The influence of parental variables and child behavioral inhibition on social anxiety in preschool children: The moderator effects of gender

Catarina C. Fernandes[1] · Ana T. Martins[1] · Andreia Santa-Rita[2] · Luís Faísca[1]

Abstract

The main objective of this study was to examine how parental characteristics, such as social anxiety (SA) and parental over-protection, and child's behavioral inhibition (BI) interact and contribute to the manifestation of SA symptoms in preschool-ers. Parents (Mothers: $n = 319$; Fathers: $n = 263$) of children aged between 3 and 6 years answered to self-report measures of overprotection and SA and measures of SA and BI about their children. A positive moderate direct association was observed between children's BI and children's SA, independently of gender. Overprotection arises as a mediator of the effects of the parents' social anxiety on children's SA; however, this mediation depends both on the child's and parents' gender. Only mother's SA was directly related to children's SA, independently of the gender of the child. Overall, these findings extend to preschool children the previous studies that consider parents' overprotective styles and social anxiety, as well as child's BI, the main variables underlying the SA in childhood. In addition, our results highlight the importance of considering the moderator role of gender in the origin and maintenance of SA symptoms in preschoolers.

Keywords Social anxiety · Behavioral inhibition · Overprotection · Preschool children · Gender

Introduction

Social anxiety (SA) is one of the most prevalent psychological disorders in children and adolescents, with a negative impact in various domains of life and long-term implications for social adjustment (National Institute for Health Excellence and Care, 2013; Pickering et al., 2019). Besides its high prevalence, SA has an earlier age of onset and, when

left untreated, persists into adulthood, resulting in significant personal and social costs (National Institute for Health Excellence and Care, 2013). Despite the well-documented effectiveness of child anxiety treatments (James et al., 2013), research into the origins of SA remains comparatively limited. Understanding the factors that place individuals at risk of social anxiety, especially during the preschool years, considered the period prior to the typical age of onset, can provide valuable information regarding prevention.

One of the key early life predictors of social anxiety disorder is behavioral inhibition (BI; Clauss & Blackford, 2012). Kagan et al. (1987) and Rapee (2002) described BI as reactions of withdrawal, wariness, avoidance, and shyness in unfamiliar situations, usually associated with specific physiological responses (e.g., higher levels of cortisol and increased heart rate). Kagan et al. (1988) suggested that BI can be already identified in 15–20% of the two years old children. BI can manifest itself in different ways according to age: while pre-schoolers react with hesitation, inhibition of spontaneous conversation, and limited smile in unfamiliar situations, school-age children manifest extreme avoidance of adults and isolation from unknown peers (Ollendick & Benoit, 2012).

✉ Luís Faísca
lfaisca@ualg.pt

Catarina C. Fernandes
cicfernandes@ualg.pt

Ana T. Martins
atmartins@ualg.pt

Andreia Santa-Rita
andreiasantarita@sapo.pt

[1] Department of Psychology and Educational Sciences & Center for Research in Health Technologies and Information Systems (CINTESIS), Universidade do Algarve, Faro, Portugal

[2] Department of Psychology and Educational Sciences, Universidade do Algarve, Faro, Portugal

Longitudinal and meta-analytic studies on BI showed that, from an early age, inhibited children are significantly more prone to develop anxiety disorders earlier and over time than the uninhibited children (Clauss & Blackford, 2012; Frenkel et al., 2015; Hudson & Dodd, 2012; Sandstrom et al., 2020; Tang et al., 2020). However, a significant proportion of inhibited children do not subsequently develop SA (Henderson et al., 2015). In this context, it is important to identify which factors influence the longitudinal stability of the BI and its development into SA, as well as to recognize the presence of other developmental pathways to SA that may not involve BI (Chan, 2010; Ollendick & Benoit, 2012).

A considerable number of studies have identified key familial factors that increase the child's risk for SA (Chavira & Stein, 2005; Degnan et al., 2008; McLeod et al., 2007; Ollendick & Hirshfeld-Becker, 2002; Tang et al., 2020). Ollendick and Hirshfeld-Becker (2002) pointed out that the parents' influence on the development of children's SA may arise from their own SA, either through a specific genetic vulnerability to SA, or through the modeling of their social avoidance. A meta-analysis based on twelve twin studies (Scaini et al., 2014) points to the heritability of both the SA as a trait and as a disorder, although the amount of variance explained by genetic factors appears to vary widely among studies, ranging from 0.13 to 0.60 with an average of 0.41. The search for specific genes underlying the susceptibility to SA has been proven difficult (Bas-Hoogendam et al., 2018) and several candidate gene studies that reported associations with SAD and related traits have not been satisfactorily replicated (Stein & Galernter, 2014; Stein et al., 2017). A realistic hypothesis is that individual differences in the development of SA involve polygenic inheritance, which is controlled both by complex genetic mechanisms and environmental factors (Bas-Hoogendam et al., 2018; Olson, 2021).

