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Heightened HPA-axis stress reactivity and accelerated pubertal progression predicts depressive symptoms over 4-year follow up

Chun Gong[a,b], Xiaonan Duan[a,b], Puyu Su[a,b], Yuhui Wan[a,b], Yuanyuan Xu[a,b], Fangbiao Tao[a,b], Ying Sun[a,b,*]

[a] Department of Maternal, Child & Adolescent Health, School of Public Health, Anhui Medical University, Hefei, Anhui Province, China
[b] Anhui Provincial Key Laboratory of Population Health & Aristogenics Hefei, Anhui Province, China

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ABSTRACT

Pubertal timing has been suggested as biological factors implicated in the onset of depression in girls. This study aims to examine the prospective association between accelerated pubertal progression with depressive symptoms, and to further explore the possible role of individual reactivity to social stress in this association. A total of 56 girls with early puberty timing (assessed through breast Tanner stage) and 56 age-matched normal breast development girls were recruited at Wave 1 (grade 3) and followed for 4 years biennially. Hypothalamic-pituitary-adrenal axis stress reactivity was indexed by four cortisol samples collected before, during and after the Groningen Social Stress Test (GSST) at Wave 1. Depressive symptoms were interviewed through Mood & Feeling Questionnaire (MFQ) at each wave. About 42.9% (24/56) from early pubertal timing group and 19.6% (11/56) from normal control group were found accelerated breast development during 4-year follow-up. Mixed effects linear models illustrated that among accelerated breast development girls, those with heightened stress reactivity is likely to have a 6.62 (95% CI, 1.14–12.11)-point higher MFQ scores, and 41.9% (95%*CI*: 25.2 to 58.6%) higher probability for depressive symptoms, compared with girls with persistent normal breast development and moderate stress reactivity. However, no similar effects were found in girls with accelerated breast development but attenuated stress reactivity. The finding suggests that heightened cortisol reactivity to social stress may represent a useful biomarker in identifying girls at greatest risk of development of depressive symptoms following accelerated pubertal progression.

1. Introduction

Pubertal development is a nonlinear process progressing from prepubescent beginnings through biological, physical, and psychological changes to full sexual maturity (Marceau et al., 2011; Natsuaki et al., 2009). There is a long-standing interest in psychological consequences of variations in pubertal development, highlighting the elevated risks of early maturation in girls for depression and other internalizing problems (Mendle et al., 2018). An accumulation of research evidence suggests that early pubertal timing in girls is associated with symptoms of depression (Ge et al., 2003; Mendle et al., 2007; Black and Klein, 2012; Wasserman et al., 2012), anxiety (Reardon et al., 2009), subclinical internalizing problems (Mendle et al., 2007; Conley and Rudolph, 2009), and higher levels of externalizing behaviors (Dimler and Natsuaki, 2015).

Despite progress in knowledge regarding the influences of the timing of puberty on mental health vulnerability (Mendle et al., 2018), gaps in our understanding remain. The challenge with examining pubertal timing across development is that progression of pubertal development is highly variable. As Mendle (2014) suggested that early pubertal timing is only part of the picture for individual differences in pubertal development. Current debate thus focuses on early puberty starting at 7 to 8 years of age, a specific question is whether there is a subgroup that may be at risk for early menarche and reduced final height. For some girls whose pubertal development starts at 7 to 8 years of age at the first assessment, the risk seems to be no longer relevant when an earlier onset after that age is usually compensated by a slower rate of pubertal progression at the next assessment (Ibáñez et al., 2000; Negriff et al., 2015). Given the dynamic and individually variable process of pubertal development, it is necessary to take into account the

Abbreviations: GSST, groningen social stress test; MFQ, mood and feelings questionnaire; BMI, body mass index; AUCi, area under the curve with respect to the increase; HPA, hypothalamic-pituitary-adrenal; HPG, hypothalamic-pituitary-gonadal; SR, stress reactivity; CI, confidence intervals

* Corresponding author at: Department of Maternal, Child & Adolescent Health, School of Public Health, Anhui Medical University, Hefei, Anhui Province, China.
E-mail address: yingsun@ahmu.edu.cn (Y. Sun).

