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Social anxiety in teens: its relationship to puberty and the moderating effect of intelligence

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Abstract

Most children and adolescents are diagnosed with social anxiety disorder (SAD) around the same time puberty starts. SAD affects one's life on a physiological, behavioural, and cognitive level. If left untreated, it can have a chronic and unremitting course over time. In recent years, little research has been done on the direct relationship between puberty and the development of social anxiety disorder. Furthermore, previous research states that psychopathology such as social anxiety disorder is less apparent in highly intelligent individuals. By examining the relationship between puberty, intelligence, and SAD, suggestions can be made on the theoretical inclusion or exclusion of puberty and intelligence as risk factors for developing SAD. This study aimed to examine the predictability of puberty and intelligence on SAD, with the addition of intelligence as moderator. It was expected that puberty would positively predict signs of SAD, that intelligence would negatively predict signs of SAD, and that intelligence was a significant moderator in the relationship between puberty and signs of SAD, wherein, during puberty, highly intelligent children developed fewer signs of SAD than averagely intelligent children. Children (N= 120, 9-12 years; M age = 11.05, SD = 0.77; 52.5% girl; 44.2% gifted) from different schools in the Netherlands completed the Standard Progressive Raven Matrices, the Pubertal Development Scale, and the social anxiety YAM-5 questionnaires. A hierarchical multiple regression yielded no evidence for the predictability of puberty (p = .282) and intelligence (p = .405) on social anxiety disorder and similarly for intelligence as moderator (p = .542). Future research on the relationship between puberty, intelligence, and SAD should increase the representation of SAD, improve the PDS, and extend the age and intelligence range. In addition, other factors (e.g., biological, behavioural, social, and parental) underlying the development of SAD should be considered.

Keywords: Social Anxiety Disorder, SAD, Puberty, Pubertal Development, Intelligence, Highly Intelligent, Averagely Intelligent, Gifted, Standard Progressive Raven Matrices, SPRM, Pubertal Development Scale, PDS, hierarchical multiple regression

Layman Abstract

Social anxiety disorder (SAD) is a burdensome disorder where people are afraid of social situations and interactions with others. Most people who suffer from social anxiety disorder are children between 8 and 13. SAD affects one's life on a physiological, behavioural, and cognitive level. Because children with SAD do not like to interact with others, they tend to underachieve in school and be at greater risk of leaving school early, have fewer friendships, lower self-esteem, and reduced peer interactions. If left untreated, it can have a chronic and unremitting course over time. According to previous research, highly intelligent children suffer less from psychological disorders such as SAD. In recent years, little research has been done on the relationship between puberty, intelligence, and SAD. By studying this relationship, a better understanding of puberty and intelligence as possible risk factors for the development of SAD

can be created. This study aimed to test the predictability of SAD by puberty and intelligence. In addition, we tried to investigate whether highly intelligent children reported fewer signs of SAD during puberty than averagely intelligent children. Children (120 in total; aged 9-12) from different schools in the Netherlands completed the Raven intelligence test, and two questionnaires about puberty and social anxiety. The results showed that puberty and intelligence did not predict having SAD. In addition, we did not find evidence for highly intelligent children having fewer signs of SAD during puberty than averagely intelligent children. Future research on the relationship between puberty, intelligence, and SAD should increase the representation of SAD, improve the questionnaire on puberty, and extend the age and intelligence range. In addition, other factors (e.g., biological, behavioural, social, and parental) underlying the development of SAD should be considered.

Introduction

The transition from childhood into early adolescence is marked by puberty, the time when the ability to reproduce begins (Blakemore et al., 2010). The onset age of puberty is estimated at 9 to 14 for girls and 10 to 14 for boys (Wood et al., 2019). Biologically, changes in hormones are responsible for developing secondary sex characteristics, including further development of the genitalia, growth spurts, pubic hair, and for girls, the growth of breasts and the onset of menstruation (Siegler et al., 2017; Wood et al., 2019). Socially, children naturally re-orient themselves from their caregiver(s) towards their peers (Nelson et al., 2005). According to Nelson and colleagues (2016), individuals seek out and express social behaviours appropriate for their development. They explain that deprivation of developmentally appropriate behaviours will result in a longer search for and expression of those behaviours until fulfilled. Social re-orientation is thus a naturally occurring phenomenon and is thus a vital part of further adolescent pubertal development.

Around the same time children start puberty, the prevalence of social anxiety disorder diagnoses peaks (median European prevalence of 2.3%; i.e., between 8 and 13 years of age; APA, 2013; Haller et al., 2015). Social anxiety disorder (SAD) is a psychological disorder characterised by symptoms of fear or anxiety provoked by social situations, interaction with peers, and (thinking about) being negatively evaluated by others (APA, 2013). SAD affects one's life on a physiological, behavioural, and cognitive level. Anxious children can experience headaches, stomach aches, nausea (Hitchcock et al., 2009), and greater sympathetic activation (Mesa et al., 2014). Furthermore, socially anxious children can be shy, express behavioural inhibition, show impaired social skills like expressing fewer facial expressions, and report significantly more distress during social interaction and public performances. Consequently, children with SAD tend to underachieve in school and be at greater risk of leaving school early, have fewer friendships, lower self-esteem, and reduced peer interactions (Hitchcock et al., 2009; Jefferies & Ungar, 2020). If SAD is left untreated, it can have a chronic and unremitting course over time (Hitchcock et al., 2009).

