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Effects of War Exposure on Pubertal Development in Refugee Children

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Abstract

Increasing research shows pubertal development accelerates in response to threats while it decelerates in response to deprivation. Yet, these environmental stressors are unlikely to occur in isolation. We investigated how war exposure and energetic stress impact pubertal development using data from the longitudinal Biological Pathways of Risk and Resilience in Syrian Refugee Children (BIOPATH) study. Our sample included 1,576 male and female Syrian refugee children and their caregivers who lived in temporary settlements in Lebanon. We hypothesized that: 1) energetic stress suppresses pubertal development; 2) war exposure accelerates pubertal timing in boys and increases risk of menarche in girls, but only under conditions of lower energetic stress; and 3) when energetic stress is elevated, effects of war exposure on pubertal development will be attenuated. Among boys, we did not find support for Hypothesis 1, but Hypotheses 2 and 3 were supported. Exposure to morbidity/mortality threats were associated with accelerated pubertal timing; this effect was attenuated under conditions of elevated energetic stress. Among girls, we found support for Hypothesis 1, but not for Hypotheses 2 and 3. Elevated energetic

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Author Contribution Statement

BIO = Contribution to BIOPATH study; PS = Contribution to present study

CJB: Conceptualization (PS), Data curation (PS), Formal analysis (PS), Funding acquisition (PS), Methodology (PS), Visualization (PS), Writing—original draft (PS), Writing—review and editing (PS)

FSM: Data curation (BIO, PS), Investigation (BIO), Methodology (BIO), Project administration (BIO), Validation (BIO), Writing—review and editing (PS)

DS: Data curation (BIO, PS), Writing—review and editing (PS)

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Disclosures

The authors declare no competing interests or financial support.

Ethics Approval Statement

The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable standards. Ethical approval was granted by the Institutional Review Board of the Unversity of Balamand / Saint George Hospital University Medical Center, Lebanon (ref: IRB/O/024-16/1815). The study was also reviewed by the Lebanese National Consultative Committee on Ethics and approved by the Ministry of Public Health.

stress decreased the risk of menarche in girls. Neither war exposure, nor any interactions with energetic stress, predicted risk of menarche. Sensitivity analyses revealed a significant interaction between bombing exposure and the amount of time since leaving Syria. Bombing decreased the risk of menarche, but only for girls who had left Syria four or more years prior to data collection. We discuss implications for translational efforts advocating for puberty screening in medical and mental health settings to identify trauma-exposed youth.

Keywords

Puberty; Conflict; Refugees; Stress; Nutrition

In recent years, increasing evidence has linked the timing and pace of puberty to mental and physical health later in life (Binder et al., 2018; Hamlat et al., 2021; Suarez et al., 2018; Sumner et al., 2019). In particular, accelerated pubertal development is associated with internalizing and externalizing behavior, aggression, substance use, and risky sexual behavior (Joos et al., 2018). The onset of pubertal development shifts in response to environmental conditions and has been studied extensively in relation to childhood adversity following predictions of Psychosocial Acceleration Theory (PAT; Belsky et al., 1991). While a number of studies have demonstrated how psychosocial stress accelerates pubertal development, it may respond differently to different kinds of adversity. For example, deprivation can delay pubertal timing while threats accelerate it (Colich et al., 2020; Sumner et al., 2019). However, much of the literature on pubertal development has been conducted in W.E.I.R.D. populations (Henrich et al., 2010). In the current study, we investigate effects of exposure to war on pubertal development in Syrian refugee children living in temporary settlements in Lebanon. We apply life history theory to advance understanding of differential impacts of certain kinds of adversity and, importantly, demonstrate that context must be taken into account when interpreting effects on pubertal development, especially in low- and middle-income countries (LMICs) where resources are more scarce. We begin with a brief description of the Syrian Civil War which exposed millions of families to conflict and displacement. We then discuss how evolutionary developmental theory elucidates patterns of child development and behavior that may follow different kinds of exposures. We conclude by discussing the few available studies on war exposure and pubertal development which sets the stage for the current study.

Syrian Refugee Context

For over 10 years, Syria has faced a brutal civil war characterized by state-sponsored chemical warfare, barbaric atrocities by terrorist groups, and millions of Syrian families fleeing the country in one of the worst humanitarian crises of the modern era. The Syrian conflict led to widespread displacement, resulting in 6.8 million refugees and 6.7 million internally displaced people (UNHCR, 2022). Nearby countries, including Lebanon, Jordan, Turkey, Egypt, and Iraq, have received the majority of refugees, many of whom are still living in informal temporary settlements and struggling to meet basic needs. Nearly half of Syrian refugees are children who, along with their families, have faced tremendous trauma and loss of family members and friends, along with social institutions such as education that

help stabilize child and adolescent development. These experiences, along with the stress associated with displacement, raise concerns about the long-term effects on the development and well-being of Syrian children.

Evolutionary-Developmental Models of Child Development

Evolutionary models of human development offer a unique lens for understanding biological embedding of exposure to adversity (Belsky, 2008; Ellis et al., 2009, 2022). Life history theory describes how bioenergetic and material resources are allocated toward trade-offs impacting evolutionary fitness (Stearns, 1992). Life history strategies have been characterized as falling along a fast-slow continuum (but see Del Giudice, 2020 for a critique of this approach). Slower strategies are comprised of later maturation, lower fertility, larger offspring, longer interbirth intervals, and longer lifespan. In contrast, faster strategies are reflected in earlier maturation, higher fertility, smaller offspring, shorter interbirth intervals, and shorter lifespan.

Belsky et al. (1991) drew on evolutionary-developmental models, including life history theory and parental investment theory (Trivers, 1974), to propose Psychosocial Acceleration Theory which suggested that childhood environments influence reproductive strategies. According to this framework, marital discord, high stress, and inadequate financial resources contribute to poor parenting characterized by harshness, rejection, insensitivity, and inconsistency. This leads to children developing insecure attachment, mistrust, and antisocial interpersonal orientations (e.g., opportunism, aggression, anxiety, depression). These children are more likely to experience earlier maturation and accelerated pubertal timing which increases the probability of engaging in earlier sexual activity and having more unstable, short-term pair bonds. Ultimately, this pattern impacts parenting behavior by limiting parental investment and repeating the pattern in the following generation. In contrast, more positive rearing environments should forecast secure attachment, trust, interdependent and prosocial interpersonal orientations, later maturation, later sexual activity, and more stable long-term pair bonds. This pattern increases parental investment and self-propagates in subsequent generations.

While Psychosocial Acceleration Theory has offered a strong framework for identifying environmental risk factors that may influence pubertal development, it does not consider several elements which might make it more generalizable in LMICs where >90% of adolescents reside (United Nations, 2022). For instance, individuals in LMICs are more likely to come from collectivistic cultures where interdependence with an in-group is promoted. Families in collectivistic cultures are more likely to live with extended family spanning several generations, meaning that these children may be more buffered from marital discord than children in nuclear families which are more characteristic of individualistic cultures. LMICs also systematically differ in other ways as well, as populations in these settings experience more political and economic instability, armed conflict, and interpersonal violence (Song & Shaheen, 2013), all of which could impact pubertal development. Subsequent evolutionary-developmental theoretical work distilled environmental risk into two fundamental dimensions, harshness and unpredictability, which are broadly applicable in diverse human contexts (Ellis et al., 2009; 2022). Harshness is

characterized by elevated morbidity and mortality and energetic stress (i.e., resource scarcity or nutrition deprivation) while unpredictability refers to temporal and spatial variance in harshness. Figure 1 below demonstrates how developmental trajectories are impacted by variation in harshness (modified with permission from Ellis et al., 2009). Key dimensions include energetic stress, age-specific morbidity and mortality, social competition, and sensitivity to resource allocation decisions (Ellis et al., 2009, 2022).

Of particular relevance to the current study are outcomes anticipated by elevated morbidity and mortality, as well as elevated energetic stress. For populations facing elevated adult-specific or overall morbidity/mortality, reproductive success is enhanced by accelerating maturation and increasing the rate of reproduction. When morbidity/mortality is juvenile specific, adaptive calibration of development depends on whether effects of morbidity and mortality are mitigated by resource allocation decisions of parents or offspring. When mitigation is possible, reproductive success is enhanced by delaying maturation and acquiring resources to improve condition and competitiveness. When mitigation is not possible, reproductive success benefits from accelerated maturation to increase time at reproductive maturity. Cross-cutting these conditions is resource scarcity/energetic stress. In populations facing resource scarcity and energetic stress, development is delayed to prioritize basic maintenance over growth due to low energy availability.

Pubertal Development and War Exposure

Over the last two decades, accumulating evidence has supported life history predictions about pubertal development, particularly with regard to the accelerating effects of child abuse (e.g., Mendle et al., 2011; Negriff et al., 2015), father absence (e.g., Neberich et al., 2010; Tither & Ellis, 2008), and traumatic stress (e.g., Gur et al., 2019; Hamlat et al., 2021). However, few have investigated the impact of war and its sequelae. Following the Bosnian war, Tahirović (1998) investigated age at menarche among girls who had lived in Srebrenica, where the Bosnian Serb Army massacred thousands of Muslim men and boys during the Srebrenica genocide. The community was under siege for over three years, leading to poor socioeconomic conditions, starvation, and disease. Compared with girls living in peaceful areas of Bosnia and Herzegovina, refugee girls from Srebrenica experienced significantly later menarche (14.43 years vs 13.04 for controls; Tahirović, 1998).

Prebeg and Bralić (2000) compared ages of menarche among girls from Šibenik, a town in Croatia that faced prolonged war conditions during the Croatian War of Independence (1991–1995). They compared age of menarche of girls living in Šibenik prior to the onset of the war (in 1981 and 1985) to those who had been exposed (in 1996). Prior to the war, age of menarche decreased by one month as access to nutrition improved, following widespread secular trends. After the war, age of menarche increased by three months, reversing this trend. These authors could not conclusively link these changes to war exposure, however, as other countries had observed slightly later onset in the same decades even in the absence of war.