Ollendick and Hirshfeld-Becker (2002) pointed out that the parents' influence on the development of children's SA is strongest in early childhood since parents represent the main socializing agent, and this influence may result from their own SA. For instance, a recent longitudinal study by Aktar et al. (2018) showed that SA in parents is linked with social avoidance in preschool children. The role of social learning in this transmission has also been demonstrated in studies showing parental modeling effects on child SA behavior (de Rosnay et al., 2006; Murray et al., 2008). In addition, Ollendick and Hirshfeld-Becker (2002) also highlighted the contribution of parenting behaviors and attitudes (such as overprotection, rejection, criticism, non-optimal fostering of social interaction), as well as the quality of the parent–child relationship (insecure attachment). Over-controlling parenting (Becker et al., 2010; Hudson et al., 2008) as well as overprotecting (Festa & Ginsburg, 2011; Lieb et al., 2000) limit the child's autonomy at early ages, such as toddler and preschool (Buss et al., 2021), and could increase the child's anxiety by reducing his or her cognitive sense of being able to cope with the environment. Such parenting practices may tacitly communicate to the child that the world is unsafe and unpredictable, and that danger and challenges are around every corner (Bögels & Brechman-Toussaint, 2006). On the other hand, parental practices characterized by flexibility of rules, strategies to support social participation, and appreciation of new situations with affective and behavioral restraint, appear to be associated with children less inhibited and more socially adapted (Degnan et al., 2010; Hane et al., 2008). In sum, the existence of an overprotective parental style toward the social world has been associated with the emergence of SA in children.

Parents' SA and parenting practices cannot be considered independent contributors to the child's SA. Anxious parents tend to be less socially active and may overprotect their children by restricting them or preventing them from being socially involved, diminishing the opportunity to develop social skills and thus perpetuating social fear (Caster et al., 1999; Norton & Abbott, 2017). It has been shown that parents with anxiety disorders appear to have less secure, more restrictive, and overprotective parenting styles (Lindhout et al., 2006). Parents with SA are even more likely to show criticism and doubting of their children's competencies than parents with other types of anxiety (Budinger et al., 2013). Overprotective parenting has been described as having a moderator or a mediator role in the transgenerational transmission of SA. A ten-year longitudinal study that followed a sample of adolescents found that the risk of developing SA was increased in offspring of parents with SA who reported overprotection, rejection, or lack of emotional warmth (Knappe et al., 2009). In a prospective study, Borelli et al. (2015) found that maternal overcontrol acted as a mediator underlying the association between maternal and child anxiety. Although the role of overprotection as a mediator between mother and child anxiety has been recognized, evidence is mainly based on samples of older children and adolescents; studies with children at an early age, such as preschool children, are scarce.

On the other hand, although the literature points to a strong relationship between both parental behaviors, their child's BI, and social anxiety symptoms in childhood, different levels of SA may be related to similar parenting behaviors (McLeod et al., 2007), and not all behavioral inhibited children will present SA symptoms later (Clauss & Blackford, 2012). One possible factor moderating these associations is the gender of both children and parents (Rork & Morris, 2009).

According to Bögels and Perotti (2011), most of the studies failed to consider the differential role of mothers and fathers in the development of the child's SA, since the literature has focused mostly on mothers. Only a handful

of studies have considered both parents to understand the phenomenon. Current literature has begun to emphasize the differential contribution of maternal and paternal variables to the child's anxiety. For example, Pereira et al. (2014) concluded that preventing school-aged child's anxiety requires reducing the exposure to the overt manifestation of mothers' anxious cognitions and behaviors as well as promoting fathers' role in child's exposure to risky situations, such as encouraging independence and helping children to confront gradually anxiety-provoking situations.

Few studies have explored the association between the child's SA and the parenting behaviors displayed specifically by the mother and father. Bögels et al. (2011) and Bögels and Perotti (2011) found that paternal behavior may have a stronger influence on the child's SA than maternal behavior. On the contrary, other authors have provided evidence for a significant relationship between maternal variables (SA and overprotection) and child's SA, but no significant relation between paternal factors and child's SA (Bögels et al., 2001; Pereira et al., 2014; Xu et al., 2017). In a longitudinal observational study with preschoolers, Majdandžić and colleagues showed that father's challenging parenting behavior (in which the child is encouraged to push his limits) diminished the risk of SA in their 4-year-old offspring, while mothers' challenging behavior contributed to higher levels of SA (Majdandžić et al., 2014). These mixed results suggest that, in order to better understand the cumulative and intersecting influences of parental overprotection and child's BI in the development of SA, it is necessary to consider the parenting behaviors of each caregiver and the parent–child relation according to gender (Bögels & Perotti, 2011; Bögels & Phares, 2008; Morris & Oosterhoff, 2016).

Regarding the child's gender, several authors pointed that girls are encouraged to adopt a gender role whose expression of fear and avoidance of feared objects and situations is socially acceptable; contrariwise, boys are encouraged to play a confrontational role in fear situations and to show active coping behaviors (Palapattu et al., 2006). Van der Bruggen et al. (2008) found a positive association between parental variables (overprotection and anxiety) and child's SA for girls, but not for boys, and attributed this difference to a greater susceptibility of girls to parenting behaviors. Graham and Weems (2015) concluded that girls may be more focused on mothers' reactivity in situations of fear, interpreting and coding information differently from boys. However, these studies were carried out with school-aged children and adolescents. Considering preschool children, Gagne et al. (2013) found that, when compared to boys, fear and shyness were higher in girls as young as three years, suggesting that gender differences in fearfulness are evident very early in development and seem to emerge between toddlerhood and preschool age. Overall, such evidence suggests that gender should be considered in the study of BI and SA symptoms at an early age.

Given the scarcity of studies addressing these questions, the impact of vulnerability factors in the development of SA, considering the moderator role of the gender of both child and parents, requires a more in-depth study to understand the weight these variables have in children's SA and how they interact (Graham & Weems, 2015). In this context, our main objective was to understand how parental variables (SA and overprotection) and child variables (BI) are related to SA levels of the children aged between 3 and 6 years, considering the gender of both children and parents. Thus, we propose an initial model that integrates the influence of these different variables (Fig. 1).