As the period in which SAD diagnoses peak seems to overlap with the start of pubertal development, puberty may be a high-risk period for developing SAD. Very little research has been done on the direct relationship between puberty and social anxiety. A 2007 study by Deardorff and colleagues found a relationship between puberty and social anxiety, with gender significantly moderating this relationship. Significant results were found for girls only. Blumenthal and colleagues (2009) found a relationship between early pubertal development and social anxiety, with the moderating effect of problematic peer relations. An explanation for puberty possibly being a high-risk period for developing SAD can be derived from how adolescents process social information. Research indicates a mismatch during puberty between the development of the affective (AN) and cognitive-regulatory (CRN) parts of the brain (Nelson et al., 2005). Regions being part of the AN reach maturation much earlier in

adolescence than the regions part of the CRN do. This means that children can have powerful emotional responses but lack the ability to regulate them and have trouble contextualising, planning, or inhibiting context-inappropriate behaviour. The mismatch in the development of the AN and CRN during puberty could lead to behavioural difficulties for some individuals or, worse, contribute to the development of psychological disorders like SAD. Therefore, it is hypothesised that puberty is associated with the development of SAD.

Based on this developmental mismatch, it is also hypothesised that highly intelligent (HI) children are less at risk of developing SAD than children that are averagely intelligent (AI). This can be explained by the difference in executive functioning (EF) in HI and AI children. Executive functions encompass cognitive processes, including inhibition, working memory, and cognitive flexibility with which goal-directed behaviour is produced (Best & Miller, 2012; Diamond, 2013). These cognitive processes make higher-order executive functions such as reasoning, problem-solving, and planning possible (Diamond, 2013). Several studies on EF and intellectual ability showed that HI children outperform AI children on executive tests (Arfa, 2007) and excel in working memory, cognitive flexibility, and in higher-order EF, such as reasoning and problem-solving (Rocha et al., 2020). Because executive functioning is part of the CRN explained earlier, and HI children excel in EF, one could deduce that the mismatch found between the affective and cognitive regulatory parts of the brain is less apparent in HI children than in AI children. Indeed, a recent systematic review by Francis and colleagues (2016) reported that HI children exhibit superior social-emotional functioning than AI children and show less evidence of anxiety, depression, behavioural problems, and overall psychopathology. In other words, HI children with powerful emotional responses should be better able to regulate them and contextualise, plan, or inhibit context-inappropriate behaviour, making them less susceptible to developing SAD during puberty.

Knowing when and which individuals might be more at risk for developing SAD symptoms can give caretakers (i.e., parents, teachers) the power to recognise and intervene early on to help prevent (further) development of SAD symptoms (and perhaps other forms of psychopathology; Aune et al., 2020; Colizzi et al., 2020; Dodge et al., 2014; Forbes et al., 2019; McArdle et al., 2011). Based on the results of the current study, suggestions can be made on the theoretical inclusion or exclusion of intelligence as a risk factor for developing SAD. Suppose intelligence is recognised as a risk factor. In that case, it will allow for better social-emotional guidance and education by educators, therapists, and caregivers for children and adolescents in general or within specific intelligence groups. The intelligence level can be utilised to consider a child's self-regulatory abilities. This study will try to answer the following questions:

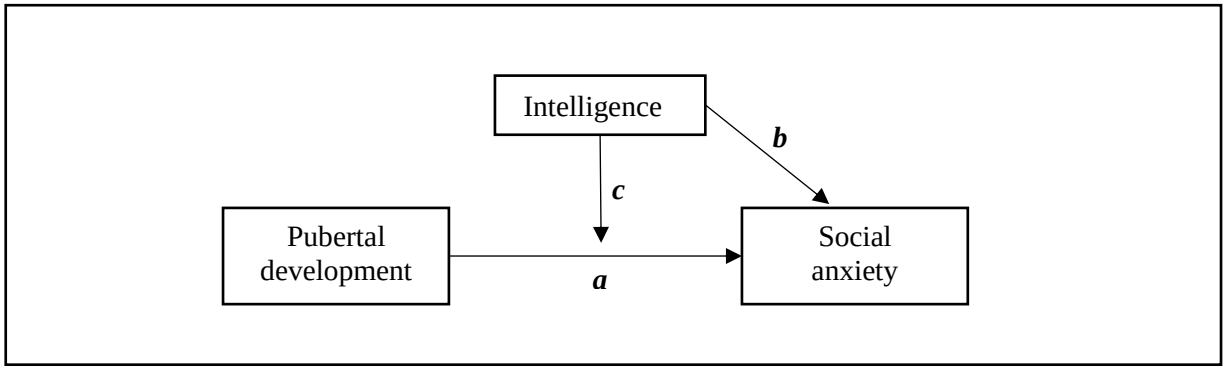
- *Is puberty predictive of signs of SAD?*
- *Is intelligence predictive of signs of SAD?*

- *Is intelligence a significant moderator in the relationship between puberty and signs of SAD?*

It is expected that puberty positively predicts signs of SAD (path *a*; Figure 1), that intelligence negatively predicts signs of SAD (path *b*), and that intelligence is a protective factor in the relationship between puberty and signs of SAD (path *c*), wherein, during puberty, HI children develop fewer signs of SAD than AI children.