Other studies examining menarche in relation to World War II focused on birth cohorts without directly measuring war exposures. These studies also tended to find delays in age at menarche, such as in Poland (Liczbińska et al., 2018) where girls born during World

War II (WWII) experienced later menarche (*mean* = 14.4 years, *SD* = 1.3) than girls born before (*mean* = 14.2 years, *SD* = 1.3) or after WWII (*mean* = 13.9 years, *SD* = 1.3). In the Netherlands, a study which focused on effects of the "Dutch Famine" (van Noord & Kaaks, 1991) found that mean age of menarche was 14.40 years for girls born in 1930, whose expected mean age of menarche would have occurred in the year of the famine. They estimated that, in the absence of the famine, girls' mean age of menarche would have been 13.75 years. Finally, in Japan, an analysis of growth characteristics before, during, and after WWII (Schneider et al., 2021) revealed that the pubertal growth spurt occurred 0.51 and 0.46 years later for war-affected boys and girls, respectively. These trends could reflect the impact of resource scarcity on pubertal development, which is more likely to result in delayed puberty. This interpretation is consistent with several studies which have found associations between war exposure and stunted physical growth (e.g., in Nigeria, Rwanda, Eritrea, and Ethopia, Akresh et al., 2012a, 2011, 2012b; in Laos, Clarkin, 2012; and in Nordic countries, Angell-Andersen et al., 2004). The magnitude of these effects on growth vary. For instance, the adult heights of girls exposed to the Nigerian civil war between 0–3 years of age were reduced by 0.75 cm while for girls exposed between 13–16 years of age the height reduction reached 4.53 cm. For girls between four to 12 years of age at the time of exposure, there was no evidence of height reduction (Akresh et al., 2011).

In contrast, Pesonen et al. (2008) investigated reproductive traits, including age at menarche, in the Helsinki Birth Cohort Study. Former child refugees who experienced parent separation as a result of the Soviet-Finnish wars experienced earlier menarche compared with children who did not experience separation because of the war. In general, the refugee group trended toward accelerated development and faster life history traits: men experienced earlier first child birth and shorter interbirth intervals while women had more children by late adulthood.

The Current Study

The seemingly conflicting results from previous research could be explained if both war exposures and nutrition deprivation are considered. The extant literature, for the most part, relied on archival data and year of birth to approximate war exposure. When war exposure was measured more directly, it lacked granularity (e.g., asking just three questions in Prebeg & Bralić, 2000), or was not used at all in relation to age at menarche (Tahirović, 1998). No study examined war exposure and undernutrition in conjunction, despite the likelihood of co-occurrence.

It is increasingly important from a global health standpoint to account for both psychosocial stress and energetic stress when measuring pubertal development, especially in LMICs where political conflict and resource scarcity are more common. Some life history research conducted in Cebu, Philippines has investigated relative effects of nutrition versus parental instability and/or sibling death, finding that nutrition but not psychosocial stressors predicted rates of maturation in boys and girls (Gettler, et al., 2015; Kyweluk et al., 2018). Yet, these studies did not test whether nutrition deprivation moderates effects of psychosocial stress on pubertal development.

We address these limitations using longitudinal data from the Biological Pathways of Risk and Resilience in Syrian Refugee Children (BIOPATH) study which collected questionnaire data and biological samples from 1,600 Syrian refugee children and their caregivers living in tented settlements in the Beqaa region of Lebanon in 2017 and 2018 (McEwen et al., 2022). We tested the following hypotheses:

- **I.** Refugee children experiencing higher energetic stress in the absence of war exposure will experience:
  - **A.** Delayed pubertal timing (in boys) or,
  - **B.** Decreased risk of menarche (in girls).
- **II.** Refugee children facing lower energetic stress and higher exposure to war events will experience:
  - **A.** Accelerated pubertal timing (in boys) or,
  - **B.** Increased risk of menarche (in girls).
- **III.** Refugee children facing higher energetic stress and higher exposure to war events will experience:
  - **A.** Attenuated pubertal timing acceleration (in boys) or,
  - **B.** Attenuated increases in risk of menarche (in girls).
- **IV.** Effects of war exposure will be especially strong for war events most closely associated with morbidity and mortality threats.

Methods

Participants

A detailed description of recruitment and sample characteristics for the BIOPATH study is provided in McEwen et al. (2022). In short, Syrian refugee child-caregiver dyads were recruited in refugee camps in Lebanon, with the following inclusion criteria: 1) family has a child between 8 and 16 years of age; 2) family left Syria within previous four years; and 3) at least one primary caregiver (i.e., a caregiver who spend the most time with the child) could be identified to answer questions about the child.

The first year of data collection included 1,600 child-caregiver pairs and 1,009 (63%) of these were followed up one year later. Five cases were excluded due to: data missing due to tablet failure; child less than 8 years old and unable to understand questions; family not living in a tented settlement; and/or family participated twice. After data cleaning (Supplemental Materials pp. 2–5), our sample consisted of 1,576 children (748 male and 828 female). Baseline sample characteristics are described in Table 1, disaggregated by sex. The average age of child participants was 11.48 years for boys (*SD* = 2.32, *range* = 7–17) and 11.37 years for girls (*SD* = 2.45, *range* = 7–17), reflecting a wider age range than specified by eligibility criteria. There were no significant differences between boys and girls at baseline. Age distributions are displayed in Figure S1 in the Supplemental Materials.

The study received IRB approval from the Unversity of Balamand / Saint George Hospital University Medical Center, Lebanon (ref: IRB/O/024-16/1815), sponsorship from Queen Mary University of London, and governmental approvals from the Lebanese Ministry of Public Health. Informed consent was obtained from parents, and assent was obtained from the child. Families were compensated for participation in the study.

Measures

Below we describe the measures used in the current study. The analytical models included measures of war exposure, energetic stress, pubertal development, and a covariate measuring the number of years since leaving Syria. The imputation model included all of these variables, as well as several auxiliary variables used to inform the imputation, including hair hormones, biometric data, family demographics, refugee environment, and environmental sensitivity (described in Supplemental Materials p. 4).

War Exposure—The War Exposure Questionnaire is a 25-item questionnaire developed by the Institute for Development, Research, Advocacy and Applied Care (IDRAAC) to measure exposure to war, adapted for use with Syrian refugees (Karam et al., 1999). The items are rated on a binary yes/no scale and ask about several war events including explosions, bombardment, destruction of property, kidnapping, beating, torture, injury, and death. For the current analysis, we constructed subscales based on conceptual similarity and theoretical predictions (Table 3 below). Based on predictions from Life History Theory that morbidity/mortality cues are most likely to accelerate pubertal development, we created a morbidity/mortality subscale by summing items that referenced injury or death. The remaining items were grouped based on conceptual similarity, resulting in a bombing subscale and a kidnapping subscale. We included all three constructs in our models to specifically test whether morbidity/mortality cues uniquely predict pubertal development when other types of war exposures are held equal.

Energetic Stress—Child height in cm, weight in kilograms (kg), hip and waist circumference in cm were transformed using the World Health Organization's (WHO) Child Growth Standards SPSS macro, which produced several metrics including BMI-for-age *z*-scores. These growth standards were developed as part of the WHO's Multicentre Growth Reference Study (2006). BMI-for-age *z*-scores are with reference to the population of the WHO study which included diverse samples from Brazil, Ghana, India, Norway, Oman, and the USA. BMI-for-age was retained as an indicator of bioenergetic stress based on known relations between body fat and pubertal development (Bygdell et al., 2020; Frisch & Revelle, 1970, 1971; Ohlsson et al., 2020) and because this metric is the most direct measure of energy balance in our sample.

Pubertal Development—Pubertal development was measured with two of five items on the Pubertal Development scale (Petersen et al., 1988). Items were selected based on feedback from our Lebanon-based partner, the Institute for Development, Research, Advocacy and Applied Care (IDRAAC) which has clinical experience with similar populations and which recommended using items without an immediate sexual association. Boys were asked two questions: 1) Have you begun to grow hair on your face? 2) Have

you noticed a deepening of your voice? Response options on a 4-point Likert-scale ranged from “1 = has not yet started growing” to “4 = seems complete”. Girls were also asked two questions: 1) Have you begun to menstruate (started to have your period)? 2) If yes, how old were you when you started to menstruate? The first question requested a yes/no response, while the second requested age in years.

For male children, responses were re-coded into one of four stages of pubertal development using the scoring criteria of the Pubertal Development Scale. Having only two of five items meant that no child could be classified as post-pubertal (the fifth pubertal stage) even though such children might be present in the sample (n=9 boys rated changes being complete across both years). It is also possible that having additional items may have increased variability in pubertal staging, and/or increased the precision of our results. Once data were recoded, ordinal pubertal development scores were regressed on age and the standardized residuals were retained as estimates of relative pubertal timing. Negative values indicate delayed pubertal timing while positive values indicate accelerated pubertal timing.

Among female children, responses to the menarche questions across both waves were transformed into person-period data, such that each girl had a binary observation for each discrete time interval beginning at birth. Girls who reported having not yet achieved menarche were assigned a zero, while girls who reported having achieved menarche were assigned a one for the specified interval. Girls who had not reached menarche by the end of data collection were censored. We dropped time intervals in which there was no variance, ultimately retaining intervals corresponding to between eight and 15 years of age.

As a result of these different approaches to handling pubertal data from boys and girls, it must be noted that the language around and interpretation of our findings will depend on sex. Specifically, whereas models involving male pubertal data describe effects on pubertal timing relative to other boys in this sample, models involving female pubertal data describe effects on “risk of” menarche due to our modeling approach (see Data Analysis below). These differences are reflected in our language. Pubertal timing, pubertal delay, and pubertal acceleration only apply to boys. For girls, the outcome is not acceleration or deceleration relative to peers, but rather increased or decreased risk of achieving menarche. When describing both boys and girls, we use the more general term, pubertal development.

Number of Years Since Leaving Syria—A single item measured the amount of time since participants had left Syria. Participants were able to report whether they had left Syria: 0–12 months ago (1), 12–24 months ago (2), 24–36 months ago (3), 36–48 months ago (4), or more than 48 months ago (5). While this is an ordinal variable, we elected to treat it as an interval variable because the lower-bound values are precisely 12 months apart and start at zero.

Data Analysis

Data cleaning was conducted primarily in IBM SPSS version 27 (IBM Corp., 2020). Multiple imputation and analytic models were completed using MPlus version 8.7 (Muthén & Muthén, 2022) using data in their original units (i.e., not mean-centered nor standardized). Height and weight data were collected on roughly 58% of the sample in year one, and

99% in year two. For the current study, we imputed the variables in the analytical model to preserve relations among variables (Nguyen et al., 2017), including interactions such as those between BMI-for-age (energetic stress) and war constructs (von Hippel, 2009). We imputed data for boys and girls separately, using several auxiliary variables including: Wave 2 measures of the imputed variables, age; hair hormone data (log transformed cortisol, testosterone, DHEA, and cortisol-DHEA ratios); transformed BMI-for-age, height-for-age, and weight-for-age; and weight, height, waist, and hip measures. We also included variables linked to resource scarcity, including family demographics, refugee environment, and the child's environmental sensitivity. Twenty imputed datasets were generated and used in structural models.

We conducted path analysis for boys and structural equation modeling for girls. All variables were observed for boys while for girls, pubertal development was a latent variable. This distinction was based on our approach to modeling for girls which used survival analysis; this approach is appropriate for time-to-event data and accommodates censored data. We used discrete time intervals because girls most often reported age at menarche in whole-year numbers, resulting in data "ties" which means that several girls share event times (i.e., achieving menarche at age 13, for instance). Discrete time survival mixture analysis treats event history variables as indicators of a latent class variable and is highly versatile (Muthén & Masyn, 2005).

