this project was unsuccessful (Downes 1990; Laudan 1980; Lugg 1985; Shah 2007). But in popularizing new approaches to philosophy of science, unlike those wielded by the logical positivists, it was a great success, and set the scene for the flourishing of historically-informed philosophy of science that would occur during from the late 1960s. We can see this flourishing in the many philosophical accounts of discovery that followed these pioneers (Brown 1994; Darden 1976; Maxwell 1974; Nersessian 1984; Siegel 1980; Simon 1973; Thagard 1982; Thagard 2003).

Section two: the discovery of McArdle disease

Let's interrupt this philosophical story to discuss an individual discovery in some detail. This is the discovery of McArdle disease, which occurred in Britain and the USA during the 1950s. In contrast to the short example of discovery given in the introduction, the recurring motif

here is vagueness: of what is discovered, and by whom, and when. We will examine the implications of this vagueness in section three.

McArdle disease is a rare disorder of glycogen metabolism characterized by fatigue and cramps on exertion. Skeletal muscle contains large quantities of glycogen, a polymer of glucose, which provides a reserve of energy to power the muscle during exertion. Two enzymes maintain this energy store. At rest, glycogen synthase adds glucose molecules to glycogen, while a second enzyme called myophosphorylase removes them during exertion. McArdle disease is caused by a functional lack of this myophosphorylase enzyme. Thus individuals with the disorder can form glycogen normally, but are unable to break glycogen back to glucose. This means that their skeletal muscles function normally until called to perform strenuous activity, upon which exertion their muscles rapidly run short of energy, leading to fatigue and pain.

McArdle disease is named after Brian McArdle (1911–2002), whose 1951 paper (McArdle 1951) was the first case-report of this (apparently novel) condition. This case report recounts the extensive clinical investigation of a 30 year old male patient, George W, at Guy's Hospital in London. He had presented to the hospital in September 1947 complaining of lifelong fatigue, stiffness and pain on exertion:

> "For as long as the patient could remember, light exercise of any muscle had always led to pain in the muscle and, if the exercise were continued, to weakness and stiffness. For example walking a few hundred yards, particularly if fast or uphill, provoked pain in the calves and thighs, and lifting heavy weights resulted in pain in the

> arms. Even chewing sometimes gave rise to pain in the masseters. The pain, at first dull and aching, increased with continued exercise, while the muscles became progressively stiffer and weaker. Usually all the symptoms rapidly disappeared on resting, but when he continued the exercise not only did the symptoms increase in severity, but they persisted longer when he was finally forced to rest."
> (McArdle 1951: 13)

Despite an approximately normal physical examination, McArdle suspected that this individual was suffering from a disorder of muscle metabolism. This suspicion was compatible with George W.'s biochemical investigations. First, the level of lactate (a breakdown product of glucose) found in the blood during exercise was significantly less than expected, indicating that the patient was utilizing less glucose than a normal control subject. Secondly, the patient experienced electrically silent muscle cramps during exercise, indicating that his muscles were failing to contract normally.

On the basis of these findings, McArdle made three claims about George W.'s illness. First he claimed that these symptoms were both real – rather than spurious – and arose from the operations of a physical – rather than mental – disease. Second, he claimed that this disease process was novel and not, say, an unusual presentation of a known disease. Third, he claimed that the etiological process responsible was some kind of defect in the glucolytic pathway. More specifically, McArdle suggested that (for reasons detailed in the next paragraph) a deficiency of an enzyme known as glyceraldehyde phosphate dehydrogenase (GPD), which is involved in producing energy from glucose, was the cause of the disease.

