Age and puberty differences in stress responses during a public speaking task: Do adolescents grow more sensitive to social evaluation?

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Summary During adolescence pubertal development is said to lead to an increase in general stress sensitivity which might create a vulnerability for the emergence of psychopathology during this period. However, the empirical evidence for increasing stress sensitivity is scarce and mixed.

Biological responses (salivary cortisol and alpha-amylase) were investigated during a social-evaluative stressor, the Leiden Public Speaking Task, in 295 nine to 17-year olds. Specific attention was paid to different elements of the task, that is anticipation to and delivery of the speech. Biological reactivity to the speech task increased with age and puberty, particularly during anticipation.

Current findings support the idea that biological stress sensitivity increases during adolescence, at least in response to a social-evaluative situation. The increasing stress sensitivity appears related to both age and pubertal maturation, but unique contribution could not be distinguished. The importance of measuring anticipation is discussed.

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1. Introduction

Adolescence has been described as a period of increased stress sensitivity (e.g., Andersen and Teicher, 2008). As a result adolescents are expected to show temporarily increased emotional responding, which Dahl refers to as 'normative affective changes' (Dahl, 2004, p. 7). Whereas infants and children are in someway buffered from stress (e.g., by reliance on the primary caregiver), it seems that the end of childhood is marked by the emergence of adult-like, somatic responses to stress (Gunnar and Vazquez, 2006). Several researchers (see for instance Dahl and Gunnar, 2009) attribute this change in stress sensitivity to puberty.

To study changes in stress sensitivity most research to date has focused on changes in basal levels of different systems (e.g., Kiess et al., 1995; Netherton et al., 2004). However, it is also informative to investigate age differences in the
resulting stress responsivity. In a recent commentary, Spear (2009) commented on the value of studies that assess “patterns of somatic activation in response to stressors and other challenges during puberty and the broader adolescent period” (p. 91). Two recent studies (Gunnar et al., 2009b; Stroud et al., 2009) investigated age and pubertal effects on stress responses to a social stressor, that is, an adapted version of the (Child) Trier Social Stress Test (TSST; Kirschbaum et al., 1993). The TSST involves an impromptu speech followed by an arithmetic task in front of an audience.

Gunnar et al. (2009b) used the TSST child version in a sample of eighty-two 9—15-year olds. For a subset of this sample (n = 52) information on puberty was also available. Stress responsivity was investigated through endocrinological data. Although the task resulted in the expected higher levels of cortisol, developmental effects observed for cortisol responsivity were weak. Fifteen-year olds responded more strongly than 11-year olds (p < .10) and puberty was marginally correlated with cortisol responsivity (p < .10). Gender differences were not obtained, except for the finding that among 13-year olds, girls had a stronger cortisol response than boys.

Stroud et al. (2009) used social exclusion tasks in addition to an elaborated version of the TSST. Two developmental groups were created based on age and pubertal status (39 children, 7—12 years and 43 adolescents, 13—17 years). The age ranges served as a proxy for Tanner stages I—III (early-mid puberty) and stages IV—V (late puberty). Participants were randomly assigned to either the TSST or social exclusion tasks. Stroud et al. measured changes in several biological stress parameters—including cortisol and alpha-amylase. In line with Gunnar, the task elicited a physical response and adolescents showed increased physical responding compared to children. For the TSST a statistically significant age effect was observed for cortisol, but not alpha-amylase, while for social exclusion tasks the opposite was observed. Gender effects were not studied, because of a lack of power.

Based on these two studies preliminary evidence has been provided for increased biological stress responsivity during adolescence. However, the reported effects are rather weak (Gunnar et al., 2009b) and inconsistent across biological parameters (Stroud et al., 2009). This might be due to: (i) limited statistical power as a result of relatively small samples per developmental group, and (ii) inadequate assessment of pubertal development. Stroud et al. used age as a proxy for puberty, while Gunnar et al. assessed pubertal development for a subset of their sample. The latter makes it difficult to draw firm conclusions about the contribution of puberty to stress sensitivity.

