The Trouble with Neurodiversity:
Etiologies, Normativity, and the Autistic Struggle for Identity

by

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Abstract

Scientific research into the etiology of autism has lead to an explosion in proposed agents implicated in the development of autism over the past 70 years. Genetics, neurotoxins, vaccinations, viral infections, parenting practices, neurological abnormalities, among others, have been proposed to explain what increasingly appears to be a heterogeneous and overdetermined condition. These proposed etiologies and the treatments they suggest pose a peculiar problem for the neurodiversity movement, an activist group of autistics and nonautistics who hope to promote a positive understanding of autism. In broad terms, the neurodiversity movement opposes cure-oriented research and activism typical of the scientific community and mainstream autism advocacy organizations. They hope to counter this trend by promoting autism as a positive identity – a normal human variation, rather than a pathology.

The tension between these two modes of thought provides a rich terrain for exploring the possibilities of identity formation even as human behaviour increasingly falls under the rubric of medical science. The scientific research discussed in this thesis simultaneously constructs and is constructed by an understanding of autism as a pathology, and in so doing challenges the claims of the neurodiversity movement both directly and indirectly: reproductive technologies and proposed treatments for autism force parents to make judgements about the worth of autistic persons, for example. This thesis draws on literature from bioethics, philosophy of medicine, and disability studies to situate both the neurodiversity movement and the scientific community in debates about normality, normativity, suffering, and the nature of disease. I argue that while the neurodiversity movement's emphasis on normality is ultimately misplaced, the movement nevertheless has much to teach us about rights, identity, authority, and self-determination.
Preface

This document is the original, unpublished work of the author, Josef Garen.
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1. Neurodiversity and its discontents

1.1 Introduction

Autism spectrum disorders (ASD), as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), are highly heritable neurodevelopmental disorders characterized by “Persistent deficits in social communication and social interaction” and “Restricted, repetitive patterns of behavior, interests, or activities.” In severe cases, autistic persons may have deficits in language use, intellectual impairment, and difficulties with sensorimotor skills. With as many as 1 in 88 children in North America diagnosed at some point on the autism spectrum and prevalence continuing to rise, ASD is often characterized as an “epidemic” and an “urgent global public health concern.” The number of diagnoses has increased dramatically since the condition was first described in the 1940s by Leo Kanner and Hans Asperg, but this increase has not been uniform. Reported rates of autism prevalence vary widely across the globe, and popular media has focused particular attention on purported autism “hotspots” in technology meccas such as Silicon Valley.

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5 This may be largely due to the extreme difficulty in carrying out cross-cultural studies of illness prevalence. See Elsabbagh, Mayada, et al., “Global Prevalence of Autism and Other Pervasive Developmental Disorders.” *Autism Research* 5, no. 3 (2012).
The geographical and temporal variability in the prevalence of the condition has many proposed explanations, from social factors such as increasing public familiarity with autism and broadening diagnostic boundaries, to physiological explanations including genetics, vaccinations, and environmental pollutants. Hundreds of millions of research dollars are devoted annually to this question, coming from governmental organizations such as the National Institutes of Health as well as various non-governmental organizations and charitable societies such as Autism Speaks/Cure Autism Now, the Organization for Autism Research, and Talk About Curing Autism. Much of this research, especially that funded by the latter organizations, is explicitly aimed at finding a cure – some method of ameliorating the symptoms of existing autistic persons or preventing new cases of autism from occurring.

In response to this dominant pattern of cure-oriented autism advocacy led by parents and family members of autistic children and the concomitant media portrayal of autism as a “tragedy,” a loose association of autistic bloggers, authors, and activists have come together to combat what they perceived to be an oppressive hegemonic framework of pathologization. The Neurodiversity Movement, as it is called, began in the late 1990s and grew through online message boards, chatrooms, and newsgroups, attempting to promote a different vision of autism. They saw autism not as a “tragedy” to be suffered through, nor as a disease or disability, but rather as a positive identity – a neurologically different way to be a person, and a difference that ought to be respected and cherished, just as we respect the differences between men and women, gay and straight people, and so forth. Neurodiversity advocates reject the dominant medical, scientific, and popular positions that autism ought to be cured, treated, or prevented when

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possible, promoting instead the acceptance and self-determination of autistic persons above all.

There is tension between these two different ways of characterizing autism as, most obviously, the “neurodiverse” and the “popular” differ on the question of whether autism ought to be cured. But there are subtler ways in which scientific research, especially etiological research, and the neurodiversity movement are at cross purposes. The goal of this thesis is to examine the ways in which scientific developments in autism etiology complement, contradict, and problematize the neurodiversity position, and vice versa. Knowing the origin of a condition not only changes how we think and talk about the condition, but it also suggests treatments for the condition, and in some cases can even change the nature of the condition itself. In this Chapter, I attempt to draw the battle lines in the debate between the scientific and neurodiverse concepts of autism. I begins in Section 1.2 by drawing on a variety of neurodiversity publications in an attempt to describe the core beliefs of the neurodiversity movement. These include the belief that autism is not a pathology and ought not be cured, that autism is a natural human variation, and that autism is an identity, as well as endorsements of nondiscrimination and the social model of disability. My hope is to provide an accurate and representative picture of what is ultimately a somewhat heterogenous and disunified movement, without aiming for complete generality.

In Section 1.3, I attempt to communicate the flavour and diversity of current scientific investigations into the etiology of autism. As whole volumes have been written on this topic, I can only briefly discuss the various factors that have been implicated in the production of autism, focusing primarily on the neurophysiological and biogenetic aspect of autism research. Genetics,

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8 This point is treated at great length in Hacking, Ian, The Social Construction of What? (Cambridge, MA: Harvard University Press, 1999). See especially Ch. 4.
industrial pollutants, vaccinations, viral infections, and immunological variables will be treated, as they are currently the most fashionable and productive avenues of scientific research and public debate. The state of the science paints a picture of autism as overdetermined, with individual cases of autism caused by heterogenous interactions of genetic predispositions with social and environmental factors; indeed, autism itself is beginning to look less like a unitary disorder, so much as a *family* of disorders that share certain biological pathways and cognitive and behavioural expressions.

Chapter 2 draws on the debates outlined in the first Chapter to address the central questions of this thesis. In it I will attempt to analyze the impact that etiological research has on the claims of the neurodiversity movement, and explicate the ways in which the tension between the “dominant” scientific view of autism and the neurodiversity view traces out the contours of much broader debates in bioethics, disability studies, and the philosophy of medicine dealing with the nature of disease and the goals of medicine. I focus my analysis on the normative standards and evaluative judgements implicit in the scientific research discussed in Chapter 1, also discussing the ethical dimensions of technologies currently proposed or in development that deal with autism in one way or another. I hope to use the neurodiversity debate to demonstrate the ways in which scientific knowledge of this kind is inherently value-laden. I close this thesis with some lingering questions and thoughts on future research directions.

1.2 Neurodiversity

The concept of “neurodiversity” was first employed in the late 1990s in response to a number of perceived problems in the way autism was understood in the popular imagination and depicted in
the media. Autism was frequently portrayed as a “tragedy,” with autistic persons regularly suffering bullying, abuse, and workplace discrimination; correspondingly, autism was mainly regarded as a medical problem to be addressed through the rubric of behavioural therapies, medication, and special education. Promoting an attitude of acceptance and respect toward autistic persons and the “neurodiverse,” Judy Singer used the term in her 1999 essay “Why can't you be normal for once in your life?” writing that

the key significance of the 'autism spectrum' lies in its call for and anticipation of a politics of neurological diversity, or neurodiversity. The 'neurologically different' represent a new addition to the familiar political categories of class/gender/race and will augment the insights of the social model of disability.⁹

Though Singer is often cited as coining the term “neurodiversity” in this essay, earlier, in 1998, Harvey Blume wrote an article for The Atlantic titled “Neurodiversity: On the Neurological Underpinnings of Geekdom” which discusses the supposedly elevated rates of autism spectrum disorders, especially Asperger syndrome, in Silicon Valley. Blume's article trades on the stereotype of the mildly autistic person as being talented with technology and computers, suggesting that Asperger syndrome may be beneficial for the development of future technologies and thus “Neurodiversity may be every bit as crucial for the human race as biodiversity is for life in general.”¹⁰ During the decade of the 2000s, the idea spread quickly across the internet, being adopted by bloggers, activists, websites and organizations dedicated to autism self-advocacy. Today, these include large advocacy organizations such as the Autism Self Advocacy Network,¹¹

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⁹ Singer, Judy. “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 Disability Discourse, ed. Mairian Corker and Sally French (Philadelphia: Open University Press, 1999), 64.

¹⁰ Blume, “Neurodiversity.”

¹¹ See http://autisticadvocacy.org/.
Aspies for Freedom,\textsuperscript{12} and Autism Network International,\textsuperscript{13} as well as countless individual bloggers, webpages, newsgroups and the like.

The neurodiversity movement poses a bit of a problem for anyone who would like to make categorical statements about it, composed as it is of a heterogeneous collection of individuals and organizations who all state their beliefs in different ways. Nevertheless, the position of the individuals and organizations who align themselves with this ideology share many commonalities which can be roughly broken into five interrelated claims. These are:

1. Autism is a natural variation among humans, not a disease or disability,

2. Autism does not need to be (or cannot be) cured,

3. Autism is an integral part of a person's identity,

4. Autistic persons deserve the same rights and social acceptance as anyone else, and

5. Autism, like many other conditions, is best understood through the social model of disability.

It would be overstating the case to say that all of these tenets are universally accepted within the movement, but as each one is espoused quite commonly by prominent organizations and members of the neurodiversity community, we shall consider this list to be representative of the general position of the movement and note contentions and disagreements where they occur.\textsuperscript{14}

\textsuperscript{12} See http://www.aspiesforfreedom.com/.
\textsuperscript{13} See http://www.autreat.com/.
Each of these claims will be discussed in turn.

1.2.1 Natural human variation

The first claim in this taxonomy is *Autism is a natural variation among humans, not a disease or disability.*\(^{15}\) John Elder Robison, an activist and advocate with Asperger syndrome who has written several books including the bestselling autobiography *Look Me in the Eye*, as well as formerly sitting on the advisory committee for Autism Speaks,\(^{16}\) writes that

> neurodiversity is the idea that neurological differences like autism and ADHD are the result of normal, natural variation in the human genome. This represents a new and fundamentally different way of looking at conditions that were traditionally pathologized; it’s a viewpoint that is not universally accepted though it is increasingly supported by science. . . . We are not sick. We are different.\(^{17}\)

This claim is echoed in neurodiversity publications across the Internet, from simple statements on personal blogs stating that “[neurodiversity holds that] Autism is a natural human variation,”\(^{18}\) to the National Symposium on Neurodiversity at Syracuse University, whose website states that

> neurodiversity is viewed is a concept and social movement that advocates for viewing autism as a variation of human wiring, rather than a disease.\(^{19}\)

The neurodiversity movement characterizes autism as a *variation* among humans,

\(^{16}\) Robison recently resigned from his position in protest of an op-ed piece published by Suzanne Wright, one of the founders of Autism Speaks. Wright's article was perceived by Robison and many autistic persons as “pro-cure.”
choosing to use the language of difference in lieu of the traditional medicalized language of diseases and disabilities to emphasize their opposition to the dominant, medicalized concept of autism. They describe these differences or variations as *natural* or *normal*, often making explicit reference to the genetic cause of the condition, or its neurological underpinnings. (This characterization of autism as a variation in “brain wiring” or a “neurological variation” is, of course, where the term “neurodiversity” gets its name from.) In short, this tenet posits a natural, endogenous, and fully “biologized” account of autism. This fact, as well as the possible meanings of the words “normal” and “natural” used in this context will be discussed further when we turn our attention to the etiological research currently underway.

