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Research Article

Autistic and ADHD traits and their relationships with atypical sensory processing and anxiety

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Abstract

Autism and attention deficit hyperactivity disorder (ADHD) frequently co-occur and show a positive correlation when examined as traits in the general population. Both are associated with anxiety, and Atypical Sensory Processing (ASP) may play a role in this relationship. A cross-sectional design was used to examine ASP as a mediator between autistic / ADHD traits and anxiety and explore the role of ASP in their shared variance. A total of 224 adults from the general population completed self-report surveys of ADHD traits, autistic traits, ASP, and anxiety. All measures correlated positively, and ASP was a partial mediator between autistic traits and anxiety and between ADHD traits and anxiety. In a partial correlation, ASP accounted for the majority of the shared variance between ADHD and autistic traits. Mediation of anxiety via ASP appeared to stem from the shared variance between ADHD and autism but not their unique variance. These results highlight the importance of considering ASP as a source of anxiety for those with higher autistic and ADHD traits. ASP is suggested as a transdiagnostic factor that may help explain the high co-occurrence of ADHD and autism. Areas for further research are discussed.

Keywords: autism, attention deficit hyperactivity disorder, sensory processing, anxiety

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Introduction

Autistic Spectrum Conditions (henceforth 'autism') and attention deficit hyperactivity disorder (ADHD) are neurodevelopmental conditions that frequently co-occur (Kotte *et al.,* 2013; Rong *et al.,* 2021). Both can be conceived of as clusters of personality trait extremes, which are positively correlated and normally distributed throughout the population (Li *et al.,* 2019; Lundin *et al.,* 2018; Panagiotidi *et al.,* 2017). Understanding conditions in this way allows for investigation into their interactions with various constructs in non-diagnostic samples.

An approach to understanding the co-occurrence of ADHD and autism, either as diagnosed conditions or sub-clinical traits, is to examine their commonalities. A review by Antshel *et al.* (2016) presents evidence from brain imaging, genetic, neurocognitive, and behavioural studies, showing a mixture of both overlap and specificity. For example, a neuroimaging study using resting-state Functional Magnetic Resonance Imaging (fMRI), identified shared structural differences between autistic and ADHD children in the right striatum/pallidum when compared to neurotypical controls, with further differences unique to each condition in other areas of the brain (Di Martino *et al.,* 2013). A genetic overlap appeared evident in both social-communication challenges and behavioural responses of reaction time, whereas weak correlations were observed between other sub-domains of autistic and ADHD traits (Pinto *et al.,* 2016). Finally, using event-related potential methodology to examine social cognition, both autistic and ADHD participants demonstrated an attenuated response to face stimuli, however this was observed at a later stage of emotion processing in the ADHD group (Tye *et al.,* 2014). These examples suggest it is plausible that mechanisms accounting for the overlap between ADHD and autism may be distinct from those accounting for each condition alone. Such evidence has led to calls for more studies to examine features that are transdiagnostic across both conditions (Mikami *et al.,* 2019). Two such shared features that may be involved in this overlap are anxiety and atypical sensory processing (ASP).

ASP refers to the atypical perception and modulation of sensory input and may include both heightened (hypersensitivity) and reduced (hyposensitivity) sensitivity across a range of sensory modalities (Robertson & Simmons, 2012). For instance, hypersensitivity to touch might cause certain sensations, such as the feeling of a clothing label against the skin, to be experienced as painful. Conversely, hyposensitivity to touch could result in stimuli that most people find painful going unnoticed. ASP is widely reported in autism, with some studies suggesting prevalence between 82% and 97% in autistic children (see Dellapiazza *et al.,* 2018 for a review). It is recognised as a core feature in autism diagnoses (American Psychiatric Association, 2013) and is correlated with autistic traits in those with and without a diagnosis (Mayer, 2016; Robertson & Simmons, 2012). Studies have also found evidence connecting ASP with ADHD (Delgado-Lobete *et al.,* 2020; Ghanizadeh, 2011; Little *et al.,* 2017; Mimouni-Bloch *et al.,* 2018) and traits of ADHD have been found to

correlate positively with ASP in adults in the general population (Panagiotidi *et al.,* 2018).

