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Intrinsic Kinds in Internal Medicine

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Abstract

What do we mean when we diagnose a patient with a disease? What does it mean to say that two people have the same disease? In this paper, I argue that diseases are natural kinds, using a conception of kinds derived from John Stuart Mill and Ruth Millikan. I demonstrate that each disease is a natural kind and that the shared properties occur as a result of the pathogenesis of the disease. I illustrate this with diverse examples from internal medicine and compare my account to alternative ontologies.

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1. Introduction

What do we mean when we diagnose a patient with a disease? What does it mean to say that two people have the same disease, or that one disease causes another disease? Rather than the standard question of what it is in virtue of which a given state counts as a disease, this paper addresses the question of what kind of things diseases are.[1] I address this question by looking at individual diseases and do not attempt to make any claim about a property or feature shared by all diseases together.

[1] There has been a great deal of attention given to the problem of separating states of disease from states of health—see, for example, Boorse (2011) and Ereshefsky (2009). Natural kinds are sometimes invoked in connection with this question and it is assumed that if diseases are not natural kinds, they must be value judgments. Sulmasy (2005) criticizes Reznek (1987, 1995) and D'Amico (1995) for this assumption.

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I argue that diseases are best understood as kinds using an account of natural kinds, based on the work of John Stuart Mill and Ruth Millikan. In section 2, I start with an overview of natural kinds in the literature and then look more specifically at Millikan's account. In section 3, I then explain how diseases should be understood as what I label "intrinsic kinds" and that the grounding for the kinds is the pathogenesis of the disease. In section 4, I consider two possible objections regarding hereditary diseases and infectious diseases but show that both are indeed intrinsic kinds on my account. This is an area that has received limited attention in the philosophy of medicine. In section 5, I compare my account to alternative ontologies, some of which are consistent with my account, provided a few modifications are made.

Before moving on, it is worth noting that in the philosophy of medicine, the word "disease" has been used to encompass all medical conditions. It is then often caveated that this is not the standard use of the word by either medics or laypeople. Some authors suggest that "medical conditions" might be better terminology (Simon 2011) and others attempt to separate diseases from medical conditions (for example, Williams 2007).[2] For my purposes here, and in line with the general usage in the philosophy of medicine, I use the word "disease" as equivalent to a medical condition, or roughly anything that can go wrong with a person's health. I do limit discussion here to physical health conditions in humans but I see no reason why it should not be applicable to mental health conditions and, indeed, to diseases of other organisms. For simplicity, I start with diseases of internal medicine, which include a vast range of diseases affecting all internal organs of the human body.

2. Kinds of Kinds

Natural kinds have been conceived in different ways in the literature. While some accounts have very strict requirements for a thing to be considered a natural kind, other accounts are more permissive. Here, I provide an overview of natural kinds and focus on the work of Mill and Millikan. I then clarify the terminology and explain the concept that I will be using for diseases.

2.1 Overview of Natural Kinds

In general, natural kinds are groupings or categories whose instances share many properties and so allow multiple projections. Natural kinds are often said to occur in the world for natural reasons, rather than simply reflecting human interests. Because they follow patterns in nature, they provide a basis for scientific inquiry. The standard examples of natural kinds are from scientific disciplines—for example, chemical elements or biological species.

Alexander Bird and Emma Tobin (2022) describe the range of positions on natural kinds. At one end of the spectrum are strong conventionalists, who would argue that there are no natural divisions in nature. And, at the other extreme, the position might be that there are natural kinds and they are a type of entity with an internal essence and sharp boundaries. As will become clear from my exposition, I do not agree with the conventionalists but I also allow natural kinds to be less definite than the latter position.

[2] Or, as Boorse suggests: "Readers who wish to preserve the much narrower ordinary usage of 'disease' should therefore substitute 'theoretically unhealthy condition' throughout" (Boorse 1975, 50).

2.2 Mill and Millikan

Mill introduced the term "Kind" (with a capital *K*) in 1843.[3] He wrote: "The class horse is a Kind, because the things which agree in possessing the characters by which we recognise a horse, agree in a great number of other properties as we know, and, it cannot be doubted, in many more than we know" (Mill 1868, 255). That is, horses form a kind because of the multiple shared properties among all horses. Horses have four legs, oval-shaped hooves, long tails, long necks, and elongated heads. They share the same internal anatomy and have a stay apparatus that allows them to sleep standing up. They also share properties of behavior—they are social animals and they like to eat hay. The list of shared properties continues. The point is that horses share multiple properties and if we recognize that something is a horse from just one or two features, we can project many other features. Because we know that something is a horse, we also know how it will walk, what its mane will look like, and so on. Mill held that there were an "indefinite and inexhaustible" number of properties that are shared by a true kind (1868, 275).

A category such as horse can be contrasted with properties that do not pick out kinds. Square things, for example, do not form a kind because the only similarities between the members relate to being square. No generalizations can be made from the fact that something is square beyond the shape and any features logically entailed by the shape (each square has four sides of equal length and four angles of 90 degrees). While there are many features in common between all horses that can be inferred from knowing that something is a horse, the same does not hold for square things. Being square is not a projectible predicate and square things do not form a kind.

