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INVESTIGATING THE LONGITUDINAL RELATIONSHIP BETWEEN SOCIAL MOTIVATION AND DEPRESSION IN AUTISTIC ADULTS

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Abstract

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Autism affects individuals across the lifespan, yet there tends to be limited research and services for autistic adults. This is especially concerning given that autistic adults have high mental health needs, with depression being one of the most common and clinically significant co-occurring conditions.

We explored the longitudinal relationships between social motivation, social access (i.e., having opportunities for meaningful social interactions), loneliness, and depression in N=303 autistic adults ages 18-65. Participants completed online surveys about social behavior and wellbeing three times over 3–4 months. We hypothesized that an interaction between higher social motivation and lower social access at Time 1 would predict depressive symptoms at Time 3 via the mediator of loneliness at Time 2. Our hypothesis was not supported, though loneliness significantly mediated the relationship between T1 low social access and T3 depression. We discuss the non-significant interaction in light of challenges measuring social motivation, defining and measuring "social access," and possible bidirectional effects of social motivation and depressed mood that unfolded pre-study. The findings still highlight the importance of social access to mood in this population and supporting meaningful social opportunities for autistic adults universally, not just those who desire social experiences.

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Chapter 1

Introduction

Autism spectrum disorder (ASD) is a neurological and developmental disorder that is characterized by impairments in reciprocal social communication and social interaction (e.g., failure to initiate or respond to social interactions) and restricted, repetitive patterns of behavior, interests, or activities (American Psychiatric Association, 2013). Autism is referred to as a "spectrum" disorder because there is a wide variety in the type and severity of traits individuals can experience (National Institute of Mental Health, 2022).

Autism and Depression in Adults

Autism can be diagnosed at as early as age two and then throughout the lifespan (National Institute of Mental Health, 2022). However, there is little research on and even fewer services available for autistic adults (Mason et al., 2022), even though approximately 100,000 autistic individuals who age into legal adulthood every year in the United States tend to continue to have high service needs (Shattuck, 2019). Most interventions for the autistic community are designed for children, leaving autistic adults and their behavioral health needs severely underserved (Pallathra et al., 2019).

In addition to behavioral health needs that align directly with autism symptoms, autistic adults tend to also have high mental health needs (Lever & Geurts, 2016). Per recent meta-analyses, the autistic population is four times more likely to experience lifetime depression than non-autistic individuals (Hollocks et al., 2019; Hudson et al., 2019). Indeed, depression is one of the most common and clinically significant outcomes among autistic adults (Hofvander et al., 2009; Howlin, 2004; Joshi et al., 2013). In this

community, depression is associated with distress, decreased quality of life, increased service use, increased caregiver burden (Joshi et al., 2013), and increased self-injury and suicidality (Cassidy et al., 2018). In sum, given the high rates of co-occurring depression among autistic adults, the significant challenges to well-being and quality of life that accompany depression, and the lack of resources for this population, it is imperative to learn more about this co-occurrence, including how to intervene most effectively. In particular, research into the potential causal mechanisms of depression in autistic adults may indicate important novel directions for improving mental health prevention and treatment in this population.

Potential Etiology of Depression in Autistic Adults

Shared Etiology with Non-Autistic Populations

When investigating etiological mechanisms leading to depression among autistic adults, we may first consider the same factors that contribute to the onset of depression in the general population: These known demographic or mechanistic factors may similarly contribute to depression, but in fact be more prevalent in the autistic population. For instance, rates of non-binary and other gender minorities are widely suggested to be higher among the autistic community (George & Stokes, 2018), and gender minority status is associated with increased depression in both autistic people (Murphy et al., 2020; Schiltz et al., 2021) and in the general population (Borgogna et al., 2019; Fox et al., 2020). As a side note, while autistic populations tend to skew male at a rate of approximately 4:1 (Zeidan et al., 2022), female sex is associated with depression in both autistic (Oswald et al., 2016; Schwartzman et al., 2021; Gotham et al., 2015) and non-autistic (Kessler et al., 1993; Piccinelli & Wilkinson, 2000) individuals.

Stress related factors such as bullying, traumatic and other negative life events, and un- or under-employment, correlate with depression in both the autistic population (Bruggink et al., 2016; Fung et al., 2015; Griffiths et al., 2019) and nonautistic population (Amiri, 2021; Lee, 2021; Strohacker et al., 2021), as well. Again, autistic adults tend to report more of these negative life events than do neurotypical controls (Bruggink et al., 2016; Griffiths et al., 2019), particularly bullying (Griffiths et al., 2019; Humphrey, & Symes, 2010; Iyanda, 2022; McLeod et al., 2019; Toseeb et al., 2018) and unemployment (Griffiths et al., 2019; Lallukka et al., 2020; Maslahati et al., 2021). Similarly, loneliness, or the unmet desire for social connectedness, is associated with depression in both the autistic (Hedley et al., 2018; Mazurek, 2014) and general population (Erzen & Cikrikci, 2018; Hawkley & Cacioppo, 2013). Again, evidence further suggests higher levels of loneliness in the autism population than the general population (Jobe & White, 2007; Lasgaard et al., 2009; Whitehouse et al., 2009).

Emotion regulation and personality factors may contribute to depression among both autistic (Cai, Richdale, Uljarević et al., 2018) and non-autistic individuals (Aldao, et al., 2016; Villalobos et al., 2021). Findings demonstrate that, compared to the general population, autistic individuals experience more impairments in emotion regulation (Cai, Richdale, Dissanayake et al., 2018; Cai, Richdale, Uljarević et al., 2018; Samson et al., 2015) and are more likely to utilize maladaptive emotion regulation strategies (Bruggink et al., 2016; Cai, Richdale, Dissanayake et al., 2018; Cai, Richdale, Uljarević et al., 2018). Irritability is also related to depression in both populations (Mayes et al., 2011) and may be particularly prevalent among autistic individuals, with two studies reporting that more than half of their sample of autistic individuals demonstrated irritability (Green

et al., 2000; Mayes & Calboun, 1999). Lastly, neuroticism has been shown to be associated with depression in the autistic (van Oosterhout et al., 2021) and general population (Jylhä, & Isometsä, 2006); again, adolescents and adults on the spectrum score higher on neuroticism measures compared with controls (Lodi-Smith et al., 2019). Taken together, factors related to demographics, stress, emotion, and personality that contribute to depression in non-autistic adults may also play a causal role in depression in autistic adults. Importantly, many of these shared potential causal factors may be more prevalent among the autistic population, contributing to the increased rates of depression in autism.

Etiological Factors Specific to Autism

We may also consider that the increased rates of depression in autistic adults, as compared to the general adult population, could be influenced by risk factors that are *specific* to autistic individuals insofar as being related to the characteristic ways autistic individuals think, experience emotion, and/or interact with others and the world around them. For the purpose of this thesis, such factors that potentially involve differences in brain function or behavior related to autism phenomenologically will be referred to as "internal factors." On the other hand, factors involving intersectional processes between autistic individuals and their community or society will be categorized as "external factors." Internal factors that have been associated with increased risk for depression in autistic adults include sensory hypersensitivity (Rossow et al., 2021), perseverative or repetitive thinking focused on negative content (Gotham et al., 2018), alexithymia (i.e., the inability to recognize or label one's own emotions that is reported in about 50% of autistic individuals; Oakley et al., 2020), anomalous reward processing (i.e., motivation

toward and experience of pleasure from rewarding things or activities; Han et al., 2019), and intolerance of uncertainty (Cai, Richdale, Uljarević et al., 2018).

External factors that correlate with depression in autistic adults include lower perceived social support (Han et al., 2019), expectation of social rejection (Botha, 2020; Keenan et al., 2019), internalized stigma (Botha, 2020; Cage et al., 2018; Cooper et al., 2017), "camouflage fatigue" (i.e., the negative health outcomes hypothesized to be related to masking autistic traits in one's daily social environment; Cage et al., 2018; Hull et al., 2017; Lai et al., 2017), and lower levels of connectedness with the autism community itself (Botha, 2020; Cooper et al., 2017). Overall, it is likely that both internal factors (e.g., thought processes like repetitive negative thinking) and external factors (e.g., minimal or unsuccessful interactions with the world around the individual) that are specific to autistic phenomenology or identity may contribute to elevated depression rates in autistic adults.

Again, understanding the etiological mechanisms leading to depression among autistic adults – and how these mechanisms interact with each other – may open doors to improving our ability to treat and even prevent depression in this population. The current thesis focuses on social motivation (related to the internal factor of aberrant reward processing), and its interaction with social access (related to the external factor of reduced opportunities for meaningful social experiences), as potential causal contributors to depression in autistic adults. Before elaborating on our hypothesis for the potential causal role of social motivation, we first outline its theoretical underpinnings, in terms of the different processes that constitute reward processing and how they play a role in social development.

Reward Processing: "Wanting" vs. "Liking"

Reward processing, also referred to as hedonic capacity, denotes the neural and experiential ability to both seek out and experience pleasure (Gard et al., 2007). Within psychology and neuroscience research fields, the concept of reward is divided into *anticipatory* and *consummatory* pleasure, distinct processes that differ temporally. Anticipatory reward is defined as the hypothetical pleasure involved in "wanting" a reward that one does not yet have, while consummatory reward is defined as the active pleasure of "liking" a granted reward while "consuming"/experiencing it (Berridge & Robinson, 2016; Treadway & Zald, 2011). As an example, if an individual experiences a craving for chocolate prior to eating chocolate, the anticipatory reward would be the craving itself, including the motivation and desire to find a piece of chocolate, while the consummatory reward would be the pleasure derived from actively eating the chocolate.

Evidence from animal models and neuroimaging research suggests that these two reward processes involve distinct neural mechanisms. Generally, anticipatory pleasure is generated by large and robust neural systems that heavily rely on mesolimbic dopamine, while consummatory pleasure is generated by scarce, smaller, more fragile neural systems that do not depend on dopamine (Berridge & Robinson, 2016). More specifically, anticipatory pleasure is mediated by subcortically weighted neural systems, such as mesolimbic dopamine projections to forebrain targets (e.g., the nucleus accumbens and other parts of the striatum; Berridge et al., 2009; Berridge & Robinson, 2016). Further, the brain substrates for anticipatory pleasure are diverse, more widely distributed, and more easily activated than the substrates for consummatory pleasure (Berridge et al., 2009). By contrast, consummatory pleasure occurs when a coordinated

network of small, fragile brain "hedonic hotspots" is stimulated (Berridge & Robinson, 2016; Nguyen et al., 2021). Findings suggest that activation of one of these hotspots leads to recruitment and activation of other hotspots, so that the entire network acts as a unified circuit (Nguyen et al., 2021). These hotspots can be neurochemically stimulated by opioids or endocannabinoid neurotransmitters (the brain's natural heroin-like and marijuana-like substrates) to amplify "liking reactions," whereas stimulations by dopamine fail to increase active "liking" (Berridge & Robinson, 2016). Taken together, consummatory pleasure is mediated by activation of small hedonic hotspots that then recruit each other to form cortical circuits (Berridge et al., 2009; Berridge & Robinson, 2016), compared to the more robust and diverse anticipatory reward circuitry. These neurobiological findings provide evidence that the two reward processes are distinct. Thus, from this framework of separating anticipatory and consummatory reward processes, the scientific literature has gone on to examine *impairments* in reward processing, such as those that may occur in autism and/or contribute to depression among autistic individuals.

Impairments in Reward Processing

Impairments in reward circuitry may involve reductions in the capacity to experience pleasure, which is referred to as anhedonia (Treadway & Zald, 2013). Evidence suggests that trait levels of anhedonia exist among non-clinical populations and can serve as a risk factor for developing psychological disorders (Chan et al., 2012; Harvey et al., 2007; Trøstheim et al., 2020). In addition to being a risk factor, anhedonia is a symptom or characteristic of numerous mental health disorders, such as substance use disorders, schizophrenia, depression, and pain disorders (Trøstheim et al., 2020).

Notably, the neural and genetic mechanisms underlying anhedonia appear to be similar across disorders, insofar as they relate to dysfunction in the mesolimbic dopamine system and its interactions with endogenous opioids (Trøstheim et al., 2020). Across disorders, inflammation or excessive immune activity also may contribute to anhedonia, suggesting that anhedonia develops in order to serve an adaptive role of conserving energy and accelerating healing in response to mental or emotional pain (Fries et al., 2018). In sum, anhedonia varies in nonclinical populations and can be both a risk factor and a symptom of psychological disorders. This transdiagnostic symptom may share common causal mechanisms (e.g., similar underlying genetic factors and/or evolutionary function) across disorders (Trøstheim et al., 2020).

Despite similarity in causal mechanisms leading to anhedonia across disorders, evidence suggests that the actual presentation of anhedonia differs by disorder. Specifically, there appear to be three distinct patterns to the clinical phenomenology of anhedonia:

1. <u>Reduction in consummatory pleasure with intact anticipatory pleasure:</u> Anhedonia may consist of intact anticipatory pleasure but reduced consummatory pleasure: Individuals may still feel motivated for a reward (i.e., intact anticipatory pleasure) that they no longer find as pleasurable upon consumption / experiencing (i.e., reduced consummatory pleasure) as they had in the past. For example, individuals experiencing substance dependence may demonstrate this pattern, in that they continue to feel motivated to obtain the drug (intact anticipatory pleasure) despite

experiencing increasingly less pleasure from the drug itself (reduced consummatory pleasure; Berridge & Robinson, 2016).

2. Reduction in anticipatory pleasure with intact consummatory pleasure: Anhedonia can also be characterized by intact consummatory pleasure but reduced anticipatory pleasure; in other words, individuals feel less motivated for a reward (i.e., reduced anticipatory pleasure; often measured by how much effort they are willing to expend to get the reward) despite still enjoying the reward when they receive it (i.e., intact consummatory pleasure; Treadway & Zald, 2013). Evidence indicates that depression is characterized by this pattern of aberrant reward processing (Dichter, 2010).

3. Reductions in *both* anticipatory and consummatory pleasure: Finally, some researchers propose that reductions in consummatory pleasure may precede and cause reductions in anticipatory pleasure: Individuals experience less enjoyment during activities (i.e., reduced consummatory pleasure), which causes them to experience less motivation to do these activities in the future (i.e., reduced anticipatory pleasure; Treadway & Zald, 2013). A recent meta-analysis concluded that schizophrenia is characterized by this pattern of impairments in reward processing (Visser et al., 2020).

Table 1

Patterns of Reward Processing Impairments

	Anticipatory Reward	
Consummatory Reward	Intact	Impaired
Intact	Typical functioning	Pattern 2 (e.g., depression)
Impaired	Pattern 1 (e.g., substance use)	Pattern 3 (e.g., schizophrenia)

Reward Processing in Depression

Importantly, findings show that anhedonia in major depressive disorder tends to be characterized by the second pattern of anhedonia outlined above, namely that of decreased anticipatory pleasure but intact consummatory pleasure (Dichter, 2010; Treadway & Zald, 2013). For example, neuroimaging evidence indicates that individuals with depression show reduced activation of striatal regions and the right caudate during reward anticipation (Smoski et al., 2009). Neurobiological findings similarly demonstrate impairments in anticipatory pleasure in depression: Again, motivational anhedonia correlates with compromised dopamine function (Treadway & Zald, 2011), and the very manipulation of the dopamine system leads to the efficacy of antidepressants (Treadway & Zald, 2013), with mesolimbic dopamine blockades selectively influencing "wanting" but not "liking" rewards (Dichter, 2010). Of note, reward network *hyperactivation* during reward anticipation has been shown to mark remitted depression post-treatment (Dichter, Kozink, McClernon et al., 2012). All together, these findings highlight the presence of

abnormalities in anticipatory pleasure in depression but not necessarily consummatory pleasure.

Reward Processing in Autism

Like depression, autism as a condition may involve impairments in reward processing, as well. Because autism is characterized by pervasive social impairments, researchers often distinguish between non-social and social reward processing when discussing reward processes within autism.