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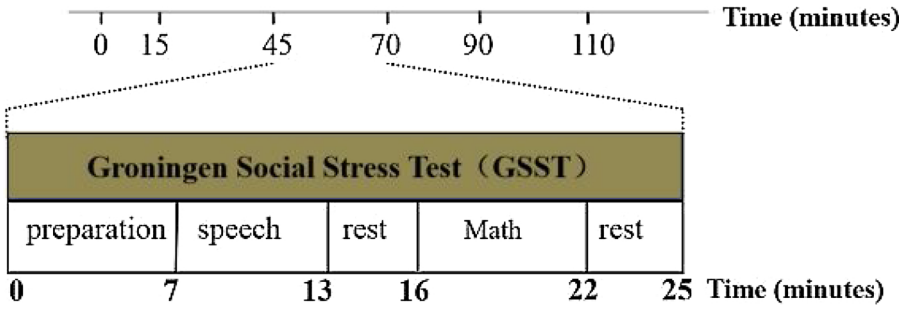

Fig. 1. The timeline of Groningen Social Stress Test (GSST) in this study.

longitudinal pattern of pubertal timing across adolescence rather than measuring pubertal timing at one point in time. This study is designed to repeatedly assess pubertal development at a regular interval over a relatively long time—from before the first signals of puberty (thelarche for girls) until most of the participants have reached full physical maturation, capturing the progression and variation of early pubertal timing more accurately. By analyzing a 4-year longitudinal data from a Chinese cohort of girls who were followed from 8 to 9 years of age at baseline, we hypothesized that only girls with accelerated pubertal progression may be at highest risk for subsequent psychopathology.

Cortisol reactivity represents one of the key biological indicators of stress reactivity. Clinical and epidemiological studies indicate a possible association between the dysregulated cortisol reactivity under social stress with development of depressive symptoms (Booij et al., 2013; Zorn et al., 2017) despite findings regarding HPA-axis responses to psychosocial stressors have been equivocal (Booij et al., 2013; Suzuki et al., 2013; Dieleman et al., 2010). Considering the functional cross-talk between the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes, we hypothesized that accelerated maturing adolescent girls exhibit higher levels of depressive symptoms might be partly due to altered patterns of HPA reactivity to stressors. Thus, another objective of this study was to probe HPA-axis response to psychosocial stress among girls varying in pubertal progression pattern with the goal of identifying the role of stress reactivity dysregulation in the longitudinal association between pubertal development with depressive symptoms over 4-year interval.

2. Method

2.1. Participants

As an ongoing longitudinal study examining psychosocial determinants of growth and development in Anhui Province, China, participants (n = 1296, girls: 568) were recruited of grade 2 and grade 3 from four large elementary schools of Bengbu city, Anhui Province, China in March 2013. Girls (n = 82, 14.4%) who were classified as early pubertal timing (Method Section 2.4 for detailed criteria) were invited and 56 of them agreed to participate in our psychosocial stress test. Fifty-six age-matched normal pubertal development girls were matched as control group. Subsequent wave of data collection took place 2 years (Wave 2) and 4 years (Wave 3) later. We secured approval from Institutional Review Boards at Anhui Medical University and then obtained written informed consent from parents and school teachers, as well as child assent.

2.2. Groningen social stress test

The Groningen Social Stress Test (GSST) is a social stress test that reliably stimulates cortisol secretion (Bouma et al., 2009; Oldehinkel et al., 2011). Experimental sessions took place on weekdays, in soundproof rooms with blinded windows at selected locations in the participants' school; lasted about 2˜3 h; and started between 14:00 h and 15:30 h at Wave 1. At the start of the session, the test assistant, blind to the participants' pubertal timing, explained the procedure. Participants were, on the spot, instructed to prepare a 6-min speech about themselves and their lives and deliver this speech in front of a video camera. After speech, participants were instructed to subtract 17 repeatedly, starting with 13,278 (4-min). They were told that their videotaped performance would be judged on content of speech as well as on use of voice and posture, and rank-ordered by a panel of peers after the experiment. Stress was further provoked by negative feedback by the research assistant, including remarks such as, "No, wrong again, begin at 13,278", or "You are too slow, be as quick as you can, we are running out of time.

