

# Natural kinds, normative kinds and human behavior

Tipos naturais, tipos normativos e comportamento humano

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## ABSTRACT

The main thesis of this paper is that a large part of human behavior cannot be understood in terms of natural kinds but by appealing to normative kinds. In the first section we explain the distinction between natural kinds and normative kinds. In the second section we focus on the notion of "human behavior", proposing a distinction between type A and type B behaviors and pointing out that psychology deals with type B behaviors, which are also included as diagnostic criteria for mental disorders. In the third section we analyze the strategies used in biomedical research to find specific etiologies ("essences") in order to explain such disorders. We argue that their results are inconsistent and that the lack of biomarkers that are clinically useful to refine the diagnoses is due to the fact that, unlike certain neuropathologies, there are no physiological essences behind such disorders. On the other hand, we argue that, as we are dealing with type B behaviors, we must interpret mental disorders as normative kinds.

**Keywords:** human behavior, natural kinds, normative kinds.

## RESUMO

A tese principal deste artigo é que uma grande parte do comportamento humano não pode ser entendida em termos de tipos naturais, mas deve recorrer a tipos normativos. Na primeira seção, explicamos a distinção entre tipos naturais e tipos normativos. Na segunda seção, ressaltamos a noção de "comportamento humano", propondo uma distinção entre comportamentos do tipo A e tipo B e salientando que a psicologia lida com comportamentos do tipo B, que também são incluídos como critérios de diagnóstico para transtornos mentais. Na terceira seção, analisamos as estratégias utilizadas na pesquisa biomédica, a fim de encontrar etiologias específicas ("essências") para explicar esses transtornos. Argumentamos que seus resultados são inconsistentes e que a falta de biomarcadores clinicamente úteis para refinar os diagnósticos se deve ao fato de que, diferentemente de certas neuropatologias, não existem essências fisiológicas por trás desses transtornos. Por outro lado, argumentamos que, ao lidarmos com comportamentos do tipo B, devemos interpretar os transtornos mentais como tipos normativos.

**Palavras-chave:** comportamento humano, tipos naturais, tipos normativos.

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## Introduction

The idea that some of the conceptual tools we deploy cut out the world “by its joints” – *i.e.* they refer to natural kinds – is an idea implicit in our everyday conception of the world and also a guide for scientific research. The scientific knowledge thus acquired affects our daily life, as we appeal to scientific information to make decisions both individually and socially. The advances of modern science in certain areas (especially in chemistry and biology) and the improvement in our everyday lives due to this knowledge are paradigmatic cases of the success provided by the assumption that the world contains natural kinds whose essence is accessible to us through scientific research. And it encourages us to extend the idea of natural kind to other scientific fields. In the case of medically relevant biological categories we have discovered the “essences” of many of our everyday categories (*e.g.*, mainly diseases, such as measles, tuberculosis, dengue, etc.). These essences are the hidden mechanisms behind the superficial similarities that we observe and use as evidence of the existence of a natural kind. Not all the categorizations we elaborate involve natural kinds, because not all the similarities we observe can be explained by deeper unobservable mechanisms. But, in any case, as cognitive psychologists found (Keil, 1989; Medin, 1989; Murphy, 2000) following Quine’s 1967 philosophical intuition, we are cognitively biased towards looking for the hidden mechanisms behind superficial similarities we find.

When it comes to social sciences and humanistic disciplines, the idea of natural kind seems inappropriate. Many authors contrast natural kinds with other types of categories, such as human or interactive kinds (Hacking, 1991b, 1995), human-made kinds (Ereshefsky, 2004), normative kinds (Griffiths, 2004), practical kinds (Zachar, 2000), historical kinds (Bach, 2012), objective types (Haslanger, 2012) or social kinds (Mason, 2016), in order to mark a difference between the taxonomies used in natural sciences and other kinds of categorization practices.

Human behavior is the object of study of a particular science: psychology<sup>3</sup>, that seems to be half a way between these two kinds of scientific disciplines. For almost two centuries, psychology has sought to find a place in the concert of other

sciences, assuming – either explicitly or implicitly – ideas that have been successful in other fields: in this paper we want to focus on the idea of “natural kind.” The question that guides this work is whether it is possible to adopt the idea of natural kind when dealing with psychological categories that involve human behavior.<sup>4</sup> In order to understand human behavior in our everyday life, we appeal to mental concepts<sup>5</sup> constituting a network that is usually called folk psychology.<sup>6</sup> The most basic folk-psychological categories are transcultural: basic emotions (such as fear, disgust, joy, sadness, etc.), desire, belief, bodily sensations (such as pain, itch), memory, learning. Besides the discussion about whether these concepts are innate or not and whether they constitute a theory or not, in our daily lives we use them to understand and explain the actions of our fellow humans. On the other hand, throughout their history, the psychological sciences added a set of technical concepts that are part of the scientific theories that seek to explain human behaviors. Some examples of technical terms historically coined in various scientific theories are: “Oedipus complex,” “autism,” “neurosis,” “hysteria,” “working memory,”<sup>7</sup> “Alzheimer’s disease,” “Attention Deficit Hyperactivity Disorder (ADHD).”

The thesis that we are going to defend in this paper is that a large part of human behavior cannot be understood in terms of natural kinds but by appealing to normative kinds. The plan of this paper is as follows. In the first section we will explain the distinction between natural kinds and normative kinds. In the second section we will focus on the notion of “human behavior,” showing the diversity of types of behaviors and drawing a distinction between type A (involuntary bodily changes) and type B (intentional movements) behaviors; pointing out that psychology seems to be dealing with type B behaviors, which are also included as diagnostic criteria for mental disorders. In the third section we will analyze the strategies that are used in biomedical research in order to find specific etiologies (“essences”) which would explain such disorders. We will argue that their results are inconclusive and that the absence of biomarkers that are clinically useful to refine the diagnoses responds to the fact that, unlike the case of certain neuropathologies, there are no biological essences behind such disorders. On the other hand, we will argue that, as we are dealing with type B behaviors, we must interpret mental disorders as normative kinds.