A limited body of research has examined ASP in relation to autism and ADHD together (Cheung & Siu, 2009; Dellapiazza *et al.,* 2021; Sanz-Cervera *et al.,* 2017; Scheerer *et al.,* 2024). In particular, Little *et al.* (2017) found higher ASP in both autistic and ADHD child groups compared to typically developing controls. Similar findings were observed in adults (Ohta *et al.,* 2020) and is further supported with resting-state fMRI research (Itahashi *et al.* 2020) which found distinct sensory processing profiles of functional connectivity that held across autistic / ADHD groups, regardless of diagnosis. Furthermore, neural correlates observed between the autistic and ADHD groups were selective to sensory related areas of the brain, thus supporting a picture of ASP as transdiagnostic in ADHD and autism.

Autism, ADHD, and ASP are all related to anxiety. A recent meta-analysis indicates an estimated 27% of autistic adults also have an anxiety disorder (Hollocks *et al.,* 2018). Similarly, 25% of those with an ADHD diagnosis are estimated to have a co-occurring anxiety disorder (D'Agati *et al.,* 2019). Associations between ADHD and anxiety continue into older adulthood (Michielsen *et al.,* 2013) and may be reciprocal, such that higher levels of either may exacerbate symptoms of the other (Murray *et al.,* 2020). ASP plays a role in anxiety for autistic people. A qualitative study involving young autistic adults and health professionals, described a range of perceived sources for anxiety in autism with a key theme relating to sensory issues (Trembath *et al.,* 2012). Furthermore, ASP is associated with the positive relationship between ADHD and anxiety. Reynolds and Lane (2009) compared ADHD children with and without sensory over-responsivity with typically developing children. They found significantly higher physiological and psychological anxiety in the ADHD group with sensory over-responsivity compared with both the ADHD group without and typically developing control groups.

Two causal models have been proposed linking autism, ASP (specifically sensory over-responsivity; SOR) and anxiety (Green & Ben-Sasson, 2010): the Primary Anxiety model and the Primary SOR model. The Primary SOR model, which has empirical support (Carpenter *et al.,* 2019; Green *et al.,* 2011), posits aversive sensory experiences create negative associations with places, situations, or objects; thus, anxiety develops though the anticipation of threatening experiences associated with these. Amos *et al.* (2018) tested both models using Structural Equation Modelling, examining stress, anxiety, autistic traits, and ASP in healthy adults. They found the best fit for the Primary SOR model, with stress and SOR fully mediating the relationship between autistic traits and anxiety. However, model fit overall was poor for both models. To date, we are unaware of any equivalent studies examining ASP mediating anxiety in relation to ADHD severity or traits. Simultaneous examination of ADHD traits, autistic traits, ASP, and anxiety may help shed light on their relationships. More specifically, examining the role of ASP (both hypersensitivity and hyposensitivity) in the overlap

between ADHD and autistic traits, may illuminate how and why these traits / conditions are related. In addition, understanding sources of anxiety is of importance for supporting the wellbeing of both clinical and sub-clinical populations.

Using quantitative analysis of self-report measures in a general population sample, the aims of this study are: (a) to determine whether ASP partially mediates the relationship between autistic traits and anxiety; (b) to determine whether ASP partially mediates the relationship between ADHD traits and anxiety; and (c) to understand the role of ASP in the shared variance between traits of ADHD and autism, and how this relates to anxiety. In respect of aims (a) and (b), it is hypothesised that the levels of anxiety that correlate with ADHD traits and autistic traits will be partially mediated by ASP. In respect of research aim (c), it is expected that a proportion of the shared variance between ADHD and autistic traits will be accounted for by ASP. Further, it is expected that ASP will play a role in mediating between this shared variance and anxiety.

Method

Participants

The sample consisted of 277 participants. The required sample size for this study was based on *a priori* power calculations for the mediations. These were run using a web-based application (https://schoemanna.shinyapps.io/mc_power_med/; Schoemann *et al.,* 2017) which indicated that a minimum of 162 participants were required to achieve power of 80%. Participants were recruited online using social media and using the Northumbria University Department of Psychology research participation pool management software (Sona Systems; www.sona-systems.com). Participants recruited using Sona Systems received credits for completing the study. No other incentives were offered. To avoid selection bias, study adverts did not mention autism or ADHD. Participants were required to be 18 years or older and have self-reported normal hearing and vision. There were no additional exclusion criteria for this research study. The study was approved by the Faculty of Health and Life Sciences Ethics Committee at Northumbria University (ref: 49736). All participants provided informed consent.

One hundred and seventy-three participants (77%) identified as female, 50 (22%) as male and one (<1%) as Genderqueer. Ages ranged from 19 to 79 years (M_{age} = 44.51 years, SD_{age} = 13.37 years). One hundred and eighty-three participants had a highest education level of a degree or above (82%), 29 (13%) had reached A-level or equivalent, and 12 (5%) had a GCSE or equivalent. Seven participants (3%) reported a diagnosis of ADHD, 5 (2%) of autism and 62 (28%, including 3 of the participants with autism and 4 of the participants with ADHD) had other mental health diagnoses.

