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ORIGINAL ARTICLE

How Might Comorbid Conditions Co-occurring With Child Autism Impact Parenting Stress?

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Abstract

Purpose Many Autistic individuals present with comorbid conditions, including internalising and externalising behaviours, sleep issues, intellectual disabilities, and gastrointestinal dysfunction. We investigated the impact of these child comorbidities on parenting stress in an effort to elucidate the underlying mechanism and how they interact with autistic core symptoms. In total, three theoretical models were tested, being the Amplification, Additive, and Mediation Hypotheses.

Methods Participants were 453 parents of an Autistic child reporting on their child's core symptoms, comorbid conditions, and their parenting stress.

Results Correlation analyses reveal moderate associations between the comorbid conditions and parenting stress, but uncovered a weak link between core symptoms and parenting stress. Regression analyses revealed that, when key variables were allowed to adjust for one another, comorbid conditions were found to be independent predictors of parenting stress. A subsequent path analysis indicated that internalising and externalising behaviours partially mediated the relationship between core symptoms and parenting stress. There was no evidence to support the Amplification Hypotheses, and limited evidence to support the Additive and Mediation Hypotheses.

Conclusion The findings reinforce the argument that Autistic children require multidisciplinary services and interventions that stretch beyond their primary diagnosis. Further suggestions for future research into child comorbid factors and parenting stress are discussed.

Keywords Autism spectrum disorder · Comorbid conditions · Parenting stress

Introduction

The characteristics of Autism Spectrum Disorder (ASD) are clearly defined in the DSM-5-TR (APA, 2022), and a plethora of research has reported links between the severity of an autistic child's core symptoms and both parenting stress and parental well-being (e.g., Shepherd et al., 2024). The transactional model (Hastings, 2002) argues the importance of parenting factors in the ASD context, describing a bi-directional relationship between parenting stress and difficult child behaviours whereby suboptimal parenting can adversely impact child well-being (O'Connor et al., 2025). However, in addition to the extra parenting demands associated with child ASD core symptoms, the literature indicates that autistic children are more likely to have one-or-more comorbid (sometimes referred to as co-occurring) conditions such as gastro-intestinal issues, internalising or externalising behaviours, and sleep disorders (Bashore et al., 2025; Micai et al., 2023). Research into the effects of comorbidities indicates that the impact of some comorbidities on parenting stress exceeds the impact of ASD core symptoms (e.g., Olson et al., 2022), and consequently there have been calls that comorbid conditions need to approached more seriously at the clinical level (Lord et al., 2018).

Autistic children frequently experience co-occurring conditions that can complicate their autism diagnosis and adversely affect their well-being (Micai et al., 2023). In a systematic review of studies reporting the prevalence of comorbid conditions in autistic children and adolescents, Muskens et al. (2017) grouped gastrointestinal disorders,

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intellectual disability, epilepsy, and sleep issues as *medical* comorbidities on account of their somatic origin. They also found evidence that such comorbidities were far more prevalent in autistic children and adolescents than non-autistic controls, emphasising the importance of increasing the awareness of comorbidities in clinical practice in order to optimise intervention effectiveness. Previously, Romero-Gonzalez et al. (2016) employed the term *psychiatric* comorbidities to group together classifications from the DSM-5-TR that they considered to be frequently co-occurring with ASD, including anxiety, obsessive compulsive disorder, attention deficit hyperactivity disorder (ADHD), aggression, and self-harm behaviours. In the ASD literature psychiatric comorbidities are typically classified as internalising (e.g., anxiety, emotional dysregulation, depression, and withdrawal) or externalising (e.g., impulsivity, hyperactivity, aggression, defiance, and other conduct issues) behaviours, both of which are more prevalent in autistic than non-autistic children (Siu et al., 2019).

A consensus is forming in the literature that co-occurring behavioural challenges are more important contributors to parenting stress than the severity of child autism (Weiss et al., 2012; Davis & Carter, 2008). Furthermore, Mello et al. (2022) reported significant associations between autism symptom severity and both internalising and externalising behaviours, as well as between maternal-rated autism symptom severity and parenting stress. However, when undertaking a regression analysis, they noted that the significant relationship between autism symptom severity and parenting stress was eliminated when internalising and externalising behaviours were added to the model. They argued that core ASD symptom severity is a poor predictor of parenting stress, echoing previous findings (Weiss et al., 2012; McStay et al., 2014; Siu et al., 2019), which supports the notion that challenging behaviours (e.g., self-injury, aggression, and elopement) may fully mediate the relationship between child autism symptoms and parenting stress. However, Wang et al. (2020), conducting a network analysis, concluded that there were widespread positive associations between core and 'non-core' (i.e., comorbid) ASD symptoms. They suggested that interventions targeting comorbid conditions such as internalising or externalising behaviours might potentially improve core ASD symptoms, which implies that comorbid conditions may in fact increase ASD-related challenging behaviours. Within the current study we refer to the proposition that comorbidities can intensify core ASD symptoms, and thus amplify parenting stress, as the Amplification Hypothesis (*re*: Fig. 1a).

The Amplification Hypothesis states that the existence of one-or-more comorbid conditions can exacerbate the child's core ASD symptoms, which in turn leads to greater parenting stress and decreased parental well-being. Hsiao (2014) reviewed a number of studies and reported that, consistent with the gut-brain axis model, many ASD cases associated with gastrointestinal problems (e.g., diarrhoea, chronic constipation and abdominal distress) are associated with amplified ASD symptom severity. Similarly, comorbid ASD-anxiety disorders presenting with heightened social-interactional impairments and rigidity and repetitiveness behaviours, appear in about 40% of ASD diagnoses (Zaboski & Storch, 2018). Similar propositions have been suggested in relation to sleep disorder (Whelan et al., 2024; Lindor, 2019). The 'Amplification' hypothesis predicts positive bivariate correlations between child ASD symptom severity and comorbid severity, which is typically reported in the literature (e.g., Buchwald et al., 2025). Furthermore,

