Exploring profiles of hormone exposure: Associations with cognition in a population-based
cohort of early adolescents

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Highlights

1. Latent profile analysis (LPA) will be used to empirically derive constellations of hormone exposure characterized by testosterone, DHEA, and estradiol in a population sample of adolescents.

Abstract

During adolescence, increases in pubertal hormones lead to reproductive maturity and drive changes in cognitive development. However, little is known about how to best characterize interindividual differences in hormone concentrations. The goal of the proposed study is to examine the antecedents and consequences of membership in empirically derived hormone subgroups. Data will be drawn from over 11,000 youth (approximately 50% male, ages = 9 - 11 years) from the Adolescent Brain Cognitive Development study. Latent profile analysis stratified by sex will be used to derive profiles of hormone exposure based on basal testosterone, dehydroepiandrosterone (DHEA), and estradiol (girls only). Profile membership will then be related to pubertal status, race/ethnicity, socioeconomic status, and body mass index (BMI). Additionally, profile membership will be examined as a prospective predictor of adolescents' cognition (i.e., inhibitory control, visuospatial processing, decision-making) assessed two years later. Findings will clarify disparate associations between hormones and behavior in adolescence and underscore the importance for youth-centered approaches that focus on patterns of individual characteristics.

Key words: Adolescent Brain Cognitive Development Study, Decision-making, DHEA, Estradiol, Inhibition, Latent Profile Analysis, Testosterone, Visuospatial Processing

Pubertal development is the biological hallmark of adolescence, leading to reproductive maturation and changes in cognitive processing (Peper & Dahl, 2013; Romeo, 2003). Increases in hormone levels lead to the growth and development of secondary sex characteristics (Biro et al., 2012), but individual differences in pubertal onset suggest that hormones can have time-sensitive, graded responses on the brain, creating meaningful individual variation in cognitive ability and behaviors. (Vijayakumar et al., 2018). Despite wide acknowledgement of these individual differences (Deardorff et al., 2019; Herting et al., 2020; Mendle et al., 2019), there is a notable lack of research utilizing person or youth-centered approaches to understand the heterogeneous nature of hormone exposure. Thus, the goal of the current study is to examine the relationship between constellations of hormone exposure and cognition in a population-level sample of early adolescents drawn from the Adolescent Brain Cognitive Development (ABCD) Study.

Hormones and Cognition During Adolescence

Puberty initiates a cascade of hormonal, neural, and behavioral changes (Blakemore & Choudhury, 2006), beginning with the activation and development of the adrenal system (adrenarche) and the hypothalamic-pituitary-gonadal (HPG) axis (gonadarche; Archibald et al., 2003). Adrenarche (typically the earliest sign of puberty) begins with the release of androgens, such as dehydroepiandrosterone (DHEA), the secretion of which continues to increase into early adulthood (Witchel & Topaloglu, 2019). DHEA and other androgens are responsible for the development of secondary sex characteristics such as pubic hair growth, body odor, and acne (Auchus & Rainey, 2004). Gonadarche begins when the production of gonadotropin-releasing hormone (GnRH) increases at pubertal onset (Abreu & Kaiser, 2016). The release of GnRH – secreted first in early infancy - reactivates the HPG axis, ultimately triggering a rise in

testosterone and estradiol in sex-specific ways with males experiencing 20 - 30-fold increases in testosterone over 2 - 5 years and females experiencing 4 - 9-fold increases in estradiol (Peper & Dahl, 2013).

Girls tend to begin puberty one to two years before boys (Farello et al., 2019), but even within sex, there are large interindividual differences in pubertal onset (Graber et al., 2010; Mendle et al., 2010) that have long-term implications for behavior (for review, see Ullsperger & Nikolas, 2017). Although some of these differences can be captured by clinician or self-reported pubertal status, concerns remain. First, while hormone levels and pubertal stage are correlated, hormone concentrations independently predict cognitive and socioemotional processing over and above pubertal status or age (Forbes & Dahl, 2010; Shirtcliff et al., 2009). Second, some youth may exhibit higher hormone levels, but lower physical maturation (or vice-versa; Herting et al., 2020) due to variation in tissue sensitivity (Greenstein, 1978), genetic factors (Parent et al., 2016), or environmental context (Greenspan & Lee, 2018). Finally, even short-lasting or cyclical hormone cycles (that cannot be captured by self-report or clinic measures) may have lasting downstream effects on cognition and behavior during adolescence (Romeo, 2003). Thus, hormone levels may have unique influences on the brain and behavior independent of (*or obscured by*) pubertal status.

