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Journal

Aggressive Behavior, 50(2)

Authors

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Publication Date

2024-03-01

DOI

10.1002/ab.22139

Peer reviewed

HHS Public Access

Author manuscript

Aggress Behav. Author manuscript; available in PMC 2025 March 01.

Published in final edited form as:

Aggress Behav. 2024 March ; 50(2): e22139. doi:10.1002/ab.22139.

Temporal relation between pubertal development and peer victimization in a prospective sample of US adolescents

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Abstract

Peer victimization typically peaks in early adolescence, leading researchers to hypothesize that pubertal timing is a meaningful predictor of peer victimization. However, previous methodological approaches have limited our ability to parse out which puberty cues are associated with peer victimization because gonadal and adrenal puberty, two independent processes, have either been conflated or adrenal puberty timing has been ignored. In addition, previous research has overlooked the possibility of reverse causality—that peer victimization might drive pubertal timing, as it has been shown to do in non-human primates. To fill these gaps, we followed 265 adolescents (47% female) prospectively across three-time points (M_{age}: T1 = 9.6, T2 = 12.0, T3 = 14.4) and measured self-report peer victimization and self- and maternal-report of gonadal and adrenal pubertal development on the Pubertal Development Scale. Multilevel modeling revealed that females who were further along in adrenal puberty at age 9 were more likely to report peer victimization at age 12 (Cohen's $d = 0.25$, $p = .005$). The relation between gonadal puberty status and peer victimization was not significant for either sex. In terms of the reverse direction, the relation between early peer victimization and later pubertal development was not significant in either sex. Overall, our findings suggest that adrenal puberty status, but not gonadal puberty status, predicted peer victimization in females, highlighting the need to separate gonadal and adrenal pubertal processes in future studies.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

Keywords

adrenal puberty; gonadal puberty; peer victimization; pubertal development; puberty

1 | INTRODUCTION

Peer victimization in children and adolescents is a public health concern given that it predicts a range of adverse socioemotional and physical health outcomes in adolescence and adulthood (Moore et al., 2017; Schacter, 2021; Wolke, Copeland, Angold, & Costello, 2013). Defined as experiencing negative physical, verbal, cyber, or indirect aggression (e.g., rumors, rejection) from a peer or peer group (Olweus, 1991), peer victimization occurs in up to 35% of adolescents in the United States (Modecki et al., 2014). Given its high prevalence and long-term negative mental and physical health consequences for children such as suicidal ideation and behaviors (Holt et al., 2015), and psychosomatic symptoms (e.g., headache, abdominal pain, sleeping problems; Gini & Pozzoli, 2009; Zimmer-Gembeck et al., 2019), there has been an international effort to understand its etiology and develop comprehensive anti-bullying programs. Anti-bullying interventions have had some success in reducing peer victimization, but further research is needed to identify additional risk factors so that interventions can be tailored to protect specific groups (Gaffney et al., 2021). One risk factor of interest is pubertal development, given that the peak of peer-to-peer aggression in early adolescence often coincides with pubertal development.

Studies show that exhibiting a different maturational trajectory than peers is a risk factor peer victimization (see Table 1 for a summary of studies on pubertal timing and peer victimization), although very few studies have investigated which specific pubertal cues make children targets. For instance, studies show that adolescents who self-report more advanced pubertal development (compared to less advanced) than their peers report significantly higher rates of peer victimization (Carter et al., 2018; Craig et al., 2001; Reynolds & Juvonen, 2011; Skoog & Kapetanovic, 2022; Su et al., 2018). In addition, several studies have reported that adolescents that report delayed pubertal development also are at higher risk (Haltigan & Vaillancourt, 2018; Jormanainen et al., 2014). While these studies clearly show that atypical pubertal timing is a risk factor, the findings are not entirely consistent and there is a key gap in our knowledge—namely, which specific pubertal cues confer peer victimization risk. Variations in findings could potentially be linked to differences in the methods used to assess puberty. Previous researchers have investigated the influences of overall puberty status (i.e., aggregate score of gonadal and adrenal puberty status), relative-to-peers puberty status, or the timing of gonadal milestones (i.e., age of menarche, first ejaculation), which do not necessarily indicate the presence or progression of visible gonadal cues As it stands, it is unknown whether specific cues of gonadal puberty (e.g., breast development, voice-deepening) or adrenal puberty (e.g., auxiliary hair growth, skin changes) are the most salient predictors of peer victimization.

The importance of these unanswered questions come into sharper relief when one considers that gonadal and adrenal puberty are dissociable processes, marked by unique neurophysiological cues and behaviors that emerge at different ages and develop at varying

rates in boys and girls. Colloquial understandings of puberty best match onto gonadal puberty, which is the cascade of hormonal events that culminates in the maturation of the reproductive system such as widening of the pelvis, breast budding, follicle development, first menstruation, and subcutaneous fat deposits in females, and voice-deepening, growth of facial hair, testicular enlargement, and production of fertile sperm in males (Abreu & Kaiser, 2016). The onset of gonadal puberty is around age 9–10 in females and 10–11 in males, and is completed typically between ages 14 and 16. Given that females usually undergo gonadal puberty initiation approximately 1–2 years before males (Abreu & Kaiser, 2016), the early presence of these cues in females could be especially asynchronous from the puberty progression of their male and female peers, potentially increasing their risk of peer victimization. Similarly, when males experience delayed pubertal progression, their asynchronous development may become conspicuous compared to their male and female peers, also increasing their peer victimization risk. Of no less import, however, are the often overlooked adrenal puberty processes, which are unrelated to reproductive capacity but instead foster brain development that spurs social and cognitive changes in adolescence (e.g., increased social learning capacity; Byrne et al., 2017; Campbell, 2006). Physical markers of adrenal pubertal development include axillary hair growth (i.e., underarm, arms, legs), oil on the skin, body odor, and changes in skeletal structure. It begins between ages 5–7 and plateaus in emerging adulthood (Campbell, 2006). In addition to the neurophysiological and temporal independence of gonadal and adrenal puberty, adrenal puberty could have a theoretically distinct relationship with peer victimization than gonadal puberty given that adrenal puberty is hypothesized to prompt the seeking out of novel social experiences and more frequent engagement with new people (via rising DHEA-S; Campbell, 2006). Given that peer victimization may increase with more frequent interaction with new people, it makes sense to examine the associations between adrenal puberty development and peer victimization separately from gonadal puberty development. Yet no previously published study, to our knowledge, has examined these unique associations.

