ARTHRITIS AND NATURE'S JOINTS

NEIL E. WILLIAMS (University at Buffalo)
forthcoming: **MIT Press *Carving Nature at its Joints – Topics in Contemporary Philosophy V. 8.***

Abstract *The thought that diseases form natural kinds tends not to sit well with the essentialist treatment of natural kinds. The essentialist's candidates for the essences of diseases—etiological properties—rarely satisfy the essentialist's requirement that they be necessary and sufficient for membership within the kind. Consequently philosophers of medicine have tended to back away from treating diseases as natural kinds. However, this retreat was too hasty: there are good reasons for thinking that diseases form natural kinds. The problem lies with the essentialist treatment of natural kinds, and not with treating disease as forming natural kinds. A similar revolution has taken place within the species debate, where the notion of natural kind has been 'taken back' from the essentialist. Borrowing the revised treatment of natural kinds from the philosophers of species and modifying it slightly, I offer a proposed treatment of disease kinds in terms of homeostatic property clusters.*

1. DISEASE KINDS AND ESSENCES

It should come as a surprise to almost no-one that the thought that diseases constitute natural kinds does not generally sit well with the essentialist picture of natural kinds as championed by Kripke and Putnam in the 1970s.[1] According to that essentialist picture, in order for a class of entities to be a natural kind it is required that all and only members of the class instantiate some very specific property or properties, and that these properties explain the presence of any other properties typically associated with being a member of the kind. These privileged properties constitute the *essence* of the kind. In the case of diseases, the essentialist claims that the properties in question are etiological: the essence of a disease kind is whatever underlying physical condition causes the instances of the disease and the associated symptoms.[2]

There are well known examples of diseases for which the essentialist picture appears to work just fine, such as tuberculosis: originally identified and classified on the basis of the name-giving tubercle, the stereotyped "nominal" essence classification of tuberculosis gave way to "real" essence classification with Koch's

[1] The question here—and throughout the paper—is that of whether the instances of a *specific* disease type form a natural kind. That is, do all the instances of *x* (where *x* is some type of disease, such as rheumatoid arthritis, or tuberculosis) form a natural kind? This is not to ask whether all the various types of disease together constitute a single natural kind. For more on the latter discussion see D'Amico (1995) and Reznek (1987, 1995).

[2] See Putnam (1975: 241, 1975: 311) and Mackie (1976: 99) for statements to this effect. When essentialists speak of 'the' cause of a disease instance, what they really mean is 'that causal factor which stands out against the causal field'; any effect will have numerous causes, but we might think of 'the' cause as perhaps the most salient amongst them. For convenience I too will speak in terms of 'the' cause of a disease instance, with the understanding that it is the most salient amongst the causes. See Mackie (1965).

1882 discovery that the *Mycobacterium tuberculosis* bacterium was the cause of the disease. Alongside tuberculosis, the essentialist can list cholera, meningitis, plague, botulism, malaria, syphilis, and a number of others; but despite this list of disease types that fit the essentialist model, the "success" stories for the essentialist treatment of disease kinds have been rather limited.[3] For every disease type that appears to satisfy the essentialist desiderata there are a dozen or more that do not. For instance, contrast the case of tuberculosis with that of rheumatoid arthritis (RA). The American Rheumatism Association (ARA) presently defines an instance of rheumatoid arthritis as one displaying at least four of the seven stipulated diagnostic criteria.[4] The current classification of the disease is entirely in terms of its clinical picture. Not only is there no known cause, even the diagnostic criteria are only rough guides (recent studies report that more than seventy percent of those who test positive for rheumatoid factor may not have the disease).[5]

Optimistic essentialists might reply that diseases like RA are only pseudo-counterexamples to essentialism: essentialism is a *metaphysical* thesis concerning the constitution of natural kinds, and nothing about the case of RA shows that there is not some underlying physical cause which is present in all and only instances of RA. It would be fallacious to assume on the basis of our not *knowing* the cause of RA that there is no cause to be known. In fact, the ARA even stipulate that their understanding of RA is subject to revision in light of future understanding, and anticipate that future classification will be based directly on improved understanding of the underlying disease pathology. Perhaps all is well in the essentialist camp.

But what happens if RA fails to have a tidy causal structure, or has multiple causes? Even as we learn more about the biological details of the disease, the possibility of multiple causes does not get ruled out. Nor can we rule out RA having a cause that is also the cause of other types of disease.[6] Should it turn out that RA has many causes, no cause, or no cause unique to RA, then the essentialist is forced to tell us that RA is not a natural kind. Should RA turn out to have no cause at all, then it is not a disease type but a syndrome (a collection or pattern of symptoms lacking a joint cause). In fact, even in the heralded case of tuberculosis, it turns out that the disease has at least *two* causes (infection by *Mycobacterium tuberculosis* or infection by *Mycobacterium oivis*), so neither can count as the essence.[7] But the

---

[3] A common feature among the stock of success cases is that they tend to be diseases caused by an identifiable disease agent such as a virus, parasite or bacterium; diseases whose causes are immunological, intra-cellular, genetic, or deficiency based tend not to make the essentialist's list. The difference can be explained—in part—by the tendency to focus on disease treatment and prevention, combined with the relative ease with which we single out a disease agent as *the* cause in the success cases (from amongst the various causes at work in any given instance), in contrast to the difficulties experienced in the latter cases.

[4] These are: (i) morning stiffness; (ii) arthritis of three or more joint areas; (iii) arthritis of hand joints; (iv) symmetric arthritis; (v) rheumatoid nodules; (vi) serum rheumatoid factor; (vii) typical radiographic changes. Arnett et al. (1988).

[5] Newman et al (1996: 8).

[6] Given the huge variability in human genetic constitution, level of nutrition, state of immune system, environment and general health, it would not be at all surprising if similar causes are capable of producing instances of different disease types in different people. Should RA turn out to have no cause at all, then it is not a disease but a syndrome (that is, a collection or pattern of symptoms lacking a joint cause).

[7] I am assuming here that: (i) disjunctive essences are eschewed by essentialism, and (ii) all instances of tuberculosis, regardless of cause, admit of such overwhelming similarity that having two causes does not constitute an adequate reason for thinking that there are two kinds of tuberculosis. As I interpret it, essentialism requires that the causal role must be

problems for essentialism do not end there. In the purportedly successful cases where the causal agent has been identified, disease kind membership depends crucially on there being clearly delineated bacterial and viral kinds, and they too face problems of classification.[8] And even if those difficulties should be surmountable, it is now 130 years after Koch's discovery of the cause of tuberculosis and there remains bitter disagreement amongst medical practitioners about how the disease name 'tuberculosis' should be used, indicating widespread concern with the essentialist's picture of natural kinds.[9] Rather than continuing to consider ways that the essentialist picture of the metaphysics of natural kinds lets us down when it comes to disease kinds, or how our present knowledge and handling of diseases suggests something other than the essentialist's metaphysical picture of disease kinds, it should suffice to say that the essentialist's account of natural kinds looks ill suited for dealing with disease kinds.

The problem is that the essentialist picture provides us with only two options, and neither is desirable: (i) satisfy the essentialist desiderata by locating some etiological feature both necessary and sufficient for kind membership, or (ii) give up on the thought that similarity between disease instances can be understood in terms of natural kinds.

The facts about disease largely rule out the first option; at best only a small subset of the recognised disease types would qualify as natural kinds. This is unsatisfactory as it conflicts with the strong intuition that most of the disease types we identify pick out genuinely natural groupings of disease instances, an intuition that plays a pivotal role in determining the methodologies we adopt in investigating, treating, and preventing diseases, not to mention disease classification. Moreover, if only a very small class of disease types satisfies the essentialist desiderata, we must forego 'naturalness' as even a rough means for distinguishing genuine diseases from fabricated ones. That is, we run the risk of seeing all non-essentialist disease groupings as non-natural nominalisations, and so lose a relatively easy way of saying that many of the groups of symptoms we treat together we do so because they form a natural unit, whereas other groupings are of our own making.[10]

The second option is clearly no better; whereas the first option severely restricts the number of disease kinds, the second option rules them out altogether. As I have suggested, the thought that diseases form natural kinds is the theoretical foundation on which our methods of treating, investigating, and preventing disease

---

uniquely satisfied, and so cannot continue to be indicated vaguely along the lines of 'whatever happens to cause the disease' when we know exactly what *they* are. In this way disease kinds follow what Putnam says of 'jade' type cases: the discovery that instances of jade have *two* microstructures (jadeite and nephrite) producing the same unique textural qualities makes for two kinds, not one: "if H_2O and XYZ had both been plentiful on Earth, then we would have had a case similar to the jadeite/nephrite case: it would have been correct to say that there were *two kinds of* '*water*' (1975: 241).