**1.2.2 Identity**

The second claim of the neurodiversity movement is *Autism is an integral part of a person's identity.*\(^{20}\) Jim Sinclair, an autistic author and self-advocate well-known in the neurodiversity community, explains in the essay “Don't Mourn for Us” that

> Autism isn't something a person *has*, or a “shell” that a person is trapped inside. There's no normal child hidden behind the autism. Autism is a way of being. It is *pervasive*; it colors every experience, every sensation, perception, thought, emotion, and encounter, every aspect of existence. It is not possible to separate the autism from the person – and if it were possible, the person you'd have left would not be the same person you started with.\(^{21}\)

Many prominent autistic figures have made similar remarks about their own experiences with

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autism. Temple Grandin, an animal scientist, professor, best-selling author, and autistic self-advocate, is often quoted in support of this claim as saying, “If I could snap my fingers and be nonautistic, I would not. Autism is a part of what I am.” 22 Autistic blogger Lisa D. expresses similar sentiments, writing

I am autistic, and my autism is not separate from my identity. “Autistic” is part of what defines me, just like “college student” and “American” and “short.” If it's okay for me to say that being female is part of who I am, then why can't I say that about autism? Or is it because disability is something that's so terrible that we need to reject it and pretend it doesn't exist? 23

Lisa D. here points to the way that neurodiverse persons deliberately blur the line between “disability” and identity. Reappropriating the “autism” label is empowering, giving autistic persons the ability to take pride in and ownership of their “quirks,” and to feel as though they belong to a small, but perhaps uniquely privileged, community. 24

Somewhat parenthetically, it should be noted that this sense of ownership and identity surrounding autism has motivated many activists to oppose the use of “person-first” language when speaking about autism. That is, the use of phrases such as “person with autism” have come under fire from neurodiversity advocates as they are seen to imply that autism is a disease,

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24 While it is a central tenet of the neurodiversity movement that autism is an identity, I should clarify that not all _autistic_ people feel this way. Most notably, perhaps, the autistic author Donna Williams has stated that “Autism is not me. Autism is just an information processing problem that controls who I am.” Quoted in Grandin, “Thinking in Pictures,” 50.
illness, or condition “overlaid” upon, and separable from, an otherwise healthy person. These individuals feel that this usage denies their identity and implicitly endorses the “cure mentality.” It has been suggested, rather, that “identity-first” language such as “autistic person,” “ASD person,” or simply “autistic” (used as a noun) be employed instead, which are seen to accord with their view that autism is an integral part of the identity of the person.\(^{25}\) While I have no desire to make premature judgements through slipshod word choice (these kinds of identity claims are, after all, partly what is at stake in this discussion), I will follow the convention among neurodiversity publications and use identity-first language in the following pages.

Individuals who have received a formal diagnosis of ASD or who have self-diagnosed themselves with the condition, especially as adults, often feel that the label powerfully recontextualizes and, crucially, explains behaviours and experiences in their lives that they were previously unable to understand. Dawn Prince-Hughes, an autistic primatologist and author writes of her experience being diagnosed as an adult that “everything suddenly made sense. I looked back over my life, perhaps the way people do before they die, and thought of all the painful memories that could now be explained. . . . It made me feel both better and worse knowing that I hadn't meant to disturb or hurt anyone.”\(^{26}\) Experiences such as difficulty fitting in with peers, the inability to participate in unstructured activities, and characteristically autistic behaviours such as rocking, hand-wringing, or echolalia previously described as “crazy” take on


new meaning in a life narrative newly structured around a nosological category.\textsuperscript{27}

It is unclear, however, whether a diagnosis of ASD really has the power to explain any of these things. Although, as stated above, mention may be given to genetics or differences in “brain wiring,” a diagnosis of ASD, as with all conditions in the DSM-5, is ultimately based on behavioural characteristics. As the DSM-5, like its predecessors, is explicitly agnostic about the etiology of the conditions it lists,\textsuperscript{28} the conditions it purports to diagnose are implicitly defined by the presence of certain constellations of symptoms. The categories of disorder in the DSM-5 are purely descriptive; in this sense, the label of “autism” is just that – a name that denotes a family of behaviours. To say, then, that a diagnosis of autism “explains” the behaviours of the person would be akin to saying that a person's shyness is explained by the fact that she is shy. In the case of autism, as in the majority of mental illnesses defined in this manner, however, no causally effective pathology has yet been identified, thus etiological research may have an important role to play in helping autistic persons to understand their own experiences, as will be discussed further in the following sections.

\textbf{1.2.3 Anti-cure}

Following closely from previous two claims is the claim that \textit{Autism should not be cured, treated or prevented}.\textsuperscript{29} Given that autism is a natural human variation, “neurodiversity activists reject the idea that autism should be cured, advocating instead for celebrating autistic forms of communication and self-expression, and for promoting support systems that allow autistic people

\textsuperscript{27} This point is treated at greater length in Hacking, “Genetics,” and Hacking, Ian, “Kinds of People: Moving Targets,” \textit{Proceedings of the British Academy} 151 (2007).

\textsuperscript{28} American Psychiatric Association, \textit{DSM-5}.

to live as autistic people.” Casting autism as a variation, advocates claim that in the same sense as we would think it ridiculous to say that we ought to “cure” someone of their gender, sexual orientation, or skin colour, we ought also to think it ridiculous to try to “cure” autism.

This claim may also be stated as *Autism cannot be cured*. This follows from the second claim in this taxonomy, that autism is an integral part of the person's identity. In Sinclair's view, the integrality of autism to one's identity implies that a “cure” would annihilate the individual. He claims that parents who wish their autistic child could be cured are, in effect, wishing that “the autistic child [they] have did not exist, and [they] had a different (non-autistic) child instead.” This statement has been challenged by parents of autistic persons who pursue potential cures and treatments; seeing their actions as motivated by their love and concern for their child's well-being, they understandably resent being told that their actions indicate that they do not truly love *their* child, but rather the child they “love” is an imaginary, nonautistic one.

Neurodiversity advocates oppose painful, uncomfortable, and potentially dangerous treatments such as chelation therapy and certain forms of applied behavioural analysis (ABA), for example. Especially opposed are treatments aimed at “normalizing” the autistic child, and many advocates feel that characteristically autistic “stimming” behaviours targeted by these treatments such as hand-flapping and rocking are harmless, and may even be beneficial for the person in question. Not all potential treatments are opposed by all members of the movement,

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32 Chelation therapy is an effective treatment for some forms of heavy metal toxicity which has been used by some parents with the belief that autism results from some kind of chemical poisoning, such as that from adjuvants in vaccinations. It has not been approved for treatment of autism and has the potential for harmful side effects; chelation therapy has resulted in the death of at least one autistic child. See Atwood, Kimball C., et al., “Why the NIH Trial to Assess Chelation Therapy (TACT) Should be Abandoned,” *The Medscape Journal of Medicine* 10, no. 5 (2008): 115.
However. Certain behavioural interventions and medications, for example, aimed at ameliorating the most disabling symptoms of autism, such as self-injurious or obsessive-compulsive-like behaviours, are often considered beneficial to the well-being of autistics. Therapy and education aimed at improving the autistic persons ability to function in society, communicating with “neurotypicals,” gaining employment appropriate to their abilities, and so forth, are also generally looked favourably upon. Therapy, cure, and prevention methods will be discussed at greater length in Chapter 2.

1.2.4 Rights and acceptance

The next claim is Autistic persons deserve equal rights, appropriate accommodations, social acceptance, and self-determination. The Autism Self Advocacy Network characterizes the neurodiversity movement as one which

promotes social acceptance of neurological difference as part of the broad landscape of human diversity and seeks to bring about a world in which Autistic people enjoy the same access, rights, and opportunities as all other citizens. Acceptance of difference is essential to understanding, accepting, and benefiting from the contributions of everyone in our society, thus allowing all people to live up to their potential.

Many autistic persons feel ostracized or discriminated against due to their condition (or due to

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33 When neurodiversity activists decide which kinds of treatments are acceptable and which are not, one can see this act as an act of essentialism – they are actively engaged in defining the essence of autism. “Characteristically autistic behaviours” such as echolalia and restricted patterns of interest are not to be interfered with – they belong to the essence of autism. “Accidental symptoms” such as self-injury do not, even though they may be equally caused by the same underlying condition. This point is treated at much greater length in Nadesan, Majia Holmer, Constructing Autism: Unravelling the Truth' and Understanding the Social, (New York: Routledge, 2005). See especially Chapter 7.


social conditions, as will be discussed in the next subsection), and the neurodiversity movement is thus explicitly aimed at addressing these problems. This includes educational campaigns designed to increase public awareness of autism, attempts to reduce bullying in school environments, reintegrating autistic children into “mainstream” classroom environments, and helping autistic persons secure employment and gain necessary workplace accommodations. High-profile, successful autistics such as Temple Grandin are often employed to help change public perceptions and reduce prejudicial attitudes.

Perhaps the most important part of this claim is the insistence on self-determination. Many decisions, such as those relating to therapeutic options, school placement, and institutionalization, are made on behalf of autistic persons in ways that are seen to interfere with their autonomy. Thus the neurodiversity movement is explicitly one of self-advocacy, arguing that autistic persons are in the best position to make choices in their own lives, and there ought to be no decisions made about autistic persons without the inclusion of autistic persons in the decision making process.

1.2.5 Social model of disability

Many neurodiversity advocates also explicitly embrace the social model of disability,\textsuperscript{36} wherein it is understood that one's body or mind is never disabled in isolation – only in the context of one's social and physical milieu does disability emerge. The social model draws a distinction between impairments, which relate to the physical and mental capacities of individuals, and disabilities, which are seen as socially constructed. These disabling social conditions are embedded in the norms, expectations, and institutional practices in one's environment.

In this capacity, autism is frequently compared with homosexuality. In a homophobic society, it is argued, homosexual people will routinely appear mentally disordered or disabled due to hegemonic social conditions that systematically disable homosexuals as compared with heterosexuals. Workplace discrimination, hate crimes, the inability to marry, and so forth all contribute to construct homosexuals as ill. These observations may go a long way toward explaining why (but not excusing the fact that) homosexuality was still “officially” considered a mental illness until the 1970s. Neurodiversity advocates argue that a similar kind of discriminatory social framework is in place today with regard to autistic and other neurodiverse persons. Social stigma, popular misconceptions, and inadequate accommodations for autistic persons in particular may make it difficult for them to secure gainful employment or forge meaningful personal relationships. These pervasive social conditions moreso than any features inherent to autism itself, it is argued, systematically construct the condition as “disabling” and contribute to the oppression of autistic persons. Thus, the neurodiversity movement is one which explicitly addresses the normative dimension of medical and social practices.

... These various claims overlap and intersect with one another in important ways, and the choice to break the neurodiversity position into five separate subclaims is somewhat arbitrary. Although many of the writers and organizations mentioned above are speaking specifically about autism and autism spectrum disorders, and the word “neurodiversity” is most often seen in that context, the concept and the movement have been expanded by some activists to encompass an inclusive attitude toward people with many different types of neurological differences. Many activists make this explicit, as in the case of the blog *Neurodiversity Now*, which states that
“Neurodiversity is a concept where neurological differences are to be recognized and respected as any other human variation. These differences can include those labeled with Dyspraxia, Dyslexia, Attention Deficit Hyperactivity Disorder, Dysequilibrium, Autistic Spectrum, Tourette Syndrome, and others.” 37 This deliberate inclusiveness 38 is no doubt important to the neurodiversity movement, but will be mostly glossed over in the following pages, as I will be treating the etiology of autism.