While the first two of these claims – that the symptoms were caused by a novel, physical disease – became accepted without serious controversy, McArdle's claim that the disease was caused by GPD deficiency was more problematic. As I noted above, GPD metabolizes glucose, rather than glycogen. This puts McArdle's third claim strangely at odds with the title of McArdle's paper (Myopathy Due to a Defect in Muscle Glycogen Breakdown). It is worth therefore briefly quoting McArdle's argument in favor of this claim. In contrast to the empirical evidence provided to support the other claims, the evidence in favor of GPD deficiency was largely analogical. George W.'s symptoms were noted to be similar to the effects of iodoacetate poisoning. As this agent was known to affect GPD function, McArdle rather tentatively concluded:

> "Theoretically, the phenomena following iodoacetate poisoning of this enzyme [GPD] could also be caused by interference with the other components of the enzyme system. It is suggested therefore that it is the glyceraldehyde phosphate dehydrogenase system that is the site of the biochemical lesion in the muscles of G.W." (McArdle 1951: 32).

It was not until the late 1950s that subsequent cases of McArdle disease were reported. Towards the end of the 1950s, two other groups of researchers reported cases of McArdle disease. One was patient D.G., a 19 year-old man with a life-long reduction in his tolerance for physical exercise, who came to the attention of clinicians at the University of California, Los Angeles (Mommaerts et al. 1959; Pearson et al. 1959; Pearson, Rimer and Mommaerts 1961). The second patient, A.D., was a 54 year-old man investigated at Harvard Medical

School in Boston (Schmid and Mahler 1959a; Schmid and Mahler 1959b; Schmid et al. 1959; Larner and Villar-Palasi 1959; Schmid and Hammaker 1961).

Many of their symptoms were broadly similar to those described by McArdle, although with minor variations in degree and kind. For example, the severity of the condition appeared to increase with age. Understanding this variability was important for the understanding of McArdle disease in general terms, rather than on the basis of isolated, individual, cases. The most important point of difference between this general account and McArdle's account concerns the etiology of the syndrome. Rather than abnormal GPD, it was argued that the characteristic symptoms were due to a lack of myophosphorylase (Mommaerts et al. 1959: 792). This claim was based on the following evidence. First, in skeletal muscle samples from patients, very little phosphorylase activity was detected (Mommaerts et al. 1959: 793-5). Second, normal function was restored if myophosphorylase was added, indicating that this lack of activity was due to an absence of functional myophosphorylase, rather than any sort of regulatory problem (Mommaerts et al. 1959: 793-5). Third, the defect could similarly be bypassed if downstream products of glycogen metabolism were added (Mommaerts et al. 1959: 793-5), indicating that the metabolic problem was specific to the glycogenolysis pathway, rather than the glucolytic pathway. Finally, it was noted that muscle glycogen stores were very much higher than normal (Mommaerts et al. 1959: 793-5), suggesting that the pathology resulted from a primary problem with the way that glycogen was broken down to glucose (catabolism), rather than a problem from the way that glycogen was made (glycogen synthesis).

In conclusion, from a total of nine papers, detailing the investigation of three individuals with McArdle disease, the following was claimed about the condition. First, that it was a real

(rather than spurious), physical (rather than mental), and etiologically distinctive disease with a variety of clinical features. Second, that the etiology responsible was an isolated absence of myophosphorylase, and that the disorder could be characterized in such terms. Third, that various consequences of this abnormality could be identified, including clinical features (fatigue, muscle weakness, myoglobinuria), heritability and possible avenues for treatment. These diverse features could all be explained by reference to the mechanistic consequences of myophosphorylase deficiency [cross reference here to Illari's chapter, please]. Finally, a range of further disease features were identified for further investigation, including the progressive nature of the disease, muscle wasting and so on.

Section three: philosophers of discovery

In this section, I will sketch out two accounts of discovery, and use them to discuss the McArdle case. As these accounts aim their responses against Hempel-like ways of understanding discovery (which we might term the 'creative' view), it will be helpful to distinguish two slightly different positions that have already been sketched out in the introduction and in section 1. These are:

> **Point conception of discovery:** a sudden moment of inspiration in which a new idea is revealed to a researcher

> **Positivist conception of discovery:** discovery is a psychological process, and as such is not amenable to logical analysis

First comes the account of discovery produced by Thomas Kuhn (1962a). Kuhn argues against the point conception of discovery. Far from being sudden moments of individual

inspiration, Kuhn argues that discoveries are typically evolving processes that are necessarily extended in time. Because of this duration, they are therefore subject to historical (and philosophical) analysis.