Measures

Participants completed the Broader Autism Phenotype Questionnaire (BAPQ; Hurley *et al.,* 2006), which is a 36-item measure of personality dimensions characteristic of autism. Example questions include "*I like being around other people*" and it is scored on a 6-point Likert scale (ranging from 1 ("*very rarely*") to 6 ("*very often*")). The BAPQ has been used to examine autistic-like traits in the general population and demonstrates good reliability for its three scales of *aloofness* (α = .89), *pragmatic language* (*α* = .76) and rigidity (*α* = .84; Ingersoll *et al.,* 2011). An excellent combined internal consistency of *α* = .94 was found in our sample.

Participants also completed the Adult ADHD Self-Report Scale (ASRS; Adler *et al.,* 2006) which measures ADHD-associated traits. The ASRS has 18 items and captures frequency of symptoms, for example ("*how often do you feel restless or fidgety*?"), using a 4-point Likert scale. The ASRS demonstrates good internal consistency for the *inattention* (*α* = .77) and *hyperactivity* (*α* = .74) sub-scales and good overall reliability (*α* = .83) for the combined items (Panagiotidi *et al.,* 2018). An excellent combined internal consistency (*α* = .91) was found in our sample.

Participants completed the Glasgow Sensory Questionnaire (GSQ; Robertson & Simmons, 2012), which measures hypersensitivity and hyposensitivity to stimuli across 7 sensory modalities (vision, audition, gustation, olfaction, touch, balance and proprioception). Participants are asked to respond to 42 items, such as "*do you find certain noises/pitches of sound annoying*?" with a 5-point scale of 1 ("*never*") to 5, ("*always*"). The GSQ has excellent internal consistency (*α* = .93; Panagiotidi *et al.,* 2017), with comparable consistency (*α* = .94) observed in our study.

Subjective anxiety was measured using the 7-item General Anxiety Disorder Scale (GAD-7; Spitzer *et al*., 2006). Respondents are asked to rate symptoms over the previous two weeks, such as ("*feeling anxious or on edge*") on a scale from 0 ("*not at all*") to 3 ("*nearly every day*"). The GAD-7 has good internal consistency (*α* = .85; Hinz *et al.,* 2017) in the general population and an excellent internal consistency (*α* = .92) was observed in our sample. Higher scores on all the above measures indicate higher levels of the underlying construct. Scores for each measure were calculated based on published methods.

Procedure

Participants completed an online study using Qualtrics XM (Qualtrics, Provo, UT). Participants completed demographic questions and the BAPQ, ASRS, GSQ and GAD-7, before they were provided with a debrief and further information and support.

Data analysis

Data were analysed using SPSS (Version 27.0, IBM Corp., Armonk, NY). Partial responses were removed, data was visually inspected for task engagement, checks were performed for normality, and scatterplots were generated to identify outliers. Scores were calculated for all subscales and totals for each measure. No outliers were found which affected the direction of relationships.

Descriptive statistics were generated and bivariate correlations conducted on the total scores for each measure of ADHD traits, autistic traits, ASP, and anxiety. Correlations of sub-factors for each measure were also investigated, to ensure the direction of relationships was consistent. Group differences were checked using *t*-tests or Mann-Whitney *U*-tests, as appropriate and correlations re-run controlling for confounds. To assess the extent to which the relationship between ADHD traits and autistic traits can be accounted for by ASP, partial correlations were run and compared, both with and without ASP as a control variable.

In the last stage of the analysis, suitability of the data for bootstrapping mediation was tested and mediations were run using SPSS PROCESS Model 4 (Hayes, 2013), with 10,000 bootstrapped samples and 99% confidence intervals (CIs), to allow for multiple comparisons. For the first two mediations, the variables of autism and ADHD traits were the main predictors, with ASP as the mediator and anxiety as an outcome. Then, since it is not possible to directly test mediation of the shared variance between two predictors, the unique variance of autistic traits was tested by controlling for ADHD traits within the mediation, and the unique variance of ADHD traits was tested by controlling for autistic traits. These results were compared to those for the basic mediations. Age was entered as a covariate in all analyses. Variance Inflation Factors for all the regressions were equal to 1, suggesting multicollinearity was not an issue.

Z-scores for Skew were outside of the range for a normal distribution for the GSQ total and its hyposensitivity and hypersensitivity subscales, and for the GAD-7 (Kim, 2013). On this basis, non-parametric analytical methods were used as appropriate.

