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The Journey from Discovery to Scientific Change: Scientific Communities, Shared Models, and Specialised Vocabulary

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ABSTRACT

Scientific communities as social groupings and the role that such communities play in scientific change and the production of scientific knowledge is currently under debate. I examine theory change as a complex social interaction among individual scientists and the scientific community, and argue that individuals will be motivated to adopt a more radical or innovative attitude when confronted with striking similarities between model systems and a more robust understanding of specialised vocabulary. Two case studies from the biological sciences, Barbara McClintock and Stanley Prusiner, help motivate the idea that sharing of models and specialised vocabulary fill the gap between discovery and scientific change by promoting the dispersal of important information throughout the scientific community.

1. Introduction

Scientific communities allow for the communication of scientific ideas and the formulation of scientific knowledge. How, precisely, this is done is a topic currently under debate. In section 2, I review both historical and contemporary notions of scientific community, scientific discipline, scientific fields and other organisational structures of science. This literature highlights the immense importance of social factors within scientific groupings. In section 3, I focus on two important social aspects of scientific communities, namely shared models and specialised vocabulary. I argue that although there may be many important social factors, the sharing of both models and vocabulary is necessary for the communication of ideas within a scientific community.

Section 4 further illuminates the necessity of these important components and emphasises the communication process within communities. I review two individual cases of radical thinking within the biological sciences. If we follow Barbara McClintock and Stanley Prusiner's journeys, a common pattern emerges. Initially, some radical thinkers are not able to communicate their ideas to the scientific community. It is through the sharing of models, the sharing of specialised vocabulary, and a precise fit between the idea and a particular community that an idea can come to be accepted as scientific knowledge. In this way, I argue for a more fluid model of the scientific process, by attempting to better explain the journey between scientific discovery and scientific change.

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2. Scientific Communities, Disciplines, and Fields

Discussions regarding scientific practices and theory choice all, in some way, concern communication. This article focuses on theory change as expressed through the communication of ideas within a scientific community, and the formation of those ideas into scientific knowledge. To truly understand questions regarding science, scientific methodology and scientific practice, philosophers of science must better understand the goings-on in scientific communities, how ideas are communicated within a community, when choices are made and how they are then taken up within the community, and when ideas become accepted as scientific knowledge. These are basic methodological questions that concern scientific communication within communities.

2.1. Scientific Community: Historical

Historically, the nineteenth century brought about a steady growth of scientific organisation and specialisation via an increased number of scientific institutions, societies and journals (Vickery 2000, 121). However, Struan Jacobs (2002, 157) argues that it is unlikely that the terms 'scientific' and 'community' were coupled until well into the twentieth century, when science began to be viewed as having a social component.[1] As such, scientific communities became social objects. The term 'scientific community' has been enthusiastically adopted by philosophers, historians, scientists, sociologists and the like, who have examined, in detail, specific scientific communities and scientific communication is various ways throughout the better part of the twentieth century.[2]

Brian Vickery points out that scientific progress has been marked by the formation of new technical terms. New scientific terms are needed as meaning within scientific circles must be 'steadily fixed and rigorously limited' (Vickery 2000, 99). Historically, scientists quickly began to understand that commonly used words, though easier to explain and readily understood, were often misused outside of the specialised field they were originated (Vickery 2000, 100). Thus, scientists began to move toward specialised terminology and vocabulary. For example, eighteenth-century scientists often utilised once common words in Latin or Greek with slight modification in meaning, such as 'thorax', 'artery', 'embryo', 'cardiac', etc. (Vickery 2000, 101).

In their historic piece, Derek de Solla Price and Donald Beaver attempted to define and study the form of academic organisation known as the 'Invisible College'. They focused on the idea that each specialty in the sciences seemed to come equipped with a core group of productive and committed 'in group' (Price and Beaver 1966, 1011). By analysing this mode of scientific organisation, Price and Beaver found that the core group could directly communicate with one another through several important avenues, most notably select conferences and journals. Within Price and Beaver's study, the core group of individuals show high rates of collaboration with one another, thus aiding in the communication and formulation of ideas within this unique social structure (Price and Beaver 1966, 1016–1017).

Others argue that the scientific community is simply global, in that scientists or experts worldwide make up the scientific community. Sally Gregory Kohlstedt (1976) argued for the formation of the scientific community via major scientific association formations. That is, since nearly every scientist is affiliated with at least one major scientific association,

such as the American Association for the Advancement of Science, these in combination form a global community of scientists.

Lindley Darden and Nancy Maull delineate a smaller, more specialised level of scientific organisation, namely the scientific field.[3] Fields like genetics and biochemistry are main scientific units and are related by the theories they share. For example, genetics and cytology are related via the chromosomal theory of Mendelian heredity, whereas the operon theory relates the fields of genetics and biochemistry (Darden and Maull 1977, 43). Fields, according to Darden and Maull, make up larger areas of knowledge, such as biology. As a result, a field is simply a group of scientists that share the same problem, the same goal, the same facts, the same techniques and methods, may have the same laws and theories and could share a special vocabulary.[4]

2.2. Scientific Community: The Importance of Social Features

Most philosophers, sociologists, historians, etc., to some extent realise the importance of social features regarding scientific communities. For example, a discipline, according to William Bechtel, is defined by its object of study (domains, phenomena and model systems), cognitive features and tools (problems, theories and techniques), or by social structure (turf, professional organisation, genealogies of training and journals). Bechtel (1993, 290) adds that smaller groups, groups that make up disciplines and fields, tend to form very specific models.[5]

A select group of academics has made social features of primary focus both within and during the creation of scientific communities. That is, in order to understand the delineation of scientific communities, one must first understand the social structure of the global scientific community, and through this method we will uncover the way in which to delineate more localised communities of scientists.

