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The long reach of puberty: mechanisms underlying sex-dependent links between pubertal timing and adult internalizing symptoms

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Abstract

Background. Pubertal development variations have consequences for adolescent internalizing problems, which likely continue into adulthood. Key questions concern the extent of these links between pubertal timing and adult symptoms, as well as the underlying mechanisms.
Methods. Longitudinal data were available for 475 female and 404 male participants. Pubertal timing was indicated by age at mid-puberty for both groups and age at menarche for female participants (both assessed continuously). Adult self-reported outcomes of recent and lifetime depression and anxiety were predicted from pubertal timing, also controlling for adolescent (then childhood) internalizing problems. Emerging adulthood self-esteem, body dissatisfaction, education level, and age at sexual initiation were examined as mediators of the pubertal timing-adult internalizing link. Multilevel models tested hypotheses.
Results. Pubertal timing had persisting and sex-dependent psychological associations. Specifically, in female, but not male, adults, early puberty was associated with all adult internalizing outcomes, and for past year and lifetime depression symptoms, even after controlling for adolescent internalizing problems. Pubertal timing links with past-year depression symptoms were mediated by age at sexual initiation, while all other persisting pubertal timing links with adult symptoms were mediated by body dissatisfaction. Most findings concerning depression held when childhood internalizing problems were also a covariate.
Conclusions. Leveraging data spanning four developmental periods, findings highlight the associations between pubertal variations and adult internalizing symptoms by revealing underlying sex-dependent behavioral pathways. Only for female participants did pubertal timing affect depression and anxiety in established adulthood, with body dissatisfaction and age at sexual initiation as unique developmental mechanisms.

Introduction

Pubertal development has a profound influence on psychological functioning, particularly internalizing behaviors. Indeed, the biopsychosocial experience of puberty can actuate psychopathology (e.g. depression and anxiety) in youth who are vulnerable. Variations in pubertal development, especially its timing in comparison to peers, are also associated with individual differences in psychological health. For instance, it is well-established that early puberty in female youth is associated with internalizing problems in adolescence (Dorn & Beltz, 2023; Ullsperger & Nikolas, 2017). There is also increasing suggestion that off-time puberty has adverse consequences for male youth, with late maturers reporting heightened depression and anxiety (Graber, 2013). Moreover, increasing evidence shows that some puberty-linked internalizing problems persist into adulthood (Dorn & Beltz, 2023).

Yet, crucial questions remain unanswered about the so-called long arm of pubertal development. Are persisting links from pubertal timing to adult depression and anxiety straightforward extensions of adolescent – or even childhood – internalizing problems? What mechanisms maintain, exacerbate, or eliminate long-term associations of puberty with depression and anxiety symptoms? Finally, are these processes similar across the sexes? (We investigated associations with puberty (a biological and sex-related process), and participants were identified by assigned sex in the parent study.)

Although answers to these questions require full longitudinal data from male and female participants spanning childhood to established adulthood, some insights can be gleaned from

recent empirical work on the persistence of depression. Hypothesized mediators focus on negative body image and life experiences resulting from self- and other reactions to early physical development in female youth. In one longitudinal investigation, links between early menarche and past-week adult depression symptoms (~28 years old) were mediated by discontinued high school education or ever experiencing physical or sexual assault (Mendle, Ryan, & McKone, 2019). Studies of adults providing retrospective reports of pubertal timing and mediators assessed at the same time as outcome measures also show some associations: Depression in male late-maturing young adults (~20 years old) statistically occurred via low self-perceived masculinity, including body image (Beltz, 2018); internalizing problems in very early-maturing young adult females (~21 years old) were mediated by body image dysphoria and low best friend intimacy (Thériault, Otis, Hébert, Gurreri, & Lambert, 2019). Other studies using longitudinal or retrospective designs have failed to find mediation (e.g. teen pregnancy did not mediate the link between early menarche and adult problems; Mendle et al., 2019).

It is difficult to draw strong inferences from these studies. No study used a true longitudinal design with contemporaneous assessment of adolescent pubertal development (in fact, all used retrospective reports with two only assessing menarche), internalizing outcomes assessed in established adulthood, control for adolescent psychological health (as adolescent internalizing problems are strongly associated with internalizing disorders in adulthood, including major depressive disorder [MDD] and generalized anxiety disorder [GAD]; van der Ende, Verhulst, & Tiemeier, 2020), and mediating mechanisms assessed between puberty and adulthood. Furthermore, only one study included male participants (Beltz, 2018).

We aimed to address this substantial knowledge gap by using full longitudinal data to determine the extent to which variations in pubertal timing are linked to adult depression and anxiety in both sexes; we covaried adolescent (and childhood) internalizing problems, and considered multiple intervening mechanisms in emerging adulthood (approximately late teenage years into late twenties) that might account for both recent and lifetime symptoms. We focused on mechanisms previously suggested to be important in prior research, which have established gender-related links with both adult internalizing symptoms and puberty (Dorn & Beltz, 2023; Graber, 2013). First, low self-esteem in adolescence has been linked to an increased risk of depression and anxiety in adulthood (Boden, Fergusson, & Horwood, 2008; Steiger, Allemand, Robins, & Fend, 2014), as well as to early maturation in female youth (Reynolds & Juvonen, 2012; Stojković, 2013). Second, body dissatisfaction has been shown to contribute to anxiety (Vannucci & Ohannessian, 2018) and depression (Sharpe et al., 2018), and is associated with early maturation in female youth (Duncan, Ritter, Dornbusch, Gross, & Merrill Carlsmith, 1985; Shope, Freeman, & Culbert, 2022). Third, educational attainment has been linked to positive well-being (Hergenrather, Zeglin, McGuire-Kuletz, & Rhodes, 2015; Sareen, Afifi, McMillan, & Asmundson, 2011), with potential pubertal timing influences that differ by sex (lower education is associated with late maturation in male youth, but with early maturation in female youth; Koivusilta & Rimpelä, 2004; Cavanagh, Riegle-Crumb, & Crosnoe, 2007). Fourth, early sexual initiation has been associated with adolescent depression and earlier pubertal timing in both sexes, though these associations appear stronger in female than male youth (Baams, Dubas, Overbeek, & van Aken, 2015; Spriggs & Halpern, 2008; Wesche, Kreager, Lefkowitz, & Siennick, 2017).

We hypothesized that (a) in female participants, pubertal timing would be negatively associated with internalizing symptoms (recent and lifetime depression and anxiety), and (b) in male participants, pubertal timing would be positively associated with symptoms (e.g. late maturation would confer higher risk than early maturation for depression) – and that these associations would persist after controlling for adolescent (and childhood) internalizing problems. For persisting associations, we then studied potential mechanisms occurring during emerging adulthood, hypothesizing that persisting internalizing symptoms would be mediated by low self-esteem, body dissatisfaction, lack of college degree, and for female participants, early sexual initiation.