Results

Fifty-three participants were excluded due to incomplete responses, leaving 224 complete responses for analysis. Using the self-report cutoff scores for males and females (as suggested by Sasson *et al.* (2013)), 67 participants (30%: 9 males, 1 genderqueer, and 57 females) were at, or above, the normative score for the Broader Autistic Phenotype (BAP). According to the ASRS, 77 participants (35%) had symptoms highly consistent with adult ADHD (Adler *et al.,* 2006) that might warrant further investigation if used clinically. Thirty-seven participants (17%) scored above both the BAP cutoff and ASRS screener. Descriptive statistics for the main research variables are provided in Table 1.

Table 1: Descriptive statistics (n = 224).

	Mean	SD	*Z* skew	*Z* kurtosis
ASRS Total	33.05	12.39	3.08	-0.37
BAPQ Total	107.42	26.17	3.32	1.38
GSQ Hypo	20.20	11.61	4.03[a]	1.52
GSQ Hyper	25.77	13.87	4.91[a]	0.57
GSQ Total	45.97	24.14	4.25[a]	0.92
GAD-7 Total	6.78	5.61	4.55[a]	-1.31

Abbreviations: ASRS: Adult ADHD Self-Report Scale; BAPQ: Broader Autism Phenotype Questionnaire; GSQ: Glasgow Sensory Questionnaire; GAD-7; 7-item General Anxiety Disorder Scale; SD: Standard Deviation
[a]: Standardised Skew values outside of the normal range according to sample size (Kim, 2013).

Bivariate Spearman correlations were calculated for the main research variables (in Table 2). As hypothesised, all variables measuring self-reported ADHD traits, autistic traits, ASP and anxiety, were positively correlated with one another, and significant (all *p*-values < .001).

Table 2: Correlations (Spearman rho) for main research variables (n = 224)

	BAPQ Total	GSQ Hypo	GSQ Hyper	GSQ Total	GAD-7 Total	Age (years)
ASRS Total	.52***	.62***	.66***	.68***	.62***	-.27***
BAPQ Total	-	.57***	.58***	.60***	.51***	-.27***
GSQ Hypo		-	.79***	.94***	.49***	-.22***
GSQ Hyper			-	.95***	.56***	-.20***
GSQ Total				-	.56***	-.22***
GAD-7 Total					-	-.26***

Abbreviations: ASRS: Adult ADHD Self-Report Scale; BAPQ: Broader Autism Phenotype Questionnaire; GSQ: Glasgow Sensory Questionnaire; GAD-7; 7-Item General Anxiety Disorder Scale
Note: *** *p* < .001

Correlations were all in the moderate range, with the strongest between the ASRS and the GSQ total scores ($r(222) = .68$, $p < .001$). This was marginally stronger than the relationship between the BAPQ and GSQ total, ($r(222) = .60$, $p < .001$). The ASRS also showed the strongest correlations with the GAD-7, ($r(222) = .62$, $p < .001$), compared to a slightly weaker moderate correlation between the BAPQ and GAD-7, ($r(222) = .51$, $p < .001$). Both the hyposensitivity and hypersensitivity subscales of the GSQ were strongly positively correlated with each other and moderately positively correlated with the other measures, suggesting they are both measuring the same underlying construct. Thus, only the total measure was considered from this point forward.

Correlations between the sub-factors of all measures, including all sensory modalities of the GSQ, were also positively correlated: ranging from weak (BAPQ aloof vs GSQ Olfactory subscales; $r(222) = .28$, $p < .001$) to moderate (BAPQ pragmatic language subscale vs. ASRS total; $r(222) = .67$, $p < .001$). It is of note that the latter correlation is stronger than the correlations between BAPQ pragmatic language and the other two BAPQ subscales (BAPQ aloofness ($r(222) = .57$, $p < .001$); BAPQ rigidity ($r(222) = .55$, $p < .001$)). A correlation matrix, including sub-factors, is provided (Supplementary Material 1).

