Accelerated pubertal development as a mechanism linking trauma exposure with depression and anxiety in adolescence

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Abstract

Exposure to early-life adversity (ELA) is associated with elevated risk for depression and anxiety disorders in adolescence. Identifying mechanisms through which ELA contributes to the emergence of depression and anxiety is necessary to design preventive interventions. One potential mechanism linking exposure to ELA with psychopathology is accelerated pubertal development. Exposure to trauma—specifically interpersonal violence—is associated with earlier pubertal timing, which in turn predicts adolescent-onset depression and anxiety disorders. We review the recent literature on adversity and accelerated pubertal development, exploring specific associations between trauma and accelerated pubertal development as a mechanism linking adversity with depression and anxiety disorders in adolescence. Finally, we suggest future directions for research exploring mechanisms linking ELA with accelerated pubertal development as well as pubertal timing and psychopathology in adolescence.

1. Introduction

Exposure to early-life adversity (ELA) is associated with elevated risk for numerous forms of psychopathology across the lifespan, including depression and anxiety disorders. Approximately one-third of adolescent depression onsets and one-sixth of anxiety disorder onsets are explained by exposure to ELA [1]. Identifying mechanisms through which ELA contributes to the emergence of depression and anxiety is imperative to designing preventive interventions.

One potential mechanism linking exposure to ELA with psychopathology is accelerated biological aging. Specifically, exposure to ELA may alter the pace of development, resulting in faster aging of physiological systems. One salient metric of biological aging early in development is pubertal timing, or the timing of the onset of pubertal development. Early pubertal timing is consistently associated with elevated risk for mood and anxiety disorders [2]. In this paper, we review evidence for the role of individual differences in pubertal

Conflict of Interest

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timing as a mechanism underlying vulnerability to depression and anxiety following ELA. Specifically, we highlight recent research suggesting that exposure to trauma—specifically interpersonal violence—is associated with earlier pubertal timing, which in turn predicts adolescent-onset depression and anxiety disorders. We discuss potential mechanisms that may underlie these associations and potential future directions for this research.

2. Early Life Adversity and Accelerated Pubertal Development

The idea that early environmental experiences impact the pace of biological aging is central in life history theory [3]. These models suggest that the timing of life history events—such as age of sexual maturity, number of offspring, length of parental investment, etc.—is determined by trade-offs in the prioritization of time and energy invested in growth relative to reproduction and survival [4]. Early environmental experiences are thought to modulate the pace of development in order to maximize the chances of survival and reproduction later in life. Environments characterized by harshness or threat (e.g., trauma or exposure to violence) may accelerate the onset of puberty in order to maximize the opportunity for reproduction prior to mortality. However, in environments characterized by material and social deprivation, the onset of puberty may be delayed in order to ensure adequate resources are available to support reproduction [3].

A commonly used metric of biological aging in youths is the timing and pace of pubertal development, including age of menarche in females and pubertal stage controlling for chronological age in males and females. Exposure to ELA is associated with earlier pubertal timing in numerous studies [5]–[9], although others have reported no such associations [10], [11] or even delayed pubertal timing following ELA [7], [12], [13]. We have argued that discrepancies across studies may be due to the treatment of ELA as a homogenous construct [14].

2.1. Specificity to Trauma/Threat Exposure

One potential explanation for variability in the association of ELA with early pubertal timing is that distinct types of ELA influence the pace of development differently. Dimensional models of ELA argue that the wide range of experiences classified as ELA can be organized into underlying dimensions of environmental experience that have unique influences on development [3], [15]. These models identify core dimensions of environmental experience that occur in numerous forms of adversity [15]. Dimensions proposed in existing models including *threat/harshness* (which encompasses experiences involving trauma/threat of harm to the physical integrity of the child, such as abuse and exposure to violence), *deprivation* (which involves an absence of expected inputs from the environment during development, such as cognitive and social stimulation and responsive caregiving), and *unpredictability* (which involves temporal variation in caregiving). Dimensional models argue that these aspects of the early environment influence emotional, cognitive, and neural development through some shared pathways as well as others that are distinct and vary as a function of the type of adversity experienced [15]. It is important to note that a central issue in much work exploring the impact of specific dimensions of early life adversity on pubertal timing is a failure to assess and adjust for co-

occurring forms of ELA. Such an approach is critical when evaluating potential specificity in associations with pubertal timing, because experiences of ELA are highly co-occurring [1][16].

