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Characterization of Puberty in an Australian Population-Based Cohort Study

Nandita Vijayakumar, Ph.D. [a,b,*], Hanafi Mohamad Husin, M.Biostat. [a], S. Ghazaleh Dashti, D.D.S., Ph.D., M.P.H. [c,d], Lisa Mundy, Ph.D. [a,b], Margarita Moreno-Betancur, Ph.D. [c,d], Russell M. Viner, Ph.D. [e], Anne-Lise Goddings, M.B.B.S., Ph.D. [e], Ellie Robson, Ph.D. [a], Susan M. Sawyer, M.B.B.S., M.D. [a,1], and George C. Patton, M.B.B.S., M.D. [a,1]

[a] *Centre for Adolescent Health, Murdoch Children's Research Institute and Royal Children's Hospital, Parkville, Victoria, Australia*
[b] *Deakin University, Centre for Social and Early Emotional Development, School of Psychology, Faculty of Health, Geelong, Victoria, Australia*
[c] *Clinical Epidemiology & Biostatistics, Royal Children's Hospital, Parkville, Victoria, Australia*
[d] *Department of Paediatrics, The University of Melbourne, Parkville, Victoria, Australia*
[e] *Population, Policy and Practice Research Department, UCL Great Ormond Street Institute of Child Health, London, United Kingdom*

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A B S T R A C T

Purpose: Current knowledge of the characteristics of puberty beyond age at menarche and thelarche is limited, particularly within population-based cohorts. Secular trends and concerns of the health effects of early puberty reinforce the value of contemporary studies characterizing the timing, tempo, duration, and synchronicity of puberty.
Methods: The Childhood to Adolescence Transition Study is a unique Australian cohort of individuals followed annually from late childhood to late adolescence, with up to eight assessments of pubertal stage from 9 to 19 years of age (N = 1,183; 636 females). At each assessment, females reported their Tanner Stage of breast and pubic hair development, while males reported on genital/pubic hair development. Nonlinear mixed-effects models characterized pubertal trajectories and were used to derive each individual's estimates of timing, tempo, and synchronicity. Parametric survival models were used to estimate the overall duration of puberty.
Results: Timing of mid-puberty (Tanner Stage 3) ranged from 12.5 to 13.5 years, with females developing approximately 6 months before males. Pubertal tempo (at mid-puberty) was similar across sex (between half and one Tanner Stage per year), but the overall duration of puberty was slightly shorter in males. Most females exhibited asynchronous changes of breast and pubic hair development.
Discussion: Estimates of pubertal timing and tempo are consistent with reports of cohorts from two or more decades ago, suggesting stabilization of certain pubertal characteristics in predominantly White populations. However, our understanding of the duration of puberty and individual differences in pubertal characteristics (e.g., synchronicity of physical changes) remains limited.

IMPLICATIONS AND CONTRIBUTION

Pubertal timing and tempo in a contemporary Australian sample were similar to reports from cohorts of two or more decades ago, suggesting stabilization of these features. However, the overall duration of puberty may be increasing over time. Implications for health and interpretation of the impact of COVID-19 are discussed.

Conflicts of interest: The authors have no conflicts of interest to declare.

* Address correspondence to: Nandita Vijayakumar, Deakin University, School of Psychology; Murdoch Children's Research Institute, Melbourne, Victoria, Australia.
E-mail addresses: nandi.vijayakumar@mcri.edu.au; nvijaya@deakin.edu.au (N. Vijayakumar).
[1] Co-senior authors.

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Puberty represents the most transformative period in growth and development outside of pregnancy and the early years of life [1]. It is shaped by the release of hormones that trigger rapid somatic growth and support sexual and reproductive maturation. These changes are accompanied by neurobiological maturation, which together contribute to marked psychosocial shifts and facilitate the transition to adult roles and responsibilities [2]. Crucially, characteristics of puberty predict several health outcomes, including mental illness and health risk behaviours [3], obesity, certain cancers, and potentially cardiovascular disease [4]. However, this research has predominantly relied on age at menarche or thelarche as markers of individual differences in the timing of puberty in females. Our understanding of pubertal characteristics beyond these markers is limited, particularly within population-based cohorts. Advancing this knowledge could serve to improve our understanding of the most pertinent risk factors for negative health outcomes, and thus inform interventions to reduce health disparities in the population.

Interindividual differences in physical maturation are typically based on the timing of recognized pubertal markers, with most research focused on the late event of menarche in females [5]. While there has been recent stabilization of the secular trend toward earlier menarche (particularly in predominantly White populations), the timing of secondary sexual development such as breast budding (thelarche) and pubic hair development (pubarche) may be shifting [6,7]. Combined with suggestions of later completion of these processes [8], the tempo and duration of puberty may also be changing. As such, there is a need for longitudinal characterization of puberty based on secondary sexual characteristics, within samples that are designed to capture early through late puberty. A focus on these visible characteristics will also support examination of puberty in males, who have been understudied given the lack of comparable, salient markers of pubertal maturity [3].

The development of secondary sexual characteristics is most often conceptualized as progression through five “Tanner” stages of development [9]. Individual differences in development can be captured through markers of timing (e.g., age at mid-puberty [Tanner Stage {TS} 3] for breast or pubic hair development) and tempo (i.e., rate of change over time). A small literature using prospective longitudinal designs has characterized nonlinear S-shaped trajectories of secondary sexual characteristics, with the timing of mid-puberty typically at 12–13 years of age and a tempo of 0.5 to 1 stages per year around mid-puberty [10–14]. Females reached mid-puberty roughly 1–2 years earlier than males, while estimates of tempo were similar across sex [10,11]. Even fewer studies have characterized synchrony in the development of different pubertal indicators, noting that a minority of individuals experience asynchronous maturation of physical changes (e.g., differential trajectories of breast and pubic hair development in females) [15–17]. The duration of puberty also varies dramatically across different definitions in the literature [18].

