Autism and Depression

To what extent does autism play a role in the onset of depression?

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Contents

Introduction

Autism Spectrum Disorder (ASD), commonly known as autism, is a neurodevelopmental disorder characterized by difficulties in social interactions, communication impairments, and restrictive, repetitive patterns of behaviour and interests. It is an umbrella diagnosis that consists of 4 formerly distinct subgroups, such as Asperger syndrome and autistic disorder. Symptoms of ASD typically appear in the first two years of life and last a lifetime (American Psychiatric Association, 2013). However with appropriate support, symptoms of ASD can be managed and ASD individuals can live equally fulfilling lives.

Major Depressive Disorder, otherwise known as depression, is a serious affective disorder characterized by a persistent feeling of sadness and recurring thoughts of death, usually accompanied by a loss of interest in activities and diminished self-esteem (American Psychiatric Association, 2013). If left untreated, depression could lead to deteriorating relationships, increased chances of risky behaviours and even death by suicide.

It has been widely reported that individuals with ASD often exhibit symptoms of depression and have elevated rates of mood disorders such as major depressive disorder (Magnuson & Constantino, 2011). Despite this, relatively few research studies have explored why this might be the case. Findings of potential causational mechanisms can have immense implications in depression prevention efforts and treatment. This is particularly important for ASD individuals, as they are often exposed to unique risk factors due to the challenges they face. Moreover by uncovering possible causes, this allows treatment strategies to be tailored according to one's condition. Additionally, risk factors for depression in a neurotypical population might not apply to ASD– some risk factors might be magnified while others might

be diminished. Thus, it is important to analyze existing research to identify potential causes, and to mitigate vulnerability to depression in ASD. In this essay, I will examine to what extent does ASD play a role in the onset of depression.

One hypothesis is that vulnerability to depression is influenced by core characteristics and life experiences related to having autism. Social challenges associated with ASD can often lead to peer rejection and alienation, which can serve as risk factors for depression (Whitehouse et al., 2008). Moreover, some have suggested that an awareness of being different from their peers may be related to depression (Hedley & Young, 2006). On the other hand, research has found that depression is more prevalent in relatives of ASD, raising questions on comorbidity and whether there are shared genetic factors linking the two conditions (Bolton et al., 1998). It is important to note that comparing socio-cognitive and biological factors is not the focus of the current research– I will be critically evaluating the contribution of both factors respectively, how and the extent of which it influences vulnerability to depression.

Influence of Socio-Cognitive Factors

In this section of the essay, I will be focusing on the influence of socio-cognitive factors on individuals with high-functioning autism. The term, High-Functioning Autism (HFA) refers to individuals on the spectrum without intellectual disabilities (IQ >80) (Attwood, 1998). This decision to narrow down my research to HFA was made due to several factors. For instance, research into depression in populations with more severe forms of ASD are hard to conduct and are contestable in nature. This is due to communication difficulties and struggles with understanding and articulating their own emotions, which interferes with self-report reliability. Moreover, it is unclear as to how depression manifests in individuals with more severe forms of ASD (Magnuson & Constantino, 2011). Overlapping clinical features and a lack of standard assessment tools make it difficult to compare between research studies. After careful consideration of available research, I have decided to narrow down my focus to HFA individuals for the influence of socio-cognitive factors in vulnerability to depression.

HFA individuals struggle with making friends, thus this may make them more vulnerable to loneliness and depression

It is well-documented that friendships serve as a protective factor from loneliness and depression in neurotypical populations– people with more intimate friendships tend to feel less lonely and depressed (Nangle et al., 2003). Hence it can be hypothesised that the same applies to individuals with HFA. As social impairments are at the core of the disorder, HFA individuals often struggle with making and maintaining friendships, which may lead to increased feelings of loneliness and depression.

In a study by Whitehouse et al. (2008), participants that consisted of controls and adolescents with Asperger's were asked to complete questionnaires designed to measure the quality of their best friendship, feelings of loneliness and symptoms of depression. Results showed that as predicted, adolescents with Asperger's who reported poorer qualities of friendships and social relationships tended to feel more lonely and depressed. This suggests that although individuals with Asperger's may have less desire to make friends (Attwood, 1998), friendships may be equally as important for both groups.

However, further analysis in Whitehouse et al (2008) found that quality of friendships was predictive of loneliness in the control group, but not in the group of adolescents with Asperger's. That is, for individuals with Asperger's, their quality of friendship did not reflect future ratings of loneliness. Assuming both findings are correct, this raises questions on how individuals with Asperger's interpret friendships and social relationships.