Hanson (1960a), on the other hand, takes aim primarily at the positivist conception of discovery. Hanson suggests that, although there is no simple inductive logic of discovery, it does not follow that there is no logic capable of accounting for discoveries. In fact, he suggests that a kind of logical inference called retroduction might be a viable candidate for such a logic of discovery.

In both cases, I will illustrate by means of the McArdle case. Finally, I make some concluding remarks linking these two accounts of discovery to the developing philosophy of scientific practices approach. It is worth emphasizing that Kuhn and Hanson's accounts are different and (strictly speaking) incompatible. However, they share the intention of showing that discovery itself, far from being just an act of creative imagination, could be an object of philosophical scrutiny.

Even a glance at the first page of Kuhn's paper shows that he disagrees with the model of point discovery: "To the historian discovery is seldom a unit event attributable to some particular man, time, and place." (Kuhn 1962a: 760). Kuhn begins with a puzzle. Knowing who discovered something is important. Historians (and others, like the Nobel committee) spend a great deal of effort trying to find out about discoveries. Despite this attention, though, often it has not proven possible to pinpoint "the time and place at which a given discovery could properly be said to have 'been made.'" (Kuhn 1962a: 761). Why is this?

Kuhn's response to this puzzle is as follows: this apparent failure of finding out arises because of a category mistake about discovery. Far from being point events, occurring in a particular place at a particular time, most discoveries occur gradually, and involve more than one researcher:

> "…there is no single moment or day which the historian, however complete his data, can identify as the point at which the discovery was made. Often, when several individuals are involved, it is even impossible unequivocally to identify any one of them as the discoverer." (Kuhn 1962a: 763).

The key to understanding Kuhn's point here is the phrase 'most discoveries', because it indicates a classification of kinds of discovery. These are **troublesome** discoveries, and **expected** discoveries. Kuhn elucidates:

> "The troublesome class consists of those discoveries…which could not have been predicted from accepted theory in advance and which therefore caught the assembled profession by surprise." (Kuhn 1962a: 761)

Expected discoveries, on the other hand, are those that are predicted by theory. Kuhn's examples here are rather schematic, but a good example might be the discovery of a new element that fills a blank spot on the periodic table, or the discovery of a planet via some mathematical prediction. The point is that the discoverer in these cases both a) "knew from the start what to look for" (Kuhn 1962a: 761) and b) had "criteria which told them when their

goal had been reached." (Kuhn 1962a: 761). As a result, this kind of discovery does not suffer from the problems that characterize troublesome discoveries. A consequence of some interest to the historian is that resolving questions about, say, the date of an expected discovery, or of the identity of its discoverer, are only limited by the available evidence.

Things are rather different for the troublesome discovery. Kuhn illustrates by giving three examples – the discovery of oxygen, the discovery of Uranus, and the discovery of X-rays. In these cases, neither of Kuhn's two characteristics of expected discovery (a) and b) above) were the case. Instead, even when there is plenty of historical evidence available, the kinds of questions that we like to answer about discoveries cannot be answered in the unequivocal manner that we associate with expected discoveries. As Kuhn suggests, this is largely a matter of recognizing that discovery is usually a more complex process than stories of point discovery would suggest. Discoveries – far from being points, or “Eureka” moments – have a proper internal history. Kuhn suggests that this history of discovery occurs in three phases:

1. Awareness of anomaly (Kuhn 1962a: 762-3)
2. Making the anomaly behave (Kuhn 1962a: 763)
3. Adjustment, adaptation, and assimilation (Kuhn 1962a: 763)

Anomalies are "nature's failure to conform entirely to expectations." (Kuhn 1962a: 762). For a discovery to occur, a scientist must first notice something novel or unexpected. This unexpected finding is an anomaly. Kuhn gives more detail at this point about exactly what is involved in this process. First, a scientist must have available the necessary tools – conceptual or tangible – to make some anomaly occur. Examples might include developing the necessary astronomical theories to be able to recognize that an orbital perturbation is really a

perturbation, or having available photographic film that might become anomalously clouded (see Kuhn's examples). Next, once the anomaly has been produced, the scientist must have the "individual skill, wit, or genius to recognize that something has gone wrong in ways that may prove consequential." (Kuhn 1962a: 763).