In addition, it might be useful to distinguish between different components of responses to social stressors, that is the anticipatory response to an upcoming stressor and the immediate response to the stressor at hand. Most stress studies try to avoid any form of anticipation within their design, as this might blunt the response to the task itself (Nicolson, 2008). Anticipation is thought to be kept to a minimum when participants have no foreknowledge about the upcoming task. In laboratory public speaking protocols this is accomplished by asking participants to give an impromptu speech; participants are not aware that the experiment includes giving a speech or they do not know ahead of time what their speech should be about (see Gunnar et al., 2009a). However, the distinction between an anticipation effect of an impending speech task and the immediate effect of the speech task itself might be especially important for revealing developmental differences.

Because peers and their opinions become more important during adolescence (Nelson et al., 2004), older adolescents might start to worry about a speech task in advance whereas younger adolescents might respond more strongly while doing the speech. Furthermore, adolescents’ advanced cognitive abilities allow them to reflect on upcoming events, which would contribute to more worry before the actual speech and increased anticipatory stress responses. For instance, Muris et al. (2002) showed that among 3—14-year olds participants elaborated on their worries more with increasing age and cognitive development. Hence, anticipation might be particularly sensitive to developmental influences.

1.1. Current study

The main focus of the current paper is whether age and pubertal differences can be observed in stress responsivity as a result of pending social evaluation in a public speaking task. For this reason, a large sample of 9—17-year-old girls and boys was recruited to investigate differences in responsivity related to age and pubertal development. The Leiden Public Speaking Task (Leiden PST; Westenberg et al., 2009) used in the study allowed for a differentiated investigation of an anticipation effect of an impending speech task and the immediate effect of the speech task itself.

Biological responsivity was studied with two components of the human stress system: cortisol as a measure of the response of the hypothalamic—pituitary—adrenocortical axis (HPA-axis), and alpha-amylase as a measure of sympathetic nervous system (SNS) activity. The two branches of the stress response work on different timeframes. Cortisol responds slowly and its peak can be detected around 20 min after a stressor’s onset (Nicolson, 2008). It is a suitable measure of enduring stress rather than a short stressor. In contrast, alpha-amylase is released at times when the body needs the most energy, at the time of action (Granger et al., 2007). Consequently, cortisol might be more sensitive to developmental differences during anticipation, whereas alpha-amylase might be more sensitive to developmental differences during the task.

Although gender differences related to biological responsivity have been observed in studies with adults, gender differences have not been observed in youth (e.g., Dedovic et al., 2009). Hence, explicit attention was given to potential gender effects on biological stress responsivity in the current sample of youth.

2. Methods

2.1. Participants

Data used in the current study are part of the Social Anxiety and Normal Development study (SAND; e.g., Miers et al., 2009; Sumter et al., 2009; Westenberg et al., 2009) which was approved by the Leiden University Medical Ethical Committee, the Netherlands and carried out in accordance with the Declaration of Helsinki. Parents provided active
Participants were 144 girls (48.8%) and 151 boys (51.2%). The participants were between 9 and 17 years of age, with a mean age of 13.10 (SD = 2.23) for boys and a mean age of 13.18 for girls (SD = 2.32; t (293) = −0.29, ns.). Participants were assigned to four age groups, namely 9–10 years (n = 68), 11–12 (n = 79), 13–14 (n = 71), and 15–17 (n = 77). The sample included children from all educational streams in the Dutch school system representing varied levels of intelligence in the whole sample and within all age groups.