Non-Social Reward Processing. Neurobiological findings indicate that individuals on the autism spectrum show non-social reward processing impairments in line with the combined pattern described above (i.e., impairments in both anticipatory and consummatory pleasure; see Table 2). Autistic individuals demonstrate reward circuitry hypoactivation (e.g., reduced nucleus accumbens and orbitofrontal cortex activation) for non-social anticipatory and consummatory rewards (e.g., in anticipation of and while receiving monetary incentives, respectively; Dichter, Felder, Green et al., 2012; Dichter, Richey, Rittenberg et al., 2012) in comparison to neurotypical controls. However, contrary to findings using monetary incentives (a method commonly used in non-social reward research), autistic individuals do *not* show reward circuitry hypoactivation in anticipation of object images associated with their special interests (another diagnostic feature of autism), and actually show increased activation of the ventromedial prefrontal cortex (a region of the prefrontal cortex involved with reward valuation; Moretti et al., 2009) in anticipation of and in response to these images (Dichter, Felder, Green et al., 2012). Thus, with regards to non-social rewards, autism is characterized by both anticipatory and consummatory pleasure impairments that depend

on the type of stimuli present, and thus can present as either hypoactivation or anomalous hyperactivation of reward circuits.

Social Reward Processing. Evidence suggests individual variability in anticipatory social reward processing (commonly referred to as social motivation) and consummatory social reward processing among the autism spectrum, with some evidence that the drive for social rewards tends to increase with age in autistic individuals (Deckers et al., 2014; Deckers et al., 2017). Autism as a condition, however, has been characterized by overall impairments in social motivation, which may help explain the hallmark social impairments of autism (Dichter, Felder, Green et al., 2012). In fact, anomalous social reward processing has been proposed as an underlying cause of the phenomenon of autism itself (Social Motivation Theory; Chevallier et al., 2012).

In order to best conceptualize social reward impairments in autism, we must first summarize social motivation in typical development. Chevallier et al. (2012) outlined an integrated model, describing the basis of social motivation in typical development at the behavioral, biological, and evolutionary levels. At the behavioral level, typical social motivation involves social orienting (i.e., humans prioritize attention toward objects with social importance, including other people's faces), seeking and liking (i.e., people not only orient to the social world, they find it rewarding), and social maintaining (i.e., people want to engage with others over long periods of time; Chevallier et al., 2012). Moreover, according to Chevallier's model, individuals engage in these prosocial behaviors not because they expect direct benefits for their efforts, but because they find the social behaviors inherently rewarding (Chevallier et al., 2012). Taken together, from

infancy on, humans feel motivated to engage in social interactions because they derive pleasure from the social world and the prosocial behaviors themselves.

At the biological level, typical social motivation reflects activity in the amygdala, ventral striatum, and orbital and ventromedial regions of the prefrontal cortex (Chevallier et al., 2012), as reflected in neuroimaging studies across the lifespan (Isaacowitz et al., 2021). Lastly, from an evolutionary perspective, collaboration and maintaining social interactions have benefits for increasing both individual and species fitness: survival depends on activities – e.g., exchanging information, sharing food, or helping one another – that would be impossible without collaboration (Chevallier et al., 2012).

Social motivation impairments in autism are then conceptualized in counterpoint to this integrated model of neurotypical social motivation in humans. At the behavioral level, individuals on the autism spectrum demonstrate impairments in social orienting starting in the first year of life, such as infrequent orienting to one's own name, decreased eye contact, and social aloofness (Osterling et al., 2002). Further, eye-tracking experiments indicate that when children watch static social photographs (e.g., photographs of friends chatting), children on the spectrum look more at the background than at the characters (Riby & Hancock, 2008). Regarding seeking and liking, children with autism may not show a preference for socially salient sounds compared to non-social control noise (Klin, 1991), and preference for collaborative activities is reduced in autism (Chevallier et al., 2012). Social maintaining impairments in autism include that autistic individuals are less likely to hide affect or offer spontaneous gestures of greetings or farewells (Chevallier et al., 2012), and half of the adult autistic population reports having no particular friends (Howlin et al., 2004).

At the biological level, individuals on the spectrum show abnormal orbitofrontal-striatum-amygdala circuit activity in response to social stimuli (e.g., faces, social approval; Chevallier et al., 2012). Autistic individuals also demonstrate disrupted oxytocin functioning (Chevallier et al., 2012), which is a hormone that leads to activation of reward pathways during prosocial behaviors (Dichter & Rodriguez-Romaguera, 2022). Social motivation impairments in autism may occur due to neuroinflammation, as animal studies demonstrate that neuroinflammation may disrupt social motivation via effects on striatal dopamine functioning (Dichter & Rodriguez-Romaguera, 2022). Finally, an evolutionary perspective on social motivation impairments in autism may help explain why other interpersonal dispositions remain intact in autism (e.g., attachment, sexual drive), as these other interpersonal dispositions are more essential to survival and reproduction and result from different evolutionary pressures (Dichter & Rodriguez-Romaguera, 2022).

Taken together, Chevallier et al.'s integrated model of social motivation helps explain the deficits in social motivation in autism from a behavioral, biological, and evolutionary perspective. The evidence on social motivation impairments in autism come together in the Social Motivation Theory of autism (Chevallier et al., 2012), which proposes that, for individuals on the spectrum, disruptions in brain mechanisms that typically mediate social motivation from infancy decrease autistic children's motivation to engage in social behaviors during early development (Dichter & Rodriguez-Romaguuera, 2022). This decreased motivation then results in fewer experiences with social rewards, which in turn prevents the development of social skills and neural networks important for social communication (Dichter & Rodríguez-Romaguera, 2022).

This dynamic interplay between reduced motivation for early social orienting and reduced opportunity for social reward thus creates a developmental cascade in which infants and toddlers predisposed to autism have fewer opportunities to develop basic social skills – at both behavioral and neurobiological levels – and thus lack the foundation on which neurotypical children build more complex social skills throughout development. Chevallier et al.'s Social Motivation Theory may therefore help explain both the reduced engagement with the social environment that is characteristic of early development in autism (Aldridge-Waddon et al., 2020), as well as the presentation of often lifelong social deficits.

As noted above, however, Chevallier et al.'s social motivation theory of autism may not capture the whole picture of social motivation (i.e., social anticipatory reward) within the autism spectrum. The evidence reviewed above supports initial reports of autism, as described by psychiatrist Leo Kanner in the 1940's, as a condition marked by extreme self-focus and lack of interest in others (Kanner, 1943). However, clinical anecdotes and research findings over time have converged to build a much more variable picture of social motivation in autistic individuals: Despite evidence for overall social reward deficits in autism, social motivation remains individually variable in the autistic population, and generally tends to increase with age (Deckers et al., 2014; Deckers et al., 2017). In summary, then, social anticipatory pleasure may be high for some autistic individuals despite a general reduction in this population overall, and is thought to increase across development for many individuals with autism.

Summary of Reward Processing in Autism. Overall, evidence suggests that autism is associated with impairments in non-social reward processing (both anticipatory

and consummatory pleasure) and anticipatory social reward processing (i.e., social motivation), with consistent findings of individual variability in these constructs. Combining these findings indicates that social motivation deficits in autism may reflect blunted anticipatory responses to rewards more broadly (Dichter, Felder, Green et al., 2012; Dichter & Rodriguez-Romaguera, 2022). Further, given the hyperactivity in pleasure response to objects related to special interests, there may be a reward processing bias in autism that favors specific categories of nonsocial rewards at the expense of social rewards (Dichter, Richey, Rittenberg et al., 2012). Importantly, though, evidence indicates that this reward processing bias does not apply to all autistic individuals; levels of social motivation vary and appear to increase with age among the autistic population, with many autistic individuals even showing high levels of social motivation (Deckers et al., 2014; Deckers et al., 2017). Thus, more research is needed to better understand the phenomenon of social motivation (i.e., anticipatory social reward) in autism. In addition, there seems to be a lack of evidence for social consummatory reward deficits in autism (Kohls et al., 2012). While evidence for non-social reward processing deficits appears to be more consistent in the autistic population, more information is needed on social reward processing in autism.

Table 2

Overview of Reward Processing in Autism

	Social Reward	Non-social Reward
Anticipatory Reward	**Impaired** ● Behavioral level: ○ Social orienting (e.g., no preference for socially salient sounds) ○ Seeking and liking (e.g., reduced preference for collaboration) ○ Social maintaining (e.g., less likely to hide affect) ● Biological level (e.g., oxytocin neuropeptide signaling abnormalities) ● Evolutionary level (e.g., other interpersonal domains more relevant to survival are intact)	**Impaired** ● Reward circuitry hypoactivation compared to controls during anticipation of monetary incentives ● Some evidence for hyperactivation compared to controls in anticipation of seeing images of restricted interest objects
Consummatory Reward	**Intact?** ● Minimal published evidence for impairment; common anecdotal experience indicates that autistic people have individually variable levels of social enjoyment.	**Impaired** ● Reward circuitry hypoactivation compared to controls while receiving monetary incentives ● Reward circuitry hyperactivation in response to images of restricted interest objects

Note. See text for associated citations for these findings (pp.10-15).

Social Motivation and Depression in Autistic Adults

At this point, we have summarized evidence for anomalous reward processing in both depressed and autistic populations separately. We turn now to a discussion of how impairments in social reward processing may play a specific role in depression *among* autistic adults.

As described previously, some autistic adults have intact or even high levels of social motivation. In the context of social deficits that are a key characteristic of autism itself, socially motivated autistic individuals may be less likely to get their social needs met compared to individuals in the general population. In other words, social impairments intrinsic to autism may prevent a subset of autistic individuals from forming and maintaining the social connections they desire. Indeed, studies have reported higher rates of loneliness among autistic adults compared to the general population (Jobe & White, 2007; Lasgaard et al., 2009; Whitehouse et al., 2009). Meanwhile, as noted previously, loneliness correlates with depression in autistic adults (Hedley et al., 2018; Mazurek, 2014) and prospectively predicts depression in non-autistic samples (Cacioppo et al., 2006). Thus, loneliness seems to be a valuable construct to explore in relation to social reward processing and depression in autism. Importantly, for the purpose of this thesis, we are defining loneliness as the subjective distress associated with the perception that one's social needs are not being met, per either quantity and quality of one's social relationships (Hawkley & Cacioppo, 2010).

Han and colleagues observed that self-reported pleasure in social and nonsocial rewards significantly moderated the relationship between autism traits (as a proxy indicator of social access, or the opportunities for meaningful social interaction) and loneliness in a sample of autistic adults and non-autistic adults who varied on depressive symptoms (Han et al., 2019); loneliness was then observed to be the strongest predictor of depression symptoms across diagnostic cohorts (autistic, non-autistic depressed, and non-autistic non-depressed). Specifically, participants with a lower self-reported capacity for pleasure showed high levels of loneliness regardless of their level of autism traits;

individuals with a greater capacity for pleasure demonstrated a positive correlation between loneliness and autism traits that increased in strength with increases in the capacity for social pleasure (Han et al., 2019). As self-report of social pleasure in hypothetical situations is used synonymously with social motivation (Gooding & Pflum, 2014), Han et al. interpreted their findings to indicate that, for autistic individuals who *want* social interaction, the discrepancy between their high desire for and low opportunity for access to social interaction likely led to higher levels of loneliness, which then contributed to depression. Of note, Han et al. also found that autistic adults who reported low social motivation still experienced elevated loneliness, and again, loneliness was strongly associated with depressive symptoms (Han et al., 2019). Similar findings were reported by Ee et al. (2019).

A recent review by Smith and White (2020) corroborated Han et al. (2019)'s findings on those with intact social motivation. Specifically, Smith and White conducted a systematic review investigating symptoms of depression in adolescents and young adults with autism. Based on the accumulated findings, they proposed a social motivation model of depression in autism whereby the presence or absence of social motivation moderates the relationship between social communication deficits and loneliness, with loneliness then mediating the development of depression (Smith & White, 2020). In line with Han et al. (2019)'s findings, Smith and White concluded that the interaction between social anticipatory pleasure (i.e., social motivation) and social access contributes to loneliness; loneliness is then a precursor to depression. In other words, for autistic individuals with intact social motivation, the discrepancy between their desire for social

interaction and their low social communication skills may have led to feelings of loneliness, which in turn may contribute causally to depression (Smith & White, 2020).

By contrast, Smith and White (2020)'s social motivation model suggests that individuals with reduced social motivation are less likely to develop depression because there is little discrepancy between their levels of social motivation and their ability to experience success in social relationships (i.e., both social motivation and social ability are low; Smith & White, 2020). This proposition was not supported empirically in Ee et al. (2019) or Han et al. (2019), in which participants with low levels of social motivation still reported high levels of loneliness and associated depressive symptoms regardless of autism trait level (i.e., a proxy marker of social impairment). One reason that Han et al.'s observations did not support Smith & White's theory may be due to the fact that the Han et al. data were cross-sectional: A competing interpretation suggests that already-depressed individuals may have reported low levels of social motivation due to their current depressive state, versus the opposite temporal pattern in which individuals with *a priori* low social motivation could be at greater risk for developing depression. Among cross-sectional data, it is not possible to assess whether low social motivation precedes or follows from depressive symptoms.

In summary, findings on the relationships between social motivation, social success (or lack thereof) as related to social ability, loneliness, and depression in autistic adults are inconsistent, and longitudinal research is necessary to clarify these relationships. While it seems that findings are more consistent in suggesting that autistic individuals who have intact social motivation but reduced social access (i.e., opportunities for meaningful social interactions) are at increased risk for depression via

loneliness, it is less clear the extent to which autistic individuals with *reduced* social motivation are at risk for depression. Further, to our knowledge, the relationships between social motivation, social access, loneliness, and depression have not been investigated longitudinally in autistic adults.

Objectives of the Current Study

In this Master's thesis project, we aimed to investigate the longitudinal relationship between social motivation, social access, loneliness, and depression in autistic adults. In contrast to previous literature that looked at autistic traits or social communication abilities as proxy measures of social success, we focused on self-reported *social access*, which we defined via two key components: (1) having opportunity to engage in social interactions, regardless of type or modality (e.g., in person with one friend, within an online chat group, etc.), and (2) perceiving one's social interactions to be meaningful and fulfilling. In other words, we would consider an individual to demonstrate high social access if they have sufficient opportunity to engage in social interactions that are meaningful to them. As an example, we would not consider an individual to demonstrate social access if they were surrounded by people at work (i.e., high social opportunity) but did not feel connected to these coworkers or find these interactions to be fulfilling (i.e., low social meaning). Likewise, we would not consider an individual to demonstrate social access if they experienced a meaningful and fulfilling social connection with a friend that they only had contact with once every few years (i.e., low social opportunity).

In line with both Han et al. (2019) and Smith and White (2020), the current study tested whether an interaction between social motivation and social access prospectively

predicted depressive symptoms, such that those with intact social motivation and reduced social access were more likely to show increased loneliness and depressive symptoms over time. Further, the current study also investigated whether low social motivation predated and prospectively predicted depression longitudinally regardless of social access, in line with Han et al.'s (2019) interpretation and Chevallier et al.'s Social Motivation Theory (2012), or if, by contrast, low social motivation would be associated with lower outcome depressive symptoms, thus appearing to protect against depression as suggested by Smith and White's social motivation model of depression in autism (2020).

We hypothesized an observed interaction between social motivation and social access, such that higher Time 1 social motivation combined with lower Time 1 social access indices would correlate with higher Time 2 loneliness and Time 3 depressive symptoms, and that this interaction would predict depression symptoms prospectively via the mediator of loneliness. This was our sole stated hypothesis, as the inconsistent prior literature made our planned analyses of those with low social motivation an exploratory investigation at this time.

As noted, studies on the association between social motivation and depressive symptoms have tended to be cross-sectional and have yielded inconsistent findings within the autism literature. Given that social impairments are a characteristic feature of autism, and notably high rates of depression exist in the autistic population, it was valuable to interrogate social motivation and social access as potential intervention targets to improve mental health in this population

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Chapter 2

Methods

The current study was part of a broader study examining longitudinal predictors of depression in autistic adults, with a focus on female sex and masking ("camouflaging") of autism symptoms in daily life as potential predictors. This parent study aimed to investigate the role of "camouflage fatigue" in depression in this population, with specific inquiry into the relationships between camouflage behaviors, social anxiety, and suicidal ideation. The study took place entirely online over the course of approximately 3–4 months; participants completed an approximately 25-45 minute survey battery largely focused on mental health and well-being, which included but was not limited to variables of interest for the current thesis. Participants completed a variation of this battery every four weeks, with two weeks allowed for completion, yielding three assessment periods (hereafter referred to as survey modules) total within approximately 3–4 months. Of note, the time estimates to complete the survey modules applied to the broader study; out of the several domains within each survey module (see Figure 1), the current thesis project focuses only on the instruments listed in Table 5.

