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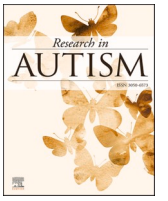

The presence and impact of Autistic child comorbid conditions and their relationship to parent well-being

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ABSTRACT

Purpose: The diagnosis of Autism Spectrum Disorder is rising globally, and as a long-life condition associated with high support needs, parents of Autistic children experience greater parenting stress and lower quality of life than parents raising typically developing children. However, while research has investigated the relationship between the severity of child autism symptoms and parenting stress, studies into comorbid conditions that likewise impair child function are not as common and often focus on a small subset of conditions. The aim of the current study was to estimate the frequency of the five most common comorbidities reported in the autism literature (Anxiety, ADHD, Intellectual Disability, Gastrointestinal Issues, Sleep Disorder) and relate them to parenting stress and health-related quality of life (HRQOL).

Methods: Using an internet-based survey, parent reports of their Autistic child's comorbid conditions and the impact these have on their child's function, parenting stress, and parental HRQOL were obtained from 453 parents residing in New Zealand. A global measure of parenting stress was obtained using the 18-item Parenting Stress Scale, while HRQOL ratings were obtained using the 36-Item Short Form Survey (SF-36).

Results: While many parents indicated the presence of comorbid conditions in their Autistic child, a substantial proportion were not formally diagnosed. A Linear Mixed-Effects Model indicated that child anxiety, Intellectual Disability, and ADHD had the greatest impact on both child and parent, however, subsequent multivariate analyses clarified that sleep disorder and Gastrointestinal Issues had the largest effect on parental stress and HRQOL, followed by ADHD. This result was robust irrespective of whether parents were asked if the comorbidity was present (vs. absent), diagnosed (vs. undiagnosed), or when related to child (i.e., functional) and parent (i.e., stress) impact.

Conclusion: Evidence that child sleep disorder, Gastrointestinal Issues, and ADHD are most detrimental to parental well-being indicate that interventions targeting these comorbidities should be prioritised. Coupled with increased child function as a direct result of intervention, better parental outcomes should increase child well-being and family quality of life, indicating that future research into the diagnostic barriers associated with comorbid conditions would be useful.

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1. Introduction

Autism Spectrum Disorder (ASD) is a form of neurodivergence classified by the DSM-5-TR as a neurodevelopmental disorder (American Psychiatric Association, 2013). Emerging in early childhood, the characteristics of autism are described by two primary domains of symptoms: 1) persistent deficits in social communication and social interaction across multiple contexts, and 2) Restricted, repetitive patterns of behaviour, interests, or activities. The DSM-5-TR requires that presentations are not better explained by intellectual disabilities, acknowledging that core ASD symptoms are not caused by intellectual disability, but rather that intellectual disabilities may co-occur with ASD. Finally, the DSM-5 specifies that other co-occurring conditions (e.g., Attention Deficit Hyperactivity Disorder (ADHD), anxiety, sleep dysfunction) need to be accounted for based on their commonality with the ASD diagnosis.

Raising a child is a challenging task that most parents adjust to (Hayes & Watson, 2013), though parenting a child with a disability is associated with greater reports of inconsistent and dysfunctional parenting, particularly with a child diagnosed with ASD (Rudelli, Straccia, & Petitpierre, 2021). Parenting difficulties are traditionally conceptualised within the coping-and-stress framework (Folkman & Lazarus, 1985) and captured using parenting stress measures. Parenting stress is the distress experienced when the demands of the parenting role exceed the parent's perceived resources to cope with those demands (Abidin, 1995). Decades of research has attempted to estimate the relationship between an Autistic child's core symptoms and parenting stress, with a plethora of inconsistent findings being reported (Buchwald et al., 2025). Research into the primary sources of parenting stress have focused primarily on either the core symptoms of ASD or challenging behaviours (Lin, Iao, Lee, & Wu, 2021; Weiss, Cappadocia, MacMullin, Viecili, & Lunsky, 2012), the latter of which are categorised as comorbid (or co-occurring) conditions[1].

In a diagnostic context, comorbidity refers to the presence of one or more additional medical or psychiatric conditions that co-occur alongside a person's primary diagnosis (van den Akker et al., 1996). Comorbidities that commonly co-occur with ASD include sleep disorders, gastrointestinal dysfunctions, intellectual disability, seizures, hormonal issues, and metabolic disorders (Bauman, 2010; Bougeard, Picarel-Blanchot, Schmid, Campbell, & Buitelaar, 2021; Silva & Schalock, 2012; van Steensel, Bögels, & de Bruin, 2013). Furthermore, ASD is also frequently diagnosed with concurrent psychiatric and mental health disorders, most of which are subsumed under the two broad categories of internalising or externalising behaviours. Internalising behaviours are those which are privately experienced by the individual (Belardinelli, Raza, & Taneli, 2016), and common examples in the ASD context include anxiety, emotional dysregulation, depression, and withdrawal (Jensen & Steinhausen, 2015). Externalising behaviours are defined as behaviours projected outwards and are visible to others, and with ASD typically manifest as impulsivity, hyperactivity, aggression, defiance, and other conduct issues (Lin et al., 2021; Mello, Rivard, Morin, Patel, & Morin, 2022). Of note, parents of children with ASD tend to report higher levels of internalising and externalising behaviours than parents of typically developing children (Siu et al., 2019).