As a primary objective of this paper is to examine whether patterns of pubertal development vary as a function of energetic stress, we conducted a series of nested model comparisons which tested interactions sequentially. Conventional model comparison statistics, such as the log likelihood ratio and chi-square difference test are unavailable for imputed data. Mplus uses the Wald X^2 test for nested model comparisons with imputed data (Asparouhov & Muthén, 2010). Thus, we took the following approach. First, models were tested with covariates only and all interactions set to zero (the restricted model). A single interaction parameter was freed and the path was tested using the Wald test. This procedure was completed for all interactions, and any significant interactions would be retained in the final model. We also conducted sensitivity tests to evaluate the robustness of our models by including the number of years since leaving Syria, in case our observations of relations between war and energetic stress could be explained by differences in temporal proximity to those exposures. That is, while years may have passed since the last war exposure in Syria, energetic stress would have been current at the time of data collection.

Data Availability

Researchers interested in accessing the data used in this study should contact Professor Michael Pluess at Queen Mary University of London, UK.

Preregistration—This study is not preregistered.

Results

Sample Characteristics

War Exposure—Examination of prevalence of specific war events revealed remarkable similarities across boys and girls (Table 2 below). For both boys and girls, the most experienced war event was being unable to go outside because of bullets or bombardment, followed by witnessing nearby explosions. It should be noted however, that this measure cannot distinguish among children who experienced a war event many times versus just once.

Descriptive statistics for the war constructs are shown in Table 3. Boys and girls experienced bombing and mortality events at similar rates, but an independent *t*-test revealed that boys experienced significantly more kidnapping events than girls ($t(1496.46) = 3.09$, $p = 002$). For both boys and girls, kidnapping and mortality were positively skewed (Figures S2–S4 in Supplemental Materials)

Resource Scarcity / Energetic Stress—Sociodemographic data indicate that participating Syrian refugee families face significant economic barriers (Tables S1–S2 in Supplemental Materials). For instance, 47.3% of families with male children and 47.4% of families with female children have income between zero to 15 USD (LBP 0–23,000) per week. Around a third of families receive cash assistance (33.4% for boys and 38.7% for girls) and nearly two-thirds receive food assistance (65% for boys and 63.9% for girls). The distributions and descriptive statistics for baseline BMI-for-age are displayed in Figure 2 below.

Pubertal Development—Table 4 below displays descriptive statistics about pubertal stages for boys and girls. As expected, age increased with each stage of puberty.

Because our measures of pubertal development consisted of fewer items than are typically used in the literature, we examined associations between pubertal development and other key variables expected to be associated with it, such as age, biometrics (weight, height, hip and waist circumference), and hormone levels, in order to increase confidence in our measures. These findings are shown in Table 5 below.

These analyses revealed strong associations between puberty items and biometric measures among both boys and girls. Only the association between pubertal stage and DHEA was non-significant in boys. Taken all together, we felt confident that our measures captured variation in pubertal development. With that said, we do acknowledge that using so few items of pubertal development is not ideal and may reduce the precision of our findings.

Analytical Models

Male Syrian Refugee Children—The models for pubertal timing in year one and year two were tested separately. To test whether boys facing energetic stress experience delayed puberty (Hypothesis I), we tested whether lower BMI-for-age predicted delayed pubertal timing. We found no association with pubertal timing in year one ($\beta = 0.00$, $SE = 0.03$, $p = 0.95$, *95% CI*: −0.06 – 0.06) nor in year two ($\beta = -0.05$, $SE = 0.04$, $p = 0.25$, *95% CI*: −0.13

– 0.03). Tabulation of these results are available in Table S4 of the Supplemental Materials. Taken all together, Hypothesis I was not supported in boys.

We then tested whether the effect of war exposure on pubertal timing depends on energetic stress (Hypotheses II and III). Tabulated results of these models are presented in Tables S4–S6 in the Supplemental Materials. The final model for boys, is displayed in Figure 3 below (tabulated in Table S5 in Supplemental Materials).

The results reveal that mortality significantly predicted pubertal timing acceleration in year one (β = 0.04, *SE* = 0.02, *p* = 0.05, *95% CI*: 0.00 – 0.08), as did the mortality*BMI-for-age interaction (β = 0.03, *SE* = 0.01, *p* = 0.02, *95% CI*: 0.01 – 0.06). BMI-for-age, bombing, and kidnapping were not associated with pubertal timing in year one. The overall model effect size for pubertal timing in year one was not significant (R^2 = 0.02, *p* = 0.14). We found no significant effects of any predictor, nor any interaction, for pubertal timing in year two.

In order to interpret the positive interaction between mortality and BMI-for-age, we plotted the interaction at different values of BMI-for-age ranging between −2 SD to +2 SD (Figure 4 below). Analysis of simple slopes revealed that among boys with BMI-for-age below zero (i.e., below average), there was no relationship between mortality exposure and pubertal timing. For boys with BMI-for-age average or above, mortality exposure significantly predicted pubertal timing acceleration, and the magnitude of its effect increases with higher BMI-for-age (Table S7 in Supplemental Materials). This finding aligns with both Hypotheses 2 and 3 which predicted an attenuated effect of war exposure for boys facing higher energetic stress, but not for boys facing lower energetic stress. Additionally, the theoretically specified mortality war construct was the only one to show this interaction, which is consistent with our prediction based on Life History Theory that children would be especially sensitive to cues of morbidity and mortality.

We also plotted the interaction using the Johnson-Neyman technique to reveal the regions of significance for the interaction (Figure S5 in Supplemental Materials). This analysis confirmed the results of our simple slopes analysis. Mortality exposure only accelerates pubertal timing when BMI-for-age is just about average or above (i.e., zero or above).

Female Syrian Refugee Children—To test whether girls facing higher energetic stress have a lower risk of menarche (Hypothesis I), we first tested a model with only BMI-for-age as a predictor. This test revealed a significant, positive relationship between BMI-for-age and risk of menarche (β = 0.27, *SE* = 0.07, *p* = <0.001; *95% CI*: 0.13 – 0.41) which supports our hypothesis (Table S8 in Supplemental Materials). That is, for a one standard deviation increase in BMI-for-age (because BMI-for-age is a standardized score), there is a 0.27 increase in log odds of menarche. As well, for a one standard deviation *decrease* in BMI-for-age, there is a 0.27 *decrease* in log odds of menarche, which aligns with our predictions. To make these findings more easily interpretable, we can easily convert log odds coefficients to the more familiar hazard odds ratio with the following hazard probability equation:

$$h(j) = exp(b)$$

Where h = hazard probability, j = time interval, and b = the log odds coefficient. Using this equation, we find that the hazard odds ratio is 1.31 which suggests that a one unit increase in BMI-for-age corresponds to 31% higher odds of achieving menarche,

To test whether the effect of war exposure on pubertal timing depends on energetic stress (Hypotheses II and III), we followed the same procedures as with boys. Results of these models are available in Tables S9–S10 in Supplemental Materials. The final model is displayed in Figure 5 below (tabulated in Table S11 in Supplemental Materials). The results reveal that BMI-for-age positively predicts girls' risk of menarche (β = 0.27, *SE* = 0.07, *p* = <0.01, *95% CI:* 0.12 – 0.41, *Hazard OR* = 1.31). Contrary to our expectations, there was no significant effect of mortality exposure, nor any significant effects of interactions between war constructs and BMI-for-age. However, time since leaving Syria showed a positive relationship (β = 0.36, *SE* = 0.02, *p* = 0.02, *95% CI:* 0.06 – 0.66, *Hazard OR* = 1.43) and a significant interaction emerged between bombing and time since leaving Syria (β = −0.10, *SE* = 0.05, *p* = 0.05, *95% CI:* −0.20 – −0.00, *Hazard OR* = 0.90). The overall model effect size was significant, but small in magnitude (R^2 = 0.06, p = 0.02).

To further probe the detected interaction, we tested the simple slopes and plotted the interaction. The tests of simple slopes, depicted in Table S11 in the Supplemental Materials, showed a significant, negative relationship between bombing and risk of menarche only for girls who left Syria four or more years ago (simple slopes β = −0.24, *SE* = 0.10, *p* = 0.02, *95% CI:* −0.44 – −0.07, *Hazard OR* = 0.79). As shown by the plot of the interaction below (Figure 6), the risk of menarche girls with more temporal proximity to the war (i.e., less time having passed since leaving Syria) was increased, but this effect was not significant. But for girls further away in time from the war, the effect of bombing decreased girls' risk of menarche.

General Discussion

Our study has revealed intriguing findings about pubertal development in war-affected refugee children living in low resource settings. We observed several novel findings with regard to unique effects of different kinds of war events and sex differences in response to these exposures. We also showed that energetic stress (BMI-for-age) can play a critical role in determining whether effects of war exposure impact pubertal development. As there is a lot to unpack, we begin by summarizing our results for boys followed by those for girls. Then we will take an eagle-eye view to explain our interpretations of the patterns we observed.

Our tests of the effects of war exposure and energetic stress on pubertal timing in boys revealed several key findings. First, we did not find support for Hypothesis I which predicted that energetic stress delays pubertal timing in boys. At the same time, our tests of Hypotheses II and III indicate that energetic stress does in fact impact pubertal development in year one, particularly by moderating the effects of morbidity/mortality exposures on pubertal timing. This interaction showed that the effects of morbidity/mortality threats on pubertal timing in year one are attenuated under conditions of elevated energetic stress. In the absence of energetic stress, we find that exposure to morbidity/mortality threats

accelerates pubertal timing, as is commonly reported in the pubertal timing literature. That this finding emerged with specific respect to the morbidity/mortality war construct, but not the bombing or kidnapping constructs, is consistent with our expectations that morbidity/mortality threats are especially salient in terms of their influence on pubertal timing, as predicted by Life History Theory.

However, by year two, all of the observed effects were absent. Pubertal timing in year two was not predicted by any war exposure construct, nor by any interaction of war with energetic stress. One possible explanation is that pubertal development "caught up" by year two. Recall that pubertal timing is a standardized *z*-score which reflects boys' pubertal stage regressed on age. Thus, pubertal timing is relative to the child's age and relative to the sample. In year one, boys exposed to more morbidity/mortality-related war events had, on average, higher pubertal timing *z*-scores than boys exposed to fewer such events. For pubertal timing in year two, we recalculated boys' pubertal timing for their age in year two. The results for year two indicate that the pubertal timing *z*-scores did not vary as a function of war, energetic stress, or their interactions. That is, by year two, boys exposed to morbidity/mortality war events did not particularly stand out relative to their peers.

