Chapter 20
The Evolutionary Etiologies of Autism Spectrum and Psychotic Affective Spectrum Disorders

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Lay Summary The mental and behavioural traits that have evolved in humans, such as complex sociality, make us vulnerable to corresponding mental disorders, such as disorders that involve too little, or too much, social thinking. Autism can be considered as a disorder where complex sociality does not develop, while schizophrenia, bipolar disorder, and depression can be considered as the opposite: pathologically overdeveloped social thought and behaviour, as seen, for example, in paranoia and hearing voices. Evolutionary biology is fundamentally important in understanding, defining, and treating mental disorders because it helps us to determine what the brain has evolved to do, which informs us about the different ways that brain functions can become dysregulated in disease.

20.1 Introduction

20.1.1 The Standard Medical Model and the Reification of Psychiatric Disorders

The standard medical model for understanding and treating disease focuses on determining its proximate physiological and developmental causes, in terms of how functional systems have become dysregulated [1]. High blood glucose levels, for example, may be due to type 1 diabetes, which results from specific, well-characterized physiological and molecular biological causes and, as a result, can be

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unambiguously diagnosed. Understanding the normal functioning of blood glucose regulation, or any other physiological system, thus represents a key precondition for determining aetiology and effective treatments.

How can the standard medical model be applied to psychiatric disorders? The medical model assumes that illness can be objectively and unequivocally quantified. By contrast, the causes and patterns of brain functions that underlie psychiatric disorders are only dimly understood. Psychiatric disorders are, instead, abstract, heuristic, descriptive constructs that are more or less useful for guiding research, diagnoses, and treatments. The clearest evidence for such artificiality is the Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria for diagnosing psychiatric disorders, which comprise detailed lists of symptoms, some set of which are considered necessary and sufficient to infer the presence of disease.

Despite these considerations, it is commonplace for psychiatric conditions to be reified—that is—considered as real, for research, medical, and societal purposes [2]. Such pragmatic reification can be considered as innocuous, but it is not: it constrains and biases how researchers think about mental disorders and their associated research agendas and leads to misconceptions of psychiatric disorders as objectively defined, purely pathological 'diseases' that people 'have' comparable in some fundamental way to diseases like diabetes, cancer, or atherosclerosis that can be objectively and physiologically quantified in terms of their causes and effects.

Under current paradigms, determining the 'causes' of mental disorders often becomes conflated with characterizing mental pathologies or deficits, at levels from genes, to neurodevelopment and function, to cognitive functions, and to deleterious environments. By contrast, according to the standard medical model, mental disorders should instead be conceptualized and analysed, in terms of what functional mental systems have become dysregulated and what forms such dysregulations take. In this regard, for example, to better understand autism, we must also better understand the development of neurotypical social cognition, and to understand bipolar disorder and depression, we must also understand the adaptive functions of normal, contextual variation in mood.

20.1.2 The Evolution of Mental Adaptations

Adaptive functions of the human mind and brain, like those of glucose regulation, have, of course, evolved. Most generally, this meaning of 'adaptive' means that such systems show, and have for many, many past generations shown, genetically based variation among individuals that has influenced survival and reproduction. Such variation has thus been subject to natural selection, which leads, across generations, to increases in, or maintenance of, the adaptive 'fit' or 'match' between organismal phenotypes and aspects of their environments. For example, the beaks of Darwin's finches are 'fit' in their sizes and shapes for different food sources. Similarly, specific regions of the human neocortex adaptively function to recognize individual faces (the fusiform gyrus), or to infer the thoughts and intentions of other

humans (the medial prefrontal cortex). Specific mental adaptations, like the insulin pathway, are real and quantifiable and the subject of intense interest in disciplines such as cognitive neuroscience.

Natural selection of human physiology and morphology is expected, under basic evolutionary considerations, to have led to the maximization of functional robustness, homoeostatic ability, and efficiency, as well as optimal flexibility under variable circumstances, all in the service of survival and reproduction. But what, then, is natural selection—the driver of adaptation—expected to maximize with regard to human cognition, emotion, and behaviour? We usually think of mental disorders as centrally involving unhappiness of the subject as well as their social circle, which motivates the seeking of help from the medical community. However, natural selection is by no means expected to maximize happiness, simply because increased happiness is by no means a primary means or route to increased survival and reproduction [3]. Instead, natural selection is predicted, by basic theory, to maximize condition-dependent human striving for the goals that have led, across many past generations in relevant environments, to high survival and reproduction, relative to other humans.

In the context of striving, human emotional systems have evolved to motivate and modulate goal-seeking, dynamically across different circumstances. Such motivation is mediated by the human 'liking' and 'wanting' reward systems, as well as by unhappiness or dissatisfaction with current situations. Human cognitive systems, by contrast, represent sets of evolved mechanisms for information processing, causal thinking, and decision-making that subserve identification of appropriate goals and tactics for reaching them. Both emotional and cognitive systems develop across infancy, childhood, and adolescence, whereby genes, environments, and gene-by-environment interactions mediate neurodevelopment. To understand human psychiatric disorders from an evolutionary perspective, it thus becomes necessary to connect these psychological trajectories and adaptations with their corresponding maladaptations (lacks of fit of phenotypes to the environment), expressed as developmental, emotional, and cognitive dysfunctions that revolve around human striving and cognition. What adaptations, then, are dysregulated in major human mental disorders and how?