Figure 1

Moderation model of Puberty, Social Anxiety, and Intelligence



Note. Path *a* represents the positive prediction of SAD by puberty. Path *b* represents the negative prediction of SAD by intelligence. Path *c* represents the moderating effect of intelligence on path *a*.

Methods

Participants

The sample included 120 children (9-12 years; *M* age = 11.05, SD = 0.77; 52.5% girl; 44.2% gifted). Participants were recruited from Dutch primary schools. Table 1 shows the sample demographics in more detail.

Table 1

Demographic characteristics

	N(%)	*Mean (SD)*
Gender		
Female	63(52.5)	
Male	57(47.5)	
Education		
Regular	67(55.8)	
Gifted	53(44.2)	
Age		11.05(.077)

Procedure

The current cross-sectional study is part of an ongoing study called 'BrightWave' by Zanolie and colleagues. BrightWave focuses on the social-emotional development of typically developing and gifted children. Children (gifted and regular; grades five and six) were recruited from different primary schools in the Netherlands. A pop-up lecture on the BrightWave study was given to enthuse the children into participation. Participants were only included if written informed consent from parents and children was provided. Voluntary participation allowed participants to decide whether or not to continue with the study. The local ethics committee of Leiden University has approved the current study (CEP19-0920/499, date of approval: 21-20-2019).

Data were collected in two sessions. The first session (duration: 120 min) took place in school and consisted of several tasks and questionnaires, including the Puberty Development Scale (PDS; online) and the Raven non-verbal intelligence test (SPRM; pen-and-paper test). After participants finished the first session, participants received a gift (Leonardo da Vinci's Self-Supporting Bridge). The second session (duration: 60 min) took place at home via an online meeting between participants and the research assistants. This session consisted of several questionnaires, including the Youth Anxiety Measure for DSM-5 (YAM-5; online).

Measures

Intelligence testing

Intelligence was measured using the Standard Progressive Raven Matrices (SPRM; McCallum, 2003). The SPRM is a non-verbal pen-and-paper test of intelligence that consists of five sets of 12 items each, resulting in 60 items. Each set and the items within each set become progressively more difficult. Each item is a geometric design from which one piece is missing. The child needs to select the missing piece out of six or eight options that they think completes the geometric design. The duration of the test was 20 minutes. The total intelligence score was calculated by adding correct answers, with a total score range of 0-60. The SPRM has good reliability (Cronbach's alpha of .88-.93) and validity (.73-.89; Abdel-Khalek, 2005; McCallum, 2003). In the current study, Cronbach's alpha for the SPRM was .896.

Puberty

Pubertal development was measured with the Pubertal Development Scale (PDS; Petersen et al., 1988; Robertson et al., 1992). The PDS measures the bodily development of boys and girls and consists of three questions that cover the development of body hair, growth spurts, and changes in complexion, and two gender-specific questions. Male participants answered questions regarding voice change and facial hair; female participants answered questions regarding breast growth and menarche. The answering options ranged from 'no/not yet started' (1) to 'growth or development is complete' (4); the question on menarche could be answered with no (1) or yes (4). In addition, participants could choose the 'I don't know' (coded 0) option. Total pubertal development scores were calculated by adding the answers given to the five questions and ranged from 0 to 20 for the boys and 1 to 20 for the girls. The PDS has strong internal consistency (Cronbach's alpha .91-.96) and test-retest reliability (intraclass correlation coefficient .81-.92; Koopman-Verhoeff et al., 2020). In the current study, Cronbach's alpha for the boys and girls were .321 and .751, respectively. These reliability statistics were based on participants that did not use the 'I don't know'-option (N=63). When considering all participants, Cronbach's alpha for the boys and girls were .103 and .038, respectively.

Social Anxiety

Symptoms of social anxiety were measured using the self-report questionnaire of the Youth Anxiety Measure for DSM-5, which assesses all anxiety disorder symptoms in children and adolescents listed in the DSM-5 (YAM; Muris et al., 2017). Out of the 50 questions the YAM-5 consists of, the six questions representing social anxiety were used. For example, "I am afraid I'll do something embarrassing", of which the answering options ranged from never (1) to always (4). The total social anxiety score was calculated by taking the mean of the six questions representing social anxiety disorder. Except for selective mutism, measuring the inability to speak or reciprocally respond when spoken to by others (APA, 2013), the YAM-5 has good reliability (Cronbach's alpha .86-.93) and validity (correlation of .36-.64) and appears to be a useful tool for assessing anxiety symptoms in children and

adolescents (Ivaki et al., 2021; Muris et al., 2016). In the current study, Cronbach's alpha for the social anxiety subscale was .712.

Statistical Analysis

Data analyses were performed using IBM SPSS (version 23; IBM Corp., 2016). Hierarchical multiple regression was used to test whether puberty and intelligence predict SAD symptoms and if intelligence is a moderator in this relationship. Gender was controlled for, as higher rates of SAD are found in girls than in boys (APA, 2013).

