21 Autistic Spectrum, Normal Variation, and Harmful Dysfunction

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> If I could snap my fingers and be nonautistic, I would not. Autism is part of what I am.
> —Grandin (2006)

Introduction

Since the pioneering work of Leo Kanner (Kanner 1943), the diagnosis of autism has been based on the so-called autistic triad, that is, the reunion of three types of features: impairment of social development, impairment of communication, and display of rigid and repetitive behavior (Baron-Cohen 2008). This mix of deficiency and oddity has been the basis on which autism has been identified as a developmental *disorder*. Research on autism has not only increased our knowledge on this condition in innumerable ways but also resulted in a more problematic picture of it. First, instead of a sharp contrast between autistic and nonautistic people, research has pointed out the presence of autistic features within some parts of the nonautistic population—which has led to the introduction of the notion of a broader autistic phenotype (Piven et al. 1997). Second, we have now good reasons to recognize a marked heterogeneity within what is called, since the work of Allen (1988) and Wing (1997), the autistic spectrum: high-functioning autism, where general intelligence is preserved, is quite different from "classic autism," and specialists are not unanimously convinced that the term "disorder" applies to it.[1] This evolution toward a greater recognition of preserved (or even enhanced) abilities of individuals in high-functioning autism has coincided with the development of the neurodiversity movement, which developed in the 1990s through the activity of online groups of high-functioning autistic persons (Ortega 2009; Jaarsma and Welin 2011).[2] The advocates of neurodiversity claim that nonautistic or, as they call them, "neurotypical" people have a negative bias against autistic persons and that we should stop confusing mere difference with genuine deficiency. So we have moved from a world where autism was a relatively well-defined medical category to a different universe marked by clinical heterogeneity, elusive boundaries, numerous first-person

accounts of how autistic persons experience the world (Grandin 2006, 2009), and finally, controversy about the very application of the term "disorder" to autism.

According to Jerome Wakefield, "a disorder is a harmful dysfunction, wherein harmful is a value term based on social norms, and dysfunction is a scientific term referring to the failure of a mental mechanism to perform a natural function for which it was designed by evolution" (Wakefield 1992a, 373). Purely normative views of mental disorders, according to Wakefield, fail to recognize that function and malfunction are independent from our values, norms, and preferences; they miss the crucial point that there are objective, natural facts that ground our medical judgments. In this chapter, I want to confront the harmful dysfunction view (mentioned hereafter as HD analysis) and the contemporary debate about autism that revolves around two key questions: how should we explain autism, and should we think of it in terms of difference and normal variation, on one hand, or deficiency and disorder, on the other hand?

In a very thorough examination of HD analysis (Poland 2002), Jeffrey Poland has made a useful distinction between two projects that, according to him, coexist within the HD analysis: one is a descriptive project, "to reconstruct as accurately as possible the commitments of contemporary mental health practice regarding the concept of mental disorder," and a normative project, "to identify a set of conceptual commitments that ought to inform contemporary mental health practice." The descriptive project deals with scientific psychiatry as it is, the normative project, with scientific psychiatry as it should be. The content of this chapter will echo this distinction, and I shall answer two different questions. First, there is the descriptive issue: is research about autism and its explanation concerned with the discovery of dysfunctional mechanisms, where "dysfunctional" has the precise meaning that is attached to it within the framework of HD analysis, and are dysfunctions currently described independently from background normative judgments? The answer I intend to provide is negative: in the literature, with few exceptions, "dysfunction" of psychological or neural mechanisms is not understood as their failure to perform what they have been "designed" to do (section I). Moreover, factual judgments about impaired performance and dysfunction are usually inseparable from implicit evaluative claims (section II). My second question is as follows: is the definition of disorders as harmful dysfunctions helping us to settle the debate about deficiency versus diversity, by providing us a standard for the application of the notion of disorder?[3] To this second question, my answer will also be negative, especially because in the context of developmental disorders like autism, what counts as "harm" is less dependent on "social values" than what is required by HD analysis (section III). The suggestion that goes with these negative answers is offered as revisionary rather than radical: I do not suggest that we should give up the core of HD analysis (combining dysfunctions and their harmful consequences) but rather that we should analyze functional talk and harm in a different way. In a nutshell, I shall advocate a more mechanistic view of dysfunctions and an understanding of harm in terms of diminished ability.

