Reference, Truth, and Biological Kinds

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Abstract  This paper examines causal theories of reference with respect to how plausible an account they give of non-physical natural kind terms such as ‘gene’ as well as of the truth of the associated theoretical claims. I first show that reference fixism for ‘gene’ fails. By this, I mean the claim that the reference of ‘gene’ was stable over longer historical periods, for example, since the classical period of transmission genetics. Second, I show that the theory of partial reference does not do justice to some widely held realist intuitions about classical genetics. This result is at loggerheads with the explicit goals usually associated with partial theories of reference, which is to defend a realist semantics for scientific terms. Thirdly, I show that, contrary to received wisdom and perhaps contrary to physics and chemistry, neither reference fixism nor partial reference are necessary in order to hold on to scientific realism about biology. I pinpoint the reasons for this in the nature of biological kinds, which do not even remotely resemble natural kinds (i.e., Lockean real essences) as traditionally conceived.
1. Introduction: Reference and conceptual change

There are occasions in the history of science that are of particular interest with respect to the metaphysical question of how concepts relate to the world. I am thinking of such episodes where some newly discovered thing generates controversy as to how exactly it should be classified. A recent example has been widely publicised: the question of whether trans-Neptunian object 2003 UB313 is or is not a planet. In the history of biology, there are many cases like this. Here are two examples. First, at the beginning of the 19th century, naturalists argued as to whether a newly discovered creature from Australia was a mammal or not. A very strange creature indeed, the duck-billed platypus *Ornithorhynchus anatinus* (first named *Platypus paradoxus*) appeared to have features from mammals and from reptiles and birds. In fact, some British naturalists, on being shipped the first specimens from Australia, thought it was a colonial prank.¹ Here is a second example: At the dawn of molecular biology in the 1940s, scientists discussed whether bacteria and viruses have genes. Both questions have been settled by the scientific community in the meantime: the platypus’ status as a mammal is secure, and bacterial and viral genes are all over the scientific journals. By contrast, UB313 didn’t make it and took poor Pluto down as well.

Cases like these may be seen as supporting a certain philosophy of language. According to a position known as “meaning finitism,” the extension of a term is not determined. This indeterminacy is such that, whenever a new case arises, there is no fact of the matter as to whether it belongs to the concept’s extension or not. The inclusion or exclusion of any referent of a concept is always subject to negotiation by the scientific community, meaning finitists argue (Barnes 1982, Bloor 1997, Kusch 2002). Clearly, meaning finitists will see cases like the platypus and the microbial genes as confirming instances for their philosophy of language: They will argue that, prior to the closure of these processes of negotiation, there was no fact of the matter as to whether the platypus belonged to the class *Mammalia*. By the same token, it was not determined whether bacteria contain any entities that are of the same kind as the genes of higher organisms. However, it must be stressed that a mere lack of consensus among a group of speakers alone does not prove that there are no reference-constituting facts, that is, facts that make it so that some thing falls

under a concept. If a group of speakers disagree whether some thing instantiates a certain concept or not, this could mean two things: It could mean that there is no fact that makes a certain thing belong to a concept before a relevant group has made a collective decision. But it could also mean that it is merely not known, or not known with certainty, if some reference-constituting facts actually obtain or not. According to some philosophers, there are reference-constituting facts associated with a term that may not be accessible or transparent to the relevant linguistic community.

Taking that second line is a challenge. Anyone who wants to argue that there are reference-constituting facts must be able to give a philosophically adequate answer of what determines the reference of scientific terms. And note that my concern is not whether the reference of terms can be determinate under some ideal conditions. It is rather whether, in these historical situations at hand, there were reference-constitutive facts that eluded the scientific community or where it was not known with certainty whether some such facts obtained or not.

I would like to examine whether a certain kind of theory of reference is able to establish the existence of such elusive reference-constituting facts about scientific terms, namely causal theories of reference. The first causal theories of reference have been developed by Saul Kripke (1980) and Hilary Putnam (1973). Such theories claim that the reference of terms may be fixed by the ostension of samples of some natural kind. After an initial "baptism", the term remains rigidly attached to whatever shares a common essence with the original samples. For example, the term "water" is rigidly attached to a certain molecular structure, H\textsubscript{2}O, which provides the underlying essence. This essence may be unknown, but the interesting cases are those where this essence is later discovered. According to the original version of the causal theory, such later discoveries of essences leave reference unchanged. I shall refer to this view as "reference fixism".

As is widely recognised today, the original version of the causal theory faces severe difficulties. Probably the most serious one is known as the "qua" problem. This problem arises because a sample may instantiate different kinds. A sample of water may also be viewed as instantiating the kind of liquids or hydrogen compounds, for example. Nothing in the original apparatus of the causal theory can distinguish between them. For this reason, many authors have modified the theory to allow certain content-bearing mental states to be involved in reference fixing (e.g., Nola 1980; Sankey 1994; Psillos 1999; Stanford and Kitcher 2000). Such theories are known as "causal-descriptive theories of reference". Because this quite a mouthful, I will refer to them simply as
“causal theories of reference”.

One of the goals of this paper is to show that under the assumptions of such a theory of reference, reference fixism about biological kind terms fails. The main example I shall use is the case of genes. After giving some historical background (Section 2), I will show that a refined causal theory of reference fails to establish reference fixism about the term “gene” (Section 3). Further, I would like to show that the reasons for this failure are philosophically interesting; they tell us something about the nature of kinds in biology and perhaps also in other special sciences. I will locate the reasons for the failure of reference fixism in the salient sameness of kind relations that underlie the classification of biological entities (Section 4). In Section 5, discuss the notion of partial reference and the attempt to use it as a basis for a realist semantics.\(^2\) I show that, in the context of biology, partial reference theory has consequences that are opposed to its realist goals. Finally, I will show that the failure of reference fixism and of partial reference is not a problem for realism about biological theories (Section 6).