An important limitation of much of this literature has been the use of clinical or at-risk samples (e.g., parental substance use) [12,19] which limit inferences that can be made to the broader population. Furthermore, small samples covering a limited age range and/or consisting of few repeat assessments [11,12,19] are not adequately designed or powered to capture the full process of intraindividual pubertal maturation. Finally, longitudinal studies have been based on cohorts from two or more decades ago. Growing concerns about the health implications of pubertal timing and other pubertal phenotypes (including potential exacerbation of pubertal trends during COVID-19 [20,21]) reinforce the importance of characterizing puberty in a contemporary sample.

We also have limited understanding of how physical changes during puberty correspond with underlying hormonal changes. Adrenal hormones (dehydroepiandrosterone [DHEA] and dehydroepiandrosterone sulphate [DHEA-S]) released via the hypothalamic-pituitary-adrenal (HPA) axis are primarily responsible for the development of secondary sexual characteristics (e.g., pubic and axillary hair), while gonadal hormones (testosterone, estradiol, and progesterone) released via the hypothalamic-pituitary-gonadal (HPG) axis support the development of gonads and attainment of reproductive maturation. Only one study has investigated correspondence between intraindividual changes in hormones and physical maturity, reporting positive correlations between estimates of timing based on testosterone/DHEA and genital/pubic hair changes, but negligible correlations for estimates of tempo, in 11–16-year-old males [19]. Further research into such characterizations during puberty will be informative for future investigations into the mechanisms of pubertal associations with health outcomes.

The primary goal of the present study is to address the need for comprehensive characterization of physical development during puberty in a contemporary longitudinal sample. A secondary goal is to explore the correspondence between physical and hormonal changes during puberty. These are undertaken using the Childhood to Adolescence Transition Study (CATS), a unique Australian population-based cohort of individuals followed annually from late childhood to late adolescence [22]. Measurements of TS are available across eight annual waves, spanning the ages of 9–19 years. In females, stages of breast and pubic hair development were measured separately, reflecting maturation of the HPG and HPA axes, respectively. In males, a combined measure of genital and pubic hair development was used, reflecting the difficulty of separating the contribution of the two neuroendocrine axes in pubic hair development [23]. We aimed to estimate timing, tempo, and duration of pubertal maturation in both sexes, as well as synchrony of pubic hair and breast changes in females. As secondary analyses, we present correspondence between individual’s hormones (DHEA, DHEA-S, and testosterone) and TS trajectories in terms of timing and tempo. Given the availability of androgen hormones alone, we hypothesized that correspondence will be strongest for pubic hair relative to breast development in females. Correspondence may be weaker in males as the measure of genital/pubic hair development is a phenotype of both the neuroendocrine axes.

Methods

Study population and design

Participants were derived from CATS, an ongoing longitudinal cohort study with annual data collection since mid-primary school. Recruitment occurred through 43 schools in Melbourne, Australia, which were selected using a stratified random sampling approach. In 2012, all students in Grade 3 (aged 8–9 years) were invited to participate (N = 2,289), of which 54% were recruited into the cohort (N = 1,239). Further details on cohort design are reported elsewhere [22]. The study was approved by the Royal Children’s Hospital Human Research Ethics Committee.

Participants were followed annually from waves 1 to 10. Data collection occurred within schools until wave 4, with alternative arrangements for those who were unable to attend. In waves 5 and 6, participants either completed questionnaires at school, online, via post, or over the phone (through Computer-Assisted Telephone Interviews, with the assistance of paper/electronic questionnaires sent to participants). From wave 7, all data collection was completed online, via post or Computer-Assisted Telephone Interviews. The current analyses include 1,183 participants (636 females) with any available data on TS between waves 3 (mean age = 10.92 years, standard deviation [SD] = 0.40, 9.82–13.00) and 10 (mean age = 17.82 years, SD = 0.39, 16.87–19.43), when self-report measures were administered. This sample excluded 56 (31 females and 25 males) of the full cohort who did not have any available data on TS across these waves. Most of the analytic sample identified as Anglo-Celtic or European (70%) and resided in socioeconomically advantaged neighborhoods (62% in fourth and fifth quantile). Refer to Table 1 for further demographic characterization (and Table A1 for comparison of the included and excluded participants).

Measures

Pubertal stage was measured using the self-reported Sexual Maturity Status (SMS) [24] from waves 3 to 10. The SMS consists of a series of line drawings of female and male bodies corresponding to the TSs and has been shown to have good validation with physical examination [24]. Participants were asked to select pictures that best matched their current physical maturity. In the SMS, females chose from five stages for (1) pubic hair and (2) breast development, which are largely driven by different neuroendocrine systems (HPA vs. HPG) [25]. However, males chose from five stages of genital and pubic hair development combined, as clear distinction between neuroendocrine systems is not present in males (i.e., both systems contribute to pubic hair development) [25,26]. Thus, the SMS provides information on adrenarchal and gonadarchal maturation separately in females, but an overall index of pubertal maturation (that combines across adrenarchal and gonadarchal processes) in males. Females were also asked if, and when, they had started menstruating in waves 3 to 9. First report of age at menarche was examined in subsequent analyses.