First, assumptions of normality, linearity, independence of residuals, homoscedasticity, multicollinearity, and outliers were checked. Second, a hierarchical multiple regression analysis was performed to assess (1) the predictiveness of puberty and intelligence on signs of SAD after controlling for the influence of gender and (2) the moderating effect of intelligence on the relationship between puberty and SAD (alpha level of .05). Participants that did not complete the SPRM, PDS, or YAM-5 and did not fall in the age range of 9-12 were excluded from data analysis (total excluded N=26). The syntax for the data analyses performed in SPSS can be found in the appendix.

Results

Assumption checks

Before performing a hierarchical multiple regression, assumptions – multicollinearity, outliers, normality, linearity, independence of residuals, and heteroscedasticity – were checked. *Multicollinearity* was assessed using correlational and collinearity statistics. With the inclusion of the interaction variable of pubertal development and intelligence in Model 3, the assumption of multicollinearity was violated (Tolerance < .10 for all predictors; Pallant, 2016). This violation does not come as a surprise. In models with two main variables and an interaction variable, the main variables are likely to be highly correlated with the interaction variable. Violation of this origin is no reason for concern, as the *p*-value for the interaction variable is not affected by multicollinearity (Allison, 2012). With the exclusion of the interaction variable in Model 2, multicollinearity was not violated with Tolerance values of .908 and .998 for pubertal development and intelligence, respectively. Table 2 shows the correlations between all the variables. As expected, the interaction variable is medium to strongly correlated with pubertal development (r = .927) and intelligence (r = .320). On the other hand, the correlation between pubertal development and intelligence is very weak (r = -.040). Similar weak correlations are found between the dependent variable of social anxiety and pubertal development (r = .134), intelligence (r = .070), and the interaction variable (r =.145). For the analysis, correlations of at least r = .300 are preferred (Pallant, 2016).

Table 2

Correlations

	Social anxiety	Pubertal development	Intelligence	Pubertal development *x* Intelligence
Social anxiety	-	-	-	-
Pubertal development	.134	-	-	-
Intelligence	.070	-.040	-	-
Pubertal development *x* Intelligence	.145	.927***	.320***	-

Note. Statistical significance of ***p < .001.

The independence of the residuals was checked using the Durbin-Watson statistic. The value of 1.864 insured no violation of this assumption (Field, 2018). *Normality* was checked using a normal P-P plot, with the expected social anxiety score set against the observed social anxiety scores. Visual

inspection of the P-P plot showed no violation of normality. *Linearity* was checked using partial regression scatterplots, and a scatterplot of the regression standardised residuals against the regression standardised predicted values. Visual inspection of the scatterplots indicated no violation of linearity.

Outliers were checked using the Mahalanobis distance and Cook's distance. With three predictors, the maximum value of the Mahalanobis distance exceeded the critical value of 16.27, indicating the presence of outliers. By sorting the Mahalanobis distance in the data file (descending), 3 data points were found with values exceeding the critical value of 16.27. Fortunately, Cook's distance values of all outliers were lower than 1, indicating no *influential* outliers and no removal of the outliers. Lastly, *heteroscedasticity* was checked by investigating the same scatterplot used for checking linearity. Visual inspections of these scatterplots showed no heteroscedasticity.

Pubertal development as a predictor of social anxiety

Hierarchical multiple regression was performed to examine the predictability of puberty and intelligence on social anxiety. Variables were entered in three steps. Gender was included in all steps, with the addition of pubertal development and intelligence in step 2 and pubertal development, intelligence, and the interaction variable in step 3. See Table 3 for descriptive statistics of all measures.

Table 3

Descriptive statistics of social anxiety, pubertal development, and intelligence

Measures	*Min. score*	*Max. score*	*Mean (SD) Boys (N=57)*	*Mean (SD) Girls (N=63)*	*Mean (SD) Total (N=120)*
Social anxiety	1	3.17	1.66(.41)	1.81(.57)	1.74(.50)
Pubertal development	3	18	7.23(2.12)	8.93(3.17)	8.13(2.84)
Intelligence	25	55	43.30(5.61)	43.06(5.76)	43.18(5.67)

Note. Min. and Max. scores indicate the actual minimal and maximal choices that participants made.

Model 1, consisting of only gender, explained 2.0% (R^2 = .020, p = .120) of the variance in social anxiety (Table 4). Model 2 was used to answer the first and second research question of the predictability of social anxiety by puberty and intelligence. After the entry of pubertal development and intelligence in Model 2, the total variance explained by the model was 3.5% (R^2 = .035). The additional 1.5% explained by the two predictors was not significant (p = .408). Similar insignificant

values were found for the two separate main effects of pubertal development (B = .018, p = .282) and intelligence (B = .007, p = .405), meaning a rejection of the first and second hypothesis.

Model 3 was used to assess the significance of the interaction variable of pubertal development and intelligence[1]. The addition of the interaction variable in Model 3 explained an additional 0.3% (R^2 = .038, p = .408) of social anxiety. The interaction variable was not significant (B = -.002, p = .542). Intelligence was thus not a significant moderator in the relationship between pubertal development and social anxiety. Figure 3 summarises the hypothesised model with the associated statistics.

