Growing up to be fearful? Social evaluative fears during adolescence

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Chapter 3

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

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Abstract

Background — 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.

Methods — Self-reported nervousness and biological responses (salivary cortisol and alpha-amylase) were investigated during a social-evaluative stressor, the Leiden Public Speaking Task, in 295 nine to seventeen year olds. Specific attention was paid to different elements of the task, that is anticipation to and delivery of the speech.

Results — Biological reactivity to the speech task increased with age and puberty, particularly during anticipation. In contrast, subjective experience of stress did not increase with developmental maturity. Gender differences were not observed for biological responsivity but were observed for self-reported nervousness. Older males reported less nervousness than younger ones, whereas no age effect was observed for the girls.

Conclusions — 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 pubertal maturation, but might also be due to cognitive development. The discrepant findings between biological stress sensitivity and self-reported nervousness might have clinical implications and should be the focus of future research.

Introduction

Adolescence has been described as a period of increased stress sensitivity. As a result adolescents should 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 it seems that the end of childhood is marked by the emergence of adult-like, somatic responses to stress (Gunnar & Vazquez, 2006). Several researchers (see for instance Dahl & Gunnar, 2009) attribute this change in stress sensitivity to puberty. Puberty causes many changes in the body on different levels. Among others there are hormonal, physiological, and motivational changes, and the emergence of secondary sex characteristics. All of these changes are said to make adolescents more sensitive to stress.

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, Goodyer, Tamplin, & Herbert, 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, Wewerka, Frenn, Long, & Griggs, 2009; Stroud et al., 2009) investigated age and puberty effects on subjective and objective stress responses to a social stressor, that is an adapted version of the (Child) Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). The TSST involves an impromptu speech followed by an arithmetic task in front of an audience.

Gunnar et al. (2009) used the TSST child version in a sample of 82 nine to fifteen year olds. For a subset of this sample (n=52) information on puberty was also available. Stress responsivity was investigated through subjective and endocrinological data. The task resulted in the expected higher levels of self-reported distress (i.e., I feel completely relaxed vs. totally freaked out) and cortisol. Although no age differences were observed in self-reported distress, weak age effects were observed for cortisol responsivity. Fifteen year olds

responded more strongly than eleven year olds ($p < .10$) and puberty was marginally correlated with cortisol responsivity ($p < .10$). Gender differences were not obtained for subjective nor objective measures, 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 positive and negative affect and two biological stress parameters – cortisol and alpha-amylase. In line with Gunnar, the task affected subjective experience (i.e., the task resulted in expected higher levels of negative and lower levels of positive affect) and elicited a physical response. No age differences were observed in subjective experience, but adolescents did show increased physical responding compared to children. For the TSST a statistically significant response 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 the evidence for increased stress responsivity during adolescence seems mixed. On the one hand age effects were absent for self-reported distress, but the findings did provide preliminary evidence for an increase of biological stress responsivity, although the reported effects are rather weak (Gunnar et al., 2009) 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. This 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, Talge, & Herrera, 2009). 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 opinion become more important during adolescence (Nelson, Leibenluft, McClure, & Pine, 2004), older adolescents might start to worry about a speech task in advance whereas younger adolescents might respond more strongly while doing the speech.

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 scale study was conducted, including enough 9 to 17 year old girls and boys 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.

Subjective stress experience was investigated in terms of self-reported nervousness. The biological response 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 minutes 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, Kivlighan, El-Sheikh, Gordis, & Stroud, 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 self-report data have consistently shown that girls report greater social-evaluative concerns than boys, gender differences related to biological responsivity appear absent in youth (e.g., Dedovic, Wadiwalla, Engert, & Pruessner, 2009). Hence, explicit attention was given to potential gender effects on both subjective and biological stress responsivity.

Method

Participants

Data used in the current study are part of the Social Anxiety and Normal Development study (SAND; e.g., Miers, Blöte, Bokhorst, & Westenberg, in press; Sumter, Bokhorst, & Westenberg, 2009; Westenberg et al., 2009) which was approved by the Leiden University Medical Ethical Committee, the Netherlands.

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. Parents provided active consent; written assent was obtained from participants themselves.