At waves 1, 3, and 4, participants provided a saliva sample via the passive drool method. Collection typically occurred in classrooms, with 83% of saliva samples provided between 9 A.M. and 10 A.M. across waves. Those unable to attend school sessions provided early-morning samples at home using saliva collection kits (4.5% of saliva samples). Samples were assayed in duplicate to measure DHEA, DHEA-S, and testosterone using highly sensitive salivary enzyme immunoassay kits. The average value for each hormone concentration was used in subsequent analyses. Further details on saliva collection and hormone measurements are reported by Dashti et al. [27].

Statistical analyses

Participants' demographic characteristics were described using summary statistics. All analyses were stratified by sex and performed using Stata 17.0 [28]. In females, analyses were undertaken separately for breast and pubic hair development. In males, analyses combined genital and pubic hair development.

Estimates of timing and tempo. For each TS measure, we used the **menl** command in Stata to fit nonlinear mixed-effects models with subject-specific random intercept and age slope, as described by Marceau et al. [11]. Lower and upper asymptotes of the models were, respectively, set to 1 and 5, reflecting normative progression through TSs. Models' fixed effects provide estimates of the population mean age at which individuals are halfway between TS-1 and TS-5 (i.e., mid-puberty or TS-3, taken as a measure for average timing) and the population mean progression rate at TS-3 (measure of average tempo). The estimated subject-specific random intercept (denoted by $\widehat{u_{0i}}$) and random slope (denoted by $\widehat{u_{1i}}$) for individual $i = 1, \ldots, n$ estimate their deviation from the population means for age and progression rate at TS-3, respectively. Models assume that u_{0i} and u_{1i} follow a

Table 1
Descriptive statistics for participants with any available data on Tanner Stage (N = 1,183)

	Female (N = 636)							Male (N = 547)						
	n	%	Mean	SD	Median	IQR		n	%	Mean	SD	Median	IQR	
Age at study entry (wave 1)	636	100	9.0	0.4	8.9	8.7	9.2	547	100	9.0	0.4	9.0	8.7	9.3
Number of waves participated (waves 1–10)	636	100	8.1	2.5	9.0	7.0	10.0	547	100	8.0	2.5	9.0	6.0	10.0
Age at menarche	576	91	12.3	1.5	12.0	11.0	13.3							
Ethnic background														
Anglo-Celtic/European	328	71						250	70					
East/South East Asian	55	12						42	12					
South Asian/Middle Eastern	25	5						29	8					
Sub-Saharan African[a]	<10	<2						<10	<2					
Aboriginal and Torres Strait/Pacific Islander[a]	<10	<2						<10	<2					
Other[a]	<10	<2						<10	<2					
Multiple backgrounds	35	8						22	6					
SES (SEIFA IRSAD)														
First quantile–least advantaged	90	14						70	12					
Second quantile	63	10						41	7					
Third quantile	92	14						99	17					
Fourth quantile	170	27						168	29					
Fifth quantile–most advantaged	221	35						194	34					

SD = Standard deviation; IQR = Interquartile Range; SES = Socioeconomic Status; SEIFA = Socioeconomic Index for Areas; IRSAD = Index of Relative Socioeconomic Advantage and Disadvantage.

[a] Actual frequencies not shown due to possible reidentification.

normal distribution with mean 0 and variances $\sigma^2_{u_0}$ and $\sigma^2_{u_1}$, respectively. We used $\widehat{\sigma}^2_{u_0}$ and $\widehat{\sigma}^2_{u_1}$, estimated from fitted models, to derive the intervals within which 95% of the individual trajectories would fall, respectively, in terms of timing and tempo at TS-3 (probability interval). Using the individual estimates of timing, for females, we also described the correlation between self-reported age at menarche and timing of breast and pubic hair development.

We fitted these mixed-effects models from participants with any available TS data across waves 3 to 10 (even if missing data for one or more waves), an approach that yields unbiased estimates of model parameters under a "missing at random" assumption. We also conducted sensitivity analyses that excluded participants with only one to two waves of available data (subgroup 1) and those that reported no change in TS across all waves or highly inconsistent changes in TS (defined as decrease by two or more stages in subsequent waves and/or repeated decreases by one TS; subgroup 2). The sample size for each of these subgroup analyses is presented in Table 2.

Duration of puberty. For each TS measure, we used the **stintreg** command in Stata to fit two parametric log-normal survival models and estimate the mean age at reaching the pubertal milestones of TS-2 and TS-5, respectively. CATS data were collected annually; therefore, the exact age when each participant entered these pubertal milestones was unknown. Such data are referred to as interval-censored: observations are considered left-censored for participants who reached a milestone before a visit (i.e., missing observation of TS-1 or TS-4); interval-censored for those who reached the milestone between two visits; and right-censored for those who did not reach a milestone during a visit (i.e., missing observation of TS-2 or TS-5). The **stintreg** command in Stata accommodates for such interval-censored data. Both sets of models were only estimated in participants who had observations of TS-1 and TS-2, as well as TS-4 and TS-5 (339 [51%] females for breast, 410 [61%] females for pubic hair, and 412 [72%] males for pubic hair). Table A2 provides further details on the nature of censored data and number of participants excluded from analyses. The duration of puberty was taken as the difference in mean age at TS-5 and TS-2, and associated confidence intervals (CIs) were obtained via bootstrapping with 1,000 replications.

Synchrony of Tanner Stage development. For females, we estimated the proportion of individuals with asynchronous TS development, defined as a difference between the subject-specific timing (i.e., the estimated age at reaching TS-3 for an individual) for breast and pubic hair development of 6 months or more.