2.2. Leiden Public Speaking Task (Leiden PST)

The Leiden PST is modelled on a classroom presentation that the age group is familiar with. The participants are requested to speak for 5 min about the type of movies they like or do not like in front of a video camera and a pre-recorded audience of age peers and a female teacher. A week before the actual speech participants are invited to the university; they visit the lab spaces where the speech takes place. They are provided with instructions about the speech and are asked to prepare for it as they would for a presentation at school. The fact that participants are informed about the speech task ahead of time allows for a differentiated investigation of the elevated stress related to the upcoming speech (i.e., anticipation response) and the immediate response caused by the speech itself (i.e., task response). The Leiden PST has been shown to result in elevated levels of self-reported nervousness and physical responses during the task as well as in anticipation to the task in young adolescents (ages 13–15; Westenberg et al., 2009). Hence, recovery levels are taken as the best approximation of rest-state levels and are used as a ‘baseline’ for investigation of the anticipation effect (see Westenberg et al., 2009).

Full details of the task are provided by Westenberg et al. (2009). Briefly, the procedure entails five phases: (1) participants watched a 25-min nature video in order to settle down psychologically and physiologically, (2) 3-min instructions were provided by the researcher to highlight the social-evaluative aspect of the task (e.g., the videotaped speech would be evaluated by age peers at a later date), (3) 5-min rehearsal time, (4) 5-min speech, and (5) a 30-min post-task/recovery phase with various assessments and watching a 10 min clip from the nature film. All sessions started at 14:15 h to minimize diurnal effects.

2.3. Measures

2.3.1. Biological stress parameters

A total of seven saliva samples were collected to assess cortisol (nmol/l) and alpha-amylase (U/l). Saliva samples were collected by passively drooling into plastic vials (IBL-SaliCap®, Germany) directly or through a straw. The first saliva sample was taken after the nature video (i.e., pre-speech sample). Five saliva samples were taken after the speech task, at 5–10 min intervals, to account for the fact that individuals differ in the timing of the cortisol response to a stressful event (Gunnar and Talge, 2007). Following Newman et al. (2007) the maximum value after the speech was taken as the best approximation of the individual stress response (i.e., speech sample). The seventh, and last, saliva sample was taken at the end of the recovery period (i.e., recovery sample).

The determination of cortisol in saliva was performed with a competitive electrochemiluminescence immunoassay ECLIA using a Modular Analytics E170 immunoassay analyzer from Roche Diagnostics (Mannheim, Germany). The sample volume was at least 5 μl. For cortisol missing values due to insufficient volume ranged between 0 and 2.7% for all samples per timepoint. Outliers (>30 nmol/l) were removed at individual time points rather than excluding all samples of the relevant participant; three pre-speech samples, one speech and two recovery samples. The remaining values were log transformed because the raw scores were strongly skewed.

The determination of salivary alpha-amylose (sAA) was performed with an enzymatic colorimetric assay using the maltolheptoside (EPS) substrate on a P-module clinical chemistry analyzer (Roche, Germany) in 400-fold diluted saliva samples. For sAA missing values due to insufficient volume ranged between 0 and 2% for the samples. Outliers (>3 SDs) were removed at individual time points rather than excluding all samples of the relevant participant. Five pre-speech samples were removed and four recovery samples. sAA values were log transformed because the raw scores were strongly skewed.

2.3.2. Pubertal development

Pubertal status was measured with a self-report questionnaire, the widely used Pubertal Development Scale (PDS; Petersen et al., 1988). Based on the PDS manual (Crockett, 1988) Tanner stages were assessed for girls and boys on the following characteristics, for boys this was pubic and facial hair development and for girls pubic hair growth, breast development, and menarche were used. For instance girls who reported some breast or pubic hair development but not menarche were assigned to the beginning pubertal stage (Tanner II). On the other hand girls who reported similar stages of development of these secondary sex characteristics but had also experienced menarche were assigned to Tanner stage III (mid-pubertal). This way Tanner stages could be determined for 284 participants: pre-pubertal (n = 46), beginning (n = 50), mid (n = 76), advanced (n = 73), and post-pubertal (n = 39). Because girls mature at a faster pace than boys, girls were overrepresented in the post-puberty group. As limited differences were expected between participants from the advanced and post-pubertal stages in their biological responses and the fact that it would not be possible to study gender differences in the most advanced groups Tanner stage IV and V were combined.