2. The case of the gene

I would like to use the gene concept as an example, but I believe that some of the results may be of more general relevance. The history of the gene concept is extremely complex. Here are just some stages in its historical development (Carlson 1966; Portin 1993; Waters 1994, 2004; Weber 2005).

\(^2\) Such accounts are usually developed with the aim of countering forms of anti-realism that are based on Laudan’s pessimistic meta-induction and/or Kuhnian considerations that involve incommensurability (Laudan, L., 1984: A Confinuation of Convergent Realism. In J. Leplin (Ed.), Scientific Realism pp. 218-249, Berkeley: University of California Press. Kuhn, T. S., 1970: The Structure of Scientific Revolutions 2nd ed., Chicago: The University of Chicago Press). This kind of challenge begins by observing that there are historical predecessors of our contemporary scientific theories that were empirically successful, yet their theoretical vocabulary contains either terms such as “phlogiston” or “ether” that are thought to have no reference. In response to this challenge, realists have tried to show that at least some of the terms of these theories successfully referred (e.g., the terms “dephlogisticated air” or “transversal electromagnetic wave”) and that this referential success supported important truths.
<table>
<thead>
<tr>
<th>Time</th>
<th>Concept</th>
<th>Mendelian behavior</th>
<th>Gene-trait relation</th>
<th>Functional role</th>
<th>Material basis</th>
<th>Structure</th>
<th>Individuation</th>
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<tr>
<td>late 19th c.</td>
<td>pangene</td>
<td>no</td>
<td>?</td>
<td>pangogenesis</td>
<td>particulate</td>
<td>open</td>
<td>?</td>
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<tr>
<td>1900–1919</td>
<td>unit-character</td>
<td>yes</td>
<td>one-one</td>
<td>trait-determination</td>
<td>open</td>
<td>open</td>
<td>via trait</td>
</tr>
<tr>
<td>1915–1950s</td>
<td>classical</td>
<td>yes</td>
<td>many-many</td>
<td>phenotypic difference maker</td>
<td>chromosomes</td>
<td>subgenes (?)</td>
<td>complementation</td>
</tr>
<tr>
<td>1950s</td>
<td>neo-classical</td>
<td>optional</td>
<td>many-many</td>
<td>cistron</td>
<td>DNA</td>
<td>linear</td>
<td>complementation</td>
</tr>
<tr>
<td>1960s</td>
<td>molecular</td>
<td>optional</td>
<td>many-many</td>
<td>protein coding</td>
<td>DNA</td>
<td>colinear w/ protein</td>
<td>via gene product</td>
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<td>1970s–present</td>
<td>contemporary</td>
<td>optional</td>
<td>many-many</td>
<td>protein/RNA coding</td>
<td>DNA / RNA</td>
<td>intron/exon (optional)</td>
<td>via gene product(s)</td>
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I shall try to simplify this story by trying to answer the simple question that I raised at the beginning: Did the term "gene" around 1940 refer to bacterial and viral genes, even though the latter had not yet been discovered?

Let us assume that the reference of the term "gene" was originally fixed with the help of a few experimental systems, in particular the fruit fly *Drosophila*. This was the main model organism used by Thomas Hunt Morgan and his associates to develop the classical theory of the gene in the years 1910-1915 (Morgan, et al. 1915). In their writings, these geneticists introduce the term "gene" by describing certain patterns of inheritance of certain trait differences in the fruit fly. These patterns include the segregation of certain traits according to the Mendelian ratios, and the independent assortment of pairs of traits.

They also show that these patterns of genetic transmission can be explained by assuming the existence of independent factors or genes that are located on the fly chromosomes. Genes that are located on the same chromosomes tend to be transmitted together, a phenomenon that was termed "linkage". But with a certain frequency, this linkage was broken. Morgan and his associates argue that the observed frequencies can be explained by assuming that the genes are arranged linearly on the chromosomes. They are also very careful in pointing out that the relationship of factors and traits was many-many: most genes affect many traits, and most traits are affected by many genes.

It is tempting to suggest that the experimental practices of these early geneticists rigidly attached the term "gene" to the things that were causally responsible for the trait differences, behaved in accordance with these Mendelian patterns and everything else that shares some kind of essence with these things. This is what a causal theory of reference suggests for this case. I will work out this suggestion in more detail in the following part, using a refined version of the causal theory of reference due to Stanford and Kitcher (2000). Then I will show that such an attempt to defend reference fixism about the term "gene" fails.

### 3. Reference Fixism: Stanford and Kitcher

Let us assume that the reference of the term “gene” was fixed by the following means (this is a slightly modified version of a causal-descriptive theory of reference that has been developed by Stanford and Kitcher 2000):

a) A range of experimental systems consisting of different strains of fruit flies and a few other organisms showing both instances and counter-instances of Mendelian inheritance
b) A complex conjunctive predicate $\Phi(x)$ composed of predicates $\phi_1 x \& \phi_2 x \& \ldots \& \phi_n x$ such that each instance satisfies $\Phi(x)$ and each counter-instance fails to satisfy $\Phi(x)$

We are not assuming that the constitutive predicates $\phi_n x$ are purely observational. In other words, inferences are permitted when applying these predicates.

And these might be the relevant $\phi$-properties in our present example:

- $\phi_1$: is arranged linearly on chromosomes
- $\phi_2$: segregates and assorts in accordance with Mendel’s laws (three kinds of Mendelian inheritance according to T.H. Morgan 1917: autosomal, sex-linked and due to unusual distribution of chromosomes)
- $\phi_3$: exhibits linkage to other factors located on the same chromosome
- $\phi_4$: crosses over with a frequency roughly proportional to the distance between two factors
- $\phi_5$: complements alleles residing at different loci
- $\phi_6$: mutates spontaneously or under the influence of ionising radiation or certain chemicals
- $\phi_7$: causes heritable phenotypic differences when mutated (difference makers, not total causes!)

The question now is of this apparatus is sufficient to attach the term “gene” to a class of things sharing an essence. What might this essence look like?