Increases in testosterone, estradiol, and DHEA during adolescence drive structural and functional brain growth and reorganization, especially in regions associated with memory, motivation, decision making, and sensation seeking (Sisk & Zehr, 2005). For example, higher testosterone levels predict better inhibitory control and spatial abilities (Ordaz et al., 2018; Shields et al., 2019). Davison and Susman (2001) found that higher testosterone was associated with better spatial abilities in boys both before and during puberty. In girls, however, higher

testosterone was only related to better spatial abilities *during* puberty, suggesting that activational effects of testosterone may vary by both sex and developmental stage. Higher levels of endogenous testosterone have been linked with worse decision making and more impulsivity in adolescent samples as well (Peper & Dahl, 2013; Poon et al., 2019).

Although less studied, estradiol and DHEA are also associated with cognitive processes. For example, higher estradiol is linked to better performance on sustained attention (Stevens et al., 2005), working memory (Dumas et al., 2013), and inhibitory control tasks (Amin et al., 2006), but worse performance on spatial ability tasks (Davison & Susman, 2001). Chung et al. (2019) further found that higher estradiol was related to better selective attention and working memory during an emotion processing task *independent of puberty-specific effects*. Similarly, higher DHEA has been linked to better cognitive flexibility and appraisal as well as less risky decision-making, particularly when dealing with social stressors (Crum et al., 2017; Goldenberg, 2017).

Together, these studies highlight the importance of considering hormone effects on cognitive processes, but thus far, studies have been limited by small sample sizes and cross sectional designs (Uban et al., 2018); varying age ranges that do not account for developmental differences that may exist even among same-aged adolescents (e.g., it may not be appropriate to compare same-aged boys and girls given well known differences in pubertal onset; Parent et al., 2003); and a focus on single hormones. Dual axis perspectives, for example, highlight that pubertal hormones should be considered in concert as multiple, interacting processes (e.g., Marceau et al., 2015). Although functional “cross-talk” between testosterone, DHEA, and estradiol has been considered in animal models (see Shirtcliff et al., 2015), little human research has taken this approach, potentially ignoring important hormonal consequences for behavior.

Capturing Individual Differences in Hormone Exposure through Youth-Centered Approaches

Previous studies have relied on variable-centered approaches that assume the same-aged adolescents exhibit similar patterns of basal hormone concentrations. However, theoretical perspectives suggest that adolescence is a period of substantive heterogeneity - that is, a single underlying distribution may not equally characterize all youth (Connell & Frye, 2006; Crone & Dahl, 2012). Not only do same-aged adolescents demonstrate quantitatively different basal hormone concentrations (Herting et al., 2020; Shirtcliff et al., 2009), not all adolescents demonstrate similar associations between hormone concentrations and cognition. For example, higher testosterone is often linked to impulsive behavior (e.g., Peper & Dahl, 2013), but this association does not hold for all adolescents (Herlitz et al., 2013). This may be because of a potential interactive effect of another hormone (e.g., DHEA might modulate testosterone; Goddings et al., 2012), but could also be due to unique environmental influences obscured by standard variable-centered approaches.

Adolescence marks the onset of novel psychosocial stressors that reshape the activation of the HPG axis (Marceau et al., 2012). Stressors experienced during this period can increase the arousal or reactivity of testosterone, estradiol, and DHEA leading to individual differences in basal hormone levels regardless of pubertal status (Granger et al., 2003; Marceau et al., 2015). Changes in levels of hormone exposure in response to environmental stressors can, in turn, have long term consequences for cognition and behaviors, resulting in interindividual differences in motivation and reward sensitivity. For example, Black youth tend to exhibit the highest levels of DHEA, testosterone, and estradiol compared to other race/ethnic groups (Herting et al., 2020). This could be due to a number of co-occurring factors such as race/ethnic discrimination and the