Moreover, very few studies have used longitudinal approaches allowing the consideration of reverse causality—the possibility that peer victimization could proceed and predict the timing of pubertal development. Indeed, studies in pro-social mammals support the idea that peer victimization can influence pubertal timing. For example, in non-human primates, adolescent females who experience more aggression from peers in the juvenile period and those with low social rank have delayed puberty (Wilson et al., 2013). Rodents experimentally treated with stress hormones (i.e., ACTH) experienced delayed sexual maturation compared to those treated with saline (Alves et al., 1993). These results are often explained using a broad life history framework proposing that altering pubertal timing represents an adaptive response intended to help the organism attain reproductive competence at a time that optimizes reproductive success, be it delayed or accelerated reproductive maturation (see Ellis, 2004 for a review of theoretical frameworks). For instance, it may be an advantageous strategy to delay sexual maturation in hostile environments until conditions improve and there is a greater likelihood of reproductive success. Although this relation between peer victimization and pubertal timing has not been examined directly in humans, it is noteworthy that the quality of social environment and psychosocial stressors have been linked with altered pubertal timing in girls (see Pham et

al. (2022) for a review of family environmental antecedents). These findings underscore the importance of exploring the social context, but also highlight the growing call for research dedicated to the broader social context, particularly the role of peers, in the relation.

Thus, the purpose of the present study was to examine the potentially bidirectional relation between peer victimization and gonadal and adrenal puberty in humans. Using three waves of data from a sample of U.S. adolescents, our study tested if gonadal and adrenal puberty status in early adolescence predicted subsequent experiences of peer victimization, and, vice versa, if peer victimization in early adolescence predicted subsequent gonadal and adrenal puberty development. We hypothesized that adolescents with higher puberty scores would be more likely to experience peer victimization but did not have specific predictions related to gonadal or adrenal puberty timing. Hence, we also conducted exploratory analysis to identify whether specific pubertal cues (e.g., breast development, height, acne) that predicted peer victimization. Conversely, we examined for the first time in humans if peer victimization in early adolescence predicted delays in subsequent gonadal or adrenal puberty status in males and females. We predicted peer victimization would delay gonadal puberty in females, because evidence from animal studies generally supports disruptions to gonadal puberty in females.

2 | METHODS AND MATERIALS

2.1 | Participants

Participants were 271 adolescents (47% female) followed across the pubertal transition using a lag-longitudinal design. These participants were a part of a larger cohort recruited during routine first trimester prenatal care from two obstetric clinics in southern California from 1999 to 2003. Child medical records were used to determine sex at birth. Detailed recruitment procedures of the larger lag-longitudinal study have been described elsewhere (e.g., Glynn et al., 2018), and all procedures were approved by the appropriate institutional review boards for the recruiting site and study center. Mothers provided written and informed consent and informed assent was obtained from adolescents.

Participants were included in the present study if they had completed at least on assessment in adolescence. Participants were excluded from the present study if they had significant developmental or medical disabilities that interfered with their ability to complete the survey measures (e.g., severe autism, brain injury; $n = 3$) or were taking medications known to impact pubertal development (e.g., puberty hormone blockers; $n = 1$). Moreover, participants who were diagnosed with precocious puberty by a physician ($n = 2$) were excluded.

The final analysis for this study included 265 adolescents (47% female). Adolescents were on average 9.6 ($SD = 0.7$) years of age at Time 1 ($n = 133$), 12.0 ($SD = 0.9$) years at Time 2 ($n = 187$), and 14.4 ($SD = 1.3$) years at Time 3 ($n = 190$). 196 participants completed at least two waves of data collection. Mothers reported child's race/ethnicity at the first visit. 41% reported being non-Hispanic-White, 29% Latino/a, 20% Multiethnic, 10% as Other, 6.4% Asian, and 3.8% Black. Table 2 presents all adolescent sociodemographic and study variables at each wave.

2.2 | Measures and procedures

At each of the three study visits, participants and their mothers were invited into the lab to complete questionnaires assessing pubertal development and adolescent peer victimization. These were completed separately by the youths and mothers.

2.2.1 | Peer victimization—Participants completed the 4-item peer victimization subscale developed for the Bullying Prevention Initiative (Williams & Guerra, 2007) at each study visit. Each item measured the frequency of physical, direct verbal (e.g., were teased, told mean things), indirect verbal (e.g., rumors were spread), and cyber victimization since the start of the school year. Response options and numeric coding ranged from (1) never, (2) once or twice, (3) several times, or (4) a lot. Although each of these items can be used separately to identify victims of physical, verbal, and cyber victimization, they can be averaged to create a reliable overall victimization measure. Thus, the items were averaged at each time-point to create a peer victimization score for that visit that ranged from 1 to 4, with higher scores indicating more peer victimization. This scale was chosen to model changes in the frequency of peer victimization over time.

2.2.2 | Pubertal development—Adolescents and mothers completed the 5-item Pubertal Development Scale which measures both gonadal and adrenal puberty (Petersen et al., 1988). The PDS assesses gonadal puberty with questions about growth in height, voice deepening and facial hair for boys, and growth in height, breast development, and the occurrence of menarche for females. Adrenal puberty is assessed through questions about changes in skin and body hair. Response options included (1) not yet started changing, (2) has barely started, (3) changes are underway, and (4) development seems completed such as reaching maximum height. Gonadal and adrenal puberty scores were converted to a 5-point scale that parallels physical exam Tanner stages using syntax developed by Shirtcliff and colleagues (2009). Maternal-report PDS scores were used for children younger than 12 years of age because maternal-report has been found to provide greater certainty than self-report for youths of this age (Lum et al., 2015; Terry et al., 2016), though concordance between maternal- and self-report pubertal development were high. Moreover, adolescent self-report of onset of menarche were utilized as prior research supports less recall bias compared to maternal-report of adolescent's timing of menarche (Coleman & Coleman, 2002; Koo & Rohan, 1997).