Table 4

Summary statistics of hierarchical multiple regression predicting social anxiety

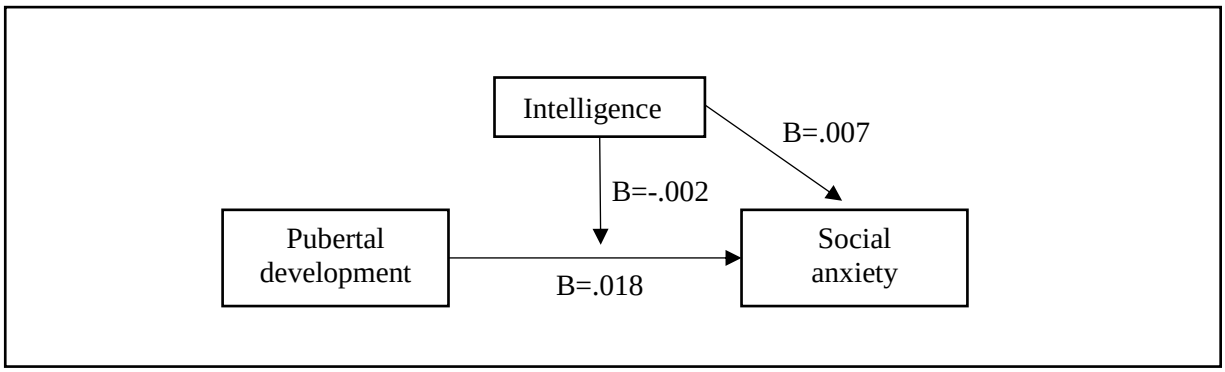
	Social anxiety			
	R^2	B	Std. Error	*p*
Model 1	.020			.120
Gender		.143	.091	.120
Model 2	.035			.408
Gender		.113	.096	.240
Pubertal development		.018	.017	.282
Intelligence		.007	.008	.405
Model 3	.038			.542
Gender		.113	.096	.242
Pubertal development		.103	.140	.462
Intelligence		.023	.028	.408
Pubertal development * Intelligence		-.002	.003	.542

Note. None of the models and individual variables showed statistical significance.

[1] Normally, we would not interpret the interaction effect in the case of an insignificant main effect. However, for assessment purposes, it has been decided to still interpret the interaction effect so the assessors can judge the statistical abilities of the student.

Figure 3

Moderation model of Puberty, Social Anxiety, and Intelligence incl. statistics



Note. None of the effects were statistically significant.

Discussion

The current study aimed to examine the predictability of puberty and intelligence on signs of SAD, with intelligence as a moderating factor. More specifically, it was hypothesised that puberty would positively predict signs of SAD, that intelligence would negatively predict signs of SAD, and that intelligence was a significant moderator in the relationship between puberty and signs of SAD. A hierarchical multiple regression was performed to test the hypotheses. The results showed no relationship between puberty and SAD. Similar results were found for the relationship between intelligence and SAD. In addition, no proof was found for the moderating effect of intelligence on the relationship between puberty and SAD.

The relationship between puberty, intelligence, and SAD

Puberty

Despite the overlap in the age range of puberty onset and the peak in SAD diagnoses (i.e., between 8 and 13 years of age; APA, 2013; Haller et al., 2015), puberty did not predict signs of SAD. Other factors underlying the development of SAD could explain this finding. Several risk factors for the development of SAD are biological factors (e.g., heredity, over-reactive amygdala, bodily changes due to pubertal development), behavioural factors (e.g., shyness, social inhibition, trait anxiety, avoidance coping, social withdrawal), social factors (e.g., stressful or humiliating life events, victim of bullying, fear of negative peer evaluation), and parental factors (e.g., parent psychopathology, overprotective and controlling parenting styles, insecure attachment, child maltreatment and adversity; APA, 2013; Detweiler et al., 2014; Olson, 2021; Pérez-Edgar & Fox, 2005). However, this does not mean that puberty cannot still play a role in the development of SAD, as going through puberty may act as a catalyser between a risk factor and SAD. Every child will undergo pubertal development, which is a vulnerable period for developing psychopathological disorders, such as SAD. This can be explained by the mismatch between the development of the brain's affective and cognitive regulatory parts during puberty (Nelson et al., 2005). The developmental mismatch results in powerful emotional responses to social stimuli and the inability to properly regulate them. It is then hypothesised that puberty will catalyse the development of SAD depending on the amount of (the previously mentioned) risk factors present at the onset of and during puberty. In other words, children meeting a lot of biological, behavioural, social, and/or parental risk factors before and during puberty will be more vulnerable to developing SAD during puberty than children meeting fewer or no risk factors.

That puberty did not predict signs of SAD could also be explained by characteristics of the current study, as our sample included limited reports of signs of SAD. According to the DSM-5 (APA, 2013), approximately 2.3% of children and adolescents in Europe report having SAD, whereas, in the current study, almost none reported having serious signs of SAD. This means that the sample was not representative of the population and that SAD was difficult to predict.

Intelligence

This study also focused on intelligence and (1) its direct effect on SAD and (2) its moderating effect on the relationship between puberty and SAD. It was hypothesised that intelligence would predict signs of SAD and that highly intelligent children would be less at risk of developing SAD than averagely intelligent children. HI children excel in their executive functioning compared to AI children (Arfa, 2007; Rocha et al., 2020), which may lessen the mismatch in the development of the AN and CRN (Nelson, 2005) and, consequently, making HI children less susceptible to developing SAD. Because the main relationship between puberty and signs of SAD was difficult to test, so was the moderating effect of intelligence on this relationship.