The different results across years of data collection suggest that effects of war exposure on pubertal timing in boys depend on when the data were collected. This may mean that temporal proximity to war exposure is important for detecting effects on pubertal timing. However, we did conduct sensitivity analyses with an item which measured the number of years since the boys had left Syria and we found no effect, neither directly on pubertal timing, nor as a moderator of the effects of war constructs on pubertal timing. In other words, we tested whether the effects of war depended on how recently the exposure occurred and did not find any indication that this might influence our results. One possibility for these disparate findings is that the single item is a poor measure of temporal proximity, at least with respect to pubertal timing in boys. Either way, future research investigating effects of adverse exposures on pubertal development should consider the possibility that temporal proximity to events may influence findings.

Our findings for Syrian refugee girls revealed a completely different pattern of effects. For one, we observed a clear relationship between energetic stress and girls' risk of menarche. As we predicted, girls facing higher energetic stress are less likely to experience menarche. However, none of our subsequent tests revealed any interactions between war constructs and BMI-for-age as we expected. In fact, the only other significant effect for girls, besides the direct effect of BMI-for-age, was a significant interaction between bombing exposure and time since the girls had left Syria. Our follow up tests to probe this significant interaction indicated that the effect of bombing predicted a lower probability of menarche, but only for girls who left Syria four or more years prior to data collection. Not only is the direction of the effect of war exposure in contrast to our expectations, but only 12% of Syrian refugee girls had left Syria more than four years prior to data collection (according to baseline data), meaning that 88% of girls are not well-characterized by this interaction.

We think one possible explanation for the different findings in boys and girls is that they were influenced by our measures of pubertal development. In particular, we were able to

calculate pubertal timing at two time points in boys and these models revealed significant effects on pubertal timing in year one, but not year two. In contrast, for girls, our best use of the data was to conduct discrete time survival analysis. To do so, we had to transform our data to create person-period variables which identified whether girls had achieved menarche or not by the end of the study. In other words, girls' pubertal development is characterized by survival (or risk of menarche) by year two. It is possible that the results may have been different if we had measured girls' pubertal stages with greater granularity and over multiple time points. Achieving menarche distinguishes girls in the later Tanner stages (4–5) from those in the earlier Tanner stages (1–3); however, further distinction is not possible. Furthermore, menarche only occurs once, further limiting its utility in measuring change over time. We do think that the possibility that the observed sex differences are due to measurement highlights a need for future research involving longitudinal measures of pubertal development. When such measures are available over multiple time points (in boys and/or girls), studies tend to focus on pubertal tempo (e.g., Hamlat et al., 2022; Sumner et al., 2023). Pubertal tempo refers to the pace of change in pubertal development, while pubertal timing focuses on the relative onset of puberty and/or relative timing of achieving different pubertal stages (Mendle et al., 2010). Understanding relations between these constructs requires further study. Some investigations involving both constructs can produce congruent results (e.g., earlier timing is associated with faster tempo; e.g., Ellis et al., 2011; Kowalski et al., 2021), but in many cases the findings are more complex (e.g., Beltz et al., 2014; Hamlat et al., 2022; Negriff et al., 2015). Ultimately, additional research is necessary to fully characterize how pubertal development changes over time following adverse exposures, as well as how these changes impact mental and physical health, as well as other outcomes, later in life.

Another possibility is that the results are due to sex differences in reporting on pubertal development. Our measures of pubertal development are likely to reflect Syrian refugee children's knowledge and understanding of the pubertal transition; however their access to reproductive health education is likely influenced by culture, gender, and religion. In general, the Western Asia and North African (WANA) region is characterized by conservative sociocultural norms that discourage youth-friendly sexual and reproductive health services and information (Gausman et al., 2019). Parents, teachers, and healthcare providers who might typically provide information on puberty in a Western context are ill-equipped to do so in communities with very conservative views of sexuality (DeJong & El-Khoury, 2006). There may be other reasons for under-reporting, particularly among girls for whom menarche potentially accompanies child marriage, early pregnancy, gender-based violence, and sexual assault, all of which are compounded by stigma and discrimination (El Ayoubi et al., 2021; Fahme et al., 2021).

Indeed, Syrian refugee adolescent girls in Lebanon report menarche as shocking, scary, and unexpected, with varying prior knowledge and access to family members who could help (El Ayoubi et al., 2021). Half of those studied were unfamiliar with its function, while the others recognized it as a sign of puberty and transition from childhood to womanhood. Nevertheless, the majority of girls were able to describe physical, personal, and social changes that occur with puberty (Korrie et al., 2021). In comparison to girls of the same age, fewer Syrian refugee boys aged 15–24 had ever discussed sexual and reproductive health

(41% versus 30%, respectively; Chahine et al., 2014). However, male adolescents are able to describe the physical changes accompanying puberty, such as hair growth, but desire more information (DeJong et al., 2017). For boys, puberty is perceived as being associated with transitioning to adulthood, sexual experimentation, moving away from parents, pregnancy, and childbirth. Although it is not entirely without stigma, as aspects of pubertal development including masturbation and sperm are considered socially unacceptable and shameful (Gausman et al., 2021), self-disclosures about one's own development are still likely to be less threatening for boys compared to girls.

Strengths and Limitations

The BIOPATH study from which our data are drawn is highly unique in several respects, thereby lending itself particularly well to the hypotheses tested in this paper. It is one of very few studies which collected data from Syrian refugee families contemporaneously with the Syrian Civil War. The sample of 1,600 refugee child-caregiver dyads living in temporary settlements in Lebanon is highly unique, as are the quality of the measures which include validated, self-report measures of a number of individual and environmental traits as well as biometric measures, hair hormones, and saliva samples. Refugees tend to be fairly mobile which can pose challenges for longitudinal data collection, but because saliva samples were collected and used in genome-wide association analysis, we were able to confirm that we were indeed collecting data from the same child over the two years of data collection. Additionally, to our knowledge, BIOPATH is the only study which has measured characteristics of puberty concurrently with development in a large sample of war-affected children who were displaced at the time of data collection.

The primary limitation of this study is our measurement of pubertal development, which consisted of just two items each for boys and girls. Our selection of items was determined by our need to use caution in how certain questions were asked due to strong cultural and religious boundaries around sexuality and reproductive health. Although we did find strong associations between these items and other measures of growth and development, our findings are likely to lack precision. For instance, we could not actually measure whether boys were post-pubertal due to the number of items available. For girls, we were unable to detect earlier stages of puberty at all as menarche typically occurs around Tanner Stage 4 (Marshall & Tanner, 1969). Future research investigating pubertal development, particularly in vulnerable groups and/or in low resource settings, should aim to collect as much information about pubertal development as possible. For example, some versions of the Pubertal Development Scale for girls include items asking about changes in height, axillary hair growth, and skin changes in addition to questions about breast growth and age at menarche (Petersen et al., 1988). To the extent possible, these items should be evaluated for fit with the population under investigation.

Future Directions

It is critical for future research on pubertal development to sample from more diverse settings. As demonstrated here, the patterns of pubertal development we identified in war-affected refugee children depart from those which are commonly found in populations living in more stable, higher resource settings. For children in the latter settings (upon whom

the majority of pubertal development research is based), threat exposures typically predict earlier and faster pubertal development, leading some to propose pubertal screening among medical and mental health providers in order to identify trauma-exposed youth (e.g., Colich et al., 2019; McLaughlin et al., 2020). However, our findings show that this recommendation would miss trauma-exposed youth who also face energetic stress. Indeed, resource scarcity in W.E.I.R.D. settings (Henrich et al., 2010) is unlikely to reach the severity that might be present in, for example, LMICs. More than 90% of the world's adolescents reside in LMICs (United Nations, 2022) but these youth are poorly characterized by the extant literature. Remediating this gap is not just a scientific endeavor, but also an ethical and humanitarian one. As we previously noted, children and adolescents in LMICs are likely to experience far more concentrated adversity, far more severe poverty, and have far less access to mental health services than youth in higher income countries. An increasing number of Western scholars are proposing that pubertal development may be a transdiagnostic mechanism (i.e., a core process linking adversity to poor outcomes) for psychopathology (e.g.,Colich et al., 2020; Hamlat et al., 2019; Mendle et al., 2020). Accumulating evidence in this area is providing the basis for critical research to identify targets for intervention in order to prevent psychopathology in trauma-affected youth. However, we think it would be remiss if these efforts failed to benefit the most vulnerable among us.

Conclusion

Exposure to trauma during childhood and adolescence is often linked to accelerated pubertal development in populations living in higher resource settings. This body of evidence is increasingly used to advocate for pubertal screening to identify trauma-affected youth and intervene in the development of psychopathology. We investigated how war and displacement impacted pubertal development in Syrian refugee children, predicting that energetic stress (nutrition deprivation) attenuates effects of war exposure on pubertal development. In Syrian refugee boys, war events linked to morbidity/mortality threats predicted accelerated pubertal timing. As predicted, this pattern is attenuated in boys facing elevated energetic stress. In Syrian refugee girls, elevated energetic stress decreased risk of menarche. Additionally, exposure to war events characterized by bombing and explosions predicted decreased risk of menarche, but only for girls who had left Syria more than four years prior to data collection. All together, these findings reveal that expected effects of trauma on pubertal development may be obscured under conditions of elevated resource scarcity. Further research is needed which investigates pubertal development in non-Western settings, particularly in LMICs where more than 90% of the world's children and adolescents live.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability Statement

Researchers interested in accessing data should contact Professor Michael Pluess at Queen Mary University of London, UK (m.pluess@qmul.ac.uk).

References

Akresh R, Bhalotra S, Leone M, & Osili UO (2012a). War and stature: Growing up during the Nigerian civil war. American Economic Review, 102(3), 273–77.

Akresh R, Verwimp P, & Bundervoet T (2011). Civil war, crop failure, and child stunting in Rwanda. Economic Development and Cultural Change, 59(4), 777–810.

Akresh R, Lucchetti L, & Thirumurthy H (2012). Wars and child health: Evidence from the Eritrean–Ethiopian conflict. Journal of Development Economics, 99(2), 330–340. [PubMed: 22962514]

Angell-Andersen E, Tretli S, Bjerknes R, Forsen T, Sørensen TIA, Eriksson JG, ... & Grotmol T (2004). The association between nutritional conditions during World War II and childhood anthropometric variables in the Nordic countries. Annals of Human Biology, 31(3), 342–355. [PubMed: 15204349]

Asparouhov T, & Muthén B (2010). Chi-square statistics with multiple imputation. Retrieved from http://statmodel.com/download/MI7.pdf on August 2, 2022.

Belsky J (2008). War, trauma and children's development: Observations from a modern evolutionary perspective. International Journal of Behavioral Development, 32(4), 260–271.