Method

Overview

Participants were enrolled in the Colorado Adoption/Twin Study of Lifespan behavioral development and cognitive aging (CATSLife) (Wadsworth et al., 2019), an extension of two longitudinal studies: the Colorado Longitudinal Twin Study (LTS) and the Colorado Adoption Project (CAP). The LTS sample consisted of monozygotic and dizygotic infant twins and their families (original sample: $N = 966$ individuals from 483 twin pairs), and the CAP sample consisted of adoptee and matched nonadoptee infants and their families (original sample: $N = 732$ individuals from 490 families). Both studies aimed to longitudinally assess genetic and environmental contributions to development from birth through adolescence and had similar data collection procedures before merging for the CATSLife. See additional details in Plomin and DeFries (1985); Rhea, Gross, Haberstick, and Corley (2006); and Wadsworth et al. (2019).

The current study utilized data from puberty and behavioral assessments conducted nearly annually from childhood to mid-adolescence and subsequently during emerging adulthood and established adulthood. Pubertal development was assessed yearly in the LTS/CAP from grades 3 through 9 (ending around age 15, with a few participants assessed until age 17). Depression and anxiety symptoms in established adulthood were assessed in the CATSLife at a median age of 29 years (range 28–43). Psychological mediators were assessed in the LTS/CAP at a median age of 20 years (range 16–33). The adolescent internalizing problems covariate was assessed in the LTS/CAP at age 16 years (range 16–18). The childhood internalizing problems covariate used for sensitivity analyses was assessed in the LTS/CAP at age 7 years.

Participants

The final sample consisted of 879 participants (54% female, from 559 families) who had data on pubertal development (described in Beltz, Corley, Bricker, Wadsworth, & Berenbaum, 2014), and at least one behavioral measure of interest at established adulthood, emerging adulthood, and adolescence. Of the 1458 participants with data on pubertal timing, 331 had missing adolescent behavioral data, and an additional 248 had no adult outcome measure; the resulting sample had data on at least one mediator. The sample was 92% White, 3% Multiracial, 2% Asian, 2% American Indian/Alaskan Native, <1% other (not reported separately due to identifiability), and 1% unknown/not reported; about 5% was Hispanic/Latine.

The procedures contributing to this work comply with the ethical standards of the relevant national and institutional

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committees on human experimentation and with the Helsinki Declaration of 1975, revised in 2008.

Measures

All measures met standard psychometric criteria, including reliability and validity in other samples, as well as internal consistency reliability in the current sample (Table 1).

Pubertal timing

Age at mid-puberty

Timing of mid-puberty was estimated via growth curve analyses from annual reports on the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), a widely used measure of pubertal status. Youth completed five items about the development of secondary sex characteristics: three items for both sexes (body hair, skin changes, and growth spurt) and two sex-specific items (breast development and menarche for female adolescents; facial hair growth and deepening voice for male adolescents). All items except menarche were rated on a 4-point scale (1 = *No development* to 4 = *Development completed*). Menarche was rated as 1 = *Absent* or 4 = *Completed.* Items were averaged to generate a composite at each assessment. Each adolescent's age at mid-puberty as measured by the PDS (score = 2.5) was then calculated via Bayes estimation from sex-stratified random effects logistic growth curve models (Beltz et al., 2014). Estimates were provided for all youth with at least two puberty assessments, but participants were excluded (as in other reports: Beltz et al., 2014; Beltz, Corley, Wadsworth, DiLalla, & Berenbaum, 2020; Chaku et al., 2024) if their estimated age at mid-puberty was three or more standard deviations from the mean.

Age at menarche

Female adolescents who indicated on the PDS that they had reached menarche were asked to report the age (years and months) at which it occurred.

Outcomes: internalizing symptoms in established adulthood

Analyses focused on assessments of the number of recent and lifetime MDD and GAD clinical symptoms. The National Institute of Mental Health (NIMH) Diagnostic Interview Schedule (DIS; Robins et al., 1999) was used to assess the number of past-year and lifetime MDD symptoms. The DIS was also used to assess the number of lifetime GAD symptoms. Assessment of past year GAD symptoms was not precise (e.g. symptom presence/absence versus count), so the Mood and Anxiety Symptom Questionnaire General Distress Anxiety subscale (MASQ GDA; Clark & Watson, 1991) was used to assess symptoms in the past 2 weeks.

Table 1. Descriptive statistics by sex

	Female $N = 475$		Male $N = 404$		Sex difference	
Measure (range of actual scores)	*M* or *N*	SD or %	*M* or *N*	SD or %	b	SE
Pubertal timing predictors (assessed at 8–17 years)						
Age at mid-puberty[1] (estimated: 10.59–18.15 years)	13.28	0.95	15.21	1.03	1.88***	0.07
Age at menarche[2] (9.85–15.67 years)	12.97	1.03	—		—	
Established adulthood outcomes (assessed at 28–43 years)						
Major depressive disorder past year symptoms[3] (0–9)	1.45	2.67	1.02	2.24	−0.44*	0.17
Major depressive disorder lifetime symptoms[3] (0–9)	2.83	3.49	2.02	3.21	−0.83***	0.24
Generalized anxiety disorder past 2 weeks symptoms[4] (11–49, α = .97)	19.06	6.21	18.10	5.14	−0.99*	0.41
Generalized anxiety disorder lifetime symptoms[3] (0–6)	1.31	2.17	0.78	1.67	−0.55***	0.14
Emerging adulthood mediators (assessed at 16–33 years)						
Self-esteem[5] (0–30, α = .90)	24.31	5.81	24.67	5.28	0.41	0.47
Body dissatisfaction[6] (0–5.62, α = .91)	1.41	1.33	0.56	0.77	−0.86***	0.10
Education level (0 = below bachelor's degree, 1 = bachelor's degree or higher)	332	70%	235	58%	−0.11**	0.03
Age at sexual initiation (0 = not early, 1 = early)	71	16%	58	16%	−0.01	0.03
Behavior covariates (assessed at 7 (childhood) and 16–18 (adolescent) years)						
Adolescent internalizing problems[7] (0–40)	6.16	6.36	5.11	5.84	−1.11*	0.44
Childhood internalizing problems[7] (0–28)	5.10	4.36	4.88	4.42	−0.24	0.35

Note: Numeric superscripts indicate measure validity references. Sex differences (0 = female, 1 = male) were assessed with random intercepts-only multilevel models to adjust for family status; *b* is the unstandardized coefficient, with its standard error (SE).
[1]Brooks-Gunn et al., 1987; Ellis, 2004; Koopman-Verhoeff et al., 2020; Mendle et al., 2019; Schmitz et al., 2004; Shirtcliff et al., 2009.
[2]Mendle et al., 2019.
[3]Allen & Becker, 2019; Robins et al., 1981.
[4]Clark & Watson, 1991.
[5]Beeber et al., 2007; Donnellan et al., 2015; Eklund et al., 2018; Martín-Albo et al., 2007; Sinclair et al., 2010.
[6]Berg et al., 2012.
[7]Marti et al., 2022.
*p < .05, **p < .01, ***p < .001.

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Mediators: measures in emerging adulthood

Hypothesized mediators available in the CATSLife were generally assessed in emerging adulthood (self-esteem, body dissatisfaction, and education level). Age at sexual initiation was not tightly developmentally constrained, as it generally occurs between puberty and emerging adulthood.

Self-esteem

Participants completed the Rosenberg Self-Esteem Scale (Rosenberg, 1965) between the ages of 16 and 21 (M = 17.32, SD = 0.57). Scores were averages of 10 items (rated on a four-point Likert scale).