Strengths and limitations

This study has several limitations. Firstly, few participants reported having serious signs of SAD. Consequently, testing the predictability of puberty and intelligence on SAD became difficult. Future research should ensure the sample is representative of the population and have enough participants reporting signs of SAD to ensure all relationships can be tested.

Secondly, large amounts of 'I don't know'-answers were reported by participants on the PDS. The 'I don't know'-option on the PDS was coded as zero in calculating the total PDS score. Consequently, the total PDS scores might have differed greatly from the true total PDS scores. To decrease the amount of 'I don't know'-answers we suggest providing visual feedback (e.g., pictures, videos, gifs) of bodily changes for each question. In addition, validation of self-report pubertal development can be given through caregiver questionnaires and saliva samples. These measures can account for the limited knowledge children at young ages (9-12) have about puberty (Cheng et al., 2021). Nevertheless, the PDS could still be used in future research as it is a valid and reliable measurement tool, definitely when one does not have access to nurses or medical personnel (Tanner Stages; Marshall & Tanner, 1969; Marshall & Tanner, 1970) or hormone assessment tools (Mendle et al., 2019).

Thirdly, children in this study had a relatively young age and were mostly at the beginning of puberty (pre and early pubertal). Our main theory was based on the developmental mismatch between affective and cognitive-regulatory parts of the brain. This mismatch might not be as apparent as it would be later in puberty. The chosen age range of 9-12 represents the start of puberty and thus the start of pubertal brain development caused by pubertal hormones (Herting & Sowell, 2017; Laube et al., 2020). Future research should extend the age range to at least 21 years.

The current study did, however, decide to base intelligence on the total score of the Raven Matrices (SPRM, McCallum, 2003). Firstly, we did not know of the methods utilised for the children being labelled as gifted. We eliminated this issue by basing the intelligence score on one test completed by all participants. Secondly, other studies operationalise giftedness in children based on their IQ scores. They are plagued by the lack of consensus in the literature regarding the definition of highly intelligent

children because IQ cut-off scores largely differ (i.e., ranging between 120 and 140; McBee & Makel, 2019). We eliminated this issue by using the total score (instead of an IQ score), including the full range of intelligence. Future research is advised to implement a broader range of intelligence by adding children with below-average intelligence scores, as the current study only included averagely intelligent and highly intelligent children.

Implications for future research

SAD is a serious psychological disorder from which approximately 2.3% of European children and adolescents suffer (APA, 2013) and that affects one's life on a physiological, behavioural, and cognitive level (Hitchcock et al., 2009; Jefferies & Ungar, 2020; Mesa et al., 2014). The current research aimed to find an explanation for the high rates of SAD during early adolescence by studying puberty and intelligence. Unfortunately, due to several limitations, an adequate understanding of the relationship between puberty, intelligence, and SAD could not be provided. However, the current study does provide recommendations for future research on the relationship between puberty, intelligence, and SAD. Aside from the methodological recommendations already mentioned (i.e., increased representation of SAD, improvement of the PDS, and extended age and intelligence range), future research should consider other factors (e.g., biological, behavioural, social, and parental) underlying the development of SAD. There are several ways of doing this. First, longitudinal research following children (and their families) from prenatal to adulthood provides a further understanding of the relationship between risk factors of SAD, the role of puberty, and the development of SAD. Second, the clinical population could give insight into their perspective on the cause of their SAD diagnosis through questionnaires or interviews. Third, performing experimental research on the effects of certain interventions (e.g., therapy, education, training) on risk factors and the relationship to developing SAD during puberty. Future research is thus focused on investigating the causal relationship between risk factors of SAD and its development and the catalysing effect of puberty on this relationship.

Conclusion

The current study aimed to examine the predictability of puberty and intelligence on signs of SAD, with intelligence as a moderating factor. No significant results were found. Methodological suggestions to improve future research would include increased SAD representation, continued use of the PDS but with improvements, and an extended age and intelligence range. Future research should also consider other risk factors influencing the development of SAD and investigate puberty as a catalyser in the relationship between these risk factors and the development of SAD. Continued research on what might contribute to the development of SAD is of the utmost importance. Knowing when and which individuals might be more at risk for developing SAD symptoms can give caretakers (i.e., parents, teachers) the power to recognise and intervene early on to help prevent (further) development of SAD symptoms (and perhaps other forms of psychopathology).