In earlier studies, moderate to substantial correlations have been observed between the PDS and physical examinations (e.g., Brooks-Gunn et al., 1987; Chan et al., 2010). Furthermore, in the current study puberty correlated significantly with age (r = .78, p < .01).

2.4. Data analysis

Preliminary analyses were conducted to test whether the Leiden PST brought about the expected changes in cortisol and sAA in the total sample. To test whether the task elicited
a stress response, a Mixed-Model ANOVA was run for both variables (cortisol and sAA), with sample time (Time: pre-speech, speech, and recovery) as within-subject variable and gender as between-subjects variable.

To test developmental effects on the stress response two sets of analyses were performed. First, Mixed-Model ANOVAs were run to test the overall effects of age and puberty on cortisol and sAA, with Gender and Developmental Group (age groups or Tanner stages) as between-subjects variables, and sample time (Time) as within-subject variable. A significant Time \texttimes Developmental Group interaction effect indicates an effect of developmental maturity on the stress response.

Second, follow-up analyses were conducted to disentangle the effect of development on both components of the stress response. Difference scores were calculated for cortisol and sAA to index both responses: the Anticipation Response was calculated by subtracting the recovery value from the pre-speech value, whereas the Task Response was calculated by subtracting the pre-speech value from the speech value. ANOVAs were then conducted to test the effect of age and puberty on both responses, with Developmental Group and Gender as between-subjects variables.

Finally, hierarchical regression analyses were conducted to investigate the relative effect of age and puberty on stress responsivity, i.e., anticipation and task responses. Only significant effects will be reported.

3. Results

3.1. Preliminary analyses: effect of the Leiden PST

Mixed-Model ANOVAs with Time as within-subject variable and Gender as between-subjects variable showed a significant main effect of Time for both variables, indicating that cortisol \((F(2, 277) = 246.15, p < .001, \eta_p^2 = .64)\) and sAA \((F(2, 275) = 167.76, p < .001, \eta_p^2 = .55)\) fluctuated during the public speaking session (see Table 1). Post hoc analyses showed that all values differed from each other for the whole sample \((p < .001)\). Specifically, speech values were higher than pre-speech values (i.e., task response), and pre-speech values were higher than recovery values (i.e., anticipation response). Main and interaction effects for gender were not found. Table 1 also presents cortisol and alpha-amylase values for all age groups separately.

### 3.2. Does stress responsivity to the Leiden PST differ between age groups and pubertal stages?

Two sets of analyses were performed to assess the effect of developmental maturity on stress responsivity. First, the findings from Mixed-Model ANOVAs revealed the expected effect of age and puberty on stress responsivity. A statistically significant Time \texttimes Age group interaction was found for both variables: cortisol \((F(6, 548) = 8.33, p < .001, \eta_p^2 = .08)\) and sAA \((F(6, 544) = 4.04, p < .01, \eta_p^2 = .04)\). A Time \texttimes Pubertal stage interaction was found for cortisol \((F(6, 530) = 6.27, p < .001, \eta_p^2 = .07)\) and sAA \((F(6, 526) = 2.32, p < .05, \eta_p^2 = .03)\). Neither main nor interaction effects for gender were obtained.

Second, developmental effects were further explored with ANOVAs that specifically tested the effect of developmental maturity on each component of the stress response (i.e., anticipation and task response).

