Neurodiversity and the Social Ecology of Mental Functions

Robert Chapman (iD)
Department of Philosophy, University of Bristol

Abstract

In psychiatry, mental dysfunction is typically framed in relation to models that seek to be continuous with physiology or evolutionary biology and that compare individual fitness to a broader functional norm. Proponents of the neurodiversity movement, however, challenge the pathologization of minority cognitive styles and argue that we should reframe neurocognitive diversity as a normal and healthy manifestation of biodiversity. Neurodiversity proponents have thus far drawn on social-relational models of disability to challenge the medical model of disability, but they have not developed an alternative functional analysis to replace conceptions of neurological dysfunction or impairment. Here I clarify and defend the neurodiversity perspective by drawing on ecological functional models that take relational contributions to collectives, and group functioning, into account alongside individual functionality. Using the example of autism as well as recent developments in the study of cognitive diversity, I apply these models to human mental functioning and argue that what I call the ecological model has greater utility for research and practice than the leading psychiatric functional analyses of mental functioning.

The concept of mental disorder is generally divided into two components. The first is dysfunction or impairment, which is described as the "objective scientific component," whereas the second is "a normative, socially negotiated component," which are the harmful consequences of the dysfunction (Murphy, 2006, p. 11). For instance, the influential definition given in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–5*) describes the harm component as a "clinically significant disturbance" associated with disability or distress; in turn, the harm must be taken to stem from "a dysfunction in the [underlying] psychological, biological, or developmental processes" (American Psychiatric Association, 2013, p. 20). More broadly, this two-component definition is reflected both in the leading theoretical models (Boorse, 1975; Wakefield, 1992a) and in the most frequently used clinical definitions of mental disorder dating back to the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980). From what is arguably the orthodox point of view then—and although each take differs on details—every disability considered to be a mental disorder is taken to be a case of socially mediated harm stemming from objective dysfunction or impairment (American Psychiatric Association, 2013; Bolton, 2008; Boorse, 2014; Wakefield, 1992a).

Nonetheless, in recent decades the pathologization of many psychiatric, cognitive, and learning disabilities has been increasingly challenged by the rise of the neurodiversity movement. The neurodiversity movement is primarily a social-justice movement aiming to end what proponents see as the default pathologization of neurodivergence (i.e., divergence from normal mental functioning) and to instead promote the acceptance and accommodation of human neurodiversity (Armstrong, 2015; Blume, 1998; Chapman, 2019b; Singer, 1999). Instead of being conceived as medical pathologies, a range of disabilities—including autism, attention-deficit hyperactivity disorder, dyspraxia, and bipolar disorder— have been reconceptualized as manifestations of humanity's "natural variation" (Jaarsma & Welin, 2012)

Corresponding Author:
Robert Chapman, Department of Philosophy, University of Bristol
Email: kn18198@bristol.ac.uk

or "dispositional diversity" (Milton, 2017). From this perspective, these cognitive styles are integral to different kinds of selfhood (Chapman, 2019b; Walker, 2014). Neurodiversity proponents (Pellicano & Stears, 2011) thus prefer identity-first language (e.g., "autistic person") rather than person-first language (e.g., "individual with autism") to emphasize how one's neurocognitive style is integral to one's selfhood—a terminological preference I shall adhere to throughout the current article.

Although views among neurodiversity proponents vary, perhaps the core theoretical underpinning of the movement comes from using social-relational models of disability. These models take at least a significant amount of neurodivergent disablement and distress to be primarily caused by social barriers and ableist norms more centrally than by the cognitive traits associated with a given disability (Chapman, 2019b). Ari Ne'eman, an autistic self-advocate who has been highly influential in the movement, stated that "none of this is meant to deny the very real fact" of disability (Ne'eman, 2010, para. 5; see also Kapp, Gillespie-Lynch, Sherman, & Hutman, 2013). However, reframing disablement primarily as a political issue and emphasizing neurodivergent strengths alongside deficits challenges the default pathologization of neurodivergent disability. There is now a growing strand of research based on this reframing that analyzes the extent to which many of the harms associated with disabilities might be more primarily explained by looking at external rather than internal factors (Chapman, 2019b; Jaarsma & Welin, 2012; Robertson, 2010; Wakefield, Wasserman, & Conrad, 2020). Although how far social-relational models can be applied is still disputed, even critics have conceded that at least some currently pathologized disabilities may be better conceived of as nonpathological (Jaarsma & Welin, 2012; Wakefield et al., 2020).

However, even if the social-relational model analysis is convincingly applied to the harm component of a given disability, the purported objectivity of the dysfunction component is still left relatively intact. This is because the social-relational model analysis in no way denies that any given trait is dysfunctional or impaired:[1] It claims only that the dysfunction or impairment is often not the key cause of most harm and disablement. Moreover, although neurodiversity proponents have questioned the objectivity of cognitive pathologization (Armstrong, 2015; Baron-Cohen, 2017; Chapman, 2019b; Milton, 2017), they have not clarified an alternative functional analysis that accounts for the claim that neurodivergence is often a manifestation of healthy "natural variation." This has left neurodiversity proponents open to the charge of relativism (Grinker, 2015) or of being antiscience (Costandi, 2019). For instance,

the neuroscientist and critic of neurodiversity Moheb Costandi recently stated that the neurodiversity perspective is "at odds with scientific understanding" (Costandi, 2019, para. 22). Likewise, philosopher of medicine Christopher Boorse dismissed refusals "to pathologize the pathological on political grounds" (Boorse, 2014, p. 687).

Here I suggest that neurodiversity proponents have, in fact, laid the grounds for an alternative functional analysis even if they have not yet clarified this analysis in enough detail. Indeed, whereas orthodox models of mental dysfunction are highly individualistic and seek to be consistent with physiology (Boorse, 1975) or evolutionary biology (Wakefield, 1992a), neurodiversity proponents have more often emphasized the need to adopt an ecological perspective (Armstrong, 2015; Blume, 1998; Singer, 1999). For instance, Judy Singer, the autistic self-advocate who coined the term neurodiversity as a sociology student in the late 1990s, focused on how neurodivergent individuals can fill an "ecological niche" (Singer, 1999, p. 66) in society because of their different ways of processing. Likewise, for Harvey Blume, a journalist who heavily influenced the movement by being the first to articulate the concept in writing, "neurodiversity may be every bit as crucial for the human race as biodiversity is for life in general" (Blume, 1998, para. 4). Or, as Thomas Armstrong, who later helped expand the concept to learning and cognitive disabilities, predicted: "Embracing the concept of neurodiversity would bring the study of mental health disorders in line with movements that have already taken place over the past 50 years around biodiversity and cultural diversity" (Armstrong, 2015, p. 350).

