Pathways to Psychopathology Among Autistic Adults

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Abstract

Purpose of Review—Autistic adults frequently require treatment of mental health problems. Increased rates of suicidality and diminished quality of life among autistic people may be partially attributable to psychiatric symptoms. Some risk factors for mental health problems in autistic people are likely the same as risk factors present in neurotypical individuals, but unique factors that are specific to neurodivergent individuals, and some even more specific to autistic people, may exist. Understanding pathways from autism to mental health problems could inform intervention efforts at the individual and societal levels.

Recent Findings—We review a growing body of research identifying risk processes across the affective, cognitive, and social domains. Consistent with the principle of equifinality, different processes appear to independently and jointly lead to heightened risk for the onset of mental health problems.

Summary—Autistic adults frequently utilize mental healthcare services, and experience heightened risk for chronic impairment as a result of mental health problems. Understanding causal and developmental risk processes in autism should inform personalized treatment. We synthesize extant research on these processes and offer suggestions for addressing them therapeutically and societally.

Keywords

Autism; Mental health; Risk; Mechanism

Introduction

The autism spectrum is a multifaceted collection of traits including symptoms that contribute to formal diagnoses [1]; many individuals self-identify as autistic without the level of symptoms that would qualify for a formal diagnosis [2]. Psychiatric symptoms, such as social avoidance, repetitive behaviors, and difficulties in emotion perception are key diagnostic features of autism, and they are also symptoms of other mental health conditions [1]. In addition, common aspects of autism such as inattention, sleep disruption, and frequent heightened levels of stress / worry are diagnostic for many psychiatric conditions, leading to inherent overlap of autism with mental health diagnoses. Many psychiatric disorders are more common among autistic adults than in the general population. Over > 70% of autistic adults meet criteria for additional diagnoses [1]. Throughout the lifespan, autistic people face heightened risk in particular for mood and anxiety problems [3]. Psychiatric problems, widely observed in autistic individuals [4, 5], directly contribute to the high healthcare costs for this population, and adversely impact their quality of life [6–8].

This review focuses on psychiatric disorders named in the Diagnostic and Statistical Manual for Mental Disorders (DSM); we consider myriad distress-linked characteristics commonly present in autistic people, such as sensory issues, pain sensitivity, synesthesia, and social exclusion, as potential vulnerability or exacerbating factors for these disorders, but do not focus on them explicitly as outcomes. We also offer recommendations as to how processes can be targeted therapeutically and societally, capitalizing on preserved strengths and capacities. Comprehensive review of all risk processes is beyond the scope of this paper. As such, the focus herein is on those processes that are likely more specific, or unique, to Autism Spectrum Disorder (ASD). The authors draw on prior research from our labs and others, direct clinical and personal family experience with this population, and personal lived experience as an autistic adult (GS).

There is a high need for care and services in autism, across functional domains (e.g., psychiatric, social, occupational), and this need goes unmet for most autistic adults. In a recent meta-analysis, ADHD was the most common co-occurring condition in autism, followed by depression and anxiety disorders, with estimated prevalence of 18.8% and 17.8%, respectively [9]. Among autistic adults with lower needs for services, up to 25% meet criteria for obsessive–compulsive disorder [10]and up to 50% meet criteria for obsessive compulsive personality disorder [11]. There has been less research on presence of personality disorders in autism; however, co-occurrence with schizoid personality disorder has been observed [12].

Autistic adults visit emergency rooms for psychiatric reasons 4 times more frequently than non-autistic adults [13], which has high costs for both those individuals and their families, and for the healthcare system [14]. Research has also documented heightened risk for

suicide and self-harm among autistic people. In a nationwide study in Denmark, autistic people had a threefold greater rate of suicide attempt [15].