From Mill, we therefore have the idea of kinds as categories in which many properties are shared and projections can be made. To this, Millikan has added that the properties cluster together for a good, non-accidental reason (1999, 2000).[4]

Millikan writes that a real kind is formed when there is a *univocal* principle that explains the similarity between any pair of members. While Mill held simply that horses form a Kind because of the multiple shared properties and projections that can be made, for Millikan, they form a kind because the properties cluster for a good reason. She distinguishes between two types of ontological grounding: historical and eternal. Historical kinds are those where properties are shared as a result of copying via descent from some historical origin. Biological species are examples of historical kinds—the similarity between horses comes from the fact that they have descended from other horses. The species is defined by reference to the historical relations among the members (Millikan 1999).

By contrast, eternal kinds are those with a shared inner core. Eternal kinds include chemical elements and their compounds but also stars, planets, asteroids, and geodes (Millikan 2000). The similarities between the instances of an eternal kind can be explained by something internal. The similarities between all samples of gold, for example, are explained by the molecular constitution, which is an internal feature, rather than a historical connection. I will explain historical kinds in more detail and then return to eternal kinds.

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[3] See Hacking (1991) for the history of natural kinds. The first use of kinds was apparently by Mill, although "natural kinds" as a phrase was first used by John Venn in reference to Mill. And the concept of categories with multiple shared properties is derived from Aristotle (Hacking 1991).

[4] Millikan's account is similar to Boyd's cluster kind account (1991) but Millikan focuses more on the different kinds of grounding, while Boyd characterizes the grounding as a homeostatic mechanism. Some of Boyd's kinds involve a process such that certain properties favor the presence of other properties, which I return to later regarding feedback loops as the pathogenesis of a disease.

Instances of historical kinds do not have a shared inner nature and instead the shared properties result from a copying mechanism. The similarities between any two horses can be explained by the fact that they are descended from other horses. Millikan compares this to the likeness between an original and a photocopy. A copying mechanism is the explanation for the similarity between the original and a photocopy (Millikan 2000). The copying process in the reproduction of a species is the explanation for the similarity between instances.[5]

Historical kinds are therefore those kinds that require a spatiotemporal connection, and the instances are produced by a copying or reproductive mechanism. Copying can be imperfect, and this can introduce variation between the instances of a historical kind. Millikan also argues that other categories that seem much further removed from classical natural kinds could form historical kinds, including dances (such as the waltz), university classes, lawyers, doctors, screwdrivers, and hard drives (Millikan 2017). She argues that these all rely on some historical lineage or spatiotemporal relation—they are all, in a sense, copied from previous instances. For my purposes, I stick to the more standard cases exemplified by historical kinds but I do not exclude the possibility of these also being natural kinds.

Returning to eternal kinds, the grounding is not a copying mechanism but is something that is internal to each instance. While horses are alike because of their shared origin, the similarity between instances of gold is because of their shared inner core. Eternal kinds do not require any spatiotemporal connection—the instances can and do come in and out of existence. The processes that create stars, planets, and asteroids do not rely on previous instances of stars, planets, and asteroids. But the processes that create instances of a biological species rely on the previous instances reproducing.

The difference between historical kinds and eternal kinds is therefore the nature of the grounding. The copying mechanism that grounds historical kinds is often imperfect and this can introduce variation. However, it would be a mistake to think that there is no variation among instances of eternal kinds. External environmental conditions, for example, will affect the properties of a chemical element, such as the boiling point or melting point.

To summarize thus far, there have been various proposed features of natural kinds with some being stricter than others. For my purposes in understanding diseases, I am interested in the clustering of properties that allow projections and occur for a good, non-accidental reason.

2.3 My Conception of Kinds: Intrinsic Versus Historical

The account of kinds that I use here is based on Millikan's framework but with different terminology. Instead of eternal kinds and historical kinds, I use intrinsic kinds and historical kinds. As I am focusing on diseases, it seems preferable to use a term that fits better with temporary and variable conditions.

Diseases may seem far removed from Millikan's eternal kind examples of chemical elements, planets, and asteroids. And Millikan does not explicitly discuss disease kinds. However, I demonstrate that there is an intrinsic similarity between instances of individual

[5] This contrasts with an eternal view of species, such as that of Devitt (2008), who argues that biological species have shared features because of shared intrinsic properties. For convincing arguments to prefer the historical kinds account for species, see Godman and Papineau (2020) and Godman, Mallozzi, and Papineau (2020).

diseases, just as there is an intrinsic similarity between instances of particular chemical elements, planets, or asteroids. Diseases are therefore intrinsic kinds.[6]

3. Disease Kinds: Intrinsic Kinds with Pathogenesis as the Grounding

For any given disease, there are shared properties among the instances (that is, among the people with the disease) and these shared properties occur due to the pathogenesis of the disease. Individual diseases form natural kinds as a result of the clustering of properties in the instances and because of the shared properties multiple generalizations can be made. The explanation for the shared properties—the pathogenesis of the disease—is something internal and therefore diseases are intrinsic kinds.[7] We can diagnose a patient with a disease or make statements about two or more people having the same disease because of this structure.