2.3. HPA axis reactivity under social stress

HPA axis responses toward the GSST were calculated by area under the curve with respect to increase (AUCi), which was assessed by four salivary samples of cortisol, referred to as C2 to C5. The protocol began with a 10-min rest period to allow the participant to adjust to the research setting. Participants then provided a baseline saliva sample (C1). They were then led into the experimental room and introduced to two research assistants to prepare a personal introduction. Sample C2 was collected before the GSST, reflecting pretest HPA axis activity during rest. At that time, participants were filling out rating scales while sitting quietly. C3 was collected immediately after the GSST, reflecting HPA axis activity at the beginning of the GSST, when participants had to deliver a speech. C4 was collected 20 min after the end of the GSST, reflecting HPA axis activity at the end of the GSST. Finally, C5 was collected 40 min after the end of the GSST, reflecting post stress HPA axis activity. The timeline of GSST was outlined in Fig. 1.

AUCi was analyzed as categorical (quartiles). The highest quartile of AUCi was defined as "heightened stress reactivity (SR)" while the lowest quartile of AUCi was defined as "attenuated SR", "moderate SR" was between these two ranges.

2.4. Early pubertal timing

Breast Tanner stage of each girl was assessed at the three waves by the same female pediatric endocrinologist. Tanner staging was done by palpation of breast tissue in addition to visual inspection. The 25th percentile age of breast TannerIIto Tanner III from 15 388 Chinese girls aged 6.0–18.9 years participated in the China Puberty Collaboration Study was used as cut-off age for early pubertal timing (Sun et al., 2012). Girls who met the following criteria, B2 < 8.0 years of age,

B3 < 9.8 years of age, B4 < 12.4 years of age, B5 < 15.1 years of age or age at menarche < 11.5 years of age, were classified as early breast development at each wave (Sun et al., 2012).

We incorporated information of pubertal assessment at Wave 1 and Wave 3 to create 4 priori-defined mutually exclusive pubertal progression trajectories: (1) persistent normal; (2) persistent early; (3) new development of early (normal at Wave 1 followed by early at Wave 3); (4) gradually normal (early at Wave 1 followed by not early at Wave 3). Persistent early and new development of early were combined as "accelerated breast development".

2.5. Depressive symptoms

At Wave 1 to Wave 3, symptoms of depression were interviewed through the Mood and Feelings Questionnaire (MFQ) (Costello and Angold, 1988). The MFQ has high criterion validity and correlates well with other measures of depression. MFQ score was analyzed as continuous and categorical that a total score of 27 and above was defined as depressive symptoms (Cao and Su, 2009).

2.6. Covariates

2.6.1. Body mass index

At each wave, height was measured with a portable stadiometer to the nearest 0.1 cm and weight with an electronic scale (Tanita TI1618) to the nearest 0.1 kg. Body mass index (BMI) was calculated as weight (in kg) divided by height squared (in m).

2.6.2. Parental education background

Parents at Wave 1 reported educational attainment during the consent process (primary school, junior school, high school and undergraduate/ graduate degree)

2.6.3. Household monthly income

Family monthly income was collected from parents at Wave 1 with the classification from "1" for " < 2,000 CNY" (ca. 291 US$) to "4" for " > 10,000 CNY" (ca. 1453 US$). The responders were asked to choose one answer from the multiple choices.

2.6.4. Family adversity

The family adversity scale is composed of the following seven dichotomous items: parental divorce; parental mental illness; parental alcoholism; interparental conflict; severe corporal punishment; severe physical illness; extreme poverty. Adolescents were defined as exposed to one item if they responded "yes". The measure of family adversity that we used was simply the sum of the 7 items with an exposure; thus, the score of family adversity ranged from 0 (unexposed) to 7 (exposed to all items).