Recruitment and Eligibility

We planned to recruit 300 autistic adults from the Simons Foundation Powering Autism Research for Knowledge (SPARK) cohort, a North American online registry of autistic adults supported by the Simons Foundation, a private funder of autism research. Through the SPARK Research Match, the Simons Foundation matches interested members of the autism community to a wide variety of research studies throughout the United States and internationally (Feliciano et al., 2018). Each individual research project

team must receive Institutional Review Board approval from their own institution prior to submitting an application to recruit through the SPARK registry. If approved by the SPARK Participant Access Committee (which includes a rotating board of SPARK team members, external scientists, and community stakeholders including autistic self-advocates), the SPARK team works together with the web design company Tempus to build and host survey research on Tempus' web portal platform. SPARK, via Tempus, handles all communication with their registrants who have previously indicated that they wish to be contacted about new research opportunities. This includes recruitment/invitation and reminder emails, as well as payment delivery via electronic gift codes funded by the individual research projects. Thus, in the case of the current parent study, the SPARK / Tempus team carried out all participant communication and hosted the survey modules on their own secure platform, and the Rowan research team designed the project, received and maintained IRB approval, funded the project, and ultimately received de-identified data only.

For the broader study (with embedded thesis project), the SPARK team advertised the study to independent autistic adult registrants (i.e., those who are their own legal guardians) who had consented to be contacted about research opportunities, and who met the following additional eligibility criteria: 18-64 years of age, had a self-reported status as a legally independent adult, were capable of fluent language use in English, and reported an autism diagnosis made by a healthcare provider.

The age range of 18-0 and 64-11 years was selected to represent the culturally typical age of "working adults" in the United States (i.e., 65 is the age when individuals become legally considered "older adults" by being insured by Medicare, collecting social

security, often retiring, collecting pensions, etc.). We excluded older adults because (1) autistic adults in this age group have received less attention in research and therefore may differ in important but unknown ways from our "working-age" adult sample, and (2) older adults have a lower chance of being diagnosed with autism in rigorous ways that included parent informant reports, so the validity of the autism diagnoses may become more questionable in this older age group.

Participants needed to have a self-reported status as a legally independent adult capable of fluent language (hereafter, verbally fluent). We had no in-person contact with the participants and did not assess their level of spoken language or reading ability; this eligibility criterion required participants to have fluent command of the language in order to understand and answer questionnaires in whatever way they saw fit (e.g., via reading or in text-to-speech format). Additionally, because many instruments within our battery have been standardized only in English, participants needed to be proficient in English (by self-report).

Finally, participants needed to self-report having an autism diagnosis made by a healthcare provider (which is also requisite for enrollment as a proband in the SPARK registry; Feliciano et al., 2018). Even though these diagnoses were not independently validated, many SPARK participants are recruited from university autism clinics, and therefore have a high likelihood of a valid autism diagnosis (Feliciano et al., 2018). Further, one study determined that in a previous version of the SPARK participant pool, 98% of the registry participants were able to produce documentation verifying a professional autism diagnosis (Daniels et al., 2012). More recently, Fombonne et al. (2021) corroborated that more than 98% of autism cases in the SPARK cohort could be

confirmed in electronic medical records. Furthermore, core clinical features recorded in the electronic medical records were in agreement with SPARK cohort data, providing further evidence of the validity of clinical information in the SPARK database (Fombonne et al., 2021).

Final Sample

A total of 303 participants completed the study. Table 3 below presents the demographic data. The average age of participants was 35.82 (SD=10.74 years, Range 18-60 years). The sample included 133 women (44%), 36 gender nonbinary individuals (12%), and 134 men (44%). The sample was predominately white, with only 12% reporting non-white race and 8% reporting Hispanic/Latino ethnicity. Similar to other SPARK studies, participants tended to have markers of higher socioeconomic status compared to clinical convenience samples of autistic adult participants, with 47% of this sample having a bachelor's degree or more education, and 41% employed full time. About 40% of the sample was single, and 21% of the sample lived alone.

Table 3

Demographics and Descriptives

Demographics Item	Mean (SD) or Number (Percentage)
Age in Years	35.82 (SD= 10.74; Range: 18-60)
Gender	
Woman	133 (44%)
Man	134 (44%)
Nonbinary or Other	36 (12%)
Autism Symptoms (CATI scores)	155.63 (SD=24.36; Range: 42-202)
Ethnicity	
Hispanic, Latinx, or Spanish	25 (8%)
Race	
Black or African American	
Asian	17 (5%)
American Indian or Alaska Native	15 (5%)
Middle Eastern or North African	20 (6%)
Native Hawaiian or Other Pacific Islander	2 (0.6%)
White	1 (0.3%)
Other	268 (88%)
	12 (4%)
Self Highest Education Level	
Some high school or less	4 (1%)
High school diploma or GED/alternative credential	30 (10%)
Trade school or vocational/technical certificate	10 (3%)
Some college but no degree	82 (27%)
Associate degree (e.g., AA, AS)	33 (11%)
Bachelor's degree (e.g., BA, BS)	82 (27%)
Some graduate/professional school but no degree	12 (4%)
Master's degree (e.g., MA, MS, MEng, MEd, MSW, MBA)	40 (13%)
Doctorate degree (e.g., MD, PhD, JD, EdD)	10 (3%)
PHQ-9 (depression symptoms)	8.76 (SD=7.54; Range: 0-32)
ODSIS (depression symptoms and impairment)	4.64 (SD=5.46; Range: 0-20)

Demographics Item	Mean (SD) or Number (Percentage)
Employment Status (Wave 1)	
Employed full-time	124 (41%)
Employed part-time	66 (22%)
Irregular/Occasional work	24 (8%)
Training/Apprenticeship	1 (0.3%)
Volunteer work	16 (5%)
Full-time or part-time student	40 (13%)
Retired	7 (2%)
Actively looking for more work and/or a different job	28 (9%)
Receiving disability benefits, unemployment, or worker's compensation	46 (15%)
None of the above	24 (8%)
Relationship Status	
Dating	25 (8%)
Divorced	20 (7%)
Married/Long Term Relationship	134 (44%)
Single	122 (40%)
Other	1 (0.3%)
NA	1 (0.3%)
Living Situation	
Alone	64 (21%)
Other	12 (4%)
Parents/relatives	73 (24%)
Residential Facility	1 (0.3%)
Roommates	19 (6%)
Spouse/partner	124 (41%)

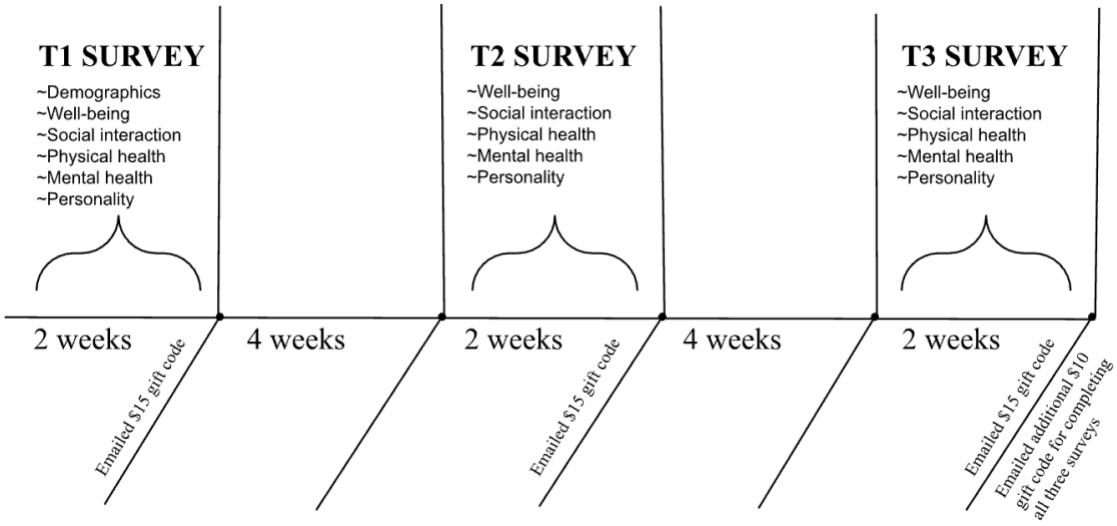
Note: Comprehensive Autistic Trait Inventory (CATI; English et al., 2021), cutoff score of 134 indicates autism; Revised Patient Health Questionnaire-9 with Impairment (PHQ-9; Kroenke et al., 2001), scores of 5,10, 15, and 20 represent mild, moderate, moderately severe, and severe depression respectively; The Overall Depression Severity and Impairment Scale (ODSIS; Bentley et al., 2014), cutoff score of 8 indicates presence of mood disorder

Procedure

SPARK registrants who consented to participate completed a 25–45 min survey module containing questionnaires on demographics, medical/psychiatric history, recent depression treatment, social interaction style, mental health symptoms (depression, anxiety, suicidality, obsessive-compulsive symptoms, anger), somatic symptoms, sensory sensitivities, well-being, and personality traits (autistic traits, circumscribed interests, neuroticism, distress tolerance, trait resilience). Demographics were collected at Time 1 only, but the remainder of the survey module was administered three times over a period of approximately 3–4 months (see Figure 1). Participants were given a two-week window to complete the module at each time point. Time 2 survey modules were administered 4 weeks after completion of each participant's Time 1 module, again with an individualized two-week window for completion, and finally Time 3 survey modules were administered 4 weeks after the completion of the Time 2 module (or 8 weeks after the completion of the Time 1 survey module for participants who did not complete the Time 2 module). Participants were compensated $15 per survey module in Amazon gift codes, with an additional $10 gift code distributed to participants who completed the module at all three timepoints. Participants thus received up to a total of $55 in emailed Amazon.com gift codes for completing all study procedures.

Figure 1

Timeline of Data Collection



Measures

Again, the broader study surveyed constructs such as mental and physical health history and current symptoms, personality traits, and markers of well-being. In this thesis, we will describe in depth only those instruments critical to our analyses: To describe our sample, we operationalized the level of autistic traits; to test our hypothesis, we measured social motivation, social access, loneliness, and depressive symptoms.

Autism traits were assessed through the Comprehensive Autistic Trait Inventory (CATI; English et al., 2021), which is a 42-item measure of autistic characteristics. Participants rated the level to which they agreed with statements (on a five point scale ranging from *Definitely Agree* to *Definitely Disagree*) across six factors: Social Interactions (e.g., "Social interaction is easy for me"), Communication (e.g., "I can tell how people feel from their facial expressions"), Sensory Sensitivity (e.g., "There are times when I feel that my senses are overloaded"), Repetitive Behavior (e.g., "I have a

tendency to pace or move around in a repetitive path"), Social Camouflage (e.g., "I rely on a set of scripts when I talk with people"), and Cognitive Rigidity (e.g., "I feel discomfort when prevented from completing a particular routine"; English et al., 2021). In a sample from the general population that included autistic individuals, the CATI was shown to have convergent validity with the Autism Spectrum Quotient and the Broad Autism Phenotype Questionnaire at the total scale and subscale level, as well as higher internal reliability for total-scale scores compared to these other measures of autism traits (English et al., 2021).

For our sample, the average CATI score (autism symptoms) was 155.63 (SD=24.36, Range 42-202), and about 80% of the participants scored at or above the cut-off for an autism classification on this instrument (CATI score of 134 is cut-off for autism; English et al., 2021).

In order to measure social motivation, we utilized the Anticipatory and Consummatory Interpersonal Pleasure Scale (ACIPS; Gooding & Pflum, 2014), which is a 17-item self-report questionnaire measuring social reward. The scale asks individuals to rate the level to which items are true of them on a 6-point scale (*Very false for me* to *Very true for me*), with lower scores indicating reduced capacity to experience interpersonal pleasure (Gooding & Pflum, 2014). The scale addresses social reward across several contexts, including more passive vs. active forms of social experiences (e.g., "I enjoy looking at photographs of my friends and family" vs. "I like talking with others while waiting in line"), in large groups (e.g., a party) vs. 1:1 interactions, and across familiar vs. unfamiliar people, in order to assess social motivation dimensionally (Gooding & Pflum, 2014). The ACIPS total score has been shown to reliably measure social reward per its

negative and significant correlation with scores on the revised Social Anhedonia Scale, a commonly used self-report measure (Gooding & Pflum, 2014).

The ACIPS originally included subscale scores for anticipatory pleasure ("wanting"; e.g., "I look forward to seeing people when I'm on my way to a party or get-together") and consummatory pleasure ("liking"; e.g., "I enjoy joking and talking with a friend or coworker."). Though the ACIPS was created with the goal of distinguishing between anticipatory versus consummatory reward, neither the original validation study (Gooding & Pflum, 2014) nor subsequent data from this instrument have supported the ability to differentiate these temporal states in a self-report questionnaire. As individuals are asked to rate their anticipated pleasure in a hypothetical reward that is not currently being consumed at the time of rating, the ACIPS total score is commonly used as a measure of anticipatory social reward (i.e., social motivation). The ACIPS has been used in autistic adult samples (Feller et al., 2021; Han et al., 2019; McKenney, 2022; Novacek et al., 2016; Zhang et al., 2020) and has been shown to demonstrate high internal consistency in this population (Han et al., 2019; McKenney, 2022; Novacek et al., 2016).

There are various ways to conceptualize the "consummatory" aspect of social interaction that complements the "wanting" of social motivation. In previous literature, this has been operationalized by number and quality of friendships, sheer frequency of opportunities to interact with others, or self-rated perceived social connectedness, support, or satisfaction (among other definitions). As mentioned before, we focused on "social access," which we defined as a combination of both opportunity for social interactions *and* finding meaning in social interactions. Importantly, both components (i.e., opportunity and meaning) are necessary to elevate "social access" scores. For

instance, if an individual frequently interacted with co-workers, but did not feel a sense of belonging or connectedness in any social interactions, we would not consider this to be high social access. Similarly, if an individual had one very close friend with whom they felt a meaningful connection but that person was rarely able to "spend time" together (across any modality), we would not consider this to be high social access.

Our two components of social access were primarily measured through the Patient-Reported Outcomes Measurement Information System (PROMIS; Cella et al., 2010), which is a set of person-centered measures evaluating physical, mental, and social health in adults and children, as charged and funded by the National Institute of Health. PROMIS has been validated and shown to be a psychometrically sound measure of life domains (HealthMeasures, 2022). *Opportunity for social interaction* was assessed through a demographics survey question that asks about the frequency of social events, as well as the PROMIS Satisfaction with Participation in Discretionary Social Activities scale items (Cella et al., 2010) that ask about satisfaction with the current level of social activity. *Meaning derived from social interaction* was measured by the PROMIS Emotional Support items (Cella et al., 2010), which includes items asking about the degree to which participants feel they have someone to confide in and how well they think others know them, the PROMIS Global Items (Hays et al., 2009) assessing social ability and social satisfaction, as well as the PROMIS Loneliness items (Cella et al., 2010) asking about social connection. Table 4 summarizes the specific items we used to operationalize social access. Under Statistical Analyses below, we discuss the methods of reducing these items across instruments into a single metric.