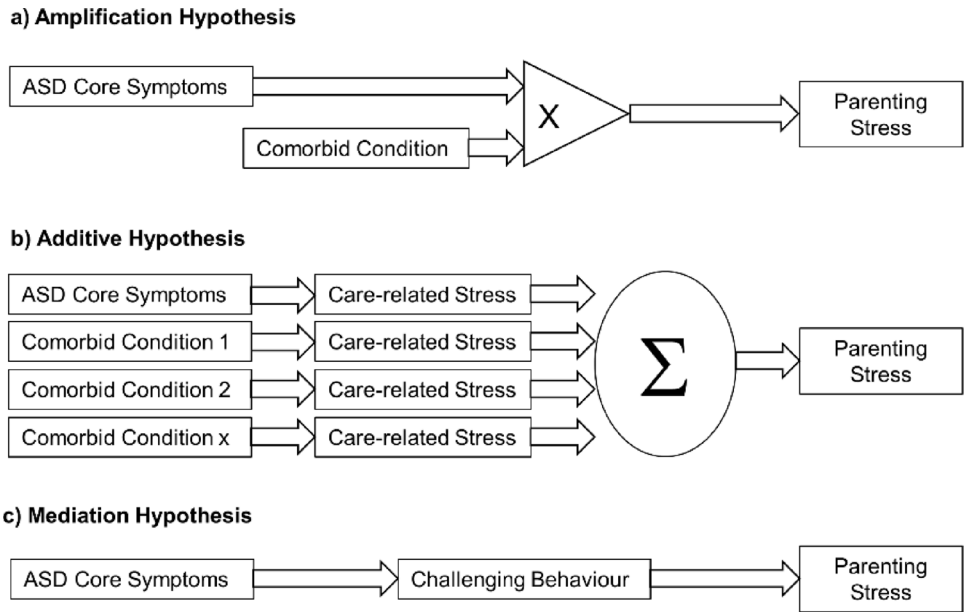

Fig. 1 A schematic representation of the three hypotheses accounting for the effects of child comorbid conditions on parenting stress

in the scheme of the Amplification Hypothesis it may be useful to conceptualise comorbid conditions as moderating the relationship between child core ASD symptoms and parenting stress, more specifically, strengthening the relationship between them.

Several studies have sought to determine if a proportional relationship exists between the number of comorbid conditions present in an autistic child and parental outcomes. Derguy et al. (2018) found that a greater number of comorbid conditions present in autistic children corresponded to lower parental well-being. Zablotsky et al. (2013) noted that mothers of autistic children with two or more comorbidities reported lower psychological well-being than mothers whose autistic child had no reported comorbid conditions. The results of these studies may likewise elucidate the mechanism upon which child comorbidities may influence parental well-being and parenting stress, that is, a simple summative or addictive effect. For example, a child with co-occurring ASD and ADHD may require a parent to be extra vigilant, or to engage in more care-related activities like play and cleaning, than if the child were not to experience with ADHD. Thus, in this scheme an increase in comorbidities may result in an increase in care-related tasks that stretch coping mechanisms, and combine to increase parenting stress. Support for the Additive Hypothesis (*re*: Fig. 1b) is, however, inconsistent, with some studies finding that parenting stress does not increase when ASD and ADHD diagnoses co-exist, compared to either condition in isolation (e.g., Miranda et al., 2015).

As noted, many studies have reported significant associations between the severity of child ASD and parenting stress (e.g., Osborne & Reed, 2009), however, when comorbidities are included in the analysis the statistical significance of these relationships is typically eliminated (e.g., McStay et al., 2014), though not always (e.g., Plant & Sanders, 2007). Thus, it may be that comorbid conditions fully mediate the relationship between child ASD severity and parenting stress. Siu et al. (2019), using a mixed sample of autistic and non-autistic children, conducted a hierarchical regression analysis and, noting evidence of a mediation effect once child problem behaviours were entered, proceeded to undertake a path analysis to test for indirect effects. Their model indicated that, after bootstrapping, both challenging and prosocial behaviours mediated the effect of child core ASD symptoms on parenting stress.

One difficulty with the approach taken by Siu et al. (2019) is that a mediation model needs to be theory-driven because it implies causality. In explaining their results and providing a theoretical framework to elucidate mechanisms, Siu et al. make the point that autistic children have greater difficulty contextualising their behaviours and, consequently, more readily enact externalising behaviours outside of the home than typically developing children. Such an approach is a reformulation of the Amplification hypothesis, being a reversal whereby the challenges in social cognition typified by ASD amplify externalising behaviours via impaired emotional regulation and difficulties managing interpersonal relationships. However, in contrast to the Amplification Hypothesis, the fully mediated relationship implies that child core ASD symptoms do not have a direct effect on parenting stress and instead indirectly influence parenting stress by exacerbating externalising and internalising behaviours.

Furthermore, this 'Mediation Hypothesis' (*re*: Fig. 1c) explains why internalising behaviours contribute less to parenting stress than externalising behaviours, as the latter constitute primary 'here-and-now' behavioural challenges while the former constitute "ASD 'background' challenges" (Siu et al., 2019, p.866). However, although externalising behaviours are generally found to be more predictive of parenting stress than internalising behaviours (Barroso et al., 2018), the majority of studies focus on children under 12 years and overlook developmental trajectories (Gotham et al., 2015). Finally, while the mediation approach effectively explains the relationship between child ASD severity and parenting stress, it is more specific in scope than the aforementioned Amplification and Additive Hypotheses, given its focus on challenging behaviours.