2.7. Statistical analyses

We explored the MFQ and depressive symptoms trajectories of adolescents across 3 repeated assessments in Wave 1 to Wave 3 using linear (MFQ score) and logistic (depressive symptoms: yes/no) growth modeling (Stata command: xtmixed and xtlogit, using mixed-effects maximum likelihood regression and unstructured covariance matrix; Stata, version 13 [StataCorp LLC]). The following model was established:

MFQ_*ij* = b_0 + b_1(Puberty_*ij* × SR_*ij*) + u_0*i* + b_2 BMI_*ij* + b_3 Age_*ij* + b_4 Income_*i* + b_5 Parent_education_*i* + b_6 Adversity_*i* + (u_0*i* + u_1*i* wave_*ij* + e_*ij*)

For i = id for each girl and j = wave. The (b_0 + ... + b_6 Adversity_*ij*) models the fixed effect on MFQ score. The (u_0*i* + u_1*i* wave_*ij* + e_*ij*) represents random effects occur at individual- and wave-level.

We compared the overall and wave-specific mean MFQ across three waves, as well as the intercepts of MFQ scores in the interaction terms of pubertal progression and stress reactivity.

With regard to the effects of pubertal progression pattern on the risk of depressive symptoms, we reported average marginal effects calculated from the logistic growth models. The marginal effect is interpreted as the average increase in the probability of depressive symptoms among girls with gradually normal or accelerated breast development, compared to girls with persistent normal breast development. All the analyses included demographic and familial covariates commonly associated with depressive symptoms: age, BMI, household monthly income, parental education background and family adversity.

3. Results

3.1. General information

As summarized in Table 1, girls in the early puberty and control group did not differ in age, baseline BMI and MFQ scores, as well as parental educational background, family income and whether the single child in the family. Of 112 study girls with a mean (SD) age of 8.27 (0.4) years, sizeable proportions were born to fathers of undergraduate education background (38.4%) and being the only child in the family (75.9%); only 7.1% of households had incomes below the China median. Fig. 2 indicated that compared to normal puberty group (2.52 ± 1.22), AUC increment after GSST was higher in early puberty group (4.02 ± 1.28) ($t = 6.316$, $P < 0.001$).

Of the 56 girls with early puberty timing and 56 girls with normal puberty timing at Wave 1, 24 (42.9%) and 11 (19.6%) of them were found accelerated breast development at Wave 3, respectively. Thirty-two girls had early breast development at Wave 1 but normal breast development at Wave 3 were classified as "gradually normal group".

Table 1
Demographic information of early puberty and control girls at baseline (n = 112).

	n	Early puberty group (n = 56)	Control group (n = 56)	t/χ^2 Value
Age, years	112	8.26 ± 0.4	8.27 ± 0.4	0.138
Body mass index, kg/m²	112	16.91 ± 2.7	17.10 ± 2.5	0.538
Baseline MFQ score	112	9.36 ± 4.1	8.66 ± 4.1	1.662
Father education				
≤Primary school	2 (1.8)	2 (3.5)	0	4.929
Junior school	20 (17.8)	13 (23.2)	7 (12.5)	
High school	47 (42.0)	21 (37.5)	26 (46.4)	
≥Undergraduate	43 (38.4)	20 (35.7)	23 (41.1)	
Mother education				5.205
≤Primary school	3 (2.7)	2 (3.6)	1 (1.8)	
Junior school	24 (21.4)	16 (28.6)	8 (14.3)	
High school	59 (52.7)	24 (42.9)	35 (62.5)	
≥Undergraduate	26 (23.2)	14 (25.0)	12 (21.4)	
Single child in the family	85 (75.9)	42 (75.0)	43 (76.8)	0.049
Family average income				2.998
< 2000 CNY/month	8 (7.1)	6 (10.7)	2 (3.6)	
2000~5000 CNY/month	65 (58.0)	29 (51.8)	36 (64.3)	
5000~10,000 CNY/month	30 (26.8)	16 (28.6)	14 (25.0)	
≥10,000 CNY/month	9 (8.0)	5 (8.9)	4 (7.1)	

MFQ: Mood & Feelings Questionnaire.