Correspondence between Tanner Stage and hormonal development. First, we classified individuals based on subject-specific timing and tempo for TS development (relative to population averages). We grouped individuals into three classes of

Table 2
Nonlinear mixed-effects models of Tanner Stage development, including sensitivity analyses

Female Breast	Full; N = 636					Subgroup 1[a]; N = 554					Subgroup 2[a]; N = 497				
	Estimate	95% CI		95% PI		Estimate	95% CI		95% PI		Estimate	95% CI		95% PI	
Timing[b]	12.59	12.472	12.707	9.941	15.238	12.635	12.509	12.761	9.949	15.321	12.639	12.513	12.766	10.023	15.255
Tempo[c]	0.552	0.527	0.577	0.071	1.032	0.541	0.515	0.566	0.066	1.015	0.575	0.549	0.601	0.117	1.032
Timing SD	1.351	1.261	1.448			1.879	1.624	2.174			1.781	1.539	2.062		
Tempo SD	0.245	0.224	0.269			0.059	0.049	0.071			0.054	0.044	0.067		
Timing/Tempo correlation	−0.15	−0.268	−0.027			−0.039	−0.081	0.002			−0.054	−0.092	−0.015		
Residual SD	0.482	0.469	0.495			0.233	0.22	0.246			0.201	0.189	0.212		
AIC	7,281.1					6,967.5					5,859.9				
Female–Pubic Hair	**Full; N = 634**					**Subgroup 1; N = 551**					**Subgroup 2; N = 479**				
	Estimate	95% CI		95% PI		Estimate	95% CI		95% PI		Estimate	95% CI		95% PI	
Timing[b]	13.117	12.994	13.24	10.352	15.881	13.118	12.988	13.248	10.329	15.907	13.134	13.006	13.261	10.508	15.759
Tempo[c]	0.661	0.63	0.692	0.077	1.245	0.659	0.627	0.691	0.073	1.245	0.723	0.69	0.755	0.178	1.267
Timing SD	1.989	1.724	2.295			2.024	1.744	2.349			1.794	1.549	2.078		
Tempo SD	0.089	0.074	0.107			0.089	0.074	0.108			0.077	0.062	0.096		
Timing/Tempo correlation	−0.074	−0.128	−0.02			−0.067	−0.123	−0.012			−0.074	−0.123	−0.025		
Residual SD	0.303	0.287	0.32			0.302	0.286	0.319			0.237	0.223	0.251		
AIC	8,209.5					7,860.7					6,168.9				
Male–Genital/Pubic Hair	**Full; N = 547**					**Subgroup 1; N = 473**					**Subgroup 2; N = 409**				
	Estimate	95% CI		95% PI		Estimate	95% CI		95% PI		Estimate	95% CI		95% PI	
Timing[b]	13.431	13.332	13.53	11.395	15.466	13.48	13.378	13.582	11.458	15.502	13.464	13.359	13.569	11.476	15.452
Tempo[c]	0.782	0.749	0.815	0.235	1.329	0.791	0.757	0.825	0.246	1.336	0.848	0.813	0.884	0.302	1.395
Timing SD	1.079	0.923	1.261			1.064	0.908	1.248			1.029	0.877	1.207		
Tempo SD	0.078	0.063	0.097			0.077	0.062	0.097			0.078	0.062	0.098		
Timing/Tempo correlation	−0.103	−0.145	−0.06			−0.105	−0.148	−0.063			−0.106	−0.146	−0.065		
Residual SD	0.284	0.268	0.301			0.28	0.264	0.297			0.212	0.199	0.226		
AIC	6,439.5					6,120.7					4,784.6				

CI = Confidence Interval; PI = Probability Interval; SD = Standard Deviation; AIC = Akaike Information Criterion.

[a] Subgroup 1 excludes participants with only one to two waves of available data, while Subgroup 2 excludes those who reported no change in TS across all waves or highly inconsistent changes in TS (defined as decrease by two or more stages in subsequent waves and/or repeated decreases by one TS).

[b] Timing is defined as the random intercept at Tanner Stage 3 in mixed-effects models.

[c] Tempo is defined as the random slope at Tanner Stage 3 in mixed-effects models.

comparatively 'early,' 'on time,' or 'late' based on how their random intercept ($\widehat{u_{0i}}$) compared with the tertiles of the estimated u_{0i} distribution ($N(0, \hat{\sigma}^2_{u_0})$: individual was classified as early if $\widehat{u_{0i}} \leq q_{0L}$, on time if $q_{0L} < \widehat{u_{0i}} \leq q_{0U}$, or late if $q_{0U} < \widehat{u_{0i}}$, where q_{0L} and q_{0U} represent the lower and upper tertiles of the estimated distribution. Similarly, we grouped individuals into two classes of 'slow' versus 'fast' tempo, based on how their random slope ($\widehat{u_{1i}}$) compared with the median of the estimated u_{1i} distribution ($N(0, \hat{\sigma}^2_{u_1})$: individual was classified as slow if $\widehat{u_{1i}} \leq 0$ and fast if $\widehat{u_{1i}} > 0$.

Classification of individuals based on their subject-specific timing and tempo of hormonal measures (relative to population averages) was previously undertaken using CATS data by Dashti et al. [27]. Briefly, linear mixed-effect models with subject-specific random intercepts and age slopes modelled longitudinal changes in hormone measurements (DHEA, DHEA-S, and testosterone). Models were used to derive individual estimates of adrenarche timing and tempo at 9 years of age, and individuals were grouped into (comparatively) 'early,' 'on time,' or 'late' and into 'slow' versus 'fast' based on how their timing and tempo, respectively, deviated from the estimated population averages.