In this section, I explain the shared properties between instances, what the pathogenesis of a disease is, and why there is variation between instances of a disease.

3.1 The Shared Properties

Each disease has many different properties shared by the instances. Here, I go through some of the most common properties shared between instances.

3.1.1 The Signs and Symptoms

The signs are the features that are elicited from clinical examination (such as a particular rash, fever, or heart murmur) and the symptoms are experienced by the person (for example, pain or difficulty breathing). Signs and symptoms cluster when they are shared by many people with the same disease. A tender and swollen calf with reddened skin is often shared by individuals with deep vein thrombosis (DVT), for example. Many signs and symptoms will not alone be diagnostic of a condition (the tender and swollen calf with reddened skin could be caused by cellulitis, rather than a clot, for example) but there is still a clustering of the properties. Not everyone with a DVT will have the calf changes and some people may have the changes even though they do not have a DVT. However, there is a still clustering of properties between the instances of a DVT.

As well as variation within a particular disease (for example, that one instance of measles could present without a rash), there is also variation in whether disease kinds have signs and symptoms. Some diseases may have clinical signs but no symptoms (for example, hypertension) and others may have neither signs nor symptoms (for example, in the early stages of a disease). Signs and symptoms generally cluster in diseases but some diseases lack them.

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[6] Fagerberg has also argued for a conception of diseases based on Millikan's real kinds—see Fagerberg (2022).

[7] I note that I start with the assumption that there are shared properties between instances of a disease, which is required for them to form a kind. In this section I focus on the explanation for the shared features and thus whether diseases are intrinsic kinds or historical kinds. It is sometimes suggested (especially regarding psychiatry) that "disease" categories are just arbitrary groupings but such claims are best understood as denying that the categories in question are diseases—see, for example, Bracken and Thomas (2010) regarding critical psychiatry. Writers such as Gräsbeck (1984), who are keen to point out that disease taxonomies are subject to change, and that no two people have the exact same disease, do not suggest that there are no shared properties between instances of a disease, or that the groupings are arbitrary.

3.1.2 Results of Investigations
These are the changes seen in radiological imaging or blood tests. Certain patterns of changes are seen on chest X-rays in pneumonia and different ones in states of fluid overload, for example. Again, results of such investigations are not absolute but there is a clustering of results among many diseases. The combination of particular signs and symptoms with certain investigation results cluster together and allow inferences about the disease.

3.1.3 The Course of the Disease
There are also shared properties related to the course of the disease—how it will unfold (that the chickenpox lesions will blister before they scab over) and what the prognosis will be (the likelihood of recovery and recurrence). Again, this is not absolute but there is clustering within particular diseases.

3.1.4 Response to Treatment
Again, there is a clustering of properties such that particular types of bacterial infections will respond to particular antibiotics and other types of diseases will respond to other treatments (the airway tightening in asthma will often improve with beta2-agonist inhalers). There is a clustering of properties related to the response to medical treatment.

3.1.5 Pathogenesis
Most importantly, the pathogenesis of a disease is also a property that is shared among the instances, and it is the property that explains the other properties. It is the property that determines what disease the person has and is therefore unique to each disease. The pathogenesis determines the shared signs and symptoms, what the investigation results will show, what the course of the disease will be, how it will respond to treatment, and so on. The next section characterizes the pathogenesis more precisely.

3.2 The Pathogenesis
I use pathogenesis to refer to the underlying cause of the clustered properties of a disease. Below, I list a few examples of the pathogeneses of different diseases to illustrate the concept. These examples are simplified but still demonstrate the reason for the similarities between instances of a disease.

Asthma is a disease characterized by wheezing, shortness of breath, and a cough, which tends to be worse at night. These properties cluster along with various investigation results and the response to treatments. The pathogenesis of asthma is the inflammation and tightening of the airways; it is a property that is shared between the instances and it is the explanation for the other shared properties. Although there can be a difference in severity of symptoms (both day to day and during an exacerbation), there are shared symptoms between people with asthma and these symptoms are shared as a result of the underlying airway changes.

Alternatively, for cystic fibrosis, the pathogenesis is the defective CFTR (cystic fibrosis transmembrane conductance regulator) protein, which disrupts the ion transportation and mucus production. This causes excess thick and sticky mucus, which in turn causes problems in the respiratory and digestive systems. Again, the properties in common between instances of cystic fibrosis result from the shared genetic abnormality.

Those with rheumatoid arthritis have shared properties because of certain inflammatory processes. Those with macular degeneration experience visual loss as a result of retinal degeneration. Shared properties among individuals with multiple sclerosis occur because of the demyelination of the central nervous system. And so on, as we work through lists of diseases. There are similarities among people with any given disease and the similarities can be explained by the pathogenesis of the disease.