1.3 Controversy and criticism

Neurodiversity itself is not an uncontested concept. Even among autistic persons, there is not complete consensus on what the term means, and some reject the concept entirely, as one autistic person who defines neurodiversity as “The idea that we autistic folks are not 'abnormal,' just a different kind of normal. (This is bullshit.)” 39 Similarly a blogger by the name of Johnathan describes himself as “An Autistic who wishes a cure could be found, though I know that might not happen in my lifetime,” making it his goal with his blog to “try to mostly show what a scam neurodiversity is.” 40 The pro-cure position is well represented by parents of autistic children and organizations such as Autism Speaks 41 and Talk About Curing Autism, 42 but pro-cure autistic individuals seem to have a weaker Internet presence than does the neurodiversity movement.

Perhaps this is because they are less common than autistic persons who adhere to the

38 There is an interesting exclusive side to this type of neurodiversity, insofar as it includes some but not all of the “neurologically different” under its banner, and in so doing may commit some of the same marginalizing normative judgements of which its advocates are otherwise so critical. This point perhaps needs further investigation but is beyond the scope of this thesis.
39 Quoted in Kapp et al., “Deficit,” 64.
41 http://www.autismspeaks.org/
42 https://www.tacanow.org/
neurodiversity ideology, or perhaps because their views are already well represented by the dominant stream of scientific research and pro-cure activism.

One objection raised by critics of the neurodiversity programme is a potential for self-selection bias in the available literature. It has been suggested that since autism self-advocates must be capable, at the very least, of communicating in some modality – typically in print – that higher-functioning individuals, such as those with Asperger syndrome, are therefore overrepresented in the neurodiversity movement, and especially in neurodiversity publications. The worry is that the de facto exclusion of low-functioning individuals results in a failure of the movement to represent the full spectrum of diversity amongst autistic persons; thus, when self-advocates claim that autism is not a disability but an identity, that autism ought not be cured, or that autism is a normal human variation, they may be (implicitly or explicitly) speaking on behalf of all autistic persons, or speaking about autism as if it were a unitary, homogenous condition, and thereby marginalizing the experiences of those autistic people who are unable to advocate for themselves due to their communication difficulties.

The problem here is twofold. The demedicalized understanding of autism proposed by neurodiversity advocates carries the risk of marginalizing people who are severely impaired by their condition, as the “disability” label can actually be quite helpful for low-functioning ASD persons. This label may enable them to gain access to disability benefits and other services that they need to carry out the basic functions of life. Conversely, retaining our current social understanding of autism as an illness stigmatizes autistic persons and perpetuates disabling social conditions, making it difficult for even high-functioning individuals to obtain meaningful

employment, for example. This is to say that both the current medical model of ASD and the proposed demedicalized autistic identity position risk making overgeneralizations about autism as a condition, and in so doing, marginalizing the needs and life experiences of those people who don't fit the cast.

To this end, Jaarsma and Welin argue that the neurodiversity claims ought to be accepted as they pertain to “high-functioning” individuals, such as those with Asperger's disorder, but not in the case of “low-functioning” autistic persons. They argue that since, in addition to deficits and disabling consequences, high-functioning autism “can also have desirable and enabling consequences, both to the individual and to society”44 and since “high-functioning autists most often can have rather independent lives in the right kind of environment,”45 we ought to honour the neurodiversity movement with respect to high-functioning autism. But low-functioning autism is seen by Jaarsma and Welin to have more undesirable effects and fewer mitigating factors, with little opportunity for a productive, independent life, and ought therefore to be considered a disability.

This narrow conception of neurodiversity, however, is problematic insofar as it draws lines somewhat arbitrarily between high and low functioning autism, relying on normative judgements of precisely the kind that the neurodiversity movement rejects. Further, although by convention high- and low-functioning autism are distinguished on the basis of whether or not the patient's IQ falls within the “normal” range or whether the patient has language delays,46 this

46 Baron-Cohen, Simon, “Is Asperger Syndrome/High-functioning Autism Necessarily a Disability?” Development and Psychopathology 12, no. 03 (2000): 490. By convention, the “normal” IQ range is two standard deviations from the average. The average IQ is defined to be 100, and the standard deviation is defined to be 15, making the “normal” range between 70 and 130. Approximately 95% of the population falls between these bounds. See Neisser, Ulric, “Rising Scores on Intelligence Tests,” American scientist 85 (1997).
kind of univariate formulation of a complex idea such as “function” has been contested. Persons on the autism “spectrum” often experience a range of deficits (and often some benefits) on different axes of ability including “spoken language, written communication, adaptive skills, different types of intelligence, need for consistency, sensory processing, and so on.”\textsuperscript{47} According to Nicolaidis, we must, therefore,

resist the temptation to categorize people as high- or low-functioning, inasmuch as such categorizations only serve to inadvertently harm our patients. We risk unnecessarily depriving patients categorized as “low-functioning” of their self-determination and opportunities to reach their potential. Similarly, we often deprive our patients categorized as “high-functioning” of necessary supports and services, or we make dangerously false assumptions about their ability to understand what we say or carry out our recommendations. Instead, we must try to understand an individual’s complex combinations of strengths and challenges, as well as the potential for wide variations in functioning.\textsuperscript{48}

This focus on the \textit{function} of autistic persons and the performativity of autism has itself been subject to criticism, as it is seen not as an objective evaluation of a person's “severity” of autism, but rather a value judgement about the person's worth or utility.\textsuperscript{49} “High-functioning” in this sense may be a euphemism meaning “functional enough to work and contribute to society,” in a manner reminiscent of the use of the word “well” to mean “well enough to work.”\textsuperscript{50}

Similar questions persist for the broader conception of neurodiversity favoured by most

\textsuperscript{47} Nicolaidis, “What Can Physicians Learn,” 507.
\textsuperscript{48} Ibid.
advocates. If autism is a simply natural human variation and another, equally valid way of being a person, why ought we worry about depriving “high-functioning” (or, for that matter, “low-functioning”) autistic persons from support and services? Clearly, what Nicolaidis and many other advocates have in mind is an approach to understanding and treating autism that goes beyond the simpleminded ill/well binary. These themes will be readdressed in Chapter 2.

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Despite this controversy, or perhaps because of it, the neurodiversity movement continues to gain supporters among the autistic and nonautistic communities alike. The movement has staked out a territory in the contentious terrain of mental health, opposing the normative judgements thereof and maintaining that autism is not pathological. This claim, however, takes place in a partial epistemological vacuum – one that is increasingly being filled by scientific research currently underway. It is to this research that we now turn.

1.4 Etiologies of autism

Seventy years after autism was first identified, its cause remains elusive. Originally thought to be a nascent form of schizophrenia, autism initially went largely unnoticed by psychiatric researchers and professionals.\textsuperscript{51} It was not until 1980, with the publication of the DSM-III, that autism first gained official recognition. At this point, autism was no longer associated with schizophrenia, but considered a “pervasive developmental disorder” – a class of idiopathic, behaviourally-defined conditions affecting young children. The 1987 revision of the Diagnostic and Statistical Manual, the DSM-III-R, modified the diagnostic criteria for autism and saw the

addition of “pervasive developmental disorder – not otherwise specified” (PDD-NOS) as a new diagnosis for people who met some but not sufficiently many of the criteria for a diagnosis of autism.\textsuperscript{52}

In 1994, the American Psychiatric Association released the DSM-IV, which again saw the expansion of diagnostic categories under the heading of pervasive developmental disabilities, adding Asperger syndrome, Rett syndrome, and childhood disintegrative disorder (CDD).\textsuperscript{53}

These five diagnostic categories, autism, Asperger's, Rett's, CDD, and PDD-NOS, shared many similarities in their symptomatology, and were thought to represent different points on an “autism spectrum,” along which the “sufferer” could vary significantly in the severity of their deficits and their ability to function.\textsuperscript{54} Thus it was decided by the APA to remove these differential diagnoses from the fifth edition of the DSM, released in 2013, in favour of the single heading, “autism spectrum disorder.”\textsuperscript{55} Throughout these years of classificatory change, the APA has remained silent on the topic of the etiology of ASD, and rightly so. Despite decades of

\textsuperscript{52} Ibid.

\textsuperscript{53} Ibid. Rett syndrome is a disorder primarily affecting girls characterized by a period of normal development for 6 to 18 months, followed by a loss of purposeful motor control, epilepsy, characteristically autistic behaviours and other symptoms. CDD follows a course similar to Rett's, with a period of normal development for up to 3 or 4 years of age followed by a rapid loss of linguistic capability and cognitive function, often accompanied by autistic behaviours. CDD remains idiopathic, and is quite rare compared to other autism spectrum disorders. Asperger syndrome, likely the most well-known of these new additions, is often referred to as a form of “high-functioning autism.” Asperger syndrome is a condition with deficits in social reciprocity and repetitive behaviours similar to those of autism, but without accompanying intellectual impairment or language delay.

\textsuperscript{54} Although some of the problems with the idea of autistic “function” were discussed in the previous Section, this language is still in common currency, and the idea of autistic “function” is still regularly used as a metric for deciding whether an autistic person deserves disability services or should be removed from the mainstream classroom to “special education,” for example. As such, the use of this term is nearly impossible to avoid; this should not, however, be taken as an endorsement on my part of the idea of autistic “function” as a useful way of thinking about autism. Similarly, the language of “deficits,” “mental illness,” and other such value-laden terminology is pervasive in the way autism is discussed in the scientific literature – hopefully I might be forgiven the use of this language in the following discussion of said literature.

\textsuperscript{55} This decision has been highly controversial. The removal of Asperger disorder has proven particularly contentious, as many people who have been diagnosed or self-diagnosed with Asperger disorder may feel that they are having their identities stripped from them. See Singh, Jennifer S., “The Vanishing Diagnosis of Asperger's Disorder,” \textit{Advances in Medical Sociology} 12 (2011).
research, autism spectrum disorders have stubbornly resisted “reductionistic” attempts to construct their etiology in terms of “the gene for autism,” mercury poisoning from child immunizations, overly aloof “refrigerator mothers,” or other univariate explanations that have held sway at different times and places.\textsuperscript{56}

In this Section, I will summarize some of the major areas of etiological research that scientists are currently undertaking with the goal of understanding autism. While I cannot hope to do justice to the depth and diversity of research presently underway, my intent with this Section is to communicate the flavour and dominant themes of current autism research, as well as to convey the heterogeneity and lack of a unified understanding of the condition. In the following pages I will discuss the dominant threads in autism etiology research, including neurophysiology, genetics, environmental pollutants, vaccinations, viral infections, immunology, and parenting practices. Volumes have been written about these subjects, of course, so I do not aim for comprehensiveness, but my concern is perhaps less with what the “actual” etiology or etiologies of autism are, so much as what is claimed to be the etiology of autism, especially when those claims bear the weight of scientific authority, real or perceived. Perhaps what is most important for how we understand autism is construction of a simple and coherent etiological narrative, the complex reality notwithstanding.

\subsection{1.4.1 Neurological underpinnings}

Research into the cause of autistic symptomatology has primarily been focused on the search for the “autistic brain” – that is, the search for abnormal developmental signatures in the brain that differentiate autistic from nonautistic persons. These studies, making use of imaging techniques

\textsuperscript{56} See Nadesan, “Constructing Autism,” especially Chs. 5 and 6.
such as MRI, PET, and CT scans, or in some cases autopsy of autistic persons' brains, have pointed to a variety of neurophysiological mechanisms that may be at play in the expression of autism. To start at a gross anatomical level, autistic persons typically have brains which are 5% to 10% larger than those of the general population. A study by Courchesne et al. reported that of those studied, 90% of two- to four-year old children with autism had a brain volume above the normal healthy average, and 37% had brain volumes at least two standard deviations above normal, thus meeting the criteria for developmental macroencephaly.57 This overdevelopment of the brain is believed to begin in the first months of the child's life, as head circumference measures of newborns show no difference between neurotypicals and those who are later diagnosed as autistic.