In relation to the McArdle case, it was George W.'s clinical features that were the anomalies. Here, though, a detailed historical story about how each clinical finding was noted is important because it illustrates Kuhn's argument about the difference between the recognition, and the production, of anomalies. While George W. had some features that could have been recognized by almost anyone likely to have met him (tiredness, pain on exertion, and so on), most of the telling anomalies required special equipment to produce and to detect. For example, the finding that George W. was suffering electrically silent muscle cramps depended on techniques capable of measuring tiny electrical currents in muscles, a technology intimately related to the development of the ECG during the first decade of the twentieth century (Porter 1997: 582). Similarly, discovering that George W. had abnormal serum levels of lactate after exertion required laboratory techniques capable of accurately measuring the concentration of small molecules in blood. Again, this was a development of the early twentieth century (Porter 1997: 582). Thus this discovery was highly dependent on the available technology.

Once an anomaly has been produced and recognized, the process of discovery moves into a second phase: that of making the anomaly behave. This involves a complicated period of negotiation in order to "make the anomaly behave" in a regular fashion. For example, once a researcher has made and recognized an anomaly, other researchers might seek to replicate it under a range of different conditions, try and understand it using their theories, compare it to

other, similar, phenomena, and so on. While Kuhn's account of the way in which anomalies are made to behave is rather schematic, a similar process can be found in the McArdle case. To recap, McArdle differed from later researchers as to the etiology of the clinical syndrome that he described:

> **McArdle:** the syndrome is caused by abnormal functioning of GPD, largely by analogy with the known effects of iodoacetate poisoning
>
> **(others)**: the syndrome is caused by abnormal functioning of myophosphorylase, largely from *in vitro* investigations of the behavior of muscle enzymes

The final stage of Kuhn's model of discovery is to fit behaving anomalies into existing knowledge. The working of this phase is best illustrated by thinking about the differences between expected and troublesome discoveries. Once expected discoveries have been discovered, they become an addition to the store of scientific knowledge. By contrast, troublesome discoveries may also transform other parts of scientific knowledge in more revolutionary ways. Again, there are resonances here with Kuhn's work on scientific revolutions (Kuhn 1962b):

> "In a sense that I can now develop only in part, they also react back upon what has previously been known, providing a new view of some previously familiar objects and simultaneously changing the way in which even some traditional parts of science are practiced. Those in whose area of special competence the new phenomenon falls often see both the world and their work differently as they emerge from the

> extended struggle with anomaly which constitutes that phenomenon's discovery." (Kuhn 1962a: 763)

That concludes this rather skeletal summary of Kuhn's article. As the above quote suggests, this work on discovery was part of a larger project for Kuhn. Discovery was important for Kuhn just because new discoveries contributed to grand changes in scientific theories – scientific revolutions. As Bîgu summarizes:

> "One of Kuhn's well-known theses is that a distinction can be drawn between periods of normal science and periods of extraordinary science. The first periods are characterized by the existence of a substantial set of shared commitments, on the basis of which the scientific activity is carried out. Still, after a period of research, anomalies, i.e. difficulties met by scientists when trying to solve the problems of normal research, become more numerous and serious. This will lead to a scientific crisis, and, possibly, to a scientific revolution." (Bîgu 2013: 331)

Perhaps it is overstating the case to say that the discovery of McArdle disease led to a revolution. Yet it was in the vanguard of diseases known to be caused by single gene abnormalities. And the consequences of trying to account for disease in genetic terms are not yet clear to us. Perhaps, to medical historians of the far future, the case of McArdle disease will have played some small role in leading to a revolution in the way that genetics might influence health.