Evolutionary biology is useful in medicine for two main reasons: (1) it teaches us how to think about human medically relevant phenotypes and diagnoses, in novel, productive ways, and (2) it indicates specific new data to collect and new approaches for therapies. In this chapter, I focus on the evolutionary biology of psychiatric disorders centrally involving social cognition, affect, and development. I first describe the primary types of causes of mental disorders, from evolutionary medical thinking. Next, I describe autism spectrum disorders and psychotic affective spectrum disorders, in the context of these causes, with reference to recent findings in genetics, neuroscience, and psychology, and in the context of which human-evolved adaptations have been subject to what forms of alteration in each case. Third, I describe and evaluate hypotheses for the relationships of these disorders with one another—relationships that define evolved axes of human

development, affect, and cognition that structure variation in adaptive and maladaptive human mental functioning. Finally, I make specific suggestions for research and clinical therapies that follow directly from these considerations.

20.2 Research Findings

20.2.1 Evolutionary Causes of Mental Disorders

The evolutionary causes of psychiatric disorders represent the 'ultimate' sources of these conditions, which indicate why, given their evolutionary history, humans exhibit particular forms of mental disorders with particular symptoms and severities. Each of the six main causes described below centres on explanations for deviations from mental adaptation and health, in the context of how maladaptations can arise, and be maintained, in populations.

20.2.1.1 Deleterious Alleles

Mutations generate novel alleles that usually cause reduced genetic function, because the perturbations randomly alter a system that would otherwise develop reasonably well. Highly penetrant mutations, with large effects, are especially likely to be highly deleterious, and considerable evidence attests to important roles for de novo, deleterious mutations, such as copy number variants or changes to highly conserve amino acid residues, in the causes of mental illness (e.g. [4]). Highly deleterious alleles that are associated with relatively severe mental illnesses include monogenic causes of autism or schizophrenia that evolve under mutation–selection balance: rare mutations arise and are selected against because their bearers exhibit greatly reduced reproduction.

Rare, deleterious alleles such as copy number variants have been estimated to account for a small percentage of cases of major mental illness [5]. Most inferred 'risk alleles' for mental disorders, such as those identified with genomewide association studies are, however, relatively common (at frequencies above 1 % or 5 %) and have small effects on risk through one dimension of their multifaceted impacts on neurodevelopment, neuronal function, and other systems. The degree to which such alleles can be considered as deleterious to health overall—given all of their effects—remains an open question; for example, neurodegenerative disease risk trades off with cancer risk, such that higher risks in one domain of disease may commonly entail lower risks in another [6]. Presumably, if psychiatric risk alleles were purely deleterious, they would indeed not be common in populations. Risk alleles may also exhibit positive effects, on health and reproduction, when expressed in genetic relatives of individuals with mental illness [7]; these findings indicate that 'risk' alleles do not simply confer increased risk of disease, but may,

depending on the context, confer benefits as well. Such considerations can help to explain the high heritabilities of psychiatric conditions including autism, bipolar disorder, and schizophrenia, on the order of 50–80 % (e.g. [8]).

20.2.1.2 Mismatched Environments

Populations and individuals are always adapted to past environments, and if environments change more rapidly than they can be tracked by selection and genetic response to selection, then populations will be maladapted. Human environments have changed radically over the past few hundred years, which is expected to lead to higher risk of psychiatric disorders to the extent that the novel environments include risk factors such as increased social stress and isolation, or toxins such as lead and mercury that degrade neurodevelopment. For example, some of the highest rates of schizophrenia are found among visible-minority (e.g. different skin colour) immigrants, who appear to be subject to relatively severe psychosocial stresses due to their novel, challenging environments [9].

20.2.1.3 Extremes of Adaptations

Some psychiatric conditions, such as generalized anxiety disorder, or some manifestations of obsessive–compulsive disorder such as excessive hygienic behaviour, clearly represent extremes of normally adaptive behaviour: anxiety functions to modulate arousal and attention under challenging conditions [10], and hygiene reduces risks of infection [11]. This conceptual framework has been generalized to connect normal personality variation along a spectrum to personality disorders and to severe psychiatric disorders, by demonstrating which aspects of personality are amplified, reduced, or otherwise distorted to generate mental dysfunction [12]. This approach has successfully described continua in personality traits from normal to maladaptive extremes, although the adaptive significance, in terms of fitness-related benefits and costs of personality variation among normal individuals, remains largely unstudied. Maladaptive extremes can also be considered more directly in the context of human evolutionary history, in that the evolution of human-specific traits, such as large brain size and language, has generated potential and scope for loss of these specific traits, as in microcephaly and specific language impairment, as well as potential and scope for dysfunctional overdevelopment, as in macrocephaly and the disordered and exaggerated components of speech in schizophrenia [13, 14].

20.2.1.4 Trade-Offs

Trade-offs have been well characterized for developmental and physiological phenotypes, whereby, for example, increased resource allocation in one domain takes away from another. For neurological and psychological phenotypes, however,

conceptual paradigms based on trade-offs have yet to be developed, despite evidence for trade-offs of verbal–social with visual–spatial skills [15], empathic with systemizing (rule-based) interests and abilities [16], neural flexibility with stability [17], as well as trade-offs between neural activation of the internally, self-directed default mode network, and the outwardly focused task-positive network [18]. Cognitive and emotional trade-offs are important because they structure the brain's functional architecture and generate coincidences of relative strengths with relative deficits; for example, Kravariti et al. [19] found that having closer relatives with schizophrenia was strongly associated with better verbal skills relative to visual–spatial skills. Trade-offs are stronger under resource-related constraints, which may commonly follow from dysfunctional neurodevelopment, and their extremes are expected to characterize some psychiatric conditions. Autism, for example, has been strongly associated with a combination of high systemizing and low empathizing, whereas some combination of dysfunctionally high empathizing and low systemizing appears to characterize some psychotic affective conditions [20], especially borderline personality disorder and depression [21].