Gender has been associated with ASRS scores (Adler *et al.,* 2018), ASP (Ujiie & Wakabayashi, 2015), and anxiety (Leach *et al.,* 2008), thus, independent sample *t*-tests were used to compare ASRS scores by gender. Mann-Whitney *U* tests used with the GSQ and GAD-7. A Bonferroni correction ($p < .017$) was applied to account for multiple comparisons, but none reached significance (all *p*-values ≥ .025); consequently, all data, irrespective of gender, were analysed together. Age has been shown to correlate with the GSQ (Panagiotidi *et al.,* 2017) and was included in our correlation matrix (Table 2). Age showed a weak negative correlation with all outcome variables, such that, as age increased, the average score on the ASRS, BAPQ, GSQ, and GAD all decreased. Consequently, the main correlations were re-run, controlling for age. All remained significant ($p < .001$) and were in the moderate range. Correlations of the main research variables were rerun, excluding all those with ADHD, autism or mental health diagnoses. All correlations remained significant *(*$p < .001$) and were in the moderate range; thus, all participants were included in the partial-correlations and mediation analysis.

To examine the proportion of shared variance between autistic traits and ADHD traits that is accounted for by ASP, two partial correlations were run and compared. The first examined the correlation between autistic traits and ADHD traits, controlling for age. A moderate positive correlation was observed, indicating that an increase in ADHD traits was associated with an increase in autistic traits ($r(222) = .48$, $p < .001$). Next, a partial correlation was run between autistic traits and ADHD traits controlling for both age and ASP. In the presence of ASP, the strength of the correlation was reduced to weak, although it remained significant ($r(222) = .16$, $p = .016$). In line with expectations, this

suggests ASP can account for the majority of the relationship between autistic traits and ADHD traits.

Mediations

To test our first hypothesis, we examined whether ASP partially mediated the relationship between autistic traits and anxiety (*Figure 1*). In line with expectations, autistic traits predicted ASP (*a* = 0.58, *p* < .001), which in turn predicted anxiety (b = 0.08, p < .001). The indirect effect (*ab* = .05) was significant (99% CI [.02, .08]). The direct effect (*c*' = 0.07) was also significant, (99% CI [0.03, 0.11]), indicating a partial mediation through ASP of approximately 41%.

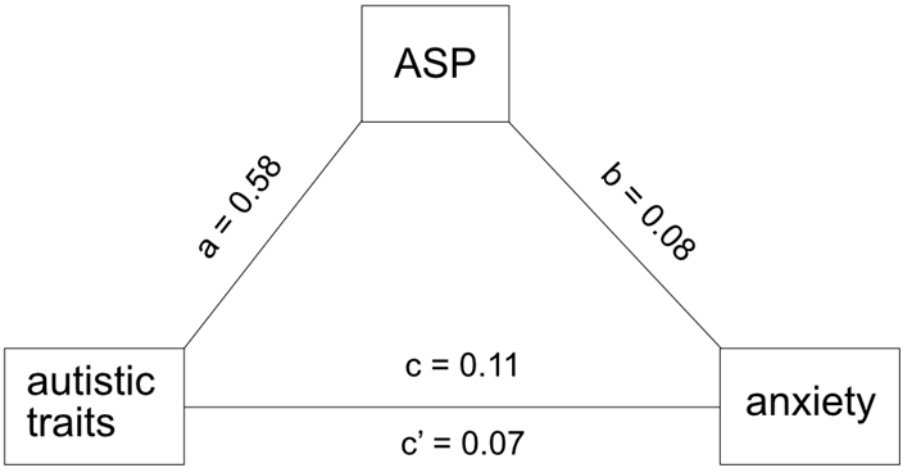

Figure 1: Atypical sensory processing (ASP) as a mediator between autistic traits and anxiety (*b* = unstandardised beta coefficients showing partial mediation; all coefficients are significant at *p* < .001).

To test the second hypothesis, we examined whether ASP partially mediated the relationship between ADHD traits and anxiety (*Figure 2*). As expected, ADHD traits predicted ASP (*a* = 1.33, *p* < .001), which in turn predicted anxiety (*b* = 0.06, *p* < .001). The indirect effect (*ab* = .08) was significant (99% CI [0.02, 0.15]). The direct effect (c' = 0.19) was also significant (99% CI [0.11, 0.28]). This indicates a partial mediation through ASP of approximately 29%.

To test our final hypothesis, the first mediation was re-run, with ADHD traits entered as a control variable (*Figure 3*). Autistic traits continued to predict ASP (*a* = .35, *p* < .001), but the path from ASP to anxiety was no longer significant (*b* = .03, *p* = .077). In this analysis, the indirect effect was no longer significant (99% CI [-0.01, 0.03]). The direct effect was significant (*c'* = .06; 99% CI [0.02, 0.09]). This shows that the unique

variance in the autistic traits, when controlling for ADHD traits, is not mediated by ASP but maintains a direct relationship with anxiety.

