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Autism's anatomy

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Autism's Anatomy

A dissection of the structure and development of a
psychiatric concept

Berend Verhoeff

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Autism's Anatomy

A dissection of the structure and development of a
psychiatric concept

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Voor Paulien, Elin en Leonard.

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Autism's Anatomy

1 | Introduction

A focus on autism

Autism makes an appealing case as a way to explore the structure and development of psychiatric knowledge. When I started my psychiatry residency training program, more than a decade ago, the phenomenon of autism had not yet been fully settled in the minds of mental health professionals, especially not in adult mental health. I remember visiting a middle aged man who had been a patient in an ambulatory psychiatric service for people with enduring psychiatric problems. He received psychiatric support from a multidisciplinary team for many years and he had seen many psychiatrists and medical doctors who had formally diagnosed him with a *DSM-IV* anxiety disorder not otherwise specified (NOS). When I visited him at the request of his psychiatric nurse and case manager, I saw an anxious and lonely man in an extremely dusty apartment. He never cleaned, never cooked, only left his house for vital necessities, collected war documentaries, hardly talked, and, apart from the mental health professionals, he only had social contact with his sister. When I visited him, he suffered from panic attacks since his landlord had threatened to remove him from his apartment due to nuisance and contamination.¹ At that time, my colleagues and I saw a suffering man in desperate need of care and support, but we did not see a man suffering from autism. We still had to learn to perceive autism. Certainly, he was not the typical *Rain Man*, but with autism-specific diagnostic questions, a developmental biography, and an autism-trained

¹ This case has been slightly modified to prevent any type of recognition of the particular patient.

psychiatric gaze, the chances are high that he would now be diagnosed with an autism spectrum disorder.

Today, autism is harder to miss. In a relatively short period, autism has become one of the most diagnosed, researched, and discussed psychiatric disorders. Estimated prevalence rates went from about 1 in 2500 (Wing et al., 1976) to 1 in 68 (CDC, 2014) in less than forty years. The possible existence of a real ‘autism epidemic’ is widely discussed among autism researchers (Leonard et al., 2010), and, the ‘dramatic increase in autism prevalence parallels [an] explosion of research into its biology and causes’ (Dawson, 2013). In 2010, federal and private foundation funding for autism research in the US exceeded US\$400 million (Pellicano et al., 2014). In 2000, funding for autism research in the US was approximately US\$50 million and compared to other developmental disorders (for example, ADHD, Down syndrome, Tourette syndrome, Fragile X syndrome and Fetal alcohol syndrome) autism research receives the most funding and has the highest annual rate of increase in funding in the US (US\$50 million annual increase between 2000 and 2010, Bishop, 2010). Even though the severity of the disorder cannot explain this growth in research funding, more and more of the NIMH and charity pie goes to autism research (ibid.). Within the UK, more than half of all autism research funding awards were given to projects in basic research areas of biology, brain and cognition. Cortical development, social cognition and animal models of autism were the most significant areas of funding between 2007 and 2011 (Pellicano et al., 2014). All this research needs to be published in scientific journals. *Molecular Autism*, *Autism Research*, *Research in Autism Spectrum Disorder*, *Good Autism Practice*, and *Autism Research and Treatment*, are all full-fledged and autism-specific international peer-reviewed journals that have only emerged over the last couple of years.

Next to all sorts of scientists and mental health workers who organize their research, clinics, careers, institutes and journals around the disorder of autism, the interest in autism has spread to the social sciences. As social scientist Chloe Silverman expressed it, ‘the idea of autism – as a metaphor, a neurological disorder, a mental state, an increasingly prevalent diagnostic category, or a species of neurological difference – has an almost ready-made appeal for social scientists, concerned as they are with questions of human identity, difference, perception and subjectivity within a social and cultural context’ (Silverman, 2008: 325). An example of a product of this appeal is *The Autism Matrix* by Eyal

et al. (2010). They give a sociological explanation for the dramatic rise in autism diagnoses. For Eyal et al., the prime suspects of the ‘autism epidemic’ are not the much-discussed mercury-based vaccine preservatives, old fathers, or the use of antidepressants during pregnancy. Instead, their explanation of the epidemic involves the emergence of autism parent organizations since the 1960s, the extensive deinstitutionalization of intellectually disabled children since the 1970s, and in greater benefits for those diagnosed with autism due to the growing availability of special services from 1991 onwards when autism was added to the Individuals with Disabilities in Education Act (IDEA).

The remarkable surge in autism prevalence also paralleled a rapid growth in popular awareness, visibility and concern regarding autism. Mark Haddon’s *The Curious Incident of the Dog in the Night-time* (2003) and an ever-growing collection of ‘autistic fiction,’ autobiographies, biographies, movies, documentaries, stories for children, stage plays and graphic novels have certainly contributed to the popular recognition of autism (see Hacking, 2009).² In less than three decades, autism transformed from a rare and largely unknown syndrome into an almost inescapable cultural experience and fascination. For three more specific reasons autism makes a good case to explore the structure and development of psychiatric disease concepts – the central entities that shape both psychiatric practice and research.

A genetic brain disease

To begin with, autism is often seen as a paradigmatic (child) psychiatric disorder. It is thought to have a large genetic component. Concordance rates among monozygotic and dizygotic twins are 50-90 percent and 0-30 percent, respectively, supporting a large genetic contribution (Berg and Geschwind, 2012). The genes or genetic variances associated with the disorder are assumed to be involved in the development of our brains. Almost without exception, autism researchers and mental health professionals see autism as a highly heritable and lifelong *neurodevelopmental* disorder. Probably more than depressive disorder, ADHD, anxiety disorders, conduct disorders or even schizophrenia, autism is primarily understood as a brain disease with which one is born and dies. Furthermore, many researchers and clinicians acknowledge the

² See Draaisma (2009) for an analysis of how popular representations of autism in novels, TV-series or movies can contribute to a harmful divergence between the popular image of autism and the clinical reality of autism.

distinctiveness and recognizability of autism. For autism expert Fred Volkmar, autism is one of the better examples of a disorder ‘that is distinctive and does not just shade off into normalcy’ (Volkmar, 1998: 45). Uta Frith, another well-known autism expert, also considers it a recognizable entity (Frith, 1989) and Leo Kanner himself, autism’s founding father, stated unreservedly that the disorder represents a ‘definitely distinguishable disease’ (Kanner, 1965: 418). Moreover, many parents of autistic children and professionals who observe, treat and interact with a lot of children diagnosed with autism develop a certain readiness to quickly perceive and recognize an autistic child as somehow different from a ‘normal’ or non-autistic child (see Chapter 2).

Against the background of psychiatry’s preferred identity as a *medical* specialty, autism’s neurobiological and distinctive image are expected to be promising for making successful translations from the biomedical sciences and the neurosciences to the clinic. Psychiatry’s need for successful translations ‘from bench to bedside’ is not particularly recent. In 1973, the historian of medicine Charles Rosenberg presented a lecture – *The crisis in psychiatric legitimacy: Reflections on psychiatry, medicine, and public policy* – that would not appear obsolete or irrelevant from a present-day perspective (published in Rosenberg, 1975).³ Rosenberg noticed a growing gap between the performance of psychiatry as a medical specialty, and the expectations from society and psychiatry itself, regarding successful psychiatric care, treatments, and (brain-based) explanations of deviant sexual, moral and social behavior. Psychiatry’s responsibilities to cure and illuminate – in Rosenberg’s prose of the antipsychiatry movement of the 1970s – ‘the clinical burdens of a society which “produces” vast numbers of individuals whose behavior is stigmatized by that society as mental illness,’ (ibid.: 249) contrasted with the sparse technical means and the lack of insight into the pathophysiological processes of these particular clinical burdens. The identification of psychiatry as a medical specialty was ambiguous, and psychiatry, while resting on its status and authority of a medical profession, was at least an unusual medical specialty. Many other areas in clinical medicine had been able to link understandings of pathological mechanisms with specific

³ As I explain later on, the work of Charles Rosenberg has been an important source of inspiration for this dissertation. Rosenberg wrote his essay *The crisis in psychiatric legitimacy*, for a meeting in Williamsburg, Virginia, in 1973 ‘to commemorate the bicentennial of the British North American colonies’ first public hospital for the mentally ill’ (Rosenberg, 1975: 245). The essay was published in 1975 in an edited book containing all the papers that were presented at this particular meeting.

therapeutic interventions that structured and defined the boundaries within which clinical decisions were being made. This was not the case for psychiatry, whose theoretical foundations were based on the analysis of clinical experiences, and who struggled to substantiate their claim to exclusive (medical) care and control – against ‘rival schools of emotional healing’ – of those defined as mentally disordered. For Rosenberg, the crisis in psychiatric legitimacy was exactly this gap ‘between the demands of medical exclusivity and the inability of psychiatry to provide either understanding or relief consistent with the pretentiousness of such demands’ (ibid.: 250). These considerations are – more than forty years and heaps of neuroscientific, epidemiological, clinical and psychopharmacological research later – still valid.

Those who followed the fierce debates surrounding the publication of *DSM-5* (APA, 2013) must have noticed that the major themes of the discussion were the free-floating boundaries of psychiatry (for example, Frances, 2013); the ‘premature’ state of behavior-based classification and diagnosis; the dubious role of big pharmaceutical and biotechnology industries in shaping psychiatry; the complete absence of useful biomarkers for treatment and prediction; the non-specificity of psychopharmacological drugs and the ambiguous evidence for the beneficial effects of these drugs; and the use (or misuse) of the diagnostic manual and psychiatry for bureaucratic, juridical, educational, insurance, and other purposes. Of course, the forces and stakeholders that shape contemporary psychiatry have shifted since the 1970s, but the ‘crisis in psychiatric legitimacy’ still comes down to a tenacious discrepancy between the expectations from a truly *medical* specialty and the performance of that same specialty. These persistent uncertainties and fragilities of a medical specialty in search of its medical identity and legitimacy are part of the broader motivation behind this dissertation.

As I have said, autism’s neurobiological and distinctive image are promising for making successful translations from the biomedical sciences and the neurosciences to the clinic. Consequently, the expectations to develop a biotechnical armamentarium and close and successful connections between biomedical research and clinical practice are particularly high for the field of autism. The illumination of pathophysiological pathways, the identification of specific diagnostic markers, and the development of ‘normalizing’ interventions on the basis of these pathophysiological mechanisms are the promising prospects of a medical discipline specialized in emotional, behavioral and

cognitive maladies. The field of autism is one of those fields in which the medical identity and legitimacy of psychiatry is very much at stake. This is all the more urgent given the rising rates of autism diagnoses and the significant economic costs of autism – a person with autism without an intellectual disability costs US\$1.4 million during her or his lifespan in the US (Buescher et al., 2014). By meeting societal and medico-techno-scientific expectations, autism could be a forerunner and important example for (child) psychiatry in general.

Attempts to fulfill these expectations are passionate and, given the considerable clinical and economic ‘burden’ of autism, it is no surprise that autism research is booming. Thousands of autism researchers worldwide devote their working days to ‘unraveling the mystery of autism’. Performance, however, is still far away from fulfilling the great expectations. Despite many premature claims of sensitive and specific diagnostic tests for autism – for instance by Duffy and Als (2012) who claimed to have developed an electroencephalogram (EEG) test that could distinguish autism from non-autism – there are no clinically useful biological tests or specific neurobiological treatments for autism on the basis of identified pathophysiological pathways.

An elusive entity

This is a second reason why autism makes a fascinating case for studying psychiatric disease concepts: autism is on the one hand culturally pervasive and an undeniable reality for patients, families, clinicians, researchers and even for social scientists while, on the other hand, it remains a big mystery in terms of its causes, its neurobiological underpinnings, its treatment, prognosis and prevention. In other words, the nature of autism remains disturbingly unknown. What is more, the *idea* of autism – or what is thought to be essential in autism – is far from stable. In its brief history, autism has been a disorder of affective contact (Kanner, 1943); a language disorder (Rutter and Bartak, 1971); a disorder in processing and integrating perceptual information (Wing and Wing, 1971); a disorder of executive functioning (Hill, 2004); a disorder of reading other people’s minds (Baron-Cohen, 1995); a disorder of weak central coherence (Happé and Frith, 2006); an excess of ‘systemizing’ drives at the expense of ‘empathizing’ drives (Baron-Cohen, 2002); a disorder of social motivation (Chevallier et al., 2012); a disorder of social cognition (Wing et al., 2011); and more. The core features of the concept of autism are not as stable as

many autism researchers presume. Nevertheless, the multiplicity of manifestations, cultural representations, explanatory frameworks and ‘essential’ characteristics of autism is, especially among social scientists of autism (for example, Murray, 2008; Fitzgerald, 2012), increasingly acknowledged. Given this multiplicity of autism realities, I suggest that if we want to understand how psychiatric knowledge develops, it is important to understand what it is that keeps the very idea of autism together and autism research thriving. That will be a central theme in this dissertation.

How is it possible to keep on talking about autism; to keep a stable image of autism; to keep on doing fundamental research on autism; to keep on perceiving clear cases of autism; and to keep on organizing psychiatric care around the category of autism, while the idea of autism is notoriously elusive, heterogeneous and variable in the way it is and has been conceptualized? How can autism be both a ‘successful,’ recognizable and well-researched disease, and yet be notoriously unstable at the same time? These questions kept on wafting into my mind during the process of thinking and writing about autism. Both constancy and change seem to characterize the entire historical trajectory of autism. One of the things I realized was that a certain degree of conceptual flexibility has been a prerequisite for a certain form of continuity, stability and unification among autism researchers, clinicians and other ‘exoteric’ communities in the broad field of autism (see Chapter 4). This dissertation argues that an exploration of the interactions between changing ideas *of* autism and the steady search *for* autism – that is, the search for behavioral, cognitive and biological common denominators or ‘the essence’ of autism – helps to understand one of the ways in which psychiatric knowledge develops.

A social disorder

A third and final reason for me to focus on autism is that it is not only seen as a paradigmatic psychiatric disorder, but also widely contested as a *medical* category. As Rosenberg also pointed out in his 1975 article on psychiatric legitimacy, to a far greater degree than other medical specialties, psychiatry is shaped by social values and attitudes toward deviance, aging, child development, and ethnicity, as well as by the needs of social policy and by other decisions made outside the medical profession of psychiatry. The emergence of psychiatry in the nineteenth century, more recent trends toward deinstitutionalization, shifts in (psychoanalytical or biological) explanatory

frameworks, and a panoply of now-discarded treatments (of which lobotomy and insulin coma therapy are prominent examples), reflected specific social needs and shifting ideologies, rather than an outcome of an ‘expanding body of knowledge or the crystallization of particular techniques’ (Rosenberg, 1975: 247). Rosenberg held that *despite* the fact that psychiatry lacks the technical means and knowledge to delineate its field, it still needs to deal with a large variety of emotional pain and psychological disabilities. And *because of* the fact that psychiatry lacks the technical means and knowledge to delineate its field, it is prone to border disputes and it has a diffuse societal responsibility. In other words, the lack of technical means and knowledge to delineate the field makes psychiatry particularly vulnerable for all sorts of social influences.

Given that autism is currently characterized and diagnosed on the basis of deficits in *social* behavior (for example, deficits in social-emotional reciprocity, non-verbal communication and in developing relationships), it does not come as a surprise that some argue that the rising numbers of people with autism reflects social changes. Among other things, a growing medicalization and pathologization of diversity; increasingly narrow social norms; shifting educational ideals toward mental flexibility, teamwork and oral presentation; and growing expectations from and demands on children’s social skills, are potential causes for the ‘autism epidemic’. For example, Molloy and Vasil (2002) see Asperger syndrome – now incorporated into autism spectrum disorder (APA, 2013) – as a socially constructed category because of its value as a category for organizing special education. Fein (2015) argues that a changing sociocultural milieu ‘in which friendship and other extra-familial relationships are increasingly determined by individual choice, with affiliations formed around likeability and the negotiation of mutual positive affect’ contributed to the increase in autism diagnoses for those ‘who are slower to develop nonverbal awareness, perspective taking, and emotional self-regulation (Fein, 2015: 82).

Notwithstanding the value of these type of studies, I am not primarily interested in disentangling all the (social) factors that might have contributed to the ‘autism epidemic’. Instead, I am interested in what we can learn about the structure of psychiatric knowledge from the almost paradoxical situation in which autism can both be extremely prone to border disputes and vulnerable to socially-induced diagnostic expansion and yet simultaneously be a paradigmatic psychiatric disorder that awaits neuroscientific discovery. How does psychiatric

research deal with these social issues and the boundaries of mental (ab)normality? Why is it that certain forms of social interaction, eye-contact, body language, imaginative play, and so on, are considered deviant, and at what point do they become deviant? Again, due to its inherent social component, autism makes a rewarding case to explore these questions in this dissertation.

A historical approach

This PhD dissertation consists of seven independent articles that have been published in or were submitted for publication to diverse scientific journals in the fields of philosophy, history and sociology of medicine and biology. I tried to publish in autism journals, but my manuscripts never entered the peer review process. A common response I got from the editors was that they are ‘accepting very few review or conceptual articles, and those that are accepted are typically solicited by the editorial team’ (personal e-mail). With the prevailing idea of autism as a heritable brain disease, historical, philosophical and sociological studies of autism are indeed not very urgent for autism research itself. However, as I argue in this dissertation, a closer connection to and collaboration with social, historical and conceptual studies can be very valuable for the field of autism – even for the basic brain sciences that study autism. Hopefully, this collection of articles will support my future attempts to bring autism research and the human and social sciences closer together.

For those who will read the entire dissertation, I apologize for quite a bit of overlap between the chapters. Nevertheless, the diversity in journals reflects the diversity of the perspectives that I have tried to incorporate in exploring expert ways of thinking about autism and what these ways of thinking might tell us about the structure and development of psychiatric knowledge. The seven articles, diverse and autonomous as they are, are tied together by the aim to render present knowledge and practices in the field of autism intelligible. Unfortunately, studies that try to understand the social, cultural, political, historical and economic determinants of current psychiatric thought (for example, Dehue, 2008; 2014; Young, 1997) are sometimes equated with radical constructionist accounts that see psychiatry as a pathologizing and medicalizing vehicle for social control (Szasz, 1972). I want to make clear that, similar to the work of above-mentioned authors, this dissertation is not a discourse of

suspicion that tries to underplay the seriousness of psychiatric problems or that tries to uncover malicious practices or conspiracies between medical imperialists and Big Pharma. Nor is it a critical reflection that tries to uncover ‘bad’ psychiatric research. Lastly, I do not attempt to present abstract philosophical reflections on the nature or social construction of psychiatric disease, on a general distinction between health and disease, or on something like the mind-body problem in mental health. Instead, my aim is to present an anatomy of the notion of autism – a critical reflection that recognizes and explores the often implicit philosophies that are operative in the field of autism itself.

To achieve this aim, one of the assumptions guiding this dissertation is that it is necessary to elucidate the past in order to understand the concepts and traditions that determine our current ways of seeing and knowing. People did not simply open their eyes and notice autism all around them. Being able to see autism requires much more than that. It would be overambitious to analyze all the historical conditions that make it possible to perceive autism, to talk about autism, to shape ‘autistic’ identities and experiences, and to investigate, discover and know autism in a particular way. Besides, some of the important social and cultural spaces that made it possible for autism to emerge as a distinct disorder – for instance, compulsory education and the rise of mental hygiene movements in the early twentieth century – have already been scrutinized (see Nadesan, 2005). Nonetheless, the importance and role of underlying concepts of disease and the ways in which actual research findings effect new conceptualizations of autism are often overlooked in historical and sociological studies of autism. These aspects will get substantial attention in this study and I took inspiration from a combination of works of several distinguished theorists, historians and philosophers of science.

Specifically, I draw on the works, concepts and methods of Ludwik Fleck (1935/1979), Georges Canguilhem (1966/1991), Charles Rosenberg (2007), Ian Hacking (2002) and Nikolas Rose (2013). Apart from the fact that they explore similar themes in the fields of medicine and the life sciences, these authors share a sincere interest in and profound knowledge of the field they reflect upon. They all want the objects of their own discussions to do well and they do not merely attempt to dismantle the hidden premises and assumptions that support particular scientific practices and truth claims. Most of these authors share the assumption that internal and external aspects of science can only

artificially be disconnected. Scientific ideas and results are always connected to the societal context in which they appear and they inherently depend upon collaborations and communicative interactions between scientists, scientific communities, and external groups of people. Furthermore, these authors theorize that epistemological and ontological matters cannot be easily distinguished. Scientists not only try to discover the world or produce knowledge of the world, but they also bring novelty to the world. It is in the spirit of these authors, who are sometimes grouped as ‘historical epistemologists’ (Rheinberger, 2010), that this dissertation is written.

The next six chapters contain several conceptual histories of autism (Chapters 3, 4 and 5) and analyses of the often implicit philosophies that are operative in the field of autism today (Chapters 2 and 4). Chapter 3 argues that the histories written by the discipline itself play a significant epistemological role and the chapter demonstrates – in an alternative history of the concept of autism – that there have been major shifts in the type of symptoms, signs and impairments that were thought to be essential for autism. Chapter 4 offers an account of the reframing of autism as a *neurodevelopmental spectrum disorder* by using the conceptual tools of philosopher of science Ludwik Fleck. In Chapter 5 I compare current understandings of autism with psychoanalytical understanding of autism, and I argue that the history of autism needs to account for two rather different kinds of autism, which are based on different understandings of psychiatric disease. These two kinds of autism are embedded in and reveal two very different ‘styles of psychiatric thought’. As I have said, my aim is not to reject present ways of thinking about autism or recent aims of autism research, but I do point out some difficulties that I think are inherent in current ways of thinking about psychiatric ailments. For a substantial part, these difficulties will be discussed in Chapters 2 and 6. In addition, I do hope to open up new ways to investigate and intervene with the behavior we are accustomed to explain by the elusive entity called autism, even when new conceptualizations of autistic behavior will certainly generate new problems. Chapter 7 offers a concrete attempt to explore new ways of thinking about psychiatric disease through the work of neurologist Kurt Goldstein.

Generally, I hope that *Autism’s Anatomy* will contribute to the awareness of the contingency of psychiatric concepts that guide and shape everyday practices, both in the lab, in the clinic, and in society at large. This will create space for possible alternatives in thinking about psychiatric disease. In the final

chapter (Chapter 8), I draw conclusions about the structure and development of the concept of autism and I come back to the larger motivation behind this study: the issues regarding the objectives, preferred medical identity and legitimacy of contemporary psychiatry.

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2 | What is this thing called autism?⁴

To act, it is necessary at least to localize. For example, how do we take action against an earthquake or hurricane? The impetus behind every ontological theory of disease undoubtedly derives from therapeutic need. When we see in every sick man someone whose being has been augmented or diminished, we are somewhat reassured, for what a man has lost can be restored to him, and what has entered him can also leave. (Canguilhem, 1966/1991: 39)

How does one find a law for irregular phenomena? – this is the fundamental problem of medical thinking. (Fleck, 1927/1986: 39)

Abstract

Currently, autism is a widespread and diverse neurodevelopmental disorder that includes both severely impaired and institutionalized patients and the fairly geeky but brilliant university professor. Despite its heterogeneity, autism is often presented as a distinct nosological entity with a unifying autism essence. This chapter argues that the common belief about the ontological status of autism is that autism constitutes a *natural kind*. There are, however, two major difficulties with a natural kind approach in autism research. First, how can we continue to speak about autism as a distinct disease while the condition is marked by such a sheer diversity of symptoms, traits, biological markers and cognitive profiles? And second, recent historical works on autism illustrate that there is something fundamentally social and historical about how autism is defined, diagnosed and treated. I argue that the dominant natural kind approach in autism research is problematic, as autism can only be understood in relation to ideas about what kind of behavior is deviant and in need of correction or

⁴ This chapter has been published as Verhoeff B (2012) What is this thing called autism? A critical analysis of the tenacious search for autism's essence. *Biosocieties* 7(4): 410-432.

support. Furthermore, locating and maintaining autism within the biological realm of the individual obscures an array of social, cultural and psychological issues in understanding the contemporary phenomenon we call autism.

Introduction

Autism has many faces. Besides autistic disorder, we are now familiar with Asperger's syndrome and 'high functioning' autism, currently all united under the banner of the autism spectrum. As autism researchers and clinicians increasingly highlight the dimensional nature and complex behavioral, biological and neurocognitive heterogeneity of autism (see, for example, Happé and Ronald, 2008), autism has become a broad and diverse disorder covering both completely mute and unresponsive individuals, and socially awkward geniuses. At the same time, despite battles over its causes and how it should be classified in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*,⁵ the idea of autism as a distinct nosological entity with a particular essential core deficit remains pervasive. Many influential clinicians and autism researchers emphasize the recognizability and distinctiveness of autism: '[T]here is little dispute that in many ways autism is one of the better examples of a "disorder" that is distinctive and does not just shade off into normalcy' (Volkmar, 1998: 45) and 'Autism is a recognizable entity' (Frith, 1989: 15). Leo Kanner himself, autism's intellectual father, was also convinced that he delineated a distinct entity: '[W]e can state unreservedly that, whether or not autism is viewed as a member of the species schizophrenia, it does represent a "definitely distinguishable disease." This disease, specific – that is, unique, unduplicated – in its manifestations, can be explored per se' (Kanner, 1965: 418).

In this chapter, I illustrate that in the expanding field of autism research, the common – if sometimes tacit – belief about the ontological status of autism is that autism constitutes a *natural kind*. In spite of the disputable philosophical status of the very notion of a natural kind (see Hacking, 2007a), a natural kind perspective matters in scientific practice. Natural kinds make good objects of

⁵ *DSM-5* was published in 2013 by the American Psychiatric Association, after the publication of this article in *Biosocieties*. Different Work Groups, consisting of approximately ten to fifteen experts in a particular field, were responsible for formulating specific criteria for each disorder.

scientific discovery, are related with the search for mechanistic explanations, and allow for inductive generalizations.⁶ Current autism research fits this scientific image strikingly well. It is guided and regulated by the depiction of autism as a *bona fide* scientific and physical object that can be discovered and identified with systematic biomedical and neuroscientific investigation. Autism is imagined to exist as an objective entity independent of its embodiment in particular individuals. Conceptualized in such a way, the idea of autism as a distinct disease with a specific etiology, pathophysiology, course and treatment seems inescapable.

However, I argue that a natural kind approach in autism research is thorny in at least two ways. First, is a problem widely acknowledged among autism researchers and clinicians, that is, the issue of heterogeneity: how can we continue to speak about autism as a distinct disease while the condition is marked by such sheer diversity of symptoms, traits, biological markers and cognitive profiles? Natural kinds⁷ – classic examples being *water* and *gold* – typically refer to classes of things that are homogeneous and have clear boundaries. Moreover, the individual members of a natural kind must share some underlying structure or property that characterizes the kind in all possible cultures, historical periods and worlds in which it could exist (Dupré, 1981). Natural kinds seem to carve up the world at naturally privileged joints. However, despite an impressive list of genes, anatomical abnormalities and other biological markers associated with autism (see, for example, Anagnostou and Taylor, 2011; Lichtenstein et al., 2010), a diagnosis of autism is still based

⁶ Inductive generalizations are ‘formulated on the basis of observed instances of a category and yet purport to license inferences about unobserved instances as well’ (Samuels, 2009: 51). On observing 100 glasses of water that all boil at 100° Celsius it is reasonable to expect that all glasses of water boil at 100° Celsius. Such inferences about unobserved characteristics are reliable if the observed characteristics of a category covary not by accident, but because of some common factor that explains the co-variation of important characteristics. This is part of what defines a natural kind: that the members share a similar structure that determines a number of properties equally shared by the members of the natural kind.

⁷ Different accounts of natural kindhood have been associated with many different classes of things, such as chemical elements and biological species. Some accounts are very stringent and others are more permissive (see Boyd, 1991), but it is not my intention to get drawn into metaphysical disputes about what exactly defines a natural kind. I will use the term rather loosely to illustrate how a certain ontological assumption shapes and regulates scientific practices.

on behavioral characteristics and there is a broad consensus that unlike in Down syndrome or Huntington's chorea, people with autism do not possess a unitary and essential causal property that determines all observable characteristics of autism (Happé et al., 2006).

An obvious question that arises is how the idea of autism as a distinct natural entity remains pervasive while empirical evidence against an essential structure underlying autism accumulates? An answer to this question might be found by analyzing social and political forces that locate and maintain autism within the biological realm of the individual rather than between the individual and the requirements of the environment. Social and political structures involved in the production of suffering and disability remain unchallenged as long as autism is a biological thing to be identified with microscopes and MRI scans. Notwithstanding the relevance and potential explanatory value of 'constructionist accounts in terms of medical imperialism, medicalization of social problems, energetic proselytising by parents and support groups, the egregious power of the drug companies with their disease awareness campaigns ...' (Rose, 2009: 79), it will not be the primary theme of this chapter.

Rather, in the first out of two parts of this chapter, I discuss the way in which some central developments and discussions in the field of autism are both conditional upon and further legitimize the particular understanding of autism as a reputable natural entity. After that, the way in which the supposed essence of autism is currently conceptualized will be discussed and I give an account of the actual strategies by which autism researchers attempt to deal with autism's heterogeneity. These strategies are related to different research programs and some of the debates around the proposed changes to the autism category in *DSM-5*.

A second difficulty with a natural kind approach in autism research emerges with a more philosophical, historical and sociological viewpoint on autism. In the growing field of critical autism studies there are extensive discussions about defining autism as a disease (Ortega, 2009), and about the emergence of autistic cultures, subjectivities and (self-)advocacy movements (Silverman, 2008). In particular, some recent historical works on autism by Nadesan (2005), Eyal et al. (2010) and Silverman (2011) forcefully illustrate that there is something fundamentally social and historical about how autism is defined, diagnosed and treated. They all argue that autism's emergence, historical transformations and fluctuating boundaries reflect social, cultural and political processes. Drawing

amply on Ian Hacking's notion of 'interactive kinds,' Eyal et al. (2010: 209) for example argue that 'classifying and naming autism set in motion processes that act on the phenomena classified and possibly change them in ways that, in turn, react back on the classification, leading to its revision'.

With a particular emphasis on Hacking's notion of an interactive kind, the second part of this chapter explores the extent to which autism's historical variability threatens a natural kind approach in autism research. Despite the importance that the idea of an interactive kind may have for explaining historical changeability of autism and for drawing attention to the power and social effects of labeling humans, I suggest that this idea will not suffice to criticize a dominant natural kind approach in autism research. Nevertheless, supported by more historical, socio-cultural and philosophical analysis, I argue that autism can only be understood in relation to ideas about what kind of behavior is unacceptable, deviant, and in need of correction or support. Autism cannot avoid being related to a cultural norm of a social, empathic and engaged individual, and any account of autism begins with a need to demarcate, locate and treat particular discontents and impairments that have appeared. Therefore, the idea of an essential core or a natural autism entity that is waiting to be identified is misguided, and the corresponding dominant neuroscientific approach to autism obscures an array of social, cultural and psychological issues important in understanding the phenomenon we call autism.

The analysis in this chapter is based largely on texts. Manuals, articles, reviews and books from multiple disciplines provide the foundation of the arguments. I also conducted interviews with some of the members and one ex-member – Fred Volkmar – of the *DSM-5* neurodevelopmental disorders (ND) Work Group that was responsible for the formation of criteria and diagnostic categories of autism and related disorders.⁸ The interviewees were selected on the basis of their expertise and authority regarding the topic of a subsequent article that will specifically cover the emergence of the *autism spectrum disorder* (ASD) category and the disappearance of Asperger's disorder as a specific disease entity in *DSM-5*. Thus, whilst this chapter does not explore these interviews in depth, I will occasionally use quotes from these interviews to illustrate some of my arguments.

⁸ See: <http://www.dsm5.org/MeetUs/Pages/Neurodevelopmental%20Disorders.aspx>, accessed 10 April 2012.

Part I: A natural entity

An autism epidemic?

In 2009, the Center for Disease Control and Prevention (CDC, 2009) published a study that estimated that the prevalence of autism in the United States had increased from 4 per 10,000 in 1989, to 66 per 10,000 in 2002, to 90 per 10,000 in 2006. That is from 1 in 2,500 children to 1 in 110 children in less than twenty years. The CDC added that other recent population based studies documented even higher prevalence rates of autism of >1 per cent of children in areas of Japan, Sweden, the United Kingdom and the United States (for example, Baird et al., 2006; Kogan et al., 2009). The substantial and steady rise in children diagnosed with autism has been of scientific concern. Multiple review articles and meta-analyses have tried to explain what caused this tremendous increase in people diagnosed with autism (see, for example, Fombonne, 2009; Waterhouse, 2008; Wing and Potter, 2002).

At this point, I am not particularly concerned with the specific explanations given for this rise nor with adding a new one, but deliberation on the potential increase of autism requires a certain notion of autism. Although different autism researchers might slightly differ in the explanations they give for the rise in autism cases, there is a striking similarity in the way the problem is posed and possible explanations are presented. The articles by Eric Fombonne, psychiatrist and autism epidemiologist, are exemplary. He reframes the problem of the rise in autism cases into a deceptively simple question with two possible outcomes. Either there is a true increase in the incidence of autism, or there is no true increase in the incidence of autism and only an increase in recognition of it.

Fombonne (2005: 292) argues that while the rates of people diagnosed with autism have gone up, ‘this increase most likely represents changes in the concepts, definitions, service availability and awareness of autistic-spectrum disorders in both the lay and professional public’. He concludes that current evidence ‘does not support the hypothesis of a secular increase in the incidence of autism’ (ibid.). Fombonne’s idea of a secular or true increase in autism assumes an idea of a true autism not at the level of concepts, definitions or diagnostic criteria, but at some ‘underlying’ material level. Concepts, definitions and criteria of autism may change, but true autism is a stable and material, but still largely unidentified, disease. Francesca Happé – prominent autism

researcher and member of the *DSM-5* ND Work Group – makes a similar argument: ‘there has been a big change in diagnostic criteria, a broadening, the introduction of Asperger’s syndrome, a widening of understanding, better diagnostic services, better services to follow diagnosis ... whether they are sufficient to explain the [rising] numbers or whether there is a real increase, I think nobody knows’.⁹

Current hypotheses about such a real increase in autism point at several causal factors including antibiotics, environmental pollutants, *de novo* genetic mutations, preservatives in vaccines and other neurotoxins.¹⁰ While none of these factors have been affirmed as a cause for the increase in autism rates, they are among the possible candidates for causing a ‘genuine’ autism increase. ‘A true risk’, Michael Rutter (2005: 2) argues, ‘due to some, as yet to be identified, environmental risk factor cannot be ruled out’. On this view, changing ideas about autism cannot explain a true increase in autism, for the reason that autism is imagined as an entity (or multiple entities) existing in individuals prior to and independent of our ideas about it.

A critique of thinking about autism as a natural entity does not imply that the rising number of people diagnosed could not for instance be caused by environmental pollution or *de novo* genetic mutations. In addition, claims and worries about an autism ‘epidemic’ do not depend exclusively on conceiving of autism as a natural phenomenon. However, the assumption of autism as a natural entity limits the scope of explanations, solutions and interventions in discussions and research on the possibility of an autism epidemic. Social and cultural forces, that are discussed in the second part of this chapter, such as shifting social norms and new ways of engaging with suffering and diversity remain out of sight in a narrow investigation of a ‘true’ increase in autism in biological terms. A good example of a sociological analysis of the more general rise in psychiatric disease incidences is given by Rose (2006), who discusses contemporary ‘demands for disease recognition, moves to screening, fears of risk and hopes for prevention’ as forces related to lower thresholds ‘at which individuals are defined, and define themselves, as suitable cases for treatment’ (p. 481). Such an analysis does not deny an involvement of biological or genetic

⁹ From an interview with Francesca Happé in London on 14 December 2010.

¹⁰ See for the controversial but extremely influential MMR vaccine hypothesis in the retracted article by Wakefield et al. (1998).

factors, but broadens the perspective on current rising numbers of a variety of diseases from hypertension to obesity and ADHD.

Retrospective diagnosing

Another indication of the natural kind assumption in autism research comes from the currently popular practice of retrospective diagnosing (see also Chapter 3). While the first official case descriptions of autism date back to Leo Kanner's 1943 study, many autism experts have been occupied with demonstrating that there are striking cases of autism in history before Kanner introduced the syndrome. Uta Frith, another leading autism researcher, describes various cases, of which the case of Victor, 'the wild boy from Aveyron' is among the most prominent. At the turn of the nineteenth century, Victor was found living wild in the woods in South-Central France. The French physician Itard published detailed accounts of his behavior. Frith finds in Itard's early accounts clear evidence for serious impairment in reciprocal social interactions and stereotypical behavior. She assumes that Victor must have been autistic and concludes that 'autism is not a modern phenomenon, even though it has only been recognized in modern times' (Frith, 1989: 16).

Other historical accounts of feral children, eccentric geniuses and religious figures have proven productive for retrospectively diagnosing autism. To name a few, there are *The Blessed Fools of Old Russia* (Challis and Dewey, 1974), the extraordinary case of Hugh Blair of Borgue in eighteenth-century Scotland (Houston and Frith, 2000) and – according to Michael Fitzgerald's (2005) endless list – Isaac Newton, Ludwig Wittgenstein and Albert Einstein.¹¹ These are just a few examples that for many provide convincing evidence that autism is indeed not a contemporary phenomenon, but something that can be recognized in different cultural and historical contexts. Frith, Fitzgerald and others explicitly distinguish the existence of labels from the existence of distinct diseases. According to Frith, retrospective diagnosing helps to 'distil those features that are the essence of the disorder beyond our immediate time and cultural context' (Frith, 1989: 17). She tries to find what she calls 'the unchanging core of autism' (Houston and Frith, 2000: 4). It is this assumed transhistorical, essential core of autism that is being recognized in the many

¹¹ Michael Fitzgerald, a Professor of Child Psychiatry, just about specializes in diagnosing historical legends with autism. He argues for a link between autism and a kind of pure and innate artistic and philosophical brilliance (2005).

recent examples of peculiar historical figures, and these examples strengthen – for autism experts and authorities, but also for clinicians, lay people, patients and their families – the idea of autism as a natural entity. Though, as we shall see, what this actually means is far from settled.

Autism as a brain disorder

Recent articles and reviews on autism usually start with stating that autism is a ‘neuropsychiatric condition,’ a ‘brain disorder’ or a ‘neurodevelopmental disorder’ (see also Chapter 4 and, for example, Happé et al., 2006; Volkmar and Pauls, 2003). Autism is considered to be ‘among the most heritable of all mental disorders’ (Lichtenstein et al., 2010): recent reviews estimate the heritability of autism to be more than 90 percent (Freitag et al., 2010; Losh et al., 2008). The emphasis on genetics, brain activity and neurotransmitters is not specific to autism but part of a broader neuroscientific shift in psychiatry that hopes – amongst other things – to carve psychiatric classifications at their ‘natural joints’ (Hyman, 2007). Social, cultural and historical conditions of possibility for this neuroscientific shift have been explored by theorists in great detail (see, for example, Rose, 2007), but fall outside the scope of this chapter.

Against earlier psychogenic theories of autism, of which the ‘refrigerator mother’ theory¹² commonly attributed to Bruno Bettelheim (1967) is among the most remarkable, the biological nature of autism is currently treated as a given starting point for solving ‘the autism puzzle’ (Schaaf and Zoghbi, 2011). Thinking about autism is far removed from unconscious desires of mothers, family dynamics and defense mechanisms of the child. Notwithstanding an increasing interest in ‘gene-environment interactions’ and epigenetics (Rutter, 2011), what there is to be explained can and should ultimately be explained at the material and mechanical level of the brain in terms of genetic polymorphisms and synaptic connectivity. This biological depiction of autism goes beyond arguing that the etiology of autism involves biological factors. Indeed, it is not only about what causes autism, it is about what autism

¹² The popularization of this term ‘refrigerator mother’ is often attributed to Bruno Bettelheim and his widely read book *The Empty Fortress* (Bettelheim, 1967). The psychoanalytically founded claim in this book is that the precipitating factor for autism is the unconscious wish, of particularly the mother, that the child should not exist (see Chapter 5). It were, however, Kanner (1949) and Eisenberg and Kanner (1956) who originally mentioned ‘a frosty atmosphere’ and ‘emotional refrigeration’ in relation to the environment and the parents of their autistic patients.

fundamentally is: a disorder of the brain, identifiable and discoverable in this particular organ at the biological and mechanical level commonly studied by the biomedical and neurosciences. The self-evident supposition of autism as a brain disorder is another facet in thinking about autism as a natural entity.

Autism today

Ubiquitous heterogeneity

What does this natural entity called autism look like? As I already mentioned in the introduction, present-day autism researchers and clinicians increasingly emphasize the heterogeneity of autism. Lord and Jones (2012: 491) recently stated that ‘the most significant scientific challenge to the concept of autism as one “disease” or even “diseases” is the heterogeneity of the genetic findings’, and, in addition, Lord (2011: 166) pointed out that ‘anyone who has met more than one person with an ASD is stuck by the differences between these individuals’. *DSM-IV* (APA, 1994) diagnostic criteria for autism are based on the characteristic autism triad of impairments that consists of impairment in social interaction, communication and impairment due to restricted behavioral patterns. With the introduction of the idea of a broad autism spectrum, Wing and Gould (1979) already highlighted that this characteristic ‘triad of autism impairments’ could occur at varying levels of severity in different individuals with autism.

Impairment in social interaction, for instance, can be expressed as a total avoidance of social interaction, or it can be expressed as a lack of understanding of social conventions and cues in a person who does show interest in other people and friendships. Impairments in communication can range from a complete lack of speech to fluent speech with particularities in intonation, pitch and rhythm, and restricted interests and repetitive behavior can be as diverse as spinning a coin for hours, or having an obsessive interest in train tables or fantasy books. Apart from the possible variety in visible signs and symptoms, the course of autism also varies from children who seem to develop normal for two years and then show a decline, to children who show an abnormal development from birth on. Some children improve with age, others have a stable course and some get worse (Willemsen-Swinkels and Buitelaar, 2002). Furthermore, autism has been associated with numerous other conditions such

as epilepsy, intellectual disabilities, sleep disorders, disruptive behaviors, anxiety, depression, hyperactivity and attention difficulties.

In their article with the telling title ‘Time to give up on a single explanation for autism’, Happé and colleagues (2006) not only argue that there is no single cause for all the core features of autism, but they also present empirical data – contra Wing – of behavioral fractionation of the communication impairment, the social impairment and the rigid and repetitive behaviors. In a population-based study, they found ‘modest-to-low correlations between autistic-like behavioral traits in the three core areas’ (ibid.: 1218). The clustering of the three symptom domains turned out to be not as strong as was previously thought, and many children only have symptoms – in varying levels of severity – from one or two symptom domains. This finding adds another level of heterogeneity to the spectrum idea of autism, now that there is variation and fractionation along at least three partly independent dimensions of impairment.

Happé and colleagues furthermore claim that it is useless to search for a monolithic genetic or neuroanatomical explanation for the three core aspects of autism as a whole. Family and twin studies of the genetic structure of the triad of impairments and neuroimaging research all suggest an equal fractionation of underlying genetic abnormalities and neural substrates. For future research, they recommend neuroimaging and molecular genetic studies to focus on specific symptom domains, instead of the so far unsuccessful ‘search for genes [and neural substrates] “for autism” as a whole’ (2006: 1219).

Have genetic studies indeed been unsuccessful in their search for autism genes? The first advances in identifying genes associated with autism came from studying syndromic autism (autism in combination with other syndromes that cause congenital malformations or dysmorphic features). Identified single-gene syndromes, such as fragile X syndrome, PTEN macrocephaly syndrome, Rett syndrome and tuberous sclerosis, are assumed to account for 5 to 7 percent of all autism cases (Miles, 2011). Another 5 percent of autism cases have been associated with genetic metabolic disorders, such as phenylketonuria, creatine deficiency and mitochondrial disorders. For the remaining – nonsyndromic – cases, recent whole genome studies on autism (Levy et al., 2011; Sanders et al., 2011) further affirmed the genetic heterogeneity in autism. These studies argue that there are numerous genetic mutations – *de novo* and transmitted – associated with autism that are extremely rare. The number of genes associated with autism may be a couple of hundreds or more, of which

the most common mutations were found in just over 1 percent of the children with autism, and not exclusively in children with autism (Schaaf and Zoghbi, 2011).

Autism's unifying essence

With this amount of diversity at clinical and biological levels, and with the absence of a clear boundary between autism and normality, what is it that unifies all the cases of autism other than the diagnostic criteria? In what sense and at what level is autism still a distinct natural entity? For many prominent autism experts, autism remains to be understood as a highly recognizable disease entity with a unifying fundamental nature. Happé and Ronald (2008: 299) for instance argue that their idea of the fractionable triad is not an attack on the validity of the diagnosis of autism since 'it is quite compatible to assert that ASD results when a number of independent impairments co-occur, and to assert that the resulting mix has a special quality, distinct prognosis and response to intervention'.

In a paper on classifying autism in the *DSM-5*, Wing, Gould, and Gillberg (2011: 769) state that 'the fundamental problems underlying all autistic conditions and the Triad of Impairments, is absence or impairment of the social instinct present from birth ... [w]e hope that research work into the behavioral neurology of the social instinct will be carried out in the near future'. Happé adds to this that '[autism] is one of the most recognizable syndromes. It is a clearly recognizable syndrome when you see it ... the receptionist who works in the clinic spots the autism as they come in the door'.¹³ Its recognizability, she argues, lies in the overlapping essential deficit in social cognition: '[autism] is a disorder of social cognition ... social problems are the real core'. On this issue Susan Swedo – chair of the *DSM-5* ND Work Group – argues similarly: 'yes, there is something that is essentially autism and the essential is a delay or failure of the development of social cognition ... autism is a core deficit, it is a pathway that did not develop'.¹⁴

Various distinct cognitive theories have been suggested as fundamental in comprehending all the problems and impairments people with autism are faced with. A defective theory of mind (Baron-Cohen, 1995), weak central coherence (Happé and Frith, 2006), impaired executive functioning (Hill, 2004), and

¹³ From an interview with Francesca Happé in London on 14 December 2010.

¹⁴ From an interview with Susan Swedo in New York on 29 October 2010.

dominant systemizing capacities compared to empathizing capacities (Baron-Cohen, 2002) are the familiar cognitive theories that have all been proposed as the essential uniform defect in autism. However, none of these theories have succeeded in explaining all the problems in autism. Happé and colleagues (Happé et al., 2006: 1219) admit the ‘failure to find a single cognitive account for the three core features of autism’. In a recent review, Chevallier et al. (2012) have a new attempt by proposing ‘the social motivation theory of autism’. They argue that autism can be seen as an ‘extreme case of early-onset diminished social motivation’ that explains a range of autism characteristics, ‘including cascading effects on the development of mature social cognitive skills’ (p. 237). Furthermore, they argue that ‘these deficits appear to be rooted in biological disruptions of the orbitofrontal-striatal-amygdala circuitry, as well as in dysregulation of certain neuropeptides and neurotransmitters’ (ibid.).