This period of overdevelopment results in abnormally high volumes of white matter in cerebral and cerebellar structures, and abnormally high volumes of grey matter in the cerebrum, especially the frontal lobes.58 The rapid development of these brain structures appears to cease prematurely within the first 2 to 4 years of life, often leaving older autistic children with smaller than normal brain volumes.59 Periods of overdevelopment such as these can lead to localized neuronal hyperconnectivity, which fact may explain some of the behavioural, cognitive, and affective disturbances observed in autistic persons, especially sensorimotor overstimulation.60

Other studies have focused on the amygdala, a part of the limbic system, as the locus of abnormal development implicated in the development of autism. The amygdala is a complex and

still poorly understood structure which is involved in numerous different tasks, including memory consolidation, danger response, fear, anxiety, and other emotions. Its role in autism was hypothesized by Baron-Cohen et al. due to the involvement of the amygdala in social interaction and emotional intelligence.\textsuperscript{61} The amygdala is also implicated in obsessive-compulsive behaviours commonly observed in autistic persons. Most research has found abnormally large or overdeveloped amygdalae in autistic children,\textsuperscript{62} however a recent study by Inui has pointed toward damage or underdevelopment of the fetal amygdala as a potential cause of autism.\textsuperscript{63}

Other potential neurological culprits include the poor regulation of neurotransmitters, including dopamine, oxytocin, and especially serotonin, which has repeatedly been found at elevated levels in sample autistic populations.\textsuperscript{64} Some research has also pointed to disturbances in the ability of neurons to migrate to the cerebral cortex during pregnancy.\textsuperscript{65} Many of the purported causes of autism are also implicated in other mental illnesses, perhaps explaining the high comorbidity of autism with other conditions. The neurological overdetermination of autism, its overlapping etiologies and high comorbidity with other mental illnesses has cast some doubt on whether our psychiatric nosologies truly represent something about the way the brain works, or whether they are simply convenient classificatory fictions.\textsuperscript{66}

\begin{thebibliography}{9}
\bibitem{65} See Schmitz, Christoph, and Payam Rezaie, “The Neuropathology of Autism: Where Do We Stand?” \textit{Neuropathology and Applied Neurobiology} 34, no. 1 (2008) for a review of these topics.
\bibitem{66} See Nadesan, “Constructing Autism,” especially Ch. 6.
\end{thebibliography}
1.4.2 Genetics

Autism is one of the most heritable of mental illnesses. Studies on monozygotic twins have demonstrated concordance rates between 70% and 90%, and while twin studies cannot rule out environmental factors entirely – indeed, environmental influences are needed to explain why monozygotic twins are not always either both autistic or both neurotypical – numerous studies have consistently shown heritability to be approximately 80%. There are three general types of genetic variations that can give rise to autism: single-gene disorders, broad phenotypic variation stemming from multiple genes, and de novo genetic mutations.

Single-gene or “Mendelian” disorders that can cause autism include Fragile X syndrome and Rett syndrome. Fragile X syndrome is caused by a mutation of the FMR1 gene on the X chromosome and may result in many different types of intellectual impairments, including ADHD, ASD, or OCD, as well as other physical and psychological effects. As it is a disorder of the X chromosome, boys are preferentially affected at a rate of 1 in 3600, compared with approximately 1 in 6000 in girls. Similarly, Rett syndrome is a disorder caused by mutations to the MECP2 gene on the X chromosome affecting synthesis of the MECP2 protein, which causes autism-like symptomatology. Rett syndrome, however, almost exclusively affects girls – male fetuses without a functioning MECP2 gene typically miscarry before reaching full term, whereas females with the disorder typically retain a functioning copy of the MECP2 gene, providing them with sufficient protein synthesis to survive, but not to experience normal neurological

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67 Monozygotic concordance refers to the probability that, given one autistic twin, the other twin will also be autistic in a set of identical twins. See Eapen, Valsamma, “Genetic Basis of Autism: Is There a Way Forward?” Current Opinion in Psychiatry 24, no. 3 (2011).

68 Ibid. “Heritability” describes the proportion of phenotypic variation attributable to genetic, rather than environmental, variations.

Between 10-15% of cases of autism are thought to stem from single-gene conditions such as these. Beyond Mendelian single-gene disorders, most cases of autism are thought to involve a variety of genes, with “each of these variations being common and distributed continually in the general population, but resulting in varying clinical phenotypes when it reaches a certain threshold through complex gene–gene and gene–environment interactions.” In these cases, no “genetic disorder” is present, but it is thought that the confluence of many factors, which in isolation would create modest or no effects, produces clinically significant symptomatology when present in a single individual. The number of distinct genes that can contribute to autistic symptoms in children is believed to be immense – a recent study claimed as many as 400 different loci may be implicated. These individual “predisposing” genes are rarely claimed to explain more than a few percent each of the variability in symptomatology between subjects and controls, and despite the innumerable studies claiming to find associations between particular genes and ASD, the replicability of such studies has generally been poor, owing the exceeding rarity of these genes, or perhaps publication bias.

The third type of genetic influence is so-called de novo genetic mutations. De novo mutations are those that are possessed by the child but are present in neither parent, and therefore exist as the result of new mutations in the germ cells of one of the parents. These mutations, usually inherited from the father, often result in severe and specific deficits in the affected child.

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72 Eapen, “Genetic Basis.”
Although autism resulting from de novo mutations constitutes only about 15% of cases, the probability of these mutations and the corresponding risk of autism increases with the father's age at the time of conception. The heterogeneity of genetic influences implicated in the development of autism-like symptoms, as well as the presence of many “autistic genes” among nonautistic populations suggests that the role genetics play is one of predisposing children toward developing autism. This idea of “genetic susceptibility” to autism suggests that genetic factors are responsible for some children having, for example, a poor ability to process heavy metals, or a malfunctioning immune system which “overreacts” to viral infections early in life – these environmental factors are then believed to push the child “over the edge,” as it were.

### 1.4.3 Heavy metals and other environmental pollutants

As it is thought that genetics alone is unable to explain the observed increase in the prevalence of autism and other neurodevelopmental disorders, researchers have begun to look at environmental factors, including heavy metals and other common industrial chemicals in the environment. In two recent review articles, Grandjean and Landrigan identified 11 industrial chemicals found in the environment that have been implicated in the development of autism, ADHD, and other conditions. These chemicals include lead, methylmercury, polychlorinated biphenyls, arsenic, toluene, manganese, fluoride, chlorpyrifos, dichlorodiphenyl-trichloroethane, tetrachloro-
ethylene, and polybrominated diphenyl ethers. Many of these chemicals are transmitted through breastmilk to the infant, whence they are able to cross the blood-brain barrier. Other of these chemicals are present in the bloodstream of the mother and may pass unimpeded through the placenta.

Exposure to such contaminants in critical developmental periods affects the function and development of the brain in various unsavoury ways. Prenatal exposure to methylmercury has been shown through use of fMRI to be associated later in life with unusually high levels of activation in certain cortical regions in response to sensorimotor tasks, congruent with the “overstimulation” reported by many autistics in response to everyday sensorimotor stimuli. Fetal exposure to phthalates, a class of chemical compounds used in many consumer products such as cosmetics and children's toys, as well as being commonly used in packaging, has also been shown to be associated with autism. Phthalates are absorbed into the body through skin contact and consumption, and travel freely through the placenta to reach the fetus where they act as endocrine disruptors. These chemicals impede the thyroid's ability to distribute hormones throughout the body by “competing” for receptor sites on the transport proteins.

Other environmental contaminants which may play a role in the increase in autism prevalence include commonly-used organophosphate pesticides; perfluorooctanoic acid, a chemical used in the manufacture of many products such as Teflon and Gore-Tex, and various

81 De Cock, Marijke, Yolanda G. H. Maas, and Margot van de Bor, “Does Perinatal Exposure to Endocrine Disruptors Induce Autism Spectrum and Attention Deficit Hyperactivity Disorders? Review,” Acta Paediatrica
types of air pollution including particulate matter from automobile traffic. The number of environmental pollutants known or suspected to play a role in the development of ASD grows seemingly without bound. On this point, Grandjean and Landrigan emphasize that

The number of chemicals that can cause neurotoxicity in laboratory studies probably exceeds 1000, which is far more than the estimated 200 that have caused documented human neurotoxicity. However, in the absence of systematic testing, the true extent of the neurotoxic potential of industrial chemicals is unknown. The physiology of brain development and experimental evidence suggest that developmental neurotoxicity is likely for all of them, except perhaps for some of the compounds that require metabolic transformation to become neurotoxic, in which immature metabolism may provide some degree of protection. The few substances proven to be toxic to human neurodevelopment should therefore be viewed as the tip of a very large iceberg.

While they are speaking here of neurodevelopmental disabilities generally, some of the chemical pollutants alluded to by these authors have already been claimed to contribute to the rise in autism prevalence, and more will almost certainly be added to the list as the science develops.

1.4.4 Vaccines

One industrial chemical that has received particular scientific and media attention is thiomersal, an organomercury compound commonly used as a preservative in vaccines. The popular notion that vaccines increase the risk of childhood autism originated in a 1998 paper published in *The Lancet* titled “Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive

101, no. 8 (2012).


developmental disorder in children” which argued for, among other things, a possible link between the measles, mumps, and rubella (MMR) vaccine and the onset of neurodevelopmental disorders such as autism. Over the next several years, through the ensuing media coverage, the idea that vaccines increase the risk of autism took hold in the public imagination despite mounting scientific evidence to the contrary. Eventually Andrew Wakefield, the principal investigator of the original study, was revealed to have falsified data and to have had financial conflicts of interest, leading to the official 2010 retraction of the paper.

In response to public outcry, thiomersal, believed to be the cause of this purported connection, was removed from vaccines in North America and Europe by 2000, an action that is often interpreted by anti-vaccine organizations as an admission of guilt on the part of vaccine manufacturers. This act did not placate the troubled parents, however, and blame quickly shifted to other organomercury adjuvants used in vaccines, or to the administration of particular pathogens or combinations of pathogens. For example, administering the particular combination of pathogens in an MMR vaccine at a critical period in the child's development is believed by some anti-vaccine activists to precipitate the development of autism by “overwhelming” the child's fragile immune system in those predisposed.

There is no scientific evidence to support these claims. Hundreds of individual trials, reviews and meta-analyses carried out by scientist in different fields have consistently found no association between childhood vaccinations and increased prevalence of autism. Nevertheless,

the influence and visibility of anti-vaccine organizations such as Generation Rescue and prominent anti-vaccine spokespersons such as Jenny McCarthy in the popular press have had a significant influence on public perceptions of ASD, and thus it bore mention.

1.4.5 Viral infections and immunology

The antivaccine movement, though thoroughly discredited, draws some apparent support from the legitimate scientific research being carried out in the field of immunology. Maternal viral infections during gestation or infections during the first few years of life of the infant have been postulated as potential catalysts for autism and other mental illnesses, though the “mechanism, by which viral infection may lead to autism, be it through direct infection of the central nervous system (CNS), through infection elsewhere in the body acting as a trigger for disease in the CNS, through alteration of the immune response of the mother or offspring, or through a combination of these, is not yet known.”

Although this is a relatively new area of research in autism etiology, several recent studies in animal models have established a link between maternal immune system activation during pregnancy and the development of autism-like symptoms in offspring. Elmer et al. performed a study on rats showing a relationship between MCHI protein concentration and maternal viral infection. MCHI (major histocompatibility complex class I) proteins play a

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crucial role in the development of the central nervous system, including negatively regulating the
development of synapses between cortical neurons. These proteins were found in much higher
amounts in the cortical neurons of offspring whose mothers’ immune systems had been activated
by viral infection during gestation, causing changes in synaptic density in the cerebral cortices of
the rats. As synaptic density in the cerebral cortex is one of the potential indicators of autism, a
similar abundance of MCHI protein in human children could catalyze the development of autism.