Belsky J, & Pluess M (2009). Beyond diathesis stress: differential susceptibility to environmental influences. Psychological Bulletin, 135(6), 885. [PubMed: 19883141]

Belsky J, Steinberg L, & Draper P (1991). Childhood Experience, Interpersonal Development, and Reproductive Strategy: An Evolutionary Theory of Socialization. Child Development, 62(4), 647–670. 10.1111/j.1467-8624.1991.tb01558.x [PubMed: 1935336]

Beltz AM, Corley RP, Bricker JB, Wadsworth SJ, & Berenbaum SA (2014). Modeling pubertal timing and tempo and examining links to behavior problems. Developmental Psychology, 50(12), 2715. [PubMed: 25437757]

Binder AM, Corvalan C, Mericq V, Pereira A, Santos JL, Horvath S, Shepherd J, & Michels KB (2018). Faster ticking rate of the epigenetic clock is associated with faster pubertal development in girls. Epigenetics, 13(1), 85–94. [PubMed: 29235933]

Boyne MS, Thame M, Osmond C, Fraser RA, Gabay L, Reid M, & Forrester TE (2010). Growth, body composition, and the onset of puberty: Longitudinal observations in Afro-Caribbean children. The Journal of Clinical Endocrinology & Metabolism, 95(7), 3194–3200. [PubMed: 20427487]

Buyken AE, Karaolis-Danckert N, & Remer T (2009). Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers. The American journal of clinical nutrition, 89(1), 221–230. [PubMed: 19056586]

Bygdell M, Kindblom JM, Jansson J-O, & Ohlsson C (2020). Revisiting the critical weight hypothesis for regulation of pubertal timing in boys. The American Journal of Clinical Nutrition, 113(1), 123–128. 10.1093/ajcn/nqaa304

Chahine A, Al-Masri M, Samra SA, & Abla Z (2014). Situation Analysis of Youth in Lebanon Affected by the Syrian Crisis: April, 2014. United Nations Population Fund.

Clarkin PF (2012). War, forced displacement and growth in Laotian adults. Annals of Human Biology, 39(1), 36–45. [PubMed: 22149011]

Colich NL, Platt JM, Keyes KM, Sumner JA, Allen NB, & McLaughlin KA (2020). Earlier age at menarche as a transdiagnostic mechanism linking childhood trauma with multiple forms of psychopathology in adolescent girls. Psychological Medicine, 50(7), 1090–1098. 10.1017/s0033291719000953 [PubMed: 31020943]

Colich NL, Rosen ML, Williams ES, & McLaughlin KA (2020). Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. Psychological Bulletin, 146(9), 721. [PubMed: 32744840]

DeJong J, & El-Khoury G (2006). Reproductive health of Arab young people. The BMJ, 333(7573), 849–851. [PubMed: 17053245]

El Ayoubi LEL, Abdulrahim S, & Sieverding M (2021). Sexual and reproductive health information and experiences among Syrian refugee adolescent girls in Lebanon. Qualitative Health Research, 31(5), 983–998. [PubMed: 33733937]

Ellis BJ, Figueredo AJ, Brumbach BH, & Schlomer GL (2009). Fundamental Dimensions of Environmental Risk. Human Nature, 20(2), 204–268. [PubMed: 25526958]

Ellis BJ, Sheridan MA, Belsky J, & McLaughlin KA (2022). Why and how does early adversity influence development? Toward an integrated model of dimensions of environmental experience. Development and Psychopathology.

Ellis BJ, Shirtcliff EA, Boyce WT, Deardorff J, & Essex MJ (2011). Quality of early family relationships and the timing and tempo of puberty: Effects depend on biological sensitivity to context. Development and Psychopathology, 23(1), 85–99. [PubMed: 21262041]

Fahme SA, Sieverding M, & Abdulrahim S (2021). Sexual and reproductive health of adolescent Syrian refugee girls in Lebanon: A qualitative study of healthcare provider and educator perspectives. Reproductive Health, 18(1), 1–16. [PubMed: 33388066]

Frisch RE, & Revelle R (1970). Height and Weight at Menarche and a Hypothesis of Critical Body Weights and Adolescent Events. Science, 169(3943), 397–399. [PubMed: 5450378]

Frisch RE, & Revelle R (1971). Height and weight at menarche and a hypothesis of menarche. Archives of Disease in Childhood, 46(249), 695. [PubMed: 5118059]

Gausman J, Othman A, Hamad IL, Dabobe M, Daas I, & Langer A (2019). How do Jordanian and Syrian youth living in Jordan envision their sexual and reproductive health needs? A concept mapping study protocol. BMJ open, 9(1), e027266.

Gausman J, Othman A, Otoom M, Shaheen A, & Langer A (2019). Youth as navigators: A study protocol to incorporate narrative and visual methods into research on adolescent sexual and gender development among Syrian and Jordanian Youth. International Journal of Qualitative Methods, 18, 1–8.

Gettler LT, McDade TW, Bragg JM, Feranil AB, & Kuzawa CW (2015). Developmental energetics, sibling death, and parental instability as predictors of maturational tempo and life history scheduling in males from Cebu, Philippines. American Journal of Physical Anthropology, 158(2), 175–184. 10.1002/ajpa.22783 [PubMed: 26239159]

Del Giudice M (2020). Rethinking the fast-slow continuum of individual differences. Evolution and Human Behavior, 41(6), 536–549. 10.1016/j.evolhumbehav.2020.05.004

Gur RE, Moore TM, Rosen AF, Barzilay R, Roalf DR, Calkins ME, ... & Gur RC (2019). Burden of environmental adversity associated with psychopathology, maturation, and brain behavior parameters in youths. JAMA Psychiatry, 76(9), 966–975. [PubMed: 31141099]

Hamlat EJ, Prather AA, Horvath S, Belsky J, & Epel ES (2021). Early life adversity, pubertal timing, and epigenetic age acceleration in adulthood. Developmental Psychobiology, dev.22085. 10.1002/dev.22085

Hamlat EJ, Laraia B, Bleil ME, Deardorff J, Tomiyama AJ, Mujahid M, ... & Epel ES (2022). Effects of early life adversity on pubertal timing and tempo in Black and White girls: The National Growth and Health Study. Psychosomatic Medicine, 84(3), 297–305. [PubMed: 35067653]

Hayes AF (2022). Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. Guilford publications.

Henrich J, Heine SJ, & Norenzayan A (2010). The weirdest people in the world? Behavioral and Brain Sciences, 33(2–3), 61–83. 10.1017/s0140525×0999152x [PubMed: 20550733]

IBM Corp. (2020). IBM SPSS Statistics for Windows, Version 27.0. IBM Corp.

Joos CM, Wodzinski AM, Wadsworth ME, & Dorn LD (2018). Neither antecedent nor consequence: Developmental integration of chronic stress, pubertal timing, and conditionally adapted stress response. Developmental Review, 48, 1–23.

Karam EG, Al-Atrash R, Saliba S, Melhem N, & Howard D (1999). The War Events Questionnaire. Social Psychiatry and Psychiatric Epidemiology, 34(5), 265–274. [PubMed: 10396169]

Korri R, Hess S, Froeschl G, & Ivanova O (2021). Sexual and reproductive health of Syrian refugee adolescent girls: a qualitative study using focus group discussions in an urban setting in Lebanon. Reproductive Health, 18(1), 1–17. [PubMed: 33388066]

Kowalski AJ, Addo OY, Kramer MR, Martorell R, Norris SA, Waford RN, ... & Stein AD (2021). Longitudinal associations of pubertal timing and tempo with adolescent mental health and risk behavior initiation in urban South Africa. Journal of Adolescent Health, 69(1), 64–73.

Kyweluk MA, Georgiev AV, Borja JB, Gettler LT, & Kuzawa CW (2018). Menarcheal timing is accelerated by favorable nutrition but unrelated to developmental cues of mortality or familial instability in Cebu, Philippines. Evolution and Human Behavior, 39(1), 76–81. 10.1016/j.evolhumbehav.2017.10.002

Li W, Liu Q, Deng X, Chen Y, Liu S, & Story M (2017). Association between obesity and puberty timing: A systematic review and meta-analysis. International Journal of Environmental Research and Public Health, 14(10), 1266. [PubMed: 29064384]

Liczbińska G, Czapla Z, Piontek J, & Malina RM (2018). Age at menarche in Polish University students born before, during and after World War II: Economic effects. Economics & Human Biology, 28, 23–28. [PubMed: 29197239]

Marsh HW, Nagengast B, & Morin AJS (2013). Measurement Invariance of Big-Five Factors Over the Life Span: ESEM Tests of Gender, Age, Plasticity, Maturity, and La Dolce Vita Effects. Developmental Psychology, 49(6), 1194–1218. [PubMed: 22250996]

Marshall WA, & Tanner JM (1969). Variations in pattern of pubertal changes in girls. Archives of Disease in Childhood, 44(235), 291. [PubMed: 5785179]

McEwen FS, Popham C, Moghames P, Smeeth D, Villiers B. de, Saab D, Karam G, Fayyad J, Karam E, & Pluess M (2022). Cohort Profile: Biological Pathways of Risk and Resilience in Syrian Refugee Children (BIOPATH). Social Psychiatry and Psychiatric Epidemiology. 10.1007/s00127-022-02228-8

McLaughlin KA, Colich NL, Rodman AM, & Weissman DG (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. BMC Medicine, 18(1), 1–11. [PubMed: 31898501]

Mendle J, Beam CR, McKone KM, & Koch MK (2020). Puberty and transdiagnostic risks for mental health. Journal of Research on Adolescence, 30(3), 687–705. [PubMed: 32109337]

Mendle J, Harden KP, Brooks-Gunn J, & Graber JA (2010). Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. Developmental Psychology, 46(5), 1341. [PubMed: 20822243]

Mendle J, Leve LD, Van Ryzin M, Natsuaki MN, & Ge X (2011). Associations between early life stress, child maltreatment, and pubertal development among girls in foster care. Journal of Research on Adolescence, 21(4), 871–880. [PubMed: 22337616]

Muthén B & Masyn K (2005). Discrete-time survival mixture analysis. Journal of Educational and Behavioral Statistics, 30, 27–58.

Muthén LK, & Muthén BO (n.d.). Mplus User's Guide. Eighth Edition. Muthén & Muthén.

Neberich W, Penke L, Lehnart J, & Asendorpf JB (2010). Family of origin, age at menarche, and reproductive strategies: A test of four evolutionary-developmental models. European Journal of Developmental Psychology, 7(2), 153–177.