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Syntax

```
* Encoding: UTF-8.
**Encoding: UTF-8.
**STUDY: BrightWave project, Thesis of Axante Drijvers.
**GOAL: Determining predictability of puberty and intelligence on SAD and the moderating effect of intelligence.
**SPSS Location: J:\Workgroups\FSW\BRIGHTWAVE_2019\Persoonlijke mappen Studenten\2022\2022_Axante.

**********STEP 1: Screening and Cleaning the Data.**********

**STEP 1A: Letting SPSS know that the missing data for the PDS = 99.
**STEP 1B: Changing coding PDS_4mB from 0 = no and 1 = yes to 1 = no and 4 = yes.
**STEP 1C: Select cases with age 9-12 & completed PDS & completed YAM.
DATASET ACTIVATE DataSet1.
USE ALL.
COMPUTE filter_$=((9 >= age < 13) & (PDS_1 >= 1) & (1 >= ya11aa01 <= 4)).
VARIABLE LABELS filter_$ '(9 >= age < 13) & (PDS_1 >= 1) & (1 >= ya11aa01 <= 4) (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

**STEP 1D: Creating and calculating total_score_PDS.
IF  (geslacht = 1) total_score_PDS=PDS_1 + PDS_2 + PDS_3 + PDS_4jA + PDS_4jB.
EXECUTE.
IF  (geslacht = 2) total_score_PDS=PDS_1 + PDS_2 + PDS_3 + PDS_4mA + PDS_4mB.
EXECUTE.

**STEP 1E: Calculating total_score_PDS by hand for all participants with values of 99 and counting 99 as 0 points.
**STEP 1F: Create and calculate by hand the Puberty Category Scale
   boys   puberty   girls
   3          pre       2 and no menarch
   4/5        early     3 and no menarch
   6-8        mid       >3 and no menarch
   9-11       late      <=7 and menarch
   12         post      8 and menarch.
**STEP 1G: Obtaining descriptives for onderwijs, geslacht, age, pubertal development, intelligence, and social anxiety.

**Onderwijs.
DATASET ACTIVATE DataSet1.
FREQUENCIES VARIABLES=onderwijs
  /STATISTICS=MINIMUM MAXIMUM
  /ORDER=ANALYSIS.

**Geslacht.
FREQUENCIES VARIABLES=geslacht
  /STATISTICS=MINIMUM MAXIMUM
  /ORDER=ANALYSIS.

**Age.
DESCRIPTIVES VARIABLES=age
```

```
  /STATISTICS=MEAN STDDEV MIN MAX.

FREQUENCIES VARIABLES=age
  /ORDER=ANALYSIS.

**Pubertal development.
DESCRIPTIVES VARIABLES=total_score_PDS
  /STATISTICS=MEAN STDDEV MIN MAX.

* Chart Builder.
GGRAPH
  /GRAPHDATASET NAME="graphdataset" VARIABLES=total_score_PDS MISSING=LISTWISE
REPORTMISSING=NO
  /GRAPHSPEC SOURCE=INLINE.
BEGIN GPL
  SOURCE: s=userSource(id("graphdataset"))
  DATA: total_score_PDS=col(source(s), name("total_score_PDS"))
  GUIDE: axis(dim(1), label("total_score_PDS"))
  GUIDE: axis(dim(2), label("Frequency"))
  GUIDE: text.title(label("Simple Bar of total_score_PDS"))
  ELEMENT: interval(position(summary.count(bin.rect(total_score_PDS))),
    shape.interior(shape.square))
END GPL.

DESCRIPTIVES VARIABLES=total_score_PDS
  /STATISTICS=MEAN STDDEV MIN MAX.

USE ALL.
COMPUTE filter_$=(geslacht = 2).
VARIABLE LABELS filter_$ 'geslacht = 2 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

DESCRIPTIVES VARIABLES=total_score_PDS
  /STATISTICS=MEAN STDDEV MIN MAX.

FILTER OFF.
USE ALL.
EXECUTE.

**Intelligence.
DESCRIPTIVES VARIABLES=total_score_SPM
  /STATISTICS=MEAN STDDEV MIN MAX.

* Chart Builder.
GGRAPH
  /GRAPHDATASET NAME="graphdataset" VARIABLES=total_score_SPM MISSING=LISTWISE
REPORTMISSING=NO
  /GRAPHSPEC SOURCE=INLINE.
BEGIN GPL
  SOURCE: s=userSource(id("graphdataset"))
  DATA: total_score_SPM=col(source(s), name("total_score_SPM"))
  GUIDE: axis(dim(1), label("total_score_SPM"))
```

```
  GUIDE: axis(dim(2), label("Frequency"))
  GUIDE: text.title(label("Simple Bar of total_score_SPM"))
  ELEMENT: interval(position(summary.count(bin.rect(total_score_SPM))),
    shape.interior(shape.square))
END GPL

USE ALL.
COMPUTE filter_$=(geslacht = 1).
VARIABLE LABELS filter_$ 'geslacht = 1 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

DESCRIPTIVES VARIABLES=total_score_SPM
  /STATISTICS=MEAN STDDEV MIN MAX.

USE ALL.
COMPUTE filter_$=(geslacht = 2).
VARIABLE LABELS filter_$ 'geslacht = 2 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

DESCRIPTIVES VARIABLES=total_score_SPM
  /STATISTICS=MEAN STDDEV MIN MAX.

FILTER OFF.
USE ALL.
EXECUTE.

**Social Anxiety.
DESCRIPTIVES VARIABLES=YA11AAsoc
  /STATISTICS=MEAN STDDEV MIN MAX.

* Chart Builder.
GGRAPH
  /GRAPHDATASET NAME="graphdataset" VARIABLES=YA11AAsoc MISSING=LISTWISE
REPORTMISSING=NO