Body dissatisfaction

Body dissatisfaction was assessed with the 8-item Shape Concern subscale of the Eating Disorder Examination Questionnaire (Fairburn & Beglin, 1994), a 28-item self-report measure of the frequency and severity of behaviors associated with eating disorders in the past 28 days. It was completed between the ages of 21 and 33 (M = 23.65, SD = 2.24).

Education level

Participants reported on the level of education obtained by early adulthood (21–25 years) (McClelland, Acock, Piccinin, Rhea, & Stallings, 2013; Wadsworth et al., 2019). Education was scored to reflect the presence or absence of a bachelor's degree or higher.

Age at sexual initiation

At age 17 or 21, participants reported the age of their first sexual experience if it had occurred (Bricker et al., 2006). Age at sexual initiation was categorized to be early or not, with age 16 serving as the cut-off based on the sample mode. Consistent with other work (Beltz et al., 2020; Chaku et al., 2024; Liang & Chikritzhs, 2013; Upchurch, Lillard, Aneshensel, & Li, 2002), participants reporting ages younger than 13 (n = 8) were excluded.

Covariate: internalizing problems in adolescence and childhood

To determine whether pubertal timing was uniquely linked to adult depression and anxiety and mechanisms that might mediate links, we controlled for parallel adolescent internalizing problems associated with pubertal timing. Caregivers reported on adolescent internalizing behavior problems at age 16 using the Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL internalizing broadband scale raw score, which includes anxiety, depression, and withdrawn behaviors, was used, although CBCL is caregiver-reported and the adult measures are self-reported. In sensitivity analyses controlling for childhood internalizing problems (as described in Sensitivity Analyses), the caregiver-reported CBCL internalizing broadband scale raw score at age 7 was used.

Analytic plan

First, descriptive information was examined: (a) sex differences in study variables were tested using multilevel models, with nesting in families accounted for with random intercepts; (b) zero-order correlations among the study variables were calculated for descriptive purposes.

Second, hypotheses were tested with a series of multilevel models accounting for nesting in families via random intercepts for recent and lifetime depression and anxiety in established adulthood (Figure 1). All multilevel models were conducted in SPSS 29, separately by sex, controlling for initial study (due to some sample differences between included participants from the LTS and CAP; Supplementary Table 1) and age, and estimated with maximum likelihood. Type I error was .05.

For each outcome, several models were considered. Model 1 ($X \rightarrow Y$; Figure 1a) provided a test of pubertal timing (X) associations with internalizing symptoms in established adulthood (Y). If significant (showing continuing links with pubertal timing), three additional multilevel models were tested to examine potential persistence and underlying mechanisms (mediators). Model 2 (X adding $C \rightarrow Y$; Figure 1a) added adolescent internalizing problems; and (C) as a covariate to determine whether pubertal timing predicted adulthood depression and anxiety above and beyond adolescent behavior. Models 3 and 4 added potential mediators (M: self-esteem, body dissatisfaction, education level, and age at sexual initiation) of pubertal timing associations with internalizing symptoms. The measure of adolescent internalizing problems was not included in Model 3 ($X \rightarrow M \rightarrow Y$; Figure 1b), but it was added in Model 4 (X adding $C \rightarrow M \rightarrow Y$; Figure 1b) as a covariate. Two equations are estimated in mediation: In the first (Mdl_M: mediator model), X predicts M; in the second (Mdl_Y: outcome model), X and M predict Y. As pubertal timing is a continuous predictor (i.e. age), its multilevel model coefficients are interpreted in yearly increments (e.g. 'early' puberty reflects an increase/decrease in symptoms with every one-year decrease in pubertal timing).

Multilevel mediation models were tested using MLmed in SPSS (Rockwood & Hayes, 2017), estimating within-family and between-family effects, and using the Monte Carlo method to estimate the sampling distribution of indirect effects, with inferences made via 95% confidence intervals; if the intervals did not include zero, then mediation was inferred (in the context of covariates). The primary hypotheses concerned between-family mediation. Within-family mediation results are also reported for completeness in the Supplementary Material; hypothesized mechanisms may be amplified within families, but power is reduced.

Sensitivity analyses

Sensitivity analyses controlling for both adolescent and childhood internalizing problems (Supplementary Figure 1) were conducted to ensure that significant pubertal timing links with adulthood outcomes were not explained by prepubertal behaviors. The age 7 CBCL internalizing broadband scale raw score was used to measure childhood internalizing problems, ensuring that these problems were captured before adrenarche and thus would not be impacted by puberty.

Missing data

Potential influences of missingness on results were assessed by comparing the 879 participants included in this study to the 579 participants from the parent study excluded from analyses. Participants with available data did not differ from those with missing data in terms of race or ethnicity but were more likely to be female and nonadoptees (Supplementary Table 2). Female participants included in analyses had later menarche, were less likely to have early age at sexual initiation, and had lower levels of adolescent internalizing problems than those excluded (Supplementary Table 3). Male participants included in analyses had more recent MDD symptoms and were less likely to have early age at sexual initiation than those excluded (Supplementary Table 4).

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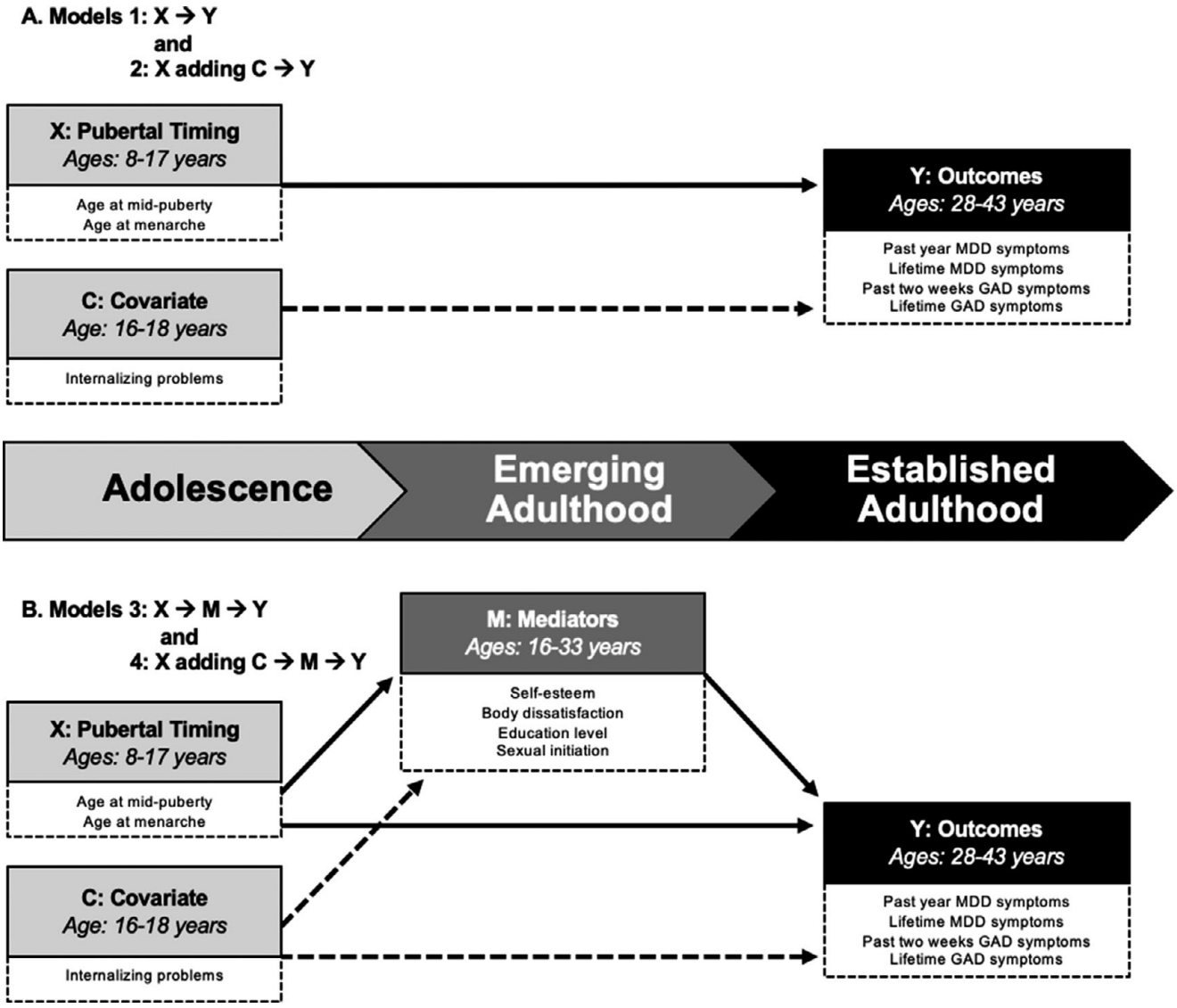