We have argued that threatening early environments (i.e., environments characterized by trauma/violence/threat of harm) may be particularly likely to lead to accelerated pubertal development, as they signal that the environment is dangerous and that morbidity and mortality risk is high [14]. In contrast, environments characterized by deprivation may lead to delayed pubertal development, as they signal that environmental resources may not be adequate to support reproduction. Indeed, across two independent samples children exposed to trauma exhibited earlier pubertal timing, whereas children who experienced deprivation did not, controlling for co-occurring ELA [13], [17]. In a meta-analysis spanning 43 studies and over 100,000 participants, ELA experiences characterized by threat (i.e., trauma/ violence exposure/threat of harm) were associated with earlier pubertal timing, but no association with pubertal timing was observed for poverty or experiences characterized by deprivation (i.e., neglect or institutional rearing) [14]. These findings support our hypotheses regarding threatening early environments and accelerated development and highlight the importance of considering the nature of the early environmental experiences and controlling for co-occurring dimensions of adversity, when examining the influence of adversity on the pace of development.

These findings highlight one potential pathway through which trauma contributes to risk for adolescent depression and anxiety. Early pubertal timing is associated with elevations in depression and anxiety during adolescence [2], [18]–[20]. Our work suggests that early pubertal timing may be one mechanism that accounts for the powerful association between trauma and adolescent psychopathology, demonstrating that earlier pubertal timing helps to explain the link between trauma and later depression and anxiety [13], [17].

3. Mechanisms Linking ELA and Accelerated Pubertal Development

The mechanisms through which threatening early environments influence pubertal timing remain unknown. One plausible mechanism involves alterations in physiological stress response systems, such as the hypothalamic-pituitary-adrenal (HPA) axis, that in turn influence biological systems responsible for sexual development, including the hypothalamic-pituitary-gonadal (HPG) axis. Most work examining associations between HPA and HPG axis function in early development has been done in animal models. These studies suggest that higher levels of corticosterone suppress the release of sex steroids and can even halt ovulation (i.e. Kamel & Kubajak, 1987). The few studies examining these associations in humans are consistent with animal models. For instance, Shi et al. (2011) found that lower daily cortisol output in childhood (pre-puberty) was associated with earlier pubertal timing in females but not males. Similarly, lower daily cortisol output predicted earlier pubertal development in females only (Negriff, Saxbe, et al. 2015; Saxbe et al., 2015).

There is mixed evidence for how ELA influences HPA-axis responsivity. The strongest support exists for the association between early-life trauma and blunted cortisol response

both diurnally and in response to stressors [24]. Given associations between trauma and blunted cortisol reactivity and diurnal patterns [25], [26], it is plausible that trauma-related alterations of the HPA-axis may influence regulation of the HPG-axis in ways that accelerate pubertal development (Belsky, Ruttle, Boyce, Armstrong, & Essex, 2015; Negriff, Saxbe, et al., 2015; Saxbe et al., 2015). More specifically, blunted HPA-axis function following early-life trauma could lead to an earlier influx of adrenal and gonadal hormones responsible for pubertal onset. Future research should directly examine whether altered HPA-axis function is a mechanism linking ELA with HPG-axis function and pubertal development.