Though our focus is psychiatric symptoms associated with autism, consistent with the prevailing medical model, autism is also associated with preserved capacities and strengths [16]. The medical model, which has dominated clinical science and intervention research, views the 'problem' as residing within the affected or disabled person. In contrast, the social model of disability recognizes that many of the problems experienced by a person with a disability, including autism, are the result of oppression or exclusion by society. Thus, increasingly, autism advocates and researchers highlight autistic strengths such as capacity to focus on details, maintain interest in specific topics, and organize information in different ways than other people do [17]. A strengths-based approach to service delivery and treatment involves helping clients embrace their diversity and wholeness while striving to attain goals and greater quality of life [18].

Following the NIH's Research Domain Criteria (RDoC) [19], we focus on three clusters of risk: affective, cognitive, and social. Figure 1 shows an fMRI meta-analysis of neural systems involved in autism, which suggests that these three domains are of specific relevance to autism. The figure reflects an automated meta-analytic aggregation across 244 functional MRI studies in which autism was mentioned, conducted using public data from the Neurosynth [20] engine as of 2/1/2023. The blue "uniformity map" (regions significantly associated with the mention of autism) and red "association map" (regions more uniquely associated with autism) strongly represent networks subserving primarily affective (e.g., amygdala, including structures involved in motivation such as the basal ganglia), cognitive (e.g., dorsolateral prefrontal cortex), and social (temporo-parietal junction) features. As such, we focus on these three domains in the ensuing sections. Finally, for those risk factors thought to be mutable, information on targeted treatments as well as potentially useful social change is provided, using an experimental therapeutics approach to connect basic science to clinical application [19, 21].

Affective: Emotional Information Processing

There is tremendous heterogeneity in emotion processing patterns within the autism spectrum. Though emotional processing impairments are neither universally observed nor emblematic of the diagnosis, there is a fairly specific strength and profile pattern associated with ASD (see [22] for review). Challenges in emotional processing tend to be more pronounced in the context of social stimuli and when emotions or tasks are complex or cognitively demanding [22]. Alexithymia, or impaired ability to identify and report on, or describe, one's own emotional state [23], is present in up to 50% of autistic adults [24] and is associated with a host of psychiatric conditions [25], including co-occurring conditions such as depression and social anxiety disorder in ASD [26–28]. Alexithymia, along with emotional acceptance, has been found to explain considerable variance in the association between ASD and anxiety among autistic adults [29].

Emotion dysregulation is a term used to describe diminished ability to manage one's experienced or expressed emotion in the service of goal-directed behavior [30]. Dysregulation includes multiple sub-strategies such as heightened emotional reactivity,

persistent negative affect, and reliance on ineffectual or maladaptive coping strategies [31–33]. Considerable research as shown that emotion dysregulation is common in ASD, and associated with the presence of psychiatric concerns including anxiety, depression, and suicidality [34–36].

Neural features of autism that could result in emotion dysregulation have been identified as early as infancy [37], with alterations in developmental trajectories of emotional regulatory function that are associated with neural changes observed within the first two years of life [38]. Features such as increased structural connectivity along pathways connecting brain regions involved in emotional processing (particularly cingulum, and superior longitudinal fasciculus [39]) have been found in young children. Other abnormalities in connectivity with temporal regions of the corpus callosum, subserving more complex emotional processing [39], have been observed in toddlers later diagnosed with ASD [40]. Thus, it is likely that alterations in neural processing of emotion begin early and continue through the lifespan; they may translate to frank symptomatology or be labeled as different from neurotypical processing for a given individual as a child, adolescent, or adult.

Autistic adults display characteristic patterns of brain activity across a wide variety of emotional information processing tasks including decreased reactivity in regions such as the striatum and dorsolateral prefrontal cortex in response to emotion regulation [41], decreased superior temporal and medial prefrontal activation during implicit emotion processing [42], and increased reactivity in structures associated with emotional information processing such as the amygdala, when viewing faces, which is further associated with gaze avoidance to those faces [43, 44] as well as symptomatology of psychiatric conditions associated with emotional avoidance such as social anxiety [45].