Cross-tabulation across timing/tempo groups for TS and each hormone determined correspondence across different indices of pubertal maturation.

Results

Figure 1 illustrates population average TS trajectories in females and males. Table 2 shows estimates of the population averages (fixed effects) for timing and tempo (with 95% CI and probability interval) and estimated SDs and correlation parameters (random effects) for each TS measure. For females, the population averages for timing and tempo at mid-puberty (TS3) were, respectively, 12.6 years and 0.6 TS/year for breast development and 13.1 years and 0.7 TS/year for pubic hair development. For males, the population averages for timing and tempo at mid-puberty were, respectively, 13.4 years and 0.8 TS/year for genital/pubic hair development. Across measures, the between-individual variation in pubertal timing (i.e., SD of random intercepts) was of moderate magnitude compared with the population average of timing. Similarly, between-individual variation in pubertal tempo (i.e., SD of random slopes) was of moderate magnitude compared to the population average of tempo (Table 2). For females and males, the correlations between timing and slope were negative but weak (all coefficients < -0.15). Results were similar in sensitivity analyses that were limited to subgroups 1 and 2. In females, the timing of breast and pubic hair development were correlated with age at menarche at 0.549 and 0.334, respectively.

For females, the estimated duration of breast and pubic hair development was 6.5 years (CI: 6.1–6.9) and 5.6 years (CI: 5.2–5.9), respectively. Estimated duration of genital/pubic hair development in males was 4.9 years (CI: 4.6–5.1).

When comparing estimates of the timing of breast and pubic hair development in females, most started breast development more than 6 months before pubic hair development (n = 299; 47% [CI: 43–51]), followed by synchronous development (n = 224; 35% [CI: 32–39]). A small subset started pubic hair development more than 6 months before breast development (n = 111; 18% [CI: 15–21]).

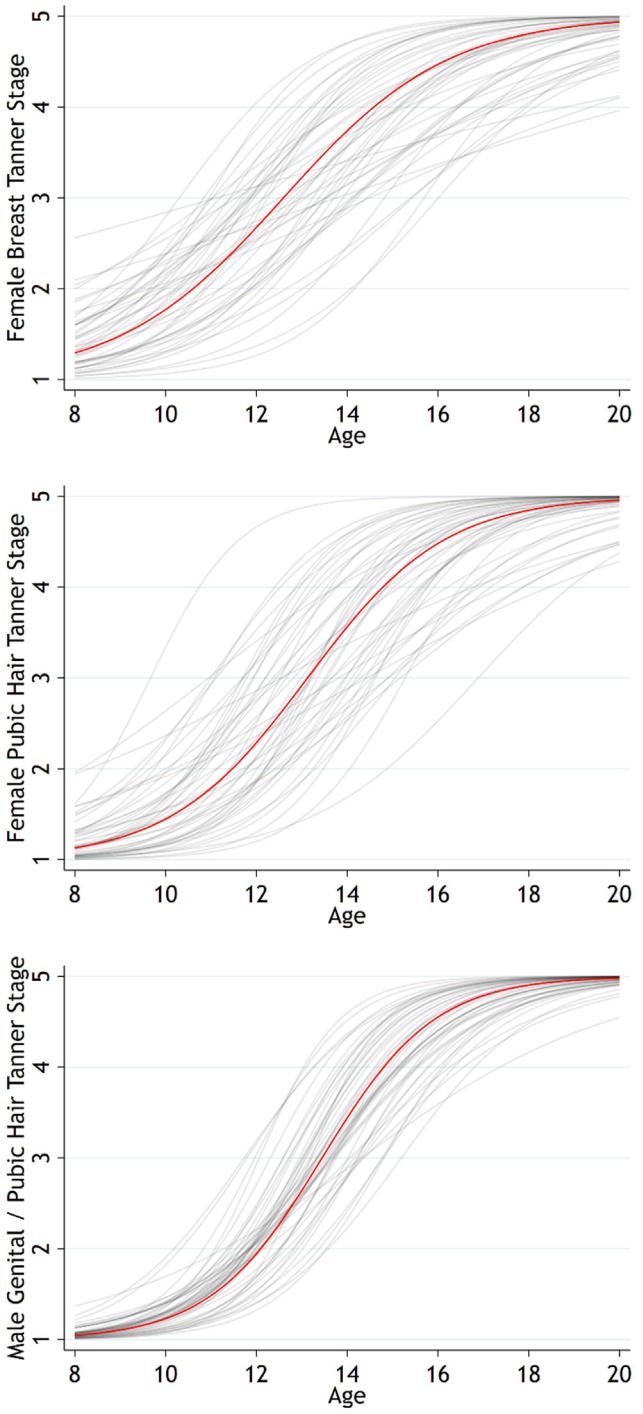

Figure 1. Predicted population-average trajectories (red) and 50 random individual trajectories (gray) for: (A) breast development and (B) pubic hair development in females and (C) genital/pubic hair development in males.