[8] Franklin (2007).

[9] In practice its use remains ambiguous between definitions that treat the presence of tubercles as contingent or not, and that similarly treat having been caused by the tubercle bacillus as contingent or not. Flier and Robbé (1999). It is worth noting that disputes over the use of 'tuberculosis' do not directly undermine the essentialist's metaphysical treatment of kinds, but something about the essentialist picture is clearly unsatisfactory to a significant number of medical practitioners.

[10] There are also certain social issues that arise. Though largely fallacious, it is not uncommon for various institutions (legal, governmental, or financial) to confer on those diseases that are less obviously 'natural' a diminished status, and use this as a basis for similarly diminished resources.

are predicated. Without this foundation, our inductive and explanatory practices concerning disease are without grounding.[11] If we are to continue our practice of treating disease types as natural kinds—without unduly restricting the class of disease types—then I suggest that we have no choice but to abandon the traditional essentialist picture of natural kinds when dealing with disease kinds and replace it with a notion that is more disease kind friendly.[12]

In what follows that is exactly what I attempt to do. That is, I will propose a treatment of natural kinds suited for dealing with disease kinds. What I will propose is that disease kinds be interpreted as a specialized case of *homeostatic property cluster kinds* (HPC) of the sort proposed by Richard Boyd.[13] In the HPC account of kinds, natural causal processes produce stable groups of properties that contingently 'cluster' together, and the clusters determine kind membership. Consequently, part of the present task will be to specify the sorts of properties that are included in the property clusters. However, as diseases are not substances, treating disease kinds as HPC natural kinds will require modifying the HPC theory in what should be a welcomed direction.

Before I proceed I offer two words of caution: (1) in order to make the range of diseases slightly more manageable, the central focus will be on *human* diseases, and will exclude mental illnesses. I am optimistic that the account will be applicable to the latter group, but considering mental illnesses brings in a range of additional worries that are best left out of the present discussion. Likewise I expect the present treatment to apply to diseases in multi-cellular organisms in general, but I will nevertheless ignore non-human diseases.[14] (2) This is intended as a *metaphysical* account of what constitute natural kinds of diseases, and should not be interpreted as a guideline for diagnosis.[15] It is an interesting question how disease kinds relate to our attempts to diagnose what type of disease a particular patient might have, but this is (mostly) a distinct issue about the application of disease kinds to a specific clinical picture and is largely an epistemic matter, and one I lack the space to go into here.

The procedure will be as follows: in section 2 I continue my defence of the claim that we should treat disease kinds as natural kinds (and so therefore the lack of fit with the essentialist picture of natural kinds means we need a replacement

---

[11] Could we not just rely on artificial or conventional groupings of disease instances to support our practices (in the absence of natural groupings)? No—it is the 'naturalness' of natural kinds that justifies what Goodman dubbed 'projectability': the inference that the properties of a subset of the members of a kind would apply to the remainder (1954). Though this inference is not infallible, if a grouping is entirely up to us we have no basis for the inference at all. See Mill (1884: Book 4).

[12] Why replace the essentialist picture—why not just have *many* kinds of natural kinds? For the most part this is sage advice: a single notion of natural kind applied without revision to all the various disciplines and sub-disciplines that utilize natural kinds would contradict most of what the discipline would otherwise have taken to be natural kinds. However, when the domain is restricted just to disease, I can see no benefit to having multiple notions of natural kinds: this would undermine most of our medical practices and make the sharing of medical information a near impossibility.

[13] Boyd (1989, 1991, and 1999). See also Griffiths (1999), Millikan (1999), and Wilson (1999).

[14] I am also naively optimistic that the present treatment will be applicable to *botanical diseases*, but it would take someone with greater botanical knowledge than I possess to tell me if that is even remotely possible.

[15] A number of prima facie objections can be avoided by keeping this distinction in mind while considering what follows.

account). Section 3 contrasts essentialism with the HPC theory, providing a brief overview of each and the HPC framework that the present treatment adopts for disease kinds. As no treatment of disease kinds can be fully divorced from an account of what a disease is, section 4 provides an account of what diseases are. Section 5 contains the proposed treatment of disease kinds.

2. WHY TREAT DISEASE KINDS AS NATURAL KINDS?

If natural kinds are as the essentialist claims they are, then few or none of the disease types we recognise constitute natural kinds. I suggest that we resolve this incongruence by adopting an alternative picture of natural kinds. But why not just drop the notion and make do with something else? Hacking, for instance, states that the student of kinds will want a theory of kinds within which natural kinds "take their proper, rather limited place."[16] Most kinds, he thinks, are social rather than natural, and have more to do with their roles as tools than as classifications. Perhaps we should think of disease kinds in this way: membership in a disease kind is more a matter of our efforts to control our environment than of natural grouping. Something strikes me as remotely correct with this picture, but I cannot help but think that our disease oriented practices strongly favour the naturalness of kinds over the picture of disease kinds as mere tools. In fact, I am confident that naturalness is required for the ways we employ natural kinds in medical thinking. Let us consider then why we ought to treat disease kinds as *natural* kinds.

The first reason is simple: essentialism is but one way of approaching natural kinds. Essentialists clearly agree that disease kinds are natural kinds; rejecting the essentialist treatment of natural kinds does not require rejecting the shared intuition that disease kinds are natural kinds. That is, we can maintain the intuition that diseases form natural kinds without requiring that each disease kind has some property possessed by all and only instances of the kind, and which explains the presence of any other property associated with that natural kind. One gets a sense from much of the kinds literature (especially as it concerns diseases) that the essentialist picture of natural kinds is the *only* picture of natural kinds; but this impression is mistaken. The thought that there are kinds in nature is a basic and useful one, but nothing about this thought requires that to be natural the kinds must be kinds that satisfy the essentialist's desiderata.

A second reason for treating disease kinds as natural kinds is that this thought plays a significant role in determining and justifying how and why we go about our medical investigations in the ways we do. When we find a group of similar symptoms arising in a statistically significant number of instances, we treat that statistical significance as an indication that the group of symptoms are *unified*; that is, that they are all indicators of the same disease. We then engage in various activities that only make sense if we are treating that group as forming a natural group; that is, they only make sense if we are taking the collections of symptoms to hang together in virtue of mechanisms outside of our conceptual organisation. For starters, we try to find out what causes the symptoms. For each instance in which the symptoms arise we assume that there is some single salient cause of these symptoms. If we discover a cause in one instance, we then look for that cause in others. We might discover that the same group of symptoms has different causes in

[16] Hacking (1991: 109).

different instances, in which case we think of that group as having multiple potential causes. Or we might discover that some subset of the symptoms is highly correlated with some other cause, and come to treat that as a distinct disease. These are the actions of people treating diseases as natural kinds.

Nor do these actions end there. Prevention is about preventing the underlying process that produces a particular group of symptoms from arising; cure is a matter of locating the cause of these symptoms and attempting to arrest or reverse its effects; diagnosis is a matter of trying to match a patient's clinical picture with those clinical pictures associated with specific types of disease, despite what is typically impoverished data about which symptoms are present in a given case.[17] If we learn that a remedy is effective in a significant number of instances, then we continue to apply that remedy to further instances, and track its success. For instance, one of the SAARDs (slow-acting anti-rheumatic drugs) used in the treatment of RA is the injection of gold. That we would attempt a treatment in one instance because it has worked in others is a clear indication that we believe the instances are members of a naturally occurring group and so are inductions are justified; that we are successful as often as we are is a reasonable indictor that we are correct.[18]

What this boils down to is what Boyd has identified as a largely realist commitment to the causal structure of the world, and of a "deference to nature" with regards to which symptoms to take to be connected.[19] "Kinds useful for induction or explanation must always "cut the world at its joints" in this sense: successful induction and explanation always require that we accommodate our categories to the causal structure of the world."[20] It is in virtue of this causal structure that we have developed the understanding of disease that we have, and that we seek explanations in the way we do. We take treatments to be repeatable, and information gathered from one instance of a disease to be relevant to further instances of that disease, because we take similarity of disease instances within disease types to be a naturally occurring feature of our world. In short, we treat medical information as *projectable*, and we do so on the grounds that disease kinds are natural kinds.