20.2.1.5 Conflicts

Genetically based conflicts, whereby two parties exhibit different optima for some genetically based phenotype, generate risk of maladaptation because one party may more or less 'lose' the conflict, resources are wasted on conflictual interactions, and conflict mechanisms generate novel targets for dysregulation and disease [22]. The forms of evolutionary genetic conflict most salient to psychiatric conditions include parent–offspring conflict (e.g. [23]), genomic imprinting conflict [24, 25], and sexual conflict [26]. Dysregulated genomic imprinting, for example, underlies the expression of Prader–Willi syndrome, one of the strongest genetic causes of psychosis [27], and this syndrome represents only an extreme case of such psychiatric effects [13]. Similarly, a recent epidemiological study of over two million individuals demonstrated that unaffected sisters (but not brothers) of individuals with schizophrenia and bipolar disorder exhibit higher fertility than controls, a pattern that is uniquely predicted by a hypothesis of 'sexually antagonistic' alleles that impose costs on males but benefit females [7].

20.2.1.6 Defences Mistaken as Symptoms

This last 'cause' of disease is only apparent: some psychiatric symptoms represent conditionally adaptive defences for alleviating problematic conditions, rather than deleterious manifestations of disease. Thus, in the same way that fever represents a conditionally adaptive bodily response to infection, with health benefits that usually outweigh its costs, some psychiatric symptoms can be interpreted as conferring benefits, relative to their absence or reduction. Examples of such phenomena include the following: (a) repetitive behaviour in autism, which serves to dampen

excessively high levels of autonomic and sensory arousal [28], (b) dissociation, as a psychological mechanism to reduce deleterious effects of trauma [29], (c) delusion formation in psychosis, as a means to mentally cope with the exaggerated and disordered perceptions of salience (causal meaning) [30], and (d) mild depression (low mood), as a conditionally adaptive response to circumstances that favour disengagement from failing or unreachable goals—which escalates to full depression if useless goal-seeking persists [31]. The danger of conceptualizing defences, like fever, as purely deleterious symptoms is that treating them is expected to make the situation specifically worse unless the underlying cause of the disorder (and defence) is addressed, such as the sensory hypersensitivity in autism, the trauma in dissociation, or the challenging life events and personal motivational structure that underlie liability to low mood and depression.

These six causes of aetiology and symptoms of psychiatric conditions converge in their emphases on determining what evolved genetic, developmental, neural, cognitive and emotional systems are altered, and how they are altered, in psychiatric conditions. These causes also provide our framework for determining how nominal, DSM-designated psychiatric conditions are related to one another in their causes, as independent and separate, partially overlapping, or diametric to one another in the same general way as the development or activity of any biological system or pathway can be altered in two opposite directions.

20.2.2 Autism Spectrum Conditions

Autism is defined, and commonly reified, as a combination of deficits in social reciprocity and communication with high levels of restricted interests and repetitive behaviour (Fig. 20.1). The degree to which this combination represents a cohesive syndrome, with causally shared rather than independent symptoms and causal factors, remains unclear [32]. Beyond these two commonalities, autism presents diverse features, with overall intellectual abilities varying from very low to above average, cognitive enhancements (above neurotypical) in sensory and visual–spatial abilities in a substantial fraction of individuals and a sex ratio that is highly male-biased overall but much less so among more severely affected individuals [33].

The most straightforward connection between the major features of autism, and human evolution, is that our evolutionary history has been characterized by elaboration of the ‘social brain’: the distributed, integrated set of neural systems that subserve the acquisition, processing, and use of social information. It is these social brain phenotypes that are specifically underdeveloped in autism. As such, autism can be conceptualized as the expression of maladaptive extremes of social brain underdevelopment, which, in principle, may be caused in a proximate way by reduction or loss of any of the myriad systems that are necessary or sufficient for human social brain development. Autism thus exhibits many single-gene, syndromic causes due to deleterious mutation, but it is also commonly underlain by combined effects from the hundreds or thousands of genes bearing alleles that affect