For many cognitive psychologists, clinicians and neuroscientists researching social cognition, and – as we will see in the next section – to a lesser degree for molecular geneticists, an essential nature beneath the diversity of apparent signs and symptoms is still assumed to unite the multiplicity of autism. Expressed in terms of a deficit in social cognition, social intuition or social motivation, and imagined somewhere in the brain, identifying autism is widely presented as a complex scientific challenge, often metaphorically referred to as ‘the autism puzzle’ or ‘the autism enigma’.¹⁵ This rhetoric of puzzles and enigmas is reliant upon the idea of autism as one or more distinct disorders that can be discovered, but it also has a number of other effects: it is effective in soliciting funding and broad public support, it serves as a magnet for researchers invested in resolving mysteries, and legitimizes the already highly specialized and costly searches taking place at increasingly complex neurobiological levels. As the assumed natural entity of autism cannot be identified at a concrete genetic or neuroanatomical level, the attempts to reveal autism’s mystery continue at even more minute and detailed molecular and epigenetic levels (Rutter, 2011). Preliminary ideas about how this natural entity is envisioned and how this relates to attempts of autism researchers to deal with heterogeneity issues are discussed in the next section.

¹⁵ A recent special issue of *Nature* called ‘The autism enigma’ attempts to sort fact from fiction in autism research (*Nature* 2011, volume 479).

Dealing with variety and autism in *DSM-5*

Lumping

Attempts to deal with heterogeneity and to identify the essential underlying nature of autism can be roughly divided into two familiar types of strategies: *lumping* and *splitting* (see McKusick, 1969). Lumping refers to the attempt to identify a single unifying underlying process or structure that offers an all-encompassing account of autism. The numerous and diverse genetic variations and associated neuroanatomical structures in different autism patients obviously pose a problem for a lumping strategy. However, a possible solution is provided by the idea of a more homogeneous mechanism or structure at a different level – sometimes referred to as a ‘final common pathway’ – that connects multiple distinct etiological and pathological processes with overt behavioral autism characteristics.

Geschwind and Levitt (2007: 103) suggest a potential unifying model for autism in which particular areas of the brain that normally connect to the frontal lobe are partially disconnected during development. Their concept of developmental disconnection, they argue, ‘can accommodate the specific neurobehavioral features that are observed in autism, their emergence during development, and the heterogeneity of autism etiology, behaviors and cognition’. More specifically, recent genome studies by Gilman et al. (2011) and Levy et al. (2011) suggest that the many different genes associated with autism could play a functional role, via the molecules they express, in a larger biological network that is related to the regulation and formation of synapses and neural development. Corresponding to a more general network-based approach to human diseases (see Barabasi et al., 2011), this larger biological network responsible for neuronal motility, axon guidance and synaptogenesis, could be disturbed by many different molecules expressed by different genes, but leading to similar deficits of neural development. These specific deficits in neural development are in turn thought to underlie the typical symptoms identified as autistic. In a similar vein, Sakai et al. (2011) developed a protein interaction network that ‘provides a framework for identifying causes of idiopathic autism and for understanding molecular pathways that underpin both syndromic and idiopathic ASDs’, and Peça and Feng (2012) illustrate how multiple families of genes involved in tuberous sclerosis, fragile X syndrome, Angelman syndrome and non-syndromic ASD, are functionally united by common cellular pathways

involved in the organization of glutamatergic synapses. Hence, a common neurodevelopmental abnormality at a complex biological level could still be the glue that holds the overall idea of autism together.

Nevertheless, a lumping strategy still needs to deal with the diversity of clinical presentations of autism. What makes it reasonable to assume that the diverse manifestations are the result of a common underlying problem? The *DSM-5* ND Work Group clearly opts for a lumping approach in the proposal to fold Asperger's disorder into a single ASD category.¹⁶ As stated by one of its prominent members, the aim in proposing the new ASD category is to 'recognize the *essential* shared features of the autism spectrum' (Happé, 2011: 541, emphasis added). At the same time the Work Group members recognize a 'vast heterogeneity within this spectrum' (ibid.: 540). They attempt to deal with it by specifying individual levels of symptom severity and by assigning nonspecific dimensional descriptors (co-existing conditions), such as intellectual or language difficulties, mood disorders and motor or sleep problems, to an ASD diagnosis.

Although core deficits – in social cognition and intuition – unite all ASD patients, their diagnosis will be individualized by describing overall levels of impairment from co-existing difficulties to account for the unique problems of each ASD patient. Particularly, levels of intellectual functioning and language competence are considered to have a major influence on autism's manifestations and on clinical decision-making. Lord and Jones (2012: 493) affirm that the 'level of expressive language is a crucial dimension in the diagnosis of ASD that needs to be considered separately in its own right, and as it affects ASD symptoms'. Other commonly associated conditions like hyperactivity, aggression, eating abnormalities, anxiety and sleep disorders, are equally not part of the core deficits in autism, but are thought to have a huge impact on how autism develops, impairs and is expressed. Therefore, these conditions are able to account for much of the heterogeneity seen in ASD. The idea of *dimensions*, both within the autism core in terms of severity of symptoms and as referring to co-existing conditions and impairments, is crucial to the reorganization of autism in *DSM-5*. It is able to unite extreme clinical heterogeneity with the idea of one single all-encompassing autism category.

¹⁶ See: <http://www.dsm5.org/proposedrevision/Pages/Default.aspx>, accessed 10 April 2012.

Splitting

Splitting autism into subgroups is another approach that attempts to deal with the problem of heterogeneity. Folstein (2006), for instance, argues for two ‘true’ autism phenotypes, Kanner’s autism and Asperger’s disorder. She regards Kanner’s autism as characterized by a child’s severe inability to relate himself to other people and situations, language deficits and an intolerance of change of any type, such as changes in a certain routine or furniture arrangement. Asperger’s disorder is a (genetically) related milder variant, but Folstein argues that both autism diagnoses should exclude a number of other conditions. Children with profound mental retardation, children with dysmorphic facial features or specific genetic conditions, such as tuberous sclerosis or Rett syndrome and children who have suffered certain kinds of severe encephalitis at an early age should be excluded from the two autism categories. Folstein argues that while these children might exhibit autism-like behavior, they are etiologically very heterogeneous and need to be considered separately from autism in neurobiological studies.

Waterhouse (2008: 283) suggests that we should accept that all the behavioral and biological variation in autism cannot be encompassed by any single theory and there ‘must be phenotypic and genotypic subgroups that have not yet been discovered’. This idea of multiple autism subgroups has led geneticist to a research approach that attempts to identify genes and regions on the genome that are associated with more homogeneous subgroups within the autism spectrum. By splitting the autism spectrum into smaller phenotypes, for instance of children with autism who also suffer from epilepsy, language delay, or extreme resistance to change, geneticists were able to identify new regions on the genome associated with only a subgroup of autism cases (see, for example, Molloy et al., 2005; Abrahams and Geschwind, 2008). However, apart from some single-gene syndromes such as fragile X and Rett syndrome, no convincing and meaningful subgroups have been identified so far. Furthermore, it is still largely unclear how the earlier mentioned whole genome studies (Levy et al., 2011; Sanders et al., 2011), that suggest the involvement of hundreds of rare genetic mutations, relate to the breakup of the autism spectrum into distinct (genetic) disorders. Future ASD research, Geschwind (2011) argues, should focus on identifying subtypes of ASD based on clusters of (interacting) genes and converging molecular pathways that relate to specific deficits in brain circuits that in turn relate to distinct autism phenotypes.

For (molecular) geneticists and neuroscientists, functional genomics, epigenetics and systems biology are among the new hopes in a new phase of solving the autism puzzle. Whether they are more committed to the idea of multiple disease entities (splitting) or to the idea of one broad autism entity (lumping), they all share a faith in discovering objective disease categories. The idea of splitting up autism into a number of genetically or neurobiologically defined disorders is not in conflict with a natural kind approach in autism research, as it is equally driven by the idea of discovering and delineating unified natural entities by means of systematic neuroscientific investigation. However, in contrast with most cognitive psychologists, neuroscientists researching social cognition and clinicians, (molecular) geneticists are generally not that much committed to the idea of autism's unifying deficit in social cognition or intuition. Since their focus is on genes, molecules and neural circuits in defining and categorizing distinct disorders that underlie the superficial autism features, the idea of a unifying essential deficit in social cognition becomes less urgent. But, as I have mentioned above, how autism should be split up on the basis of neurobiological data, what the corresponding (autism) phenotypes will be and how they might be related to each other is still largely undetermined (see Geschwind, 2011; Berg and Geschwind, 2012).

DSM-5 debates

For as long as autism is not yet carved at its natural joints and the search for distinct biological mechanisms continues, the behavior-based DSM classification system remains the main point of departure both for clinical practice and for fundamental neurobiological research. The recent proposal to create one ASD category for the *DSM-5* has aroused heated debates and high emotions. Particularly the incorporation of Asperger's disorder into ASD has encountered forceful resistance from psychiatrists, researchers, patients, patients associations and other stakeholders (see, for example, Ghaziuddin, 2011). However, the Work Group argues that 'there is little evidence to support the current diagnostic distinction between Asperger disorder and high-functioning autism' (Happé, 2011: 541).

Among the opponents of the ND Work Group's decisions is autism expert Fred Volkmar. He resigned from the Work Group in 2009 based on his objections to the single ASD category and on his worry that less children will qualify for extra support and therapeutic services. Volkmar, supporting a

splitting approach, argues that Asperger's disorder should be in *DSM-5* as a separate category 'both for research and clinical purposes'. He suggests that 'there is a big difference with autism ... [Asperger's children] have different needs, they are more verbal and look for social relationships, you can use that as a medium for intervention, and they often have a different profile of strengths and weaknesses'.¹⁷ From a research perspective the Asperger's disorder category is important because of 'the potential differences from autism in neuropsychology, and I also think genetics is probably stronger in Asperger's'. The problem with *DSM-5* is that 'they get rid of all the distinctions [...] If you could really find a homogeneous subgroup, you can find genes, but if you just mess them all up as a spectrum we are not able to do that'. Furthermore, Volkmar worries that 'a lot of the people with Asperger's disorder will probably lose their diagnosis altogether [...] because if you look at the proposed criteria, they have put up the threshold. That is one of those funny things, you think spectrum means broader, but it is actually narrower'.¹⁸

As a senior author, Volkmar recently backed up his earlier worries in a provocative study (McPartland et al., 2012) that mapped the new *DSM-5* criteria for ASD onto a dataset of child and adult patients collected more than 20 years ago for the field trial of *DSM-IV*. They conclude that almost 40 percent of individuals currently diagnosed with an ASD, mostly individuals with Asperger's disorder and PDD-NOS, will no longer meet criteria for an ASD in *DSM-5*. The proposed changes, the authors predict, 'could exert detrimental effects on service eligibility' (p. 382). These alarming conclusions generated substantial media attention and parental concerns with *DSM-5*, because of the fear that a significant proportion of individuals with a current autism diagnosis will be excluded from medical, educational and social services.

In a commentary in the same journal issue, the entire ND Work Group fires back and argues that the McPartland et al. study justifies 'neither alarming headlines nor dramatic conclusions' (Swedo et al., 2012: 347). So far, the Work Group has been careful to stress that 'no individual currently diagnosed with Asperger's disorder or PDD-NOS who needs support will lose that support' (Happé, 2011: 541), and they believe the data used in McPartland's analysis 'have too many inherent limitations to assess definitely the criteria proposed ... and do not support such dramatic conclusions' (Swedo et al., 2012: 347). The

¹⁷ From an interview with Fred Volkmar in New Haven CT 27 October 2010.

¹⁸ Ibid.

issue of service eligibility illustrates a certain tension that arises with the production of a diagnostic manual intended to serve a whole range of different purposes that extend beyond structuring research and clinical practice. The very sensitive topic of eligibility for therapeutic services and support is one important aspect in redefining autism that intermingles with the scientific interest of identifying distinct biological mechanisms. As we will see in the second part of this chapter, such contextual issues pose profound difficulties for a natural kind approach in autism research.

Part II: Historical variability

Recent social studies on the emergence and development of autism suggest that there is something fundamentally social and historically variable about how autism is defined, treated and diagnosed. Silverman (2011: 29) argues that although practitioners may maintain that they have gained the ability to recognize autism on sight in a similar way as one might learn to recognize the distinctive style of a particular artist, ‘much about the diagnostic criteria, practices of identification, modes of treatment, and daily experience of autism has changed, and changed radically’. Indeed, in Kanner’s days, autism was a very rare disorder characterized by a child’s extreme withdrawal and remoteness from affective and communicative contact with other people (Kanner, 1949). These days, as we have seen, autism is a widespread neurodevelopmental disorder with a variety of social and behavioral impairments.

In *The Autism Matrix*, Eyal et al. (2010) explain the historical changes in autism and the exponential rise in the number of autism diagnoses in terms of a process of deinstitutionalization of mental retardation in the mid-1970s, parental activism, a greater availability of services from 1991 onwards ‘when autism was added to the Individuals with Disabilities in Education Act’ (p. 19), and the reorganization of expertise. In a similar vein, Nadesan’s *Constructing Autism* (2005) explores the role of early-twentieth-century child guidance and mental hygiene movements, ‘which together brought childhood into focus as a legitimate sphere of psychiatric inquiry’ (p. 6). She provides a social history of the material institutions, professional identities and cultural values that enabled the emergence and transformation of autism as a psychiatric disorder.

These historical studies approach autism not as a distinct entity in nature that can be discovered, but as something mutable and produced by its socio-historical context. However, a natural kind approach cannot easily be discarded on the basis of autism's historical variability, as autism scientists could argue that human understandings of a (supposed) 'true' nature of autism might improve as science progresses. One might argue, as the retrospective diagnosticians do, that we just get better at recognizing what has always been there. Furthermore, any scientific discovery or progress requires particular social, cultural and institutional conditions of possibility. Probably, some highly 'social' history could be told about the discoveries of the periodic table elements. Nevertheless, I argue that the dominant natural kind approach in autism research is limited. This, however, requires further examination of the precise character of autism's historical variability.

Interactive kinds

In their explanation of the historical variability of autism, Nadesan (2005), Eyal et al. (2010) and Silverman (2011) draw on Ian Hacking's notion of 'interactive kinds'.¹⁹ Silverman (2011: 29) argues that diagnostic categories are interactive, mutable things, 'they make groups of subjects visible and distinct by describing them, but they then set them free to carry on their business, to resist, reshape, and reform that definition through their own actions'. Likewise, for Eyal et al. (2010: 23), the key concept for understanding historical changes in autism is 'looping':

Certain human conditions, says Hacking, are interactive in the sense that the very act of naming, classifying, diagnosing, and assigning them to treatment loops back to modify the condition thus named. [...] We suggest thinking of the autism epidemic, therefore, neither as a naturally

¹⁹ Much of Hacking's work (see Hacking, 2007b) attends to the way in which classifying and describing people brings about a feedback mechanism that changes the kinds of people under study. Individuals react to being classified and as a result of their behavioral changes, the very people and the classifications that are supposed to cover them go through a process of alteration. This feedback mechanism, Hacking argues, results in 'interactive kinds' having histories totally different from the histories of what he calls 'indifferent kinds'. Indifferent kinds, such as tigers and gold, are unaffected by how we classify them.

occurring event, nor as a socially constructed fiction, but as a final spiral in an increasingly widening vortex of looping processes.

Eyal and his colleagues convincingly illustrate this interactive process by arguing that as Leo Kanner and Bernard Rimland – another eminent first generation autism researcher – insisted on the rareness and distinctiveness of autism in the mid-1960s in order to destigmatize the condition, to defeat the by that time dominant psychogenic hypothesis and to remove the burden of guilt from parents, they also gave rise to an active parents' movement. This meant, they argue, 'that the diagnosis was becoming less rare, and in due course, less distinctive' (2010: 210). After that, as therapies emerged, what happened between autistics and therapies 'began to redefine what autism really was and what it was not' (ibid.). Therapies identified certain central behaviors and characteristics like head-banging, self injury and extreme social remoteness as treatable and therefore 'incidental to the syndrome, thus trimming away at its edges and blurring its boundaries' (ibid.). This, they argue, led to revisions of diagnostic criteria in subsequent DSM editions, and eventually completed the 'loop'.

Yet can we conclude on the basis of these interactive processes that autism cannot be understood as a discoverable entity in nature, or could it still be argued that these changes reflect a better understanding of what autism really is?²⁰ Or, as Cooper (2004) astutely points out in arguing that interactive kinds can still be natural kinds, could autism be changeable and still be a natural kind in the same sense that bacteria – given that they are natural kinds – mutate as they are affected by antibiotics? Changes in the behavior of people diagnosed with autism due to therapeutic processes or due to reactions on being classified do not contradict a natural kind approach. Although these behavioral changes might be evidence for social, cultural and psychological factors in affecting behavior, they are not evidence for a *necessary* dependence on social or cultural factors for the existence of autism as a distinct category, in a sense that the

²⁰ Contrary to what Eyal et al. seem to suggest, there are several ways to evaluate whether a particular cluster of symptoms makes a more valid category than another cluster of symptoms. Instead of only using an 'objective' marker 'in order to check their validity' (2010: 19), psychometric techniques such as 'confirmatory factor analysis' are used, next to more conventional 'validators' such as familial aggregation and precipitating factors. For a recent book on psychiatric taxonomy, see Kendler and Parnas (2012).

existence of the category of domestic animals necessarily depends on how we created it.²¹

Ian Hacking himself (1999; 2007b) has been struggling with autism and this issue of whether natural, or indifferent kinds – as he prefers to call them – and interactive kinds are mutually exclusive. On the one hand, Hacking represents autism as an interactive kind. The doctrine of the refrigerator mother had a severe impact on the family and the person diagnosed with autism and this doctrine and the subsequent changes in the family ‘contributed to a rethinking of what childhood autism is – not because one found out more about it, but because the behavior itself changed’ (Hacking, 1999: 115). A looping effect changed those diagnosed with autism ‘in such a way as to change the very concept of autism’ (Hacking, 2007b: 304).

On the other hand, Hacking leaves open the possibility of autism being – ‘in traditional jargon’ – a natural kind. With the identification of ‘one or more fundamental neurological or biochemical problems’ (Hacking, 1999: 116), then, ‘the more obvious it will seem that we are in the realm of indifferent, “natural” kinds’ (ibid.: 120). After all, for Hacking, it is obvious that ‘there were autistic children before Kanner singled them out’ and ‘if, as is widely supposed, autism is a congenital neurological deficit, then there were certainly autistic children who were dismissed as retarded, feeble-mind, and so on, a long previous litany of dismissive epithets’ (2007b: 304). For Hacking, autism’s interactiveness and historical variability eventually do not threaten a natural kind approach in autism research. As ‘many kinds of mental illness are interactive kinds, and yet are also indifferent kinds’ (1999: 119), autism can still be empirically discovered at fundamental neurological or biochemical levels.

To sum up, the notion of an interactive kind has proven to be a great conceptual tool for analyzing developments in the concept of autism. Particularly, in the works of Eyal et al. (2010), Nadesan (2005) and Silverman (2011), this notion facilitated valuable sociological insights in how ideas about autism emerged and changed and how autism became such a prevalent and much discussed condition. However, the idea of autism as an interactive kind is insufficient to criticize the dominant natural kind approach in autism research. Instead of highlighting autism as an interactive kind, I will now shed light on autism’s variability in a somewhat different way, and challenge Hacking’s hypothetical identification of autism as a natural (indifferent) kind. I argue that

²¹ For the entire argument, see Cooper (2004).

delineating autism and identifying autism at neurobiological levels are fundamentally reliant upon what is considered to be abnormal, harmful and impaired in a child's relation with the world, and thereby, fundamentally reliant upon where we draw the lines. In other words, the 'natural joints' at which autism researchers intent to carve up autism are reliant upon often very implicit commitments to normative expectations of individual children and their behaviors.

The undesirability of autism

As Nadesan (2005: 19) illustrates in her history of the emergence of autism, this event in the 1940s 'must be understood in relation to a matrix of professional and parental practices marking the cultural and economic transitions to the twentieth century'. The formalization of compulsory education and the creation of the child guidance movement led to increased forms of social surveillance over childhood, an increased public concern over 'deviant' children who posed a threat to social stability and an increased demand for more nuanced understandings of childhood pathology. Consequently, understandings of normality and pathology in mental health shifted and community clinics and special schools for children newly recognized as in need of psychiatric evaluation and support emerged. Autism could only emerge as a diagnostic category because, according to Nadesan (*ibid.*: 53), 'it was within these schools and clinics that a new cadre of experts ... encountered a class of children who escaped the increasingly narrow parameters of normality but whose apparent pathologies could not be satisfactorily explained by the extant psychiatric categories'.

Autism, as a problem of sociality, was able to fill the space that came into being by the increasing need to medically structure, treat and regulate childhood deviance. Nadesan's analysis provides an historical example of a widespread consensus among philosophers of psychiatry (see, for example, Bolton, 2008) that how the line between what is normal and what is pathological is drawn depends on social, cultural and individual values and circumstances. There is no plausible way in which this distinction can be made by referring only to biological or statistical measures (Canguilhem, 1966/1991). Even someone like Jerome Wakefield (1992), who is considered to be on the farthest naturalist side concerning mental disorders, acknowledges that a biological dysfunction needs

to be harmful in order to become a disorder, and harm cannot be understood independent of sociocultural circumstances.²²

The evaluative and contextual nature of pathology suggests that the kind ‘mental disorder’ as a whole does not constitute a natural kind, and a quick inference could be that the subclassification of mental disorder into specific mental disorders inherits this lack of natural kindhood. However, many scholars in the philosophy of psychiatry object to such an inference. In defense of a mechanistic model of psychiatric disorders, Kendler et al. (2011: 1147) argue that ‘psychiatric kinds are grounded in common features of the causal structure of the world, not merely imposed upon the world by psychiatrists through their classificatory practices’. Arguing for a natural kind approach to delusions, Samuels (2009) concludes that delusions do not have to be pathological. There is no necessary, but only a contingent connection between pathology on the one hand, and delusions on the other. He states that without some reason to suppose that this connection is a necessary one, the normativity of pathology does not pose a threat to the natural kind thesis in delusions. Cooper (2005: 76) uses an analogy to clarify this point:

...we should think of mental disorders in a way analogous to the way we think about weeds. Weeds are unwanted plants, thus whether a daisy is a weed is at least in part a value-judgement. Still, types of plant that are generally considered to be weeds – daisies, buttercups, stinging nettles – are natural kinds. Similarly, I argue that the claim that schizophrenia is a disorder is in part a value-judgement, but that it may well be the case that schizophrenia and depression are natural kinds.

For Cooper, Samuels and Kendler and colleagues, the general kind ‘mental disorder’ should be considered independent from kinds such as schizophrenia and autism. The pathological aspect of specific mental disorders is not seen as something inherent in psychiatric categories and as a consequence, the possibility of a natural kind approach is repossessed. From a different perspective, a similar position is taken by a group of autism self-advocates – the ‘neurodiversity’ movement – who ‘believe their condition is not a disease to be

²² Wakefield’s notion of *harmful dysfunction* has been influential in the philosophy of psychiatry, but also controversial since his idea of a value free biological dysfunction is highly problematic. For a discussion on this topic see Bolton (2008).

treated and, if possible, cured, but rather a human specificity (like sex or race) that must be equally respected. For them, an atypical neurological “wiring” and not a pathological cognitive organization accounts for their difference’ (Ortega, 2009: 426). A current diagnosis of autism, however, requires a *failure* to develop *appropriate* relationships, a *lack* of seeking to share enjoyment or *impairments* in the use of behaviors that regulate social interaction (APA, 1994). Instead of attributing the impairments and failures to the ‘atypically wired’ autistic individual, the neurodiversity movement locates the source of failure and impairment in the lack of acceptance, respect and societal tolerance for autistic difference.

Nevertheless, irrespective of the possible sources of distress and impairment, and unlike daisies or buttercups, delineating and conceptualizing autism has always been related to the medical commitment to treating suffering, impairment and ‘abnormality’ wherever it occurs. A necessary relation between diagnosing autism and undesirable conditions, often expressed in terms of impairment, disability or distress, can be further illustrated by paying some more attention to diagnostic assessments and instruments. In a clear and accessible book *Autism and Asperger Syndrome: The Facts* (2008), written for a wide audience by autism professor Simon Baron-Cohen, the process of making an autism diagnosis is spelled out. Baron-Cohen explains that, besides standardized diagnostic instruments such as the ADI (*Autism Diagnostic Interview*), a clinician conducting the diagnostic interview needs to ask questions that are evidence for the required social difficulties: ‘Have they found it difficult to make and keep friends? ... Do they show a lack of normal *social awareness*? ... Have they found it hard to understand and respond appropriately to other people’s feelings?’ (Baron-Cohen, 2008: 38). Answers to these types of questions with value-laden terms such as ‘lack of’ or ‘appropriately’ profoundly depend on experiences of impairment or dysfunctioning in the social sphere and on implicit social norms related to making friends, being socially aware and being able to empathize appropriately.

As I mentioned earlier, behavior-based diagnostic manuals – like the DSM – are the main point of departure both for clinical practice and for fundamental neurobiological research, and an official DSM diagnosis for autism even explicitly requires, among other things, qualitative impairments in social interaction and in communication (APA, 1994). In *DSM-III-R* (APA, 1987: 38), one of the criteria for impairment in communication was ‘lack of imaginative

activity ... and lack of interest in stories about imaginary events'. This criterion changed in *DSM-IV* (APA, 1994: 70) into 'lack of varied, spontaneous make-believe play or social imitative play', and in the proposal for *DSM-5*, this criterion changed again into 'difficulties in sharing imaginative play'.²³ These changes in criteria reflect changes in ideas about abnormal and impaired imaginative play in children. In spite of autism's inevitable relation with (variable) ideas about abnormality and impairment in social behavior, Baron-Cohen naively hopes that in the spirit of a natural kind approach 'diagnosis will not depend on the vagaries of a clinical interview or of direct observations of behavior, which invariably includes some subjective elements. Instead, it will be based on a biological marker or set of markers ... measured in the blood or in other bodily tissue or cells' (Baron-Cohen, 2008: 41).

The works of Nadesan (2005), Eyal et al. (2010) and Silverman (2011) provide more examples of how the variable needs of clinicians, parents, researchers and society guided the demarcation and structuring of particular problems with sociality, and how autism emerged and developed in close relation to historically and socially variable ideas about deficiency, abnormal behavior and unmet needs of children. Nadesan (2005: 3), for instance, argues that high-functioning forms of autism and Asperger's disorder emerged 'in the context of new standards for parenting that emerged mid-twentieth century'. Within new cultural and social conditions in the 1960s that led to a shift in 'emphasis on the "psychological" adjustment of personality ... towards an emphasis on "cognitive" fitness of intellect' (p. 109), the popularization of models of intellectual and linguistic development and the ethological idea of 'critical periods' of developments created a need and responsibility for engineering a child's intellectual and emotional development. In combination with a growing public's interest in education and an effort to ensure a child's future success, this led to an increased sensitivity of parents to any 'delays' in a child's cognitive and social development, an increased need for expert advice, new needs and space for medicalizing and pathologizing childhood deviance, and a further expansion of autism's boundaries (Nadesan, 2005).

I suggest that it is a combination of, on the one hand, the context dependent undesirability of autism, and, on the other hand, the context sensitive nature of the problems (with sociality) that autism covers, that explain

²³ See: <http://www.dsm5.org/proposedrevision/Pages/Default.aspx>, accessed 31 August 2012

what makes autism particularly vulnerable to change. Current ideas about autism are relational to some sense of discontent about how a child relates to its environment. This is why, for autism at least, Cooper's weed analogy does not hold good. Shifting ideas of how a child should behave in the social world go hand in hand with shifting scientific ideas on autism, whereas varying ideas on what should be recognized as a weed, will not affect the characteristics of daisies or buttercups. The boundaries of autism are not set by nature, but by the need to frame discontent in a particular way: a way that marks, delineates and converts a capricious category of problems into a suitable case for treatment, a way that facilitates a structured narrative that is able to understand and explain what previously remained uncertain. The needs and discontents of a society regarding how an individual interacts with others, makes friends, initiates chit-chat, seeks to share enjoyment, empathizes and figures out implicit social norms, will be continuously in flux and make autism inevitably hard to pin down. Not a single gene, biological network, neural process or cognitive theory can do anything about the variability of autism.

Conclusion

During autism's relatively short history, it has repeatedly been presented as a distinct nosological entity. All through the many theories that have been suggested and rejected, from refrigerator mothers to vaccine preservatives and genetic mutations, researchers and clinicians have always tried to get a grasp on the true nature of autism.

Discussions on the rising rates of people diagnosed, on how autism should be classified in the *DSM-5*, and other pressing issues like which promising new research directions should be invested in and where treatment and interventions should be directed at are all shaped within a framework that assumes autism to be a natural category. Currently, this 'thing' called autism is imagined at a neurobiological level and autism researchers continue their search at increasingly complex levels of epigenetics, molecular biology and neural networks, to find what they are looking for: the biological boundaries of autism. Despite ubiquitous heterogeneity, the rhetoric of the autism puzzle and the prospect of finding one or more underlying unified mechanisms fuels the

tenacious search for autism's neurobiological nature. This search comprises the central challenge for contemporary autism researchers.

There is, however, a serious concern for a natural kind approach in autism research. No matter how sophisticated and multicausal the underlying mechanisms are presumed to be, autism can only be understood in relation to ideas about what kind of behavior is unacceptable and in need of correction or support. Autism's emergence, the historical transformations and fluctuating boundaries necessarily reflect certain desired standards of a child's connection with the world. Autism has always been related to a cultural norm of a social, communicative, empathic and engaged individual, and recent historical studies on autism show that various changing accounts of autism cannot avoid being related to historically and socially variable needs to demarcate, locate and treat particular discontents and impairments that have apparently appeared. In making this point, I certainly do not argue that autism does not include severely disturbed individuals that immensely suffer, disrupt entire families, and need continuous care. Seriousness or persistency is not at issue here. But I do argue that despite the widely experienced sense of a distinct and unique syndrome, there is no transhistorical essence or 'true' autism core to be revealed in nature, and that there is a remarkably persistent desire to locate suffering, disruption and the requirement for care as a natural phenomenon, rather than implicated in the demands of a social world.

This is not just of theoretical importance. Attempts to carve nature at its joints, to dig up historical cases of autism and to clarify whether there is a 'true' autism epidemic are only intelligible with the presumption that autism can be defined and demarcated in neurobiological terms. Rejecting this presumption by emphasizing the social and cultural elements in delineating autism reveals these attempts to be seriously limited. Furthermore, apart from the enormous costs that are involved in neurobiological research, a natural kind approach in autism research obscures an array of social, cultural and psychological issues important in understanding how the phenomenon we call autism has emerged, developed and become one of the most prevalent mental disorders in children in less than thirty years. What have become the prevailing values and implicit norms in social life and children's behavior? How does modern society deal with diversity and suffering; what does it mean to be a child diagnosed with autism and how does it shape a child's sense of self? What are the social and

political forces that enable to locate and maintain autism within the biological realm of the individual?

These questions – and others – have become relatively unimportant in the scientific search for autism's natural boundaries at neurobiological levels. In practice, they have been dismissed to the supposedly non-scientific disciplines such as sociology, anthropology and philosophy, and thus designated as fundamentally irrelevant to the work of scientists. However, clinicians and neurobiological researchers urgently need to engage these questions. Acknowledging that autism is not an entity (or multiple entities) contained within an individual and that it does not have a unifying core or fundamental nature, de-inevitabilizes current biomedical perspectives, taxonomies, research purposes and intervention targets. Acknowledging the human and contextual elements in the production of the 'thing' called autism makes it possible to renegotiate autism's boundaries, to put the above-mentioned questions on the agenda in autism research and to better connect with the concerns of those involved. For this purpose, a closer collaboration between the social, medical and neuroscientific disciplines is absolutely indispensable. Only from a multidisciplinary point of view can we properly approach the question whether the current use of the category of autism to delineate, structure and respond to the 'autistic' problems so many children and adults are faced with today is effective, useful and ethically warranted.

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Supplement²⁴

Challenging a mechanistic model of mental disorders

A fundamental question in the philosophy of psychiatry is: What kind of things are psychiatric disorders? This issue is being discussed extensively in a philosophically oriented literature, but there is still no consensus as to the best answer. Can psychiatric disorders best be conceived of as; objects that exist in nature independent of psychiatric classifications (natural kinds, see, for example, Haslam, 2003; Cooper, 2004); scientifically constructed tools or instruments that help to achieve important goals (practical kinds, see, for example, Zachar, 2002); or maybe as kinds that are brought into being by societies and cultures through the practice of classifying human behavior as distinct kinds (socially constructed kinds, see, for example, Young, 1995)?

Current assumptions, understandings and practices in the field of autism, I suggest, are compatible with a permissive account of natural kinds, namely the *mechanistic property cluster* (MPC) account of natural kinds recently proposed by Kendler, Zachar and Craver (2011) as *the* model for understanding psychiatric disorders in general. However, despite the attractiveness of a value-free mechanistic model, the MPC model has certain limitations. In this supplement to the previous chapter, I illustrate how these limitations relate to the traditional separation of two types of demarcation problems in (the philosophy of) psychiatry – between distinct mental disorders on the one hand, and between normality and pathology on the other hand. A mechanistic model of psychiatric

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disorders only concerns the former demarcation problem while it is indifferent with respect to the latter. Due to this limitation, this model is unable to account for the way in which social and cultural norms, and shifting boundaries of normality and pathology shape and transform autism as a psychiatric entity.

Mechanistic property cluster (MPC) kinds

In a recent essay in *Psychological Medicine*, Kendler, Zachar and Craver (2011) argue for a mechanistic model for understanding psychiatric disorders. Inspired by the philosopher Richard Boyd (1991; 1999) they suggest that psychiatric disorders can best be viewed as *mechanistic property cluster* (MPC) kinds. Boyd developed the concept of homeostatic property clusters (HPC) to challenge a stringent essentialist model of natural kinds in which a necessary and sufficient property or structure (an essence) directly and causally determines all key features of a kind (Kendler and colleagues replaced the term ‘homeostatic’ with ‘mechanistic’ to avoid possible confusion due to different meanings of the term ‘homeostatic’).

According to Boyd, there are scientifically important kinds – biological species for instance – that are characterized by a cluster of often co-occurring characteristics and by the underlying mechanisms that bring about their co-occurrence. These clusters do not have invariable and exclusive essences and the members of a kind do not need to overlap in a fixed set of characteristics. Rather, kind membership is defined by some set of empirically discoverable causal mechanisms that explain, in the case of biological species, ‘the imperfectly shared and homeostatically related morphological, physiological and behavioral features which characterize its members’ (Boyd, 1991: 142). Similar stable patterns of often complex causal mechanisms that involve interactions between multiple possible levels of explanation – such as physiology, behavior and environment – instantiate the imperfect co-occurring characteristics of the members of a species. They are considered imperfect because ‘kind definitions must conform to the (sometimes messy and complex) causal structure of the world’ (Boyd, 1991: 143). Members of a species need not share all their characteristics, and differences between species can be vague. However, this doesn’t imply that there are no stable explanatory mechanisms to be discovered underlying common characteristics of individual members of a species.

Kendler, Zachar and Craver (2011) suggest that Boyd's HPC model of kinds should be the key model for understanding what kind of things psychiatric disorders are. They ask us to consider a multi-dimensional matrix that reflects human mind/brain states. The properties included in this matrix may include genes, neural systems, psychological states, symptoms themselves and environmental inputs. They argue that there are only a finite number of mind/brain states that 'are cohesive and temporally stable, some proportion of which represents "psychiatric syndromes"' (p. 1147). For them, psychiatric disorders are best conceived of as sets of symptoms that are connected through a system of causal mechanisms. Ultimately, these causal mechanisms are what define and sustain the disorder.

This MPC model of mental disorders is attractive for several reasons. It corrects an empirically inadequate 'gene X causes disorder Y' (essentialist) model, it is compatible with the multicausality, fuzzy boundaries and heterogeneity of most psychiatric disorders, and it provides (unlike pragmatist models) prescriptive guidance for the investigation of objective causal structures that will inform psychiatric nosology in the attempt to carve nature at its joints (*ibid.*). These joints are not located at the boundaries of single genes, infective agents or local lesions, but at the boundaries of causal mechanisms (Samuels, 2009). Thus, the MPC model facilitates the prospect of discovery and 'true' delineation of specific disorders. Since MPC kinds are grounded in the natural features of the world and are 'not merely imposed upon the world by psychiatrists through their classificatory practices', psychiatric categories will become scientifically valuable in terms of prediction, explanation and control. Even though there is no single causal mechanism or essential property that explains all the superficial properties of a kind, 'the identity of the disease across time and across cultures is grounded in the similarity of the complex mutually reinforcing network of causal mechanisms in each case' (Kendler et al., 2011: 1147).

Furthermore, as both Kendler, Zachar and Craver (2011) and Samuels (2009) – who defends an MPC model for delusions – underline, MPC kinds allow 'that the same cluster of symptoms might arise from different etiological, underlying or sustaining mechanisms in different cases' (Kendler et al., 2011: 1147). There need not be a one-to-one relation between an underlying neurobiological causal mechanism and the resulting cluster of psychiatric symptoms. However, distinct etiological or pathophysiological mechanisms in

different members of the same kind must share a similar causal mechanism at another biological level. Biological heterogeneity is allowed, as long as more homogeneous mechanisms can be identified at other biological levels. Much of the research in autism is, despite profound genetic heterogeneity, directed at identifying unifying neural mechanisms that underlie all – or a subgroup of – autism cases. According to Samuels (2009), identifying such unifying mechanisms comprises perhaps *the* fundamental explanatory challenge for an MPC approach to psychiatric disorders. Kendler, Zachar and Craver (2011) conclude that we are ‘far from being able to define plausible stability-producing mechanisms for most psychiatric disorders’ (p. 1148).

However, as Chapter 2 also illustrated, contemporary researchers in the field of autism generally, but usually unknowingly, follow the prescriptive guidance of the MPC model. A common neurodevelopmental abnormality is still assumed to unite all – or a subgroup of – autism patients. Functional genomics, epigenetics, molecular genetics and systems biology are among the new hopes in the search for autism’s unity. Current developments in autism research fit strikingly well with the MPC model proposed by Kendler et al. (2011). Autism researchers and clinicians need to deal with multiple causes and (genetic) heterogeneity, but autism research is nonetheless directed at identifying ‘objective’ causal mechanisms that should inform nosologists in their attempt to carve autism’s boundaries at its supposed natural joints. However, a convincing mechanistic approach to autism requires a clear separation of two familiar types of demarcation problems in psychiatry.

Two demarcation problems

The first demarcation problem concerns the question of whether and when a certain constellation of signs and symptoms legitimately reflects a distinct category. Is schizophrenia, for instance, a valid disease category and to what extent is schizophrenia distinct from schizo-affective disorder, delusional disorder or any other (‘normal’) state or trait? A central term in this debate is *validity*. This is a complex construct with several meanings and subtypes, which I do not discuss here in detail. Rather, I briefly focus on how this term has been used in psychiatric nosology.

Robins and Guze (1970) were the first to propose a formal method to improve the validity of psychiatric categories. In their influential article on establishing diagnostic validity for schizophrenia, they proposed five phases in the evaluation of a putative diagnostic category that they thought were an indication of its validity: clinical description, laboratory studies, delimitation from other disorders, follow-up studies, and family studies. These validators were used to show that ‘apparent “schizophrenia” with a good prognosis is not a mild form of schizophrenia, but is a different illness’ (p. 987). Their findings provided the basis for the distinction between schizophrenia and schizophreniform disorder in *DSM-III* (APA, 1980). Kendler (1990) expanded the set of validators and distinguished between antecedent validators (familial aggregation, premorbid personality, and precipitating factors), concurrent validators (including psychological tests), and predictive validators (diagnostic consistency over time, rates of relapse and recovery, and response to treatment).

A common assumption underlying discussions about validity and proposals to increase the validity of psychiatric categories is that a ‘truly’ valid psychiatric disorder reflects genuine underlying (pathophysiological) differences in relation to other disorders and normal brain functioning. Kendell and Jablenski (2003) argue that while the diagnosis of psychiatric disorders is still based on clinical observation, a distinct syndrome will be valid if we reasonably expect that it can be defined by physiological, anatomical, chromosomal, histological or molecular abnormalities. Besides increasing reliability, since *DSM-III* the aim of psychiatric classification systems has been to create psychiatric categories that facilitate the identification of genes, neurotransmitter mechanisms and other neurobiological markers related to psychiatric disorders. In line with this aim, the ultimate goal of psychiatric taxonomy, as the research agenda for *DSM-5* Kupfer et al. (2002) concluded on this issue, has become ‘to translate basic and clinical neuroscience research relating brain structure, brain function, and behavior into a classification of psychiatric disorders based on etiology and pathophysiology’ (p. 70).

The MPC model of psychiatric kinds is in line with this effort. By informing nosologists, the MPC model attempts to increase the validity of psychiatric categories, where validity depends on whether a certain psychiatric category captures genuine underlying differences. The MPC model is supposed to bring us closer to the ultimate goal of current psychiatric nosology, which is a system

based on etiology and pathophysiology with neuroscience providing the foundation for classification and possibly individual diagnosis. However, the MPC model and conventional discussions on validity are largely indifferent towards another central demarcation problem in psychiatry. This second demarcation problem which will be discussed below, concerns a more general question: How can the distinction between normal and pathological mental functioning be made?

In a comprehensive monograph on this contested topic, Bolton (2008) discusses several possible ways to make this distinction. One way, for instance, is to conceive pathological mental functioning as ‘a matter of breakdown of meaningful connections in mental life’ (p. 16). Examples of a breaking down of meaningful connections include emotions that are excessive or have no appropriate object, behavior that is not under the control of the person’s will, and beliefs that have no basis in experience. Another possibility, inspired by the work of Jerome Wakefield (1992), is to conceive of pathological mental functioning as ‘not functioning as it has been naturally designed to do in the evolutionary process’ (Bolton, 2008: 17). Despite the value of some of the theories he discusses, Bolton concludes that there is not one single theory that adequately distinguishes all forms of mental pathology from normality. Furthermore, in line with a widespread consensus among philosophers of psychiatry, Bolton concludes that how the line between what is normal and what is pathological in mental functioning is drawn depends on social, cultural and individual values and circumstances. Even Jerome Wakefield (1992), who is considered to be on the naturalist side concerning mental disorders, acknowledges that a biological dysfunction needs to be *harmful* in order to become pathological, and harm cannot be understood independent of sociocultural circumstances.

In defending the value-free MPC approach for delusions, Samuels (2009) is well aware of the two potentially conflicting demarcation problems. However, he argues that the normativity of pathology is not necessarily but only contingently connected with delusions. Without some reason to suppose that this connection is a necessary one, this normativity does not pose a threat to the MPC model regarding delusions (ibid.). Kendler, Zachar and Craver (2011) are equally aware of the evaluative nature of mental pathology as they acknowledge that ‘values are intimately involved in determining which psychiatric kinds deserve clinical attention’ (p. 1147). However, values are not

only involved in determining whether the condition we have come to call autism deserves clinical attention, they are also involved in defining and delineating this psychiatric kind in the first place. Taking the distinction between normality and pathology into account is crucial for understanding the way in which autism emerged, transformed and is currently defined as a diagnostic entity.

Limitations of a mechanistic model

As Cooper (2010) convincingly argues, culture-bound syndromes that emerge in highly specific social and historical contexts can still be distinct ‘natural’ disorders. For instance, similar to different kinds of igneous rocks that are created under specific environmental conditions, a mental disorder can be influenced by cultural and environmental factors such as diet, lifestyle or environmental pollution, and still be a distinct natural (MPC) kind grounded in a network of causal mechanisms. Social and cultural factors can be considered as causal agents that become part of the entire network of causal mechanisms associated with the particular kind. Biology and culture may interact, Cooper argues, ‘so as to produce cases of a disorder that are recognizably and reliably similar to each other and such disorders can usefully be recognized by psychiatric classification systems’ (ibid.: 331).

Following Cooper’s argument, putative culturally and historically specific *causal* factors (for example, child-rearing practices or environmental toxins) and, as a hypothetical consequence, varying prevalences or manifestations of autism all over the world would not necessarily threaten a mechanistic (MPC) model of autism. However, the fundamental requirement of the model, that the *identity* and *boundaries* of a particular disorder are *set* by causal mechanisms, is particularly problematic for autism. As Kendler, Zachar and Craver (2011) argued, ‘the identity of the disease ... is grounded in the similarity of the complex mutually reinforcing network of causal mechanisms in each case’ (p. 1147). ‘An MPC kind’ is their best answer to the ontological question: What kind of thing is a psychiatric disorder? However, the historically and culturally variable boundaries of ‘impairment of social interaction’ or ‘a lack of ability to understand and use the rules governing social behaviour’ – now considered essential features of autism – are clearly not set by causal mechanisms. This

issue of setting the boundaries of autism is not just a matter of demarcating a coherent cluster of signs and symptoms, it is also a matter of demarcating normality from pathology.

Social and cultural values and norms not only influence whether a certain cluster of symptoms is considered as a disorder, but they play, in autism at least, a necessary role in what becomes a recognizable cluster of symptoms in the first place. Defining autism as a nosological entity incorporates the (shifting) needs and discontents of a society regarding how an individual interacts with others, empathizes, makes friends, seeks to share enjoyment, initiates small-talk, and figures out implicit social norms. This blurs the boundaries between the two discussed demarcation problems as demarcating autism (and identifying neurobiological dysfunctions related to autism) *necessarily* involves demarcating undesirable conditions. An MPC model of autism that attempts to ground the identity and boundaries of autism in causal mechanisms has to ignore these normative dimensions.

Mental disorder in *DSM-5*

Both in the definition of mental disorder in *DSM-IV* (APA, 1994) and in the proposal by Stein et al. (2010) for a modified definition of mental disorder for *DSM-5*, the two discussed demarcation problems are reflected in separate criteria (see also Broome and Bortolotti, 2010; Verhoeff and Glas, 2010). In particular, criterion A, that a mental disorder is ‘a behavioral or psychological syndrome or pattern that occurs in an individual’, implicitly concerns the first demarcation problem of whether a certain cluster of features legitimately reflects a distinct disease (Stein et al., 2010: 1761). Criterion B – ‘the consequences of which are clinically significant distress (for example, a painful symptom) or disability (i.e. impairment in one or more important areas of functioning)’ – refers to the second general problem of demarcating normality from pathological mental functioning.