Figure 1. Multilevel models testing hypotheses of pubertal timing associations (*X*) with outcomes in established adulthood (*Y*) via mediators in emerging adulthood (*M*), and covarying behavior problems in adolescence (*C*). *Note*: (a) Model 1 assesses the links between pubertal timing and established adulthood outcomes (solid arrow). Model 2 incorporates an adolescent behavior covariate (dashed arrow). (b) Model 3 assesses emerging adulthood mediations of the associations pubertal timing and established adulthood outcome links (solid arrows). Model 4 adds an adolescent behavior covariate (dashed arrows). There was minor overlap in assessment periods due to scheduling constraints in the original studies, with 4% of participants having their adolescent internalizing problems covariate assessment overlap with their self-esteem mediator assessment. All four models controlled for age and initial study. MDD, 'major depressive disorder'; GAD, 'generalized anxiety disorder'.

Results

Descriptive statistics

Descriptive data are shown in Table 1. Most expected sex differences were found: Compared to male participants, female participants had earlier pubertal timing, higher levels of major depressive and generalized anxiety symptoms in established adulthood, higher body dissatisfaction and likelihood of attaining a bachelor's degree during emerging adulthood, and higher levels of internalizing problems in adolescence. Zero-order correlations among study variables are shown in Supplementary Table 5.

Persistence and mechanisms of pubertal timing associations with internalizing symptoms in established adulthood

The results of hypothesis testing using multilevel models to predict internalizing symptoms and between-family mediation are shown in Tables 2 (MDD) and 3 (GAD) for female participants and in Supplementary Table 6 for male participants. Within-family mediation results for female participants are in Supplementary Tables 7 and 8. As noted, for outcomes with nonsignificant associations with pubertal timing (Model 1), Models 2–4 were not tested.

Predicting female participants' internalizing symptoms

Major depressive disorder

Recent (past year) MDD symptoms were predicted by age at mid-puberty, with younger age associated with higher adult means (Model 1). This remained significant after controlling for adolescent internalizing problems (Model 2). Age at menarche showed a similar pattern of results but was not significant in Model 1.

The link between age at mid-puberty and past-year MDD symptoms was significantly mediated by age at sexual initiation (Model 3): early timing was associated with early sexual initiation,

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Table 2. Multilevel model results for female participants: between-family associations between pubertal timing (*X*) and major depressive disorder symptoms in established adulthood (*Y*) via mediators in emerging adulthood (*M*), covarying adolescent internalizing problems (*C*), initial study (*S*), and age (*A*)

			X→*M*		*M*→*Y*		*C*→*Y*		*X*→*Y*		*M* indirect effect		Random effect			
Mdl	M	N	b	SE	b	SE	b	SE	b	SE	b	[95% CI]	Est	SE	S	A
Past year symptoms																
***X* = Age at mid-puberty**																
1	—	473	—	—	—	—	—	—	**−0.27***	**0.12**	—	—	1.01	0.58		
2	—	473	—	—	—	—	**0.32****	**0.12**	**−0.25***	**0.12**	—	—	0.81	0.58		
3	SE	356	0.59	0.38	**−0.08****	**0.03**	—	—	−0.07	0.16	−0.04	[−0.13,	Mdl_M: 8.32**	3.10		
												0.01]	Mdl_Y: 0.84	0.61		
	BD	318	**−0.28****	**0.09**	0.07	0.14	—	—	−0.21	0.17	−0.02	[−0.11,	Mdl_M: 0.48**	0.15		
												0.06]	Mdl_Y: 0.47	0.66		
	EL	473	0.04	0.03	−0.05	0.30	—	—	**−0.32***	**0.14**	−0.002	[−0.03,	Mdl_M: 0.11***	0.02	+	+
												0.03]	Mdl_Y: 1.05	0.58		
	AS	442	**−0.08*****	**0.02**	**1.15****	**0.40**	—	—	−0.21	0.15	**−0.09**	**[−0.19,**	Mdl_M: 0.05***	0.01		
												−0.02]	Mdl_Y: 0.78	0.66		
4	SE	356	0.41	0.36	−0.06	0.03	Mdl_M: **−0.27*****	**0.06**	−0.05	0.16	−0.02	[−0.08,	Mdl_M: 5.93*	2.80		
							Mdl_Y: 0.05	0.03				0.02]	Mdl_Y: 0.75	0.61		
	BD	318	**−0.25****	**0.09**	−0.02	0.13	Mdl_M: **0.04****	**0.01**	−0.18	0.17	0.004	[−0.07,	Mdl_M: 0.44**	0.14		
							Mdl_Y: **0.09****	**0.03**				0.08]	Mdl_Y: 0.25	0.64		+
	EL	473	0.03	0.03	0.11	0.30	Mdl_M: **−0.01****	**0.004**	−0.27	0.14	0.003	[−0.02,	Mdl_M: 0.11***	0.02		+
							Mdl_Y: **0.06****	**0.02**				0.03]	Mdl_Y: 0.86	0.57		
	AS	442	**−0.08*****	**0.02**	**1.00***	**0.40**	Mdl_M: **0.01****	**0.003**	−0.18	0.15	**−0.08**	**[−0.16,**	Mdl_M: 0.04***	0.01		
							Mdl_Y: **0.06***	**0.02**				**−0.01]**	Mdl_Y: 0.61	0.65		
***X* = Age at menarche**																
1	—	435	—	—	—	—	—	—	−0.13	0.13	—	—	1.23	0.63		
Lifetime symptoms																
***X* = Age at mid-puberty**																
1	—	473	—	—	—	—	—	—	**−0.45****	**0.16**	—	—	3.09**	0.92		
2	—	473	—	—	—	—	**0.38***	**0.16**	**−0.42****	**0.16**	—	—	2.91**	0.91		
3	SE	356	0.59	0.38	**−0.11****	**0.04**	—	—	−0.28	0.22	−0.06	[−0.17,	Mdl_M: 8.32**	3.10		
												0.02]	Mdl_Y: 2.66**	0.98		