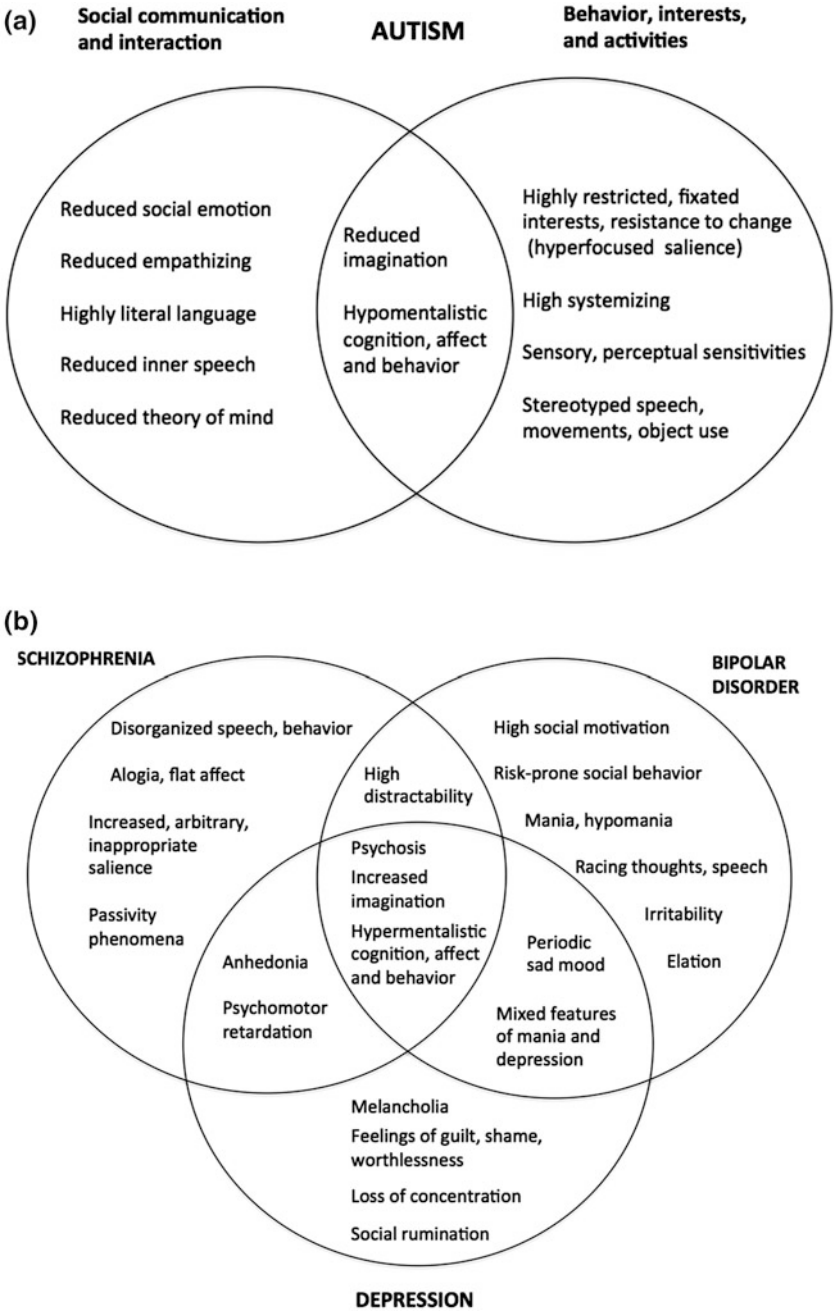

Fig. 20.1 Phenotypes that describe **a** the autism spectrum and **b** the psychotic affective spectrum, based on DSM-V diagnoses, evolutionary considerations, and the hypothesized relationships between the two sets of disorders

social brain development [34]. As such, there can be no primary, proximate physiologically based cause of autism (as there may be, for example, for type 1 diabetes), and the search for causes becomes a differential characterization, subdivision, and prioritization of the diverse genetic, epigenetic, and environmental influences that converge on underdevelopment of the social brain.

As social cognition is underdeveloped under all psychologically based theories for autism, it can also be conceptualized, and studied, in terms of developmental heterochrony, whereby child cognitive development is not completed in autism, and childhood characteristics, including reduced social cognition, are retained into adulthood [35, 36]. In this context, other human-elaborated traits including highly developed, regulated social striving and goal-seeking, guided by perceived reward-associated or cost-associated (aversive) salience (inferred, causative meaning) of social stimuli, remain underdeveloped as well on the autism spectrum. External stimuli may thus have salience predominantly in terms of perceived sensations, or specific, highly restricted non-social interests, especially foci of highly selective attention [37]. Frith [38] indeed sees a weak drive to discern meaning in the world as epitomizing the weak central coherence theory of autism, which has been supported by a wide range of evidence.

A central, unresolved question in the study of autism is whether a single, central, psychological, or cognitive-level factor can explain the apparently inexplicable combination of reduced sociality with restricted interests and repetitive behaviour. In the context of social brain underdevelopment, increased restricted interests and repetitive behaviours, and sensory, visual–spatial, and mechanistic cognition enhancements in autism, can be explained by several hypotheses.

First, increases in asocial phenotypes may pre-empt the development of social phenotypes, such as by directing perceived salience, interests, and brain specializations along asocial paths. Such effects, which are notably represented by a theory for autism aetiology based on enhanced perceptual functioning [39], may be mediated by overdevelopments of sensory perception and mechanistic, systemizing cognition [33].

Second, increased asocial cognition may itself be a direct result of reduced social cognition, as a compensatory or trade-off-based neurodevelopmental mechanism akin to the overdevelopment of non-visual senses among the blind.

Third, some such asocial cognition and behaviour, and phenotypes such as insistence on sameness and stimulus over-selectivity, may, as noted above, represent defences that aid in coping with challenging symptoms such as increased perceptual sensitivity or avoidance of stress from dealing with inexplicable social cognitive tasks.

Finally, one possible resolution, based on reduced expression of a phenotype virtually unique to humans, is that autism is, in part, underpinned psychologically by underdeveloped imagination, defined as ‘the faculty or action of forming new ideas, or images or concepts of external objects not present to the senses’. This hypothesis, originally described by Rutter [40], and Wing and Gould [41], can, in principle, jointly explain social and asocial alterations in autism, including reduced

pretend play, reduced social imagination as expressed in theory of mind, restriction of interests and repetition of behaviour, and insistence on sameness.

Determining the degree to which these hypotheses are correct, in general or for any particular individual, is crucially important to autism therapy, especially to prevent enhancements or conditionally adaptive defences of autistic individuals from being treated as deleterious symptoms.

20.2.3 Psychotic Affective Spectrum Conditions

Psychotic affective spectrum conditions include a set of DSM disorders, mainly schizophrenia, bipolar disorder, and depression, which broadly overlap in their symptoms, neurological and psychological correlates, and genetic and environmental risk factors [42] (Fig. 20.1). All of these conditions exhibit substantial genetic components and mediation in part by rare, penetrant risk factors, although most genetic risk appears to be underlain by many alleles each with small effect.

