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The unique contribution of blushing to the development of social anxiety disorder symptoms: results from a longitudinal study

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Background: Self-conscious emotional reactivity and its physiological marker – blushing has been proposed to be an etiological mechanism of social anxiety disorder (SAD), but so far, untested in longitudinal designs. This study tested, for the first time, whether self-conscious emotional reactivity (indexed as physiological blushing) contributes to the development of SAD symptoms over and above social behavioral inhibition (BI), which has been identified as the strongest predictor of SAD development in early childhood. Methods: One hundred fifteen children (45% boys) and their mothers and fathers participated at ages 2.5, 4.5, and 7.5 years. Social BI was observed at all time points in a stranger approach task, and physiological blushing (blood volume, blood pulse amplitude, and temperature increases) was measured during a public performance (singing) and watching back the performance at ages 4.5 and 7.5. Child early social anxiety was reported by both parents at 4.5 years, and SAD symptoms were diagnosed by clinicians and reported by both parents at 7.5 years. Results: Higher social BI at 2.5 and 4.5 years predicted greater social anxiety at 4.5 years, which, in turn, predicted SAD symptoms at 7.5 years. Blushing (temperature increase) at 4.5 years predicted SAD symptoms at 7.5 years over and above the influence of social BI and early social anxiety. Conclusions: That blushing uniquely contributes to the development of SAD symptoms over and above social BI suggests two pathways to childhood SAD: one that entails early high social BI and an early onset of social anxiety symptoms, and the other that consists of heightened self-conscious emotional reactivity (i.e. blushing) in early childhood. Keywords: Social anxiety disorder; blushing; self-consciousness; behavioral inhibition.

Introduction

We have all, on occasion, experienced jitters in certain social situations: giving a public speech, attending a job interview, or going on a date. The feelings and sensations accompanying these events might have been overwhelming for most of us: bumping heart while walking to the stage or cheeks turning red and burn while talking to our crush. We suddenly start thinking how stupid or ridiculous we may appear to others and how others will reject us. This common condition is called social anxiety. It appears when we are concerned with others’ opinions of us, which may be negative. Some scholars argue that being socially anxious from time to time is beneficial – caring about what other people think of us helps us behave in ways that promote bonding with others (Gilbert, 2001). Although occasional social anxiety may be adaptive, experiencing intense and frequent social anxiety may become a burden for an individual and for a society at large.

Extreme and persistent fear of others’ negative evaluation, which leads to avoidance of social situations or suffering, is a mental disorder called social anxiety disorder (SAD; American Psychiatric Association, 2013). SAD is one of the most prevalent mental disorders in the Western world – one in around every ten people suffer from SAD during their lifetime (Furmark, 2002). The disorder causes considerable distress and interference with everyday social functioning and is associated with general reduction in quality of life, poor academic and professional performance, and high comorbidity with other disorders such as depression and substance abuse (Fehm, Beesdo, Jacob, & Fiedler, 2008). Moreover, SAD entails substantial economic and societal costs in the form of financial dependency, decreased work productivity, lower employment rates, and increased health service use (Lipsitz & Schneier, 2000; Patel, Knapp, Henderson, & Baldwin, 2002).

Social anxiety disorder has an early onset with an average age of 10–13 years (Rapee & Spence, 2004). However, the first social anxiety symptoms may occur much earlier – already at preschool ages – and may pose risk for later SAD development (Furhiss, Beyer, & Guggenmos, 2006). Once developed, SAD has usually a chronic course when left untreated and low recovery rates compared with other anxiety disorders, especially in childhood and adolescence (Hudson et al., 2015). The serious individual and societal burdens and poor treatment outcomes highlight the importance of investigating the etiology of SAD development. The temperamental factor behavioral inhibition (BI) has been highlighted as the single largest predictor of SAD in early childhood.
childhood (Clauss & Blackford, 2012). BI refers to a temperamental style of extreme withdrawal and avoidance in novel situations such as meeting an unfamiliar person or interacting with a new toy (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). In these situations, behaviorally inhibited children avoid unfamiliar events and/or people. When confronted with novel situations, these children withdraw and seek the proximity of their caregivers, and display nonverbal signs of fear, such as freezing or body tension and facial expressions of fear (wide-open eyes, stretched lips, and/or jaw drop), vocalizations (e.g. anxious whining or fearful exclamation), and verbalizations of fear (e.g. saying ‘Go away’). They rarely approach novel objects or unknown people, and when they do, the approach is delayed and cautious (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Kagan et al., 1984).