<sup>3</sup> We are including under the label “psychology” both normal and abnormal/pathological behaviors; hence, we are including psychiatry as part of psychology.

<sup>4</sup> In fact, all social sciences and humanistic disciplines are concerned with human behavior. While there is some bibliography about the notion of natural kind in sociology, gender studies, etc., there are not many works that consider this idea specifically regarding categorizations in psychology. In general, as we shall see, it is assumed that psychology is a natural science and that therefore it deals with natural kinds, whose essences are biological; however, recently this orthodox view has begun to be put into doubt, as we will see in the next paragraph.

<sup>5</sup> We will use the expressions “mental concepts” and “psychological concepts” indistinctly.

<sup>6</sup> Lewis, 1972, p. 212; Fodor, 1987, Goldman, 1993, p. 15; Hutto, 2008, p. ix.

<sup>7</sup> In some cases, some folk-psychological concepts such as “memory” are redefined or divided into sub-species such as “working memory”, “long term memory”, “episodic memory”, etc.

# 1 Natural kinds and normative kinds

We classify the things around us in order to guide our actions in the world. We categorize things as being “of the same type” insofar as dealing with that set of things is simplified thanks to such categorization, because the things belonging to that category correspond in the same way to our interests and practical purposes. Very often we classify things as being of the same type because we think that they all have something in common that allows us to predict their behavior by studying some of them and projecting our knowledge onto similar objects. This is the idea of a “natural kind”. Paradigmatically, modern science aims at increasing knowledge through research on natural kinds. This strategy has brought enormous benefits to humanity. Thus, modern science has managed to increase its predictive ability by refining ordinary categories and finding new forms of categorization that sometimes coincide,<sup>8</sup> sometimes divide<sup>9</sup> and sometimes come into conflict with our ordinary concepts.<sup>10</sup> Natural sciences aim to find the mechanisms that unify each natural kind, mechanisms that are understood as the hidden essences that allow us to explain the superficial features and behaviors of the objects that belong to a certain kind. In paradigmatic cases, these essences are described by some scientific discipline involving new concepts. A paradigmatic example of a natural kind is “water”, whose essence –  $H_2O$  – was established with the development of chemistry in the eighteenth century; another example is tuberculosis, whose “essence” is given by the presence of Koch’s bacillus in the organism.

According to Putnam (1975), natural kind concepts consist of at least two components: an essence and a stereotype. The stereotype is the set of superficial, observable features that every competent speaker – or, alternatively, everyone who has mastery of the concept – knows; it allows us to rightly apply the term to the objects in the world. In the case of “water”, this stereotype will include that: it is a tasteless, colorless, odorless liquid; it quenches thirst; we can find it in lakes, rivers, seas and oceans; it freezes at 0 degrees Celsius and boils at 100 degrees (always at sea level), etc. This set of traits is usually presented as a whole in nature, and they are the ones that we seek to explain through scientific research, finding an essence which unifies these features.

The essence, therefore, is what we aim to discover through empirical research, in order to explain the superficial observational features of a given kind. Putnam defended a realistic reading of these essences, but the notion of natural kind can also be adopted from a non-essentialist position.

According to a non-essentialist view – developed, among others, by Boyd (1991) and Hacking (1991a) – when we are in the presence of clusters of properties that regularly appear together, we tend to look for deeper, unobservable causes, and to propose new (scientific) redescrptions of the phenomena in order to account for what is behind and unifies those observable features. According to this non-essentialist view, this hunger for deeper explanations is a characteristic of our cognitive apparatus, without compromising with the idea that reality actually has any “joints” to be found. In what follows we will remain neutral about this metaphysical question, as it is enough for us to assume that these natural kind concepts include a set of prototypical, superficial, observable features that we think need deeper cohesion and that natural science has the responsibility to find the deep explanatory nature of these clusters of properties. Thus, we will only be concerned with the cognitive bias that led us (individually and as members of the scientific community) to look for deeper explanations of observable phenomena. We will use the expression “essence” (in quotation marks) to emphasize this hunger for an explanation couched in terms belonging to more basic levels, and “stereotype” to refer to the cluster of observable, superficial, phenomenological properties that natural science seeks to explain. It is important to remark that in the course of scientific development we can find divided “essences” for a given stereotype (like in the case of jade), or we can be urged to exclude certain objects from the extension of a natural kind term, even if the stereotype is present, because it does not have the proper “essence” (for example, whales from the kind fish), or we can even be led to modify or refine the stereotype. In sum, some objects could be excluded from a category because of a scientific discovery about the nature of its “essence” and also the stereotype of a natural kind can change when an “essence” is scientifically determined; typically, in the case of medical terms, the “essence” will determine new elements of the stereotype, i.e. of the diagnostic criteria for the disease (including, for example, the result of blood tests in the proper diagnosis of certain diseases).

In the case of psychology, mental concepts have a “stereotype” that includes human behavior. It is not necessary to be a behaviorist to realize that it is imperative to include open behaviors among the evidence we must consider when dealing with mental concepts. Thus, leaving aside for a moment the question of the reference of concepts such as “pain”, “belief”, “fear” or “attention”, it seems clear that in order to use these concepts appropriately and apply them to our fellows, we must take into account the observable (linguistic and non-linguistic) behavior that they display. Within orthodox functionalism – which is predominant in cognitive sciences, including cogni-

<sup>8</sup> For example, water =  $H_2O$ .