One notable philosopher interested in these sorts of questions was Thomas S. Kuhn. For Kuhn, paradigm shifts occur when one phase of normal science is abandoned. During this time of crisis, many ideas are floated and a novel idea emerges. Kuhn writes:

> Copernicus saw as counterinstances what most of Ptolemy's other successors had seen as puzzles in the match between observation and theory. Lavoisier saw as a counterinstance what Priestley has seen as a successfully solved puzzle in the articulation of the phlogiston theory. And Einstein saw as counterinstances what Lorentz, Fitzgerald, and others had seen as puzzles in the articulation of Newton's and Maxwell's theories. (Kuhn 1962, 79–80)

As more scientists begin working on the novel idea, a new normal science forms and is eventually taken up by a large majority of the community.

On Kuhn's view, fields of study become disciplines when a range of previously unrelated phenomena are unified under a set of principles that are verified under different methods. At the top of Kuhn's hierarchy of scientific organisation resides the global scientific community. One level below is a professional grouping, comprised of physicists, geologists, chemists, etc. Below that are important specialty groupings or subgroupings, such as solid-state physicists, organic chemists, etc. Finally, microgroups often numbering between a few to a hundred members worldwide (Jacobs 2006, 164). For Kuhn, scientists may belong to more than one micro-community (lowest, most exclusive community), but could not belong to more than one professional grouping or subgrouping.

Since Kuhn, many philosophers have attempted to further explain this picture of science. For example, while attempting to better understand Kuhn's notion of scientific community, K. Brad Wray (2011) argues that it is the research community that is the locus of scientific change. By focusing on older scientists who create new theories and are less resistant to theory change, Wray argues that values and social structures play a large role in theory change. Utilising a Kuhnian framework, Fred D'Agostino (2010, 127–134) models hierarchies within scientific communities and specifically aims to better understand communication patterns.

Wray and D'Agostino are not alone in attempting to fill out Kuhn's notions, indeed many academics have furthered the pursuit by focusing on the importance of social features both within and during the creation of scientific communities and other scientific groupings.[6] For example, Warren Hagstrom (1965), while studying the difference in communication behaviours among scientists working in the same field, identified a typology of scientific researchers as communicators. He argues that the influence of scientific colleagues on the conduct of one another's research is of primary focus when understanding the social or organisational stratification of the scientific community. Hagstrom concludes that it is the group of colleagues, or those engaged in one another's research, either physically or intellectually, that make up the important grouping for a scientific community because it is this dynamic or social influence that produces the kind of conformity to scientific norms and values that we have come to associate with the term 'scientific community'.

John Ziman (2000) is after highly specialised international communities of experts that form spontaneously to generate scientific knowledge. Ziman, sceptical of philosophical attempts to define science in traditional ways, strives to place science in a context of social and epistemic practices, a context in which scientific ideas emerge and are sustained. As such, he identifies numerous key components to the scientific pursuit, including agreed upon methods of quantification, methods of experimentation, acceptable technologies, modes of verification, etc. For Ziman, every scientific discipline has its own criteria for what counts as science (Ziman 2000, 85).

Steve Fuller (2000, 99) contends that scientific knowledge cannot be independent from its social context. For Fuller, a discipline is bounded by its specific procedure for understanding knowledge claims. So, rules that govern and restrict word usage, what can be borrowed from other disciplines and appropriate contexts of justification/discovery are required for the formation of a discipline (Fuller 2002, 191). In fact, one way of understanding interdisciplinary work is to look for a metalanguage that is constructed so that an interdisciplinary community can share ideas and insights (Fuller 2002, 194). What science needs is not categorisation and specialisation, but a better understanding of scientific organisation and communication.

3. Scientific Communities, Shared Models, and Specialised Vocabulary

The notion of scientific community I am after within this article is one of a communicative grouping in which a scientific community's aim is theory change as expressed through the communication of ideas and the formation of those ideas into scientific knowledge. Drawing from the literature discussed above, two features required for the communication of ideas within a scientific community become apparent, namely the importance of shared model systems among scientists (Bechtel 1993; Bechtel and Richardson 1993; Fuller 2002)

and sharing specialised vocabularies (Darden and Maull 1977; Vickery 2000; Darden 2006; Wray 2011).

Like Bechtel's disciplines, which focus on a common goal among its constituents, or Darden and Maull's fields, which are picked out by a common problem, scientific communities share a common phenomenon of interest and thus create relevantly similar representations or models of that shared phenomenon. Furthermore, the communication of ideas within a scientific community requires a specialised vocabulary that is utilised by individual scientists and shared by the community of scientists. One way this kind of language becomes adopted both by the individual and the community is through the use of shared models. Indeed, it is my claim that a scientific community is created along with the creating of a shared model system and specialised vocabulary. The very act of sharing these defining features creates the community. These two necessary but not sufficient features of scientific communication will be our focus.[7]

3.1. The Sharing of Models

Scientific communities share model systems. For example, some philosophers of science have recently argued that explanations within the biological sciences take the form of descriptions of the mechanism responsible for a given phenomenon (Craver and Darden 2013). Instead of always appealing to laws, biologists will sometimes appeal to a mechanism to explain a phenomenon of interest. The explanation consists of representing the mechanism and showing how it realises or produces the phenomenon (Illari and Williamson 2012). That is, to explain why, scientists explain how. They provide a model of the mechanism underlying the phenomenon in question. Scientists are able to provide a successful explanation by identifying and manipulating variables in a model of a mechanism thereby determining how those variables are situated and make a difference in the mechanism (Darden 2007). In other words, the explanation consists of explaining how those variables act and interact to produce the phenomenon of interest.[8] So, models of mechanisms explain how a phenomenon is produced, how some task is carried out and how the mechanism behaves.