Although there may be a variety of fruitful ways of developing the ecological emphasis (e.g., as a methodological tool for studying cultural evolution or as a rhetorical tool for disability advocacy), here I argue that it allows us to not only challenge but also pose a viable alternative to the purportedly objectivist evolutionary *functional analysis* underlying psychiatric pathologization. More specifically, here I propose an *ecological model* of mental functioning as an alternative to the leading physiological and evolutionary models. This model is based on systems functioning rather than fitness and selection, and it takes relational and collective functioning into account alongside individual functioning. To the extent that the ecological model is convincing, it will provide an alternative functional analysis that allows a much broader range of neurodivergent functions than orthodox models.

Although I hope to influence psychological research and practice, it is worth noting that I write here as a philosopher of science interested in clarifying the fundamentals of the concept of neurodiversity—especially

how the neurodiversity movement seeks to bring about a "paradigm shift" (Chapman, 2019b; Walker, 2014) in how we conceive of psychological disability. This seems important to get right because, although it is true that the neurodiversity movement is primarily a social-justice movement, it must also have a viable theoretical underpinning to successfully bring about a scientific paradigm shift. Given that my concern is with returning to and clarifying the fundamentals, I begin by focusing on the two leading theoretical models of mental dysfunction: Christopher Boorse's biostatistical theory and Jerome Wakefield's harmful dysfunction analysis. These are the models that are typically taken to underpin the validity of the definitions of mental disorder found in the *DSM* and other manuals. By combining the neurodiversity perspective with existing critiques of the orthodox view, I argue that both of these models encounter conceptual, epistemic, and practical issues regarding their limitation to individual fitness that diminish their utility and make the claims to objectivity hard to sustain. I then clarify the conceptual foundations of the ecological model and argue that it provides some leeway with the problems I associate with the evolutionary models. Drawing on the example of autism as well as research on the benefits of cognitive diversity in groups, I argue that an ecological perspective has greater utility for both research and clinical practice. To the extent this turns out to be convincing, the neurodiversity movement will have offered an alternative for both components of mental disorder as traditionally conceived—thus establishing the basis for a paradigm shift in the sciences of psychological ability and disability.

Boorse's Biostatistical Theory

Boorse's biostatistical theory was first developed in the 1970s in response to concerns regarding the scientific status of our health and disease concepts, which had been criticized within the antipsychiatry movement and more broadly. Since then, this model has proven to be one of the most robust and influential objectivist analyses of mental functioning (Boorse, 1975, 1977, 1997, 2014). Although Boorse acknowledged that psychiatric practice is value-laden, he distinguished between social norms and physiological-fitness norms to defend the objectivity of his analysis. According to Boorse, health is defined in terms of physiologically normal species fitness levels. By contrast, a dysfunction or "pathological condition is a state of statistically species-subnormal biological part-function, relative to sex and age" (Boorse, 2014, p. 684). If Boorse's model is viable, then determining function or dysfunction is an "objective matter, to be read off the biological facts of nature without need of value judgements" (Boorse, 1997, p. 4).

To account for the above depiction, Boorse made two key moves. First, he adopted a goal-oriented systems definition of functions, whereby the proper function of any biological part is defined in light of the typical causal contribution it makes toward the goals of the biological system. Goals refer to the disposition of biological systems to adjust behavior to survive and reproduce (i.e., the elements of Darwinian fitness) rather than to intentionally held goals (Boorse, 2002, pp. 68–69). For an organism, the function of any given subsystem (e.g., mental faculties, organs) is therefore defined by its "causal contributions" (Boorse, 2002) toward its organismic goals of survival and reproduction. The function of the heart, for instance, is to pump blood because this is the role it plays in overall organismic fitness.

Second, to delineate function from dysfunction, Boorse proposed that the line between function and dysfunction should be determined through recourse to a biostatistical fitness average across the appropriate reference class. From Boorse's perspective, the appropriate reference class from which to determine the norm should consist of all members of the same species, age, and sex. This is because he deems each to be "a natural class of organisms of uniform functional design" (Boorse, 1996, p. 7). Dysfunction of any given organ, or subsystems of the organ (e.g., modules in the brain), and hence medical pathology, is thereby determined in terms of interreference with statistically normal functioning for the reference class. For instance, if autistic individuals have a social-processing ability below the species norm or have failed to meet age-specific developmental milestones, and to the extent that such factors are important for individual fitness, then these processing abilities will be taken to be objectively dysfunctional.

The purported benefit of this model is that by focusing on fitness it can distinguish physiological norms from social norms, making its analysis objective. Nonetheless, many issues have been noted with Boorse's concept of the reference class that may undermine the claim to objectivity. Most notably, Boorse's sole reliance on species, age, and sex to form the reference class may lead to the undue pathologization of minorities. For instance, minority sexualities, or communities that have adapted to local environments, might be unduly pathologized in Boorse's account (Cooper, 2002; Kingma, 2007). This problem also applies to neurological and cognitive variation, which seems much more varied than Boorse's model has room to acknowledge (Amundson, 2000; Chapman, 2019b, 2020a). In practice, an additional concern is that adhering to such a model may lead to viewing minority neurotypes through an overly negative lens, which might also lead to social facts (such as low IQ as a result of inadequate education)

being unduly reified as being natural (Dinishak, 2016). Overall, getting the reference class right is vital because it determines who is counted as healthy or pathological, yet it is not clear how statistical models can objectively verify the appropriate reference class given the complexity and variation in human functioning (Cooper, 2002; Kingma, 2007).

From another angle, Boorse's individualism has also been problematized (Delehanty, 2019; Valles, 2018). According to Boorse, the function and dysfunction of a mechanism can be determined only in relation to individual fitness goals. This limitation to individual goals is important to consider because, as evolutionary psychiatrist Randolph Nesse put it, "whether a condition is considered a [dysfunction] or not depends on whether the benefits are considered from the point of view of the gene, the individual, or the social group" (Nesse, 2001, p. 43). This point precisely mirrors the one raised by neurodiversity proponents in regard to how neurodiversity may be functional as a group trait (Hoffman, 2017), as I return to below. Boorse (1976, pp. 83–84) did acknowledge that in ecology organisms or groups can be taken to fulfill ecosystem functions, but he nonetheless argues that among the biological sciences "it is only the subfield of physiology whose functions seem relevant to health" because medicine is generally concerned with the functioning of the individual biological organism (Boorse, 1977, p. 556). Nesse (2001) corroborated here that it is the "individual who seeks help for suffering and disability" rather than, for example, the gene or group (p. 43).

The problem is that medical theory and practice increasingly look beyond the individual organism. As Delehanty (2019) pointed out, Boorse's framework does not fit well with the rise of population-health science, wherein "we see an emphasis not on the health of individuals but on the health of groups or populations" (p. 6; see also Arah, 2009; Valles, 2018). When it comes to psychological health specifically, we see a further move away from individual physiology. For instance, widely used forms of group therapy and systems therapy focus on the family or social group as the unit of treatment. Likewise, it is also not unheard of for clinicians or researchers to talk of, for example, "collective trauma" (Wessel & Moulds, 2008), "dysfunctional communities" (Taylor & de la Sablonnière, 2013), or "relational dysfunctions" (Hoffman, 2017). Physiological individualism is also rejected in contemporary accounts of cognitive and psychiatric disability, which increasingly focus on how minds are embedded, scaffolded, and extended rather than being reducible to individual neurology (Drayson & Clarke, 2020; Hoffman, 2016). In line with these contemporary accounts, ecological approaches to the psychological and behavioral sciences have become increasingly accepted by researchers in recent decades (even if these approaches have not yet been extended to functional analysis; Fuchs, 2017; Oishi & Graham, 2010). Given such developments, Boorse's justification for physiological individualism seems increasingly dated, especially when it comes to conceptualizing mental functioning. A functional analysis that really did fit the context of medicine would take collectives, and the complex relationship between society and biology, into account.