emerging salience of race/ethnic identity, that in turn, may increase stress responses and influence pubertal processes (Carter et al., 2017; Deardorff et al., 2019; Quintana, 2007; Rivas-Drake et al., 2014). Lower socioeconomic status (SES; measured by parent education and financial difficulties) has also been associated with higher testosterone in boys and higher estradiol in girls (Assari et al., 2020). This too may represent a proxy for stressful occurrences such as living in neighborhoods with scarce resources or high levels of environmental toxins (Stumper et al., 2020; Worthman et al., 2019). Thus, variable-centered approaches overlook the possibility that certain subgroups of youth, defined by unique biological and contextual experiences, may demonstrate qualitatively distinct patterns of hormone concentrations that explain disparate associations with cognitive abilities.

Person-centered approaches have the potential to uncover these subgroups and link subgroup membership to different contextual factors and life experiences. Person-centered research on hormone concentrations is limited, but several recent studies have reported qualitatively distinct patterns of salivary cortisol that uniquely predict behavioral adjustment in youth and young adults over and above traditional variable-centered approaches. For example, Hoyt and colleagues (2021) found specific combinations of cortisol parameters related to differences in slope, area under the curve (AUC), and cortisol awakening response (CAR) predicted mental health trajectories. Similarly, Bendezu and colleagues (2018) found that distinct cortisol subgroups predicted differences in coping styles and in another study, found that membership in different cortisol-DHEA profiles was uniquely associated with later behavioral problems (Bendezu et al., 2021).

As seen in the cortisol literature, approaches which include multiple *pubertal* hormones could provide insight into cross-hormonal patterns that index maladaptive or resilient

functioning. Although the current literature has mostly considered functional cross-talk between hormones *across axes* (e.g., cortisol and testosterone; see Dekkers et al., 2019 for review), a few studies have also considered links between testosterone and DHEA, finding that DHEA may modulate levels of testosterone, potentially optimizing cognitive functions related to attention or working memory (Nguyen et al., 2017; Nguyen et al., 2013). These studies have primarily studied multiple hormones via interaction effects which are helpful for elucidating the effect of one hormone on an outcome at different values of another. However, these approaches fail to capture how constellations of hormones can jointly influence behavioral processes in non-additive ways (Brand et al., 2005). This is important because increases in testosterone, DHEA, and estradiol reflect different processes both within and across sex. For example, in boys, testosterone reflects gonadal processes and DHEA reflects adrenal processes (Witchel & Topaloglu, 2019). In girls, however, estradiol reflects gonadal processes and testosterone and DHEA reflect adrenal processes (Yu et al., 2020). Limited research suggests that adrenal and gonadal processes may predict different outcomes, but they are difficult to disentangle in traditional, variable-centered approaches, adding to growing literature that multimodal pubertal measures are needed to elucidate the effects of hormone concentrations on cognition and behavior.

The Proposed Study

The Adolescent Brain Cognitive Development (ABCD) study presents a unique opportunity to examine patterns of hormone exposure in a population-level sample of early adolescents. The goal of the current study is to derive hormone profiles in boys (consisting of testosterone and DHEA) and girls (consisting of testosterone, DHEA, and estradiol) using latent profile analysis (LPA), a person-centered technique appropriate for the analysis of

interindividual differences. We will then test demographic predictors of profile membership and additionally, examine how hormone profiles prospectively predict differences in cognition assessed two years later. Specific questions and hypotheses are elaborated on in Table 1. As LPA is a data-driven approach, the hypotheses for the second and third research questions are contingent on the final profile solution. If those profiles do not emerge from the data, all following analyses will be considered exploratory and use a more stringent threshold for evidence (described in Table 1). The results of this study will inform ongoing scientific research regarding the organization of hormone concentrations in early adolescents. Further, conducting this study as a registered report with shared analytic code applied to an openly available, population-level dataset will further increase the replicability and reproducibility of this work.

Methods

Data for these analyses will be drawn from the upcoming ABCD 4.0 Data Release (estimated: Fall 2021). The ABCD Study is an ongoing, longitudinal study of over 11,000 youth, enrolled at ages 9-11 from 21 study sites across the U.S. Detailed information on study design, sampling, recruitment, and retention efforts for the ABCD Study have been previously outlined (see Ewing et al., 2018; Garavan et al., 2018) and additional information on study protocols can be found at abcdstudy.org. Individual study sites provided Institutional Review Board approval. All parents and youth provided consent and assent respectively.