Even in diseases that are poorly understood, we can often still pick out a feature that explains the other shared properties. For example, Parkinson's disease is a degenerative neurological condition. Exactly why it occurs is unknown but the death of the dopaminergic brain cells is known to cause the signs and symptoms; that is, the loss of these cells explains the tremor, balance problems, and memory loss characteristic of the disease. These symptoms all cluster in Parkinson's disease and they cluster because of the death of the dopaminergic brain cells. This loss of brain cells is therefore the pathogenesis of the disease (although the exact reasons for it occurring are unknown).

Some diseases might seem to be caused by a feedback loop and it might therefore seem difficult to pick out what the pathogenesis of the disease is.[8] On my conception, it is still possible to pick out a single thing that I would consider to be the pathogenesis. Systemic lupus erythematosus (SLE) is an example of an internal medicine disease that may be caused by a feedback loop. Mark J. Shlomchik, Joseph E. Craft, and Mark J. Mamula (2001) describe a feedback loop that augments the autoimmune response and causes the clinical features seen in SLE. The pathogenesis of SLE or other conditions caused by a feedback loop system would then simply be the feedback loop. It may be that several separate factors are required for the feedback loop to start but, once it has started and the feedback continues, this process can be considered the pathogenesis on my account.[9] Feedback loops are therefore not problematic for a conception of diseases individuated by pathogenesis.[10]

It should now be clear that the pathogenesis is the explanation for the clustering of properties in diseases. This concept is sometimes referred to as the etiology of a disease but the etiology sometimes also refers to a more distal cause of a disease. Etiology can be used to refer to a single feature that occurs prior to the development of a disease—for example, that smoking is the cause of lung cancer. Marc Lange (2007) suggests that this is problematic because smoking can contribute to both lung cancer and emphysema, although they are clearly different diseases. This is not what I am referring to as the pathogenesis or cause of the disease. On my conception, it is easy to separate lung cancers (plural because

[8] Although I am focusing on conditions in internal medicine, this is often said to be the case for mental health conditions, such as depression. Godman, Mallozzi, and Papineau (2020), for example, suggest that there is no single cause for depression as it relies on reciprocal causation.

[9] See (Fagerberg 2022) for a more detailed argument regarding feedback loops as the grounding for both psychiatric and somatic medical kinds.

[10] It might be thought that Boyd's homeostatic property cluster (HPC) account offers a better description of the feedback loops because of the word "homeostatic" and the idea that such mechanisms would maintain the clustering of properties (Boyd 2010). However, on closer inspection, Boyd's account does not require feedback processes for the clustering.

there is distinct clustering for different forms of lung cancer) from emphysema. The pathogenesis of adenocarcinoma of the lung, for example, involves the abnormal cell growth in the cells lining the lungs. It causes the clustering of symptoms when it results in malignant transformation, causing local invasion in the lung. By contrast, emphysema is characterized by the destruction of the alveolar walls and therefore loss of lung elasticity, which leads to difficulty breathing. Both adenocarcinoma and emphysema are caused by smoking but they are very different diseases, with different property clusters. The bit that I want to pick out as the pathogenesis is the thing that causes the clustering—the malignant transformation and local invasion in lung adenocarcinoma and the loss of lung elasticity in emphysema. Smoking, of course, contributes to both diseases but it is distal to the pathogenesis—prior to the changes that explain the signs and symptoms, and so on.

Further, for infectious diseases, it is sometimes said that two conditions are the same if they are caused by the same infective agent and it is noted that tuberculosis is responsible for both Pott's disease of the spine, as well as the more common respiratory condition. Lange (2007) suggests that we now recognize that they are the same disease as we know that they are both caused by *mycobacterium tuberculosis*. I do not deny the involvement of the same bacteria in both but different properties cluster for spinal tuberculosis and respiratory tuberculosis and, on my conception, they are therefore different diseases. Although there is the same distal cause, there is a different pathogenesis. Pott's disease generally occurs secondary to respiratory tuberculosis but, as there is unique clustering, it should be considered its own disease kind. I will return to the general problem of associating a disease with the infective agent that is present in section 4. For the moment, it suffices to say that the pathogenesis is more than just an infective agent or a risk factor, such as smoking.

It might be argued that the pathogenesis of a disease might explain the clustering of signs and symptoms, the prognosis, and so on, but does it really explain all the properties that cluster? For example, does the pathogenesis explain the difficulty in getting medical insurance or the stigma associated with certain diseases? I would respond that—like all intrinsic kinds—diseases can be influenced by contingent environmental conditions. Such environmental conditions include the atmospheric pressure that determines the melting point of gold. Other environmental conditions include the social factors that might affect the clustering of properties related to gold, such as value and fashion. In the same way, people with leprosy have experienced stigma because of contingent environmental or social conditions, rather than directly as a result of the pathogenesis of the disease.