The Current Study

Parents typically experience high levels of parenting stress when raising their autistic child, with parenting stress leading to poorer parent-child interactions and a less sensitive parenting style. As a result, parenting stress can impede the outcomes of interventions and supports, hinder the development of their autistic child, and negatively impact the well-being and mental health of both child and parent. Reducing parenting stress is therefore an important goal to promote the development and quality of life of autistic children, and understanding the causes of parenting stress is central to its reduction. The literature indicates that the presence of comorbidities in autistic children can induce parenting stress above-and-beyond that related to core ASD symptoms. However, no studies to date have considered the mechanism by which the extra demands created by comorbid conditions serve to influence parenting stress. The objective of the current study is to seek evidence in support of the three aforementioned hypotheses: the Amplification, Additive, and Mediation Hypotheses. Specifically:

Hypothesis 1 *In line with the Amplification Hypothesis, comorbid conditions will moderate the relationship between child ASD symptom severity and parenting stress.*

Hypothesis 2 *In line with the Additive Hypothesis, the cumulative impact of child comorbidities on parenting stress will explain significant variability in parenting stress above-and-beyond that explained by child ASD symptom severity.*

Hypothesis 3 *In line with the Mediation Hypothesis, it is hypothesised that child comorbid conditions (i.e., externalising-, internalising-, and prosocial-behaviours) will fully mediate the relationship between child ASD symptom severity and parenting stress.*

Method

Participants

Participants were 404 female (M_{age} = 50.3 years, SD=9.45) and 49 male (M_{age} = 44.5 years, SD=8.85) parents of an autistic child and who were currently residing in New Zealand, and whose child had been formally diagnosed by a paediatrician (n=285), a registered psychologist/psychiatrist (n=121), or another health professional (e.g., MD, n=47). The parents reported on an autistic child identified as either male (n=348, M_{age} = 12.6 years, SD=6.86) or female (n=101, M_{age} = 12.8 years, SD=6.60), with four parents not disclosing the gender of their child. In terms of parent education, most reported a tertiary qualification from a technical institute (32.5%) or a university (53.3%), while 14.2% reporting finishing their education at a secondary school. The average age at which parents first noted ASD-like behaviours in their child was 1.76 years (SD=1.49), with the mean diagnostic age being 4.92 years (SD=2.92).

Measures

Parent-Rated Child Autism Symptoms

A modified version of the Autism Impact Measure (AIM: Kanne et al., 2014) was completed by parents in order to obtain a profile of their autistic child's core ASD symptoms. The original AIM instrument comprised both impact and frequency response scales, however, for reasons for brevity the measure was modified in the present study to only include the impact dimension when assessing the effects of ASD symptoms on child functioning. Each of the 25 items required a response on a 5-point Likert-scale ranging from 1 ('not at all') to 5 ('severely'), and four key dimensions are obtained: Restricted/Ritualized Behaviours (8 items), Odd/Atypical Behaviours (5 items), Communication/Language Impairment (5 items), and the presence of Social-Emotional Reciprocity skills (7 items). A total score is obtained by summing these four dimensions.

Parent-Rated Child Comorbid Conditions

Parents were asked to indicate whether their autistic child possessed one-or-more of the following comorbid conditions, whether formally diagnosed or not: anxiety, attention-deficit-hyperactivity disorder (ADHD), intellectual disability (ID), gastro-intestinal (GI) issues, or a sleep disorder. These five comorbidities were selected using a binary response scale (yes vs. no), and chosen on the basis of their high prevalence, as guided by Lanyi (2021), Micai et al., 2023, and Khachadourian et al. (2023). If a parent selected 'yes' then they were also asked, on a five-point scale (1=Very Mildly to 5=Very Severely), the degree to which the comorbidity affected their child's functioning. If a parent responded 'no' it was assumed the comorbidity was absent and a score of zero was assigned in regards to the impact on their child's function. The ratings obtained for each of the five comorbidities, ranging between 0 and 5, were then summed to create a total score with a theoretical range of 0 to 25. As this total score represents the cumulative effect of the five comorbidities upon child function it is denoted Comorbid Impact Total Score (CITS), and is differentiated from Comorbid Number which represents how many of the five comorbidities the parents reported their child to have.

Additionally, to allow a direct comparison to findings reported in the literature, continuous measures of child challenging behaviours were obtained using the Strengths and Difficulties Questionnaire (SDQ: Goodman, 1997). For the present study, parents were asked to record their response regarding a specific behaviour using one of three possible answers: "not true", "somewhat true", or "certainly true" based on their child's everyday behaviour over the past six months. The SDQ categorises the emotional and behavioural strengths and difficulties of a child into five subscales: emotional symptoms, conduct problems, hyperactivity, peer problems, and prosocial behaviour. An internalising behaviours score is obtained by summing the emotional symptoms and peer problems subscales, while the hyperactivity and conduct problems subscales are summed to obtain an externalising behaviours score. The calculation of externalising and internalising subscales increases sensitivity by not treating challenging behaviours as a homogenous construct (O'Connor et al., 2025), while the inclusion of the prosocial behaviour scale allows the model of Siu et al. (2019) to be tested.

Parenting Stress

The degree of self-reported parenting stress was estimated using the 18-item Parenting Stress Scale (PSS) which measures stress attributable to raising children (Berry & Jones, 1995). In the current study parents responded to each item using a five-point scale (1 = Strongly Disagree to 5 = Strongly Agree). Total scores were computed to meet the objectives of the current study, with higher scores indicating greater levels of stress.

Procedure

The survey was presented using the Qualtrics application. Here, participants were first presented information about the study and informed that engagement was voluntary and that they would remain anonymous. After confirming their eligibility, participants were informed that by completing the survey they were providing informed consent for the use of their data. Recruitment involved national autism support agencies within New Zealand, who promoted the study on their social media outlets. Ethical approval had been sought and granted by the University's Ethics Committee Auckland University of Technology Ethics Committee: 21/218 prior to data collection beginning.

Statistical Analysis

All analyses were performed in R v. 4.5.1, beginning with descriptive statistics (means and standard deviations), reliability analyses (Cronbach's alphas), and correlation coefficients (Pearson's *r*). Preliminary covariate screening was undertaken using a bivariate analysis to identify variables co-varying with the main variables of interest, and to detect any potential collinearity issues. On the basis of this screening, parent gender and age, the number of children in the house, and the age at which the child's autism-like behaviours emerged were selected as covariates to include in the regression models.