Participants

The initial sample will include all youth (approximately 50% male) with at least one hormone sample (of testosterone, DHEA, or estradiol) at baseline. Hormone samples will be evaluated for quality (described below) and focal analyses will be conducted with those who have high quality hormone data.

Measures

Hormone Measures.

Testosterone, DHEA, and estradiol (girls only) were collected via salivary measures at baseline. Detailed information about biomarkers are available at (Uban et al., 2018). Briefly, participants were instructed not to eat, drink, or chew gum for 30 minutes prior to the collection of the saliva sample. Then, saliva was collected via passive drool from each participant under the instruction of a trained research assistant and immediately placed on ice after collection. Collection time of the saliva samples were not standardized across ABCD sites but instead varied between 7:00 AM to 7:00 PM. Saliva samples then were shipped to Salimetrics (Salimetrics, 2011) and assayed in duplicate to avoid multiple freeze-thaw cycles.

Single hormone values will be derived following best practices outlined by (Herting et al., 2020). Youth will be excluded from the sample if they did not provide a saliva sample at baseline or if their salivary sex does not match the master sex reported in ABCD. Replicate values will be averaged to create a single hormone value. If one of the replicates is higher or lower than the detectable limits of the assay, that replicate will be discarded and the single replicate will be used as the final value. If both replicates are outside the upper or lower bounds of the assay, both replicates will be discarded, and that participant will be excluded from the analysis. Before analysis, all hormone data will be standardized and thus, comparable with scores close to zero representing “average” hormone concentrations, scores above zero representing relatively higher hormone concentrations, and scores below zero representing relatively lower hormone concentrations. This procedure is common in latent profile analyses (Jung & Wickrama, 2008).

Cognitive Tasks.

Inhibitory control will be measured using the Toolbox Flanker Task, a variation of the Eriksen Flanker task (Eriksen & Eriksen, 1974). The task assesses the degree to which participants' responses are influenced by surrounding stimuli when trying to identify the target during congruent and incongruent trials. In each trial, four stimuli (two arrows on the outer left and two arrows on the outer right) either face the same way (congruent trial) or to the left or right (incongruent trial) of the middle arrow. Adolescents have to push the arrow that corresponds to the middle arrow. The ABCD study provides a composite measure of the Flanker that incorporates both speed and accuracy. Uncorrected scores will be used for analysis.

Visuospatial processing will be measured using the Little Man Test (LMT; Acker, 1987). In the LMT, youth are presented with a male figure holding a briefcase. The figure is presented in several different orientations (e.g., right side up, upside down, facing towards or away from the participant). Youth must press an arrow key that corresponds to the hand the figure is holding the briefcase with. Participants complete 32 trials of the task. The ABCD study provides a measure of task performance incorporating both accuracy and reaction time (Luciana et al., 2018). Uncorrected scores will be used for analysis.

Decision making will be measured using the Game of Dice Task (GDT; Brand et al., 2005). In the GDT, participants guess the number they will roll on a set of fictional dice. Each outcome of the dice roll is associated with a fixed amount of fictional, potential monetary reward or gain. After each dice roll, participants receive feedback on both the outcome of the dice roll and their winnings. Certain rolls are associated with a higher probability of reward, with a smaller monetary gain (i.e., a "safe" decision), while others are associated with a higher probability of loss, but a larger potential monetary reward (i.e. "risky" decision). The ABCD study has adapted this task and provides measures of participants' monetary winnings or losses,

as well as the number of “safe” or “risky” decisions. The number of risky decisions will be used in this analysis.

Predictors of Profile Membership.