I. Psychological Theories of Autism: What Is Dysfunctional, and in What Sense?

If we analyze some of the cognitive theories of autism that have been the most widely discussed in recent years, it seems to be an easy task to reconcile their construal of autism with HD analysis. My reason to start with psychological theories is that their common ambition has been to go *beyond* mere diagnostic criteria and to characterize underlying psychological *mechanisms*, that is, to offer causal explanations of autism involving some kind of disturbance at the psychological level. These theories are the mindblindness theory (Baron-Cohen 1995), the weak coherence theory (Frith 1989; Frith and Happé 1994), and the executive dysfunction theory (Ozonoff et al. 1991; Hill 2004)—for the sake of simplicity, I shall leave aside the empathizing-systemizing theory (Baron-Cohen 2009) that refines on the mindblindness theory.

1. According to the *mindblindness theory*, what is central to autism is the inability to ascribe correctly beliefs and desires to people in order to explain and predict their behavior or, as it is often said, to "read their minds." In its stronger form, the theory postulates that there is a mental mechanism dedicated to the ascription of mental states to others that takes the form of a cognitive module (a psychological faculty with its own cognitive domain and its specific operations, which entails the possibility of its own internal breakdown). This capacity is called in the literature the theory of mind (ToM) module (Baron-Cohen 1995). The idea is roughly that in the autistic mind, because of a defective ontogenetic history that does not lead to its proper development, the ToM module is not working the way it is supposed to.

2. According to the *executive dysfunction theory*, to understand autism, we have to focus on what is called executive function, that is, the ability to control action—where action may be the movements of the body but also the thoughts of the subject. The creation and execution of plans, the ability to stay focused on a given topic or to shift attention, presuppose the integrity of the executive system. Concerning autistic disorders, the executive dysfunction theory aims, in particular, at explaining repetitive behaviors and narrowed interests.

3. According to the *weak coherence theory*, there is a standard human form of information processing that is crucial to the construction of complex structures; it makes possible both to build and to recognize organized wholes, to memorize complex patterns (rather than their discrete elements). Autistic people would be specifically impaired in these information-processing mechanisms, and their levels of performances in all sorts of cognitive tasks (pattern recognition, memory of meaningful sentences, parsing of sentences) would be evidence of that.

In each case, what is postulated by the theory is that there is a psychological mechanism that fails to perform its function, whether it is the function of controlling action, of ascribing beliefs and desires to others, or of integrating details within coherent wholes. Accordingly, autism would be based on the dysfunction of some kind of

psychological mechanism, and in each case, the dysfunction would produce *harmful* results (because one values the ability to control one's actions, the understanding of others as well as related social skills, or the ability to process information in a coherent manner). Explaining autism would consist in identifying the underlying dysfunction(s) that lead(s) to clinically significant, harmful consequences. Of course, advocates of each theory underline that there is much more to the symptoms of autism than what a rival theory can account for, but all such theories and their more recent counterparts share the same goals.