Wakefield's Harmful Dysfunction Analysis

The other leading account of mental dysfunction is Wakefield's harmful dysfunction analysis, which was first developed in the 1990s and purported by proponents to avoid the problems associated with statistical models (Wakefield, 1992a, 1992b, 2015). For our purposes, the key difference is that Wakefield seeks to be consistent with evolutionary biology more centrally than physiology. Given this aim, he adopts the *etiological* definition of functions, whereby the proper function of a mechanism is defined in reference to natural history rather than current biostatistical norms. As Boorse (2014) explained, "on my view, a functional trait must serve [fitness] in the present, while on Wakefield's, it must have served [fitness] in the past and been selected for that effect" (p. 687). The proper function of any given biological mechanism will therefore be the effect for which it was naturally selected. From this perspective, when it comes to psychiatric or cognitive disability specifically, dysfunction refers to the "failure of a psychological mechanism to perform" its naturally selected proper function (Wakefield, 2015, p. 999). For instance, it may be that "psychotic disorders involve failures of thought processes to work as designed, anxiety disorders involve failures of anxiety-and-fear-generating mechanisms," and so forth (Wakefield, 2015, p. 1000).

To some extent, this model may help to avoid the concerns about overpathologization noted with the biostatistical theory. From Wakefield's point of view, it may be that some "conditions that are considered disorders are in fact naturally selected variants" and hence not in fact disorders (Wakefield, 2015, p. 1001). This could include minority communities with idiosyncratic selection histories. It is notable that some neurodiversity proponents have stressed the possible evolutionary basis for certain forms of neurodiversity (e.g., Armstrong, 2015). Moreover, this analysis—or some updated variation of it (Hoffman, 2017)—may have more room to conceptualize group dysfunction, by reference to multilevel selection or relational dysfunction. Indeed, the

harmful dysfunction analysis has been used to challenge what Wakefield took to be the undue pathologization of ordinary sadness or anxiety in the *DSM* (Wakefield, 2015). Wakefield et al. (2020) also expressed sympathy for at least a minimal conception of neurodiversity that allows what Wakefield considers to be mild manifestations of autism to count as natural human variations.

Nonetheless, there are still problems with this approach. Cooper (2002) argued that whichever time frame is adopted for deciding which historical selection determines the function (e.g., the recent past or the distant past) will itself be somewhat arbitrary and yet will also determine what is thereby counted as the proper function. Another issue is that relying on theorizing about selection history and its relationship to any given individual behaviors is imprecise at best (Bolton, 2008). When it comes to clinical practice, Murphy and Woodfolk (2000) argue that attempts to determine what is a genuine underlying dysfunction or not will rely on a "cluster of normative concepts, *expectable*, *proportionate*, *appropriate*, and *normal*, which we suspect cannot be unpacked without making value judgments" (p. 246). And when it comes to conceptualizing general dysfunctions, as Varga (2011) notes, in practice, "just like Boorse, Wakefield must also identify the evolutionarily designed response to the environment via recourse to a statistical norm" (p. 5). This identification is imperative because—in the absence of being able to accurately verify natural history—theories regarding historical selection will often be based on the existing population for its justification (see also Boorse, 2002, p. 101). Given these epistemological issues, similar concerns to those associated with Boorse's model may therefore reemerge, including the possibility of unduly pathologizing minorities by projecting current societal values into speculation about selection history.

From the neurodiversity perspective, the problem may be understood as further exasperated by systemic epistemic ignorance resulting from broader ableist power structures. Epistemic ignorance about disability arising from structural ableism has been associated with a widespread inability to understand the lived experience of disability and the causes of disablement and a widespread dismissal of the perspectives and testimonies of disabled individuals (e.g., Barnes, 2016). In line with this recognition of epistemic ignorance, neurodiversity proponents do not just contest that much of the disablement and distress they face is a product of ableist social structures and attitudes; they also contest that what we even recognize as good or desirable is itself distorted for the same reason (Rodogno, Krause-Jensen, & Ashcroft, 2016). Indeed, as autism researcher Laurent Mottron noted,

Even researchers who study autism can display a negative bias against people with the condition. For instance, researchers performing functional magnetic resonance imaging (fMRI) scans systematically report changes in the activation of some brain regions as deficits in the autistic group—rather than evidence simply of their alternative, yet sometimes successful, brain organization. Likewise, variations in cortical volume have been ascribed to a deficit when they appear in autism, regardless of whether the cortex is thicker or thinner than expected. When autistics outperform others in certain tasks, their strengths are frequently viewed as compensatory of other deficits, even when no such deficit has been demonstrated empirically. (Motton, 2011, p. 33)

Given that application of the etiological account relies on backward-looking speculation that may be biased by broader systemic epistemic ignorance, the *propensity* for neurodivergent functioning (and flourishing)[2] may be systematically unrecognized because it falls outside what is currently considered typical. Although the significance of propensity has been independently pointed out in research on biological functions (Bigelow & Pargetter, 1987), it seems especially important to consider whether we want to minimize the possibility of unduly pathologizing neurodivergence.

Conceptualizing the Ecological Model

As demonstrated above, the leading physiological and evolutionary accounts are too individualistic, encounter multiple conceptual and epistemic problems, and may reinforce the undue pathologization of minorities. However, there is no obvious reason not to apply an ecological rather than evolutionary functional framework to human thinking. Indeed, although it is true that the human mind is the product of evolution and surely can be analyzed as such, it is also the product of, and sustained by, socioecological scaffolding and is always situated in a broader culture (Fuchs, 2017; Oishi & Graham, 2010). Consequently, whether we adopt an analysis more in line with individual biology (as the *DSM*, Boorse, and Wakefield do) or an analysis more in line with ecology (as neurodiversity proponents suggest) is not necessarily set by the subject matter. Given the problems with the evolutionary framings, it seems especially worth asking whether the neurodiversity framing might provide a viable alternative, particularly because this question aims to acknowledges a much broader range of functioning than the orthodox accounts and hence purports to avoid the issue of undue pathologization. In this section I clarify a version

of this idea by adapting accounts of ecological functions to apply to mental functions. I then spend the rest of the article arguing that the ecological model may have greater utility than the accounts reviewed above.