However, a cohort study of 7379 participants carried out in Denmark investigated the
relationship between autism and early childhood infections. While the study found a higher
incidence of ASD diagnoses among children who were admitted to the hospital at some point for
bacterial or viral infections, the results were not significantly different by type of infection – that
is, children hospitalized with measles were not statistically more likely to be diagnosed with
ASD than children hospitalized with upper respiratory tract infections, for example. For this
reason, the authors of the study conclude that the relationship between hospitalization for
infectious diseases and ASD diagnoses is not a causal one. As this line of research is fairly
young, few definitive conclusions have been reached.

1.4.6 Parenting practices and behavioural interventions

The final area of research discussed here deals with parenting practices and the social
environment of the child, suggesting these factors may play a role in autistic symptomatology. In
retrospect this perhaps seems obvious; if behavioural interventions have any effect on the
severity of autistic deficits, then social-environmental practices affect children in ways more or

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90 Atladóttir, Hjórdís Ósk, et al., “Association of Hospitalization for Infection in Childhood with Diagnosis of
Autism Spectrum Disorders: A Danish Cohort Study,” Archives of Pediatrics & Adolescent Medicine 164, no. 5
(2010).
less conducive to the expression of autism. This area of research has been slow to develop, due to the understandable stigma attached to such explanations. This dates as far back as Leo Kanner's 1943 paper where he postulates that cold and aloof mothers are to blame for their children's autism, coining the term “refrigerator mother.”91 While the “refrigerator mother,” with its misogynistic overtones, is now universally dismissed by the scientific community as a root cause of autism,92 a recent study by Smith et al. has shown a relationship between maternal warmth and praise and the amelioration of certain symptoms among autistic adults, including increased social reciprocity and decreased repetitive behaviours.93

This is echoed by other researchers who have found that parental limit-setting behaviour is associated with fewer behavioural problems from their autistic children,94 and that parental mindfulness is associated with decreased aggression and self-harm,95 for example. The research suggests, however, that the role of social factors such as parental warmth is one of mitigation, rather than prevention. Autism is not caused by a particular parenting style, however overbearing or neglectful. Rather, it is thought that particular parenting styles and behavioural interventions may be adopted with the knowledge that the child is already autistic, in an attempt to achieve the “best” outcome for the child, where “best” is typically understood to mean that the child scores higher on measures of social reciprocity, verbal behaviour, and intelligence, or displays fewer characteristically autistic behaviours such as hand flapping or self-harm.96

91 Kanner, Leo, “Autistic Disturbances of Affective Contact,” *Nervous Child* 2, no. 3 (1943).
96 Numerous studies have been conducted demonstrating the effectiveness of diverse therapies such as applied
This list is not exhaustive. In a recent review article on the relationship between genetic and environmental factors in autism, Meek et al. noted

As is evident through a brief review of autism genetic research, there are likely multiple genetic influences and distinct biological pathways involved in the development of autism. While the field faces many challenges, including genetic and phenotypic heterogeneity, the dearth of research incorporating both genetic and social environmental measures may be among the most limiting factors to the field’s progress.97

The picture of autism painted by scientists is an interactional one – in most cases of autism in which a single-gene disorder like Fragile X or Rett syndrome is not present, it is thought that genetic predisposition combined with a variety of different environmental “insults” all contribute to precipitating the development of autism in the child. This is an especially intuitive hypothesis in cases where children show a period of normal development followed by rapid deterioration of motor, cognitive, and linguistic abilities, as in the case of CDD.

Indeed, it is likely that the category we now call “autism spectrum disorders” does not represent a common underlying pathology, but rather that there are many distinct etiologies that find common expression in the constellation of symptoms we now classify as autistic, potentially

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97 Meek, “A Review.”
explaining why there is such a heterogeneity among so-called autistic persons in the expression of their symptoms, as well as why there is such a wide spectrum of “function.” Clearly, the field is still far from constructing a coherent narrative describing the cause of autism, and much more research will be required to do this, especially focusing on the interactions between genetics, environment, and upbringing. But even these inchoate etiologies suggest new ways to understand autism, as well as new “treatments,” “therapies,” or other medical interventions aimed at preventing new cases of autism or ameliorating symptoms in those already diagnosed. These new ways of understanding autism, and how they conflict, concur, and interact with the claims of the neurodiversity movement will be the subject of the following Chapter.
2. (De)medicalizing autism

Prima facie, this research into the cause of autism need not be any more committed to pathologizing autism than research into the cause of blue eyes need be committed to pathologizing eye colour. Searching for the genetic, neurological, and environmental influences that affect the course of autism development in children is no doubt a legitimate area of scientific inquiry, in this sense. But overwhelmingly researchers cast their investigations in the language of health and illness, systematically constructing autism as a mental health problem to be addressed through medical interventions.

In a recent review article on the relationship between vaccines and autism, for example, Tomljenovic and Shaw begin with the claim that “Autism spectrum disorders (ASD) are serious multisystem developmental disorders and an urgent global public health concern,”98 while Sebat et al. have described autism as a “major burden to society.”99 Grandjean and Landrigan, whose work was discussed above, devote a section of their review article to the economic costs associated with autism and other neurodevelopmental disorders, claiming that ASD is responsible for the loss of some 7 million IQ points in the United States alone, corresponding to a loss of some $126 trillion in potential lifetime earnings in 2008 dollars.100 Their concern, it seems, is motivated by the potential loss of economic productivity, insisting on an economically instrumental valuation of persons. They are motivated to propose prevention measures for conditions such as ASD not because they cause suffering to individuals, but because they hinder the growth of the economy.

98 Tomljenovic and Shaw, “Do Aluminum Vaccine Adjuvants Contribute.”
99 Sebat et al., “Strong Association.”
Research conducted on these terms constructs and is constructed by a pathological understanding of autism – indeed, one that goes beyond treating autism as an individual affliction, but as a scourge on society as a whole. The use of rhetorical strategies such as these exposes these researchers as committed to pathologizing autism, but at least it has the benefit of being overt – one can hardly call autism a “burden to society” or “public health concern” and deny their belief in its status as disease. But the research has other implications, perhaps more insidious, that also construct autism as pathological – and in so doing raise important questions about suffering and disease. In this chapter, I will discuss the ways, both direct and indirect, in which this etiological research contributes to a pathological construction of autism. I will then address the role of normality in the neurodiversity program, and the complex relationship this concept has with what we might call the nature of disease. I will argue that the insistence on normality ultimately commits the neurodiversity movement to a problematic model of disease, and that they might be better served by remaining agnostic on the questions of normality and etiology. I close with some final thoughts on identity and authority.

2.1 Genetics and reproductive technology construct autism as a disease

Etiologies suggest treatments. The state of our knowledge of the heterogeneous genetic factors that dispose children to developing autism motivated Joseph Buxbaum of the Mt. Sinai School of Medicine to state in 2005 that a prenatal test for autism would be available within 10 years.101 This statement was widely criticized by the neurodiversity movement for its perceived eugenic overtones, and lead to the creation of an “Autism Genocide Clock” posted on the blog of a

neurodiversity advocate that counted down the days until the proposed test was supposed to become available.\textsuperscript{102} Predictions of this kind are unreliable at best, of course, and the genetics of autism has proven much more complex than Buxbaum initially supposed. Nevertheless, Campbell made a similar suggestion in 2010, claiming that we have already sufficient knowledge of the genetic determinants of autism to warrant genetic testing for Mendelian disorders such as Rett syndrome, and we should be able to produce a clinically useful genetic test for other forms of autism in the near future.\textsuperscript{103} Indeed, a nascent form of preimplantation genetic selection is already underway in the state of Western Australia, where sex selection techniques for in vitro fertilization patients have been approved as a method of reducing the likelihood of having a child with autism for parents thought to be at high risk. As boys are four times more likely than girls to be autistic, the Reproductive Technology Council of Western Australia approved the use of sex selection for female children as an effective method of reducing the risk of having an autistic child.\textsuperscript{104} While the method is crude – a bit like trying to avoid seeing one's ex-wife at a cocktail party by moving to a different country – it nevertheless clears the path for future, more closely targeted genetic tests.

The existence of a test of this kind puts expecting parents in the position of deciding on the genetic future of their children, a thought that no doubt makes many people uneasy, as it seems to foster an attitude of control and discourage the kind of “openness to the unbidden”

\textsuperscript{102} The clock has since been removed. See Ventura33, “Autism Research and Prenatal Testing,” \textit{The Ventura33 Fanfiction Universe} (blog), n.d., http://www.ventura33.com/clock/.
some see as necessary to love and appreciate a child. But the promise of genetic engineering to allow us to prevent debilitating genetic disorders and freely select benign character traits for our children is a tempting one. On this point, Savulescu and Kahane argue that as a corollary to our duty to provide our children with the best lives we can, parents have a moral obligation, when possible, “to select the child, of the possible children they could have, whose life can be expected to go best.” According to this “Principle of Procreative Beneficence,” as they call it, parents have a duty to use any genetic selection techniques available to them in an effort to give their future children the “best possible future.” While it is not at all clear what they have in mind by the “best possible future” or what it means for someone's life to “go best” – the authors gesture vaguely toward “well-being” and “advantages” – they make it clear that those conditions we typically call developmental disabilities, such as Down syndrome or autism, are excluded.

Jaarsma and Welin disagree with this assessment. They argue that not only do we not have a duty to select against an autistic fetus, but that parents who so desire ought to be allowed to select for a mildly autistic child. This is a qualified claim however – as discussed above, they support a narrow conception of neurodiversity, in which they honour the identity claims of high-functioning autistics only, not low-functioning ones. But although Jaarsma and Welin seem to agree with at least a restricted version of the neurodiversity claim, this agreement is based on the attributes thought to accompany high-functioning autism. A high-functioning autistic may have social deficits, flattened affect, and repetitive behaviours, for example, but these “hypo-

empathizing” behaviours may be compensated for by a “hyper-systematizing” cognitive style that gives high-functioning autistic persons exceptional abilities in scientific, mathematical, and creative domains.

Savulescu and Kahane's assessment differs in its prescriptions from Jaarsma and Welin's, but ultimately both are based on an instrumental valuation of persons according to the normative standards of independence, employability, and so forth – in other words, “to function.” Savulescu and Kahane appear to recognize this fact, noting that often the worst consequences of disabilities are due to prevailing social conditions, yet they would prefer to see such difficulties addressed through the medium of preventative genetic medicine rather than social change. Given this commitment, as well as the ambiguity in what it might mean for someone's life to “go best,” we could understandably infer from their principle that we have a “significant moral obligation” to select for male over female children in a patriarchal culture, straight over gay children in a homophobic culture, light-skinned over dark-skinned children in a racist culture, and so forth, given the myriad privileges associated with each of these characteristics. In this sense, Savulescu and Kahane's commitment to “procreative beneficence” is also a tacit commitment to reinforcing social ills such as racism and sexism, not to mention discrimination against the disabled or the neurodiverse.

No doubt this is not what Savulescu and Kahane have in mind, and they probably would not advocate, if asked, selecting against black or gay or female babies, feeling that these are problems to be addressed through social change and not genetic change, but in so doing they are dividing the arena into health problems and social problems, drawing (what they feel to be) a clear line between them.\footnote{Savulescu and Kahane cannot argue that female children ought not be selected against because gender is not a}
be considered health problems rather than social problems (although there might be some social problems associated with then). But by classifying autism under the rubric of health and not social problems, they have precisely begged the question – they have decided the issue in advance against neurodiversity.