Figures 2 and 3 (and Tables A3 and A4) show the cross-tabulation of categories between timing and tempo for TS and hormonal development. In females, there was reasonable correspondence of timing categories; earlier and later developers based on the timing of hormone changes were also characterized

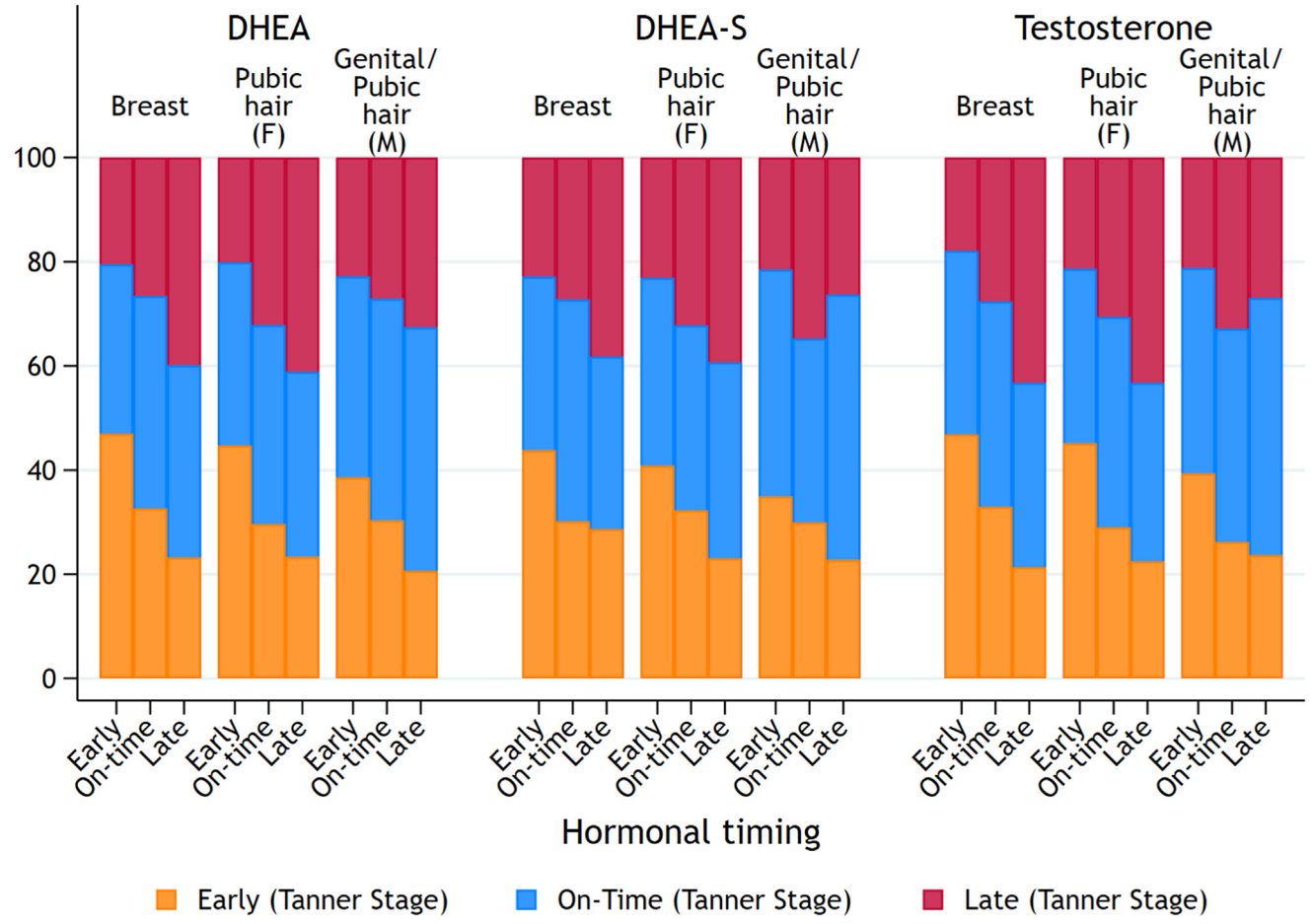

Figure 2. Correspondence between estimates of hormonal and Tanner Stage timing. F = females, M = males.

as earlier and later developers (respectively) based on the timing of TS breast and pubic hair development. While similar correspondence between genital/pubic hair and hormones was identified for earlier developing males, there was less correspondence when considering later developers. Furthermore, no clear correspondence was identified for any estimates of tempo, with roughly equal numbers of faster maturers based on hormones categorized as slower and faster maturers based on TS (across females and males).

Discussion

These findings summarize the characteristics of physical development during puberty in a contemporary (population-based) sample of Australian males and females who were longitudinally assessed between 9 and 19 years of age. Females reached mid-puberty between approximately 12.5 and 13.0 years of age on average, while males reached this milestone approximately six months later. Estimates of mean pubertal tempo were similar in both sexes, ranging between half and one TS per year (at mid-puberty). The mean duration of puberty ranged from approximately 5 to 6.5 years, with longer estimates in females relative to males. Females most commonly exhibited asynchronous maturation, characterized by earlier maturation of breasts relative to pubic hair. Finally, there was reasonable correspondence between estimates of timing based on physical and hormonal development (particularly in females), but there was little correspondence in estimates of tempo.