2.2.3 | Covariates—A set of potential covariates were chosen because they have been identified in previous research as predictors of peer victimization (Tippett & Wolke, 2014; van Geel et al., 2014) or pubertal development (Deardorff et al., 2014; Huang & Roth, 2021; Pham et al., 2022; Ramnitz & Lodish, 2013). The following potential covariates were examined: Body mass index (BMI), father absence, ethnicity, and income-to-needs ratio. BMI was assessed in-lab at each visit by measuring youth's height/weight then calculated using the Child and Teen's Body Mass Index (weight kg/height m^2). Cohabitation status was assessed using maternal report of father presence or absence from the home at each visit. This variable was coded as 0 for father absent and 1 for living with father. Income to needs ratio was calculated for each child's family at each visit by maternal report of household income and household size relative to the cost of living for the reported income

year. Potential covariates were included in subsequent models if they were associated with both peer victimization and pubertal development.

2.3 | Data analytic plan

A four-step data analytic strategy was implemented. First, Pearson's correlations were used to explore simple associations between peer victimization and pubertal status at each time point. Second, we used growth curve modeling to establish the trajectories of peer victimization and pubertal development over time, testing linear and unrestricted models. Third, to identify potential third variables (i.e., confounding variables), multilevel modeling was used to determine sociodemographic factors that met our third variable criteria (i.e., associated with both peer victimization and pubertal development at $p < .05$) and were entered into subsequent models. Finally, we tested the potential of reverse causality-specifically, that early pubertal development (e.g., adrenal, gonadal, and the occurrence of menarche or not) predicts peer victimization or changes in victimization across adolescence. Then, we tested the reverse, that early peer victimization predicts puberty or alterations in pubertal development, adjusting for any potential third variables. The onset of puberty differs for boys and girls (Abreu & Kaiser, 2016), and initial models revealed that sex moderated trajectories in pubertal timing, so all analyses were run separately by child sex.

Multilevel modeling was used because initial models indicated that the intraclass correlations (ICC), or the shared variance in measures within-people across time, were high (see Section 3). Moreover, multilevel modeling has advantages over other methods (e.g., repeated measures analysis of variance [ANOVA]) for longitudinal data analyses because it accounts for shared-variance across observations and accommodates missing data (Shek & Ma, 2011). Linear and unstructured models were tested to model changes in pubertal development and victimization over time (because we only had three time points for each measure, we could not test for quadratic or cubic trajectories, although unstructured models, which treats time as a repeated measure category, is highly flexible and allows for the modeling of differential rates of change between time point). Linear models showed the best fit for all outcomes. Predictors were entered at level 2 of the model, while time-varying outcomes were modeled at level one. Because covariates were collected at each time point and changed over time, these factors were entered at level 1 of the models. See Supporting Information Materials for further details on model specification, including the rationale for choosing multilevel modeling over random-intercept cross-lagged models.

All analyses with continuous outcomes were conducted in SPSS v24.0 using the MIXED command and employed the Restricted Maximum Likelihood estimator (Shek & Ma, 2011). All analyses with dichotomous variables (i.e., onset of menarche or not at each time point) was conducted in STATA v17.0 with the xtgee command. An unstructured covariance structure (SPSS syntax UN) and REPEATED command for time were used to help account for shared variance in outcome measures across time. Predictors and BMI were z-scored before analyses to ease model interpretation. Unadjusted model estimates are presented in text. In Figure 1, high and low scores were defined as values one standard deviation above or below the specified time-period mean, respectively.

Results with a $p < .05$ and 95% confidence interval (CI) that did not overlap with 0 were interpreted as statistically significant. Models were centered at the mean age for each of the three visits (T1 = 9.6, T2 = 11.9, T3 = 14.4) to examine intercept differences as a function of earlier peer victimization and/or pubertal development on latter peer victimization and or/pubertal development.

3 | RESULTS

3.1 | Preliminary analysis

Table 2 presents descriptions of adolescent sociodemographic, pubertal development, and peer victimization scores at each assessment. Pearson's correlations were conducted to assess the degree of relatedness between peer victimization and pubertal development at each time point (see Table 3). Adrenal puberty status at T1 in females was correlated with peer victimization at T2 ($r = .387$, $p = .004$). Pubertal development and peer victimization were not significantly correlated at any other time point.

Before any predictors were included in multilevel models, we ran basic models to calculate the ICC see Table S1 for ICCs). Models revealed that 42% of the variance in peer victimization and 30%–41% of the variance in pubertal development (e.g., adrenarche = 41%, gonadarche = 30%) was due to shared variance within the same person over time (see S1). Thus, multilevel modeling is recommended when ICCs are high because it has advantages over other methods (e.g., repeated measures ANOVA) for longitudinal data analyses because it accounts for shared-variance across observations and accommodates missing data (Shek & Ma, 2011). Linear multilevel growth modeling allowed us to describe the growth trajectories in peer victimization and pubertal development. Peer victimization scores declined slightly over time in males and females, though non-significantly. As expected, all pubertal development measures significantly increased over time in males and females (e.g., gonadal and adrenal PDS scores).

Overall, growth modeling showed that males and females reported similar rates of victimization at each time point. Both sexes also had a similar non-significant decrease in victimization over time (see Table S2 for intercepts and slopes). Furthermore, females reported higher average gonadal and adrenal pubertal development scores at each time point compared to males. Growth curve modeling revealed that females had steeper increases (i.e., faster development) in gonadal and adrenal PDS scores than males over time.

3.1.1 | Covariate analyses—Sociodemographic characteristics of the sample, along with their associations with peer victimization and pubertal development are presented in Table S3. The only variable that met our initial third variable criteria was father absence. Father absence in adolescence predicted higher peer victimization in males. Furthermore, father absence predicted higher gonadal and adrenal puberty scores in females, and a trend for higher gonadal puberty status in males ($p = .058$). However, there was a strong theoretical rationale to include BMI as a covariate because of previous research linking BMI to both peer victimization and pubertal development in western societies (Waasdorp et al., 2018). Moreover, higher BMI predicted higher gonadal and adrenal PDS scores for males

and females, and BMI predicted earlier age of menarche. Thus, all subsequent models were adjusted for father absence and BMI.

