ARTICLES
https://doi.org/10.1038/s41559-018-0567-6

nature ecology & evolution

Corrected: Author Correction

Childhood ecology influences salivary testosterone, pubertal age and stature of Bangladeshi UK migrant men

Kesson Magid[1*], Robert T. Chatterton[2,3,4], Farid Uddin Ahamed[5] and Gillian R. Bentley[1]

Male reproductive investment is energetically costly, and measures of human reproductive steroid hormones (testosterone), developmental tempo (pubertal timing) and growth (stature) correlate with local ecologies at the population level. It is unclear whether male reproductive investment in later life is 'set' during childhood development, mediated through adulthood, or varies by ethnicity. Applying a life-course model to Bangladeshi migrants to the United Kingdom, here we investigate plasticity in human male reproductive function resulting from childhood developmental conditions. We hypothesized that childhood ecology shapes adult trade-offs between reproductive investment and/or other fitness-related traits. We predicted correspondence between these traits and developmental timing of exposure to ecological constraints (Bangladesh) or conditions of surplus (United Kingdom). We compared: Bangladesh sedentees ($n = 107$); Bangladeshi men who migrated in childhood to the United Kingdom ($n = 59$); migrants who arrived in adulthood ($n = 75$); second-generation UK-born and raised children of Bangladeshi migrants ($n = 56$); and UK-born ethnic Europeans ($n = 62$). Migration before puberty predicted higher testosterone and an earlier recalled pubertal age compared with Bangladeshi sedentees or adult migrants, with more pronounced differences in men who arrived before the age of eight. Second-generation Bangladeshis were taller, with higher testosterone than sedentees and adult migrants, and higher waking testosterone than Europeans. Age-related testosterone profiles varied by group, declining in UK migrants, increasing in sedentees, and having no significant relationship within UK-born groups. We conclude that male reproductive function apparently remains plastic late into childhood, is independent of Bengali or European ethnicity, and shapes physiological trade-offs later in life.

Globally, men in wealthy, developed regions generally have higher testosterone than those living in less affluent ones[1–3]. While some researchers link such variation to 'ethnic', 'racial' or genetic traits[4–6], ecological and behavioural variables associated with energy availability, such as abundant nutritional intake, pathogen load and sedentary lifestyles, also potentially contribute to interpopulation differences in reproductive phenotypes[7–12]. Developmental exposure to energetic variables during childhood may further explain adult variation in reproductive steroid hormones. Evidence supporting this 'developmental hypothesis' connects early infancy, pre-birth or childhood experience with sex steroid levels in later infancy[13,14], developmental timing as measured by adult height and pubertal age[15–18], or adult reproductive function[3,18–21].

Migration studies support the developmental hypothesis. Children migrating from less to more affluent regions show rapid postnatal growth and earlier sexual maturation[19,22,23]. Levels of salivary progesterone, ovulation rates and menopausal age of Bangladeshi women who reached adulthood in more ecologically constrained environments were lower compared with those who migrated to a less challenging one[19,24,25], and early-childhood migration (age 0–8 versus 9–16 years) was associated with more robust ovarian function[19,23].

We lack comparable migrant studies among men, but—based on the above findings—we predicted that men with different life histories would express varying degrees of reproductive investment depending on differential developmental conditions. We expected that males encountering improved ecologies before or during developmental transitions would invest in more costly reproductive effort associated with competition and/or sexual signalling, mediated by testosterone[26–28]. Based on ecological developmental histories, we presumed that individual trade-offs between testosterone-mediated traits and other energetic demands would lead to population-level differences. Considering male variation in reproductive function, hormonal variations in non-clinical populations are unlikely to impact fecundity[29,30], but instead relate to trade-offs between traits associated with survivorship and reproductive effort[2,3,31–34].

We therefore designed a cross-cultural study to distinguish whether global variations in male reproductive phenotypes (measured by salivary testosterone levels, pubertal age and stature) reflect: (1) developmentally plastic, organizational responses to childhood ecology or (2) current, activational responses to local ecology. We selected a generally homogenous, ethno-cultural group of Bangladeshis of Bengali ethnic origin, some of whom migrated from a less to more affluent region (specifically, Sylhet, northeast Bangladesh to London, United Kingdom).