For SAD, BI in social contexts (e.g. meeting a stranger) may be of particular interest because SAD entails impairments in social, rather than nonsocial functioning (American Psychiatric Association, 2013). Past research has consistently shown that BI in early childhood predicts later development of SAD (Chronis-Tuscano et al., 2009; Hirshfeld-Becker et al., 2007). However, not all people with SAD have a history of BI suggesting that other individual factors, independent from BI, also contribute to the SAD development.

Recent theoretical models suggest that heightened self-conscious emotional reactivity plays an important role in SAD. Heightened self-conscious emotional reactivity, consisting of increased excitability of self-conscious emotions such as embarrassment, shyness, and shame, has been assumed to be a symptom of SAD (Bögels et al., 2010; Hofmann, 2007; Stein, 2015) and a key factor involved in the development of SAD (Hudson & Rapee, 2000). A physiological marker of heightened self-conscious emotional reactivity is blushing (Crozier, 2004). Blushing refers to reddening of the face, and sometimes neck and chest, in social situations, when a person is exposed to social attention (Leary, Britt, Cutlip, & Templeton, 1992). Blushing is seen as a core physiological symptom of SAD (American Psychiatric Association, 2013; Bögels et al., 2010), and a recent meta-analysis confirmed that blushing is indeed related to social anxiety (Nikolić, Colonnese, de Vente, Drummond, & Bögels, 2015). Although it is known that heightened self-conscious emotional reactivity (and blushing, as its physiological marker) is a symptom of already developed SAD (American Psychiatric Association, 2013; Nikolić et al., 2015) and a factor that can maintain SAD (Hofmann, 2007), it is not yet clear whether it has an important role in the development of SAD, as proposed in some models (Hudson & Rapee, 2000).

Self-conscious emotional reactivity (and blushing) occur when a person realizes that there is a possibility of making a negative impression on others whose approval that person seeks (Leary et al., 1992). Thus, people blush when they experience that they may be evaluated negatively. In these situations, people typically experience acute self-consciousness – the feeling of being exposed to other people’s attention (Leary et al., 1992). Blushing, then, reflects high level of ambivalent arousal – there is an urge to flee from the exposure situation and hide from negative evaluation, but at the same time, the person who blushes experiences social interest and fears that fleeing may have social consequences; thus, the person needs to stay and inhibit the fleeing tendency (Nikolić, Colonnese, de Vente, & Bögels, 2016; Van Hooft, 2012).

The ability to blush is assumed to emerge when children acquire self-awareness – a sense of public self (Buss, 1980; Darwin, 1872; Leary et al., 1992), which appears in the second year of life (Lewis, 1995, 2000). At this age, children may experience embarrassment due to being exposed and being in the center of attention and they may blush as a consequence of this exposure (Lewis, 1995, 2003). Around the age of 3-4, children also develop internal standards against which they can evaluate their own behaviors and may blush and experience evaluative self-conscious emotions, such as embarrassment, shyness, and shame when they think they may be judged negatively by others (Lewis, 2001, 2003; Stegge, 2013). Because fear of negative evaluation is also a core feature of SAD (American Psychiatric Association, 2013), the proneness to self-conscious emotional reactivity (i.e. blushing) and being sensitive to others evaluations in toddlerhood and early childhood may predispose a child to SAD because it prompts evaluative social fears (Hudson & Rapee, 2000). Actually, a few scholars proposed that self-conscious emotions and blushing may be an early indicator of the development of social anxiety as it occurs earlier in life, before fears and concerns typical of social anxiety may be assessed (Hudson & Rapee, 2000; Nikolić, Colonnese, de Vente, & Bögels, 2016; Voncklen & Bögels, 2009). Normal evaluative concerns (i.e. fear of negative evaluation) develop at the same time as evaluative self-conscious emotions, that is, around the age of 3-4, but they become prominent between the ages of 6 and 12 (Gullone, 2000). At this time, they can significantly impair children’s social functioning, leading to a disorder (Rapee & Spence, 2004).