3.2 | Primary analysis

3.2.1 | Early pubertal development predicting peer victimization—Early adrenal puberty prospectively predicted later peer victimization in females (see Figure 1). Specifically, higher adrenal PDS scores at age 9 predicted an increase in mean peer victimization at age 12 (Coeff. = 0.15, *SE* = 0.05, 95% CI = [0.05, 0.26], *p* = .005, Cohen's *d* = 0.25). Adrenal PDS scores at T1 also predicted a trend for more peer victimization at T1 (Coeff. = 0.15, *SE* = 0.08, 95% CI= [−0.02, 0.31], *p* = .090) and T3 (Coeff. = 0.16, *SE* = 0.09, 95% CI = [−0.01, 0.34], *p* = .070) in females only. The same pattern of results was observed when the covariates BMI and father absence were included in the model.

Adrenal PDS scores at T2 were not associated with peer victimization at any time point, and adrenal PDS scores at T1 and T2 did not predict changes in peer victimization over time. Furthermore, gonadal PDS scores were not associated with peer victimization at any time point and did not predict changes in peer victimization over time in males and females. Further, the occurrence of menarche was not associated with peer victimization or changes in peer victimization at any time point in females. These relations were not changed by the inclusion of covariates in the models.

3.2.2 | Early peer victimization predicting pubertal development—Overall, early peer victimization did not prospectively predict gonadal and adrenal pubertal trajectories in males and females. Peer victimization was not associated with gonadal PDS scores at any time point and did not predict changes in gonadal puberty trajectories in males and females. Peer victimization was not associated with the occurrence of menarche at any time-point in females. Peer victimization was not associated with adrenal PDS score at any time point nor did it predict adrenal puberty trajectories in males and females. These relations were not changed by the inclusion of covariates in the models.

3.3 | Testing specific indicators of pubertal development

Post hoc analyses were conducted to explore whether specific pubertal cues at age 9 were driving the association with peer victimization at age 12 (Figure 2). Overall, unadjusted models revealed that changes in skin (Coeff. = 0.12, *SE* = 0.05, 95% CI = [0.01, 0.23], *p* = .040) and body hair (Coeff. = 0.12, *SE* = 0.06, 95% CI = [0.01, 0.23], *p* = .037) at age 9 predicted peer victimization at age 12 in females only, with effect sizes of *d* = 0.19 and *d* = 0.19, respectively. There was a trend in which adolescent females that were further along in breast development were more likely to experience peer victimization (Coeff. = 0.10, *SE* = 0.06, 95% CI = [−0.01, 0.21], *p* = .087) as well a significant cross-sectional association in which advanced breast development at age 9 predicted peer victimization at age 9 (Coeff. = 0.18, *SE* = 0.08, 95% CI = [0.02, 0.34], *p* = .028). No other pubertal cues were significantly related to peer victimization in our sample.

4 | DISCUSSION

This is the first study, to our knowledge, to separately test the effects of gonadal and adrenal puberty status on peer victimization. We found that higher adrenal puberty status in early adolescence, but not gonadal pubertal status, predicted peer victimization in females. Specifically, females farther along in adrenal puberty status at 9 years old were more likely to report experiencing higher levels of peer victimization at age 12, a finding that remained significant after statistically adjusting for BMI, father absence. It is important to note that, if our study had used an overall PDS score that combines cues of both gonadal and adrenal puberty, as several previous studies have done (Carter et al., 2018; Craig et al., 2001), we would have found a significant and positive association between early overall pubertal status and peer victimization in females (Coeff. = 0.17, $SE = 0.06$, $p = .006$) but not males. However, using the composite PDS score makes it difficult to tell whether gonadal or adrenal puberty status is driving the relation, highlighting the significance of examining these cues individually. By separating the unique contribution of gonadal and adrenal pubertal development, we could establish that only adrenal pubertal cues, not gonadal puberty status, predicted victimization in females (but not males).

As detailed in Table 1, two out of three previously published studies have found a positive association between gonadal pubertal measures and peer victimization, a relation we did not detect (Skoog & Kapetanovic, 2022; Su et al., 2018; but see Jormanainen et al., 2014 for results that vary by gender). Discrepancies with previous findings may be explained by differences in puberty measures. Previous studies that have examined gonadal puberty have found an association with the puberty measures age of menarche and first ejaculation or first voice-deepening, by categorizing adolescents age of onset as “early,” “on-time,” and “late.” Until the current study, no study had utilized the gonadal subscale of the PDS, which combines breast development and the occurrence of menarche in girls, and facial hair and the occurrence of voice-deepening in males, in addition to growth spurt for both females and males, to create a composite gonadal pubertal progression score. Considering our findings within the context of the literature, we conducted an exploratory post hoc analysis to examine what specific items of the PDS at age 9 that were driving peer victimization at age 12 in our sample. There was a trend in which adolescent females that were further along in breast development were more likely to experience peer victimization, as well a significant cross-sectional relation in which advanced breast development at age 9 predicted peer victimization, but a lack of overall effect of gonadal puberty, suggests that early gonadal puberty cues that are more visible like breast development may be more detectable to peers than other gonadal cues (e.g., first menstruation) and thus more likely to predict peer victimization. However, it is crucial to approach these results with caution considering the trend did not reach conventional statistical significance and our limited statistical power from analysis conducted separately by sex. Still, we speculate that females may have been targeted for early breast development because of noticeable changes in sexual appearance that stood out against the less-mature physical forms of their peer group (Skoog et al., 2016). Because girls’ gonadal puberty changes typically occur 1–2 years before boys (Abreu & Kaiser, 2016), early breast development would be an asynchronous cue from both girls and boys, enticing negative responses from an expanding pool of potential perpetrators. Potential

explanations for the association between breast development and peer victimization, but null association with other aspects of gonadal puberty, should be tested in future research.