If Kuhn's account of discovery is mainly concerned with showing that discoveries are usually not point events, Hanson aims at describing a logical structure of these processes. To do this, he uses a form of inference that he terms "retroductive reasoning". This form of inference is as follows:

1. Some surprising, astonishing phenomena p_1, p_2, p_3... are encountered.
2. But p_1, p_2, p_3... would not be surprising were an hypothesis of H's type to obtain. They would follow as a matter of course from something like H and would be explained by it.
3. Therefore there is good reason for elaborating an hypothesis of type H – for proposing it as a possible hypothesis from whose assumption p_1, p_2, p_3... might be explained. (Hanson 1960a: 104)

Let's illustrate using the McArdle case, before moving on to discuss why this might be an effective rejoinder to the positivist philosophers with whom Hanson was arguing.

First, we need some surprising phenomena. As with the discussion of Kuhn's account of discovery above, I will take these to be the various clinical features belonging to George W. Next, we need some kind of hypothesis that is capable of explaining these clinical features. Happily for us as philosophers (although maybe not as medical practitioners) we have at least two of these H's:

H_1: the syndrome is caused by abnormal functioning of GPD (McArdle)
H_2: the syndrome is caused by abnormal functioning of myophosphorylase (Mommaerts and colleagues)

Having two competing hypotheses is useful to see how Hanson intended this logical framework to operate. As he admits in the final paragraph of his paper, the aim here is to provide a way of discarding hypotheses in a logically definite way: "With such a rich profusion of data and technique as we have, the arguments necessary for eliminating hypotheses of the wrong type become a central research inquiry." (Hanson 1960a: 106). And we can see, by a careful study of the McArdle disease literature, that H_2 does a superior job to H_1 in explaining how our surprising phenomena come about. Thus we can give a logical account of how part of the discovery of McArdle disease came about.

Is this an effective rejoinder to the positivist? Well, on the one hand it does seem to provide a way of understanding how part of the process of discovery might happen in logical terms. On the other hand, this is clearly not the algorithmic kind of discovery that Hempel characterized as the "narrow inductivist view" (Hempel, 1966, 15 TK). Retroductive reasoning also does not offer us a comprehensive logical account of discovery, because we still have to create some hypotheses before we can examine them against our surprising phenomena. Perhaps this just moves the context distinction further forward, such that part (albeit a smaller part) of discovery remains a creative, psychological, non-logical process.

Conclusion

In this chapter, I have tried to sketch out the beginnings of discovery as a topic for philosophers of science. This work has continued, with an increasing number of philosophers treating discovery as an object of philosophical inquiry. With reference to the philosophy of medicine, one important facet of this work has been the various attempts to produce algorithmic methods by which discovery from data could be (somewhat) automated. Herbert Simon, for example, became interested in logical

ways of producing discoveries from patterns in data (Simon 1973). Note, though that Simon does not claim to produce these discoveries in a completely certain way (as proponents of what Hempel called the "narrow inductivist account of scientific inquiry" (Hempel, 1966: 11)). Instead, Simon speaks of a "recommended strategy" that – alone "does not guarantee the achievement of the goal." (Simon, 1973: 474). Simon's work is also intimately connected to Kuhn and Hanson's rejection of the purely psychological view of discovery popularized by Popper and others:

> "…we see that we must reject Popper's assertion that the 'question how it happens that a new idea occurs to a man…may be of great interest to empirical psychology; but it is irrelevant to the logical analysis of scientific knowledge'" (Simon 1973: 474)

Simon's 1973 paper is a modest start to a grand project about digital automation of discovery from data. Moving through various technologies, this work has been the subject of much interest in the philosophical community. Just to pick one example of this work on the interface of philosophy and computer science, Paul Thagard and David Croft note that the retroductive inferential pattern that Hanson described for discovery is also to be found in the way in which technological innovations in computer science occur:

> "Although abductive inference to explanatory hypotheses is much more central to scientific discovery that to technological innovation, inference to possible solutions to technological problems seems to involve very similar representations and processes. We can therefore

> conclude that scientific discovery and technological innovation are cognitively very much alike." (Thagard and Croft 1999: 137)

This consilience between fields is, I think, another good reason to think that there is more to discovery in medicine than a sudden flash of inspiration.