In ecology, etiological accounts of functioning are less popular than systems accounts, mainly because reproduction and selection are less relevant and harder to conceptualize at the level of the ecosystem. Thus, systems-based analyses have generally been preferred, more in line with Boorse's definition of functions (Boorse, 2002; Dussault, 2019; Dussault & Bouchard, 2017; Maclaurin & Sterelny, 2008; Nunes-Neto, Moreno, & El-Hani, 2014). Perhaps the most relevant account comes from Dussault and Bouchard (2017), who suggest that the function of any given unit of biodiversity is best defined by its contribution toward a "systems' ability to thrive and perpetuate themselves in the future" (p. 1117). Here the emphasis is on how units of biodiversity (organisms, species, etc.) contribute to the ecosystem's "propensity to persist" (p. 1122) in the face of change. From this point of view, functions are *relational* and contextual rather than intrinsic (p. 1118), which means that no unit can be functional or dysfunctional as such. Moreover, the propensity for an effect will be intrinsic to the unit, but the function or dysfunction will always be relational and depend on actual behavior at a given time.

Turning from ecology to human mental functions, the emphasis on the propensity to persist fits well with claims made by neurodiversity proponents in at least two ways. First, note the following claim from Blume (1998): "Neurodiversity may be every bit as crucial for the human race as biodiversity is for life in general. Who can say what form of wiring will prove best at any given moment?" (para. 4). The phrases "will prove" and "at any given moment" indicate a future-oriented perspective, moving away from selection history or present averages toward what is likely to prove adaptive given our changing environment. For instance, one company leader (Aspinal, 2020) recently reported that having autistic employees has helped his company survive during the coronavirus lockdown in 2020 despite this not being the reason why he employed them. This may be analogous to how genetic diversity is adaptive for the propensity of the species to persist given the likelihood of new viruses, regardless of whether the genetic diversity itself was an adaption (i.e., a product of selection). Second, in an important respect this fits well with the social-relational model analysis because one way of putting the core insight of the social-relational model is that the minority propensity for adaptiveness is often stifled both through a combination of societal disablement and epistemic oppression. For instance, it has often been assumed that people with developmental or cognitive disabilities will be naturally ineducable, and

therefore they are not given access to education. Because a propensity account can take contingently stifled propensity resulting from oppression or marginalization into account, it may have the benefit of moving toward acknowledging contingently stifled propensity, which may help to avoid the reification of social facts.

Beyond the forward-looking nature of this account, equally relevant to propensity is that in ecology functional roles are multilevel and relational rather than restricted to individuals.[3] That is, they are attributed either when some part of the ecosystem plays a role within biodiversity or ecosystem functioning or when biodiversity itself, or its component parts, plays a role (Nunes-Neto et al., 2014). It is worth noting here that neurodiversity proponents have indicated that we can similarly understand mental functional propensity on at least three levels. First, mental traits can contribute to the persistence propensity of the individual (Robertson, 2010). This is much like what we see in the individualist accounts but aims to be more sensitive to minority modes of functioning that fall outside of species norms by focusing on strengths as well as limitations. Second, we can also take specific cognitive dispositions to contribute toward what Singer (1999) called an ecological niche. (Here I focus on what I call "niche contribution," rather than the typical "niche construction," to emphasize the specific functional roles enabled by disability-inclusive niche construction: Although Singer's point also regarded how the niche construction of environments either enables or disenables, I do not focus on the theme of construction here because it is already covered by the social-relational model of disability.) This would be when they contribute to a specific role in the collective, for instance, such as when Blume (1998) claimed that "cybernetics and computer culture, for example, may favor a somewhat autistic cast of mind" (para. 4). Third, we can understand the functional propensity traits as emerging at the group level. By analogy, genetic diversity in a species is adaptive, but here the robustness of the species is an emergent group trait that is not derived from any single member of the species. Likewise, as Hoffman (2017) has proposed, it may be that there are collective cognitive traits that are more or less adaptive for the collective, in relation to the environment, that emerge from the group rather than being directly traceable to individual members. The ecological model allows for any trait to contribute to relational functions or dysfunctions at any level. Indeed, it may contribute to functions or dysfunctions at all levels, or it may contribute to both functions and dysfunctions simultaneously.

A tentative definition of function from the neurodiversity perspective could thus be as follows: To contribute to a function, a mental trait, cognitive style, or group must have the propensity to perform an effect

that contributes either to individual or group persistence, or both. By contrast, dysfunction will occur whenever there is a relational clash between any of these levels that hinders the propensity to persist. From this point of view, it is not only that cognitive traits can contribute to both individual and collective functions or dysfunctions but also that groups themselves have traits that are functional or dysfunctional in relation to the group or individual persistence. This would be partially analogous to how we talk of malfunctioning at the ecosystem level. I give some examples to show how this kind of analysis may help to capture the complexity of psychological ability and disability in the following sections.

The Ecological Model and the Autism Spectrum

In recent decades, many propensity traits that may be beneficial both for the individual and as niche contributions have been noted with a variety of psychological disabilities traditionally framed as merely deficient (Armstrong, 2010, 2015). However, the most well-established example is that of autism, which has traditionally been framed only in terms of deficits but that is increasingly associated with individual benefits and may also be reconceptualized through Singer's concept of the ecological niche. On the one hand, autistic cognition is associated with a variety of traits that may be beneficial to the individual. These include capabilities for hypersystemizing (Baron-Cohen, 2006; Greenberg, Warrier, Allison, & Baron-Cohen, 2018), hyperattention to detail (Fitch, Fein, & Eigsti, 2015), intense ability to focus (Murray, Lesser, & Lawson, 2005), and reduced susceptibility to framing effects (De Marinto, Harrison, Knafo, Bird, & Dolan, 2008). In turn, these same traits may also be a factor in niche contributions for the collective. In his original article on neurodiversity, Blume (1998) predicted that autism may be particularly well suited to "computer culture." Since then, autistic traits have been associated with the evolution of folk physics (Badcock, 2009), and it has been found that autistic individuals, and individuals with autistic traits, are overrepresented in engineering and the sciences (Wheelwright & Baron-Cohen, 2001). Although this finding does not apply to all autistic individuals, having a subset of members who have a neurological adaptedness to working with mechanistic pursuits may be considered highly adaptive not only from the individual but also as a niche role in the group, insofar as human groups often rely on technological solutions to solve complex problems.

More strikingly, even some autistic "deficits" may contribute to functions at the group level despite being associated with individual disability. For instance,

autistic individuals tend to be less spontaneously attuned to neurotypical social worlds than neurotypical individuals are (Chapman, 2019a; Milton, 2012). Although this can be disabling for autistics, it has also been associated with increased originality of thought (Happe & Vital, 2009) and a form of moral agency that lends itself to being freer from subtle social pressures (Baron-Cohen, 2011). For instance, Baron-Cohen (2011) associates the autistic cognitive style with a tendency toward being "super moral" in terms of following social rules with less restraint from social pressures. An example might be the autistic climate activist Greta Thunberg, who hypothesized that her activism has been successful because of, rather than despite, her autistic social-cognitive style (Silberman, 2019). Although these same traits are both beneficial and disabling for her as an individual, Thunberg's role as a climate-change activist is arguably a vital niche from the group perspective. Depending on the goals of the group, autistic inability to spontaneously attune to a neurotypical social world therefore has the propensity to fulfil niche-functional roles at the group level despite being associated with disability at the individual level.