<sup>9</sup> This is the case of jade, which has now a divided reference (“jadeite” and “nephrite”) because there are two different chemical structures behind what we phenomenologically identify as jade.

<sup>10</sup> For example, whales were considered big fishes, because they look phenomenologically alike to fishes, but now we learned that they belong to a mammal clade (assuming that clades are the essences of species; see Dupré, 1993 for a discussion about this topic).



tive neurosciences – psychological concepts refer to internal states of the individual that have a causal effect on behavior and are detectable by their effects (under normal conditions, *i.e.* in everyday life, and also in scientific experiments). But we also attempt to access these internal states by other more direct means when we “open the black box” and look for the neural correlates of these internal states; neurological states that supposedly constitute the neurophysiological “essence” of mental states. In parallel with the case of water, some scientists (particularly the cognitive neurosciences) assume that pain, belief, fear, attention and all the other mental concepts have their associated stereotype, including the prototypical behaviors caused by these mental states, and a biological essence that can be known via (neuro)scientific research.

Griffiths (1997) was the first to cast doubt over whether a subset of these psychological concepts (the concept “emotion” and the concepts corresponding to particular emotions such as “fear”, “love”, etc.) could be considered natural kind concepts. In asking this question, Griffiths took for granted that in order to give an affirmative answer it was necessary to find through empirical research an “essence” that stood behind those concepts, that is, a “nature” that unifies the phenomena in question. Griffiths held that “emotion” is not a natural kind because it would be impossible to find a single “essence” for all of the different emotions (he argued that some of them would be better explained by biology, while others would be better explained by the social sciences). But he also argued that basic Darwinian emotions (fear, disgust, anger, joy, sadness and surprise) are in fact natural kinds in so far as biology had located an essence for them: a neurophysiological “affective program.”<sup>11</sup>

There has also been interest in posing a similar question about other types of psychological concepts: those we use to name psychiatric disorders. Hacking (1991b) considers the example of the concept “child abuse,” which seems to be a key notion in the diagnosis of multiple personality disorders, and shows how this concept varied both in extension and intension in the last half century, noting that those changes were not linked to an increase in knowledge of a certain phenomenon through the natural sciences, but due to changes in the cultural and social norms that establish which set of practices were acceptable in a society and which were not. Thus, this is an example of what Griffiths (2004) called a “normative type.” His idea is to highlight that what is behind the phenomenological observable properties of certain kinds may well be

normative types, that is, normative practices that a society has imposed on itself in the course of its history.

As we said above, there are several authors who contrast natural kinds with other types or kinds, such as human or interactive kinds (Hacking, 1991b, 1995), human-made kinds (Ereshefsky, 2004), normative kinds (Griffiths, 2004), historical kinds (Bach, 2012), objective types (Haslanger, 2012), practical kinds (Zachar, 2000), or social kinds (Mason, 2016), to mark the difference between the sciences concerned with human behavior and the natural sciences. In this paper we propose to use the category of “normative kind” to include all those categories that involve normative standards behind their stereotypical features.<sup>12</sup> In the next section we will say more about them.

As an example of a normative kind we can consider ADHD, which is very frequently diagnosed in school-age children. The diagnostic criteria used in this case involve the child’s behavior and the diagnosis is exclusively based on the evaluation that adults (teachers, parents, etc.) perform on the child’s behaviors, indicating what her achievements are, which activities she is able to do and which she cannot, how good she is in doing her tasks compared with the average of other children of the same age, etc. We can contrast this situation with usual physical diseases and with the diagnostic criteria used when we need to diagnose them. Consider, for example, the case of dengue. In this case we have a set of symptoms such as high fever, headaches, vomiting, muscle and joint pains, and a characteristic skin rash, which leads the physician to ask for a blood test in order to find the antibodies to the dengue virus or its RNA (the “essence”) that produces the symptoms. The final diagnosis depends upon the result of the blood test. As Griffiths (2004) points out, the diagnosis of ADHD allows educators, parents and psychiatrists to single out behaviors or a behavioral style that is unacceptable to the teachers and the educational institutions to which the child belongs. But by treating these concepts as “natural kinds” we are assuming that there is a scientific discipline that is responsible for explaining and understanding these phenomena, authorizing the agents involved in the case to perform certain practices, such as medicating the child, and carrying out various types of therapies. The question about the biological essence of ADHD is still an open question, but in our opinion there are solid reasons to believe that the search for biological “essences” in normative kinds such as this is hopeless, as we will argue analyzing in detail another example in #3.

<sup>11</sup> Elster (1999) argues against the idea of thinking of emotions as natural kinds. In the case of propositional attitudes, Pérez (2005) argues in favor of considering them as natural kinds, while Sehon (1997) argues against this idea.

<sup>12</sup> Notice that a normative kind can be applied both to humans and non-humans; in fact, it is applied to all those beings that are constrained in their actions by normative standards. Whether there are non-human beings in the extension of a normative kind is an open question (we are not trying to present a category that is *a priori* applied only to human beings, as Ereshefsky [2004] does). The distinction we have in mind does not pose a division of cultural or social vs. biological behaviors, but of intentionally described behaviors vs. a physical description of the (same?) behavior. In our view, as long as we need an intentional description of what is going on, we are no longer concerned with the biological realm. It is plausible to think that our normative kinds are co-extensive to Ereshefsky’s (2004) “human-made kinds”, but we believe that the label “normative kinds” makes more explicit the precise difference we find between natural and human-made kinds.