By way of contrast, consider what would be the case were we not treating disease kinds as natural kinds. Though we would certainly recognise symptoms as arising naturally, we would not be annexing those symptoms to a single type. Remedial practices of clusters of symptoms would no longer be a matter of treating those symptoms *jointly*, but rather *severally*. This might still have some success, but not because some single cause or process had been stopped or reversed, but because numerous independent and unconnected processes had been stopped or reversed. This sort of difference parallels that which we find in uses of broad-spectrum antibiotics as opposed to narrow-spectrum antibiotics. In those cases where we have

---

[17] This picture of prevention, cure, and diagnosis is indicative of the ideal cases; that is, where we have a strong understanding of the disease type in question. In cases where we have limited understanding of a disease, or very poor information about a patient's condition, we are forced to take other forms of action, though I submit that even these other actions suggest that we are thinking of instances of diseases as forming natural kinds.

[18] This is not to claim that we get it right even close to most of the time, nor that our diagnoses are frequently on target. It is simply a claim about what is going on when we approach treatment, prevention, diagnosis, and so on, in this way, and what seems to be the case when we are successful.

[19] Boyd (1991: 140).

[20] Boy (1991: 139).

identified some specific bacterial infection as a (typical) cause of a disease, treatment proceeds via narrow-spectrum antibiotics that target just that specific type of bacterial infection. But where the cause is unknown (and the effects typically quick and fatal) broad-spectrum antibiotics are applied as a sort of anti-bacterial carpet bombing. If symptoms were treated severally and not jointly, then there would be fewer differences in how we understand the application of narrow-spectrum versus broad-spectrum antibiotics. To be clear, this might (unfortunately[21]) be all we are doing when we treat diseases, but it is not what we *think* we are doing, and that attitude is sufficient evidence of our treating disease kinds as forming natural kinds.

A final reason in support of offering an alternative picture of natural kinds for disease rather than just giving up on thought that disease kinds are natural kinds comes from the directly parallel response to essentialism with regards to species. Having argued that the essentialist picture of natural kinds did not sit well with any of the various biological classifications, biologists and philosophers of biology had originally backed away from treating biological kinds as natural kinds, and initiated a retreat from natural kinds in general.[22] But not long afterwards it was recognised that the natural kind concept was a fruitful and central concept in the biological sciences, and that the problem was with the essentialist picture of natural kinds, not with treating biological kinds as natural kinds. Consequently some biologists and philosophers of biology have started to 'take back' the notion of natural kind, offering alternative conceptions better suited to biological classifications.[23] After all, biological kinds—species in particular—are paradigmatic natural kinds; if the essentialist picture of natural kinds was ruling them out, then it was the picture that needed to change.

My recommendation is that a similar revolution be initiated within the medical sciences. As the essentialist picture of natural kinds is no more appropriate for disease kinds than it is for other biological kinds, the natural kind concept must be taken back and a new version devised that is better suited for medical science and disease classification.

3. TWO APPROACHES TO NATURAL KINDS

Like its essentialist predecessor, the proposed treatment of disease kinds is realist, and continues to be a metaphysical treatment of natural kinds, but is otherwise quite different. As I have said, the new approach is a specialised version of the HPC theory of kinds. In this section I will give a brief overview of the HPC theory.[24] This overview is presented against a backdrop of the essentialist view. I do this partly because it makes it easier to see how the two accounts differ, but also because there are additional features of essentialism we have not yet considered (concerning the

---

[21] This would be unfortunate because it would mean that even our successes in treating diseases would not provide as much information as they seem to.

[22] This is the response given in Dupré (1981).

[23] Boyd is leading this reclamation within the biological sciences; see his (1989, 1991, and 1999). Though I endorse the HPC account of natural kinds for some sorts of natural kinds, I remain neutral regarding the question of whether species should be understood in terms of HPC kinds.

[24] The account of the HPC theory that follows is a modification of that found in Boyd (1989, 1991, 1999). One notable difference is that the present version omits the conventionalist and constructivist undertones suggested in Boyd's formulation—undertones that tend to compromise the extent to which his HPC account is a realist account of kinds.

larger model of science in which essentialism is embedded) that are similarly inappropriate for dealing with disease kinds.

We have already seen the central theses of essentialism: they are that all members of a natural kind instantiate some very restricted set of properties (typically a single property), and that the having of this property is both necessary and sufficient for kind membership. This privileged set of properties constitutes the 'essence' of the kind. On the assumption that substances can be sorted into clearly demarcated classes in virtue of their instantiating (or failing to instantiate) these essential properties, the essentialist's kinds are likewise clearly demarcated, and are disjoint. Where a substance appears to be a member of two kinds, this can only be so if the one kind is a subset of the other. Hence, according to the essentialist, all kinds are parts of a single, immutable, hierarchical structure.

We have also seen that the essential properties will explain the other properties typically had in common by the members of the kind (referred to at times as the 'nominal' essence, 'stereotype', or 'surface properties' of the kind). The basic picture is this: members of a natural kind will tend to have numerous properties in common, but only a small sub-set is had by all, and fewer still make up the essence of the kind. Whereas the essential properties must be had by all members of the kind, the other properties will only *tend* to be had by all members—they will be had in most cases, but need not be had by all. In fact, essentialism is compatible with its being that case that no member of the kind has any of the surface properties. Consequently, the surface properties play no role in determining kind membership. What will be the case, however, is that the presence of the surface properties is *explained* by the presence of the essential properties. For instance, in the case of gold, common surface properties such as instances of gold being shiny, malleable, heavy, and so on, are to be explained via the essence of the kind—in this case being composed of atoms with the atomic number 79. How exactly this explaining takes place is not clear, but the general response has to do with the ways in which essential properties slot into the laws of nature. Presumably the having of essential properties in the right sort of law-like environment provides an account of why the surface properties are what they are. From here we begin to get a fuller sense of the essentialist's metaphysical picture: because the essential properties explain the surface properties through their interaction with the laws of nature, it follows that the essential properties will be categorical properties, and that they will tend to be intrinsic rather than relational. Consequently any dispositional properties we might find amongst the surface properties of the kind are to be similarly explained by the categorical properties in the essence; likewise for any relational properties.[25]

Moving onto the wider framework surrounding the essentialist picture of natural kinds, we find frequent reference to the roles of natural kinds within the system of natural laws. We have already seen that they connect via the essential properties, but it is also part of the wider framework that the laws of nature range over natural kinds. This gives the essentialist an additional criterion by which to assess whether a kind is natural or not: only natural kinds are appropriate subjects of the laws of nature.[26] Like the hierarchical structure of natural kinds, the natural laws

---

[25] The form of essentialism defended in Ellis and Lierse (1994) and Ellis (2001) is constructed directly in opposition to this wider account of laws, but they will be the first to admit that the common features of 'traditional' essentialism are as I have presented them, which is all I am interested in.

[26] This is not to suggest that a functional kind member like a table is not subject to the laws of nature, it simply does not obey those laws *qua* table. (Some other natural kinds, perhaps

are unchanging and exceptionless: the kinds and the laws mesh perfectly to form a seamless closed system.

The homeostatic property cluster (HPC) theory of natural kinds diverges in a number of significant ways from its essentialist counterpart. Where the essentialist demands determinateness and structure, the HPC theorist permits indeterminateness. Where the essentialist draws sharp boundaries, the HPC theorist sometimes draws sharp boundaries, but other times draws rough ones. The HPC theorist likewise tolerates change, and greatly inflates the number of properties involved in determining the kind. Let us start with this final difference. In place of the essentialist's restricted set of essential properties that are necessary and sufficient for kind membership, the HPC theorist suggests that there is a "cluster" of properties. Like the essentialist, the HPC theorist distinguishes purely accidental properties arising in the kind from those important to kind determination, including only the latter in the cluster, but the set of important properties is much larger than the very restricted set the essentialist uses for the same purpose. In a very rough sense, we might think of the HPC theorist's cluster as incorporating both the essentialist's essential properties as well as a number of the surface properties.[27] However, within the cluster no particular property is either necessary or sufficient for membership within the kind. What matters is the *extent* to which the properties a substance instantiates overlap with the properties in the cluster, which can be satisfied to varying degrees. The extent to which a substance is a member of a class or its complement will vary accordingly. It follows that HPC natural kinds are not required to have sharp boundaries or determinate cut off points between them, but rather can have ranges of peaks and valleys, where peaks indicate the highest degree of property overlap with the cluster, and valleys the least.[28] According to Boyd, this indeterminacy is incapable of revision; to insist on anything sharper would render the kinds "unnatural" as they would no longer correspond to their causal engagement with the world. In effect, a sharpening of natural kinds would make them artificial: they would be sharper than the world itself.[29]

As well as dulling the essentialist's sharp boundaries, the HPC theorist makes the metaphysical privilege the essentialist affords the essential properties a largely

---

those that constitute the table, are presumably at work: electrons or molecules or something of the sort).