It is important to note, however, that not all individuals will exhibit the strengths typically associated with their disability more generally. From the neurodiversity perspective, concepts such as autism are taken as politically useful classifications but are not taken to have biological validity (Chapman, 2020a). Examples of group-based niche contributions must be taken as rules of thumb that have some level of arbitrariness rather than as a hard-and-fast claim about the proper function of any given group or its members. Still, it is important to recognize such associations even if they are generalities, especially because they have often been overlooked given the prevalence of deficit-based models.

This also raises the question of how the social-ecological analysis would frame the issue of functionality for multiply disabled autistic individuals with high support needs. For the model to have utility it must be helpful for neurodivergent individuals who do not have the cognitive strengths just noted. It should be noted here that a core part of the neurodiversity perspective is to view individuals as valuable and deserving of acceptance and rights regardless of their functional propensities.[4] With this caveat, I suggest that the social-ecological analysis would have greater utility than a more deficit-based evolutionary framing when it comes to multiply disabled autistic individuals.

The utility relates to how dysfunction is always relational in the ecological model. This relational perspective provides room for us to acknowledge both how individual traits contribute to individual disability and also the societal need to accommodate disabled

individuals—yet without committing to the further claim that these individuals, or fundamental parts of their way of being, are intrinsically pathological. This allows some flexibility with regard to where intervention toward the dysfunction is aimed, leaving it open whether the focus should be more on the individual or the context yet without the dehumanizing and stigmatizing effects of pathologizing fundamental aspects of an individual's selfhood. In practical terms, this allows the benefits of both social-model and medical-model interventions where appropriate, yet in a way that avoids locating the dysfunction in the individual's way of being.

Relatedly, as noted above, because the ecological model is compatible with social-relational models of disability, it is part of the analysis that the propensity of these individuals may have been stifled (e.g., by being excluded from adequate education) and moreover that we may have an epistemic ignorance of the propensities of such individuals. In this case, this analysis would encourage assuming, and searching for, propensity even if it is not obviously apparent. Consider, for instance, how "severely" autistic neurodiversity advocate Mel Baggs has argued that they have a different kind of experiential "richness" (Baggs, 2010) that is no less valuable for being so different. Likewise, as Nakoi Higashida, who has also been given the "severe autism" label, stresses, "functioning in our society is difficult for neuro-atypicals, but encountering difficulties is not the same thing as being unhappy" (Higashida, 2017, p. 261). As such testimonies indicate, it may be that autistic well-being and concerns are different to neurotypical forms in a way that is largely unrecognized (Rodogno et al., 2016). Because it is attentive to our epistemic ignorance of minority forms of functioning, it would be part of neurodiversity paradigm science to orientate toward studying such overlooked propensities, such as the intense joy often anecdotally ascribed to nonverbal multiply disabled autistic individuals (e.g., McCafferty, 2018) instead of focusing only on deficits. This may be helpful for multiply disabled autistics because of its effects on research and in turn clinical and public understanding, which would help to foster a perspective that recognizes and accommodates their disabilities without viewing them through an overly negative lens. Overall, the ecological model therefore allows us to acknowledge the dysfunctions autism can contribute to, as the orthodox models do, but it also helps direct us toward recognizing the variety of functions autism contributes to at both the individual and group levels.

Emergent Group Functions

Beyond niche contribution of individuals to collectives, the ecological model can also take account of emergent effects that exist at the group level. Niche contributions are specific and identifiable, whereas group propensities may be emergent in the sense that they stem from but are not reducible to the traits of the individuals that make up the collective. Philosopher and neurodiversity proponent Ginger Hoffman (2017) hypothesized that a group with greater neurological diversity may have epistemic and practical benefits compared with a neurologically uniform group because the former will have a greater variety of cognitive resources to draw on. Moreover, according to Hoffman, to the extent that this is so, we might say that a neurologically uniform group could be comparatively dysfunctional at the group level, even if all the individual members were neurotypical. If this is feasible, it would have the interesting implication that a group of neurotypical individuals may in some cases have less functional propensity than a group of neurodivergent individuals, even if the former are considered individually more functional on orthodox accounts.

Hoffman's argument is based on the notion that a more neurologically diverse group would have a greater range of cognitive resources to draw on, thus making it likely to be superior when it comes to complex problem solving. Interestingly, this argument gains some support from recent research into the benefits of cognitive diversity, which has precisely been measured as a group trait. For instance, Torchia, Calabro, and Morner (2015) found an association between cognitive diversity and creativity in a study of company boards, and Liao and Long (2016) found that cognitive diversity in groups significantly increased team performance. In line with these findings are those from Reynolds and Lewis (2017), who assessed how well different groups deal with environmental complexity and reported that, from the group perspective, "cognitive diversity is what we need to succeed in dealing with new, uncertain, and complex situations" (para. 23). Furthermore, on the basis of an analysis of historical examples, Syed (2019) suggested that cognitive diversity can help to avoid confirmation bias and minimize blind spots, which may help to explain why cognitively diverse teams tend to perform better.

This does not mean that increased cognitive or neurological diversity will necessarily increase the functional propensity of all groups. Aggarwal, Wooley, Chabris, and Malone (2019) found that although optimum levels of cognitive diversity led to increased group intelligence, past a certain point diversity levels began to hinder communication, leading to diminished group intelligence. Moreover, Chen, Liu, Zhang, and Kwan (2019) found that high levels of cognitive diversity increased group reflexivity but could also contribute to conflict. Interestingly, however, Wang, Kim, and Lee

(2016) found that transformational leadership helps to harvest the benefits of cognitive diversity while minimizing its associated problems. Relatedly, as neurodiversity proponent Jorn Bettin (2019) wrote,

> Neurodiversity friendly forms of collaboration hold the potential to transform pathologically competitive and toxic teams and cultures into highly collaborative teams and larger cultural units that work together more like an organism rather than like a group of fighters in an arena. (para. 2)

More research clearly needs to be done to understand how to best cultivate the group benefits of cognitive diversity. Still, from what we do know, it seems that although extreme levels of cognitive diversity may lead to mixed results, relatively cognitively diverse groups will often perform better than cognitively uniform groups. Moreover, problems associated with more extreme levels of cognitive diversity may arise from contingent social factors such as inadequate leadership rather than from the cognitive makeup of the group as such. What has been found so far therefore gives preliminary support to Hoffman's hypothesis, meaning that neurodiversity may contribute to emergent group-functional propensities even in cases in which specific niche contributions have not been identified. The ecological model has the benefit of acknowledging emergent group functions that may be overlooked in individualist accounts.