Of course, models within the biological sciences need not be mechanistic. Model building is incredibly diverse (Lynch and Woolgar 1988). Mathematical models (Lynch 1988; Kaplan and Craver 2011), linguistic descriptions, computer simulations, causal modelling (Hitchcock 2009) and the like are abundant. Physical models, like those used by James D. Watson and Francis Crick to model DNA have a long history within science. Visual displays, textbook illustrations and sketches among scientists are all used to produce explanations of phenomena (Latour 1988; Lynch 1988; Sheredos et al. 2013; Abrahamsen and Bechtel 2015).

More generally, models are idealised structures that we use to represent the world via resemblance relations between the model and real-world target systems (Giere 1988). So, the scientist uses the model to gain a better understanding of a complex system by understanding a simpler, hypothetical system that resembles the real-world system in relevant respects (Glennan 2005; Craver 2006; Gross and Harmon 2014).

Take, for example, protein folding. There are many kinds of scientists attempting to understand the mechanisms behind protein folding. Some scientists focus on different proteins and how they fold in certain environments. Others are interested in the way in

which two proteins might interact and cause each other to fold differently. Still others are interested in how a single protein folds in a similar pattern regularly, and so on. So, scientists are interested in different aspects of the shared model.

While there are many ways to study proteins, the groups that do so share the same set of models. For instance, most scientists interested in protein folding acknowledge the thermodynamic hypothesis. This hypothesis states that a protein's native structure is determined by its amino acid sequence in a particular environment (Anfinsen 1973). Important environmental conditions include solvent concentration and composition, temperature and the like. These environmental factors dictate how the protein folds, in that the protein will most likely fold in a way that requires the least energy.[9] Moreover, what kinds of things count as proteins, how proteins act in different environments, how proteins are produced, how proteins interact with one another, how proteins denature, how proteins act to temperature change, etc. are all built into the shared model systems. Although scientists have different interests regarding protein folding, the entire community shares a model system that is dictated by common constraints. Each model depicting a different aspect of protein folding will entail these and other agreed upon constraints.[10]

Similarity between models, either based on the modelling of the same or similar phenomenon or the use of similar parameters when modelling, is required for membership within a scientific community. Like members of fields and disciplines, member of scientific communities can, and usually are, members of multiple communities. For instance, a scientist interested in protein folding can be in multiple communities based on her modelling practices and interests, such as the chemical bonding between amino acids and the 'spontaneous' folding of prion, or malfunctioning, proteins.[11] In this way, a scientist in one of these communities may indeed be part of the other community if her other projects, interests and models are similar. So, if a scientist is interested in the effects a certain plant has on cognition, given its interaction with chemical receptors in the brain, she would rightfully be part of both communities. Although different communities are hard pressed to communicate to other communities, individuals are capable of being members of multiple communities.[12]

3.2. The Sharing of Specialised Vocabulary

Scientific communities are built around shared models that allow for the communication of novel ideas between the discoverer and her community. One benefit of sharing models or creating models that are very similar is the sharing of specialised vocabulary. Distinct scientific communities either use a different specialised vocabulary or use vocabulary in a different way. For instance, scientists studying the role that chemical receptors play in cognition will utilise very different models and specialised vocabulary than those studying the medicinal effectiveness of a certain new plant species.

This shared vocabulary can be a great source of flexibility and creativity for the community. For instance, a biological model may include imaginary biological populations or hypothesise a molecule not yet discovered. These imaginary objects are a lot like imaginary objects of literary fiction. For example, a book club discussing a Sherlock Holmes book is able to communicate to one another and discuss the many literary figures in the book. Peter Godfrey-Smith argues that 'these are imaginary things that we can, somehow,

talk about in fairly constrained and often communal way' (Godfrey-Smith 2007, 735). So, a community can even communicate hypothetical biological entities within an already provided framework. In much the same way, scientists utilising the same model can talk about the model and understand one another regarding certain aspects of the biological world because of the shared model (Gross and Harmon 2014). This new shared vocabulary would not be possible with those that have not read the book or do not share the model. This shared vocabulary seems integral and enables discussion, communication and the discovery of novel phenomena within a scientific community by providing a common ground or framework.

History reveals the importance of specialised vocabulary and utilisation of shared models. Take for instance, the development of the periodic table of the elements.[13] This shared model invoked very specific terminology, such as 'electron', 'proton', 'molecular weight', 'electron shell', etc., and provided an understanding of how these terms interact and explain chemical interactions. Chemists, armed with shared terminology, could communicate about elements not yet discovered. The shared model provided the vocabulary and flexibility needed to both hypothesise the existence of unknown elements as well as discuss elements prior to discovery (Scerri 2006).

3.3. The Sharing of Models and Specialised Vocabulary

The term 'scientific community' is often used to indicate that those within the community have something in common, they share something that is important to the group dynamic. Shared models offer both explicit and implicit communicative practices that give character and meaning to a given community. Shared models permit error, novel vocabulary and encourage learning via one another. Shared models are communicative practices of a people within a given community. One important trademark of a community is a practice that needs to be learned for outsiders to make sense of the work being done in a community (Arnett and Holba 2012, 212). Model sharing has this very trademark.