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 five minutes 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 one 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 all participants are informed about the speech task a week before 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 to 15; Westenberg et al., 2009).

Full details of the task are provided by Westenberg et al. (2009). Briefly, the procedure entailed five phases: (1) participants watched a 25 min nature video in order to settle down psychologically and physiologically, (2) three-minute 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) five-minute rehearsal time, (4) five-minute speech, and (5) a 30-minute post-task/recovery phase with various assessments and watching a 10 min clip from the nature film. All sessions started at 14:15 to minimize diurnal effects.

Following Westenberg et al. (2009), recovery levels were taken as the best approximation of rest-state levels, whereas pre-speech levels would be influenced by the anticipatory stress response. Hence, the anticipation response was indexed by a positive difference between pre-speech and recovery. The task response was indexed by a positive difference between speech and pre-speech.

Measures

Self-reported nervousness. Self-reported nervousness was measured with visual analogue scales (VAS; Davey, Barratt, Butow, & Deeks, 2007) at three different moments during the task. The participants indicated how nervous they felt by placing a vertical mark on a 100 mm line anchored by two labels, this is not nervous at all (0) and very nervous (100). VAS-ratings were obtained after the nature video (i.e., pre-speech value), after speech task (i.e., speech value), and at the end of recovery (i.e., recovery value).

Biological stress parameters. A total of seven saliva samples were collected to assess cortisol and alpha-amylase. 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 to 10 minute intervals, to account for the fact that individuals differ in the timing of the cortisol response to a stressful event (Gunnar & Talge, 2007). Following Newman, O'Connor, and Conner (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).

Saliva samples were collected by passively drooling into plastic vials (IBL-SaliCap®, Germany) directly or through a straw. 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 5uL. For cortisol missing values due to insufficient volume ranged between 0 and 2.7% for all samples. 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-amylase (sAA) was performed with an enzymatic colorimetric assay using the maltoheptaoside (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 *SD*s) 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.

Pubertal development. Pubertal status was measured with a self-report questionnaire, the widely used Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). To assess Tanner stages three characteristics of pubertal change were used for girls (pubic hair growth, breast development, and menarche) and two for boys (pubic and facial hair development;

see Crockett, 1988). 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. To equalize the gender distribution the advanced and post-pubertal group (n = 112) were combined. Puberty correlated significantly with age (r = .78, p < .01).

Data analysis

Preliminary analyses were conducted to test whether the Leiden PST brought about the expected changes in self-reported nervousness, cortisol and sAA in the total sample. To test whether the task elicited a stress response, a Mixed-Model ANOVA was run for all three variables, with sample time (Time: pre-speech, speech, 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 effects of age and puberty on self-reported nervousness, 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 x Developmental Group interaction effect indicates an effect of developmental maturity on the stress response.

Second, because the current research questions focused on disentangling anticipation and task responses, follow-up analyses were conducted to directly investigate the effect of development on both components of the stress response. For the three dependent variables difference scores were calculated 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.

Results

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 all variables, indicating that self-reported nervousness ($F(2, 285) = 658.33$, $p < .001$, partial η^2 (η_p^2) = .82), 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 for each variable differed from each other (*p*s < .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. Effect of Leiden Public Speaking Task on self-reported nervousness, cortisol (LN) and alpha-amylase (LN).

	Pre-speech value	*Speech value* M (SD)	*Recovery value*	*Post hoc differences*
Nervousness (N=287)	37.39 (23.70)	61.80 (26.73)	7.20 (11.31)	all differ at p < .001
Cortisol (N=279)	1.98 (0.55)	2.19 (0.52)	1.75 (0.60)	all differ at p < .001
Alpha-amylase (N=277)	12.49 (1.00)	12.82 (1.04)	12.24 (1.07)	all differ at p <. 001

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 the stress response. First, the findings from Mixed-Model ANOVAs revealed the expected effect of age and puberty on the stress response. A statis-

tically significant Time × Age group interaction was found for all variables: self-reported nervousness ($F(6, 556) = 3.48$, $p < .01$, $\eta_p^2 = .04$), 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 three-way interaction Time × Age group × Gender was significant only for self-reported nervousness ($F(6, 556) = 2.52$, $p < .05$, $\eta_p^2 = .03$). Follow-up analyses showed that the Time × Age group interaction was significant for boys ($F(6, 286) = 4.60$, $p < .001$, $\eta_p^2 = .09$), but not for girls ($F(6, 268) = 1.26$, *ns)*.