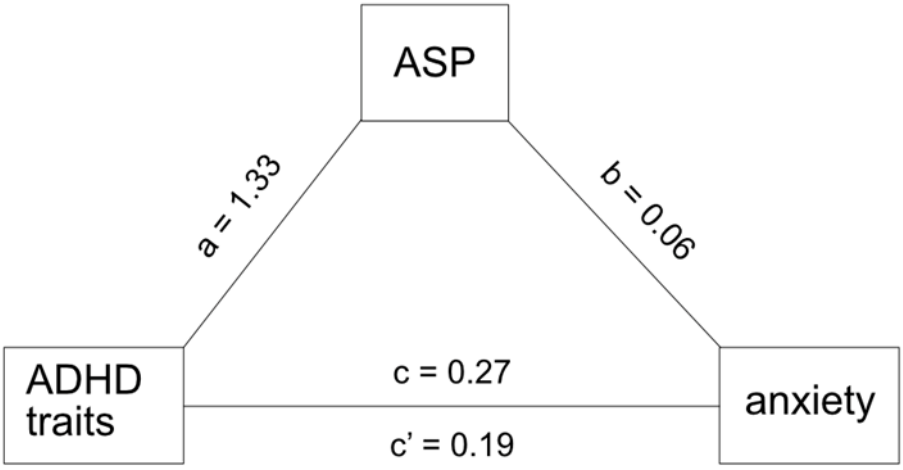

Figure 2: Atypical sensory processing (ASP) as a mediator between ADHD traits and anxiety (*b* = unstandardised beta coefficients showing partial mediation; all coefficients are significant at $p < .001$).

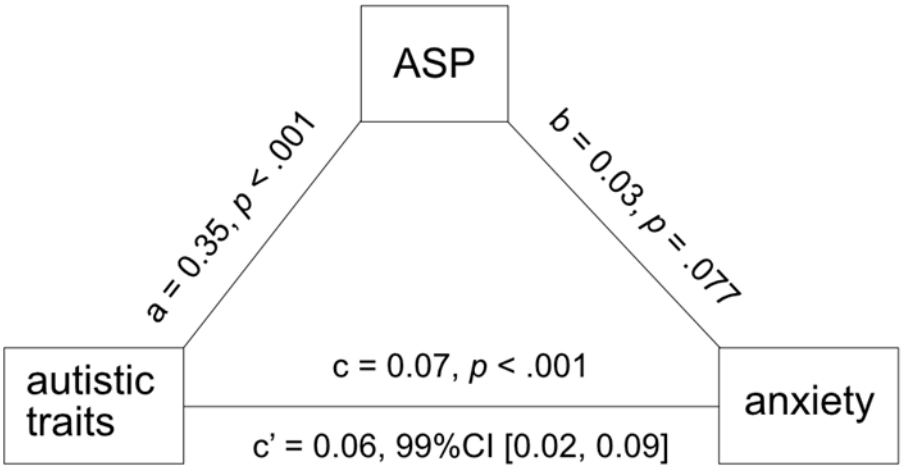

Figure 3: *Atypical sensory processing (ASP) as a mediator between ADHD traits and anxiety* (*b* = unstandardised beta coefficients).

Subsequently, the second mediation was also re-run, controlling this time for autistic traits (*Figure 4*). ADHD traits continued to predict ASP (*a* = .95, *p* < .001). However, ASP no longer predicted anxiety (*b* = .03, *p* = .077). Once again, adding the control variable meant that the indirect effect (*ab* = .03) was not significant (99% CI [-0.02, 0.09]). The only significant relationship between the variance unique to autistic traits

and anxiety was in the direct effect (*c'* = .17; 99% CI [0.09, 0.25]). Thus, the unique variance in ADHD traits, when controlling for autistic traits, is not significantly mediated by ASP but has a direct relationship with anxiety. In line with our hypothesis for research aim (c), these final two mediations suggest that the mediating effect of ASP on anxiety applies to the shared variance between autistic traits and ADHD traits, rather than their unique variance.

