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Autism's anatomy

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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’.

⁵⁶ This chapter has been published as Verhoeff B (2015) Two kinds of autism: a comparison of distinct understandings of psychiatric disease. *Medicine, Health Care and Philosophy* DOI 10.1007/s11019-015-9655-4.

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 pathophysiologicals, 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, Ereshfsky (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.

References

- Anthony EJ (1958) An aetiological approach to the psychopathology of childhood: autism. *British Journal of Medical Psychology* 31: 211-227.
- APA (1952) *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.
- APA (1980) *Diagnostic and statistical manual of mental disorders, 3rd edition*. Washington, DC: American Psychiatric Association.
- APA (1994) *Diagnostic and statistical manual of mental disorders, 4th edition*. Washington, DC: American Psychiatric Association.
- APA (2013) *Diagnostic and statistical manual of mental disorders, 5th edition*. Washington, DC: American Psychiatric Association.
- Baron-Cohen S (2000) Is Asperger syndrome/high-functioning autism necessarily a disability? *Development and Psychopathology* 12: 489-500.
- Bettelheim B (1967) *The Empty Fortress: Infantile Autism and the Birth of the Self*. New York, NY: Free Press.
- Bleuler E (1911/1950) *Dementia Praecox or the Group of Schizophrenias*. New York: International Universities.
- Boorse C (1975) On the Distinction Between Disease and Illness. *Philosophy and Public Affairs* 5: 49-68.
- Bracken P et al. (2012) Psychiatry beyond the current paradigm. *The British Journal of Psychiatry* 201: 430-434.
- Canguilhem G (1966/1991) *The normal and the pathological*. New York: Zone Books.
- Cohen H (1955) The Evolution of the Concept of Disease. *Proceedings of the Royal Society of Medicine* 48(155): 1-6.
- Cuthbert B and Insel TR (2010) The Data of Diagnosis: New Approaches to Psychiatric Classification. *Psychiatry: Interpersonal and Biological Processes* 73(4): 311-314.

- Dawson G (2008) Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology* 20(3): 775-803.
- Decker HS (2013) *The making of DSM-III; a diagnostic manual's conquest of American psychiatry*. Oxford: Oxford University Press.
- Eisenberg L and Kanner L (1956) Early infantile autism, 1943–55. *American Journal of Orthopsychiatry* 26: 556–566.
- Ereshfsky M (2009) Defining “health” and “disease”. *Studies in the History and Philosophy of Biology and Biomedical Sciences* 40: 221–227.
- Evans B (2013) How autism became autism: The radical transformation of a central concept of child development in Britain. *History of the Human Sciences* 26(3): 3-31.
- Eyal G, Hart B, Onculer E, Oren N and Rossi N (2010) *The Autism Matrix*. Cambridge: Polity Press.
- Fleck L (1935/1979) *Genesis and development of a scientific fact*. Chicago, IL: University of Chicago Press.
- Frances AJ (2013) *Saving Normal: An Insider's Revolt Against Out-of-Control Psychiatric Diagnosis, DSM-5, Big Pharma, and the Medicalization of Ordinary Life*. New York: William Morrow.
- Freud S (1905/2001) Three Essays on the Theory of Sexuality, in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, vol. 7, trans. J. Strachey. London: Vintage.
- Freud S (1950) Extracts from the Fleiss papers. *Standard Edition* 1: 173–280.
- Gould J and Ashton-Smith J (2011) Missed diagnosis or misdiagnosis? Girls and women on the autism spectrum. *Good Autism Practice* 12: 34-41.
- Grob GN (1991) Origins of DSM-I: A Study in Appearance and Reality. *American Journal of Psychiatry* 148: 421-431.
- Grünbaum A (1984) *The Foundations of Psychoanalysis*. Berkeley: University of California Press.
- Happé F and Frith U (2014) Annual research review: Towards a developmental neuroscience of atypical social cognition. *Journal of Child Psychology and Psychiatry* 55(6): 553-577.
- Hermelin B and O'Conner N (1963) The response and self-generated behavior of severely disturbed children and severely subnormal controls. *British Journal of Social and Clinical Psychology* 2: 37-43.