Objections that can be raised against this type of explanation are not necessarily a problem for HD analysis. For instance, concerning the mindblindness theory, the reduced social interactions of autistic children could be explained by a low-level deficit in the perception of social cues, rather than by a higher-level deficit in mentalizing per se (Gerrans 2002). It would be the lack of relevant input, rather than the dysfunction of a dedicated mechanism, that would carry the burden of the explanation: the theory of mind module, then, would become an unnecessary theoretical construct. But HD analysis is not committed to the prediction that, for any mental disorder, the underlying dysfunction is the dysfunction of a specialized mental mechanism (Wakefield 2000). If we give up the theory of mind module, this does not by itself compromise the application of the harmful dysfunction view to autism. However, this kind of immunity to refutation may be a sign of epistemic weakness, rather than a strength. The notion of a dysfunctional "mental mechanism" that would have been "designed" reflects the conviction that first, there must be some objective basis of mental disorders that is independent from our values and expectations and, second, that mental mechanisms are the product of evolution, like other biological mechanisms. Speaking of dysfunction, then, is rejecting relativism and being committed to the idea that mental disorders have a biological nature, but it is not much more than that.

Now, to use Poland's distinction quoted above, is it legitimate to say that mental disorders are currently defined in terms of harmful dysfunctions where "dysfunction" refers to the failure of a mental mechanism to perform "a natural function for which it was designed by evolution"? Mindblindness theory of autism can be (and has been) construed within an evolutionary framework.[4] It is possible to give to "dysfunction" the kind of meaning that *befits* HD analysis. But as it has been pointed out several times by critics of Wakefield's views (Murphy and Woolfolk 2000; Poland 2002; Murphy, this volume), functional talk can have a different meaning, and functional ascriptions may answer different kinds of explanatory purposes. Cummins's view of functions (Cummins 1975) according to which the function *F* of a component *C* is its contribution to the explanation of a capacity of the system in which *C* is embedded seems appropriate for psychological as well as physiological mechanisms. When a scientist tries to explain perseverations and narrowed interests in terms of the dysfunction of an executive system (which would usually monitor and controls action planification), the function

of such a system is not seen as the reason why such a system has been recruited by natural selection. In more mechanistic terms (Machamer et al. 2000), its function is the contribution it makes to the behavioral and psychological repertoire of similar individuals belonging to the same species. The ascription of a dysfunction is not based on Darwinian speculation but on the contrast between a shared ability and an (intriguing) disability. So when we think of an "executive dysfunction theory of autism," we do not (have to) care about the past contribution of this system to the reproductive success of our ancestors. Research related to the executive function theory does not even try to address these issues. What counts is the explanatory value of a functional analysis that postulates an executive system and how the dysfunction of such a system (its failure to do what it usually does in humans, not what it has been designed to do) is able to help us to understand where autistic features come from. Even the mindblindness theory is, in practice, a theory of the *ontogeny* of mental mechanisms (Baron-Cohen 1995). Evolutionary psychologists can speculate about the origins of the theory of mind module, but this entity is useful in cognitive psychology only if it fits in a plausible mechanistic decomposition of the mind. The reference to an evolutionary background remains quite idle in this context.

Moreover, to be able to distinguish between functioning and malfunctioning in Wakefield's sense, we would have to know *when* an evolved mechanism is not doing what it has been designed to do and what the corresponding normal range of variation is in terms of level of performance (Schwartz 2007). Talking about failure and disability leads us to ignore the fact that cognitive tests usually do not offer evidence of a complete *lack* of ability in autistic persons, even for tasks where they are known to be at a disadvantage. For instance, in a task of sentence comprehension for which autistic and nonautistic subjects were tested (Just et al. 2004), error rates were only slightly different: error rates were 8% and 13% for the autistic group (for active and passive sentences), when they were 5% and 7% for the control group. We can interpret this result as the sign of a cognitive dysfunction as evidence of an impaired linguistic ability. But we could also consider (as the different pattern of brain activation in the autistic group suggests) that autistic people use a different cognitive strategy that is quite effective in a *majority* of cases: where shall we find reasons to justify our claim that an error rate of 5% indicates a level of performance that deserves to be called normal (the mind, then, is working "as designed") and that an error rate of 8% is equivalent to a cognitive dysfunction? There is a danger of circular reasoning here, because tests are there to determine what is impaired in the autistic mind, but the slight difference in the results is interpreted as a sign of disturbance *because* the subjects are known to be autistic and their faculties are presumed to be impaired. Adding evolutionary considerations will not help us to break this kind of circle.