The separation of the two demarcation problems in different criteria is compatible with an MPC model of psychiatric kinds, in which a behavioral or psychological syndrome or pattern (cluster) reflects underlying (psychobiological) mechanisms. Whether it ‘deserves clinical attention’ (criterion B) can be approached as a separate issue. However, for autism, as we

have seen, these two problems are inextricably linked to each other. The phrase ‘in an individual’ in criterion A is particularly problematic for autism. As Broome and Bortolotti (2010) indicated, the phrase ‘in an individual’ is complex, controversial and carries conceptual baggage. It may seem evident that certain psychological states and behavioral patterns belong to or reside in an individual. However, as the case of autism illustrates, the recognition and description of an autistic behavioral pattern or particular autism signs and symptoms is profoundly embedded in a social and cultural context. Defining autism depends on historically and culturally variable ideas about deficiency, abnormality and dysfunction, and on the need to demarcate and treat particular discontents and impairments that have appeared. The case of autism, generally considered to be one of the most ‘biological’ of all mental disorders, illustrates Broome and Bortolotti’s (2010) suggestion: ‘that at the very least the claim that a disorder occurs “in an individual” warrants further examination’ (p. 1784).

Conclusion

The mechanistic property cluster (MPC) model, which attempts to define and delineate autism in terms of causal mechanisms, is attractive for several reasons: it corrects an empirically flawed essentialist model; it is compatible with the multicausality, heterogeneity and fuzzy boundaries of many mental disorders; it provides prescriptive guidance for the investigation of objective causal structures; and it ‘satisfies the intuitions of reductionist psychiatrists’ (Kendler et al., 2011: 1148). Current autism research fits the MPC model strikingly well, as autism research – despite the acknowledged heterogeneity of the condition – is guided and regulated by the depiction of autism as a scientific and natural object that can be discovered and identified with systematic neuroscientific investigation. However, the MPC model of natural kinds (needs to) neglect(s) the way in which autism relates to ideas about what kind of behavior is inappropriate and in need of correction or support. As Chapter 2 argued, normative issues concerning disability and impaired social interaction have been and still are inextricably linked to how we recognize and understand autism.

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3 | Autism in flux²⁵

Autism is not a modern phenomenon, even though it has only been recognized in modern times. In view of the short history of psychiatry, and the even shorter history of child psychiatry, we know that a disorder recently described is not necessarily a recent disorder. An increase in diagnosed cases does not necessarily mean an increase in cases. There are tantalizing hints of Autism in the medical records of history.

A case description by the apothecary of Bethlem Hospital, the London mental asylum, often been quoted and never contested, as early evidence of Autism. The case was that of a five-year old boy who was admitted in 1799. It was particularly noted that this boy never engaged in play with other children or became attached to them, but played in an absorbed, isolated way with toy soldiers. (Frith, 1989: 16)

Since 1938, there have come to our attention a number of children whose condition differs so markedly and uniquely from anything reported so far, that each case merits – and, I hope, will eventually receive – a detailed consideration of its fascinating peculiarities. (Kanner, 1943: 217)

Abstract

This chapter argues that a new relation between past and present – a supposed historical continuity in the meaning of autism – is created by histories written by the discipline itself. In histories of autism written by ‘practitioner-historians,’ a sense of scientific progress and an essentialist understanding of autism legitimize and reinforce current understandings and research directions in the

²⁵ This chapter has been published as Verhoeff B (2013) Autism in flux: a conceptual history from Leo Kanner to DSM-5. *History of Psychiatry* 24(4): 442-458.

field of autism. Conceptual discontinuities and earlier complexities and disputes concerning classifying and delineating autism are usually left out of the positivist narrative of autism. In an alternative history of the concept of autism, I demonstrate that there have been major shifts in the type of symptoms, signs and impairments that were – and are – thought to be essential and specific for autism.

Introduction

With the latest version of the American Psychiatric Association's *Diagnostic and Statistical Manual for Mental Disorders* (APA, 2013), the history of autism enters a new era. The Neurodevelopmental Disorders (ND) Work Group – responsible for the formation of criteria and diagnostic categories of autism and related disorders – folded Asperger's disorder, along with PDD-NOS²⁶ and autistic disorder, into the new category of *autism spectrum disorder* (ASD). The main argument for the creation of one ASD category was that 'there is little evidence to support the current diagnostic distinction between Asperger disorder and high-functioning autism' (Happé, 2011: 541). Studies that compared clinical and demographic characteristics, neuropsychological profiles, comorbidity and prognosis between autism and Asperger's disorder largely did not support a diagnostic distinction between the two diagnostic categories (Witwer and Lecavalier, 2008). As stated by one of its prominent members, the *DSM-5* ND Work Group's aim in proposing the new ASD category was to 'recognize the *essential* shared features of the autism spectrum' (Happé, 2011: 541, emphasis added).

In the Work Group's reflection on the current state of autism research, autism is depicted as a discoverable entity with particular transhistorical essential features and autism research is presented as a growing body of scientific knowledge in relation to that particular entity. With this conception of autism and autism research, current developments and decisions concerning autism in *DSM-5* have become a logical and inevitable outcome of the present state of autism research. Furthermore, the dominant positivist understanding of autism underlies recent debates on the existence or non-existence of an autism

²⁶ Pervasive Developmental Disorder Not Otherwise Specified, also considered part of the autism spectrum.

epidemic and it underlies the current tenacious search for autism's neurobiological essence (see Verhoeff, 2012/Chapter 2). This particular understanding of autism is being reinforced by the histories written by the discipline itself, depicting a more or less chronological, linear and progressive development towards current understandings of autism. However, this chapter illustrates that this is a limited representation of autism and its history.

For a better understanding of what is happening in the field of autism today, it is essential to explore its historical development in more detail. As stated by Polish medical doctor and philosopher of science Ludwik Fleck, 'at least three-quarters if not the entire content of science is conditioned by the history of ideas, psychology, and the sociology of ideas and is thus explicable in these terms' (Fleck, 1935/1979: 21). The primary aim of this chapter is exactly to explore the historical development of scientific ideas about autism in more detail, with a particular focus on how autism has been conceptualized since Leo Kanner (1894-1981) first described autism as a distinct nosological entity. Thereby, a new perspective on the idea of an autism spectrum and on the rise and fall of Asperger's disorder emerges.

In the first part of this chapter, some historiographical points are made and I demonstrate a specific way in which the discipline's history is being reorganized according to current understandings of autism. I argue that a new relation between past and present – a supposed historical continuity in the meaning of autism – is created through historical perceptions and references to classic autism studies. The second part of this chapter presents an alternative history of the concept of autism that challenges the general sense of continuity and progress. Instead of emphasizing the widely presumed historical stability of the core meaning of autism, I focus on these historical irregularities that are often overlooked, denied or misunderstood.

Even so, it is often noted that autism has gone through major changes since Leo Kanner first described the syndrome in 1943. Explanatory theories, interventions, public awareness and specialist services have changed considerably over the past seventy years. In addition, a progressive broadening of the concept of autism is generally accounted for in, for instance, explanations for the extraordinary rise in people diagnosed with autism (see, for example, Fombonne, 2005; Wing and Potter, 2002). As is visible through an evident widening of diagnostic criteria for autistic disorder in subsequent versions of the DSM, the broadening of the concept of autism is unambiguous.

However, the exact nature and scope of this broadening remain largely opaque and unexplored. I suggest that there has not just been a dimensional broadening, but that there have been major shifts in the specific type of symptoms, signs and impairments that were – and are – thought to be essential and specific for autism.

Recurrent histories of autism

Books on the history of autism are not as numerous as autism novels, parent guides, autobiographies or textbooks. Of the small number of histories that are available, there is a wide variety in depth and specific focus of historical attention. Some recent histories of autism have as their object the pioneers in research, treatment and care (Feinstein, 2010), the role of parents and parent organizations (Silverman, 2011), or the social and cultural conditions of possibility for autism to emerge (Eyal et al., 2010; Nadesan, 2005). However, most histories of autism have been written by autism researchers and experts – or ‘practitioner-historians’ – often as an introductory first chapter to the topic in text- and handbooks, or as a short introduction in review articles or empirical studies. A common denominator of these latter histories is that they approach their main object – autism – as a static, decontextualized ‘thing’, discoverable by science. Moreover, these histories present a more or less chronological, linear and progressive development towards an ‘inevitable’ current understanding of autism, while mentioning the myths, mistakes, struggles and scientifically unsound convictions of earlier darker periods. For example:

We have come a long way from the era of myths and legends, through the unhappy deviation into psychoanalysis, to the practical realism of the present day that is accepted by most, though sadly not all, professionals in the field. We now know that there is a wide spectrum of autistic conditions, with Kanner’s and Asperger syndromes each forming only a part of these. (Wing, 1997: 20)

Such histories might seem to be little more than the memory of a science and a subordinate distraction from what the topic is really about, but if we follow the French historians and philosophers of science, particularly Gaston

Bachelard (Gutting, 1987) and Georges Canguilhem (1994), a discipline's history has become a central part of the discipline itself. In a discipline's history there is 'the tendency to see the history of the subject in the light of today's truth, which is easily confused with eternal truth.' (Canguilhem, 1994: 42). A discipline's history serves as an important legitimization of a science with the current knowledge about a subject matter as a necessary endpoint of scientific development; it confirms the idea, favorable to the discipline, of a science as a rational inquiry with a coherent accumulation of knowledge through history. The historical awareness of scientists is 'recurrent' in a sense that:

The history they construct for themselves is always constructed from the present and its criteria for truth. The perspective of the present reorganizes the past according to its own relevances. A mutation in current conceptions of scientific truth brings elements from past and present into new relations ... Hence, as science corrects its errors, it rewrites its history. (Rose, 1998: 166)

In the field of autism, two types of frequently combined 'recurrent histories' can be distinguished. The first one I call 'positivist histories'. These emphasize the progressive and cumulative nature of knowledge about autism and the maturation of the research field, while judging earlier theories, interventions and perspectives in the light of current expert convictions.

'The history of ideas on autism; Legends, myths and reality' (1997) by Lorna Wing, British psychiatrist and autism expert, makes a clear example of a positivist history of autism. She depicts the idea of a spectrum of autistic conditions as something in nature that was out there waiting to be discovered through a laborious process in which plain myths and Freudian mistakes had to be defeated by proper science. This resulted in 'the increasing knowledge of the nature of autistic disorders' (ibid.: 20). Earlier etiological hypotheses of autism attributing an important role to psychogenic causes, of which the infamous 'refrigerator mother'²⁷ received most attention, are discarded as 'unhappy deviations' since current research has shown that 'complex genetic factors are important in the causation but there are other physical causes that can lead to

²⁷ The creation and popularization of this term 'refrigerator mother' is often attributed to the for many years most popular public autism expert Bruno Bettelheim and his widely read book *The Empty Fortress* (1967).

autistic conditions' (ibid.). Earlier ideas on autism are neatly divided into plain myths or reality, and earlier legends in the field are deemed either right or wrong. Current knowledge about autism acts as an endpoint and norm to judge history, while knowledge about autism cumulated in a linear and chronological way towards the contemporary view that 'autistic spectrum conditions are developmental disorders caused by physical abnormalities in parts of the brain' (ibid.: 20).

The recursive process in positivist histories of autism can be noticed by focusing on how autism researchers refer to earlier criteria and understandings of autism. In choosing the diagnostic criteria for autism in *DSM-IV* (APA, 1994), Lynn Waterhouse et al. (1992) provide a history of the development of the *DSM-III-R* (APA, 1987) criteria for autism. They state that the *DSM-III-R* criteria 'represent a reorganization and elaboration of *DSM-III* criteria that reflect a return to the criteria of Kanner's (1943) original case study descriptive accounts of impaired sociability, impaired social communication, and the presence of stereotypies or repetitive behaviors' (Waterhouse et al., 1992: 531). Besides the suggested fundamental continuity in diagnostic criteria for autism, the 'triad of autistic impairments' popularized by Wing and Gould (1979), is rather unproblematically projected onto Kanner's original description of early infantile autism. The '*extreme autistic aloneness*' mentioned by Kanner (1943: 242) is nevertheless quite different from impaired sociability, just as impaired social communication is different from the severe deficits in language development that were illustrated by Kanner, such as complete muteness and (delayed) echolalia. In addition, for Kanner, 'the child's behavior is governed by an *anxiously obsessive desire for the maintenance of sameness* that nobody but the child himself may disrupt' (ibid.: 245). An extreme resistance to changes in restricted routines and rituals or changes in furniture arrangements is again in many ways distinct from the 'presence of stereotypies or repetitive behaviors'.

The point here is that in a brief historical reference, Kanner's work is confidently connected to recent criteria of autism, with a suggestion of both continuity and refinement. Fred Volkmar (1998: 46) similarly claims that the *DSM-IV* (APA, 1994) and *ICD-10* (WHO, 1993) diagnostic systems have a 'fundamental continuity with the original description of autism made by Leo Kanner (1943)'. Volkmar suggests that Kanner emphasized as central to the definition of autism: 'the characteristic impairment of social interaction' (ibid.). Earlier 'truth' about autism is thus colored in the light of current 'truth' and the

connections between them are being reinterpreted to legitimize and reinforce the current status of autism research.

The increasingly popular practice of retrospectively diagnosing historical figures with autism is part of what I call ‘essentializing histories’ of autism. These particular histories are not primarily concerned with the development of the scientific field, but above all with affirming the validity and trans-historical continuity of the specific object under study: autism. Apart from being a recognizable disorder (Frith, 1989; Volkmar, 1998), autism is currently first and foremost ‘a highly heritable neurodevelopmental disorder’ (Mosconi et al., 2010) and ‘among the most heritable of all mental disorders’ (Lichtenstein et al., 2010). If autism is such a recognizable phenomenon and if it is a current fact that autism is a heritable brain disorder, traces of autism must be found in distant pre-Kannerian times.

Indeed, traces have been found. Historical accounts of feral children, eccentric geniuses, religious figures and even fictional literary protagonists have proven productive for retrospectively diagnosing autism. To name only some of those diagnosed, there are *The Blessed Fools of Old Russia* (Challis and Dewey, 1974), the extraordinary case of Hugh Blair of Borgue in eighteenth-century Scotland (Houston and Frith, 2000) and – according to Michael Fitzgerald’s (2005) endless list – Isaac Newton, Michelangelo, Ludwig Wittgenstein and Albert Einstein.²⁸ According to Frith (1989: 17), retrospective diagnosing helps to ‘distil those features that are the essence of the disorder beyond our immediate time and cultural context’. She points out that ‘autism is not a modern phenomenon, even though it has only been recognized in modern times’ (Frith, 1989: 16) and she and Houston try to find what they call ‘the unchanging core of autism’ (Houston and Frith, 2000: 4). However, it is this assumed trans-historical, essential core of today’s autism that is being recognized in the many recent examples of peculiar historical figures, and these historical cases, in turn, support the legitimization of the current ‘tenacious search for autism’s essence’ (Verhoeff, 2012/Chapter 2) at neurobiological

²⁸ Michael Fitzgerald, professor of Child and Adolescent Psychiatry, diagnosed most of these historical geniuses with Asperger’s disorder. From the perspective of ‘recurrent histories’ of autism, it will be interesting to follow up on what will happen with his huge amount of retrospective diagnoses of Asperger’s disorder now that Asperger’s disorder stops being an official diagnosis in *DSM-5*.

levels. It is only in present times that these peculiar historical figures have become part of the history of autism.

Three phases in the history of the concept of autism

The following history of autism will not explicitly search for explanations, causes, events, decisive scientific moments or the final truth about autism. Nor will it trace, like the histories of Nadesan (2005) and Eyal et al. (2010), the socio-political, economic, ideological or technical factors that might have made the emergence and reshaping of autism possible. These histories give important and detailed accounts of the role of parental activism; the availability of new treatments and services; the demand for educable conditions; and the reorganization of expertise, in understanding shifts in thinking about autism. However, the aims for the second part of the present chapter are different, and the primary aim of the following history of autism is to give a basic description of the development of scientific notions of autism.

With landmark scientific articles, definitions, diagnostic criteria, case studies and descriptions of characteristic phenomena, ideas about what makes a typical case of autism will be reviewed. However, a straightforward and unambiguous identification of earlier notions of autism is a chimera. Ideas about autism are not fixed but constantly in flux. There is not a single test, definition, article or researcher that marks a definite idea of autism in a specific period. Hence, a history of the concept of autism always remains an imperfect approximation of a general (scientific) sense of the meaning of autism at a particular moment in time. Yet in the history of ideas about the characteristic features of autism three periods can be distinguished. However, these periods are not marked by clear events and probably reflect more gradual and partial – rather than radical or revolutionary – changes, and they are above all a heuristic for structuring the historical analysis and for helping to bring to light important modifications in thinking about autism.

Phase I

Extreme autistic aloneness and insistence on sameness (1943-1965)

Most histories of autism start with Leo Kanner, a pioneer in the field of child psychiatry and the founder of the first clinic for children with psychiatric

problems at Johns Hopkins University in Baltimore. He introduced the diagnostic category of childhood autism in his famous and canonical case series of eleven children with ‘autistic disturbances of affective contact’ (1943). This was indeed the moment that autism, as a separate and unique psychiatric entity, became visible for the first time. However, if we want to retrieve the specific meaning of this new disorder, it might be better to start with the well-known Swiss psychiatrist Eugen Bleuler. Bleuler had already coined the term ‘schizophrenia’ in 1908, and was the first to use the word ‘autism’ somewhere around 1910 (Kuhn, 2004). Bleuler began using the term ‘autism’ to refer to what he considered to be one of the most important symptoms of schizophrenia.²⁹ Next to association loosening, ambivalence and affect inappropriateness (Berrios, 1996), autism was one of the primary symptoms of schizophrenia, and it was characterized by ‘a definite withdrawal from the external world’ (Bleuler quoted in Kanner, 1973: 94).³⁰ In the decade after its introduction Bleuler’s use of the term started to develop and expand to include a more moderate and non-pathological form of ‘autistic thinking’ that included daydreaming and fantasy (see Bleuler, 1919).

For Frith (1991), and for many other contemporary autism researchers, Bleuler’s schizophrenic autism and autistic thinking are unrelated to the disorder that came to be referred to by the same name. As Frith notes, ‘autistic thinking in Bleuler’s sense has nothing to do with autism as we know it’ (1991: 38). Maybe, if we think of the recursive processes that are active in rewriting the history of autism, it is because of the current disconnection between autism and schizophrenia, that Bleuler seems to have disappeared from the discipline’s history of itself.³¹ However, it was not without reason that Kanner borrowed Bleuler’s by then popular term.³² As Nadesan (2005: 40) notes, it is not surprising that ‘Leo Kanner and Hans Asperger elected to describe their patients in terms of the concept of autism. Autism was a phrase with wide

²⁹ Bleuler looked upon schizophrenia not as a distinct disease entity but as a common name for a group of particular symptoms. He referred to this group by speaking of the ‘group of the schizophrenias’ and not of schizophrenia in singular.

³⁰ For more on Bleuler’s conception of schizophrenia and autism, see, for example, Bleuler (1911); Berrios (1996); Gundel and Rudolf (1993).

³¹ For instance, Bleuler is not mentioned in the history of autism by Wing (1997).

³² The nature of autism as a symptom of schizophrenia and the nature of autistic thinking were actively debated in psychiatric circles in the 1920s and 1930s, for example by Ernst Kretschmer and Hans Gruhle (Gundel and Rudolf, 1993).

currency and applicability, particularly in German psychiatry'.³³ Bleuler's autism, which describes a certain break with reality combined with other dissociations of affect, provided the framework for Kanner to introduce a new diagnostic category.

In a discussion on the problems of nosology and psychodynamics of early infantile autism, Kanner (1949: 418) considers that 'the extreme isolation from other people, which is the foremost characteristic of early infantile autism, bears so close a resemblance to schizophrenic withdrawal that the relationship between the two conditions deserves serious consideration' and early infantile autism 'may be looked upon as the earliest possible manifestation of childhood schizophrenia' (p. 419). In one of his later articles, Kanner (1973: 94, original italics) explains that 'in my search for an appropriate designation, I decided on the term *early infantile autism*, thus accentuating the time of the first manifestations and the children's limited accessibility'. Kanner recognized in his autistic patients a remoteness from affective contact with other people, similar to the remoteness of Bleuler's schizophrenic patients.

However, unlike Bleuler's schizophrenic patients, who withdrew from previous participation, Kanner's children never participated in the first place. They began 'their existence without the universal signs of infantile response' (ibid.). Furthermore, as Kanner pointed out, 'they develop a remarkable and not unskillful relationship to the inanimate environment' (ibid.: 95). Instead of a turning away from the external world, Kanner's autistic patients 'can cling to things tenaciously, ... They are so concerned with the external world that they watch with tense alertness to make sure that their surroundings remain static' (ibid.). Despite a fundamental *aloofness* that relates the two autisms, and despite Kanner's explicit recognition of an intrinsic relationship between his autism and

³³ Hans Asperger (1944) introduced the term 'autistic psychopathy' only one year after Leo Kanner (1943) introduced his 'early infantile autism' and there is lots of speculation and debate (for example in Feinstein, 2010; and Eyal et al., 2010) about how it is possible that these two clinicians came up with very similar and new childhood disorders almost at the same time, while they were 'separated by an ocean and a war'. Hacking (2006: 4) argues that this is certainly not a coincidence, as 'Asperger, a generation younger than Kanner, had trained under August Homburger, the author of one of Kanner's main German textbooks, who wrote about childhood schizophrenia and other developmental disorders. They came from the same medical culture (each had served in the Austrian army, although in different wars)'.

Bleuler's (group of) schizophrenia(s), he concluded that his early infantile autism 'does not seem to fit in with Bleuler's criteria for autism' (ibid.).

Let us get back to Kanner's landmark case series that for the first time illustrated the new psychiatric condition he designated *early infantile autism*. Donald T. – his first and most detailed case – was a remarkable little boy who was happiest when left alone. He didn't seem to notice when someone entered or left the room he was in, and he was indifferent to visiting relatives, potential playmates, and he even failed to pay the slightest attention to Santa Claus in his full regalia. When petted he showed no apparent affection and he gave the impression to be self-sufficient. At the age of two he developed an obsession for spinning blocks and pans and virtually all round objects that could be spun. A spinning pan for instance, could keep him fascinated for hours and, when interfered with, he had destructive temper tantrums. The majority of his actions were endless repetitions performed in exactly the same way in which they had been carried out originally. Furthermore, he never spontaneously spoke just to chat or to share his thoughts. When he spoke, he seemed either to ejaculate irrelevant utterances randomly, such as 'chrysanthemum' or to parrot what he had heard said to him at some other time (Kanner, 1943).

After the description of ten more cases, Kanner's article (1943) ends with a discussion of a number of common characteristics of early infantile autism that appear essential. Among them are:

an extreme autistic aloneness ... [a] limitation in the variety of spontaneous activity ... performances [and verbal utterances that] are monotonously repetitions ... the child's behavior is governed by an anxiously obsessive desire for the maintenance of sameness ... [the child has] excellent rote memory. (Kanner, 1943: 242-243, original italics)

Certain language problems are also mentioned. In later articles, Kanner and Eisenberg (1956: 557) present a somewhat stricter definition of early infantile autism:

In the light of experience with a tenfold increase in clinical material, we would now isolate these two pathognomonic features, both of which must be present: extreme self-isolation and the obsessive insistence on the preservation of sameness, features that may be regarded as primary,

employing the term as Bleuler did in grouping the symptoms of schizophrenia.

Kanner investigated the phenomenon – fundamental for autism – of ‘obsessive desire for the preservation of sameness’ in more detail in a separate study (Kanner, 1951). He argued that the autistic child desires ‘to live in a static world, a world in which no change is tolerated ... The slightest change of arrangements, sometimes so minute that it is hardly perceived by others, may evoke a violent outburst of rage’ (1951: 23). Furniture arrangements, the arrangement of toy building blocks, beads or sticks, the precise route to school, and the position of the dishes on the table are typical examples of things that must not be changed. Autistic children, Kanner concluded, find ‘security in sameness, a security that is very tenuous because changes do occur constantly and the children are therefore threatened perpetually and try tensely to ward off this threat to their security’ (p. 26).

The frequently described problems with language and speech were not considered to be core features of autism. Although they are ‘often the most striking and challenging of the presenting phenomena, [they] may be seen as derivatives of the basic disturbance in human relatedness’ (Eisenberg and Kanner, 1956: 557). Furthermore, they argued that simple repetitive activities may be seen in severely retarded children and may offer a diagnostic problem, but ‘the presence of elaborately conceived rituals together with the characteristic aloneness serves to differentiate the autistic patients’ (ibid.: 558).

Robinson and Vitale (1954) discussed another diagnostic problem for autism at the annual meeting of the American Orthopsychiatric Association in 1953. They presented three cases of children with circumscribed interest patterns and ‘a limited establishment of interpersonal relationships’ (1954: 755). These children were all introvert, had average or above average intelligence, good language skills and circumscribed interests in rather unusual topics. Tom, for instance, was unable to participate in activities with other children, but developed an intense interest in and a hunger for knowledge about chemistry and finance by the age of eight years. Nine-year-old Billy had an ‘amazing knowledge of trolley routes and an unusual interest in and knowledge of calendars and maps’ and John, also nine years old, ‘preferred to play by himself’ and was ‘particularly interested in astronomy’ (pp. 758-759). The behavior of these children shows a striking similarity with that of the children described by

Hans Asperger (1944) and they would probably be diagnosed with Asperger's disorder or high-functioning autism in present times (Gillberg, 1998).

However, clearly unaware of Hans Asperger's earlier cases, Robinson and Vitale explicitly distinguished these children from autistic children in 'that they have not presented the early infantile incapacity for emotional responsiveness' (Robinson and Vitale, 1954: 760). Furthermore, they 'present a lesser degree of "withdrawal from contact with people" and a lesser measure of the "obsessive desire for the preservation of sameness"' (ibid.). In a response to their article, Leo Kanner himself agrees that autism is a different condition and he adds that the rituals and fixated patterns in autism are qualitatively and etiologically distinct from the circumscribed interest patterns described by Robinson and Vitale. For as far as autistic children exhibit a circumscribed interest, it 'has often been foisted on the children by their parents' (Kanner in Robinson and Vitale, 1954: 766) and is not seen as a core feature of the syndrome.

Debates on the possible causes of autism erupted soon after Kanner's original article was published. Initially, Kanner emphasized that autistic children 'have come into the world with innate inability to form the usual, biologically provided affective contact' (1943: 250). Somewhat later, with the growing psychoanalytic influence in American psychiatry, he came to place a greater emphasis on psychogenic factors like the obsessive traits, emotional coldness and lack of affection that he saw in the parents (Eisenberg and Kanner, 1956). Arguing that 'early infantile autism is a total psychobiological disorder', Kanner often defended a middle course between a psychogenic and an innate biogenic origin of autism, which required 'a comprehensive study of the dysfunction at each level of integration: biological, psychological, and social' (Eisenberg and Kanner, 1956: 564). Various psychiatrists and psychologists argued that autism was mainly a psychogenic disorder (for example, Bettelheim, 1967; Despert, 1951), whereas others regarded autism primarily as an organic brain disorder (Rimland, 1964; Rutter, 1968).

However, discussions on etiology aside, Kanner's first description of autism as a diagnostic entity characterized by extreme emotional withdrawal and tenacious insistence on sameness, remained largely unchallenged for approximately the first two decades after its introduction. Despite discussions on the possible nosological relations between early infantile autism and (childhood) schizophrenia (Kanner, 1965), and despite some unsuccessful proposals to lump autism together with childhood schizophrenia, mental

retardation and organic brain disease under the broad term ‘atypical child’ (see Rank, 1949; Szurek, 1956), it was not until the 1960s that the concept of autism started to change.

Phase II

Language and other perceptual and cognitive abnormalities (1960-1980)

From the 1960s on, as early infantile autism slowly entered the public arena and organized research communities started to replace the earlier observation-based case descriptions with the first epidemiological and experimental studies with autistic children, the concept of autism was altered significantly. Influenced by new types of investigation and new scientific methods, an important shift in emphasis occurred: from severe affective withdrawal as the essential defect in autism, towards language and other cognitive and perceptual abnormalities as essential and primary in autism. Whereas Eisenberg and Kanner (1956: 557) regarded ‘the vicissitudes of language development’ as derivatives of the fundamental disturbance in affective contact, many autism researchers in the 1960s and 1970s not only observed accurately, but also actively tried to engage with the autistic child. They argued that the basic defect in autism was the inability to develop a normal use and understanding of language, in combination with a global defect in the integration of other sensory stimuli. In spite of the popular but barely empirically studied, often vague and contradictory hypotheses that autism had a psychogenic basis, deficits in language, speech and cognition became fundamental features and key characteristics in diagnosing and recognizing autism.

This new way of thinking about autism was backed up by empirical evidence from several new studies and methods in autism research. For instance, the first longitudinal studies on autism (Lockyer and Rutter, 1969) showed that Kanner’s primary disturbance in affective contact and the profound withdrawal tend to lessen considerably as the autistic child grows older, while other symptoms like language deficits and intellectual shortcomings tend to persist. The first systematic psychological experiments with autistic children (for example by Frith, 1970; Hermelin and O’Connor, 1970) that tested intellectual (language and performance), receptive, integrative, and expressive abilities suggested that language and speech problems were not due to profound affective withdrawal or motivational failure, but instead due to a poor understanding of the meaning of spoken words.

Furthermore, these experimental studies showed that the autistic child: had difficulties with the use and understanding of gesture; was particularly unresponsive to verbal stimuli; lacked the ability to associate words semantically; had difficulties with grammatical aspects of language; and made little use of concepts in memorizing. Additional difficulties with the transformation of information from one sensory modality to another and in perceiving temporal patterns in visually presented stimuli 'suggested a central defect in the processing of any sort of coded, meaningful, or temporally patterned stimuli' (Rutter and Bartak, 1971: 27). A new discourse, which was profoundly influenced by the at that time emerging cognitive and computer sciences (see Gardner, 1987) and that made use of terms like codes, processing, stimuli and sensory modalities, became dominant in investigating, recognizing and thinking about autism. Furthermore, in addition to the familiar observations and parents' descriptions of the child's behavior, (neuro)psychological tests and experiments became an integral part of the diagnostic process.

More than a disorder of language, 'the central problem, present in even the most mildly handicapped autistic people, appears to be a specific difficulty in handling symbols, which affects language, nonverbal communication, and many other aspects of cognitive and social activity' (Ricks and Wing, 1975: 214). In a review of concepts of autism, Rutter (1968: 21) explicitly states that 'contrary to earlier views, infantile autism is *not* anything to do with schizophrenia, and is *not* primarily a disorder of social relationships'. Language and cognitive defects were thought to constitute the primary handicap in autism, with 'the social and behavioral abnormalities arising as secondary consequences' (Rutter and Bartak, 1971: 29). Kanner's cardinal resistance to change and insistence on routines were also thought to be secondary and were explained by deficits in processing and integrating visual perceptual information (Wing and Wing, 1971).

This major shift in thinking about autism was reflected in the first formal diagnostic criteria for autism in *DSM-III* (APA, 1980). Besides a 'pervasive lack of responsiveness to other people', the second cardinal criterion became 'gross deficits in language development', and 'if speech is present, peculiar speech patterns such as immediate and delayed echolalia, metaphorical language, [and] pronominal reversal' were central in diagnosing autism (p. 89). The third and final criterion became 'bizarre responses to various aspects of the environment, for example, resistance to change, peculiar interest in or attachments to animate

or inanimate objects' (ibid.: 90). The dominant theory of a basic defect in the use and understanding of language, in combination with a global defect in the integration of other sensory stimuli caused by deficits in processing and integrating perceptual information, reshaped and redefined how autism could be detected, recognized and how the stereotypical autistic child was imagined. In spite of the ambition (of those involved in putting together the *DSM-III*) to produce 'theory neutral' descriptions of observable behavior distinctive for each disorder (Spitzer and Cantwell, 1980), autism's diagnostic criteria undeniably reflected theoretical commitments.

Phase III

Deficits in social cognition and instinct (1980-present)

A second major shift in thinking about autism started somewhere in the early 1980s. I will highlight two events in the autism research field – with Lorna Wing playing a significant role in both of them – that anticipated, influenced and illustrate this second shift.

The first event is one of the first epidemiological studies on autism, which was carried out in London. This study investigated the prevalence of social, language, and cognitive impairments found in intellectually disabled children, and it investigated to what extent these distinct impairments tended to occur together (Wing and Gould, 1979; Wing, 1981b). From a larger sample, 132 children were selected on the basis of exhibiting at least one impairment in social interaction, language development, intellectual functioning, or repetitive stereotyped behaviors. The value of this epidemiological approach, Wing (1981b: 32) argued, 'was that it allowed the examination not only of "pure" syndromes but also of borderline and partial forms, within the context of a larger, geographically defined population'. The children were split into two groups. The first group consisted of sociable children 'in whom social interaction was lively, positive, and a source of great pleasure' and the second group consisted of socially impaired children 'who were inappropriate in their social interaction' (ibid.: 34). This second group consisted not only of socially aloof and unresponsive children, but also included children who interacted passively and 'amiably accepted approaches from others without initiating ... [and children who] made peculiar one-sided approaches to others, approaches that were not adapted to the responses of the person approached' (ibid.). The study subsequently demonstrated that the children with social impairments, the

second group, all had 'repetitive stereotyped behavior and almost all had absence or abnormalities of language and symbolic activities. Thus, the study showed a marked tendency for these problems to occur together' (Wing and Gould, 1979: 25). In contrast, most of the children in the first sociable group had symbolic pretend play, and repetitive activities and language impairment were found in a minority of the sociable and severely retarded children (*ibid.*).

Wing argued that 'the abnormalities of social interaction, verbal and nonverbal communication, and imaginative activities so consistently occurred together ... that they could be referred to as "the triad of social and language impairment"' (1981b: 37) and she suggested that there was no clear division between Kanner's autism and other less severe forms of social impairment. The nature of autism, Wing (1981b: 38) suggested, can only be understood 'in the wider context of the triad of language and social impairment' and Wing and Gould (1979: 25) argued that 'the clustering of the social, language, and behavioral abnormalities ... provided support for the main division into the socially impaired and the sociable though severely retarded groups'. This somewhat circularly justified division between 'socially impaired' and 'sociable' now provided a new basis for categorization, and social impairment became a central distinguishing aspect in the study of autism. Furthermore, social impairment was not understood as 'extreme autistic aloneness' but as a subtle deficit in the use and understanding of the 'unwritten rules of social behavior' (Wing, 1981b: 42).

The second important event was the introduction of the work of the Austrian pediatrician Hans Asperger (1906-1980) into the Anglo-Saxon autism literature. Although Van Krevelen (1971) and a few others had already discussed Asperger's cases of *Autistische Psychopathie* (Asperger, 1944) in English, an article by Wing (1981a) followed by Frith's 1991 translation of Asperger's original cases aroused substantial international interest in what became known as Asperger's syndrome. Based on four cases, Asperger (1944/1991) described a 'particularly interesting and highly recognizable type of child' (p. 37). This type of child exhibited 'the essential feature of ... a disturbance of adaptation to the social environment' (*ibid.*: 87). Apart from this essential feature, Asperger described a variety of behavioral and physical peculiarities in his cases, such as: odd, idiosyncratic or pedantic speech; absence of a sense of humor; little facial expression and limited gestures; an almost 'aristocratic' appearance; an over-sensitivity to criticism; manipulative, vindictive and antisocial acts; difficulties in

learning simple practical skills; an absence of feelings of shame or guilt; hyper- or hyposensitivity for noise; extreme egocentrism; a lack of affection; clumsy movements; single-mindedness, as manifested in peculiar and limited interests; a gift for logical, abstract and original thinking; and more. Despite acknowledging certain similarities, Asperger considered his autistic personality disorder to be basically different from Kanner's autism:

Kanner's early infantile autism is a near psychotic or even a psychotic state, though not identical with schizophrenia. Asperger's typical cases are very intelligent children with extraordinary originality of thought and spontaneity of activity though their actions are not always the right response to the prevailing situation. (Asperger, 1979: 48)

Furthermore, Asperger argued that his children developed highly grammatical speech, while Kanner's children 'generally avoid communication ... [and] do not develop speech or develop it very late' (*ibid.*). In contrast, Wing (1981a) argued that Asperger's cases and Kanner's cases were essentially similar. Despite the variations in terms of severity of impairments, Wing argued that both disorders shared a common and essential characteristic: the impairment of two-way social interaction.³⁴ This impairment:

is not due primarily to a desire to withdraw from social contact. The problem arises from a lack of ability to understand and use the rules governing social behaviour. These rules are unwritten and unstated, complex, constantly changing, and affect speech, gesture, posture, movement, eye contact, choice of clothing, proximity to others, and many other aspects of behavior. (Wing, 1981a: 116)

³⁴ Timimi et al. (2011) highlight the differences between Asperger's cases and Wing's (1981a) case descriptions of children she diagnosed with Asperger's syndrome. For example, most of Wing's cases spoke late, whereas Asperger's cases spoke early. Furthermore, 'most of Wing's cases were described as having little capacity for analytical thought whereas Asperger's cases were thought by him to be highly analytical. None of Wing's cases could be described as manipulative, mendacious, cheeky, confrontational or vindictive (terms of description used by Asperger about his cases)' (p. 61). Timimi et al. (2011) argue that Wing's understanding of Asperger's syndrome was fundamentally different from Asperger's own understanding of the condition he delineated. This created a misleading link between Asperger's and Kanner's cases.

A new way of distinguishing and thinking about autism arose, influenced by: an international introduction of Asperger's work; a growing awareness of the existence of Asperger's new 'type of child'; the recognized and emphasized similarities between Kanner's and Asperger's cases; and by Wing's epidemiological study that argued that certain problems affecting early child development tend to cluster together. From a rare disorder characterized by a pervasive lack of responsiveness and gross deficits in language development, autism became first and foremost a disorder of sociality. The lack of intuitive skills that make complex social interaction possible is now central in yet again a new way of conceptualizing autism. Next to psychiatric and psychological assessments and tests, information on a child's (dys)functioning at school, among peers and in other social activities gets a central position in diagnosing autism.

This way of thinking about autism became more explicit and formalized in the revised version of *DSM-III*. In only seven years, the criteria for autistic disorder in *DSM-III-R* (APA, 1987) changed remarkably compared to the earlier criteria for autism in *DSM-III* (APA, 1980). A 'pervasive lack of responsiveness to other people' (*DSM-III*) changed into 'qualitative impairment in reciprocal social interaction' as, for instance, manifested by 'no or abnormal seeking of comfort at times of distress' or 'gross impairment in ability to make peer friendships' (*DSM-III-R*). Likewise, 'gross deficits in language development' were no longer central to or necessary for an autism diagnosis and were replaced by a new domain of 'qualitative impairments in verbal and nonverbal communication'. This domain included 'marked abnormal nonverbal communication' and 'marked impairment in the ability to initiate or sustain a conversation with others, despite adequate speech' (*DSM-III-R*). Furthermore, whereas *DSM-III* required an onset before 30 months of age, autism lost its adjective 'infantile' and became 'autistic disorder' in *DSM-III-R*. Autistic disorder could now be diagnosed not only during infancy but also during childhood when (latent) more subtle problems with social interaction and communication become visible.

***DSM-5* and the short life of Asperger's Disorder**

Debates about whether Asperger's disorder is a distinct nosological entity, identical with 'high-functioning' autism or part of a broader autism spectrum started right after the publication of Wing's (1981a) influential article (see Macintosh and Dissanayake, 2004). On the eve of the publication of *DSM-IV* (APA, 1994), Rutter and Schopler (1992: 476) argued: 'As there is an obvious research need to compare autism with Asperger syndrome, we suggest that there is a need for a ... category for Asperger syndrome in order to encourage and facilitate that research'. Mainly for research purposes, Asperger's disorder became a distinct DSM diagnostic category in 1994 (Szatmari, 1992). However, the concept of autism had already been influenced by Asperger's 'type of child', and the essential 'disturbance of adaptation to the social environment' (Asperger, 1944: 87) of Asperger's children had become *the* central characteristic of autism in the 1980s.

In order to assess the validity of viewing Asperger's disorder as a distinct category and to compare Asperger's disorder with autistic disorder on multiple characteristics such as course, prognosis, neuropsychological profiles and underlying neurobiological markers, Rutter and Schopler (1992: 476) acknowledged that it would be necessary 'to define the syndrome in such a way that there is no overlap with autism'. However, despite the fact that Michael Rutter was a member of the Work Group responsible for creating criteria for autism and related disorders in *DSM-IV*, criteria for Asperger's disorder and autistic disorder hardly differ in *DSM-IV*. Both disorders require 'qualitative impairment in social interaction' and 'restricted repetitive and stereotyped patterns of behavior, interest, and activities'. The major differences are that for a diagnosis of Asperger's disorder 'there are no clinically significant delays in language' (APA, 1994: 75) and that for autistic disorder 'qualitative impairments in communication' are required. But, as we have seen, delays in language were no longer at the core of or necessary for a diagnosis of autistic disorder, and most people who meet criteria for Asperger's disorder also show 'marked impairment in the ability to initiate or sustain a conversation' sufficient to meet the impairments in communication criteria for autistic disorder. Because of this significant overlap of the two disorders, in combination with 'the precedence rule: diagnose Asperger disorder only if criteria for autistic disorder are not met' (Happé, 2011: 541), several autism researchers even concluded that a diagnosis

of Asperger's disorder is impossible using *DSM-IV* criteria (Mayes et al., 2001; Szatmari et al., 1995).

The problems with applying the *DSM-IV* Asperger's disorder criteria resulted in wide variation in how the term Asperger's disorder was, and still is, used in clinical practice and research (Happé, 2011). Lord and colleagues (2012) recently argued that the best predictor of an Asperger's disorder diagnosis is not the characteristics of the individual, but the specific clinic the individual goes to. In defense of the decision to fold Asperger's disorder into the new category of *autism spectrum disorder* (ASD), Work Group member Happé explains that 'There is no evidence of differential treatment response or etiology to date, and claims for a distinct neurocognitive profile in Asperger disorder have received mixed results' (Happé, 2011: 540). Happé concludes that 'there is little evidence to support the current diagnostic distinction between Asperger's disorder and high-functioning autism' (Happé, 2011: 541).

However, as the history of the concept of autism reveals, even before Asperger's disorder became an official diagnostic category in the 1990s, Hans Asperger's 'autistic psychopathy' and Lorna Wing's interpretation of it, influenced a shift in thinking about autism in the early 1980s. Wing (2005: 198) later acknowledged that she 'always considered Asperger's syndrome to be part of the autistic spectrum. It shares the impairments of social interaction, social communication and social imagination and the repetitive pattern of activities and interests that characterize the spectrum'. It is not just the lack of empirical 'evidence to support the diagnostic distinction' between autistic disorder and Asperger's disorder, but this earlier broad interpretation of autism as 'the absence or impairment of the social instinct' (ibid.: 201) that made the separate category of Asperger's disorder scientifically fragile. The impossible task of contrasting a new category with an already very broad conceptualization of autism made defining Asperger's disorder as a distinct diagnostic category rather prospectless even before it officially existed. Additionally, the seemingly recent introduction of an 'autism spectrum' is not as new as it might seem, but just a new term for an already accepted and established understanding of autism as a wide and heterogeneous disorder of social contact.

The *DSM-5* Work Group for neurodevelopmental disorders might make history repeat itself by the introduction of, next to ASD, a new category of *Social Communication Disorder* (SCD). Even though history is often unsuited for predicting future developments, the introduction of this category resembles the

introduction of Asperger's disorder in *DSM-IV* and PDD-NOS in *DSM-III-R*. As a residual category for those who did not fully fit the autistic disorder category, PDD-NOS was thought 'to describe the (*very rare*) children who appear to merit special diagnostic notation' (Cohen et al., 1986: 217, emphasis added), but PDD-NOS became far more commonly diagnosed than autistic disorder (Chakrabarti and Fombonne, 2001). Today, the *DSM-5* will include SCD 'to describe the *rare* individuals who display significant social/communication impairments of ASDs without restrictive/repetitive behaviors' (Mahjouri and Lord, 2012, emphasis added). However, Lord argues elsewhere that 'How many individuals fall into this group is not clear ... No data are yet available about its reliability, validity or prevalence' (Lord and Jones, 2012: 499). It is unclear if and how SCD is different from ASD, for instance, in terms of etiology, symptom profile and management. Furthermore, as Ozonoff (2012) pointed out, it seems 'logically and internally inconsistent for the *DSM-5*, so pioneering in its dimensional approach, to introduce a *separate category* that is so qualitatively similar to another condition' (original italics). The way in which SCD enters the *DSM-5* is highly reminiscent of the way in which Asperger's disorder entered *DSM-IV*, and I will not be surprised if it becomes far more commonly diagnosed than ASD and if it then – just like Asperger's disorder – disappears again to be incorporated into the qualitatively similar autism spectrum.

Conclusions

This historical analysis of the concept of autism might not be directly helpful for a deeper understanding of *why* ideas about autism have changed, but it is a necessary first step towards such an understanding as it illustrates how autism as an object of scientific inquiry and clinical practice evolved and mutated. More than just a broadening of the concept or an inclusion of milder forms of an essentially similar deficit, what is considered essential in autism has gone through major changes, from profound affective withdrawal and aloofness, to language and other perceptual and cognitive abnormalities, to deficits in social cognition and intuition. In addition, the rise and fall of Asperger's disorder is not an inevitable result of scientific scrutiny, but deeply bound to earlier conceptualizations of autism and Asperger's disorder.

Contrary to the assertions of ‘practitioner-historians’ who argue that ‘Ever since Kanner’s first descriptions in 1943 ... there has been agreement on the core symptoms’ (Sponheim, 1996: 513), this history makes room for unacknowledged discontinuities and irregularities, which are often found in the same canonical texts that are used for the positivist and essentialist narrative of autism. In this narrative, a sense of progress indirectly legitimizes current understandings, research directions, and decisions concerning classifying autism in *DSM-5*, while conceptual discontinuities and earlier complexities and disputes concerning classifying and delineating autism are left out. One tends to see and describe those aspects of earlier accounts of autism that corroborate current views of autism and thereby confirm the validity of autism as a recognizable disease characterized by a particular essential or core deficit.