These data beg the question of why, if autistic individuals have increased reactivity to emotional information, they have been labeled as low in empathy. Recent findings suggest that, in actuality, autistic individuals have comparatively high levels of emotional reactivity to others' emotions (affective empathy), but may have decreased differentiation of attribution for specific emotion categories (cognitive empathy). Practically speaking, it may be clear to the autistic adult that someone is in distress, and they experience empathic distress, but subtle distinctions regarding the nature of the distress (e.g., differentiating between another person's anxious worry and sad rumination) may be less likely to occur [46].

Similarly, autistic adolescents and young adults have been found to show less frequent or less effective use of strategies to manage emotionality such as reappraisal, and potentially increased use of coping strategies that tend to be less potent, such as rumination [47, 48], which have been associated with increased amygdala reactivity in non-autistic depressed individuals [49]. Moreover, the brain's salience network, of which the amygdala is a key component, has been associated with sustained negative information processing in adult autism [50]. Similarly, alterations in the functional connectivity (synchronization of brain activity across regions) between amygdala and prefrontal cortex (key pathway in regulatory processes in socioemotional processing) have been reported in autism [51]. Connectivity differences have also been reported in autistic individuals during task-free

resting state, which may be indicative of pervasive alterations in emotional information processing that could increase vulnerability to other psychiatric conditions that contribute to broader problems with cognitive and socioemotional processing [52]. Yet, a growing body of data suggests that such observations may be a byproduct of comorbid depressive symptomatology or PTSD [53, 54] with psychophysiological reactivity characteristic of sustained rumination-like emotional information processing [55] being apparent only in autistic individuals who are depressed [56]. Thus, no differences are apparent in ecological observations of repetitive negative thinking between non-depressed autistic and non-autistic adults, and of note, repetitive negative thinking in autism is not related to the presence of other repetitive behaviors [57]. That said, commonly reported aspects of autistic symptomatology, such as "autistic inertia", by which autistic individuals who have an internal experience like an intense emotion may feel "stuck" in that state and unable to mobilize coping resources to ask for help or otherwise regulate their reactions [58].

Another aspect of emotional functioning, which is affected in autism, regards motivation, particularly how reward and punishment are processed. There is a broad reward processing difficulty, reflected in differential neural reactivity, in ASD, which is not specific to social stimuli [59]. Specifically, the anticipation or 'wanting' of the stimulus, or reward [60], which is strongly associated with dopaminergic function [61], is diminished. Diminished anticipation of reward has been associated with stress and affective instability as well as cognitive difficulties in the executive function domain such as working memory and cognitive flexibility [62, 63]. For instance, one must be able to maintain information in memory related to a task and target outcome in order to anticipate reward [64], and reward cues indicate where to direct attention [65]. Aberrant reward processing has long been associated with anhedonia, or loss of pleasure, which is a hallmark of clinical depression. There are also known changes in reward processing linked to the onset of puberty [66, 67]. The research base on the role of motivation and reward processing more generally in relation to mental health in autism is not deep.

Thus, in contrast to previous models, which suggested concepts such as blunted affect, lack of awareness of affect, and low empathy in autism be addressed, neural data suggest that autistic individuals have increased affective reactivity, acute awareness of their heightened affective state, and normative affective empathy. Thus, helping autistic individuals accept and learn to work with their increased reactivity (e.g., via mindful acceptance strategies, and through using preserved capacities) can help prevent or ameliorate stress, anxiety, and depression [68]. Additionally, strategies such as redirecting sensory attention from interoception to external stimuli (e.g., via vibration or fidget toys) and, potentially, teaching cognitive empathy, could be useful. Similarly, increasing societal awareness of the lower threshold for affective reactivity in autism, and differences between affective and cognitive empathy could help to create pathways for autistic individuals to interact as themselves without having to continuously feel shame about, or mask their natural reactions.

Cognitive: Executive Function and Cognitive Load

Many differences in cognitive, sensory, and motor processing have been observed in autistic, compared to non-autistic, individuals (see [69] for a review), from frank learning disabilities

and increased need for structure in some autistic individuals, to differences in how sensory information is processed [70]. Considerable work has been done to understand these features at the subjective, behavioral, neural, and molecular levels [71]. These features can, in some cases, lead to mental health issues.