To sum up, it is true that occasionally, the dysfunction of the theory of mind module has been conceived in the literature on autism as "the failure of a mental mechanism

to perform a natural function for which it was designed by evolution." But on one hand, we have to remain cautious about the kind of just-so stories that proliferate in evolutionary psychology (Richardson 2010). And on the other hand, concerning the cognitive explanations of autism in general, there is no systematic reference to such an evolutionary background, and more important, such a background plays no special role when the merits and flaws of these cognitive explanations are discussed. We could add that what is true of psychological explanations would also be true of neurocognitive accounts of autism, like the so called broken-mirror view (Ramachandran 2011): it is quite easy to speculate both on the evolutionary history of mirror neurons and on the cognitive impact of the disruption of the mirror system, but in the end, all that matters is how relevant the functioning of the mirror system is to the causal explanation of autistic symptomatology (Hickok 2014).

II. Normality and High-Functioning Autism

In the previous section, I have tried to establish that cognitive explanations of autism are developed without being integrated to an evolutionary framework where dysfunction has the precise meaning that Wakefield is ascribing to it. But now I would like to follow a different strategy. Let us suppose that, in conformity with HD analysis, what mental mechanisms usually or typically do in the general population is what they have been designed to do. In nonautistic people, key mental mechanisms would function as designed, and in autism, the same mechanisms would fail to do what they are designed to do. Does it help us to solve the difficult problems linked today to the autistic phenotype in its wide diversity?

Let us take, for instance, the weak coherence theory. As we have seen, it links key aspects of autism with a definite kind of malfunction of psychological mechanisms. We could call its main hypothesis the central coherence view of cognitive processing (CCV). CCV is explicitly presented as a view of "*normal* [emphasis added] information processing" (Frith and Happé 1994). The meaning of "normal" here is statistical: when Frith and Happé mention, for instance, "the ease with which we recognize the contextually appropriate sense of many ambiguous words used in everyday speech," *they* clearly refers to what *most of us* are able to do *most of the time*. In the same paper, central coherence is even called a "universal feature of human information processing." But here normal has also, obviously, an *evaluative* character: processing information the way we do yields special benefits; it enables us to construct complex representations, to get the meaning of a joke, to memorize a meaningful sentence, and so on. Central coherence is not only the standard way of information processing; it is supposed to be the *right* way. Ascribing impaired or *dysfunctional* cognitive capacities to autistic people presupposes a given account of what it is like to be normal. In this case, humans would have evolved an ability for information processing along a principle of central

coherence that is intrinsically *adaptive*. In the spirit of HD analysis, then, failure to reach central coherence would be a cognitive dysfunction.

However, one key discovery about autistic people has been that weak coherence, that is, relying on an alternative mode of information processing, instead of being always a source of impairment, may yield marked benefits in several contexts. There are now numerous examples of the "unusual strength" of autistic children on a large number of cognitive tasks: perception of detail, memory for word strings (rather than meaningful sentences), memory for unrelated items, recognition of upside-down faces, and so on (Frith and Happé 1994; Happé and Vital 2009). Looking for what is dysfunctional in autistic children, cognitive research on psychological mechanisms has revealed that in some areas, autism is not causing any form of obvious harm, as it may be a source of ability, excellence, or talent.