- Insel TR et al. (2010) Research Domain Criteria (RDoC): Toward a New Classification Framework for Research on Mental Disorders. *American Journal of Psychiatry* 167(7): 748-751.
- Jaarsma P and Welin S (2012) Autism as a natural human variation: Reflections on the claims of the neurodiversity movement. *Health Care Analysis* 20: 20-30.
- Kanner L (1943) Autistic disturbances of affective contact. *Nervous Child* 2: 217-250.
- Kapp SK, Gillespie-Lynch K, Sherman LE and Hutman T (2012) Deficit, difference, or both? Autism or neurodiversity. *Developmental Psychology* 49: 59-71.
- Kaufman I, Rosenblum E, Heims L and Willer L (1957) Childhood psychosis: 1. Childhood schizophrenia: Treatment of children and parents. *American Journal of Orthopsychiatry* 27: 683-690.
- Kim YS et al. (2011) Prevalence of Autism Spectrum Disorders in a Total Population Sample. *American Journal of Psychiatry* 168: 904-912.
- Lidz T (1966) Adolf Meyer and the development of American Psychiatry. *American Journal of Psychiatry* 123(3): 320-332.
- Lotter V (1966) Epidemiology of autistic conditions in young children. *Social Psychiatry* 1: 124-137.
- Mahler M (1952) On child psychosis and schizophrenia: autistic and symbiotic infantile psychosis. *The Psychoanalytic Study of the Child* 7: 286-305.
- Matson JL and Nebel-Schwalm MS (2007) Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities* 28(4): 341-352.
- Meyer A (1908) The problems of mental reaction-types, mental causes and diseases. *The Psychological Bulletin* 5(8): 245-261.
- Micale MS (2014) The ten most important changes in psychiatry since World War II. *History of Psychiatry* 25: 485-491.
- Miller G (2010) Is pharma running out of brainy ideas? *Science* 329: 502-504.
- Nadesan MH (2005) *Constructing Autism: Unravelling the 'Truth' and Understanding the Social*. New York: Routledge.
- Ortega F (2009) The cerebral subject and the challenge of neurodiversity. *BioSocieties* 4(4): 425-445.
- Osbourne T (1998) Medicine and ideology. *Economy and Society* 27: 259-273.

- Pellicano E, Dinsmore A and Charman T (2014) What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism* 18: 756-770.
- Raz M (2014) Deprived of touch: How maternal and sensory deprivation theory converged in shaping early debates over autism. *History of the Human Sciences* 27(2): 75-96.
- Rose N and Abi-Rached JM (2013) *Neuro: The new brain sciences and the management of the mind*. Princeton, NJ: Princeton University Press.
- Rosenberg CE (1975) The crisis in psychiatric legitimacy: Reflections on psychiatry, medicine, and public policy. In Kriegman G, Gardner RD and Abse DW (eds) *American psychiatry, past, present, and future: Papers presented on the occasion of the 200th anniversary of the establishment of the first state-supported mental hospital in America*. Charlottesville: University Press of Virginia, 135-148.
- Rosenberg CE (2002) The tyranny of diagnosis: Specific entities and individual experience. *The Milbank Quarterly* 80(2): 237-260.
- Rosenberg CE (2003) What is disease? in memory of Owsei Temkin. *Bulletin of the History of Medicine* 77(3): 491-505.
- Rutter M (1968) Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* 9(1): 1-25.
- Sadler JZ (2011) Psychiatric molecular genetics and the ethics of social promises. *Journal of Bioethical Inquiry* 8(1): 27-34.
- Silverman C (2012) *Understanding autism: Parents, doctors, and the history of a disorder*. Princeton, NJ: Princeton University Press.
- South M, Ozonoff S and McMahon WM (2007) The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism* 11(5): 437-451.
- Temkin O (1977) *The double face of Janus and other essays in the history of medicine*. Baltimore, MD: John Hopkins University Press.
- Tustin F (1969) Autistic processes. *Journal of Child Psychotherapy* 2: 23-42.
- Verhoeff B (2013) Autism in flux: a conceptual history from Leo Kanner to DSM-5. *History of Psychiatry* 24(4): 442-458.
- 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.
- Waterhouse L (2013) *Rethinking Autism: Variation and Complexity*. Amsterdam: Academic Press (Elsevier), 399-443.

- Wing L and Gould J (1979) Severe impairments of social interaction and associated abnormalities in children: epidemiology and classification. *Journal of Autism and Developmental Disorders* 9(1): 11–29.
- Yirmiya N and Charman T (2010) The prodrome of autism: early behavioral and biological signs, regression, peri- and post-natal development and genetics. *Journal of Child Psychology and Psychiatry* 51(4): 432-458.
- Young A (1997) *The Harmony of Illusions: Inventing Post-Traumatic Stress Disorder*. Princeton, NJ: Princeton University Press.