[27] This is very rough indeed; HPC theorists (Boyd particularly) tend to give us very little indication of what exactly belongs in the cluster. I aim for greater clarity with regards to HPC disease kinds; see section 5 below.

[28] This is not to suggest to that HPC kinds are always fuzzy, or that sharp cut offs cannot exist in nature, only that sharp cut offs are not required for establishing kinds. More often than not HPC natural kinds will have fairly sharp boundaries; what matters—and what distinguishes them from the essentialist's kinds—is that sharp boundaries are not required, and that the kinds are shaped through natural causal processes.

[29] Boyd also suggests that the indeterminacy of HPC kinds is partly a product of our own inductive and explanatory practices through which *we* engage with the causal nature of the world. As these practices are themselves somewhat inexact (relying on generalisations from test scenarios, laboratory conditions, sampling, and so on), and themselves involve causal processes, Boyd claims that the kinds that emerge will be similarly imprecise.

I think that Boyd's suggestion here is overly constructivist, and threatens the naturalness of kinds that justifies our inductive and explanatory practices. It is therefore important to keep separate the disjointness that arises in our picture of the kinds from any disjointness that naturally arises from the interaction of substances with the world. The former is just good old healthy fallibilism, and if not separated from the latter compromises the realism of the HPC kinds.

epistemic matter. For our own purposes of determining whether a given substance is a member of this or that kind, we treat certain properties as more important than others, but this importance need not be reflected in the kinds themselves; the privileged properties are reliable *indicators* of kind membership, but they do not determine kind membership. The range of the kind reflects the full cluster of properties, not this or that property. In removing the metaphysical force of essential versus surface property distinction, the HPC theorist also removes the explanatory relation the essentialist takes the two to share. In its place we are told that the co-occurrence of the cluster as whole is explained by the causal mechanisms of the world: it is *those* properties that group together naturally, where 'natural' is understood causally. It is the cluster-producing effects of these homeostatic mechanisms that lend their name to the HPC account: the clusters are formed through a natural balance of environmental processes that act on members of the kind.[30] The property clusters are as they are because they reflect the causal structure of the world.

The next difference between essentialism and the HPC theory concerns the type of properties within the cluster. The cluster can include the intrinsic categorical properties the essentialist includes, but may also add dispositional and relational properties. The HPC theory adopts a permissive approach to cluster properties in general, not endorsing any particular metaphysics concerning the laws of nature. This is in direct contrast to the essentialist, whose account brings with it strong metaphysical commitments concerning the laws of nature and causal powers.[31] This demonstrates yet another point of departure: though the HPC theory is realist about there being a causal nature to reality, it remains neutral concerning its particular features. This allows it to be compatible with a range of different approaches to the laws of nature, including those that reject them outright.

A final departure concerns the ahistoricity of the essentialist's natural kinds. The essentialist imagines an immutable hierarchical structure. The HPC theorist, on the other hand, allows that kinds can, and do, change with time. On the assumption that species are amongst the natural kinds in biology and are subject to evolutionary changes, it stands to reason that the natural kinds in biology will change with time. The properties associated with a particular species of organism reflect the organism's place in the world, and are, in part, a reflection of the organism's interaction with the world. Internal mutations and changing environmental factors will produce changes within the species of organism, but need not be changes that constitute the appearance of a new species. As the species change under environmental pressures, so too must natural kinds; this variability within the species is accommodated through the flexibility of the property clusters.

Despite the many differences between the essentialist and HPC treatments of natural kind, it is important to note that both are similar in that each offers an *a posteriori* treatment of the knowledge of natural kinds. According to both theories, natural kind classifications are a matter of discovery; the natural kinds exist prior to—and independent of—our knowledge of them, and through our interactions with the members we may come to learn about the kinds. Essentialism is, at its most

---

[30] Boyd writes that: "the presence of some of the properties in F tends (under appropriate conditions) to favour the presence of the others, or there are underlying mechanisms or processes that tend to maintain the properties in F, or both" (1999: 143).

[31] I think it is fair to say that (traditional) essentialism carries with it a number of neo-Humean metaphysical commitments. The essentialism favoured by Ellis and Lierse (1994) rejects this aspect of essentialism.

basic, a metaphysically realist and robust account of kinds, and as such assumes that we start out in the dark regarding our knowledge of it. In this respect, HPC kinds are no different.[32]

The metaphysical picture the HPC theorist paints of natural kinds lacks the demand for sharp edges that the essentialist endorses: it permits rough edges, it allows colours to blend into one another, and its features are often obscured, but it is realist nonetheless. Whereas the essentialist offers a rigid and eternal structure of natural kinds integrated with the laws of nature, the HPC theorist's realism is centred on the more modest assertion that the world has some sort of causal basis, and that this will produce kinds. The causal features of the world are not always perfectly tidy, so it is no surprise the HPC kinds that emerge might be similarly dappled.

4. WHAT IS A DISEASE?

To be able to talk about what makes two disease instances members of the same disease natural kind, and therefore what constitutes a disease natural kind, it is imperative we have a working concept of disease. But deciding what a disease is—that is, defending a particular disease concept—is a substantial undertaking unto itself, and one I lack the space to fully undertake here. Consequently I will partly rely on the concept of disease I have defended elsewhere and present a brief and slightly revised version of it here.[33]

One important preliminary aspect of the disease concept I offer is that it is value-free.[34] According to the present account, calling something a disease does not depend on our having any presuppositions about what is good or bad for an organism; hence, the present account is a version of what is sometimes referred to in the literature as a 'naturalist' account of disease. It is naturalist in that the standards against which diseases are understood are statistically determined, where the negative effects typically associated with a disease type are not effects that *we* deem to be bad, but rather those that reduce the organism's ability to deal with environmental pressures. Why is a value-free treatment of disease important? It matters because some objectors are bound to insist that a value-laden notion of disease undermines the suggestion that diseases can be understood as natural kinds. The argument might be something like this: if what counts as a disease depends on

---

[32] The form of realism presented here for HPC kinds marks a departure from the realism Boyd depicts them as having. Boyd's understanding of 'realism' is captured by the following: "When we ask about the "reality" of a kind or of the members of a family of kinds—or when we address the question of "realism about" them—what we are addressing is the question of what contribution, if any, reference to the kind or kinds in question makes to the ways in which the classificatory and inferential practices in which they are implicated contribute to the satisfaction of the accommodation demands of the relevant disciplinary matrix" (1999: 159).

[33] See Williams (2007). A number of the concepts I use here in presenting the account of disease (homeostasis, cellular dispositionality, standard conditions) get much a fuller treatment there. Mention of 'homeostasis' as applied to cellular processes is not to be confused with the homeostasis that forms HPC kinds, though there is bound to be occasional overlap.

[34] That is, as value-free as any physical or biological science happens to be, in the sense of being independent of our evaluative judgements.

our judgements about what is a good or bad effect, or what is a normal or irregular process, then what counts as a disease is up to us as much as it is anything else, and therefore we cannot claim that what constitutes a disease kind can be given by nature, independent of our evaluative judgements. As it happens, the argument is fallacious: even if the extension of 'disease' depends on our value judgements it would not follow that specific diseases did not form natural kinds.[35] For those who remain suspicious, the easiest way to satisfy the objector is to make use of a value-free concept of disease, avoiding the worry altogether, as I do.

With regard to our present concerns, the central feature of the account is that it considers diseases in contrast with naturally occurring processes, primarily at the cellular level. Groups of cells form causally interactive groups—'networks'—that collaboratively manifest various homeostatic dispositions. The cells themselves possess a wide range of dispositions, some small number of which are routinely manifested in response to the cell's environment; that environment is comprised of other cells within the network and various chemicals. When environment is typical, the dispositions that are manifested are similarly typical, and they give rise to standard cellular processes.[36] That is, they result in the ebb and flow of molecules and energy, in and out of the cells, in the cycles of excess and shortage that we recognise as typical homeostasis. But not all the cell's dispositions are for these typical manifestations. Either as a result of problems within individual cells (cellular misinformation; DNA problems), or owing to environmental changes (chemical changes; increased or decreased energy; invasion of foreign bacterial or viral agents; chemical absences), cells will manifest other dispositions. In sufficient numbers, or if repeatedly manifested for a sufficiently prolonged period, the manifestations of these other dispositions give rise to processes that deviate significantly from the standard homeostatic processes of the cellular network. These distorted processes are disease processes.[37]

The previous paragraph suggests two responses to the question of what a disease is. The first is that a disease is a process; specifically, a process of cellular network interaction that deviates significantly from the standard, where that process—like the standard processes from which it deviates—is the manifesting of various cellular dispositions. According to this response, a disease type is to be identified with a type of disease process. The second response suggested by the above paragraph is that a disease is a set of dispositions, namely those dispositions whose manifestations result in a disease process. So which is it to be: are diseases sets of dispositions or processes?