However, in describing the reshaping of the concept of autism, the historicity, provisionality and plurality of knowledge and truth about autism becomes apparent. As a consequence, such a historical reflection destabilizes the present ‘truth’ about autism as a neurodevelopmental spectrum disorder of social cognition localized in an individual’s brain, and as it destabilizes the present ‘truth’ it creates space for other possible perspectives and conceptualizations of autism in the present and future. The point is, however, not to dismantle the very idea of autism, but merely to correct a positivist and essentialist understanding of autism as a discrete and stable entity in nature that we get to know and understand better and better as science progresses and knowledge accumulates.

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4 | Stabilizing autism³⁵

One cannot speak of anything at any time; it is not easy to say something new; it is not enough for us to open our eyes, to pay attention, or to be aware, for new objects suddenly light up and emerge out of the ground. (Foucault, 1972: 44f)

It is also inadequate to define syphilis phenomenologically rather than conceptually, in the manner that animals and plants might be defined on the basis of their characteristics. For it is naïve to think that, although its historical development has been tortuous and complicated, we can today arrive at the concept of the disease entity “syphilis” simply and safely merely by using current techniques of observation and experiment. (Fleck, 1935/1979: 21)

Abstract

Using the conceptual tools of philosopher of science Ludwik Fleck, I argue that the reframing of autism as a neurodevelopmental spectrum disorder is constrained by two governing ‘styles of thought’ of contemporary psychiatry. The first is the historically conditioned ‘readiness for directed perception’ of, and thinking in terms of, ontologically distinct diseases. The clinical gaze of mental health professionals, the bureaucratic needs of health administration, the clinical and scientific utility of disease categories, and the practices of autism-oriented advocacy groups all imply a bias toward thinking about autism and related disorders as ontologically distinct psychiatric and scientific entities. Second, within the ‘neuromolecular style of thought’, mental disorders are more and more located at the neurobiological levels of the brain. In autism research, one of the biggest challenges is the identification of autism’s neurobiological

³⁵ This chapter has been published as Verhoeff B (2014) Stabilizing autism: A Fleckian account of the rise of a neurodevelopmental spectrum disorder. *Studies in History and Philosophy of Biological and Biomedical Sciences* 46: 65-78.

singularity. However, at a moment when biological and categorical approaches toward autism face serious empirical difficulties, a balance is established that holds together these two styles of thought. With a need to account for some of the most persistent uncertainties and conflicts in autism research, namely ubiquitous heterogeneity and a failure to identify disease specific biomarkers, the reframing of autism as a neurodevelopmental spectrum disorder satisfies the scientific, institutional and socio-political needs for stability and homogenization.

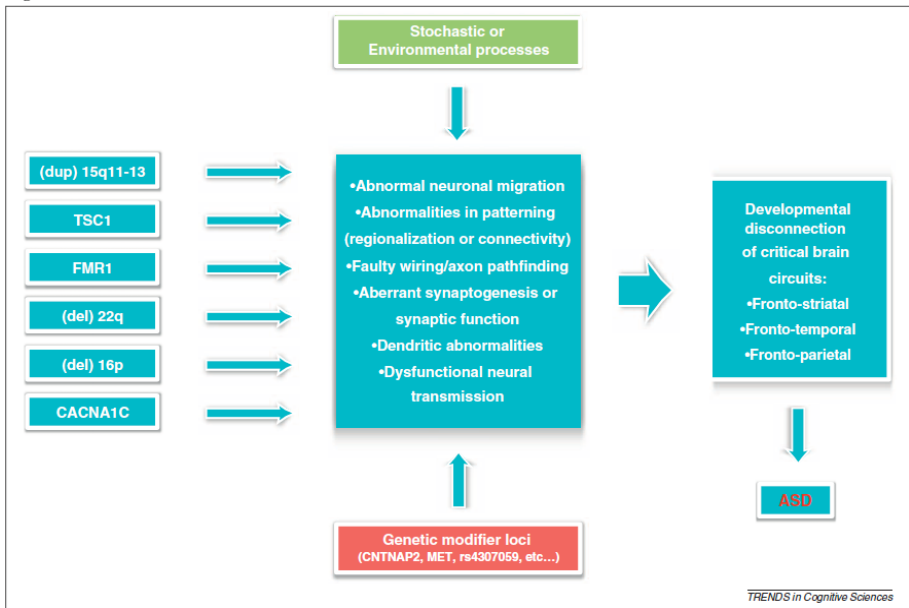
Introduction

Today, autism is understood to be both a ‘spectrum’ disorder and a ‘neurodevelopmental’ disease of the brain – at least by leading autism experts and those who are responsible for organizing the criteria and categories of autism and related disorders in the latest version of the *Diagnostic and Statistical Manual for Mental Disorders (DSM-5)*. According to one of the members of the *DSM-5* Neurodevelopmental Disorders Work Group, ‘the term *autism spectrum disorder* ... reflects current widespread consensus that autism is best considered as existing on a spectrum with variable manifestations across life span, gender, and intellectual level and/or language ability’ (Happé, 2011: 540). In *A Parent’s Guide to Autism Spectrum Disorder (ASD)*, the American National Institute of Mental Health (NIMH) summarizes that ‘the term “spectrum” refers to the wide range of symptoms, skills, and levels of impairment, or disability, that children with ASD can have’ (NIMH, 2011: 1). In moving from representing autism as a single disorder to autism as a spectrum disorder, the biological and hereditary nature of autism remains unchallenged. However, the earlier search for a discrete ‘autism-gene’ (Bonora et al., 2003) or a distinct deficit in a tentative cognitive module (Baron-Cohen et al., 1985) nowadays appears naïve. In line with the spectrum approach, more complex images of the etiology and pathophysiology of autism have replaced ‘simple’ essentialist notions of autism.

Besides a spectrum disorder, autism has become a *neurodevelopmental* disorder in which multiple deficits in the growth and development of the brain are assumed to underlie the diversity of abnormalities in social interaction and mental flexibility. Genes still play a role, but no longer as central determining factors. Several dozen or maybe even a hundred susceptibility genes are thought

to be involved – via the different molecules they express and in interaction with each other and environmental processes – in the disturbance of larger biological networks and pathways responsible for neuronal motility, axon guidance and the regulation and formation of synapses (see, for example, Voineagu et al., 2011). As Geschwind (2011) explicitly illustrates in a working model (see Figure 1), deficits in neurodevelopment and the neuroscientist's laboratory have become 'obligatory passage points' (Callon, 1986) for producing truths about autism.

Figure 1.



This model illustrates how autism risk alleles, such as those shown at the left, and environmental factors act on systems of neurodevelopment and lead to autism. Geschwind (2011: 413) argues that although gene expression and proteomic molecular studies identify molecular and biological pathways that provide a source of convergence, 'the ultimate convergence must lie in neural systems'. At this neurodevelopmental level, 'the convergent process will likely be disconnection of the circuits outlined in the far right box because these systems are thought to underlie the core deficits of ASD'. Adapted with permission from Geschwind (2011).

However, while medical and scientific authorities emphasize that autism is a neurodevelopmental spectrum disorder, other kinds of experts in the social sciences analyze autism – its emergence, treatments, theories, institutions and conceptual transformations – from a social, cultural and historical perspective (for example, Eyal et al., 2010; Silverman, 2011). Most social studies of autism do not deny the seriousness or biological basis of the conditions that are labeled as autism (see, for example, Nadesan, 2005; Grinker, 2007; Eyal et al., 2010), but focus primarily on socio-cultural, economic and political factors that are involved, for instance, in the recognition, spread, interpretation and remediation of autism. Eyal et al. (2010), for example, acknowledge that autism is usually explained in biological terms, but – in their analysis of the ‘autism epidemic’ – they focus mainly on social factors and cultural changes in medical practice such as the reorganization of expertise, processes of deinstitutionalization of mental retardation, parental activism and an increase in the availability of services. Like most other social autism studies (for example, Nadesan, 2005; Grinker, 2007), Eyal et al.’s study does not integrate specific scientific practices and theoretical content into (social) explanations and understandings of conceptual transformations of the scientific object of autism and, according to Fitzgerald (2012: 52), ‘the authors situate their account exactly on the fault-line of biological and cultural explanation’.³⁶

In these social histories of autism, cultural change and scientific explanations are generally approached separately, and the specific ways in which scientific practices and developments are socially and historically situated and intermingle with social and institutional developments remain rather unanalyzed. This is not the place to discuss whether this is a significant limitation of these particular histories of autism. However, as this chapter argues, in order to understand certain aspects of the recent history of autism – in particular, the rise of a new view of autism as a ‘neurodevelopmental spectrum disorder’ – one needs to combine attention to developments, aims, challenges and failures of contemporary biomedical autism research with an

³⁶ In a series of interviews with autism neuroscientists, Fitzgerald (2012) is particularly intent on taking autism neuroscience seriously. However, as he acknowledges, his ‘unglamorous empirical sociology’ stays at the level of ‘neuroscientists *own* accounts of their daily assumptions and practices’ and does not try to explain changes in the conceptualization of autism (p. 210, emphasis in original).

investigation of the historical and cultural matrices in which autism as a medical category emerged and currently circulates.³⁷

Drawing upon the work of Ludwik Fleck (1935/1979) – who is viewed today as a pioneer of the historical and sociological approach to the production of scientific knowledge (Fagan, 2009; Hedfors, 2007; Löwy, 2004a) – and drawing upon contemporary studies inspired by Fleck (for example, Löwy, 2004a; 2004b; Rose and Abi-Rached, 2013; Rosenberg, 2003), I will propose a theoretical framework for an integrated account of the current views of autism. Fleck's notions of thought styles, thought collectives and passive and active elements of knowledge are especially well adapted to describing 'medical facts' (such as those of contemporary autism research) that is 'entities that are developed and stabilized through multilevel interactions and circulation in heterogeneous networks, that are dynamic and historical, and that are at the same time strongly material and cultural' (Löwy, 2004a: 443). Using Fleck's conceptual tools, this chapter aims to understand the rise of a neurodevelopmental spectrum disorder in a way that neither ignores the 'hard residue of material reality' (or 'passive elements of knowledge'), nor ignores the importance of historical, social and institutional ('active') elements of and constraints on thinking about autism (Fleck, 1979).

The third section of this chapter, which follows this introduction and some general remarks, briefly discusses the central concepts introduced by Fleck. Instead of providing a neat way to disentangle scientific from cultural aspects of current understandings of autism, Fleck's conceptual tools enable the development of a cohesive account of the rise of a neurodevelopmental autism spectrum disorder. In this account the modified concept of autism becomes a solution for particular persistent uncertainties and conflicts in contemporary

³⁷ An interest in the material practices of biomedicine and medical cultures is certainly not new for historians or anthropologists of medicine and, according to Löwy (2011), is at least thirty years old. On the 'practice turn' in the history of medicine, see also Pickstone (2011). Löwy (2011: 122) furthermore points out that this 'practice turn' in the history of medicine and the adoption of methods of inquiry of historians and sociologists of science and technology came at a cost, namely a neglect of an earlier generation of historians of medicine. In general, she argues, 'we know much more about biomedical "laboratory life" than about the life of the clinics' and in a plea for 'more medicine into biomedicine studies', she suggests involving theoretical insights of scholars from earlier generations, such as Fleck, Temkin, Canguilhem and Foucault. Using the theoretical insights of Fleck, I hope this contemporary history of autism heeds Löwy's call for more medicine into (the history of) biomedicine.

autism research, namely ubiquitous heterogeneity and a failure to identify specific biomarkers for autism. This solution, I argue, which involves a reworking of the very object of explanation, must be understood as closely bound to and resulting from two organizing and constraining ‘styles of thought’ of contemporary Western psychiatry. The first ‘style of thought,’ which will be discussed in the fourth section, is the self-evident way of medical thinking in terms of ontologically distinct disease entities. This style of thought plays an important role in connecting, integrating and facilitating scientific, diagnostic, therapeutic, and bureaucratic practices. Something similar to what others have called the biological or ‘neuromolecular’ style of thought (Rose and Abi-Rached, 2013) will be discussed in the fifth section as a second central aspect of contemporary Western psychiatry. Before all that, I want to make some general remarks.

Why autism?

The recent shift in representing, classifying, studying and treating autism as a neurodevelopmental spectrum disorder should not be imagined as an autism-specific, unique or isolated phenomenon. Similar new ways of investigating, conceptualizing and categorizing psychiatric conditions in terms of neurodevelopment and spectra have been progressively emerging over the last decade. An increase in the number of scientific articles about the psychosis spectrum, the bipolar spectrum, the obsessive-compulsive disorder spectrum (see, for example, Ruhrmann et al., 2010), and new models that describe ADHD, depression, anxiety and schizophrenia (see, for example, Lewis and Levitt, 2002) as neurodevelopmental disorders indicate a broader transformation in thinking about psychiatric disorders.³⁸

³⁸ In addition, the spectrum concept is certainly not new in psychiatry. Kety et al. (1968), for instance, used the spectrum metaphor to introduce a ‘schizophrenia spectrum of disorders’ that linked patients diagnosed with schizophrenia with ‘uncertain schizophrenia’ and ‘inadequate personality’ observed in biological relatives. Even earlier, Kretschmer (1921) proposed dimensional models for schizophrenia (schizothymic, schizoid, schizophrenic) and for affective disorders (cyclothymic temperament, cycloid psychopathy, manic-depressive disorder) to emphasize a hypothetical common pathophysiology. Furthermore, levels of severity (mild, moderate,

However, autism does make a special case that is worth exploring in detail. As the first DSM category with the word ‘spectrum’ in its name (APA, 2013) and the most researched and funded neurodevelopmental disorder (Bishop, 2010), autism is an important forerunner in this broader shift and can serve as a paradigm case for understanding recent developments in psychiatry.³⁹ As we will see, psychiatry is on the one hand fragmented and mainly organized around specific disease categories, but on the other hand, there are general tendencies or ‘styles of thought’ in psychiatry and medicine that transcend the level of individual disease categories. A focus on autism reveals and demonstrates the dynamics between these more abstract levels of general psychiatric thought and the concrete level of disease specific research and practice. Although these dynamics and conceptual developments are not specific for autism, thinking in terms of a spectrum is most accepted and stabilized – compared to other mental disorders – in both professional and lay perceptions of autism. In addition, autism’s highly developed neuroscientific research makes it possible to explore with autism how general psychiatric and medical styles of thought are reflected in cutting-edge psychiatric research, and how, in turn, specific developments within the advanced field of autism affect psychiatry at large.

Furthermore, despite a general tendency in medicine towards ‘personalized medicine’ (see Hamburg and Collins, 2010), a persistent use of broad categories like obesity and coronary heart disease (see, for example, Jones, 2013), which gather heterogeneous phenomena, could be seen as comparable to the recent spectrum approach in psychiatry. These broad disease categories have their own historical, material, professional and organizational constraints and, comparable to the spectrum categories in psychiatry, they also function as intermediaries between several communities of practice in order to maintain a shared identity across sites (Bowker and Star, 1999).⁴⁰ Yet, in contrast with the

severe) could already be specified for several disorders (for instance, depression and intellectual disability) in all earlier DSM editions.

³⁹ There have also been earlier dimensional and spectrum concepts related to autism (for example, Rutter, 1968; Wing and Gould, 1979; Wing, 1996). How they are both similar to and importantly different from more recent thinking in terms of the ‘autism spectrum’ will be discussed in more detail in part 6, ‘Rise of a neurodevelopmental spectrum disorder’.

⁴⁰ Bowker and Star would call these categories ‘boundary objects,’ as they ‘inhabit several communities of practice and satisfy the informational requirements of each of them.... Such objects have different meanings in different social worlds but their

spectrum categories, the unity of these broad and heterogeneous categories is somewhat less puzzling and found in ‘straightforward’ phenotypes (a Body Mass Index of more than 30 kg/m² in obesity, or blood pressure above 100-140 mmHg in hypertension) or in a particular organ that is affected (heart coronaries in coronary heart disease). Whereas, the meaning of a ‘spectrum’ in psychiatry and what it is that keeps a spectrum category together is very ambiguous. As we will see, unlike the broad categories in general medicine and besides being intermediaries between several communities, an important and specific function of the idea of an autism *spectrum* is legitimizing and stabilizing the continuous search for autism’s neurobiological foundation and to hold the heterogeneous (and fuzzy) category of autism together as a valid, researchable, clinically useful and convincing diagnostic entity.

Moreover, this Fleckian approach to autism should not be read as a critical attack or *normative* evaluation of the neurodevelopmental spectrum idea of autism, or of the autism research field in general. Nor is it a proposal for an alternative model to conceptualize and investigate autism. Instead, it aims to understand the recent history of autism in a novel way that combines cutting-edge scientific developments with social, historical and institutional determinants and constraints in a cohesive account. Nevertheless, in revealing a particular way in which ‘biomedical facts’ about autism and the very idea of autism itself came into being and developed, this analysis does open up space for thinking about the intersections between ‘the clinic,’ ‘the lab,’ and society and how they might be connected in future autism research and clinical practice.

Ludwik Fleck

In his pioneering study, *The Genesis and Development of a Scientific Fact* (1935/1979), Fleck offers an account of scientific facts not as things to be discovered or revealed, but as products of scientific practices, technologies and historically and socially preconditioned ways of seeing and thinking. Fleck’s epistemology concerned the acquisition of scientific knowledge, and his history of syphilis and the Wasserman reaction illustrates the dense social, material and

structure is common enough to more than one world to make them recognizable, a means of translation’ (Bowker and Star, 1999: 297).

cognitive network in which scientific knowledge arises, stabilizes and changes. According to Fleck, science and its products are fundamentally rooted in a specific and unique history. Within a particular scientific field or ‘thought collective’ – defined by Fleck as ‘a community of persons mutually exchanging ideas or maintaining intellectual interaction’ (ibid.: 39) – tradition, education, and familiarity produce a ‘*readiness for stylized (that is, directed and restricted) perception and action*’ (ibid.: 84).

This readiness for directed perception, for which being educated and experienced within a particular scientific field is epistemologically fundamental, is ‘the main constituent of [a] thought style’ (ibid.: 92). Fleck illustrates how the production of scientific knowledge is determined by and can only arise in a context of collectively structured background knowledge. Ways of making observations, shaping and establishing the very object of explanation, defining the set of problems, and using specific methods, techniques and languages are all constrained and appear inevitable within a particular style of thought. Furthermore, besides historical and socio-cognitive constraints on scientific thought, an additional source of ‘resistance’ to ‘the free unfolding of ideas’ (Fleck, 1979: 84) arises due to intrinsic properties of the material world:

[T]here are always other connections which are also to be found in the content of knowledge that are not explicable in terms either of psychology (both individual and collective) or of history. For this very reason these seem to be “real,” “objective,” and “true” relations. We call them the passive connections in contrast with the others which we call active. In our history of syphilis the combination of all venereal diseases under the generic concept of carnal scourge was thus an active association of the phenomena, explained in terms of cultural history. In contrast, a restriction of the curative effect of mercury ... that “sometimes mercury does not cure the carnal scourge but makes it even worse” represents a passive association with respect to the act of cognition. (Fleck, 1979: 10)

Obviously, Fleck’s example of a passive connection can only be formulated using the concept of carnal scourge that contains passive as well as active elements. Fleck recognizes that the ‘passive resistance’ of material elements of the natural world and the ‘active resistance’ of scientific thought created by

historical, social and psychological connections ‘cannot be separated from each other completely either logically or historically’ (ibid.: 95). Furthermore, ‘the more developed and detailed a branch of knowledge becomes, the smaller are the differences of opinion’ (ibid.: 83) and the greater the number of active and passive connections will be. As a result, a dense network of active and passive connections that constitutes a particular style of thought seems to achieve a kind of inevitable stability, in which conventionalism is considerably restricted and the active elements – ‘as a result of education and training as well as through [the scientists] participation in the communication of thoughts within his collective’ (ibid.: 141) – become invisible. At this point, a ‘tendency to reify and objectivize the conceptual creations of scientific thought arises’ (ibid.: 144), and a ‘resistance to anything that contradicts [the closed system of knowledge]’ helps to guard a kind of ‘harmony of illusions’ (ibid.: 27).

However, change in style of thought, that is change in readiness for directed perception, can be generated by communicative interactions within and between thought-collectives: ‘communication never occurs without a transformation, and indeed always involves a stylized remodeling, which intracollectively achieves corroboration and which intercollectively yields fundamental alteration’ (ibid.: 111). According to Fleck, thought collectives have an internal interactive structure made up of an ‘esoteric’ circle of experts and specialists, and an exoteric circle of ‘educated amateurs’. Once scientific facts are stabilized within the inner ‘esoteric’ circle of a particular thought collective, they often move outside and encounter ‘other scientific communities, but also other social groups: users of science, practitioners, politicians, the public at large. This is a bi-directional process. Proto-concepts originating in society at large, such as ‘syphilitic blood’, affect the development of scientific facts, while such facts in turn influence society and culture’ (Löwy, 2004b: 513).

Fleck’s approach makes it possible to analyze the reframing of autism as a ‘neurodevelopmental spectrum disorder’ as a complex dynamic phenomenon rooted in multilevel interactions between ‘passive elements’ – such as the structure of neural pathways and the heterogeneity of biomarkers – and ‘active elements’, like the cultural history of psychiatry as a medical profession or historical modes of classifying mental disorder.

Distinct disease pictures: the contemporary style of medical thought

Within medicine and the medical sciences, Fleck (1927/1986) argued,

[A]bnormal morbid phenomena are grouped round certain types, producing laws of a higher order, because they are more beautiful and more general than the normal phenomena which suddenly become profoundly intelligible. These types, these ideal, fictitious pictures, known as morbid units, round which both the individual and the variable morbid phenomena are grouped, without, however, ever corresponding completely to them – are produced by the medical way of thinking, on the one hand by specific, far-reaching abstraction, by rejection of some observed data, and on the other hand, by the specific construction of hypotheses, i.e. by guessing of non-observed relations. (Fleck, 1927/1986: 40)

With the assumption that the development of science is only a matter of time, technical possibilities and accident, Fleck continues, we would never be able to grasp ‘why a phenomenon which is accessible to everybody had been observed at the given moment for the first time, and even almost simultaneously by several researchers’ (ibid.: 41). For instance, despite the repeatedly emphasized recognizability and distinctiveness of autism (see, for example, Frith, 1989; Kanner, 1965; Volkmar, 1998), the clinical unit of autism emerged markedly recent (in 1943). Yet, many pre-Kannerian historical figures, eccentric geniuses and religious figures, such as Newton and Michelangelo, have been retrospectively diagnosed as cases of autism or Asperger’s disorder (Fitzgerald, 2005).

Furthermore, Hans Asperger – separated from Leo Kanner by an ocean and a war – introduced his ‘autistic psychopathy’ only one year after Leo Kanner introduced his fairly similar ‘early infantile autism’ (Asperger, 1944).⁴¹ For Hacking (2006), this was certainly not a coincidence as Asperger and Kanner,

⁴¹ I will not go into detail about the particular cultural, social and political conditions that made the specific emergence of autism possible (see, for instance, Nadesan, 2005), but the term ‘autism’ was coined by Swiss psychiatrist Eugen Bleuler around 1910 as one of the symptoms of schizophrenia. By the time Kanner introduced his new diagnostic entity, autism was a term with wide use in German psychiatry (see Evans, 2013; Nadesan, 2005; Verhoeff, 2013/Chapter 3).

although they never met nor corresponded, came from the same medical culture – the same ‘esoteric circle’.⁴² Without the idea that distinct scientific periods possess specific styles of thinking, the rise and development of certain definite clinical units would become unintelligible. However, thinking in terms of ontologically distinct diseases is ‘by no means the only logical possibility. As history shows, it is feasible to introduce completely different classifications of disease’ (Fleck 1979: 21).

From idiosyncrasies to distinct disease entities

The insightful work of historian of medicine Charles Rosenberg reveals some of these ‘completely different classifications of disease’. He illustrates that the perception that corporeal or psychological pain and suffering can and should be thought of as specific disease entities existing outside their unique appearances in particular individuals did not become pervasive until the late nineteenth century (Rosenberg, 2006).⁴³ Before this period, disease was not imagined as a set of different entities, each with specific signs and symptoms, and a specific etiology, course and pathophysiology. Instead, disease was a variable physiological state of the individual patient that resulted from ‘a cumulative interaction between constitutional endowment and environmental circumstances’ (Rosenberg, 1977: 487). Owsei Temkin, an earlier important historian of medicine, called this the ‘physiological’ understanding of disease, distinct from the later ‘ontological’ understanding of diseases as existing

⁴² Hacking analyzes the development of human categories like multiple personality disorder (Hacking, 1995), obesity and also autism (Hacking, 2007) from a particular perspective. With his term ‘interactive kinds’ he attends to the way in which people react to being classified and described, and how, as a result of these reactions, the very people and the classifications that are supposed to cover them go through a process of alteration. Eyal et al. (2010) use Hacking’s feedback loop to explain the rise in people diagnosed with autism. In these accounts, the contribution of particular scientific content and styles of thought to the development of human categories remains largely unanalyzed.

⁴³ This does not imply that there are no earlier examples of hypothetical disease entities. Even psychological ailments had been viewed as conditions such as melancholy or hysteria and ‘humeral explanations of temperamental peculiarity are as old as Western medicine itself’ (Rosenberg, 2006: 412). However, the disease concepts they brought into play were fundamentally different from those that became habitual by the end of the nineteenth century. See Thomas Sydenham’s *Epistolary Dissertation on the Hysterical Affections* of 1682 as an earlier attempt at classifying forms of madness.

independently of their unique appearances in particular individuals (Temkin, 1977).

In the former period, the body, health and disease were understood in holistic terms of equilibrium, physiological adjustment and vital powers, in which food, water, air, climate, living conditions and even morals played a necessary and irreducible role. Disease was understood in individual rather than general terms and 'health was a consequence of a symbiotic relationship between nature, society and the individual' (Grob, 1998: 192).⁴⁴ Clinical treatment and physicians' importance was largely independent of any nosological system and involved the ability to recognize and deal with the multitude of environmental circumstances and the idiosyncrasies of each patient.⁴⁵

This perception of disease gradually changed around 1870 with a new emphasis on diseases as discrete entities with specific underlying mechanisms. Earlier nineteenth century postmortem pathology, the clinical use of 'instruments of precision' such as the thermometer, blood and urine chemistry and microscopy, and the growing status of what we now call the biomedical sciences (histology, physiology, biochemistry) all contributed to this new ontological way of thinking about disease. A lesion-based notion of disease, and later germ theories of infectious disease in combination with new forms of knowledge arising from bacteriology and laboratories of biochemists and physiologists, reinforced the idea that diseases could be delineated in precise and measurable terms. In addition, and of particular importance for psychiatry, 'the late-19th-century vogue for heredity and evolution constituted another significant factor, linking biology and behavior, mind and body, past and present ... Like germ theory, heredity provided ... a reassuringly somatic mechanism with which to explain a variety of unsettling emotions and problematic behaviors' (Rosenberg, 2006: 413). Alcoholism, neurasthenia, hypochondria, anorexia, homosexuality and kleptomania all emerged as distinct

⁴⁴ Even something as widespread and terrifying as the yellow fever epidemic of 1793 in Philadelphia 'could be construed as the consequence of a peculiarly tainted microenvironment – presumably something in the atmosphere – coupled with an individual idiosyncrasy, which explained why some succumbed, some recovered, and others never fell ill during a local outbreak' (Rosenberg, 2007: 3).

⁴⁵ The regulation of secretions – by extracting blood or promoting urination, defecation or perspiration – in order to recover a damaged equilibrium, was the physician's most powerful therapy.

diseases by the end of the nineteenth century, and heredity seemed a determining factor, rather than one of the many interacting individual and environmental factors that determined health and disease in earlier times.⁴⁶

Autism as a distinct disease

Part of this new style of medical thought was Emil Kraepelin's very influential system of classification of psychiatric disorders, published in the 6th edition of the *Lehrbuch der Psychiatrie* (1899). Kraepelin, often regarded as the founder of modern psychiatry, introduced 16 categories based on patterns of symptoms with each containing a particular 'essence' and (in theory) specific biological disturbances (Berrios and Hauser, 1988).⁴⁷ There is, however, no clear historical path from Kraepelin's biomedical model of nosological entities to the contemporary 'neo-Kraepelinian' era of biological psychiatry and DSM-based education, clinical practice and research. Throughout the twentieth century, the ontological understanding of disease had not been as steady and monolithic in psychiatry as it was in the rest of medicine. For instance, Adolf Meyer's 'mental reaction-types' and his holistic 'psychobiological' understanding of human behavior (Meyer, 1908), Freudian psychoanalytical theories, and Karl Menninger's emphasis on interpersonal, social, and environmental factors in personal maladjustment (Menninger, 1963) were all very influential perspectives that were not based on disease entities and explicitly opposed the Kraepelinian nosology of distinct diseases of organic or hereditary origin.

Even though holistic and psychodynamic approaches were never completely hegemonic – for much of the twentieth century, American psychiatry was a divided and ambivalent specialty (Grob, 1998) – nosological systems were of minor importance in American psychiatry until the late 1970s. Closer to an individual understanding of disease and in the spirit of Meyer's 'genetic-dynamic' framework, which 'shaped several generations of American psychiatrists,' (ibid.: 202) American psychiatrists were generally more 'interested

⁴⁶ Social and political aspects such as managing deviance and rationalizing health policies and the relation with this idea of distinct disease entities are not discussed here, but these aspects undoubtedly played a role in the emergence of this new medical way of thinking. I will pay some attention to this in discussing autism in relation to regulatory practices.

⁴⁷ There is, of course, much more that can be said about the emergence of academic psychiatry in the late nineteenth century. See, for example, Berrios and Porter (1995) and Berrios (1996).

in the person and his life experiences rather than, like so many of our continental colleagues, primarily in a disease process' (Lidz, 1966: 321).

Remarkably, it was within this Meyerian and psychodynamic, anti-Kraepelinian psychiatric culture that Leo Kanner, who was appointed by and worked together with Meyer himself at the Johns Hopkins Hospital in Baltimore (Neumärker, 2003), proposed the distinct diagnostic entity of 'early infantile autism' on the basis of descriptions of the behavior of 11 children with similar symptoms. Very much against the established psychoanalytic style of thinking and theorizing about psychological problems in children in terms of unconscious thought processes, libidinal instincts and destructive impulses and fantasies (Evans, 2013), Kanner's descriptive approach in child psychiatry was clearly Kraepelinian. He presented autism as 'a "unique syndrome," not heretofore reported' (Kanner, 1943: 242) and he hypothesized that children with autism came 'into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps' (*ibid.*: 250).

The fact that Kanner had been educated in Berlin within the intellectual and institutional milieu of German medicine (Neumärker, 2003)⁴⁸ and that he was very well aware of Kraepelin's work and nosological system, might have played a role in his apparently anachronistic perception and introduction of a new disease entity. A few years after this introduction, he confidently argued that 'now that early infantile autism has a well-defined symptomatology and the syndrome as such can be recognized with relative ease, it is ready to apply for a place in the existing psychiatric nosology' (Kanner, 1949: 416). With an explicit reference to Kraepelin's scientific psychiatry, the major challenge for his distinct syndrome became 'to find a common denominator' and to identify 'the intrinsic nature of the condition as related or unrelated to the intrinsic nature of other conditions' (*ibid.*: 416-417). However, at least until the 1960s, many professionals in the field of child psychology and psychiatry were not quite ready to adopt this 'new' style of medical thinking about autism as a distinct disease entity. Instead of delineating syndromes and discovering the underlying nature of distinct diseases, they continued to employ psychodynamic terms such as 'autoerotism, primary narcissism and symbolic thinking to understand

⁴⁸ In search of better professional opportunities, Kanner moved to the USA in 1924 when he was 29 years old (Neumärker, 2003).

infantile psychopathology and problems with developing relationships' (Evans, 2013: 10).

Autism slowly started to settle as a recognizable syndrome in the 1960s and 1970s. A growing need for epidemiological studies and new experimental and neuropsychological tests required reliable behavioral descriptions (*ibid.*). Kanner's earlier descriptions became the central point of reference for further empirical studies (see, for example, Lotter, 1966). The term autism was less and less used to refer to the (psychoanalytically interpreted) symptom of disturbance in engagement with external reality, and instead came to stand for the entire syndrome that Kanner delineated. Again in line with the Kraepelinian tradition and Kanner's initial project, autism research became mainly directed at unraveling the 'intrinsic nature' and 'primary defects' of the disorder (Rutter, 1968). The arrival of *DSM-III* (APA, 1980) marked a definite neo-Kraepelinian turn, and autism was included as a distinct category in the new and soon prevailing nosological taxonomy in psychiatry.⁴⁹ From now onwards, the focus of child psychiatrists and of psychiatry in general, predominantly shifted towards an ontological understanding of disease and a biomedical model of mental disorder with 'intellectual inspiration derived from Kraepelin, not Freud' (Bayer and Spitzer (1985) quoted in Young, 1997: 99).

Constraining elements of ontologically distinct disease entities

Fleck pointed out some of the characteristic difficulties that arise and only make sense within this particular style of medical thought,

As soon as medical thinking has found a certain ideal type in an infinite plurality of apparently atypical morbid phenomena, it faces a novel problem: how to reduce them to a common denominator, to obtain, by way of analysis, certain common elements, some component bricks from which the observed phenomena could be reproduced. In this way elements of morbid anatomy and morbid physiology arise. However, combinations of the motifs obtained in this way ... never do adequate justice to the entire wealth of the individual features of the disease. (Fleck, 1927/1986: 41)

⁴⁹ See Young (1997, Chapter 3) and Mayes and Horwitz (2005) for more context on the *DSM-III* and the revolution in the classification of mental illness.

This outline of the logical consequences of medical thinking in terms of disease entities matched later developments in autism research prophetically. As I already mentioned, the search for a ‘common denominator’ started soon after Kanner described the plurality of morbid phenomena he associated with autism. Out of a number of initial characteristics, such as limited spontaneous and varied activity and monotonous and repetitive verbal utterances and behavior (Kanner, 1943), Eisenberg and Kanner (1956) later chose two necessary and sufficient features: ‘extreme self-isolation and the obsessive insistence on the preservation of sameness, features that may be regarded as primary’ (1956: 557). Soon after, these two features were considered too general and were no longer believed to capture the entire complexity of autism. Influenced by new types of investigation and new experimental and epidemiological methods, autism researchers decided that ‘the central problem, present in even the most mildly handicapped autistic people, appears to be a specific difficulty in handling symbols, which affects language, nonverbal communication, and many other aspects of cognitive and social activity’ (Ricks and Wing, 1975: 214). For some time, it was not social withdrawal but language deficits that were essential and necessary for an autism diagnosis (Rutter, 1968). This shifted again in the 1980s and at present, it is no longer language problems but deficits in social cognition and social interaction that have become essential in autism (Verhoeff, 2013/Chapter 3; Wing et al., 2011). In this process of redefining autism in terms of its essential features and primary deficits – that, as we will see, also takes place at biological levels – autism appears in modified forms, with new properties, and with new relations to and distinctions from other mental disorders.

Furthermore, besides the desire to reduce complex clinical pictures to primary and essential elements, ‘the omnipresence of conflict and negotiation at the boundaries of particular ills’ (Rosenberg, 2003: 500) is inherently related to the contemporary style of medical thought. In fact, part of the search for a common denominator is the ongoing intraprofessional controversy surrounding the proper categorization and demarcation of autism. Initially, controversy arose over whether autism was a subcategory of schizophrenia. Leo Kanner considered that early infantile autism ‘may be looked upon as the earliest possible manifestation of childhood schizophrenia’ (Kanner, 1949: 419). Later expert discussions, for instance, concerned if and how autism differs from other language disorders (Rutter, 1978), whether autism and Asperger’s

disorder are essentially similar (Schopler, 1996), whether and how autism and Asperger's disorder differ from schizoid and schizotypal personality disorders (Tantam, 1988), how repetitive behavior in autism is different from repetitive behavior in mental retardation (Bodfish et al., 2000), if autism and psychopathy share an underlying cognitive profile (Rogers et al., 2006), how lack of empathy is different in autism compared to lack of empathy in psychopathy (Jones et al., 2010), how autism and ADHD are both similar and different (Gargaro et al., 2011), whether social communication disorder is part of the autism spectrum (Ozonoff, 2012), and so on. Without the contemporary style of thinking in terms of ontologically distinct diseases, these constant comparisons between disorders and the difficulties inherent in demarcating categories would be unthinkable or meaningless.

The continuous dynamic of searching for common ground, negotiating the essence and borders of autism, relating autism to other conditions, being unable to do adequate justice to the complexity of the clinical picture, and shifting emphasis to other 'component bricks' at phenotypical – but also cognitive and biological – levels, has been and still is an important driving force of autism research. It can even be argued that the conceptual changes, new categories, the construction of hypotheses, guessing about non-observed relations and the continuous search for autism's essence are necessary for the persistence of autism as a legitimate object of scientific scrutiny. With the idea of autism as an ontologically distinct disease, paradoxically, stability and endurance requires conceptual adjustments and consequently, a shifting image of autism.

Social entities and the bureaucratic imperative

Conflicts and negotiation at the boundaries of particular disorders are not restricted to intraprofessional controversies. Distinct categories inevitably involve including some people as well as excluding others. Wide-ranging issues such as insurance reimbursements, accountability for crime, responsibility for unwanted behavior or pain and suffering, but also issues concerning medical evidence, objectivity and authority are at stake in delineating diseases. They are a logical consequence of the (social) reality of distinct diseases. Each with their own ideas, backgrounds and interests, medical doctors, patient advocacy groups, governments, insurance companies, pharmaceutical companies, etcetera, play their part in establishing and shaping the boundaries of particular diseases.

Although Fleck mainly focused on the role of perception and cognition in his history of the Wasserman reaction, he also mentioned the role of consensus conferences and legislation in the ‘genesis and development of a scientific fact’ (Löwy, 2004b). The ‘thought collective’ of serologists, he clarified, ‘standardized the technical process with genuinely social methods, at least by and large, through conferences, the press, ordinances, and legislative measures’ (Fleck, 1979: 78, quoted in Löwy, 2004b: 519). Rosenberg (2003) further emphasized the practical importance of disease categories in their relationship to the management and administration of health care. ‘Disease’, he argued, ‘does not exist in the domains of the clinical and bureaucratic practice as a general quality or experience: without a specific diagnosis it remains largely invisible – unreadable – to the world of clinical medicine’ (2003: 499). Rationalizing tendencies within medicine, such as diagnostic procedures (for example, DSM categories), standardized treatments, clinical trajectories, and the governing rules of evidence-based medicine and randomized clinical trials, in combination with the needs of bureaucratic systems (for instance, regarding the Individuals with Disabilities Education Act (IDEA) or reimbursement procedures) create an additional constraint toward the construction and preservation of discrete disease categories. ‘Disease categories serve’, Rosenberg explains, ‘as integrating mechanisms, facilitating countless microdecisions and thus linking different parts of the health-care system in a way that seems both necessary and proper’ (ibid.).

Other important actors are disease and disability (self-) advocacy groups that are typically organized around specific disease categories. Autism has been a very significant locus of organization and activism. Starting in the 1960s in the UK and the US, parents have organized to share experiences and information, and to lobby for better services for their children (Silverman, 2011). In addition, partially as a reaction to dissatisfaction with psychoanalytical approaches and Bruno Bettelheim’s by that time popular theory that autism was caused by cold, emotionally distant mothers (Bettelheim, 1967), parent advocacy groups lobbied for and contributed to a reconceptualization of autism as a biological disorder. Organizations like the US-based *Autism Speaks*, which is the biggest and probably most influential autism advocacy group, have a substantial impact on research directions through their research funding programs. From different perspectives and with different convictions and priorities, exoteric communities of parents of children with autism have, for instance, worked together to shape

research on the genetics of autism (Silverman, 2007) and to investigate the possibility that autism is caused by vaccines (Hobson-West, 2007).

Furthermore, other exoteric autism organizations and societies have played a significant role in raising public awareness about autism using, amongst many other means, worldwide fundraising and awareness-raising events.⁵⁰ These advocacy groups have been crucial in the identification of autism as an object of urgent global mental health concern. Meanwhile, other social movements, such as the neurodiversity movement, believe that autism is not a disease to be treated but rather ‘a human specificity (like sex or race) that must be equally respected’ (Ortega, 2009: 426). Although these various exoteric groups have different priorities, their activities are bounded by and organized around the central category of autism. Through autism awareness campaigns, activism on services and research funding, and the identity politics of the autism self-advocacy movement, autism groups not only reshape but also stabilize and popularize the very idea of autism.

The DSM-trained clinical gaze of mental health professionals, together with the bureaucratic needs of health administration, the clinical and scientific utility of disease categories and the organization of autism-oriented advocacy groups, all imply a bias toward seeing and thinking about autism as an ontologically distinct psychiatric and scientific entity. Furthermore, the borders of different autism communities are highly porous and open to multidirectional pressures and exchanges of ideas and individuals. Susan Swedo, for instance, is not only the chair of the *DSM-5* Neurodevelopmental Disorders Work Group and a researcher in the field of neuropsychiatry, but also a member of the scientific advisory board and a reviewer of grants for *Autism Speaks*. Similarly, founder of the UK-based *National Autistic Society*, Lorna Wing, is also one of the most influential autism researchers and the mother of an autistic child, and Bernard Rimland, research psychologist and father of an autistic child, founded the *Autism Society of America* and was a widely acknowledged autism authority. Fundamental research, clinical practice, autism advocacy and personal involvement have always been closely linked in the history of autism, with autism as a central and connecting object that intellectually unifies the multiple levels and various aims and practices of this broad and heterogeneous thought collective.

⁵⁰ See: <http://www.autismspeaks.org/what-autism/world-autism-awareness-day>, accessed February 4, 2014.

Neurobiological specificity: the neuromolecular style of thought

Beginning at the end of the nineteenth century, germ theories – in combination with the rise of pathological anatomy, chemical pathology, and studies of normal and abnormal physiological functioning – constituted a strong case for a reductionist and mechanism-oriented way of thinking about the body and poor health. Similar to the ontological understanding of disease, this way of thinking has not been as steady in psychiatry as in the rest of medicine. Only in the last twenty to thirty years have psychiatric ailments been primarily thought of as biological disorders, with the brain being the central organ in which disease-specific somatic mechanisms are localized.⁵¹ In the context of recent developments in the neurosciences, life sciences and biomedicine – for instance, in molecular genomics and neuroimaging – mental disorders are more and more located at the neurobiological level of the brain.⁵² Contemporary psychiatry is part of a thought collective that consists of a broad range of neuroscientific disciplines, from neurogenomics to neuroaesthetics and social

⁵¹ Developments in the production and use of psychotherapeutic drugs played a significant role in the emergence of a brain-centered, neuromolecular seeing and understanding of human beings and their cognitive, emotional, and volitional states. The introduction of novel psychotherapeutic drugs in the 1950s and 1960s played an important role in a new way of linking the brain and psychiatric disorders – through a ‘neuromolecular gaze’. As Rose and Abi-Rached (2013) explain, throughout the 1950s, a number of decisive studies discussed the effects of novel psychotropic drugs, notably chlorpromazine, reserpine and imipramine, on mood and on psychotic and behavioral disturbances. Gradually the idea emerged that these drugs produced their clinically useful effects by acting upon the level of neurotransmission by influencing the amounts of monoamines (a group of neurotransmitters including serotonin, norepinephrine and dopamine) in the brain. A consensus began to arise that specific disorders were caused by anomalies in specific classes of neurotransmitters (Healy and McMonagle, 1997; Moncrieff, 2008).

⁵² The brain as locus of mental pathology and target for treatment is certainly not a recent phenomenon. In mid-twentieth century asylums, brain-directed treatments were widespread. These treatments included electroshock therapy, insulin-induced coma and, to a lesser extent, brain surgery (see Sargant and Slater, 1948). However, if, where and how these unspecific and harsh therapies worked remained mysterious. Furthermore, mid-twentieth century corporeal understandings of emotion, behavior and cognition were very different from the recent pervasive and ‘techno-somatic’ understandings of specific mental disorders as being grounded in specific anomalies in the depths of the brain (Pickersgill, 2009).

neuroscience, unified by a particular style of thought.⁵³ Rose and Abi-Rached (2013) enumerate some of the key structuring principles of this ‘neuromolecular style of thought’:

All mental processes reside *in the brain* (where else could they reside!), and each mental process will reflect, or be mediated by, or have something variously described as a correlate, an underpinning, or a basis, in brain events. Thus any mental state or process (normal or abnormal), will have a relation – exactly what relation is in dispute – with a potentially observable material process in the organic functioning of the neuromolecular processes in the brain. (Rose and Abi-Rached, 2013: 43)

Furthermore, within the neuromolecular style of thought, neural brain processes can and should be anatomized at a molecular level and investigations should proceed in a reductionist mode by exploring the fundamental elements of brain functioning. A key element of brain functioning is communication along and between neurons, that is neurotransmission. Besides different types of neurons and neurotransmitters, neurotransmission also involves ‘the function of multiple other entities: ion channels, transporters, receptors, enzymes that catalyze or metabolize neurotransmitters at different rates, and so forth’. These entities are involved in all sorts of processes of neuronal motility, axon guidance, synaptogenesis and other aspects of neurodevelopment, and ‘variations in each of these elements have functional significance and can in principle account for processes at higher levels’ (ibid.).

Two major influences on this new way of thinking about mental disorders were the ‘dopamine hypothesis of schizophrenia’ (Carlsson and Lindqvist, 1963), which suggested there was a link between high levels of dopamine in the

⁵³ There are many kinds of explanations for the rise of a ‘biological psychiatry’ and the current dominance of the neurosciences in psychiatry. Often mentioned explanations are; the promising introduction of chlorpromazine as the first antipsychotic drug; dissatisfaction with unsuccessful psychoanalytic treatments of schizophrenia and the decline of psychoanalytic prestige in general; the infamous power of drug companies with their focus on neurobiological anomalies and their close ties with academic psychiatry; biopolitical forces oriented towards the medicalization of social problems; the seductive allure of ‘objective’ neuroscientific explanations and brain pictures; the activism of parents and support groups and the hopes and promises for a ‘genuine’ cure; and more (see, for example, Rose, 2007).

brain and schizophrenia, and the ‘catecholamine hypothesis of depression’ (Schildkraut, 1965), which suggested a link between depression and a deficiency of norepinephrine at particular adrenergic receptor sites in the brain. According to Rose and Abi-Rached (2013: 37), these studies marked an important moment ‘when a new language was assembled together, one that would come to shape the [neuromolecular] style of thinking’ that connected neuroscience with clinical psychiatry through pharmacology.⁵⁴ The research path of neuropsychiatry headed for a future in which psychiatric classification and diagnoses could be based on the specific neurochemical malfunctions of neurotransmitter systems. This ‘neuromolecular gaze’ not only provided psychiatry with new types of explanations, but shaped what counted as a valid explanation and constrained the kind of problems that needed to be solved.