(*Continued*)

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Table 2. (*Continued*)

			X→M		*M→Y*		*C→Y*		*X→Y*		*M* indirect effect		Random effect			
Mdl	M	N	b	SE	b	SE	b	SE	b	SE	b	[95% CI]	Est	SE	S	A
	BD	318	**−0.28****	**0.09**	**0.42***	**0.19**	—	—	−0.26	0.25	**−0.12**	**[−0.27,**	Mdl_M: 0.48**	0.15		
												−0.01]	Mdl_Y: 3.21**	1.04		
	EL	473	0.04	0.03	0.32	0.40	—	—	**−0.64****	**0.19**	0.01	[−0.02,	Mdl_M: 0.11***	0.02	+	+
												0.06]	Mdl_Y: 3.28***	0.90		
	AS	442	**−0.08*****	**0.02**	**1.28***	**0.54**	—	—	**−0.49***	**0.21**	**−0.10**	**[−0.22,**	Mdl_M: 0.05***	0.01		
												−0.02]	Mdl_Y: 4.20***	0.96		
4	SE	356	0.41	0.36	**−0.09***	**0.04**	Mdl_M: **−0.27*****	**0.06**	−0.26	0.22	−0.04	[−0.13,	Mdl_M: 5.93*	2.80		
							Mdl_Y: 0.04	0.04				0.03]	Mdl_Y: 2.60*	0.98		
	BD	318	**−0.25****	**0.09**	0.32	0.19	Mdl_M: **0.04****	**0.01**	−0.22	0.24	−0.08	[−0.22,	Mdl_M: 0.44**	0.14		
							Mdl_Y: **0.10***	**0.04**				0.01]	Mdl_Y: 2.95**	1.02		
	EL	473	0.03	0.03	0.51	0.40	Mdl_M: **−0.01****	**0.004**	**−0.58****	**0.19**	0.01	[−0.02,	Mdl_M: 0.11***	0.02		+
							Mdl_Y: **0.08***	**0.03**				0.07]	Mdl_Y: 3.06***	0.89		
	AS	442	**−0.08*****	**0.02**	**1.14***	**0.54**	Mdl_M: **0.01****	**0.003**	**−0.46***	**0.20**	**−0.09**	**[−0.20,**	Mdl_M: 0.04***	0.01		
							Mdl_Y: 0.06	0.03				**−0.01]**	Mdl_Y: 4.05***	0.95		
***X* = Age at menarche**																
1	—	435	—	—	—	—	—	—	−0.28	0.17	—	—	3.28*	0.99		

Note: Estimates are unstandardized. All significant fixed direct ($p < .05$) and indirect (confidence intervals that do not include 0) effects are bolded. Alternating models are shaded grey for ease of distinguishing them. Initial study (LTS or CAP) and age links with model outcomes are only noted if significant; all significant study and age links were in positive directions (+). Residual effects ranged from 0.08 to 25.04 and all but four were, as expected, larger than random effects. *Mdl*, model; Model 1: *X* → *Y*; Model 2: *X* and *C* → *Y*; Model 3: *X* → *M* → Y; Model 4: *X* and *C* → *M* → *Y*; *M*, emerging adulthood mediator; *C*, covariate; *Y*, established adulthood outcome; *S*, initial study; *A*, age at established adulthood; SE, self-esteem; BD, body dissatisfaction; EL, education level; AS, age at sexual initiation; Mdl_M, mediator model within mediation analysis; Mdl_Y, outcome model within mediation analysis.
*$p < .05$, **$p < .01$, ***$p < .001$.

Table 3. Multilevel model results for female participants: between-family associations between pubertal timing (*X*) and generalized anxiety disorder symptoms in established adulthood (*Y*) via mediators in emerging adulthood (*M*), covarying adolescent internalizing problems (*C*), initial study (*S*), and age (*A*)

			X→M		M→Y		C→Y		X→Y		*M* indirect effect		Random effect			
Mdl	M	N	b	SE	b	SE	b	SE	b	SE	b	[95% CI]	Est	SE	S	A
Past 2 weeks symptoms																
***X* = Age at mid-puberty**																
1	—	464	—	—	—	—	—	—	**−0.63***	**0.29**	—	—	6.11	3.24		
2	—	464	—	—	—	—	**0.88****	**0.29**	−0.56	0.29	—	—	5.47	3.17		
3	SE	347	0.60	0.39	**−0.29*****	**0.07**	—	—	−0.56	0.40	−0.17	[−0.44,	Mdl_M: 8.57**	3.15		
												0.04]	Mdl_Y: 3.97	3.64		
	BD	310	**−0.29****	**0.09**	**1.30*****	**0.33**	—	—	−0.36	0.43	**−0.37**	**[−0.72,**	Mdl_M: 0.46**	0.15		
												−0.11]	Mdl_Y: 6.44	3.79		
	EL	464	0.04	0.03	−0.40	0.71	—	—	**−0.95****	**0.34**	−0.01	[−0.10,	Mdl_M: 0.11***	0.02	+	+
												0.05]	Mdl_Y: 6.73*	3.20		
	AS	434	**−0.08*****	**0.02**	0.17	0.95	—	—	**−0.97****	**0.36**	−0.01	[−0.18,	Mdl_M: 0.05***	0.01		
												0.15]	Mdl_Y: 7.79*	3.49		
4	SE	347	0.41	0.37	**−0.25*****	**0.07**	Mdl_M: **−0.26*****	**0.06**	−0.51	0.39	−0.10	[−0.32,	Mdl_M: 6.50*	2.86		
							Mdl_Y: 0.12	0.06				0.08]	Mdl_Y: 3.84	3.57		
	BD	310	**−0.26****	**0.09**	**1.18*****	**0.34**	Mdl_M: **0.04****	**0.01**	−0.31	0.43	**−0.31**	**[−0.63,**	Mdl_M: 0.43**	0.15		
							Mdl_Y: 0.12	0.07				**−0.08]**	Mdl_Y: 6.01	3.74		
	EL	464	0.03	0.03	−0.01	0.72	Mdl_M: **−0.01****	**0.004**	**−0.85***	**0.34**	−0.0003	[−0.06,	Mdl_M: 0.10***	0.02	+	+
							Mdl_Y: **0.15****	**0.05**				0.06]	Mdl_Y: 6.11	3.14		
	AS	434	**−0.08*****	**0.02**	−0.30	0.95	Mdl_M: **0.01****	**0.003**	**−0.89***	**0.36**	0.02	[−0.12,	Mdl_M: 0.04**	0.01		
							Mdl_Y: **0.19*****	**0.05**				0.18]	Mdl_Y: 6.78*	3.37		
***X* = Age at menarche**																
1	—	428	—	—	—	—	—	—	−0.46	0.30	—	—	7.19*	3.38		
Lifetime symptoms																
***X* = Age at mid-puberty**																
1	—	473	—	—	—	—	—	—	**−0.20***	**0.10**	—	—	0.99*	0.39	+	+
2	—	473	—	—	—	—	**0.29***	**0.10**	0.18	0.10	—	—	0.76	0.39	+	+
3	SE	356	0.59	0.38	**−0.08****	**0.02**	—	—	−0.11	0.14	−0.05	[−0.12,	Mdl_M: 8.32**	3.10		
												0.01]	Mdl_Y: 1.09**	0.38	+	+