The sharing of models also implies that these scientists, ones that are within a scientific community, are interested in or pick out certain common values. Judging from the work of Andrea Woody (2004), it seems that model systems shared by a community not only allow for communication through the sharing of vocabulary, but also represent the community's particular epistemic aims and values.[14]

One well-known example of this can be found during the discovery of the DNA double helix. While Watson and Crick were utilising a physical model, Rosalind Franklin was utilising X-ray crystallography to model the physical form of DNA. Although they were utilising different models, the models were similar, in that they both targeted the same phenomenon. Moreover, they were both constructed with similar constraints, namely the physical spatial relations that base pairs had to one another, Chargaff's rules, the hydrophobic and hydrophilic nature of certain areas of the DNA structure, etc.[15]

It is well documented that the two research groups were indeed able to discuss their similar findings by utilising a common understanding and vocabulary. For instance, Watson and Crick attended a presentation by Franklin, where she explained the hydrophobic nature of different parts of the DNA molecule. As a result, Watson and Crick corrected their erroneous model based on the findings and explanation of Franklin's relevantly similar model (Watson 2012).[16] Individuals within a scientific community

utilise models that share similar attributes and rely on specialised vocabulary to better understand a shared phenomenon of interest.

3.4. The Journey from Discovery to Scientific Change

By focusing on shared models and specialised vocabulary, we may now be in a better place to understand rough boundaries between scientific communities. Scientific communities are united via the sharing of similar models and vocabulary. Similar, here, means that the models must at least aim at representing the same phenomenon or cluster of phenomena, must have the shared goal of explaining that phenomenon or cluster of phenomena, and the way in which they represent or model that phenomenon or cluster of phenomena is directed by shared constraints. These models then allow for the cultivation of a unique or specialised vocabulary, thus enabling discussion and allowing for communication within the community.

This similarity of modelling and adopting specialised vocabulary is perhaps best explained by understanding how these features breed trust within a scientific community. Trust is an important social factor within science, as a scientific community requires trust in one another regarding ideas, findings, methodology, etc. (Shapin 1994; Ziman 2000, 96–98). Believing in fellow experts and using/trusting their ideas is paramount to forming scientific knowledge (Hardwig 1985, 1991; Kusch 2002). As such, scientists form teams based on testimony and trust, or, as Hardwig argues, scientists are 'epistemically dependent' on one another with in this grouping (Hardwig 1985, 347; 1991, 695–696).[17]

Science is done in a community, and scientific knowledge and the communication of that knowledge to members is likewise done in a community of individuals that share many similar aims, goals, models and commitments (Welbourne 1993; Kusch 2002). By having a common goal, a common phenomenon, common vocabulary and a common model, scientists begin to form trust in one another's work.[18] Thus, trust within a scientific community is aided by the sharing of similar models and specialised vocabulary, which in turn delineates how similar models need to be in order to aid in the community's pursuit of knowledge.

Similarly, trust plays a role in the shifting of a scientific community. While the first reaction of scientists within a community is one of 'categorical conservatism', or preference for entrenched categories (Goodman 1955), eventually, as data mount, it is natural and rational for scientists within a community to revise tightly held ideas, hypotheses, models, terms, etc. That is, an important part of scientific practice is the ability for scientists to depart from mainstream or dominate epistemic judgements when it proves advisable (Goldman 1993).

Philip Kitcher (1990, 7) maintains that cognitive diversity within scientific communities is beneficial, in that multiple theories are being progressed and knowledge is being cultivated even if it is believed that one theory, method or model is clearly more meritorious. By differentiating between belief in a theory and pursuit of a theory, it is rational for each scientist to believe the theory that is better supported by the available evidence and still hold that it may not be rational for each scientist to pursue that theory (Laudan 1977, 108–114; Kitcher 1990, 8). Something similar can be found in Kuhn (1977), when he concerns himself with conservative and innovative approaches within a paradigm. Kuhn ultimately insisted that both new ideas and those furthering ideas within the paradigm were necessary for each community.[19]

What follows is an attempt to understand the balancing of these two attitudes. Scientific communities are epistemic communities based on communal trust and clustered around shared models and specialised vocabulary, which allows for the communication of ideas to the community. That is, individuals will be motivated to adopt an innovative attitude when confronted with striking similarities between model systems and a more robust understanding of specialised vocabulary from trusted experts within the community.[20] In this way, the sharing of models and specialised vocabulary fill the gap between discovery and scientific change by promoting the dispersal of important information throughout the scientific community.

It is important to note that I do not mean to argue that communities are always fully functioning. When scientific communities are functioning properly, communication is aided by shared model systems and a shared specialised vocabulary. However, communication breakdowns happen when either or both components are missing, as we will see in the next section.[21]

Similarly, I do not mean to argue that members of a community always fully accept the model, its parameters, background hypotheses, joint vocabulary, etc. Instead, as our subsequent case studies will show, a scientific community can allow for a continuum of acceptance. For example, D'Agostino (2010, 91) argues that a scientific community can accommodate what he calls 'shallow consensus' or the idea that while agreement among scientists within a community can be ephemeral or superficial, it can nonetheless be productive.[22] That is, the acceptance of shared models and specialised vocabulary need not be wholly complete for these defining features to aid in the communication of knowledge.

However, a lack of defining features, such as a shared model system and specialised vocabulary, will result in a breakdown of communication and greatly hinder communication of scientific ideas within the community. Although communication and consensus within a scientific community is flexible, both shared model systems and specialised vocabulary are required for the formation of a scientific community and thus the communication of scientific ideas within the community. Although shared models and specialised vocabulary are merely two of many factors affecting the communication of scientific knowledge and the creation of a scientific community, they are necessary factors.