A Time × Pubertal stage interaction was found for self-reported nervousness ($F(6, 534) = 2.42$, $p < .01$, $\eta_p^2 = .03$), cortisol ($F(6, 530) = 6.27$, $p < .001$, $\eta_p^2 = .07$) and sAA ($F(6, 526) = 2.32$, $p < .05$, $\eta_p^2 = .03$). Interaction effects for gender were not 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)

1. Anticipation response. The findings for anticipation are presented in Figure 1A through Figure 1C. A significant Gender x Age group and Gender x Puberty interaction was observed for self-reported nervousness (respectively, $F(3, 282) = 3.21$, $p < .05$, $\eta_p^2 = .03$ and $F(3, 271) = 3.79$, $p < .05$, $\eta_p^2 = .03$, see Figure 1A). Follow-up ANOVAs showed age and puberty effects for boys (i.e., $F(3, 146) = 6.31$, $p < .001$, $\eta_p^2 = .12$ and $F(3, 138) = 7.74$, $p < .001$, $\eta_p^2 = .12$), but not for girls (respectively, $F(3, 136) = 0.65$, ns and $F(3, 133) = 0.96$, *ns*). In contrast to our expectations, follow-up polynomial analyses showed a significant linear decrease with age (Linear Contrast Estimate (LCE) = -14.42, $p < .001$) and puberty (LCE = -16.10, $p < .001$) for boys. Furthermore, boys from the two oldest age groups and the two most advanced pubertal stages reported less anticipatory nervousness than the two younger age groups and boys from the pre- to beginning pubertal stages (*p*s < .05, post hoc Bonferroni).

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 Figure 1B). Interaction effects with gender were not significant. 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 (ps < .05, Bonferonni), 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, Bonferroni) and beginning to mid pubertal youth (ps = .05, Bonferroni).

In contrast, no age and puberty effects were observed for sAA (respectively, F(3, 269) = 2.18, *ns* and F(3, 260) = 0.90, *ns*, see Figure 1C). Furthermore, the 2 (Gender) x 4 (Age group) ANOVA also showed a main effect for gender (F(1, 269) = 4.00, p < .05, η_p^2 = .02), but no gender by age group interaction effect. Girls showed a stronger sAA anticipation response than boys.

2. Task Response. The findings for the task response are presented in Figure 1D through Figure 1F. A significant Gender x Age group interaction was observed for self-reported nervousness (F(3, 284) = 3.52, p < .05, η_p^2 = .04; see Figure 1D). An age effect was observed for boys (F(3, 145) = 4.42, p < .01, ηp2 = .08), but not for girls (F(3, 129) = 1.29, *ns*). A significant cubic pattern was found for boys (cubic CE = -16.10, p < .01). Post hoc tests showed that the 13 to 14 year old boys had a stronger task response than 11-12 year old (p < .01), 9-10 year old (p = .09), and oldest boys (p = .10). The puberty effect for self-reported nervousness in relation to the task response was not significant (F(3, 273) = 1.19, *ns*, see Figure 1D) and no Gender x Puberty interaction was observed (F(3, 273) = 0.73, *ns*).

Age and puberty effects for cortisol were not significant (respectively, F(3, 279) = 1.23, *ns* and F(3, 269) = 0.57, *ns*, see Figure 1E). Interaction effects with gender were not significant.

Finally, for sAA the age effect was significant (F(3, 276) = 3.04, p < .05, η_p^2 = .03, see Figure 1F), but no gender by age group interaction was found. A significant linear increase was observed for age (LCE = .12, p < .05). Post hoc tests confirmed that the 13 to 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, *ns*, see Figure 1F).