Blurred boundaries and neurobiological underpinnings

Rose and Abi-Rached (2013) furthermore argue that the neuromolecular image of the brain blurred two historically important boundaries in psychiatry. They first mention the ‘Cartesian boundary’, which had been crucial since the birth of psychiatry as a distinct medical discipline in the mid-nineteenth century (see Davidson, 1999), between organic and functional disorders. The strict distinction between disturbances of mental functioning caused by identifiable lesions in the brain (organic) or caused by stressful events, life history or suppressed desires (functional) blurred when all mental disorders or disturbances of mental functioning must, at a fundamental level, be related to neuromolecular anomalies in the brain. Something similar applies to the distinction between ‘states’ and ‘traits’, a distinction that often characterizes the division between psychiatry and psychology. If both states (that is, intermittent periods of illness) and traits (that is, pervasive features of personality of character) ‘essentially were variations of the same molecular mechanisms, that

⁵⁴ Even though the catecholamine hypothesis of depression and the dopamine hypothesis of schizophrenia ‘proved to be wrong, perhaps fundamentally so’, these ‘two founding myths of the psychopharmacological imaginary’ (Rose and Abi-Rached, 2013: 37) and the style of thinking they accompanied, would come to dominate the field of psychiatry. For a couple of decades, ‘the biogenic amine system in the brain would increasingly become the obligatory passage point of all accounts of mental disorder’ (ibid.). Whether there were environmental, genetic, psychodynamic, social or biographical factors in the etiology of mental disorder, they would have their effects through this biogenic amine system.

distinction blurred, and along with it the distinction between personality disorders and psychiatric illnesses – perhaps, even, the disciplinary divide between psychology and psychiatry when it comes to intervention’ (Rose and Abi-Rached, 2013: 46).

Interestingly, the recently published *DSM-5* (APA, 2013) removed the common multi-axial classification system consisting of personality disorders and intellectual disabilities on a distinct axis from other mental disorders (such as anxiety disorders and schizophrenia), in favor of a nonaxial system. Even though, particularly for practicing mental health professionals, the neuromolecular style of thought should not be thought of as universally accepted and distributed, in general, mental disorders are no longer conceptualized in Freudian psychodynamic terms or as psychological reactions to personal and social adversities. In essence, mental disorders have become disorders of the neuromolecular structures of the brain.

Part of this neurobiological image of mental disorders consists of the neuropsychiatric efforts and hopes to demarcate mental disorders in neurobiological terms using genetic biomarkers and patterns of brain activation. The initial goal, which eventually was found to be premature, for the new *DSM-5* classification system of mental disorders was to ‘translate basic and clinical neuroscience research relating brain structure, brain function, and behavior into a classification of psychiatric disorders based on etiology and pathophysiology’ (Kupfer et al., 2002: 70). Steven Hyman, the former director of the NIMH, acknowledged in 2007 that ‘it is probably premature to bring neurobiology into the formal classification of mental disorder’, however, he argued, ‘it is not too early to use neurobiology as a central tool to rethink the current approach to mental disorder’ and he expressed a hope that future manuals ‘can usefully incorporate information about brain structure and function’ (Hyman, 2007: 725). In similarly promising terms, current NIMH director Thomas Insel argued that ‘reclassifying disorders based on brain function could yield a system of diagnosis based on biomarkers – biological signs such as brain activity patterns or chemical or structural changes specific to the condition’ (Insel, 2010: 50-51). He rather boldly predicted that ‘today’s developing science-based understanding of mental illness very likely will revolutionize prevention and treatment and bring real and lasting relief to millions of people worldwide’ (ibid.: 51).

For autism in particular, the expectations that neuroscience will solve diagnostic and therapeutic problems – that is, distinguish autism subtypes; demarcate pathology from normality; gain understandings of etiology; screen presymptomatic individuals; and develop effective treatments that aim at specific neurobiological underpinnings – have been and still are high. Since approximately the 1970s, with the decline of psychoanalytic prestige, the revival of Kraepelinian thinking, and in the spirit of the emerging neuromolecular style of thought, autism has become a yet-to-be-identified biological thing. However, much like every other mental disorder, autism has turned out to be extremely hard to pin down on a specific neurobiological basis.

Autism and neuroscience

Despite the overarching neuroscientific tendency in psychiatry, autism research has its own particular link with neuroscience. Autism parents have played an important role in pushing autism research in a predominantly biological direction (Eyal and Hart, 2010; Silverman, 2011). This involvement is often interpreted as a reaction to Bettelheim's influential psychoanalytically founded theory that autism was caused by 'the parent's wish that his child should not exist' (1967: 125). However, it was a few years before Bettelheim's book that the previously mentioned autism parent and researcher Bernard Rimland proposed the first consistent neurological theory of autism when he suggested that autism was caused by malfunctions in specific parts of the brainstem (Rimland, 1964). From that period onward, biological and neurocognitive research on autism expanded exponentially.

Another important source of support for the biological approach to autism, against psychoanalytical theories, was the first and very influential autism twin study (Folstein and Rutter, 1977). This study brought 'the importance of genetic factors in the aetiology of autism ... [and its] very high heritability' (p. 307) to the attention of autism research and later heritability studies confirmed extremely high heritability rates of between 85 and 92 per cent (see Miles, 2011). This study marked the beginning of an extensive search for autism genes carried out by disciplines ranging from epidemiology to molecular genomics and with a variety of statistical methods ranging from twin and linkage studies, to association studies with candidate genes and whole-genome association studies (see, for example, Abrahams and Geschwind, 2008). This ambitious and complex search yielded no easy route from gene(s) to disorder. The number of

genes associated with autism may be a couple hundred or more; they are probably not specific for autism and the most common mutations are found in less than one per cent of the children with autism (Schaaf and Zoghbi, 2011). Sanders et al. (2011) even stated that between 130 and 234 submicroscopic chromosomal deletions and duplications (copy number variants or CNVs), which vary widely in function, are linked to autism. These rather ambiguous results further complicated the search for autism's underlying neurobiology.⁵⁵

In the meantime, at different neurobiological and cognitive levels – situated in the black box between genes and the phenotype – various lines of neuroscientific research have attempted to provide specific and unifying accounts of autism. The 1980s and 1990s saw the rise of a couple of key cognitive theories of autism (Hollin, 2013; Verhoeff 2013/Chapter 3). For example, a defective 'theory of mind', which is the inability to attribute mental states to other individuals (see Baron-Cohen et al., 1985); 'weak central coherence', which refers to a lack of conceiving a meaningful whole picture in combination with an overemphasis on detail (Frith and Happé, 1994); and impaired 'executive functioning', which refers to problems in planning and other problem-solving capacities (Ozonoff et al., 1991), have all been proposed as a cognitive 'common denominator' and, therefore, as being fundamental to autism. However, despite their influence on thinking about autism, it is now widely accepted that none of these cognitive theories can explain all the behavioral phenomena associated with autism (Happé and Ronald, 2008).

Other searches for neurobiological singularity have focused on structural anomalies in the entire brain and in specific brain areas. For instance, abnormal enlargement of total brain volume (Sparks et al., 2002), increased white matter compared to gray matter (Herbert et al., 2004), enlargement of the frontal lobes (Carper et al., 2002), enlargement of the amygdala (Schumann et al., 2004) and enlargement of the cerebellum (Hardan et al., 2001), have all been associated with autism. Yet again, none of these studies has been able to get a grip on the specific neurobiology of autism (Amaral et al., 2008). Furthermore, individuals with autism may or may not have neurotransmitter abnormalities in dopamine, serotonin, or glutamate systems, or abnormalities in the neurohormones oxytocin and vasopressin (Insel, 2010). The most recent focus in autism

⁵⁵ See Jeste and Geschwind (2014) for a recent review on genetic findings in ASD research and their expectation to identify specific autism subgroups based on 'genetic classifiers'.

neuroscience is on functional, instead of structural, problems of the brain. With functional neuroimaging and molecular genetics studies, abnormalities in neural networks (Peca and Feng, 2012), mechanisms of synaptogenesis (Persico and Bourgeron, 2006), and problems in the connection between different brain areas are the new hopes for a coherent neurobiological account of autism. Yet, despite current efforts, the neurobiological basis of autism remains unidentified (Rutter, 2011; Waterhouse, 2013).

Where does that leave us for a neurobiology of autism? To quote Rose and Abi-Rached (2013: 138) again: ‘each of the pathways that neuropsychiatry has attempted to trace through the brain seems to run, not into the bright uplands of clarity, but into the murky, damp, misty, and mysterious forests of uncertainty’. As with other mental disorders, the ‘hard residue of material reality’ resists a straightforward translation of autism into biological terms. Currently, despite the neuroscientific dominance of autism research, no clear autism biomarkers have been found that support diagnostic practices, distinguish autism subtypes, guide the development of new treatments, or demarcate pathological from normal conditions. Autism diagnosis and classification remain behaviorally based. Yet, still guided by an ontological (Temkin, 1977) understanding of autism and not less constrained by the neuromolecular style of thought, neuroscientific research on autism has readapted its attempts to identify neurobiological singularity towards developmental processes, epigenetics and neuroplasticity. The ambiguous findings of many genes, multiple brain areas, different cognitive profiles and heterogeneous diagnostic features need to be connected in a novel way. In the neuromolecular style of thought and with the need to take neurobiological heterogeneity and dimensions of time, plasticity and interactions with the environment into account, autism has become a neurodevelopmental spectrum disorder.

Rise of a neurodevelopmental spectrum disorder

Besides neurobiological heterogeneity, there is another profound uncertainty that surrounds autism research: the heterogeneity of the phenotype itself – the clinical picture with all the signs and symptoms associated with autism. This problem of heterogeneity relates to the increasing struggle to hold autism

together as a convincingly steady diagnostic category (see also Fitzgerald, 2012; Hollin, 2013).

A crisis of heterogeneity

Many autism experts have come to argue that ‘the central challenge to understanding autism has been its heterogeneity’ (Waterhouse, 2013: 3). Diagnostic features including social interaction impairment, repetitive and restrictive behaviors, and sensory abnormalities as well as non-diagnostic features such as savant skills, intelligence, language skills, perceptual problems, motor disorders, neurological disorders (epilepsy), ADHD symptoms and environmental risk factors vary widely in form and severity among those diagnosed with autism. ‘If you’ve seen one child with autism, you’ve seen one child with autism. Autism’s like a snowflake’, is the often heard quote from autism researcher Robert Schultz (Scott, 2011). Autism researcher Lynn Waterhouse (2013) devoted an entire monograph to this issue of heterogeneity.

She states that variation in patterns of impaired sociability has been found for all developmental stages. For instance, some infants later diagnosed with autism ‘paid no attention to social stimuli as babies, smiled infrequently, and vocalized little. However, this was not true for all, because many infants later diagnosed with autism did smile, vocalize, and pay attention to other people as infants’ (Waterhouse, 2013: 7). Some toddlers with autism do not respond to their name, never reach to be picked up, or have limited eye contact and social interaction with parents. Yet, other toddlers with autism do not lack this type of interactive behavior. The same is true for autistic children who may or may not initiate social interaction, show empathy, or use appropriate gestures along with speech. Likewise, other diagnostic features such as rigid interests and repetitive behaviors have also been found to be heterogeneous. Preoccupation with restricted interests; non-functional routines or rituals (an inflexible need for the same route or the same clothing); and repetitive motor mannerisms (for example, flapping hands or spinning in circles) are all found in various forms, combinations and levels of severity (ibid.).

Guided and constrained by an ontological understanding of autism and the idea of neurobiological specificity, attempts to understand and deal with variation have alternated between searching for biologically and behaviorally homogeneous subgroups (see, for example, Jeste and Geschwind, 2014; Lui et al., 2008; Ingram et al., 2008), and reducing the complex clinical picture of

autism to a common (underlying) denominator (see Verhoeff 2012/Chapter 2). However, as I have shown above, researchers have not been able to identify either a unique, unifying brain (or cognitive) deficit or meaningful subgroups. 'There is now', Waterhouse (2013: 23) argues, 'a large pile of competing orphaned, and unsynthesized theories of autism subgroups, and theories of unifying brain deficits and unifying patterns of genetic and environmental risk factors for autism'.

The vast clinical heterogeneity in the manifestation of autism, including course, prognosis, response to treatment and co-morbid conditions, in combination with the difficulties of identifying specific neurobiological markers or cognitive profiles, created serious scientific challenges. It destabilized the understanding of autism as a separate neurobiological disease and produced a need for a reworking of the very idea of autism. Yet, bound by the stylized readiness for perceiving and thinking about autism and related conditions as ontologically distinct diseases, and by autism's bureaucratic function in integrating different parts of the health-care system, reframing autism is a delicate process. It is a process of stabilization and harmonization that takes place at the nexus of 'exoteric' and 'esoteric' circles inhabited by neurobiological researchers, psychiatrists, medical practitioners, health administrators and patients and their advocates. The emergence of the 'autism spectrum' as the new central object of study can be seen as an important moment in this process, in which the ontological understanding of disease and neuromolecular styles of thought persist, whereas this new image of autism can account for many of the difficulties and ambiguities produced by recent autism research.

The spectrum idea of autism

As I stated earlier, the meaning of the term 'spectrum' in relation to autism is ambiguous, and the shift towards scientific endorsement, popularization and institutional acceptance of the autism spectrum is more gradual than radical. Autism researcher Michael Rutter had already mentioned in 1968 that in autism 'there seems to be a continuum from severe persistent social withdrawal to mild transient withdrawal' (Rutter, 1968: 20). Wing and Gould (1979: 26) also noticed a 'continuum of severity' in the social, language and behavioral impairments associated with autism and Lorna Wing introduced and popularized the idea of a triad of social impairment and the differences in

symptom severity, associated levels of impairment and clinical manifestation of these three cardinal features of autism as ‘the autistic spectrum’ (Wing, 1996). More recently, autism researchers have also used the spectrum concept to refer to the continuity of autistic traits between the clinical and the general population, and to refer to the variety of associated features (instead of the cardinal features described in DSM criteria) such as epilepsy and savant skills (see, for example, Williams et al., 2008).

Furthermore, the idea of the autism spectrum is currently used to reflect the variable manifestations across life span within and between individuals with autism (Happé, 2011). Lastly, it is important to note that Lai et al. (2013) mention another meaning of the autism spectrum, suggesting that the idea of a spectrum not only refers to phenotypical variation, but also has come to reflect the earlier mentioned neurobiological, genetic, cognitive and etiological (including potential environmental contributors) variation in autism. A spectrum of genes, neural networks, molecular mechanisms, neurocognitive and neurodevelopmental profiles, and etiological elements (Geschwind, 2011; Happé, 2011) has become part of the idea of an autism spectrum.

In the last couple of decades, the spectrum metaphor has repeatedly been used to indicate different levels of heterogeneity related to autism. In a cumulative manner, the use of the term expanded from different levels of symptom severity to diversity in symptoms, manifestations, impairment, course, prognosis, response to treatment and cognitive and neurobiological markers. Hacking (2009: 47) argues that the spectrum metaphor is problematic, as it ‘suggests that you can arrange autistic people on a line, from more to less’. Without discussing the possible function of the spectrum metaphor or what it is that keeps the autism category together, Hacking prefers to speak of an ‘autistic manifold’ which is, I suggest, more or less what the ‘spectrum’ in autism research and practice has come to signify: heterogeneity at multiple levels. Nevertheless, the assumption that the autism spectrum or potential subgroups within the spectrum (for example, Asperger’s disorder) have distinct brain-based pathophysiologies which explain, produce and underlie the symptoms, impairments and suffering experienced by autistic patients – that is, the ontological understanding of autism within the contemporary style of medical thought – remains unchallenged.

The recent ‘official’ shift to ‘autism spectrum disorder’ (APA, 2013) might not seem very different from earlier dimensional perspectives of, for instance,

Rutter (1968) and Wing (1996). However, despite the clear continuities in recognizing heterogeneity, I argue that it does indicate an important change in thinking about autism. With the latest advent of promising functional neuroimaging studies, molecular genetics studies and genome-wide association studies (GWAS), the persistent failures to identify autism's neurobiological pathophysiologies has become particularly challenging for the validity of autism as a neurobiological disorder. That is to say, within an ontological understanding of disease, the homogeneity of these underlying genetic and neurobiological elements is basically what indicates the validity and legitimacy of a separate disease category. In light of these disappointments, reframing autism as a spectrum disorder converts problematic and ubiquitous heterogeneity – including the 'existentially' challenging neurobiological and cognitive heterogeneity – into an intrinsic feature of the disorder. This reframing ensures the existence of an autism category; it postpones the problem of validity; and creates new opportunities for further neuroscientific research directed at fundamental and specific brain-based disease mechanisms.

As a comparison, the diversity in severity, symptoms and course in Down syndrome or Huntington's disease is widely acknowledged, but these well-defined (valid) genetic disorders are not considered spectrum disorders. The shift towards an autism spectrum disorder is not just about describing and acknowledging variation in severity and symptoms, but involves a reworking of the very object of explanation. With the reframing of autism as a somewhat vague and multi-interpretable spectrum disorder, diverse hypotheses concerning the now inherent neurobiological variation, for instance, aimed at complex networks of genes (Szatmari, 2011; Jeste and Geschwind, 2014) or converging molecular pathways (Sakai et al., 2011), have come into being. These are not just new explanations of autism; the object of investigation has been modified, facilitating further research and a range of new types of hypotheses and explanations regarding the validity – that is, neurobiological specificity – of the new autism spectrum.

In a discussion on the *DSM-5* category of ASD, Rutter (2011: 399), for instance, concluded that 'at the moment ... it is highly likely that there are meaningful subcategories of autism spectrum disorders but that these are not well identified'. Holt and Monaco (2011: 455) suggested that 'genetic research is validating the view that ASDs should not be considered a set of discrete disorders, but a continuous range of individually rare conditions', and Boucher

(2011) hypothesized that the autism spectrum reflects many brain deficits and varied etiologies that must in some way interact to converge on a single specific brain abnormality. As members of the *DSM-5* Task Force, Kupfer and Regier (2011: 673) stated that ‘the proposal for a single “autism spectrum disorder” category ... was born from data suggesting that these disorders [Asperger’s disorder, autistic disorder, PDD-NOS] share a pathophysiological substrate’. Somewhat more specifically, Kana et al. (2011: 428) argued that ‘given the complexity, heterogeneity, and the developmental nature of ASD, a global explanation or a set of explanations seems optimal. We believe that disrupted cortical connectivity may be one such explanatory model that provides a comprehensive outlook’.

Thus, while the powerful autism-oriented advocacy organizations, the need to regulate administrative and clinical practices, and the DSM-trained gaze of clinicians, autism experts and other mental health professionals are important forces that hold autism together as a diagnostic category, a reworking of the construct of autism towards a spectrum preserves the existence of autism as a scientific object in search of neurobiological specificity. Despite all the ambiguity and uncertainty that surrounds the neurobiology of autism and pressured by the need for successful translations between fundamental neuroscientific research and goal-oriented clinical applications, the move towards an autism spectrum protects autism’s threatened scientific status as a valid neurobiological disorder – a status that equally legitimizes many of the more exoteric practices and communities in which autism circulates.

In sum, at a moment when biological and categorical approaches towards autism face serious empirical difficulties, a delicate balance is established that holds together and integrates the dominant neuromolecular style of thought and the current style of medical thought in terms of ontologically distinct diseases. The emergence of an autism spectrum satisfies the scientific, institutional and socio-political needs for continuity, homogenization and unification of neuroscientific research, clinical practice and the bureaucratic space of special services, reimbursements and health administration. Both within and between the ‘exoteric’ and ‘esoteric’ circles of the autism field, stability and commensurability is at stake in reframing autism. However, this need for stability and commensurability comes at a cost, as the particular objectives of specific research groups, clinicians, and advocacy groups might not be best achieved through a unifying autism spectrum disorder.

Concluding remarks

As current director of the NIMH Thomas Insel argues:

With no validated biomarkers and too little in the way of novel medical treatments since 1980, families need science to provide more than hope. Genetics and neuroscience finally have the tools to transform the diagnosis and treatment of mental illness. But first, it is time to rethink mental disorders, recognizing that these are disorders of brain circuits likely caused by developmental processes shaped by a complex interplay of genetics and experience. (Insel and Wang, 2010: 1971)

Yet, as Insel and Wang try to open up space to rethink mental disorders, ‘the free unfolding of ideas’ (Fleck, 1979: 84), or the amount of freedom to rethink mental disorders is rather limited as mental disorders have already become ‘disorders of brain circuits’. Contemporary academic psychiatry remains committed to the neuromolecular style of thought and following autism, psychotic disorders, bipolar disorder, obsessive-compulsive disorder, and other, are gradually modified and appear in neurodevelopmental and spectrum terms that still hold disease categories together. The case of autism shows that despite the failure to identify specific and clinically relevant biological markers, the current transformation in thinking about specific psychiatric disorders as a neurodevelopmental spectrum disorders enables the continuity of neuropsychiatric research and releases tension from the general disappointments and unfulfilled promises and hopes for a neuroscientific foundation of psychiatry in general.

Why is this a good moment to disentangle the prevailing styles of thought that directed the emergence of a neurodevelopmental autism spectrum? As Fleck pointed out, ‘the more developed and detailed a branch of knowledge becomes, the smaller are the differences of opinion’ (1979: 83). A dense network of active and passive connections that constitutes a particular style of thought seems to achieve a kind of inevitable stability and a ‘tendency to reify and objectivize the conceptual creations of scientific thought arises’ (ibid.: 144). However, now that we are still close to this process of reframing and stabilizing autism, the dominant socially and historically preconditioned ways of seeing and thinking about autism become less compelling and no longer seem

inevitable. In the current scientifically uncertain times, in which ‘neuropsychiatry [has] not been able to “self-vindicate”’ (Rose and Abi-Rached, 2013: 138), and in which the communities that are involved are increasingly diverse in their practices, aims and interests, the harmony of autism as a central structuring concept is not self-evident and the traces of labor invested in keeping this heterogeneous field together are not yet erased. A consideration of these traces of labor and the historically contingent processes through which the neurodevelopmental autism spectrum comes into being makes us realize where our contemporary psychiatric categories, our preconditioned ways of seeing and thinking, and our ways of organizing, treating and investigating psychiatric ailments come from.

The recent emergence of a neurodevelopmental autism spectrum illustrates the multitude of interactions within its heterogeneous ‘thought collective,’ and can be seen as an exemplary case of the outcome of a close intertwining of abstract understandings of disease, the clinical gaze of health professionals, techniques that enable the visualization of neurobiological phenomena, epidemiological studies, the practical needs of clinicians, specific advocacy groups and regulatory practices. A network of active and passive elements of knowledge, taking neurobiological research seriously and recognizing the socially and historically contingent determinants and constraints of particular styles of thought, effected the reframing of autism as a neurodevelopmental autism spectrum and the new way of seeing this particular phenomenon.

In this paradoxical process of stability through change, the modified concept of autism fosters new hypotheses and spaces of experimentation in the continuous search for autism’s neurobiological foundation. However, whether this search will result in valuable translations of fundamental research into clinical practice is very uncertain. Given the diversity of the aims of clinicians, individual patients, neuroscientific researchers, epidemiologists, health administrators, advocacy groups, insurance companies, and other communities involved, it might be time to give up some of the coherence across these communities and to let go of the effort to keep this broad field together by a single autism spectrum. This should include a consideration of the benefits and disadvantages of an ontological understanding of mental illness and the related search for autism’s neurobiological specificity.

Supposing, for argument’s sake, that these conclusions regarding the rise of a neurodevelopmental autism spectrum disorder are sound, there remains the

question of how ‘Fleckian’ they are. One could argue that a true Fleckian account requires a more fine-grained sociological analysis of the different thought collectives, their members, how they interact, how they disagree and dispute and how, taking these dynamics into account, the spectrum approach has become the new way forward in the field of autism. It is certainly true that some collectives have not been sufficiently heard. The perspectives and influences of (and arguments against) conflicting positions, for instance of clinicians who oppose the ontological understanding of autism and of Asperger’s disorder advocacy groups that make a plea against the autism spectrum and for a separate Asperger’s disorder, have remained rather unanalyzed. Instead, with Fleck’s conceptual tools I focused on those historically constrained ideas, practices and ways of perceiving that keep the autism field together and the research going. An analysis of how particular styles of thought directed the reworking of the idea of autism resulted in a story of stability and commensurability in which the multiplicity of ways in which autism is experienced and understood disappeared out of sight. For a more sociologically oriented approach of how these differences are negotiated within and between the thought collectives, indeed, a complementary study would be indispensable.

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5 | Two kinds of autism⁵⁶

As history shows, it is feasible to introduce completely different classifications of diseases. Furthermore, it is possible to dispense with the concept of a disease entity altogether, and to speak only of various symptoms and states, of various patients and incidences. This latter point of view is by no means impractical because, after all, the various forms and stages as well as the various patients and constitutions must always be treated differently. It is evident that the formation of the concept “disease entity” involves synthesis as well as analysis, and that the current concept does not constitute the logically or essentially only possible solution. In this context it is not possible to regard things simply as given. (Fleck, 1935/1979: 22)

Abstract

This chapter argues that the history and philosophy of autism need to account for two kinds of autism. Contemporary autism research and practice is structured, directed and connected by an ‘ontological understanding of disease’. This implies that autism is understood as a disease like any other medical disease, existing independently of its particular manifestations in individual patients. In contrast, autism in the 1950s and 1960s was structured by a psychoanalytical framework and an ‘individual understanding of disease’. This implied that autism was not a distinct disease but an idiosyncratic and meaningful response of the child to a disturbed development of the ego. These two kinds of autism are embedded in and reveal two very different ‘styles of psychiatric thought’.

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Introduction

An important event in the history of autism is the shift from psychoanalytical to cognitive and neurobiological explanations of autism. This shift, which occurred roughly over the course of the 1960s and 1970s, paralleled the broader decline of psychoanalytical prestige and the rise of a brain-centered biological approach in Western psychiatry (see Decker, 2013; Micale, 2014). Additionally, this shift encompassed a radically different understanding of the general idea of psychiatric disease. In this chapter, I demonstrate how the change from psychoanalytical to cognitive and neurobiological understandings of autism not just reflected different explanatory frameworks for a similar phenomenon, but also involved a change in the underlying concepts of disease, which affected a range of diagnostic, therapeutic and scientific practices across the entire field of autism. One could argue that – in the spirit of philosopher of science Ludwik Fleck (1935/1979) – psychoanalytical and neurobiological approaches to autism operate in distinct ‘styles of psychiatric thought’. This epistemological point sheds light on the discontinuity not just of explanatory theories of autism, but of the entire idea of autism itself. At least ‘two kinds of autism’ can be identified in the history of autism.

The history and philosophy of medical and psychiatric thought – in particular the work of eminent historians of medicine (Temkin, 1977; Rosenberg, 2003) – are used to explain these two kinds of autism. To begin with, I introduce the historically informed distinction between ontological and individual understandings of disease. This distinction serves as a broad framework to situate psychoanalytical and neurobiological understandings of autism. As I argue, an ontological understanding of disease is consistent with the contemporary idea that autism, autism spectrum disorder, autism spectrum disorders, or whatever the prevailing nomenclature indicates, are diseases like any other medical disease that exist independently of their particular manifestations in individual patients. Specific pathophysiologies, nowadays imagined as evolving, multilevel neurobiological circuits, cause and sustain the visible signs and symptoms, and the suffering, impairment and dysfunctioning experienced by each individual autistic patient.

In contrast, psychoanalytical understandings of autism were based on a very different individual understanding of disease, since autism was not seen as a distinct disease but as an idiosyncratic and meaningful response of the child to

a disturbed development of the ego. Subsequently, I exemplify how these two different understandings of autism are linked to distinct scientific, therapeutic and diagnostic practices. Even though it is clear and uncontested that the psychoanalytical theories of ‘maternal deprivation’ (Raz, 2014) and ‘refrigerator mothers’ (Bettelheim, 1967) have been harmful, stigmatizing and not supported by empirical studies, it is not the aim of this study to compare different theories of autism in terms of right or wrong. Instead, the aim is to show that for a better understanding of the history of autism, of the development of the concept of autism and of the development of knowledge about autism we need to account for two different kinds of autism that comprise radically different understandings of disease and styles of psychiatric thought.

Two understandings of psychiatric disease

A straightforward way to look at psychiatry is to see it as a medical specialty concerned with diagnosing and treating psychiatric diseases. However, as many historians, philosophers and social scientists have illustrated, a more comprehensive perspective recognizes that psychiatry ‘has been – and is being – shaped by social values and needs and consequent decisions of social policy to a far greater degree than most other specialties in medicine’ (Rosenberg, 1975: 246). Psychiatry has an important social function. The creation of asylums, psychiatric associations and diagnostic manuals, and many major trends towards deinstitutionalization, the enormous use of psychopharmacological agents, and the domination of neuroscientific research reflect, among other things, (responses to) particular social, political and economic needs and priorities (see, for example, Young, 1997). Likewise, the very notion and understanding of ‘psychiatric disease’ – the central and legitimizing object of psychiatry’s clinical and scientific practices – is profoundly historical and social.⁵⁷

⁵⁷ From a metaphysical point of view, what is considered to be a ‘disease’ is far from settled and tracks longstanding and contentious debates in the philosophy of medicine regarding notions of health, disease, normality and dysfunction (see, for example, Ereshefsky (2009) for a flavor of these debates). I will not rerun these longstanding discussions. When I discuss the ‘ontological understanding of disease,’ I am not concerned with the abstract philosophical question regarding a ‘true’ nature of disease,

For present purposes, the crude historical and thematic distinction between ontological and individual understandings of disease helps to clarify the ways in which autism is and has been understood.⁵⁸ As Rosenberg wisely warns, the distinction between the two concepts of disease has been (mis)used for value-imparting narratives. The ontological view has become too easily associated with a ‘celebratory history of postmortem and laboratory-initiated progress, a reductionist trajectory of increasing understanding and mastery of nature, while the physiological [individual] has been associated with skeptical, clinical, holistic points of view’ (2003: 494). The seductively utile distinction between these two concepts of disease that, according to Cohen (1955) and Temkin (1977), runs through the whole history of medicine from Hippocrates and Galen to Sydenham, Pasteur, Kraepelin and Freud will serve here primarily as an analytic tool. I agree with Temkin, that the question ‘does disease exist or are there only sick persons?’ is an abstract one and, in that form, does not allow a meaningful answer. Disease is not simply either the one or the other. Rather it will be thought of as the circumstances require’ (Temkin, 1977: 455).⁵⁹ Which circumstances and needs deserve more (or less) attention and, consequently, whether current dominant concepts of disease are appropriate is of course open to debate.

As far as I can tell, Lord Cohen of Birkenhead (Cohen, 1955) was the first to notice that ‘from the earliest times to the present day two main concepts have dominated all writings on the nature of disease’. As he explains:

These are (i) disease as a distinct entity; when a healthy man A falls ill he becomes A plus B, where B is ‘a disease’. This view maintained that there are innumerable Bs, each with its individual and recognizable characters. And (ii) disease as a deviation from the normal; a healthy man A, through the influence of any number of factors ($x_1, x_2, x_3, \dots, x_\infty$) – physical

independent from how researchers or clinicians investigate and have come to think about disease.

⁵⁸ This distinction should not be confused with the much used distinction between disease and illness (see Boorse, 1975) that, within the ontological-individual typology, would be part of the ontological understanding of disease.

⁵⁹ Temkin, for instance, points out that it is no coincidence that ‘Sydenham, the ontologist, lived at the time of the great plague of London, and the plague, I understand, has little concern with individual variations’ (Temkin, 1977: 455).

or mental – is changed and suffers: he is dis-eased ($\neg A$). The appropriate formula is $A^{X1, X2, X3 \dots X^\infty} \rightarrow A$ when ill. (Cohen, 1955: 1-2)

Cohen adds that there are many terms used to contrast these two concepts: ‘e.g., *ontological* – indicating the independent self-sufficiency of diseases running a regular course and with a natural history of their own, as opposed to the *biographical* or *historical* which records the history of the patient’ (ibid.). For Cohen, other contrasting terms that have been used to specify these two concepts of disease, such as Platonic and Hippocratic, realist and nominalist, rationalist and empirical, or conventional and naturalistic, are of little importance. However, despite the persistent appeal and convenience of this broad distinction, there are many evolvments and complexities related to this seemingly transhistorical distinction between understandings of disease as ‘specific entities’ and ‘individual sickness’ (Temkin, 1977) that I refer to as the ‘ontological’ and ‘individual’ understandings of disease.⁶⁰ Without attempting to refine this schematized typology, some of these evolvments and complexities deserve more attention as they relate to current thinking about autism.

Ontological understanding of disease

The way in which autism experts and authorities think about autism has been constantly changing over the last couple of decades. For example, diagnostic criteria; modes of classification (lumping or splitting; dimensional or categorical); cognitive deficits that are considered ‘fundamental’; the ‘essential’ genes and neural networks that are involved; environmental causes; the existence of meaningful subgroups; and the relation to other neurodevelopmental disorders are all unstable and continuously matters of professional debate. However, there is also an important continuity in thinking about autism. This continuity, I suggest, is guaranteed by an underlying ontological understanding of disease.

It may be surprising that current understandings of autism would be deemed ontological. No one sincerely argues that the entrance of a certain extracorporeal being called autism – analogous to a demoniac etiology of

⁶⁰ Somewhat confusingly, Temkin (1977: 442) also used the term ‘physiological’ next to ‘individual’ to contrast with the ontological understanding of disease. In this context, I suggest that ‘individual’ is the more appropriate term to group understandings of disease that focus on the human person as a whole.

disease – is responsible for the disease. As Temkin argued, even the nineteenth century bacteriologists ‘had to deviate from this ideal of medical ontology’ (Temkin, 1977: 443), as they had to relate the parasite or bacterium to how it damaged organs or tissues in order to explain the symptoms in tuberculosis, typhoid fever, or small pox. It was the specific interaction between the germ and the host that accounted for specific disease patterns. Yet two centuries before the first germ theories of disease, Sydenham (often regarded as the ‘arch-ontologist’ of modern times) already claimed that ‘nature, in the production of disease, is uniform and consistent; so much so, that for the same disease in different persons the symptoms are for the most part the same’ (Sydenham quoted in Temkin, 1977: 443).⁶¹ Although he criticized the search for the specific causes of each disease, Sydenham thought of diseases as Platonic ideal entities that could be ‘reduced to certain and determinate kinds with the same exactness as we see it done by botanic writers in their treatise of plants’ (Sydenham quoted in Cohen, 1955: 2).

Later ontologists focused less on delineating clinical pictures and pathognomonic syndromes, and instead directed their attention to the source of particular patterns of clinical symptoms. Throughout the nineteenth century, organs, tissues, physiological processes, local lesions and bacteria, became essential to the identification and classification of distinct diseases. The source and identity of specific diseases were not only to be found outside the body. Diseases were not necessarily expressions of a foreign life – external things that invade the body. Nonetheless, despite being linked to the internal mechanisms and processes of the human body, diseases were thought of as entities independent of their expression and embodiment in particular individuals.⁶² As

⁶¹ Even before that, there are signs of generalization and an ontological understanding of disease when Rhazes of Persia differentiated between smallpox and measles in the ninth or tenth century.

⁶² In contrast with Rosenberg (2003), Osbourne (1998) does not regard modern concepts of disease as ‘ontological’. Instead, he argues that if ‘medicine is to be characterized as reductive this should not be in terms of its ontological fixation but for something quite different ... namely a certain ... predilection for monist explanations’ (1998: 267). The distinctive character of modern medicine, he continues, ‘is *not* that it is ontological but, on the contrary, *that it has rid itself of any constitutive ontology*’ (ibid.). For Osbourne, an ontological understanding of disease seems to require that the disease is thought to be identical with a foreign thing, ‘a morbus that attacks the body’ (ibid.). To me, this interpretation of the ontological conception appears much too stringent.

Rosenberg has frequently and forcefully asserted in his articles on the history of disease concepts:

Recognizably modern notions of specific, mechanism-based ailments with the characteristic clinical courses were a product of the 19th century. Pathological anatomy with its emphasis on localized lesions, physical diagnosis, the beginnings of chemical pathology, and studies of normal and abnormal physiological function all pointed toward the articulation of stable disease entities that could be – and were – imagined outside their embodiment in particular individuals and explained in terms of specific causal mechanisms within the sufferer's body. (Rosenberg, 2002: 242)

This image of disease, often equated with the biomedical model of disease, is not very far removed from contemporary ideas about autism and psychiatric disorders in general. Indeed, this ontological understanding of disease pervades thinking about autism at scientific, clinical and exoteric levels (see Verhoeff, 2014/Chapter 4). However, substantively different from the ontological orientation of earlier bacteriologists and geneticists, the current image of autism is increasingly complex and needs to account for the multiple genes, neurodevelopmental circuits and epigenetic processes associated with autism. Nevertheless, all of the following imply and reinforce an ontological understanding of autism: the fundamental biomedical search for autism's specific neurobiological mechanisms; epidemiological studies regarding identifying the prevalence and determinants of autism; clinical trials that assess treatments by defining autism and control groups; diagnostic procedures that delineate autism from ADHD or obsessive compulsive disorder; the prescription of particular treatments on the basis of an autism diagnosis; classification practices directed at demarcating a homogeneous autism phenotype with a consistent course, prognosis and response to treatment; and the clinical and lay narratives in which autism provides meaning and serves as an explanation, cause and exculpation for disturbing behaviors and experiences. In all these narratives and practices, autism has an identity and natural history of its own, independent of the individuals and context in which it occurs.

This way of thinking about autism is exemplified by current discussions on missed diagnoses of autism in girls and women, whose autism disorder is

thought to be masked by better social skills, social play and the fact that they tend to imitate social actions more than boys do (Gould and Smith, 2012). Similarly, it is assumed that adults with autism had autism when they were young, even if an autism diagnosis was never made, and that beneficial circumstances must have kept symptoms latent. These narratives underscore the centrality of thinking about autism as having an autonomous (neurobiological) existence separate from the symptoms and disabilities it produces.

From a different perspective and with very different intentions, a comparable position is taken by the autism ‘neurodiversity’ movement – a group of autism self-advocates – who ‘believe their condition is not a disease to be treated and, if possible, cured, but rather a human specificity (like sex or race) that must be equally respected. For them, an atypical neurological “wiring” and not a pathological cognitive organization accounts for their difference’ (Ortega, 2009: 426). Instead of attributing impairments and failures in communication and social interaction to the ‘atypically wired’ autistic individual, the neurodiversity movement locates the source of impairment and suffering in the general lack of acceptance, respect and societal tolerance for autistic difference (see also Jaarsma and Welin, 2012). The autism neurodiversity movement accepted and incorporated the psychiatric term of autism and the medical way of thinking about autism as a neurobiological condition. Clearly different from the dominant medical perspective is that they do not consider the condition of autism to be a deficit or a pathological condition (see Kapp et al., 2012).⁶³

However, a precise image of the neurobiological condition of autism (pathological or not) remains elusive (see Waterhouse, 2013, Chapter 8). Unlike many medical diseases, autism still has no biological markers that support diagnostic practices, facilitate treatment decisions, differentiate between autism subtypes, hint at targets for new treatments, or demarcate autism from other conditions or ‘normalcy’.

⁶³ Autism expert Simon Baron-Cohen (2000) similarly argues that autism is not necessarily a disorder. I do not take a position in this discussion as I merely try to describe the different positions and ideas about disease and abnormality in relation to autism.

Individual understanding of disease

Similar to the many ways in which the nature of diverse disease entities has been understood, the individual understanding of disease has several versions. Central to each version is the idea that disease and symptoms can only be understood by taking the particular circumstances of the whole person into account. It is the history of the individual patient and not a natural history of a putative disease entity that is central to this understanding of disease. From the ancient Greeks till early modern medicine, Rosenberg argues, ‘disease concepts were focused on the individual sufferer. They were symptom oriented, fluid, idiosyncratic, labile, and prognosis oriented. Diseases were seen as points in time, transient moments during a process that could follow any one of the variety of possible trajectories’ (Rosenberg, 2002: 242).⁶⁴ Humoral imbalances, idiosyncrasies, and unique living conditions and environmental circumstances were paramount in understanding disease.

In twentieth century psychiatry, at least two versions of an individual understanding of disease have been popular. The first is Adolf Meyer’s explanatory framework which describes emotional and behavioral ailments as ‘mental reaction-types’. Meyer’s ‘genetic-dynamic’ psychiatry attempted to integrate the life history and meaningful experiences of the individual with physiological and biological data. He saw ‘mental reactions’ as the central topic of psychiatric research and practice and regarded them as ‘reactions of the person as a whole ... [that] are necessarily physical, but contrasted with *non-mental* reactions, and distinguished by the qualitative feature of consciousness in the modes of their hanging together. They are *attitudes and reactions of the person as a whole*’ (Meyer, 1908: 258, emphasis in original). Meyer explicitly opposed Kraepelinian nosological psychiatry and the idea of ontologically distinct psychiatric disorders. Instead, he considered the unique life history of each individual to be the crucial element in the etiology and understanding of psychiatric problems. With this perspective, Meyer had ‘no use for the essentially “one person, one disease” view’ of Kraepelin’s nosology (Meyer, quoted in Grob, 1991: 426). Meyer was particularly influential in the first half of the twentieth century in the United States, where he trained several generations

⁶⁴ According to Temkin (1977: 445), Hippocrates was ‘outstanding for having seen disease as a process in time, not a mere stationary picture’ and for taking into account ‘the peculiar nature of each individual’.

of psychiatrists at Johns Hopkins School of Medicine between 1910 and 1941 (Lidz, 1966).

Even more influential than Meyer's reaction-types – certainly beyond the realm of psychiatry – was Freud's psychoanalytical framework for thinking about the intricacies of the human mind. For Freud and for the many psychoanalytical schools of thought that followed, the boundaries between psychological health and illness were never very clear. The typical Freudian phobias, paranoia, masochism, narcissism, etcetera, were certainly not distinct diseases. The psychological inconveniences that brought (and still bring) people to the couch of the psychotherapist were thought to be symptoms produced by (repressed) conflicts between unconscious urges (id) and the conscious rational self (ego). These conflicts, like the one symbolized as the archetypical Oedipus complex, are inherent in all civilized human beings. According to Freud, everybody has a degree of neuroticism. Yet, the extent of one's inability to cope with others, oneself or the aims of society and the specific manifestations of suppressed intrapsychic conflicts depend on the particular life experiences – often from early childhood – of the individual. Consequently, the general aim of psychoanalysis is to find, make conscious and interpret the sources of conflicts and the situations in which they arise. It is not some universal disease entity, but the patient's detailed biographical history that is essential to the psychoanalytical understanding of disease. Symptoms have significance only in relation to the unique actions, experiences and emotions of the whole individual.

Between approximately 1930 and 1970, the word autism was frequently used by child psychological professionals in both Britain and the USA within this psychoanalytical framework.⁶⁵ Unlike Kanner's more or less coherent syndrome of 'early infantile autism' (1943), diagnostic terms such as childhood psychosis, childhood schizophrenia, 'atypical child,' and autism were used rather loosely and interchangeably in a psychoanalytical approach to infantile psychopathy

⁶⁵ Eugen Bleuler, who had coined the term autism in 1911, attributed its etymological roots to Freud, and ultimately to the British sexologist Havelock Ellis, through the term 'autoerotism'. Freud had used this term in 1899 in a letter to Wilhelm Fliess: 'The lowest sexual stratum is auto-erotism, which does without any psychosexual aim and demands only local feelings of satisfaction. It is succeeded by allo-erotism (homo- and hetero-erotism) but it certainly also continues to exist as a separate current' (Freud, 1950: 280).

and problems with developing relationships. Primary narcissism, infantile unconsciousness, symbolic thinking and the ‘psychotic child’s ego’ were the theoretically-laden terms in which a child’s failure to develop a stable relationship to reality was expressed. Within this framework, autism referred to infantile hallucinations, fantasies and emotional withdrawal and was understood – for example, by psychoanalyst Elwyn James Anthony – as ‘an inability to form a coherent and stable sense of self; [and] an inability to ‘cathect’ internal experiences accurately’ (Evans, 2013: 11).

The source of disturbed infantile ego development was typically the absence of a warm, intimate and stable relationship with the mother; ‘maternal deprivation’ was one of the most dominant explanations for this particular conception of autism (see Raz, 2014) and treatment focused on restoring ego functions (for example reality testing and object relations) through psychotherapy (also for the parents) and interventions in the family or educational environment. Instead of seeing autistic behavior as the result of an underlying (biological) disease process, symptoms like extreme isolation and repetitive behavior were interpreted as reactions or defense mechanisms used to protect an underdeveloped or damaged ego. Despite being bounded by Freud’s rigid explanatory framework and terminology, autistic symptoms carried meaning only by taking the history, circumstances and actions of the whole child into account. The psychoanalytical understanding of autism was not just a psychogenic theory of autism. It involved a particular perspective on normal (child) development, treatment and recovery, the interpretation of symptoms, and the notion of autism itself.

In the next section I juxtapose in more detail the distinct understandings of autism by focusing on specific scientific, therapeutic and diagnostic practices.⁶⁶ Based on a comparison between child psychotherapists in the late 1950s and 1960s and contemporary clinical psychiatrists, with regard to their gaze, cognitions, and modes of interaction with patients, it is clear the two groups are directed and limited in radically distinct ways – that is, each is mediated by a different style of thought. I will not elaborate on the multifaceted and very fascinating shift in the prevailing ‘style of psychiatric thought’ that occurred in the 1970s when, to put it bluntly, Meyerian and psychoanalytical frameworks made room for the neo-Kraepelinianism of *DSM-III* (APA, 1980) and

⁶⁶ For more on how these practices relate to the regulatory and bureaucratic practices in more exoteric communities of psychiatric health care, see Verhoeff (2014)/Chapter 4.

biological psychiatry (see Decker, 2013, for a history of *DSM-III*).⁶⁷ For the sake of clarity, I distinguish clinical (i.e., diagnostic and therapeutic) and scientific aspects of the two kinds of autism. In each subsection, current (neurocognitive) approaches to autism are contrasted with the largely discarded psychoanalytical theories and practices that were widespread in the 1950s and 1960s.