Table 4

Items Representing the Construct of Social Access

Item	Location of Item	Social Access Component
On average, how often do you participate in social events (i.e., spending time together with someone in-person or over a live video chat) with friends, neighbors, or people from outside your family?	Demographics Survey	Social Opportunity
"I am satisfied with my current level of activities with my friends."	Well-being survey PROMIS Satisfaction in Discretionary Activities Items	Social Opportunity
"I am satisfied with my current level of social activity."	Well-being survey PROMIS Satisfaction in Discretionary Activities Items	Social Opportunity
"I am satisfied with my ability to do things for my friends."	Well-being survey PROMIS Satisfaction in Discretionary Activities Items	Social Meaning
"In general, how would you rate your satisfaction with your social activities and relationships"	Well-being survey PROMIS Global Items	Social Meaning
"In general, please rate how well you carry out your usual social activities and roles. (This includes activities at home, at work and in your community, and responsibilities as a parent, child, spouse, employee, friend, etc.)."	Well-being survey PROMIS Global Items	Social Meaning
"I have someone who will listen to me when I need to talk."	Well-being survey PROMIS Emotional Support Items	Social Meaning
"I have someone to confide in or talk to about myself or my problems."	Well-being survey PROMIS Emotional Support Items	Social Meaning
"I have someone who makes me feel appreciated."	Well-being survey PROMIS Emotional Support Items	Social Meaning
"I have someone to talk with when I have a bad day."	Well-being survey PROMIS Emotional Support Items	Social Meaning

Item	Location of Item	Social Access Component
"I feel that people barely know me."	Well-being survey PROMIS Loneliness Items	Social Meaning
"I feel isolated from others."	Well-being survey PROMIS Loneliness Items	Social Meaning
"I feel that people are around me but not with me."	Well-being survey PROMIS Loneliness Items	Social Meaning
"I feel isolated even when I am not alone."	Well-being survey PROMIS Loneliness Items	Social Meaning
"I feel that people avoid talking to me."	Well-being survey PROMIS Loneliness Items	Social Meaning

To measure loneliness, we used the Scale of Loneliness (SoLo; Williams et al., 2022), a 13-item measure developed by our research team in collaboration with colleagues from Vanderbilt University and University of California-Los Angeles. Items include "When I am around other people, I still feel lonely and isolated" and "I am satisfied with how often other people include me in their activities and plans." This scale was created in response to our observations that existing loneliness scales appeared to be conflated with general sadness and/or asked about social isolation without tapping into respondents' feelings or beliefs about this state. As social isolation is not necessarily perceived as negative by all individuals on the autism spectrum, we created this scale to operationalize loneliness as longing for social experiences or connectedness, and/or dysphoria or distress specific to social isolation.

Depression levels were measured by two instruments, the first being the Revised Patient Health Questionnaire-9 with Impairment (PHQ-9; Kroenke et al., 2001). This is a

10-item adapted version of the original nine-item depression scale (Kroenke et al., 2001) that modifies the language in several of the original PHQ-9 questions and includes an additional impairment question. The Revised PHQ-9 (PHQ-9-R) asks participants to rate over the past 2 weeks how many days the problems depicted in the items have bothered them, using a four-point scale ranging from 0 (rarely or not at all) to 3 (almost every day). The PHQ-9-R was created by Nicolaidis et al. (2020) as part of a community-based participatory study on determining the best practices for adapting questionnaires for the autistic community (Nicolaidis et al., 2020). Items include "Feeling down, depressed, or hopeless" and "Little interest or pleasure in doing things." Although the Revised PHQ-9 has not been published and validated, the original PHQ-9 has been used in the autism population (Alshahrani et al., 2021; Arnold et al., 2020; Cassidy et al., 2021; Pilunthanakul et al., 2021), and the scale shows good convergent validity among the autistic population against the Warwick-Edinburgh Mental Well-being Scale and the World Health Organization Quality of Life—BREF psychological well-being domain (Arnold et al., 2020). The PHQ-9 also demonstrates excellent reliability for the total score (α = 0.91), and good internal consistency (Cronbach α values above 0.8 in several studies) among autistic samples (Arnold et al., 2020; Pilunthanakul et al., 2021).

Depression was also measured using the Overall Depression Severity and Impairment Scale (ODSIS; Bentley et al., 2014), a five-item instrument measuring depression-related severity and impairment participants have experienced over the past week. Although to our knowledge the ODSIS has not been used in the autism population, the measure has demonstrated excellent internal consistency, convergent and discriminant validity, and cross-cultural validity in non-clinical populations and other clinical

populations, such as major depressive disorder, panic disorder, social anxiety disorder, and obsessive-compulsive disorder (Ito et al., 2015). We included both the Revised PHQ-9 and the ODSIS in order to collect both symptom and impairment measures associated with depressed mood.

Importantly, among the focal measures for this project, all stated instructions specified a similar time frame for participants to reflect on in their responses: Survey instructions directed participants to think about the past 1-2 weeks, so that their window of ratings would not overlap across the three survey modules.

Table 5

Measures Organized by Construct

Construct	Measures
Autism Traits	Comprehensive Autistic Trait Inventory (CATI; English et al., 2021)
Social Motivation	Anticipatory and Consummatory Interpersonal Pleasure Scale (ACIPS; Gooding & Pflum, 2014)
Social Access	PROMIS Satisfaction with Participation in Discretionary Social Activities (Cella et al., 2010)
	PROMIS Emotional Support items (Cella et al., 2010)
	PROMIS Global Items (Hays et al., 2009)
Loneliness	Scale of Loneliness (SoLo; Williams et al., 2022)

Statistical Analyses

To test our hypothesis, we planned to test model fit of a moderated mediation model, all in which social motivation moderates the relationship between social access and loneliness, with loneliness mediating the relationship between social access and depression. A moderated mediation model evaluates the influence of moderator W on the mediated (M) relationship between X and Y (see Figure 2). Put another way, the model assesses whether the relationship from X→ M→Y depends on W (Washburn, 2017). The moderation can occur at any of the paths in the mediation model (i.e., paths a, b, c in Figure 2, or any combination of the three; Washburn, 2017).

We used Bayesian analyses with post-hoc testing against Regions of Practical Equivalence (ROPE; Kruschke & Liddell, 2017) to test our hypothesized moderated mediation model (Figure 3 panel B) against the baseline model (Figure 3 panel A) to assess which was better able to prospectively predict depressive symptoms. In the baseline model, Time 2 loneliness mediates the relationship between Time 1 social access and Time 3 depressive symptoms (hereafter "depression" for ease of reading), with no inclusion of social motivation. Our hypothesized model adds on the additional moderation variable of social motivation at Time 1 as a moderator of the relationship between Time 1 social access and Time 2 loneliness.

Figure 2

Basic Moderated Mediation Model (Washburn, 2017)

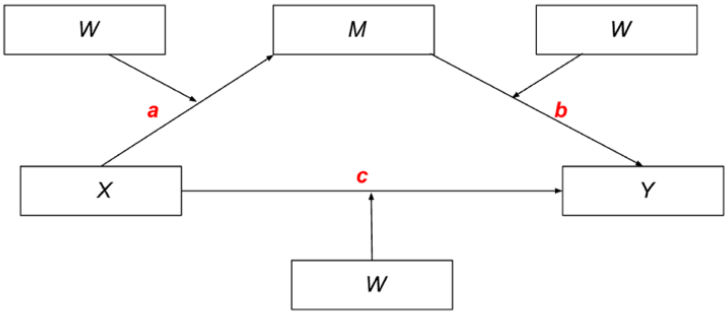
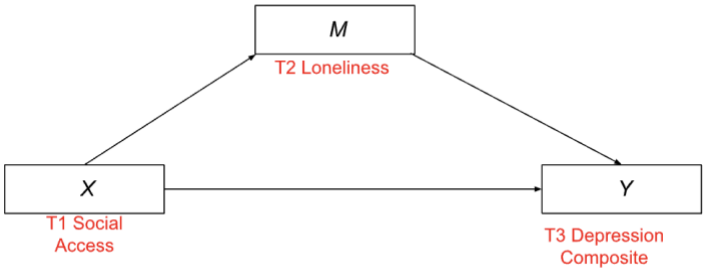


Figure 3

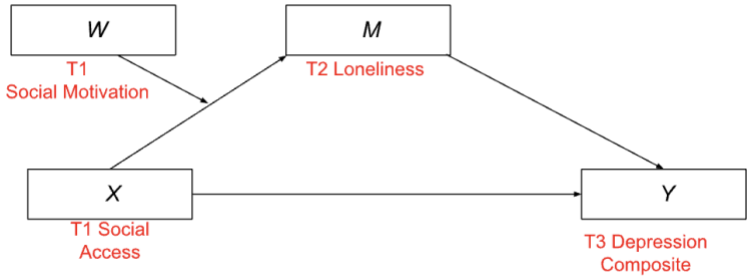
Baseline Model and Hypothesized Moderated Mediation Model

A. Baseline Model



Note. Baseline model showing Time 2 loneliness (measured by the Scale of Loneliness) mediating the relationship between Time 1 social access (measured by the PROMIS Satisfaction in Discretionary Activities, Emotional Support, Loneliness, and Global items) and Time 3 depression (measured by the PHQ-9 and ODSIS).

B. Hypothesized Moderated Mediation Model



Note. Hypothesized model showing an interaction between Time 1 social access (measured by the PROMIS Satisfaction in Discretionary Activities, Emotional Support, Loneliness, and Global items) and Time 1 social motivation (measured by the ACIPS) predicting Time 2 loneliness (measured by the SoLo), and Time 2 loneliness subsequently predicting Time 3 depression (measured by the PHQ-9 and ODSIS).

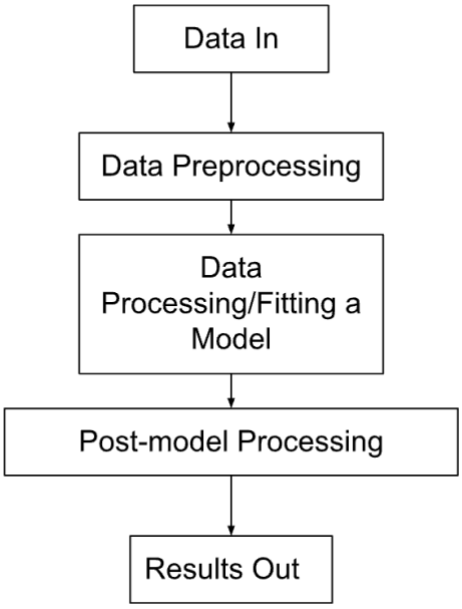
The general procedure we used to test the above models included data preprocessing, fitting the models, and post-model processing, as represented in Figure 4. We first preprocessed the data, which included addressing missing data, assessing the fit of the measures we used for our four variables (i.e., social motivation, social access, loneliness, and depression), and calculating scores on general factors (G factors; i.e., latent variables that influence all indicators within a bifactor model [see Toland et al., 2017 for more information]) for the constructs of interest (see Appendix A, Figure A1). Next, we processed the data by fitting these G factors to the baseline model and

hypothesized model (Figure 3) within the Bayesian framework (see Appendix A Figure A2 for an overview of this data processing stage). Finally, in post-processing we further evaluated the final model by comparing the posterior values derived from the final model to a set of null values to assess their practical significance and determine support for the final model against the various models tested.

Note that Appendix A provides more in-depth background on Bayesian analyses and the specific theory behind our analytic strategy and decisions at each step of the procedure outlined in Figure 4. We summarize the main points below; see Appendix A for additional details on analytical theory and methods.

Figure 4

Overview of Data Analysis



Data Preprocessing

We utilized multiple imputation models to fill in the missing data (Enders, 2023). This strategy generates replacement values ("imputations") for the missing data, as repeated numerous times and based on the statistical characteristics of the data (e.g., the associations among the distributions of variables in the data set) (Li et al., 2015). To complete the multiple imputation models in R, we used the missForest package, which uses random forest imputation (Stekhoven & Bühlmann, 2012) to compute these multiple guesses (Stekhoven, 2022). We completed the multiple imputations ten times to yield ten different imputed datasets.

Next, we tested the fit of the different measures that operationalized our four variables of interest (social motivation, social access, loneliness, and depression). A combination of the confirmatory fit index (CFI), root mean square error of approximation (RMSEA), Tucker-Lewis indices (TLI), and standardized root mean square residual (SRMR) were used to determine the global fit of the measures. "Good fit" was operationalized as a CFI and TLI > 0.97 (Cai et al., 2021), RMSEA < 0.089, and SRMR < 0.05 (Maydeu-Olivares & Joe, 2014). Additionally, item misfit at the local level was evaluated by examining standardized residuals, such that $|r_{res}| > 0.1$ (Maydeu-Olivares, 2014), and a Q_3 value above the empirical cutoff value (Christensen et al., 2017; Yen, 1984) indicated the need to delete an item from the model.

We next used a plausible value imputation algorithm to compute the latent traits (i.e., "G factors") of social access, depression, social motivation, and loneliness. This algorithm mapped each measure onto the same, standardized scale (a Z-score with M=0, SD=1), and weighed each item differently according to how well the item assessed the G factor. The plausible value imputation was able to calculate the G factors for the four

latent variables by estimating the factor score five different times for each model (in each imputed data set, for a total of 50 draws per construct), with each estimation providing a small margin of additional error to account for the fact that our estimates are not error-free and that the latent variable is not observed.

Fitting the Models

In the Bayesian approach, candidate explanations of new data have prior credibility of being the best explanation for the new data (Kruschke & Liddell, 2017). We subsequently shift the credibility toward the candidate explanation that best accounts for the new data, and our "best estimate" explanation then becomes a weighted combination of prior beliefs and observed data (Kruschke & Liddell, 2017). In more technical terms, the Bayesian approach combines two probability density functions (PDFs); the PDF from before collecting data (known as a "prior" belief) and the PDF from the data collected (Koch & Koch, 1990). Combining the prior PDF with the PDF of the data leads to an updated belief about the data, which is referred to as a "posterior" distribution or "posterior belief" (Koch & Koch, 1990). The posterior distribution then represents the most credible values and range of reasonable parameter values (Kruschke & Liddell, 2017). If we were to run a study again, this posterior would then become the new prior, and the process would repeat, leading to a continuous cycle of posterior beliefs becoming updated prior beliefs to test with more data (Kruschke & Liddell, 2017). The goal of a Bayesian analysis is thus to generate a posterior distribution that is informed by our prior beliefs and the observed data.

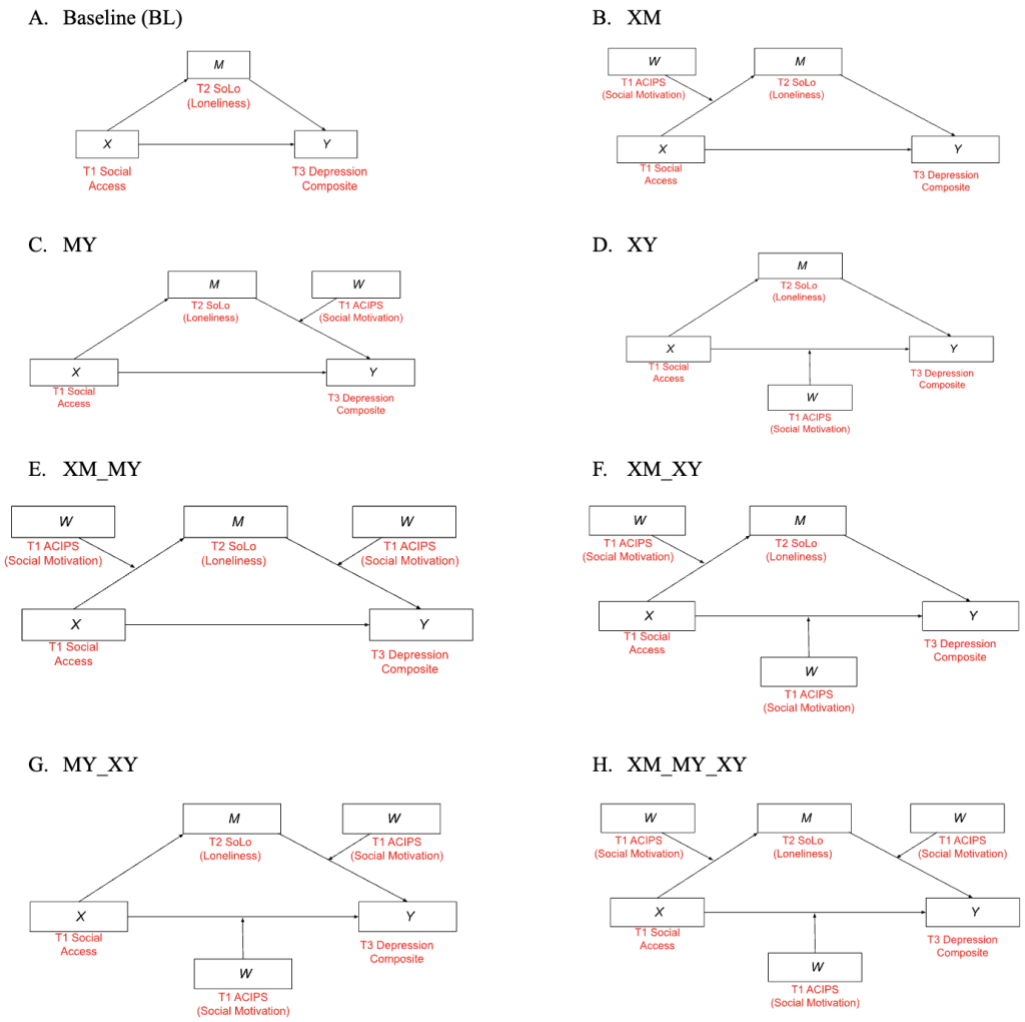
We chose to analyze our data within a Bayesian framework because the approach was well suited to test multiple parameters with non-normal likelihoods, and because, as

a longitudinal replication of a cross-sectional study, we had existing prior explanations for our data.