4. Potential Mechanisms Linking Accelerated Pubertal Development to Psychopathology

Extensive evidence suggests that earlier pubertal timing is associated with elevated risk for psychopathology in adolescence, including depression and anxiety disorders [2], [28]. Numerous mechanisms are likely involved in this association (for reviews see Ge & Natsuaki, 2009; Graber, 2013). Early conceptual models focused on differences in the pace of maturation among physical, emotional, and cognitive systems as underlying the association between early pubertal timing and psychopathology [31]. For example, earlier pubertal timing may lead to changes in physical appearance and body size and shape that are not in line with societal ideals, leading to psychological distress and risk for psychopathology [32]. More recently, these ideas have been applied to theories of brain development—suggesting that earlier puberty leads to changes in neural networks involved in emotion processing before the maturation of prefrontal control networks that increases risk for adolescent psychopathology [33], [34].

Despite substantial interest in how puberty shapes neurodevelopment, evidence for the impact of pubertal *timing* specifically on brain development is sparse. Findings from the Imaging brain development in Childhood to Adolescence Transition Study (iCATS; Simmons et al., 2014) reveal wide ranging effects of early adrenarche on brain function and structure. For instance, early adrenarche was associated with decreased frontal white matter [36], changes in neural responses to emotional faces in the salience network and ventromedial prefrontal cortex [37], and altered fronto-amygdala connectivity [38], with divergent patterns for males and females. These patterns of neural function, along with increased volume of the pituitary gland, mediated the association between early adrenarche and increases in anxiety symptoms [38], [39]. Altogether, these findings suggest that alterations in pubertal timing influence brain structure and function, particularly fronto-limbic circuitry. Work in this area remains due to the difficulty of disentangling the influence of alterations in pubertal timing from normative changes associated with pubertal development and chronological age. More research is needed to determine how other facets of pubertal timing, such as changes in body morphology or sex steroids associated with gonadarche, influence neural function and structure in ways that contributes to risk for adolescent psychopathology.

5. Limitations and Future Directions

Despite substantial evidence linking ELA and early pubertal timing with risk for psychopathology in adolescence, much work is needed to identify mechanisms underlying these associations. For example, symptoms of psychopathology may contribute to accelerated biological aging following ELA [40]–[42]. Understanding whether interventions, designed to treat psychopathology in children and adolescents exposed to adversity, lead to changes in pubertal timing will help to clarify the direction of these associations.

Understanding of the impact of pubertal timing on neurodevelopment remains limited, due in part to gaps in knowledge of normative patterns of brain development. For instance, reliable patterns of neurodevelopmental maturation in networks frequently examined in relation to puberty, such as amygdala-PFC connectivity, have not been established (although see Bloom et al., 2021). Establishing these normative patterns is essential in order to understand how pubertal timing influences these trajectories. Alternatively, more global metrics such as "BrainAGE" may do a better job of distinguishing departure from typical developmental trajectories (Figure 1). These methods will allow investigation into whether pubertal development accelerates the pace of brain development globally or only in particular brain networks (e.g., those involved in emotional processing or that have higher concentrations of sex steroid receptors). With the help of large longitudinal samples such as the Adolescent, Brain, Cognition and Development Study (Jernigan, Brown, & Dowling, 2018) and the Human Connectome Project-Development [45], we will be better able to distinguish how pubertal timing influences neurodevelopmental trajectories in ways that contribute to risk for adolescent psychopathology.

Additionally, it is important to consider the possibility of sex differences in the associations among ELA, pubertal timing, and different domains of psychopathology. A full review of the literature on sex differences in the impact of pubertal timing is beyond the score of this review; however, it is important to note that a recent meta-analysis did not find support for sex differences in the association between pubertal timing and psychopathology generally [2].However, that analysis did not examine whether the association of pubertal timing with different types of psychopathology, such as internalizing and externalizing problems, varied by sex. Given significant sexual dimorphisms in the development of biological systems beginning in early development and continuing throughout sexual maturation and adulthood [46] as well as sex differences in rates of depression and anxiety that emerge in adolescence [47], it is likely that there may be sex differences in the association between pubertal timing and depression and anxiety in adolescence.