### 3.2.1. Anticipation response

The findings for anticipation are presented in Fig. 1A and C. Age and puberty effects were observed for cortisol (respectively, \(F(3, 271) = 5.26, p < .01, \eta_p^2 = .06\) and \(F(3, 262) = 4.15, p < .01, \eta_p^2 = .05\), see Fig. 1A). Interaction effects with gender were not significant, neither were the gender main effects. Follow-up polynomial contrast analyses demonstrated that cortisol effects were as expected, namely a positive linear pattern for age and puberty (respectively, LCE = 1.81, \(p < .001\) and LCE = 1.73, \(p < .01\)). Post hoc tests showed that the oldest age group showed more anticipation than the two youngest age groups \((p < .05, \text{Bonferroni})\), and the 13–14-year olds \((p = .06)\). Likewise, the advanced/post-pubertal group showed more anticipation in cortisol than the pre-pubertal \((p < .05, \text{Bonferroni})\) and beginning to mid-pubertal youth \((p = .05, \text{Bonferroni})\).

### Table 1: Effect of Leiden Public Speaking Task on cortisol nmol/l (LN) and alpha-amylase U/l (LN) for all age groups and whole sample.

<table>
<thead>
<tr>
<th></th>
<th>Pre-speech (^{a})</th>
<th>Speech (^{b})</th>
<th>Recovery (^{c})</th>
<th>Post hoc differences</th>
<th>Multivariate test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cortisol</strong></td>
<td></td>
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<tr>
<td>Age group 1</td>
<td>2.11 (.45)</td>
<td>2.30 (.47)</td>
<td>2.03 (.52)</td>
<td>(b &gt; a, c \text{ at } p &lt; .001)</td>
<td>Wilks’ (\Lambda = .55, F(2, 62) = 25.13, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 2</td>
<td>1.96 (.58)</td>
<td>2.18 (.49)</td>
<td>1.83 (.51)</td>
<td>(\text{all differ at } p &lt; .05)</td>
<td>Wilks’ (\Lambda = .29, F(2, 74) = 88.69, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 3</td>
<td>1.82 (.52)</td>
<td>2.11 (.55)</td>
<td>1.56 (.62)</td>
<td>(\text{all differ at } p &lt; .05)</td>
<td>Wilks’ (\Lambda = .26, F(2, 65) = 91.64, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 4</td>
<td>2.03 (.61)</td>
<td>2.18 (.58)</td>
<td>1.58 (.65)</td>
<td>(\text{all differ at } p &lt; .01)</td>
<td>Wilks’ (\Lambda = .26, F(2, 70) = 98.53, p &lt; .001)</td>
</tr>
<tr>
<td>Whole sample</td>
<td>1.98 (.55)</td>
<td>2.19 (.52)</td>
<td>1.75 (.60)</td>
<td>(\text{all differ at } p &lt; .001)</td>
<td>Wilks’ (\Lambda = .36, F(2, 277) = 246.16, p &lt; .001)</td>
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<td><strong>Alpha-amylase</strong></td>
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<tr>
<td>Age group 1</td>
<td>12.17 (.14)</td>
<td>12.42 (.13)</td>
<td>11.98 (.15)</td>
<td>(\text{all differ at } p &lt; .03)</td>
<td>Wilks’ (\Lambda = .51, F(2, 60) = 28.56, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 2</td>
<td>12.50 (.10)</td>
<td>12.79 (.11)</td>
<td>12.34 (.10)</td>
<td>(\text{all differ at } p &lt; .01)</td>
<td>Wilks’ (\Lambda = .47, F(2, 73) = 40.73, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 3</td>
<td>12.51 (.14)</td>
<td>13.00 (.14)</td>
<td>12.24 (.15)</td>
<td>(\text{all differ at } p &lt; .01)</td>
<td>Wilks’ (\Lambda = .38, F(2, 64) = 51.46, p &lt; .001)</td>
</tr>
<tr>
<td>Age group 4</td>
<td>12.74 (.11)</td>
<td>13.04 (.11)</td>
<td>12.37 (.04)</td>
<td>(\text{all differ at } p &lt; .001)</td>
<td>Wilks’ (\Lambda = .31, F(2, 72) = 81.82, p &lt; .001)</td>
</tr>
<tr>
<td>Whole sample</td>
<td>12.49 (.06)</td>
<td>12.82 (.06)</td>
<td>12.24 (.06)</td>
<td>(\text{all differ at } p &lt; .001)</td>
<td>Wilks’ (\Lambda = .45, F(2, 275) = 167.76, p &lt; .001)</td>
</tr>
</tbody>
</table>
In contrast, no age and puberty effects were observed for sAA (respectively, \( F(3, 269) = 2.18, \text{ns} \) and \( F(3, 260) = 0.90, \text{ns} \), see Fig. 1C). The 2 (Gender) \( \times 4 \) (Age group) ANOVA showed a main effect for gender (\( F(1, 269) = 4.00, p < .05, \eta^2_p = .02 \)), but no gender by age group interaction effect. Overall, girls showed a stronger sAA anticipation response than boys.