One possible explanation is that individuals with Asperger's have a different understanding of friendship. For example, studies have observed that children with HFA struggle with relating to the emotional aspects of friendships and base their intimacy of friendship upon physical proximity (Bauminger & Kasari, 2003). Individuals with Asperger's might perceive their friendships to be closer than reality, hence might have reported their peers as friends even if the other person did not think the same way, raising doubts on the validity of the study.

Another explanation is that individuals with Asperger's might experience loneliness differently. In a study by Bauminger and Kasari (2003), neurotypical children described loneliness as both being alone (social-cognitive loneliness) and feelings of sadness

(emotional loneliness) whereas the majority of HFA children defined loneliness only as a lack of friends. This suggests that perhaps HFA individuals struggle with associating emotions with their loneliness. It raises doubts on whether loneliness in HFA is dependent upon the quality of friendships, and the effect loneliness has on HFA individuals.

Although these explanations are merely speculation and should be taken with a grain of salt, it is the best explanation currently available for the contradictory findings. It is important to recognize that there is always the possibility that such findings were based on flawed research or evidence. This illustrates the challenges of self-report in research into ASD and reflects the need for accurate tools of assessment and measurement. Nonetheless, it is a general trend that adolescents with Asperger's who reported more intimate friendships tend to feel less lonely and depressed, highlighting the importance of healthy relationships in depression mitigation.

It must be noted that participants with Asperger's in Whitehouse et al. (2008) were individuals who received a DSM-IV based diagnosis of Asperger syndrome. Following changes made to DSM-V in 2013, Asperger syndrome was eliminated as a separate diagnosis and is now issued under ASD (American Psychiatric Association, 2013). Although the differentiation of HFA and Asperger's remains a topic of debate, both groups share considerably similar features regarding social impairment and intelligence (Attwood, 1998). Thus in the context of this essay, it is reasonable to generalize research that specifies individuals with Asperger's to HFA.

Although HFA individuals may comprehend loneliness differently, they are still equally as vulnerable to its negative consequences of loneliness

As previously mentioned, Whitehouse et al. (2008) proposed that friendship serves as a protective function against loneliness and depression in HFA individuals– HFA individuals with more intimate friendships tend to feel less lonely and depressed. However, alternative explanations suggest that this might not be the case. Loneliness reflects a difference between the quality of desired and actual friendships– as individuals with HFA are portrayed to prefer social isolation (Attwood, 1998), they may experience loneliness differently.

Mazurek (2013) investigated the nature of loneliness in adults with ASD. Questionnaires regarding loneliness, life satisfaction and well-being were sent via email to participants, who received a US$15 gift card upon completion. Results found that those who reported higher levels of loneliness exhibited more severe symptoms of depression. Moreover, they were also less satisfied with their lives and tended to suffer from diminished self-esteem. Contrary to popular belief that individuals with ASD are not affected by loneliness, loneliness in ASD was found to be associated with negative emotional experiences, and was a risk factor for depression in this study.

An interesting finding is that such correlations remained significant even after controlling for ASD severity (Mazurek, 2013). Although individuals with HFA may lack an emotional understanding of loneliness and might prefer social isolation, they experience loneliness in the same way (independent of their understanding) and are equally as vulnerable to the consequences of loneliness.

Alternatively, it is possible that underlying depression may have provoked feelings of loneliness and social isolation, or that there may be a common cause of depression and loneliness. Correlation does not imply causation– it is crucial to recognize the lack of causation in non-experimental studies. This is particularly relevant for research into mental health, as it is often not ethically possible to manipulate independent variables in human subjects. Further longitudinal studies, such as that by Whitehouse et al. (2008) are required to uncover such interactions. Nevertheless, existing evidence supports the hypothesis that even for individuals with HFA, friendship plays a crucial role in depression prevention and mitigation.

Self-esteem in HFA children may be a risk factor for depression, as opposed to actual social competence

So far, we have established that even for individuals with HFA, friendship is crucial in mitigating feelings of loneliness and depression. By the same logic, those who are less socially competent would struggle more with maintaining friendships, hence would be generally more vulnerable to depression. However, there is a level of complexity to this relationship. In a correlational study with HFA children by Vickerstaff et al. (2007), participants, their parents, and teachers completed questionnaires regarding social competence, self-esteem and depression. Results showed that, as opposed to actual social competence rated by parents and teachers, only self-perceived social competence correlated with depressive symptoms. In other words, participants who believed themselves to be poor at socializing were the ones who felt more depressed, as opposed to those that actually struggled with social relationships.