## 2 Human behavior

As we argued in the last paragraph normative kinds involve normative standards within the stereotype. But it is not easy to see why. As we said above, the stereotype of all psychological concepts involves human behaviors. Hence, in order to find the hidden normativity we should clarify what we understand by “human behavior.” Trying to answer this question, Kim (2006) identifies three different types of events that are considered public behaviors, namely:

(i) **Physiological reactions and responses:** for example, perspiration, salivation, increase in the pulse rate, increase in blood pressure

(ii) **Bodily movements:** for example, raising and waving a hand, opening a door, throwing a baseball, a cat scratching at the door, a rat turning left in a T-maze

(iii) **Actions involving bodily motions:** for example, typing an invitation, greeting a friend, checking a book out of the library, going shopping, writing a check, signing a contract (Kim, 2006, p. 59).

It is clear that it is possible to find an explanation in biological terms of human behaviors of type (i). But there seems to be solid reasons to doubt that we can find biological explanations of type (iii) behaviors. Type (ii) behaviors are ambiguous: they can be read as cases of type (i) behaviors or as type (iii) cases, since “raising a hand” can be understood as (i) John’s hand raised (because someone else moved it, for example), or as (iii) John raised his hand (i.e. an intentional action that John performed). In our view there is not a third genuinely different type of behavior other than (i) and (iii), so in the rest of the paper we will be considering two types of human behaviors: type A behaviors, which include all physiological responses as well as all involuntary bodily movements, and type B behaviors, which include all intentional behaviors.

Our claim is that all kinds that include type B behaviors in their stereotype are normative kinds, as opposed to natural kinds, which include only type A behaviors. In what follows we will argue that there are strong reasons to think that we cannot find biological “essences” of normative kinds.<sup>13</sup> There are three reasons why biology will not suffice and why intentional/psychological categories will not be replaced by purely biological ones. Two of them are connected with the normative character of type B behaviors.

In the first place we should have in mind that type B behaviors are intentional actions, that is, the type of actions that folk psychology – and, by extension, scientific psychology (following Fodor, 1987) – seeks to explain and predict. And according to a longstanding philosophical tradition, folk-psychological categories have a special normative nature, because the classical explanation for human action comes through practical syllogism, which presupposes the rationality of the agent. In this line, Davidson (1970) and Dennett (1987; 1991) consider that folk psychology (and therefore scientific psychology, as long as it follows the path of folk psychology) is constituted by normative principles, such as the principle of charity or the principle of rationality. This normative character, which governs the use of psychological concepts, is precisely what prevents (as argued by Davidson, 1970; 1973) the formulation of genuine causal laws involving psychological states and thus any attempt to reduce psychology to the natural sciences. As Davidson (1973) says, “detailed knowledge of the physics or physiology of the brain, indeed of the whole man, would not provide a shortcut to the kind of interpretation required for the application of sophisticated psychological concepts” (Davidson, 1973, p. 258), including among them “intention, belief, [...] desire, [...] action, decision, memory, perception, learning, wanting, attending, noticing, and many others” (Davidson, 1973, p. 246). Assuming a similar view, Graham (2010) defines mental disorders in terms of a “disability, incapacity or impairment in the rational or reason-responsive operation or exercise of one or more fundamental mental faculties or basic psychological capacities of a person” (Graham, 2010, p. 156), arguing that because of this presupposed rationality and values, they cannot be reduced to brain disorders (Graham, 2010).

Secondly, it is important to notice that even those who deny the normative character of folk psychology but accept the functionalist thesis – as all cognitive scientists do – that proposes the multiple realizability of mental states also hold that psychological laws cannot be reduced to (that is, they are not able to be rewritten in terms of) the neurophysiological realizators of these mental states, given the existence of normal conditions (or *ceteris paribus* clauses) in psychological laws, that prevent their reduction to biological ones (Fodor, 1974; 1987; 1991). In sum, even those who argue that it is possible to find scientific (causal) laws that link psychological states with behaviors do not believe that these laws can be rewritten in biological terms.

In the third place, it is important to point out that, in the case of scientific laws that involve type B behaviors, the determination of which behaviors are considered “normal” (that is, how the “normal conditions” are determined) does not depend upon statistical analyses. On the contrary, the behaviors that are considered “normal” are the expected behaviors in the context of the social norms established by the culture / society in which the individual is immersed (for example, it is considered an abnormal behavior for a school child not being seated on a chair for eight hours a day as educational policies prescribe). In other words, in the case of type A behaviors, it is very clear what it means to consider them as “abnormal” (e.g. the rising of the heart rate or the pulse above a certain level),

<sup>13</sup> By “biological essences” we mean purely neuro, endocrino and/or genetic facts that explain the stereotype features of that kind.

because these are behaviors that conspire against the health of the individual. But that is not clearly the case concerning type B behaviors (e.g. if we think about homosexual behaviors, to give an example of something that at some point in our cultural history – not very long ago – was considered a disease); it seems that they are “abnormal” in another sense. The fact that they are considered “abnormal” by a certain society at a certain moment is related to the values and practices that are approved or disapproved by that society, not to their relevance to the health of the individual.<sup>14</sup> The explanation of this kind of behaviors requires more than just biological facts.

In sum, it is not clear that there are laws involving type B behaviors. If such laws existed, it is not clear either that they could be reduced to laws involving internal states of the subject couched in non-psychological terms (that is, in biological, neurophysiological, chemical, genetic or hormonal terms, etc.). There are good reasons to think that the “essences” of psychological states cannot be wholly formulated (unlike the “essence” of water) in terms of another (more basic) scientific discipline. And finally, even if there were laws that might be statistically representative of the typical actions of human individuals, there is no guarantee that such statistical generalization does not hide norms, values and social practices that do not depend upon the biology of the subjects, but are the result of the actual history of the society / culture in which individuals are immersed.