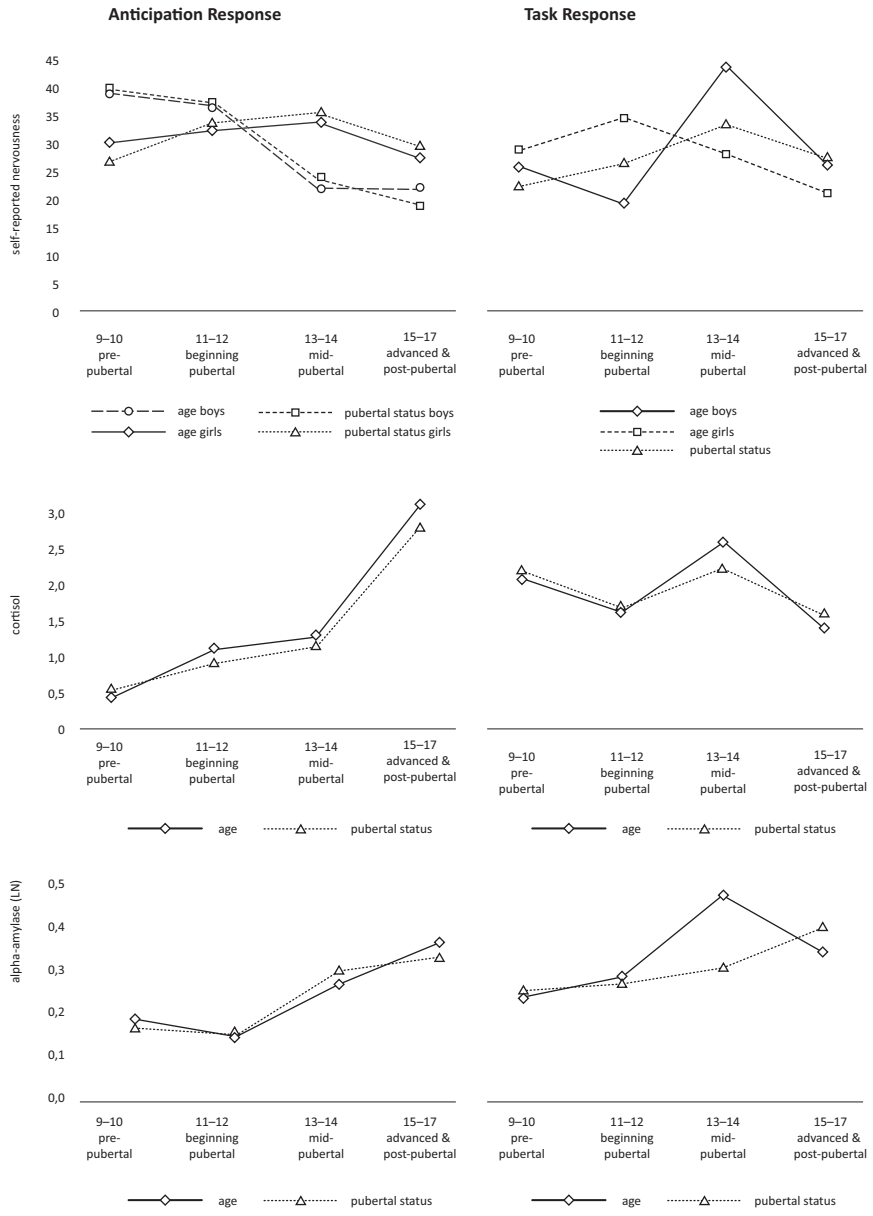

Figure 1. Age and puberty effect on anticipation and task response (from top to bottom self-reported nervousness, cortisol and alpha-amylase). Note that the four age groups are not identical to the four pubertal stages which are assessed with the Pubertal Development Scale.

Discussion

This research investigated developmental effects on stress responsivity. A large sample was recruited to test age and puberty effects on subjective nervousness and 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, whereas subjective experience of stress did not increase.

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. (2009) 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 be most pronounced in anticipation of a known stressor.