In what follows, I focus on two radical thinkers from the biological sciences, to add to contemporary notions of scientific community and scientific change. I argue that two shifts must occur within a community for the communication of a radical idea to occur. First, the community must become less attached to the currently held model, and second, a select group within the community must link their research to the radical idea using shared models and shared vocabulary. When this happens, scientific research can then be communicated to a scientific community.[23]

4. Radical Thinkers

4.1. Barbara McClintock

Barbara McClintock was working during a time in biology when it was widely accepted that 'once information passed into a protein it couldn't get out' (Crick 1957). So, the mantra 'DNA to RNA to protein' became fixed. In 1948, McClintock publicly introduced

her model system regarding genetic control and introduced the concept of transposition (Keller 1983, 133; Comfort 2001, 9). McClintock argued that there are controlling elements within genes that allow for genomic rearrangement and, in some cases, the ability of genes to react to the environment. Perhaps most surprisingly, McClintock argued that genes can 'jump' to different locations throughout the genome.

McClintock began, through her observations and research, building a model of genetic mobility and genetic control in maize plants. By noticing and documenting genetic patterns and chromosomal behaviour, McClintock hoped to both explain and predict certain patterns within maize. McClintock's goal was to model genetic control, thus explaining chromosomal behaviour and genetic mobility.[24]

McClintock's model-building process is quite interesting. McClintock began by searching for controlling genetic factors. Soon, she located what she called dissociator (*Ds*) and activator (*Ac*) genes. To construct her model, McClintock ran empirical tests aimed at locating these two loci, *Ds* and *Ac*, and quickly noted that the loci seem to be moving around throughout the genome.

> She discovered a house in which the lights flickered in a complex but recognizable pattern. Rather than ignoring this variability … she imagined that the lights had switches. Mutable or mosaic genes, in which the light alternated on-again, off-again, would then have nothing to do with the bulb; rather the switch had gone haywire … The genetic factor or element controlling mutation rates in the twin sectors seemed to be such a switch. Between 1946 and 1948, McClintock's primary goals were to identify and isolate this element. By the spring of 1948, she had accomplished this, but was surprised by two wrinkles: there were two elements, and they moved. She soon saw that they provided her with an explanation of mutable genes. And mutable genes were simply ordinary genes to which something funny had happened. They became her key to the genetics of development. (Comfort 2001, 98)

Moreover, McClintock uncovered a common pattern that she then based her model upon:

> By April 1948, she had concluded that both *Ac* and *Ds* could be found in two states, high and low. The high state of an *Ac* locus resembled the dosage effect: one high-state *Ac* was like several low-state *Ac*-loci. Both produced kernels with few small speckles. … She soon saw parallels between the states of *Ac* and *Ds* and the qualitative and quantitative genes. The states of *Ac* and *Ds* could be caused by a common mechanism: exchange of subunits that influence the degree of expression. (Comfort 2001, 131)

By understanding the effects of *Ac* and *Ds* as additive, in the same way as the effects of some genes are additive, McClintock constructed experiments meant to test this possibility and utilised this analogy to help build her model. McClintock spent a great deal of time attempting to understand how this additive relation results in different kernel patterns. She wrote,

> I am viewing the mechanism of building up the units of a block to be similar to the mechanism of building-up of blocks in changes of the Ac locus. With this mechanism, one builds up blocks and reduces them in the same mitosis—one chromatid gains what another loses. (McClintock 1948)

In other words, McClintock observed that *Ac* and *Ds* control gene exchange among chromosomes. One gene or group of genes can jump to another chromosome.

Throughout her experimentation, McClintock was in the process of constructing a model, both mathematical and diagrammatic, to explain how the transposition of genes

results in differing patterns on maize kernels (Comfort 2001, 132). McClintock concluded that both *Ac* and *Ds* are able to transpose. Thus, the explanation for *Ac* and *Ds*, as controlling genetic factors, can account for the exchange of chromosome parts between chromosomes (Comfort 2001, 114).

In this way, McClintock's models were at odds with the popular view of genetics at the time, where genetic variation was supposed to be random rather than under the control of the organism. Commonly referred to as the change-in-the-gene model, it was held by the community that genes were autonomous and that any mutation that happened was a result of chemical changes, not structural changes. McClintock's integrative genome, where genes acted in clusters and were controlled by regulatory elements (*Ac* and *Ds*), challenged this model directly. Not only do genes work together, according to McClintock's models, there is an internal mechanism that controls which genes are expressed and which are not. Moreover, there is also internal control regarding the mobility of parts of genes and exchange of these parts.[25] McClintock's model system was in direct dissent with the common model shared among the community at that time.

McClintock's community did not lack a response. McClintock was described as obscure, incomprehensible, mystical and mad, while others within the community were simply dismissive (Comfort 2001, 162–169). McClintock concluded that 'transposition was absolutely nonsensical to biologists then' (Keller 1983, 136).

McClintock was unable to properly communicate her findings to her community for several reasons. Perhaps most notably, McClintock's style of research and resulting models were very different from those within her community. For many years McClintock spent hours observing corn plants and maize genetics, resulting in an intimate knowledge of the organism that others simply did not have (Rhodes 1992, 45–69). Scientists at the time knew very little about maize genetics and were thus unable to follow McClintock's intricate arguments and detailed analysis of her models that were necessary to promote her radical claims (Keller 1983, 9, 50; Comfort 2001, 166). As a result, McClintock's projects, interests and resulting models were very different from those being utilised from her community.