Two styles of psychiatric thought

Diagnosing: Autistic disorder versus autistic withdrawal

Currently, a valid psychiatric disorder is thought to have a characteristic pathophysiological mechanism, clinical course and response to treatment. As Rosenberg (2002) puts it, diseases have ‘a natural history that – from both the physician’s and patient’s perspective – [form] a narrative. The act of diagnosis inevitably [places] the patient at a point on the trajectory of that ineluctable narrative’ (2002: 243). In contemporary psychiatric practice, the diagnostic process is crucial and clearly separated from treatment. Every first visit to a psychiatrist or clinical psychologist starts with a series of questions and tests – the diagnostic interview – that aims at describing and structuring all the different symptoms and signs in order to diagnose or exclude a particular psychiatric disorder.⁶⁸ A comprehensive diagnostic assessment for autism may include screening tools such as the Modified Checklist for Autism in Toddlers

⁶⁷ The contemporary dominant ‘style of psychiatric thought’ has not remained unchallenged and ‘critical psychiatry,’ ‘postpsychiatry,’ ‘the recovery movement’ and ‘values based practice’ are examples of oppositional voices at the margins of academic and clinical psychiatry (see, for example, Bracken et al., 2012). However, what is generally missed in these critical accounts is how different scientific, clinical, philosophical and institutional aspects hang together in a coherent and constraining way.

⁶⁸ In his essay *The Tyranny of Diagnosis* (2002) Rosenberg points to the fact that, for about the past two centuries, diagnosis is central to the definition and management of what we call disease. He argues that diagnosis constitutes ‘an indispensable point of articulation between the general and the particular, between agreed-upon knowledge and its application. It is a ritual that has always linked doctor and patient, the emotional and the cognitive, and, in doing so, has legitimated physicians’ and the medical system’s authority while facilitating particular clinical decisions and providing culturally agreed-upon meanings for individual experience’ (2002: 240).

(MCHAT), diagnostic tools such as the Autism Diagnostic Interview (ADI) or the Autism Diagnostic Observation Schedule (ADOS), and furthermore may consist of physical examinations, hearing tests, intelligence tests, chromosomal analysis, MRI scans, electroencephalographs (EEG), and more. Leo Kanner was the first to articulate descriptive diagnostic criteria for autism (Kanner, 1943), and thereafter, many attempts to capture and specify the essential behavioral and cognitive characteristics of autism in formal diagnostic criteria followed (for instance by Eisenberg and Kanner, 1956; Lotter, 1966; Rutter, 1968; Wing and Gould, 1979; APA, 1980; APA, 1994; APA, 2013).

Despite the basic assumption that autism is a psychiatric disorder that can, in principle, be diagnosed, the clinical reality is often complex and inexact. A diagnostic verdict may be postponed, require extra information, be fiercely debated in clinical meetings (has the patient autism, ADHD, or a conduct disorder?), or it may just be complicated since many patients have comorbid conditions. Furthermore, individual variation within autism is huge and an autism spectrum disorder (APA, 2013) allows for wide variations in severity, different levels of impairment and heterogeneity of symptoms. Nevertheless, ideally, a diagnostic assessment of autism has two possible outcomes: either there is autism, or there is no autism that explains or coincides with the behavioral or emotional problems that brought someone to the clinic. Of course, there are children who only have a few signs of autism and not the full clinical picture. *DSM-III* (APA, 1980) included a category of ‘atypical autism’ and *DSM-IV* (APA, 1994) included ‘pervasive developmental disorder not otherwise specified (PDD-NOS)’ for those who did not meet the full criteria of autistic disorder, but who did have severe impairments in one of the domains associated with autism. The use of these ‘atypical’ residual categories only highlights the centrality of the conception of autism as an ideal ‘typical’ disease entity.

To deduce an ideal disease entity from often messy and idiosyncratic manifestations is one of the clinician’s core competences. The status and authority of a psychiatric clinician depends on this ‘clinical gaze’. Despite the widely acknowledged variation within the autism spectrum, autism experts (and parents of autistic children as well) often claim that they can distinguish autism from non-autism within a few minutes, whereas a medical student or a fresh psychiatrist might miss what an expert considers to be a clear case of autism. By education and experience, those who work or live with autistic children

develop, in Fleck's terms, a 'readiness for directed perception' (1979: 92) of autism.

The importance of the (descriptive) diagnostic process and systematic classification may seem self-evident today, but during times of psychoanalytical dominance in the field of child psychiatry and psychology, it was not. Even after Kanner *described* the behavioral symptoms of 'early infantile autism' as a distinct syndrome as early as 1943, the descriptive mode in child psychiatry did not become pervasive until the 1970s (Evans, 2013). Instead, child psychologists and psychiatrists were used to attributing unconscious thought processes to infants in theoretically-laden terms such as primary narcissism, symbolic thinking, hallucinatory confusion, autoerotism and autistic, unrealistic thinking. These terms could only be understood within the Freudian psychoanalytical framework in which psychopathology was typically viewed as a disturbance in the development of the ego. In particular, psychological problems in infants were believed to be rooted in anxiety caused by the infant's failure to develop, through 'appropriate' relations with the external world, a coherent and differentiated identity and self-consciousness.

Psychoanalytic theorists after Sigmund Freud, such as Anna Freud, Melanie Klein, Margaret Mahler, Donald Winnicott and Bruno Bettelheim, focused less on Sigmund Freud's stages of psychosexual development to explain psychopathology. Instead they developed an ego psychology in which mothering and 'object relations' were crucial for a child's ego development. Especially after World War II, child psychologists tended to focus more on the child's early interactions with the mother and the external world (see Nadesan, 2005). Child psychosis, childhood schizophrenia and autism were the somewhat undefined and interchangeable terms that referred to the postulated failures of the 'psychotic ego' to form a stable sense of self and to relate the self (the 'ego') to other people and objects (Anthony, 1958).

The term 'autism' did not represent a distinct syndrome with particular observable features. Within the psychoanalytical framework, autism was roughly understood as a particular type of 'withdrawal' towards a particular 'autistic position' (Bettelheim, 1967: 46). Child psychoanalysts did not tend to use the term autism as a noun, but they often used the adjective 'autistic' in combination with a variety of psychoanalytically interpreted actions and states such as 'autistic reactions,' 'autistic defenses,' 'autistic withdrawal,' 'autistic thinking,' 'autistic position,' 'autistic barriers' and so on (see, for example,

Anthony, 1958; Mahler, 1952; Tustin, 1969). Autism was not the *cause* of particular behavior, but autistic behavior was the *result* of some complex disturbance in ego development. Moreover, the basic aim of psychoanalysts was not to *diagnose* a particular disorder, but to *analyze* the child's disturbed development of the ego and its disturbed relation with the external world. Systems of classification played a minor role in psychoanalytic practice and, in the psychodynamic spirit of Adolf Meyer, the *DSM-I* (APA, 1952) mainly classified psychological 'reaction types' (Grob, 1991). For instance, a 'psychotic reaction' was defined as one in which 'the personality, in its struggle for adjustment to internal and external stresses, utilizes severe affective disturbance, profound autism and withdrawal from reality, and/or formation of delusions or hallucinations' (APA, 1952: 12).

Different psychoanalytical theorists had slightly different ideas about what caused the disturbed development of the ego. The Austrian psychoanalyst Margaret Mahler, for instance, described autistic thinking as a form of psychosis that resulted from poor ego differentiation (between the id, ego and superego) that in turn resulted from – what she called – a disturbed 'symbiotic process' (Mahler, 1952). With successful ego differentiation, a state of 'normal autism' or 'symbiosis' preceded individuation. Mahler defined this symbiotic state as 'hallucinatory or delusional omnipotent somatopsychic fusion with the representation of the mother and, in particular, the delusion of a common boundary between two physically separate individuals' (cited in Nadesan, 2005: 96). According to Mahler, successful individuation depended on the mother's empathic support for this delusion of symbiosis. In this process, the infant slowly and carefully separates from the mother by building up its representations of the mother. The mother was seen as crucial for assisting the infant with progressively differentiating the ego and interacting with the external world. For Mahler, maternal absence and a lack of the mother's support and emotional availability could result in a disturbance of this process of 'separation-individuation' that in turn could lead to anxiety, autistic withdrawal as a primitive defense mechanism and an inability of the infant to transform from a state of symbiotic fantasy to a state of differentiation between the self and the other.

Similar to Mahler, the British psychoanalyst Elwyn James Anthony used a 'barrier hypothesis' to explain a child's defense mechanisms of withdrawal, perseveration, rigidity, repetition, raised sensory thresholds, pseudo-deafness,

and the like (Anthony, 1958). He argued that in the delicate process of normal ego development, the infant's constitutional self-protecting barrier is enhanced by a barrier of the mother, which gradually makes room for an 'autonomous ego barrier'. Anthony distinguished two types of barrier disturbances that resulted in autistic defenses of what he interchangeably called an 'autistic state of mind' or a 'psychotic ego'. The first was the development of an 'abnormally thick barrier' that blocked external sensations and held the infant in a state of primary narcissism. The second was the development of an 'abnormally thin barrier' that resulted in an excessive amount of stimulation of the fragile ego of the infant. As a defense, the infant then withdraws and develops a secondary psychotic barrier which protects the infant from over-stimulation (Anthony, 1958; Evans, 2013). Similar to Mahler's symbiotic fantasy, the 'psychotic ego' of the child failed to enable the development towards a coherent sense of self, self-awareness, and a conscious distinction between inner fantasy and outer reality.

These psychoanalytical ideas about autism were clearly derived from Bleuler's use and introduction of the concept of autism. For Bleuler, autism was one of the core symptoms of schizophrenia and he regarded 'autistic thinking' or 'derealistic thinking' as an infantile defense used to escape unsatisfying realities by substituting them with hallucinations, fantasies and delusions (Bleuler, 1911/1950). Bleuler derived his concept of autism from the term 'autoerotism' – a term used by Freud to describe self-comforting fantasies of infants in a stage of development that preceded the infant's interaction with others (Freud, 1905/2001). Like Bleuler and Freud, the psychoanalytical theorists of the 1950s and 1960s assumed that – not only in abnormal but also in normal development – hallucinatory thinking and fantasy preceded the formation of 'real' connections with other people and objects. As Evans (2013: 4) convincingly argues, 'whereas "autism" in the 1950s referred to excessive hallucinations and fantasy in infants, "autism" in the 1970s referred to a complete lack of an unconscious symbolic life'. In sum, both at a substantive and a conceptual level, the psychoanalytical conception of autism is very different from the dominant present conception of autism as a brain disorder. These differences go far beyond the simple assertion that psychogenic theories differ from neurobiological theories of autism. In the next subsection I specify the differences in the role of symptoms for both kinds of autism.

The role of symptoms: Expressions of disease versus meaningful reactions

In the contemporary diagnostic process, personal experiences, feelings, behaviors and relational or emotional difficulties are reframed using a specialized vocabulary as general symptoms and signs *of* a particular disorder. Next to recognizing typical clusters of signs and symptoms as disease pictures, mastering this specialized vocabulary is also one of the basic requirement for becoming a modern psychiatrist. Symptoms of a particular disorder are typically divided into primary or core symptoms and secondary symptoms. Primary are those symptoms that are necessary and characteristic for a particular disorder. They are considered primary, because they are thought to be directly caused by a specific disease process. For example, specific neurodevelopmental mechanisms are believed to cause, sustain, underlie and explain the core autistic signs and symptoms.

Repetitive behavior (for instance hand-flapping or repetition of sounds or words) and deficits in social-emotional reciprocity (APA, 2013) are now considered primary and essential in autism spectrum disorder, whereas intellectual or language deficits are considered secondary (South et al., 2007). Secondary are those symptoms that are not the direct result of the specific disease process, but either – due to some sort of a chain reaction – the result of primary symptoms (self-injurious behavior, depression, anxiety and hyperactivity are currently seen as secondary in autism (see Matson and Nebel-Schwalm, 2007)) or, in an often unexplained way, frequently associated with core symptoms (for instance epilepsy and motor abnormalities in autism). The primary and secondary subdivisions of autism symptoms have been rather variable throughout the history of autism. In the late 1960s and early 1970s, language deficits were generally thought of as primary, whereas deficits in social interaction were thought to be a consequence of the primary language deficits (Rutter, 1968). In sum, despite the fact that the autistic symptoms are thought to cause the daily impairments and distress of those diagnosed with autism, within the biomedical framework of current practitioners, symptoms are mainly regarded as the observable markers – the epiphenomena – of the disease that hides below the surface, waiting to be revealed by the technologies of the biomedical and brain sciences that reach under the skin.

In psychoanalytically informed approaches to ‘autistic’ behavior, the behavioral symptoms are not epiphenomenal expressions of the disease. In

contrast, the various forms of autistic behavior and thought, such as withdrawal, perseveration, delusional fantasy, obsessions and body-rocking are understood as a form of ego defense against anxiety. Autistic symptoms are unconscious but meaningful and anxiety-reducing defenses of an underdeveloped ego in reaction to ego-threatening stimuli. Autistic symptoms are not caused by a disease, but are a self-protecting and understandable manifestation of the infant's entire self and they are meaningful in the sense that they can only be understood in relation to the life history of the child and as a way to cope with the particular difficulties in the development, differentiation and formation of the child's self. In a review of Bruno Bettelheim's (in)famous book on autism, *The Empty Fortress* (1967), Peter Gay, a biographer of Freud, recognizes this irreducible and idiosyncratic behavior of the autistic child:

Obviously (Bettelheim is enough of a Freudian to be convinced of this) all aspects of autistic behavior are meaningful; all of it – the twiddling, the peculiar modes of defecating, the silent rocking, the refusal to eat – is a kind of language, even if it is directed at no one. But since symptoms vary so enormously, and since the therapist has no way of checking his hunches with the patient, as he does in psychoanalysis, the interpretation of the 'language' autistic children have available to them demands the utmost concentration, intelligence, empathy, and persistence. (Gay quoted in Silverman, 2012: 69)

For a psychoanalyst, there are no primary or secondary symptoms. All symptoms have a particular meaning in the troubled development of the child's psychic life, no matter how bizarre or isolated the child's behavior may seem. Behind the seemingly meaningless expressions of the autistic child, lies the child's delicate struggle for a coherent self in a threatening sphere of libidinal impulses and drives, fears of annihilation, fears of separation, phantasies of omnipotence, moral confusion, sensory overload and demands of the super ego.

Treating autism

In current times, a particular diagnosis is coupled to a particular treatment. Guidelines, treatment algorithms for psychopharmaca, and the Randomized

Controlled Trials (RCTs) of Evidence-Based Medicine (EBM) that they are based on all rely on specific diagnostic disease entities. Moreover, the U.S. Food and Drug Administration (FDA) approves psychoactive drugs strictly for distinct diseases (instead of, for instance, for isolated symptoms) and treatments are often not reimbursed without the appropriate diagnosis. Similarly, eligibility for special education services is a particularly powerful incentive for parents to seek an autism or ADHD diagnosis. Having no specific diagnostic label means receiving no treatment, no support from the health care system, and maybe even more important, no visibility, recognition or meaningful (medical) explanatory narratives in personal, familial, social, professional and educational spheres. Again, in Rosenberg's terms, 'it is almost as though the disease, not its victim, justifies treatment' (2002: 255).

The ideal objective of current autism treatments is a correction or normalization of the specific dysfunctions or abnormalities that are assumed to define the disease. Philosopher of science Georges Canguilhem turned this idea around and argued that 'to act, it is necessary at least to localize. ... The impetus behind every ontological theory of disease undoubtedly derives from therapeutic need' (Canguilhem, 1966/1991: 39).⁶⁹ Indeed, diseases have often been demarcated through the specific response to a specific treatment (for example quinine in distinguishing malaria) and this made a strong (but circular) argument for the existence of disease-specific pathophysiologies (Rosenberg, 2002).⁷⁰ This argument is closely related to the common assumption in current psychiatric practice that cure is the highest goal of treatment, and this generally means bringing the patient back to an earlier (normal or healthy) state.

Nowadays, it is generally thought that autism patients were never in a 'normal' state and thus a complete cure would mean creating a new normal state by eliminating the inherited autism-specific dysfunctions. However, since the identification of these specific dysfunctions seems very far away, this therapeutic goal is considered naïve and might never be feasible due to the assumed complex developmental nature of the disorder. Furthermore, this

⁶⁹ This idea resembles Canguilhem's other counter-intuitive idea that 'the abnormal, while logically second, is existentially first' (Canguilhem, 1966/1989: 243).

⁷⁰ Nobody understood this better than the pharmaceutical industry when it launched *antidepressant* and *antipsychotic* medication that acted on separate monoaminergic pathways that were later (incorrectly) claimed to be the specific dysfunctional neurotransmitter pathways in depressive and psychotic disorders (see Rose and Abi-Rached, 2013: 36-37).

therapeutic goal is ethically problematic from a neurodiversity perspective. Today, autism treatment focuses not on cure, but on symptom control, care, support and helping the patient to cope with everyday difficulties. Psychotropic drugs, social skills training, applied behavioral analysis (ABA), mindfulness, psychoeducation and psychomotor therapy are examples of contemporary interventions for supporting people with autism. Currently, aims and hopes are oriented towards prevention and very early intervention as the best treatment (see, for example, Dawson, 2008).

Diagnosing and treating are not easily separated in psychoanalytic practice. The process of observing and interacting with the child, in combination with the interpretation of the variety of autistic reactions in terms of unconscious desires, fears, ego differentiation and hidden motives, was seen as therapeutic in itself. Especially the careful and devoted interaction with the child was seen as an important contribution to the child's autonomy and stable ego formation (see, for example, Kaufman et al., 1957). Bruno Bettelheim's Orthogenic School at the University of Chicago was at the same time a place where 'milieu therapy'⁷¹ was offered and a place where insights into the child's behavioral and emotional disturbances were obtained (Silverman, 2012).

Eyal et al. (2010: 143-147) notice some more interesting differences between contemporary autism treatments and autism treatments in the 1950s and 1960s. Contemporary therapies, they argue, recruit parents as co-therapists, whereas in earlier therapies the therapist took the role of a substitute parent. For instance, the children at the Orthogenic School in Chicago and at other similar schools for atypical children were removed from their families for extended periods of time and a 'loving environment' was created that was modeled upon the family. In a completely new environment, teachers, nurses, and therapists played the roles of loving and caring mothers and 'big sisters' and replaced the families from which the children were removed. Nowadays, autism treatments actively involve parents, for instance in psychoeducation, not to attempt to construct a completely new 'milieu,' but instead to create a 'prosthetic environment' for the child that enables the child to cope with and avoid everyday difficulties (ibid.).

Furthermore, contemporary therapies are 'guided for the most part by a moral narrative of construction, laying down the building blocks of

⁷¹ Milieu therapy was thought to produce psychological change through the creation of a therapeutic environment or milieu that encompassed all aspects of life. Bruno Bettelheim's Orthogenic School was such an environment.

development, while 1950s-1960s autism therapies were guided by a moral narrative of discovery, drawing the child outside the fortress represented by autism' (ibid.: 143). Behind the defenses the child has erected, there hides a creative, empathic and social child that needs to let go of her or his haven of withdrawal and come out of her or his autistic shell. It was the aim of psychoanalytical therapies to carefully penetrate the 'fortress' in a long-term process, in order to *discover* the child within and to lead the child out. In contrast, important contemporary autism therapies, such as ABA and social skills training, do not use an image of discovery, but of construction. Today's autism therapies try to create and then improve skills that were not already there. In smaller chunks of time and with possibilities to measure intermediate progress, they lay down building blocks of further development and they teach certain social and communication skills the child simply does not have (Eyal et al., 2010).

Science: Discovering versus analyzing

Whether it concerns the prevalence of autism, comparing and evaluating autism treatments, or attempts to identify pathophysiological mechanisms, etiological factors, or other biomarkers of autism with neuroimaging or (epi)genetics, autism research basically relies on the distinct diagnostic categories of the DSM or ICD classification systems. These systems of classification are increasingly being criticized for being invalid (Cuthbert and Insel, 2010) and for expanding the boundaries of abnormal social behavior (Frances, 2013). It is, however, not the general idea of defining specific psychiatric diseases in terms of neurobiology or causal mechanisms that is being challenged. Rather, it is the lack of predictive value of DSM categories in terms of response to treatment and course, as well as scientists' inability to identify disease-specific pathophysiologies or even useful biomarkers using current diagnostic manuals that is mainly being criticized.

One of the most influential alternatives to current diagnostic categories, the National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC), conventionally argues that 'identifying syndromes based on pathophysiology will eventually be able to improve outcomes' (Insel et al., 2010: 749). However, the descriptive diagnostic systems based on clinical presentation, NIMH's director Thomas Insel argues, will not lead to an accurate understanding of pathophysiology as medical history has shown that

‘disorders once considered unitary based on clinical presentation have been shown to be heterogeneous by laboratory tests – for example destruction of islet cells versus insulin resistance in distinct forms of diabetes mellitus ... [and conversely] that syndromes appearing clinically distinct may result from the same etiology, as in the clinical presentations following syphilis or a range of streptococcus-related disorders’ (Insel et al., 2010: 748).⁷²

Irrespective of the method that will turn out to be most fruitful for delineating autism on the basis of pathophysiology, the major challenges in the field of autism regarding classification, accurate diagnosis, better treatment and prevention are thought to be best surmounted after the neurocognitive basis of autism has been discovered. This order of importance is reflected in the type of autism journals that have high impact factors (for example *Molecular Autism*⁷³) and the type of research that is dominant, gets published and granted (i.e., neuroimaging and genetics, see Pellicano et al., 2014). Although (contingent) environmental and individual factors are thought to be important in so far as they affect the disease process, the identification and existence of specific malfunctioning causal mechanisms is independent of person and context. This means that autism – in theory – in patient X in Spain in the nineteenth century is similar to autism in patient Y in Japan in the twenty-first century. Again, it is apparent that this conception of autism is guided by the medical model of disease; just as epilepsy and cervical cancer might have variable environmental causes and variable social and cultural consequences, they are considered to be identifiable independent of cultural, historical or personal context. Thus, irrespective of cultural, historical and personal context, autism is a neurodevelopmental brain disorder that, nevertheless, might have place- and time-specific consequences and impairments. Cross-cultural prevalence studies for autism, in which cultural issues are secondary concerns, are an illustration of this assumption (see, for example, Kim et al., 2011).

Furthermore, as I have already mentioned, detecting early pre-syndromal signs of autism is an important goal of autism research. Early detection, it is thought, will facilitate a better understanding of the beginning of the disease process; it will facilitate new ways to early intervention and it might prevent

⁷² For more on how RdoC will try to base classification on pathophysiology, see <http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml>. Accessed 24 March 2014.

⁷³ *Molecular Autism* has the highest impact factor in 2014 of all autism journals.

autism from developing into a full-fledged psychiatric disorder. The frequency of gazing at faces, vocalizations to others, shared smiles, as well as deviancies in head growth and amygdala activity in facial emotional recognition tasks using fMRI, have all been mentioned as indicative prodromal signs in infancy of a later autism disorder (Yirmiya and Charman, 2010). However, as of yet all the investigated prodromal signs are neither necessary nor specific for autism and seem to reflect ‘normal’ variation or general disturbances in neural development.

The scientific status of psychoanalysis is widely contested (see, for example, Grünbaum, 1984) and I will not rerun the extensively debated issue of whether psychoanalysis can be regarded as a form of science. For current purposes it will suffice to mention that contemporary Anglo-American psychiatric reasoning tries to understand psychiatric problems through epidemiological, statistical and biotechnological methods. Following up on the natural sciences, mechanistic explanations, inductive generalizations and the discovery of objects (psychiatric disorders) are the main objectives of contemporary psychiatric research. Instead of drawing upon group averages and quantitative analysis, psychoanalytic theory drew on internal psychology and individual cases for theoretical refinements. Psychoanalytic ‘science’ was not about *discovering* an entity or making universalizing, law-like claims, but about *analyzing* the (often familial) factors and processes that influence the very personal ego development, and about *understanding* the meaningful and unconscious defenses of each individual child in her or his personal context.

A more systematic and standardized method of collecting data on autistic children only started to emerge in the course of the 1960s with autism researchers such as Lotter (1966), who conducted the first epidemiological study on autism, and Hermelin and O’Conner (1963), who started using neuropsychological tests and statistical methods to support their theories on autism.⁷⁴ Gradually, autism was no longer seen as an idiosyncratic reaction to a disturbed ego development, in which symptoms represented logical and meaningful responses to a pathogenic (maternal) environment. Instead, autism became a yet-to-be-identified neurodevelopmental disorder characterized by statistically correlated behavioral and cognitive deviations, grounded in the social brain (Happé and Frith, 2014).

⁷⁴ See Evans (2013) for a closer look at this development of autism research in the 1960s.

Conclusions

Autism is often presented as a discoverable disease that we get to know and understand better and better as science progresses and knowledge accumulates (see Verhoeff, 2013/Chapter 3). This chapter has argued that such an image of autism profoundly depends on a particular understanding of disease – an ontological understanding of disease – which is certainly not the only possible way to think about mental ailments. This study shows that the history and philosophy of autism need to account for at least ‘two kinds of autism’ that imply radically different concepts of disease. Moreover, these two kinds of autism are embedded in and reveal two very different and relatively coherent ‘styles of psychiatric thought’. That is to say, the ontological (disease-centered) and individual (person-centered) understandings of autism play a central role in a kind of matrix of distinct scientific and clinical practices that mark the dominant ways of thinking about autism in specific periods. In these time-specific matrices with separate diagnostic practices, systems of classification, ideas about ‘normal’ mental functioning and child development, and modes of treating children, interpreting symptoms and improving theoretical models, ideas about autism were and are shaped, directed and restricted in completely divergent ways.

As it seems, small cracks are appearing in the contemporary disease-centered, neuroscientific hegemony of psychiatric thought: the neurosciences’ ability to solve diagnostic and therapeutic issues is no longer self-evident (Bracken et al., 2012); disproportional financial investments in neuropsychiatry are being ethically challenged (Sadler, 2011); and the lack of biomarkers or targets for new treatments in psychiatry has already resulted in a decline in investments in the development of new psychopharmaceuticals (Miller, 2010). In order to address some of the uncertainties and difficulties that plague the field of autism research and practice today (for instance regarding the heterogeneity of autism, the lack of validity of the disease category, comorbidity issues, the ‘autism epidemic’ and the lack of autism-specific interventions), awareness of the historical contingency of our ways of thinking about psychiatric disease can make room for new understandings of ‘autistic’ behavior and new styles of psychiatric thought. Psychoanalytic understandings of autistic behavior are unlikely to return. However, finding a fruitful way to move beyond

a disease-centered approach, it seems to me, is the most urgent challenge facing the contemporary field of autism.

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6 | Fundamental challenges for autism research⁷⁵

Abstract

One of the central aims of autism research is to identify specific neurodevelopmental mechanisms that cause and explain the visible autistic signs and symptoms. In this short chapter, I argue that the persistent search for autism-specific pathophysiologies has two fundamental difficulties. The first regards the growing gap between basic autism science and clinical practice. The second regards the difficulties with demarcating autism as a psychiatric condition. Instead of the unremitting search for the neurobiological basis of autism, I suggest that basic autism research should focus on experiences of impairment and distress, and on how these experiences relate to particular (autistic) behaviors in particular circumstances, regardless of whether we are dealing with an autism diagnosis or not.

Introduction

Without much hesitation, autism or autism spectrum disorders are considered to be disorders of neurodevelopment. Consequently, one of the central aims of autism research is to identify the specific neurodevelopmental mechanisms that cause, sustain, underlie and explain the visible autistic signs and symptoms. It is commonly thought that fundamental questions (for example, how to classify

⁷⁵ This chapter has been published as Verhoeff B (2015) Fundamental challenges for autism research: the science–practice gap, demarcating autism and the unsuccessful search for the neurobiological basis of autism. *Medicine, Health Care and Philosophy* 18(3): 443–447.

autism; how to better diagnose autism; how to treat and cure autism; how to prevent autism; etc.) can best be answered after there is a better understanding of the neural basis of autism (Insel and Daniels, 2011). In view of this biomedical framework, it is no surprise that the majority of autism research indeed focuses on ‘basic science’ – ‘on neural and cognitive systems, genetics and other risk factors’ (Pellicano et al., 2014: 757).

In this short conceptual analysis, I argue that this focus upon the search for autism-specific pathophysiologies implies two rather underestimated difficulties. These difficulties pose an urgent challenge for contemporary autism research. The first challenge regards the gap between (basic) autism science and the day-to-day difficulties of those diagnosed with autism. Paradoxically, despite the unremitting hope that autism neuroscience will lead to translational benefits for autistic patients, the tenacious effort to identify the underlying neurobiology of autism (see, for example, Jeste and Geschwind, 2014) seems to widen the gap between autism science and clinical practice.

The second challenge relates to the difficulties with demarcating the boundaries of autism and, at a somewhat more philosophical level, the boundaries of health. Obviously, this longstanding philosophical problem regarding the distinction between health and disease will not have an easy solution. However, the dramatically increasing prevalence of autism with current estimates of one in 68 children (Centers for Disease Control, 2014), the alarming indications and prophecies of medicalization, overdiagnosis, false epidemics and rising healthcare costs (see Frances and Widiger, 2012), and – from a different angle – the emergence of neurodiversity movements that proclaim that autism is not a disease to be cured but atypical brain wiring that needs to be respected illustrate the significance of integrating ideas about health and disease in the field of autism (Jaarsma and Welin, 2012; Kapp et al., 2012). Of course, these very complicated issues are not fully explored in this short chapter. Nevertheless, I try to argue why these fundamental issues deserve more explicit attention in the dynamic field of autism research.

The Science-Practice Gap

There is a substantial gap between the scientific perception and investigation of autism as a neurodevelopmental or biological ‘thing,’ and the clinical and individual experience of autism as a heterogeneous and variable cluster of symptoms associated with impairment of particular forms of social behavior (APA, 2013). Of course, there is nothing suspicious about this gap as such. In order to make generalizing (scientific) claims, reductions of ‘real-world’ problems into measurable and researchable objects are inevitable. Science and everyday practice will never completely coincide. However, attempts to reduce a particular conception of autism to specific neurobiological and cognitive circuits that are thought to underlie and ultimately define autism, have not been very successful yet.

Despite the dominance of (social) neuroscientific research in the field of autism (Pellicano et al., 2014), efforts to identify reliable diagnostic biomarkers, meaningful (biological) subgroups, autism-specific genes or neural circuits, and targets for brain-based and psychopharmacological interventions remain disappointingly unproductive. Current candidate biomarkers for autism – such as particular genetic variants, different brain structures, brain functions, and neuropeptides – are not found in all autism cases (poor sensitivity) and they tend to be associated with many other neurodevelopmental disorders and ‘normal’ conditions (poor specificity). In short, they are not valid or clinically useful (Walsh et al., 2011). In addition, the diagnostic category of autism has proved to be rather variable in time and heterogeneous in its manifestations (Verhoeff, 2013/Chapter 3; Waterhouse, 2013). A general sense of uncertainty and dissatisfaction with autism research is well exemplified by the following comment by autism expert Michael Rutter (2014: 55): ‘It seems decidedly odd that after more than half a century of both research and clinical experience with autism spectrum disorders (ASDs), there continue to be arguments on the nature of autism’. In other words, the nature of autism remains disturbingly unknown.

The usual response to this uncertainty about the nature of autism is an appeal to complexity; autism researchers try to ‘explain the enigma’, ‘unravel the mystery’, and ‘solve the puzzle’ of autism (see Frith, 1989). This appeal to complexity legitimizes further research and, together with the optimistic hope of actually unraveling this mystery of autism in the near future, it guarantees the

flow of autism research funds. Potential unifying accounts of autism varied from cognitive deficits (for example, a defective theory of mind or weak central coherence) in the 1980s, to genetic and structural abnormalities in the 1990s, to the functional and neurodevelopmental disturbances of the twenty-first century (for example, Geschwind and Levitt, 2007). And today, the uncertain search for a common denominator continues at increasingly complex levels of molecular genetics and neural connectivity (see Auffray, 2014). Hypotheses regarding distinct neural circuits that involve many genes, different brain areas, connectivity patterns, developmental trajectories and functional brain networks are the new promises for a neuroscientific basis of the autism spectrum. The ‘enigmatic’ image of autism; the faith in the very existence of a complex neurobiological basis of autism (Kiser et al., 2015); the growing socio-economic ‘burden’ of autism (Buescher et al., 2014); and the high hopes for and prophecies of specific biological treatments for autism, resulted in a significant growth in basic autism research in the past few decades (Bishop, 2010). In the future, these factors will only further attract funding for autism neuroscience in order to unravel the mystery of autism and, accordingly, they will enable promising careers for autism neuroscientists (Dawson, 2013).

However, instead of clarifying and alleviating the devastating behavioral and cognitive difficulties and distress of those diagnosed with autism, basic autism research increasingly complicates the neurobiological image of autism. Furthermore, the numerous attempts to identify specific pathophysiological mechanisms and cognitive deficits, and the construction of the symptom-based autism category affect each other constantly (Verhoeff, 2014/Chapter 4). While autism researchers are digging deeper into the unrestricted complexities of the brain, on the clinical side of the divide, autism has become a common, broad, heterogeneous, and – in clinical terms of prognosis, course and response to treatment – unspecific category for people with restricted patterns of behavior and deficits in social interaction (APA, 2013). In a dynamic process, the search for common neurobiological (causal) mechanisms has to rely on this heterogeneous category of autism symptoms, and, the other way around, the lack of decisive and distinctive findings from basic autism research played an important role in conceptualizing autism as a broad spectrum disorder (Happé, 2011).

The tentative, probabilistic, multilevel and multifactorial hypotheses regarding the neural basis of the elusive category of autism do not give much

hope for future clinically valuable translations from the neurosciences (see also Waterhouse, 2013, Chapter 8). Instead, current ideas about the biological nature of autism seem to be moving away from the everyday, very diverse and contextual ailments of those diagnosed with autism. Thus far, the very idea of autism, an autism spectrum, or several autisms, in combination with the idea of specific neurobiological mechanisms that are supposed to underlie these clinical syndromes, has driven basic autism research further and further into the infinite complexities of the brain. Whether and how the complex molecular levels at which autism is currently imagined will ever become clinically valuable is very uncertain and should be a topic of urgent debate in the field of autism. This debate should include a critical evaluation of the scientific and clinical benefits of the autism (spectrum) phenotype and of current attempts to identify its neurobiological foundation.

Demarcating autism

Another challenge for autism research regards the issue of demarcating autism as a psychiatric condition. What makes autism a pathological condition? Where does autism stop and normality begin? What is appropriate social interaction? Who is a suitable case for treatment? Whose treatment should be reimbursed? Undeniably, these types of questions concerning the boundaries of particular ailments are as old as the discipline of medicine itself. However, today, the biomedical and neurosciences are expected to solve these challenging issues. Demarcating autism can and should be done – it is thought – by identifying the underlying malfunctioning neurobiological circuits. For it is in these brain circuits, in their neural connections, in their systems of neurotransmission, in their genetic, cellular and molecular processes and their patterns of activity that ‘true’ psychiatric syndromes should be delineated (see Cuthbert and Insel, 2013). This approach would not only enable nosologists to solve persistent debates about whether it is better to lump autism as a single entity or to split autism into various subtypes according to distinct neuronal and cognitive pathways, but it would also distinguish between the dysfunctional and the normal neural circuitry of the social brain.

However, until now, autism neuroscientists have not been able to point out how and when parts of the brain work improperly. What we currently know

about neurobiological ‘abnormalities’ in autism derives merely from associations with the autism phenotype, and not from conceptions of (failures of) normal biological or cognitive functioning provided by the neurosciences. Theoretically challenging ideas about brain dysfunctions or dysfunctional neural pathways have not provided psychiatry with concrete methods to demarcate its territory. Nevertheless, the neurosciences are saddled with this daunting task of ultimately demarcating autism. And because the burden of truly defining autism and its specific neurobiological substrate lies on the brain sciences, revisions of diagnostic criteria are mainly directed at creating a valid category that facilitates the identification of pathophysiological mechanisms (APA, 2013). In this search for specific neurobiological dysfunctions, demarcating the healthy from the suitable cases for treatment is of lesser importance. For instance, difficult demarcation issues like how to separate appropriate from inappropriate ‘back-and-forth conversation’ or a normal need for regularity from abnormal ‘insistence on sameness’ (APA, 2013) are not addressed in a theoretical or methodical way.⁷⁶ Instead, creating a valid disease category is primarily focused on clustering separate signs and symptoms into a statistically coherent whole.

In the meantime, prevalence rates of autism keep rising (Centers for Disease Control, 2014). More and more children are recognized as autistic, as socially impaired, as restricted in their interests, and as neurodevelopmentally disordered. Simultaneously, criticisms of the medicalization and pathologization of normal childhood, of the lack of tolerance and acceptance of human diversity, and of the (Big Pharma-induced) creation of false epidemics have become commonplace. The field of autism does not convincingly answer these criticisms and these criticisms tend to erode public confidence and trust in autism research and practice. Why is it that certain forms of social interaction, eye-contact, body language, imaginative play, and so on, are considered deviant, and at what point do they become deviant? This issue remains implicit and hardly debated in the process of classifying autism. It is not demarcating

⁷⁶ Of course, specific cases of autism need to be demarcated in some way. In clinical practice, this is done with DSM criteria including the criterion of clinical significance. That is, symptoms must cause clinically significant distress or impairment in social or occupational functioning. However, as the definition of mental disorder in *DSM-5* (APA, 2013) illustrates, distress and impairment in mental disorder are secondary and need to be caused by biological or psychological dysfunctions. The clinical significance criterion is seen as a currently necessary but imprecise and unscientific threshold for mental disorders.

abnormal or unhealthy behavior as such, but creating a valid cluster of signs and symptoms and identifying a neurobiological substrate that is paramount in classifying autism. This approach has lost its vital connection with present-day clinical and societal concerns.

Furthermore, this disconnection between constructing a valid category of particular behaviors and ideas about ‘the pathological’ or the need for psychiatric treatment, made the emergence of neurodiversity movements possible (Jaarsma and Welin, 2012; Kapp et al., 2012). For these movements, autism has nothing to do with being healthy or unhealthy. Autism, they claim, is not a disease to be cured but a valid biological category of atypical brain wiring that needs to be respected. According to Jaarsma and Welin (2012: 28), ‘some autism ... can be seen as a natural variation on par with for example homosexuality’. This idea will not help to demarcate autism, but it does bring us to the daunting demarcation problem that needs more attention in the field of autism: how do we separate those (with autism) who need medical treatment and support from those (with autism) who only need acceptance and respect.

Discussion

This chapter merely touched upon some major uncertainties in the field of autism. But, relative to the enormous number of autism studies that are being conducted, these grand themes are rarely discussed and deserve an active debate. A recent special issue in *Autism* titled *Autism and Society* advocated that ‘high-quality research into the social dimensions of autism is as necessary and valuable as basic scientific research into autism’ (Singh and Elsabbagh, 2014: 754). It is hard to disagree with this, but I think it is also time to reconsider the objectives and fundamental assumptions of basic autism research itself. I argue that current scientific perceptions of autism as a complex neurodevelopmental disorder drift away from the diversity of the problems and experiences of those diagnosed with autism. Today, much autism research centers its hope on the neurosciences, but in order to reconnect with the growing socio-cultural, economic and clinical concerns regarding, among other things, the ‘autism epidemic,’ autism research should not wait for the neurosciences to illuminate this phenomenon.

Instead of the persistent search for the neurobiological basis of autism, I suggest that basic autism research needs to focus more on notions and experiences of impairment, disability, suffering and distress, and on how these experiences relate to particular (autistic) behaviors in particular circumstances, regardless of whether we are dealing with an official autism diagnosis or not. Obviously, this is not to exclude neuroscientific or fundamental research. Rather, basic autism research could, for instance, focus on the neurobiological mechanisms that are involved in distinct behavioral difficulties and patterns of impairment and distress that occur in specific social, familial and cultural contexts, instead of explaining these vital experiences away by referring to the elusive entity of autism. This would require a diagnostic system that is not based on abstract disease entities, but on concrete behaviors and types of distress.

By focusing on the various types of impairment, capabilities, experiences and resilience of the ailing individual, autism research will need to emphasize what it means to be healthy or diseased, and it will need to stress the contextual elements of autistic behavior that cannot be explained by neurobiology alone. In doing so, autism research will come closer to clinical practice and the everyday struggles of those we have come to call autistic. Furthermore, with such a focus on various and contextual types of impairment and suffering, autism research will be equipped to constructively contribute to heated public debates concerning the medicalization and pathologization of childhood. These suggestions are, obviously, preliminary and not much more than an invitation to rethink some of the fundamental objectives and assumptions of basic autism research.

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7 | Kurt Goldstein on autism⁷⁷

A physician's thought and activity are incomprehensible without the concepts of the normal and the pathological. (Canguilhem, 2008: 121)

An organism that actualizes its essential peculiarities, or – what really means the same thing – meets its adequate milieu and the tasks arising from it, is “normal.” Since this realization occurs in a specific milieu in an ordered behavioral way, one may denote ordered behavior under this condition as normal behavior. (Goldstein, 1934: 325)

Abstract

Autism research is facing profound difficulties. The lack of clinically valuable translations from the biomedical and neurosciences, the variability and heterogeneity of the diagnostic category, and the lack of control over the ‘autism epidemic,’ are among the most urgent problems facing autism today. Instead of encouraging the prevailing tendency to intensify neurobiological research on the nature of autism, I argue for an exploration of alternative disease concepts. One conceivable alternative framework for understanding disease and those we have come to call autistic, can be found in the work of neurologist Kurt Goldstein (1878-1965). His person-centered approach provides radically new ways to investigate and intervene with the behavior we are accustomed to explain by the elusive entity called autism.

⁷⁷ This chapter is under review at *History and Philosophy of the Life Sciences*.

Introduction

Autism research seems to be hitting a wall. Up to now, the search for autism's neurobiological foundation has been largely unsuccessful. The increasingly complex neurodevelopmental image of autism (see, for example, Jeste and Geschwind, 2014; Happé and Frith, 2014), the lack of clinically valuable translations from the biomedical and neurosciences, and the variable, heterogeneous and irreducibly sociocultural diagnostic category of autism (Verhoeff, 2013/Chapter 3) expose some fundamental uncertainties of contemporary autism research. Bridging the gap between research and practice turns out to be much harder than expected (Bauman and Schumann, 2013). Moreover, the field of autism is unable to establish some form of control or vision regarding the expanding boundaries of abnormal social behavior (see Frances, 2013).

These difficulties partly stem from the idea of autism as a distinct biomedical disease and emerge when asking the fundamental question that guides, directs and dominates autism research, namely: 'What is autism?' And today, an answer to this question seems further away than ever. Nevertheless, answering this question becomes more and more pressing because of the growing need for better treatments, more precise diagnoses, classifications and prognoses, and early detection, intervention and prevention of autism.⁷⁸ Accordingly, despite the fundamental difficulties and uncertainties in autism research, neuroimaging, neurogenetics, social neuroscience and also epidemiological studies of autism are flourishing (see Bishop, 2010). Furthermore, autism is increasingly present in the popular imagination and an increasingly popular topic in social, historical and science studies (for example, Silverman, 2008; Evans, 2013). In short, autism's (social) reality is pervasive and the search for autism's nature is, albeit disappointingly fruitless, more active than ever.

However, irrespective of the seemingly unavoidable idea of autism as a diagnosable psychiatric disorder and irrespective of what future autism research might bring us, this paper explicitly tries to sidestep the idea of autism as a distinct diagnostic category and the persistent attempts to understand and

⁷⁸ Part of this need is motivated by financial rewards; a recent economic analysis estimated the cost of autism was more than that of any other medical or psychiatric condition (Buescher et al., 2014).

explain its identity. In this paper, I will not investigate – from whatever scientific, social, philosophical, pragmatic, phenomenological, historical or cultural perspective – autism’s reality. Instead, I will propose an alternative to the pervasive ‘ontological understanding of psychiatric disease’ in which psychiatric diseases exist independently of their unique appearances in particular individuals (see Temkin, 1977; Verhoeff, 2014/Chapter 4). Something of a conceivable alternative framework for understanding disease and the difficulties, impairments and suffering of those we have come to call autistic, I suggest, can be found in the work of neurologist Kurt Goldstein (1934/1995). In his ‘so-called holistic, organismic approach’ (p. 18), in which it is not a disease entity but the ‘performance of an organism’ (p. 42) that is central, symptoms ‘are only expressions of the organism’s attempt to deal with certain demands of the environment’ (p. 35). For Goldstein, disease, normality and recovery are states that cannot rely on a statistical norm or an assumed disease entity, but are founded upon the person’s abilities. Symptoms and signs are not merely manifestations of an underlying disease process, as ‘no phenomenon should be considered without reference to the organism concerned and to the situation in which it appears’ (p. 40).

Goldstein’s ‘individual understanding of disease’ entails a different way of thinking about normal human functioning and abnormality. His understanding of disease challenges the divides that mark contemporary psychiatric thought – the divides between a disease and its milieu and between biological processes and social factors and contexts. Goldstein provides new ways to think about, investigate and intervene with the behavior we are now so accustomed to explain by the as yet unidentified entity called autism. In the section ‘Goldstein versus Kanner,’ I discuss Goldstein’s uninfluential article *Abnormal mental conditions in infancy* in which he explicitly opposes Leo Kanner’s understandings of autism. The section ‘Disease, health and the milieu’ uses the work of Georges Canguilhem to enrich and clarify Goldstein’s unorthodox notions of health and disease. But before that, and before I discuss Goldstein’s holism, his ideas about the ‘abstract attitude’ and ‘catastrophic reactions,’ and his person-centered approach in relation to the behaviors and impairments we have come to call autism, I want to say a bit more about recent developments in autism research. I hope to show why it might be time to start thinking beyond the idea of autism as a distinct diagnosable disease.

Autism in the brain

What is autism? After more than seventy years of both research and clinical experience with autism, this question remains almost as puzzling as when Leo Kanner introduced the syndrome in 1943 (Kanner, 1943). Very recently, eminent child psychiatrist Michael Rutter concluded that it is ‘decidedly odd that’ after such a long period of thorough and committed autism research, ‘there continue to be arguments on the nature of autism’ (Rutter, 2014: 55). Likewise, Christopher Gillberg – a well-known autism expert – acknowledged that ‘the fact is that we do not know what autism “is.” I have been in the field for forty years, and I can honestly say that I do not believe we are any closer now than [we were] twenty years ago to a real understanding of what it is about autism that makes experienced clinicians “certain” that it is autism regardless of whether operationalized criteria for the disorder are met or not’ (Gillberg in Waterhouse, 2013, ix). Nonetheless, despite (or maybe because of) the persistent elusiveness of autism, autism research has never been more vigorous. Additionally, the close linkages between diagnostic practices, the clinical and popular gaze of the – I must admit convincing – prototypical *Rain Man*-like autism case, the scientific attempts to identify unifying neurobiological mechanisms, and the biomedical understanding of psychiatric ailments in general make the very reality of autism something of a *fait accompli* (see Verhoeff, 2014/Chapter 4). Notwithstanding the persistent elusiveness that surrounds the unsolved puzzle of what we have come to call autism, the idea of autism as a distinct disease category plays a pivotal role in directing and structuring childhood mental health care.⁷⁹

Currently, the burden of delivering on the hopes and promises for a better understanding, treatment and prevention of autism lies on the (social) neurosciences. Mental disorders in childhood are increasingly conceptualized as ‘neurodevelopmental, meaning that they are linked to abnormal brain development’ (Insel, 2014: 1727). In the field of autism, unraveling ‘the social brain’ (Kennedy and Adolphs, 2012) and tracing the atypical neurodevelopmental trajectories related to delayed or abnormal social cognition has become the most promising route to progress (Happé and Frith, 2014).