Limitations and Implications

In proposing the ecological model I have aimed to show that there is room for a viable functional analysis on the neurodiversity paradigm that can be used as an alternative to comparatively individualistic, fitness-based evolutionary models. To summarize, there are three key differences between evolutionary accounts and the ecological model. First, my account is oriented toward the future, whereas the respective accounts of Boorse and Wakefield are oriented toward the present or past. Second, the ecological model is multilevel in that it takes group propensities and niche contributions into account alongside individual propensities. Third, the ecological model has a relational conception of functions rather than taking them to be intrinsic. Framed in this manner, the claim that many minority ways of being associated with individual disability are natural human variations (rather than dysfunctions) is feasible.

It is important to stress that I do not mean to present the ecological model as a rival to evolutionary models per se. After all, my model is specifically for

understanding human mental functioning only, not for understanding biological functioning. It may also be that which model is preferable will be determined by the specific case. As Maung (2018) has argued, rather than there being a single "correct" model of health or functions, different models will be more or less useful in different instances. I suspect the ecological model will be more helpful for thinking about autism, dyslexia, and many other mental disabilities but much less so for thinking about, say, infant anencephaly, which in my view would be best framed as an inherently dysfunctional neurological disorder. By being able to take a range of functions that are obscured or overlooked by more individualistic evolutionary accounts, the ecological model allows us to recognize a greater variety of cognitive differences without unduly pathologizing them.

It is also important to note that I do not claim to have solved all the problems I associated with the evolutionary models. For instance, I do not claim that I can precisely draw the line between function and dysfunction any more easily than the evolutionary accounts can. Whatever way function and dysfunction are measured, some reference class or another will be proposed, and it is hard to avoid at least some level of arbitrariness when determining the appropriate reference class, which makes the possibility of error impossible to wholly eliminate.[5] Despite this, I take the possibility of error to be less of an issue for my approach. Being able to objectively demarcate exactly who is functional or dysfunctional is necessary for medical research projects that categorize humans on the basis of those who are mentally well and those who are mentally disordered. But because the ecological model rejects this approach and instead frames functions and dysfunctions as relational, then the issue of where we draw the line becomes far less pressing. Hence, although this issue is not solved on this account, it is dissolved to some extent, because the importance of getting the distinction exactly right stems from the high stakes involved in the medical approach.

Beyond its theoretical benefits, part of my argument for the ecological model is a pragmatic one: It may serve as a useful basis for directing future scientific research, clinical understanding, and the public understanding of disability. For scientific researchers, adopting this analysis will lead them to ask not "What is wrong with this individual or group in relation to those who are normal?" but rather "How can we understand the strengths, limitations, struggles, or potential of this group or individual in the wider social context?" Arguably, this reorientation might help to capture the complexity of disability and ability in their broader social context in a way that is more nuanced than

existing models. In turn, the ecological model might also allow great flexibility when it comes to psychotherapeutic practice or the development of individual self-understanding because it is better at capturing the complexity of psychological ability and disability in their broader contexts than the orthodox accounts. Finally, when it comes to public understanding, the shift away from individual pathology to relational dysfunction and function may help to alleviate the stigma surrounding disability. Although it is by no means necessary that an individual should benefit the community for their rights and value to be recognized—as mentioned above, it is a core component of the neurodiversity perspective to affirm the value and rights of disabled people regardless of functional propensities—a reorientation toward recognizing neurodivergence as part of humanity's social ecology would be a positive one. The concept of neurodiversity, as Judy Singer (2020) has recently written, orients us toward seeing that "the more neurodiversity is respected and facilitated within a culture, the more stable, adaptable and sustainable that culture is" (para. 31). Building this insight into scientific and clinical understandings would truly bring about a paradigm shift in the science of psychological ability and disability.

Transparency

ORCID iD

Robert Chapman (iD) https://orcid.org/0000-0003-1220-6068

Acknowledgments

Notes

1. Used in this way at least, the terms "impairment" and "dysfunction" are largely synonymous. Each refers to individual biological limitations associated with disabilities (Barnes, 2016; Boorse, 2010). The key difference is that the former is more often used in disability studies, whereas the latter is more often used in psychiatric theory. Here I stick with the latter, but my argument applies to either.

2. Indeed, it is notable that in a recent critique of the neurodiversity perspective, Wakefield et al. (2020) fall back on general claims based on implicit species norms, for instance, that "humans are social animals" (p. 14) and that people with some forms of autism would be excluded from "any plausible understanding of human flourishing" (p. 13). Although it is beyond the scope of this article, it is worth noting that neurodiversity proponents precisely challenge dominant assumptions about flourishing as well as functioning (Rodogno et al., 2016).

3. As noted above, there is some conceptual space for group functions in the etiological account, but because this seems to rely on group selection, it is generally thought to be very minimal and has generally been seen as controversial. For a recent and interesting discussion of collective dysfunction on the etiological account that does not rely on group selection, see Hoffman (2017).

4. Many proponents of the medical view would, of course, also agree (e.g., Wakefield et al., 2020). However, some (e.g., Barnbaum, 2008) have argued that profoundly disabled autistic individuals should not be considered persons who automatically qualify for human rights. From the neurodiversity perspective, the medical-deficit framing of neurodivergence lends itself to this way of thinking even if there is no necessary connection.

5. Although I do not have room to explore this here, unlike Boorse's focus on what he took to be natural kinds, I would take a more relaxed approach to the reference class. After all, autism and other neurodivergent disabilities, despite being defined by rough clusters of cognitive, biological, and behavioral traits, are not natural kinds, but they are socially and politically useful classifications (Chapman, 2020b).

References

Aggarwal, I., Wooley, A. W., Chabris, C. F., & Malone, T. W. (2019). The impact of cognitive style diversity on implicit learning in teams. *Frontiers in Psychology*, *10*, Article 112. doi:10.3389/fpsyg.2019.00112

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Amundson, R. (2000). Against normal function. *Studies in History and Philosophy of Science C: Biological and Biomedical Sciences*, *31*, 33–53. doi:10.1016/S1369-8486(99)00033-3

Arah, O. A. (2009). On the relationship between individual and population health. *Medicine, Health Care and Philosophy*, *12*, 235–244. doi:10.1007/s11019-008-9173-8

Armstrong, T. (2010). *Neurodiversity: Discovering the extraordinary gifts of autism, ADHD, dyslexia, and other brain differences*. Cambridge, MA: Da Capo.

Armstrong, T. (2015). The myth of the normal brain: Embracing neurodiversity. *AMA Journal of Ethics*, *17*, 348–352. doi:10.1001/journalofethics.2015.17.4.msoc1-1504

Aspinal, D. (2020, April 16). Two thirds of our team is autistic: That's helping us survive the coronavirus. *CNBC*. Retrieved from https://www.cnbc.com/2020/04/16/our-it-team-is-autistic-thats-helping-us-survive-the-coronavirus.html

Badcock, C. (2009). *The imprinted brain: How the genes set the balance between autism and psychosis*. London, England: Jessica Kingsley.

Baggs, A. (2010). Up in the clouds and down in the valley: My richness and yours. *Disability Studies Quarterly*, *30*(1). doi:10.18061/dsq.v30i1.1052

Barnbaum, D. (2008). *The ethics of autism: Among them but not of them*. Bloomington: Indiana University Press.