  /GRAPHSPEC SOURCE=INLINE.
BEGIN GPL
  SOURCE: s=userSource(id("graphdataset"))
  DATA: YA11AAsoc=col(source(s), name("YA11AAsoc"))
  GUIDE: axis(dim(1), label("mean social anxiety score"))
  GUIDE: axis(dim(2), label("Frequency"))
  GUIDE: text.title(label("Simple Bar of mean social anxiety score"))
  ELEMENT: interval(position(summary.count(bin.rect(YA11AAsoc))), shape.interior(shape.square))
END GPL.

USE ALL.
COMPUTE filter_$=(geslacht = 1).
VARIABLE LABELS filter_$ 'geslacht = 1 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
```

```
FILTER BY filter_$.
EXECUTE.

DESCRIPTIVES VARIABLES=YA11AAsoc
  /STATISTICS=MEAN STDDEV MIN MAX.

USE ALL.
COMPUTE filter_$=(geslacht = 2).
VARIABLE LABELS filter_$ 'geslacht = 2 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

DESCRIPTIVES VARIABLES=YA11AAsoc
  /STATISTICS=MEAN STDDEV MIN MAX.

FILTER OFF.
USE ALL.
EXECUTE.

**********STEP 2: Preliminary Analyses.**********

**Descriptive Statistics of onderwijs, geslacht, and age.
FREQUENCIES VARIABLES=onderwijs geslacht
  /STATISTICS=STDDEV MINIMUM MAXIMUM MEAN MEDIAN
  /ORDER=ANALYSIS.

DESCRIPTIVES VARIABLES=age
  /STATISTICS=MEAN STDDEV MIN MAX.

**Findings:
    N:          120
    onderwijs:  HB 53, 44,2%; regulier 67, 55,8%
    geslacht:   Jongen 57, 47,5%; Meisje 63, 52,5%
    age:        Min 9,30; Max 12,67; Mean 11,05; SD 0,77.

**********STEP 3: Reliability Analyses.**********

**STEP 3A: Cronbach's Alpha calculation of YAM-5.
RELIABILITY
  /VARIABLES=ya11aa01 ya11aa03 ya11aa05 ya11aa07 ya11aa10 ya11aa12
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA.

**STEP 3B: Cronbach's Alpha calculation of PDS for boys and girls separately.
USE ALL.
COMPUTE filter_$=(geslacht = 1).
VARIABLE LABELS filter_$ 'geslacht = 1 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

RELIABILITY
```

```
  /VARIABLES=PDS_1 PDS_2 PDS_3 PDS_4jA PDS_4jB
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=PDS_1 PDS_2 PDS_3 PDS_4jA PDS_4jB
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA
  /MISSING=INCLUDE.

USE ALL.
COMPUTE filter_$=(geslacht = 2).
VARIABLE LABELS filter_$ 'geslacht = 2 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

RELIABILITY
  /VARIABLES=PDS_1 PDS_2 PDS_3 PDS_4mA PDS_4mB
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=PDS_1 PDS_2 PDS_3 PDS_4mA PDS_4mB
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA
  /MISSING=INCLUDE.

FILTER OFF.
USE ALL.
EXECUTE.

**STEP 3C: Cronbach's Alpha calculation of Raven.
RELIABILITY
  /VARIABLES=A1 A2 A3 A4 A5 A6 A7 A8 A9 A10 A11 A12 B1 B2 B3 B4 B5 B6 B7 B8 B9 B10
B11 B12 C1 C2 C3
    C4 C5 C6 C7 C8 C9 C10 C11 C12 D1 D2 D3 D4 D5 D6 D7 D8 D9 D10 D11 D12 E1 E2 E3 E4 E5
E6 E7 E8 E9 E10
    E11 E12
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=A1 A2 A3 A4 A5 A6 A7 A8 A9 A10 A11 A12 B1 B2 B3 B4 B5 B6 B7 B8 B9 B10
B11 B12 C1 C2 C3
    C4 C5 C6 C7 C8 C9 C10 C11 C12 D1 D2 D3 D4 D5 D6 D7 D8 D9 D10 D11 D12 E1 E2 E3 E4 E5
E6 E7 E8 E9 E10
    E11 E12
  /SCALE('ALL VARIABLES') ALL
  /MODEL=ALPHA
  /MISSING=INCLUDE.

**********STEP 4: Hierarchical Multiple Regression.**********
```

```
**STEP 4A: Create interaction variable PDS-SPM.
DATASET ACTIVATE DataSet1.
COMPUTE int_PDS_SPM=total_score_PDS * total_score_SPM.
EXECUTE.

**STEP 4B: Checking assumptions.
DATASET ACTIVATE DataSet1.
REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING PAIRWISE
  /STATISTICS COEFF OUTS CI(95) R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT YA11AAsoc
  /METHOD=ENTER geslacht
  /METHOD=ENTER geslacht total_score_PDS total_score_SPM
  /METHOD=ENTER geslacht total_score_PDS total_score_SPM int_PDS_SPM
  /PARTIALPLOT ALL
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS DURBIN NORMPROB(ZRESID)
  /SAVE MAHAL COOK.

**Findings:
    Multicollinearity: low correlation SAD-PDS .134; no multicollinearity
    Normality, linearity: normality and linearity
    Independence of residuals: 1.864; positive autocorrelation, relatively normal
    Homoscedasticity: Hetero
    Outliers: 3 outliers; Mah_1 exceeds the critical value of 16.27; max. Cook's d < 1.

**Evaluating the model
    R Square: .038
    R Square change: .018
    Sig. F Change: .540
    ANOVA: F(4,115)=1.151, p=.336

**********THE END.**********
```