Self-conscious emotional reactivity is thought to be distinct from BI (Buss, 1986; Crozier, 1999). Buss (1986) made a clear distinction between self-conscious shyness (which we refer to as self-conscious emotional reactivity) and fearful shyness (which reflects the same construct as BI) by stating that origins, elicitors, and manifestations of these two types of shyness are different. Fearful shyness appears in infancy, it is evoked by novel social situations (e.g. meeting an unknown person) and intrusiveness (e.g. fast approach), and it is characterized by crying, inhibition, fearful reactions,
escape, and high autonomic arousal, whereas self-conscious shyness develops in early childhood as a reaction to an exposure or being in the center of attention, available for others' scrutiny (e.g., when being overpraised, teased, performing in front of others, evaluated by others). This kind of shyness manifests as blushing, feeling foolish, or flustered. Fearful shyness originates in fearful temperament, whereas self-conscious shyness originates in self-awareness (Buss, 1986; Schmidt & Buss, 2010). Empirical studies confirmed this distinction by showing that fearful and self-conscious shyness are uncorrelated throughout childhood (Eggum-Wilkens, Lemeray-Chalfant, Aksan, & Goldsmith, 2015) and have different neurophysiological underpinnings and behavioral manifestations (Poole & Schmidt, 2019), suggesting that these two factors may be independent predictors of SAD symptoms. Although behavioral inhibition was thoroughly investigated as a predictor of SAD development, it is not yet clear whether self-conscious emotional reactivity and blushing, as its physiological marker, contribute to the development of SAD. Only a few cross-sectional studies provided evidence that blushing is related to social anxiety symptoms in children (Nikolić, de Vente, et al., 2016; Nikolić van der Storm, Colonnese, Brummelman, & Bügels, 2019); however, blushing has never been investigated longitudinally, as a factor contributing to SAD development. More importantly, it is unknown whether blushing contributes to the development of SAD over and above the contribution of early social BI. Here, we investigated, to our knowledge for the first time, whether heightened self-conscious emotional reactivity indexed as blushing in early childhood predicts SAD symptom development in later childhood over and above the influence of early childhood BI. We hypothesized that early social BI predicts SAD symptom development from early to late childhood. Moreover, we assumed that heightened self-conscious emotional reactivity predicts SAD symptom development from early to late childhood over and above the contribution of early social BI.

Method

Participants

One hundred fifteen children (45% boys) participated in the current study. Participants were part of a bigger longitudinal study on the development of anxiety with 153 couples who started at the prenatal measurement. Children visited the research laboratory separately with their mother and father on each measurement occasion. During the laboratory visit, the child was 4 months, 1 year, 2.5 years, 4.5 years, and 7.5 years old. The stranger approach task from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith & Rothbart, 1996) was conducted. The stranger approach task was introduced by the experimenter: 'And now, famous [name of the child]! One song is going to be played. You have to choose a song to perform. After deciding on the song, the child sat on the podium during the recovery, with the father on the stage. If the child rejected this option, the assessment was stopped. If the child was calm and stopped singing, and when this happened, the experimenter offered to sing along with the child. Some children at the age of 4.5 (but none at the age of 7.5) displayed noticeable distress (e.g., starting to cry) during the procedure. When this happened, the experimenter offered to sing along with the child and the possibility that the father could join the stage and sing along. If the child rejected this option, the assessment was stopped. If the presence of the father on the stage did not result in the reduction in distress, the assessment was also stopped and the child was calmed down by the experimenter. Following the performance, the child sat on the podium during the recovery, after which the child was asked to watch his/her recorded performance (if the child sang a song) with the father and the test leader on a television screen. Physiological blushing was measured during this procedure.