Pubertal status, race/ethnicity, SES, and Body Mass Index (BMI) will be examined as predictors of profile membership at baseline. Pubertal status will be assessed via the Pubertal Development Scale (PDS; Crockett & Petersen, 1987). The PDS is a 5-item questionnaire that assesses sex-specific physical changes (i.e., breast development, body hair) on a five-point scale (1 = pre-pubertal; 2 = early puberty; 3 = mid-puberty; 4 = late puberty; 5 = post-puberty). Both youth and their caregivers provided reports of youth pubertal status, but because youth tend to overestimate their pubertal status in early adolescence (Shirtcliff et al., 2009), the parent report will be used in the subsequent analysis. Race/ethnicity will be dummy coded to create five binary variables (White, Black, Hispanic, Asian, Other/Multi-race). White race/ethnicity will be used as the reference group in subsequent analyses. SES will be conceptualized via parent-reported yearly household income (1 = < \$50,000; 2 = > \$50,000 and < \$100,000; 3 = > \$100,000) and maternal education (1 = < high school diploma; 2 = high school diploma/General Education Degree (GED); 3 = Some college; 4 = Bachelor’s Degree/Post Graduate Degree). High income (> \$100,000) and having a Bachelor’s or Post Graduate Degree will be used as the reference group in the subsequent analyses. BMI will be calculated using measures of height and weight provided by ABCD. Open source code will be used to derive BMI percentile based on youth age and biological sex using growth charts provided by the Center for Disease Control (<http://www.cdc.gov/growthcharts>).

Control Variables

Following guidelines and best practices outlined in (Herting et al., 2020) for the analysis and processing of hormone data in ABCD, the following indicators will be included as controls in the LPA to ensure data quality and limit the influence of errors common to the collection of hormone samples: collection time, duration of salivary collection, time taken to freeze the saliva sample, use of hormonal birth control (girls only), caffeine usage, physical activity on day of lab visit, and wake-time on the day of the lab visit.

Analytic Plan

Data will be downloaded from the Data Exploration and Analysis Portal (DEAP). All variables will be assessed for normality and outliers in Stata Version 16.1 (StataCorp, 2007). Normality will be assessed via normal probability plots (quantile-quantile plots) of the data. If the data appear skewed, a transformation will be attempted in proceed with the proposed inferential analyses. Outliers will be determined using the *bacon* command, which is appropriate for use with population-level data (Weber, 2010). Briefly, the *bacon* command uses the Mahalanobis Distance (i.e., a metric that calculates the distance between every point and center in dimensional data) to determine multivariate outliers. Outliers identified by the *bacon* command will be excluded from the focal analyses. Then, descriptive statistics (means, medians, standard deviations/SDs) and correlational analysis will be conducted to describe the sample and report associations between hormonal data (testosterone, DHEA, estradiol), cognitive variables (Flanker, Little Man, Game of Dice tasks), and predictors of profile membership (pubertal status, race/ethnicity, SES, BMI). Additional correlational analyses will be conducted at this point to assess how hormone levels vary as a function of each control variable.

RQ1. What are the population-level profiles of hormone exposure in adolescents?

LPA will be used to identify patterns of hormone exposure using MPlus Version 8.0 (Muthén, 2007). As is common in the puberty literature, the LPA will be stratified by sex to account for differences in the meaning and interpretation of hormone levels (Marrocco et al., 2016; Neufamy et al., 2009). Based on current recommendations and to minimize overfitting, the full sample will be used to fit the profile solution (see He & Fan, 2019; Whittaker & Miller, 2021). All models will control for age, collection time, duration of salivary collection, time taken to freeze the saliva sample, use of hormonal birth control (girls only), caffeine usage, physical activity on day of lab visit, and wake-time on the day of the lab visit. The mixture complex command will be used to account for the nested structure of the data (individuals nested within families; families nested within site) and derive robust standard errors. Means and variances will be freely estimated, and hormone variables will be allowed to freely correlate with each other within profile. See the supplemental materials for analytic code.

Models will be run iteratively, starting with a one profile solution, and then adding additional profiles until the sample-adjusted Bayesian Information Criteria (saBIC) increases across two models in a row, until one of the profiles in a given solution represents less than 5% of the sample, or until model convergence issues occur (Jung & Wickrama, 2008; Whittaker & Miller, 2021). Following prior research (Ferguson et al., 2019; van der Nest et al., 2020), the final solution will be selected based on fit indices (lower saBIC scores), theoretical relevance (e.g., Have expected profiles emerged from the data?) and parsimony (e.g., Is an additional profile simply a variation of another?). See Figure 1 for an analytic decision-tree for evaluating the final model solution.