The pathogenesis is therefore the feature that explains the other shared properties in disease kinds—sometimes caveated by the environmental conditions. But it is important that the explanation for the similarities between instances is always something internal. This is why diseases are intrinsic kinds, rather than historical kinds. After reviewing reasons for the variation among instances of disease, I demonstrate that external pressures can be involved in hereditary conditions and infectious diseases but both are intrinsic kinds, rather than historical kinds.

3.3 The Variation

It is often pointed out that the same disease is experienced differently by different individuals, and this is sometimes used as a reason for which diseases cannot be consistent

with an essentialist picture of natural kinds—see, for example, Jensen (1984) or Williams (2011). From the above, it should be clear that even if there does not exist a list of necessary and sufficient conditions for each disease, there is still a clustering of properties as a result of the internal pathogenesis. This is sufficient for natural kind status on my account but it is worth highlighting some of the reasons that there is variation in experiences and outcomes of disease.

Of course, not everyone will experience a disease in the same way and no one expects that they would. Despite a clustering of properties, there are often differences among individuals with the same disease. In many cases, we cannot fully predict how a particular person will experience a disease but we know that diseases are often more severe in extremes of age, or if there are concurrent health conditions, or medications contributing to immunosuppression. For example, the immunosuppressed state from AIDS allows more opportunistic infections, as well as more severe illness. Other variations in disease presentation appear to be related to socioeconomic factors, with studies consistently finding worse results among those from lower socioeconomic groups (Mackenbach et al. 2000). Further considerations might include lifestyle factors, such as smoking and alcohol consumption, increasing age, and obesity. It is also recognized that there are unknown confounders—reasons for which diseases are more or less severe, or medications are more or less effective—that are unknown.

It is perfectly consistent with my account to accept the variation between instances but still maintain that diseases form natural kinds. Stefan Dragulinescu frames the difference between a paradigmatic natural kind, such as gold, and that of Graves' disease as a "difference in degree," rather than an "ontological gap" (Dragulinescu 2010, 361). He concludes that there is a difference in degree because we are less certain how an instance of Graves' disease will behave than an instance of gold. It is certainly easier to control the relevant variables for chemical elements (for example, atmospheric pressure) than it is for the unknown variables in instances of disease. However, chemical elements and disease kinds are both intrinsic kinds and variation between instances does not lessen the clustering of properties that occurs for a good, non-accidental reason.

4. Hereditary Diseases and Infectious Diseases Are Not Historical Kinds

It might be tempting to consider hereditary diseases or infectious diseases as historical kinds because there seems to be a spatiotemporal connection involved.[11] However, I demonstrate here that both are better understood as intrinsic kinds despite the involvement of copying mechanisms. With the examples of Huntington's disease and measles, I show that the properties that cluster together to form the disease kinds are not themselves copied.

4.1 Hereditary Disease

Hereditary diseases rely on a form of copying but the properties of the diseases considered above (signs, symptoms, prognosis, and so on) are not themselves copied. Instead, in each person with the condition, the shared properties occur as a result of the underlying genetic

[11] Although they mention it only briefly, Godman and Papineau (2020) suggest that infectious diseases might have both a historical and an internal explanation.

change. The explanation for the shared clustering of properties (the pathogenesis) is therefore intrinsic. Below, I work through an example to clarify this.

Huntington's disease is caused by a defect in the huntingtin gene (Imarisio et al. 2008). This defect is inherited in an autosomal dominant manner, so that if one parent has Huntington's disease, there is a 50 percent chance that any offspring will be affected. The genetic mutation is present from birth but symptoms resulting from Huntington's disease do not generally occur for several decades since they correlate to progressive neuronal dysfunction and loss.

The shared features that eventually develop in people with Huntington's disease occur because of changes that themselves result from the defect in the huntingtin gene. But these shared properties are not themselves copied. Classical symptoms include uncontrolled movements and progressive cognitive problems. While someone may have inherited the faulty gene that causes Huntington's disease from their parent, and copying processes will have been involved here, the symptoms themselves are not copied. The chorea is not copied from parent to offspring—it is caused anew in each person as a result of the neurons degenerating.

For each person, the explanation for the properties that cluster in Huntington's disease is internal. We can contrast this to historical kinds in which the properties themselves seem to be the result of copying. This is particularly clear for a historical kind, such as a chain of Starbucks. One instance is like another because it has been copied from the original: the machinery is copied, the seating is copied, the cups are copied, and so on. There are shared properties because they have been copied. But it is not like this in Huntington's disease—the features are not each copied like this. Only the gene is copied but the gene itself does not cause property clustering until there is neural degeneration. The genetic change seems to be a necessary precursor to the disease but not the disease itself.