Measures

Social behavioral inhibition. Child social BI was coded in the stranger approach task at 2.5, 4.5, and 7.5 years of age.
Children's SAD symptoms. When children were 7.5 years old, their diagnostic status was determined by a semi-structured clinical interview for DSM-5 childhood diagnoses, the Junior SCID (Wanie, Braet, Boigels, & Roelofs, 2020), which is the DSM-5-based adaptation of the Kid-SCID (Hien et al., 1998) used to reliably assess common psychiatric disorders in children and adolescents. The interviews were conducted by two research assistants with a master's degree in Pedagogy who were trained to administer the Junior SCID. The interviewer administered the Junior SCID to both parents. To assess inter-rater reliability, one trained assistant, who was blind to the original interview and scoring, coded the audio-taped sessions (12% double-coded). The inter-rater reliability was good: intraclass correlation = .84. In the present study, we focused on the number of SAD symptoms, rather than on the diagnosis, for two reasons. First, the number of symptoms allows for dimensional assessment of SAD, which is in line with DSM-5 recommendation (American Psychiatric Association, 2013). Second, because our sample was a community sample and a diagnosis was not expected in more than 8% of the sample at this age (Kashdan & Herbert, 2001), we obtained more power for our analyses by assessing the disorder as a continuous variable.

Child SAD symptoms at 7.5 years were also reported by both parents using the SCARED-C, a valid and reliable instrument to assess child anxiety disorders according to DSM (Birmaher et al., 1997). The modified social anxiety subscale (Boigels & van Melick, 2004) consists of nine items and possesses good reliability, \( r = .80 \) (Boigels & van Melick, 2004). Internal consistency in our sample was also good, \( r = .86 \) for mothers and \( r = .83 \) for fathers. Mothers’ and fathers’ ratings were highly correlated, \( r (80) = .61, p < .001 \), standardized, and averaged into a composite score.

Statistical analyses
We applied structural equation modeling (SEM) to investigate the predictive role of blushing and social BI in the development of SAD symptoms. Maximum-likelihood estimation was used to obtain model parameters and their standard errors as well as a chi-square measure of overall goodness of fit. We used the following fit indices to judge the model fit: nonsignificant chi-square value (\( x^2 = .05 \)), the root mean square error of approximation (RMSEA), and the comparative fit index (CFI). RMSEA of 0.01, 0.05, and 0.08 indicates excellent, good, and mediocre fit, respectively, and CFI values above .95 indicate good fit (Hu, & Bentler, 1999).

We planned to run three models in which the concurrent and longitudinal effects of (a) DC reactivity, (b) AC reactivity, and (c) cheek temperature reactivity on SAD symptoms next to the concurrent and longitudinal effects of BI on SAD symptoms were estimated. The concurrent and longitudinal effects of social BI on blushing and of blushing on social BI were not estimated (fixed to zero), because we assumed that these two processes are independent from each other. Also, the effects of earlier social anxiety on subsequent blushing and social BI were set to zero, because we did not have specific hypotheses about the existence of these effects. Next to these effects, to assess stability, the model included the effects of social BI at one time point on social BI at subsequent time points. The stability of blushing and SAD symptoms was also modeled.
Estimation of these parameters in the model controls for the stability of the constructs; thus, any effects of the predictors on the outcomes add predictive power to the explanation of the variance in the outcome over and above the power which can be obtained from the stability of the construct. For example, if social BI and blushing predict SAD symptoms at 7.5, they do so over and above social anxiety at 4.5. This means that, in this case, social BI and blushing would predict the development of SAD symptoms from 4.5 to 7.5. The model was identified by fixing one coefficient per latent factor to 1.00. The model included three time points with social BI being measured at all the time points and blushing and social anxiety being measured at the last two time points. We opted for the most parsimonious models in which we estimated only the hypothesized effects. If this resulted in a good fit, the hypothesized models were retained.