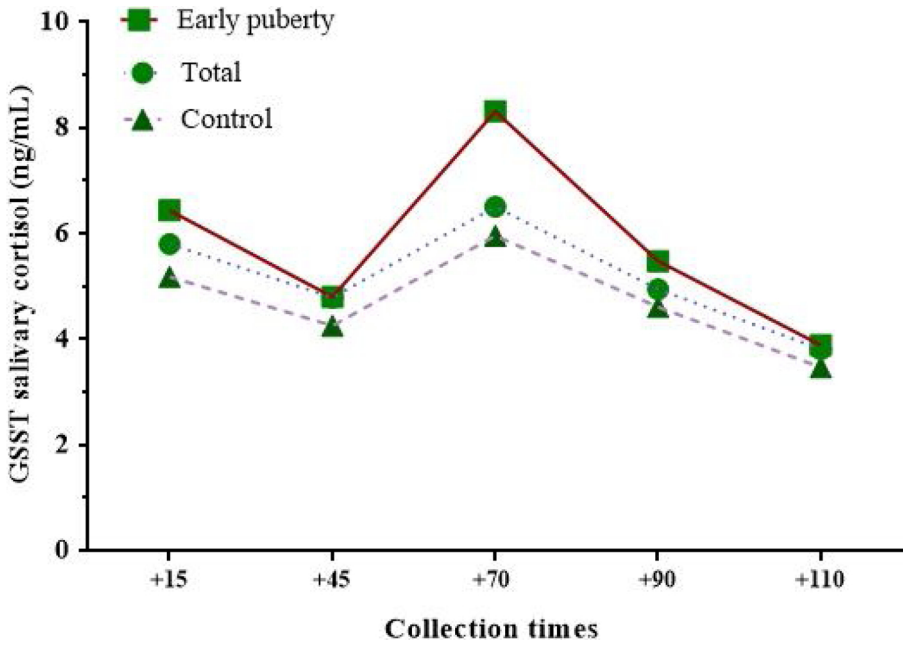

Fig. 2. Salivary cortisol response to GSST by pubertal timing GSST: Groningen Social Stress Test.

Table 2
Mean Mood & Feelings Questionnaire (MFQ) scores and Prevalence of Depressive Symptoms for Girls with Different Pubertal Progression.

	Pubertal Progression Pattern			
	Accelerated breast development (n = 35)	Gradually normal (n = 32)	Persistent normal (n = 45)	*F* value
MFQ score				
Wave 1 (grade 3)	12.83 ± 3.8^{**,a}	10.51 ± 4.7	8.79 ± 5.8	6.296***
Wave 2 (grade 5)	15.54 ± 7.7^{**,a}	12.62 ± 6.4	11.03 ± 7.2	3.781*
Wave 3 (grade 7)	18.49 ± 8.2^{***,a}	14.626 ± 6.7^{**,a}	11.58 ± 7.1	8.096***
Total	15.52 ± 7.2^{***,a}	12.58 ± 6.2	10.46 ± 6.8	16.25***
***P* for time trend**[b]	**0.04**	**0.015**	0.868	
Depressive symptoms, %				
Wave 1 (grade 3)	0	0	0	
Wave 2 (grade 5)	28.6 (10)	15.4 (6)	13.2 (5)	3.286
Wave 3 (grade 7)	51.4 (18)	20.5 (8)	18.4(7)	11.858**
Total	26.7 (28)	12.0 (14)	10.5 (12)	10.53**
***P* for time trend**[b]	0.399	0.914	0.614	

MFQ: Mood & Feelings Questionnaire.
*** $P < 0.001$.
** $P < 0.01$.
* $P < 0.05$.
[a] Compared with persistent normal group.
[b] Adjusted for age, body mass index, parental education, household monthly income, family adversity and stress reactivity to social stress.

Forty-five girls exhibiting normal breast development both at Wave 1 and 3 were classified as "persistent normal group".