In terms of estimated comorbid occurrence, ADHD is the most common condition, affecting approximately 28 %-35 % of Autistic children (Khachadourian et al., 2023; Simonoff et al., 2008), though some non-clinical estimates put the rate closer to 70 % (Mayes, Calhoun, Baweja, & Waschbusch, 2021). Using the Strengths and Difficulties Questionnaire to measure externalising behaviours exhibited by Autistic children, Grasso, Lazzaro, Demaria, Menghini, and Vicari (2022) reported that the highest mean scores occurred in the hyperactivity domain and the lowest mean scores with conduct problems. Anxiety disorders, classified as an internalising behaviour, present another commonly occurring comorbidity with ASD (Lord, Elsabbagh, Baird, & Veenstra-Vanderweele, 2018), with estimates reaching 42 % depending on measurement criteria, information source, and symptom specification (Mayes et al., 2021; Mohammadi et al., 2019). An additional neurodevelopmental condition that is prevalent in about 30 % of Autistic children is the simultaneous presence of an intellectual disability (Polyak, Kubina, & Girirajan, 2015). Furthermore, around 11 % of children with ASD complain of frequent stomach-ache and/or nausea (Mayes et al., 2021), with some research indicating gastrointestinal issues in between 13.8 % and 36.8 % of Autistic children (Lanyi, Mannion, Chen, & Leader, 2022). Finally, children with ASD are significantly more likely to present with dysfunctional sleep patterns than typically developing children (Li, Liu, Chen, Bi, & Liang, 2020), with estimates of severe sleep problems reported to be between 25–40 % in this group (Sivertsen, Posserud, Gillberg, Lundervold, & Hysing, 2012; Soke, Maenner, Christensen, Kurzius-Spencer, & Schieve, 2018).

An Autistic child's comorbid conditions can adversely impact parent outcomes, with a number of cross-sectional studies indicating that externalising behaviours are the dominant contributors to parenting stress (e.g., Bonis, 2016; Huang et al., 2014; Lovell & Wetherell, 2016). One study reported that the severity of both internalising (i.e., anxiety) and externalising (i.e., ADHD) behaviours exhibited by an Autistic child were associated with higher parenting stress (Lanyi et al., 2022), but typically it is externalising behaviours that have the strongest relationship (Zaidman-Zait et al., 2017). Longitudinal studies (e.g., Rodriguez et al., 2019; Totsika, Hastings, Emerson, Berridge, & Lancaster, 2015; Zaidman-Zait et al., 2014) investigating causal (i.e., non-, uni-, or bi-directional) relationships between challenging child behaviour and parenting stress have yielded inconsistent findings, and causation may be dependent on the developmental stage of the Autistic child (Lin et al., 2021). However, evidence to support the transactional relations model (Lin et al., 2021; Hastings, 2002), in which challenging child behaviours and parenting stress form a positive feedback loop that creates a deteriorating spiral terminating with the parent unable to effectively self-regulate, has come from a number of studies (e.g., Lecavalier, Leone, & Wiltz, 2006; Osborne & Reed, 2010).

While the bulk of the parenting stress literature has focused upon externalising and internalising behaviours exhibited by Autistic children, a small number of studies have reported the effects of other co-occurring conditions. For example, intellectual disability is a

[1] Throughout the manuscript we use the term 'comorbidity' to retain consistency across the literature.

risk factor for lower parental well-being, though is not always an independent predictor once other factors such as ASD severity and challenging behaviours are controlled for (e.g., Totsika, Hastings, Emerson, Lancaster, & Berridge, 2011). In a review, Martin, Papadopoulos, Chellew, Rinehart, and Sciberras (2019) analysed six studies reporting the association between child sleep problems, parenting stress and parent mental health, concluding that child sleep dysfunction was associated with poorer parent mental health and higher parenting stress. While a review by Enea and Rusu (2020) reported conflicting results regarding the relationship between child sleep problems and parenting stress, more recent studies seem to confirm the link between impaired child sleep and parenting stress (e.g., Mannion & Leader, 2023; Mannion, Whelan, & Leader, 2024). Responding to the focus on challenging behaviours and a dearth of studies on other comorbid conditions, Mannion and Leader (2024) obtained parent-reports of gastrointestinal issues in their Autistic children and linked them to measures of parenting stress. Specifically, they reported a positive correlation between GI Symptoms Inventory scores and parenting stress. In an earlier intervention study evaluating the effectiveness of probiotic therapy with Autistic children, Guidetti et al. (2022) noted significant reductions in pre-post parenting stress scores, providing further evidence of a link between child gastrointestinal problems and parenting stress.

Beyond parenting stress, another common outcome measure encountered in the literature is health-related quality of life (HRQOL), which assesses how an individual's physical, mental, and social well-being are affected by their health. Caregiving demands are typically higher for parents raising an autistic child, and when combined with elevated parenting stress and concerns about their child's future, are linked to poorer physical and mental health outcomes (Hayes & Watson, 2013). In the ASD context, Lanyi et al. (2022) reported negative associations between child comorbid psychopathology and parental HRQOL, while John, Lam-Cassettari, Dissanayake, and Eapen (2025) similarly reported a proportional relationship between child adaptive functioning and parental quality of life. Performing a network analysis on parent-reported data, Shepherd, Buchwald, Siegert, and Vignes (2024) found that both child ASD symptoms and externalising behaviours were predictors of psychological HRQOL, but that only child symptoms had a direct effect on physical HRQOL.

2. The current study

The ASD-related literature argues that the existence of child comorbid conditions adversely effects parent outcomes (e.g., Lovell & Wetherell, 2016), which in turn can negatively affect the child via transactional effects. Furthermore, there have been calls to elucidate the factors contributing to parenting stress in the ASD context in order to develop more targeted interventions able to support families with Autistic children and deliver better outcomes for Autistic children (Hayes & Watson, 2013; Weiss et al., 2012). Best practice dictates that comorbidities presenting in Autistic children should be systematically investigated and treated (Lord et al., 2018) and given the reliance upon parents to deliver ASD-related interventions (DePape & Lindsay, 2015), these additional child conditions become an important factor when considering parenting stress and parent well-being.

While a substantial amount of research has been conducted on the relationship between child core-ASD symptoms and parenting stress, fewer studies have focused on child comorbidities, and then typically on challenging behaviours such as ADHD or anxiety. Furthermore, while a small number of existent studies focus on sleep dysfunction and gastrointestinal issues, they typically investigate the comorbidity in isolation, and thus do not account for potential interactions or symptom overlap between comorbid conditions. In addition, the literature consistently reports the barriers parents face to obtain a diagnosis for their autistic child (Crane, Chester, Goddard, Henry, & Hill, 2016), and the same is likely true for their child's comorbid conditions. As such, while comorbidity research often includes Autistic children with a conferred diagnosis of one-or-more concurrent conditions, those with subclinical symptoms or without a formal diagnosis are excluded. To bridge a gap in the literature, the current study asks parents if they judge their Autistic child to have one-or-more comorbidities, in addition to inquiring if a formal diagnosis has been granted.