My answer is that they are both. Or, more correctly, that some diseases are best thought of as sets of dispositions, whereas others are best thought of as

---

[35] Reznek (1987, 1995) argues that 'disease' does not name a natural kind, because he believes the concept to be value-laden; however, he nevertheless admits that individual diseases could turn out to be natural kinds.

[36] What counts as 'standard' is a statistical matter, determined within the relevant comparison classes. Here I follow Boorse (1975, 1977, 1997).

[37] For the sake of brevity I have passed over two other important conditions that are necessary for the distorted process to count as a disease process: the first is that the cellular network is incapable of remedying itself (without producing further distortions in other networks; and where those further networks are either incapable of self-remedy without distorting some further network, and so on), and the second is that the process tends to reduce the organism's ability to cope with environmental pressures.

processes.[38] The difference, I suggest, turns on the sort of dispositions involved and the circumstances of their manifestation. In the typical case—that which results in standard homeostasis—we have a subset of the cell's dispositions manifesting in response to a typical cellular environment. Here we can isolate two key components: the set of dispositions involved, and the conditions (circumstances) in which those dispositions are manifested. Call the set of *all* dispositions possessed by a typical cell S_A, and the typical circumstances in which the cell finds itself *c*. There is some subset of S_A, call it S_C, comprised of just those dispositions that are manifested in circumstances *c*, where the manifestation of the dispositions in S_C result in standard homeostasis.

I submit that a disease is best thought of as a process if it arises from a change in the conditions, such that some other subset of the cell's dispositions are manifested. Instead of the typical circumstances *c*, we have a different cellular environment—circumstances *x*—meaning that the subset of dispositions that are manifested is no longer S_C, but S_X, where S_X does not result in homeostasis.[39] The cell has not changed (these are all dispositions typical cells possess; they are members of S_A), but changes to the cell's environment have changed which dispositions are manifested. In cases where the triggering of these typical-for-the-cell-to-have-but-not-typical-for-the-cell-to-manifest dispositions result in a disease process, we have a case in which the disease in question is best considered a process.

On the other hand, a disease is best thought of as a set of dispositions in those cases where a disease process arises (if ever it does) from dispositions that are not typical for the cell to have.[40] That is, where the set of dispositions the cell possesses is not S_A. This can either be due to the addition of *novel* dispositions, or because of the *absence* of typical ones.[41] In neither case does it matter whether the conditions (cellular environment) are standard or not, as it is the presence (or absence) of the dispositions that matters for having the disease. However, as either change is bound to be more apparent if the dispositions in question are ones that manifest in typical conditions (circumstances *c*), we will only look at those cases.[42]

---

[38] My current understanding of 'disease' differs slightly from that I defended in 2007. Whereas I previously argued that the dispositions for the non-standard manifestations had to be manifested in order for the disease to be present in a person (and so diseases were to be identified with certain kinds of processes), I now believe that *some* diseases are such that the mere presence of certain dispositions, even if they are never manifested, suffices for the having of the disease, and so *some* diseases are dispositions. My revised view has been influenced by discussions with Barry Smith and Marc Lange (thanks to Marc for making pre-published material available to me; see Lange *forthcoming*).

[39] It is an empirical question how much S_X must differ from S_C before homeostasis is no longer maintained, the answer to which may plausibly vary for different cell types. For ease of exposition we shall assume the extreme case in which S_X and S_C are disjoint, and no disposition in S_C is such that it will be manifested in circumstances *x*.

[40] It is of course not required that the disease process does in fact arise, for in these cases having the relevant dispositions is sufficient for having the disease. But, it must be the case that the process that would result is a disease process, as outlined above.

[41] The absence of a disposition for *m* in some circumstances *e* is just to have a disposition for *some manifestation other than m* (and that does not include *m* as a part) in circumstances *e*; hence the absence of a disposition is just the having of some other disposition (for those same circumstances). In what follows I will take this as understood and speak of absences of dispositions as dispositions.

[42] Having novel dispositions for disease processes that only manifest themselves in atypical circumstances is still to have a disease, as is lacking dispositions that manifest in atypical

If we change S_C by *adding* to it novel dispositions (those not typically found in S_A) whose manifestations arise in circumstances *c*, then we can expect that with sufficient change to S_C (one or more dispositions added) that the resultant manifestations will no longer support homeostasis. In this case the disease is a matter of having certain atypical dispositions. On the other hand, we can get the same effect if we *remove* a sufficient number (one or more) dispositions from S_C, in which case the disease is a matter of lacking certain typical dispositions.

Some examples are bound to help. Classical phenylketonuria (PKU) is a genetic disease characterised by the inability to metabolise phenylalanine (phe), an amino acid found in breast milk, bread, meats, potatoes, and numerous other common foods. The inability comes from a mutation of the gene for phenylalanine hydroxylase (PAH), an enzyme required for metabolizing phe.[43] Because of the enzyme deficiency, the ingestion of phe results in increased levels of phe in the bloodstream, leading to such symptoms as impaired cognition and microcephaly. However, by maintaining a lifelong phe-free diet, persons with the PAH enzyme deficiency can avoid this process and the associated symptoms. Nevertheless, despite avoiding the process and the symptoms, people with the enzyme deficiency have the disease.

PKU is a disease type best thought of as a set of dispositions. Most notable within that set are the inability to produce sufficient PAH and the inability to metabolise phe. Combined with the conditions arising from normal diet, these inabilities would lead to a disease process and various symptoms, but it is the absence of these two dispositions that is significant for having PKU. As Lange rightly asserts, phenlyketonurics maintain phe-free diets because they *have* the disease, not to avoid it.[44] For those who need further convincing, consider a hypothetical therapy involving the use of symptom-suppressing drugs. Though a successful drug regimen can halt a disease process and prevent the production of symptoms, no-one would consider such a treatment a *cure* of the disease. The organism is still diseased, even if the drugs successfully block or counter the symptoms, as would be apparent if we stop administering the drugs. In the case of PKU the disease process is prevented through a strictly controlled diet, but with a regular diet the problems would surface. A phe-free diet is not a cure for PKU: no disease process is required for the disease to be present.

Contrast the situation with PKU with that of scurvy. In humans, ascorbic acid (vitamin C) is required for the enzymes that synthesize collagen to operate. As collagen is the most abundant protein in the human body (found in tendons, artery walls, skin, bones and so on), this is obviously a vital process. Nonetheless, humans do not produce ascorbic acid, and so require diets rich in vitamin C (found in many fruits and vegetables). As foods high in vitamin C are part of most typical diets, cases of persons significantly deficient in vitamin C are rare. But, if and when this occurs, the deficiency results in a disease process that can involve such

---

circumstances, but on the assumption that things are typical most of the time, these diseases are far less likely to get noticed.

[43] Lange (*forthcoming*) discusses PKU at length, arguing that PKU is an incapacity (a type of disposition), not a process. Lange considers only PKU, asking what sort of disease concept suits it best, but makes no claim about other disease types. As far as I can tell, the dispositional concept does not apply to all disease types: some are best thought of as dispositions, others are best thought of as processes. That said, all disease types will involve dispositions and have associated processes and symptom; the difference is whether or not the process must be present to be an instance of the disease.

[44] Lange (*forthcoming*).

symptoms as spots on the skin, teeth loss, and bleeding from mucous membranes, and in all cases is fatal if not treated.

Despite the similarities between PKU and scurvy (deficiency; control through diet), scurvy is a disease type best thought of as a process. This is the case even though scurvy results from a vitamin deficiency and various cellular dispositions. And the reason is simple: persons that do not produce ascorbic acid but maintain diets rich in vitamin C *do not have scurvy*. Only if one's diet is atypical—perhaps due to a lengthy trip at sea without adequate means for storing fruits and vegetables, when the lack of ascorbic acid means that dispositions typically manifested (collagen synthesis) are no longer manifested—does one have scurvy. If the process has not been initiated, then scurvy is not present.[45] Likewise, unlike PKU, scurvy is *cured* if the process is stopped (which can be achieved by consuming a diet rich in vitamin C). Scurvy is a disease type best thought of as a process.

This gives us the main picture regarding the ontology of disease, but two lesser details are worth pointing out. The first is that even in cases where the disease type is identified with a process, that the process occurs does not require or guarantee that the associated symptoms will result. Symptoms, though part of a disease process, are not to be identified with the process. The relationship between symptom and disease process is that of part to whole; the appearance of a symptom may be one terminus of a disease, or it can be one of the stages of the disease process. Consequently it is possible to have certain parts or stages of the disease process that occur in the absence of symptoms. Additionally, as most of us would expect, even if a process is allowed to continue, different conditions within the diseased individual can mean that only some symptoms appear.