(*Continued*)

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Table 3. (*Continued*)

			X→M		M→Y		C→Y		X→Y		*M* indirect effect		Random effect			
Mdl	M	N	b	SE	b	SE	b	SE	b	SE	b	[95% CI]	Est	SE	S	A
	BD	318	**−0.28****	**0.09**	**0.37*****	**0.11**	—	—	−0.05	0.14	**−0.10**	**[−0.21,**	: 0.48**	0.15		
												−0.03]	Mdl_Y: 1.02**	0.37	+	+
	EL	473	0.04	0.03	−0.23	0.25	—	—	**−0.24***	**0.12**	−0.01	[−0.04,	Mdl_M: 0.11***	0.02	+	+
												0.01]	Mdl_Y: 0.96*	0.39	+	+
	AS	442	**−0.08*****	**0.02**	0.28	0.33	—	—	**−0.26***	**0.13**	−0.02	[−0.08,	Mdl_M: 0.05***	0.01		
												0.03]	Mdl_Y: 1.23**	0.42	+	+
4	SE	356	0.41	0.36	**−0.06***	**0.02**	Mdl_M: **−0.27*****	**0.06**	−0.09	0.13	−0.02	[−0.08,	Mdl_M: 5.93*	2.80		
							Mdl_Y: **0.06****	**0.02**				0.02]	Mdl_Y: 1.00**	0.38	+	+
	BD	318	**−0.25****	**0.09**	**0.31****	**0.11**	Mdl_M: **0.04****	**0.01**	−0.03	0.14	**−0.08**	**[−0.17,**	Mdl_M: 0.44**	0.14		
							Mdl_Y: **0.07****	**0.02**				**−0.01]**	Mdl_Y: 0.85*	0.36	+	+
	EL	473	0.03	0.03	−0.09	0.25	Mdl_M: **−0.01****	**0.004**	−0.19	0.12	−0.003	[−0.03,	Mdl_M: 0.11***	0.02		+
							Mdl_Y: **0.06*****	**0.02**				0.02]	Mdl_Y: 0.79*	0.39	+	+
	AS	442	**−0.08*****	**0.02**	0.11	0.33	Mdl_M: **0.01****	**0.003**	−0.22	0.12	−0.01	[−0.06,	Mdl_M: 0.04***	0.01		
							Mdl_Y: **0.07*****	**0.02**				0.04]	Mdl_Y: 0.93*	0.43	+	+
***X* = Age at menarche**																
1	—	435	—	—	—	—	—	—	−0.14	0.10	—	—	1.07**	0.41		+

Note: Estimates are unstandardized. All significant fixed direct ($p < .05$) and indirect (confidence intervals that do not include 0) effects are bolded. Alternating models are shaded grey for ease of distinguishing them. Initial study (LTS or CAP) and age links with model outcomes are only noted if significant; all significant study and age links were in positive directions (+). Residual effects ranged from 0.08 to 33.27 and all but three were, as expected, larger than random effects. *Mdl*, model; Model 1: *X* → *Y*; Model 2: *X* and *C* → *Y*; Model 3: *X* → *M* → *Y*; Model 4: *X* and *C* → *M* → *Y*; *M*, emerging adulthood mediator; *C*, covariate; *Y*, established adulthood outcome; *S*, initial study; *A*, age at established adulthood; SE, self-esteem; BD, body dissatisfaction; EL, education level; AS, age at sexual initiation; Mdl_M, mediator model within mediation analysis; Mdl_Y, outcome model within mediation analysis.
*$p < .05$, **$p < .01$, ***$p < .001$.

which, in turn, was associated with more MDD symptoms. After adjusting for adolescent internalizing problems (Model 4), age at sexual initiation remained a significant mediator. The direct effect ($X \rightarrow Y$) of age at mid-puberty on MDD symptoms remained significant in Models 3 and 4 in which age at sexual initiation was a significant mediator. Self-esteem, body dissatisfaction, and education level were not significant mediators. Covariates (initial study, age) were rarely significant, except for models including education level.

Similar patterns arose for lifetime MDD symptoms. Lifetime MDD symptoms were predicted by age at mid-puberty (Model 1), and this remained significant after controlling for adolescent internalizing problems (Model 2). Age at menarche did not significantly predict lifetime MDD symptoms but showed a similar pattern of results.

The link between age at mid-puberty and lifetime MDD symptoms was significantly mediated by both body dissatisfaction and age at sexual initiation (Model 3): early timing was associated with heightened body dissatisfaction and early sexual initiation, both of which, in turn, were associated with more MDD symptoms. After adjusting for adolescent internalizing problems (Model 4), age at sexual initiation remained a significant mediator, but body dissatisfaction did not. The direct effect of age at mid-puberty on MDD symptoms was no longer significant in Model 3, in which body dissatisfaction was a significant mediator. In contrast, the direct effect ($X \rightarrow Y$) of age at mid-puberty on MDD symptoms remained significant in Models 3 and 4 in which age at sexual initiation was a significant mediator. Neither self-esteem nor education level was significant mediators. Covariates were not significant, except for models including education level.

In sensitivity analyses controlling for both adolescent and childhood internalizing problems, results for MDD symptoms generally showed the same pattern of findings as those controlling for only adolescent internalizing problems (Supplementary Tables 9 and 10; Supplementary Figure 2), despite their smaller sample size (N_{main} = 473 versus $N_{sensitivity}$ = 385).

Generalized anxiety disorder

Both recent (past 2 weeks) and lifetime GAD symptoms were predicted by age at mid-puberty, with younger age associated with more anxiety in established adulthood (Model 1). These associations were no longer significant after controlling for adolescent internalizing problems (Model 2), although the coefficients were similar between Models 1 and 2 for each outcome. Age at menarche showed similar patterns of results for both past 2 weeks and lifetime GAD symptoms but was not significant in Model 1.

The links between age at mid-puberty and both the past 2 weeks and lifetime GAD symptoms were significantly mediated by body dissatisfaction (Model 3): early timing (each one-year decrease of the continuous measure) was associated with heightened body dissatisfaction, which, in turn, was associated with more GAD symptoms. Body dissatisfaction remained a significant mediator after adjusting for adolescent internalizing problems (Model 4). The direct effects ($X \rightarrow Y$) of age at mid-puberty on both the past 2 weeks and lifetime GAD symptoms became nonsignificant in Models 3 and 4. Self-esteem, education level, and age at sexual initiation did not significantly mediate the links between age at mid-puberty and past 2 weeks and lifetime GAD symptoms. Covariates were not significant for past 2 weeks symptoms, except in models including education level; they were significant in all models of lifetime symptoms.