We hypothesized that (1) child's SA is positively associated with (1a) child's BI, (1b) parents' SA, and (1c) parental overprotection. We also hypothesized that (2) parental overprotection partially mediates the effect of (2a) parental SA and (2b) child's BI on child's SA. Finally, we hypothesized that (3) the intensity of the association between

Fig. 1 Hypothesized structural model
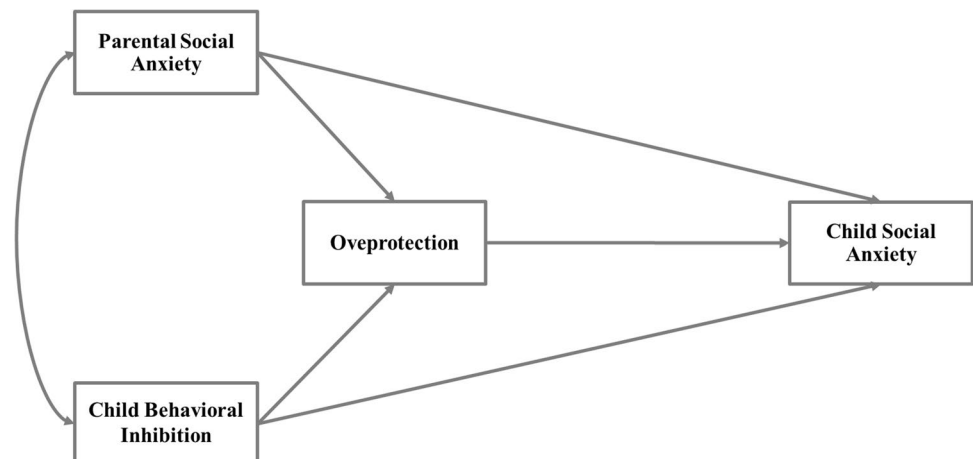
the variables described in the model is moderated by the gender of the parents and child.

Method

Participants

The sample was composed by the 319 mothers (M *age* = 35.81 years, *SD* = 5.37; 66.4% with high school or bachelor's degree) and the 263 fathers (M *age* = 38.20 years, *SD* = 6.05; 56.3% with high school or bachelor's degree) of 330 children recruited at several kindergartens and preschools in Algarve, Portugal (M *age* = 4.05 *yrs*, *SD* = 0.98; range: 3 to 6 years; 49.1% girls). Children and/or parents who had been previously diagnosed with a major psychological or medical condition were excluded from participation after a preliminary screening process by phone. Participants who had not completed more than 20% of the items of any of the questionnaires were also excluded; this criterion led to the exclusion of 38 of the initial 368 participants.

Measures

Behavioral Inhibition Questionnaire (BIQ parent version; Bishop et al., 2003; Portuguese version: Fernandes et al., n.d.) is a 30-item scale measuring child's BI characteristics as reported by their parents. BIQ comprises three dimensions: *social novelty inhibition*, *situational novelty inhibition,* and *physical challenges*. Social novelty (14 items) is measured regarding three contexts: peers (six items; e.g., "Is comfortable asking other children to play"), adults (four items; e.g., "Happily chats to new (adult) visitors to our home"), and performance situations (four items; e.g., "Dislikes being the center of attention"). Situational novelty (12 items) is measured in two contexts, namely preschool/separation (four items; e.g., "Takes many days to adjust to new situations") and unfamiliar situations (eight items; e.g., "Settles in quickly when we visit the homes of people we don't know well"). The physical challenges consist of four items (e.g., "Happily explores new play equipment"). Answers are given on a 7-points rating scale. The BIQ Portuguese version presents excellent internal consistency indexes for the total score as well as for the social and situational subscales ($\alpha > 0.90$). Only the physical challenges subscale presents a less satisfactory internal consistency ($\alpha = 0.61$). In the present study, the total BIQ score was used to assess the child's inhibited behavior frequency in a large range of contexts.

Social Interaction and Performance Anxiety and Avoidance Scale (SIPAAS, Pinto-Gouveia et al., 2003) is a 44-item self-report questionnaire, rated on a 4-point scale, to assess adults' level of anxiety and avoidance in social situations. SIPAAS comprises two subscales: the distress/anxiety subscale and the avoidance subscale, both showing high internal consistency (for anxiety, $\alpha = 0.94$; and for avoidance, $\alpha = 0.92$). In the present study, we used only the anxiety/distress subscale.

Parental Overprotection Measure (POM; Edwards et al., 2008; Portuguese version: Fernandes et al., n.d.-b) is a 19-item self-report questionnaire designed to assess parenting behaviors that restrict the exposure of the child to the perceived threat. Items have a behavioral or situation-specific focus (e.g., "I give my child extra attention when he/she clings to me" and "I try to protect my child from making mistakes") and parents are asked to rate in a 5-point scale the extent to which each item represents their usual response towards the child. POM total score has been found to have strong test–re-test reliability, good construct and predictive validity, and high internal consistency in a sample of parents of 3–5 years old ($\alpha = 0.87$) (Edwards et al., 2008) and 7–12 years old ($\alpha = 0.89$) (Clarke et al., 2013) children. In the Portuguese version, internal consistency was higher than 0.87 for all age groups.

Preschool Anxiety Scale (PAS; Spence et al., 2001; Portuguese version: Almeida & Viana, 2013) is a parent-rated scale (28-item) measuring the frequency of symptoms of preschoolers aged three to six in five anxiety disorders: obsessive–compulsive disorder (five items), social anxiety (six items), separation anxiety (five items), physical injury fears (seven items), and generalized anxiety (five items). Parents are asked to rate each item regarding their children's level of anxiety, using a 5-point scale. Although the whole scale was applied, we only used the *Social Anxiety* subscale. The original scale demonstrated a good internal consistency both for the PAS total score ($\alpha = 0.89$) and for the *Social Anxiety* subscale ($\alpha = 0.74$). The Portuguese version presented acceptable internal consistency for the *Social Anxiety* subscale ($\alpha = 0.70$).