Estimates of the timing of mid-puberty in the CATS cohort (between 12.5 and 13.5 years of age) are consistent with prior longitudinal studies that have used similar measures and nonlinear modelling of TS, including cohorts from two or more decades ago [10,11], as well as the more recent Danish National Birth Cohort Puberty Cohort (with similar characteristics to the CATS cohort) [29]. Females reached mid-puberty based on pubic hair development less than 6 months earlier than males. This sex difference is considerably smaller than the reports of at least 12 months that were typical in older cohort studies [30]. However, smaller differences have also been reported in more recent cohorts employing similar methodology to our study [10,11]. The stabilization in pubertal timing also broadly aligns with stabilization of secular trends for age at menarche over the last few decades in certain countries [31,32]. Mean age at menarche in CATS was 12.3 years, consistent with another Australian cohort that is a decade older (Raine Study) [33]. Estimates are also comparable with recent reports in Europe [34], which generally range between 12 and 13 years [35]. While contemporary estimates can be younger in the United States, they are closer to 12 years in non-Hispanic Whites [36]. Thus, across multiple metrics, our findings add to the suggestions of potential

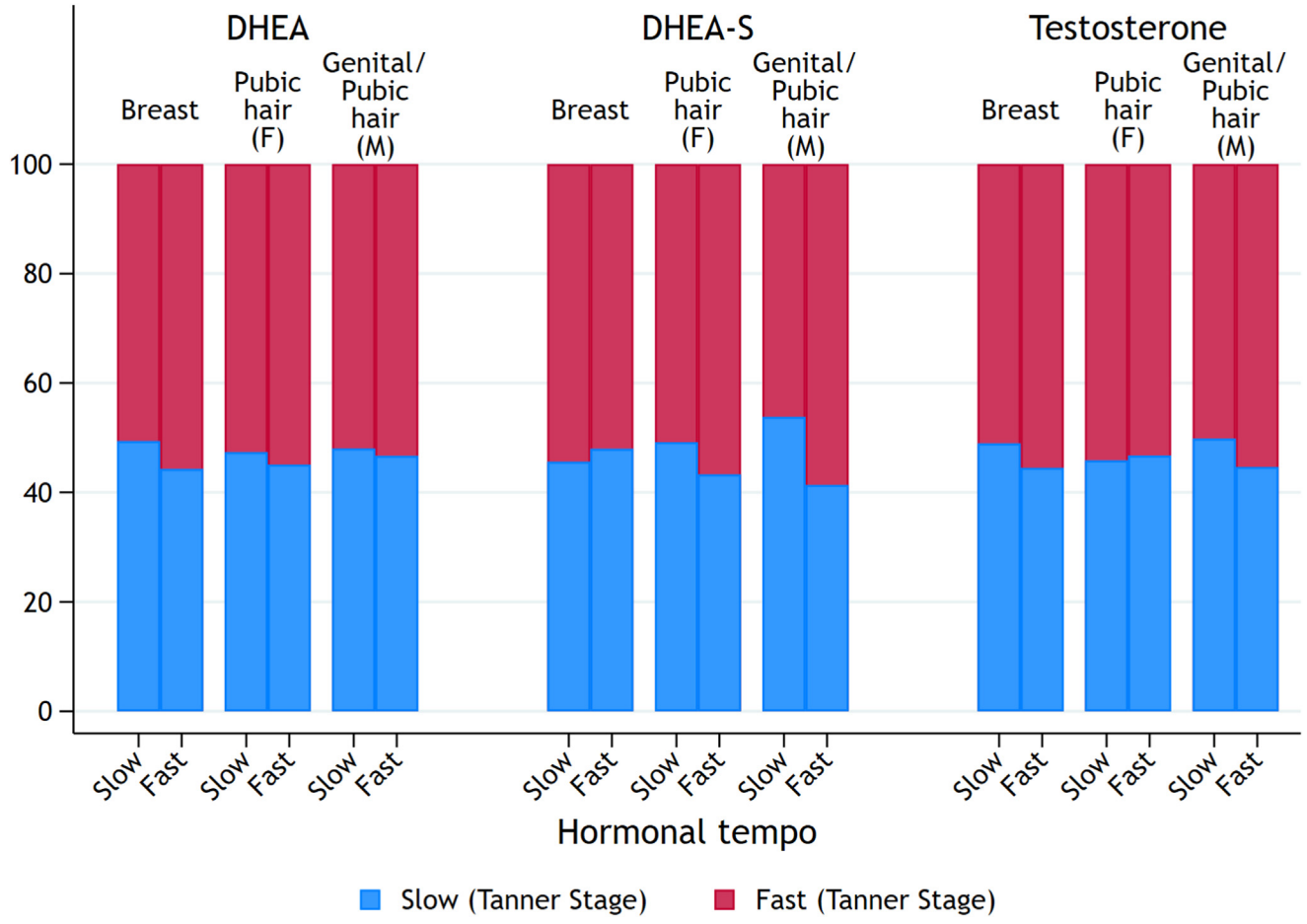

Figure 3. Correspondence between estimates of hormonal and Tanner Stage tempo. F = females, M = males.

stabilization of pubertal timing over the past few decades, particularly in predominantly White populations. Importantly, this knowledge of pubertal timing in a contemporary cohort aids the interpretation of recent reports of increased cases of early and precocious puberty in females during the COVID-19 pandemic [21] and its implications for future health outcomes in the current generation of adolescents.

Findings of pubertal tempo in CATS are also consistent with prior estimates at mid-puberty, which typically range between half and one TS per year for both sexes [10–12]. We also identified negative associations between timing and tempo, such that early developers exhibit slower patterns of maturation. Although small in size, this negative correlation was consistent across males and females. Prior longitudinal research using similar modelling approaches has reported positive associations between timing and tempo in females, such that early developers exhibit faster maturation [10,12], although negative associations have been reported in males [11,12]. Other studies have failed to identify any meaningful associations in either one or both sexes [10,11,19]. Of note, our sensitivity analyses yielded reduced estimates of variance in the tempo of breast development and the magnitude of negative correlations between timing and tempo of breast development. This suggests that models may be sensitive to design aspects of longitudinal cohorts that vary across studies (e.g., age range captured, number of waves, and intervals between waves). Thus, our understanding of the relationship between timing and tempo remains limited. There is most support for earlier pubertal timing increasing risk for several mental health problems (e.g., depression, anxiety, health-risking behaviors) [3], but few have investigated the impacts of pubertal tempo with mixed results [10–12,37,38]. The present study provides contemporary thresholds for early timing and fast tempo that can inform continued investigations into the health consequences of these pubertal trajectories.