The second detail is that we need to distinguish diseases from other medical conditions that are sometimes spoken of as if they were diseases. These conditions are what I call 'disorders'. Disorders are purely *structural* deviations from the standard: broken arms, cataracts, hernias, and so on. These are all medical conditions that may require treatment, sometimes even immediate and extensive treatment, but they are not diseases. They are states of the organism that differ significantly from the canonical structure in terms of topography, shape, proportion, and number. Disorders will not figure directly in the account of disease kinds (it is their exclusion that is relevant); I mention them because disorders can sometimes be partial causes of diseases, and can often be symptoms, and to make clear that to be a disease is not simply a matter of being something that gets or requires medical attention or treatment.[46]

Though I have offered two accounts of disease types in terms of sets of dispositions and processes, it is not that case that what determine disease kinds will differ much between the two. In fact, we shall see that there is a good deal in common terms of the kinds and how they are constituted. With a basic picture of what it is to be a disease in mind, we can turn to the proposed treatment of disease natural kinds.

---

[45] What of the reply, on behalf of someone who takes all diseases to be dispositions, that we all have scurvy all the time? The answer is that this is a desperate move, and the kind of thing one would only suggest if in the grips of a theory. The rest of us know better; I do not have scurvy, nor do I suffer from numerous other diseases (and death) that would arise were I to stop eating.

[46] Consequently it is clear that definitions of 'health' that treat it as merely the absence of disease are way off the mark.

5. NATURAL DISEASE KINDS

I have suggested that essentialism runs into problems when dealing with disease kinds, and, because it is desirable to treat disease types as natural kinds, we should look for an alternative account of natural kinds. The proposed alternative is a version of the HPC theory of natural kinds according to which kind membership is a matter of instantiating some or all of some set of properties. Having provided a basic understanding of what a disease is, I now want to put forward a recommendation as to which properties matter for the determination of disease kinds; that is, what sort of properties make up the clusters for disease kinds.[47]

Before we can do this, there is a prior issue that must be considered. Both disease concepts I have offered make extensive use of the notion of a disease process. However, the natural kinds literature focuses almost exclusively on natural kinds of substances, so extending talk of natural kinds to processes is something of a departure. Consequently, something must be said about the treatment of processes as natural kinds. In doing so not only will it become apparent that treating processes as natural kinds is a welcome development of natural kinds theories; it will also help to highlight which properties are relevant to the determination of disease kinds (which properties make up the clusters), and why the two concepts of disease do not require separate treatments when looking at disease kinds.

Despite the prevalence of substance-based examples in treatments of natural kinds (essentialist and HPC accounts in particular), applying the natural kind concept to processes is less of a departure than it initially seems. Ellis and Lierse agree.[48] In their brief discussion, Ellis and Lierse (and later Ellis alone) claim that processes form natural kinds, and that this is in accord with the natural kind tradition. Ellis provides the following examples: physical processes like chemical reactions, radioactive decay, and osmosis; and biological processes like meiosis and mitosis.[49] Moreover, it is clear that the ways in which we study these processes, as well as track them, learn about them, and attempt to control and manipulate them, demonstrate our treating them as natural kinds. Even Mackie, in his discussion of Kripke and natural kinds, speaks straightforwardly of natural kinds of processes, and suggests that he thinks of disease types as natural kinds of processes.[50] I submit that

---

[47] I will say little about the homeostasis that produces and maintains the property clusters. The basic picture is one of cellular processing and of interactive networks of cells, in addition to various environmental conditions. The ways these operate within the body provide the conditions that support disease. For more on these processes and their connection to disease see Williams (2007).

[48] Ellis and Lierse (1994) and Ellis (2001). Despite sharing their enthusiasm for natural kinds of processes, I reject their essentialist treatment of them.

[49] Ellis (2001: 162).

[50] He says first that "[i]f our archetypes or typical specimens of rusting are in fact oxidation of iron, then if any process, however superficially like rusting it was, were not the oxidation of iron it would not be rusting," and then following his claim that the essence of malaria is the malarial parasite and the essence of measles the measles virus, that "[w]hat such examples show is that it is not the difference between substances and non-substances that matters here, in the sense of the distinction between items which are supposed to 'subsist by themselves' and items which are not, but rather the difference between cases where it is useful or fruitful to think and speak preferentially of a possibly unknown or inadequately known 'nature'." Mackie (1976: 99 and 100).

treating processes as natural kinds is not only compatible with standard natural kind notions, it is part of them.

Nevertheless, it might be objected that processes "are really just the ways in which things of different kinds are bound to behave, given their circumstances and the laws of nature," such that kinds of processes just supervene on substances and the laws of nature, and should not be treated as kinds in their own right.[51] For instance, Sulmasy adopts just this sort of approach in his understanding of disease types, arguing that diseases are not themselves natural kinds, but are *had by*, and sometimes *caused by* natural kinds.[52] Ellis's response to this line of objection is to claim that the objection would only stand if it could be shown that the processes are independent of the intrinsic behavioural dispositions of the substances, and if it could be explained why the laws of nature discriminate between substances as they do. I do not think we need go nearly that far. I cannot see why, even if a process is nothing over and above a substance's obeying certain laws of nature, this should preclude its being a natural kind. That processes depend ontologically on substances, and perhaps only arise in accordance with the laws of nature, is orthogonal to the question of whether they form natural kinds: nothing about being a natural kind demands the members be fundamental existents. Consequently I cannot take seriously the thought that there cannot be natural kinds of processes.

What is it then for two processes to be of the same kind? An obvious suggestion is that they follow the same pattern: they should have a similar initiating cause, follow a similar progression, and result in some similar outcome. We would have few problems treating two such processes as processes of the same kind. But it would be asking too much to demand that any two instances of the same kind of process must run their full course. Two processes can be members of the same process kind even if one should fail to run to completion. Consider a simple chemical example: a small sample of gold is submerged in an adequately large quantity of *aqua regia* and begins to dissolve. Given sufficient time the gold will go into solution. Call the process that would occur if the gold and solution were left until the gold had fully dissolved the 'completed' process. Now imagine a similar case in which the procedure is interrupted, and the (now slightly smaller) sample of gold is removed while there is still something left of it. This is surely the same kind of process as the completed one, only now it has been cut short. So what makes it the same? First, that it has a similar initial cause (immersion in *aqua regia*); second, that it has at the outset a similar set of dispositions (concerning the gold's solubility and the reciprocal dispositions of the solvent) regardless of whether they are manifested or not; and third, it has a similar terminus (no more solid gold). In the completed process the dispositions in question are all manifested; in the truncated process only some are, but the rest are present and *would have been manifested* were it not for the interruption.[53] Likewise, the end result is what would have occurred without the interruption.

---

[51] Ellis (2001:163).

[52] Sulmasy (2005). For the most part we can ignore the details of Sulmasy's account; for starters, he claims that diseases are states of affairs, which I reject, but more importantly his argument against treating diseases as natural kinds is that they lack essences. Naturally I agree, but endorse an altogether different response.

[53] For the sake of convenience I am overlooking all of the difficulties that arise when speaking counterfactually about dispositions and their manifestations. I should not, however, be interpreted as suggesting a counterfactual analysis of dispositions; I am merely pointing out what, *ceteris paribus*, would tend to occur.

Another example—perhaps even more familiar than the last—is the biological process of human pregnancy. It has a well known set of initial causes (you can do it the old fashioned way or enlist the aid of a fertility specialist); it manifests certain dispositions and produces new ones; it has a typical course that runs around nine months, and ends with the appearance of a tiny human. That is the 'completed' process that we typically desire—but shorter processes, without the desired outcome, nevertheless constitute processes of the same natural kind. Notice how easily we recognise members of this natural kind, despite the different ways that the process can be initiated and the variety of ways in which the process can unfold.

As biological processes, disease processes follow a similar basic pattern to that we find in the case of pregnancy. A disease process has some sort of initiating cause (either the triggering of atypical dispositions by atypical circumstances, or by removing or adding to the subset of dispositions triggered by typical circumstances), it has some course that takes place within the organism, and if uninterrupted will result in some set of symptoms. And just as we recognise that pregnancies can unfold in a variety of ways without failing to be members of the same process kind, disease processes can develop in a variety of ways—including abrupt interruptions and successful pharmaceutical efforts to stop them—and still be members of the same disease process kind. And though there are many ways that a particular kind of process might unfold, experience teaches us that the processes we treat as natural kinds tend to proceed along inductively reliable lines. This speaks to the homeostatic unity of the properties involved, and to the naturalness of the ensuing cluster of properties.