Importantly, although we were interested in assessing whether our proposed moderated mediation model predicted depression better than the baseline model (Figure 3, Panels A vs. B), we also assessed social motivation as a moderator for every potential path. This led to eight total models to compare (see Figure 5) – the Baseline unmoderated model, our hypothesized moderated mediation model, and six additional moderation mediation models to explore the moderator's potential effect on each path. Testing all eight models, versus just the Baseline and Hypothesized, allowed us to compute an inclusion Bayes factor ($BF_{\text{inclusion}}$) for each specific part of the model (i.e., a specific coefficient of the model) as averaged across all models.

Figure 5

The Eight Models Tested in Bayesian Framework



Our first step in comparing the eight models was to determine priors of the regression slopes of each model, using the findings from Han et al. (2019) to inform these priors.

We then entered the eight models and their corresponding priors into R and computed the Bayes factors for each model. Per convention, we classified the strength of

the Bayes factors (*BF*) as 3<*BF*<10 indicating moderate evidence for a given model, 10<*BF*<30 indicating strong evidence for a given model, and *BF*>30 indicating very strong evidence for a given model (Wagenmakers et al., 2011). We then selected the model with the highest BF_{model} and highest inclusion Bayes factor as our final model.

Post-Model Processing

We next determined a range of null values known as the region of practical equivalence (ROPE) (Kruschke & Liddell, 2017), which defines a region of values that are practically equivalent to the null values and thus translate to a zero effect size (Kruschke & Liddell, 2017). Posterior values from the final model then were compared to this ROPE range to determine if they fell inside the ROPE (i.e., evidence against our final model) or outside the ROPE (evidence in support of our final model). The Bayesian ROPE analysis calculates several important values, including the median of the distribution (Mdn), which indicates the direction of the relationship (i.e., positive or negative) between the variables in the path being examined. The P_d, or probability of direction, provides the probability of the sign of the relationship (i.e., the P_d indicates the probability that the sign of the Mdn observed is true). If the P_d>0.975, this is evidence of statistical significance (as a P_d>0.975 is equivalent to two-tailed p<0.05) (Makowski et al., 2019).

The Bayesian ROPE analysis also generates the Bayes Factor ROPE (BF_{ROPE}), which compares the evidence for the null hypothesis that the parameter of interest (i.e., the posteriors generated in the data processing phase of analysis) is *inside* the ROPE (i.e., H_0: Parameter X is practically equivalent to zero; BF_{ROPE}<$^1/_3$ provides evidence for H_0) to the evidence for the alternative hypothesis that the same parameter is *outside* the ROPE

(H_A: Parameter X is large enough to be practically significant; BF_{ROPE}>3 provides evidence for H_A) (Makowski et al., 2019). Taken together, then, the P_d is used to assess statistical significance, while the BF_{ROPE} is used to assess practical significance.

Finally, we tested our exploratory question regarding the longitudinal relationship between low social motivation and depression through a Bayesian framework that used a t-regression. Specifically, we ran 50 imputations (10 datasets x 5 plausible values per dataset) and set the priors the same as in all other models. We first predicted Time 2 depression from age (Z-score), sex, gender minority status (cisgender or vs. other), T1 ACIPS score (plausible value), and T1 depression score (plausible value). We then repeated this same process for predicting Time 3 depression instead of Time 2 depression. Importantly, these analyses allowed us to hold T1 depression scores constant, in order to comment on T1 social motivation as a more independent predictor of T2 or T3 depression.

Chapter 3

Results

Data Preprocessing

Filling in Missing Data: Multiple Imputation Models

Overall, there were zero scores missing in Wave 1, 44 SoLo scores (14.5%) missing in Wave 2, and 65 depression scores (21.5%) missing in Wave 3. We used the multiple imputation modeling to generate replacement values for these missing data, as described in the Methods and in Appendix A.

Testing Fit of Measures

Table 6 summarizes the final fit index values for each construct (i.e., social access, loneliness, social motivation, and depression). The items across scales that we used to define social access (see Table 4) collectively demonstrated good CFI, RMSEA, SRMR, and TLI values of 0.985, 0.055, 0.017, and 0.979 respectively. No items were removed.

For the novel loneliness measure (SoLo), items 2 (“I am happy with how connected I am to the people in my community”), 6 (“I often feel upset that I am left out of social things going on around me”), and 7 (“I spend a lot of time wishing I were with other people”) were cut, as the CFI improved from 0.986 to 0.994, the RMSEA improved from 0.064 to 0.046, the SRMR improved from 0.051 to 0.044, and the TLI improved from 0.979 to 0.990 following removal of these items.

For social motivation (ACIPS), item 9 (“I enjoy watching films about friendships or relationships with my friends”) was removed due to the data misfitting. Social motivation then demonstrated good CFI, RMSEA, SRMR, and TLI values (0.987, 0.054, 0.044, and 0.984 respectively).

Lastly, no depression items required removal, as the CFI, RMSEA, SRMR, and TLI demonstrated good fit (0.985, 0.067, 0.011, and 0.975 respectively).

Table 6

Final Fit Index Values for Constructs of Interest

Variable	CFI	RMSEA	SRMR	TLI
Social Access (items listed in Table 4)	0.985 (good)	0.055 (good)	0.017 (good)	0.979 (good)
Loneliness (SoLo)	0.994 (good)	0.046 (good)	0.044 (good)	0.990 (good)
Social Motivation (ACIPS)	0.987 (good)	0.054 (good)	0.044 (good)	0.984 (good)
Depression (PHQ-9 and ODSIS)	0.985 (good)	0.067 (good)	0.011 (good)	0.975 (good)

Data Processing

Selection of Priors

We derived the width of the normal distributions for regression slopes based on the results from Han et al. (2019). The priors for the parameters are listed in Table 7.

Table 7

Priors Based on Han et al. (2019)

Parameter	Prior Distribution	Distribution Parameters
B0 [Intercept]	Student t distribution	df = 3, mu = 0, sigma = 2.5
B1 [Slopes (main effects)]	Normal distribution	(mu = 0, sigma = sqrt(2)/2)
B2 [Slopes (interactions)]	Normal distribution	(mu = 0, sigma = sqrt(2)/4)
Nu [t degrees of freedom parameters]	Gamma distribution	(alpha = 2, beta = 0.1) with a lower bound of 1
Sigma [Residual standard deviation parameters]	Exponential distribution	(lambda = 1)

Note. Based on Han et al. (2019), prior probabilities for all models were set to 0.125.

We were able to justify these priors based on Han et al. (2019) as follows:

- For the relationship between loneliness and depression in Han et al. (2019), the Adjusted $R^2 = 0.33$:
    - $R^2 = 0.33$ (partial $r \approx 0.5744$, so $beta_{Std} \approx 0.7018$ based on the formula [$r_p = beta_{Std}/sqrt(beta_{Std}^2 + 1)$])
- We then set the prior to be Normal (0, sqrt (2)/2) for all of the main effects.
    - We set the prior this way in order to make the normal values not uncommon for the regression slopes (but also to allow for bigger effects with a not insignificant probability).

- Since interactions are typically much smaller in magnitude, we set the prior on those to be Normal (0, sqrt (2)/4) to allow for a SD of half the size of the main effect.

Selection of Final Model

The results of the Bayesian analysis are shown in Table 8 and Table 9. Our results suggested that the baseline model (BL) yielded the highest Bayes factor. For the BL model – which again included no moderation by social motivation at any path – the BF_{model} was 4.261 (moderate evidence). Notably, the model with the second highest individual Bayes factor was our hypothesized model, XM (BF_{model}=2.69), in which social motivation moderated the path between social access and loneliness; direct comparison of this model with the baseline model found weak and inconclusive evidence favoring the baseline model (*BF*/BL=0.746).

The $BF_{inclusion}$ for the moderation of the XM path (i.e., social motivation moderating the path between social access and loneliness) was equal to 0.762 (inconclusive evidence slightly favoring the lack of an effect), which indicates that the (posterior) odds of the XM path being moderated were 0.762 times as high (i.e., 1.31 times *lower*) than they were *before* observing the data. Alternatively, inclusion Bayes factors for both the MY path (i.e., loneliness to depression) ($BF_{inclusion}$ = 0.232) and the XY path (i.e., social access to depression) ($BF_{inclusion}$ = 0.232) both demonstrated conclusive evidence *against* moderation effects when averaged across all tested models (Table 9).

In sum, the results of the Bayes Factor analysis indicated that of all eight models, the baseline model – in which the path between T1 social access and T3 depression was mediated by T2 loneliness, with no moderation by social motivation of any path – was the best at predicting depressive symptoms in our short-term longitudinal data (i.e., the BL model yielded the largest Bayes factors in comparison to the other seven models). This was supported by inclusion Bayes factors that conclusively rejected the moderation of both the MY and XY paths when averaged across all models. Although the evidence *against* the hypothesized moderation of the XM path was not conclusive using either the inclusion Bayes factor or the direct model comparison (Wagenmakers et al., 2011), we nevertheless found insufficient evidence to suggest an interaction between T1 social motivation and T1 social access in predicting T2 loneliness in the current analysis. Thus, the baseline model, in which no interaction terms were included, was retained as the final model in all future analyses.

Table 8

Bayes Factor Results for All Individual Models

Model	**Prior Probability**	**Posterior Probability**	BF_{model}	**1/*BF***	***BF*/BL**
BL	0.125	0.378	4.261	0.235	1.0000
XM	0.125	0.278	2.694	0.371	0.7464
MY	0.125	0.087	0.664	1.505	0.2161
XY	0.125	0.085	0.649	1.540	0.2196
XM_XY	0.125	0.071	0.531	1.885	0.1638
XM_MY	0.125	0.069	0.518	1.930	0.1612

Model	Prior Probability	Posterior Probability	BF_{model}	1/*BF*	*BF*/BL
MY_XY	0.125	0.018	0.127	7.904	0.0454
XM_MY_XY	0.125	0.015	0.107	9.352	0.0339

Note. Compare to Figure 5 for schematic definition of the various models.

Table 9

Inclusion Bayes Factors for All Three Moderated Paths (Averaged Across All Models)

Moderated Path	Prior Probability	Posterior Probability	$BF_{inclusion}$	$BF_{exclusion}$
XM	0.5	0.423	0.762	1.31
MY	0.5	0.188	0.232	4.31
XY	0.5	0.188	0.232	4.32

Note. XM=Path between T1 social access and T2 loneliness; MY=Path between T2 loneliness and T3 depression; XY=Path between T1 social access and T3 depression

Post-Model Processing

Results of the ROPE Analysis

As shown in Figure 6, **A1** represents the path from T1 social access (X) to T2 loneliness (M). Based on the results displayed in Table 10, the median of the distribution (Mdn) value indicates that the relationship between T1 social access (X) and T2 loneliness (M) is -0.564. Thus, lower levels of social access at T1 correlated with higher levels of loneliness at T2. The P_d, or probability of direction, indicates the probability of the sign of this relationship. Because P_d for A1 is >0.9999, there is essentially 100% certainty that the A1 path is negative (i.e., the relationship between T1 social access and

T2 loneliness is negative). The fact that P_d>0.975 further indicates that this path is statistically significant. The ROPE value was set to -0.1 to 0.1 per standards described in Methods. The BF_{ROPE} for A1 was $6.71*10^8$, indicating that there is very strong evidence that the parameters for A1 fell outside of the ROPE and that the path is practically significant. In total, then, the ROPE analysis indicates that lower levels of Time 1 social access significantly predicted higher levels of Time 2 loneliness.

B2 shows the path from T1 social motivation to T3 depression. The median of distribution value for this path was -0.094 with a P_d of 0.8991, indicating a high probability that this relationship is negative. As such, lower social motivation at T1 was more likely to be correlated with higher depression at T3. ROPE values were set to the standard -0.1 to 0.1. The BF_{ROPE} was 0.112, which provided moderate evidence that the B2 values fell inside the ROPE compared to outside the ROPE (i.e., $^1/_{10}<BF_{ROPE}<^1/_3$). Taken together, the ROPE analysis suggested that T1 social motivation did not significantly predict T3 depression (i.e., P_d <0.975 and $BF_{ROPE}<^1/_3$).

A2 represents the relationship between T1 social motivation and T2 loneliness for the XM model. The Mdn value for A2 was 0.409, and the P_d was >0.9999, together indicating that there is near 100% certainty that the relationship between T1 social motivation and T2 loneliness is positive. Thus, in our sample, higher levels of social motivation at T1 correlated with higher levels of loneliness at T2, with the P_d indicating that this relationship is statistically significant. The ROPE value was set to -0.1 to 0.1. The BF_{ROPE} was $7.74*10^5$, suggesting very strong evidence that the A2 posterior parameters fell outside the ROPE and reached practical significance. Based on these data,

we were able to reject the interval null for A2. Thus, higher T1 social motivation significantly predicted higher T2 loneliness.

B1 represents the path from T2 loneliness to T3 depression. The Mdn value for B1 was 0.267 with a P_d of 0.9996, suggesting a high probability that this relationship was positive (i.e., the lonelier participants were at T2, the more depressed they were at T3). The standard -0.1 to 0.1 ROPE was again used for this part of the model. The BF_{ROPE} was 6.97, indicating moderate evidence that the parameters for B1 were located outside the ROPE. Taken together, the P_d and the BF_{ROPE} indicate that this path reached statistical and practical significance. Thus, higher levels of T2 loneliness significantly predicted higher levels of T3 depression.

The **Direct Effect** represents the direct path between T1 social access and T3 depression. The Mdn value for this relationship was -0.255 with a P_d of 0.9981, suggesting a high probability that the relationship is negative. Thus, the more social access individuals had at T1, the less depressed they were at T3. We used the standard ROPE values (-0.1 to 0.1). The BF_{ROPE} was 2.89, suggesting weak evidence that the direct effect posteriors fell outside the ROPE. Overall, then, the direct effect from T1 social access to T3 depression was statistically significant but not practically significant (i.e., P_d>0.975 but the BF_{ROPE} did not reach practical significance with the ROPE test).

Lastly, the **Indirect Effect** represents the path from T1 social access to T3 depression through the mediator of T2 loneliness. The Mdn value for this path was -0.149, with a P_d of 0.9996 (suggesting a high probability that the path was negative). Because the indirect effect was expected to be smaller in magnitude than the direct effects or main effects, we used a narrower ROPE of -0.05 to 0.05 in order to recognize

smaller effect sizes as practically significant. The BF_{ROPE} was 20.4, suggesting strong evidence that the indirect posterior parameters fell outside the ROPE compared to within the ROPE. Taken together, the indirect effect from lower T1 social access to higher T3 depression through higher T2 loneliness was statistically (P_d>0.975) and practically significant (BF_{ROPE}>3).

The Total Effect represents the combined direct effect from X to Y and the indirect effect from X to Y through M (Agler et al., 2017). The Mdn value for the total effect was -0.405 with a P_d of >0.9999, suggesting a high probability that the path was negative. Thus, the total relationship between Time 1 social access and Time 3 depression was negative, such that lower levels of social access at Time 1 corresponded with higher levels of depression at Time 3, when combining direct evidence and that through the mediation path. The standard -0.1 to 0.1 ROPE was used. The BF_{ROPE} was $1.24*10^4$, indicating very strong evidence that the parameters for the total effect were located outside the ROPE. Thus, the total effect (i.e., combining the direct effect and the indirect effect of the model) was statistically (P_d>0.975) and practically significant (BF_{ROPE}>3).