Intellectual disability, in particular, is associated with certain types of psychopathology [72], though there is debate regarding the extent to which autism serves as a vulnerability factor for psychopathology above and beyond the intellectual disabilities themselves [73, 74]. There is more evidence that cognitive ability moderates the risk for psychopathology and perhaps influences symptom expression (e.g., anxiety and depression among those without cognitive impairment; aggression, ADHD, and pica among those with cognitive impairment).

Differences in executive function (EF), decision-making, and intolerance of uncertainty can lead to psychopathology. For example, severe EF deficits can be clinically impairing and disabling to daily functioning [75]. Autistic adults without cognitive impairment often struggle with cognitive flexibility, working memory, and phonemic fluency, along with relative strengths in planning, decision-making, and semantic verbal fluency [76]. ASD has been uniquely linked to cognitive inflexibility [77], or the ability to shift between different thoughts or tasks [78]. Decreased cognitive flexibility is associated with depression above and beyond ASD features or emotion regulation difficulty [79], potentially through the association of decreased flexibility with increased rumination in autism, which is a vulnerability factor for depression [53]. Alternatively, difficulty with cognitive flexibility and insistence on sameness can breed inflexible depressogenic cognitions (e.g., rigid or 'black or white' thinking) [80]. Similarly, autistic youth with reduced EF exhibit elevated symptoms of anxiety [4].

Consistent with the attentional control theory of anxiety, which suggests that top-down cognitive functioning is reduced during times of high anxiety in order to permit resources to be reallocated to addressing threat, it is possible that executive dysfunction may increase the risk for heightened reactivity and engagement of cognitive biases [81]. Likewise, executive dysfunction can also be associated with increased load on cognitive systems. Focus and cognitive energy are diverted to process stimuli that may be subconsciously disregarded by non-autistic individuals. The experienced cognitive load is only increased by the compensatory strategies that must be consciously applied to function despite these neurological deficits. This additional cognitive labor contributes to a state of chronic stress, well known to contribute to the development of other psychiatric disorders [82]. The accumulation of these effects over time needs more attention in adult outcome research. Alternately, executive control is mediated primarily by prefrontal resources that are implicated in emotion regulation [83] yielding strong relationships between emotion and basic cognitive function [84, 85]. Having low EF is associated with risk for and presence of disorders of decreased emotion regulation in people who are not autistic [86, 87]. Executive dysfunction can thus be associated with unmitigated emotion dysregulation. In autism, such dysregulation can present as anxiety, depression, meltdowns, or intense reactions to stimuli which might not be considered overtly emotional.

Although research evidence has not been uniformly conclusive [88], some autistic adolescents experience anxiety about decision-making, which may be partly due to increased attention to negative outcomes of previous decisions [89]. Emotional exhaustion and anxiety can result from a tendency to collect too much information, and feeling unable to reach a decision [90]. Intolerance of uncertainty, conceptualized as inability to tolerate the emotional and cognitive sequelae triggered by lack of sufficient information in a given situation [91], is commonly seen in autistic people, and contributes to anxiety, depression, and impaired coping [48, 92, 93]. Recent research supports intolerance of uncertainty as a transdiagnostic risk process for the experience of anxiety and depression. Emotion dysregulation has been found to mediate the relationship between intolerance of uncertainty and symptoms of both anxiety and depression [94]. Additionally, alexithymia can lead to confusion and intolerance of uncertainty [28]. There is support, based on cross-sectional data, for a model in which atypical sensory processing (e.g., sensory seeking and sensitivity), along with need for sameness and consistency, drive intolerance of uncertainty, which then partially mediates the experience of anxiety [95].

In light of the aforementioned cognitive-domain risk processes, it is plausible that intervention targeting specific EF processes (e.g., cognitive flexibility) may be helpful. A pilot study of a problem-solving program for autistic young adults showed promising findings with increased problem-solving ability and reduced subjective distress [96]. Likewise, approaches based on the principles of behavioral exposure that facilitate increasing a person's tolerance of uncertainty might promote greater ability to 'sit with' unpredictability. Cognitive-behavioral approaches in this domain have demonstrated impact with autistic children and adolescents [97], but need upward translation to adults [98].