We suspect one reason that early gonadal pubertal status was not related to peer victimization in our sample of males is that our study was restricted to early- to mid-adolescence. One study suggests late-maturing boys are more likely to be targeted than early maturing boys based on age of first ejaculation recorded at age 15 (Jormanainen et al., 2014). Thus, it is possible that our study did not detect a significant relation in males because the adolescents in our study ranged in age from 9 to 14, before the age at which lack of gonadal pubertal development in boys would be highly unusual. Moreover, our study includes items about facial hair and voice-deepening, but not age of first ejaculation, so differences in gonadal puberty measures could also explain discrepancies. It seems reasonable that late-maturing boys would be more likely to be targeted than early developing boys because they would have the greatest pubertal asynchronicity from their peers given boys' gonadal pubertal onset is 1–2 years after girls. More research is needed to test if gonadal puberty status in late adolescence is a more relevant period to boys' peer victimization risk than puberty status in early adolescence, as well as ascertain if certain gonadal puberty cues are more likely to predict peer victimization.

Several possible proximal mechanisms could explain why females with higher adrenal puberty status were more likely to experience peer victimization. Our post hoc analysis revealed that adolescent girls were specifically targeted for changes in skin and body hair, though it is important to exercise caution considering these analyses were exploratory and we did not make statistical adjustment for multiple comparisonsNonetheless, first, it is plausible that adolescents were targeted for skin and hair changes specifically because of the high visibility of these cues. Skin and body hair changes may also drive peer victimization because they accompany increased body odor, which has been identified by researchers as the content of bullying in several qualitative studies (Jette, 2012; Ramsey, 2010). Secondly, it is equally plausible that puberty-related body odor changes may act as a chemosignal (a form of social communication in mammalian species) to peers that could increase aggressive behaviors (Pause, 2012). In line with the idea that chemosignals may subtly influence social behavior in humans, one study found that mothers could smell when their child was in pre- or late-puberty, and that puberty status corresponded with salivary testosterone levels (Schäfer et al., 2020), which is one androgen expressed alongside DHEA in sweat glands (Mostafa et al., 2012). Future studies should systematically examine if changes in body odor, or chemosignals, are perceptible by peers and an underlying factor in peer victimization. Higher adrenal puberty status could be associated with peer victimization because the changes are conspicuous to peers but could also be related to peer victimization through less obvious changes in social behavior.

Yet another pathway through which adrenal puberty may drive may influence peer victimization is through adrenal-puberty related social and cognitive changes (Campbell, 2006; Del Giudice, 2018; Kotler & Haig, 2018). Specifically, it has been argued that rising DHEA-S levels, such as those occurring in adolescence, have effects on the brain to reduce fearfulness, increase the retention and processing of social experiences, and spur greater plasticity in the brain connections related to social cognition (Campbell, 2006). In line with

this view, animals that are acutely treated with DHEA-S tend to have an impairment of the fear response to conditioned stimuli (Fleshner et al., 1997) and enhanced memory (Wolf & Kirschbaum, 1999). It is plausible, then, that adrenal puberty corresponds with behaviors that encourage more frequent interaction with unfamiliar peers increase peer victimization risk. However, no studies, to our knowledge, have sought to explicitly examine the extent to which adrenal pubertal development is linked to social changes (e.g., reduced fearfulness, enhanced memory), and if these, social changes in turn increase victimization. There is a body of literature that implicates the degree of social competence, or the ability to start conversations with others, develop and preserve friendships easily, and solve problems in social contexts (Asher, 1983), is related to peer victimization (Carter et al., 2018; Craig et al., 2001; Troop-Gordon, 2017). It is unclear if adrenal-linked social behavioral skills correspond with low or high social competence measures as it is untested. Still, we believe there is conceptual overlap between social competence and the social changes proposed to be associated with a biochemical adrenal puberty that warrants future research to test how features of each are related. Overall, these novel findings underscore the importance of separating gonadal and adrenal puberty cues because only adrenal puberty status predicted peer victimization in females in our study.

Unlike studies in non-human primates, we found no evidence of reverse causality—early peer victimization did not predict subsequent gonadal or adrenal puberty timing in males or females in our study. We believe this possibility deserves additional human research since our study utilized an existing dataset that was not designed to test this question specifically. Future research interested in this topic should measure peer victimization earlier in adolescence, before the onset of both adrenal and gonadal puberty, given that they are likely sensitive periods that precede and predict the timing of HPG axis development. Our first peer victimization measure occurred around age 9 and may have been too late in pubertal development to detect a relation. Moreover, future studies should include objective measures of peer victimization because our self-report measure may have precluded forms of bullying that were unrecognized or normalized such as passive-aggressive methods. Naturalistic observations of social interactions have been used in animal studies that find a relation between peer harassment and pubertal timing, and we believe it is possible that the use of a similar thematic coding of social interactions between adolescents could be successful in more accurately capturing a relation in humans. Given physical aggression is a frequent form of bullying in animal studies (De Almeida et al., 2015), we ran an additional post hoc analysis to test if the physical victimization (a subscale in the peer victimization measure) in early adolescence, predicted subsequent gonadal or adrenal puberty timing in our sample. The physical victimization subscale was not related to gonadal or adrenal puberty, but physical aggression may have been too uncommon in our sample as it was reported by only 35.5% of adolescents. Therefore, studies may need to focus on younger adolescent samples that are at higher risk for physical victimization to document associations.

Although our study had several strengths, including our use of a well-characterized longitudinal dataset and our distinction between gonadal and adrenal puberty cues, the results of this study should be considered alongside several limitations. First, our study relied on maternal- and adolescent self-reports of physical markers of pubertal development.

Although the PDS scale has been validated and corresponds with objective puberty status (Shirtcliff et al., 2009), future studies should consider an objective measure of puberty status, such as physician examination, to ensure accurate measurement of adrenal and gonadal puberty cues. Secondly, peer victimization was relatively uncommon in this study when we looked at the physical, verbal direct, verbal indirect, and cyber peer victimization subscales individually. Most participants reported never experiencing peer victimization or only “once or twice” for each subscale, and no adolescent reported frequent verbal indirect victimization (see Table S4). Thus, we created a composite peer victimization measure to increase variability and could not reliably look at the unique associations between pubertal status and different types of peer victimization. Additionally, our measure of peer victimization did not explicitly inquire about experiences of peer sexual victimization or forms of social rejection, such as a peer commenting on appearance in a sexual way or excluding them from social activities, respectively. Because our measure of peer victimization cast a wide net, it is likely that we captured aspects of sexual and social peer victimization, but were unable to make any inferences about the potential relation with adrenal puberty without direct measurement. Future research that measures specific forms of peer victimization, in addition to separating gonadal and adrenal puberty cues, could extend this work.