We assumed that there would be fewer ecological constraints on males in the United Kingdom compared with Bangladesh. Despite improvements, Bangladesh still ranks globally among the poorest quartile of countries, with high indicators of maternal undernutrition and stunting (36%) among children aged <5 years[35,36]. However, the Bangladeshi populations studied here originate from the land-owning, middle-class not normally subject to nutritional

[1]Department of Anthropology, University of Durham, Durham, UK. [2]Department of Obstetrics and Gynecology, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA. [3]Department of Physiology, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA. [4]Department of Pathology, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA. [5]Department of Anthropology, University of Chittagong, Chittagong, Bangladesh. *e-mail: kesson.magid@durham.ac.uk

or energetic constraints. Instead, they contrast with migrants in developmental exposure to infectious and/or parasitic diseases and environmental instability (that is, political unrest, periodic flooding, and poor public health provision and sanitation)[37,38], which cross social and economic boundaries[37,39–42]. Limitations on energy availability during growth can lead to trade-offs in reproductive function[7,11,12,24,25]. In London, migrants join other British Bangladeshis, 65% of whom are classified as living in income poverty, which is higher than for other UK ethnic groups[43].

We selected timing of migration as a dependent variable (encompassing developmental exposure to environments with abundant resources) to predict hormonal (salivary testosterone) and maturational/growth (recall of pubertal timing and standing height) markers of adult reproductive function. Developmental milestones of birth, middle childhood (concurrent with adrenarche: the prepubertal adrenal secretion of androgens) and puberty informed our hypotheses. Groups comprised: (1) 'sedentees' (men who never left Sylhet); (2) 'adult migrants' (British Bangladeshis from Sylhet who migrated as adults (post-puberty) to London, United Kingdom); (3) 'child migrants' (British Bangladeshis who migrated to the United Kingdom as children (aged ≤19 years)); (4) 'second-generation migrants' (UK-resident Bangladeshis born and raised in the United Kingdom, with parents originating from Sylhet); and (5) 'British Europeans' (UK-born men of European ethnicity who grew up in the United Kingdom and reside in similar neighbourhoods and socioeconomic conditions as the migrants).

We tested two childhood developmental hypotheses. First, that men who experienced fewer ecological constraints before puberty express greater adult reproductive investment than men with more constrained childhood experiences. The second hypothesis refined the first, focusing on early childhood before the age of nine, and proposing that fewer ecological constraints experienced between birth and eight years would lead to greater adult reproductive investment compared with men with more constrained early-childhood experiences. Both hypotheses predicted that, compared with adult migrants and sedentees, men who grew up in the United Kingdom would have: (1) significantly higher levels of salivary testosterone; (2) earlier recalled age markers of puberty[44]; and (3) greater stature.

Our third, non-childhood developmental hypothesis proposed that adult male reproductive traits (for example, salivary testosterone) remain plastic during the life course, reflecting either cumulative exposure to adult ecological conditions or responses to current, local ecology. This predicted: (1) significantly higher salivary testosterone in adult migrants compared with sedentees; and (2) correlation within adult migrants between the number of years spent in the United Kingdom and salivary testosterone, adjusting for age.

Our final, non-developmental hypothesis proposed that biological and cultural traits associated with ethnicity explain interpopulation variations in reproductive traits. This predicted: (1) higher salivary testosterone; and (2) an earlier age at puberty and taller stature in UK-born British Europeans compared with second-generation British Bangladeshis.

Results

Table 1 presents descriptive statistics for all groups. Adult migrants were significantly older than all other groups, averaging 48.4 years (95% confidence interval (CI) = 44.6 to 52.3). Second-generation men were the youngest, averaging 24.5 years (95% CI = 22.5 to 25.8). Age at migration and recruitment correlated for child migrants (Pearson's correlation coefficient (r) = 0.44, t = 3.3, d.f. = 46, P = 0.001), but not adult migrants (r = 0.13, t = 1.0, d.f. = 62, P = 0.3). Men who arrived aged <9 years were younger at recruitment than those who arrived aged 9–19 (28.0 and 36.9 years, respectively; t = 3.1, d.f. = 33.6, P = 0.004).