The veracity of the Amplification Hypothesis was probed using two sets of moderation analyses using hierarchical multiple regression containing interaction terms (*re*: Hypothesis 1). The first set consisted of two regression analyses each containing four steps, with the PSS Total score designated the dependent variable. In the first block covariates were introduced, while Block 2 introduced the AIM Total score. To afford comparisons with previous studies (e.g., Mello et al., 2022; Siu et al., 2019) Block 3 consisted of either the SDQ's externalised behaviours (Model 1) or internalised behaviours (Model 2) scores, with the focus on whether any statistical significance observed in Block 2 (i.e., AIM Total score) remained. Finally, Block 4 introduced the interaction term between the moderator and the AIM Total score and, if significant, would be taken as evidence to support the Amplification Hypothesis. The second set of moderation analyses contained a single regression analysis similar to those used in the first set, with the only difference being that the SDQ subscales were removed and instead the Comorbidity Impact Total Score was designated the moderator. Again, a significant interaction term would marshal evidence for the Amplification Hypotheses.

A pair of hierarchical multiple linear regression were used to test the Additive Hypothesis (*re*: Hypothesis 2). In Block 1 covariates were entered, Block 2 the AIM Total score, and in Block 3 the number of comorbidities endorsed by parents (ranging from 1 to 5) or the impact of comorbidities on child function scores were included. Evidence for the Additive Hypothesis would be marshalled through a significant increase in the amount of variance explained (ΔR^2) in the model between Blocks 2 and 3.

The Mediation Hypothesis (*re*: Hypothesis 3) was tested using a path model taken from Siu et al. (2019: their Fig. 1). In the current study AIM Total scores predicted PSS Total scores through the three SDQ subscales (i.e., externalising-, internalising-, and prosocial-behaviours). A single analysis was conducted in R using the lavaan package (v. 0.6–16: Rossel, 2012), with 2000 bootstrap samples indicating the statistical significance of indirect paths by providing bias-corrected 95% confidence intervals. The goodness of fit of the model was gauged using the Comparative Fit Index (CFI), Tucker-Lewis Index (TLI) and Standardized Root Mean Square Residual, where indices of > 0.95, > 0.90, and < 0.08 indicate a satisfactory fit, respectively.

Results

All analyses were undertaken in R (v. 4.4.2), with test assumptions scrutinised using Tabachnick and Fidell's (2019) guidelines, which also guided when variables were to be mean-centred. Descriptive statistics for the main outcome variables, when continuous, are displayed in the penultimate and ultimate rows of Table 1. The mean number of child comorbid conditions reported by parents was 2.68 ($SD = 1.08$, Min = 0, Max = 5). For those reporting one-or-more comorbidities, Fig. 2 displays mean PSS scores as a function of total comorbidities (i.e., Comorbid Number). A one-way ANOVA indicated significant differences in PSS scores across the five groups ($F(4,439) = 9.868$, $p < .001$), with *post hoc* testing indicating that those reporting a single comorbidity having significantly less stress than those reporting two-or-more. All other pairwise comparisons were non-significant ($p > .05$).

Table 1 Pearson's correlation coefficients for predictor and continuous variables (below and to the left of the major diagonal), with partial correlation controlling for participant gender and age (above and to the right of the major diagonal), while cronbach's alpha (in bold font) occupies the major diagonal

Measure	1. PSS	2. AIM Total	3. CITS	4. Externalising	5. Internalising	6. Prosocial
1. PSS	**0.90**	0.13*	0.36***	0.46***	0.37***	−0.32***
2. AIM total	0.12*	**0.89**	0.34***	0.31***	0.26***	−0.32***
3. CITS	0.36***	0.33***	-	0.49***	0.488***	−0.25***
4. SDQ externalising	0.43***	0.36***	0.50***	**0.79**	0.34***	−0.29***
5. SDQ internalising	0.37***	0.28***	0.49***	0.31***	**0.63**	−0.17***
6. SDQ prosocial	−0.32***	−0.32***	−0.24***	−0.25***	−0.156**	**0.76**
Mean	51.58	76.02	8.66	19.44	18.88	8.86
SD	12.46	15.38	4.45	4.01	3.15	2.49

PSS Parenting stress scale, *AIM Total* Autism impact measure total score, *CITS* Comorbid impact total score, *SDQ* Strengths and difficulties questionnaire

*p<.05; **p<.01; ***p<.001

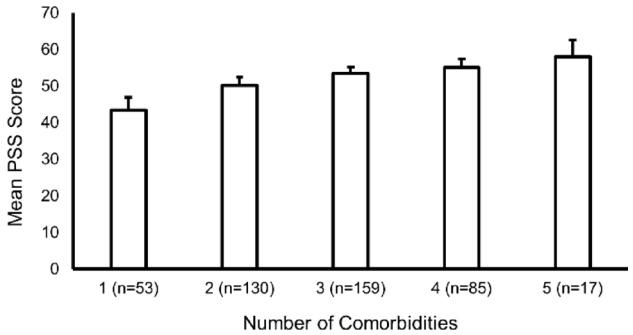

Fig. 2 Mean PSS score as a function of the number of child comorbid conditions endorsed by parents for their child

Correlational Analyses

Prior to conducting further regression and path analyses a correlation analysis was conducted across continuous measures to ensure sufficient variability existed. Table 1 presents bivariate coefficients (below the major diagonal) and partial coefficients controlling for participant age and gender (above the major diagonal). The major diagonal itself contains estimates of internal consistency, presented as Cronbach's alpha coefficients. All correlation coefficients were statistically significant, though the coefficient between the PSS and AIM total score was notably weak.