These results have several important implications. They mean, first, that coherence comes at a price (as in the case of neglected details) and that we have to contextualize success and failure. For instance, it is well known that there are side effects to the context sensitivity of cognitive processing that is typical of "central coherence." In the Titchener Illusion (see figure 21.1), when asked to compare the respective size of two (identical) circles, nonautistic people are mistakenly influenced by the size of other adjacent figures, while autistic people are not (Happé 1999). This means that autistic people, in a given

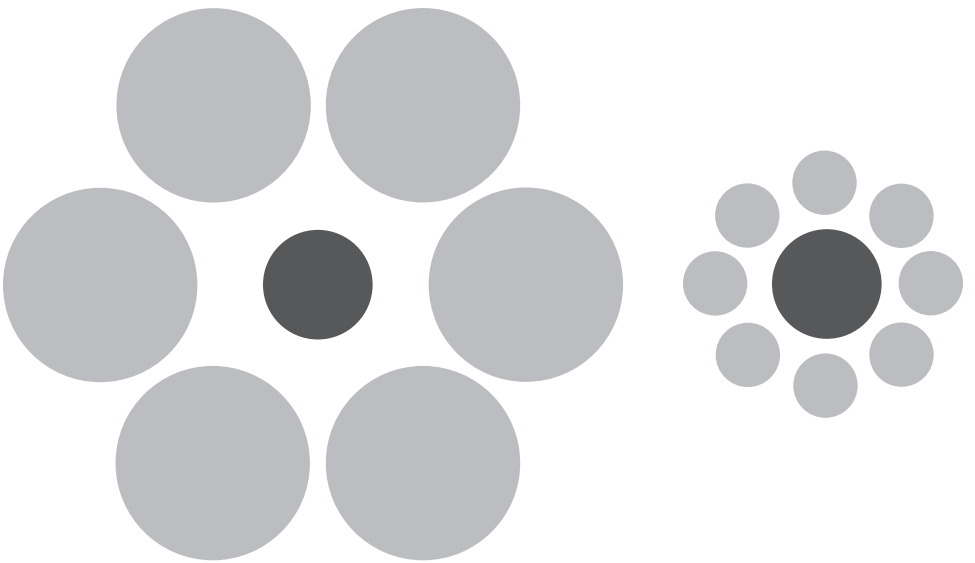

Figure 21.1
The Titchener Illusion. Context sensitivity leads to errors of judgments (the circles at the center are judged to have a different size). Autistic people do not succumb to this illusion (Happé 1999).

context, may make a perceptual judgment that is *correct* just because they *do not* rely (or not preferably) on the central coherence mode of information processing. Not succumbing to an illusion is hardly a clear sign of disturbance and impairment.

These results also mean that instead of thinking in terms of cognitive deficits in autism, we may have to think in terms of cognitive style (Happé 1999) and a different trade-off between abilities that are impaired and other abilities that may be enhanced, at least in high-functioning autism. This blocks any narrow, chauvinistic view of normality because if we look for dysfunctions and impairments on the sole basis of diminished performance, then an alternative view should be considered: autistic persons could label nonautistic people "coherentists" (with a pejorative meaning). They would dig in the scientific literature to list all the tasks where coherentists are clearly at a disadvantage to justify their own claims: strong coherence is a clear sign of a (widespread) abnormality. And in fact, this characterization of nonautistic people *has* been suggested. See this definition of the neurotypical syndrome (NT, that is, nonautistic people) that mimics and mocks the scientific description of autistic disorders:

> Definition of NT:—Neurotypical syndrome is a neurobiological disorder characterized by preoccupation with social concerns, delusions of superiority, and obsession with conformity … Neurotypical individuals often assume that their experience of the world is either the only one, or the only correct one. … NT is believed to be genetic in origin. Autopsies have shown the brain of the neurotypical is typically smaller than that of an autistic individual and may have overdeveloped areas related to social behaviour. How common is it?—Tragically, as many as 9625 out of every 10,000 individuals may be neurotypical. … There is no known cure for Neurotypical Syndrome. However, many NTs have learned to compensate for their disabilities and interact normally with autistic persons. (Posted on the website of the Institute for the Study of the Neurologically Typical, quoted by Brownlow 2010)