Research directly examining the impact of Autistic children's comorbid conditions on parent outcomes is limited and tends to focus primarily on either internalising or externalising behaviours (Davis & Carter, 2008), or comorbid conditions in isolation, eliciting calls for further research in this area (Lanyi et al., 2022; Liu et al., 2021; Mannion et al., 2023, 2024). The current study relied on parent report and was guided by three objectives: (1) to further document the presence of comorbidities associated with a diagnosis of ASD; (2) to document the impact of these comorbidities on child function and parenting stress; (3) to explore the relationship between Autistic child comorbid conditions and parent HRQOL, and; (4) undertake bivariate and multivariate analyses to explore how comorbidities differentially contribute to parenting stress and HRQOL.

3. Method

3.1. Participants

A total of 404 females (M_{age} = 50.3 years, SD = 9.45) and 49 males (M_{age} = 44.5 years, SD = 8.85) who reported parenting an autistic child that was either male (n = 348, M_{age} = 12.6 years, SD = 6.86) or female (n = 101, M_{age} = 12.8 years, SD = 6.60) participated in the study. In terms of education, 64 (14.2 %) participants reported having finished secondary school, 147 (32.5 %) achieved a qualification from a technical college, and 241 (53.3 %) possessed a university degree, indicating that the sample was relatively well educated. Most participants identified primarily as European / Caucasian (n = 363, 80.8 %), and approximately 1-in-5 (n = 96, 21.2 %) reported that they were single (i.e., solo) parents. For their children, the mean age at which parents first noted ASD-like traits was 1.76 years (SD = 1.49) and the mean diagnostic age was 4.92 years (SD = 2.92), with the majority of diagnoses being conferred by a paediatrician (n = 285) or either a psychologist or psychiatrist (n = 121).

3.2. Measures

3.2.1. Comorbidity measures

The presence of one-or-more comorbidities was represented using binary variables, for example, *does your child experience anxiety*. In the case of binary variables, parents were asked to indicate whether their Autistic child possessed one-or-more of the following comorbid conditions: anxiety disorder (anxiety), attention-deficit-hyperactivity disorder (ADHD), intellectual disability (ID), gastro-intestinal (GI) issues, or a sleep disorder. These conditions were selected from Lanyi et al. (2022) and Khachadourian et al. (2023) on the basis of their high estimates, and with anxiety and ADHD representing internalising and externalising behaviours, respectively. Each comorbidity was presented serially, and parents who selected 'present' were immediately asked if a professional diagnosis had been obtained (e.g., *Has your child's ADHD been formally diagnosed?*). Irrespective of diagnostic status, two five-point scale items were then presented, asking the parents to rate the degree to which the comorbidity interferes with their child's everyday functioning (e.g., *How much does your child's ADHD interfere with their everyday functioning?*) rated from 'very mildly' to 'very severely', and how much the comorbidity contributes to their parenting stress levels (e.g., H*ow much does your child's gastrointestinal problems contribute to your levels of parenting stress?*) rated from 'not at all' to 'extremely'. These two items are denoted 'Child-Impact' and 'Parent-Impact', respectively, so as not too cause confusion with the Parenting Stress Scale that is also reported in the current study. Note that participants who failed to endorse a specific comorbidity weren't asked to provide a Child- or Parent-Impact rating, however, for the regression analyses only they were automatically assigned a value of zero with the Child- and Parent- Impact scores. This was done on the basis that this comorbidity was not affecting child function or contributing to parenting stress given it was not present.

3.2.2. Health-related Quality of Life (HRQOL)

The SF-36 Short-Form Medical Outcomes Scale (Ware & Sherbourne, 1992) was employed to estimate parent HRQOL. The SF-36 contains 36 items which can be condensed into two summary measures: the Physical Component Summary (PCS) probing how well an individual can physically function in daily life and the impact of physical conditions on day-to-day activities, and the Mental Component Summary (MCS) measuring psychological quality of life and limitations due to emotional dysregulation. Four domains are used to calculate the PCS (Physical Function, Role Physical, Bodily Pain, General Health) and the MCS (Vitality, Social Function, Role Emotion, Mental Health). Prior to calculating the PCS and MCS each of the eight domains were transformed to a 0 (worst HRQOL) to 100 (best HRQOL) scale, allowing the two summary measures to be calculated using standardised scoring algorithms (Ware, Koskinski & Keller, 1994) and the New Zealand weightings published by Frieling, Davis, and Chiang (2013). In their final form, the PCS and MCS scores below 50 can be interpreted as below average HRQOL and scores above 50 interpreted as above average HRQOL. In the current study the Cronbach's alpha coefficients were $\alpha_c = .915$ and $\alpha_c = .907$ for the PCS and the MCS, respectively.

3.2.3. Parenting stress scale

The Parenting Stress Scale (PSS) is an 18-item self-rated scale that contains both positively and negatively worded statements estimating stress levels attributable to raising children (Berry & Jones, 1995). In the current study the PSS was used to estimate stress associated with caring for an Autistic child, and parents responded to each question using a 5 point Likert-type scale (1 = strongly disagree to 5 = strongly agree). Following reverse coding, total scores were computed, with higher scores indicating greater levels of stress. The overall Cronbach's coefficient obtained in the current study was acceptable (α_c =.905).

3.2.4. Procedure

Participants were recruited with the assistance of national autism support agencies within New Zealand, who advertised the study on their social media outlets. Upon clicking a link to a Qualtrics survey, participants were introduced to the study and its inclusion criteria, and were invited to complete the survey after reading a participant information page. Participants were informed that engagement was voluntary and that they would remain anonymous, and that ethical approval had been sought and granted by the University's Ethics Committee.