Once the final profile solution is selected, two sensitivity analyses will be conducted to assess the robustness of the profiles. First, given well-known diurnal rhythms of pubertal

hormones (Matchock et al., 2007), the sample will be restricted to those who provided a saliva sample in the morning (between 7:00 AM - 12:00 PM) to assess the effect of saliva collection timing on profile robustness. Second, if outliers were discovered during the descriptive analyses, the LPA will be re-run with that excluded data. If robust to the sensitivity analysis, inferential analyses related to the second and third research questions will continue. If not robust to these sensitivity analyses, additional exploratory analyses will be performed to examine the extent of the differences in profile composition and to determine the effect of saliva collection timing and outliers on subsequent inferences.

R2. How do sociodemographic factors (i.e., pubertal status, race/ethnicity, SES, BMI) predict profile membership?

Once the final LPA solution is selected, the most likely latent profile membership will be exported to Stata as an observed variable using the estimated posterior probabilities. Nested chi squares and ANOVAs will be used to compare profile membership by pubertal status, race/ethnicity, SES, and BMI adjusting for the multilevel structure of the data. Significant omnibus effects (at $p < .05$) will be followed up with post-hoc analyses (using a Bonferroni correction to account for multiple comparisons) to assess for significant pairwise differences ($p < .05$). If the hypothesized profiles do not emerge from the data, these analyses will be run using a more stringent threshold for evidence ($p < .005$).

Corrected p -values, standard deviations, confidence intervals, and effect sizes will be reported for all tests. Effect sizes for chi-square tests will be assessed with Cramer's V where .1 is considered a weak association, .4 is considered a medium association, and .5 is considered a strong association (Acock & Stavig, 1979). Effect sizes for ANOVAs will be assessed with a

partial eta squared (η^2) where .01 is considered a small effect size, .09 is considered a medium effect size, and .25 is considered a large effect size (Richardson, 2011).

R3. How is profile membership associated with cognition (i.e., inhibitory control, visuospatial processing, decision-making)?

The three-step Bolck, Croon, and Hagnaars (BCH) procedure in MPlus will be utilized to explore associations between profile membership and distal outcomes assessed two years later. As recommended by Asparouhov (2014), after the estimation of the final LPA solution (Step 1), the most likely profile membership will be calculated taking classification uncertainty into account (Step 2), and then applied to a secondary model where profile membership will be used to assess mean differences on distal outcomes (Step 3). The nested structure of the data will be accounted at all three analytic steps. Distal outcomes will be assessed with and without adjusting for mother's education, income, and race/ethnicity to fully elucidate the effects of profile membership on cognitive outcomes.

The statistical significance of profile-specific mean differences will be evaluated with Wald tests of mean equality, which are appropriate for continuous distal outcomes (Asparouhov, 2014). As Bonferroni corrections are not available for Wald tests in MPlus, the type I error inflation will be adjusted for using the Benjamini-Hochberg False Discovery Rate procedure (Benjamini, 1995) against a nominal alpha of 0.05. Again, if the hypothesized profiles do not emerge from the data, these analyses will be run using a more stringent threshold for evidence ($p < .005$). Additionally, an effect size (Cohen's d) will be calculated by dividing the mean difference score between two profiles by the distal outcome's standard deviation (SD) where 0.2 is considered a small effect, 0.5 is considered a medium effect and 0.8 is considered a large

effect size (Fritz et al., 2012). Full information maximum likelihood (FIML) will be used to address missing data.

Power Analyses

Power analysis within latent profile analysis framework is a developing field: The required sample size is dependent on the number of profiles and the distance between the profiles, which is unknown in advance (Tein et al., 2013). Some simulation studies suggest that samples between 300 - 500 may represent a minimal sample size for the detection of small effects (Finch & Bronk, 2011; Nylund et al., 2007), but caution will be taken when interpreting differences between profiles and when interpreting associations between profile membership and outcome variables. As described in the analytic plan, the significance value will be set at an alpha of .05 and corrected for multiple comparisons. Further, effect sizes will be reported and interpreted in terms of meaningfulness where small effects may be potentially consequential in the short-term, medium effects may evince some explanatory and practice usefulness in the long-term, and large effects may be potentially powerful in both the short and long-term (Funder & Ozer, 2019).