It is, then, extremely difficult to tell if "weak central coherence" can be understood in terms of *dysfunction* in Wakefield's sense. One possibility would be that there is a continuum between central coherence and weak coherence and that weak coherence as it is manifested in high-functioning autism and autistic talent is an instance of normal variation in human populations with a different trade-off between abilities that are diminished and abilities that are enhanced. Another possibility would be that the cognitive phenotype of high-functioning autism is in fact an instance of *harmless dysfunction*: the autistic mind deviates from an evolved, adaptive mode of functioning that corresponds to central coherence, but within current social environments, in the case of high functioning autism, this alternative mode of functioning is not detrimental and does not by itself cause significant harm. But, as significant as this alternative may seem in terms of conceptual analysis, we do not seem to be in a position to choose between these two very different descriptions of what high-functioning autism is. In particular, substituting brain mechanisms to psychological mechanisms to ground the ascription of a "dysfunction" would be a helpless move, because objective differences

within the autistic brain, which do not resemble the focal lesions of traditional neuropsychology (Baron-Cohen and Belmonte 2005), can be understood as the basis of an alternative mode of cognitive functioning: to distinguish what is dysfunctional and what is atypical, all will depend on the way we describe what the autistic brain does. For instance, "*reduction* in the connectivity between specialized local neural networks in the brain and possible *overconnectivity* within the isolated individual neural assemblies" (Rippon et al. 2007) is one way of characterizing the specific features of the autistic brain. But "overconnectivity within the isolated individual neural assemblies" could also be the basis on which, for instance, attention to detail supervenes (Grandin 2009, 1439–1440). One central problem of the research on the autistic brain is to go beyond what is merely plausible (Machamer et al. 2000) and to offer evidence that brain findings are *actually* related to the clinical picture. But it is also that we need to disentangle what is related to autism in general and what is related to the most unwelcomed aspects of severe autism. Pointing at overconnectivity, without further qualification, offers no evidence of natural dysfunction.

What counts for mental medicine is how we draw the line between a type of weak coherence (or more broadly an autistic phenotype) that counts as harmful and another that does not and how we explain the difference between the two. Talking of mental or neural mechanisms that perform or fail to perform their evolved function does not seem to help us much in that.

III. Harm without Values

In his seminal paper "The Concept of Mental Disorder" (Wakefield 1992a), Wakefield claims that "only dysfunctions that are socially disvalued are disorders," and it is quite obvious that autism today is a social problem as well as a purely medical question. As we have seen above, the claim of the neurodiversity movement has been that we should stop thinking of autism as a pervasive developmental disorder, waiting for a cure, and rejecting the very notion of an underlying dysfunction. The idea of dysfunction is linked to the decomposition of a whole into components among which one or several is (are) unable to perform its (their) function. This is precisely what has been challenged by activists like Jim Sinclair in his famous essay "Don't Mourn for Us" when he denies that autism is something that people *have* (they would *have*, for instance, an impaired theory of mind comparable to a broken leg or a cardiovascular disease) and that there would be "a normal child behind the autism" that, in principle, could be freed from its problem (Sinclair 1993). From the viewpoint of neurodiversity advocates, the "pervasive" character (to use Sinclair's word) of autism is assumed to be, not the simultaneous impairment of several cognitive areas, but a different mode of feeling and thinking "that colors every experience, every sensation, perception, thought, emotion, and encounter, every aspect of existence" (Sinclair 1993). Such a shift from