Of course, today we have the molecular gene concept according to which genes are DNA sequences that determine the linear structure of a protein or RNA molecule. Could we not view this coding property as something like an essence that is shared by all genes, including bacterial and viral genes? In asking this question, it is important to note that causal theorists of reference will not be worried about the fact that Morgan and his associates did not know the molecular essence of genes. Causal theories of reference were developed for precisely such cases.

The crucial question is whether the classical gene concept picked out a molecular, relational essence. The question is far from being trivial. Reasons can be given both for affirming or for denying such a thesis for referential continuity. Many of the genes isolated in Morgan’s lab were later described at the
molecular level. I have shown that the classical gene concept and the associated operational criteria were actually used for isolating molecular genes in *Drosophila* (Weber 2005, Ch. 6). The molecular concept, on the other hand, was worked out mainly by using bacteria and bacteriophage as model organisms.

However, none of this really proves that the reference of the term "gene" as it was introduced by classical geneticists, extended to bacteria in 1940. For bacteria did not exhibit the patterns of inheritance known from fruit flies and other higher organisms. They have no chromosomes in the classical, cytological sense of the term. They don’t exhibit Mendel’s laws. Something like phenomena of linkage and crossing-over can be observed, but only under highly contrived experimental conditions. These include, for example, double infections of bacterial cells with two different strains of virus. What is interesting to note is that Seymour Benzer, who was the first to apply the technique of complementation analysis to bacteriophages by using this technique, had strong reservations about the term "gene" (Benzer 1955).

The only property that bacteria showed from the beginning was random mutation. This was shown in a classic study by Max Delbrück and Salvador Luria that was published in 1943 (Luria and Delbrück 1943). In the conclusion section of their paper, Delbrück and Luria wrote: “Naming such hereditary changes ‘mutations’ of course does not imply a detailed similarity with any of the classes of mutations that have been analyzed in terms of genes for higher organisms. The similarity may be merely a formal one.” Clearly, they were reluctant to draw any close parallel between the processes they studied in bacteria and those studied by *Drosophila* geneticists. Of course, this will not worry causal theorists of reference because, in their view, reference-constituting facts may obtain irrespectively of what scientists actually believe.

However, what causal theorists of reference must show is that the scientists’ mental states together with the experimental systems originally used when some term was introduced uniquely pick out some essence, for example to DNA sequences that have the coding property. What made it so that the classical term ‘gene’ referred exactly to the set of DNA sequences that share the coding property in their cellular context?

One suggestion might be that the coding property is the function that explains all of the properties traditionally associated with genes, in other words, the φ-properties according to our present account. I mean “function” in a minimal causal role sense, that is, not in the sense of proper function. We could further modify Stanford’s and Kitcher’s theory of reference. They suggest that natural kind terms refer to “the set of those things having the inner constitution that is a common constituent in the total causes of the presence of each
of the $\varphi$-properties in each of the samples. This is not applicable to our case, because genes are not structural kinds. We need to substitute function for structure.

So is there some function that is a common constituent in the total cause of each of the $\varphi$-properties? This does not seem right. Some of the phenomena studied by classical geneticists, in particular the Mendelian regularities, are explained simply by the way in which the chromosome align and separate in the formation of germ cells, not by the coding property.

Note also how important the Mendelian behavior was for the initial referential success of classical geneticists. To drop the Mendelian behavior from the list of properties involved in reference fixing means also to drop the chromosomal location of genes. But this allows the *qua*-problem to run amok. Because then it is not at all clear what functional properties the geneticists were ostending when they introduced the term "gene" into discourse. The reference of "gene" then might include all sorts of things that are involved in heredity, including cytoplasmic factors. If classical geneticists succeeded in referring to anything, it was something that is located on a chromosome and, therefore, exhibits the Mendelian patterns.

Stanford and Kitcher suggest that "a principal motivation for causal theories lies in the possibility of discovering that some members of a natural kind lack properties originally used in picking out that kind". They suggest that this was the case in the example they have studied, which is the chemical term "acid". So Stanford and Kitcher, it seems, would allow that some referents of a kind term are later shown to lack some of the properties that once were crucial for referential success. This would allow bacteria to have genes, even though they lack Mendelian inheritance. However, I will show now that their account cannot be modified in a way that would allow us to say that the classical gene concept picked out the molecular essence of genes.

4. Sameness of Kind: Why Reference Fixism Fails

In section 3, I raised some scepticism concerning the idea that the classical term "gene" may have referred to some molecular essence prior to the advent of molecular biology. Now, it is time to provide some metaphysical grounds for this scepticism.

Genes are no kind like those that have been discussed in physics and chemistry. Here are some differences, most of which have been consistently ignored in discussions of reference and biological kinds:
(1) Genes are a relational kind. To be a gene is not an intrinsic property of some chemical substance. Some DNA sequences are only genes because there exist cellular contexts that contain specific biochemical machinery of gene expression. While some of the machinery can recognize DNA sequences from other species, most genes are only properly expressed by cells derived from the same species (unless the sequences are tampered with by genetic engineers). Thus, while to be a H2O molecule is an intrinsic property that a thing can possess independently of anything else, to be a gene is not an intrinsic property.  

(2) Genes are a functional kind, in the sense that they are individuated by their causal role in a system. This is partly responsible for the relational character of genes mentioned above. In the molecular sense, genes are also structural kinds, because only things made of nucleic acid are called “genes” today. Thus, genes are a mixed-functional kind (Waters 2000).  

(3) Genes are a variable kind. All water molecules are the same. By contrast, genes vary enormously both within and between species.  

(4) Genes are a generic kind. Genes come in billions of subkinds such as “the human PAX6 gene” or “the Drosophila melanogaster white gene”, etc. The generic kind of gene and these subkinds are related in the same way as the kind “species” is related to the kind “Homo sapiens”. Every species taxon (e.g., H. sapiens) is an instance of the species category (to use Ernst Mayr’s terms). By the same token, every specific gene (e.g., the human PAX6 gene) is an instance of the generic kind “gene”. I shall use the terms specific gene kinds and generic gene kind to distinguish these.  