Procedure

The study was initially approved by the Portuguese National Commission for Data Protection (CNPD) and the Directorate-General for Education. The data collection protocol was explained to the coordinators of each preschool institution. An informed, voluntary, written consent was obtained from parents before participation in the study. Finally, parents were asked to complete the questionnaires. The questionnaires were delivered to the parents in a closed envelope and were completed at home. Mother and father were asked to respond individually to the POM and SIPAAS questionnaires. Regarding child's measures (BIQ and PAS), parents were instructed to complete the questionnaires together.

Data Analyses

Descriptive analysis, correlation analysis, and *t*-tests were performed with SPSS 26. Measurement internal consistency was assessed by Cronbach's alpha coefficient. Cohen's *d* was used to estimate the magnitude of the differences between groups.

To test the hypothesized model (Fig. 1), we conducted a Structural Equation Model analysis using SPSS AMOS 20. Model goodness of fit was evaluated using several indices, including the model's χ^2 test, the comparative fit index (*CFI*), and the root mean square error of approximation (*RMSEA*). A multi-group analysis was also carried out to identify differences between boys and girls regarding the influence of parental variables and child's BI on children's SA levels. When the model imposing equivalent structural weights on both genders failed to adjust, the critical ratios for the differences between parameters were used to identify which structural weights should be freed. After verifying the fit, indirect effects were estimated, and their significance was evaluated with a 95% bias-corrected percentile confidence interval (BCCI) based on 5000 bootstrap samples. Through the application of BCCI, it is possible to avoid

power problems due to nonnormal asymmetric sampling distributions of indirect effects (Mackinnon et al., 2004). Finally, we used bootstrap confidence intervals to test the moderating role of gender on indirect effects (moderated mediation model). All the results were presented using non-standardized parameters.

Results

Descriptive Results

Means and standard deviations of the main variables of interest in the study are presented in Table 1. All measures used in the study showed an acceptable level of internal consistency (Cronbach's alpha $\alpha > 0.7$). A comparison of means showed no differences between boys and girls regarding the studied variables.

Correlations between parents' and children's variables were computed attending gender (Table 2). Significant

Table 1 Descriptive statistics and mean comparisons between gender (t-test)

		Cronbach's α	Girls Mean ± DP	Boys Mean ± DP	*t*	*p*	*Cohen's d*
Parental Variables							
Social Anxiety (1–4)	*Mother*	0.95	2.06 ± 0.44	2.07 ± 0.48	-0.22	0.82	0.02
	Father	0.96	1.84 ± 0.45	1.85 ± 0.44	-0.23	0.82	0.02
Overprotection (0–4)	*Mother*	0.92	2.04 ± 0.75	2.16 ± 0.72	-1.46	0.15	0.16
	Father	0.92	2.07 ± 0.72	2.13 ± 0.80	-0.63	0.53	0.08
Child Variables							
Social Anxiety (0–4)		0.75	1.18 ± 0.61	1.08 ± 0.67	1.41	0.16	0.16
Inhibition (1–7)		0.90	3.37 ± 0.72	3.36 ± 0.79	0.14	0.89	0.01

Parental variables. *Social Anxiety*: SIPAAS (Social Interaction and Performance Anxiety and Avoidance Scale); *Overprotection*: POM (Parental Overprotection Measure); **Child variables**. *Social Anxiety*: PAS (Preschool Anxiety Scale – social anxiety subscale); *Inhibition*: BIQ (Behavioural Inhibition Questionnaire)

Mother: *N* = 156 girls and *N* = 163 boys; Father: *N* = 132 girls and *N* = 131 boys

Table 2 Pearson correlation for children and parents' variables, considering gender

		Mother		Father	
		Social Anxiety	Overprotection	Social Anxiety	Overprotection
Mother	*Social Anxiety*	1			
	Overprotection	0.335**	1		
Father	*Social Anxiety*	0.409**	0.218**	1	
	Overprotection	0.184**	0.432**	0.277**	1
Girls	*Social Anxiety*	0.348**	0.266**	0.128	0.071
	Inhibition	0.249**	0.138	0.166	0.142
Boys	*Social Anxiety*	0.305**	0.269**	0.221**	0.313**
	Inhibition	0.233**	0.178*	0.136	0.058

* $p < 0.05$; ** $p < 0.01$

correlations were observed in almost all variables, especially in the measures related to the mother.

Model Assessment Considering Parent and Child Gender

To test the hypothesized model (Fig. 1) a Structural Equation Model (SEM) was used. The purpose of the analysis was to characterize the relationship between these variables while considering both the gender of the parents and child. In this sense, a multi-group analysis was performed to test the moderating effect of the child's gender on these relationships. Since it was not possible to obtain the parental report of both parents for all the children, the models for mother and father were analyzed separately. In the present study, the BI-SA path estimates will necessarily be similar for the father and mother models, since these variables were based on the answers given by both parents simultaneously.

Mother Model

The first model assumed that the associations between variables were equivalent for boys and girls, and consequently all regression weights were constrained to be equal across gender groups (Table 3, Model 1). This model does not fit the data well ($X^2(5) = 11.35$, $p = 0.045$, $CFI = 0.971$, $RMSEA = 0.063$), so it was decided to release some restrictions. Through the inspection of critical ratios, it was found that the weight of the path between SA mother and Overprotection is significantly different regarding child gender ($Z = -3.08$, $p < 0.001$), and thus these regression weights were freely estimated for girls and boys. This parameters release provided a better fit for the model ($X^2(4) = 2.89$, $p = 0.577$, CFI = 1.00, RMSEA = 0.00; $\Delta X^2(1) = 8.46$, $\Delta p = 0.004$). This new model (1a) explains 43% of SA variance in girls and 37% of SA variance in boys. The final model obtained is shown in Fig. 2.