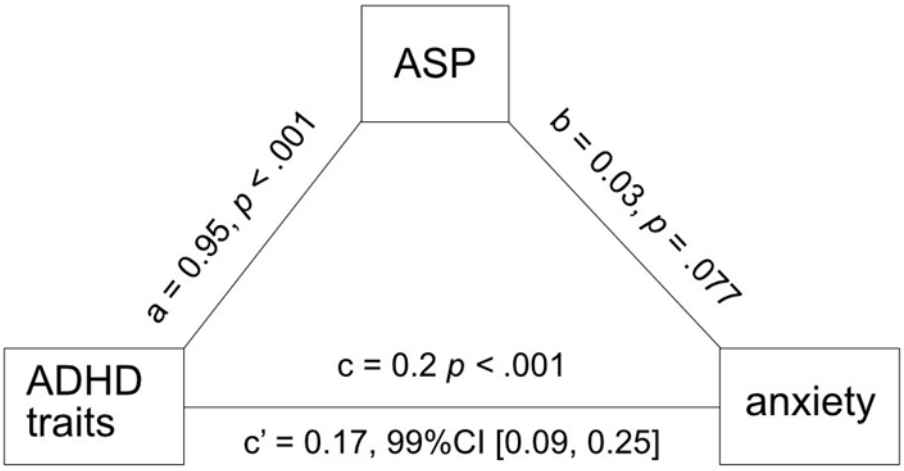

Figure 4: *Atypical sensory processing as a mediator between ADHD traits and anxiety, controlling for autistic traits* (*b* = unstandardised beta coefficients)

Discussion

This study had three main aims: (a) to test ASP as a mediator between autistic traits and anxiety, (b) to test ASP as a mediator between ADHD traits and anxiety and, finally, (c) to examine the role of ASP in the relationship between ADHD and autistic traits, including investigating ASP as a mediator between the overlap of ADHD / autistic traits and anxiety. All hypotheses testing these aims were upheld and the results revealed further insights into the relationships between the research variables.

In line with the first hypothesis, higher autistic traits were associated with higher anxiety, and ASP was a partial mediator of this relationship. This is consistent with literature suggesting that a proportion of the anxiety that co-occurs with autism may result from ASP (South & Rodgers, 2017; Trembath *et al.,* 2012). In the present study, mediation through ASP accounted for more than a third of the relationship between autistic traits and anxiety, indicating a substantial role for ASP in this relationship. These findings are consistent with Amos *et al.* (2018) who demonstrated sensory over-responsivity (hypersensitivity) as a mediator between autistic traits and anxiety. The current study extends these findings, to show that this relationship holds when combining measurements of hyposensitivity as well as hypersensitivity.

Our second hypothesis was also confirmed, with ASP also acting as a partial mediator between ADHD traits and anxiety. This novel finding highlights the importance of considering ASP in the context of the anxiety experienced by those higher in ADHD traits. It is consistent with research in children, suggesting those with ADHD plus sensory over-responsivity are more prone to anxiety than those with solely ADHD or typically developing controls (Reynolds & Lane, 2009). As with autistic traits, the mediation of anxiety is partial, highlighting that ASP is not the only variable connecting ADHD traits with anxiety.

It is interesting to note that, whilst the numbers diagnosed with ADHD or autism in our study were closer to population averages, the numbers at or above normative trait scores were higher. This is consistent with other studies that have found considerable ADHD symptom burdens in undiagnosed adults (e.g. Pawaskar *et al.,* 2019). Given both their association with anxiety and with other adverse health outcomes (Bishop *et al.,* 2019; McMorris *et al.,* 2018), our findings emphasise the extent to which neurodevelopmental differences may be affecting the wellbeing of substantial, but undiagnosed, proportions of the population. In addition to underscoring the need to explore interventions, this provokes a wider question around how we support and accommodate this diversity in workplaces, educational establishments and society in general.

A prerequisite of the analyses in this study was that all the main research variables would be positively correlated. This was found to be the case, replicating and extending findings from Robertson and Simmons (2012), and Panagiotidi *et al.* (2017; 2018). Positive modest correlations were apparent in subscales of both hyposensitivity and hypersensitivity in the GSQ. This supports other literature suggesting similar patterns of both hyposensitivity and hypersensitivity associated with ADHD and autistic traits (Little *et al.,* 2017; Ohta *et al.,* 2020). The strongest of the main correlations was between ASP and ADHD traits. This is consistent with findings from Ohta *et al.,* (2020) where similar or higher ASP was observed in their ADHD sample compared with their autistic sample. Given that ASP is now recognised as a core feature of autism (American Psychiatric Association, 2013), our findings lend weight to investigating ASP as a core feature of ADHD as well.