(5) Genes are sortal kinds. You can count genes, and a statement of the form “there are less than 50000 human genes” (meaning specific gene kinds) or “this plasmid contains three genes” (meaning tokens of arbitrary specific gene kinds) are complete without requiring extra sortal terms. By contrast, statements  

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3 Neumann-Held, E. M. (1999). The Gene is Dead - Long Live the Gene! Conceptualizing Genes the Constructionist Way. In P. Koslowski (Ed.), Sociobiology and Bioeconomics. The Theory of Evolution in Biological and Economic Thinking pp. 105-137). Berlin: Springer. has argued that genes ought to be conceptualized as containing all the biochemical machinery necessary to express them. I don’t see the need for such a radical departure from molecular biological uses of the term “gene”. That genes are relational with respect to this machinery does not mean that they contain it (in a mereological sense).
of the form “there are about 1’000’000’000 H_2O in this sample” is incomplete without addition of general sortal term such as “molecule”. This sortal term, by the way, is unfit for gene talk, as one molecule of DNA or RNA may contain an arbitrary number of genes.

Another way of expressing this characteristic is by pointing out that the term “gene” is a count noun (see Rosenberg 2006, 114). Most of the natural kind terms that have been discussed in the philosophy of science are not count nouns, but mass nouns. Examples include “oxygen” or “water”. Even “acid” is a mass noun. You can count acid-types (sulphuric acid, acetic acid), but not acid-tokens, unless you introduce another sortal expression such as “molecule”. Examples of count nouns in the physical sciences are “atom” or “electron” but – curiously – these are not the terms that have been discussed the most in debates over reference and concepts in science.

I would like to claim that some of these characteristics are responsible for the difficulties of applying a causal theory of reference to the kind “gene”. For such theories to work, it is instrumental that there is a Lockean real essence (i.e., an inner constitution or common structure) that furnishes the salient sameness of kind relation. This real essence had better be nomologically linked to the properties used to identify instances of the kind (Locke’s “nominal essence”). The relational nature of genes is not compatible with there being such nomological connections. Gold atoms and the laws of physics (should those be in some sense independent of the intrinsic properties of gold, which some metaphysicians doubt, see Ellis 2001) make it so that lumps of gold exhibit the same properties in many different contexts in which they can exist. This is not so in the case of the gene. DNA or RNA as a chemical compound may satisfy this requirement, but not any piece of DNA or RNA contains genes. The gene-making relations are context-dependent. A piece of human DNA will not be biologically active in most cellular contexts, even if in its original context (a human cell) it contains a fully functional gene. Therefore, with respect to their biological (as opposed to purely chemical) properties, genes lack the kind of context-independent nomological relations to other properties.

Does this matter at all? This will depend on whether there is some sort of unique causal role that all and only genes share and that, perhaps, could constitute their relational essence. According to molecular biology, there appears to be such a role: The causal determination of the linear sequence of
either RNA or protein molecules (in the appropriate cellular environment).\textsuperscript{4} The question is if this causal role is specific enough to delimit all and only genes. There are reasons for doubt. First of all, the notion of “causally determining the linear sequence of a biomolecule” is in need of explication. Probably the best explication for this causal notion is this: The salient sense of causal determination here is to be explicated in terms of \textit{causally specific actual difference-making causes}. Ken Waters (forthcoming) has recently used James Woodward’s manipulationist theory of causation in order to explicate this concept. This account starts by differentiating between \textit{potential} and \textit{actual} difference-making causes in a population of entities (e.g., the population of proteins in a cell). Actual difference-making causes are those that actually vary in the population and that account for the variation of the dependent variable. Potential difference-making causes are capable of this, but they don’t \textit{actually} vary in the population. Where the actual difference-making cause fully accounts for the variation in the dependent variable, Waters speaks of \textit{the} actual-difference-making cause. If the independent variable accounts for the variation in the dependent variable only partially, Waters refers to the former as a actual difference-making cause (whether a given variable is “independent” or “dependent” is to be analyzed in accordance with Woodward’s theory of causation. Basically, independent variables (causes) are those that can be manipulated such as to change the value of another variable (effects) in a way that does not alter the value of any other variables that could do the same.

According to Waters, this apparatus can be used to specify a unique role for certain nucleic acids in determining the linear structure of other nucleic acids or proteins, for example, prokaryotic genes (where there is no post-transcriptional modification). An additional causal concept is needed to single out a unique role for eukaryotic genes: the concept of causal specificity. Waters borrows this notion from Lewis (2000). Briefly, specific causes are causes where a multiplicity of different states of an independent variable are causally linked to a comparative multiplicity of states of the dependent variable. Using

\footnotesize{\textsuperscript{4} As Ken Waters has argued, in molecular biology the use of the term “gene” is context-sensitive: depending on the stage of gene expression that is being talked about, a gene may include or exclude certain DNA sequences. For example, in a context where biologists talk about primary transcript, they will mean the term “gene” in a sense that includes the introns (non-coding intervening sequences that are spliced out after transcription). By contrast, in a context where they speak about mRNA or finished proteins, the gene will exclude the introns (Waters, C. K. 1994. Genes Made Molecular. \textit{Philosophy of Science}, 61, 163-185.). This may be an extra complication for a causal theory of reference, but it seems to me that it fades in comparison to those that I discuss in the text.}
this notion, Waters argues that eukaryotic genes are the only causally specific actual difference-making causes in RNA- and protein synthesis.

I have argued elsewhere that the notion of causal specificity admits of degrees (Weber 2006). Causal specificity may be viewed as special kind of invariance in the sense of Woodward (2003), namely a relationship such that a change in the independent variable (e.g., a DNA sequence) would bring about a change in a dependent variable (e.g., protein sequence) in a way as it is specified in the relationship. Causally specific relationships are such that they relate discrete variables. Now, depending on how many different values these variables can take, the relationship is more or less causally specific.