Schizophrenia, as well as other conditions that involve psychosis, can be understood most directly and simply in terms of dysfunction of the human adaptive system for assigning salience (causal meaning) to external, and internally generated, stimuli [30, 43]. Salience assignment, which is underpinned by a dedicated neural system involving the anterior cingulate cortex and insula, is fundamental to cognition, behaviour, and goal-seeking, in that it mediates subjective causal understanding of perceptual inputs. Psychosis thus involves overdeveloped and inappropriate salience, usually in the contexts of social interactions, agency, intentionality, self-other associations, and other aspects of mentalistic (social and mind-related) thought, apparently due to the primacy of social cognition in human goal-directed behaviour [24]. Paradigmatic manifestations of psychosis thus involve paranoia, other social delusions, megalomania, belief that events always refer to the self, alterations to self-other distinctions, and assignment of mind, agency, and intentions to inappropriate subjects and inanimate objects. Such reality distortions are mediated by top-down cognitive processes, and they can be considered as attempts to 'make sense' of the excessively high and inappropriate salience assignment, for external stimuli, that is driven by hyperdopaminergic neurotransmission [30, 44, 45]. Hallucinations, in turn, can be understood as misinterpreted and exaggerated internal perceptions, mediated by overdeveloped salience of internal representations, such that given certain neurophysiological alterations, thought, inner speech, and imagination come to be considered as external percepts. Like delusions, hallucinations are usually expressed as social phenomena, especially auditory hallucinations.

Schizophrenia is predominantly considered as a disorder of cognition, whereby the causal meanings that guide striving become overdeveloped and dysfunctionally overmentalistic. Bipolar disorder and depression, by contrast, represent mainly disorders of emotion, the set of neural and hormonal systems that motivate and modulate striving and goal-seeking across different contexts. Understanding such

mood disorders requires consideration of the adaptive significance of condition-dependent variation in human emotions, especially with regard to the social interactions that permeate human thought and behaviour [46]. In this context, considerable evidence indicates that low mood is normally adaptive in situations where individuals benefit by disengaging from unreachable or unprofitable goals, as it facilitates such disengagement and motivates alternative behavioural patterns of goal-seeking that should be more advantageous [47]. High, positive mood, in comparison, represents an emotional mechanism whereby human reward systems motivate continuation of beneficial behaviour, because one's goals are being reached. Depression, then, can be conceptualized and studied as overly low and overly stable mood, a maladaptive extreme of an adaptation, whereby individuals fail to disengage from deleterious thought patterns and striving [3, 47]. Conversely, mania represents an emotional opposite to depression, as the expression of inability to emotionally restrain high mood and intensity of striving, even if and when its consequences become detrimental [48, 49]. Behaviours associated with mania and hypomania can, moreover, be directly interpreted in the context of extreme striving for social dominance, power, and influence, which, if successful, leads to substantial benefits [50, 51]. This evolutionary perspective can explain shifts between mania and depression in bipolar disorder, in that mania is expected to foster pursuit of goals that become more and more risky, unreachable or unsuccessful, eventually prompting the generation of mixed states and descent into depression.

In bipolar disorder, then, cognitive salience systems, and choices of goals, commonly remain functional, but the homoeostatic regulation of the emotions that underlie goal pursuit becomes dysregulated, towards overly low or overly high moods and their sequelae. Moreover, like schizophrenia, mania and depression both centrally involve extremes of social, mentalistic thought and behaviour, here in the context of guilt, shame, embarrassment, perceived social defeat, and social rumination in depression, and social dominance pursuit and pride in mania. Affective psychoses, which comprise psychosis with alterations of mood, may thus be mediated by self-punishment-driven, or reward-driven, overattributions of social salience, in the context of emotionality that becomes sufficiently strong to dysregulate salience. These considerations can help to explain well-documented, otherwise-inexplicable associations of bipolar disorder with high social motivation and achievement [49, 52, 53]. Moreover, bipolar disorder, as well as schizophrenia and schizotypy, have been associated across a wide diversity of studies with increased social imagination, divergent thinking, creativity, and goal attainment, especially in the arts and humanities [54–59]. Imagination can indeed be considered, under Bayesian models of cognition and learning, as directly associated with causal cognition and inference of meaning, such that salience, causal thinking, and imagination should tend to increase, or decrease, in concert with one another [60].

20.2.4 The Relationship Between Autism Spectrum and Psychotic Affective Spectrum Disorders

Bleuler invented the term 'autism' to describe withdrawal from reality and social interactions in schizophrenia, but Kanner was careful to point out that his conceptualization of autism referred to children who had never participated in social life [61]. The relationship between autism and schizophrenia, and psychotic affective disorders more generally, has since been considered in terms of two main hypotheses: (1) partial overlap, with some degree of shared social cognitive deficits and genetic risk factors; (2) a diametric (opposite) relationship, based, at a psychological level, on underdevelopment of social cognition and affect in autism, normality at the centre, and dysfunctional forms of their overdevelopment in psychotic affective conditions [62] (Fig. 20.2). The partial overlap hypothesis is data-driven and motivated primarily by the prominence of social deficits especially in autism and schizophrenia. By contrast, the diametric hypothesis follows directly from evolutionary and neurodevelopmental considerations, under the premises that human evolution has been characterized primarily by elaboration of social cognition (generating increased scope for altered development of specific phenotypes) and that the neurodevelopmental systems that underlie it, like all biological systems, can vary and be perturbed in two opposite directions towards lower or higher expression (Fig. 20.2).