There are several important unanswered questions for future research. One consideration is to examine the role of the adolescent’s social context. While this study exclusively focuses on biological changes, adolescence is also a time of dramatic social transitions such as increases in group size, group composition of opposite-sex peers, autonomy, and reductions in adult supervision (Connolly et al., 1999). Studies have drawn attention to the importance of the social context and how it relates to peer victimization, such as the buffering effect of having more friends (Furman & Rose, 2015; or decreased risk with more adult supervision (Blosnich & Bossarte, 2011). Despite research highlighting the importance of biological and social changes separately, a paucity of work aims to integrate these aspects of adolescence into a comprehensive model of peer victimization risk. Thus, we recommend that future research considers features of the social transition as potential predictors, compounding factors, or moderators of the relation between adrenal puberty and peer victimization to better predict peer victimization. One challenging factor in creating an integrative approach is the lack of research on the temporal relation between puberty and social changes and, thus, would a first step would be to untangle the temporal relation. For example, one possibility would be to longitudinally examine how increases in DHEA-S correspond with the timing and tempo of adrenal puberty physical cues and timing of social transitions. An additional consideration for future research would be to capture the perpetrator’s identity, social status, and motivations to try to answer questions about the evolutionary functions of bullying adrenally advanced peers in particular. Bullying has been hypothesized to be an adaptive tool used by conspecifics to shape developing social hierarchies and enhance access to critical group resources such as mates, protection, food, and access to alloparents in competitive environments (Volk et al., 2012). However, it is untested if there are benefits to bullying adrenally advanced peers and if targeting these adolescents serves one of the proposed functions of bullying (i.e., social dominance, resources, reproduction). If bullies benefit from selecting these adolescents specifically, studies might expect a positive

correlation between bullying behavior and proxies of success, such as social status (e.g., peer-reported dominance). Assessment of the perpetrator could refine the larger theoretical framework and research regarding the etiology of peer victimization. Last, we included concurrent father absence as a covariate in our study based on existing empirical evidence (Ellis & Garber, 2000; Rowe, 2000) and statistical support, although most research in the life-history framework centers on father absence before the puberty. As such, we urge caution in interpretation of our findings within the life-history framework and highlight the need for further research on concurrent predictors of puberty timing.

Our work has several potential implications for bullying prevention programs and public health. First, many widely used anti-bullying interventions do not include puberty as a risk factor for peer victimization in their online training modules for children, youth, parents, administrators, teachers, and schools (Finland Ministry of Education and Culture; NYC Service; U.S. Department of Health & Human Services). It is possible that peer victimization might be reduced by educating children and adolescents about the general risk of early pubertal development, the differences in gonadal and adrenal puberty, and the natural variations that occur between people to prepare them for when noticeable differences emerge between peers. Teachers and school personnel should also be aware that pubertal development is a risk factor and that early puberty cues may confer victimization risk. It has been well-established that negative or stressful events, like peer bullying, during puberty increase vulnerability to negative developmental outcomes such as depression (Troop-Gordon, 2017). Normalizing puberty differences could not only reduce peer victimization but also alleviate the psychological distress of puberty-related peer victimization and expectantly improve long-term mental and physical health outcomes. Given that adolescent girls with skin changes (e.g., acne, oil) were more likely to experience peer victimization in our study, and that acne affects roughly 85% of adolescents in Western civilizations (Bhate & Williams, 2013), anti-bullying programs may benefit from a direct focus on skin problems in adolescents. National rates of adolescent acne and skin problems have been increasing over recent decades (Lynn et al., 2016). It would be interesting to test if rising levels of skin problems in childhood and adolescence coincide with an overall increase in the prevalence of peer victimization over time. In sum, the current study contributes to previous work by underscoring the importance of separating gonadal and adrenal puberty cues because adrenal puberty status, but not gonadal puberty status predicted peer victimization which may have implications for bullying prevention programs and public health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

The authors would like to thank the study participants and dedicated research staff that collected the data used in the current study. We also wish to thank Kendra Leak for her invaluable assistance. This study was supported by grants from the National Institute of Health: HD-40967 (LMG), P50 MH096889 (EDP, LMG, CAS), R01 HD51852 (CAS), R01 NS041298 (CAS).

Funding information

National Institutes of Health

DATA AVAILABILITY STATEMENT

Supplementary Online Materials (SOM), including analysis scripts, are available on the Open Science Framework (OSF). Data not available due to restrictions.

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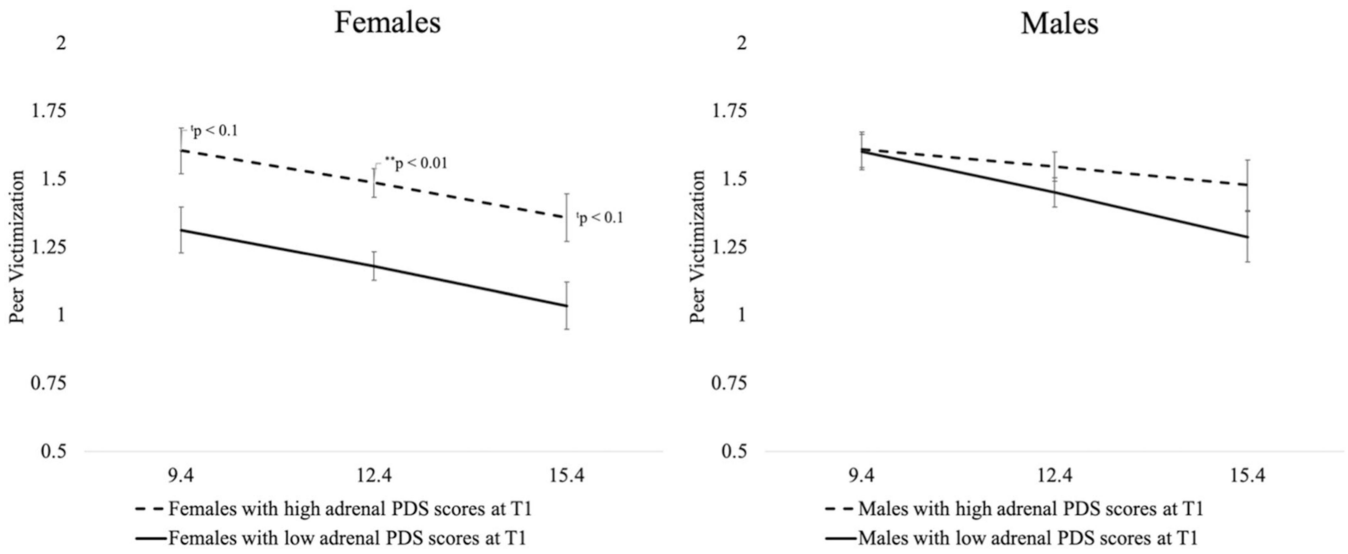

FIGURE 1.