Secondly, McClintock utilised new terms and often adopted and extended common terminology (Comfort 2001, 130). For instance, 'dissociator genes', 'transposons' and 'activator genes' were newly coined terms, whereas other terms were adopted from the community but used differently. McClintock used 'quantity' and 'quality' not only as properties of genes, as was common in the community, but also to refer to properties of alleles, an uncommon practice within the community (Comfort 2001, 130). The vocabulary that scientists use is interpreted via the objects in the models that scientists use and the relations in which these objects stand to each other. Thus, different models yield different interpretations of bits of otherwise uninterpreted language. In other words, different models yield different specialised vocabulary that is very difficult to convey to those not utilising similar models.

Among other differences, McClintock constructed a new model system, introduced new terminology and utilised the community's terms differently (McClintock 1956, 58–74).[26] As a result, McClintock found it very hard to communicate ideas to other scientists within her community.

Fortunately, new findings began to undermine molecular biology's simplistic ideas. McClintock's community became increasingly dissatisfied with the accepted dogma that

genes are just 'beads on a string'. Gradually, the community became focused on genetic mutations and how the chromosome can react to an ever-changing environment. Finally, by the mid- to late 1970s, molecular biology began to look more complex (Comfort 2001, 258–260). New interest and findings in *Drosophila* and *E. coli* soon appeared that seemed similar to McClintock's. Spontaneously occurring mutations in *E. coli* as well as *Salmonella typhimurium* bacteria research showed instances of genetic mobility, of the same type that McClintock modelled years earlier. Also, bacterial research uncovered transduction, thus opening the possibility for transposons (Comfort 2001, 202–208). As scientists began to draw the parallels between their work and McClintock's work in print, jumping genes were found in *Drosophila*, thus supporting McClintock's research in maize (Keller 1983, 187; Green 1992, 117–122; Comfort 2001, 181). In this way, McClintock's controlling elements in the gene were backed by research being done by the community in other organisms.

Scientists were utilising, not only McClintock's model system, but also the novel vocabulary it carried with it (Comfort 2001, 203–208). For example, Jacques Monod and François Jacob wrote:

> Long before regulator genes and operators were recognized in bacteria, the extensive and penetrating work of McClintock (1956) had revealed the existence, in maize, of two classes of 'genetic controlling elements' … the parallel is so striking that it may justify the conclusion that the rate of structural gene expression is controlled in higher organisms as well as in bacteria and bacterial viruses, by closely similar mechanisms, involving regulator genes, aporepressors, operators, and operons. (Monod and Jacob 1961, 394–395)

Research within the community was pointing to the controlling elements of the gene that McClintock had been trying to push along with transposons, and in this way her work increased the scope of the central dogma; it added a crucial form of feedback to the unidirectional command that went from RNA to DNA to protein (McClintock 1961). The community began constructing models like those used by McClintock, and, as a result the community began utilising the terminology introduced through her models (Goldschmidt 1951).

The community accepted McClintock's research only after thirty years of miscommunication and misunderstanding (Fedoroff and Botstein 1992). It was not until the community shifted, or begin allowing for alternative ideas, that McClintock's ideas became of interest. Notice, the molecular biology community was slowly moving towards a more complex picture of the gene, its mutability and its interaction with the environment. But, this only occurred after a small subset of the community became interested in other organisms that behave similarly, in the relevant ways, to McClintock's organism (Green 1992, 117–122). In other words, some scientists began to construct similar models and incorporate McClintock's new vocabulary. As a result, the community, or at least part of it, was in a better situation to understand McClintock's research and results and to link her research to theirs publicly.

Often, individual scientists have a relatively hard time communicating radical ideas to their respective scientific communities. This was evident within McClintock's journey. Moreover, it is not until the community shifts and allows for other scientists to link their findings to the original radical idea is an individual able to communicate a radical idea to a scientific community. A lot of this work is done by the sharing of similar

models and specialised vocabulary. I hope I have begun to sketch this pattern in the case of Barbara McClintock. If my hypothesis is correct, then we should expect similar cases within biology. Drawing from a more contemporary and far less settled case, I propose that something similar may happen regarding prion research.

4.2. Stanley Prusiner

In 1982, biochemist and neurologist Stanley Prusiner proposed a hypothesis concerning infectious proteins. He identified them as abnormal prions, proteinaceous infectious particles, capable of converting normal prions, or naturally present proteins in mammals, into an abnormal form causing a fatal disease of the central nervous system in both animals and humans. The unique sequences of amino acids in a prion make it possible for these molecules to have two different, stable tertiary structures. One type, called a cellular ('healthy') prion protein (PrPc), has functional structure folds with many α-helices. The abnormal prion protein (PrPSc) has many β-plated sheets. They are the same protein but just folded differently. Prusiner's team of research scientists suggest that PrPSc converts α-helices into β-plated sheets (Pan et al. 1993) and thus transmits the disease (Prusiner 1998).

Some scientists have come to accept Prusiner's claims. Recent literature shows that medical research utilising Prusiner's hypothesis is currently underway (Prusiner 1999; Collinge et al. 2009). For example, prion diseases are now thought to include diseases such as Creutzfeldt–Jakob disease (CJD), variant Creutzfeldt–Jakob disease (vCJD), Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia and kuru.[27] Scientists modelling these diseases utilise strikingly similar mechanisms to explain disease propagation and disease contraction. Scientists studying infectious proteins have found that these diseases propagate via a pathway that allows a single (or small number of) diseased protein (PrSc) to chemically alter nearby healthy proteins. In this way, β-sheet plaques form, thus causing the loss of memory and neuronal functioning (Prusiner 1998, 1999).