Lastly, the **proportion mediated** indicates the ratio of the indirect effect to the total effect, providing an estimate of the extent to which the total effect is accounted for by the pathway through the mediated variable (Ananth, 2019). In other words, the proportion mediated speaks to how much of the total effect operates through the mediator (Ananth, 2019). The proportion mediated suggested a Mdn value of 0.370, suggesting that the proportion of the BL model that consisted of the mediation effect was 37% with a range of 11% to 69%. For this path, we set the ROPE to be from −infinity to 0.5 so that

we could test the probability that the effect of Time 1 social access on Time 3 depression l was less than 50% mediated (i.e., the ROPE indicates up to 50% mediated). The P_{ROPE}, which represents the probability that the posterior parameters fell within the ROPE, was 0.7978, suggesting a high (79.8%) chance that the posterior parameters fell within negative-infinity to 0.5, and thus that loneliness mediated less than 50% of the effect of social access on depression in the BL model.

In sum, combining these values shown in Table 10 suggests that A1, A2, and B1 performed well in the ROPE analysis (i.e., we were able to reject the interval null). Thus, we can be more certain that the paths from T1 social access to T2 loneliness (A1), T1 social motivation to T2 loneliness (A2), T2 loneliness to T3 depression (B1), and the indirect effect from T1 social access to T3 depression were significant. However, the BF_{ROPE} indicates that the path from T1 social motivation to T3 depression (B2) was not significant. The direct effect from T1 social access to T3 depression was close to being practically significant, as there was over a 95% chance the effect exceeded the ROPE. Lastly, the fact that the proportion mediated was less than 50% (i.e., the proportion mediated was 37%) indicates that, although the indirect effect was significant, this was only a partial mediation. In other words, social access may be influencing depression through other mechanisms (i.e., mediators) in addition to loneliness.

Figure 6

Conceptual Diagram of Components of BL Tested Against the ROPE

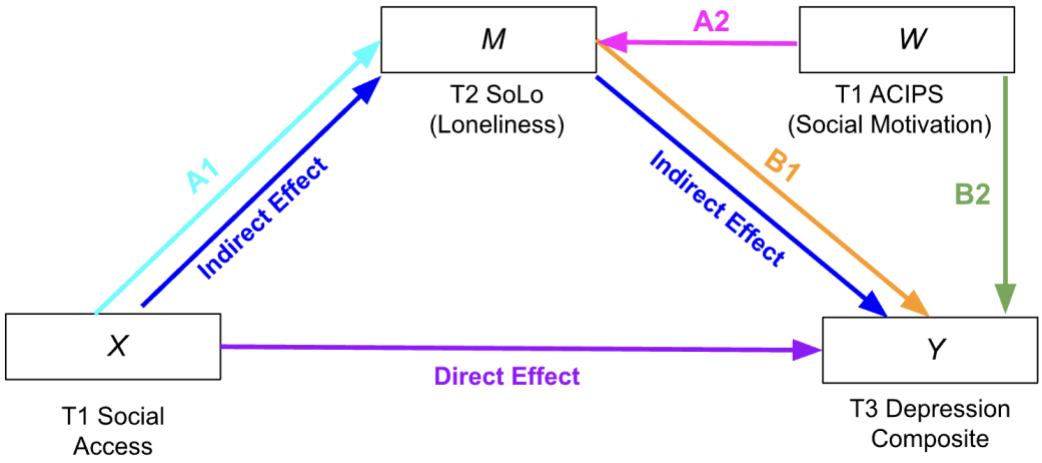


Table 10

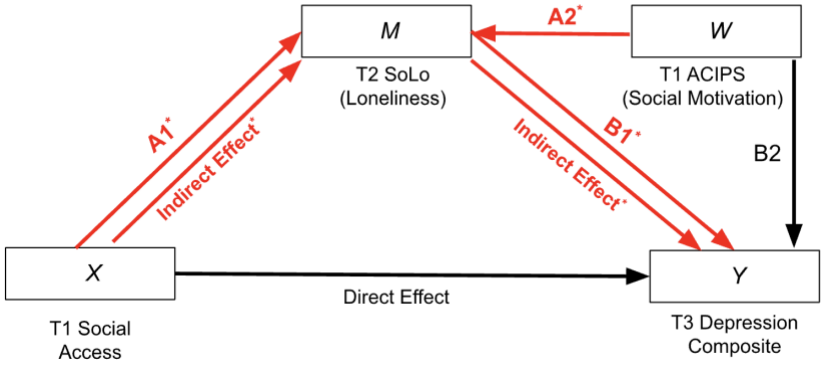
Corresponding Results of the ROPE Analysis

Section of BL	Mdn	P_d	ROPE	BF_{ROPE}
A1	-0.563	>0.9999	{-0.1,0.1}	6.71e+08
A2	0.409	>0.9999	{-0.1,0.1}	7.74e+05
B1	0.267	0.9996	{-0.1,0.1}	6.97e+00
B2	-0.094	0.8991	{-0.1,0.1}	1.12e-01
Direct Effect	-0.255	0.9981	{-0.1,0.1}	2.89e+00
Indirect Effect	-0.149	0.9996	{-0.05,0.05}	2.04e+01
Total Effect	-0.405	>0.9999	{-0.1,0.1)	1.24e+04
Proportion Mediated	0.370	0.9996	{-inf,0.5}	*NA*

Taken together, the results suggested that the BL model was the best of the eight tested models in being able to predict depressive symptoms at Time 3. The inclusion Bayes factors for the moderation of the XM, MY, and XY paths all indicated weak evidence for moderation of those paths, further supporting the BL model as the best model. Examining the BL model further through the ROPE analysis revealed that we can be more confident in the significance of certain BL paths (see Figure 7), including the effect of Time 1 social access on Time 2 loneliness (A1), the effect of Time 1 social motivation on Time 2 loneliness (A2), the effect of Time 2 loneliness on Time 3 depression (B1), and the indirect effect of Time 1 social access on Time 3 depression through the mediator of Time 2 loneliness. The ROPE analysis also demonstrated that we can be less confident that the effects of Time 1 social motivation on Time 3 depression (B2) and the direct effect of Time 1 social access on Time 3 depression (direct effect) were significant.

Figure 7

Significant Relationships in the Baseline Model (red)



Post-Hoc Exploratory Analyses

We were also interested in testing our exploratory question regarding the longitudinal relationship between low social motivation and outcome depression when controlling for initial depressive symptoms. Specifically, we aimed to determine whether low social motivation still correlated with higher depression (as it did in Han et al.'s 2019 findings) in our now longitudinal data, or if low social motivation was associated with lower depressive symptoms due to the lack of a discrepancy between social motivation and social access (i.e., in support of Smith & White's 2020 findings).

Starting with the model predicting Time 2 depression, the results suggested that the Mdn for the path from T1 social motivation to T2 depression (after controlling for age, sex, gender minority status, and T1 depression scores) was -0.047 with a P_d of 0.798, together indicating a high probability that this relationship was negative, though not reaching statistical significance (i.e., P_d<0.975). Thus, lower levels of T1 social motivation predicted higher levels of T2 depression, even controlling for T1 depression

scores. ROPE values were set to the standard of -0.1 to 0.1. The BF_{ROPE} for the T1 social motivation effect was 0.028, suggesting weak evidence that the T1 social motivation parameters fell outside the ROPE. Thus, the Bayes Factor suggests that this model was practically insignificant (i.e., $BF_{ROPE}<1/3$).

Findings for the T3 depression model were similar: The Mdn for the path from T1 social motivation to T3 depression (after controlling for age, sex, gender minority status, and T1 depression scores) was -0.025 with a P_d of 0.663 (statistically non-significant), suggesting a 66.3% chance that the relationship was negative. As such, lower levels of T1 social motivation predicted higher levels of T3 depression even when accounting for T1 depression. ROPE values were again set to the standard of -0.1 to 0.1. The BF_{ROPE} was 0.019, indicating weak evidence that the T1 social motivation parameters fell outside the ROPE. Overall, then, similar to the T2 Depression Model, the Bayes Factor indicated that the T3 Depression Model was practically insignificant (i.e., $BF_{ROPE}<1/3$).

In addition, because the baseline model was the best fitting model in the tests of our primary hypothesis, we also conducted post-hoc exploratory analyses on the relationship between T1 social access and T2 and T3 depression scores after controlling for age (Z-score), sex, gender minority status (cisgender or vs. other), and T1 depression score (plausible value). Similar to the previous post-hoc analysis, we tested these exploratory questions through a Bayesian framework that used a t-regression. We ran 50 imputations (10 datasets x 5 plausible values per dataset) and set the priors the same as in all other models. The Mdn for the path from T1 social access to T3 depression (after controlling for age, sex, gender minority status, and T1 depression scores) was -0.194 with a P_d of 0.998 (Mdn= -0.157 with a P_d of 0.994 when predicting T2 depression),

indicating a very high likelihood that the relationship between T1 social access and T2 or T3 depression was negative. Thus, lower levels of social access at T1 corresponded with higher levels of subsequent depression. ROPE values were once again set to the standard of -0.1 to 0.1. The BF_{ROPE} was 1.426 for T3 depression (0.546 for T2 depression), suggesting weak and inconclusive evidence that the T1 social access values fell outside the ROPE. In total, then, the Bayes Factors indicated that the paths from T1 social access to T2 or T3 depression, after controlling for demographic variables and T1 depression, were statistically (P_d>0.975) but not practically significant (i.e., the Bayes factors for each path were nonzero but did not quite reach practical significance with the ROPE test).

Chapter 4

Discussion

Autistic adults experience significantly higher rates of lifetime depression that correlates with worse outcomes (e.g., suicidality). Better understanding of the etiology of depression in this population could translate into treatment targets, and ideally from there, to more efficient, effective interventions. The current study aimed to test whether a discrepancy between high social motivation and low access to meaningful social opportunity interacts to prospectively predict loneliness and subsequent depression among autistic adults. We further investigated the association between initial low social motivation and depressive symptoms at outcome, in order to contribute to the open question of whether low social motivation is associated with increased loneliness and depression, in line with previous cross-sectional findings (Han et al., 2019), or in fact *protects* against loneliness and depression (Smith & White, 2020).

In our short-term longitudinal data collected through the SPARK online registry, we found little evidence of moderation by social motivation in our models of social access at Time 1 predicting depressive symptoms at Time 3 via Time 2 loneliness as a mediator. Instead, our data supported the baseline model of loneliness mediating the relationship between social access and depression, with no moderating effects of social motivation at any path of the model. Taken together then, we did not find sufficient evidence to suggest that an interaction between high social motivation and low social access prospectively predicts loneliness and depression. Further, our exploratory analyses on low social motivation at Time 1 fell short of significance but appeared to support Han

et al.'s findings that low social motivation does not protect against subsequent depression.

Primary Findings

To test our main hypothesis, we assessed whether high social motivation moderated the longitudinal relationship between low social access and loneliness, and whether this interaction predicted subsequent depression. Though our hypothesis was not supported (in that the baseline, unmoderated model of loneliness mediating the relationship between social access and depression was the best fitting model), our proposed moderated mediation model showed the next best fit of 8 tested models, second only to the baseline. Our Bayesian approach allowed us to average the effect of each path across all eight tested models; the moderated path from Time 1 social access to Time 2 loneliness was non-significant across the aggregate data, providing further evidence that an unmoderated path from social access to loneliness was favored. Using Region of Practical Equivalence (ROPE) analyses to examine the individual paths within the baseline model indicated that:

- Higher levels of **social motivation** at Time 1 significantly predicted higher levels of **loneliness** at Time 2
- Lower levels of **social access** at Time 1 significantly predicted higher levels of **loneliness** at Time 2
- Higher levels of **loneliness** at Time 2 significantly predicted higher levels of **depression** at Time 3
- Lower levels of **social access** at Time 1 significantly predicted higher levels of **depression** at Time 3 via the mediator of **loneliness** at Time 2

In essence, the individual "pieces" that led to our hypothesis were each supported independently, in that both high social motivation and low social access prospectively predicted loneliness, which prospectively predicted depressive symptoms. However, the hypothesized *interaction* between these pieces at Time 1 had only "weak and inconclusive" support in this study.

Our post-hoc analyses suggested that lower levels of Time 1 social access were (statistically) significantly associated with higher levels of Time 2 and Time 3 depression even when controlling for age, sex, gender minority status, and Time 1 depression. That this association failed to reach practical significance may have been due to lack of inclusion of the loneliness mediator in these post-hoc analyses: In our primary model testing (without these covariates controlled), the direct effect from Time 1 social access to Time 3 depression was statistically but not practically significant, but the indirect effect through loneliness was both statistically and practically significant. Not surprisingly, the proportion mediated suggested that other factors in addition to loneliness are also likely mediating the relationship between social access and depression. We speculate on some of these under Future Directions below.

Low Initial Social Motivation and Outcome Depressive Symptoms

We also tested the longitudinal relationship between initial low social motivation (that was not tied to existing depression) and depressive symptoms at outcome. These analyses were deemed "exploratory" insofar as either direction of hypothesis could be supported by previous literature: Cross-sectional data indicated that low social motivation was still associated with higher depression symptoms in autistic adults (Han et al., 2019; Ee et al., 2019), however Smith and White's 2020 social motivation model suggested that

low social motivation would have a protective effect against depression in autism. Per our ROPE analyses, lower levels of social motivation at Time 1 were associated with higher levels of depression at Time 3, though these analyses fell short of statistical significance. Our post-hoc analyses similarly suggested that lower levels of social motivation at Time 1 corresponded with higher levels of depression at Time 2 and Time 3 even when controlling for Time 1 depressive symptoms (as well as demographic covariates), but these relationships did not reach statistical significance either. Nonetheless, the direction of the relationship between these constructs was more in line with Han et al., 2019, in that low social motivation did not appear to protect against depression.

Measurement Challenges

We considered several explanations for our hypothesized moderation mediation model failing to reach significance. First, we may have not accurately operationalized and/or measured the constructs of social access and social motivation. Further, our short-term longitudinal approach may have failed to capture the social constructs early enough in time to avoid "contamination" by previous depressive episodes or cycles of perceived social failure. We will discuss these measurement challenges, both psychometric and chronological, in turn.

Psychometric Challenges

As we noted in both the Introduction and Measures sections, social access can be defined in various ways (e.g., number and quality of friendships; amount of social opportunity in terms of frequency counts; perceived social support or satisfaction). We defined social access as *both* having opportunities for social interactions *and* having interactions or relationships perceived as meaningful, as characterized by feeling socially

connected. Thus, we combined two facets of social engagement into one definition, and it is possible that one or the other (i.e., sheer opportunity to interact or "meaningfulness" of social interactions) is the more relevant construct in terms of interacting with social motivation to predict loneliness and depression. In other words, it is possible that the key component to satiating social motivation is the frequency of social opportunity, regardless of whether one derives meaning from those social interactions, or conversely, that the only salient construct here is the sense of social belonging or deriving meaning from social interactions, regardless of frequency. Perhaps by including both, we measured one or both constructs insufficiently, or introduced too broad a range of endorsed scores, diluted by the presence of a less salient construct and ultimately obscuring our ability to observe significant associations.

Furthermore, while the first part of our definition of social access was more quantifiable (i.e., frequency of social participation), the second part of our definition (i.e., the level of meaning and belonging experienced in these social interactions) may have been less clearly quantified in our self-report survey. Importantly, it may be that we did not appropriately define and measure "meaning" in social interactions or relationships via our social meaning items (e.g., "I have someone who makes me feel appreciated"; "I have someone who will listen to me when I need to talk"). We may have failed to adequately capture the degree of experienced social "meaning" with our standardized, Likert-scale format.

We also may not have accurately measured social motivation with the self-report instrument used (the ACIPS). The scientific literature is currently unclear as to how validly and reliably individuals can self-report their levels of social motivation (Fulford et

al., 2018). Social motivation is also more complex than motivation for basic rewards, such as money and food, making it more challenging to operationalize social motivation through self-report or even laboratory tasks (Fulford et al., 2018). Many self-report questionnaires on social motivation depend on recent interactions and ongoing relationships (Fulford et al., 2018). Given that autistic adults may be experiencing low levels of recent interactions and ongoing relationships, it is possible that the self-report questions may reflect the absence of meaningful interactions, rather than an inherent motivation or objectively observable drive to interact with others and maintain social relationships (Fulford et al., 2018). Due to these challenges with self-report survey questions, researchers are moving toward momentary, real-time assessments of social motivation in self-report (Han et al., 2019) or more objective, observable paradigms, such as the Cyberball game (Silva et al., 2020).