Compared with sedentees, British Europeans and all migrants were significantly taller (except adult migrants) and heavier, with higher body mass indices (BMIs) (Table 1). Age at recruitment negatively predicted the height of child migrants (n = 40) compared with sedentees (regression coefficient of population (β) = −0.379 s.d., 95% CI = −0.749 to −0.008, n = 106, P = 0.045), while no significant secular trends for height were observed within other residence groups (Fig. 1). Across all groups, older men recalled reaching puberty later (β = 0.28 s.d., 95% CI = 0.155 to 0.410, n = 237, P = 0.00002). After correcting for age at recruitment, men with higher salivary testosterone recalled reaching puberty earlier (waking: β = −0.172 s.d., 95% CI = −0.301 to −0.044, n = 219, P = 0.01; evening: β = −0.130 s.d., 95% CI = −0.258 to −0.003, n = 220, P = 0.047; Supplementary Table 10). However, no such relationship was observed when restricting the same analysis to Bangladeshis resident in the United Kingdom or only to sedentees. All intergroup regressions included age at recruitment and all salivary testosterone analyses included BMI as covariates, with established predictive relationships with adult testosterone[34,45–49]. Testosterone regressions confined to child migrants included BMI imputed at the population mean (23.9 for n = 8, 24% of cases), and were replicated with complete cases.

Regression findings supported both childhood developmental hypotheses and their associated predictions with few exceptions, while the third and fourth hypotheses based on adult ecology or ethnicity were largely not supported (Table 2). The experience of UK ecological conditions before adulthood led to higher testosterone, an earlier age at puberty and taller stature compared with men who experienced similar conditions after puberty. Second-generation men who spent all of their childhood in the United Kingdom had the highest waking (153.5 pg ml^{-1}, 95% CI = 133.8 to 173.2, n = 25) and evening (119.4 pg ml^{-1}, 95% CI = 98.3 to 140.5, n = 28) salivary testosterone of any group (Fig. 2), significantly higher than adult migrants (waking: 90.7 pg ml^{-1}, 95% CI = 80.7 to 100.7, n = 53 P = 0.0001; evening: 75.0 pg ml^{-1}, 95% CI = 65.5 to 83.5, n = 53 P = 0.03) and sedentees (waking: 100.9 pg ml^{-1}, 95% CI = 91.4 to 110.4, n = 103, P = 0.0002; evening: 76.2 pg ml^{-1}, 95% CI = 68.3 to 84.2, n = 102, P = 0.007). Child migrants had the second highest salivary testosterone levels, which were higher for waking (141.4 pg ml^{-1}, 95% CI = 119.2 to 163.5, n = 26) and evening (100.1 pg ml^{-1}, 95% CI = 84.6 to 115.7, n = 27) samples than sedentees (P = 0.002 and P = 0.02, respectively) and adult migrants (P = 0.0003 and P = 0.07, respectively).

Age at migration predicted an earlier recalled age at puberty for migrants who arrived before completing puberty (≤ age 19), but not for those who migrated as adults (Fig. 3); however, this relationship was not significant in child migrants after including recruitment age in the model (β = 1.10 s.d., 95% CI = −0.106 to 2.30, n = 19, P = 0.071). Both child migrants (15.8 years, 95% CI = 14.5 to 17.1, n = 19) and second-generation men (14.2 years, 95% CI = 13.5 to 14.8, n = 21) recalled earlier ages at puberty compared with sedentees (16.1 years, 95% CI = 15.7 to 16.5, n = 103) and adult migrants (16.4 years, 95% CI = 15.9 to 16.9, n = 49), but these differences were only significant for the second-generation men (P = 0.003 and P = 0.02, respectively; Table 2). Similarly, second-generation men (n = 49) averaged 8.6 cm (95% CI = 6.6 to 10.7, n = 106, P = 4^{-15}) taller than sedentees and 7.1 cm (95% CI = 4.0 to 10.2, n = 65, P = 0.0001) taller than adult migrants, while child migrants (n = 44) averaged 4.3 cm (95% CI = 2.2 to 6.5, P = 0.0007) taller than sedentees, and a non-significant 2.8 cm (95% CI = −0.4 to 6.0, P = 0.12) taller than adult migrants.

Ecological conditions in the United Kingdom predicted higher testosterone, an earlier age at puberty and taller stature if experienced during early childhood (ages 0–8 years) compared with men only exposed to these conditions in middle childhood and puberty (>9 years). Within child migrants, age at migration negatively predicted evening salivary testosterone independent of the number of years spent in the United Kingdom (waking: β = −0.553 s.d.,