SAD symptoms reported by the parents at 7.5 and number of SAD symptoms rated by a clinician at 7.5 were used as indicators of the latent SAD symptoms variable at 7.5. Blushing during performance and blushing during watching back for AC, DC, and temperature increases were used as indicators of the latent blushing variable, both at the age of 4.5 and 7.5. Because DC, AC, and temperature indices of blushing were not correlated, they were analyzed in separate models.

Results
Preliminary analyses
All variables were checked for outliers (±3SD) and winsorized in the case outliers were detected. The variables were checked for normality and transformed in the case of severe violation (George & Mallery, 2010), which was the case for number of SAD symptoms rated by the clinician. Descriptive information is presented in Table 1. There were no significant differences between boys and girls in any of the outcomes (all ps > .290); thus, sex was not included in the analyses.

Because it was possible that socially anxious children would become more self-conscious (and, thus, would blush more) already during the baseline period preceding the performance (e.g. due to the electrode attachment), we investigated whether blushing indexed as absolute cheek temperature during baseline was associated with the levels of social anxiety at 4.5 years and SAD symptoms at 7.5 years. Baseline cheek temperature at 4.5 years was not significantly associated with social anxiety at 4.5 years, \( r(df = 58) = -.12, p = .365 \), and with SAD symptoms at 7.5 years, \( r(df = 60) = -.14, p = .284 \). The same nonsignificant association between baseline cheek temperature at 7.5 years and social anxiety at 4.5 years and SAD symptoms at 7.5 years was found, \( r(df = 66) = .10, p = .418 \) and \( r(df = 72) = .15, p = .212 \), respectively. We did not examine this for AC and DC reactivity as the absolute values of these measures are not meaningful (Drummond et al., 2007).

The missing data appeared for several reasons. First, because of the multiple measurement points, it was possible that families visited the laboratory at a certain but not all measurement points. Also, some children refused to take part in the performance task (\( n = 7 \) at 4.5 years and \( n = 9 \) at 7.5 years), and even when they did perform, physiological equipment failed for some children. Some children did not explicitly refuse to take part in the performance task, but once announced, they did not sing a song (\( n = 22 \) at 4.5 and \( n = 3 \) at 7.5 years). The analyses of missing data are reported in Appendix S1. Importantly, the analyses showed that, although the missingness on some variables was dependent on other variables in the model (e.g. children for whom AC reactivity at 4.5 years was missing had higher social anxiety and SAD symptoms than children for whom AC reactivity at 4.5 years was available), children with missing data on one variable at one measurement point (e.g. BI at 2.5) did not differ on that variable measured at other time point(s) (e.g. BI at 4.5 and 7.5) from children for whom data were available. This was true for all variables except for the DC increase. Because the missingness depended on

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Means, standard deviations, and zero-order correlations among study variables</th>
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<tr>
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<td>( n )</td>
</tr>
<tr>
<td>Social BI 2.5</td>
<td>107</td>
</tr>
<tr>
<td>Social BI 4.5</td>
<td>108</td>
</tr>
<tr>
<td>Temp. 4.5</td>
<td>66</td>
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<tr>
<td>AC 4.5</td>
<td>73</td>
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<tr>
<td>DC 4.5</td>
<td>47</td>
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<td>SA 4.5</td>
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<tr>
<td>Temp. 7.5</td>
<td>68</td>
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<tr>
<td>AC 7.5</td>
<td>77</td>
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<tr>
<td>DC 7.5</td>
<td>75</td>
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<tr>
<td>SAD sympt. 7.5</td>
<td>106</td>
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Standardized composites of the increase in the performance and watching back for AC, DC, and temperature increases are reported. Standardized composite of clinician and parental rating of SAD symptoms are reported at 7.5. AC, blood pulse amplitude increase; BI, behavioral inhibition; DC, blood volume increase; SA, social anxiety; SAD sympt., social anxiety disorder symptoms; Temp., temperature increase.