### 3 Autism Spectrum Disorder: A study case

In this section we will take a look at a paradigmatic psychological/psychiatric concept: “Autism Spectrum Disorder” (ASD). The diagnostic criteria for mental disorders such as this are, usually, type B behaviors. In this section we will analyze the strategies used in biomedical research in order to find specific etiologies (“essences”) to explain such disorders. We will argue that these research programs are inconclusive and that the lack of biomarkers that are clinically useful for refining the diagnoses is due to the fact that, unlike in certain neuropathologies, there are no physiological essences that can fully explain ASD symptoms. In our view, as we are dealing with type B behaviors as part of the stereotype for ASD, we had better interpret this mental disorder as a normative kind.

In the previous paragraphs we pointed out that we could find biological causal agents for diseases whose symptoms (or

diagnostic criteria) are type A behaviors. In this regard, we described why dengue might be interpreted as a natural kind. We can also extend this label to certain diseases that concern the brain, such as meningitis. Such diseases – dengue and meningitis – are caused by a specific external agent (a virus, a bacterium, or a toxic substance), which precedes and triggers the symptoms (fever, headache).

There are also some neurocognitive disorders that have specific anomalies in certain areas as brain correlates. For example, in Alzheimer’s and Parkinson’s diseases a specific physical deterioration is observed as a predecessor of cognitive deficits.<sup>15</sup> In general, neurodegenerative diseases represent a spectrum characterized by the accumulation of certain proteins, which form aggregates and precipitate in certain neuronal populations, and are subdivided on the basis of clinical presentation, deposition of the protein and cellular and sub-cellular pathology (Brett & Kearney, 2017). In other words, these diseases are triggered by specific internal factors. In this sense, they count as natural kinds because there is a biological explanation for some type A behaviors that constitute the stereotype, for example the tremors that are characteristic consequences of the progressive neuronal deterioration that accompanies Parkinson’s disease.

In our opinion, the cases mentioned above are examples of natural kinds; the stereotype includes type A behaviors (fever, headache, tremors) that can be explained by an essence which can be completely described in biological terms, either by external factors, as in the case of meningitis, or by internal factors, such as the protein aggregate that is characteristic of neurodegenerative diseases. Moreover, if the stereotype retains, from its characterization prior to the discovery of the biological “essence,” type B behaviors such as memory loss (a cognitive deficit), that is because memory loss is directly associated with a specific physiological deterioration that precedes and triggers it, affecting the functions of certain neuronal populations on which these impaired cognitive functions depend.

In contrast, mental disorders – so called because their specific triggering agents are unknown – do not seem to be diagnosed in relation to type A behaviors. In these cases, there are no precise symptoms such as fever, headache, or tremors. Even cognitive deficits such as those associated with biological dysfunctions – for example, memory loss – do not appear to be present. On the contrary, we cannot do anything but to appeal to type B behaviors in order to diagnose these disorders. But interpreting them as natural kinds implies that we will be able to find behind type B behaviors a biological causal agent

<sup>14</sup> It is not even clear that it is statistically “abnormal” to carry out homosexual behaviors (nor is it clear enough what behaviors should exactly count as “homosexual”). There are no reliable statistics (to our knowledge) regarding how common homosexual behavior is in our society, and even if it were true that it represents a minority in statistical terms, it would not be clear that its statistically low presence is not the *result* of negative social pressures that these behaviors usually receive, as opposed to their being based on facts about our biology. (Notice that we are talking about “homosexual behaviors”, not about gender identity.)

<sup>15</sup> DSM-4 classified them as Alzheimer’s dementia and dementia due to Parkinson’s disease. But they have been reclassified in DSM-5 as neurocognitive disorder (NCD) due to Alzheimer’s disease and neurocognitive disorder due to Parkinson’s disease. The change is due, in part, to the fact that, although it is not understood how the deterioration of cognitive functions is triggered, there is a concrete physical deterioration.



capable of explaining them, and we have called into question this possibility in the previous section.

Given that specific etiologies for mental disorders are not known, there are no blood tests or similar diagnostic methods; therefore, the diagnosis is based on behavioral observation (and self-report). In order to create a unified criterion, there are reference diagnostic frameworks. The two broadly accepted ones are the Diagnostic and Statistical Manual (DSM) edited by the American Psychiatric Association (APA) and the International Classification of Diseases (ICD) approved by the World Health Organization. The descriptive perspective they assume raises the need to develop new theoretical and etiological approaches in order to validate these diagnostic criteria (Tsou, 2016).<sup>16</sup> New classification proposals that incorporate biomarkers as an objective condition for disorders have become the new scientific challenge for the upcoming DSM and ICD updates (Lord & Jones, 2012); however, there is skepticism regarding this project (Hyman, 2014).

In what follows we will analyze, as a paradigmatic example, the Autism Spectrum Disorder (ASD). It is a paradigmatic case due to its great interindividual variability. In effect, the word “spectrum” in this kind of cases refers to the high heterogeneity that usually characterizes the presentation of clinical cases.