This suggests that low self-worth and self-esteem might be a risk factor for depression. On the surface, this seems contradictory, as more intimate friendships prevent depression, yet this is unrelated to actual social competence. However, better social competence does not always equate to more intimate friendships. This is because even in groups with better social competence, social deficits that are the core of ASD still exist. Their social struggles will still interfere with their ability to form successful relationships. In other words, an HFA individual might be "socially competent", but he will still struggle socially, hence may still experience trouble making friends.

The strength of Vickerstaff et al. (2007) lies in their research methodology. As they measured social competence reported by both parents and teachers (external perspective) and participants themselves (internal perspective), this allowed them to contrast self-perceived and actual social competence, shedding light on the cognitive functions of HFA individuals. This also highlights the need for using a range of methods to account for limitations to self-report. However, the sample size was limited and participants were children (aged 7-13 years) who might not have experienced depression given their young age. Repeats with larger sample sizes and more mature participants would be required to ensure generalizability.

Self-esteem alters vulnerability to depression by interfering with people's automatic thoughts

As previously stated in Vickerstaff et al. (2007), low self-esteem might be a risk factor for depression in HFA individuals as opposed to actual social competence. As such findings are consistent with research into neurotypical populations, it may be the case that conventional cognitive explanations to depression might apply to HFA individuals. Aron Beck's (1967) cognitive theory of depression suggests that deeply rooted mental beliefs (schemas) about ourselves interfere with people's subconscious, automatic thoughts, which in turn changes how they process their experiences. In this case, HFA individuals are exposed to more negative social experiences, contributing to diminished self-esteem and self-worth. Such negative self-belief provokes pessimistic, irrational automatic thoughts, which changes how they process their experiences, thus making them more vulnerable to depression

If Beck's explanation applies to HFA individuals, then one would expect that knowledge and awareness of their disability would alter one's self-esteem, which would make them more vulnerable to depression. This is because as social deficits are a defining feature of ASD, knowledge of their disability leads to an increased mental focus on negative social experiences, magnifying its impact on self-esteem. This is indeed the case as seen in Butzer and Konstantareas (2003), who observed that children with Asperger's who were more aware of their disability (based on parental report) tended to report higher levels of depressive symptomatology. Moreover, awareness of their disability could lead to a self-fulfilling prophecy, where one's beliefs and expectations of their behaviour influence their actions.

This leads to increased social difficulties and distress, further contributing to the low levels of self-esteem in individuals with autism.

Social comparison in children with Asperger's greatly affects self-esteem, therefore may link to depression

Hedley and Young (2006) extended this further by proposing that social comparisons are responsible for one's self-esteem and self-worth, thus vulnerability to loneliness and depression. In this study, Hedley and Young found that children with Asperger's who perceived themselves to be different and tended to compare themselves socially with their peers exhibited more symptoms of depression in general. Social comparison theory explains this by stating that humans evaluate their own ability and determine their self-worth by comparing themselves to others (Suls & Wills, 1991). Children with Asperger's that compare themselves socially with their peers more often are more frequently reminded of their social deficits, which leads to a lower evaluation of self-worth, thus contributing to depression.

Compelling evidence shows that Aron Beck's (1967) cognitive theory of depression is a competent explanation to the findings of Vickerstaff et al. (2007). Although there are various limitations, particularly with causational mechanisms and generalizability, it can be established that self-perception is a detrimental factor in depression vulnerability. As HFA individuals are exposed to more negative social experiences, this leads to lower self-esteem, which provokes negative, irrational automatic thoughts. Such automatic thoughts influence how they process their experiences, thus leading to increased vulnerability to depression.

So far, we have examined the ways in which ASD influences vulnerability to depression through critically evaluating a range of socio-cognitive factors. For instance, friendship in children with Asperger's prevents loneliness– as HFA individuals struggle with making friends, this may make them more vulnerable to loneliness and depression (Whitehouse et al., 2008). Moreover, self-esteem plays a crucial role in regulating people's automatic thoughts, which influences how they process their experiences (Beck, 1967). As HFA individuals are exposed to more negative social experiences, this influences their self-esteem, thus vulnerability to depression (Vickerstaff et al., 2007). It is important to recognize the complexity of socio-cognitive influences in depression vulnerability. This is particularly relevant for research into ASD, as it is often difficult to distinguish relative contributions of genetic and socio-cognitive factors. In the following section of this essay, I will be evaluating the influence of biological factors, namely genetics and neurochemical influences in the onset of depression.