Further, the measures we used were developed for and validated within the general population, and thus their item content and wording were likely geared toward neurotypical individuals. More specifically, even though these measures have been used and shown to have internal consistency in autistic populations (e.g., Novacek et al., 2016), the items on the questionnaires still reflect neurotypical values around social experiences. For instance, several questions on the ACIPS ask participants to rate their motivation to spend time with *groups* of people, which reflects the neurotypical viewpoint that socializing with a group of friends is "normal" behavior. Given the social challenges that are characteristic of autism, though, autistic individuals may experience motivation to interact with a single *individual* or a small group of people rather than a *group* of individuals. Further, given the high co-occurrence of social anxiety and autism

(Spain et al., 2018), it could be that autistic adults not only feel unmotivated to pursue group social activities, but they actually may avoid such interactions. Taken together, our measure of social motivation may have *underestimated* the degree of social motivation our participants experienced due to the inclusion of several group related social questions. On the other hand, these items may have served to expand the variability of self-reported social motivation within our autistic adult sample.

Finally, there may be an aspect of social motivation or social access that we have not yet considered that may be more salient to the experiences of autistic adults than self-reported social motivation, opportunity, and sense of belonging. In sum, while our hypothesis yielded results that were either statistically significant or very close to being practically significant, our null findings may be due to the psychometric challenges of defining and measuring these nuanced social constructs.

Chronological Challenges

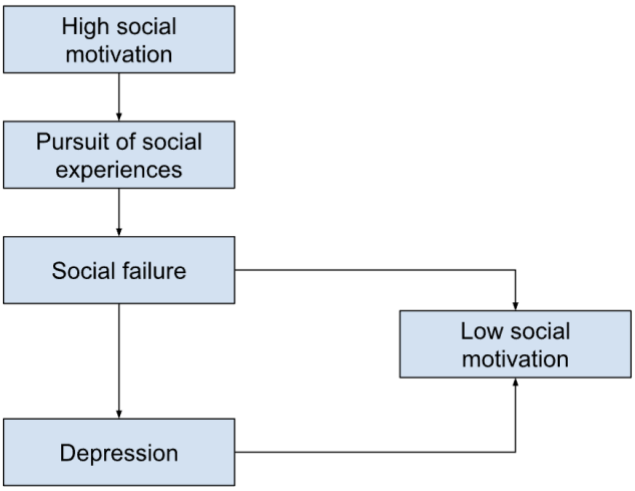
Beyond these psychometric challenges, we may not have found support for our hypothesis because we were not able to index inherent social motivation that we would expect to be neurologically mediated and trait-like per individual. Rather, we may have been surveying social motivation in adults who already had been influenced by at least two decades' worth of social feedback and potential mental health problems. It is reasonable to assume that participants endorsing low social motivation at Time 1 included both those with more stable, trait-like low social motivation, as well as those with either more transient or long-term "acquired" reductions in social motivation as a result of previous or ongoing social failure, anhedonia, or depression. In the latter scenario, autistic adults with higher innate levels of social motivation likely acted on

social motivation at previous stages of their lives to engage socially, then experienced real or perceived social failure in their social interactions. Indeed, there is evidence that this relationship between higher social motivation and greater social failure occurs among children and adolescents on the autism spectrum (Sedgewick et al., 2016). One research team interpreted their findings to suggest that a high level of social motivation among adolescent girls on the autism spectrum appeared to facilitate their ability to initiate social contact and make friends, but the core social and communication difficulties they face prevented them from recognizing and responding to subtle social nuances, making them an "easy target" for relational conflict and social neglect (Sedgewick et al., 2016). Indeed, several teams have proposed that among individuals on the autism spectrum, adolescence can be a time marked by a combination of higher social motivation (due to interventions or maturation) and increased awareness of social failures (due to the gap between the adolescents on the spectrum and their peers becoming more evident) (Kasari & Sterling, 2013). Humans are not motivated to pursue things that we do not find reinforcing: This real or perceived social failure, especially if repeated, may feed back to cause a more stable reduction in social motivation due to a long-term lack of reinforcing experiences. Perceived social failure could also contribute directly to depression, by activating core beliefs that often increase risk for depression (e.g., "I am unlovable;" Otani et al., 2018), with the anhedonia related to depression then acting to further reduce social motivation (American Psychiatric Association, 2013). As summarized in Figure 8, then, there could be paths from high social motivation→ pursuit of social experiences→ social failure → low social motivation + depression, as well as social failure→ depression→ low social motivation. In that case, our current high social motivation

responders may be only a subset of those with initial high social motivation, and our low responders would be heterogeneous across innate and acquired reduced social motivation.

Figure 8

Potential Pathways for "Acquired" Low Social Motivation



Importantly, this heterogeneity could obscure our ability to comment on the discrepancy between high social motivation and low social access as a contributor to loneliness and then depression, and thus could explain both (1) why we did not see the proposed interaction as related to increased depressive symptoms, and (2) why we did not find evidence to suggest that low social motivation protects against depression. This represents a limitation in the measurement of social motivation, now as related to the chronology of our entry point into the system. Similar work should be undertaken in children as a comparison prior to potential social-failure feedback loops.

In addition to not capturing social motivation early enough in one's lifetime, the 3–4 month time frame we chose to use may have been too short to see our predicted longitudinal relationships develop. It could be that, had we continued the study for 1-2 years, the hypothesized patterns would emerge.

Though our current findings suggest the opposite, it remains possible then that, autistic adults with more intrinsic, stable, trait-like reduced social motivation may be less likely to internalize social failures and/or have to cope with unmet social needs, protecting them from experiencing isolation, loneliness, and ultimately depression.

Potential for Model Confounds and Other Limitations

In addition to measurement challenges, we may have not observed our predicted interaction because our model failed to account for confounding variables related to the predictor, mediator, and/or outcome in this proposed moderated mediation mechanism.

One such potential confound is not taking into account the reference group when asking individuals to rate their own social motivation. For example, we did not account for any variables related to cultural norms around social interaction, but indeed culture may have played a role in how much individuals value social interaction and the types of social interactions individuals view as meaningful. For example, participants may come from cultures that place greater emphasis on familial relationships compared to friendships outside the family. Given that several of the social access questions specifically ask about "friends" or individuals outside the family (e.g., "I am satisfied with my ability to do things for my friends;" "On average, how often do you participate in social events…with friends, neighbors, or people from outside your family?"), our measures may have underestimated the fulfillment of social needs for these participants.

Perhaps these participants experience high amounts of social meaning and interaction with people within their familial unit; this may be true of many autistic adults regardless of their cultural background. Additionally, participants may have compared themselves to individuals from their own culture in order to inform their answers. For example, Participant 1 may rate themselves as experiencing less social motivation than Participant 2 if Participant 1 is comparing themselves to a more outwardly socially motivated, collectivist cultural group, when in fact an outside observer from an individualistic culture may still perceive Participant 1 as more socially motivated than Participant 2. Similarly, participants may have compared themselves to members of their own family to select their answers, and indeed family cultures may differ in their views on the importance of social interaction, the types of social interactions that are seen as meaningful, and the amount of social interaction that is viewed as appropriate. For instance, coming from a family that greatly values social interaction and is very socially active could influence participants to underestimate their levels of social motivation and social access if they do not feel as motivated as their family members or do not participate in as many social activities as their family members.

Further, this study relied solely on self-report, which poses additional measurement challenges if we did not achieve measurement invariance by gender, culture, or other variables – in other words, if the self-report measures we used were not equally biased by potential confounds. For example, if self-report measures of social motivation and social access were influenced by culture, IQ, or insight more so than the measures of loneliness or depression, this discrepancy could skew the results. Conversely, if all self-report measures were biased by culture to a similar degree, this

would have less of an effect on the overall results, (i.e., the relative relationships between self-report variables would not be affected if the bias was evenly distributed across all self-report measures), compared to an outlying biased instrument. Failure in measurement invariance might also look like men and women having systematic patterns of differences in how they respond to a set of items, which may have obscured findings.

Because this was an online study, we had little way of assessing IQ or level of social insight, but these functioning variables may represent additional confounds. It could be that autistic adults who are more aware of societal norms for "acceptable" social interactions experience a discrepancy between their levels of desired and achieved social engagement that leads to loneliness and depression. In our study, we grouped all autistic adults together, when perhaps we would have observed our hypothesized moderated mediation model if we had adjusted for autistic trait level or social insight.

Another potential confound is chronological age. We recruited participants between the ages of 18-64, and while we did include age as a dimensional covariate in our post hoc analyses, we did not assess whether different categorical age groups within this broad range had similar patterns of results. However, there is some evidence to suggest that age is an important consideration for our constructs: social motivation appears to increase with age in autistic individuals (Deckers et al., 2014; Deckers et al., 2017); depressive symptoms increase with age in the general population (Alonso et al., 2021); and loneliness appears to demonstrate a complex, nonlinear shape in the general population, with higher loneliness among young adults (<30 years), middle age adults (around 50-60 years) and very old age (>80 years) (Hawkley et al., 2022). As such, perhaps we would have observed our hypothesized relationships specifically in the age

group of around 50-60 years, as these individuals may be more likely than the younger participants to experience higher social motivation, higher depressive symptoms, and higher loneliness while also having fewer built-in social opportunities (e.g., younger individuals are more likely to live at home with family, Fry, 2020; attend college, Hanson, 2023; connect with others over social media; Perrin, 2015). Thus, combining participants from different age groups into one sample could have further "diluted" our results.

In sum, it is possible that we did not observe our hypothesized findings for several reasons. Psychometric challenges, such as not properly defining and measuring social access and social motivation, may have contributed to our results. Further, we may have been measuring low social motivation that is a remnant of social failure and/or depression when we intended to measure more trait-like reduced social interest, or not following our sample for a sufficiently long window in which to have observed these patterns between variables, thus obscuring our findings due to the chronology of when we measured these constructs. Lastly, we may have failed to account for confounding variables in our proposed moderated mediation model, including those relating to social insights via individual functioning or cultural norms.

Clinical Implications

Despite the various potential limitations we have outlined above, our results still have several important implications. **First, the findings underscore the importance of having meaningful social opportunities for psychological wellbeing in autistic adults.** Lower social access prospectively predicted loneliness, which prospectively predicted increased depressive symptoms. The fact that we did not observe a moderating effect of

social motivation suggests that it may be important to support meaningful social access for autistic adults universally, versus using resources selectively to target individuals who explicitly desire social interaction.

Taken together, it could be helpful to foster opportunities for social interaction among all autistic adults, whether or not they express a desire for social interactions. Note, this would need to be balanced with what we have learned about "fit" and self-determination when evaluating the quality of adult outcomes (Howlin, 2021). Perhaps autistic adults would benefit most, then, from support to define what a *meaningful* interaction would look like for the individual, with concrete help to achieve that. One idea could be to help autistic adults find others who share their special interests (Finke et al., 2019).

Second, our findings suggest that low social motivation does *not* protect against depression. As such, we did not find support for this particular aspect of Smith and White's social motivation model of depression (2020), in that low social motivation was not associated with lower depressive symptoms prospectively in our sample. The autism field is moving increasingly toward a strengths-based approach that emphasizes that autism characteristics (e.g., stimming) should not be modified or eliminated but rather are forms of neurodiversity that should be supported (Ferenc et al., 2022; Kapp et al., 2019). The current findings, however, suggest that low social motivation may not be merely a benign individual difference, but may be associated with deleterious mental health outcomes. It is unclear if the mechanism underlying this prospective association is reduced engagement in social activities or relationships, or rather a neurological link between any form of anhedonia (reduced motivation) and depressed mood. However, at

least in terms of long-term "acquired" low social motivation, rather than stable trait-like low social motivation, it seems possible that levels of social motivation could be modifiable and ideally treatment-sensitive.

Future Directions

Future studies can aim to address various study challenges and limitations we have outlined. Starting with the psychometric measurement challenges, future studies can determine better methods to define and measure social motivation and social access for autistic adults. This might include testing and addressing deviations in measurement invariance, developing self-report measures of salient social constructs in collaboration with the autistic community, and replicating the present study with observable or objective measures of social motivation and social access. Future work can also aim to start with younger samples and otherwise think creatively about disentangling trait-like low social motivation from "acquired" low social motivation and evaluate their respective differences in contributions to depressive symptoms. Perhaps conducting a longitudinal study tracing older school age participants as they transition into adolescence could help tease apart these two forms of low social motivation.

We also need to consider other constructs that, together with loneliness, mediate the relationship between social access and depression. Our baseline and best fitting model demonstrated only a partial mediation of less than 50% of the variance explained. This finding indicates that social access may be contributing to depression through other mechanisms besides loneliness. By identifying and incorporating these constructs in our model, we may be able to better explain the longitudinal, and plausibly causal, pathways

between having insufficient meaningful social opportunity and developing depression symptoms.

Other untested constructs that may be worth evaluating include maladaptive cognitive schema – which could issue from or be exacerbated by perceived social failure, as noted above – or social comparison, the process of comparing oneself to others (Swallow & Kuiper, 1988). Evidence suggests that upward social comparisons (i.e., when people compare themselves to others they see as superior; Wang et al., 2017) may play a role in depression in non-autistic individuals through the development of negative self-evaluations or connecting social performance success to one's self-worth (Swallow & Kuiper, 1988). Additionally, with the increase in social media use in today's society, which often portrays unrealistically positive self-portrayals of users, novel evidence suggests connections between social media use, upwards social comparison, envy, and depression in neurotypical individuals (Alfasi, 2019; Appel et al., 2016; Li, 2019). Indeed, data indicates that upward social comparison may play a role in depression among individuals on the autism spectrum, as well: One study on children and adolescents on the autism spectrum determined that scores on the Social Comparison Scale subscale of perceived group membership significantly and independently predicted depression scores, such that those who perceived themselves as being more dissimilar to others reported higher depressive symptoms (Hedley & Young, 2006). Thus, it could be that autistic adults experience upward social comparisons to other more "socially successful" individuals because they experience social challenges *and* live in a neurotypical world that emphasizes social activity as normal and desirable behavior. Further, in the current world, social comparison may be especially hard to avoid given the

pervasiveness of social media. Social media may serve a constant reminder for many autistic adults that they are not social in the same way that society has deemed acceptable, which may then contribute to loneliness and depression.

In line with Behavioral Activation theory (Dimidjan et al., 2014), it may be valuable to investigate factors associated with social interaction that protect against depression regardless of the quality of the interactions. For example, through increased physical activity, daily structure, time spent out of the house, etc., the pursuit of social interactions could still be protective from depression, even if the social interactions themselves are not emotionally beneficial. Lastly, given the highlighted importance of social access indicated by the current findings, future studies can aim to develop interventions to support autistic adults in developing and maintaining consistent and sufficient opportunities for meaningful social engagement. For example, UCLA's *Program for the Education and Enrichment of Relational Skills (PEERS)* is a manualized evidence-based social skills program originally designed for adolescents that has been found to be effective for improving social skills, knowledge, and engagement among autistic young adults (Laugeson et al., 2015). Future studies can continue to refine and modify these types of programs for younger and older adults.

Conclusions

The current study sheds light on the longitudinal relationships between social motivation, social access, and loneliness, and how these constructs play a role in the development of depressive symptoms in autistic adults. Importantly, our study did not find evidence that an interaction between high social motivation and low social access predicts higher loneliness and higher depressive symptoms in these individuals.

Nonetheless, our current findings suggest that loneliness mediates the relationship between low social access and depression, highlighting the importance of meaningful social engagement in the psychological wellbeing of autistic adults. Our data further suggest a (non-significant) pattern in which low social motivation prospectively predicts increased depressive symptoms, indicating that low social motivation does not protect against depression. Overall, the findings from the current study have the potential to advance our understanding of etiological mechanisms that lead to depression in autistic adults, a clinical population with both social challenges and high rates of depression. This is an important step toward developing more effective resources to enhance psychological well-being and quality of life in this underserved population.