Further, despite the genetic element in most cases, not all cases even involve copying since *de novo* mutations occur and account for around 10 percent of instances. There is therefore not a necessary historical component (although there is a necessary genetic component). There is also not a connection between all cases of Huntington's disease. It is not the case that all instances are connected to previous instances. Even those that are not the result of *de novo* mutations are not connected to other instances if some of those were *de novo*. The 90 percent of non-*de novo* cases include the offspring of *de novo* cases. Not all cases of Huntington's disease have a common ancestor. By contrast, there is a connection between all instances of horses as they are all from a shared origin. There are no sporadic instances of horses. As such, Huntington's disease appears very different to the historical kinds such as those of biological species. Something cannot be a horse unless it is derived from horses but a person can be an instance of Huntington's disease without any historical lineage to other instances.

To summarize, hereditary conditions such as Huntington's disease contain features that are present from birth, including a genetic mutation that is normally copied from a parent. However, the features of the disease themselves occur because of the changes that result from the genetic mutation. A copying mechanism creates the likelihood of the disease but the disease itself is something that happens in each person anew as neuronal changes occur. The best explanation for the shared properties between instances of Huntington's disease is the pathogenesis, which is the process that occurs in each person and causes the characteristic symptoms. Hereditary conditions are intrinsic (not historical) kinds.

4.2 Infectious Diseases

I now demonstrate that infectious diseases also form intrinsic kinds, rather than historical kinds. Copying mechanisms are involved in the replication of the microbe but the diseases themselves are intrinsic kinds. I work through the example of measles to demonstrate this. It was already demonstrated above that the same *mycobacterium tuberculosis* can cause different diseases, depending on which body system is affected (respiratory or spinal).

Like all viruses, the measles virus requires a host cell for replication and persists by copying. The measles virus (*measles morbillivirus*) is therefore a historical kind. For *measles morbillivirus* (unlike many genetic diseases, such as Huntington's disease), there is a common ancestor for all instances. There are not sporadic ways to form the virus equivalent to the *de novo* huntingtin protein mutations. All cases of measles are therefore historically linked to the original evolution from rinderpest centuries ago (Furuse, Suzuki, and Oshitani 2010).

The measles virus itself is a historical kind, with the instances being the virus particles. It is a kind because of the multiple shared properties between the virus particles and the grounding is historical because of the common heritage. However, it is important to distinguish the measles virus from the disease that is caused by measles (with the characteristic signs and symptoms). While the measles virus is a historical kind, the disease caused by the virus measles is an intrinsic kind.

The disease measles is an intrinsic kind, with the instances being the people who have the condition. It requires the virus and the properties cluster as a result of a combination of the virus and the immune response. The pathogenesis of the disease is the interaction between the virus and the immune system that happens in each person with the disease. The signs and symptoms are not copied from person to person. The rash, for example, develops in response to the inflammation in the skin from the measles virus and happens each time anew—it is not itself copied. The disease measles is therefore an intrinsic kind since the explanation for the shared properties is internal.

It is important to separate a third category: subclinical measles. This is spread by *measles morbillivirus* but is asymptomatic. It often occurs in the vaccinated population and does not cause any signs or symptoms—the characteristic clustering of properties does not occur in subclinical measles (Perry and Halsey 2004). There is still an antibody response that can be found from blood tests but the person exhibits no features of measles. I would say that this person does not have a disease because there is no clustering of properties. It is not that they have measles without a fever, or measles without a rash, but instead they have none of the features of measles.

Subclinical measles can only be found by looking for it—by testing a well population. And because subclinical measles is not associated with onward spread, it is not generally something to look for—that is, subclinical measles is not a disease and it is not associated with spreading measles. This could be contrasted with other infectious diseases for which there is good reason to look for subclinical or asymptomatic infection. Take the recent example of Covid-19 caused by SARS-CoV-2. Many people tested positive for SARS-CoV-2, although they never developed symptoms of Covid-19 (the respiratory illness that results from SARS-CoV-2). Here, there was a good reason to test and identify these people because it was thought that subclinical infections could be responsible for spreading the disease. The

reason to test and diagnose was for population-based health, rather than for the benefit of the individual with presence of SARS-CoV-2. However, a good pragmatic reason to test someone does not suggest that the asymptomatic carrier has a disease. Indeed, I would say that asymptomatic SARS-CoV-2 is also not a disease.

It therefore seems that for at least some infectious agents (including *measles morbillivirus* or SARS-CoV-2) there can be the presence of the microbe without the presence of the disease.[12] This is further reason to separate the historical kind measles from the disease that results from it. I discussed viruses above but bacterial and fungal infections (as well as parasites and prions, and so on) all follow a similar pattern. The infective agent is itself an instance of a historical kind. But the disease that occurs is an intrinsic kind.

To summarize this section, it seems that the microbes that cause infectious diseases are themselves historical kinds. However, the infectious diseases are intrinsic kinds because the clustering of properties occurs in each person due to internal factors, such as the individual immune system. Having argued that diseases form intrinsic kinds (including for diseases that rely on copying mechanisms, such as hereditary diseases and infectious diseases), I next consider alternative accounts of disease ontology.

5. Alternative Accounts

In this section, I consider several points that have been discussed in alternative conceptions of the ontology of diseases. I use the examples of a dispositional account of diseases and the relationship between diseases and features of disease to clarify my own account.