The Amplification Hypothesis

Moderator analyses were used to test the Amplification Hypothesis (i.e., Hypothesis 1), with each analysis consisting of four blocks, and with the PSS total scores assigned as the dependent variable. For the first set of regressions, the SDQ's Externalising (*re*: Table 2a) and Internalising (*re*: Table 2b) subscales were designated as moderators, though neither interaction term was significant in the final step of the models. However, while child ASD symptom severity (i.e., AIM Total) was a significant predictor of PSS scores when entered into the second step of the model, this significance was eliminated when either the Externalising or the Internalising scores were entered in the third step. Subsequently, the analysis was repeated using the Comorbid Impact Total Score (CITS) as the moderator (*re*: Table 2c), though the outcome was also a non-significant moderating effect.

The Additive Hypothesis

A pair of hierarchical multiple linear regressions were used to test the Additive Hypothesis (i.e., Hypothesis 2). As seen in Table 3, the first two steps of the model are equivalent to those presented in Table 2, but the third step presented either Comorbid Number (*re*: Table 3a) or Comorbid Impact Total Score (*re*: Table 3b). In Step Three of both models we note two interesting results, the first being that the entry of the Comorbid Number and Comorbid Impact Total Scores contributed significantly to the R^2 statistic, approximately 6% and 12%, respectively. The second finding, however, was that the measure of child ASD symptom severity (i.e., AIM Total) failed to retain its significance in the third block, suggesting an absence of independent effects.

The Mediation Hypothesis

To test the Mediation Hypothesis (i.e., Hypothesis 3) proposed by Siu et al. (2019), a path analysis was conducted (*re*: Fig. 3, standardised path coefficients). The model explained the covariation in the data sufficiently well (CFI = 0.98, SRMR = 0.026), with the observed and predicted correlations being close. However, a low TLI (= 0.77) is suggestive of model over-complexity or poor explanatory power relative to degrees of freedom. In terms of indirect effects, the analysis indicated siginficant paths to parenting stress through autism severity and: internalizing problems (B = 0.048, SE = 0.014, β = 0.052, p<.001);

Table 2 Test of the amplification hypothesis

(a) SDQ externalising behaviours								
Variable	Block 1		Block 2		Block 3		Block 4	
	B (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β
Constant	52.87 (1.80)		53.78 (1.78)		53.23 (1.61)		53.39 (1.61)	
Gender	−1.50 (1.92)	−0.04	−2.51 (1.89)	−0.06	−1.86 (1.71)	−0.05	−1.76 (1.71)	−0.04
Age	−0.02 (0.07)	−0.01	0.01 (0.06)	0.01	0.21 (0.07)	0.15***	0.21 (0.06)	0.15***
Children	−1.19 (0.61)	−0.10*	−1.16 (0.59)	−0.10	−1.35 (0.54)	−0.11*	−1.34 (0.54)	−0.11*
Emergence	0.30 (0.21)	0.07	0.27 (0.20)	0.06	0.31 (0.18)	0.07	0.29 (0.18)	0.07
AIM total			0.36 (0.078)	0.21***	0.07 (0.08)	0.05	0.08 (0.08)	0.05
SDQ externalising					1.49 (0.15)	0.48***	1.50 (0.15)	0.48***
interaction							−0.02 (0.02)	−0.06
Overall model	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)
	1.714	0.015	5.449***	0.58	22.814***	0.236	19.862***	0.240
	(4,444)	(0.015)	(5, 443)	(0.043)	(6, 442)	(0.179)	(7, 441)	(0.004)
(b) SDQ internalising behaviours								
Variable	Block 1		Block 2		Block 3		Block 4	
	B (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β
Constant	52.87 (1.80)		53.78 (1.78)		54.47 (1.69)		54.56 (1.70)	
Gender	−1.50 (1.92)	−0.04	−2.51 (1.89)	−0.06	−3.22 (1.79)	−0.08	−3.19 (1.79)	−0.08
Age	−0.02 (0.07)	−0.01	0.01 (0.06)	0.01	−0.03 (0.07)	−0.02	−0.03 (0.07)	−0.02
Children	−1.19 (0.61)	−0.01*	−1.16 (0.59)	−0.10	−1.17 (0.57)	−0.010*	−1.19 (0.57)	−0.10*
Emergence	0.30 (0.21)	0.07	0.27 (0.20)	0.06	0.10 (0.20)	0.02	0.10 (0.20)	0.02
AIM total			0.36 (0.08)	0.21***	0.12 (0.08)	0.07	0.12 (0.08)	0.07
SDQ internalising					1.39 (0.19)	0.35***	1.40 (0.19)	0.35***
Interaction							−0.01 (0.03)	−0.02
Overall model	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)
	1.714	0.015	5.449***	0.58	13.743***	0.157	11.805	0.158
	(4,444)	(0.015)	(5, 443)	(0.043)	(6,442)	(0.099)	(7,441)	(0.001)
(c) Comorbid impact total score								
Variable	Block 1		Block 2		Block 3		Block 4	
	B (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β
Constant	52.87 (1.80)	-	53.78 (1.78)	-	54.06 (1.69)	-	54.15 (1.69)	-
Gender	−1.50 (1.92)	−0.04	−2.51 (1.89)	−0.06	−2.79 (1.79)	−0.07	−2.73 (1.79)	−0.07
Age	−0.02 (0.07)	−0.01	0.01 (0.06)	0.01	−0.031 (0.066)	−0.02	−0.035 (0.066)	−0.03
Children	−1.19 (0.61)	−0.10*	−1.16 (0.59)	−0.10	−0.150 (0.566)	−0.12**	−1.52 (0.567)	−0.12**
Emergence	0.30 (0.21)	0.07	0.27 (0.20)	0.06	0.388 (0.194)	0.09	0.383 (0.194)	0.09
AIM total			0.36 (0.078)	0.21***	0.142 (0.081)	0.08	0.139 (0.081)	0.08
CITS					0.963 (0.132)	0.34***	0.980 (0.135)	0.35***
Interaction							−0.011 (0.015)	−0.03
Overall model	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)
	1.714	0.015	5.449***	0.58	13.872***	0.158	11.958	0.160
	(4,444)	(0.015)	(5, 443)	(0.043)	(6,442)	(0.101)	(7,441)	(0.001)