*\( p < .050 \); **\( p < .010 \).
other variables included in the model, but not on the missing variable itself, we could assume that the missing was at random (Schafer & Graham, 2002) for all but the DC reactivity models and we could apply maximum-likelihood estimation, for which the missing at random assumption needs to be met (Enders, 2001), to analyze the incomplete data. Thus, we were able to obtain unbiased estimates of values on all variables for all children, including those who refused to perform, did not sing, or had missing data due to other reasons such as not wanting the electrodes attached, not coming to the laboratory visit at one point, or having the devices or the tasks failed. Because DC increases were not missing at random and the sample size was too small to run our SEM only on complete cases, we did not perform the planned analyses with DC reactivity.

Main analyses

We ran SEM in IBM SPSS AMOS 25.0 (Arbuckle, 2014). We used maximum-likelihood estimation which works by estimating a likelihood function for each individual based on the variables that are present so that all the available data are used. Importantly, model fit information is based on all cases. In our case, therefore, all the models were based on the full sample (N = 115). We ran two models, one with social BI and AC increase and the other with social BI and temperature increase predicting SAD development. In both models, early social anxiety reported by parents at 4.5 and SAD symptoms reported by the clinician and the parents at 7.5 were predicted.

The hypothesized model with AC increase as an index of blushing fitted the data well, $\chi^2 = 23.42$, $df = 23$, $p = .437$, RMSEA = .01, 95% CI [.00, .08], CFI = 1.00. All standardized and unstandardized parameter estimates with their standard errors and $p$ values are presented in Table S1, and the significant standardized parameter estimates for the structural model are presented in Figure S1. In the model, physiological blushing at 4.5 and 7.5 years did not significantly predict social anxiety at 4.5 years and SAD symptoms at 7.5 years after accounting for social BI. Social BI at 2.5 years significantly predicted social BI at 4.5 years and at 7.5 years. Social anxiety at 4.5 years significantly predicted SAD symptoms at 7.5 years. In addition, social BI at 4.5 years significantly predicted social anxiety at 4.5 years, but did not directly predict SAD symptoms at 7.5 years. Also, there was a trend toward a significant effect of social BI at 2.5 years on social anxiety at 4.5 years. The indirect effect of social BI at 2.5 years on SAD symptoms at 7.5 years via social anxiety at 4.5 years was found according to Sobel test = 2.36, $p = .018$. The indirect effect of AC at 4.5 years on SAD symptoms at 7.5 years via social anxiety at 4.5 years was not significant.

The hypothesized model with the temperature increase as an index of blushing fitted the data well, $\chi^2 = 20.81$, $df = 23$, $p = .592$, RMSEA = .00, 95% CI [0.00, 0.07], CFI = 1.00. The significant standardized parameter estimates for the structural model are presented in Figure 1, and all standardized and unstandardized parameter estimates with their standard errors and $p$ values are presented in Table S1. For clarity, the error variances are not depicted in Figure 1 although they were estimated as discussed earlier. As shown in Figure 1, the stability of social BI and social anxiety was again found. Also, higher social BI at 2.5 and 4.5 years significantly predicted social anxiety at 4.5 years, but not SAD symptoms at 7.5 years. The indirect effect of social BI at 2.5 years (but not 4.5 years) on SAD at 7.5 years via social anxiety at 4.5 years was, however, significant according to Sobel test = 2.01, $p = .044$. After accounting for the influence of social BI on social anxiety, blushing at 4.5 years significantly predicted social anxiety at 4.5 years. Most importantly, blushing at 4.5 years significantly predicted SAD symptoms at 7.5 years over and above the influence of earlier social anxiety at 4.5 years and social BI at 2.5, 4.5, and 7.5 years. Indirect effect of blushing at 4.5 years on SAD symptoms at 7.5 years via social anxiety at 4.5 years was not significant.