A central prediction of the diametric hypothesis is that autism and psychotic affective conditions (especially schizophrenia, for which most of the relevant data are available) should exhibit opposite phenotypes and genetic risk factors. A suite of such evidence is described in Table 20.1, which provides support for the

Under-development on autism spectrum	**Uniquely-human or human-elaborated trait**	**Over-development on psychotic-affective spectrum**
No speech, literal speech	(1) Language	Auditory hallucination
Reduced sense of self	(2) Sense of self	Megalomania, delusions of reference
Low mentalistic skill	(3) Mentalistic skill	Paranoia, other social delusions
Basic emotions	(4) Social emotionality	Shame, guilt, embarassment increased in depression
Mechanical logic	(5) Logical, analytic skill	Thought disorder, loose associations
Reduced goal pursuit	(6) Complex, regulated goal pursuit	Mania, hypomania
Reduced empathizing, empathic abilities	(7) Empathic drive, skills	Enhancements in borderline personality disorder and mild depression
Over-selective attention, restricted interests and repetitive behavior	(8) Drive for causal meaning, salience	Hyper-salience in schizophrenia prodrome, and in psychosis

Fig. 20.2 The autism spectrum and the psychotic affective spectrum can be conceptualized as diametric disorders, with regard to the direction of alterations in uniquely human or human-elaborated phenotypes that comprise their core features

Table 20.1 Diametric genetic risk factors, phenotypes, and correlates of autism spectrum and psychotic affective spectrum conditions

Trait	Autism spectrum	Psychotic affective spectrum	Comments
Copy number variants	Duplications of 22q11.2 increase autism risk [63, 71]	Duplications of 22q11.2 decrease schizophrenia risk; deletions of 22q11.2 greatly increase schizophrenia risk [72]	Deletions of 22q11.2 suggested to increase ASD risk but pattern not found in ASD CNV cohorts [63]
Copy number variants	Duplications of 1q21.1 increase autism risk and increase head size [63, 73]	Deletions of 1q21.1 increase schizophrenia risk and reduce head size [71, 73]	Deletions may increase autism risk, or be false positive [63]
Copy number variants	Deletions of 16p11.2 increase autism risk and increase head size [74]	Duplications of 16p11.2 increase schizophrenia risk and reduce head size [71, 74]	Duplications may increase autism risk, or be false positive [63]
Copy number variants	Duplications of 15q11.2 (BP1-BP2) increase autism risk [75]	Deletions of 15q11.2 (BP1-BP2) increase schizophrenia risk [71]	Deletions and duplications of CYFIP1, a key gene in this CNV region, cause opposite alterations to dendritic spine complexity [76]
Birth size (weight, length)	Smaller size protects against autism; larger size increases autism risk [64]	Larger size protects against schizophrenia; smaller size increases schizophrenia risk [64]	Each of the patterns of risk has been replicated across many other studies
Brain size	Larger brain size in children with autism [77, 78]	Smaller brain size in schizophrenia [79]	Autism involves faster brain growth in early childhood, in particular
Neurological function	Congenital blindness increases risk of autism [80, 81]	Congenital blindness protects against schizophrenia [82, 83]	
Neurological function	Sensory abilities increased in autism [39, 84–90]	Sensory abilities decreased in schizophrenia; sensory deprivation induces features of psychosis [91–98]	Strong, highly consistent pattern in schizophrenia; substantial although somewhat mixed evidence in autism
Neurological function	Prepulse inhibition increased in autism [99, 100]	Prepulse inhibition decreased [101]	Findings highly consistent for schizophrenia, variable for autism
Neurological function	Mismatch negativity increased in autism [102]	Mismatch negativity decreased in schizophrenia [103, 104]	Findings highly consistent for schizophrenia, variable for autism
Neurological function	Mirror neuron system activation decreased in autism [105, 106]	Mirror neuron system activation increased in actively psychotic individuals with schizophrenia [107]	Same protocol used to measure mirror neuron function, in autism and schizophrenia [107]; other studies of schizophrenia usually show reduced activation [108] but do not involve actively psychotic subjects

(continued)

Table 20.1 (continued)

Trait	Autism spectrum	Psychotic affective spectrum	Comments
Neurological function	Default mode system activation reduced in autism, in association with reduced self-referential and imaginative cognition [109–112]	Default system overactivated in schizophrenia, in association with reality distortion and increased imaginative cognition [110]; also less deactivation of this system [113]	Some studies of autism show reduced deactivations of default system that may be associated with reduced activation [110]; Immordino-Yang et al. [114] also contrast autism and schizophrenia as opposite with regard to the default network
Neurological function	Reduced connectivity within default mode in autism [115, 116]	Increased connectivity within default mode in schizophrenia [117–119]	Some mixed results in both autism and schizophrenia, but two reviews support opposite nature of the alterations [120, 121]
Neurological function	Increased local brain connectivity, decreased long-range connectivity, in association with early brain overgrowth [78]	Decreased local brain connectivity, increased long-range connectivity, in association with increased cortical thinning, in childhood-onset schizophrenia [78]	Findings based on the review of neuroimaging findings [78]
Neurological function	Temporal–parietal junction region shows reduced activation in autism and underlies mentalizing reductions [122, 123]	Temporal–parietal junction region shows increased activation in schizophrenia and underlies some psychotic symptoms [124]	
Emotionality and motivation	Reduced social motivation in autism [125]	Increased social motivation in mania, hypomania [49, 51]	Motivation in general decreased in negative symptom schizophrenia, depression
Emotionality and motivation	Cognitive empathic abilities reduced in autism [126]	Some cognitive empathic abilities enhanced in borderline personality disorder and subclinical depression [21, 127]	Cognitive empathic abilities lower in schizophrenia, bipolar disorder, and depression, in association with general cognitive deficits (e.g. [128, 129])
Emotionality and motivation	Reduced social emotion in autism [130]	Increased social emotion expression in bipolar disorder and depression (e.g. guilt, shame, embarrassment, pride) [50, 131]	Reduced general expressed emotionality in negative symptom schizophrenia
Cognitive function	Decreased inattentional blindness in autism [132]	Increased inattentional blindness in schizophrenia [133]	