Influence of Biological Factors

ASD is a neurodevelopmental disorder. As it is a product of physiology, it can be genetically inherited. Family and twin studies have shown that ASD is genetically inherited to a large extent, but research into identifying specific genes has been inconclusive (Szatmari et al., 1998). Research into depression, however, has been able to identify specific genes (such as different functional polymorphisms of the 5-HTT gene) that moderate a person's sensitivity to adverse life events, hence vulnerability to depression (Caspi et al., 2003). As ASD is genetically inherited and individuals with ASD have elevated rates of depression (Magnuson & Constantino, 2011), this raises questions on the comorbidity of ASD and depression. Are there shared genetic factors linking the two conditions? If ASD and depression frequently occur together, could depression be one way in which ASD symptoms are manifested?

First-degree relatives of ASD individuals are more likely to have a history of depression, which suggests a common genetic predisposition between ASD and depression

In an attempt to investigate the genetic relationship between ASD and depression, Bolton et al. (1998) interviewed parents and siblings of ASD adults for a history of mood disorders. Results show that first-degree relatives of ASD individuals were more likely to have a history of depression than the general population.

There are several explanations to the findings of Bolton et al. (1998). First, this could be explained by the elevated psychological stress associated with raising an ASD child. However, the vast majority of parents with a history of depression reported an onset age

before the birth of the child with ASD (Bolton et al., 1998). This suggests that the burden of raising an ASD child cannot fully account for the elevated rates of depression.

Another explanation is that as ASD and depression frequently occur together, depression could be a symptom of ASD. If this is the case, one would expect relatives of ASD individuals that display more symptoms of ASD (but not to a clinically significant level) to be more depressed in general. However, this does not seem to be the case as the relatives who displayed ASD symptoms were different individuals than the ones who were predisposed to depression (Bolton et al., 1998). Such findings support the argument that despite overlapping clinical features, the two disorders have distinct causes, and that depression is not a manifestation of ASD symptomatology.

Other studies have argued that perhaps the two disorders are not genetically related. For instance, ASD children with depression are more likely to have a family history of depression than nondepressed ASD children, suggesting that depression might be inherited separately (Ghaziuddin & Greden, 1998). However, parents may pass on both a genetic vulnerability to depression and corresponding environments that encourage depression. For example, a child could inherit both a genetic predisposition to stress and a familial environment with high expectations. This raises uncertainty on the extent in which the disorder is actually inherited. Family studies are based on data about families spanning several generations, hence cannot differentiate between the effects of genetic and shared environmental factors. Further research into molecular genetics is required to identify specific genes responsible and to isolate genetic influences.

Genetic predisposition depends on environmental factors to be triggered

Despite evidence into the genetic comorbidity of ASD and depression, it is important to recognize that genetic predisposition depends on environmental factors to be triggered. In the same way significant life events act as stressors in neurotypical populations, research has observed that depressed children with HFA experienced significantly higher rates of stressful life events before the onset of their depression (Ghaziuddin & Greden, 1998). Moreover, the effects of life stressors are often magnified for HFA individuals, due to their sensitivity to changes in routine and familiarity, and a lack of social support network. Such stressors may even exacerbate ASD features. This further highlights the vulnerability of HFA individuals, both from a genetic perspective and vulnerability due to their inherent challenges.

Predisposition to serotonin imbalance might affect depression vulnerability in ASD individuals

In the absence of a concrete explanation for the elevated rates of depression in relatives of ASD individuals, research has shifted to identifying genetic causes of depression in neurotypical samples, and establishing whether this is particularly relevant for ASD individuals.

One of such is the serotonin hypothesis, which states that serotonin (a neurotransmitter) imbalance in the brain is a factor that causes depression. This is based on two types of evidence: Firstly, depression-inducing side effects were found in certain drugs that were known to deplete levels of serotonin in the brain. Secondly, drugs such as MAO inhibitors

and SSRIs that were shown to enhance synaptic concentration of serotonin in animal studies appeared to be effective against symptoms of depression (Cowen & Browning, 2015). As serotonin levels are inherited and reduced serotonin levels are associated with increased depression severity (Leventhal et al. 1990), low serotonin levels in ASD individuals would suggest potential genetic comorbidity between the two disorders.