Discussion

The goal of this study was to investigate whether self-conscious emotional reactivity indexed as physiological blushing contributes to the development of SAD symptoms over and above the strongest predictor of SAD in early childhood – social BI. We found that blushing indexed as temperature increase has a unique contribution to the development of SAD symptoms from early to late childhood over and above earlier social anxiety and social BI. In addition, we found that social BI in toddlerhood and early childhood predicts the onset of early social anxiety after which, it does not influence the further development of SAD symptoms, except indirectly. Our findings suggest that there are two pathways to SAD: one starting already in toddlerhood, which consists of having high social BI contributing to the occurrence of early social anxiety, which may later on develop into the disorder; and the other starting in early childhood, consisting of heightened self-conscious emotional reactivity (i.e. blushing) that influences the development of SAD in late childhood.

This study was the first to show that blushing contributes to the development of SAD symptoms in childhood, thereby confirming the theoretical assumption that blushing plays an etiological role in SAD (Hudson & Rapee, 2000). Possibly, around the age of 4, when children acquire abilities to
understand that they can be judged by others, they may start blushing in social situations due to the concern about being negatively evaluated and this uncomfortable feeling of the hot face may signify to them that the situation is threatening and should be avoided, eventually leading to SAD.

We found that blushing predicts the development of SAD symptoms only when it is indexed as temperature reactivity, not as blood pulse amplitude reactivity. Similarly, a direct effect of temperature, but not other physiological indices of blushing, on social anxiety was found in a study with older children (Nikolić et al., 2019). This might be because the slow changes in temperature may reflect blushing that results from social stress due to prolonged social exposure (also called creeping blush; Leary et al., 1992; Nikolić et al., 2019), which may be especially uncomfortable because it is lengthy and uncontrollable, and, thus, may lead to avoidance of similar situations in the future and result in increasing social anxiety. In contrast, blood pulse amplitude may indicate sudden blush that comes and goes quickly during social interactions and that is related to social sensitivity, rather than anxiety (Nikolić et al., 2019). Another possible explanation is that only with the temperature increase, people can detect that their cheeks are getting hot and that they blush (Cooper & Gerlach, 2013) and it may be that this awareness of getting hot in the face may cause distress and avoidance in children.

Our findings also clarified the path from early social BI to SAD symptoms in late childhood. It is already known that children with high BI in toddlerhood and/or early childhood have higher risk of developing SAD later in childhood or adolescence (Chronis-Tuscano et al., 2009; Hirshfeld-Becker et al., 2007), but the mechanism of this pathway was not clear. The zero-ordered correlations between social BI and SAD symptoms in our study confirmed that social BI significantly predicts SAD symptoms. Unlike past studies, the current study also explained the mechanism of this prediction: Social BI in toddlerhood and early childhood influences the occurrence of social anxiety in early childhood. These early symptoms, in turn, predict the development of SAD symptoms in late childhood. Thus, early social BI predicts the development of SAD symptoms in late childhood indirectly, through its influence on social anxiety in early childhood. Of note, social BI was stable from 2.5 to 4.5 and to 7.5 years, which is in line with the majority of work on stability of BI in toddlerhood and early childhood (e.g. Kagan, Reznick, & Snidman, 1988). However, social BI was not stable from 4.5 to 7.5 years. Although some studies found similar low stability at this age (e.g. Scarpa, Raine, Venables, & Mednick, 1995), other studies found higher stability of BI (e.g. Asendorpf, 1994). Lower stability of BI in our study compared to other studies may be explained by the fact that we investigated only the social part of BI unlike other studies, which investigated and combined both social and nonsocial BI. Social BI may be shaped by social experiences specifically starting at the age of 4, when Dutch children start with school and are exposed to situations with unfamiliar people more often. Future studies can investigate constructs that originate from fearful temperament, similarly to BI, but are also shaped by social experiences to understand how fearful temperament expresses and influence the development of SAD. For example, examining social reticence—socially wary behaviors in the company of unfamiliar peers in early childhood (e.g. Coplan et al., 1994; Degnan et al., 2014; Rubin, Burgess, & Hastings, 2002) may shed new light on the development of SAD.