a medical view to a different perspective where support and recognition of autistic people become crucial outside of medical institutions has important consequences. As statistics show that autists often confront problems like massive unemployment and low income, companies like the Danish society Specialisterne and networks like the Autistic Self Advocacy Network vindicate the application of the principle of equality of opportunities. The question, then, is social justice rather than medical explanation. And if we read recent studies and reports, it is quite obvious that the climate is changing: some major, global companies have specifically targeted people with autism in their recruitment policy (Erbentraut 2015). The underlying philosophy is not only that diversity (including "neurodiversity") is essential to innovation. It is also that people with autism have specific assets: they can be outspoken, which is perceived as a good thing in a context where constructive criticism may be attenuated by office politics. And, interestingly, what has been seen as defects, oddities, and the result of cognitive impairments in clinical contexts is redescribed in terms that underline the positive aspect of the same well-known features and their potential benefit in terms of professional achievement (Walsh et al. 2014). What was called narrow interests is now perceived as the ability to stay focused on a given task. Attention to detail is not presented as the inability to grasp large, coherent wholes but as the perception of elements that will be missed by the ordinary viewer. People with autism are at a disadvantage in the context of standard job interviews: but this tells us something about the standard of job interviews, not against people with autism. As a result, there may be an ongoing change in the appraisal of autism: in a different social setting, it seems to be *valued* as it has never been before. According to the proponents of neurodiversity, autism would be harmful only in some circumstances and for external reasons—because of the negative attitudes that are the product of deeply rooted prejudices. It may *cause* harm, then, in a society that mistakenly perceives it as a disorder and a source of impairments, but it is not harmful in itself. Is there a way to reconcile the medical view and the claims of neurodiversity? And how does the HD view relate to this debate? I suggest we give a closer look to the relations between disorders, values, and harm.

First, even if this changing attitude toward autism is confirmed, a reappraisal has not in itself the power to transform a disorder into a nondisorder, as if functions and dysfunctions were dependent on our values. In this I would side with Wakefield: more positive attitudes toward autism in general do not change the boundaries of what disorders are, even if they may change the *representation* of disorders. These attitudes coincide with the outcome of scientific research that has unveiled *facts* that were previously neglected: autistic talent has been "unmasked" by scientific research and by the exposure of exceptional cases. But this does not prove wrong the view that in many cases autism may be harmful. This means also that the claims of the neurodiversity movement suffer from the same flaw that plagues the traditional, medical view: claiming that all forms of autism are instances of normal variation is just another

brand of essentialism. Advocating the rights of autistic persons in general is compatible with the recognition of the wide disparities within the autistic spectrum and of the vulnerability and disabilities that are the consequences of severe autism, which make the medical research as important as it has ever been. The reasonable, narrow view of neurodiversity—high-functioning autism is an instance of normal variation—does not entail the broad view—any kind of autism is a form of normal variation (Jaarsma and Welin 2011). Progress in terms of social integration does not offer evidence that the medical view is wrong in itself and that in all cases, disability is nothing more than a disvalued difference.

But then, what is needed is a view of harm that is more factual and not dependent on "social values." The deprivation of something that is both widespread and useful is in itself harmful, in this narrow sense, because it is a source of disadvantage. For a given subject, dysfunctions result in harm when they reduce significantly and repeatedly his autonomy, the range of his opportunities, or the probability of success of his actions (Forest and Le Bidan 2016). Jerome Wakefield acknowledges this sense of harm from time to time as in the example of kidneys (Wakefield 1992a, 384) when he says that "a dysfunction in one kidney often has no effect on the overall well-being of a person and so is not considered to be disorder": in this case, clearly, the absence of harm (and, as a consequence, the absence of a disorder) has little to do with "present cultural standards" but only with the lack of detrimental effects of the dysfunction on the ordinary life of the individual. To say that "to be considered a disorder, the dysfunction must also cause significant harm under present circumstances and according to present cultural standards" leaves open the possibility of the presence of harm under present circumstances *without* reference to cultural standards, as in the case of kidney dysfunction. In the very same sense, impairing language acquisition in a neurodevelopmental disorder is causing significant harm because in this case, the range of opportunities is severely reduced and results in a disadvantage for the child. And to know this, we don't need an evolutionary scenario about the benefits of language mechanisms. And we don't need to think of social values, because we cannot figure out a society where failing to learn how to speak would not be intrinsically detrimental to a human child.