The description made by DSM-5 for ASD is based on two central criteria, both of which include type B behaviors. Criterion A includes persistent deficits in social communication and social interaction, and criterion B includes restricted and/or repetitive patterns of behavior, interests or activities. It is interesting to note that the latest update of DSM incorporated two new diagnostic labels: Disorder in Social Communication (SCD), for those people who only meet criterion A, and Disorder of Stereotyped Movement (SMD) for those who only manifest criterion B. Although both criteria can coexist by coincidence in an individual, ASD, SCD and SMD are considered as three different disorders because it is assumed that each of them has a characteristic and different etiology. For this reason, ASD has not been replaced by SCD plus SMD. That is, both criteria would be present in ASD, but due to a different specific etiology (Inui *et al.*, 2017, p. 2).

As we will show in detail below, it has not yet been demonstrated that there is a causal relationship between the physiological correlations that have been associated with autistic behaviors and people who have been diagnosed with this disorder. In other words, despite great efforts to find a specific etiology triggering ASD, a biological “essence” that might ex-

plain the stereotype has not been found yet. We will review the main initiatives that have been developed to find the biological “essence” of ASD and the difficulties that such studies have had; the first one involves the search for specific genes, while the second focuses its efforts on trying to find specific brain structures and neuronal activation patterns. We are aware that we do not have a knockdown argument because it will always be possible to argue that more time is needed in order for these research programs to find the results they are looking for. But the failure in these cases, along with the fact that with the same technologies in other fields – such as neurodegenerative diseases like Alzheimer’s and Parkinson’s – these research programs were successful, plus the *a priori* arguments we made in the previous section about the difficulties of understanding in biological terms some specific behaviors (type B), leads us to suggest that we had better think of these behavioral patterns as revealing normative kinds.

### 3.1 Searching for genetic “essences”

In order to find the molecular mechanisms that cause ASD, numerous lines of research were looking for anomalies, both at the genomic and cerebral levels, comparing “control” subjects with those who were diagnosed with ASD. Given the fact that all these studies only include a small number of clinical cases, several initiatives have emerged which concentrate the data that has been obtained from those studies, increasing their statistical power. Psychiatric disorders are characterized by high comorbidity. For this reason, the Cross-Disorder Group of the Psychiatric Genomics Consortium (PGC) used genome-wide genotype data from case-control groups for schizophrenia (SZ), bipolar disorder (BP), major depressive disorder (MDD), autism spectrum disorders (ASD) and attention-deficit / hyperactivity disorder (ADHD) in order to examine the genetic etiology shared by such disorders (Docherty *et al.*, 2016, p. 3). Mental disorders are also characteristically polygenic. That is, in each disorder there will be thousands of genes involved, each one having a very small effect. In this sense, the predominant hypothesis to explain comorbidity is that there may be genetic overlaps between two disorders, resulting in a pleiotropy that would explain why a person might be diagnosed with two disorders (*ibid.*, p. 2). In the same direction, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria (RDoC) project, which aims to “understand the nature of mental health and illness in terms of varying degrees of dysfunctions

<sup>16</sup> Although Tsou considers that certain disorders described in DSM are not natural kinds, he affirms the existence of others that are, such as schizophrenia and depression (Tsou, 2016). His criterion to distinguish disorders that would actually count as natural kinds from those that are not does not depend, as we suggest, on the presence of type B behaviors in the stereotype, but on the possibility of discovering underlying biological mechanisms that account for the stereotype. But currently there are no biomarkers with clinical application for mental disorders, such as schizophrenia, contrary to what Tsou suggests. It is still a project under development to increase the statistical power of the information available about persons diagnosed with schizophrenia in order to find biomarkers that are clinically useful (see <https://www.natureindex.com/institution-outputs/united-states-of-america-usa/international-schizophrenia-consortium-isc/537ab-da2140ba03066000008>, as well as the Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia, based on the search for cognitive and imaging biomarkers, <http://cntrics.ucdavis.edu/>).

in general psychological / biological systems.”<sup>17</sup> A database was created for this purpose, integrating information at many levels: from genomics, through neuronal circuits, to behavior and self-report. Numerous studies have compared the genomes of people diagnosed with ASD, finding variants in the number of gene copies [CNVs], as well as rare single-nucleotide variants (SNVs), both inherited and *de novo* (not present in either parent). However, the heterogeneity of clinical cases has an impact on the genetic studies; there is a multiplicity of genetic patterns that have been associated with ASD, in addition to the lack of specificity of the genes supposedly involved (overlapping with other disorders) (Lord & Jones, 2012, p. 2). In addition, the supposed genetic risk factors found for ASD can be found in the general population, that is, in people who have not manifested any psychiatric symptoms (Robinson *et al.*, 2016). Moreover, the classic studies in twins analyzing inheritance are not univocal. In fact, there are works that suggest that the inheritance of ASD would be approximately 38%, while shared environmental factors would influence 58% (Goldani *et al.*, 2014, p. 2). In this case it was concluded that the epigenetic factors were predominant.<sup>18</sup> A more recent large population-based epidemiological study of roughly 2.5 million families seems to have provided clear evidence for a genetic component of ASD, estimating the disease’s heritability at around 50% to 95%, making it one of the most heritable of neuropsychiatric disorders. Nevertheless, in all these studies, the phenotypic concordance between monozygotic twins is incomplete, indicating that nongenetic environmental factors do play a role in the etiology of ASD (Chahrour *et al.*, 2017, p. 335).

Given the results mentioned so far, we think that there are reasons to cast a doubt on the idea that there is a genetic essence for ASD. In the first place, we would like to emphasize that epigenetic factors, even if they have a clear influence, are never considered as the main causal factor. They are usually interpreted in a biological way – as the gene “environment” – resulting in an “autistic phenotype.”<sup>19</sup> But, in our view, given the fact that epigenetics incorporates non-biological factors (Fine *et al.*, 2017), both genes and epigenetic factors broadly constructed – and not genes alone – are the emergence base of what we prefer to call the “autistic stereotype” (instead of “autistic phenotype”).