Similarly, scientists are beginning to draw parallels between traditional prion disease and thyroid diseases due to similar receptor protein conformations (Tatzelt, Prusiner, and Welch 1996; Welch and Brown 1996). That is to say, the scientists studying various prion diseases and the infectious protein hypothesis in general, utilise strikingly similar models to better understand both disease pathway and possible active conformations of the protein (Prusiner 1999).

Moreover, these models often utilised the same specialised vocabulary. For instance, terms like 'prion', 'plaques', 'amyloid', 'miniprion' and 'unconventional virus' are used throughout the literature to denote commonalities among the disease models and mechanism representations. For example, 'plaques' are consistently understood across models as the clustering of abnormal (high levels of β-plated sheets) proteins in a location of the brain, or the chain of 106 amino acids known formally as PrP106 is known as a 'miniprion' (James et al. 1997; Prusiner 1999).

The utilisation of shared/similar models and specialised vocabulary has begun a new line of inquiry regarding the possible similarity of prion diseases to other human-based diseases, such as Alzheimer's disease and Parkinson's disease (Prusiner 2001). Although not contagious, both Alzheimer's and Parkinson's diseases are being modelled by scientists and are represented in similar ways, often utilising this specialised vocabulary. As a result,

some scientists are beginning to believe that human-centred diseases like Parkinson's and Alzheimer's disease 'propagate through the nervous system in much the same way' as prion diseases (Miller 2009, 1337). Researchers are hopeful that the prion concept can integrate much of what's known about neurodegenerative diseases (Prusiner 2014).

Despite this budding research and the general success of the infectious protein hypothesis, Prusiner still finds it difficult to communicate his ideas to other scientists (Prusiner 2014).[28] Prusiner admits he struggles to explain the prion model, in part due to introducing new and radically different ideas as well as new terms and vocabulary.

Indeed, some within his community remain sceptical (Prusiner 2014). Lacking any nucleic acid, prions violate a tightly held 'universal' biological rule, namely the idea that only nucleic acids can duplicate themselves. In other words, the infectious prion hypothesis violates a tightly held belief of the community. Others argue that there are concerns regarding Prusiner's hypothesis as well as discrepancies in linking his work to scientific fact (Zaitsev 2009). Laura Manuelidis (2007) suggests that prions may simply be part of the late stage of a disease, not part of the cause. Still others maintain that the composition of the prion particle is unresolved (Aguzzi and Weissmann 1997; Chesebro 1998).[29] The abundance of scientific data and arguments among an impressive segment of scientists, contradicting and challenging Prusiner's hypothesis, warrants continued reconsideration within the community.

While the fate of the infectious protein hypothesis is still undetermined, it may provide an interesting challenge to the schema offered above. Prusiner's community may be shifting, in that it seems as though the community is willing to utilise Prusiner's findings, especially after some scientists in the community have found that Prusiner's work may be applicable to human diseases. If my schema is correct, then the shared models used by those studying human-based prion diseases and diseases having possible relevant similarities to prion diseases will begin to aid in the communicating of Prusiner's ideas to his community by allowing for a sharing of important ideas and vocabulary. The combination of the changing community and the utilisation of the prion model system by other researchers may yet result in acceptance of Prusiner's hypotheses.

When taken together, these two biological cases illuminate much.[30] First, it is often the case that a radical thinker is hard pressed to communicate new findings precisely because the radical thinker is utilising vastly different model systems than others within their intended community. As such, the radical thinker develops a new specialised vocabulary, in that the individual often develops new terms, new ways to use old terms and new ways to explain a certain phenomenon. A radical thinker often undergoes several attempts at communicating his findings to an intended community and fails. It is only after the intended community begins seeking new model systems, and develops a renewed interest in a radical thinker's model does the communication of ideas begin within a scientific community.

5. Conclusion

Within the present article, I examine theory change as the complex social interaction among individual scientists and their scientific community regarding the communication of scientific ideas. By following the journey from discovery to communicating that discovery, it becomes apparent that communicating a radical idea to a scientific community is

often a struggle that can greatly hinder scientific change. It is my belief that two necessary components are required when communicating radical or new ideas within a scientific community, namely shared models and the ability to generate a specialised vocabulary. I argued that the sharing of models and specialised vocabulary allows for the ability of community members to communicate their findings and thus greatly aids in achieving scientific knowledge.

To test my hypothesis, I offer two individual cases of radical thinking within the biological sciences, one in which a common pattern arises and another more speculative case in which the common pattern is beginning to form. The case studies show that, initially, radical thinkers are not able to communicate their ideas to the scientific community. It is through the sharing of a model, the sharing of a specialised vocabulary and a precise fit between the idea of an individual and a particular community that an idea can come to be scientific knowledge. Although I focus on only one way in which scientific change and communication may happen, I hope to enrich our current understanding of scientific discovery, scientific community, scientific change and scientific knowledge.