Early adrenal puberty prospectively predicts higher rates of peer victimization in girls, not boys. Unadjusted models. Bars display the *SE*. Low adrenal PDS = −1 *SD* for that time-period. High adrenal PDS = +1 *SD* for that time-period.

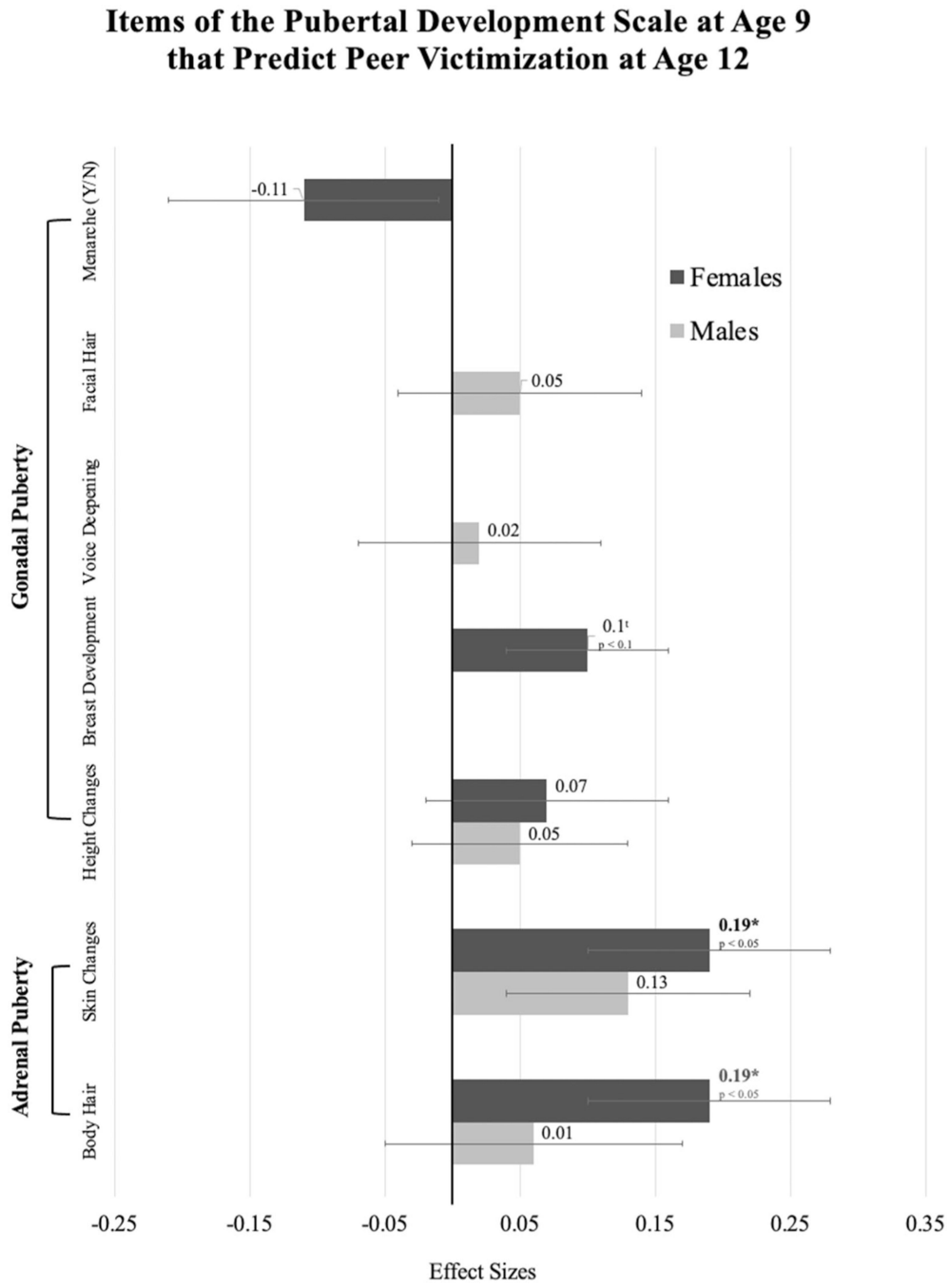

FIGURE 2.

Unadjusted exploratory post hoc analysis: Comparison of PDS items at age 9 that predicted peer victimization at age 12 and effect sizes. Bars display the effect size and *SE*.

TABLE 1

Literature review of studies that assess how pubertal development relates to peer victimization[a].