Whatever their utility, these ethical musings bring into focus the implicit valuations that technologies of this type inherently carry. A useful comparison would perhaps be the prenatal (postimplantation) genetic test by amniocentesis commonly used to test for Down syndrome, the use of which places parents at an increased risk of miscarriage. To parents who intend to keep and care for the child no matter its condition, such a test makes no sense – there is little merit in taking the risk unless it were assumed that the parents intended to abort the fetus should the test come back positive. These tests only make sense in a context that allows for selection (in this case, elective abortion); thus to take the test is to accept the value implicit in those selective procedures. But even the very existence of a genetic test for a condition that might be considered “undesirable” forces upon prospective parents a choice of whether or not to use it, with the knowledge that even refusing the test constitutes a tacit judgement on the worth of children with said condition.

Preimplantation genetic tests would allow for selection without increased risk of miscarriage or the need for elective abortion, but they do not necessarily avoid making implicit normative judgements. The sex-selection technique approved in Western Australia exemplifies this as, contra Jaarsma and Welin, the technique has been approved to select against autistic

disability, as they are very careful not to draw any moral distinctions that line up with distinctions of disability or illness to avoid claims of discriminating or eugenics. Nevertheless, they admit that selecting a smarter fetus, for example, is a moral obligation, even if the alternative is a child of average intelligence. Why not, in that case, select for a more privileged gender, sexuality, or race? See Savulescu and Kahane, “Moral Obligation,” 284-9.
children only, leading some neurodiversity advocates to accuse the state of practicing eugenics. Here the value judgement is explicit – autism is regarded as an undesirable disability, and the technology is made available to those parents who wish to avoid the tragic fate. But suppose we indulged the fantasy of Jaarsma and Welin, and allowed selection for mildly autistic children. Since ASD typically trades in predispositions rather than “genes for autism,” might we feel like selecting for children with Asperger syndrome would put them at risk for more severe forms of ASD? Or, perhaps we should go further and allow selection for any and all forms of ASD, however severe. Would doing so be honouring the neurodiversity programme? Or would the very existence of a genetic selection procedure somehow challenge the autistic claim to identity and normality?

We might be inclined to wonder why there is such intense interest in the possibility of a prenatal genetic test for autism in the first place. That such time, energy, and money is spent in the search for the genetic determinants of autism suggests that there is significant interest in making such a test available for use. It is not difficult to imagine that most parents, given the choice, would opt for a neurotypical child, given the well-known difficulties associated with raising autistic children. The presence of such a test, then, could serve not only to reinforce a


111 That parents might wish to select for a child with Asperger syndrome or even “low-functioning” autism is not so far-fetched. By way of comparison, members of the Deaf community have already used preimplantation genetic tests in efforts to increase their chances of having deaf children. These Deaf persons have often expressed sentiments similar to those of the neurodiversity movement, including the belief that deafness is an identity, rather than a disability, yet their actions have proven controversial. Deaf community members claim that since deafness is an identity like any other they have done nothing wrong in choosing an identity for their children, while others among the bioethics community and general public have expressed outrage that parents would deliberately attempt to bring a disabled child into the world. See Sandel, The Case Against Perfection, 1-5.

112 Many of the most vehement opponents of the neurodiversity movement are parents of autistic children themselves, as they are all too familiar with the stresses and challenges concomitant to raising an autistic child. See, for example, Rao, Patricia A., and Deborah C. Beidel, “The Impact of Children with High-Functioning Autism on Parental Stress, Sibling Adjustment, and Family Functioning,” Behavior Modification 33, no. 4 (2009), and Davis, Naomi Ornstein, and Alice S. Carter, “Parenting Stress in Mothers and Fathers of Toddlers
pathological understanding of ASD, but also as a way of moving the discussion away from the rights and acceptance claims of the neurodiversity movement (Claim 4) and into the realm of preventative medicine instead. Providing the opportunity for prospective parents to genetically select against autism or other “disabilities” devalues the lives of persons living with those conditions selected against, treating them as medical problems to be addressed through the framework of preventative genetic medicine, and ignoring the social and institutional barriers that create the disabilities experienced by autistics. Under this framework, the “disability itself” is viewed as the problem, not the failure of social justice. The existence of such a genetic test, or even its proposal, is not value-neutral – rather, it serves to reinforce the battle lines drawn between the neurodiversity movement and the popular and scientific conceptions of autism.

2.2 Environmental pollutants construct autism as a disease

The discovery that industrial pollutants, heavy metals, and other environmental toxins are implicated in the development of autism raises similar questions and normative challenges. Would we feel more or less disposed to regard autism as an identity if it were caused by environmental “insults”? Further, given that exposure to toluene and methylmercury may precipitate the development of autism in genetically predisposed children, for example, ought we attempt to eliminate those chemicals from the environment? Grandjean and Landrigan think so. Claiming that the increase in neurodevelopmental disorders represents a “pandemic,” and using the language of disease and disability, they propose a strategy based on the Precautionary


Principle aimed at the elimination of neurotoxic industrial chemicals causing developmental disabilities such as autism and ADHD.\textsuperscript{114}

Their view is certainly not unprecedented. As mentioned above, thiomersal was removed from vaccines in 2000 due to public outcry, despite virtual scientific consensus that the chemical posed no risk for the development of autism or other neurological conditions. Even after the removal of thiomersal, some anti-vaccine activists still contend that childhood vaccines are implicated in autism, opting to prevent their children from being vaccinated over taking the purportedly increased risk of ASD. Here we have a clear indication of their commitments – not only do we have a duty to remove potentially toxic chemicals from the environment of children who may be at risk of developing autism due to exposure, but further, autism is such an undesirable condition that it is worth placing one's own children and the children of others at risk for serious infectious disease to avoid it. Here, the identification of a causally efficacious agent – even one based on scanty evidence and conjecture – in the development of autism stabilizes and reinforces the belief that autism is pathological.

The presence of thiomersal and other mercury compounds in vaccines and their dubious relationship with autism has, understandably, been the subject of much controversy, as well as the subject of a number of best-selling books in which thiomersal has been compared with the thalidomide scandal of the mid-20\textsuperscript{th} century.\textsuperscript{115} There are parallels between the two cases, naturally. In both, we see the introduction of a foreign substance into the body of the affected person or the mother during pregnancy resulting ultimately in (alleged) physiological or

\textsuperscript{114} Grandjean and Landrigan, “Neurobehavioural Effects.”
\textsuperscript{115} Thalidomide, of course, is a drug that was marketed to pregnant mothers in the 1950s and 1960s to help reduce morning sickness, but was quickly pulled from circulation when it was shown to cause marked birth defects in the children of mothers who had used the drug. See Dachel, Anne, “Thalidomide / Thimerosal,” \textit{Age of Autism} (blog), September 5, 2012 (5:46 a.m.), http://www.ageofautism.com/2012/09/thalidomidethimerosal.html.
psychological changes in the child. But we also have a similar dynamic in play in the case of, for example, vaccinations (disregarding the thiomersal connection) – an administered external chemical agent affects the physiology of the child, in this case triggering their immune system to produce antibodies for specific communicable diseases. In order for the comparison between thalidomide and thiomersal (or the comparison between gross anatomical birth defects and autism) to have the desired rhetorical force, the authors must assume that the effects of thalidomide were pathological and, in so doing, they are able to argue, or perhaps simply assume, that autism is pathological as well. And the comparison seems to make sense, at least superficially – but we're willing to accept this argument so readily only because most of us already believe that thalidomide-induced birth defects (and autism) are pathological.

There is a distinctly essentialist flavour to this kind of argument. There seems to be a kind of essential person lurking under the surface here, and conditions such as autism or birth defects are seen to get in the way of who that person was meant to be, in some sense. Perhaps this could be cast in terms of genetic endowment; we might think that thalidomide-induced birth defects or chemically- or virally-induced autism disturb the essential person, imposing a detrimental trait upon someone's otherwise preordained genetic destiny. But surely it can't be as simple as this, if the idea of a “genetic disease” such as Huntington's is to make any sense. Nor can such a naive focus on the genetic account for the critically important role of the environment in not only creating diseases and impediments, but also those people we call “healthy.” This kind of ghostly essential person is closely related to the concept of normality, and it also makes an appearance in the arguments of the neurodiversity movement, in particular their insistence on autism as normal and as an integral part of identity. This point will be discussed at greater length in the following
If we assume that some of the aforementioned chemicals are truly implicated in the development of autism spectrum disorders, what, then, is our duty? Do we follow the lead of the anti-vaccine activists in one arena, and many scientists and activists in another, to recommend that these chemicals be banned immediately to safeguard our children's health? Answering this question would seem to also stake out a claim on the question of autism as a pathology. If autism were truly just a neurological difference, to be valued no greater or less than any other human differences, it is difficult to see why should it matter whether the condition is caused by anthropogenic chemicals, genetics or viral infections. That so many people in both the scientific and lay communities seem to feel that there is in fact a difference between chemically-induced autism and genetic autism, however, reveals something about the way we think about disease and the important role etiology has to play in distinguishing health from illness and normality from abnormality.

2.3 Neurodiversity and “normality”

Bearing the weight of scientific authority, these kinds of etiological claims and their associated normative flavours have an uneasy relationship with the neurodiversity movement, not least because the claims are often in direct contradiction with one another. As discussed in Section 1.2, the first claim of the neurodiversity movement is that ASD is a natural human variation, often couched in the language of genetics. A claim of this type predicates the validity of autism as an identity on a shaky foundation of speculative causation – a foundation that is vulnerable to collapse as the science moves toward constructing a coherent etiology that does not square neatly
with these preconceptions. And while scientists point to sole genetic causes in the case of syndromes such as Rett's, the most general picture of autism painted by scientists is more complex, resulting from an interplay of genetic predisposition and myriad environmental factors.

Many neurodiversity advocates avoid mentioning ultimate causes such as genetics or pollutants, but maintain that autism represents a variation in “brain wiring.” This turn of phrase is delightfully vague, but there is no doubt that it is true under an appropriate interpretation. To the extent that the ontology or the functional organization of a mind is responsible for the differences in behaviour, affect, and cognition experienced by autistic persons, and to the extent that these ontological or organizational differences derive from differences in a person's brain, we should have no prima facie objection to this kind of description. Yet, as Nadesan points out, this kind of “reductionistic” focus on neurophysiology is misleading, and can serve to move attention away from the social forces involved in the production of autism, broadly construed. In this sense, etiological investigation into the interplay of these kinds of social forces may be seen to undermine the authority of neurodiversity advocates who have completely “neurologized” autism.

These observations point toward an apparent inconsistency in the neurodiversity programme. That is, a commitment to the genetic, neurologic, or any particular origin of autism as a premise for the acceptance of the neurodiversity claims not only puts one at risk of losing

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116 I must resist the urge to digress at length on the philosophical topics that ultimately bear only very minimally on the topic at hand, and also resist the temptation to uncritically adopt a potentially controversial position such as mind-brain identity simply because it is currently fashionable among philosophers of mind and neuroscientists. I wish to remain silent on these topics, but I take it as uncontrovertial that there is some kind of causal relationship between brain and mind, and that we have no prima facie reason to believe that this kind of relationship would be necessarily incapable of producing the kinds of cognitive, affective, and behavioural differences characteristic of autism.

117 “Social forces” could include not just such obvious things as parenting practices and socioeconomic factors, but also more broadly include diet and environmental pollutants, for example, insofar as these factors are thoroughly embedded in social practices. See Nadesan, “Constructing Autism,” especially Ch. 6.
legitimacy as the science develops, but also commits oneself to a problematic model of pathology. This line of argument implies that if it were the case that autism were an abnormal genetic variation, or a nongenetic variation (i.e. if autism had some other etiology), then we would be justified in calling it pathological. John Elder Robison, quoted above in support of the first neurodiversity claim, has made this argument more explicitly, claiming “Autism that’s a result of chemical poisoning is a very different thing from the condition I grew up with . . . Being born different is one thing; crippling ourselves through preventable injury or ingestion of chemicals is something else entirely. No one wants to accept that.”