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Appendix

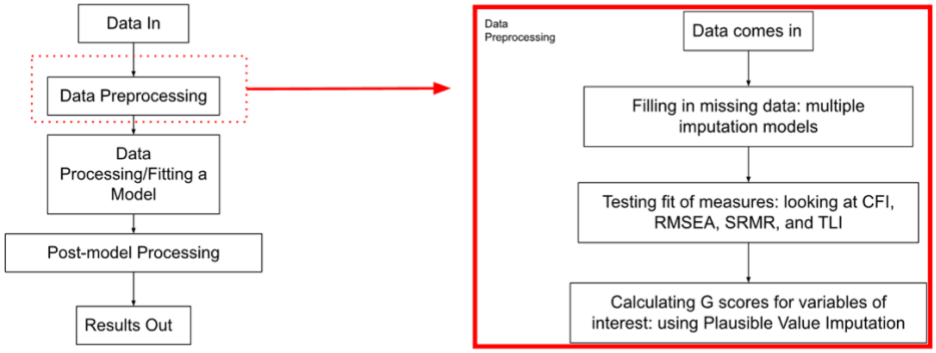
Statistical Analysis Details

Data Preprocessing

Before assessing the eight models (Figure 5 of main text), we first needed to complete data preprocessing (see Figure A1), which included addressing missing data, assessing the fit of the measures we used for our four variables (i.e., social access, social motivation, loneliness, and depression), and calculating scores on general factors (G factors; i.e., latent variables that influence all indicators within a bifactor model [see Toland et al., 2017 for more information]) for the constructs of interest.

Figure A1

Overview of Steps in Data Preprocessing Stage



Filling in Missing Data: Multiple Imputation Models

We utilized multiple imputation models to fill in the missing data (Enders, 2023). In the first of two stages (Li et al., 2015), multiple imputation modeling generates replacement values ("imputations") for the missing data and repeats this procedure numerous times, resulting in many data sets with replaced missing information (Li et al., 2015). These multiple guesses at the missing entries are based on the statistical characteristics of the data (e.g., the associations among the distributions of variables in the data set) (Li et al., 2015). In the second stage, these multiple imputed data sets are combined as if there were no missing data (Li et al., 2015). By doing so, we are able to maximize the data from our sample. Rather than eliminating participants from our sample who had missing data, we were still able to include these individuals in our analyses through multiple imputation. However, the algorithm factors in that there was more error in these participants' values compared to participants we did have full data for, as the algorithm is guessing at the values the participants would have reported.

In order to complete the multiple imputation models in R, we used the missForest package, which is an R package that uses random forest imputation (Stekhoven & Bühlmann, 2012) to compute these multiple guesses (Stekhoven, 2022). We completed the multiple imputations ten times to yield ten different imputed datasets.

Testing Fit of Measures

After filling in the missing data, we tested the fit of the different measures that operationalized our four variables of interest (social motivation, social access, loneliness, and depression).

A combination of the confirmatory fit index (CFI), root mean square error of approximation (RMSEA), Tucker-Lewis indices (TLI), and standardized root mean square residual (SRMR) were used to determine the global fit of the measures. CFI measures the amount of deviation between the estimated chi square value and the expected chi square value for the sample under the assumption that the model is correct (Van Laar & Braeken, 2021). RMSEA is an absolute fit index, as it assesses how far a hypothesized model is from a perfect model, while the CFI and TLI are incremental fit indices that compare the fit of a hypothesized model to that of a baseline model, or model with the worst fit (Xia & Yang, 2019). The SRMR represents the difference between the observed correlation and the expected correlation matrix (Ringle et al., 2022).

"Good fit" was operationalized as a CFI and TLI > 0.97 (Cai et al., 2021), RMSEA < 0.089, and SRMR < 0.05 (Maydeu-Olivares & Joe, 2014). Additionally, item misfit at the local level was evaluated by examining standardized residuals, such that $|r_{res}| > 0.1$ (Maydeu-Olivares, 2014), and a Q_3 value above the empirical cutoff value (Christensen et al., 2017; Yen, 1984) indicated the need to delete an item from the model.

Calculating G Factors: Utilizing Plausible Value Imputation

We then tested the models by calculating composite scores, or latent construct measures for our four variables of interest (social access, social motivation, loneliness, and depression). This step allowed us to map all measures used for each variable onto the same, standardized scale.

We used a plausible value imputation algorithm to compute the latent traits (i.e., "G factors") of social access, depression, social motivation, and loneliness. This algorithm mapped each measure onto the same, standardized scale (a Z-score with M=0,

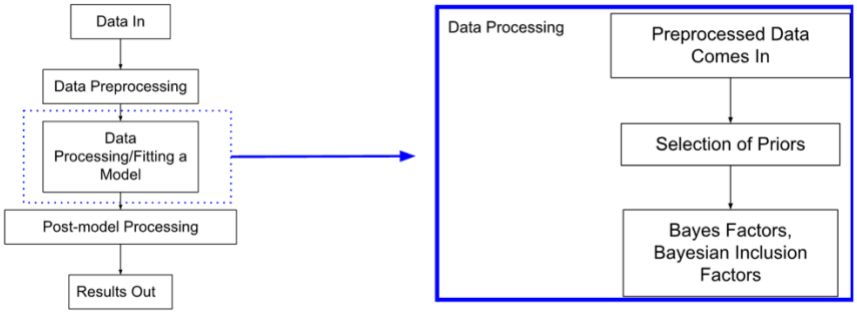
SD=1), and weighed each item differently according to how well the item assessed the G factor. Specifically, plausible values are multiple imputed values taken from a latent regression or population model (Khorramdel et al., 2020). The plausible value technology completes several draws (mostly five) from the posterior distributions of latent variable scores for each participant that are then used as latent variables in a subsequent statistical model (e.g., a regression model or structural equation model), generating a structural model containing a complete data set (Gorter et al., 2015). Every draw adds a little bit of error to the model, which importantly accounts for the fact that the latent trait score estimates are not free of error and that the latent variable is not actually observed (Gorter et al., 2015). Indeed, the literature recommends drawing five sets of plausible values to address the uncertainty corresponding with the plausible values for the missing data (Gorter et al., 2015). Thus, the plausible value imputation was able to calculate the G factors for the four latent variables by estimating the factor score five different times for each model (in each imputed data set, for a total of 50 draws per construct), with each estimation providing a little bit of error to account for the fact that our estimates are not error-free and that the latent variable is not observed.

Data Processing

With our preprocessed data (i.e., missing data were filled in; G factors calculated for all four variables of interest) we next fit these G factors to the baseline model and hypothesized model (see Figure 3 of main text) within the Bayesian framework (see Figure A2 for an overview of this data processing stage).

Figure A2

Overall Steps of Data Processing



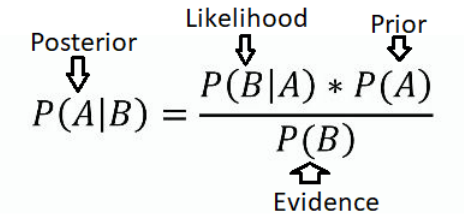
Background Information on Fitting Bayesian Models

Before describing the steps in Figure A2, it is crucial to outline the Bayesian approach. In the Bayesian approach, we have new data that we are attempting to explain (Kruschke & Liddell, 2017). We then have candidate explanations of this new data that have prior credibility of being the best explanation for the new data (Kruschke & Liddell, 2017). We subsequently shift the credibility toward the candidate explanation that best accounts for the new data, and our "best estimate" explanation then becomes a weighted combination of prior beliefs and observed data (Kruschke & Liddell, 2017). In other words, the Bayesian approach involves taking our beliefs about the data before collecting the data and combining these beliefs with what the data are actually telling us to give us a more updated belief about the data (Kruschke & Liddell, 2017).

In more technical terms, the Bayesian approach combines two probability density functions (PDFs); the PDF from before collecting data (known as a "prior" belief) and the PDF from the data collected (Koch & Koch, 1990). Combining the prior PDF with the PDF of the data leads to an updated belief about the data, which is referred to as a

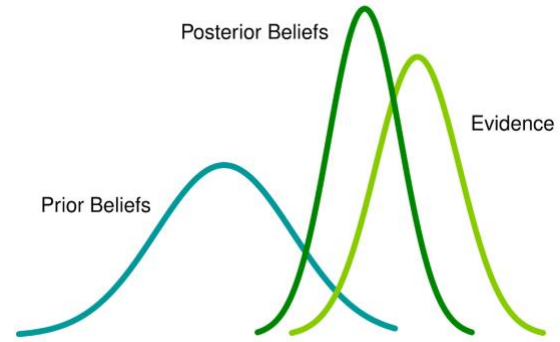
"posterior" distribution or "posterior belief" (Koch & Koch, 1990). The posterior distribution then represents the most credible values and range of reasonable parameter values (Kruschke & Liddell, 2017). If we were to run a study again, this posterior would then become the new prior, and the process would repeat, leading to a continuous cycle of posterior beliefs becoming updated prior beliefs to test with more data (Kruschke & Liddell, 2017).

The goal of a Bayesian analysis is thus to generate a posterior distribution that is informed by our prior beliefs and the observed data. Represented as an equation (below), the posterior we aim to determine is defined as the likelihood multiplied by the prior, and this product then divided by the evidence. More specifically, in the equation below, "A" represents our prior belief, and "B" represents the observed data. Thus, our ultimate goal is to determine the posterior, P(A|B), which represents the probability of our prior belief given our observed data. The likelihood, P(B|A), represents the probability of our observed data given our prior beliefs. P(A) represents the probability of our prior belief, and P(B) represents the probability of our observed data, also known as the evidence. (Kruschke & Liddell, 2017).



(Castellanos, 2019)

Shown graphically below, Bayesian analysis determines a posterior distribution that falls in between the prior beliefs and our observed data (i.e., the evidence).



(NSS, 2023)

We chose to analyze our data within a Bayesian framework for several reasons. First, Bayesian analyses are superior at handling multiple parameters (Kruschke & Liddell, 2017). Because we were testing a moderated mediation model, we needed to calculate the posteriors of various combinations of parameters, and Bayesian analyses easily allowed for this by multiplying posteriors together (Hinne et al., 2020). Bayesian analyses also allowed us to use different non-normal likelihoods (e.g., student-t error terms), which made the regressions more robust to outliers (Zhang et al., 2021). Other strengths of the Bayesian approach include that the posterior distribution is directly interpreted; we "read off" the most credible parameter values and range of parameter values (Kruschke & Liddell, 2017). As such, there was no need to create sampling distributions from null hypotheses, and it was unnecessary to figure out the probability that fake (hypothetical) data would be more extreme than the observed data (Kruschke & Liddell, 2017). Instead, measures of uncertainty were based *directly* on the priors

(Kruschke & Liddell, 2017). Taken together, the Bayesian approach was desirable for our study because the approach was well suited to test multiple parameters with non-normal likelihoods, and the approach did so in a simple way that could be directly interpreted (i.e., we could ask questions that we actually wanted answered, like "what is the [posterior] probability that effect X is greater than 0.5 [conditional on our data and priors]?").

From a more general perspective, the Bayesian approach is beneficial because it inspires meta-analytic thinking, as it encourages us to continually "update" our probability estimates (König, & van de Schoot, 2018). In this way, the approach also reinforces the idea that our study was one of a large number of studies that *could* be done and helps prevent thinking that a single study has discovered the "end-all" answer (König, & van de Schoot, 2018). In sum, the approach encourages us to replicate studies and continuously update our beliefs rather than assuming that a given study has determined the final answer.

Preprocessed Data Comes In

The preprocessed data refers to the G factors for the four variables (social motivation, social access, loneliness, and depression).

Importantly, although we were interested in assessing whether our proposed moderated mediation model predicted depression better than the baseline model (see Figure 3 of main text), we also assessed social motivation as a moderator for every potential path (i.e., the eight models shown in Figure 5 of the main text). We assessed all eight possible moderated mediation models because doing so allowed us to compute an

inclusion Bayes factor for the best model, which looks at the evidence for a specific part of the model (i.e., a specific coefficient of the model) averaged across all models.

Selection of Priors

Our first step in testing the eight models was to determine priors of the regression slopes of each model. We were able to utilize the findings from Han et al. (2019) to inform and justify these priors.

We also selected to use a student-t likelihood function to improve the robustness of our models to outliers (Lange et al., 1989):

$$f(y) = \frac{1}{\sqrt{2\pi}\sigma}\exp\left(-\frac{1}{2}\left(\frac{y-\xi}{\omega}\right)^2\right)\left(1+\operatorname{erf}\left(\alpha\left(\frac{y-\xi}{\omega\sqrt{2}}\right)\right)\right)$$

Selection of Final Model

We then entered the eight models and their corresponding priors into R and computed the Bayes factors for each model. Importantly, we classified the strength of the Bayes factors as $3<BF<10$ indicating moderate evidence for a given model, $10<BF<30$ indicating strong evidence for a given model, and $BF>30$ indicating very strong evidence for a given model (Wagenmakers et al., 2011):

The BF_{model} value tests the hypothesis that the tested model is the best model. A $BF_{model}>3$ indicates evidence that a given model is the best model, while a $BF_{model}<1/3$ or a $(1/BF_{model})>3$ is evidence that some other model is better than the tested model.

We then selected the model with the highest BF_{model} and highest inclusion Bayes factor to be our final model.

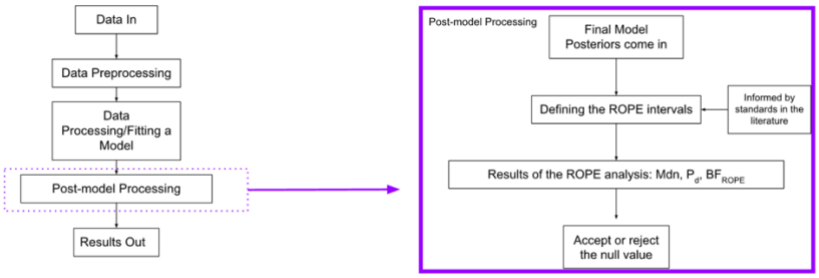
Post-Model Processing

Figure A3 below outlines the specific analyses steps we completed to further evaluate the final model.

We aimed to assess where the posterior values derived from the final model fell in relation to a set of null values. If the posterior we observed fell within the range of null values, this would be less support for the final model. However, if the posterior values fell outside of the range of null values, then this would be further evidence that the final model predicted depression the best among the eight models tested.

Figure A3

Post-Model Processing Outline



Defining the ROPE Intervals

In order to test whether the posterior values derived from the final model fell outside or within the range of null values, we first identified the range of null values to

compare to the posterior values. This selected range of null values is known as the region of practical equivalence (ROPE) (Kruschke & Liddell, 2017). In other words, the ROPE defines a region of values that are practically equivalent to the null values (also called the interval null), with the standard for null values falling between -0.1 and 0.1 (Kruschke & Liddell, 2017). Thus, this region translates to a zero effect size (i.e., Cohen established a convention of a "small" effect size being 0.2) (Kruschke & Liddell, 2017).

We then utilized specific ROPE outputs to determine whether the posterior values from the final model fell inside the ROPE (i.e., evidence against our final model) or outside the ROPE (evidence in support of our final model) (Kruschke & Liddell, 2017).

This Bayesian ROPE analysis then calculates several important values. The ROPE analysis calculates the median of the distribution (Mdn), which indicates the direction of the relationship (i.e., positive or negative) between the variables in the path being examined. The P_d, or probability of direction, then provides the probability of the sign of the relationship (i.e., the P_d indicates the probability that the sign of the Mdn observed is true). If the $P_d>0.975$, this is evidence of statistical significance (as a $P_d>0.975$ is equivalent to two-tailed $p<0.05$) (Makowski et al., 2019).

The Bayesian ROPE analysis also generates the Bayes Factor ROPE (BF_{ROPE}). The BF_{ROPE} compares the evidence for the hypothesis that the parameter of interest (i.e., the posteriors generated in the data processing phase of analysis) is *inside* the ROPE (i.e., H_0: Parameter X is practically equivalent to zero; $BF_{ROPE}<1/3$ provides evidence for H_0) to the evidence for the alternative hypothesis that the same parameter is *outside* the ROPE (H_A: Parameter X is large enough to be practically significant; $BF_{ROPE}>3$ provides evidence for H_A) (Makowski et al., 2019).