⁷⁹ See Rosenberg (2002; 2006) and Jutel (2013) for more on the central role of disease entities in the many aspects of modern medicine. For autism in particular, see Verhoeff (2014)/Chapter 4.

Similarly, after rethinking autism's variation, complexity and complete absence of useful biomarkers (for diagnosis, prognosis, prevention or treatment), autism authority Lynn Waterhouse suggests that advances in the field of autism research 'will depend on increased knowledge of the genetics, epigenetics, and gene–environment interactions involved in brain development, as well as ... increased knowledge of individual brain circuits, the whole brain connectome, and the mechanisms of the dynamic processes involved in brain development'. She argues that 'future discoveries should be able to better isolate specific brain circuits that when disrupted result in neurodevelopmental social impairment' (Waterhouse, 2013: xiii).

These tendencies in autism research are in line with the 'Research Domain Criteria' (RDoC) project of the National Institute of Mental Health (NIMH). This project aims to develop a classification system based upon dimensions of neurobiology that reflects advances in genetics, neuroscience and cognitive science. Current descriptive diagnostic systems (and disease categories), it is argued, lack validity because they rely upon presenting signs and symptoms that 'do not adequately reflect relevant neurobiological and behavioral systems' (Cuthbert and Insel, 2013: 1). Progress in genomics and imaging, combined with computational abilities, is thought to facilitate the identification of 'biomedical tests for routine clinical practice' and neurobiologically homogeneous populations 'that [cut] across the traditional diagnostic boundaries while simultaneously transforming them' (Kapur et al., 2012: 1178). Even though this shift in focus 'from behavioral symptoms to neurodevelopmental trajectories' (Insel, 2014) is often presented as a 'paradigm shift,' it is not as radical as it seems. Indeed, methods used to identify biomarkers and underlying pathophysiologies differ, but fundamental assumptions about the nature of psychiatric disease remain unaffected – whether with *DSM-5* or RDoC, autism is thought of as a disease like any other medical disease. More specifically, autism is a *brain* disease and it is in this organ that an answer to the question 'what is autism?' can and should be found.

Even though autism's indispensability is facing several profound difficulties⁸⁰, the search for autism is far from stagnating. The obstacles of

⁸⁰ Historical variability of the concept of autism, heterogeneity in symptomatology, the lack of successful translations from bench to bedside (no biomarkers), the expanding boundaries of autism, and an increasingly complex picture of autism's neurobiology are few of the fundamental problems of current autism research.

autism research and autism neuroscience in particular do not lie in the innovative efforts of autism researchers creating new hypotheses related to autism's neurobiology (Verhoeff, 2014/Chapter 4). It is not the neurobiological, reductionist and context-neglecting approach of autism neuroscience itself that is hitting a wall. However, whether autism neuroscience proceeds constructively and whether it produces or will produce convincing scientific accounts of autism's neurobiology is far from clear. As it seems, the limits of autism neuroscience do not lie in the innovative efforts of autism researchers creating new hypotheses related to autism's neurobiology, but they lie, today at least, in successfully translating neuroscientific results into clinical care and practice.

Goldstein's holism

Very different from current neurobiological, reductionist and ontological approaches to autism is Kurt Goldstein's holistic-organismic approach to disease. Kurt Goldstein (1878-1965), a German neurologist and psychiatrist who wrote his major theoretical work *Der Aufbau des Organismus*⁸¹ right after his flight from Nazi Germany (Goldstein, 1934/1995), based his ideas about normal and abnormal living beings on his extensive research on aphasia and on his experience with treating and training those who incurred brain injuries during World War I. A lot can be, and has been, said about the socio-political context of national fragmentation and political instability that attracted many Germans to ideals of 'wholeness' in the interwar period (see, for example, Harrington, 1998; Hau, 2000; Rosenberg, 1998). However, for present purposes I will focus on Goldstein's understanding of disease and pathological behavior and how his holistic method provides a radically different 'style' of looking at those diagnosed as autistic.

In *Der Aufbau des Organismus*, Goldstein introduces a new holistic method by which he believed 'more justice may be done to the description and understanding of the behavior of normal and pathological living beings' (Goldstein, 1995: 17). Long-term observations of and neuropsychological tests with some 2000 brain-injured soldiers made Goldstein question the adequacy

⁸¹ Later translated as *The organism: A holistic approach to biology derived from pathological data in man*.

and usefulness of the conventional approach of 'cerebral localization' for understanding brain pathology. Knowing the exact location of a brain lesion in a wounded soldier did not help the therapy, nor could the location of a lesion, according to Goldstein, explain the extraordinary complexity of pathological behavior manifested by the patient. The basic error in analyzing brain lesions, Goldstein argued, was the assumption that 'cortical injury is usually followed by a loss of circumscribed functions, such as speech, visual perception, or motor performance. ... According to this conception, [neurologists like Carl Wernicke and Paul Broca] distinguished and designated various disease syndromes by such terms as aphasia in its various forms, visual agnosia, apraxia, and so on. They assumed also that circumscribed centers controlled those particular functions' (p. 33). Within this dominant localizationist model, 'we have become so accustomed to regard symptoms as direct expressions of the damage in a part of the nervous system that we tend to assume that, corresponding to some given damage, definite symptoms must inevitably appear' (p. 35). This way of thinking about neurological and psychopathological symptoms was 'borrowed from reflexology and the prevailing association psychology' (p. 36). According to the main 'reflex theory' of the medical and psychological sciences, the organism represented 'a bundle of isolable mechanisms that are constant in structure and that respond, in a constant way, to events in the environment (stimuli)' (p. 69). The idea of separate mechanisms (or modules) and the tight relationship between brain structure and function, and between specific stimuli and specific responses 'led to the supposition that circumscribed injuries would result in disorders specific to the mechanisms involved' (p. 36).

However, Goldstein challenged these central elements of the contemporary neurological localization of (mental) pathologies and the associationist psychology of reflex. The 'atomistic method⁸²,' he extensively argued, failed to provide a deeper understanding of the way the entire organism tried to compensate for internal disturbances. It failed to explain the organism's capacity to adapt and reorganize after severe trauma, and it was unable to account for recovery of function after brain injury. Furthermore, the localizationist model did not account for the well-recognized variability of symptomatology. As Harrington (1998: 28) puts it, 'the simple fact that brain-

⁸² For Goldstein, this method 'intended to designate any method which uses a dissecting procedure and tries to derive laws from the parts studied' (Goldstein, 1963: 3).

damaged people can get better over time, can regain lost speech and movement, was simply incompatible with the nineteenth century “machine” model of the nervous system as a purely mechanical apparatus operating according to fixed laws of reflex and association’.

Instead of focusing on specific lesions, reflexes and particular disturbances, and instead of assuming that individuals with similar lesions were affected in similar ways, Goldstein focused on the ‘performance’ of the individual organism, as it was affected by the disturbance. Brain damage, and cortical injury in particular, he argued, ‘does not result in the loss of isolated performances but in systematic disintegration following the principle that certain forms of behavior will be impaired while others remain intact’ (Goldstein, 1995: 45). To understand these impaired forms of behavior; that is, to ‘provide a meaningful description of the symptoms,’ it is necessary to take ‘into account the organism as a whole’ (p. 40). Not local lesions, but performances of the entire organism – defined by Goldstein as ‘any kind of behavior, activity, or operation ... that expresses itself overtly and bears reference to the environment’ (p. 42) – are essential in understanding pathological behavior. There is, Goldstein argues, no direct relation between a brain lesion and disruption of behavior:

Whether a certain symptom will appear on account of a local injury, especially whether it will become a permanent symptom, certainly depends on many factors: on the nature of the disease process, on the condition of the rest of the brain, on the state of the circulation, and on the psycho-physical constitution of the patient. It also depends on the ‘difficulty’ of that performance, the disturbance of which represents the symptom, and, finally, on the reaction of the entire organism to the defect. (p. 207)

Just as normal behavioral reactions are expressions of the organism’s attempt to deal with certain demands of the environment, symptoms (abnormal reactions) ‘are answers, given by the modified organism, to definite demands: they are attempted solutions to problems derived on the one hand from the demands of the natural environment and on the other from special tasks imposed on the organism in the course of examination’ (p. 35). This idea that symptoms and disease must be understood in relation to particular tendencies

and performances of the entire individual, and not in a bottom-up and atomistic manner from specific disturbance to specific behavior, is central in Goldstein's holistic alternative for investigating both normal and abnormal living human beings.

The abstract attitude, catastrophic reactions and coming to terms with the world

The analysis of behavioral changes in patients suffering from brain injuries led Goldstein 'to make a distinction between two modes of behavior – the abstract and the concrete' (Goldstein and Scheerer, 1941: 1). These modes of behavior, Goldstein argued, are dependent upon two corresponding attitudes that 'are not acquired mental sets or habits of an individual, or special isolable aptitudes, such as memory, attention, etc. Rather, they are *capacity levels of the total personality*' (original emphasis). In all his patients with brain injuries Goldstein noticed a diminished capacity for abstract thought and a strong tendency towards concrete behavior. In 'concrete' performances 'a reaction is determined directly by a stimulus, is awakened by all that the individual perceives. The individual's procedure is somewhat passive, as if it were not he who had the initiative'. In 'abstract' performances, on the other hand, 'an action is not determined directly and immediately by a stimulus configuration but by the account of the situation which the individual gives to himself. The performance is thus more a primary action than a mere reaction, and it is a totally different way of coming to terms with the outside world' (Goldstein, 1940/1963: 61-62). It is not isolated performances or cognitive functions, but the general abstract attitude – an essential attribute of the human being and the basis for '*conscious and volitional* modes of behavior' (Goldstein and Scheerer, 1941: 4) – that is affected in those with cortical brain injuries: 'We venture to remark that whenever the patient must transcend concrete (immediate) experience in order to act – whenever he must refer to things in an imaginary way – he fails. On the other hand, whenever the result can be achieved by manipulation of concrete and tangible material, he performs successfully' (Goldstein, 1995: 43).

Goldstein provides numerous examples of disturbed abstract performances. One patient, for instance, is asked to drive a nail with a hammer into a piece of wood. This task does not cause any difficulties. However, when the nail is taken

away and the patient is asked to imagine that there is a nail, he is unable to make the movement of hammering. Even if he sees the nail 'he is unable to make the movement of driving the nail in' (Goldstein, 1963: 45). Another patient could use a key to open a door, but was 'unable to demonstrate how to use a key without the door present' (Goldstein and Scheerer, 1941: 7). Some patients could find their way while walking from the hospital to their home, but they could not draw a map or give a verbal account of their route. They tended to succeed in the 'sphere of immediacy,' but failed in the more abstract 'sphere of the possible':

Each problem that forces him [the patient] beyond the sphere of immediate reality to that of the 'possible,' or to the sphere of representation, ensures his failure. This manifests itself in all responses such as action, perception, thinking, volition, feeling, and so on. The patient acts, perceives, thinks, has the right impulses of will, feels like others, calculates, pays attention, retains, and so on, as long as he is provided with the opportunity to handle objects concretely and directly. He fails when this is impossible. (Goldstein, 1995: 43)

One patient could throw a ball into different boxes at distinct distances. However, he could not estimate the distances nor could he say which box was nearer or farther. Another of Goldstein's patients could count on his fingers, but was unable to state whether 7 or 4 was more 'and had no concept of the value of numbers whatsoever' (Goldstein and Scheerer, 1941: 7). For these patients, words only referred to concrete objects, but categories, concepts, analogies and metaphors were not understood since abstractions of a common property were necessary. 'They fail on a simple syllogism or on tests of finding the common denominator of several items'. This disturbance in what Goldstein called 'categorical behavior' did not imply that patients were unable to select items, for instance from a heap of colored wooden skeins, on the basis of particular characteristics such as brightness, softness, or color. However, a patient 'who seems to be choosing according to a certain attribute is not able to follow this procedure voluntarily if it is demanded of him ... [and] he does not seem to be able to hold to a certain procedure' (Goldstein, 1963: 72). The lack of a grasp of the abstract, the lack of an approach to imagined things or

hypothetical situations, and the inability to reflect on one's acting or thinking are

also the reason [the brain-injured patient] can grasp a little story as long as it concerns a familiar situation in which he himself has participated. But he will not understand a story – certainly no more difficult for the average person – requiring him to place himself, in imagination, in the position of someone else. He does not comprehend metaphors or puzzles. He can manipulate numbers in a practical manner but has no concept of their value. He can talk if there is some concrete subject matter present but cannot recount material unrelated to him or report it in purely conceptual terms. He is incapable of representation of direction and localities in objective space, nor can he estimate distances; but he can find his way around very well and can execute actions that are dependent on perception of distance and size. ... The most general formula to which change can be reduced is probably that the patient has lost the capacity to deal with that which is not real – with the possible. (Goldstein, 1995: 43-44)

Pathological phenomena, Goldstein argued, should not be considered 'as curiosities caused by illness and therefore not intelligible in the same way as the behavior of normal individuals ... they become intelligible if one takes into consideration the characteristic alterations which illness produces' (Goldstein, 1963: 35). These alterations are not just the result of a disturbance of the abstract attitude directly caused by brain pathophysiology. Goldstein emphasized that a patient's reduced performance at a concrete level of functioning was also an adaptive reaction of the whole individual to preserve or return to an *ordered* condition and to avoid a *catastrophic* reaction. For Goldstein, this is a very important second distinction that helps to understand the injured condition of the organism: the distinction between *ordered* and *disordered* or *catastrophic* behavior. Again, this distinction can only be understood by considering 'the total behavior in which the individual performance appears' (Goldstein, 1995: 48).

In an ordered situation, responses appear to be constant, correct, adequate to the organism to which they belong, and adequate to the

species and to the individuality of the organism, as well as to the respective circumstances. The individual himself experiences them with a feeling of smooth functioning, unconstraint, well-being, adjustment to the world, and satisfaction, that is, the course of behavior has a definite order, a total pattern in which all involved organismic factors – the mental and the somatic down to the physicochemical processes – participate in a fashion appropriate to the performance in question. ... The ‘catastrophic’ reactions, on the other hand, are not only ‘inadequate’ but also disordered, inconstant, inconsistent, and embedded in physical and mental shock. In these situations, the individual feels himself unfree, buffeted, and vacillating. He experiences a shock affecting not only his own person, but the surrounding world as well. He is in a condition that we usually call anxiety. (Goldstein, 1995: 48-49)

Not only in ‘normal’ individuals, but also in brain-damaged patients, Goldstein recognizes a universal tendency toward ordered behavior and an avoidance of those situations that threaten the very existence of the individual, that is, catastrophic situations. Crucial in this tendency are the (often unconscious) attempts to ‘come to terms with the world,’ which means being able to handle or cope with the tasks and demands that inevitably arise in interaction with the environment. Ordered behavior or a correct and successful performance ‘is a coming to terms of the organism with environmental stimuli by a behavioral act, be this eyelid closure under stimulation or a total movement like running toward a goal, or hearing, seeing, and so on’ (p. 42). In the organism’s coming to terms with the world, or, in other words, in meeting ‘its adequate milieu and the tasks arising from it’ (p. 325) the environment of an organism ‘is by no means something definite and static but is continuously forming commensurably with the development of the organism and its activity ... an organism can exist only if it succeeds in finding in the world an adequate environment – in shaping an environment’ (p. 85). Ferrario and Luigi (pp. 217-218) note that in defining ‘performance’ – Goldstein’s central unit of investigation – as a ‘coming to terms,’ it is obvious that ‘the organism as a whole is always called into question, and that the organismic behavior will always be holistically oriented in line with this kind of “bio-ecological” finality’.

In Goldstein’s patients with brain injuries, the loss of the abstract attitude (that is, among other things, the lack of an approach to imagined things or

hypothetical situations and the inability to reflect on one's acting or thinking) diminished the capacity to cope with (new) tasks imposed by the environment. This made his patients extra vulnerable to catastrophic reactions and Goldstein systematically described the various and powerfully motivated adaptive strategies patients had developed to avoid the overwhelming anxiety of catastrophe, and to maintain, albeit in a reduced, rigid, and automatic manner, an adequate feeling of functioning and adjustment to the world. In other words, his patients tended toward concrete and ordered behavior: 'Avoiding catastrophic situations is possible only if he is able to come to terms with the world in spite of his defects – that is, only if he finds a new milieu which is appropriate to his defective condition, a milieu from which no stimuli arise which put him into a catastrophic condition' (Goldstein, 1963: 95).

One of the strategies, or 'substitute performances' as Goldstein calls them, is the patient's 'tendency towards excessive and fanatical orderliness'. For instance, in putting several objects at random on a table, the patient 'will at once arrange them in some order' (p. 101). After an examination with one of his patients, Goldstein drops his pencil on a sheet of paper and the patient immediately 'takes up the pencil, straightens the paper carefully so as to bring its sides parallel with the side of the table, and then as carefully places the pencil parallel to the margin of the paper'. When the pencil is put in an oblique position, the patient reacts once more by putting it in back into the parallel position. 'Apparently,' Goldstein argues, 'such a state of "disorder" is unbearable to him'. Furthermore, his patients are 'punctual in their daily activities, in bathing, going to bed, etc., doing everything at the prescribed time'. All patients with brain injury, Goldstein concluded, 'have a tendency toward such "primitive" order. ... The principal demands that "disorder" makes on them are choice of alternative, change of attitude, and rapid transition from one behavior to another. But this is exactly what is difficult or impossible for them to do' (Goldstein, 1995: 54). An unstructured, 'chaotic' environment creates tasks that make these demands, and catastrophic reactions and anxiety inevitably ensue. To avoid this anxiety the patient 'clings tenaciously to the order that is adequate for him but that appears abnormally primitive, rigid, and compulsive to normal people' (p. 54).

Another strategy, when confronted with a new task which the patient cannot perform, is endlessly repeating an earlier performance. The patient 'avoids a catastrophic situation indirectly *by busying himself with those things which he*

is able to do' (Goldstein, 1963: 99, original emphasis). The things the patient tends to cling to have 'the character of stereotypy and exhibit little variation' (Goldstein, 1995: 52). They keep the patient occupied and 'so secluded from the outside world that he remains unaffected by many events of his environment' (p. 53). Unexpected stimuli are dangerous and avoided as they might create a situation that demands a particular adjustment that the patient cannot make. The patient 'tries at all costs to avoid the unknown' (Goldstein, 1963: 100). Goldstein mentions that his patients go for a walk only if they have a specific goal. 'They do not stroll about, for strolling about contains in it many dangers of abrupt stimulation. Thus the patient avoids it, and may even resist going to a known goal by an unfamiliar route'.

A final strategy that I would like to discuss is the patient's 'avoidance of emptiness' or 'abhorrence of a vacuum, a *horror vacui*' (p. 104). When Goldstein's patients were faced with an empty space or a situation which did not contain a possibility for the patient to react upon, this caused the patient to become anxious and troubled. One patient, for instance, could not write on an empty sheet of paper. However, he could write if there was a line on the paper he could write upon. Another patient was unable to read letters or words if they were not written on a line. These incapacities, Goldstein argued, did not consist in an inability to read or write without lines, but 'in the inability to do anything without clinging to a given concrete object' (p. 105). His patients tried to avoid these kinds of situations of emptiness because empty space was not an adequate stimulus and demanded an abstract attitude, which was exactly what was lacking. They evaded the difficulties that arose in emptiness by clinging to a concrete object which they could cope with, 'knowing that as soon as he gives up his point of reference he will become helpless, ineffective, disturbed, and driven to catastrophic reaction' (Goldstein, 1995: 55).

All these substitute performances imply a tremendous restriction of the environment in which the patient lives: 'a defective organism achieves ordered behavior only by a shrinkage of its environment in proportion to the defect' (p. 56). Goldstein describes the various forms of pathological behavior and substitute actions from the perspective of the whole organism and not as the direct result of brain pathophysiology. For Goldstein, many particular symptoms could only be understood as a 'means by which existence can be maintained. In this sense, they are meaningful; they enable the organism to come to terms with the environment, at least in some way' (p. 52).

Goldstein versus Kanner: A different perspective on autistic behavior

As far as I can tell, there is only one paper by Goldstein in which he explicitly evaluates Leo Kanner's presentation and interpretation of the new syndrome of 'early infantile autism' (Goldstein, 1959; Kanner, 1943). Goldstein, who wrote *Der Aufbau* in 1934, was astonished by the way in which Kanner's autistic children (Kanner, 1943) reacted 'in ways closely reminiscent of brain-injured adults with impairment of abstraction' (Goldstein, 1959: 554). The inflexible and restricted behavior, the particular relationship to persons and objects, the inclination to have temper tantrums, the lack of imagination, the inability to understand jokes and metaphors, the inability to reflect on one's action or to take someone else's perspective, and the strong dependence on others for survival and finding a way in the complex world, were features that Goldstein repeatedly observed in both 'abnormal infants' and in brain-injured adults. He was convinced that 'the so-called autism and the "desire for the maintenance of sameness" could also be understood from our point of view, namely, that we are dealing with an impairment in the mental capacity, particularly of that function which we call abstract attitude' (p. 539).

Goldstein had already noticed that 'the ["normal"] child behaves, in some respects, similar to the brain-injured patient' (Goldstein, 1995: 238). The child is frequently confronted with tasks with which he cannot come to terms in an adequate way. Stimuli originating from the child's environment 'do not yet fit the organism of the child, they demand reactions corresponding to a more mature and more integrated organism than the child actually is' (p. 249). Anxiety and a need for predictability, regularity and, for instance, bedtime rituals play a great role in a child's life. The condition of infancy, Goldstein argued, 'is suited to bring about catastrophes easily ... Because the human child is born in an immature state, it is often exposed to inadequate conditions in the environment' (Goldstein, 1959: 538). Once separated from the mother's body, the child has lost 'the "adequate" condition of the womb, and is confronted with many catastrophes. It has to find a new "adequacy" if to survive and develop. That presupposes that the infant is protected against the dangers of the external world. This protection comes ... from the activity of persons around him' (p. 540). However, for a child with an 'abnormal mental condition,' this protection is not sufficient as he does not develop 'the abstract

capacity, which increasingly enables him to organize himself and his world, and so to guarantee his existence' (p. 541).

Goldstein's theoretical constructs of 'the abstract capacity' and 'catastrophic reactions' provide him with a different interpretation and explanation of the 'autistic' behavior originally described by Kanner. Kanner, for instance, emphasized a particular type of asymmetry regarding the way in which autistic children interacted with objects and persons. Kanner argued that his children 'are able to establish and maintain an excellent, purposeful, and 'intelligent' relation to objects that do not threaten to interfere with their aloneness, but are from the start anxiously and tensely impervious to people, with whom for a long time they do not have any kind of direct affective contact' (Kanner, 1943: 249). Yet, Goldstein argued that the children's particular interaction with both objects and person can be explained by an overarching lack of the abstract capacity. This involves an inability to handle and understand whole objects and a behavioral repertoire that is restricted to 'primitive' and concrete reactions to isolated stimuli. He argued that, with only primitive reactions available, 'there is very little the infant can do with the person' (Goldstein, 1959: 544). Objects, on the other hand, present more reactive possibilities to the child than do persons: 'the infant can grasp objects, move them, squeeze them, put them into his mouth; in a word, there is a varied performance repertoire available'. For Goldstein, it is not the case that these children behave normally toward objects and abnormally toward persons because, as Kanner thought, objects 'do not threaten to interfere with their [desire for] aloneness'. Instead, 'abnormal children show only reactions to parts – to those parts they can do something with "adequately" – although it may seem as if they are reacting to the whole object' (p. 544). Objects are just more appropriate than persons in the child's attempt to prevent catastrophe and to come into an ordered relationship with the environment.

Kanner explained the awkward social behavior of the autistic child – not making eye contact or not paying any attention to other people in the same room – by assuming that for the child, the other person does not exist because he has no emotional tie to others: 'these children have come into the world with innate inability to form the usual, biologically provided affective contact with people' (Kanner, 1943: 250). An 'inborn autistic disturbance of affective contact' and 'a powerful desire for aloneness and sameness' were, for Kanner, the basic deficits in early infantile autism. Goldstein saw something else. With

his idea of primitive reaction types, the ‘autistic’ child ‘reacts only to “stimuli” and not to whole persons’ (Goldstein, 1959: 545). Goldstein did not assume a specific (defective) inborn ability for affective contact. Persons were treated instrumentally, ‘not because the child has no feeling for the former but – due to his primitive way of reacting – because he has only one way to treat both persons and objects’ (p. 546). For Goldstein it was sufficient to assume that the autistic child ‘is urged to do something, and does what he is able to do in a particular situation, resisting demands that he do something he realizes he cannot do. When the demand is not so strong as to bring catastrophe, he seems not to pay attention to it’ (p. 545). The tasks and demands that arise in interaction with other people are too complex to handle; they require particular abstract abilities.

It was also unnecessary to assume a special drive for aloneness or repetition. Kanner’s assumption of an ‘obsessive desire for the maintenance of sameness’ could be explained as being analogous to the ‘tendency to orderliness and repetition’ of Goldstein’s brain-injured patients. Goldstein assumed that the repetitive behavior and the inflexible routines were the only reaction forms which permitted the autistic child ‘to come into an adequate relationship with the “world” and so actualize himself in striving to cope with the world. He sticks to it under all circumstances, and he comes into catastrophe when something is demanded of him that cannot be fulfilled in this way’ (p. 546). Thus, a small change in a routine or a disturbance of repetitive behavior implied new demands and a potentially dangerous, disordered situation. The repetitive behavior and the ‘aloneness’ should not be understood as intentional or desired, but simply as a function of the inability to do anything else, and due to this inability the child perseverates in these activities. Goldstein did not assume several specific drives that directed particular behavior, but assumed only *one* different kind of general driving force which determined both normal as well as abnormal behavioral conditions. This general driving force was the organism’s tendency to realize its possibilities, or, in other terms, a ‘tendency to actualize itself according to the circumstances’ (Goldstein, 1995: 355). For the child with deficiencies of the abstract attitude, who has only ‘primitive’ and concrete performances at his disposal, this ‘self-realization,’ as we will also see in the next section, ‘is possible only in a reduced form’ (Goldstein, 1959: 556).

Disease, health and the milieu

No contemporary author has cited Goldstein more frequently than the French philosopher of biology and medicine Georges Canguilhem. On the issue of normality, pathology, health, and the milieu, Canguilhem was deeply influenced by Goldstein's work.⁸³ When Canguilhem famously argued that 'health is the margin of tolerance for the inconstancies of the environment,' (Canguilhem, 1991: 197) or that 'disease is a narrowed mode of life, lacking in creative generosity because lacking in boldness ... characterized by new physiological constants,' he directly referred to Goldstein's clinical experiences and theoretical interpretations. As Geroulanos and Meyers (2012) also remind us, Canguilhem worked from a perspective influenced by 'the surgeon René Leriche and ... Kurt Goldstein, both of whom helped him to question the claim – which in France dated back to Francois-Joseph-Victor Broussais and Auguste Comte – that disease and the pathological condition more generally are nothing more than modifications of the normal condition' (in Canguilhem, 2012: 2). Canguilhem shared with Goldstein an attention to and recognition of the complexity of pathological experience and behavior in a way 'that was at odds with the positivist normative conception of health deriving from Comte' (p. 3). According to Canguilhem, positivist medicine and positivist conceptions of disease and biology reduced individual reactions to expressions of a disease, and considered them merely as 'aberrations from normality that could or could not be corrected; in so doing, they also effaced the experience of suffering and even of health itself'.

As we have seen, for Goldstein, pathological phenomena were not merely normless 'disordered' conditions or deviances from a 'supra-individual' statistical or 'idealistic' norm. He argued that the statistical norm 'cannot be used to determine whether a given individual is to be regarded as normal or abnormal. The statistical norm concept cannot do justice to the individual' (Goldstein, 1995: 326). And it is only the individual as a whole to which states of health, disease and normality can be ascribed. The normal and the pathological fundamentally depend upon the capacities and experiences of the individual and not upon isolated anatomical structures or processes. Goldstein showed with his patients that an understanding of particular modifications in

⁸³ See, for example, Canguilhem, *Knowledge of Life* (2008): 113-114 and 129-132 and *The Normal and the Pathological* (1991): 181-196.

individual performances and experiences was central to understanding disease. As Goldstein put it, 'pathological phenomena are the expression of the fact that the normal relationships between organism and environment have been changed through a change of the organism and that thereby many things that had been adequate for the normal organism are no longer adequate for the modified organism' (p. 328).

Goldstein's patients were abnormal not because of a lack of ordered behavior (apart from the catastrophic reactions) or because of a deviance from a healthy norm. Instead, Goldstein saw in his patients a tendency to establish a new relationship with the environment – a new norm of life – that was characterized by a tenacious adherence to concrete and ordered behavior in a new but narrowed environment. Disease is still a norm of life, albeit inferior in several ways: 'it tolerates no deviation from the conditions in which it is valid, incapable as it is of changing itself into another norm' (Canguilhem, 1991: 183). As Canguilhem paraphrases Goldstein and generalizes this point,

The norms of pathological life are those that oblige the organism to henceforth live in a 'shrunk' milieu, which differs qualitatively, structurally, from its former milieu of life; the organism is obliged by its incapacity to confront the demands of new milieus (in the form of reactions or undertakings dictated by new situations) to live exclusively in this shrunk milieu. (Canguilhem, 2008: 132)

Health, the vital contrary of 'pathological' or 'disease' implies something more than 'normality' in the sense of a life regulated by norms.

Now, to live, already for animals and even more so for man, is not merely to vegetate and conserve oneself. It is to confront risks and to triumph over them. Especially in man, health is precisely a certain latitude, a certain play in the norms of life and behavior. What characterizes health is a capacity to tolerate variations in norms on which only the stability of situations and milieus – seemingly guaranteed yet in fact always necessarily precarious – confers a deceptive value of definite normalcy. Man is truly healthy only when he is capable of several norms, when he is more than normal. (p. 132)

Exactly opposite to the positivist conception of disease as modifications from normal (physiological) functions, disease, at least for Goldstein and Canguilhem, is a conservative, defensive and static condition. As Goldstein's cases exemplify, the inability of the organism to shift modes of behavior, leave the 'sphere of immediacy,' and adapt to different environments characterizes the pathological condition. What Goldstein learned from pathology was that 'the tendency to self-preservation is characteristic of sick people ... For the sick person the only form of actualization of his capacities which remains is the maintenance of the existent state. ... Under adequate conditions the tendency of normal life is toward activity and progress' (Goldstein, 1963: 141-142). In Canguilhem's more philosophical terms: 'the sick living being is normalized in well-defined conditions of existence and has lost his *normative* capacity' (Canguilhem, 1991: 183, emphasis added). For Canguilhem, this 'normative capacity' is a prerequisite for health and it means 'the capacity to establish other norms in other conditions'. Health is more than a 'coming to terms with the world' by avoiding catastrophic situations. Instead, through Goldstein's extensive experience with pathological conditions, he came to see the urge for new experiences, 'for the conquest of the world, and for an expansion of his sphere of activity in a practical and spiritual sense' (Goldstein, 1995: 238) as an expression of the organism's 'natural' tendency to realize or actualize its own essence. An organism is truly healthy when this 'tendency toward self-actualization is coming from within' and when it 'overcomes the disturbance arising from the clash with the world, not out of anxiety but out of joy of conquest' (p. 239).⁸⁴

As health and disease fundamentally depend upon the (modified) performances of the individual as a whole, expressed by Goldstein in terms of 'self-preservation,' 'self-actualization' and a 'coming to terms with the world,' it is only in relation to particular circumstances that an individual can be called abnormal. 'The sick person must always be judged in terms of the situation to which he is reacting and the instruments of action which the environment itself offers him ... There is no pathological disturbance in itself: the abnormal can be evaluated only in terms of a relationship' (Canguilhem, 1991: 188). Consequently, the divergence from a physiological, anatomical, behavioral or

⁸⁴ Obviously, Goldstein's (crypto-) teleological image of man is not unambiguous. For discussions on vitalism and teleology in relation to the work of Goldstein, see Ferrario and Corsi (2013).

neuropsychological constant becomes pathological only in relation to a personal ‘milieu of life’ in which certain tasks have become unavoidable for the individual living being. Furthermore, and this makes up the core of Goldstein’s holistic approach, it is always the totality of the organism that attempts to come to terms with the world or reacts catastrophically in relation to the milieu. All this implies that the ‘pathological in man cannot remain strictly biological, for human activity, work, and culture have immediate effect of constantly altering the milieu of human life’ (Canguilhem, 2008: 128).

However, the fact that, for instance, an anatomical irregularity can become pathological in one person and normal in the other, does not lead to the conclusion that a distinction between the normal and the pathological cannot be made. To individualize the norm and the normal does not erase this distinction, nor does it mean that individuals always react differently in similar situations. Goldstein’s holism – the effort to explain disease by referring to the individual’s tendency for wholeness in its way of dealing with its situation – made Canguilhem conclude ‘that human biology and medicine are, and always have been, necessary parts of an “anthropology.” ... [and] that there is no anthropology that does not presuppose a morality, such that the concept of the “normal,” when considered within the human order, always remains a normative concept of properly philosophical scope’ (p. 133).

Beyond autism: A person-centered style of psychiatric thought

Now let us get back to where we started: autism. In my attempt to sidestep the idea of autism as a distinct disease entity by searching for an alternative understanding of disease, it is not a coincidence that I came up with Goldstein’s work. Even though he mentioned autism in only one of his many papers (Goldstein, 1959), his ideas about the abstract capacity and what happens when a patient loses this capacity, fit, as we have seen, strikingly well with the behavior of those diagnosed with autism. Besides Goldstein’s own examples of cortical brain injuries, autistic behavior could even be regarded as a paradigmatic case for his particular holistic approach. However, remarkably enough, Goldstein’s interpretation of autistic behavior and his holistic approach to disease have been largely neglected in autism research, the history of autism and psychiatry in general. His paper *Abnormal Mental Conditions in Infancy* (1959)

has only been cited some thirty times, while Kanner's landmark paper on autism (Kanner, 1943) has become the central reference and starting point in autism research and has been cited more than 7000 times. Goldstein's lack of impact on autism research becomes somewhat less remarkable when we consider the fact that Goldstein and Kanner not only interpreted autistic behavior differently, but they also had very different concepts of disease and more broadly, a very different 'style of psychiatric thought' (Verhoeff, 2014/Chapter 4).

In line with Kanner's disease entity approach to autism, the current field of autism research and practice, like psychiatry in general, is structured around disease categories and the idea of specific pathophysiological processes. Goldstein's approach to medicine is indeed radically different and although it never translated into an active and established clinical or research practice,⁸⁵ his work is extremely rich and cohesive in a sense that it connects a broad range of experimental, theoretical, conceptual, therapeutic, diagnostic, anthropological and philosophical elements. Due to the cohesive and thoroughly different way of explaining and looking at behavioral ailments, Goldstein's work provides something of a conceivable alternative framework for current understandings of autism. In the spirit of Ludwik Fleck (1935/1979) and in contrast with the current biomedical approach to autism, I consider Goldstein's approach an example of a person-centered style of psychiatric thought. How such a different style comprises new ways to think about, investigate and intervene with the behavior we have come to call autism can be illustrated by pointing out some general differences with the current biomedical approach or 'style of psychiatric thought' in the field of autism.

As I mentioned in the introduction, an ontological understanding of disease, in which psychiatric diseases exist independently of their appearances in individuals, is what makes the idea of autism as a psychiatric disease possible. For Goldstein, in contrast, disease and symptoms can only be understood by taking the particular circumstances of the whole person into account. It is the history of the entire person and not a natural history of an assumed disease entity that is central in this 'individual understanding of disease'. Hence, a

⁸⁵ Nevertheless, Goldstein's work has been important for the emergence and development of humanistic psychology (Noppene, 2001).

disease category like autism, in which underlying pathophysiological processes are thought to cause and explain the visible signs and symptoms, simply does not make much sense when particular behaviors can *only* be understood with reference to the tendencies of the entire individual. Likewise, the diagnostic practice of structuring different symptoms and signs in order to diagnose or exclude a particular disorder, or to distinguish a distinct disease picture from other conditions such as ADHD, becomes unthinkable. Instead of disease entities, individual performances and experiences are the central units of a ‘Goldsteinian’ medical analysis.

Moreover, in a ‘Goldsteinian’ approach, diagnostic interviews and observations are not focused on detecting and revealing deviances from an ideal or statistical norm, such as the behavioral deviances that are expressed in the *DSM-5* (for example, ‘highly restricted, fixated interests that are abnormal in intensity or focus,’ ‘abnormal social approach and failure of normal back-and-forth conversation’ or ‘abnormalities in eye contact and body language’ (APA, 2013) as criteria for autism spectrum disorder). Nor will these symptoms, and the impairment and suffering that derive from them, be interpreted as expressions of malfunctioning neurobiological circuits that underlie social cognition and behavior. Instead, following Goldstein’s line of thought, mental health professionals will emphasize an individual’s specific attempts, possibilities and limitations to come into an ordered relationship with the surrounding world.

Furthermore, central clinical and research topics will not put emphasis on how and to what extent patients deviate from behavioral, socio-cognitive or neurobiological norms or which cluster of symptoms forms a valid disease category; instead they concern the way in which symptoms, phenomena, experiences and performances are functionally significant for the whole organism and how they relate to the (in)abilities to cope with demands from the (social) environment. Goldstein’s style of medical thought also includes a focus on the way in which particular environments, for instance at school, at home, or in the community, are inextricably linked to healthy and pathological conditions, instead of seeing environmental factors only as external etiological factors that affect distinct pathophysiological processes. It also includes a focus on (the reduction of) the capacities and possibilities to reshape one’s personal milieu, on the occurrence and avoidance of catastrophic situations, or in Canguilhem’s terms, on how individuals express and overcome a reduction in

the ‘margin of tolerance for the inconstancies of the environment’ (Canguilhem, 1991: 197). Moreover, treatments do not primarily aim at correcting dysfunctions towards a normal state, but instead they aim at creating or regaining ‘responsiveness’ or ‘normativity’. That is, enabling someone to get a grip on new situations, adapt to new circumstances, overcome difficulties, and to live a life beyond ‘the maintenance of the existent state’ (Goldstein, 1963: 141). In short, biomedical and person-centered styles of psychiatric thought each involve a distinct psychiatric gaze, with different problems to be solved that are expressed in different terms, against different background ideas about disease, normality, the environment, and what a proper treatment requires.

Conclusions

The aim of this paper was to present something of a conceivable alternative framework for understanding disease and the difficulties, impairments and suffering of those we have come to call autistic, without actually referring to the disease category of autism. I have argued that an approach in the style of Goldstein circumvents the idea of autism by providing a completely different understanding of disease. No matter how ‘syndromic,’ meaningless and neurobiologically determined the repetitive behaviors and restricted interests of autism patients may look, Goldstein shows that current understandings of autism are less inevitable as they seem. Obviously, this anachronistic move of bringing Goldstein’s approach to the present is hypothetical, and we are currently far away from a person-centered style of psychiatric thought. Today’s psychiatry does not start with the experiences, performances and impairments of the individual; rather, psychiatry is trying very hard to become ‘applied neuroscience’ (Bracken et al., 2012). However, Goldstein’s anthropocentric perspective is attractive for several reasons, and not only for the clinic but also for the research lab.

At first, Goldstein’s holism goes beyond the traditional divides that mark contemporary psychiatric thought – the divides between biological mechanisms and social factors and contexts and between ‘the disease’ and ‘its milieu’. And these are precisely the divides that seem to drive autism research and clinical practice further apart. The existing gap between the uncertain search for autism’s neurobiological specificity and the everyday, highly contextual ailments

of those diagnosed with autism does not give much hope for future clinically valuable translations from the biomedical sciences. Psychiatric (autism) research that focuses more on the development, abilities, performances, impairments and distress of the total human being that constantly shapes and is being shaped by his surrounding world, could be a first step in bridging this gap.

Secondly, Goldstein's conception of disease and normality avoids the contradictory attempts to demarcate 'abnormal' *social* behavior by identifying malfunctioning *biological* mechanisms. Currently, the boundaries of autism depend more on creating a coherent ('valid') disease entity that should enable the identification of a neurobiological substrate, than on ideas about where normal social behavior ends and abnormality starts. For Goldstein, as we have seen, there is no pathological behavior or disturbance in itself, and the 'abnormal' should be evaluated only in terms of a relationship between the individual and his surrounding world. Instead of leaving the demarcation of autism and disease to the biomedical sciences, including statistics and epidemiology, these issues become, as Canguilhem also argued, explicitly part of an anthropology and hence, a morality. This implies for psychiatry what most psychiatrists already know: that psychiatry is, and will be, much more than 'applied neuroscience'.

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8 | Conclusion: Searching for autism in a sea of flux

We are not so presumptuous as to pretend to renovate medicine by incorporating a metaphysics into it. If medicine is to be renovated, it is up to physicians to do so at their risk and to their credit. (Canguilhem, 1966/1991: 34)

Is autism a single disease? Is it a spectrum of different disorders? Are its causes genetic, environmental, social, or a mixture of these? Is it a disorder of social cognition, social motivation, or of processing and integrating perceptual information? These are questions that continue to concern those who try to solve the autism puzzle. With this dissertation I did not aim to add a piece to this puzzle but, instead, I have tried to understand what it is that makes these questions possible to ask and to determine what type of puzzle needs to be solved. I have investigated how it is possible to continue diagnosing autism, to continue doing fundamental research on autism, and to continue perceiving clear cases of autism, despite the fact that historical and conceptual perspectives reveal that the idea of autism is notoriously elusive, heterogeneous, socially-inflected and variable in the way it is and has been conceptualized. I began this dissertation with an exploration of these issues, which provided important insight into the development of psychiatric knowledge. In this conclusion I will not restate the separate conclusions of each chapter, but I will briefly return to some of the Fleckian analyses and look at a couple of broader implications for the field of psychiatry.

A brief return to Fleck

In tracing the philosophies that are operative in the field of autism today, particularly in Chapter 2, I did not run into any confident reductions of autism to well-delineated neural substrates or clear-cut genetic variations. Similar to what Fitzgerald (2012) concludes after multiple interviews with autism

neuroscientists in the UK, I found high degrees of uncertainty and ambiguity regarding the nature of autism. Among autism researchers and clinicians, one of the few certainties is that autism is a very complex and heterogeneous disorder that resists any straightforward neuro-reduction. The search for autism is no longer a search for a neatly delimited entity. It is a search for something more spread out, dynamic, and continuously affected by personal and environmental circumstances. Autism researchers are not looking for a single brain area, a brain lesion or a genetic mutation that is supposed to underlie and explain autistic behavior. Partly due to developments in the basic neurosciences, autism research has moved beyond the classical localizationism that became widespread in nineteenth-century medical thought towards an ‘extended localizationism’ that involves different brain areas, neural pathways and molecular mechanisms that underpin and sustain the capacities for human social interaction. The entire brain and its seemingly boundless potential for opening up new scales of investigating sociality has become the ‘extended’ space in which the puzzle of autism is supposed to be solved. Environmental and cultural factors might matter as well, as long as they affect the brain.

In short, the ‘autism puzzle’ is particularly tricky. To be honest, this finding is hardly remarkable. It would be somewhat incredible to conclude that autism researchers think of autism as a simple disease with a well-detectable pathophysiology and a clear etiology, course, prognosis and treatment. If that were the case, then what would explain the lack of convincing etiological factors or valuable biomarkers of autism? Certainly, this lack is not due to a lack of fundamental autism research. Instead, a convincing account of autism’s neurobiology needs to account for complex epigenetic processes; hundreds of possibly-involved genes; pleiotropy; neuroplasticity; gene-gene interactions; gene-environment interactions; multiple functional brain networks; neurodevelopmental processes; variable manifestations of autism (in time, place and person); and many contradictory findings.

Chapter 4 explained how these complexities and ambiguities played a role in the reframing of autism as a neurodevelopmental spectrum disorder. What is more, with the rise of a neurodevelopmental autism spectrum disorder, autism’s complexity and ubiquitous heterogeneity – covered by the ‘spectrum’ idea of autism – transformed into an inherent aspect of the syndrome. This new object of investigation ensured and facilitated further research and new hypotheses regarding the neurobiological specificity of autism. I argued that understanding

autism as a neurodevelopmental spectrum disorder is not a sign of explanatory success. Rather, it is a sign of explanatory quandary.

More precisely, my inquiry into the development of knowledge of autism exposed a particular dynamic. Rather than a progressive understanding of autism as a stable and discoverable object, which is the implicit message in many histories of autism (see Chapter 3), a reshaping of the idea of autism and the search for autism's essence go hand in hand. Ambiguous and heterogeneous research findings required researchers to make conceptual changes, create new categories, and construct new hypotheses regarding autism's core features in order for autism to remain a legitimate object of scientific scrutiny. A recurrent pattern in the development of knowledge of autism can be discerned. This pattern involves a search for common ground (specificity), problems with heterogeneous findings and doing adequate justice to the complexity of the clinical picture, and shifting emphasis toward new potential common ground at phenotypical – but also cognitive and biological – levels.

However, as I described in Chapter 6, the search for autism's neurobiological foundations has an underestimated downside. The connected processes of reshaping the idea of autism and searching for autism's essence resulted in increasingly complex neurobiological accounts of autism, while on the clinical side of the divide, autism has become a common, broad, heterogeneous, and clinically unspecific category. Contrary to what basic autism researchers proclaim, this development does not give much hope for future clinically valuable translations from the neurosciences.