Let us now analyze the causal influence of eukaryotic genes on proteins. DNA is an actual-difference making cause, but so are certain agents that are responsible for alternative splicing (the production of different polypeptides from a single RNA molecule by cutting and joining the exons or coding sequences is different ways). So far, these factors are causally on a par (Oyama, Sterelny and Griffiths (1999)). However, DNA is more causally specific a variable than the splice agents, because it could take a much larger number of different values (= nucleotide sequences). Thus, we may define the causal role of genes as that of being the most highly specific actual difference-making causes in the synthesis of RNA and protein in a cell.

Now add to this the properties from the nominal essence of genes according to the classical theory, i.e., chromosomal location, complementation, Mendelian inheritance, mutation, recombination. A causal theorist of reference might suggest that the term “genes”, as it was used in the classical period” denoted exactly those parts of the Drosophila chromosomes that had these ϕ-properties in the fruit flies and any other thing that shares the causal role of being the most highly specific actual difference-making cause of the linear structure of RNA and protein in the cell (but that need not have any of the ϕ-properties).

I think the problem with this suggestion is obvious: This account of the reference of “gene” attributes to Morgan and his associates mental states that they did not have. They may have had mental states bearing contents such as “difference-making cause”, perhaps even “highly specific actual difference-making cause” (perhaps implicitly so). But they did not have thoughts containing the idea that genes are the most highly specific difference-making cause of the linear structure of protein and RNA. It was not yet known at that time that genes play this biochemical role. But it is necessary to spell out this role in order to secure reference to the kind of things recognized as genes by contemporary biology. If that is left out, all we have is a bunch of fly genes
and everything else that has the same causal role. But these genes play many causal roles, so the reference of “gene” would include way too many things.

It seems to me that the general problem is this: There might be no other way to pick out a function short of actually specifying the function. I can point to a particular space-time region, say, one containing liquid water and say “I am talking about that stuff, and everything else that has the same structure” and succeed in referring. To be precise, we can succeed provided that we can solve the qua problem by specifying some appropriate \( \varphi \)-properties such as boiling temperature to exclude that I am talking about the natural kind of liquids, for example.

But if I want to fix the reference of the term “heart”, I can’t just point to my chest, saying “I am talking about that thumping thing in there, and everything else that has the same function.” This would pick out far too many things. For example, this might pick out all things that make thumping noises or that produce heat and carbon dioxide (note that I mean “function” in a minimal causal role sense). In order to succeed in referring, I need to specify what function I am talking about, for instance, the blood-pumping function. Therefore, it is not possible to refer to such an essence without already knowing it. But this is exactly what causal theories of reference would require.

As LaPorte (2003) points out, we should not judge a theory of reference on the basis of whether or not it makes reference determinate. A theory of reference shouldn’t see referential determinacy where there is none. However, the whole point of bringing causal theories of reference to the philosophy of science so far has been to establish referential continuity in the face of theoretical and conceptual change in science. The upshot of my analysis, so far, is that the case of the gene lacks such continuity, and there are in principle reasons for this, reasons that have to do with the nature biological kinds.

I now turn to examining whether the case of the gene exhibits partial reference. This is a form of referential indeterminacy, but presumably one that does not beget radical conceptual change of the kind that spells doom for scientific realism.

5. Partial reference and truth

The idea of partial reference was introduced by Field (1973). Using the transition from Newtonian to relativistic mechanics as his main example, Field argued that there is no fact of the matter as to what the term “mass” referred to prior to Einstein. It did not refer to proper mass, nor did it refer to relativistic
mass (which are taken as the real properties). Reference was indeterminate, and the Newtonian concept of mass was lacking in discriminatory capacity with respect to this distinction. Nonetheless, Field suggested that there is a relation of “partial denotation” between the term “mass” as it was used before Einstein and the real properties relativistic mass and proper mass. This means that the term did not refer to either property; rather it partially referred to both. According to Field, a similar relation obtains between the classical term “gene” and the units of recombination, of function (Benzer’s cistron), and of mutations. Before the advent of molecular biology, the term “gene” lacked discriminatory power to distinguish these different units. Thus, the term “gene” partially denoted all of them.

Stanford and Kitcher also end up endorsing partial reference towards the end of the paper. In their main example, which is the chemical term “acid”, chemists abandoned some properties as being essential for acids that were once thought to be essential. This would not be a problem if there were only one salient natural kind in the relevant domain. In the latter case, it would be possible that the properties used to identify acids (i.e., its nominal essence) could change, while the term “acid” would still refer to the same real essence. But there are several natural kinds that once were candidates for the reference of the term “acid”. Hence, reference of the term was partial.

5 T.S. Kuhn, famously, argued that these concepts are incommensurable, meaning that there is no way of expressing one concept solely in the vocabulary of the other theory. According to Carrier, M. (2001). Changing Laws and Shifting Concepts: On the Nature and Impact of Incommensurability. In P. Hoyningen-Huene, & H. Sankey (Eds.), Incommensurability and Related Matters pp. 65-90). Dordrecht: Kluwer. Kuhnian incommensurability means that it is impossible to preserve both the conditions of application and the standing inferential relations in an attempt to translate statements containing concepts into the language of a theory that contains concepts that are incommensurable to the first. Field’s argument can be seen as an attempt to salvage a weak form of realism in the face of the Kuhnian challenge.

6 It is often said that classical geneticists such as the school of T.H. Morgan thought that these units coincide. This is historically incorrect, see Weber, M. (1998). Representing Genes: Classical Mapping Techniques and the Growth of Genetical Knowledge. Studies in History and Philosophy of Biological and Biomedical Sciences, 29, 295-315.

What is the point of introducing the notion of partial reference? This becomes evident when we ask what the notion of reference was once introduced for: truth. A statement of the form $Fa$ is true exactly if $a$ belongs to $F$’s extension. Reference is the relation between a predicate’s extension and its term, and true statements are such that they predicate a predicate of a member in its extension.