Notes

1. See Jacobs (2002, 2006) for details regarding Kuhn, Peirce, Royce, Polanyi, and Fleck's use of the term 'scientific community'.
2. Examples include, but are not limited to, Peirce ([1887] 1992), Polanyi (1951), Allen (1966), Royce (1968), Havelock (1969), Price (1969), Ravetz (1971), Feyerabend (1975), Gralewska-Vickery (1976), Cole (1979), Fleck (1979), Garvey (1979), Knorr-Cetina (1981), Zuckerman, Cole, and Bruer (1991), Staley (2004), and Rehg (2009).
3. The same could be said of Darden (1991). The book offers an interesting case study, Mendelian genetics, as a way to better understand theory change in science.
4. DeLanda (2015) argues similarly while focusing on the chemical science. According to DeLanda, fields have three important components: a domain of phenomena, a community of practitioners, and instruments and techniques. As such, scientific fields are broken down similarly, in that they are based around a cluster of common phenomena and scientists within a field share the same techniques instruments and methods.
5. See Bechtel (1986) regarding the integration of scientific disciplines, and strategies and examples of such integration.
6. Examples include, but are not limited to, Watkins (1970), Ravetz (1971), Kuhn (1977), Brown (1983), Hull (1988), Hoyningen-Huene (1993), Friedman (2001), D'Agostino (2005, 2010), and Jacobs (2006).
7. I do not mean to offer a complete delineation of all the components necessary for a scientific community. Scientific communities may be different from other scientific groupings, but shared models and specialised vocabulary are minimally required to delineate a communicative cluster of scientists, what we are calling scientific communities.
8. In what is dubbed 'new mechanistic philosophy', each 'new mechanist' offers a slightly different notion of mechanism. See Machamer, Darden, and Craver (2000, 3); Glennan (2002, S445); Bechtel and Abrahamsen (2005, 423); Craver (2007, 128–129).
9. See Anfinsen (1973) and Rooman et al. (2002) for more specifics on free energy and protein folding.
10. For example, protein-folding software is now available to the public. This software, or computer simulation, contains an explicitly built model system. The users can utilise models that are all governed by specific constraints with the common goal of adding to the scientific understanding of protein folding in general (Pronk et al. 2013).
11. More on prions and protein folding in section 4.

12. Focusing on Kuhnian notions of scientific community, D'Agostino (2005) argues that individual members of scientific communities can be members of multiple communities. Perhaps most interestingly, he goes on to show how an individual member can perform multiple particular roles within the hierarchy of a single community.
13. For a more contemporary example, see Piccinini and Craver (2011) regarding mechanisms sketches and the integration of neuroscience and psychology.
14. See Woody (2004) for more discussion regarding how model systems, or as she calls them scientific representation schemes, can reflect a community's epistemic aims and pragmatic concerns.
15. Chargaff, Zamenhof, and Green (1950) discovered the ratio of the base pairs, or the pairing of base pairs that allowed for insight regarding the structure of DNA. In other words, Chargaff introduced a constraint on the community's model system.
16. Craver and Darden (2013, 161–185) argue similarly regarding shared mechanistic models.
17. For case studies that exemplify this, see Shapin (1994), Galison (1997), and Knorr-Cetina (1999).
18. One further benefit to this schema is that it can greatly reduce the time and effort spent interpreting other scientists' models and theories. In this way, the sharing of similar models and specialised vocabulary enhances a scientist's ability to contribute to scientific knowledge (Fuller 2002, 206).
19. D'Agostino (2008, 2010) utilises both social psychology and organisation theory to better understand the proper balance of these two attitudes.
20. See Galison (1997, esp. ch. 9) for a detail discussion regarding the coordinating of action and belief, as well as real scientific case studies of such occurrences.
21. It should be noted that I am not interested in scientific disagreement, broadly construed. Instead, I want to focus on how scientists communicate their findings to a community that, initially, does not have the specialised language and other skills necessary to accept the novel idea. I am sure scientific disagreement does ensue, and often, but I am interested in cases where disagreement cannot even happen yet, because, to borrow from Kuhn, the scientists are speaking a different language. Thank you to Philip Kitcher for pointing this out during personal communication.
22. Kusch (2002, 73–74) argues similarly when he discusses background communities, in which a scientist's attitude toward a model, hypothesis, term, etc. can vary. See Fuller (2002, 208–216) for a variety of types of scientific consensus.
23. I am focusing on only one way in which scientific change and communication may happen, but still hope to enrich our current understanding of scientific discovery, change and knowledge.
24. For more regarding McClintock's model-building practices and the diversity of models within her model system, see Comfort (2001, 222). There, Comfort discusses McClintock's particular mechanisms for genomic rearrangement. Also, Comfort (2001, 223, fig. 8.6) shows one of McClintock's physical model schemas. See McClintock (1978) for more particulars.
25. It should be noted that McClintock was modelling several important mechanisms at the time. I have only offered a few, but I wish to stress the point that models often come in closely related systems. McClintock's research is a good example of this.
26. Other possible factors include, presentation and communication style, particular and advanced research background, gender discrimination, etc. (Keller 1983; Comfort 2001). For the purposes of this paper, I focus on differences in models (and model building) as well as terminology, but I do not mean to argue that these are the only reasons for McClintock's difficulties. Instead, I argue that these are important contributing factors.
27. I limit the discussion to human-centred prion diseases for simplicity.
28. Most notably, Prusiner's two Nobel prizes, countless articles/findings, and numerous new research avenues.
29. There are further concerns. For example, it has been established that one of the major routes of transmission is along the gastrointestinal tract. However, recently it has been shown that

the PrP is rapidly destroyed by alimentary track fluids (Jeffrey 2006). Other studies showed that living microglia from an infected brain had no detectable prions, yet contained maximal levels of infectivity (Baker, Martin, and Manuelidis 2002). One study showed that 'PrP neither encodes nor alters agent-specific characteristics' (Arjona et al. 2004). Also, blocking the formation of prions by an antimalaria drug does not lengthen victims' lives (Collinge et al. 2009).

30. Unfortunately, length constraints only allow for illustration of two cases case. However, the same patterns occur with other scientists as well, most notably, Charles Darwin. See Hull (1973) and Roe (Forthcoming).

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