Estimates of the duration of puberty from TS-2 to TS-5 were 6.5 and 5.6 years for breast and pubic hair development in females (respectively) and 4.9 years for genital/pubic hair development in males. While these estimates are considerably larger than existing literature based on older cohorts (which range up to 4.5 years) [18], this overall pattern is consistent with the notion of earlier age of onset of secondary sexual characteristics together with later completion of development [8]. Our estimates are also more comparable with recent estimates in the Danish National Birth Cohort [29], which further supports the notion that the duration of puberty may be increasing. Nonetheless, as prior studies employed varied methods of assessing duration, it remains unclear whether these differences are attributable to cohort effects (i.e., increased length of puberty

over time) or methodological inconsistencies. Further research is warranted, particularly in large, population-representative cohorts that capture the extended period from pubertal onset to termination, as increasing duration of puberty has been identified as a risk factor for breast cancer independent to the timing of puberty [39].

Our characterization of pubertal synchronicity highlighted individual differences in the maturation of adrenarchal and gonadarchal components of puberty in females. Females most commonly reached mid-puberty based on breast development six or more months prior to mid-puberty based on pubic hair development, and the population average timing of breast development was earlier than that of pubic hair development by 6 months. This was followed by a considerable portion who exhibited synchronous maturation and comparatively few with earlier pubic hair development relative to breast development. This pattern of findings differed from prior studies of synchrony that have identified predominantly earlier pubic hair development [16] or synchronous development [17]. Nonetheless, current findings align with reports of earlier (mean levels of) timing of breast versus pubic hair development in females [40] and a greater percentage of females with higher TS for breast relative to pubic hair development at a given time point during early adolescence [38]. The relevance of pubertal synchronicity for mental health is understudied, but there is some preliminary evidence that this pattern of asynchronous pubertal development in females (particularly characterized by earlier maturation of the overt signs of puberty and later maturation of covert signs) may be a stronger risk factor for depression than pubertal timing [15].

Finally, there were discrepant findings regarding correspondence between pubertal estimates of timing/tempo based on TS and timing/tempo based on adrenal hormones. Individuals with early timing based on hormones also exhibited earlier physical maturation (i.e., most of those with higher levels of hormones during late childhood were quicker to reach TS-3). However, those with faster hormonal tempo were equally likely to exhibit fast versus slow tempo based on TS, which may be influenced by the limited variance in hormonal tempo in our sample. The only prior investigation comparing estimates of TS with hormonal development also noted positive associations for timing of testosterone/DHEA and physical changes, but no such associations for tempo, in White males [19]. They also showed that timing of testosterone but not TS predicted substance use in late adolescence, suggesting a potential physiological (rather than psychosocial) mechanism of action. Interestingly, our preliminary analyses identified similar patterns of correspondence between adrenal hormones and both adrenarchal and gonadarchal phenotypes in females, as well as combined phenotypes in males (although testosterone is related to both adrenal and gonadal endocrine axes in males). Further research is warranted that distinguishes these aspects of puberty, such as the measurement of estradiol in females, differentiation of genital and pubic hair changes in males, and comparison of late childhood and mid-adolescence (to more effectively capture adrenarchal and gonadarchal processes, respectively).

Although CATS represents one of very few contemporary longitudinal cohorts with repeated assessments of multiple indices of puberty across adolescence, these findings need to be considered in light of certain limitations. First, there was less detailed characterization of puberty in males, and as such, we were unable to tease apart adrenarchal and gonadarchal components of physical changes. Further examination into this distinction, as well as age at spermarche, is warranted. Second, analyses on hormonal correspondence are restricted to adrenal hormones. Future research should incorporate estradiol and progesterone, which are likely to be more strongly correlated with breast development in females. Moreover, measuring hormonal changes beyond the late childhood to early adolescent window will be important to adequately capture gonadarchal development, and it is also important to note that hormone levels can additionally reflect non-pubertal processes. Third, self-report measures of TS have some limitations compared to clinician ratings, including overestimation at early stages, underestimation at late stages, and poorer correspondence to underlying hormones [41,42]. Nonetheless, self-report measures remain the most feasible assessments in longitudinal population cohorts. Fourth, the lack of self-report TS measurements prior to wave 3 could lead to bias in model estimates of timing and tempo due to missed earlier TS reports (i.e., informative left censoring). Fifth, modelling of pubertal trajectories would be more accurate with shorter time intervals between assessment waves. Sixth, the CATS cohort is predominantly White. This reflects the demographics of the Australian population, but the findings may be less generalizable to other populations with greater diversity in race and ethnicity.

In conclusion, estimates of pubertal timing and tempo in CATS, a contemporary Australian population-based sample, are consistent with prior reports of cohorts from two or more decades ago. This suggests a stabilization of certain (mean levels of) pubertal characteristics in predominantly White populations over the past few decades. However, our understanding of the duration of puberty and individual differences in these trajectories (such as the correlation between timing and tempo, as well as the synchronicity of physical changes) remains limited. While we also present preliminary evidence of correspondence between the timing of adrenal hormonal and physical development during puberty, further research is needed into correspondence in the tempo of these pubertal markers.

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Supplementary Data

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