3.3. Statistical analysis

An initial frequency analysis determined the proportions of the five comorbidities in the sample (absent vs. present) and, if present, the number of endorsees who had obtained a formal diagnosis for their child (yes vs. no). For participants who indicated that their child experienced a specific comorbidity (i.e., present), item means and standard deviations for the Child Impact and Parent Impact items were calculated, in addition to the calculation of a Pearson's correlation coefficient (denoted r_{Child_Parent}). To ascertain whether there were statistical differences in the mean Child Impact or Parent Impact ratings across the five comorbidities a linear mixed-effects model (LMM) was utilised. This approach was chosen as not all participants contributed data for all comorbidities, and so the design is intrinsically unbalanced. In these models the main effects were comorbidity (5 levels) and diagnostic status (2 levels), while the interaction term between the two was also scrutinised (i.e., comorbidity x diagnosis). The LMM was conducted in R using the lme4 package to obtain fixed effects, and the lmerTest package to obtain significance values. To determine if Child Impact and Parent Impact means differed between those whose children had a formal diagnosis and those who had not, a battery of Welch's *t*-tests were conducted with the application of Tukey adjusted *post-hoc* tests.

Next, descriptive analyses provided summary statistics (means, standard deviations, Cronbach's alpha: α_c) for composite variables (i.e., the PSS, the SF-36 PCS and MCS scales), and was accompanied by a correlational analysis to examine the patterns of variability between these variables (*re*: Supplementary Table 1). Additional Welch corrected *t*-tests (*re*: Supplementary Table 2) were performed

on the composite variables (i.e., PSS, PCS, MCS) across the five comorbidities, with the grouping variable being present and diagnosed vs. present and undiagnosed. Here, Cohen's d was used to quantify effect size, with $d < 0.2$ indicating a very small effect, $d = 0.5$ a moderate effect size, and $d > 1.2$ indicating a very large effect.

To gauge the unique contribution of each child comorbid condition to parenting stress, a series of simultaneous multiple linear regression (MLR) analyses was performed. Simultaneous MLR analyses were selected to determine how each variable uniquely contributes to the variance in parental well-being while adjusting for the effects of the others, given that some of comorbidities may interact (e.g., GI and anxiety). For each MLR analysis the dependent variable was either PSS, SF-36 PCS, or SF-36 MCS scores. Predictors consisted either of the five comorbidities encoded as dummy variables (0 = absent, 1 = present), or represented as their impact on child function (i.e., Child Impact) or parenting stress (i.e., Parent Impact). For those analyses in which the dependent variable was PSS scores the Parent Impact scores were not used because of collinearity. Finally, all MLR analyses contained a selection of covariates that we identified during an initial exploratory analysis of the data, involving both univariate and bivariate analyses. Consequently, to reduce model complexity and avoid overfitting only the following covariates were entered into the model: parent age and gender, number of children in the household, and age of symptom emergence (denoted ASD Emergence).

All analyses were undertaken in R (v. 4.4.2), with test assumptions scrutinised using Tabachnick and Fidell's (2019) guidelines.

4. Results

4.1. Frequency of comorbidities

Parental endorsements of their Autistic child's comorbidities are tabulated in Table 1, with the most endorsed condition being anxiety and the least being ID. Of note, nearly half (47.9 %) of parents endorsing anxiety indicated that their child had not yet been diagnosed by a medical professional, a rate exceeded only by GI (55.7 %). Fig. 1 is a histogram displaying the total number of comorbidities reported by parents, with 103 parents (≈ 23 %) reporting no comorbidities, only two parents endorsing all five, and with a single comorbidity being the most common ($n = 138$).

4.2. Child and parent impact of comorbid conditions

Parent-rated Child Impact and Parent Impact means are reported in Table 1 for each comorbidity, with each mean having a theoretical range of 1–5. The correlations between Child- and Parent- Impact scores ranged from moderate-to-large. A Linear Mixed-Effects Model (LMM) identified a main effect of comorbidity type on Child Impact ratings, with Tukey adjusted *post-hoc* tests indicating that the mean ratings for anxiety, ADHD, and ID were significantly higher than those for GI or sleep disorder (all $p < .05$), while not differing between themselves (all $p > .05$). The LMM analysis was repeated using the Parent Impact ratings, with mean scores for GI being less than anxiety, ADHD, ID, and sleep disorder, while both anxiety and ADHD had significantly higher means than sleep disorder. For both models a main effect of diagnosis was obtained ($p < .001$), with those reporting formal diagnoses on average reporting higher levels of impact (*re*: Fig. 2). However, while the two LMMs revealed significant interaction effects between comorbidity type and diagnostic status, these effects do not appear to influence the interpretation of the main effects. Evidence to support this position comes from scrutinising the simple effects presented in Fig. 2a (Child Impact) and 2b (Parent Impact).

4.3. Predictors of PSS

Table 2 presents the results of two multiple linear regressions with PSS scores ($M = 51.55$, $SD = 12.30$) as the dependent variable and the predictor variables, adjusting for one another, listed in the first column. In the first model the comorbidities were categorical (i.e., present vs. absent), while in the second they were the Child Impact ratings. The linear combination of the predictor variables accounted for 12.6 % of the variability in PSS scores ($R = .355$, $R^2 = .126$) in the first model, and 16 % in the second ($R = .401$, $R^2 = .160$). Of the five comorbidities, child ADHD, GI, and sleep disorder were significant predictors across both models, while anxiety was significant predictor of PSS scores in the second, but not the first model. While parent factors (i.e., age and gender) failed to reach

Table 1
Prevalence (present vs. absent) of Autistic child comorbid conditions in the current sample, and if present, their impact on child function (Child Impact) and parenting stress (Parent Impact), and the relationship between them ($r_{\text{Child,Parent}}$).

	Anxiety	ADHD	ID	GI	Sleep
Present (*n*)	409 (90 %)	280 (62 %)	160 (35 %)	176 (38 %)	298 (66 %)
Undiagnosed (*n*)	196 (47 %)	80 (29 %)	40 (25 %)	98 (56 %)	114 (38 %)
Diagnosed (*n*)	210 (52 %)	199 (71 %)	118 (75 %)	76 (44 %)	179 (62 %)
Absent (*n*)	44 (10 %)	172 (38 %)	292 (65 %)	275 (62 %)	153 (34 %)
Mean Child Impact	3.36 (1.02)	3.52 (0.91)	3.48 (1.02)	2.77 (1.01)	3.07 (1.01)
Mean Parent Impact	3.54 (1.04)	3.58 (1.00)	3.49 (1.01)	2.75 (1.16)	3.33 (1.21)
$r_{\text{Child,Parent}}$	0.67^{***}	0.74^{***}	0.45^{***}	0.77^{***}	0.57^{***}

$^{***}p < .001$, $^{**}p < .01$, $^{*}p < .05$

Note: Frequencies affected by missing data in places. Percentage of diagnosed and undiagnosed cases are calculated from those indicating that the comorbidity is present.