### 3.2.2. Task response

The findings for the task response are presented in Fig. 1B and D. The age effect was significant for sAA (\( F(3, 276) = 3.04, p < .05, \eta^2_p = .03 \), see Fig. 1D). No gender by age group interaction or gender main effect was found. In line with the hypothesis, a significant linear increase was observed for age (LCE = .12, \( p < .05 \)). Post hoc tests confirmed that the 13–14-year olds showed a stronger rise in sAA from pre-speech to speech than the youngest age group (\( p < .05 \), Bonferroni); other group differences were not statistically significant. The puberty effect showed an upward trend (LCE = .14, \( p < .05 \)), but the ANOVA was not significant (\( F(3, 266) = 1.80, \text{ns} \), see Fig. 1D).

Age and puberty effects for cortisol were not significant (respectively, \( F(3, 279) = 1.23, \text{ns} \) and \( F(3, 269) = 0.57, \text{ns} \), see Fig. 1B). Interaction effects with gender were not significant, neither was the main effect of gender.

### 3.3. Relative contribution of age and puberty on stress responsivity

The relative contribution of age and puberty on the effects described above was tested with hierarchical regression analyses. Only in the cortisol anticipation response a significant proportion of variance is explained by age and puberty (\( R^2 = .05, F(2, 267) = 6.43, p < .01 \)). However, the betas for both age (\( b = .09 \)) and puberty (\( b = .14 \)) were not significant. This means that although age and puberty have a joint contribution to explaining the variance, they do not make a unique contribution.

### 4. Discussion

This research investigated developmental effects on stress responsivity. A large sample was recruited to test age and puberty effects on biological responses during anticipation and task phases of the Leiden Public Speaking Task (Leiden PST). The findings provided support for increased biological stress responsivity during adolescence.

The most consistent and strongest effects were obtained for HPA-axis activity (i.e., cortisol) during the anticipation phase: this response increased with age and pubertal status, particularly during mid-adolescence and advanced puberty. Developmental effects were also obtained for SNS activity (i.e., alpha-amylase) during the speech task but these effects were weaker and less clear-cut. The weaker effect of development during the speech task might be one of the reasons why Gunnar et al. (2009a,b) and Stroud et al. (2009) observed relatively weak stress responses during their stress tasks. They had not assessed the stress response during the anticipation of an impending speech task. The developmental effects might therefore be most pronounced in anticipation of a known stressor.
Pubertal development is presumed to be the driving force behind increasing stress sensitivity during adolescence (e.g., Dahl and Gunnar, 2009). The present findings support the idea that pubertal development explains age-related increases in stress responsivity. However, the reverse was also true: the effect for puberty was explained by the age effect. The latter finding indicates that individual differences for pubertal maturity did not have an incremental effect on stress responsivity beyond the effect of chronological age. The parallel effects of age and pubertal development may have various reasons.