Mother's final model shows an equal weight for all paths, except for the trajectory *SA mother → Overprotection*, which weight was higher for girls, despite being also significant for boys (♀ $b = 0.776$, SE = 0.123, $Z = 6.29$, $p < 0.001$; ♂ $b = 0.288$, SE = 0.115, $Z = 2.50$, $p = 0.012$).

Mother's SA has a significant total effect on their child's SA (total effect: ♀ $b = 0.299$, 95% BCCI = 0.213 – 0.406,

Table 3 Summary of fit statistics across gender for Mother and Father model

|		χ^2	df	p	χ^2/df	$\Delta\chi^2$	Δdf	Δp	CFI	ΔCFI	RMSEA
Mother model											
1	Equivalent regression weights	11.35	5	0.045	2.27	-	-	-	0.97	-	0.06
1a	Equivalent regression weights [a]	2.89	4	0.577	0.72	8.46	1	0.004	1.00	0.029	0.00
Father model											
1	Equivalent regression weights	11.57	5	0.041	2.31	-	-	-	0.95	-	0.07
1a	Equivalent regression weights [b]	2.52	4	0.642	0.63	9.05	1	< 0.001	1.00	0.036	0.00

χ^2 = chi-square statistic; df = degrees of freedom; $\Delta\chi^2$ = change in chi-square; Δdf = change in degrees of freedom; CFI = comparative fit index; ΔCFI = change in CFI

[a] Regression weights between SA parent and Overprotection were allowed to remain free across gender

[b] Regression weights between Overprotection and SA child were allowed to remain free across gender

Fig. 2 Mother's final model (unstandardized regression weights; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$)
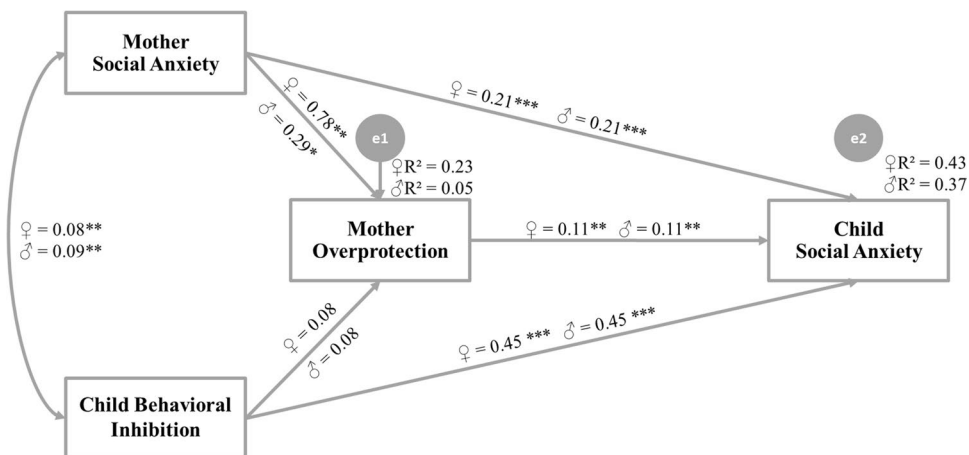
$p = 0.007$; ♂ $b = 0.245$, 95% BCCI $= 0.166 – 0.374$, $p = 0.007$). Mediation analysis revealed that 29% of the total effect of mother's SA on her daughter's SA is mediated by overprotection ($b = 0.086$, 95% BCCI $= 0.024 – 0.146$, $p = 0.018$). Regarding boys, the mediated effect represents only 13% of the total effect ($b = 0.032$, 95% BCCI $= 0.013 – 0.081$, $p = 0.005$). These results suggest partial mediation for both genders.

To further verify whether these gender differences in mediation are statistically significant, we conducted a moderated mediation analysis. The results showed that the strength of the partial mediation effect differs significantly for girls and boys (difference between indirect paths: $b = 0.054$, 95% BCCI $= 0.012 – 0.137$; $p = 0.008$). Thus, the effect of the mother's SA changed according to the gender of the children, having a greater effect on overprotection when the child is a girl. These results indicate that socially anxious mothers are more overprotective when they have a daughter, compared to socially anxious mothers of boys.

A direct positive significant association was found between child's BI and child's SA, independent of gender ($b = 0.447$, $SE = 0.038$, $p < 0.001$). Due to the absence of a reliable effect of child's BI on maternal overprotection, the indirect effect was proven to be non-significant ($b = 0.008$, 95% BCCI $= -0.002 – 0.028$, $p = 0.100$, for both genders).

Father Model

The first Father's model imposed the restriction of equality of all regression weights between boys and girls (Table 3, model 1) and did not fit the data ($X^2(5) = 11.57$, $p = 0.041$, CFI $= 0.952$, RMSEA $= 0.071$). This result supports the idea that the contribution of the variables varies according to the child's gender. Through the inspection of critical ratios comparing parameters, we decided to release the equality restriction imposed on the Overprotection→SA child path (difference between gender: $Z = 2.831$,

$p = 0.002$). When the weight of this path was freely estimated for girls and boys, it provided a better fit of the model ($X^2(4) = 2.52$, $p = 0.642$, CFI $= 1$, RMSEA $= 0$; $\Delta X^2(1) = 9.05$, $p < 0.001$). This model explains 35% of SA variance in girls and 39% of SA variance in boys. The final model obtained is shown in Fig. 3.