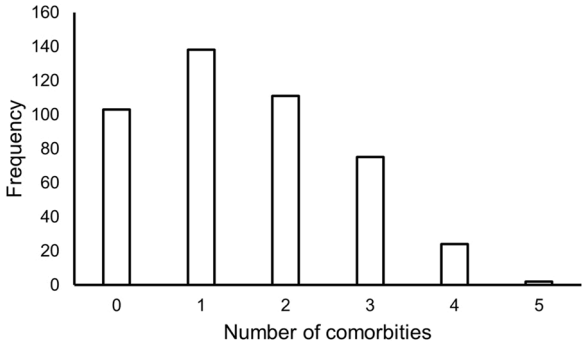

Fig. 1. Frequency of the total number of child comorbidities reported by the parent sample.

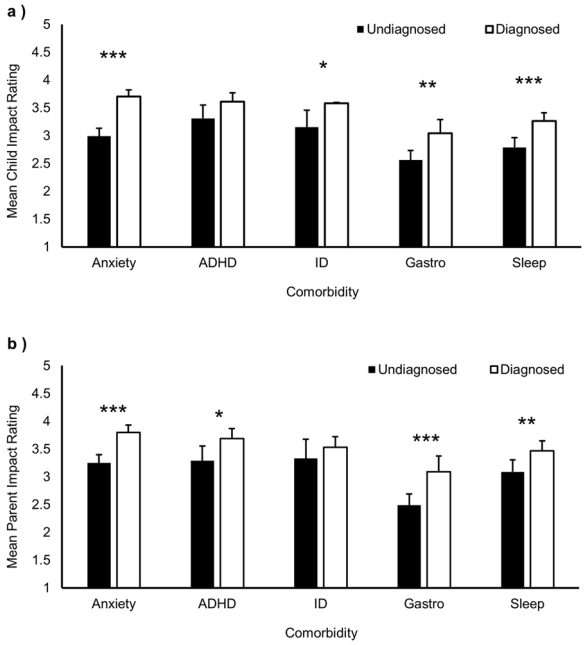

Fig. 2. Mean Child Impact (Fig. 2a) and Parent Impact (Fig. 2b) ratings displayed for each of the five child comorbidities as a function of diagnostic status. Bars are accompanied by 95 % confidence intervals and asterisks represent significant differences across diagnostic status (***$p < .001$, **$p < .01$, *$p < .05$).

significance in either model, the number of children in the family did, with the observation that the coefficients were negative, indicating that as the number of children in the family increased, PSS scores decreased.

To further explore the impact of diagnosis on parenting stress the sample was divided up, for each comorbidity, into those whose child had received a formal diagnosis and those who had not (see Table 1 for relevant proportions), irrespective of whether the parent had reported the presence of the comorbidity. The ensuing MLR analyses (R =.238, R^2 =.057) contained the four covariates and the five comorbidities dummy encoded as 1 (present, and formally diagnosed) and 0 (either absent or present, but not formally diagnosed). As per the previously described analyses, the number of children in the house was significantly associated with PSS scores ($\beta = -.108$, $p = .029$). Examining the regression coefficients for the comorbidity variables, only ADHD (β =.105, $p = .034$) and sleep disorder were significant (β =.126, $p = .012$). All other predictors in the model were non-significant ($p > .05$).

4.4. Predictors of physical HRQOL

Table 3 relates the parent-rated child comorbidity measures to the parent physical component score (PCS: $M = 43.24$, $SD = 8.18$) derived from the SF-36. In sequence, Model 1 (R^2 =.265, $p < .001$) contains the comorbidities encoded as dummy variables (i.e., present, absent), Model 2 (R^2 =.305 $p < .001$) as the impact on child function (i.e., Child Impact), and Model 3 (R^2 =.293 $p < .001$) as contributing to parent stress levels (i.e., Parent Impact). After adjusting for potential covariates and the other comorbidities, sleep emerged as a significant predictor across all three PCS models, with the presence of a sleep disorder (Model 1) or higher Child- and

Table 2
Raw (B) and standardized (β) regression coefficients predicting Parent Stress Scale (PSS) scores. 95 % confidence intervals (CI) are included, while the *t*-statistic indicates if β is significantly different from 0.

PSS Model 1	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	-2.281	1.877	-5.971	1.408	-0.057	-1.215
Parent Age	-0.006	0.070	-0.144	0.131	-.005	-.093
Number of Children	-1.404	0.590	-2.563	-0.245	-.115	-2.380*
ASD Emergence	0.405	0.206	0.000	0.809	.092	1.967
Child Anxiety (Y/N)	3.657	1.968	-0.211	7.526	.087	1.858
Child ADHD (Y/N)	2.801	1.201	0.442	5.161	.109	2.333*
Child ID (Y/N)	1.013	1.294	-1.531	3.557	.039	0.782
Child GI (Y/N)	2.635	1.211	0.254	5.016	.103	2.175*
Child Sleep (Y/N)	3.756	1.249	1.302	6.211	.143	3.008***
PSS Model 2	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	-3.088	1.816	-6.658	0.481	-.077	-1.700
Parent Age	-.042	0.067	-0.175	0.090	-.031	-.629
Number of Children	-1.513	0.569	-2.631	-0.395	-.123	-2.659**
ASD Emergence	.321	0.199	-0.070	0.713	.073	1.615
Child Impact: Anxiety	.813	0.321	0.183	1.444	.117	2.535*
Child Impact: ADHD	1.441	0.447	0.562	2.320	.161	3.220***
Child Impact: ID	.581	0.316	-0.041	1.202	.082	1.835
Child Impact: GI	.891	0.392	0.120	1.662	.106	2.271*
Child Impact: Sleep	1.125	0.365	0.408	1.842	.151	3.094***

***$p < .001$, **$p < .01$, *$p < .05$

Table 3
Raw (B) and standardized (β) regression coefficients predicting SF-36 Physical Component scores (PCS). 95 % confidence intervals (CI) are included, while the *t*-statistic indicates if β is significantly different from 0.