First, the overlap between age and puberty may be due in part to the self-report procedure for assessing pubertal development. Although the Pubertal Development Scale provides a reliable index of pubertal stage, it is still less accurate than physical examinations (e.g., Coleman and Coleman, 2002). Second, in the current study age and puberty were highly correlated. Greater diversity in pubertal development within rather than between age groups would make it possible to study the effect of puberty independently of age. In addition, a longitudinal design would be better able to disentangle age and puberty effects. Third, pubertal development contains several aspects and plays its part on different levels, e.g., hormonal, physiological, and motivational changes, and the emergence of secondary sex characteristics. The current measure mainly reflects the emergence of secondary sex characteristics. It might be that other elements of pubertal development will have a stronger effect on emerging stress sensitivity. Future studies would benefit from assessing these different pubertal parameters. Finally, if puberty is a time of temporarily increased emotional responding (Dahl, 2004), it would be expected that stress responsivity diminishes at the end of puberty. The present study showed the highest level of biological stress responsivity among the most mature groups, namely the oldest age group and the advanced/post-pubertal group. Both groups are on the edge of maturity. By including young adults in future studies the assumed transient nature of stress sensitivity could be tested. This would also allow us to test for gender differences during this latter stage of adolescent development.

At the same time, future studies should also investigate the potential influence of other developmental variables in addition to puberty. The present findings showing that developmental effects seemed strongest during anticipation, might pinpoint cognitive maturation as a prime candidate to include in those studies. Adolescents’ advanced cognitive abilities allow them to reflect on upcoming events, which would contribute to more worry before the actual speech and increased anticipatory stress responses (Muris et al., 2002). Based on Adam (2006) it is expected that such increases in worry might be reflected in increased cortisol levels. In this study adolescents reported on their mood and at the same time provided a saliva sample and it appeared that among participants who reported more worry concurrent cortisol levels were higher.

In line with the results by Gunnar et al. (2009b) and Stroud et al. (2009) no gender by development interaction effects were observed. Furthermore, main effects for gender have not been observed in the current study and several other studies on biological responsivity among youth. Adolescent boys and girls appear to respond similarly to social stressors (Dedovic et al., 2009; Gunnar et al., 2009b; Kudielka and Kirschbaum, 2005). The exception was the gender difference in sAA, the immediate task response was stronger in girls than boys. It is unclear why this difference emerged and replication in future studies will be necessary.

The absence of a main effect of gender is in contrast with the consistent gender effects observed for trait measures of social fear, which show that girls report more social fear than boys (e.g., Westenberg et al., 2004). Due to the influence of sex-role stereotypes boys may be under-reporting their social fears or girls might be over-reporting.

The current study focused on developmental differences in biological responsivity during a speech task. Several studies showed that correspondence between objective and subjective measures is limited (e.g., Mauss et al., 2004). Hence, it is unclear how the current results relate to developmental differences in subjective experience measured during an actual speech task. Future studies will need to address this issue and present a careful account on how objective and subjective measures relate to each other.

An important contribution of the current study is the distinction between anticipation and task responses. However, some uncertainty about the meaning of the anticipation effect remains. It is unclear from which moment on participants anticipated the upcoming speech (e.g., from the first time they heard about it, the morning of the speech, or on their way to the speech session). Anticipation as measured in the current study can best be viewed as short-term anticipation. To better understand how and when anticipation builds up in the week before the speech it will be necessary to include multiple assessment points during that week.

The findings of the present and other recent studies provide support for the hypothesis that adolescence is a period of increased stress sensitivity (Dahl, 2004; Gunnar and Vazquez, 2006). It has been suggested that this sensitivity creates a vulnerability for the emergence of various emotional problems and substance abuse during adolescence, especially in high-risk youth (e.g., Spear, 2009; Paus et al., 2008). The distinction between anticipation and task responses might be useful in this regard. Differences in anticipation, rather than task responsivity, might be an important predictor for psychopathology.

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Conflict of interest

All authors declare that they have no conflicts of interest.

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