To repeat, two processes do not fail to be of the same kind just because one is cut short. But there has to be a bottom limit to just how short an instance of a process kind can be, and still be counted as a process at all. I lack any good sense of just where that line ought to be drawn, but it hardly matters. What matters is the set of dispositions that are manifested and will continue to manifest if untreated. In short, when it comes to comparing disease instances, what matters is the set of dispositions involved. And this is why—when thinking of disease kinds—it ultimately makes little difference if the kind falls under the process concept or the set of dispositions concept: in both cases what matters is the set of dispositions involved.

Considering potential disease processes brings to light three ways in which instances of disease processes can be treated as similar: the first concerns the cause, the second the set of dispositions that would manifest themselves in the disease process, and the third involves the symptoms arising during (and at the end of) the disease process. All three ways lend themselves to specific property types and will be applied in the determination of disease kinds. That is, all three types of property will be included (one way or another) in the cluster of properties that determine disease kind membership. Let us take a closer look, starting with the causes.

Central to the essentialist's account of disease kinds is the thought that causes of diseases are involved in determining disease kinds. In at least this much the essentialist is surely correct: differences in causes can often mark the boundary between different disease natural kinds. As we have seen, no specific cause is necessary for a given disease kind, as many diseases have more than one cause (recall that tuberculosis can result from infection by *Mycobacterium tuberculosis* or infection by *Mycobacterium oivis*); conversely, many causes are not sufficient either (infection of an immunized organism will not result in a disease process). Therefore, though we should follow the essentialist in treating etiological features of diseases as important to determining disease kinds, we must be more liberal in their inclusion. One way to do this is to include within the cluster of properties all of the potential causes of a

disease (note that this will still be a very limited set of causes). This would capture all of the right instances.

Hence etiological features will figure importantly in the property clusters that define kinds. These properties—perhaps best construed as relational properties—will vary slightly between the two disease concepts. Where the disease type is identified with a type of process, the salient aspects of the cause are the conditions (circumstances) that trigger the atypical dispositions. Recall that the dispositions in question are not unusual for the cells and cellular networks to possess, but that it takes atypical conditions for these dispositions to be manifested. These will tend to involve changes in the cellular environment, either because other cells in the network are acting differently, or because there is an increase or decrease in this or that chemical in the cellular environment.

For those types of disease that are understood as sets of dispositions, the casual properties are those responsible for the presence or absence of the typical dispositions. Unlike the process diseases, what matters for dispositional disease types are deviations from the set of properties typically possessed by the cell and cellular networks; whatever affords this difference is the causal property we are interested in. In many cases this will be a genetic issue: right from the get go various cells may have an atypical complement of dispositions owing to genetic inheritance. But it can also be the case that cells that start out normal are changed somehow, perhaps via environmental insult or lesion. These are the causes we are interested in.

The next group of properties included in the cluster are the dispositions for the disease process. We have seen that the essentialist eschews the thought that surface or stereotype properties be used to determine kind membership. The essentialist argument against the inclusion of such properties relies on the possibility of members of the kind that fail to exhibit one or many of the surface properties associated with the kind. Samples of gold, for instance, can fail to be shiny or yellowy, hence surface properties are not necessary for membership in the kind. Nor are they sufficient: fool's gold exhibits the characteristic properties of gold, but is not a member of the kind. In the case of disease kinds, essentialism requires that the set of signs and symptoms associated with the disease type not to be included amongst the set of properties that determine the natural kind. For instance, Putnam writes: "we are prepared to classify sicknesses as cases of multiple sclerosis, even if the symptoms are rather deviant, if it turns out that the *underlying condition* was the virus that causes multiple sclerosis."[54]

Contrast this picture with that we find we find in contemporary disease classifications. If one inspects the World Health Organisation's most recently adopted version of the International Classification of Diseases (ICD-10, formally endorsed by the WHO in 1990), one finds that one of the primary classificatory criteria is symptom based.[55] When it comes to practice, and for practical purposes,

---

[54] Putnam (1975: 311).

[55] The ICD-10 can be accessed at: http://www.who.int/classifications/icd/en/. As Kendell (2001) points out, the ICD-10 classifications are steeped in convention, to the extent that the same disease type is classified as a 'different' disease type if more than one specialist treats it within his or her specialty. However, the inclusion of symptoms in determining disease kinds does not demand such conventionalism, and I suggest it be avoided.

Ironically, amongst the "diagnostic issues to consider" presented to the workgroups developing the ICD-11, it is stated that for the "[c]lustering of signs, symptoms, and operational characteristics," the workgroups are to try to "identify the features that are necessary and sufficient to define the disease/disorder" (Üstün, Jakob, et al. 2007). I chalk

the essentialist criteria are not adhered to. As one might expect, displaying most or all of the symptoms associated with a particular disease kind is strong evidence that the disease in question is a member of the kind. After all, let us not forget that the symptoms we associate with a disease tend to be found together as a result of natural processes; they are not randomly collected, accidentally grouped, or arising individually. Distinguishing diseases according to their associated symptoms is clearly both beneficial and desirable. What should be done?

Fortunately, in rejecting essentialism we also reject the essentialist's insistence that the only properties that determine kind membership must be instantiated by every member of the kind. In the essentialist's thought experiment we are asked to imagine a member of the kind that fails to exhibit this or that surface property. The essentialist takes this as evidence that the property in question is not important for determining membership in the kind. But all it actually shows is that the property in question is not *essential* for being a member of that kind. It does not show that it is not a frequent or important indicator, nor does it show that only essential properties should be used to pick out the kind. In the debate between the 'surface' properties and the 'essential' properties, the HPC kinds theorist is not forced to choose—all such properties can, and should, be included, at least to some extent.[56]

However, consider a disease process that is cut short, perhaps one that is cut off at a very early stage indeed, and well before any of the diagnostic symptoms has arisen. For instance, imagine that we have an instance of RA, but that before the disease can lead to any joint discomfort and so on, a wonder cure is ingested. We have already said that a disease process does not need to be very long to count as a disease process, so this case will count.[57] But there are no symptoms. Hence, if symptom-type categorical properties were required for kind membership, then their absence in this case would mean that this was not an instance of RA, contrary to what we have imagined. But I submit that this is an instance of RA, and a central one. One wonders what we were doing administering the wonder cure if it was not (assume that the wonder cure is incredibly expensive and in short supply).

Consider a second case, wherein the RA disease process has gone much further and some symptoms start to arise. Lacking the wonder cure, a slightly less wonderful symptom-blocker is administered. The dispositions remain, and perhaps that portion of the disease process that does not include the symptoms continues (if there is any such part of the process), but the patient suffers no obvious ill effects. But beware, if her insurance runs out the symptoms will return—this is *not* a cure! This case has a few more symptom-type categorical properties we can point to, but still too few if all the symptoms are required to be a member of the kind. And yet it seems as central an instance of the disease process as any (or what were we preventing?).

---

this up to residual essentialist thinking, and conflating the privileging of certain criteria for diagnostic purposes when they are more reliable indicators—an epistemic matter—with that of metaphysical kind determination.

[56] This is particularly beneficial in the case of syndromes, where the underlying cause is unknown, as is the case with RA. Assuming the syndrome does in fact have some as of yet unknown etiology, what we have is a cluster of symptoms that arise together as a result of the causal nature of the world. It is precisely for this reason that we group the symptoms together as a syndrome and treat them jointly, rather than treating them severally.

[57] I am assuming that RA is a disease type of the process variety. If this is false, then the relevant dispositions need not be manifested at all.

As a final example consider two different instances of RA, where each instance displays exactly four of the seven ARA diagnostic symptoms such that the two have only one symptom in common. These ought to be two 'peak' instances of RA, but the reduced number of symptoms and the lack of overlap suggest that at most one can be a peak instance, and that one or both are at least part way down the hill. This is yet another version of the same problem.

The solution is to reject the thought that symptoms contribute to the determination of disease kinds, opting instead for properties that even the non-symptom cases generally possess: the *dispositions to produce the symptoms*. These are present—at least in one form or another—in even the shortest instances of the disease process, and in the set of disease types that fall under the set of dispositions concept.

That is not to suggest that the dispositions in the cluster are all dispositions that could immediately manifest themselves in symptoms, as some of the immediately-for-symptoms dispositions will be such that other dispositions must be manifested before they can then be manifested (these are 'higher order dispositions'[58]), but through the more basic dispositions they will help to determine kind membership. If the disease process is initiated, and left to run its course, the symptoms will arise. Of course, other factors concerning the general health of the host organism will dictate the extent and sometimes severity with which the dispositions for symptoms manifest, but these are differences that the account of disease kinds is right to ignore. I submit that most of the differences between instances of the same disease kind, such as those in the four-out-of-seven RA case, can be explained by differences specific to the patient. In sum, by having dispositional properties in the cluster, we are not forced to treat the above problem cases as non-central instances of the kind, and we can maintain the role of symptoms in determining disease types (even if only indirectly[59]).