Social: Masking, Motivation, and Societal Factors

Social features common in autism, as well as societal factors broadly, are often strongly linked to mental health issues, including isolation [99], alexithymia, and their associated social consequences such as discrimination, stigma and ableism [100], and the resultant consequences in terms of access to housing, employment, healthcare, and wealth. Loneliness, in particular, may be a distal risk process for depression in this population. Specifically, both social communication impairment [8] and anhedonia, which are rooted in aberrant reward processing [101].

Social cognition, specifically processing of emotional stimuli and mental states, has been strongly linked to unipolar depression [102]. Among non-autistic adults experiencing a depressive episode, deficits have been documented in higher order social cognition (e.g., theory of mind, social perception, and metacognition) [103]. To date, little research has examined social cognition in relation to depression within autism though initial data suggests the links are not direct, and may, in fact, be mediated through executive control deficits [104]. Thus, diminished frontotemporal brain activity during social cognition tasks has been observed in autistic adults, an effect most pronounced among those with co-occurring depression [105].

There are other external and societal risk processes for depression and anxiety. Specifically, bullying and interpersonal victimization [106, 107], social rejection and social exclusion

[108], child maltreatment [109], sexual victimization [110, 111], and other adverse interpersonal experiences during adolescence can result in long-term trauma and post-traumatic stress symptoms, difficulties interpreting social situations, depression and loneliness (e.g. [112]). These negative interpersonal experiences can fuel a feedback loop, whereby autistic people experience negative interpersonal interactions and thus grow less motivated to re-experience social contact due to the negative experience, and thus become more solitary, isolated, lonely, and at increased risk for further social rejection [107].

The historically pervasive but highly contested social motivation hypothesis [113] purports that autistic people find social stimuli less meaningful and less rewarding than do non-autistic people [114]. However, more recent research suggests that in fact any motivational impairments characteristic of ASD are not necessarily specific to the social domain [60]. Other aspects of stimuli, for instance perceptual factors, may play a significant role in motivation. Studies of face processing in autism have found perceptual differences contributing to motivation in unstructured tasks. With more explicit instructions, autistic individuals perform better, as well as show significant activity in fusiform face area [115]. Moreover, motivation for face processing can also be enhanced by familiar, familial, and friendly faces than random face stimuli [116]. For a detailed examination of the social motivation hypothesis and a critique of its assumptions, see [117]. Risk for depression may be higher among autistic people with intact social motivation, due to the mismatch between desire for social engagement and deficits in ability to form quality relationships or lack of self-confidence in one's ability to do so [8].

Autistic burnout is similar to, yet distinct from, general psychological or occupational burnout [118–121]. Autistic burnout captures stress, intolerance, and chronic exhaustion that results from the compounding of cumulative stress, barriers to effective supports that could provide respite or relief, and resource depletion associated with masking autistic features while trying to perform other effortful tasks. Similarly, risk for psychopathology may be heightened through a confluence of high levels of negative social life events, lack of interpersonal reinforcement, and impoverished sense of mastery or competence. This model of depression etiology in ASD is consistent with the interpersonal model of depression [122], while identifying risk factors somewhat unique to autism.

There is specific evidence to suggest that for many autistic people, the strain of "masking" or attempting to camouflage autistic traits in order to increase social acceptance can lead to severe depletion of internal resources, anxiety, and depression [121, 123]. The cumulative stress of masking is often compounded by on-going experiences of interpersonal rejection, social isolation, societal-level stigma related to living with a disability, and barriers to receiving effective helping services.

Negative experiences with therapeutic and support services that are intended to help autistic people unfortunately can increase risk for depression and anxiety. For example, Applied Behavior Analysis (ABA) was founded on theories of operant conditioning, and based on the idea that clinicians should use reinforcement, punishment, extinction, and repetition to extinguish particular behaviors and increase communication in autistic youth [124, 125]. Evidence is now emerging to suggest that at least for some autistic youth, the

experience of participating in ABA caused psychological distress and trauma (e.g., [126–128], methodological criticisms of this nascent body of research notwithstanding (e.g., [129–131].