3.2. Interactions of pubertal progression with stress reactivity on follow-up MFQ score and depressive symptoms

Table 2 presents means and SDs of MFQ scores, as well as prevalence of depressive symptoms for the 3 different groups of pubertal progression (gradually normal, persistent normal and accelerated breast development) across three time-points. For the sample as a whole, the model indicates that, there was an increasing change in the mean MFQ scores over time ($\beta = 1.67$, 95% CI: 0.27–3.06; $P = 0.019$), and a marginally significant increasing trend in prevalence of depressive symptoms ($\beta = 1.71$, 95% CI: −0.14 to 3.56; $P = 0.07$). Mixed-effect model suggests that compared with girls with persistent normal pubertal development (10.56 ± 4.59), those with accelerated breast development had significant higher MFQ scores in average (12.90 ± 6.54) ($F = 23.210$, $P < 0.001$). Similar differences were found at each repeated assessment.

Prevalence of depressive symptoms among girls with accelerated

Table 3
Mixed-Effects Linear Regression for Interaction Between Pubertal Progression Pattern and Stress Reactivity to Social Stress on MFQ scores[a].

Pubertal progression	Difference in intercept of MFQ Scores, Coefficient (95% CI)	*P* value
Persistent normal* moderate SR	Ref	
Gradually Normal*attenuated SR	1.25 (−4.21 to 6.71)	0.654
Gradually normal* heightened SR	−0.39 (−5.88 to 5.10)	0.889
Accelerated puberty * attenuated SR	3.67 (−1.76 to 9.10)	0.185
Accelerated puberty * heightened SR	6.62 (1.14 to 12.11)	0.018

SR: stress reactivity; MFQ: Mood & Feelings Questionnaire; CI: confidence intervals.
[a] Adjusted for age, body mass index, assessment wave, parental education, household monthly income and family adversity.

Table 4
Marginal effects and standard errors for risk of depressive symptoms[a].

Pubertal progression Pattern	Depressive symptom	*P* value[b]
Moderate SR		
Gradually normal group	−0.03 (0.058)	0.603
Accelerated breast development	−0.06 (0.053)	0.250
Attenuated SR		
Gradually normal	0.03 (0.066)	0.617
Accelerated breast development	0.07 (0.073)	0.306
Heightened SR		
Gradually normal	0.08 (0.083)	0.366
Accelerated breast development	0.419 (0.085)	< 0.001

[a] Adjusted for age, body mass index, parental education, household monthly income, family adversity and stress reactivity to social stress.
[b] Compared with persistent normal group.

breast development increased from 28.6% in grade 5 to 51.4% in grade 7, while the figure was 15.4% in grade 5 to 20.5% in grade 7 among gradually normal puberty girls, and 13.2% to 18.4% among persistent normal puberty girls.

Table 3 presents the analysis of the mixed effects linear models illustrating the influence of interaction between pubertal progression pattern and cortisol response to social stress on MFQ scores. Compared with reference group (girls with persistent normal puberty and moderate stress reactivity), only girls with accelerated breast development and heightened stress reactivity are likely to have, on average, a 6.62 (95% CI, 1.14–12.11)-point higher MFQ scores, after adjusted for age, BMI, assessment wave, parental education, household monthly income and family adversity.

As presented in Table 4, girls who had accelerated breast development with a heightened stress reactivity had a higher probability to depressive symptoms and the marginal effect showed an increase in probability of 41.9% (95%CI: 25.2–58.6%) ($P < 0.001$).

We further explored the possible effect of pubertal progression trajectory on multiple domains of psychopathology with stress reactivity and several covariates controlled (Supplementary Table A). No similar effects were observed for anxiety symptoms ($\beta = 0.5$, 95% CI: 0.3, 1.2; $P > 0.05$) oppositional defiant disorder symptoms ($\beta = 0.7$, 95% CI: 0.3, 1.6; $P > 0.05$), conduct problems ($\beta = 1.2$, 95% CI: 0.5, 3.0; $P > 0.05$), as well as aggressive behaviors ($\beta = 6$, 95% CI: 0.2, 1.8; $P > 0.05$) among girls.

4. Discussion

In this 4-year longitudinal study, our findings demonstrate that accelerated breast development among girls, not early pubertal timing per se, associates with an increased risk of developing depressive symptoms. Of note, this effect is only significant among those with heightened stress reactivity (more than 40% increased risk). The results confirm previous finding of Natsuaki et al. (2009) and add supportive evidence suggesting that the heightened activation of HPA axis under psychosocial stress, as an individual vulnerability factors, may reflect difficulty in regulating emotional responses in a stressful context, which might in turn place early and rapid mature girls at greater risk for developing depressive symptoms.