The final aim of this paper was the exploration of ASP in the relationship between autistic and ADHD traits. It was hypothesised that ASP would play a role in this relationship and in mediating between their shared variance and anxiety. A partial correlation was used to examine the extent to which ASP was involved in the association between ADHD and autistic traits. When compared to a correlation without controlling for ASP, it was apparent that the larger part of the relationship between ADHD and autistic traits could be accounted for by ASP. Furthermore, by controlling first for ADHD traits and then for autistic traits in the mediations, it was possible to analyse the variance unique to both autistic and ADHD traits. When doing this, only the direct effects of autistic and ADHD traits on anxiety remained significant. Thus, it

can be inferred that the anxiety being mediated by ASP, is associated, not with the unique variance, but with the variance that is shared between ADHD and autistic traits. Two important observations are consistent with these findings. Firstly, there are specific and distinct associations with anxiety that are unique to both ADHD and autism. Whilst these associations may be multiple and complex, they are associated separately with each neurodevelopmental condition. This upholds the conception of distinct elements unique to both conditions (Antshel *et al.,* 2016) and is evidence against hypotheses suggesting both ADHD and autism sit on a single, unidimensional continuum. Secondly, within the overlap between ADHD and autistic traits, there is a relationship with anxiety that is mediated by ASP. Together with the result of the partial correlation, this evidence suggests ASP as a transdiagnostic factor in ADHD and autism, that may substantially account for the relationship between them.

Genetic and other evidence suggest that the mechanisms accounting for autism and ADHD alone may be distinct from those accounting for their overlap (Antshel *et al.,* 2016). The centrality of ASP in this overlap, as evidenced in the present study, may be reflective of distinct underlying neural mechanisms. This is consistent with Itahashi *et al.*'s (2020) fMRI study which demonstrated that these areas of overlap are specific to sensory areas of the brain. Furthermore, Ohta *et al.* (2020) found white matter alterations in the corpus callosum that were similar in ADHD and autism and also related to ASP. This neurobiological evidence is consistent with evidence from this study, suggesting that the relationship between ADHD and autism may be connected to ASP.

A notable finding from the correlations of sub-scales in this study, was that the correlation between the BAPQ Pragmatic Language sub-scale and the ASRS, exceeded the intercorrelations between the three BAPQ subscales. Findings such as this emphasise the dimensionality of autistic and ADHD traits and pose a challenge to traditional diagnostic categories. A similar argument was made by Krakowski *et al.,* (2020), who found that scores of inattention and hyperactivity / impulsivity could not differentiate between children diagnosed as autistic and those with ADHD. They used this finding to argue for a framework that considers neurodevelopmental domains across diagnostic boundaries. The findings in our study support such an approach.

The evidence reported in this study should be considered in the light of some limitations. Firstly, all measures were based on self-report and as such will be vulnerable to certain biases (Stone *et al.,* 1999). However, the instruments used are validated and showed excellent reliability. Moving forward, studies combining self-report, observational and physiological measures would be useful to confirm the observed relationships. Care was taken to avoid selection bias, by not mentioning ADHD or autism in the recruitment material. However, people who completed and shared the survey may have referenced diagnostic labels. As is common in this type of research, the sample recruited in this study was weighted towards people identifying

as female. Future studies with a more representative selection of participants would increase confidence in the generalisability of these findings.

The mediation analyses run in this study are based on assumptions of a causal connection from higher autistic and ADHD traits to anxiety, both directly and via ASP. Although our findings are consistent with such a picture, they are purely correlational. More studies using longitudinal designs, particularly with adults, are needed to test the direction of these relationships. A number of further research possibilities flow from this study. As already mentioned, interventions targeted at the management of ASP are warranted in the general population for those with higher anxiety / ADHD or autistic traits. Current interventions for ASP in autism should also be explored in relation to ADHD.

In conclusion, this study adds important detail to our understanding of the relationships between ADHD traits, autistic traits, ASP and anxiety. Mediation analyses showed that the positive association between anxiety levels and both autistic and ADHD traits was partially mediated by ASP. Our second hypothesis was also supported, showing a comparable mediation of anxiety by ASP in relation to ADHD traits and highlighting ASP as a potential source of anxiety for adults with higher ADHD traits. It supports the case for considering ASP as a core component of ADHD and suggests interventions focused on ASP (such as Mindfulness Based Cognitive Therapy and Sensory Integration Therapy; Yuan *et al.,* 2022) merit exploration in adults with higher traits irrespective of diagnosis. Finally, our study showed that ASP could account for most of the association between ADHD and autistic traits. Furthermore, when ASP is acting as a mediator of anxiety, this mediation occurs in the variance that is shared between ADHD and autistic traits and is not significant in their unique variance. This suggests that the ASP that co-occurs with neurodevelopmental traits may be important to understanding the relationship between ADHD and autism. Further studies are warranted to explore how ASP may be involved in traits and mechanisms shared between these conditions.

Data availability statement

The authors confirm that the data supporting the findings of this study are available within the article and/or its supplementary materials.

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