SF-36 PCS Model 1	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	1.037	1.283	-1.484	3.558	.039	0.809
Parent Age	-0.014	0.045	-0.102	0.075	-.015	-0.302
Number of Children	0.864	0.402	0.075	1.654	.107	2.153*
ASD Emergence	0.392	0.137	0.123	0.662	.138	2.860**
Child Anxiety (Y/N)	0.912	1.380	-1.801	3.625	.032	0.661
Child ADHD (Y/N)	-1.615	0.823	-3.233	0.003	-.095	-1.962
Child ID (Y/N)	-0.508	0.826	-2.131	1.115	-.030	-0.615
Child GI (Y/N)	-1.624	0.818	-3.231	-0.016	-.097	-1.985*
Child Sleep (Y/N)	-1.862	0.849	-3.530	-0.194	-.107	-2.194*
SF-36 PCS Model 2	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	0.797	1.267	-1.693	3.287	.030	0.629
Parent Age	-0.014	0.045	-0.103	0.075	-.016	-0.307
Number of Children	0.889	0.397	0.108	1.670	.110	2.238*
ASD Emergence	0.408	0.137	0.140	0.677	.143	2.989***
Child Impact: Anxiety	-0.072	0.305	-0.672	0.527	-.012	-0.236
Child Impact: ADHD	-0.388	0.222	-0.824	0.048	-.084	-1.749
Child Impact: ID	-0.074	0.221	-0.508	0.359	-.016	-0.336
Child Impact: GI	-0.589	0.272	-1.122	-0.055	-.108	-2.169*
Child Impact: Sleep	-0.744	0.252	-1.239	-0.250	-.150	-2.960***
SF-36 PCS Model 3	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	1.154	1.271	-1.345	3.653	.043	0.908
Parent Age	-0.024	0.045	-0.112	0.064	-.027	-0.538
Number of Children	0.809	0.398	0.027	1.590	.100	2.033*
ASD Emergence	0.403	0.138	0.132	0.674	.141	2.925**
Parent Impact: Anxiety	-0.101	0.293	-0.678	0.475	-.018	-0.345
Parent Impact: ADHD	-0.373	0.216	-0.798	0.053	-.083	-1.721
Parent Impact: ID	0.025	0.219	-0.405	0.455	.005	0.114
Parent Impact: GI	-0.437	0.263	-0.953	0.079	-.083	-1.664
Parent Impact: Sleep	-0.662	0.222	-1.098	-0.225	-.149	-2.989***

***$p < .001$, **$p < .01$, *$p < .05$

Parent-Impact scores (Models 2 and 3) being associated with lower physical HRQOL. Additionally, both the presence of gastrointestinal issues and higher impact of child function ratings were associated with lower physical HRQOL. In terms of covariates, the age at which the child's ASD behaviours were first noted by the parent, and the number of children in the household, were both positively associated with physical HRQOL, where an increase in the covariate was linked to an increase in the SF-36 PCS score.

4.5. Predictors of mental HRQOL

Table 4 mirrors Table 3 with the exception of the dependent variable now being the SF-36 mental component score (MCS: M = 33.45, SD = 9.55). Across Model 1(R^2 =.318, $p < .001$), Model 2 (R^2 =.340, $p < .001$), and Model 3 (R^2 =.401 $p < .001$), child gastrointestinal issues and sleep difficulties emerge as significant predictors of parental mental HRQOL, with both acting as risk factors for lower mental well-being. Furthermore, in Model 1, the presence of intellectual disability is linked to lower mental HRQOL, as was parental stress relating to child anxiety in Model 3. Turning to the covariates, parent age, number of children, and the child's age when parents first noted autistic like behaviours, were significant across all three models.

5. Discussion

Past studies have examined the association between autistic child comorbidities and parental well-being, with evidence suggesting a dose-response relationship between comorbid symptom severity and parenting stress. However, most investigations have focused on comorbid conditions in isolation, potentially obscuring the unique and combined effects of multiple co-occurring conditions, particularly where symptom overlap or interaction exists. Additionally, few studies have investigated the impact of Autistic child comorbid conditions upon parental HRQOL, and an exploratory investigation was conducted in order to document relationships across key outcome variables and inform future modelling.

Comorbidities are observed in approximately 70 % of Autistic individuals in New Zealand (Bowden et al., 2020), a figure that is in general agreement with our parent-report data presented here (*re*: 77 %). Aligning with international studies (e.g., van Steensel et al., 2013; van Steensel, Bögels, & Perrin, 2011; Simonoff et al., 2008), the current data indicated that the most common comorbidities were anxiety and ADHD. Furthermore, the frequencies reported in the current study for the diagnosis of sleep disorder (Liu et al., 2021; Sivertsen et al., 2012; Soke et al., 2018; Tyagi, Juneja, & Jain, 2019), ID (e.g., Polyak et al., 2015) and GI (Lanyi et al., 2022) are likewise comparable to international estimates.

In the current study we were interested in the frequency of parents reporting that their Autistic child had a specific comorbidity, and whether or not it had been formally diagnosed. For example, 90 % of parents in the sample endorsed anxiety disorder as one of their child's comorbid conditions, and yet it was an even split in terms of diagnosed vs. undiagnosed. Speculatively, the substantial number of parents reporting that their child experienced anxiety but were without a diagnosis may be explained by several factors. First, and for a number of reasons, a parent may not be engaging with health systems or actively seeking a diagnosis for their child. Second, it may be that parents do seek a diagnosis, but it is not conferred on the basis that the symptoms are subclinical. Of relevance, in a New Zealand sample of school children aged 5–11 years, 28 % of children who were clinically assessed for anxiety did not receive a

Table 4
Raw (B) and standardized (β) regression coefficients predicting SF-36 Mental Component Scores (MCS). 95 % confidence intervals (CI) are included, while the *t*-statistic indicates if β is significantly different from 0.