Results for GAD symptoms changed in sensitivity analyses controlling for both adolescent and childhood internalizing problems (Supplementary Table 11). Age at mid-puberty was no longer significantly associated with adult outcomes in Model 1, although the change in probability level for lifetime GAD (i.e. p = .046 increased to p = .058) likely reflects the reduced sample size (N_{main} = 473 versus $N_{sensitivity}$ = 385).

Predicting male participants' internalizing symptoms

None of the symptom outcomes (i.e. past year or lifetime MDD, past 2 weeks or lifetime GAD) were predicted by pubertal timing in Model 1.

Discussion

This study with full longitudinal data reveals persisting links between internalizing symptoms in adulthood and variations in pubertal development in female, but not male, participants; it also provides novel evidence about mechanisms accounting for the sex-dependent associations. Early puberty (i.e. each one-year decrease in a continuous measure) continued to be associated with psychological problems in established adulthood, above and beyond associations seen before puberty and in adolescence, through links with behaviors in emerging adulthood. The results support most of our hypotheses concerning depression symptoms (Figure 2); hypotheses concerning anxiety symptoms were sensitive to childhood behavior problems (Supplementary Figure 2). Specifically, persisting depression symptoms were mediated by body dissatisfaction for lifetime depression and by early sexual initiation for both past year and lifetime depression; past 2 weeks and lifetime anxiety symptoms were mediated by body dissatisfaction, but this association was reduced when childhood internalizing problems were considered.

Our results extend the literature, for the first time establishing these links using full longitudinal data, with psychological symptoms measured in adulthood controlled for problems in childhood and adolescence, puberty measured contemporaneously throughout the pubertal transition, and mediators assessed between puberty and established adulthood. Pubertal timing predicted depression and anxiety in female adults, above and beyond links present in adolescence (and for depression, even when controlling for childhood problems) as well as when controlling for study-related and age covariates. Body dissatisfaction and age at sexual initiation mediated select links between pubertal timing and internalizing symptoms in female adults, again beyond associations in adolescence (and beyond associations in childhood for depression). Finally, patterns of results for recent versus lifetime symptoms were also largely similar, including in the context of adolescent covariates and mediators, highlighting that persisting downstream links between puberty and internalizing symptoms are experienced in the day-to-day lives of established female adults.

Findings about developmental mediators importantly extend previous work using cross-sectional retrospective reports showing that body dysphoria measured at puberty mediated links between early puberty and internalizing problems in female young adults (Thériault et al., 2019) and is consistent with broader hypotheses about links between pubertal development and body image as well as sexual activity. For example, early maturation has been consistently linked to lower body satisfaction in female youth and higher body satisfaction in male youth (Dorn & Beltz, 2023); female young

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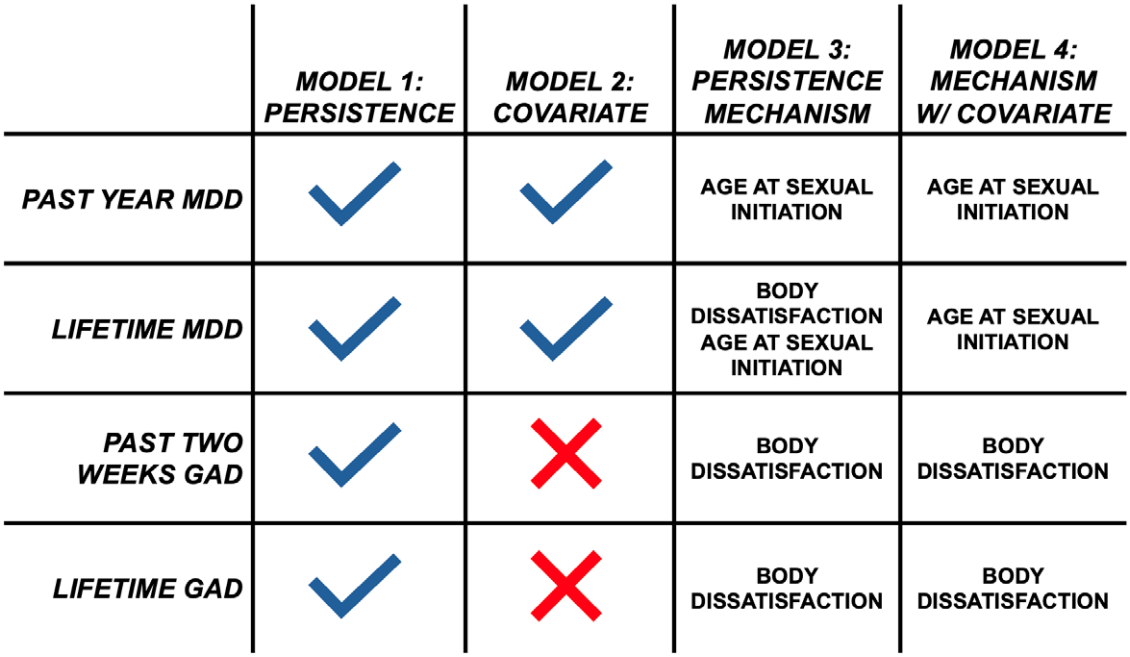

Figure 2. Summary of results for female participants: pubertal timing associations with MDD and GAD recent and lifetime outcomes and mechanisms, and controls for adolescent behavior. *Note*: Checkmarks indicate significant pubertal timing associations with the outcome (leftmost column). *X*s indicate nonsignificant pubertal timing associations. All four models controlled for age and initial study. Covariate models (Models 2 and 4) also controlled for adolescent behavior. Only significant mediators are indicated under the 'Persistence Mechanism' and 'Mechanism w/ Covariate' columns. MDD, 'major depressive disorder'; GAD, 'generalized anxiety disorder'.

adults who retrospectively reported early maturation also reported greater body surveillance, greater sex appeal self-worth, and less body appreciation (Grower, Ward, & Beltz, 2019). These findings broadly align with speculations about the psychological impacts of cultural sexual objectification of the female body (Fredrickson & Roberts, 1997). Early (versus on-time or late) pubertal timing could increase the likelihood of objectification within a peer group and potentially prolong the sensitive period during which female youth engage in negative cognitions about their bodies, ultimately accentuating internalizing problems in adulthood. In comparison, cultural sexual objectification is not as prevalent for the male body, which could explain the present study's null findings for male participants.

Nevertheless, not all investigated links were significant. Persisting associations between pubertal timing and anxiety symptoms were less evident when childhood internalizing problems were included in analyses, consistent with work questioning the developmental origins of adolescent anxiety (e.g. Reardon, Leen-Feldner, & Hayward, 2009) and the significance of the study covariates (e.g. age) on lifetime anxiety symptoms (Table 3). Self-esteem and education level were not significant mediators of any pubertal timing – adult outcome links, possibly because our assessment used an age-related dichotomous indicator of college completion (as a potential socioeconomic status indicator), whereas past research focused on high school completion (as an indicator of externalizing problems; Mendle et al., 2019). There were also limitations of the measures, including assessment ages of the mediators and method variance (i.e. reports of psychological problems were obtained from parents at early ages but participants themselves at later ages). Estimates that were similar in magnitude were significant in some cases but not others because of varying sample size (e.g. pubertal timing links with lifetime anxiety in female participants).