Although this study was the first to longitudinally assess blushing next to social BI in the development of SAD symptoms, and it did so using a multimethod
approach, it also has some limitations. First, as we had a community sample of children, the results need to be replicated in a clinical sample, using the diagnosis of SAD. Second, our sample consisted of relatively highly educated parents; thus, we cannot generalize on other populations. Third, we were not able to estimate the contribution of blood volume reactivity to SAD symptoms due to the missing data not being at random. Future studies may benefit from investigating this measure of blushing in larger samples. Finally, we measured blushing response of children while they were performing in front of a small audience consisting of strangers and their fathers. Although we believe that the results would hold if a different audience was present (e.g. strangers and the mother), the results should be interpreted having this in mind.

It is also important to note that due to the nature of the measures and the longitudinal design employed in this study, we had missing data with which we dealt by applying maximum-likelihood estimation which is recommended as superior to other missing data methods (e.g. Enders & Bandalos, 2001). Maximum-likelihood estimation requires that the data are missing at random, but typically, there is no way to directly test MAR. Thus, we could only assume that data are MAR by confirming that children with missing data on one variable at one measurement point did not differ on that variable measured at other time point(s) from children for whom data were available and by including the variables that the missingness was dependent on in the models. In doing so, we believe we were able to obtain unbiased estimates of values on all variables for all children.

The findings of our study have important clinical implications, both for prevention and treatment of SAD. First, not only higher social BI but also stronger blushing response in social situations in early childhood may pose risk for SAD development and may, thus, be targeted in early prevention programs. Second, the efforts to treat SAD should focus on heightened self-consciousness and blushing in particular. Treatments such as task concentration training, mindfulness, and self-compassion therapy that concentrate on refocusing the attention on the task at hand rather than on the self, and on acceptance of one's own flaws (such as blushing in public) may be beneficial in treating patients with SAD.

In conclusion, we found support for a novel pathway to the development of SAD symptoms through intense blushing in early childhood. Next to this, we found that social BI in toddlerhood and early childhood contributes to the early onset of social anxiety symptoms, which then poses risk for the development of SAD later in childhood. These new insights may create leverage for clinical interventions to curb SAD.

Supporting information
Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Missing data analysis.
Figure S1. Social BI and physiological blushing, indexed as blood pulse amplitude (AC) increase, predicting SAD symptoms.
Table S1. Standardized and unstandardized estimates for the model with (a) AC reactivity, and (b) temperature reactivity.

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Key points
- Social behavioral inhibition (BI) is the strongest predictor of social anxiety disorder (SAD) development, but other factors likely play a role as well.
- Physiological blushing may be involved in the development of SAD; however, studies investigating blushing as an etiological factor of SAD are currently lacking.
- We found that blushing in early childhood predicts the development of SAD symptoms later in childhood over and above social BI and early social anxiety.
- Early social BI predicted the onset of social anxiety in early childhood, which, in turn, predicted SAD symptoms later in childhood.
- Because blushing uniquely contributes to the development of SAD, clinical treatments of childhood SAD may benefit from focusing on blushing and how to cope with it.
References

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