SF-36 MCS Model 1	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	-2.431	1.467	-5.315	0.452	-0.078	-1.658
Parent Age	0.241	0.052	0.140	0.342	0.235	4.672***
Number of Children	1.225	0.459	0.322	2.128	0.131	2.667**
ASD Emergence	-0.415	0.157	-0.723	-0.106	-0.125	-2.643**
Child Anxiety (Y/N)	-2.350	1.579	-5.453	0.754	-0.071	-1.488
Child ADHD (Y/N)	-0.103	0.941	-1.953	1.747	-0.005	-0.109
Child ID (Y/N)	-2.551	0.944	-4.407	-0.695	-0.127	-2.702**
Child GI (Y/N)	-2.118	0.971	-4.026	-0.210	-0.104	-2.182*
Child Sleep (Y/N)	-2.409	0.935	-4.247	-0.570	-0.124	-2.575**
SF-36 MCS Model 2	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	-2.928	1.455	-5.787	-0.068	-.094	-1.937
Parent Age	0.248	0.052	0.146	0.350	.241	4.775***
Number of Children	1.219	0.456	0.322	2.116	.130	2.671**
ASD Emergence	-0.361	0.157	-0.669	-0.052	-.109	-2.298*
Child Impact: Anxiety	-0.633	0.350	-1.322	0.055	-.091	-1.808
Child Impact: ADHD	0.052	0.255	-0.449	0.553	.010	0.204
Child Impact: ID	-0.426	0.253	-0.924	0.072	-.078	-1.681
Child Impact: GI	-0.948	0.312	-1.561	-0.335	-.149	-3.039**
Child Impact: Sleep	-0.563	0.289	-1.130	0.005	-.097	-1.987*
SF-36 MCS Model 3	*B*	*SE*	Lower CI	Upper CI	*β*	*t-statistic*
Parent Gender	-2.337	1.417	-5.121	.448	-.075	-1.649
Parent Age	.226	.050	.128	.324	.221	4.539***
Number of Children	1.067	.443	.196	1.938	.114	2.408*
ASD Emergence	-.349	.153	-.651	-.047	-.105	-2.274*
Parent Impact: Anxiety	-1.066	.327	-1.708	-.423	-.160	-3.261***
Parent Impact: ADHD	.027	.241	-.447	.501	.005	.112
Parent Impact: ID	-.477	.244	-.956	.002	-.089	-1.956
Parent Impact: GI	-.746	.293	-1.321	-.171	-.121	-2.549*
Parent Impact: Sleep	-.820	.248	-1.306	-.333	-.159	-3.311***

***$p < .001$, **$p < .01$, *$p < .05$

diagnosis (Kercher, Beattie, Donkin, & Shepherd, 2024). Thirdly, it has been noted that anxiety may be under-identified and considered an intrinsic part of ASD (Bowden et al., 2020; Zaboski & Storch, 2018), with similar observations made for ADHD (St Pourcain et al., 2011).

5.1. Impact of comorbidities

In the bivariate analyses it was noted that anxiety, ADHD, and ID were rated significantly higher in terms of their impact on child function than either GI or sleep disorder. Consistent with this, when parents directly rated the degree to which an endorsed comorbidity contributed to their parenting stress, anxiety and ADHD contributed significantly more to stress than GI or sleep disorder. Previous research has identified internalising and externalising behaviours as the main determinants of parenting stress (Halstead, Jones, Esposito, & Dimitriou, 2021; Yorke et al., 2018), likely because both anxiety and ADHD impact either child psychological well-being and/or have observable behavioural components (e.g., expressions of distress and avoidance behaviour in anxiety; impulsivity and attention deficit in ADHD) which parents may find difficult to manage. Of further interest, the correlation coefficients obtained in the current study between the Child Impact and Parent Impact ratings (*re*: Table 1) provide strong evidence of an exposure-response relationship, aligning with the findings of Lanyi et al. (2022), and suggest that interventions aimed at reducing the impact of comorbidities on child function could potentially have a positive carryover effect on parenting stress.

Relatedly, it could be expected that higher mean Child Impact and Parent Impact ratings would be observed when children are formally diagnosed with a comorbidity than when they are not. The assumption here is that a diagnosis will only be conferred when symptoms or behaviours exceed a clinical threshold, indicating that higher levels of support are required for both child and parent. For the Child Impact rating (*re*: Figure 3a), this was found for all but ADHD, where the impact of ADHD was equivalent irrespective of a diagnosis. Why children are not conferred with ADHD diagnoses even though its impact may warrant as much may be down to several factors, including parent fear of their child being labelled, a mistrust of treatments, restricted access to clinicians, or diagnostic 'overshadowing' (Palmer et al., 2024). For parent impact, significance was noted for all comorbidities apart from ID, suggesting that the decreased daily living skills associated with ID in children may be legitimate clinical targets irrespective of whether the child has a formal ID diagnosis or not.

5.2. How the presence of a child comorbidity is related to parenting stress

While the Parent Impact rating directly related a specific child comorbidity to parenting stress, the influence of the five comorbidities on global parenting stress was also gauged using the Parenting Stress Scale (PSS). This multivariate approach adjusts for the influence of covariates and estimates the independent effect of a comorbidity on the PSS while holding the effects of other comorbidities constant. Irrespective of whether the comorbidities were binary coded (i.e., absent vs present) or represented by Child Impact ratings, the strongest associations were between sleep disorder and the PSS. Worthy of further remark is the lack of significance between ID and the PSS, with this relationship potentially being mediated by child sleep quality (Bourke-Taylor, Pallant, Law, & Howie, 2013). In addition, and consistent with previous studies (Mannion & Leader, 2024), GI was a significant predictor. However, when the sample was divided along diagnostic lines (i.e., diagnosed vs. undiagnosed / absent) sleep disorder remained the dominant predictor, echoing previous research (Mannion et al. 2024), with ADHD being the only other comorbidity to attain significance. Taken together, these findings suggest that prioritising the treatment of sleep disorder, GI and ADHD in Autistic children may decrease parenting stress and consequently reduce negative transactional effects.