References

Bîgu, D. (2013) "A similarity-based approach of Kuhn's no-overlap principle and anomalies," *Studies in History and Philosophy of Science Part A*. **44**(3): 330-8.

Brown, R.H. (1994) "Logics of Discovery as Narratives of Conversion: Rhetorics of Invention in Ethnography, Philosophy, and Astronomy," *Philosophy & Rhetoric*. **27**: 1–34.

Campbell, W. C. (1979) "History of Trichinosis: Paget, Owen and the Discovery of 'Trichinella Spiralis'," *Bulletin of the History of Medicine*, **53**(4): 520-552.

Darden, L. (1976) "Reasoning in scientific change: Charles Darwin, Hugo de Vries, and the discovery of segregation", Studies in History and Philosophy of Science Part A, Volume 7, Issue 2, 1976, Pages 127-169, ISSN 0039-3681, http://dx.doi.org/10.1016/0039-3681(76)90014-5.

Downes, S. (1990) "Herbert Simon's Computational Models of Scientific Discovery," *Philosophy of Science Association*, **1990** (1): 97-108.

Gillies, D. (2005) "Hempelian and Kuhnian approaches in the philosophy of medicine: the Semmelweis case," Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences. **36**(1): 159-181.

Greenwood, D., Slack, R. and Peutherer, J. eds. (2000) *Medical Microbiology.* 15th edition. London: Churchill Livingstone.

Hanson, N.R. (1958a) "The Logic of Discovery," *The Journal of Philosophy*, **55**: 1073–1089.

---------- (1958b) Patterns of Discovery: an Inquiry into the Conceptual Foundations of Science. Cambridge: Cambridge University Press.

---------- (1960a) "Is there a logic of scientific discovery?" *Australasian Journal of Philosophy*, **38**: 91–106.

---------- (1960b) “More on ‘The Logic of Discovery’,” *The Journal of Philosophy*, **57**: 182–188.

Hempel, C.G. (1966) *Philosophy of natural science*. Englewood Cliffs, NJ : Prentice-Hall.

Hempel, Carl G. and Fetzer, J.H. (ed) (2001). *The Philosophy of Carl G. Hempel: Studies in Science, Explanation, and Rationality*. Oxford University Press

Kuhn, T.S. (1962a) “Historical Structure of Scientific Discovery,” *Science*, **136**(3518): 760-4

---------- (1962b) *The Structure of Scientific Revolutions*. Chicago: University of Chicago Press.

Larner, J. and Villar-Palasi, C. (1959) “Enzymes in a glycogen storage myopathy,” *Proceedings of the National Academy of Sciences of the United States of America*, **45**, pp. 1234-5.

Laudan, L. (1980) “Why was the Logic of Discovery Abandoned?” in Nickles (ed.) *Scientific Discovery, Logic, and Rationality*. Boston: Boston Studies in the Philosophy of Science Volume 56: 173-183

Lugg, A. (1985) “The Process of Discovery,” *Philosophy of Science*, **52** (2): 207-220.

Maxwell, N. (1974) “The Rationality of Scientific Discovery Part I: The Traditional Rationality Problem,” *Philosophy of Science*. **41**: 123–153.

McArdle, B. (1951) “Myopathy Due to a Defect in Muscle Glycogen Breakdown,” *Clinical Science*, **10**(1): 13–33.

Mommaerts, W.F.N.M. et al. (1959) “A functional disorder of muscle associated with the absence of phosphorylase,” *Proceedings of the National Academy of Sciences of the United States of America*, **45:** 791-7.

Nersessian, N.J. 1984. “Aether/or: The creation of scientific concepts,” *Studies in History and Philosophy of Science Part A*, **15**: 175–212

Pearson, C.M., Rimer, D.G. and Mommaerts, W.F.N.M. (1959) "Defect in muscle phosphorylase: a newly defined human disease," *Clinical Research,* **7**, pp. 298.