Barnes, E. (2016). *The minority body: A theory of disability*. New York, NY: Oxford University Press.

Baron-Cohen, S. (2006). Two new theories of autism: Hyper-systemising and assortative mating. *Archives of Disease in Childhood*, *91*, 2–5. doi:10.1136/adc.2005.075846

Baron-Cohen, S. (2011). *Zero degrees of empathy: A new theory of human cruelty*. London, England: Allen Lane.

Baron-Cohen, S. (2017). Editorial perspective: Neurodiversity—A revolutionary concept for autism and psychiatry. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *58*, 744–747. doi:10.1111/jcpp.12703

Bettin, J. (2019, June 28). Organising for neurodivergent collaboration. *Autistic Collaboration*. Retrieved from https://autcollab.org/2019/06/28/organising-for-neurodivergent-collaboration

Bigelow, J., & Pargetter, R. (1987). Functions. *Journal of Philosophy*, *84*, 181–196.

Blume, H. (1998, September). Neurodiversity: On the neurological underpinnings of Geekdom. *The Atlantic*. Retrieved from https://www.theatlantic.com/magazine/archive/1998/09/neurodiversity/305909

Bolton, D. (2008). *What is mental disorder?* New York, NY: Oxford University Press.

Boorse, C. (1975). On the distinction between disease and illness. *Philosophy and Public Affairs*, *5*, 49–68.

Boorse, C. (1976). Wright on functions. *Philosophical Review*, *85*, 70–86.

Boorse, C. (1977). Health as a theoretical concept. *Philosophy of Science*, *44*, 542–573.

Boorse, C. (1997). A rebuttal on health. In J. M. Humber & R. F. Almeder (Eds.), *What is disease?* (pp. 1–134). Totowa, NJ: Humana Press.

Boorse, C. (2002). A rebuttal on functions. In A. Ariew, R. C. Cummins, & M. Perlman (Eds.), *Functions: New essays in the philosophy of psychology and biology* (pp. 63–112). New York, NY: Oxford University Press.

Boorse, C. (2010). Disability and medical theory. In D. C. Ralston & J. Ho (Eds.), *Philosophical reflections on disability* (pp. 55–58). Amsterdam, the Netherlands: Springer.

Boorse, C. (2014). A second rebuttal on health. *Journal of Medicine and Philosophy*, *39*, 683–724. doi:10.1093/jmp/jhu035

Chapman, R. (2019a). Autism as a form of life: Wittgenstein and the psychological coherence of autism. *Metaphilosophy*, *50*, 421–440. doi:10.1111/meta.12366

Chapman, R. (2019b). Neurodiversity theory and its discontents: Autism, schizophrenia, and the social model. In S. Tekin & R. Bluhm (Eds.), *The Bloomsbury companion to the philosophy of psychiatry* (pp. 371–389). London, England: Bloomsbury.

Chapman, R. (2020a). Neurodiversity, wellbeing, disability. In H. Rosquvist, N. Chown, & A. Stenning (Eds.), *Neurodiversity studies: A new critical paradigm* (pp. 57–72). New York, NY: Routledge.

Chapman, R. (2020b). The reality of autism: On the metaphysics of disorder and diversity. *Philosophical Psychology*, *33*, 799–819. doi:10.1080/09515089.2020.1751103

Chen, X., Liu, J., Zhang, H., & Kwan, H. K. (2019). Cognitive diversity and innovative work behaviour: The mediating roles of task reflexivity and relationship conflict and the moderating role of perceived support. *Journal of Occupational and Organisational Psychology*, *92*, 671–694.

Cooper, R. (2002). Disease. *Studies in History and Philosophy of Science C: Biological and Biomedical Sciences*, *3*, 263–282.

Costandi, M. (2019, September 12). Against neurodiversity. *Aeon*. Retrieved from https://aeon.co/essays/why-the-neurodiversity-movement-has-become-harmful

Delehanty, M. (2019). The changing landscape of the philosophy of medicine. *Philosophy Compass*, *14*(8), Article e12612. doi:10.1111/phc3.12612

De Marinto, B., Harrison, N., Knafo, S., Bird, G., & Dolan, R. (2008). Explaining enhanced logical consistency during decision making in autism. *The Journal of Neuroscience*, *28*, 10746–10750. doi:10.1523/JNEUROSCI.2895-08.2008

Dinishak, J. (2016). The deficit view and its critics. *Disability Studies Quarterly*, *36*(4). doi:10.18061/dsq.v36i4.5236

Drayson, Z., & Clarke, A. (2020). Cognitive disability and embodied, extended minds. In D. Wasserman & A. Cureton (Eds.), *Oxford handbook of philosophy and disability* (pp. 580–597). New York, NY: Oxford University Press. doi:10.1093/oxfordhb/9780190622879.013.10

Dussault, A. C. (2019). Functional biodiversity and the concept of ecological function. In E. Casetta, J. Marques da Silva, & D. Vecchi (Eds.), *From assessing to conserving biodiversity: Conceptual and practical challenges* (pp. 297–316). Amsterdam, the Netherlands: Springer. doi:10.1007/978-3-030-10991-2_14

Dussault, A. C., & Bouchard, F. (2017). A persistence enhancing propensity account of ecological function to explain ecosystem evolution. *Synthese*, *194*, 1115–1145. doi:10.1007/s11229-016-1065-5

Fitch, A., Fein, D. A., & Eigsti, I. (2015). Detail and Gestalt focus in individuals with optimal outcomes from autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *45*, 1887–1896. doi:10.1007/s10803-014-2347-8

Fuchs, T. (2017). *Ecology of the brain. The phenomenology and biology of the embodied mind*. New York, NY: Oxford University Press.

Grinker, R. R. (2015). Reframing the science and anthropology of autism. *Culture, Medicine, & Psychiatry*, *39*, 345–350. doi:10.1007/s11013-015-9444-9

Greenberg, D. M., Warrier, V., Allison, C., & Baron-Cohen, S. (2018). Testing the empathizing-systemizing theory of sex differences and the extreme male brain theory of autism in half a million people. *Proceedings of the National Academy of Sciences, USA*, *115*, 12152–12157. doi:10.1073/pnas.1811032115

Happe, F., & Vital, P. (2009). What aspects of autism predispose to talent? *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *364*, 1369–1375. doi:10.1098/rstb.2008.0332

Higashida, N. (2017). *Fall down seven times: Get up eight. A young man's voice from the silence of autism*. London, England: Hodder & Stoughton.

Hoffman, G. A. (2016). Out of our skulls: How the extended mind thesis can extend psychiatry. *Philosophical Psychology*, *29*, 1160–1174.