5.3. How the presence of a child comorbidity relates to parent HRQOL

Regression analyses were performed to examine how the presence and impact of a child comorbidity impacts parent HRQOL. Turning first to the physical HRQOL SF-36 score (i.e., the PCS) it emerged that the presence of GI issues and sleep disorder emerged as significant risk factors of lower physical HRQOL. Also using the SF-36 in the ASD context, Liu et al. (2021) reported strong links between child sleep dysfunction and lower parental PCS scores, arguing that children's sleep quality is a readily treatable condition with the use of pharmacological (e.g., melatonin) or non-pharmacological interventions. Mannion & Leader (2023) reported that parents of Autistic children with sleep dysfunction had lower scores on physical HRQOL measures than other parents of Autistic children, and noted that scant studies have been undertaken in the area even though child sleep issues can result in parent sleep problems and exhaustion. In further commentary, Mannion & Leader (2024) flagged the lack of research investigating child GI and parental well-being, though while finding significant effects in some domains of HRQOL they did not report significance in the physical domain as was found in the present study. However, in view of their analyses they did promote interventions alleviating child GI issues as a means of improving parental HRQOL, an appeal that is supported by the current analysis.

Considering the mental HRQOL scores it was apparent that parent age accounted for the bulk of the variability in the SF-36 MCS. This is not a novel finding, with the literature commonly pitting the 'Wear-and-Tear' Hypothesis against the Coping Hypothesis, where the former predicts parent HRQOL to decrease as the parenting journey progresses, and the latter predicting that it decreases on account of the deployment of adaptive coping mechanisms. In this study we find support for the Coping Hypothesis, with mental HRQOL increasing with parent age. In common with the SF-36 PCS scores, sleep disorder and GI were associated with lower MCS scores. Regression modelling by Liu et al. (2021) failed to uncover significant associations between child sleep dysfunction and parent MCS scores, though they did report significant differences in mean scores when comparing parents of Autistic children to those of typically developing children. Turning to GI symptoms, Mannion and Leader (2024) reported less parental satisfaction with personal

and social relationships than parents of Autistic children without GI, opining that parents have fewer opportunities for family outings and social activities due to their child experiencing pain and discomfort.

6. Strengths and limitations

The current study possesses a number of strengths, in particular the sample size, the simultaneous analysis of multiple child comorbid conditions, and comparing their unique effects on parent well-being across reported presence, diagnostic status, and impact on both child and parent function. The main limitation of the current study is its cross-sectional nature, and that cause-and-effect relationships are speculative and cannot be inferred on the basis of the current data. A further limitation is the sample profile, which being homogenous and biased towards Caucasian and well-educated females, which may limit the generalisability of the data due to a lack of representativeness. For brevity and to avoid fatigue in participants, only five comorbid conditions were selected, where ideally a greater number would have been presented, including anxiety subtypes and depression.

An additional caveat is the assumption that the comorbidities entered into the regression models are independent and can be separated statistically. While care was taken to confirm the absence of multicollinearity in the models we must acknowledge the associated risk of predictors that may lack independence. Additionally, the Child Impact and Parent Impact scores were obtained using a single item, and therefore may lack the precision of multi-item estimates. Finally, the reliance upon parent ratings of their child's characteristics may also limit the interpretability of the findings, and though this may not be as problematic as is generally assumed (Mello et al., 2022), a multi-informant approach would contribute both a validation of parent ratings and a comparison across parents and clinicians.

7. Conclusion, implications and future directions

The findings of the current study support previous research indicating that child comorbid conditions can be detrimental to parent well-being, and are of clinical relevance given the transactional relationships between child and parent. A concern raised in the current study was the substantial number of parents indicating the presence of a specific comorbidity that was undiagnosed, and therefore less likely to attract targeted treatment. Future investigations could seek to understand why such a large proportion of children go undiagnosed, for example, are the symptoms below the clinical threshold, do the parents struggle to access services, or is it that some practitioners do not view ASD and its common comorbidities as distinct diagnoses (Zaboski & Storch, 2018)?

As comorbidities present treatment challenges and are linked to adverse health outcomes for both the Autistic child and their parents, it is important to prioritise and target the most debilitating comorbid conditions. In our multivariate analyses child sleep and gastrointestinal issues consistently emerged as reliable risk factors of lower parental outcomes, irrespective of whether comorbidities were represented in terms of parent-reported presence, diagnosis vs. undiagnosed, or single-item ratings on the impact upon child function or parent stress. As such, greater clinical impact may be achieved if clinicians were to prioritise the treatment of these two comorbidities, especially as both sleep (Mazzone, Postorino, Siracusano, Riccioni, & Curatolo, 2018) and GI (Hsiao, 2014) have been suggested to aggravate the core symptoms of ASD as-well-as other challenging behaviours. Furthermore, for our global measure of stress (i.e., the PSS), ADHD was also a reliable risk factor, and while treatable to some degree with psychostimulants (e.g., Methylphenidate), non-pharmacological therapies as yet have proved less efficacious (De Crescenzo, Cortese, Adamo, & Janiri, 2017).

While the current study is novel in its inclusion of a diverse range of comorbid conditions, which were simultaneously accounted for in the multivariate models, further statistical models of increased complexity are required in order to understand how the various comorbidities interact with each other. For example, does a lack of sleep increase internalising behaviours, or might gastric problems irritate and induce externalising behaviours? Increasing model complexity to include the core symptoms of ASD and coping mechanisms is also important clinically, to determine not only the strength of the relationships between these risk factors and parental outcomes, but also the direction.

In conclusion, to effectively respond to the physical and psychological needs of their Autistic child, parents must effectively parent, something which can adversely affected by parenting stress (Bonis, 2016). While a child's core ASD symptoms contribute to parenting stress, past research has demonstrated that other, comorbid conditions can amplify parenting stress levels. While previous studies (e.g., Davis & Carter, 2008) have implicated externalising behaviours as a primary contributor of parenting stress, the current study presents evidence indicating that child sleep and gastro-intestinal dysfunction may be as equally potent in increasing parenting stress and degrading parental HRQOL. While these findings have implications for clinical practice, the relative novelty of the research requires the findings to be confirmed through further multivariate investigation, and causal relationships determined using more complex longitudinal designs.

CRediT authorship contribution statement

Daniel Shepherd: Writing – review & editing, Writing – original draft, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Sonja Goedeke:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Jason Landon:** Writing – review & editing, Writing – original draft, Project administration, Investigation, Formal analysis, Data curation, Conceptualization.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.reia.2025.202785.

Data availability

Data will be made available on request.

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