5.1 Dispositions: The Problem of Diabetes

Properties have featured in other accounts of disease. For example, Jonathan Fuller (2018) has argued that many chronic diseases are dispositional properties. In this section, I look at the implications for diabetes.

Properties can be divided into categorical properties and dispositional properties. A categorical property is one that is always manifest whenever it is present, while a dispositional property is only manifested in certain circumstances. Size and shape are categorical properties, while flammable and soluble are dispositional properties. Something only has the property of being round or five foot long when it is round or five foot long. But things are often flammable or soluble without ever catching on fire or dissolving. The disposition of a glass to shatter only refers to it possibly shattering in certain circumstances and says nothing about the actual behavior of the glass. As such, dispositions might seem "mysterious" in a way that categorical properties are not (Choi and Fara 2021).

Fuller (2018) looks at the instances of chronic diseases, or the disease "tokens," as he labels them. He purposely does not consider the question of whether diseases form natural kinds.[13] He finds that many chronic diseases are dispositional properties because the

[12] I certainly do not claim that all infectious diseases can have subclinical counterparts that are not themselves diseases because the issue is very complicated and not without controversy. See, for example, Wakefield (2014) for discussions about Typhoid Mary—an asymptomatic carrier of typhoid who was responsible for multiple outbreaks.

[13] Fuller only discusses the instances of a disease and not the disease categories, while my focus has been on the categories. I have also discussed the instances of a disease as the people with that condition. It is important to

characteristic manifestation of the disease is not always present when the person has the disease. However, he suggests that infectious diseases are categorical properties. I focus here on Fuller's examples of diseases as dispositions since these raise some important points for my argument.

Fuller argues that both type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM) are dispositional properties: "Diabetes is a disposition towards hyperglycemia" (Fuller 2018, 3208). I argue that they can be considered dispositional only if the disposition is associated with its causal base.[14] From my perspective, T1DM and T2DM are two different diseases, which could be consistent with dispositional properties. As Fuller focuses on the instances of disease, his point is that in each person with T1DM or T2DM there is a disposition toward hyperglycemia. Both T1DM and T2DM can cause high blood sugar levels (hyperglycemia) but they do so for different reasons.[15] T1DM results from the destruction of the insulin-producing beta cells in the pancreas and, without insulin, blood glucose levels cannot be moderated. It tends to affect young people and most cases appear to be auto-immune mediated. A person with T1DM produces no insulin and therefore requires exogenous insulin to drive the glucose into the cells, where it can be used for energy. Without insulin, not only can the glucose not be used by the body's cells but there is also a risk of death from dangerously high glucose levels in the bloodstream.

By contrast, T2DM causes hyperglycemia as a result of insulin resistance—insulin is produced but not used by the body's cells in the liver, fat, and muscle. T2DM is associated with obesity and globally increasing levels of obesity have increased the prevalence of T2DM. Symptoms of T2DM can sometimes be managed with diet and exercise, or oral medications. Many people will also require exogenous insulin injections like those patients with T1DM but because of the insulin resistance, they often require higher doses.

T1DM and T2DM therefore both result in hyperglycemia but for different reasons. On my conception of diseases, there are two different disease kinds. People with T1DM are instances of the disease kind T1DM because of the many shared properties that are caused by the destruction of beta cells. And people with T2DM are instances of the disease kind T2DM because of those shared properties resulting from the distinct pathogenesis.

Of course, there is some overlap in the properties since both T1DM and T2DM result in hyperglycemia. Hyperglycemia itself can cause symptoms such as excess urine, thirst, and weight loss. And this can occur in those with hyperglycemia regardless of the cause for the hyperglycemia. But the clustering of properties for each of T1DM and T2DM includes more than just those caused by hyperglycemia. This is akin to the painful red and swollen ankle that can occur because of either cellulitis or DVT, as mentioned above. There is still distinct clustering of properties as a result of the unique pathogeneses.

Properties shared by those with T1DM include a diagnosis at a young age, normally with an apparent sudden onset, a risk of other autoimmune conditions, the risk of ketoacidosis, as well as hyperglycemia and complications related to intermittent or sustained hyperglycemia. And T2DM is associated with a distinct set of properties, including a slower

be clear about the difference in the ontology of disease instances and disease categories—see for example, Whitbeck's (1977) specific criticism of King (1954).

[14] An alternative dispositional account has been proposed by Smart (2014), in which diseases are the causal processes produced by the disposition. As my discussion will show, his view is consistent with my theory.

[15] As well as T1DM and T2DM, there are other forms of diabetes, including gestational diabetes, diabetes caused by pancreatic problems (cystic fibrosis, chronic pancreatitis, pancreatic cancer, or pancreatic surgery), and drug-induced forms of diabetes. It is enough to compare the two most common forms here.

progression and diagnosis that occurs at an older age, as well as the properties associated with hyperglycemia that are shared with T1DM.