As was suggested above, I suspect that dispositional properties play an important role in any natural process kinds, diseases or otherwise. Consider the pregnancy case: the woman who has recently conceived shows almost no signs of the process that has begun. But the process has begun, and a series of dispositions are present that may be manifested in due time. Of course, there is no guarantee that these dispositions will be manifested, as they can only arise if the correct conditions for their manifestation obtains, but the potential is there. As the process unfolds, some of these dispositions will be manifested (more correctly, the unfolding of the process *just is* the manifesting of these dispositions), and new dispositions will be generated.

The moral here is that most of the properties in the cluster will be dispositional in nature. They are dispositions for disease processes, and therefore include dispositions for the symptoms associated with the disease. These are the most important properties for the determination of disease kinds.

The final class of properties within the cluster are relations that pertain to the primary location of the dispositional properties that give rise to the disease process (if or when it is initiated), where location is understood in two different ways.

---

[58] For more on higher order dispositions and their role in possibility see Williams and Borghini (2008).

[59] This assumes—uncontroversially, I should think—that the presence of a symptom indicates that the disease previously had (and may continue to have) the disposition to produce that symptom. Hence symptoms serve as guides—very good guides—for the dispositions that are (or were) present.

The first of these concerns the particular site within the organism where the dispositions are located, and where the disease process tends to occur. That is, we get distinct disease kinds roughly on the basis of which organ or organ system the process occurs in. No disease process will ever be entirely contained within one area—disease processes engage with the organism's other processes (for example, passing infection through the bloodstream), and some disease types have no specific location at all—but for many types there tends to be a primary site. In cases where a disease process spreads, distinctions will tend to be a feature of where the process begins; that is, those cells or networks that have the dispositions in question. This could be a single organ, an organ system, a part of an organ, or even the organism as a whole. Hence, though they are similar in a number of respects, we make a distinction in kinds between such diseases as bladder cancer and lung cancer.

For certain disease types it might turn out that the inclusion of locational properties is redundant. This will be the case when the set of dispositions already reflects the locale where they are found. For instance, it may be that the disposition to *x* is only found at some specific location *l*, removing the need for additional properties that spell out the location. Hence we might be able to make do without this third category of properties in the cluster. However, as there is at least the logical possibility of disease kinds that perfectly overlap in terms of their initiating causal relations and dispositions, but differ only in location, the locational properties are best included, even if they might duplicate information already contained within the cluster.

Like the first, the second locational aspect is something that might already be contained with the dispositions in question, but which we shall include regardless. This second notion of location concerns the class against which the standards are determined. This will include coarse-grained distinctions about the ages of the comparison class of the diseased organism, as well as the environment of the comparison class. As might be expected, the latter plays some role in determining such things as diet, pollutants, stress, and so on, that affect the class, and therefore have some effect on what are the standard processes in which the body engages. The former concerns a comparison with regards to general age group; something to the effect of child, adult or geriatric. The point here is similar to that made above: because diseases are dependent entities, partly constituted by the cellular processes of the organism that bears them, significant differences in the nature of the host organism make for similarly large differences in the diseases themselves. And as the healthy cellular processes differ across these wide age groups and regions, so too will the disease kinds.

6. CONCLUSION

I suggest that this is how we should think of natural disease kinds. Following Boyd, it is important that we allow room for imprecise boundaries, and not attempt to sharpen them artificially, even if most of the time they are naturally disjoint. The human body is a vastly complex system, often lacking in sharp cut offs, and slowly but surely changing over time. Consequently it would be foolish not to permit a small amount of variation and indeterminacy. In this way we can preserve our intuition that diseases constitute natural kinds.

REFERENCES

Arnett, F, Edworthy S, Bloch D, et al. (1988) "The American Rheumatism Association 1987 Revised Criteria for the Classification of Rheumatoid Arthritis". *Arthritis and Rheumatism*, 31: 315-324.

Boorse, Christopher (1997) "A Rebuttal on Health". *What is Disease?* . Humber and Almeder (Eds.). New Jersey: Humana Press, 3-134.

Boorse, Christopher (1977) "Health as a Theoretical Concept". *Philosophy of Science*, 44: 542-573.

Boorse, Christopher (1975) "On the Distinction Between Disease and Illness". *Philosophy and Public Affairs*, 5: 49-68.

Boyd, Richard (1999) "Homeostasis, Species, and Higher Taxa". *Species – New Interdisciplinary Essays*. Wilson, R (Ed.). Cambridge: MIT Press, 141-185.

Boyd, Richard (1991) "Realism, Anti-Foundationalism and the Enthusiasm for Natural Kinds". *Philosophical Studies*, 61: 127-148.

Boyd, Richard (1989) "What Realism Implies and What it Does Not". *Dialectica*, 43: 5-29.

Caplan, A L, Engelhardt, H T Jr., and McCartney, J J (Eds.) (1981) *Concepts of Health and Disease – Interdisciplinary Perspectives*. London: Addison-Wesley.

D'Amico, Robert (1995) "Is Disease a Natural Kind?". *Journal of Medicine and Philosophy*, 20: 551-569.

Dupré, John (2002) *Humans and Other Animals*. New York: Oxford University Press.

Dupré, John (1981) "Natural Kinds and Biological Taxa". *The Philosophical Review*, 90: 66-90.

Ellis, Brian (2001). *Scientific Essentialism*. Cambridge University Press, New York.

Ellis, Brian, and Lierse, Caroline (1994) "Dispositional Essentialism". *Australasian Journal of Philosophy*, 72: 27-45.

Flier, F, and De Vries Robbé, P (1999) "Nosology and Causal Necessity: The Relation Between Defining Disease and Discovering its Necessary Cause". *Theoretical Medicine and Bioethics*, 20: 577-588.

Franklin, Laura (2007) "Bacteria, Sex and Systematics". *Philosophy of Science*, 74: 69-95.

Goodman, Nelson (1954). *Fact Fiction and Forecast*. Harvard University Press, Cambridge.

Griffiths, Paul (1999) "Squaring the Circle: Natural Kinds with Historical Essences" *Species – New Interdisciplinary Studies*. Wilson, R (Ed.). 1999. Cambridge: MIT Press, 209-228.

Hacking, Ian (1991) "A Tradition of Natural Kinds". *Philosophical Studies*, 61: 109-126.

Kendell, R.E. (2001) "The Distinction between Mental and Physical Illness". *British Journal of Psychiatry*. 178: 490-493.

Kripke, Saul (1980) *Naming and Necessity*. Cambridge: Harvard University Press.

Lange, Marc *(forthcoming)* "The End of Diseases". *Philosophical Topics*.

Mackie, J L (1976) *Problems from Locke*. New York: Oxford University Press.

Mackie, J L (1965) "Causes and Conditions". *American Philosophical Quarterly*. 2: 245-264.

Mill, John Stuart (1884) *A System of Logic*. London, Longmans.

Millikan, Ruth (1999) "Historical Kinds and the Special Sciences". *Philosophical Studies*. 95: 45-65.

Newman, S, Fitzpatrick, R, Revenson, T, et al (1996) *Understanding Rheumatoid Arthritis*. New York: Routledge.

Putnam, Hilary (1975) *Mind, Language and Reality*. New York: Cambridge University Press.

Reznek, Lawrie (1995) "Dis-Ease about Kinds: Reply to D'Amico". *Journal of Medicine and Philosophy*, 20: 571-584.

Reznek, Lawrie (1987) *The Nature of Disease*. New York: Routledge.

Sulmasy, Daniel (2005) "Diseases and Natural Kinds". *Theoretical Medicine and Bioethics*. 26: 487-513.

Üstün, T, Jakob, R, et al. (2007) "Production of ICD-11: The Overall Revision Process". Accessed through WHO website at: http://www.who.int/classifications/icd/ICDRevision.pdf

Williams, Neil E, and Borghini, A (2008) "A Dispositional Theory of Possibility". *dialectica*, 62: 21-41.

Williams, Neil E (2007) "The Factory Model of Disease". *The Monist*, 90: 555-584.

Wilson, Robert (1999) "Realism, Essence, and Kind: Resuscitating Species Essentialism?" *Species – New Interdisciplinary Studies*. Wilson, R (Ed.). 1999. Cambridge: MIT Press, 187-207.