Hierarchical regression analyses predicting the relationship between child core ASD symptoms (AIM Total) and parenting stress scale (PSS) scores when the moderating variable was child Externalising Behaviours (Table a), Internalising Behaviours (Table b), or Comorbid Impact (Table c)

B = unstandardised regression coefficient, *β* = standardised regression coefficient, *SE* standard error, Gender = participant gender, Age = participant age, Children = number of children in the family, Emergence = age of child when ASD symptoms first emerged, *AIM Total* Autism impact measure total score, *SDQ* Strengths and difficulties questionnaire, *CITS* Comorbid impact total score

*p < .05; **p < .01; ***p < .001

Table 3 Test of the additive hypothesis

(a) Number of comorbidities

	Block 1		Block 2		Block 3	
Variable	*B* (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β
Constant	53.32 (4.21)	-	42.32 (5.62)	-	40.27 (5.47)	-
Gender	−1.50 (1.92)	−0.04	−1.41 (1.91)	−0.04	−1.93 (1.85)	−0.05
Age	−0.018 (0.07)	−0.01	0.021 (0.071)	0.02	−0.012 (0.069)	−0.01
Children	−1.20 (0.61)	−0.10	−1.15 (0.614)	−0.09	−1.39 (0.588)	−0.11*
Emergence	0.304 (0.208)	0.07	0.395 (0.209)	0.09	0.447 (0.203)	0.10*
AIM total			0.114 (0.039)	0.14**	0.065 (0.039)	0.08
Number of comorbidities					2.87 (0.541)	0.25***
Overall model	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)
	1.714 (4,444)	0.015 (0.015)	3.105** (5, 443)	0.034 (0.019)	7.440 (6,442)	0.092 (0.058)

(b) Comorbid impact total score

	Block 1		Block 2		Block 3	
Variable	*B* (*SE*)	β	*B* (*SE*)	β	*B* (*SE*)	β
Constant	53.32 (4.21)	-	42.32 (5.62)	-	45.07 (5.28)	-
Gender	−1.50 (1.92)	−0.04	−1.41 (1.91)	−0.06	−2.44 (1.79)	−0.06
Age	−0.018 (0.07)	−0.01	0.021 (0.071)	0.02	−0.039 (0.067)	−0.03
Children	−1.20 (0.61)	−0.10	−1.15 (0.614)	−0.09	−1.53 (0.568)	−0.13**
Emergence	0.304 (0.208)	0.07	0.395 (0.209)	0.09	0.419 (0.196)	0.10*
AIM total			0.114 (0.039)	0.14**	0.012 (0.039)	0.01
CITS					1.03 (0.131)	0.37***
Overall model	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)	*F* (*df*)	R^2 (ΔR^2)
	1.714 (4,444)	0.015 (0.015)	3.105** (5, 443)	0.034 (0.019)	13.285 (6,442)	0.153 (0.119)

Comorbidities represented by frequency (Table a) or their cumulative impact on the child (Table b)

B=unstandardised regression coefficient, *β*=standardised regression coefficient, *SE* standard error, *AIM Total* Autism impact measure total score, Gender=participant gender, Age=participant age, Children=number of children in the family, Emergence=age of child when ASD symptoms first emerged, *CITS* Comorbid impact total score

*p<.05; **p<.01; ***p<.001

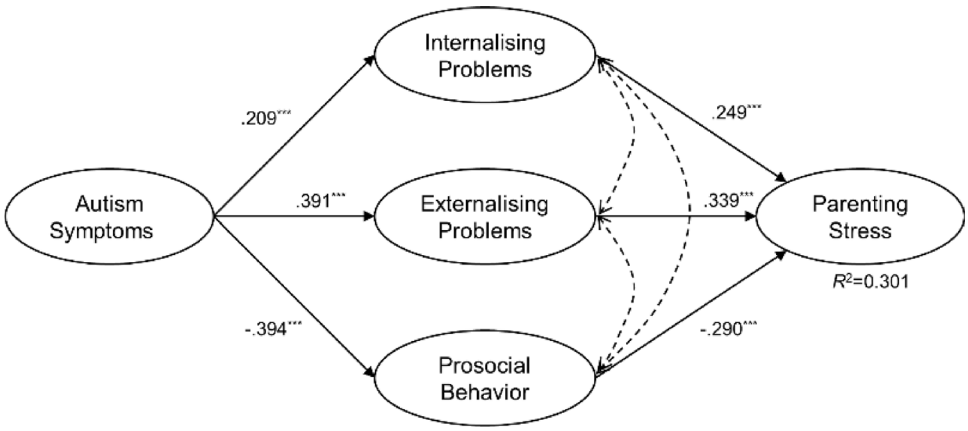

Fig. 3 Test of the mediation hypothesis. An adaptation of Siu et al.'s (2019) model and applied exclusively to the ASD context. Standardised coefficients are from the current study, and for the sake of clarity covariance statistics are not reported in the figure

externalizing problems (B=0.124, SE=0.021, β=0.133, p<.001), and; prosocial behaviours (B=0.106, SE=0.019, β=0.114, p<.001). Of note, the direct effect between AIM Total scores on PSS scores remained significant (B=0.128, SE=0.042, β=0.137, p=.002), indicating that partial mediation was occurring. Overall, the model accounted for 30% of the variability in PSS scores.

Discussion

Although the impact of comorbid conditions on child function and parenting factors are increasingly being reported in the ASD-related literature, little commentary has considered the underlying mechanism by which comorbidities may influence parenting stress. The aim of the current study was

to describe and test three potential mechanisms, namely the Amplification, Additive, and Mediation Hypotheses.