Author and year	Study design	Sample size	Puberty measure	Timing of puberty measure	Peer victimization measure	Timing of peer victimzation measure	Results in girls	Results in boys
Carter et al. (2018)	Longitudinal	655 girls; 709 boys	Total PDS	Grades 4, 5, and 6	Peer exclusion	Grades 4, 5, and 6	Earlier pubertal timing directly predicted high levels of peer exclusion cross-sectionally and longitudinally	Null
Craig et al. (2001)	Cross-Sectional	447 girls; 552 boys	Total PDS	M_{age}: 12.7 years	Peer victimization: Physical, verbal, and social	M_{age}: 12.7 years	Early puberty predicted social victimization	Early puberty predicted social victimization
Reynolds and Juvonen (2011)	Longitudinal	912 girls	Total PDS	Grades 6 (Wave 1)	Peer rumors	Grade 6 (Wave 2)	Advanced pubertal development predicted increased risk of rumors	–
Haynie & Piquero (2006)	Longitudinal	3543 girls; 3404 boys	Relative puberty status[b]	Ages 11–16 (Wave 1)	Peer physical victimization	Ages 12–17 (Wave 2)	Puberty predicted physical victimization	Puberty predicted physical victimization
Haltigan and Vaillancourt (2018)	Longitudinal	368 girls; 332 boys	Parent-report relative puberty status[b]	Grade 5, 6, 7, 8, 9, 10	Overall peer victimization: Physical, verbal, social, and cyber	Grade 5, 6, 7, 8, 9, 10	Early and later puberty development predicted higher incidence of peer victimization dependent on time-point	Later puberty development predicted higher incidence of peer victimization across time
Jormanainen et al. (2014)	Longitudinal	1167 girls; 903 boys	Girls: Age of menarche; Boys: Age of first ejaculation	Age 15 (Wave 1)	Peer victimization & social exclusion	Age 17 (Wave 1 and 2)	Null	Late pubertal development cross-sectionally associated with social exclusion. No longitudinal association
Skoog and Kapetanovic (2022)	Longitudinal	773 girls; 742 boys	Girls: Age of menarche; Boys: Age of first ejaculation OR Age of first voice-deepening	Age 13	Peer victimization: Sexual, verbal, physical harassment, or social exclusion	Ages 13, 14, 15	Early puberty timing was associated with higher levels of peer victimization cross-sectionally	Early puberty timing was associated with higher levels of peer victimization cross-sectionally
Su et al. (2018)	Cross-Sectional	227,443 girls	Age of menarche	M_{age}: 13.6 years	Peer victimization	M_{age}: 13.6 years	Early menarche predicted occasional and frequent peer victimization	–

[a] Includes studies with an outcome variable of peer victimization such as overall peer victimzation score or verbal, physical or cyber victimization score. Excludes studies with an outcome variable of peer sexual harassment or violent offenses (e.g., rape, robbery, assault).

[b] Relative puberty item compares perceived development to same-age peers: "How advanced is your physical development compared to other [girls/boys] your age?"

TABLE 2

Sample characteristics.

	Time 1		Time 2		Time 3	
	Males	Females	Males	Females	Males	Females
Variable	***M* (*SD*)/%**	***M* (*SD*)/%**	***M* (*SD*)/%**	***M* (*SD*)/%**	***M* (*SD*)/%**	***M* (*SD*)/%**
Age, years	9.5 (0.7)	9.7 (0.7)	12.3 (0.9)	12.3 (0.9)	15.1 (0.8)	15.2 (0.8)
Body mass index	18.5 (3.9)	18.5 (3.5)	20.5 (4.5)	21.0 (4.9)	21.9 (4.5)	23.1 (5.3)
BMI percentile	60.8 (29.8)	61.0 (28.5)	64.5 (30.1)	65.1 (28.8)	63.9 (28.6)	67.8 (27.6)
Income to needs ratio	509.3 (614.3)	612.3 (621.4)	579.9 (676.3)	617.5 (671.1)	580.9 (479.1)	665.01 (719.1)
Adrenal puberty	1.2 (0.5)	1.6 (0.9)	2.0 (1.0)	3.1 (1.3)	3.0 (1.1)	4.2 (0.9)
Gonadal puberty	1.4 (0.6)	2.0 (1.0)	2.2 (1.3)	3.2 (1.3)	3.5 (1.2)	4.5 (0.9)
Peer victimization	1.6 (0.6)	1.5 (0.6)	1.5 (0.6)	1.5 (0.7)	1.5 (0.6)	1.4 (0.5)
Father absent	28%	8%	34%	25%	37%	28%
Age of Menarche[a]	NA	9.9 (0.8)/3%	NA	11.8 (0.9)/31%	NA	12.3 (1.5)/50%

Note: $N = 265$. 47.2% female.

[a]Average age of menarche is reported at each visit only for adolescent females that reported first menstruation. Sample size slightly differed between variables and visits due to missing data.

TABLE 3

Correlation of study variables for males (top right) and females (bottom left).

Variable	1. T1 Peer victimization	2. T2 Peer victimization	3. T3 Peer victimization	4. T1 Adrenal PDS score	5. T2 Adrenal PDS score	6. T3 Adrenal PDS score	7. T1 Gonadal PDS score	8. T2 Gonadal PDS score	9. T3 Gonadal PDS score
1. T1 Peer victimization	-	**0.27** *	**0.17**	**−0.05**	**−0.03**	**0.17**	**0.08**	**0.02**	**0.18**
2. T2 Peer victimization	*0.32* **	-	**0.53** **	**0.15**	**0.09**	**0.04**	**0.01**	**0.01**	**−0.06**
3. T3 Peer victimization	*0.17*	*0.65* **	-	**0.06**	**0.05**	**0.17**	**0.05**	**0.16**	**0.09**
4. T1 Adrenal PDS score	*0.13*	*0.39* **	*0.22*	-	**0.25** *	**0.27**	**0.25** *	**0.22**	**0.12**
5. T2 Adrenal PDS score	*0.08*	*0.20*	*0.17*	*0.52* **	-	**0.61** **	**−0.02**	**0.67** **	**0.48** **
6. T3 Adrenal PDS score	*0.01*	*0.13* ^{t}	*0.09*	*0.32* *	*0.49* **	-	**0.33** *	**0.43** **	**0.62** **
7. T1 Gonadal PDS score	*0.12*	*0.11*	*0.09*	*0.52* **	*0.55* **	*0.34* *	-	**0.11**	**0.11**
8. T2 Gonadal PDS score	*0.01*	*0.14*	*−0.06*	*0.38* **	*0.68* **	*0.32* **	*0.55* **	-	0.36 **
9. T3 Gonadal PDS score	*−0.09*	*0.19*	*0.20* ^{t}	*0.22*	*0.44* **	*0.39* **	*0.33* *	*0.54* **	-
10. Age of Menarche (n = 96)	*0.04*	*0.14*	*0.21* ^{t}	*−0.30* ^{t}	*−0.11*	*−0.01*	*−0.49* **	*−0.30* *	*0.04*

Note: **Bold** = results for males, *italic* = results for females.

^{t} $p < .1$

* $p < .05$

** $p < .01$

*** $p < .001$.