Early timing of puberty, particularly in girls, is one of the best-researched predictors of psychological distress during adolescence (Ullsperger and Nikolas, 2017; Graber, 2013). Our results highlight the importance of individual variability in pubertal progression trajectory, a topic which has been understudied to date. Using latent growth curve modeling, Mendle and her colleagues (2011) investigated how pubertal tempo and pubertal timing indicated through parent-reported Tanner stages predicted depressive symptoms over a 4-year period in a sample of children recruited from New York City area public schools. The findings indicated that pubertal timing for girls and pubertal tempo for boys emerged as the most salient factor for depressive symptoms. Findings from our study indicated that accelerated pubertal progression instead of early pubertal timing might be important for vulnerability of depressive symptoms among girls. Specifically, we found that not all girls with advanced breast development at baseline exhibit the same risk for subsequent depressive symptoms. For girls with subsequent slow pubertal progression had much lower risk compared with those with accelerated pubertal progression. These results highlight the dynamic and nonlinear nature of pubertal development across adolescence.

Clinical and epidemiological studies, further supported by meta-analytic studies, indicate a possible association between the onset and persistence of depression and HPA axis responsiveness to psychosocial stress (Colich et al., 2015; van den Bos et al., 2017; Booij et al., 2013). Some studies highlighted the links among cortisol response to stress, depression and pubertal stage (Colich et al., 2015; Stroud et al., 2011). Colich et al. (2015) assessed cortisol reactivity and pubertal stage in a sample of 9- to 15-year-old girls and found a significant interaction between cortisol reactivity and pubertal stage in predicting the development of depression. Results suggested that for girls who were more advanced in puberty, both heightened and attenuated cortisol response to the social stress task at baseline would predict the subsequent onset of a depressive episode. Our research extends their study by incorporating longitudinal rather than one time point pubertal assessment, indicating that only heightened stress reactivity may increase susceptibility to depressive symptoms among accelerated maturing girls. These results may help to resolve discrepancies in the literature concerning the association between early pubertal timing and subsequent developmental psychopathologies.

It's interesting to note that a recent meta-analysis (Ullsperger and Nikolas, 2017) suggests that accelerated pubertal timing leads to

heightened risk for all forms of psychopathology, with similar risk amongst both boys and girls. We further explored the possible effect of pubertal progression trajectory on multiple domains of psychopathology with stress reactivity and several covariates controlled. Results indicated that adverse impacts of accelerated pubertal progression on psychopathology was specific to depressive symptoms among girls. No similar effects were observed for oppositional defiant disorder symptoms, conduct problems and aggressive behaviors, as well as anxiety symptoms among girls.

As pubertal development for girls in our study was assessed every two years, this major methodological deficit in our study limit our capacity to plot pubertal development curves and describe individuals' pubertal tempo using nonlinear mixed-effects models proposed by Marceau et al. (2011). Second, we did not explicitly assess the HPA axis cortisol reactivity during follow-up. Some investigators have implicated cortisol response to a laboratory stress-induction task increased as a function of level of pubertal development (Gunnar et al., 2009; van den Bos et al., 2017).

The present study reinforces the benefits to investigators of moving beyond pubertal timing to examine individual variability at pubertal progression. Our results suggest that heightened cortisol reactivity to psychosocial stress may represent a useful biomarker in identifying girls at greatest risk of development of depressive symptoms following accelerated pubertal progression.

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Conflict of interest

No conflict of interest exits in the submission of this manuscript, and manuscript is approved by all authors for publication.

Contributors

Conceptualization and design: Ying Sun, Fangbiao Tao

Acquisition of data: Chun Gong, Xiaonan Duan, Yuhui Wan, Yuanyuan Xu

Analysis and interpretation of data: Chun Gong, Ying Sun, Yuanyuan Xu, Puyu Su

Revising it for intellectual content: Ying Sun, Fangbiao Tao, Puyu Su, Yuhui Wan

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.psyneuen.2019.02.001.

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