Table 1 | Descriptive statistics for each residence group studied

	n	Age, years	Height, cm	Weight, kg	BMI	Waking[a], pg ml^{-1}	Evening[a], pg ml^{-1}	Recalled age at puberty, years
Sedentees	107	38.7 (14.1)	162.8 (5.6)	60.0 (9.2)	22.6 (3.2)	100.9 (49)	76.2 (40.8)	16.2 (1.9)
Adult migrants	75	48.4 (15.6)	164.3 (6.6)	67.8 (9.2)	25.1 (2.9)	90.7 (40.8)	75.0 (33.9)	16.4 (1.7)
Child migrants	59	32.1 (10.8)	167.1 (6.4)	69.0 (12.2)	24.6 (3.6)	141.4 (67.3)	100.1 (48)	15.8 (2.7)
Second-generation migrants	56	24.2 (5.6)	171.4 (5.5)	71.2 (12.6)	24.2 (3.8)	153.5 (54.6)	119.4 (59.6)	14.2 (1.4)
British Europeans	62	41.4 (16.1)	177.1 (6.3)	76.8 (10.7)	24.5 (3.2)	114.5 (52.6)	92.1 (64.9)	14.2 (1.4)
All groups	359	38.0 (15.3)	167.5 (8)	67.4 (12)	23.9 (3.4)	112.0 (55.3)	86.8 (49.7)	15.7 (2)

All values shown are means (s.d.). [a]Salivary testosterone levels.

95% CI = −1.136 to 0.029, $n = 33$, $P = 0.07$; evening β = −0.930 s.d., 95% CI = −1.480 to −0.380, $n = 34$, $P = 0.003$; Supplementary Table 2), and infant or early-childhood migrants had significantly higher evening salivary testosterone compared with late-childhood migrants (9–19 years) (Fig. 4 and Supplementary Fig. 1), although the difference was not significant when adjustments were made for recruitment age (Supplementary Table 4). Migration between birth and age 9 ($n = 8$) predicted earlier recalled age at puberty compared with migration at >19 years (β = −0.858 s.d., 95% CI = −1.637 to −0.079, $n = 40$, $P = 0.034$; Supplementary Table 6), but not compared with migration between 9 and 19 years (β = −0.919 s.d., 95% CI = −1.96 to 0.121, $n = 20$, $P = 0.10$). Combining second-generation and child migrants, exposure to UK conditions from before birth to age 9 ($n = 29$) predicted an earlier age of puberty compared with UK migrants who moved between the ages of 9 and 19 years (β = −0.969 s.d., 95% CI = −1.675 to −0.264, $P = 0.004$) or after the age of 19 (β = −0.909 s.d., 95% CI = −1.495 to −0.322, $P = 0.003$).

Child migrants were taller if they migrated earlier. Age of migration predicted the adult height of child migrants after adjusting for the number of years in the United Kingdom (β = −0.719 s.d., 95% CI = −1.22 to −0.217, $n = 37$, $P = 0.009$; Supplementary Table 11), although there were no significant predictors of height when adjusting instead for recruitment age (Supplementary Table 12).

The experience of ecological conditions in the United Kingdom at any point during adulthood did not lead to higher testosterone. Instead, the waking salivary testosterone levels of adult migrants were significantly lower than those of sedentees, suggesting fixation