Table 1. List of Research Questions and Corresponding Hypotheses

Research Question	Prediction	Type of Analysis	Effect of Interest	Threshold for Evidence
RQ1. What are the population-level profiles of hormone exposure in adolescents?	H1a. The following subgroups will emerge in boys: 1. A “typical” profile that consists of relatively average levels of testosterone and DHEA compared to all other profiles. 2. A “high” hormone profile, distinguished by relatively higher testosterone and DHEA 3. A profile typified by higher DHEA only.	Latent profile analysis stratified by sex	saBIC	Best fitting model as indicated by a lower saBIC value, theoretical interpretability, and parsimony
	H1b. The following subgroups will emerge in girls: 1. A “typical” profile that consists of relatively average levels of testosterone and DHEA, and estradiol compared to all other profiles. 2. A “high” hormone profile, distinguished by relatively higher testosterone and DHEA, and estradiol 3. A profile typified by higher DHEA only. 4. A profile typified by higher testosterone only.			
R2. How do sociodemographic factors (i.e., pubertal status, race/ethnicity, SES, BMI) predict profile membership?	H2a. Boys and girls with more advanced pubertal status will be in profiles characterized by higher hormone exposure.	Nested Analysis of Variance (ANOVA) or Chi-Squares (for race/ethnicity)	Main effect of profile membership (e.g., profile 1 vs. profile 2)	$p < .05$ using Bonferroni corrections to adjust for multiple comparisons ($p < .005$)
	H2b. Black and Hispanic boys and girls will be in profiles characterized by comparatively higher overall hormone exposure or high DHEA exposure whereas White and Asian boys and girls will be in profiles characterized by relatively average hormone exposure			
	H2c. Boys and girls from lower SES backgrounds (measured by parent education and by yearly income) will be in profiles characterized by higher hormone exposure or higher levels of DHEA.			
	H2d. Boys and girls with higher BMI be in profiles characterized by higher hormone exposure. Girls with higher BMI may additionally be in profiles characterized by higher testosterone.			

R3. How is profile membership associated with cognition (i.e., inhibitory control, , visuospatial processing, decision-making)?	H3a.	In boys, the average hormone exposure profile will have better performance on the task measuring inhibitory control compared to other profiles. In girls, the average and high hormone exposure profiles will have better performance on the task measuring inhibitory control compared to other profiles			
	H3b.	In boys, the high hormone exposure profile will have better performance on the task measuring visuospatial processing compared to other profiles. In girls, the high hormone and high testosterone exposure profiles will have better performance on the task measuring visuospatial processing compared to other profiles	Wald tests of mean equality	Mean difference score	$p < .05$ using the Benjamini–Hochberg False Discovery Rate procedure to adjust for multiple comparisons ($p < .005$)
	H3c.	In boys, the high hormone exposure profile will have worse performance on the task measuring decision-making compared to other profiles, In girls, the high hormone and high testosterone exposure profile will have worse performance on the task measuring decision-making compared to other profiles			

Notes. As latent profile analysis is a data-driven analyses, the second and third research questions are contingent on the development of the profiles. Thus, if the expected profiles do not emerge from the data, any further analyses will be considered exploratory. Further, other profiles (in addition to those specified in RQ1) may emerge from the data. If so, analyses related to those profiles will be exploratory. The threshold for evidence will be more stringent for any exploratory analyses. The thresholds for those analyses are listed in parentheses. saBIC = sample-size Bayesian Information Criteria.

Figure 1. Latent Profile Analysis Model Evaluation Decision Tree

Notes. Model development will be guided by the saBIC values which recent simulation studies suggest recover accurate profile solutions in > 80% of cases (Whittaker & Miller, 2021). Model evaluation will prioritize theoretical interpretability and parsimony (i.e., fewer solution parameters). saBIC = sample-size adjusted Bayesian Information Criteria.

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Data Accessibility: A script detailing the analysis procedure for the current study will be made available for stage 2 peer review and for public download in conjunction with the finalized manuscript.

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