The Amplification Hypothesis

To begin, the current study found no evidence supporting the Amplification Hypothesis, which argues that an autistic child's comorbid conditions serve to aggravate their core ASD symptoms which in turn increase parenting stress. Consistent across the seven moderation analyses, the Amplification Hypothesis was not supported irrespective of whether the moderator was continuous (i.e., internalising vs. externalising behaviours) or binary (i.e., yes/no responses to five specific comorbidities). This Hypothesis assumes a positive relationship between the severity of comorbidities and ASD core symptoms, which in the current study were observed when considering both internalising and externalising behaviours (*re*: Table 1). However, when the Autism Diagnostic Observation Schedule (ADOS) is used by clinicians to assess core symptoms, studies have failed to report an association between severity of child ASD and the degree of child internalising and externalising behaviours (e.g., Lin et al., 2021; Mello et al., 2022). Such findings challenge the Amplification Hypothesis, and the associations reported in the current study may be an artefact of relying on parent-reported data. Of relevance, this lack of association is not reported when parent, as opposed to clinician, ratings are used, such as the Childhood Autism Rating Scale (CARS) completed by mothers in the Mello et al. (2022) study.

In the course of testing the Amplification Hypothesis a further finding emerged that is worthy of commentary. For the three models it was observed that when the internalising (*re*: Table 2a) or externalising (*re*: Table 2b) behaviours variables were entered, or the Comorbid Total score (*re*: Table 2c), the significant relationship between the AIM Total scores and the PSS scores was lost. Though statistically significant, initial bivariate analysis indicated that the relationship between the Total AIM and PSS variables was weak to begin with (*re*: Table 1), and thus at risk when exposed to multivariate analyses. One explanation is that estimates of the relationship between parenting stress and child autism severity are weak as they are vulnerable to how either stress (Shepherd et al., 2018) and child autistic symptoms (Mello et al., 2022) are measured. An alternative interpretation, and one strongly argued in the literature (e.g., Zaidman-Zait et al., 2017; Olson et al.,2022), is that parenting stress may be better predicted by the child's challenging behaviours, and especially externalizing behaviours (McStay et al., 2014).

The Additive Hypothesis

In contrast to the multiplicative mechanism of the Amplification Hypothesis, the Additive Hypothesis posits that the combined effect of multiple stressors is equal to the sum of their individual effects, assuming an absence of interaction. Our analyses found some support for this approach, with either the total number of child comorbid conditions (*re*: Table 3a) or the total score for the impact of comorbid conditions on child function (*re*: Table 3b) significantly increasing the variance explained statistic. However, in these two models the elimination of statistical significance of the AIM Total predictor in the third block indicated an absence of independent effects. This finding may reflect a loss of unique variability due to diagnostic overlap, which introduces shared variance across variables. For example, anxiety disorders and ADHD are both associated with sensory sensitivities commonly found in ASD (e.g., Carpenter et al., 2019).

Further evidence undermining the veracity of Additive Hypothesis comes from the data presented in Fig. 2, and subsequent inferential testing. Specifically, the model would predict that as the number of child comorbid conditions increases, so too should parenting stress. Here, statistical significance was only noted between parents reporting their child has one comorbidity and those reporting two-or-more comorbidities. Alternatively, it may be that the assumption of no interaction effects between predictors is incorrect, though evidence of such could not be marshalled from the moderator analyses presented in Table 2. In the scheme of the Additive Hypothesis a child's core ASD symptoms or co-occurring conditions would independently contribute to parenting stress and there are no synergistic effects, however, the literature does not always support such assumptions (Bashore et al., 2025). For example, anxiety amplifies the effect of sensory sensitivity through hypervigilance, then a non-additive (interactive) model such as the Amplification Hypothesis would be more appropriate. In the current study evidence definitively supporting either of the Amplification or Additive Hypotheses was lacking.

The Mediation Hypothesis

Siu et al. (2019) hypothesized that child ASD symptom severity increases parenting stress, but that comorbid child behaviours might be the underlying mechanism. Further to this line of reasoning, they conjectured that prosocial behaviours by autistic children increased parent well-being by acting as an intervening variable between core ASD symptoms and parenting stress. Such a proposition is not without support in the existent literature, with Huang et al. (2014) reporting that challenging behaviours tended to drain

parents of their mental energy while prosocial behaviours displayed by their autistic child enhanced parent mental well-being. In the current study we obtained significant indirect effects (*re*: Fig. 3), and hence some support for the Mediation Hypothesis. However, an unexpected finding in light of the results obtained with the regression analyses (*re*: Tables 2 and 3) was a significant direct effect between child ASD core symptoms and parenting symptoms. Here, the more sophisticated path analyses indicated that child challenging behaviours do not fully explain the impact of ASD symptoms on parenting stress, and that mediation is partial and not full.

The model coefficients reported by Siu et al. (2019) and those in the current study are generally comparable, despite some methodological differences between the two studies. An interesting difference in the current study is that, for internalising behaviours, the path coefficient to parenting stress is slightly larger than that from ASD symptoms to internalising behaviours, while this pattern is not noted for the externalising- or prosocial-behaviours paths. This finding may indicate that when a child experiences depression or anxiety (or other internalising behaviours), the emotional impact on parents is more substantial, even compared to how strongly autism severity predicts internalising behaviours in the child. Parents are emotionally distressed by their autistic child's sadness, worry, withdrawal, or hopelessness, which are emotionally taxing and can increase aspects of parenting stress (Lecavalier et al., 2006). In contrast, externalizing and prosocial behaviours are more behaviourally rooted in autism. Although their contribution to parenting stress is still important, they are generally less emotionally intense or distressing for parents than internalising behaviours.

In Siu et al.'s sample, child age ranged from 6 to 11 years (M_{age} = 8.4), whereas the current study included a broader age span, extending to 20 years of age. However, neither study captures the dynamics between parenting stress and child behavioural challenges. For example, the literature indicates that in Autistic individuals, internalising behaviours tend to increase during puberty and young adulthood (Gotham et al., 2015), while externalising behaviours typically decrease over time (Adams et al., 2023). Yet, both the current analysis and that of Siu et al. (2019) fail to adequately document these developmental shifts. Pertinently, although parenting stress appears relatively stable, the underlying risk factors may be changing.