Father's final model shows an equal weight for all paths, except for the trajectory *Overprotection*→children's SA, which weight is significant only for boys (♀ $b = -0.018$, $SE = 0.056$, $Z = -0.32$, $p = 0.753$; ♂ $b = 0.227$, $SE = 0.060$, $Z = 3.78$, $p < 0.001$).

Father's SA has a significant total effect on their son's SA but not on their daughter's (total effect: ♀ $b = 0.057$, 95% BCCI $= -0.100 – 0.206$, $p = 0.477$; ♂ $b = 0.167$, 95% BCCI $= 0.007 – 0.334$, $p = 0.042$). This effect is partially mediated by paternal overprotection ($b = 0.102$, 95% BCCI $= 0.041 – 0.178$, $p = 0.012$) and this indirect effect corresponds to 61% of the total effect. For girls, the indirect effect is non-significant ($b = -0.008$, 95% BCCI $= -0.061 – 0.029$, $p = 0.587$): despite father's SA being strongly and positively associated with overprotection behaviors ($b = 0.450$, $SE = 0.103$, $p < 0.001$, for both genders), overprotection addressed to daughters have a negligible effect of girls' SA. A moderated mediation analysis confirmed that the strength of the mediation effect differs significantly for girls and boys (difference between indirect paths: $b = -0.110$, 95% BCCI $= -0.199 – -0.047$; $p = 0.002$). Thus, father's SA only influences the SA of their sons (but not daughters) and this effect is mainly mediated by overprotection behaviors.

The direct positive significant association between child's BI and child's SA was independent of gender ($b = 0.461$, $SE = 0.041$, $p < 0.001$). Due to the absence of a reliable effect of child's BI on father's overprotection, the indirect effect was proven to be non-significant for both genders (♀ $b = -0.001$, 95% BCCI $= -0.015 – 0.004$, $p = 0.439$; ♂ $b = 0.014$, 95% BCCI $= -0.010 – 0.048$, $p = 0.241$).

Fig. 3 Father's final model (unstandardized regression weights; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$)
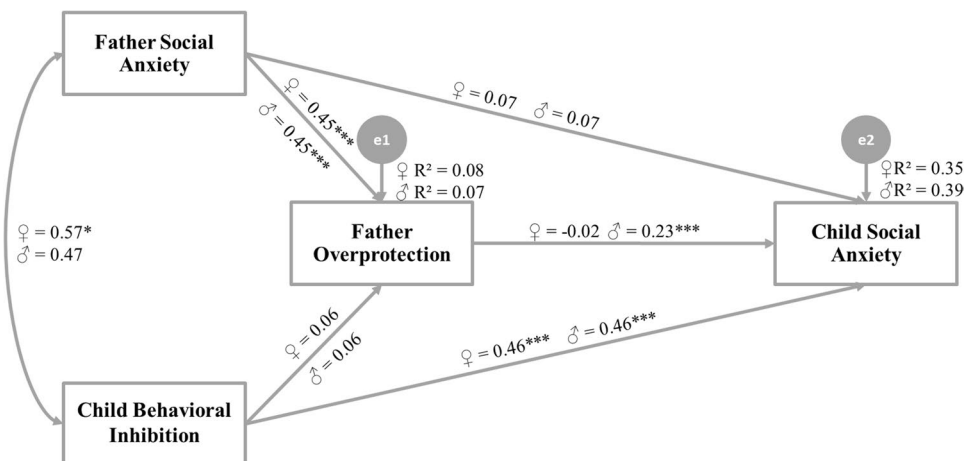
Discussion

The main objective of this study was to examine how child's BI and parental variables (SA and overprotection) relate to preschooler SA symptomatology, considering parental and child gender as moderators.

For that purpose, we hypothesized that the child's SA levels depended on both his/her BI and the parents' SA as well as on parental overprotection, which was assumed to mediate the effect of the two previous variables (Fig. 1). This model explained nearly 40% of the SA variance ($0.35 \leq R^2 \leq 0.43$, for the four models tested), suggesting that the considered variables have a relevant contribution to explaining the individual differences in the SA symptomatology of preschoolers. However, the impact of some of these variables was moderated by gender, both for the parents and the child.

As expected, a positive moderate-to-large association between BI and SA was found. Our results suggest that this effect does not depend on the child's gender. Although the BI-SA association is already recognized in the literature (e.g., Clauss & Blackford, 2012), the moderating effect of gender has been little explored. A recent study with late childhood and adolescent participants (10–16 years) showed that temperamental shyness is more strongly associated with social anxiety among girls than boys (Tsui et al., 2017). Aktar et al. (2018) found that BI assessed when children were aged 1 to 2.5 years predicted the levels of fear and avoidance showed two years later in social and non-social tasks, regardless of gender. Another longitudinal study with temperamentally inhibited preschool children reported that inhibited young girls appeared to be at slightly higher risk for developing clinical-level anxiety and internalizing problems than were inhibited boys at the age of five but this difference tends to attenuate one year later (Bayer et al., 2019). Although our study focused on the same age group as Bayer et al., we did not find gender effects on BI-SA association, which may partly be due to the fact that we used a cross-sectional design and assessed specifically child's social anxiety symptomatology. Gender differences in BI and SA trajectories during infancy are still not well known and more longitudinal studies would be pertinent to clarify their interdependence.