Partial reference is only an interesting relation to the extent in which it can support truths. The whole point of saying that Newton’s term “mass” partially referred (as opposed to complete failure of reference as suggested by Kuhn and Feyerabend) is to enable Newton and other pre-Einsteinian physicists to have said at least some true things about the world, even though their theory on the whole was false. Field suggested the following way of allowing for truth with partial reference. Assume that a scientific term such as “mass” is associated with different structures that map this term to different referents. One such structure may map the term “mass” to relativistic mass, while another may map it to rest mass. Any statement may now be true or false with respect to a given structure. For example, with respect to a structure that maps “mass” to relativistic mass, the statement “momentum equals velocity times mass” is true (by the lights of relativity theory), while the same statement is false with respect to a structure that maps “mass” to proper mass. So long as this is the case, i.e., when different structures give rise to different truth-values to statements containing partially referring terms, we can’t say that the statement is true. Its truth value is indeterminate. However, there is the logical possibility that all the structures of such a statement return the value “true”. An example would be “in a given frame of reference, the mass of the Earth is less than the mass of the sun”. No matter how “mass” is interpreted in this sentence, it comes out true (again, by the lights of relativistic mechanics). In such cases, Field allows a sentence to be true even if contains partially referring terms.

McLeish (2006) has argued that Field’s account is too restrictive on truth. It will recognize precious little truths to have been spoken in the history of science. Furthermore, Field’s account of truth under partial reference is in conflict with some strong intuitions. McLeish therefore suggests the following amendment of Field’s account. First, any partially referring term is not only associated with a set of structures that map the term to some set of referents. It also contains a structure that maps the term to the empty set. Thus, a statement like “dephlogisticated air does not exist” is true under at least one structure if that term refers partially. This is in line with our intuition that, in a sense, there is no such thing that fits the description that Priestley et al. gave
of dephlogisticated air. But at the same time there is a way of interpreting some statements made by phlogiston chemists according to which “dephlogisticated air” referred to oxygen, such that “dephlogisticated air supports respiration” is true. Of course, there is no single interpretation that makes the absurd sentence “dephlogisticated air does not exist and supports respiration” true. This is how it should be.

A second modification introduced by McLeish is to say that sentences containing partially referring terms are true if there is at least one structure that makes the statement true. Thus, a statement such as “dephlogisticated air supports the respiration of mice” may be true, namely if there is a structure that maps “dephlogisticated air” to oxygen and “oxygen supports the respiration of mice” is true. In contrast to Field’s original account, which is conjunctive, McLeish’s account is disjunctive. This makes it much more permissive with respect to truth.

McLeish’s account has several attractive features. First, it does not make reference of a term used in a statement made in the past a matter of whether that statement is true (which would put the cart before the horse. Successful reference begets truth, not vice versa). Second, it does not privilege any of the descriptions of theoretical entities or magnitudes that scientists used in the past. Thirdly, the account does not need to appeal to our intuitions as to whether some past tokening of a term referred. Thus, it avoids a certain kind of whiggism.

I want to leave open question as to whether McLeish’s theory gives a correct account of the reference of terms from the physical sciences, such as “mass” or “dephlogisticated air”. Of course, there cannot be much hope that this account has no difficulties of its own. McLeish’s account is in danger of making reference a vacuous relation. To avoid vacuity, it must be able to show how reference failure is possible. We can’t have any term from the history of science partially refer, e.g., things like Darwin’s “gemmules,” just because it may be associated with a structure from some class that contains one good structure. I will not delve on this issue here. What I would like to do instead is to show that the aim of allowing truths to be spoken in the past can be reached without partial reference, at least in biology.

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6. Classification and general truths in biology

When Priestley spoke about “dephlogisticated air”, this term may have partially referred to oxygen. Oxygen is a traditional natural kind in that all samples of oxygen share an essential property that all and only the kind members instantiate (Locke’s “real essence”, given by atomic number according to contemporary chemistry). If McLeish is right, then some of Priestley’s statements may have been true, given that there is a partial structure associated with this term that maps “dephlogisticated air” to oxygen. If some general statement endorsed by Priestley was true, e.g., “dephlogistocated air sustains respiration of mice”, then it was true of all the members of the natural kind that was partially denoted by “dephlogisticated air.”

Now contrast this example with some claim made by a classical geneticist, for example, “genes cross over with a frequency that is roughly proportional to the distance of their separation on the chromosome”. Is there a partial structure in Field’s and McLeish’s sense that maps Morgan’s use of the term “gene” to a natural kind? Perhaps there is, provided that this partial structure excludes everything that fails to exhibit this classical genetic regularity. But note that we can just as well say that there is a subkind of what is today recognized as genes that is fully (as opposed to partially) denoted by Morgan’s term gene, namely, all eukaryotic genes that reside on the same chromosome of a diploid, sexually reproducing organism. This subkind is variable; it contains different genes from the same species and genes from different species. There is nothing wrong with some use of the term “gene” refer to a subkind of what is today recognized as the class of genes. If we compare this to the oxygen case, we notice that this is not a life option there. You can’t refer to a subkind of the natural kind “oxygen”, because there aren’t any.9

We are now ready to consider the problem mentioned in the introduction, to wit, if “gene” referred to bacterial genes before the advent of bacterial genetics. If it did so refer, then it can only have referred partially. For to say that it fully referred to bacterial genes requires that we privilege some description of genes as the dominant one. This can only be done by the lights of molecular biology, which begs the question, see McLeish. However, partial reference is in danger of being a vacuous relation. Is there any way out of this dilemma? I think there is. We can simply say that Morgan et al. only referred to some subkinds of the molecular kind of genes, namely Drosophila genes and

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9 There may be different isotopes of oxygen, but these do not differ chemically. Genes, by contrast, come in different subtypes that differ biologically.
perhaps the genes of some sufficiently similar organisms. Thus, we should read general sentences from classical genetics as ranging only over subkinds that do not include things such as bacterial genes. If we attribute to Morgan’s term “gene” the full (not partial) reference of all molecular genes, this makes most of his general beliefs plainly false. (Bacterial genes show very few of the characteristics that Morgan et al. discovered in *Drosophila*.) This violates the intuition that his group of researchers discovered important truths about inheritance in sexually reproducing organisms. The flight to partial reference is cumbersome, for the reasons indicated. But we don’t need partial reference: We can say that Morgan’s sentences were not referring to bacterial genes at all. Instead, these sentences were only about the model organisms used back then plus, perhaps, a few others.