Secondly, it is not clear that ASD is caused by genetic mutations. SNVs arise in more than one form in the population and these different forms are not signs of either disorder or dysfunction, but they simply reflect natural variation (Baron-Cohen, 2017, p. 744). In addition, the presence of rare

genetic mutations that could cause severe dysfunctions was not found in people diagnosed with autism (*ibid.*). Moreover, as Chahrour affirms, “although the overall burden of *de novo* CNVs is higher in affected than in unaffected individuals, many of the same CNVs also occur in the unaffected individuals, making it difficult to determine which changes are likely to be disease-causing. The heterogeneity of CNV-associated phenotypes can also be manifested within a single family as a result of unidentified modifiers” (Chahrour, 2017, p. 336). The majority of CNVs have very low recurrence in ASD, and a specific CNV can often be unique to a single patient (*ibid.*). Indeed, it has been found that *de novo* mutations collectively explain less than 5% of overall ASD liability, which leads to the conclusion that “almost all genetic risk factors for ASDs can be found in unaffected individuals. For example, most people who carry 16p11.2 deletion, the most common large mutational risk factor for ASDs, do not meet criteria for an ASD diagnosis” (Robinson *et al.*, 2016, p. 2).

Finally, if both the genetic factors and the proposed epigenetic factors do not result in an univocal phenotype, it is necessary to call into question the idea of an “autistic phenotype,” which presumes the existence of biological mechanisms underlying the behaviors labeled as autistic. Instead, we propose that the heterogeneity of clinical cases shows that type B behaviors are not susceptible to being rewritten in biological terms. The lack of phenotypic concordance, even between monozygotic twins, works as a strong evidence that supports our proposal. In the light of all these findings, there are reasons to think that a genetic “essence” for ASD will never be found.

### 3.2 Searching for “essences” in the architecture and functioning of the brain

Autism Brain Imaging Data Exchange (ABIDE) is the main project devoted to the study of the architecture and functioning of the brain in people with ASD. Its goal is to enhance the scope of brain connectomics research in Autism Spectrum Disorder.<sup>20</sup> There is plenty of evidence of abnormal neuronal connectivity in people with ASD (Di Martino *et al.*, 2017). In this sense, non-invasive brain imaging techniques seem to offer a great promise for discovering patterns in brain structures and functioning that could be used as objective measures of this mental disorder. Among such techniques, R-fMRI is presented as the key candidate to define functional neurophenotypes (*ibid.*). The ABIDE I initiative, launched in 2012, was the first to enable a site of free access

<sup>17</sup> <https://www.nimh.nih.gov/research-priorities/rdoc/index.shtml>.

<sup>18</sup> Epigenetics refers to how the environment, which includes our practices and lifestyle, impacts on the expression of our genes.

<sup>19</sup> We would like to emphasize that the term “phenotype”, widely used in scientific literature to refer to behaviors labeled as autistic, implies *per se* an understanding of such behaviors in biological terms, because this concept refers to the visible characteristics of an individual – such as eye color – resulting from his genetic constitution and the interaction with the environment. We use quotation marks in order to stress that it seems doubtful to talk about phenotypic type B behaviors.

<sup>20</sup> [http://fcon\\_1000.projects.nitrc.org/indi/abide/](http://fcon_1000.projects.nitrc.org/indi/abide/).



to brain images obtained through functional magnetic resonance in resting state (R-fMRI) of the connectome of people with ASD. Collecting data from different research centers, it reached an N = 539 and N = 573 for people diagnosed with ASD and controls, respectively. To increase the size of the sample and to help discover the neuronal correlations in people diagnosed with autism, an ABIDE II provided a new data source, adding a N = 487 for people with ASD and a N = 557 of control subjects (Di Martino *et al.*, 2017, p. 2). Two sources of heterogeneity present in ABIDE I would make ABIDE II a more reliable source. First, it analyzes the disorders that co-occur with ASD (comorbidity), something that has been largely ignored in the neuroimaging field. In this sense, ABIDE II actively encouraged researchers to provide phenotypic information regarding co-occurring illnesses, if assessed (*ibid.*).<sup>21</sup> This is in tune with the initiatives described above regarding genomic comparisons between different disorders to analyze comorbidity. Secondly, ABIDE II considers sex bias due to the prevalence of ASD in men. This causes women to be excluded, or minimally represented, in studies. For this reason, from N = 65 for women with ASD in ABIDE I, ABIDE II has increased it to N = 138 when I and II are combined. This increase would facilitate finding risk and protection factors specific to the sexes, providing information about the molecular mechanisms underlying ASD (*ibid.*).<sup>21</sup>

In relation to neuronal correlates based on the repository available in ABIDE, one study used R-fMRI in 697 participants to detect useful biomarkers with clinical applications. It is important to note that the total number of participants resulted from the exclusion of those who did not meet the diagnostic criteria used by that particular study. ABIDE data comes from multiple simple study sites, each study having its own methodology and involving variables that limit the validity of grouping data in a common study. We emphasize this fact because, in general, the heterogeneity between the experimental procedures of each study presents a challenge for research that aim to develop brain markers for psychiatric disorders. The most common variables range from MRI acquisition protocols (e. g. scanner type, imaging sequence) to participant instructions (e. g. eyes open vs. closed) to recruitment strategies (age-group, IQ-range, level of impairment, treatment history and acceptable comorbidities) (Abraham *et al.*, 2016, p. 2).