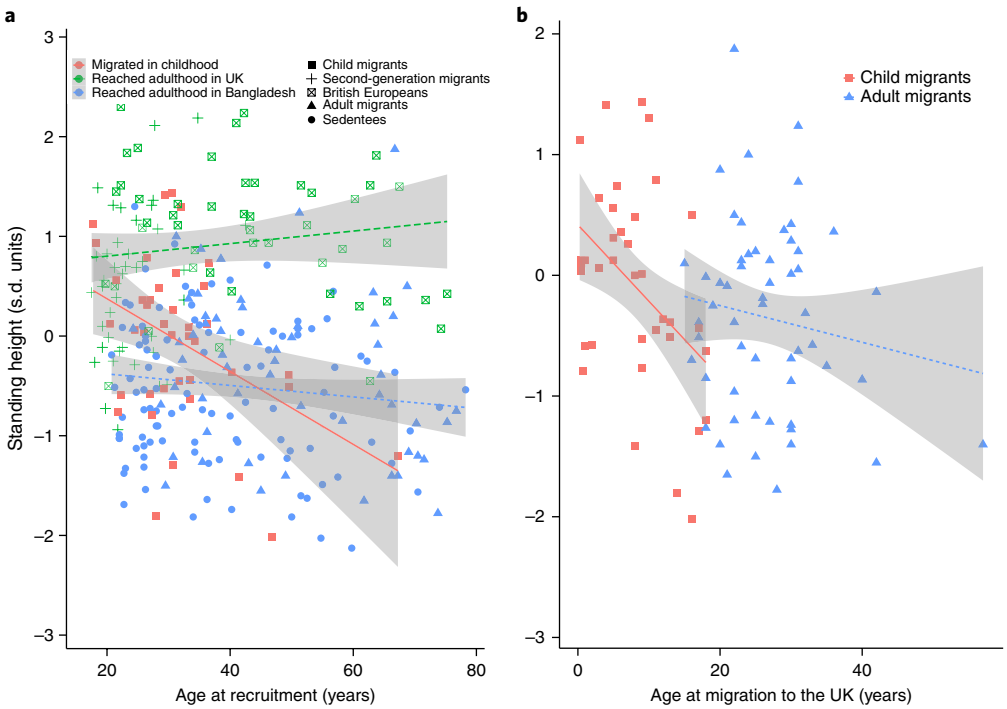

Fig. 1 | Linear regression of standing height by age at recruitment and migration. Standing height is shown in z-transformed s.d. units. Lines indicate linear regression ± s.e.m. (standard error of the mean; grey shaded areas). **a**, Age at recruitment for cohorts separated by childhood conditions. There was a significant correlation for child migrants (s.d. = −0.483, 95% CI = −0.840 to −0.125, $n = 40$, $P = 0.009$). No significant secular trends were observed for men who reached puberty in the United Kingdom (s.d. = 0.123, 95% CI = −0.033 to 0.279, $n = 96$, $P = 0.12$) and Bangladesh (s.d. = −0.078, 95% CI = −0.20 to 0.05, $n = 162$, $P = 0.22$). **b**, Age at migration, separated by migration after self-reported age at puberty, or at <19 years of age. The correlation for child migrants was significant (s.d. = −0.713, 95% CI = −1.235 to −0.191, $n = 37$, $P = 0.009$), but for adult migrants it was not (s.d. = −0.171, 95% CI = −0.491 to 0.148, $n = 56$, $P = 0.28$). Linear regressions including both age at recruitment and age at migration in the same model were non-significant for either child or adult migrants (all covariates $P > 0.1$).

Table 2 | Multiple linear regression of salivary testosterone and composite age at puberty by residence group

	Waking salivary testosterone	Evening salivary testosterone	Composite age at puberty
Constant	−0.113 (−0.297 to 0.072); $P=0.234$	−0.114 (−0.306, 0.078); $P=0.246$	0.257** (0.087, 0.427); $P=0.004$
Age(log)	−0.031 (−0.169 to 0.106); $P=0.657$	−0.021 (−0.164 to 0.121); $P=0.771$	0.229*** (0.100 to 0.358); $P=0.001$
BMI	0.173** (0.051 to 0.294); $P=0.006$	0.128* (0.004 to 0.252); $P=0.045$	NA
Adult migrants	−0.344* (−0.667 to −0.021); $P=0.039$	−0.120 (−0.454 to 0.213); $P=0.480$	−0.060 (−0.371 to 0.251); $P=0.707$
Child migrants	0.652** (0.242 to 1.061); $P=0.003$	0.505* (0.089 to 0.922); $P=0.019$	−0.110 (−0.544 to 0.323); $P=0.620$
Second-generation migrants	0.845*** (0.407 to 1.283); $P=0.0002$	0.608** (0.171 to 1.044); $P=0.007$	−0.791*** (−1.222 to −0.360); $P=0.0004$
British Europeans	0.063 (−0.272 to 0.398); $P=0.715$	0.086 (−0.260 to 0.432); $P=0.628$	−1.003*** (−1.312 to −0.694); $P=0.0000001$
Observations	251	254	237
R^2	0.177	0.088	0.242
Adjusted R^2	0.156	0.066	0.225
Residual s.e.m.	0.917 (d.f. = 244)	0.948 (d.f. = 247)	0.880 (d.f. = 231)
F statistic	8.731*** (d.f. = 6; 244)	3.995*** (d.f. = 6; 247)	14.723*** (d.f. = 5; 231)

All values are z-transformed s.d. units. Age and testosterone are also log-transformed. Reference category: Bangladeshi sedentees. *$P<0.05$; **$P<0.01$; ***$P<0.001$.