By the way, many of the specific genes that classical geneticists talked about were later re-described at the molecular level (Weber 2005, Ch. 7). Thus, there is no difficulty in saying that when Morgan talked about the *Drosophila white* gene, he referred to the same class of DNA sequences as a modern biologist (or the *Drosophila* genomic database known as “Flybase”). Many of the subkinds of the generic kind “gene” are quite stable throughout the history of genetics. However, the generic term “gene” has not been stable, as many authors have suggested (Kitcher (1982); Burian (1985); Waters (1994); Burian, et al. (1996)). The reference of this term has been “floating” incessantly as new mutants were discovered, as new model organisms and new experimental systems were developed (Weber 2005, Ch. 7).

Biology does not aspire to the kind of generality known from physics or chemistry. General claims in the latter disciplines range over the whole universe. Oxygen atoms, electrons or mass have the same properties and enter into the same nomological relations no matter where they are found. Theories that describe the interactions of fields and particles are universal. Biological theories are much more local. No-one expects there to be a universal genetics. The genetic code, which is found in most organisms on Earth, is about as universal as it gets in biology. This is a far cry from the generality of physical and chemical theories.

As a result, truth comes much easier in biology, unless biologists over-generalize. Of course, they have been known of over-generalizing. But there is no indication to think that the theory of the gene, as it was proposed by Morgan et al., was supposed to cover all life on Earth, including bacteria and archaea. There is certainly no indication in the works of these authors that would suggest that they thought their theories would have this kind of scope. Therefore, to ascribe to their term “gene” such a wide reference as to include
bacteria is both uncharitable and unnecessary. What is more, this is uncharitable and unnecessary before we even begin to consider the further difficulties that this will incur, especially those of partial reference.

In comparison to physics and chemistry, biological theories are only of restricted scope. There may be generalizations that are true of all genes (in the molecular sense), but there are also generalizations that are true of some subclasses of genes. (By contrast, there are no physical theories that are only true of some samples of oxygen, or some instances of mass). The theories of classical genetics should not be interpreted as making claims about all kingdoms of life; this takes the theory further than its own fathers would have been willing to defend. For this reason, it is best interpreted as having established full reference (as opposed to partial), but not to the full set of things that are recognized as genes today. Reference was only to some subclasses of the kind.

At this point, it may be asked if partial reference does not make a similar claim: Does it not also say that a term may partially refer to different kinds which often stand in some hierarchy of kinds? For example, according to partial reference theorists, “dephlogisticated air” also partially referred to gases, which contains oxygen as a subkind. Why should we not say that the classical term “gene” partially referred to some subclass of genes (e.g., those studied by Morgan & Co.), but it also had the full set of molecular genes as a partial referent? As long as partial reference is construed along the lines of McLeish’s disjunctive account, this still allows some sentences produced by classical geneticists to be true.

The difference becomes clear if we ask to what extent the different accounts assign the same truth-value to different sentences. Take a sentence such as “all genes are located in the cell nucleus”. If “gene” is read in the molecular sense, this sentence is false (bacteria don’t have a nucleus, and in eukaryotes there are also mitochondrial and chloroplast genes). On my analysis, the sentence is true if said or thought before the advent of molecular biology. Because I maintain that the reference of “gene” did not reach very far beyond the organisms that were experimentally accessible back then. But on McLeish’s account, this sentence may also be regarded as true, provided that there is a structure that maps “gene” to just the nuclear genes of higher organisms (even if there is also a structure that maps “gene” to the set of molecular genes that makes the sentence come out false). Thus, in this case, the two accounts assign the same truth-value. So far so good.

But now comes the rub: There are also sentences such as “all genes segregate in accordance with Mendel’s laws of segregation and independent assortment”. This sentence was known to be false as early as 1916. Many genes
don’t obey Mendel’s laws, in fact, the whole history of early 20th century genetics may be described as the discovery of a series of anomalies to these laws (Darden 1991). One of the first anomalies was sex-linked inheritance, another was linkage. I would say that while, originally, the term “gene” only referred to things that obey Mendel’s two classical laws, the reference of the term was expanded to accommodate new cases as genetics was developed (Weber 2005, Ch. 7).

Here, McLeish’s account exhibits its difference, and also its difficulties. I see no reason why the partial reference theorist should not say that things that obey Mendel’s laws of segregation and independent assortment belong to a Field/McLeish-style structure. It’s as good as the other structures that we have examined so far. But this has the undesirable consequence that the sentence “all genes obey Mendel’s laws of segregation and independent assortment” comes out true, therefore attributing to Morgan & Co. false beliefs that they did not entertain.

Could McLeish’s account not be saved from this difficulty by saying that “things that obey Mendel’s laws of segregation and independent assortment” was not among the descriptions that Morgan et al. used to refer to genes? In fact, statements can be found in their texts that explicitly exclude this description as reference-relevant.

However, this move is not open to the partial reference theorist. For the partial reference theory forbids us to privilege some descriptions in determining reference. If descriptions that widen reference are parts of a Field/McLeish structure, then so are descriptions that narrow reference. But as soon as this is accepted, the damage is done: This makes statements true that are clearly false by any lights, be it our best contemporary theories or some historical predecessor.