(continued)

Table 20.1 (continued)

Trait	Autism spectrum	Psychotic affective spectrum	Comments
Cognitive function	Overselective attention [37, 134]	Reductions in selective attention in schizophrenia and positive schizotypy [135, 136]	
Cognitive function	Enhanced Stroop task performance in autism [137]	Decreased Stroop task performance in schizophrenia, by meta-analysis [138]	Results mixed for autism, highly consistent for schizophrenia
Cognitive function	Enhanced Iowa gambling task performance in high-functioning autism [139]	Reduced Iowa gambling task performance in schizophrenia, in most studies [140]	Results mixed for autism, consistent for schizophrenia
Cognitive function	Reduced susceptibility to rubber hand illusion in autism and in healthy high ASD trait individuals [141–143]	Increased susceptibility to rubber hand illusion in schizophrenia [144]	Same general pattern also found for visual illusions, with some inconsistencies [36]
Cognitive function	Literal word interpretation, underinterpretation of social relevance, in autism [145]	Overinterpretation of word meaning and social relevance in schizophrenia [145]	
Cognitive function	Decreased induction of false memories [146, 147]	Increased induction of false memories associated with psychosis phenotypes [148–150]	Results somewhat mixed (some non-significant) for autism
Cognitive function	Semantic memory network states overly rigid in autism [151]	Semantic memory network states chaotic in schizophrenia [151]	
Cognitive function	Working memory deficits in autism [152]; extraordinary working memory enhancements in child prodigies, who score above autism range in attention to detail on autism quotient test and exhibit high rates of autism in their families [153]	Large working memory deficits in schizophrenia; highly consistent finding [154, 155]	Findings of Ruthsatz and Urbach [153] would benefit from replication; areas of excellence in child prodigies notably overlap with those found in savantism in autism [156]
Cognitive function	Hyperlexia found predominantly in autism [85, 157, 158]	Dyslexia associated with schizophrenia and schizotypy [159–161]	Williams and Casanova [162] contrast autism and dyslexia for cortical microstructure
Cognitive function	More deliberative decision-related processing in autism [163]	'Jumping to conclusions' associated with delusions in schizophrenia [164, 165]	

(continued)

Table 20.1 (continued)

Trait	Autism spectrum	Psychotic affective spectrum	Comments
Cognitive function	Bias towards hypopriors in Bayesian models of perception and cognition [166, 167]	Bias towards hyperpriors in Bayesian models of perception and cognition [44, 166]	
Cognitive function	Reduced inference of intentions in autism [168]	'Hyperintentionality' in schizophrenia and schizotypy [169, 170]	Bara et al. [171] contrast autism and schizophrenia directly in this regard
Cognitive function	Reduced theory of mind in autism spectrum children by ToM storybooks test [172]	'Hypertheory of mind' in children with more psychotic experiences by ToM storybooks test [173]	
Cognitive function	Theory of mind abilities reduced in autism, using MASC test, due to combination of hypomentalizing, lack of mentalizing, and hypermentalizing [174, 175]	Theory of mind abilities reduced in association with positive symptoms of schizophrenia, using MASC test, due to hypermentalizing [176, 177]; hypermentalizing also found in borderline personality disorder using MASC [178]	
Cognitive function	Reduced salience of social stimuli, and overly specific and inflexible salience of primary perceptual and non-social stimuli [179, 180]	Overdeveloped and arbitrary salience in prodrome and psychosis, mainly involving social phenomena [43, 45, 65]	
Cognitive function	Decreased perception of biological motion, entities, in autism; fail to see humans who are there [181]	Increased and false perception of biological motion, entities, in schizophrenia; see humans in random dots [182]	
Cognitive function	Selectively enhanced visual–spatial abilities in autism [183, 184]	Reduced visual–spatial skills, relative to verbal skills, positively associated with genetic liability to schizophrenia [19, 185]	
Cognitive function	Enhanced embedded figures test performance among healthy individuals with more autistic traits [186]	Reduced embedded figures test performance among healthy individuals with more positively schizotypal traits [186]	
Behaviour	Reduced imagination and creativity in autism [187, 188]	Increased imagination and creativity, in schizophrenia, schizotypy, and bipolar disorder and in relatives [54, 189–192]	The literature relating psychotic affective spectrum phenotypes and conditions to aspects of increased imagination and creativity is large and diverse; reduced imagination has been considered as a diagnostic criterion for autism

(continued)

Table 20.1 (continued)

Trait	Autism spectrum	Psychotic affective spectrum	Comments
Behaviour	Reduced pretend play and social play in autism [193, 194]	Higher levels of dissociation, hallucination, psychotic affective psychopathology associated with the presence of childhood imaginary companions [195–198]	
Social correlates	Autism associated with technical professions in fathers, mothers, and grandfathers [199–201]	Schizophrenia, schizotypy, bipolar disorder, and depression associated with careers and interests in arts, humanities, and literature [202, 203]	
Social correlates	Autism in family associated with technical college majors [204]	Bipolar disorder, depression in family associated with arts and humanities majors [204]	Insufficient data on schizophrenia for analysis, in [204]
Social correlates	Autism associated with higher socioeconomic status [205, 206]	Schizophrenia associated with lower socioeconomic status [207]	