The observed developmental effects for biological responsivity were not matched by similar effects for self-reported nervousness: subjective experience of stress did not increase with developmental maturity. Indeed, a negative developmental trend was observed for the males: older boys reported less nervousness than the younger ones in anticipation to the task. The absence of a positive trend is in line with Gunnar et al. (2009) and Stroud et al. (2009): they did not observe increases in subjective stress either. Spear (2009) suggests that adolescents might differ in how they use their somatic responses as information for emotional attribution. Whereas adults interpret their increased somatic responses during a public speaking situation as a sign of apprehension or fear, adolescents might interpret it as being excited or filled with adrenaline. This would also fit with the finding that adolescents desire to be very capable and bold as a way to enhance their status among peers (Dahl & Gunnar, 2009). This might be a reason for the present finding that older boys reported less nervousness in anticipation to the task than the younger ones.

The absence of an upward trend for self-reported nervousness during a social-evaluative situation contrasts with recent findings that show upward trends for self-reported fear of negative social evaluation. While most fears decline with advancing maturity, fear of negative social evaluation steadily increases (Weems & Costa, 2005; Westenberg et al., 2004). In addition, the tendency to *avoid* social-evaluative situations appears to increase as well (Sumter et al., 2009). It may be that older adolescents recognize their greater sensitivity to social evaluation in general, but that they are unable or unwilling to acknowledge this greater sensitivity when they are directly asked during a specific stressful social situation. Future studies need to devise similar indirect measures of subjective experience which might be more sensitive to developmental differences during experimental social stressors.

Gender effects were not observed for biological responsivity. This is consistent with the absence of gender differences in other studies of biological reactivity: adolescent boys and girls appear to respond similarly to social stressors (Dedovic, Wadiwalla, Engert, & Preussner, 2009; Gunnar et al., 2009; Kudielka & Kirschbaum, 2005). These findings are in contrast with 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.

Pubertal development is presumed to be the driving force behind increasing stress sensitivity during adolescence (e.g., Dahl & Gunnar, 2009). However, in the present study the effect sizes suggest that age is a better predictor of biological responses than pubertal development. This might 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 be less accurate than physical examinations (e.g., Coleman & Coleman, 2002). Moreover, in the current study age and puberty were highly correlated. Diversity in pubertal development within rather than between age groups would make it possible to study the effect of puberty independently of age.

At the same time, the present findings suggest that pubertal development might not be the sole factor behind the increasing stress sensitivity during

adolescence. The developmental effects were strongest during anticipation. This might be due in part to cognitive maturation. 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, Merckelbach, Meesters, and Van den Brand (2002) showed that among 3-14 year olds participants elaborated on their worries more with increasing age and cognitive development. Furthermore, in a study by Adam (2006) adolescents reported on their mood and at the same time provided a saliva sample. This study showed that among participants who reported more worry concurrent cortisol levels were higher. Future studies should include measures of cognitive maturity in addition to assessments of pubertal development to better understand the increase in stress sensitivity.

Finally, if adolescence is a time of temporarily increased emotional responding (Dahl, 2004), it would be expected that stress responsivity diminishes at the end of adolescence. 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.

Clinical Implications

An important contribution of the current study is the distinction that has been made between anticipation and task responses. Further studies are needed to carefully investigate the relationship between development and anticipation. Developmental differences in anticipation, rather than task responsivity, might be an important predictor for psychopathology. If anticipation responses set in relatively early in life this could serve as an indicator for psychopathology vulnerability. However, some uncertainty about the meaning of the anticipation effect remains. It is unclear from which moment on participants anticipated the upcoming speech (from the first time they heard about it, the morning of the speech, or on their way to the speech session).

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 & 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, Keshavan, & Giedd, 2008). In addition, the findings of the present and other studies indicate that adolescents do not seem to recognize their increased stress levels while being in a stressful situation. Hence, clinicians should consider that adolescents might be unaware of their own vulnerability or interpret their somatic signals differently to adults. As a result an emerging problem, or possibly deviant social fear, might go unrecognized for a long time. Longitudinal studies are needed to investigate whether responses during the Leiden PST can be used as an early indicator of future psychosocial and emotional problems.

Key Points

- It appears important to distinguish between anticipation to a speech and the actual delivery of a speech.
- Including multiple stress parameters can better inform our understanding of biological responsivity in social-evaluative situations.
- There is a need to develop subjective measures that are sensitive to age differences during social stressors.
- Understanding of normative development of social fears could further our understanding of deviant development (and the onset of social anxiety disorder).