It is important to note that we focused on a range of symptoms measured continuously, not categorical diagnoses, consistent with a dimensional approach to psychopathology (Cuthbert & Insel, 2013). This reflected the community nature of the sample (with low rates of diagnoses) and the likelihood that early puberty acts to increase problems but not necessarily to trigger frank psychopathology, as past work in this sample has shown that childhood problems do not predict pubertal timing, but that both problems and timing have largely unique influences on adolescent problems (Beltz et al., 2020). This also means that our results are not likely to change with any changes in diagnostic criteria (Chmielewski, Clark, Bagby, & Watson, 2015).

This study is novel in several ways. First, these are the only findings using a full longitudinal design – with assessments from four distinct developmental periods – to investigate pubertal timing associations with adult recent and lifetime behavioral outcomes, whether those associations reflect continuity or potential emergence (using covariates of both childhood and adolescent psychological health), and the mechanisms underlying them (using mediators measured between adolescence and established adulthood). It also leveraged sophisticated multilevel mediation analyses necessary for accounting for within-family dependencies when estimating indirect effects via confidence intervals (Rockwood & Hayes, 2017).

Second, we extended work on mediators of pubertal timing links to include male participants. We did not replicate findings of late puberty associations with adult depression in male participants, perhaps because previous work focused on early adulthood versus our focus on established adulthood, and we also had difficulty differentiating on-time from late-maturing male youth because puberty assessments ended at age 15 in the CAP/LTS.

Third, pubertal timing was described with growth curve models based on multiple assessments, leveraging past work in this sample and the Pubertal Development Scale (PDS) without making assumptions about Tanner Stages (Beltz et al., 2014; Shirtcliff, Dahl, & Pollak, 2009). Most previous studies relied on a single measure of

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menarche in female youth, thus excluding male youth. Indeed, it is surprising that persisting links between age at menarche and established adult outcomes were not detected. This might reflect the enhanced sensitivity of the age at mid-puberty measure or systematic missing data linked to menarche, as participants with earlier menarche were less likely to be included in analyses.

Limitations and considerations

The interpretation of findings should be considered in light of some limitations often shared by similar studies. First, puberty was assessed by self-reports on the PDS rather than physical exam (e.g. Tanner staging), and it is unknown if mid-puberty as assessed via the PDS is equivalent to mid-puberty as assessed by other methods. However, there are several strengths of using the PDS, including its reliability and validity (especially with longitudinal assessment (Dorn & Beltz, 2023; Shirtcliff et al., 2009)), as well as its incorporation of menarche status.

Second, the sample is limited in several ways, consisting of siblings in families within a narrow demographic range, so it is important to extend the work to more heterogeneous samples especially in light of racial/ethnic differences in pubertal timing (Deardorff, Hoyt, Carter, & Shirtcliff, 2019; Deardorff et al., 2021; Dorn & Beltz, 2023; Keenan, Culbert, Grimm, Hipwell, & Stepp, 2014). The sample was also likely selected for psychological health both in the foundation sample (given criteria for adoptive parents and longitudinal study demands), and in the participants with full data for this study (excluded participants had more psychological problems than those included); this reduces the prevalence of psychological problems in this sample and underestimates potential links with pubertal variations. Further, missing data resulted in different subsamples for different outcomes and lower power to test mediation than direct effects. It also produced variations in standard errors around similar-sized associations and, therefore, levels of statistical significance. Overall, these sample limitations likely resulted in the underestimation of associations.

Third, we considered only some aspects of the long-term consequences of pubertal timing. We did not include all mediators likely to be important, such as sexual assault (Mendle et al., 2019) and peer interactions (Dorn & Beltz, 2023; Negriff & Susman, 2011); our measure of age at sexual initiation also could have been interpreted in different ways by participants (e.g. not only as intercourse). This reflected the nature of the sample (e.g. few adverse experiences), and our reliance on existing data using a strict criterion that the mediator be assessed between puberty and established adulthood. Due to the importance of having mediators occur temporally between pubertal timing and adult symptoms (O'Laughlin, Martin, & Ferrer, 2018), we were unable to consider adolescent problems as a mediator, though it is likely that pubertal timing impacts adolescent psychosocial health, which could, in turn, influence later outcomes. We also examined a selected set of internalizing outcomes and did not consider other adult psychological outcomes, such as substance abuse or other adolescent externalizing problems also shown to be related to variations in pubertal timing (e.g. risk taking and antisocial behavior; Dorn & Beltz, 2023; Ullsperger & Nikolas, 2017). We only examined persisting associations with pubertal timing because we did not have data on potential mediators of other developmental changes, such as desisting links mediated by parental monitoring (Dorn & Beltz, 2023).

Fourth, we could not identify whether early pubertal timing increased risk, late pubertal timing was protective, or both. Although we took a dimensional approach in assessing continuous pubertal timing links via linear relations with outcomes, this assumes a similar association exists across the range of mid-puberty ages; this is a perpetual issue (Caspi & Moffitt, 1991; Hoyt, Niu, Pachucki, & Chaku, 2020).

Furthermore, we did not correct for multiple comparisons, given the dearth of information about the long-term links between puberty, potential mediators, and adult internalizing symptoms. Although this may have increased Type I error, we opted to minimize Type II error in this novel investigation of multiple mediators and outcomes (Rothman, 1990). Confidence in our findings is increased by consistency of prediction across mediators and both recent and lifetime symptoms; nonetheless, our findings require replication.

Future directions

Our results converge with others to emphasize the long-term associations between pubertal timing and psychological health, while also highlighting important opportunities for future work. These opportunities include exploring how associations extend to other psychological problems, particularly externalizing problems, and other aspects of pubertal development (e.g. variations in tempo). Additionally, future work could examine whether these associations reflect continuity or developmental change in nature or size (e.g. emerging or persisting with potential augmentation or reduction, or desist) or in manifestation (e.g. shifting from adolescent depression to adult substance use). Such work may require unique assessments, including of potential mediators temporally close to puberty and using idiographic approaches (e.g. intensive and/or personalized questions and assessment schedules; Chaku & Beltz, 2022). Few studies will have the full or intensive longitudinal data ideal to address these questions. Fortunately, some aspects of pubertal development can be retrospectively assessed using a simple measure in which adults report their pubertal timing relative to their peers. This measure has been shown to provide a good approximation to timing measured contemporaneously, with similar links to adolescent behavior (Chaku et al., 2024).

Conclusions

Our findings reveal that early puberty's links with experiences of early sexual activity and poor feelings about one's own body matter for mental health, especially for depression symptoms, in female established adults. We uniquely delineate how pubertal timing continues to influence psychological health decades later in sex-differentiated ways, using longitudinal data from four periods of the lifespan to extend other work to show that some adverse links may persist, and a mediating role of body dissatisfaction and age at sexual initiation in maintaining some links. Understanding these mechanisms linking pubertal variations to psychological health in adulthood may ultimately lead to interventions to reduce the risk for psychological health problems.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291725000820.

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Competing interests. The authors declare no competing interests exist.

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