Negriff S, Blankson AN, & Trickett PK (2015). Pubertal timing and tempo: Associations with childhood maltreatment. Journal of Research on Adolescence, 25(2), 201–213. [PubMed: 26146470]

Nguyen CD, Carlin JB, & Lee KJ (2017). Model checking in multiple imputation: an overview and case study. Emerging Themes in Epidemiology, 14(1), 1–12. [PubMed: 28184234]

Ohlsson C, Gidestrand E, Bellman J, Larsson C, Palsdottir V, Hägg D, Jansson P-A, & Jansson J-O (2020). Increased weight loading reduces body weight and body fat in obese subjects – A proof of concept randomized clinical trial. EClinicalMedicine, 22, 100338. [PubMed: 32510046]

Pesonen AK, Räikkönen K, Heinonen K, Kajantie E, Forsén T, & Eriksson JG (2008). Reproductive traits following a parent–child separation trauma during childhood: a natural experiment during World War II. American Journal of Human Biology, 20(3), 345–351. [PubMed: 18257076]

Petersen AC, Crockett LJ, Richards MH, & Boxer AM (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence, 17(2), 117–133. 10.1007/bf01537962 [PubMed: 24277579]

Pluess M (2015). Individual Differences in Environmental Sensitivity. Child Development Perspectives, 9(3), 138–143. 10.1111/cdep.12120

Pluess M, Assary E, Lionetti F, Lester KJ, Krapohl E, Aron EN, & Aron A (2018). Environmental sensitivity in children: Development of the Highly Sensitive Child Scale and identification of sensitivity groups. Developmental psychology, 54(1), 51. [PubMed: 28933890]

Prebeg Ž, & Bralić I (2000). Changes in menarcheal age in girls exposed to war conditions. American Journal of Human Biology, 12(4), 503–508. [PubMed: 11534042]

Schneider EB, Ogasawara K, & Cole TJ (2021). Health Shocks, Recovery, and the First Thousand Days: The Effect of the Second World War on Height Growth in Japanese Children. Population and Development Review, 47(4), 1075–1105.

Scott EC, & Johnston FE (1982). Critical fat, menarche, and the maintenance of menstrual cycles A critical review. Journal of Adolescent Health Care, 2(4), 249–260. [PubMed: 6749778]

Smeeth D, McEwen FS, Popham CM, Karam E, Fayyad J, Saab D, Rieder MJ, Elzagallaai AA, van Uum S, & Pluess. M. (2023). War exposure, post-traumatic stress symptoms, and hair cortisol concentrations in Syrian refugee children. Molecular Psychiatry, 28, 647–656. [PubMed: 36385169]

Song S, & Shaheen M (2013, August). Assessing the impact of violence and war on youth in low-and middle-income countries. In Child & Youth Care Forum (Vol. 42, No. 4, pp. 275–283). Springer US.

Stearns SC (1992). The Evolution of Life Histories. Oxford University Press.

Suarez A, Lahti J, Czamara D, Lahti-Pulkkinen M, Girchenko P, Andersson S, Strandberg TE, Reynolds RM, Kajantie E, Binder EB, & Raikkonen K (2018). The epigenetic clock and pubertal, neuroendocrine, psychiatric, and cognitive outcomes in adolescents. Clinical Epigenetics, 10(1), 96. 10.1186/s13148-018-0528-6 [PubMed: 30021623]

Sumner JA, Gao X, Gambazza S, Dye CK, Colich NL, Baccarelli AA, ... & McLaughlin KA (2023). Stressful life events and accelerated biological aging over time in youths. Psychoneuroendocrinology, 106058. [PubMed: 36827906]

Stride CB, Gardner S, Catley N & Thomas F (2015) ‘Mplus code for mediation, moderation, and moderated mediation models’, http://www.offbeat.group.shef.ac.uk/FIO/mplusmedmod.htm. Retrieved February 26, 2023.

Sumner JA, Colich NL, Uddin M, Armstrong D, & McLaughlin KA (2019). Early Experiences of Threat, but Not Deprivation, Are Associated With Accelerated Biological Aging in Children and Adolescents. Biological Psychiatry, 85(3), 268–278. [PubMed: 30391001]

Tahirović HF (1998). Menarchal age and the stress of war: an example from Bosnia. European Journal of Pediatrics, 157(12), 978–980. [PubMed: 9877035]

Tither JM, & Ellis BJ (2008). Impact of fathers on daughters’ age at menarche: A genetically and environmentally controlled sibling study. Developmental Psychology, 44(5), 1409. [PubMed: 18793072]

Trivers RL (1974). Parent-offspring conflict. Integrative and Comparative Biology, 14(1), 249–264.

United Nations (2022). World population prospects: The 2022 revision. Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat, New York.

United Nations High Commissioner for Refugees (2022, August 15). Figures at a Glance. https://www.unhcr.org/uk/figures-at-a-glance.html

Van Noord PAH, & Kaaks RV (1991). The effect of wartime conditions and the 1944–45 ‘Dutch famine’on recalled menarcheal age in participants of the DOM breast cancer screening project. Annals of Human Biology, 18(1), 57–70. [PubMed: 2009006]

Von Hippel PT (2009). 8. How to impute interactions, squares, and other transformed variables. Sociological Methodology, 39(1), 265–291.

World Health Organization (Ed.). (2006). WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: methods and development. World Health Organization.

Public Significance Statement

- Exposure to war and displacement in childhood is a known risk factor for poor health outcomes; however, few studies characterize how these experiences impact pubertal development. We investigated how exposure to war impacts pubertal development in Syrian refugee children living in temporary camps in Lebanon.
- Energetic stress, or nutrition deprivation, can attenuate effects of war exposure on pubertal development, which limits the utility of solely screening for pubertal development to identify trauma-exposed youth. Puberty research conducted in low resource settings must account for energetic stress when investing how adverse exposures impact puberty.
- Findings from this study highlight a critical need for longitudinal research on pubertal development, especially in low- and middle-income countries where >90% of adolescents live, to fully characterize how pubertal development is impacted by adverse experiences.

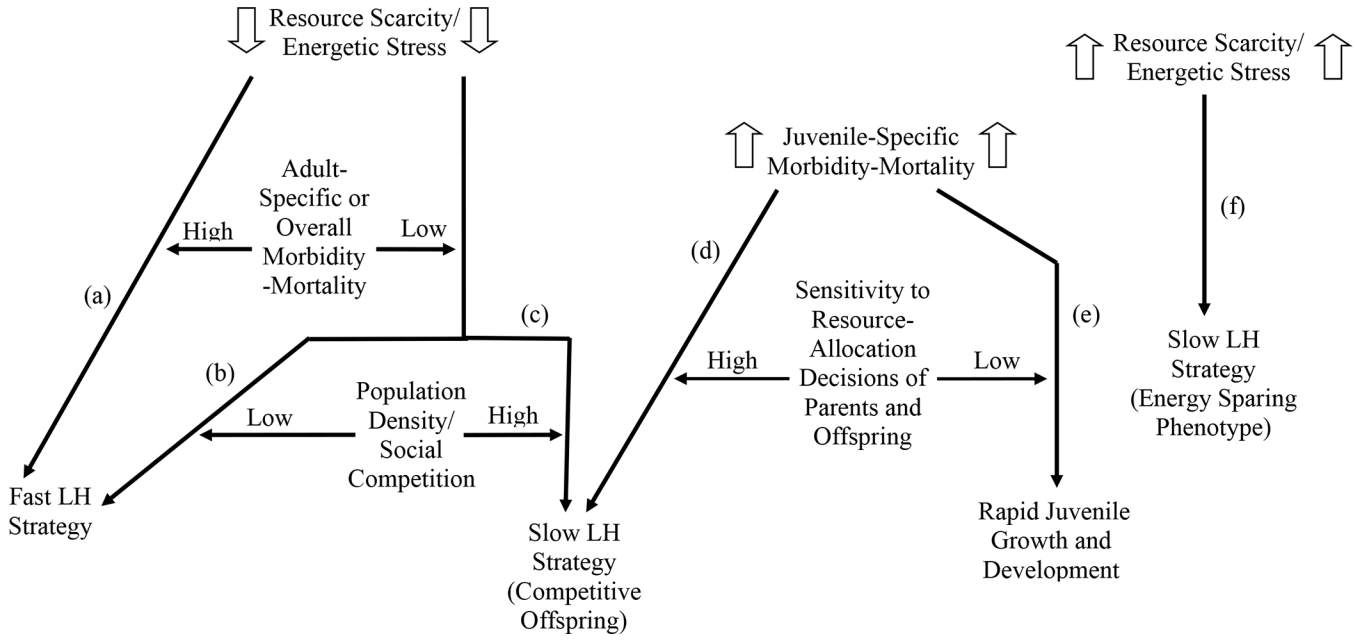

Figure 1.

Impact of environmental harshness on developmental trajectories. LH = life history. Modified with permission from Ellis et al., 2009.

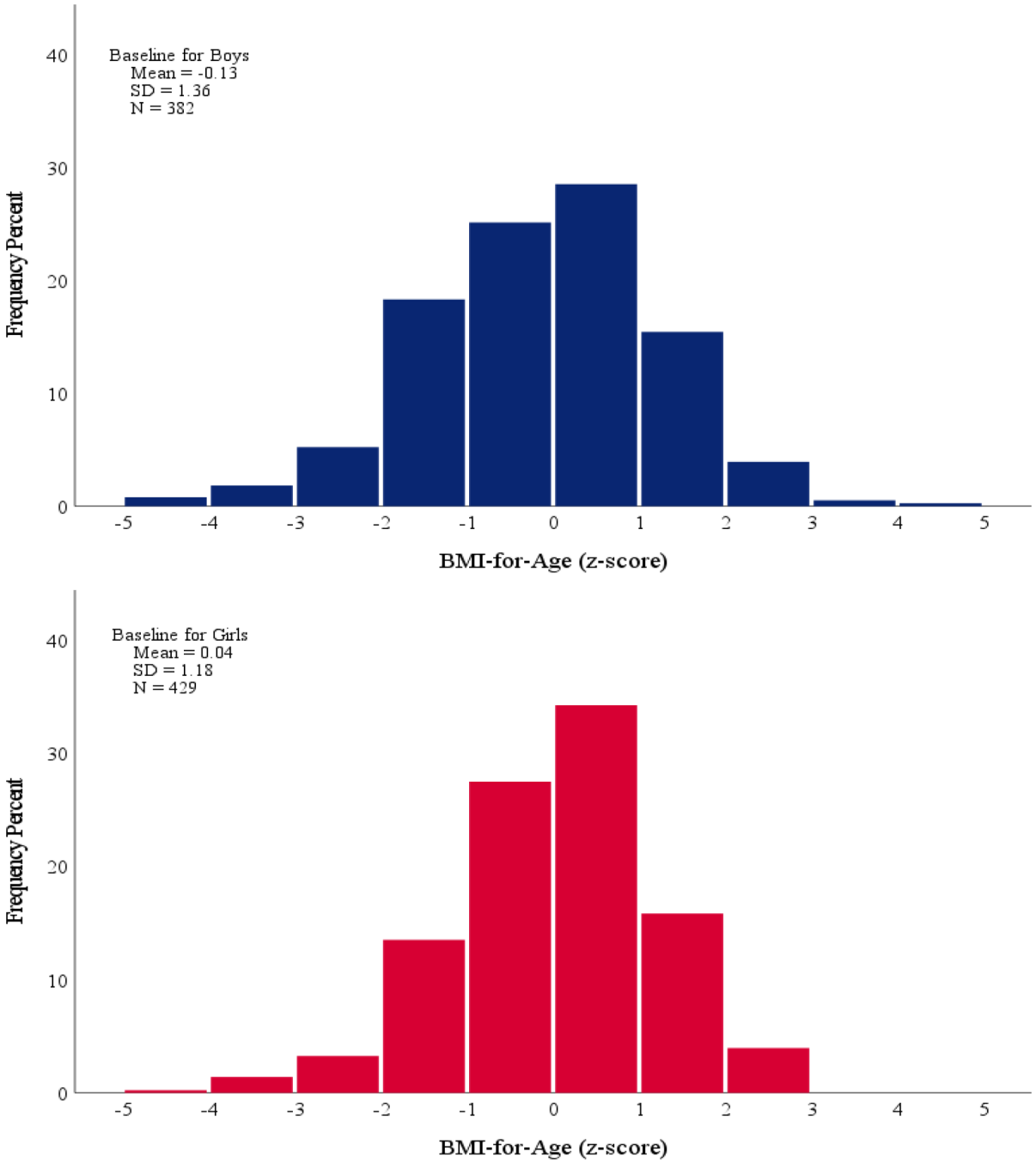

Figure 2.
BMI-for-age distributions and baseline descriptive statistics for boys (blue) and girls (red).