Intervention often includes helping the person identify sustainable support networks outside one's family [132]. Providers can help the person identify affinity groups, social networks or individuals, while also respecting that the autistic person may have variable desire and may need reprieve from social demands (i.e., wanting social discourse, but also finding it taxing). To some degree, to effectively address motivational processes therapeutically, we need to consider both individual targets and societal change. In other words, intervention that targets social motivation or social skills alone will likely be less impactful than an intervention that targets double empathy issues, interpersonal relationship health, and broader acceptance of differences in motivation and social wanting.

Some research suggests that autistic people who have a social identity as autistic may experience better self-esteem than autistic peers without that identity, and that this positive social identity buffers against depression and anxiety [133]. One study has found that autistic people who have experienced acceptance from external sources, and accept themselves as autistic, experience lower rates of depression [134]. Adults who receive an autism diagnosis in adulthood may experience self-compassion, feel more able to engage in self-care, and feel less self-critical for not conforming to societal expectations [135]. A recent study of 109 autistic adults found that those who adopted a neurodiversity-embracing attitude had higher self-esteem and better subjective well-being than autistic people who viewed autism as a disorder [136]. Finally, autistic people with mental health needs also face challenges identifying, getting access to and receiving counseling and psychotherapy [137]. Importantly, psychotherapists too rarely receive training in autism, lack general knowledge about autism, and may either refuse to treat autistic people or provide less than optimal care [138–140].

Conclusions

We have highlighted research underlying the interplay of autism with mental health conditions and have suggested that with this knowledge, there may be ways to interrupt the feedback loops which maintain them. We note that understanding of interactions between autism and mental health is in its infancy. Additionally, much of the extant research has focused on autistic people with lower service needs, and therapeutic mental health interventions have, for the most part, been explicitly targeted to this group (e.g., [141]). Our understanding of co-occurring psychiatric conditions, much less risk processes, in profoundly affected people and those with intellectual disability, as well as females, is considerably underdeveloped. It does appear, however, that degree of support need moderates the expression or type of mental health problem (e.g., mood, anxiety problems may be more common among autistic individuals without intellectual disability, whereas aggression and inattention are more problematic among those with higher needs).

Research on the processes that increase risk has begun to inform targeted prevention and intervention work. In this review, we summarized research across the broad domains of

cognitive, affective, and social functioning. Consistent with an experimental therapeutics framework [21] to intervention design and evaluation, in which the proximal processes thought to be both malleable and responsible for development or perpetuation of the target behavior (e.g., aggression) are modified, we sought to identify the most viable risk processes within each domain. We assert that this approach holds promise in terms of cost, efficiency, and clinical impact, as clinicians and scientists address the mental health crisis in this population.

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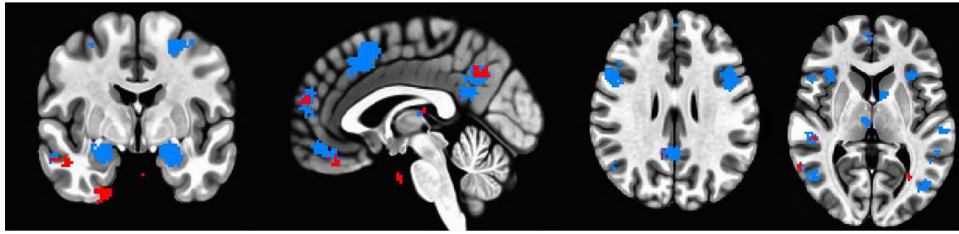
Fig. 1.

Automated fMRI meta-analysis depicting four different slices highlighting brain regions in which activity is associated with autism (from left to right: coronal, sagittal, dorsal axial, ventral axial view). Blue regions are nodes of activity reliably associated with autism in the literature, and red regions are uniquely associated with autism above and beyond other terms