Whether this is all consistent with the idea that diseases are dispositions depends on how we consider dispositions. There is a general question in the metaphysics of dispositions of whether dispositions should be identified with their causal bases or be only the tendency to manifest in certain ways whatever the cause—so-called bare dispositions (Choi and Fara 2021).

The risk here is that T1DM and T2DM could appear to be the same disease since they both have hyperglycemia as a characteristic manifestation. This would be problematic when there are differences between the two diseases. On my account, there is a principled reason to separate the diseases because of the distinct grounding for the shared properties. The grounding for T1DM is the destruction of the beta cells in the pancreas while the grounding for T2DM is the insulin resistance. If the disposition is associated with causal basis, this would work with my account. The causal basis for the disposition is the pathogenesis of the disease.[16] However, with a different view of dispositions (in which they are not associated with their causal bases), the claim that diseases are dispositions would be incorrect for T1DM and T2DM.

Several authors have argued for close links between dispositions and natural kinds and this is entirely consistent with what I have described—see, for example, Van Rooij and Schulz (2021). Dispositions can be used alongside my account, so long as the disposition is associated with its causal basis.[17] With this association, T1DM and T2DM should appear to be different diseases.

In his discussion of T1DM, Fuller (2018) is keen that the disease should not be the process by which hyperglycemia occurs as the process is not always present. He notes that when a patient is receiving adequate insulin, there is no hyperglycemic process so "we should conclude that the disease is not the process, the disease is something that remains throughout treatment" (Fuller 2018, 3216). On my conception, managed or treated diseases are still diseases, so I would agree that the disease is not something that is only present when the relevant processes or signs are occurring. Indeed, that certain treatments work form part of the property clusters for any given disease (for example, that the beta2-agonist relieves shortness of breath in asthma). The processes that produce the effects therefore do not need to be occurring for the person to have a disease.

Next, I consider whether the signs and symptoms or processes related to diseases are parts of the disease or caused by the disease.

[16] Fuller (2018), of course, recognizes that there are distinct causes for T1DM and T2DM and that the physiological basis for the dispositions involved rely on the pathological changes. However, he is also committed to a view that diabetes cannot be type reducible to either the changes behind T1DM or T2DM. This problem is avoided on my account since I do not group T1DM and T2DM together as dispositions to hyperglycemia.

[17] For Fuller (2018), there is a distinction between the causal basis for the disposition to hyperglycemia and the pathogenesis of the disease.

5.2 Are the Signs and Symptoms Caused by the Disease? Or Part of It? What About the Course?

Caroline Whitbeck argues that diseases are processes and she distinguishes diseases from "static conditions," such as cleft palate (Whitbeck 1977).[18] I am unsure about this distinction as many potentially static conditions do cause a series of progressive complaints. Benjamin Smart, for example, points out that aortic stenosis or patent ductus arteriosus could both be conceived as static impairments but both can cause advancing disease (Smart 2014). Cleft palate itself can be associated with other sequelae, such as failure to thrive or hearing problems, so perhaps it is not a static condition either. We can certainly accept that diseases frequently (if not always) involve processes.

Whitbeck then considers whether the clinical course of a disease should be considered produced by the disease or part of it and concludes that it is part of the disease. So, for Whitbeck, manifestations of a disease are all part of the disease process: "My thesis in this section has been that the signs and symptoms which are identifying marks of a disease are not properly understood as effects of the disease process but states of affairs that occur within it" (Whitbeck 1977, 634).

In contrast, Lange (2007) and Fuller (2018) have argued that the disease explains the signs and symptoms and therefore the disease cannot be the signs and symptoms. Lange states: "For a patient's disease to explain her signs and symptoms, the disease must be distinct from its clinical picture, since otherwise physicians would be calling upon that picture to explain itself" (Lange 2007, 268).

On my account, diseases are kinds because of the clustering of properties, which includes the signs and symptoms. There is an explanation for the signs and symptoms from the pathogenesis of the disease but the signs and symptoms are not somehow separate to the disease. This is equivalent to the explanation for the melting point of gold being explained by the molecular constitution. The melting point is a feature of gold, not something caused by it. Similarly, the signs and symptoms are features of the disease, not caused by the disease. Again, the intrinsic kind account provides a better explanation for the clustering of properties within diseases.

6. Concluding Thoughts

I have argued here that diseases form intrinsic kinds, which are natural kinds, where the shared properties can be explained by the pathogenesis of the condition. With diverse examples, I have demonstrated that this conception captures the way in which diseases have been used in internal medicine.

Returning to the question that I asked at the start, to diagnose a patient with a disease is to recognize that they are an instance of an intrinsic kind for that disease—that is, to recognize that the person has a series of properties because of the pathogenesis and that other people with the same pathogenesis are likely to have a similar series of properties.

I would expect that this could be extended beyond internal medicine, including to psychiatric conditions but I recognize the difficulty when the pathogenesis is less well understood.

[18] While Fuller (2018) discusses only instance of disease, Whitbeck (1977) discusses both the disease type or category (which she calls the "disease entity") and the individual diseases, which she labels "cases."

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