Why does this problem not arise in the more traditional cases such as oxygen? It seems to me that, in the latter cases, there is a smallest causally homogeneous kind the (partial) denotation of which by some scientific vocabulary is responsible for the truth of certain sentences. We are there in the tidy world of physics and chemistry, which is neatly divided into causally homogeneous kinds of truly cosmic extensions. This is not so in the messy world of biology. Here, causal homogeneity is a matter of degrees, and a matter of relations. Some class of entities may be causally homogeneous in relation to some specific mechanism (i.e., the gene expression machinery of a bacterial species) but causally heterogeneous in relation to another mechanism. Causal homogeneity is a matter of context in biology. Hence, there is no smallest causally homogeneous kind that the theory of partial reference needs in order to avoid to
make far too many statements true. Even if it works for physical and chemical kinds (which I doubt), it cannot do justice to the nature of biological kinds.

It is time to take the special character of biological kinds into account when speaking about reference and truth in biology. I suggest that taxonomies of kinds in biology should be viewed as open classification systems, much like biological systematics itself. In contrast to classifications systems such as the period table or the standard model in particle physics, there is no limit to the number of kinds that such a system could accommodate. It is always possible to introduce new taxa, to lump or split existing taxa, or to enlarge or contract existing taxa. Such classificatory choices will be informed by the theoretical goals that the classificatory system is supposed to serve (and perhaps practical goals and interests as well). The species category, for example, can accommodate an unlimited number of species. It had better be able to so, for new species arise by evolution all the time, while existing species go extinct. When a new species arises, this does not correspond to the filling of a pre-existing slot (unlike when an atom of some chemical element forms for the first time). By the same token, an extinct species does not leave an empty slot behind.

It was a mistake to model the reference of biological terms on the model of oxygen or mass, as Field (1973) or Kitcher (1982) have done. Biological systematics is a much better model. The term “gene” is more similar to the term “species” than it is to “electron” or “acid”. It is generic term that comes in many subtypes. “The human PAX6 gene” is related to “gene” like “Homo sapiens” is related to “species”. As in the case of species, new genes arise all the time by evolution. When that happens, there is no filling of a pre-existing slot. Even though not infinite in the mathematical sense, the number of possible genes is not limited in any relevant way.

Biology differs enormously from physics with respect to the generality of its theoretical claims—this is hardly news (Beatty 1995; Waters 1998; Weber 1999; Mitchell 2000). But what has not been sufficiently appreciated are the implications of this insight for the theory of reference. Today, in the age of genomics, generalizations such as those of classical genetics (Waters 2004) generalize over subkinds of all the things that are classified as genes. By contrast, in the era of classical genetics, these generalizations ranged over the whole extension of the term “gene”. This makes for a substantial reference shift. At the same time, this does justice to the intuition that Morgan and co-workers discovered important truths. What is more, none of the other accounts of reference that have been proffered in the history and philosophy of science do proper justice to this intuition. The view of reference fixism attributes to classical geneticists many false beliefs, because it has them make general claims
about genes that differ radically from the genes they had experimental access to (e.g., bacterial genes). There is no historical evidence that these scientists actually held such beliefs. The theory of partial reference, as I have shown, makes statements true that classical geneticists (correctly) thought to be false. Thus, a view of “floating reference” (Weber 2005, Ch. 7) does the best job in attributing true beliefs, and not too many false ones, to classical genetics.

Of course, we should not judge a theory of reference solely on the basis of what kinds of statements it makes true. On the other hand, intuitions about the truth of historical theories has been a major motivation to develop such theories in the first place. Clearly, the alternative theories of reference that I have discussed have problems other than what kinds of truths they support. We can now add to these problems the fact that, with regard to biological kinds, these theories are not necessary in order to hold on to the view that the historical predecessors were tracking important truths.

7. Conclusions

I have examined various theories of reference and conceptual change with respect to what they say about biological kinds, in particular the case of the gene. I have shown that genes are unlike any of the kinds that have been discussed as paradigm cases of natural kinds, such as “oxygen” or “acid”. The kind “gene” is relational, functional (or mixed-functional), variable, generic, and sortal. These properties, as I have shown, are toxic for reference fixism. There may be causal as well as descriptive elements involved when experimen
tal biologists attached the term “gene” to some class of unknown fac
tors, as Stanford and Kitcher and others have suggested, however, this causal-descriptive apparatus was never sufficient to pick out anything remotely resembling Lockean real essence, i.e., a molecular constitution or something of this sort. Reference of the term “gene” was floating; it changed with every new major model organism and investigate technique deployed. So reference fixism fails.

The theory of partial reference runs into the difficulty that it makes his
torical statements come out true that were known to be false by the relevant

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10 Ultimately, a causal element in reference-fixing will have to be involved to fence off meaning finitism. Note that my rejection of reference fixism does not commit me to meaning finitism, at least not in its full-blown form. There are many new instances of scientific concepts that are clear-cut and do not require a community choice (like in Kuhnian normal science, perhaps). But there are also new instances that require revision of the existing conceptual taxonomy (like in scientific revolutions, but not necessarily as radical).
historical actors (on McLeish’s disjunctive account). I have located this difficulty in the fact that, in biology, there is usually no smallest or most basic causally homogenous kind that could be responsible for a theory’s success in speaking truths. Thus, nothing stops such a semantic theory from assigning positive truth values to a motley of statements that may be true about some subkinds of a general kinds. So partial reference is counter-intuitive in biology on top of the other philosophical difficulties in faces.

I hope to have shown that we don’t need reference fixism or partial reference to account for the intuition that an area such as classical genetics discovered important truths. General claims made back then generalized only over parts of the domain of molecular genetics, that is, over subkinds of the contemporary gene concept. Some of these general claims ranged over the full extension of the term “gene” as it was used then. This kind of conceptual change, which I have termed floating reference, is different from partial reference in that there was no ambiguity in reference, and it is different from reference fixism in that there were substantial reference shifts associated with new developments in experimental techniques and with new model organisms. Floating reference provides a more adequate truth-conditional, realist semantics for biological science, while something like partial reference may be required for defending a realist semantics in physical science.

Finally, I have suggested that biological kinds are typically part of open classification systems that resemble biological taxonomy itself. Such systems admit lumping and splitting, as new specimens are discovered and new investigative techniques are developed. Nature’s biological joints are always in motion, and so is the language of those who try to carve them.

8. References