According to the results of this study, a 67% prediction was obtained, the largest one predicted from ABIDE (Abraham *et al.*, 2016, p. 9). The predictions were based on the intrinsic functional connectivity of three functional systems

that have been noted as having a decreased connectivity in people with ASD (Abraham *et al.*, 2016, p. 10). However, even if there are such correlations, it is not clear that we are dealing with a causal/explanatory biological essence of the symptoms (the ASD stereotype). An evidence for our claim is that no biomarkers have been found for any mental disorder that, in contrast with neuropathologies such as Alzheimer's and Parkinson's, is characterized by not having specific phenotypes.<sup>22</sup> In this sense, the need to find "candidate biomarkers" from which to refine the diagnosis is hampered by the heterogeneity that characterizes the clinical cases of psychiatric disorders. This fact is especially clear in ASD, in which case, despite having a vast amount of scientific literature that describes it in fundamentally biological terms, we have no biomarkers of clinical utility for its detection (Bargiela *et al.*, 2016, p. 3292).

At this point it is important to remark that some authors suggest that ASD is not a disorder, i.e. something that would need treatment. Instead, they have characterized it as a different way of functioning, a "cognitive style" (Happé, 1999), due to "neurodiversity" (Baron-Cohen, 2017), and not as a dysfunction. This is because people diagnosed with ASD are sometimes better than average people in certain cognitive domains, such as attention to details, memory for details, the ability to detect patterns or to systematize them (Baron-Cohen, 2017, p. 744). Moreover, Baron-Cohen argues that

*Autism is not alone in DSM-5 in being called a disorder. Since DSM-1 in 1952, when there were 106 disorders listed, there has been a steady increase, and when DSM-5 was published in 2013, the number had reached 300. It is unlikely that DSM really 'carves nature at its joints', as Plato recommended our best classificatory and explanatory theories should, if we can keep adding or subtracting diagnostic categories each time a new edition of DSM is published. Recall how homosexuality was classified as a disorder in DSM-I and DSM-II, until civil rights protests succeeded in having it declassified from DSM-III in 1980, on the grounds that it is just a natural example of the diversity of sexual orientations that exist in any population (Baron-Cohen, 2017, p. 744).*

As we can see, there are authorized voices that have put into doubt the idea that DSM includes only natural kinds. However, the notions of cognitive style and neurodiversity are still interpreted as functional differences *due to* different

<sup>21</sup> We do not accept the idea that the existence of a prevalence in one or the other sex legitimizes the view that there is a biological origin that explains ASD. We suggest that there are other possible interpretations of this prevalence that include both the gender bias in the diagnosis and the gender bias in the research studies, but developing this suggestion is beyond the scope of this paper.

<sup>22</sup> Although there are currently no biomarkers that can be used in the clinic to predict Alzheimer's and Parkinson's diseases before neuronal deterioration begins to appear, a great advance has been made in finding proteins and specific candidate genes associated with this deterioration (Redensek *et al.*, 2018; Shui *et al.*, 2017).

biological constitutions. That is, it is assumed that genetic variables and different brain structures and functions are the causal agents of the “autistic phenotype,” only questioning their labeling as “anomalous.” In short, the discussions are either about which ones are to be considered disorders or not (Happé, 1999; Baron-Cohen, 2017), or which ones are susceptible or not to be categorized as natural kinds (Tsou, 2016). However, the validity of assigning biological essences to psychological categorizations whose stereotype includes type B behavior, whatever they are, interpreting them in terms of phenotype, is not questioned.<sup>23</sup> Our proposal is, in this sense, more radical: where we have type B behavior as part of the stereotype of a given kind it would be impossible to find a purely biological explanation.

## 4 Conclusion

As we said in the previous paragraph, type B behaviors are used in order to classify mental disorders and cognitive styles. But the labels proposed have changed in short periods of time, some of them were corrected, others added, some deleted, some considered sometimes as identifying diseases and sometimes only as identifying different lifestyle patterns. As we said in paragraph 2, we hold that those categories that involve type B behaviors in their stereotypes should not be considered as natural kinds, and hence we should not look for their biological “essence.” On the contrary, we should consider them as normative kinds and acknowledge that there will always be normative standards involved in these categories, which rule out the idea of finding a purely biological essence for them. It is apparent that our experiences, the habits we acquired, the abilities we learn in our lives are incorporated into our neuronal wiring and affect the expression of our genes, our brain architecture and our neural circuits. The uniqueness of our brains transcends the normative categories created to describe them. We propose this form of reinterpretation in the case of ASD because it is a paradigmatic case: exceptions are the rule in relation to the diagnostic criteria described by DSM, and several voices have been raised against the very idea of autism being a mental disorder. In our view, a diagnosis based on type B behaviors involves normative ideals to which the real behaviors of the diagnosed people are then adjusted. In this sense we hope that the distinction between normative kinds and natural kinds will contribute to the proper treatment of these types of cases, highlighting that more reflection about the social practices in which we are involved is needed.

<sup>23</sup> Along the same lines, it is remarkable how, despite the fact that homosexuality has been excluded from DSM, some researchers insist on looking for biological bases of type B behavior patterns, such as human sexual behavior. For example, sexual orientation continues to be characterized by current neuroendocrinological discourse as a behavior dependent on prenatal androgen levels (Hines et al., 2016), that is, it is explained in terms of innate biological programming. In the same line, numerous studies have focused on the search for structures and brain functions in order to explain and classify different types of homosexuality (LeVay, 2011). In addition, non-normative gender identities continue to be in DSM, trans people being pathologized, again proposing an explanation in terms of the impact of hormones on the brain during fetal development (García-Falgueras & Swaab, 2010).

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