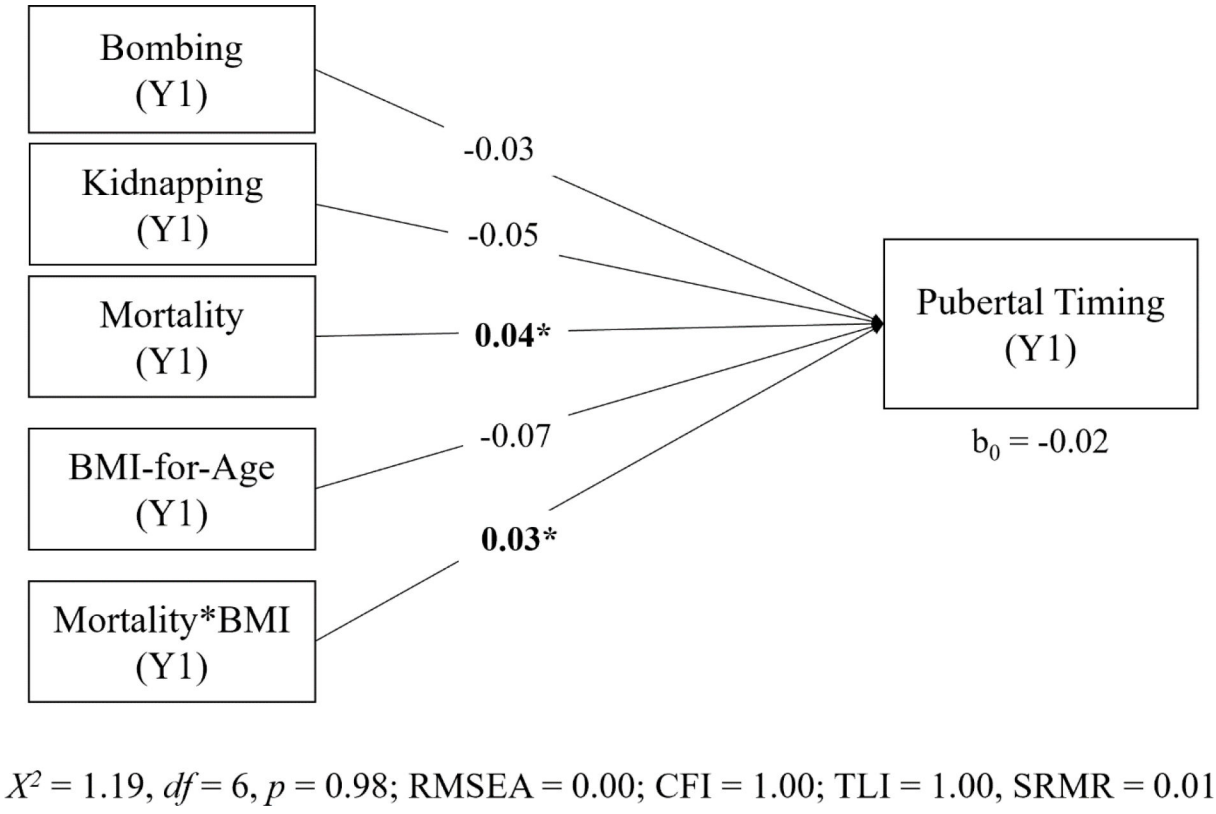

Figure 3.
Final model showing effects of war exposure and energetic stress on pubertal timing in boys.

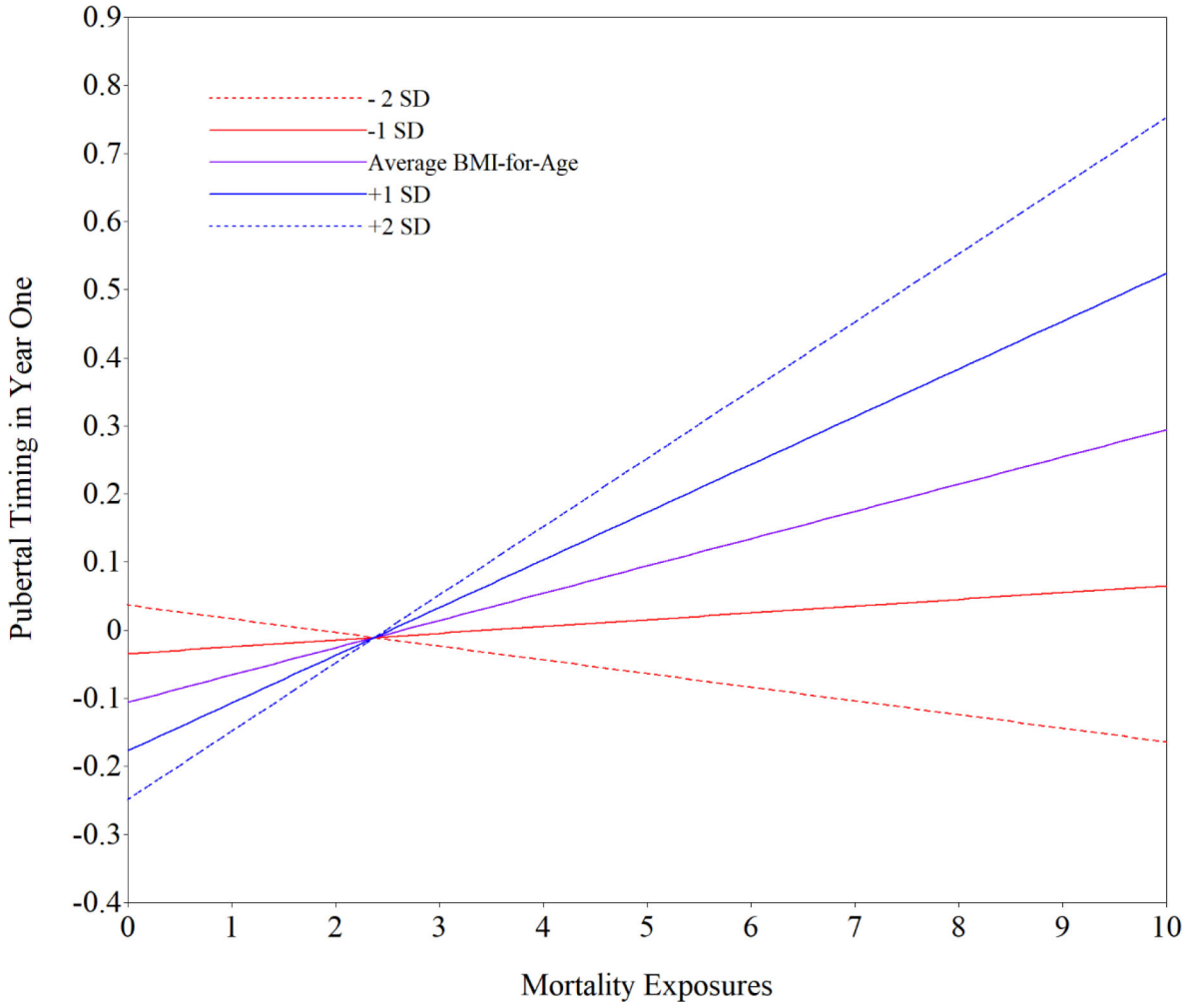

Figure 4.
Effect of mortality*BMI-for-age interaction on boys' pubertal timing in year one.

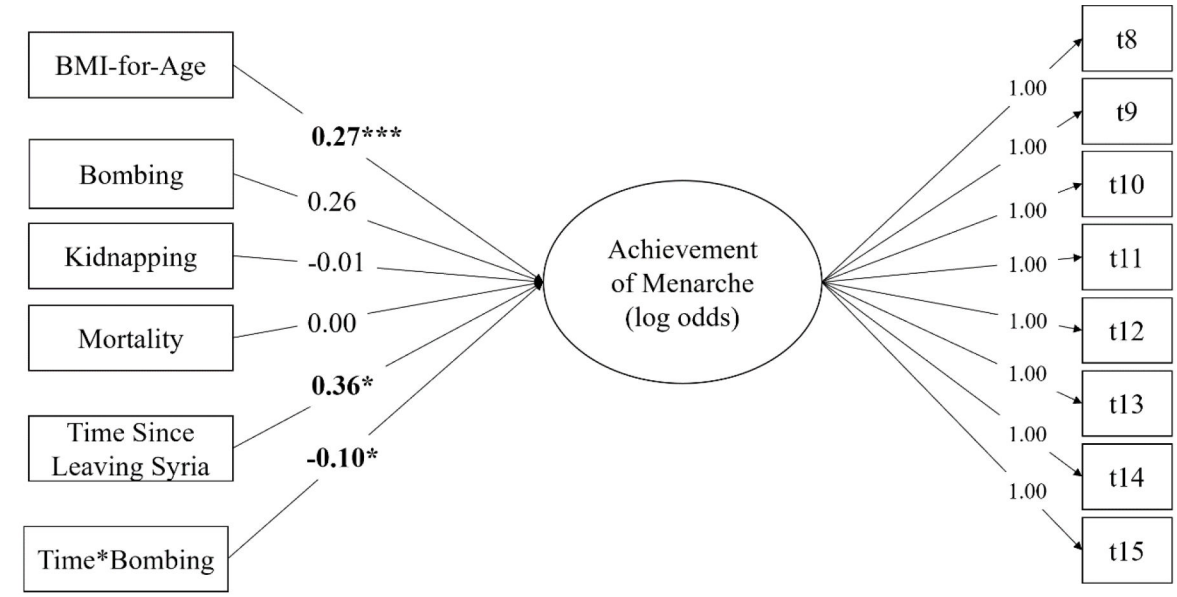

Figure 5.