The abovementioned results suggest that BI may be a predominant precursor for SA development for boys and girls, although the moderator role of child gender is not fully clarified. The strong association found between a child's BI and SA may be due in part to the sharing of genetic and environmental etiological factors (Bourdon et al., 2019). Furthermore, both constructs share similar core characteristics such as social

avoidance and shyness. Thus, early interventions for children with BI provide an opportunity to reduce the future risk of SA and subsequent negative outcomes (Sandstrom et al., 2020).

Considering that behaviorally inhibited children may elicit specific parenting behaviors (Ryan & Ollendick, 2018), our model also hypothesized a possible indirect path between BI and SA, mediated by parental overprotection. We expected that children with high levels of BI may induce overprotective behaviors, which in turn would reinforce avoidance, and prevent learning that a feared stimulus is safe (Hudson & Rapee, 2004), leading to a greater likelihood of developing SA later (Kiel & Buss, 2012; Kiel et al., 2016). However, in our study, the association between BI and overprotection was non-significant for either boys or girls in both mother and father models. Thus, the parental overprotection seems not to mediate the effect of BI on the child's SA.

Recently, Borelli et al. (2015) have found a mediation role of maternal and paternal overcontrol (a construct that partially overlaps overprotection) in the longitudinal effect of the child's avoidant characteristics on later anxiety. Although these results do not support those found in our study, there are clear differences between both studies, especially regarding the variables assessed (anxiety vs. SA; avoidant coping vs. BI; overprotection vs. overcontrol) and children's age (school-aged vs. preschool). Furthermore, children's BI may induce different parenting behaviors. For example, while Belsky et al. (2000) found that BI predicted increases in parents' encouragement of approach and discouragement of withdrawal, Kiel and Buss (2011) reported that fearful temperament predicts increases in maternal protective behavior. Parents react differently to children's BI and this heterogeneity may justify the lack of a clear relation between BI and overprotection. Although the child's gender might elicit different parental behavior and, thus, help to clarify such heterogeneity, our results did not support a gender moderation effect on the association between BI and overprotection behavior. Further studies are needed to better understand the role of specific parental overprotection behaviors in the association between children's BI and SA.

Regarding the transgenerational transmission of SA, the obtained results illustrated different pathways, depending on the gender of both parents and children.

Mother's SA seems to have a direct effect on their child's SA. Part of this influence may result from genetic transmission or modeling (Bögels & Brechman-Toussaint, 2006). In an experimental study (Gerull & Rapee, 2002), children (15–20 months) who saw their mother reacting negatively to a toy showed significantly more avoidance and persistent fear when compared to children whose mothers positively contacted the toy. This study demonstrates that signs of anxiety transmitted by the mother can contribute to signs of anxiety already in infants. More specifically for SA, Murray

et al. (2014) found that anxious mothers' narratives may be implicated in the transmission of SA to their preschool children. The authors verified that when socially anxious mothers produced more negative (higher threat attribution) and less supportive (lower encouragement) narratives in a storytelling context, their children show higher SA responses.

The abovementioned studies suggest that maternal modeling may support the intergenerational transmission of SA. In the present sample, such direct influence was equivalent for boys and girls, being gender similarity between child and mother apparently not relevant in this transmission mechanism, at least for preschool children.

It is known that parent anxiety can indirectly affect children through parenting practices (Bayer et al., 2019). Indeed, an indirect effect of the mother's SA on the child's SA, mediated by overprotection was observed in our study. This effect was significant for children of both genders but stronger for girls. The moderation by gender results from the greater impact that the mother's SA has on overprotection when the child is a girl, compared to its impact on overprotection when the child is a boy: while the mother's SA explains 23% of the overprotection over the daughter, it only explains 5% of the overprotection when the child is a boy.

Several mechanisms have been suggested to explain why socially anxious parents adopt overprotective parenting strategies with their children. Socially anxious mothers are intolerant to uncertainty and may rely on overprotective and overcontrolling behaviors in part to curb their own anxiety (Woodruff-Borden et al., 2002). Besides that, the cognitive bias toward threat and avoidance of threat of SA mothers can make these mothers more overprotective and involved than less anxious mothers (Hudson & Rapee, 2002). Furthermore, anxious parents seem to be unable to adjust their controlling behavior to the child's developmental stage, their levels of control remaining constantly high regardless of age (Ballash et al., 2006). This evidence supports the mediation effect of overprotection but does not clarify possible gender differences. However, a hypothesis can be suggested: due to the expertise associated with their gender role, mothers tend to spend more time with their daughters than with their sons (Möller et al., 2012) and, therefore, it is expected that socially anxious mothers would predict more distress to a novel social situation for their daughters than for their sons, and consequently be more protective of girls. Moreover, since they are closer and talk more about emotions with their daughters (Fivush et al., 2000), it is reasonable to expect that socially anxious mothers would provide greater overprotection over their daughters than their sons.

Despite the impact of the mother's SA, the mother's overprotection levels seem to contribute significantly to the child's SA levels, regardless of gender. Several recent studies also supported our results, showing that higher maternal overprotection is a relevant predictor of child SA symptoms (Hudson & Dodd, 2012; Lewis-Morrarty et al., 2012; Verhoeven et al., 2012). Similarly, the absence of gender differences in this effect was reported by Verhoeven et al. (2012), who found that the relation between mother overprotection and child anxiety did not differ for boys and girls. Also, Kiel et al. (2016) observed no gender differences in how overprotective behavior predicted social withdrawal, concluding that, once the mother's overprotective response emerges, the child's gender has no importance in the consequences of that protective behavior. Despite the scarce literature, we can suggest that the impact of the mother's overprotection has the same weight whether the child is a boy or a girl.