Then, it is both true that only harmful dysfunctions matter to medicine (because of their significant consequences) and false that the presence or the absence of a disorder depends on "social values." To see this, we can make the following thought experiment. Let us imagine that in a given (imaginary) society, children who meet the criteria for classical autism (difficulties with language acquisition, reduced social interactions, and repetitive behavior) receive a most favorable treatment, because they are supposed to have been chosen for some kind of higher, spiritual purpose. According to religious beliefs that shape the attitudes of members of this society, the (apparent) deficiencies of autistic children are only the sign of their (hidden) supernatural powers. But in this case, we still have the underlying cognitive dysfunctions and the reduced abilities. And

we have every reason to believe that in this case, classical autism is still a disorder, even if the child is placed in a most favorable environment. It does not seem possible, then, to claim that, underlying dysfunctions being kept constant, the presence or the absence of a mental disorder depends on social values. In many cases at least, whatever values we adopt or reject, the harmful consequences, in the sense above defined, are there to stay.

Conclusion

Hempel used to underline that functional talk presupposes a certain standard of what the "normal functioning" or the functional integrity of the corresponding system may be, and he insisted on the necessity of making such standards as explicit as it is possible (Hempel 1965). In medicine and psychiatry, in particular, it is not possible to make claims about function and dysfunction without sensible background assumptions relative to the integrity of the individual. And what psychiatry lacks, too often, is a theory of what mental health would be. Having no standard of integrity at all would lead us nowhere, but we have to be especially careful not to define these standards in an excessively narrow way and to remain sensitive to what we could call the *varieties* of mental health. One popular version of our evolutionary history is that what makes humans special is their mindreading ability, an ability that allows us, in particular, to navigate within large social groups. Another version would be our ability to construct abstract, coherent wholes and to decompose them into their elements. Autism, then, would be a disorder not just because autistic behavior is odd, or because autistic children fail to do several things, but because autistic persons deviate from a certain standard to which we give a special importance for theoretical reasons. It is, as a consequence, especially important that just-so stories that flourish in evolutionary psychology do not introduce bias in our representation of what mental disorders are. If, as I have suggested above, for a given subject, dysfunctions result in harm when they reduce significantly and repeatedly his autonomy, the range of his opportunities, the quality of his well-being, or the probability of success of his actions, we have, very roughly, a standard to judge when autism is a kind of normal variation (associated with different abilities and opportunities) and when it is a source of impairment that requests medical concern. Again, dysfunction, in this sense, is not defined in reference to an evolutionary background, and harm is not judged according to social values.

Notes

1. Baron-Cohen, 2008: "The official terminology is to use the acronym ASD, for autism spectrum disorder. I prefer the acronym ASC [Autistic Spectrum Condition], since individuals in the high-functioning subgroup are certainly different [….] but it is arguable whether these differences should be seen as a disorder" (p. 14).

2. To my knowledge, the first occurrence of the word "neurodiversity" in a publication is Blume, 1998.

3. About the second question, I want to stress that what I have in mind is not reducible to the "imprecise boundary objection" against which Jerome Wakefield has already vindicated his views (Wakefield, 1999). In his answer to Lilienfeld and Marino, he has claimed that the HD analysis, as an instance of conceptual analysis, is not aimed at resolving the question of the boundary of disorders, but aimed at "explaining shared judgments about a range of important cases that fall on one side or the other of the boundary" (379). But clearly, high-functioning autism is not a limited set of rare and exceptional, boundary cases, it covers a large part of the autistic spectrum. And as judgments about high-functioning autism are contradictory, and not "shared," the question is how to take sides in the debate in a non-arbitrary manner.

4. See the foreword by evolutionary psychologists Leda Cosmides and John Tooby to Baron-Cohen, 1995.

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Defining Mental Disorder

Jerome Wakefield and His Critics

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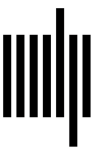

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