The neurodiversity movement is generally in agreement with Robison on this point, and it seems to make good sense – yet it is difficult to say exactly why. If we accept the claim that autism is to be respected as any other human variation – indeed, that autism is simply an identity, like being male versus being female – then there is no immediately obvious reason why the cause of the identity should matter. What is it about environmental pollutants, as opposed to genetics, that should make us feel differently? Is it the fact that it is due to chemicals? The fact that the pollutants are man-made? Or the fact that the results of exposure to these chemicals was unanticipated and accidental?

Pathologization resists simpleminded equations such as these. Autism in particular poses challenges for those who would like to make a simple delineation between “genetic autism” and “chemically-induced autism” (or what have you): There is a wide range of “function” among autistics – or, perhaps it would be better to say that there is a wide spectrum between people whose autism offers them a few quirks and eccentricities compared to neurotypicals and those whose autism colours every aspect of their lives. Similarly, the main lesson of autism genetics is

118 Robison, “Neurodiversity and Me.”
that autism generally deals in predispositions, and thus we are left not with two obvious and sharply demarcated categories of “endogenous autism” and “exogenous autism,” but an etiological spectrum – and we are certainly unable to tell the difference in practice. For these reasons, we should perhaps be hesitant to draw clear lines between persons whose autism is incidental to the person and those whose autism is integral to the person, lest we be forced to answer difficult questions about whose autism truly “counts” as an identity and whose does not. Shall we only respect the identity claims of those autistic people whose autism is wholly genetic in origin? What about someone whose autism is almost entirely genetic in origin? Even if there were a way for us to tell the difference, where ought we draw the line between natural human variation and pathology? Putting the question in these terms calls into stark relief the theoretical commitments at stake. To draw a meaningful boundary between autism that is nonpathological and autism that is pathological (or even to construct autism as uniformly nonpathological) one needs to assume that there is a meaningful distinction between pathology and health, and that this distinction lines up with some kind of identifiable property – in this case, it is assumed to line up with “normal genetic variations” on the one side, and, presumably, abnormal genetic variations and environmental insults, on the other. In other words, the boundary between pathology and health is drawn along etiological, rather than symptomatological, lines, in which some kind of abnormality of structure or function (i.e. an identifiable etiology) is taken to be evidence of pathology. According to this view, then, our understanding of the roles played by environmental toxins, viral infections, and the like bears directly on the question of whether autism is, indeed, “normal.”

119 There is something suspicious about this right from the start. Arguing that autism is not a disease because it is a normal genetic variation puts normality and disease in opposition to one another, when, in fact, disease is normal, in some sense of the word. Everyone gets sick at one point or another; isn't illness just a normal part of being human? This statement could be made more precise, but to do so would take us too far afield and
2.4 The “normal” function model

These views characterize a particular model of disease – what has been called the “normal function model,” the “species-typical function model,” or the “medical model” of disease. This model, which treats abnormalities of structure or function as constitutive of disease, plays an important role in the medical-scientific conception of health, as well as an important role in the realm of bioethics. Ideally, it gives us an objective and non-arbitrary way to distinguish disease and disability from health, according to Norman Daniels, as “disease and disability are seen as departures from species-typical normal functional organization or functioning.”

According to Daniels, this model is not only an accurate and commonsensical account of disease, but serves two important political ends as well. The first is that it allows us to distinguish treatment from enhancement. Medical interventions that are aimed at restoring a patient's capacities to “normal” are regarded as medical treatments, while interventions aimed at increasing a patient's capacities to beyond the normal range are regarded as enhancements (e.g. the use of nootropic drugs to enhance cognition). This distinction has an important role to play for Daniels in health care – on his view, a fair and just health insurance policy or national healthcare system should, at minimum, provide its clients with access to medical treatments, but not necessarily enhancements. This treatment/enhancement distinction is an important concept in bioethics with an extensive body of literature, but it bears little on the present discussion.

Secondly, the normal function model is thought to help protect against the kind of aggressive pathologization of the same type that neurodiversity advocates are critical of. The model explicitly recognizes that there is a range of “normal” persons, each with different

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capacities and endowments, thus the fact that one person is more shy than another, for example, need not immediately indicate the presence of disease. Daniels uses this model to delineate the “proper domain” of medicine, which is thought to include the amelioration of disease and disability, but not to include complete physical or mental well-being. Thus, while there may be such a thing as “pathological shyness” (i.e. social anxiety disorder), it is not the case, on this view, that all shyness is necessarily to be viewed as disordered.

The statements made by neurodiversity advocates quoted above make them implicitly beholden to this kind of model of disease. Their emphasis on autism as “natural” or “normal” as a basis for demedicalization carries with it the corollary that “unnatural” or “abnormal” variations ought to be medicalized. What the word “normal” means to neurodiversity activists is not immediately clear, however, as “normality” is a bit of a thorny concept, but ultimately proves to be crucially important.

2.5 Three ways to be normal

Robert Wachbroit distinguishes three ways that the word “normal” can be used: what he calls the “statistical,” “evaluative,” and “biological” concepts of normality.\footnote{Wachbroit, Robert. “Normality as a Biological Concept.” \textit{Philosophy of Science} 61, no. 4 (1994).} The first two of these concepts are simple enough. Statistical normality refers to the familiar measures of central tendency, such as the mean and median. On this account, something is considered normal if it is \textit{average} or within some bounds, say, within two standard deviations of average. This is a mathematically exact concept, that can be applied precisely to problems amenable to quantitative analysis. The second concept is that of evaluative normality. This concept is broader than the first, encompassing such things as ethics, conventions, cultural norms and the like. Something
that is evaulatively normal need not be common or statistically normal – for example, although left-handedness is uncommon, very few people (anymore) feel it is unacceptable. Conversely, something that is very common or statistically normal may not be evaluatively normal.

Wachbroit distinguishes a third category of normality, what he calls “biological normality.” Biological normality is an explicitly teleological stance – when we make statements about the “normal” heart or the “normal” lung, we are also making statements about the goal or the function of the structure in question, as in “The function of the lungs is to draw oxygen into and expel carbon dioxide from the body.” Function and biological normality go hand-in-hand for Wachbroit – the normal organ (or mind, or body) is one that fulfills its function; the abnormal does not.\textsuperscript{122} It is this sense of biological normality Daniels has in mind in his “normal function” account – the medical model is one that explicitly looks for “abnormalities” of structure or function against the backdrop of the hypothetical norm. And while neurodiversity advocates unequivocally agree that autism is an evaluatively normal condition, their vehement declarations of particular etiologies as “normal” makes it clear that they also adhere to the view that autism is biologically normal.

Theories that invoke concepts of “biological normality” have been the subject of much criticism.\textsuperscript{123} Distinguishing between “normal” and “abnormal” can, in many cases, feel like a

\textsuperscript{122} There is, evidently, some degree of overlap between these concepts of normality. There is certainly a degree of evaluation implicit in the decision of what constitutes statistical normality (e.g. how many standard deviations from the mean ought we think normal?). Similarly, evaluative judgements are pervasive in decisions about what constitutes biological normality or “health,” as will be discussed below. It is perhaps no surprise that normality is a thoroughly normative concept.

pretty arbitrary affair, especially with regard to etiology. Taking at face value the claim made by Robison that chemically-induced autism is not normal (that is, it is pathological) but genetically caused autism is, we would be forced to conclude that two autistic persons with otherwise identical symptoms and abilities ought to be regarded differently depending on the etiology of their condition, despite the fact that they may experience the same kinds of life challenges and mixed blessings that autism carries. Other authors have made similar remarks about other conditions – such as distinguishing between short-statured children on the basis of whether they have a human growth hormone deficiency or not.\textsuperscript{124} Such a distinction feels arbitrary and capricious given that those with similar symptomatology will experience similar challenges. Why should etiology have a role to play in deciding which conditions “count” as diseases?

The answer to the question lies in another facet of the normal function model that has received criticism – that it relies upon a “theoretical account” of the design of the organism.\textsuperscript{125} To say that such-and-such condition is biologically normal requires knowledge of the “plan” or “schema” of how the organism “ought” to function: What is it supposed to look like? What capacities is it supposed to have? This stance is explicitly teleological, and might be favourably compared to the stance an auto mechanic takes when diagnosing and repairing a car. Knowing the design specifications of the engine, the mechanic knows just how the engine is supposed to look, what each belt and hose is supposed to do. Adopting a teleological stance toward an automobile makes sense; automobiles are designed by human beings with intentions, those intentions are built into the design of the automobile, and we are able to know the “theoretical account” of the design of the automobile because we have access to design specifications and

\textsuperscript{124} Daniels, “Normal Function.”
\textsuperscript{125} Parens, “Is Better Always Good?”
operations manuals. However, as human beings were not designed by someone with intentions, but rather developed incrementally through the process of evolution, we have no design manuals to inspect. Moreover, as evolution is an unthinking natural process and famously short-sighted, it would not be overstating the case to say that humans were not designed for anything. Thus, if we are to have a “theoretical account” of the design of the human, then it is something we must glean (or, rather, assign) through observation and inference. What is a human being supposed to look like, anyway?

This turns out to be a difficult question to answer, as there is a certain kind of circularity involved in establishing what constitutes “normality.” King explains,

We think health as freedom from disease, and disease as an aberration from health. This is travelling in circles . . . When we apply statistical methods we already have in mind the idea of health. We exert selection on the cases we study. Thus, to find the “normal” blood sugar level we eliminate known diabetics. And the basal metabolic rate, in health, we determine after omitting known thyroid disease.

Here, King has in mind the idea of statistical normality, but in other cases we might be less interested in statistics and more interested in an “ideal,” or what we have called “biological normality.” King gives the example of dentistry, in which the healthy ideal is to have a full set of 32 teeth, even though the average American adult only has about 25 remaining. Thus, even though most adults may vary from the ideal, the “theoretical model” of the human has 32 teeth, and deviation from this desideratum constitutes deviation from ideal health – what we might

126 Even the use of the word “designed” here perhaps suggests too much agency on the part of evolution.
127 King, “What is Disease?” 195.
conventionally call “disease.”

While missing a few teeth may not be what most people have in mind when they use the word “disease,” the above considerations capture several important points on the issue of what diseases consist in. King explains that our concepts of disease are very closely related to our values. . . . Disease is the aggregate of those conditions which, judged by the prevailing culture, are deemed painful, or disabling, and which, at the same time, deviate from either the statistical norm or from some idealized status. Health, the opposite, is the state of well-being conforming to the ideals of the prevailing culture, or to the statistical norm.129

This is one of the major problems with the normal function model. Not every departure from the hypothetical biological norm constitutes a disease, and those departures from the norm which do qualify as diseases do so for reasons that are intimately bound up with the way we value persons. Indeed, the very definition of the hypothetical biological norm is evaluative.130 In many cases these evaluative judgements are relatively uncontroversial – few would argue that malaria is not a disease, on this or any account – but in some cases these kinds of judgements can serve to systematically marginalize persons who, for whatever reason, do not conform to these norms. These norms are intimately bound up in the culture of the time and place, especially as regards “mental illness.”131 Autism in particular has all the hallmarks of a historically contingent

129 King, “What is Disease?” 197.
131 Horwitz, “Creating Mental Illness.”
disorder, as discussed by Nadesan in her book *Constructing Autism,*\(^\text{132}\) what today we medicalize under the banner of “autism spectrum disorders,” we might at a different point in time have categorized as “eccentricities” (in the case of high-functioning individuals) or “feeblemindedness” (in the case of low-functioning individuals).\(^\text{133}\) What constitutes “normal,” then, is a bit of a moving target.