As with any body of research, it is important to consider the methodological limitations inherent in the subject under study. For instance, there are well documented recall biases associated with retrospective reporting of childhood experiences in adulthood [48]–[50]. Indeed, a recent meta-analysis comparing retrospective and prospective methods for measuring ELA exposure demonstrates very little overlap in the groups identified by each of these methods, suggesting that prospective and retrospective assessments identify fundamentally different groups of people [50]. Thus, it is important to account for the way that ELA experiences are measured when interpreting study results. In contrast, self-report

of age of menarche, even retrospectively, shows relatively high reliability [51], [52] and does not suffer from retrospective recall to the same degree [53], [54]. However, different metrics of pubertal timing have differing degrees of reliability. For best practices in measuring pubertal development see [55]–[57].

Finally, it is important to determine whether associations between ELA and pubertal timing extend to additional metrics of biological aging, or whether this association is specific to puberty. We recently found consistency in the association among threat-related adversity and early pubertal timing, cellular aging, and structural metrics of cortical development [14]. Recent evidence suggests that ELA is associated with changes in brain aging [42], [58] and the timing of the emergence of permanent molars in early childhood [59]. More work is needed to integrate across measures of biological aging, in order to fully understand the mechanisms underlying associations among ELA, biological aging and risk for adolescent psychopathology.

6. Conclusions

Accelerated pubertal development is a potential mechanism linking exposure to threat-related adversity with the onset of depression and anxiety disorders in adolescents. Mechanisms underlying these associations are still under investigation, including associations between stress systems and reproductive hormones, and the impact of alterations in pubertal timing on brain development in childhood and adolescents. These findings and future research directions present novel potential to inform early interventions for children and adolescents who have experienced ELA.

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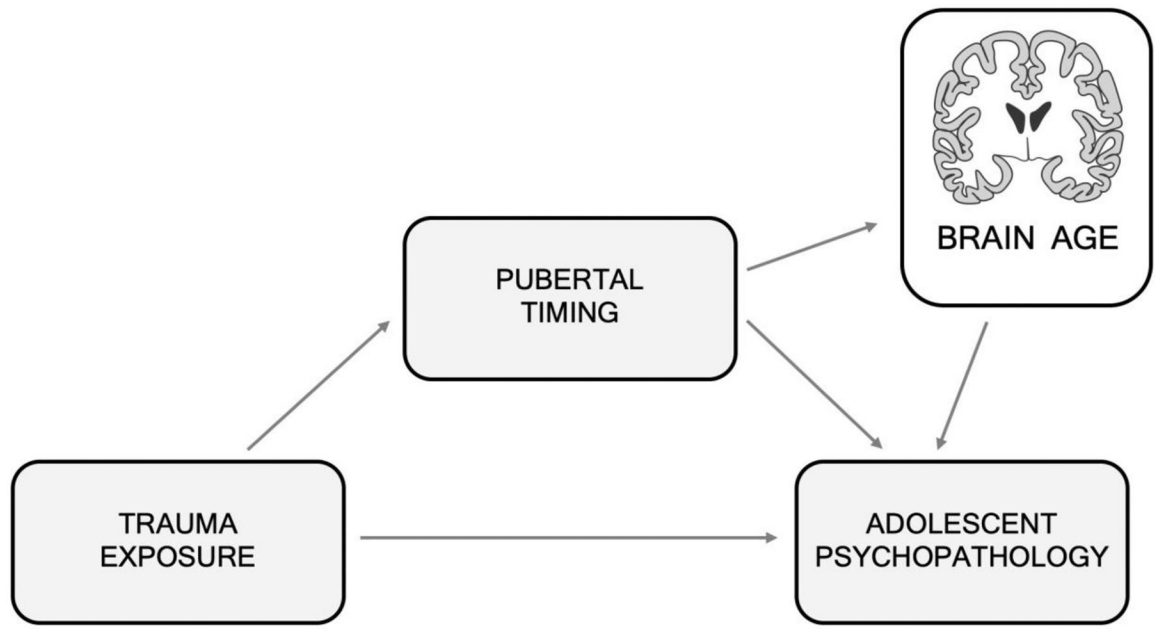
Figure 1.

A visual representation of our theoretical model linking childhood trauma exposure and adolescent psychopathology through accelerated pubertal development.