Final model showing effects of war exposure, energetic stress, and time since leaving Syria on risk of menarche in girls.

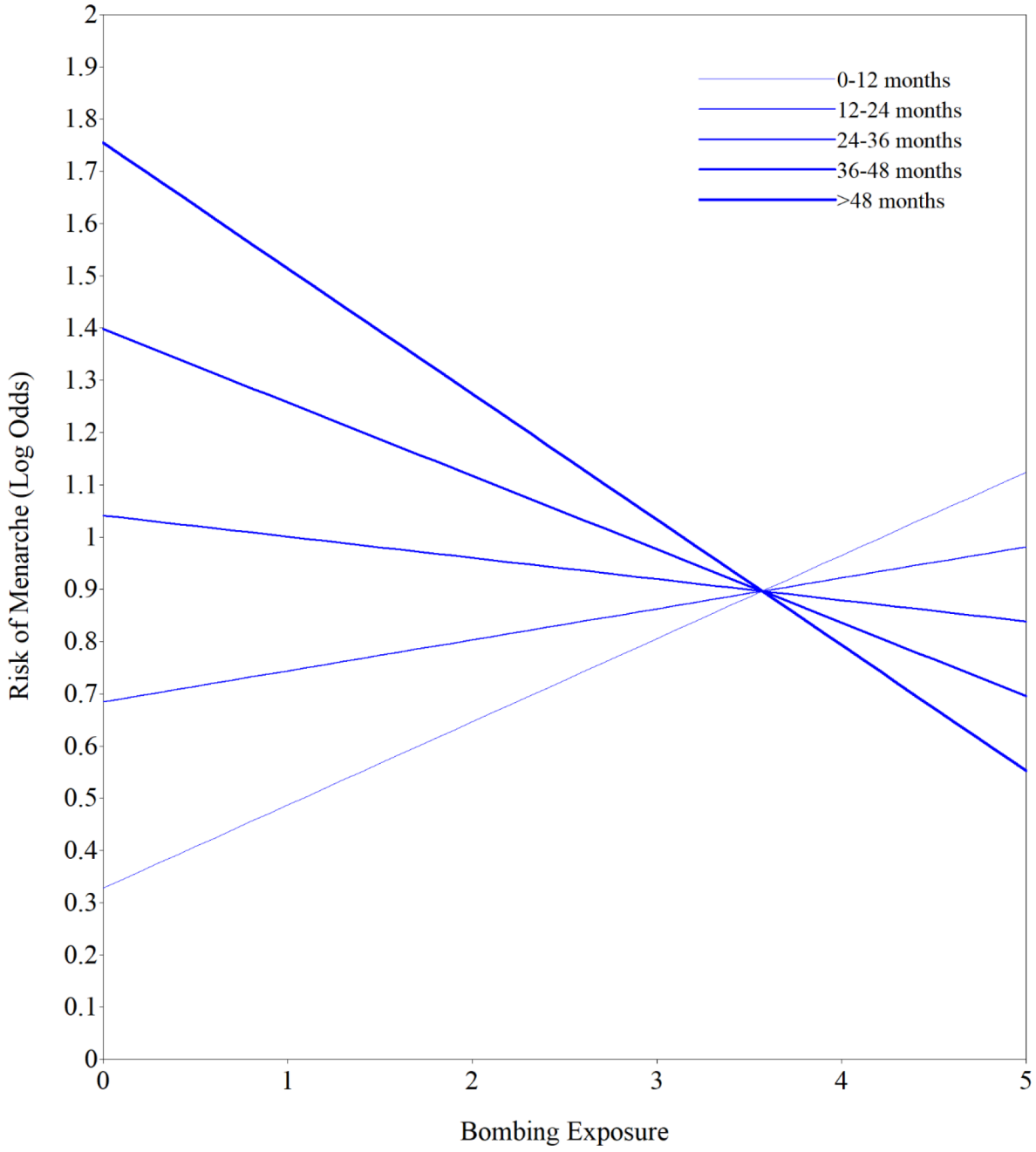

Figure 6.
Effect of bombing*time interaction on girls' risk of menarche.

Table 1.

Baseline descriptive statistics by sex.

	Boys		Girls		Test of Differences
	Mean (SD)	**N (Missing)**	**Mean (SD)**	**N (Missing)**	***t (df), p***
Child age	11.48 (2.32)	662 (86)	11.37 (2.45)	732 (96)	0.84 (1392), 0.40
Caregiver age	39.27 (8.72)	740 (8)	38.70 (8.40)	819 (9)	1.30 (1557), 0.20
Number of children in household	4.96 (2.04)	748 (0)	5.13 (2.07)	824 (4)	−1.59 (1570), 0.11
Number of people in household	7.59 (2.61)	739 (9)	7.80 (2.58)	821 (7)	−1.58 (1558), 0.12

Table 2.

Percent of Syrian refugee children exposed to war events and constructs.

		Percent Exposed	
War Event	**Construct**	**Male**	**Female**
Did you witness explosions close to you	Bombing	59.6	63.3
Were you not able to go outside house because of bullets or bombardment	Bombing	66.6	69.2
Were houses of people you know destroyed	Bombing	58.6	57.9
Did you witness it	Removed	40.4	38.4
Did you house get completely destroyed or part of it	Bombing	33.2	36.1
Did you witness it	Removed	16.3	17.6
Were you inside your house when it got bombarded	Bombing	8.7	11.8
Did you witness the kidnapping of someone	Kidnapping	10.8	8.2
Did any member of your family get kidnapped	Kidnapping	4.4	3.6
Did you witness it	Removed	1.9	1.0
Did armed persons try to kidnap you	Kidnapping	3.2	1.3
Did you witness someone getting beaten up	Mortality	30.2	25.5
Did armed people enter your house	Kidnapping	24.3	19.1
Did you witness it	Removed	19.8	16.4
Did you witness persons getting tortured	Mortality	24.3	24.2
Did you get beaten to give information about your parents	Mortality	3.1	1.9
Did you see an injured person (not on TV)	Mortality	41.3	37.3
Did you see armed persons shooting people	Mortality	23.5	22.6
Were you injured from explosions or bombarding	Mortality	2.9	1.8
Was any close person to you injured in war	Mortality	31.0	26.3
Did you witness it	Removed	17.9	13.6
Did you see a dead person (not on TV)	Mortality	27.7	26.3
Did you witness armed people killing someone	Mortality	16.2	16.5
Did a close person to you get killed	Mortality	33.7	32.5
Did you witness it	Removed	15.4	13.9

Table 3.

Descriptive statistics of war exposure for boys and girls.

	Mean (SD)	Median	N (Missing)	Min./Max.	Correlations		
					Bombing	Kidnapping	Mortality
Boys							
Bombing	2.27 (1.43)	2.00	748 (0)	0/5	1.0	0.28***	0.49***
Kidnapping	0.43 (0.71)	0.00	748 (0)	0/4		1.0	0.47***
Mortality	2.33 (2.48)	2.00	748 (0)	0/10			1.0
Girls							
Bombing	2.37 (1.46)	3.00	828 (0)	0/5	1.0	0.30***	0.49***
Kidnapping	0.32 (0.63)	0.00	828 (0)	0/4		1.0	0.46***
Mortality	2.15 (2.40)	1.00	828 (0)	0/10			1.0

*** $p < 0.001$

Table 4.

Descriptive statistics for pubertal development variables at baseline.

Pubertal Category (Boys)				
	Frequency (Missing Age)	**Mean Age (SD)**	**Minimum**	**Maximum**
Pre-pubertal	411 (58)	10.51 (1.84)	7.04	16.72
Early pubertal	96 (15)	12.75 (1.84)	8.46	16.56
Mid-pubertal	62 (7)	14.54 (1.52)	9.73	16.96
Late pubertal	19 (1)	14.61 (1.98)	9.42	17.01
Post-pubertal*	N/A	N/A	N/A	N/A
Missing Puberty Category	74 (5)	11.84 (2.07)	7.80	17.00

* Post-pubertal category not detectable in our sample due to number of items.

Menarche (Girls)				
	Mean Age (SD)	**N (Missing Age)**	**Minimum**	**Maximum**
No Menarche	10.33 (1.75)	538 (80)	7.09	16.72
Menarche	14.30 (1.62)	192 (16)	9.41	17.91
Missing Menarche	9.18 (1.17)	2 (0)	8.35	10.01

Table 5.

Correlations between pubertal development and associated variables for girls and boys.

	Boys		Girls	
Variable (Year)	**Pubertal Stage (Y1)** ***r*** **(SE)**	**Pubertal Stage (Y2)** ***r*** **(SE)**	**Menarche Achieved (Y1)** ***r*** **(SE)**	**Menarche Achieved (Y2)** ***r*** **(SE)**
Age (Y1)	0.58 (0.03)***	0.69 (0.02)***	0.71 (0.02)***	0.74 (0.02)***
Cortisol (Y1)	0.27 (0.03)***	0.32 (0.04)***	0.27 (0.03)***	0.30 (0.04)***
Cortisol (Y2)	0.18 (0.06)**	0.24 (0.05)***	0.25 (0.04)***	0.30 (0.04)***
DHEA (Y1)	−0.00 (0.04)	0.00 (0.04)	0.31 (0.03)***	0.28 (0.04)***
DHEA (Y2)	0.03 (0.06)	0.03 (0.06)	0.31 (0.04)***	0.33 (0.04)***
Testosterone (Y1)	0.30 (0.03)***	0.39 (0.03)***	0.26 (0.03)***	0.24 (0.04)***
Testosterone (Y2)	0.34 (0.05)***	0.41 (0.04)***	0.21 (0.04)***	0.23 (0.04)***
Height (Y1)	0.57 (0.03)***	0.70 (0.02)***	0.63 (0.02)***	0.71 (0.02)***
Height (Y2)	0.58 (0.03)***	0.73 (0.02)***	0.52 (0.03)***	0.65 (0.02)***
Weight (Y1)	0.53 (0.03)***	0.60 (0.03)***	0.68 (0.02)***	0.73 (0.02)***
Weight (Y2)	0.59 (0.03)***	0.68 (0.03)***	0.59 (0.02)***	0.70 (0.02)***
Waist (Y1)	0.39 (0.04)***	0.45 (0.04)***	0.52 (0.03)***	0.55 (0.03)***
Waist (Y2)	0.44 (0.04)***	0.51 (0.04)***	0.48 (0.03)***	0.59 (0.03)***
Hip (Y1)	0.43 (0.04)***	0.55 (0.03)***	0.66 (0.02)***	0.69 (0.02)***
Hip (Y2)	0.49 (0.04)***	0.57 (0.03)***	0.59 (0.03)***	0.70 (0.02)***

*** $p < 0.001$

** $p < 0.01$