The upshot of these observations is that so-called “normal function” models or the “medical model” rely heavily on value judgements and normativity to establish what does and does not “count” as normal – what, in other words, a human being is *supposed* to be like. The fact that the dominant “medical model” is fraught with assumptions, political claims, and normativity has motivated some neurodiversity advocates to adopt the “social model” of disease and disability (Claim 5). As discussed there, the social model understands an autistic person’s *disability* as produced through the interaction of their capacities with the social and physical environment. Disease, on this account, is not a “natural kind” – that is, not so much a category of biological fact as one of evaluative judgement. We do not call HIV a “disease” simply because of the presence of a virus, nor do we call teratoma a disease simply because of the presence of a tumour – we call them diseases because of the normative judgements we make about them. HIV and teratoma are, quite simply, undesirable conditions to have.

That many of the problems with the normal function model are the same problems decried by neurodiversity advocates regarding the “cure mentality” and the medical approach to autism makes it all the more surprising that such advocates would use the language of normality to defend their position. While many neurodiversity advocates avow their adherence to the social

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\(^\text{132}\) Nadesan, “Constructing Autism.”
\(^\text{133}\) Hacking, “Kinds of People.”
model, and while the social model of disability addresses the normative problems associated with
the medical model, there seems to be an incomplete adoption of the social model within and
among the positions of the neurodiversity movement.

2.6 Identity revisited

In this connection, it is perhaps also worth asking why neurodiversity advocates have set up what
seems like a false dichotomy by claiming that autism is an identity, *rather than* a pathology.
Conditions that affect the mind – what we might conventionally call “mental illnesses” or
“mental disabilities” – often have a difficult relationship with personal identity, and it is this
tension that is pointed to by neurodiversity advocates who make the claim that autism is a
positive identity – indeed, an inextricable part of the person, rather than a disorder or a disease.
When we speak about autism as a “disease” or we describe someone as a “person with autism”
we are exposing a tacit belief that autism is separable from the person, in contrast to the belief
that autistic people simply have a different kind of brain or mind from neurotypicals.
Interestingly, this sense of autism as an identity may actually be strengthened by increasing
knowledge of its etiology. As mentioned above, the current paradigmatic definition of ASD is a
descriptive one – it assigns the label to individuals who exhibit certain behavioural
(ir)regularities, without reference to cause. Knowledge of the neurophysiology of autism, in this
sense, provides an explanatory framework that the nosological category previously lacked.

134 For example, individuals with bipolar disorder, chronic depression, or anxiety disorders often struggle for years
trying to find a regimen of psychiatric drugs, exercises, and therapies that alleviate their symptoms, and when
they do so, report feeling like they are *finally themselves*. But many people, upon finding a treatment that
relieves them of the symptoms of their condition by psychometric measures, are left feeling robotic, or
otherwise “not themselves.” People with certain conditions, such as type I bipolar disorder, are notoriously
difficult to keep medicated, as many would rather suffer the negative consequences of their condition than go
through life feeling like someone other than themselves. See, for example, Karp, David A., *Is It Me or My
But it is not immediately obvious that these two categories – identity and pathology – are mutually exclusive.\textsuperscript{135} Other kinds of identification, such as "cancer survivor" or "alcoholic" blur this distinction – or disregard it entirely, though they do so as a means of engaging with, addressing, and reappropriating what is otherwise a completely undesirable condition. Those who identify as cancer survivor do not necessarily do so because they want their cancer to be respected as any other human variation, but may do so as a way of reclaiming a painful and potentially deadly condition as a means of strength and personal growth. Nor would most alcoholics embrace alcoholism as a desirable condition, but identifying as an alcoholic is seen, in some approaches, as a necessary step toward recovery and treatment. In this sense, there is an important disanalogy between these cases and the case of autism, in that embracing autism as an identity \textit{and} a pathology would mean accepting the values implicit in the pathology label. Treating autism as we treat cancer or alcoholism could be seen to locate the autist's suffering in the autism, not in the social environment.

But this also relates to the neurodiversity movement's tacit acceptance of the valuations implicit in the label of "disease," which fact speaks to their indebtedness to "normal function" models, as discussed in the previous section. But also, insofar as the neurodiversity movement implicitly accepts the ill/well dichotomy as presented, they have forced a dilemma upon themselves.\textsuperscript{136} Namely, they have to \textit{either} place autism into the category of pathology or health – or break the condition up along some simplistic, univariate axis of ability. Perhaps the obvious

\textsuperscript{135} The complex and difficult relationships people have with their "pathologies" is treated at great length in Elliott, Carl, \textit{Better than Well: American Medicine Meets the American Dream} (New York: W. W. Norton, 2003) and Elliott, Carl, \textit{A Philosophical Disease: Bioethics, Culture and Identity} (New York: Routledge, 1999).

\textsuperscript{136} Perhaps they have not forced it upon themselves so much as had it thrust upon them by the existing structures of medical, political, and social power that refuse to acknowledge the subtle and nuanced ways in which autistic and other neurodiverse persons are simultaneously enabled and disabled by their condition, and the many ways in which autistic persons differ greatly in their abilities.
solution is to reject the ill/well dichotomy entirely. Yet the language of disease has the power to be simultaneously both stigmatizing and liberating. “Biologized” accounts of mental illnesses are becoming increasingly common as neuroscience and cognitive psychology gain ground over traditional psychological or psychoanalytic explanations of those conditions, but these new accounts come with decidedly mixed blessings. Such explanations are no doubt well-intentioned, as in the case of the “brain disease” metaphor for drug addiction – an account of addiction as a disease can reduce feelings of guilt or blame for the condition, as well as making it easier for addicts to receive treatment, but it can also reduce feelings of personal responsibility (perhaps necessary for recovery) and increase social stigma.\textsuperscript{137} Social stigma may make it less likely for the patient to seek medical treatment (even if said treatment is more easily available), experience social isolation, and be a constant source of emotional pain.\textsuperscript{138} Thus, even if biogenetic accounts of autism or, for that matter, a label such as “disease” or “disability,” makes needed services more easily available to autistic persons who need them, one may feel that, on balance, such labels and explanations are causing more harm than good to autistics. Taking a stance that simultaneously embraces autism as an identity and a disease could prove a much more radical position – one that transgresses the conventional binaries of ill and well, normal and abnormal.

2.7 Conclusion

This debate between and among academics, scientists, neurodiversity advocates, and the lay public provides rich terrain for the exploration of values and normativity in medicine. There are, of course, many questions here left unasked and unanswered that bear on these matters. Among


them are such questions as these: Although normativity is pervasive in the way we conduct medicine, which may account for the current marginalization of autistic persons, is normativity categorically undesirable in medicine? Are we even able to do medicine without at least some degree of normativity, especially as regards mental illness – or conditions that are seen to affect the mind? Specifically, can we always defer to the authority of the patient to make decisions on their own behalf, even when their condition calls their competence directly into question? And does that phrasing implicitly beg the question?

Ultimately, questions such as these probe the boundaries not just of the neurodiversity movement, but also of the orthodox scientific conception of autism. We should ask: if we were to take the neurodiversity position seriously, would that mean “anything goes?” How would we deal with difficult cases such as schizophrenics who may pose a danger to themselves or others, but nevertheless refuse treatment? Can these cases be adequately dealt with through a utilitarian application of the law, or would doing so simply be a rebranding of the normativity implicit in the psychological programme of categorization, diagnosis, and treatment? On the other hand, the neurodiversity movement addresses serious problems with the psychological paradigm, and we ignore these insights at our own peril. This movement points to a kind of perceived aggressive pathologization becoming increasingly common, which at its extreme entails a kind of “weaponizing” of the DSM, so to speak: using psychometric measures of behaviour and nosologies of mental illness as a means of political, social, and economic power.

There is a need for much more scholarship and research regarding these questions, as well as the question of autism's etiology. The science, as they say, is still in its infancy – scientists are still a long way from establishing a stable narrative or narratives explaining the
origins of the condition. Increasingly, it appears that the picture being painted of ASD is one of a messy, disunified category that lumps together people with certain superficial behavioural, cognitive, affective, or other characteristics. The production of these characteristics may be overdetermined – produced by a complex dynamic interplay of social forces and biological ones, personal will and environmental determinism, genetics and socialization.

But the neurodiversity movement has an uneasy relationship with questions of autism's etiology. Some of the claims of the movement are in direct conflict with emerging scientific knowledge, while in some cases a subtler conflict arises from underlying assumptions about the nature of suffering and disease. But the ultimate goal of the neurodiversity movement, it seems, is to promote awareness and acceptance of diversity in the human sphere, and promote the self-determination and rights of autistic persons specifically, and the “neurodiverse” more generally. This is no doubt a worthy goal, and insofar as the scientific research concerns itself with causal agents and matters of fact, there need be no conflict. But to the extent that etiological research simultaneously constructs and is constructed by a conceptual framework in which autism is a “mental illness” or “developmental disability,” this discourse systematically constructs autism in a way that calls into question the competence, and therefore self-determination, of autistic persons.

The neurodiversity movement hopes to counter this trend by situating autism in the realm of health through their use of the language of “normality” and “natural genetic variation.” But just as predicating the neurodiversity claim on the inchoate etiology of autism spectrum disorders leaves it vulnerable to being undermined as the science develops, similarly, staking out the claim that autism is not a pathology because it is normal commits oneself to a problematic concept of
the nature of disease. Perhaps a more robust tactic for neurodiversity advocates would be to remain explicitly agnostic on the question of autism's etiology. Many do. By deliberately moving the focus away from etiology, neurodiversity advocates can focus the debate on the normative standards implicit in the project of constructing nosologies of mental illness.

If, in the preceding pages, I've done little more than explain the fairly obvious fact that scientific research into the etiology of autism carries with it value judgements, normative assumptions, and ethical quandaries, then so be it. That science is inherently value laden is hardly a novel statement. But the particular contours of that scientific research – and the particular values it carries – have an important role to play in shaping the lives and experiences of those who live with autism spectrum disorders daily. One wonders, though, about the stability of the neurodiversity movement. It may turn out to be the kind of activism and advocacy that can only sustain itself in a (partial) scientific vacuum – in the liminal space between naming and identifying a condition, on the one hand, and constructing a coherent etiology, perhaps in tandem with a treatment, on the other. This may seem like a pessimistic prediction, but it has not been my goal here to make judgements on whether the autism “really is” normal or pathological – in my opinion, these are the wrong questions to ask. I have instead endeavoured simply to show the ways that these two ways of thinking – the “medical” and the “neurodiverse” – are in tension with one another. It may be the case that some of these tensions can be resolved, either through a revision of the neurodiversity programme, or by a more careful rhetorical approach by the scientific and lay communities. Scientific research aimed at discovering the cause of autism may be more or less value neutral on the surface, but this research also suggests programmes for the treatment and prevention of the condition, and is often motivated explicitly by this desire.
Technologies of this kind put ordinary people into situations of forced choice, in which making ethical decisions and value judgements about the desirability of autism – and by extension, its status as pathology – are unavoidable. The normative contours of this etiological research, however well intentioned, are thus impossible to ignore.

Of course, all these observations gloss over certain other questions I feel also need to be asked. Why, for example, did I speculate that scientific research might undermine the neurodiversity movement's foundation, and not vice versa? Why has the neurodiversity movement received so much criticism from parents, scientists, and the like? Why are we so eager to let scientists speak about autism, rather than autistic persons themselves? Perhaps, then, the key insight of the neurodiversity paradigm is not that autism is “normal” nor that autistic persons deserve respect, rights, and opportunities appropriate to their abilities, but that it calls into stark relief the theoretical commitments and inherent ethical contours of neuroscientific research directed toward autism – as well as our own beliefs about who has the authority to speak on behalf of whom. With these insights clearly in mind, perhaps we can tread more carefully in the realm of mental health.
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