For phenotypes with large sets of evidence, only recent articles or reviews are cited. Crespi and Badcock [24] present additional evidence, from less recent literature

diametric hypothesis from diverse and independent sources of data. The partial overlap hypothesis is consistent with the sharing of deficits, especially in social cognition, between autism and schizophrenia, but such deficits can also be considered as deriving from opposite alterations both of which reduce performance on standard tests. Genetic risk factors, such as some genomic copy number variants and some SNPs, have also been associated with both autism spectrum disorders and schizophrenia [62]. Such findings, however, are subject to the caveat that premorbidity to schizophrenia in children and young adolescents, in the form of social deficits and associated developmental problems, can be realistically diagnosed only as autism spectrum since there is not (and never has been) a diagnostic category for schizophrenia premorbidity [63]. This structural limitation in the DSM is expected to lead to a non-negligible incidence of false-positive diagnoses of autism among children who are actually premorbid for schizophrenia, especially among individuals harbouring relatively penetrant genetic risk factors such as copy number variants. Patterns of diagnoses for well-studied CNVs indeed fit with expectations from such false-positive diagnoses [63].

The diametric hypothesis for autism and psychotic affective disorders is novel and controversial and has just begun to be subject to systematic, large-scale testing of its predictions (e.g. [64]). However, to the extent that it is correct, the study of human disorders involving social cognition should be revolutionized and provided its first solid grounding in basic evolutionary principles.

20.3 Implications for Policy and Practice

Risks for human mental disorders have evolved. Evolutionary conceptualizations of autism and psychotic affective disorders lead directly to novel, specific implications for understanding, studying, and treating these conditions.

First, autism, schizophrenia, bipolar disorder, and depression cannot justifiably be considered as ‘diseases’ under standard medical models of disease, because the neural system adaptations subject to maladaptive alteration in each case remain inadequately understood. Instead, these conditions currently represent broad-scale, heuristic descriptions for suites of related psychological and behavioural problems, none of which has currently specifiable genetic or neurological causes in the same way as do diseases like cancer or diabetes, and all of which grade smoothly in their symptoms into normality. As such, schizophrenia and related psychotic and affective disorders can best be considered as ‘syndromes’: groups of symptom dimensions that cluster in different combinations across different individuals [65]. Risks and symptoms for these psychiatric conditions have, however, evolved in close conjunction with the evolution of complex human social cognition, affect, and behaviour, which provides the basis for an ultimate understanding, and nosology, of psychiatric maladaptations. In this context, DSM descriptions of autism or a psychotic affective condition should represent starting points for differential diagnosis of their genetic, neurological, social, and environmental causes, for each specific

individual. Such causes are expected to involve some combination of effects from deleterious mutations, evolutionarily novel environments, extremes of adaptations, trade-offs, genomic conflicts, and evolved defences.

Second, autism can be considered, from an evolutionary perspective, in terms of underdevelopment of social cognition and affect, centrally involving some combination, and causal conjunction, of reduced social development with increased non-social perception, attention, and cognition. Such social and non-social alterations may have diverse proximate causes, but they appear to commonly converge, psychologically, on reductions in imagination, which can explain both lower levels of sociality and increases in restricted interests and repetitive behaviour. This conceptualization of autism is fully compatible with previously developed psychological models founded on reduced central coherence [66], lower empathizing and higher systemizing [33], and enhanced perceptual function [39].

Third, psychotic affective disorders can be considered as centrally involving dysfunctionally overdeveloped social cognition, affect, and behaviour, expressed as social hypersalience in aspects of psychosis, dysregulated social goal motivation and dominance-seeking in mania, and extremes of negative social emotionality in depression. Each of these disorders, which grade into one another, can best be understood in the individual-level contexts of the developmental causes of negatively valenced and imaginative social salience, and the motivational structure of one's past, current, and future imagined life goals, especially regarding regulation of, and impediments to, success in striving. This framework is fully compatible with current psychological, neurological, cognitive-science-level accounts of psychotic affective conditions (e.g. [30, 43, 49, 51]), but grounds them in evolutionary considerations and in their relationship to the autism spectrum.

Fourth, autism and psychotic affective conditions can be considered and analysed as diametric (opposite) disorders with regard to social development, cognition, affect, and behaviour. This diametric model provides for comprehensive, reciprocal illumination of the diagnoses, causes, and treatments of these disorders, such that insights derived from studying one set of disorders can be applied directly to the other. Most generally, cognitive behavioural treatments for autism should especially focus on enhancing phenotypes that are overdeveloped in psychotic affective conditions, including social imagination, flexible and social salience, and social motivation and goal-seeking. By contrast, treatments for psychotic affective conditions, in addition to focusing more directly on the adaptive, dynamic regulation of social cognitive salience and mood-directed striving, should involve therapies to make perception, cognition, affect, and behaviour relatively 'more autistic'. Similar considerations apply to pharmacological effects: for example, valproate during foetal development represents a well-established human cause, and animal model, of autism [67], but valproate is also used to treat bipolar disorder and schizophrenia [68]; comparably, mGlur5 pathway antagonists are being used to treat fragile X syndrome and autism [69], whereas mGlur5 agonists are being developed to treat schizophrenia [70].

The findings and inferences described here emphasize that evolutionary approaches in medicine, and psychiatry, can offer specific, well-rationalized

hypotheses and can help to direct research and treatments along novel and promising paths. Such progress should lead, eventually, to the integration of psychiatry with the standard medical model of disease, as dovetailing evolutionary and proximate approaches to the study of brain development and function uncover the adaptive significance of psychological, cognitive, and affective phenotypes and their neurological and genetic foundations.

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