Overall, our results suggest that socially anxious mothers can affect their child's SA directly; modeling and heritability may be possible mechanisms for that direct transmission. However, the mother's SA can also have a smaller indirect effect, mainly when the child is a girl, through parenting behaviors that convey the perception of the social world as dangerous. Conceivably, overprotection can make the child more aware of the threat, decrease the perceived control over the threat, withdrawing him/her from opportunities to explore the environment, and develop skills to deal with new situations (Verhoeven et al., 2012).

The influence of the father's SA on the child's SA had a different pattern from the one shown for mothers: the SA transmission was observed only for boys and was exclusively mediated through the father's overprotective behavior.

The absence of the father's direct transgenerational SA transmission was unexpected considering the putative role attributed to genetic factors (Bourdon et al., 2019). On the other hand, the transmission effects that can be attributed to modeling have not been consensual in the literature: while some studies reported a stronger direct influence of the father's SA on children, compared to the mother's SA (Burstein & Ginsburg, 2010), other studies showed a greater influence of mothers (Muris et al., 1996). The lack of father's SA influence may be partially due to the reduced opportunities that the preschool child has to observe fathers' withdrawal and avoidant behavior in daily life. Indeed, not only do fathers, as male adults, tend to display a lower affective intensity in social interactions, but also preschool children spend more time with their mothers, considered the primary caretaker, than with their fathers. Although fathers have an increasing presence in the daily care of children, mothers continue to be perceived as more involved in emotional dimensions, such as caregiving and emotional and social development (de Rosnay et al., 2006). In this sense, the greater expression of anxiety in mothers is linked to greater levels of fears and worries in children (Murray et al., 2008).

Men are socially seen as those who take more risks in the external social world (Byrnes et al., 1999) and involve their

children – especially boys – in challenging play, contributing to the development of children's social confidence (Paquette, 2004). In this sense, Bögels and Perotti (2011) suggested that fathers with SA behaviors will have a more exacerbated influence on the child's SA compared to the influence of the mother's SA since the father's avoidance and withdrawal behaviors are not expected and may be interpreted as a reliable signal of potential external threats. Our results can be interpreted according to Bögels and Perotti's model: the indirect transmission through overprotective behaviors may indicate that children interpret paternal overprotection as a strong negative signal about the external social world, thus promoting avoidant behaviors and fostering social anxiety symptomatology. However, this effect was observed only for boys. Indeed, girls are encouraged to be affectionate, sensitive, and sympathetic and expect to be protected by their parents; so, paternal overprotection may not be interpreted negatively. In contrast, boys are encouraged to control their emotions, to have autonomy, to explore the outside world, and to get involved in more socially risk-taking situations (Bögels & Phares, 2008; van der Bruggen et al., 2010). Therefore, boys may be more susceptible to a biased interpretation of fathers' overprotective behaviors (Bögels & Perotti, 2011). These results are very relevant because they suggest that parental behavior may be a key factor in the gender differences found in SA.

In summary, it was observed that the contribution of parental dimensions to children's SA is different for mothers and fathers. Contrary to what was observed in the father, the mothers' SA has a direct impact on the child's SA regardless of the child's gender, possibly through modeling of social avoidance and fear of evaluation. On the other hand, it was verified that maternal overprotection is associated with child's SA levels, independently of their gender, while paternal overprotection is only associated with boys' SA. The level of SA of mothers also has a greater impact on overprotection toward girls than in relation to boys, although its impact is significant in both genders. This result indicates that the total contribution of the mother's SA is greater for the SA of her daughters than of her sons since part of this contribution is indirectly conveyed by overprotection behaviors. For fathers, although their SA does not have a direct effect on the children's SA, it influences their overprotection behaviors towards sons and daughters. However, only boys seem to respond with social anxiety symptoms to paternal overprotection. Thus, regarding the transmission of SA from parents to children, a gender-dependent mediation through overprotection was found, especially considering the Mother-Daughter and Father-Son dyads.

These results also suggest that the overprotection behaviors of parents appear to depend more on parental characteristics (SA) than on the characteristics of the child (BI) and are affected by the gender of the parent and child. However,

although the weight of parental SA and overprotection in the development of SA in children has been recognized (Fisak & Grills, 2007; Rapee, 2001), there are still very few studies assessing the extent to which parental SA influences the SA of the child through overprotection. Future studies are needed to corroborate our results.

Despite the findings revealed by the current study, some limitations should be considered. Firstly, this is a cross-sectional study and causal associations among variables cannot be claimed. Another limitation was the exclusive reliance on measures reported by parents, providing only an indirect characterization of the child. Parents often underreport negative parenting behaviors (Bögels & Melick, 2004), suggesting that self-reports of overcontrol may underestimate the prevalence of these behaviors. Third, questionnaires were sent to the child's homes and aimed at both parents; there was no way of controlling whether the parents actually filled out the questionnaires separately or together. Finally, a close limitation can be the possible "common method variance" bias since all measures were collected at the same time and using the same informants.

Regardless of these considerations and although more studies are needed to replicate the current findings, using different sources of information about the child, this study extends previous work with the inclusion of both parents, allowing examining their influence on the development of the child's SA, and focused on mediation and moderation processes that help to understand these parental influences. These results emphasize the importance of considering gender and both parents for a deeper understanding of the factors associated with the development of SA, which may contribute to developing preventive parent education programs and improving the efficacy of interventions with SA children.

Declarations

by the Portuguese CNPD (National Commission for Data Protection; approval code 6313/2016) and by the MIME (Committee for Monitoring Surveys in School Environment, Portuguese Ministry of Education; approval code: 0516100002). Written informed consent was obtained from all participants (parents and teachers).

Conflict of Interest Statement On behalf of all authors, the corresponding author states that there is no conflict of interest.

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