---------- (1961) "A metabolic myopathy due to absence of muscle phosphorylase," *The American Journal of Medicine,* **30**, pp. 502-17.

Popper, K.R. (1959) *The Logic of Scientific Discovery*. London: Routledge.

---------- (1963) *Conjectures and Refutations*. London: Routledge.

Porter, R. (1997). The Greatest Benefit to Mankind: A Medical History of Humanity from Antiquity to the Present. London: HarperCollins.

Reichenbach, H. (1938) *Experience and Prediction*. Chicago: University of Chicago Press.

Reisch, G.A. (2005) How the Cold War transformed philosophy of science: to the icy slopes of logic. Cambridge: Cambridge University Press.

Schmid, R. and Mahler, R. (1959a) "Syndrome of muscular dystrophy with myoglobinuria: demonstration of a glycogenolytic defect in muscle," *The Journal of Clinical Investigation*. **38:** 1040.

---------- (1959b) "Chronic progressive myopathy with myoglobinuria: demonstration of a glycogenolytic defect in the muscle," *The Journal of Clinical Investigation,* **38:** 2044-58.

Schmid, R., Robbins, P.W. and Traut, R.R. (1959) "Glycogen synthesis in muscle lacking phosphorylase," *Proceedings of the National Academy of Sciences of the United States of America*, **45**: 1236-40.

Schmid, R. and Hammaker, L. (1961) "Hereditary absence of muscle phosphorylase (McArdle's syndrome)," *The New England Journal of Medicine,* **264:** 223-5.

Shah, M. (2007) "Is It Justifiable to Abandon All Search for a Logic of Discovery?" *International Studies in the Philosophy of Science*. **21**: 253–269.

Siegel, H. (1980) "Justification, Discovery and the Naturalizing of Epistemology," *Philosophy of Science*. **47**: 297–321.

Simon, H.A. (1973) "Does Scientific Discovery Have a Logic?" *Philosophy of Science*. **40**: 471–480.

Thagard, P. (1982) "Artificial Intelligence, Psychology, and the Philosophy of Discovery," *PSA: Proceedings of the Biennial Meeting of the Philosophy of Science Association 1982*, 166–175.

---------- (2003) "Pathways to Biomedical Discovery," *Philosophy of Science*. **70**(2): 235-54.

Thagard, P. and Croft, D. (1999) "Scientific Discovery and Technological Innovation," In Magnani, L., Nersessian, N.J. and Thagard, P. (eds.)(1999) *Model-Based Reasoning in Scientific Discovery*. New York: Kluwer pp. 125-137. Need date for this

Whitehead, A.N. and Russell, B. (1910) *Principia Mathematica.* Volume 1. Cambridge: Cambridge University Press

Further reading

For a general introduction to the Vienna Circle and the subsequent development of philosophy of science in the twentieth, see the extremely accessible Gillies, D. (1993) *Philosophy of Science in the Twentieth Century*. Oxford: Blackwell.

For a more detailed historical treatment of the Vienna Circle, their followers, and their subsequent influence on philosophy of science in the later twentieth century see Reisch, G.A. (2005) *How the Cold War transformed philosophy of science: to the icy slopes of logic*. Cambridge: Cambridge University Press.

For a set of neatly drawn case-studies of important biomedical discoveries, see Norrby, E. (2010) *Nobel Prizes and Life Sciences*. World Scientific Publishing: Singapore.

For far more on the role of discovery in the philosophy of Norwood Hanson, see Lund, M. (2010) *N. R. Hanson: Observation, Discovery, and Scientific Change*. Amherst, N.Y.: Humanity Books.

For more examples of the way that discovery has become part of the mainstream of philosophy of science, see the papers in Magnani, L., Nersessian, N.J. and Thagard, P. (eds.) *Model-Based Reasoning in Scientific Discovery*. New York: Kluwer pp. 125-137.

Biographical note

Brendan Clarke is lecturer in history and philosophy of medicine at the Department of Science and Technology Studies, University College London.