Hoffman, G. A. (2017). Collectively ill: Reasons for psychiatry to think that groups can possess mental disorders. *Synthese*, *196*, 2217–2241. doi:10.1080/09515089.2016.1236369

Jaarsma, P., & Welin, S. (2012). Autism as a natural human variation: Reflections on the claims of the neurodiversity movement. *Health Care Analysis*, *20*, 20–30. doi:10.1007/s10728-011-0169-9

Kapp, S. K., Gillespie-Lynch, K., Sherman, L. E., & Hutman, T. (2013). Deficit, difference, or both? Autism and neurodiversity. *Developmental Psychology*, *49*, 59–71. doi:10.1037/a0028353

Kingma, E. (2007). What is it to be healthy? *Analysis*, *67*, 128–133. doi:10.1111/j.1467-8284.2007.00662.x

Liao, Z., & Long, S. (2016). Cognitive diversity, alertness, and team performance. *Social Behavior and Personality: An International Journal*, *44*, 209–220. doi:10.2224/sbp.2016.44.2.209

Maclaurin, J., & Sterelny, K. (2008). *What is biodiversity?* Chicago, IL: University of Chicago Press.

Maung, H. H. (2018). Is infertility a disease and does it matter? *Bioethics*, *3*, 343–353. doi:10.1111/bioe.12495

McCafferty, K. R. (2018, October 5). My son's future may look different from others, but I know he is happy. *Autism Speaks*. Retrieved from https://www.autismspeaks.org/blog/my-sons-future-may-look-different-others-i-know-he-happy

Milton, D. (2012). On the ontological status of autism: The 'double empathy problem.' *Disability & Society*, *27*, 883–887. doi:10.1080/09687599.2012.710008

Milton, D. (2017). *A mismatch of salience: Explorations of the nature of autism from theory to practice*. London, England: Pavilion Press.

Mottron, L. (2011). The power of autism. *Nature*, *479*, 33–35.

Murphy, D. (2006). *Psychiatry in the scientific image*. Cambridge, MA: MIT Press.

Murphy, D., & Woodfolk, R. (2000). The harmful dysfunction analysis of mental disorder. *Philosophy, Psychiatry, & Psychology*, 241–252.

Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, *9*, 139–156. doi:10.1177/1362361305051398

Ne'eman, A. (2010). The future (and the past) of autism advocacy, or why the ASA's magazine, *The Advocate*, wouldn't publish this piece. *Disability Studies Quarterly*, *30*(1). doi:10.18061/dsq.v30i1.1059

Nesse, R. M. (2001). On the difficulty of defining disease: A Darwinian perspective. *Medicine, Health Care and Philosophy*, *4*, 37–46. doi:10.1023/A:1009938513897

Nunes-Neto, N., Moreno, A., & El-Hani, C. N. (2014). Function in ecology: An organizational approach. *Biology & Philosophy*, *29*, 123–141. doi:10.1007/s10539-013-9398-7

Oishi, S., & Graham, J. (2010). Social ecology: Lost and found in psychological science. *Perspectives on Psychological Science*, *5*, 356–377. doi:10.1177/1745691610374588

Pellicano, E., & Stears, M. (2011). Bridging autism, science and society: Moving toward an ethically informed approach to autism research. *Autism Research*, *4*, 271–282. doi:10.1002/aur.201

Reynolds, A., & Lewis, D. (2017, March 30). Teams solve problems faster when they're more cognitively diverse. *Harvard Business Review*. Retrieved from https://hbr.org/2017/03/teams-solve-problems-faster-when-theyre-more-cognitively-diverse

Robertson, S. (2010). Neurodiversity, quality of life and autistic adults: Shifting research and professional focuses onto real-life challenges. *Disability Studies Quarterly*, *30*(1). doi:10.18061/dsq.v30i1.1069

Rodogno, R., Krause-Jensen, K., & Ashcroft, R. E. (2016). Autism and the good life: A new approach to the study of well-being. *Journal of Medical Ethics*, *42*, 401–408. doi:10.1136/medethics-2016-103595

Silberman, S. (2019, September 24). Greta Thunberg became a climate activist not in spite of her autism, but because of it. *Vox*. Retrieved from https://www.vox.com/first-person/2019/5/6/18531551/greta-thunberg-autism-aspergers

Singer, J. (1999). Why can't you be normal for once in your life? From a 'problem with no name' to the emergence of a new category of difference. In M. Corker & S. French (Eds.), *Disability discourse* (pp. 59–67). London, England: Open University Press.

Singer, J. (2019, October 10). Neurodivergent from what, exactly? [Blog post]. Retrieved from https://neurodiversity2.blogspot.com/2019/09/question-neurodivergent-from-what.html

Singer, J. (2020). What is neurodiversity? [Blog post]. Retrieved from https://neurodiversity2.blogspot.com/p/what.html

Syed, M. (2019). *Rebel ideas: The power of diverse thinking*. London, England: Hachette UK.

Taylor, D. M., & de la Sablonnière, R. (2013). Why interventions in dysfunctional communities fail: The need for a truly collective approach. *Canadian Psychology/Psychologie canadienne*, *54*, 22–29. doi:10.1037/a0031124

Torchia, M., Calabro, A., & Morner, M. (2015). Board of directors' diversity, creativity, and cognitive conflict. *International Studies of Management and Organization*, *45*, 6–24. doi:10.1080/00208825.2015.1005992

Valles, S. (2018). *Philosophy of population health: Ethics and evidence in the new public health era*. New York, NY: Routledge.

Varga, S. (2011). Defining mental disorder. Exploring the 'natural function' approach. *Philosophy, Ethics, and Humanities in Medicine*, *6*, Article 1. doi:10.1186/1747-5341-6-1

Wakefield, J. (1992a). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, *47*, 373–388. doi:10.1037//0003-066x.47.3.373

Wakefield, J. (1992b). Disorder as harmful dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review*, *99*, 232–247. doi:10.1037/0033-295X.99.2.232

Wakefield, J. (2015). Biological function and dysfunction: Conceptual foundations of evolutionary psychopathology. *The Handbook of Evolutionary Psychology*, *42*, 1–19. doi:10.1002/9781119125563.evpsych242

Wakefield, J., Wasserman, D., & Conrad, J. (2020). Neurodiversity, autism, and psychiatric disability: The harmful dysfunction perspective. In A. Cureton & D. T. Wasserman (Eds.), *The Oxford handbook of philosophy and disability* (pp. 501–521). New York, NY: Oxford University Press.

Walker, N. (2014, September 27). Neurodiversity: Some basic terms and definitions. *Neurocosmopolitanism*. Retrieved from https://neurocosmopolitanism.com/neurodiversity-some-basic-terms-definitions

Wang, X. H., Kim, T. Y., & Lee, D. R. (2016). Cognitive diversity and team creativity: Effects of team intrinsic motivation and transformational leadership. *Journal of Business Research*, *69*, 3231–3239. doi:10.1016/j.jbusres.2016.02.026

Wessel, I., & Moulds, M. L. (2008). Collective memory: A perspective from (experimental) clinical psychology. *Memory*, *16*, 288–304. doi:10.1080/09658210701811813

Wheelwright, S., & Baron-Cohen, S. (2001). The link between autism and skills such as engineering, maths, physics and computing: A reply to Jarrold and Routh, Autism, 1998, 2(3): 281-9. *Autism*, *5*, 223–227. doi:10.1177/1362361301005002010