Surprisingly, Cook et al. (1994) found the opposite to be true– elevated blood serotonin levels were found in ASD individuals and their relatives. In addition, it was also found that parents of ASD children with elevated blood serotonin levels tend to be more depressed than parents of ASD children with normal blood serotonin levels (Cook et al. 1994). This suggests that perhaps elevated blood serotonin levels are responsible for the increased rates of depression in ASD families.

One possible explanation is that serotonin was found to influence the way the brain processes emotions, affecting emotional well being. As emotional processing functions differ for ASD individuals, serotonin might play a different role in emotional regulation. Moreover, in the same way serotonin produces negative effects in a small number of patients, serotonin might have adverse effects in ASD individuals due to their inherent neurological differences. In this case, the shared neurochemical predisposition would explain why depression is more prevalent in individuals with ASD.

There are numerous areas of uncertainty with this explanation. As with every correlational study, cause and effect interferences are not possible. Depression caused by other factors may induce a neurochemical imbalance, creating an accumulative effect. In addition, recent

animal studies have suggested that serotonin might be a mediator between depression and ASD symptoms. By controlling variants of the serotonin transporter gene, researchers have been able to manipulate blood serotonin levels of mice. Mice with elevated blood serotonin levels displayed abnormal social functions, communication impairments and repetitive behaviour, similar to symptoms displayed by autistic mice models (Veenstra-VanderWeele et al., 2012). On the other hand, other studies have observed that serotonin transporter knockout rats (rats with low serotonin levels) displayed behaviour that resembled depressive symptoms, such as diminished motivation, social isolation and appetite changes (Olivier et al., 2008). It is probable that genetic predisposition to serotonin abnormalities influences both how ASD symptoms manifest and sensitivity to environmental stressors (which influences depression vulnerability). Although elevated blood serotonin levels in ASD individuals have been widely documented, no animal study so far (to the author's knowledge) has recorded the effects of serotonin manipulation on both disorders. Existing evidence warrants further research to establish any neurochemical predispositions between ASD and depression.

Lastly, the serotonin hypothesis is often criticized for its basis on the treatment etiology fallacy: treatment for depression (which targets serotonin) reduces symptoms of depression, hence serotonin causes depression. This might not be the case. The fact that antidepressants, which promote brain serotonin levels do not take effect immediately shows that perhaps the influence of serotonin on depression might be indirect. As mentioned above, research has suggested that serotonin influences emotional processing in the brain, which might act as a mediator between serotonin and depression (Harmer et al., 2009). Without an adequate understanding of underlying mechanisms, it is difficult to determine how serotonin predisposition affects depression vulnerability in ASD individuals.

Conclusion

This essay examined to what extent does ASD play a role in the onset of depression. Research studies suggest that vulnerability to depression is influenced by ASD symptomatology and life experiences related to having HFA. For instance, Whitehouse et al. (2008) proposed that friendship serves as a protective factor from loneliness and depression in HFA individuals. However, alternative explanations suggested that this might not be the case, as HFA individuals have a different understanding of friendship and loneliness (Bauminger & Kasari, 2003). To eliminate such alternative explanations, Mazurek (2013) investigated the nature of loneliness in HFA individuals and found that their experience of loneliness is independent of their understanding and perception of loneliness.

Moreover, findings from Vickerstaff et al. (2007) suggested that low self-worth and self-perception might be a risk factor for depression, as opposed to actual social competence. This can be explained by Aron Beck's (1967) cognitive theory of depression. As HFA individuals are exposed to more negative social experiences, this diminishes their self-esteem, provoking irrational automatic thoughts, thus leading to increased vulnerability to depression. This is supported by findings from Butzer and Konstantareas (2003) and Hedley and Young (2006), who found that factors which encourage attention to negative social experiences contribute to depression vulnerability.

On the other hand, Bolton et al. (1998) observed that relatives of ASD individuals were at increased risk for depression, raising questions on the genetic relationship between ASD and depression. However, explanations for this phenomenon have been inconclusive. Cook et al (1994) proposed that common neurochemical predispositions might explain comorbidity

between the two disorders. Animal studies have highlighted a potential connection between serotonin genetic predisposition and the two disorders (Veenstra-VanderWeele et al., 2012; Olivier et al., 2008). However, further research is needed to establish causational mechanisms and to ensure reliability and generalizability of study findings. Additionally, despite the evidence presented in this essay, there is still a huge degree of uncertainty in the role of ASD in depression vulnerability. Nevertheless, this essay provides valuable insight into existing research, and builds on interconnected concepts to establish the role ASD plays in the onset of depression.

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