To be clear, I do not consider basic autism research invalid or mindless due to its attempts to reduce the normativity and complexity of autism or social behavior to a neural substrate. In Chapter 2 I criticized the overall neglect of autism research of cultural norms inherent in assessing social interaction, the context-dependency of impairments, and the historical transformations and fluctuating boundaries of the category of autism. Initially, I considered the 'natural kind approach' in autism research to be misguided. In the process of writing later chapters, I came to understand this neglect not so much as misguided, but as a form of resistance to anything that might contradict the perception of autism as a distinct disease (see Chapters 4 and 5). As I have argued throughout this dissertation, in both the 'esoteric' and 'exoteric' autism communities, the tendency to objectivize and reify autism is strong. Autism

creates impairments, causes disabilities, makes people suffer, and produces special needs. But it can also come with talents and exceptional qualities. Despite the still unknown neurodevelopmental nature of autism, autism has a life of its own and is thought to cause and explain all sorts of experiences.

As Chapters 4 and 5 argued, an ‘ontological understanding of disease’ is a central element of contemporary psychiatric research and practice. This concept of disease is important insofar as it fulfills a particular role in the field of psychiatry. It is not just an abstract idea, but an idea used in different places with all sorts of material, institutional, technological, ethical and rhetorical implications and functions. The idea that autism exists independently of its particular manifestations in individual patients affects diagnostic tests and decisions, patient-physician interactions, treatment options, decisions regarding special services, the interpretation of symptoms, the organization of mental health care, research designs, research goals, biographical narratives, identity formation, senses of responsibility and accountability, and so on. Without this idea, which is an explicit reification, these practices and ethical and societal connotations become rather unintelligible. Claiming that the diagnostic category of autism is merely based on conventions and nothing more than a convenient category for clinicians or researchers misses this point and ignores the historical connections that legitimize the ‘existence’ of autism in the first place. The ontological understanding of disease is such a historical connection and a condition of possibility for almost anything that happens in the field of autism today.

In sum, numerous factors including a particular medical tradition, the medically-educated and DSM-trained clinical gaze of psychiatrists and other mental health professionals, the clinical and scientific utility of disease categories, the bureaucratic needs of health administration, and the practices of autism-oriented advocacy groups all create a dense social, material and cognitive network in which autism achieves a seemingly inevitable stability. Autism has become a form to be directly perceived. This ‘*readiness for stylized (that is, directed and restricted) perception*’ (Fleck, 1979: 84) of autism makes it possible to continue to perceive a variable mixture of seemingly independent signs and symptoms as expressions of a specific, identifiable disease. The persistent search for autism’s neurobiological basis can be regarded as an almost inevitable part of the contemporary style of psychiatric thought. Current, increasingly complex neurobiological accounts of autism are neither mindless reductions of reality,

nor products of an original and creative ‘free unfolding of ideas’ (ibid.: 84). Rather, they can be understood as solutions that follow from highly stylized ‘signals of resistance’ in thinking.

Even though the stability of the very idea of autism requires conceptual adjustments and consequently, a shifting image of autism, the dense, interconnected system of knowledge about autism resists major changes. Autism research develops as it does because of the history it has; because of the historically and socially preconditioned ways of seeing and thinking about autism and psychiatric ailments in general. As Ludwik Fleck argued with regard to syphilis, ‘it is not possible to legitimize the “existence” of syphilis in any other than a historical way’ (ibid.: 23). Like syphilis, the concept of autism ‘could not be attained without the consideration of particular historical connections’ (ibid.).

Furthermore, the tenacity of the system of ‘facts’ about autism causes the things that do not fit into the system to remain unnoticed or, in Fleck’s terms, ‘laborious efforts are made to explain an exception in terms that do not contradict the system’ (ibid.: 27). For instance, the current fact that autism is a lifelong neurodevelopmental disorder challenges the experience that some children diagnosed with autism recover at an older age. This experience is sometimes ignored and other times contested – either the patient did not fully recover, or she did not have autism in the first place. Similarly, someone diagnosed with autism for the first time at an older age must have had autism as a child, even though it might have been latent due to favorable circumstances. These findings do not neatly fit into the system of factual knowledge of autism. Another type of active resistance to contradictory data is demonstrated in Chapter 3. This chapter argued that in many histories of autism, practitioner-historians tend to see, describe, recreate and focus upon earlier facts and descriptions of autism which corroborate current perspectives and thereby give them substance. Moreover, the reification of autism enabled other social and psychological phenomena, such as autism neurodiversity and self-advocacy movements and autistic subjectivities, which, in turn, have the capacity to further solidify the idea of autism.

It is important to note that the rigid and constraining structure of views on autism is not an obstacle to the production of knowledge. Quite the contrary; in fact, true statements can *only* emerge out of such a dense network of social, historical, material and cognitive connections – Fleck’s active and passive

linkages – that direct what can be seen, thought and done. Furthermore, in the dense network of autism knowledge, in which an avalanche of autism research creates countless and highly detailed and specialized (neurobiological) scientific findings about autism, the active connections – those that are explicable in terms of history, culture or psychology (both individual and collective) – tend to become invisible. This is an additional explanation for the strong tendency to reify autism. As a result of education, training and active participation in a scientific field, the corresponding thought style will appear imperative. Fleck argues that:

A universally interconnected system of facts is thus formed, maintaining its balance through continuous interaction. This interwoven texture bestows solidity and tenacity upon the ‘world of facts’ and creates a feeling both of fixed reality and of the independent existence of the universe. The less interconnected the system of knowledge, the more magical it appears and the less stable and more miracle-prone is its reality, always in accordance with the thought style of the collective. (ibid.: 102, quoted in Klaassen, 2014: 45)

Thus, the more active you are in a relatively closed (scientific) field of autism research, the more definite and independent some sort of idea of autism becomes. For example, the more you know, learn and talk about the genes, molecules and brain activities that are correlated with autism, and the more you know about autism’s prevalence and the neurocognitive distinctions with ADHD, the more you will probably neglect autism’s relation to cultural norms of social behavior, the arbitrary boundaries of clinical significance and the contextual aspects of typical autism impairments.

However, as basic autism research continues to fail to become clinically relevant, and as the extending boundaries of autism and mental ‘abnormality’ gain clinical, societal and economic concern (see Chapter 6), more interaction between relatively autonomous groups (for instance, between autism neuroscientists, clinicians, epidemiologists and health policymakers) will probably take place. At these moments of intercollective communication, the ‘active connections’ are likely to resurface. To quote Fleck again, ‘communication never occurs without a transformation, and indeed always involves a stylized remodeling, which intracollectively achieves corroboration

and which intercollectively yields fundamental alteration' (Fleck, 1979: 111). In the final section which discusses some implications of this study for psychiatry, I will not argue for further intracollective corroboration of current ways of thinking about autism, but for intercollective communication which might start and accelerate the development of new styles of psychiatric thought.

It will be clear that Fleck's epistemology does not contain the idea of universal and eternal truths. There is no neutral position outside history or a social context (a thought collective) from which autism can be identified. However, this does not imply a relativistic view of autism or truth:

Truth is not 'relative' and certainly not 'subjective' in the popular sense of the word. It is always, or almost always, completely determined within a thought style. One can never say that the same thought is true for A and false for B. If A and B belong to the same thought collective, the thought will be either true or false for both. But if they belong to different thought collectives, it will just *not* be *the same* thought! It must either be unclear to, or be understood differently by, one of them. Truth is not a convention, *but rather (1) in historical perspective, an event in the history of thought, (2) in its contemporary context, stylized thought constraint*'. (ibid.: 100)

With Fleck, truth is always relational due to its necessary place in a complex network of linkages that constitutes a thought style. Klaassen (2014: 44) uses the concept of 'relationism' to stress that 'the notion of "truth" can be of value, even if truth is (only) an event taking place in a network that is always in the process of change'. And as truth changes in an advanced scientific field, 'each new fact harmoniously – though ever so slightly – changes all earlier facts. Here every discovery is actually a recreation of the whole world as construed by a thought collective' (Fleck, 1979: 102). Chapters 3, 4 and 5 explored some of the earlier facts about autism, and they explored how these facts changed as new facts about autism involved a rewriting of the history of autism.

Fleck's 'relationist' epistemology turned out to be a fruitful framework for understanding developments in the field of autism. It can account for the observation that, from a historical perspective, autism is notoriously elusive, socially-influenced and variable. In the past seventy years, many facts about autism have been established and discarded, while in its contemporary context,

autism has an undeniable reality. Fleck's notion of thought styles allows autism to be both continuously in flux *and* a seemingly stable – albeit unknown – object with many true representations and consequences.

Thinking beyond autism

The above-mentioned insights into the structure and development of a psychiatric concept lead to a supplementary question. How should psychiatry, as a more or less autonomous, medical specialty dedicated to the care of mental ailments, respond to the developments towards increasingly technical and specialized neurobiological research into psychiatric disease entities? Despite the passionate efforts of thousands of scientists, millions of dollars for fundamental research and confident promises regarding clinical applicability of basic research findings, psychiatric disorders are notoriously hard to get a grip on. Autism research, like psychiatric research in general, persistently fails to translate laboratory findings – for instance from brain imaging experiments or genetic studies – into real-world situations (see Chapter 6).

Of course, it is problematic to speak of psychiatry as a uniform whole. Despite the general dominance of neurobiological explanatory frameworks, clinical psychiatry is rather pluralistic. There are, at least in the Netherlands, many traditions and influences that range from psychoanalysis, social psychiatry, cognitive behavioral therapy, narrative psychiatry, phenomenology, existential psychotherapy, and more. Furthermore, one specific setting or clinical focus draws more from particular traditions than another setting does. For instance, people diagnosed with a personality disorder are likely to be approached from a psychoanalytical framework, while someone diagnosed with an anxiety disorder will probably receive a cognitive-behavioral approach. Mental health professionals who work with children diagnosed with autism also use behavioral techniques (for example, applied behavioral analysis or ABA) and ecological perspectives. More often, clinical practice draws from a mixture of approaches in a way that makes disentangling the different perspectives rather artificial.

Nevertheless, despite my limited focus on autism, I think it is safe to consider the brain sciences as the fundamental sciences in contemporary psychiatry (see Chapter 4). When it comes to what type of research receives the

most funding and what type of research has the most scientific impact, status and authority, the neurosciences head the lists. True explanations of mental ailments somehow have to pass through the brain. In this sense, psychiatry is influenced by and also an important part of the broader 'neurofication' of human life (Rose and Abi-Rached, 2013). Furthermore, and maybe most importantly for psychiatry, the prominence of the brain sciences in psychiatric research ensures psychiatry's medical status and the link with other medical specialties.

Thus, despite a clinical pluralism, it is hard to deny the importance of the brain sciences for the reputation, self-image and legitimacy of psychiatry. But, it is also hard to deny that the medical identity of psychiatry, and the legitimacy of psychiatry in general, is under pressure. As I mentioned in the introduction, more than forty years after Charles Rosenberg's lecture – *The crisis in psychiatric legitimacy: Reflections on psychiatry, medicine, and public policy* – his observations are still valid (Rosenberg, 1975). Rosenberg noticed a gap between the performance of psychiatry as a medical specialty and the expectations from society and psychiatry itself regarding successful psychiatric care, treatments, and (brain-based) explanations of deviant behavior. Psychiatry's objectives to cure and illuminate mental disorders contrasted with the sparse technical means and the lack of insight into the pathophysiological processes of these particular clinical burdens. For Rosenberg, the crisis in psychiatric legitimacy was exactly this gap 'between the demands of medical exclusivity and the inability of psychiatry to provide either understanding or relief consistent with the pretentiousness of such demands' (ibid.: 250).

As Rosenberg cleverly analyzed, it was not psychiatry's ability to cure, prevent, predict and diagnose that justified the legitimacy of psychiatry as a medical specialty. Instead, its legitimacy lied in 'the very gravity and scale of the responsibility it must undertake' (ibid.: 255). Society shaped a particular role and assigned responsibilities to the psychiatrist, namely to treat those who are considered and consider themselves to be mentally ill, consistent with 'the needs of an urban, rationalistic society unwilling to tolerate certain kinds of deviance' (ibid.). A 'medical' psychiatry profited from the status and authority society grants physicians, and it incorporated, authenticated and reinforced social norms and values through a particular medical framework. According to Rosenberg, it was not effectiveness, but orientations, values, and responsibility that were the basis of psychiatry's legitimacy.

Of course, psychiatric practice and research has changed a lot since the early 1970s. To recognize this, one only needs to think of the publication and widespread impact of the ‘neo-Kraepelinian’ third edition of the Diagnostic and Statistical Manual of Mental Disorders (*DSM-III*, APA, 1980); of the development and popularity of new psychotropic drugs such as Prozac and Risperidone in the 1990s; of the rise and emancipatory role of patient movements; of increasing deinstitutionalization and new types of community-based treatment for severe mental illness; and, equally important, of the major developments in neuroimaging, molecular genetics and the cognitive neurosciences in general that profoundly affected the way we think about the nature of mental ailments. However, many of these developments were still shaped by decisions made outside the medical profession of psychiatry. Furthermore, if we are allowed to generalize the aims of autism research, direct links between understandings of pathological mechanisms and successful therapeutic interventions remain among the highest goals for psychiatry. Ideally, insights into pathophysiological processes structure and define the boundaries within which clinical decisions are being made. Currently, this is not the case.

The *DSM-III* (APA, 1980) and the rise and dominance of a ‘biological psychiatry’ can be seen as responses to the crisis described by Rosenberg. They represented the promises and hopes for productive and direct interactions between biomedical research and clinical practice, and consequently, what should justify psychiatry’s relationship with medicine. As this dissertation has demonstrated for autism, these promises and hopes have not yet been fulfilled, and current responses to the ‘crisis of psychiatric legitimacy’ and the disappointments that surround the publication of the barely innovative *DSM-5* (APA, 2013) come from various directions. The most influential response is probably the Research Domain Criteria (RDoC) initiative of the American National Institute of Mental Health (NIMH), which ‘attempts to bring the power of modern research approaches in genetics, neuroscience, and behavioral science to the problems of mental illness, studied independently from the classification systems by which patients are currently grouped’.⁸⁶ In the Netherlands, Borsboom (2013) argues for a network approach to psychopathology in which disorders result from the causal interplay between

⁸⁶ See: <http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml>, accessed April 2015.

symptoms,⁸⁷ while van Os (2014) makes a plea for idiographic (personalized) understanding rather than nomothetic (law-like) explanations of psychiatric disease.⁸⁸

However, even if new systems of classification and concepts of psychiatric disease provide new theoretical insights, better treatments and symptomatic relief for mental ailments, this might not satisfy ‘those activist critics who see in psychiatry an agent of social control’ (Rosenberg, 1975: 256). Even if psychiatry manages to overcome ‘the crisis in psychiatric legitimacy’ by developing the technical means and much closer and more successful connections between biomedical research and clinical practice – i.e., by illuminating pathophysiological pathways and developing successful and specific diagnostic markers and ‘normalizing’ interventions on the basis of these pathophysiological mechanisms – it might still not satisfy the critics who consider psychiatry a repressive (or reductionist) vehicle for social and moral control or a puppet of large commercial interests.

What I have shown using the case of autism is that psychiatry relies, and needs to rely, on particular ideas about the nature of disease and on ideas about what is considered appropriate or inappropriate about our thoughts, feelings and behaviors. Even if psychiatry manages to vindicate its medical status, it cannot escape operating in a particular style. Consequently, as psychiatry deals with sensitive topics such as deviant human behavior, emotions and personalities, it will probably, and hopefully, never escape a critical stance which constantly questions the stylized thoughts regarding the nature of psychiatric disease. In addition, whether psychiatrists like it or not, psychiatry is increasingly drawn into political and ethical discussions about intervening on the threshold between healthy and ‘abnormal’ child development, about the boundaries between mad and bad, and about the desirability of screening and prevention of what we have come to call neurodevelopmental disorders (Morgan, 2015). Politics and society are not external to psychiatry, but inevitably part of it.

⁸⁷ See Ruzzano et al. (2015) for an application of this network approach in autism.

⁸⁸ Neither Borsboom (a psychologist) nor van Os (a psychiatrist) is particularly concerned with the medical status of psychiatry. Nevertheless, they do seem to take the disappointing achievements of psychiatric research (or again, the discrepancy between expectations and performance) as a starting point for their ‘better’ models of psychiatric disease. For my review of van Os’ book, see Verhoeff (2014).

Arguably, the more general lesson that can be learned is that psychiatry should not try to neglect or reject a critical reflection on even its most fundamental assumptions. Unfortunately, as a psychiatrist myself I sometimes experience hostile ‘disciplinary defense reactions’ that try to protect ‘medical’ territories and certain scientific commitments against a contextualizing and historicizing critical mood. It may even be that the zealous quest for a truly medical psychiatry has been an obstacle to the recognition of the inevitable normativity of the foundations of psychiatry. I suggest that it is not the discrepancy between certain expectations of a truly *medical* specialty and performances consistent with these expectations that exposes the weakest spot of contemporary psychiatry, but rather these very expectations themselves and the difficulty mainstream psychiatry has incorporating and accepting a certain type of critique.

If we start to acknowledge that psychiatry is always formed by social and cultural conditions, a critical reflection on these conditions will become accepted by and relevant for psychiatry itself. What are the social, historical, financial and political factors that shape psychiatric knowledge, practices and institutions? And what are the forces that keep particular traditions and seemingly indisputable facts in place? Of course, these types of questions are already addressed by entire departments and a range of historians and sociologists of science and medicine, and this study also tried to contribute to this particular type of study. However, these questions are relatively unimportant in psychiatric science. In practice, they tend to be designated as interesting, but fundamentally irrelevant to the work of biomedical scientists and clinicians in the field of psychiatry. As an alternative, I argue that these questions are fundamental to psychiatry since a study of the contingent conditions of possibility for contemporary psychiatry shows that the practices and operative philosophies in psychiatry are not given but are actually always open to change and debate. Only by understanding this will psychiatry mature its insecure identity as an ailed medical specialty. This is one of the reasons why I suggest that psychiatry should work more closely with the human and social sciences. A more active interaction with fields such as medical anthropology, sociology, medical history and disability studies will give psychiatry a better understanding of and grip on how society shapes psychiatry and vice versa. In this respect, the current partial focus on the brain sciences keeps psychiatry somewhat ignorant and powerless.

A second reason that there should be more ‘intercollective communication’ between psychiatry and the human and social sciences follows more directly from this study. As Chapter 6 argues, the gap between basic autism research and the everyday struggles of those diagnosed with autism seems to be widening. I have argued that current scientific perceptions of autism as a neurodevelopmental disorder drift away from the diversity of problems and experiences of those diagnosed with autism. In addition, the boundaries of autism are unlikely to be drawn by the brain sciences, while there are growing socio-cultural, economic and clinical concerns regarding the still growing ‘autism epidemic’. These worrying developments create opportunities for a more positive – instead of a merely critical – role for the human sciences. They can play a positive role in the development of new styles of psychiatric thought that explicitly take personal, social, cultural and historical context into account. Of course, the brain sciences make reference to social and cultural factors, but they tend to reduce the environment – of cultural meanings, social economical situations, symbols, local practices, traditions, and so forth – to quantifiable inputs that are only interesting in the way they affect the brain (Rose and Abi-Rached, 2013). I suggest that, at least for the field of autism, new styles of thinking about mental ailments should not conceive of the milieu as merely external to an abstract disorder, but as constitutive of the impairments and disruptive behaviors of an individual. This does not imply a neglect of the brain or body, but a recognition of the embeddedness of the human being that constantly shapes and is being shaped by its surrounding world. From this perspective, which is certainly not at odds with certain current developments in the neurosciences, it is by taking the individual in his or her particular situation into account that mental ailments can exist.

Chapter 7 provided something of a first step in the development of such a new style for psychiatry. The work of Kurt Goldstein offers some ‘active connections’ in thinking about disease that can facilitate the development of new types of studies in which ‘the milieu’ becomes indispensable in defining health and disease and in thinking about recovery and intervention. Interactions with fields such as sociology, medical anthropology and medical history can be very productive as these fields have a lot to say about the ways in which particular environments – for example, schools, homes, big cities, and small towns – are inextricably linked to healthy and pathological conditions, instead of seeing environmental factors only as external etiological factors that affect

distinct pathophysiological processes. Such a new style should not lose sight of the vital – or biological – aspects of disturbed mental life that are, nevertheless, always mediated by the specific milieu the person experiences. Obviously, brain or body interventions can still play a significant role in relieving certain ailments. However, they cannot be evaluated separate from the particular circumstances in which they are used.

Furthermore, such a new style might have to let go of the very idea of autism and psychiatric disease entities in general. Paradoxically, the ‘unifying’ disease entity of autism keeps different scientific and academic disciplines rather disconnected. As I have argued in this dissertation, autism neuroscientists investigate autism’s neurobiological basis, while social studies of autism focus on topics regarding autistic cultures, neurodiversity, autistic subjectivity, and so on. Both these fields study autism, but in radically different ways – they develop independently. Psychiatry can take the lead in developing new concepts and ways of thinking about psychiatric disease that enable these different disciplines to work more closely together on shared problems. Goldstein’s person-centered understanding of disease, in which the distinction between ‘the social’ and ‘the biological’ is never absolute, is one option that deserves further scrutiny, particularly in the field of autism.

This alternative understanding of disease and style of psychiatric thought, of which I have only sketched a rudimentary form, will end psychiatry’s dominant focus on the biomedical and brain sciences, which psychiatry currently depends on for its medical identity and status. Instead, it will entail a broader focus that puts the troubles, impairments and adverse experiences, as well as the strengths and resiliencies of the entire human being at center stage. In the spirit of Canguilhem, this does not imply a neglect of ‘the biological,’ but a recognition of the inescapable normativity of life, biology and medicine. Undoubtedly, any new style of psychiatric thought will generate new types of problems and criticisms, but I expect that psychiatric research that manages to take the intricate relationship between human life and the surrounding milieu seriously has a better chance at translating into clinical practice.

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Summary

This dissertation is a collection of seven independent historical and conceptual studies on the concept of autism. I use autism as a central case to gain a specific insight into the development of scientific knowledge in the field of psychiatry. There are several reasons why autism makes an interesting and important case, and why it is important to get a better insight into the structure and development of psychiatric disease concepts. I will briefly mention four important reasons.

Firstly, autism is one of the most diagnosed, researched and discussed psychiatric disorders. Certainly, that has not always been the case. In less than forty years, prevalence rates have increased dramatically from about 1 in 2500 in 1976 to 1 in 68 in 2014. Currently there is no clear explanation for this major increase in autism cases. Next to an increase in autism research, public concerns regarding autism and a wider awareness and recognition of autism have also risen exponentially. Films like *Rain Man* and, for instance, Mark Haddon's bestseller *The Curious Incident of the Dog in the Night-time* have contributed to this popularization of autism. In a relatively short period of time, autism not only became a common psychiatric disorder, but it also became an almost inescapable cultural phenomenon. An analysis of the development of the concept of autism offers new perspectives on this 'autism epidemic'.

Secondly, autism is generally viewed as a 'true' psychiatric disorder. This means that autism is a good example of a serious brain disease with a clear genetic component. Autism researchers and clinicians conceive of autism as a distinct and largely heritable neurodevelopmental disorder. More than with any other psychiatric disorder, expectations are high that neuroscientific research on the biological foundations of autism will become clinically relevant in the near

future. This is why a large part of autism research is directed at unraveling the neurobiological pathophysiology of autism. However, as of yet, there have been no clinically relevant findings or discoveries from autism neuroscience.

This fact brings us to the third reason why autism makes a good case to use to get a better understanding of the development of psychiatric disease concepts. On the one hand, autism is an undeniable reality for patients, families, mental health professionals and autism researchers, and, on the other hand, autism remains a big mystery in terms of its causes, neurobiological foundation, prognosis, course, early signs, prevention and adequate treatment.

In other words, autism's nature remains mysterious. In addition, the 'essential' characteristics of autism – the core symptoms or phenotype – are historically rather variable. Autism has been a disorder of affective contact, a language disorder, a disorder of integrating sensory information, a disorder in understanding the intentions of others, a disorder of executive functions, etcetera. The question that arises is how it is possible that autism can be a clear, recognizable and extensively researched brain disease, while, at the same time, autism is notoriously elusive, heterogeneous and historically variable. And how is it possible that autism is a neurodevelopmental disorder when no clear disturbances have been identified in the development of the 'autistic' brain? These paradoxes are a central theme in this dissertation.

Lastly, autism makes an interesting case because it is not only conceived of as a brain disease. Critics consider the rise of autism diagnoses as a consequence of increasing medicalization and pathologization of diversity. Critics argue that societal changes have resulted in increasing attention to and higher expectations of social skills of children. At school, teamwork, oral presentations and mental flexibility have become more and more important. Furthermore, an official diagnosis is often necessary in order to qualify for all sorts of special services. Thus it seems that there are several social factors involved in the emergence of the 'autism epidemic'. Given this obvious societal component, a study on the concept of autism can provide insight into how autism experts, researchers and clinicians deal with the boundaries of this particular disorder. How do they think about the role of social factors in diagnosing autism and how do they demarcate between, for instance, normal social interaction and pathological social behavior? Again, autism makes a good case for exploring these issues.

All these issues are addressed in this study and in Chapter 2 I focus on the question of the nature – or ontology – of autism from the perspective of autism experts and researchers. This chapter argues that the common belief about the ontological status of autism is that autism constitutes a *natural kind*. There are, however, two major difficulties with a natural kind approach in autism research. First, how can we continue to speak about autism as a distinct disease while the condition is marked by such a sheer diversity of symptoms, traits, biological markers and cognitive profiles? And second, recent historical works on autism illustrate that there is something fundamentally social and historical about how autism is defined, diagnosed and treated. I argue that the dominant natural kind approach in autism research is problematic, as autism can only be understood in relation to ideas about what kind of behavior is deviant and in need of correction or support. Furthermore, locating and maintaining autism within the biological realm of the individual obscures an array of social, cultural and psychological issues involved in understanding the contemporary phenomenon we call autism.

Chapter 3 argues that a new relation between past and present – a supposed historical continuity in the meaning of autism – is created by histories written by the discipline itself. In histories of autism written by ‘practitioner-historians,’ a sense of scientific progress and an essentialist understanding of autism legitimize and reinforce current understandings and research directions in the field of autism. Conceptual discontinuities and earlier complexities and disputes concerning classifying and delineating autism are usually left out of the positivist narrative of autism. In an alternative history of the concept of autism, I demonstrate that there have been major shifts in the type of symptoms, signs and impairments that were – and are – thought to be essential and specific for autism.

Using the conceptual tools of philosopher of science Ludwik Fleck, Chapter 4 argues that the reframing of autism as a neurodevelopmental spectrum disorder is constrained by two governing ‘styles of thought’ of contemporary psychiatry. The first is the historically conditioned ‘readiness for directed perception’ of, and thinking in terms of, ontologically distinct diseases. The clinical gaze of mental health professionals, the bureaucratic needs of health administration, the clinical and scientific utility of disease categories, and the practices of autism-oriented advocacy groups all imply a bias toward thinking about autism and related disorders as ontologically distinct psychiatric and

scientific entities. Second, within the ‘neuromolecular style of thought,’ mental disorders are more and more located at the neurobiological levels of the brain. In autism research, one of the biggest challenges is the identification of autism’s neurobiological singularity. However, at a moment when biological and categorical approaches toward autism face serious empirical difficulties, a balance is established that holds together these two styles of thought. With a need to account for some of the most persistent uncertainties and conflicts in autism research, namely ubiquitous heterogeneity and a failure to identify disease-specific biomarkers, the reframing of autism as a neurodevelopmental spectrum disorder satisfies the scientific, institutional and socio-political needs for stability and homogenization.

Chapter 5 argues that the history and philosophy of autism need to account for at least two different kinds of autism. As is argued in Chapter 4, contemporary autism research and practice is structured, directed and connected by an ‘ontological understanding of disease’. This implies that autism is understood as a disease like any other medical disease, existing independently of its particular manifestations in individual patients. In contrast, autism in the 1950s and 1960s was structured by a psychoanalytical framework and an ‘individual understanding of disease’. In contrast with the ontological understanding of disease, autism was not a distinct disease but an idiosyncratic and meaningful response of the child to a disturbed development of the ego. These two kinds of autism are embedded in and reveal two very different ‘styles of psychiatric thought’.

In Chapter 6 I argue that the persistent search for autism-specific pathophysiology has two fundamental difficulties. The first involves the growing gap between basic autism science and clinical practice. The second involves the difficulties researchers face with demarcating autism as a psychiatric condition. Instead of the unremitting search for the neurobiological basis of autism, I suggest that basic autism research should focus on experiences of impairment and distress, and on how these experiences relate to particular (autistic) behaviors in particular circumstances, regardless of whether we are dealing with an autism diagnosis or not.

Chapter 7 is an exploration of alternative disease concepts. One conceivable alternative framework for understanding disease and individuals we have come to call autistic can be found in the work of neurologist Kurt Goldstein (1878–1965). His person-centered approach provides radically new ways to investigate

and intervene with the behavior we are accustomed to explain as being caused by the elusive entity called autism.

In the conclusion in Chapter 8 I illustrate that Fleck's notion of thought styles makes it possible for autism to be both continuously in flux *and* a seemingly stable – albeit unknown – object with many true representations and consequences. Numerous factors including a particular medical tradition, the medically-educated and DSM-trained clinical gaze of psychiatrists and other mental health professionals, the clinical and scientific utility of disease categories, the bureaucratic needs of health administration, and the practices of autism-oriented advocacy groups all create a dense social, material and cognitive network in which autism achieves a seemingly inevitable stability. Autism has become a form to be directly perceived. This '*readiness for stylized (that is, directed and restricted) perception*' of autism makes it possible for scientists and clinicians to continue to perceive a variable mixture of seemingly independent signs and symptoms as expressions of a specific, identifiable disease. This 'readiness' explains the strong tendency to objectify and reify autism. From this Fleckian perspective, the persistent search for autism's neurobiological basis can be regarded as an intelligible consequence of the restricted and directed way of perceiving, thinking and acting in contemporary psychiatry.

Finally, I argue that the broad field of psychiatry will always depend on certain contingent ideas about the nature and boundaries of mental ailments. I suggest that a critical, reflective attitude towards these ideas (and the circumstances under which they emerge) should be an integral part of psychiatry. A psychiatry that is mainly focused on legitimizing its own *medical* identity does not leave much room for a contextualizing and historicizing critical attitude. However, if we start to acknowledge that psychiatry is always formed by social and cultural conditions, a critical reflection on these conditions will become accepted by and relevant for psychiatry itself. Psychiatry will be less inclined to reject a critical view of some of the fundamental assumptions in psychiatry. This is one of the reasons why I suggest that psychiatry should work more closely with the human and social sciences. A more active interaction with fields such as medical anthropology, sociology, medical history and disability studies will give psychiatry a better understanding of and grip on how society shapes psychiatry and vice versa. In this respect, the current partial focus on the brain sciences keeps psychiatry somewhat ignorant and powerless.

A second reason that there should be more ‘intercollective communication’ between psychiatry and the human and social sciences follows more directly from this study. As Chapter 6 argues, the gap between basic autism research and the everyday struggles of those diagnosed with autism seems to be widening. I have argued that current scientific perceptions of autism as a neurodevelopmental disorder drift away from the diversity of problems and experiences of those diagnosed with autism. In addition, the boundaries of autism are unlikely to be drawn by the brain sciences, while there are growing socio-cultural, economic and clinical concerns regarding the still growing ‘autism epidemic’. These worrying developments create opportunities for a more positive – instead of a merely critical – role for the human sciences. They can play a positive role in the development of new styles of psychiatric thought that explicitly take personal, social, cultural and historical context into account. I suggest that, at least for the field of autism, new styles of thinking about mental ailments should not conceive of the milieu as merely external to an abstract disorder, but as constitutive of the impairments and disruptive behaviors of an individual. This does not imply a neglect of the brain or body, but a recognition of the embeddedness of the human being that constantly shapes and is being shaped by its surrounding world. From this perspective, which is certainly not at odds with certain current developments in the neurosciences, it is by taking the individual in his or her particular situation into account that mental ailments can exist.

The human and social sciences can play an important and positive role in developing new ways of thinking about mental ailments that, for instance, put the troubles, impairments and adverse experiences, as well as the strengths and resiliencies of the entire human being at center stage. Undoubtedly, any new style of psychiatric thought will generate new types of problems and criticisms, but I expect that psychiatric research that manages to take the intricate relationship between human life and the surrounding milieu seriously has a better chance at translating into clinical practice.

Samenvatting

Dit proefschrift is een verzameling van zeven onafhankelijke historische en conceptuele studies naar het begrip autisme. In al deze studies gebruik ik autisme als centrale casus om inzicht te krijgen in de ontwikkeling van wetenschappelijke kennis in de psychiatrie. Er zijn verschillende redenen waarom autisme een interessante en belangrijke casus is, en waarom het van belang is om meer inzicht te krijgen in de structuur en ontwikkeling van psychiatrische ziekteconcepten. Ik zal kort de vier belangrijkste redenen noemen.

Ten eerste is autisme één van de meest gediagnosticeerde, onderzochte, en besproken psychiatrische stoornissen. Dat is zeker niet altijd het geval geweest. De prevalentiecijfers van autisme zijn in minder dan veertig jaar omhoog geschoten van ongeveer 1 op de 2500 in 1976 naar 1 op de 68 in 2014. Er is tot op heden geen duidelijke verklaring voor deze gigantische toename. Naast een enorme toename van autisme onderzoek zijn ook de maatschappelijk zorgen over autisme en een brede bekendheid met autisme de afgelopen decennia exponentieel toegenomen. Films zoals *Rain Man* en bijvoorbeeld Mark Haddon's bestseller *The Curious Incident of the Dog in the Night-time* hebben hieraan bijgedragen. Autisme is in een korte tijd niet alleen een veelvoorkomende psychiatrische aandoening geworden, maar ook een bijna onontkoombaar cultureel verschijnsel. Een onderzoek van de ontwikkeling van het autismebegrip biedt een nieuw perspectief op deze 'autisme epidemie'.

Ten tweede wordt autisme gezien als een 'echte' psychiatrische aandoening. Hiermee wordt tegenwoordig bedoeld dat autisme een goed voorbeeld is van een hersenziekte met een overduidelijke genetische component. Autism onderzoekers en behandelaren zien autisme over het algemeen als een duidelijk

te onderscheiden en grotendeels erfelijke hersenontwikkelingsstoornis. Nog meer dan bij andere psychiatrische aandoeningen zijn er hoge verwachtingen dat neurowetenschappelijk onderzoek naar het biologische substraat van autisme in de nabije toekomst klinisch relevant wordt. Een groot deel van het autisme onderzoek is dan ook gericht op het ontrafelen van de neurobiologische pathofysiologie van autisme. Echter, tot op heden zijn er geen klinisch bruikbare bevindingen vanuit neurowetenschappelijk onderzoek naar autisme.

Dit brengt ons bij de derde reden waarom autisme een goede casus is om de ontwikkeling van psychiatrische ziekteconcepten beter te begrijpen. Autisme is enerzijds een onmiskenbare werkelijkheid voor patiënten, families, behandelaren en onderzoekers, en anderzijds een groot mysterie als het gaat om de oorzaken, het neurobiologisch substraat, de prognose, het beloop, preventie mogelijkheden en werkzame behandelingen. Met andere woorden, de precieze aard van autisme blijft een groot raadsel. Bovendien zijn de ‘essentiële’ kenmerken van autisme – de kernsymptomen – historisch weinig stabiel. Autisme was ooit een stoornis in het maken van affectief contact, een taalstoornis, een stoornis in het verwerken van zintuiglijke informatie, een stoornis in het begrijpen van de intenties van een ander, een stoornis in het plannen van taken, enzovoorts. De vraag die rijst is hoe het kan dat autisme een duidelijk herkenbare en veel onderzochte hersenziekte is, en tegelijkertijd notoir ongrijpbaar, heterogeen en historisch variabel is. En hoe kan het dat we kunnen spreken van een hersenontwikkelingsstoornis, terwijl er nog geen eenduidige stoornissen in de ontwikkeling van de hersenen zijn aangetoond? Deze schijnbare tegenstellingen vormen een centraal thema in dit proefschrift.

Tot slot is autisme een interessante casus omdat het niet slechts als een hersenziekte wordt benaderd. Critici menen dat de toename van autisme diagnoses een gevolg is van een groeiende medicalisering en pathologisering van diversiteit. Maatschappelijke veranderingen zorgen volgens critici voor toegenomen verwachtingen van de sociale vaardigheden van kinderen. Op school zijn teamwork, presentaties en mentale flexibiliteit steeds belangrijker geworden. Ook is het krijgen van een diagnose van belang om aanspraak te kunnen maken op allerlei ondersteunende voorzieningen. Er lijken dus verschillende sociale factoren een rol te spelen bij het ontstaan van de ‘autisme epidemie’. Gegeven deze sociale component geeft een studie naar het autismebegrip een belangrijk inzicht in hoe autisme experts, onderzoekers en

behandelaren nadenken over de grenzen van psychiatrische ziektes. Hoe denken zij over de rol van sociale factoren in het ontstaan van autisme en hoe bepalen zij de grens tussen bijvoorbeeld normale sociale interactie en pathologisch sociaal gedrag?

Al deze aspecten komen in deze studie aan bod en in Hoofdstuk 2 richt ik mij hoofdzakelijk op de vraag wat autisme is vanuit het perspectief van autisme experts en onderzoekers. Dit hoofdstuk beargumenteert dat de algemene opvatting over de ontologische status van autisme is dat het een natuurlijke soort is. Ik bespreek twee problemen van deze natuurlijke soort benadering in autisme onderzoek. Het eerste probleem is de enorme diversiteit aan symptomen, kenmerken, biologische markers and cognitieve afwijkingen die gevonden worden bij mensen die met autisme zijn gediagnosticeerd. Het tweede probleem is dat recente historische en sociologische studies laten zien dat er fundamenteel historische en sociale aspecten zitten aan de manier waarop autisme gediagnosticeerd, gedefinieerd en behandeld wordt. Ik beargumenteer dat de natuurlijke soort benadering van autisme problematisch is aangezien autisme alleen begrepen kan worden in relatie tot variabele ideeën over wat afwijkend gedrag is. Het lokaliseren van autisme in het brein van een individu houdt een scala aan sociale, culturele en psychologische factoren buiten beeld. Deze factoren zijn echter wel van belang voor een completer begrip van wat we tegenwoordig verstaan onder autisme.

Hoofdstuk 3 beargumenteert dat er een nieuwe relatie tussen het heden en het verleden – een vermeende historische continuïteit in de betekenis van autisme – wordt gecreëerd door de geschiedenissen van autisme die worden geschreven door de discipline zelf. In deze geschiedenissen wordt er een duidelijke vooruitgang van kennis over autisme verondersteld en een essentialistisch begrip van autisme functioneert als een legitimatie van hedendaags autisme onderzoek. Complexe discussies over het classificeren en begrenzen van autisme worden weggelaten in dit type geschiedschrijving. In een alternatieve geschiedenis van het autismebegrip laat ik zien dat er grote veranderingen zijn geweest in de manier waarop autisme werd geconceptualiseerd. Wat we onder de kernsymptomen van autisme verstaan is veel minder stabiel dan wat veel autisme onderzoekers ons doen geloven.

Met hulp van enkele begrippen van de wetenschapsfilosoof Ludwik Fleck, stelt Hoofdstuk 4 dat het herinterpreteren van autisme als een *neurodevelopmental spectrum disorder* te begrijpen is vanuit een combinatie van twee belangrijke

‘denkstijlen’ van de hedendaagse psychiatrie. De eerste is de historisch geconditioneerde ‘gereedheid tot gerichte perceptie’ van, en denken in termen van, ontologisch aparte ziektes. De klinische blik van behandelaren in de geestelijke gezondheidszorg, de manier waarop de gezondheidszorgadministratie georganiseerd is, de klinische en wetenschappelijke bruikbaarheid van aparte ziektecategorieën, en de praktijken van op autisme georiënteerde patiënten- en belangenverenigingen impliceren het idee van autisme als een ontologisch afzonderlijke ziekte-eenheid. De tweede ‘neuromoleculaire denkstijl’ houdt in dat psychiatrische problemen in toenemende mate worden gelokaliseerd op een neurobiologisch niveau in het brein. Één van de grootste uitdagingen van hedendaags autisme onderzoek is het identificeren van de specifieke neurobiologie van autisme. Echter, op het moment dat er ernstige empirische moeilijkheden ontstaan bij de biologische en categoriale benaderingen van autisme, is het hervormen van het autismebegrip noodzakelijk om deze twee denkstijlen bij elkaar en in stand te houden. Het hervormen van autisme tot een *neurodevelopmental spectrum disorder* is een manier om rekenschap te geven van de persisterende onzekerheden in autisme onderzoek, namelijk heterogeniteit op alle niveaus van genotype tot fenotype en de mislukte pogingen om autisme-specifieke *biomarkers* te identificeren. Het idee van een *neurodevelopmental spectrum disorder* voorziet in de neiging tot stabiliteit van de heersende denkstijlen en de verschillende wetenschappelijke, institutionele en sociaal-politieke groepen.

Hoofdstuk 5 beargumenteert dat de geschiedenis en filosofie van autisme rekening moeten houden met op zijn minst twee totaal verschillende soorten autisme. Zoals Hoofdstuk 4 ook al aangeeft wordt hedendaags autisme onderzoek gestructureerd en gestuurd door een ‘ontologisch begrip van ziekte’. Dit impliceert dat we autisme begrijpen als een medische ziekte met een bestaan dat onafhankelijk is van de specifieke manifestaties in individuele patiënten. Daarentegen, in de jaren vijftig en zestig van de vorige eeuw werd autisme vormgegeven binnen een psychoanalytisch kader en een ‘individueel begrip van ziekte’. Dit impliceerde dat autisme niet werd begrepen als een aparte ziekte, maar als een idiosyncratische en betekenisvolle reactie van het kind op een verstoorde ontwikkeling van het ego. Deze twee soorten autisme zijn ingebed in en onthullen twee compleet verschillende psychiatrische denkstijlen.

In Hoofdstuk 6 beargumenteer ik dat de aanhoudende zoektocht naar autisme-specifieke pathofysiologie twee fundamentele problemen kent. Het

eerste probleem is de groeiende kloof tussen fundamenteel autisme onderzoek en de klinische praktijk. Het tweede probleem is het onvermogen om autisme te begrenzen als een psychiatrische of pathologische aandoening. In plaats van een aanhoudende zoektocht naar de neurobiologische basis van autisme, stel ik voor dat fundamenteel autisme onderzoek zich moet richten op ervaringen van lijden en beperkingen, en op hoe deze ervaringen gerelateerd zijn aan bepaalde 'autistische' gedragingen in specifieke omstandigheden, onafhankelijk van een officiële autisme diagnose.

Hoofdstuk 7 is een verkenning van alternatieve ziekteconcepten. Een aantrekkelijk alternatief om ziekte en autisme te begrijpen haal ik uit het werk van de neuroloog Kurt Goldstein (1878-1965). Zijn persoonsgerichte benadering van ziekte geeft radicaal nieuwe manieren om autisme te onderzoeken en te behandelen, zonder gebruik te maken van het abstracte en ongrijpbare autismebegrip.

In de conclusie in Hoofdstuk 8 laat ik zien dat Flecks notie van denkstijlen het mogelijk maakt dat autisme zowel continu aan verandering onderhevig is *en* een ogenschijnlijk stabiele – maar grotendeels onbekende – entiteit is met vele ware representaties en werkelijke consequenties. In een hecht netwerk van verschillende sociale, materiële en cognitieve verbindingen krijgt autisme een haast onvermijdelijke stabiliteit. Autismes is een vorm geworden die direct kan worden waargenomen. Dit verklaart de sterke neiging tot het objectiveren en reïficeren van autisme. Vanuit dit Fleckiaanse perspectief is de aanhoudende zoektocht naar de neurobiologische basis van autisme een logisch gevolg van de begrensde en gerichte manier van waarnemen, denken en handelen in de huidige psychiatrie.

Tot slot beargumenteer ik dat de psychiatrie altijd afhankelijk zal blijven van bepaalde contingente ideeën over de aard en grenzen van psychiatrische aandoeningen. Dit betekent dat een kritische reflectieve houding ten opzichte van die ideeën (en de omstandigheden waaronder ze mogelijk worden) een integraal onderdeel van de psychiatrie zou moeten zijn. Een psychiatrie die hoofdzakelijk gericht is op het legitimeren van zijn eigen *medische* identiteit laat weinig ruimte voor een contextualiserende en historiserende kritische houding. Echter, wanneer we inzien dat sociale en culturele omstandigheden de psychiatrie altijd zullen vormgeven, dan wordt een kritische houding ten aanzien van die omstandigheden uiterst relevant voor de psychiatrie zelf. De psychiatrie zal dan ook minder geneigd zijn om een kritische blik op

fundamentele aannames buiten de psychiatrie te houden. Dit is één van de redenen waarom de psychiatrie meer zou moeten samenwerken met disciplines zoals medische antropologie, sociologie en medische geschiedenis. De eenzijdige focus op de neurowetenschappen laat de psychiatrie zonder grip op de manier waarop de maatschappij en de psychiatrie elkaar wederzijds beïnvloeden.

Een tweede reden voor een actievere samenwerking met de menswetenschappen en de sociale wetenschappen volgt direct uit de problemen die ik in Hoofdstuk 6 aankaart. De groeiende kloof tussen fundamenteel autisme onderzoek en de dagelijkse frustraties en beperkingen van patiënten met een autisme diagnose, en de groeiende sociaal-culturele, financiële en klinische zorgen over de almaar toenemende ‘autisme epidemie,’ vragen om de ontwikkeling van nieuwe denkstijlen in de psychiatrie. Ik stel voor dat een nieuwe manier om over psychiatrische aandoeningen na te denken – en over autisme in het bijzonder – expliciet rekening houdt met de directe persoonlijke, sociale en culturele context waarin het psychiatrische probleem ontstaat. De omgeving is dan niet extern aan een abstracte stoornis, maar onlosmakelijk verbonden met de beperkingen en gedragingen van het individu. Vanuit dit perspectief, waar ik in Hoofdstuk 7 uitgebreid op in ga, kan een psychiatrisch probleem alleen bestaan wanneer er rekening wordt gehouden met het individu in zijn of haar specifieke situatie. De menswetenschappen en de sociale wetenschappen kunnen een belangrijke en positieve bijdrage leveren aan het verder ontwikkelen van een psychiatrie die zich primair richt op de beperkingen, kwetsbaarheden en pijnlijke ervaringen van de mens als geheel. Natuurlijk zal een nieuwe denkstijl ook nieuwe problemen en kritische geluiden met zich meebrengen, maar ik vermoed dat psychiatrisch onderzoek dat serieus rekening houdt met de complexe relatie tussen de mens en zijn omgeving meer kans maakt om zich te vertalen naar de klinische praktijk.