Finally, while Siu et al. (2019) reported significant indirect paths supporting the Mediation Hypothesis, there are two points of difference from the current study. First, they used a mixed sample of Autistic and neurotypical children, and second, they appear to only report the strengths of indirect effects without consideration of whether the mediation is partial or full. A possible reason for this neglect may be the theoretical implications of full mediation, especially in terms of causality, or the existence of confounding variables masking the relationship between child ASD symptom severity and parenting stress, such as intervention history, types of social support, or parenting self-efficacy. Secondly, collinearity between ASD symptoms and comorbidities may occur due to shared symptoms (Siu et al., 2019), to the degree that some have questioned in the context of internalising behaviours whether "…ASD and anxiety are even distinct diagnoses" (Zaboski & Storch, 2018: p.32).

Limitations and Future Directions

The main limitation of the current study is its cross-sectional design, and the associations reported in the current study should be interpreted as correlational only. Because all variables were measured at a single point in time, the directionality of relationships between child autism severity, co-occurring conditions, and parenting stress cannot be definitively determined. Although emerging longitudinal evidence suggests that parenting stress may predict later child internalising and externalising behaviours (e.g., O'Connor et al., 2025), the literature remains mixed, with some studies indicating confounding gender effects (Rodriguez et al., 2019) and others reporting conflicting findings (e.g., Paynter et al., 2025). There is a clear need to replicate and extend longitudinal work (e.g., Zaidman-Zait et al., 2017) to better understand the temporal and potentially bidirectional relationships among these variables.

A further limitation is the sample profile, which being relatively homogenous and biased towards well-educated female participants may limit the generalisability of the data. As such, it is recommended that future studies aim to recruit a more diverse sample, including greater representation of fathers, individuals from lower socio-economic backgrounds, and participants from a wider range of cultural groups.

The reliance upon parent ratings of their child's characteristics and their own psychological well-being may also serve to bias the data and hence the study's findings. The advantages of parent-reported vs. clinician-reported data come from the greater amount of time parents spend with their autistic child, particularly in the family home, giving greater ecological validity. However, for a number of reasons parent reports may reflect biases, and so multi-informant designs including clinicians and teachers are recommend. Additionally, parent-reports were relied upon for information regarding their child's comorbidities, and the data analysed without respect to whether a formal diagnosis had been concerned. As a shortcoming it must be weighed against the potential for a child to go undiagnosed on account of parent

fear of their child being labelled, a mistrust of treatments, restricted access to clinicians, or diagnostic 'overshadowing' (Palmer et al., 2024).

It has been opined that comorbid and co-occurring conditions in the ASD context have been understudied (e.g., Mannion et al., 2023, 2024; Lanyi et al., 2022; Liu et al., 2021). In relation to both child and parent well-being the Transactional Model (Hastings, 2002) argues the importance of understanding how comorbid behaviours manifest and how they relate to parenting stress. In light of the findings emerging from the mediation analysis, child challenging behaviours explain part of why autism symptoms raise parenting stress, but other factors are likely involved, including parent resilience and coping strategies, or external support, and so a larger model incorporating other child and parent factors would be informative. Further, while the mediation in the current study and that of Siu et al. (2019) focused on challenging behaviours, future studies employing more comprehensive measures of conditions such as child sleep and gastrointestinal issues would be beneficial, as these have been found to interact (e.g., Bashore et al., 2025). As such, broader comorbidity assessments should be performed in order to construct more elaborate multivariate models.

Clinical Implications and Conclusion

The main clinical implication of the current findings concerns the relative potency of child comorbid conditions relative to core ASD symptom severity. While the primary diagnosis is often the centre of attention when developing an intervention plan, best practice argues for the detection and surveillance of co-occurring conditions in order to maximise treatment efficiency (Lord et al., 2018). Comorbid conditions add an additional layer of complexity to clinical presentations, and as such introduce important considerations at the level of practice. First, clinicians must be careful to avoid diagnostic overshadowing, by which clinicians attribute a child's symptoms or behaviours to their primary ASD diagnosis, without the consideration of other potential conditions (Palmer et al., 2024). Given that comorbid conditions can influence developmental outcomes independently, their identification and inclusion into intervention plans is important, which brings us to the second consideration. The high prevalence of comorbid conditions in autistic children argues strongly for a multidisciplinary and integrated approach to assessment and support. Muskens et al. (2017) go further, arguing that modifiable risk factors of comorbid conditions should also be identified and addressed in order to reduce symptoms and improve the quality of life of both child and parent.

A further implication emerging from the current study is that while comorbid conditions clearly have a strong influence on parenting stress, the results of our mediation analysis (*re*: Fig. 3) argues that interventions targeting core ASD symptoms can also serve to protect parents from care-related stressors and thus preserve the parent-child dyad. Zaidman-Zait et al. (2017) likewise came to the same conclusion, stating that early interventions need to target comorbid conditions in addition to core ASD symptoms. Parenting stress can adversely affect child behaviour and well-being (O'Connor et al., 2025), and given that ASD treatments are often intensive, expensive, and rely upon parent delivery (DePape & Lindsay, 2015), they need to be tailored to not only address child symptoms but also to reduce parenting stress.

In conclusion, comorbidities in the ASD context present diagnostic and treatment challenges, are linked to adverse child health outcomes, and amplify health care costs (Li et al., 2020). To effectively respond to the physical and psychological needs of their autistic child, parents must effectively parent, a task which is adversely affected by stress (Bonis, 2016). While core symptoms lead to diagnosis, there are also comorbid conditions that are common amongst autistic children that can perpetuate parenting stress above and beyond ASD core symptoms. In the current study we explore the mechanisms by which comorbid conditions effect parenting stress, finding that the Amplification Hypothesis was not supported, limited support for the Additive Hypothesis, and partial support for the Mediation Hypothesis. Understanding these underlying mechanisms can inform the development of interventions designed to reduce parental stress and augment the quality of life of autistic children.

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Declarations

Conflict of interest The authors declare they have no conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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