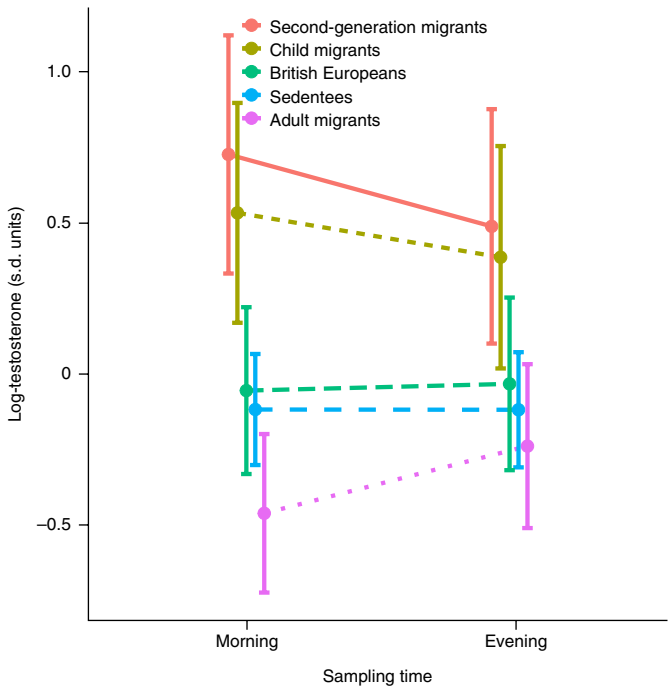

Fig. 2 | Resident group least squares mean salivary testosterone values, adjusted for age and BMI. Morning and evening salivary testosterone (log- and z-transformed, expressed in s.d. units). Each point indicates the residence group least squares mean adjusted for age and BMI. Error bars indicate 95% CI. Child migrants included men ≤19 years at the time of migration. Least squares mean values (±95% CI), calculated using the R package *lsmeans*, and sample sizes were as follows (morning, evening): second-generation: 0.728 (0.333 to 1.122), 0.489 (0.101 to 0.878), $n=25, 28$; child migrants: 0.534 (0.17 to 0.898), 0.387 (0.019 to 0.756), $n=26, 27$; British Europeans: −0.055 (−0.331 to 0.222), −0.032 (−0.319 to 0.254), $n=44, 44$; sedentees: −0.117 (−0.301 to 0.067), −0.118 (−0.309 to 0.073), $n=103, 102$; adult migrants: −0.461 (0.724 to −0.199), −0.239 (−0.51to 0.033), $n=53, 53$.

of this trait in relation to ecological conditions at some point before adulthood, or even an opposite directional effect to that seen in child migrants (Table 1). Moreover, the number of adult years spent in the United Kingdom correlated negatively with salivary testosterone (waking: β = −0.019 s.d., 95% CI = −0.034 to −0.003, $n=53$, $P=0.03$; evening: β = −0.024 s.d., 95% CI = −0.036 to −0.011, $n=56$, $P=0.0005$), while age at adult migration failed to show a relationship with testosterone (waking: β = −0.061 s.d., 95% CI = −0.443 to 0.320, $P=0.8$; evening: β = −0.093 s.d., 95% CI = −0.392 to 0.207, $P=0.5$; Supplementary Table 7).

Characteristics distinctive to European ethnicity failed to predict higher salivary testosterone or earlier age at puberty compared with Bengalis sharing similar developmental histories. Instead, waking testosterone levels of British Europeans ($n=44$) were marginally lower than for second-generation migrants (β = −0.78 s.d., 95% CI = 0.102 to 1.46, $n=25$, $P=0.02$) and no higher than sedentees or child or adult migrants at waking or in the evening (Fig. 2, Table 2 and Supplementary Table 1). The recalled age at puberty did not differ between British-European and second-generation British-Bangladeshi men, but was significantly earlier in Europeans compared with all other groups (Table 2). While British Europeans were 5.6 cm taller than second-generation migrants (95% CI = 8.1 to 2.4; $P=0.00002$), this difference was smaller compared with the other ethnic Bengali groups.

Across populations, waking and evening salivary testosterone values declined by age at recruitment. Adjusting for BMI, these declines were −0.79 (95% CI = −1.22 to −0.357, $n=251$, $P=0.001$) and −0.55 (95% CI = −0.949 to −0.156, $n=254$, $P=0.01$) pg ml^{-1} yr^{-1}, respectively. The relationship between age and salivary testosterone varied by residence group. Salivary testosterone levels declined with age in child and adult migrants, the relationship was non-significant in both UK-born groups, and testosterone levels increased with age in sedentees (Supplementary Fig. 1 and Supplementary Table 9). Within UK-resident groups, child migrants ($n=34$) showed a more pronounced age-related decline in waking salivary testosterone than Europeans (β = −0.50 s.d., 95% CI = −0.952 to −0.042, $n=44$, $P=0.01$). As a pooled group, men born in the United Kingdom had a significant decline in waking (−1.22 pg ml^{-1} yr^{-1},

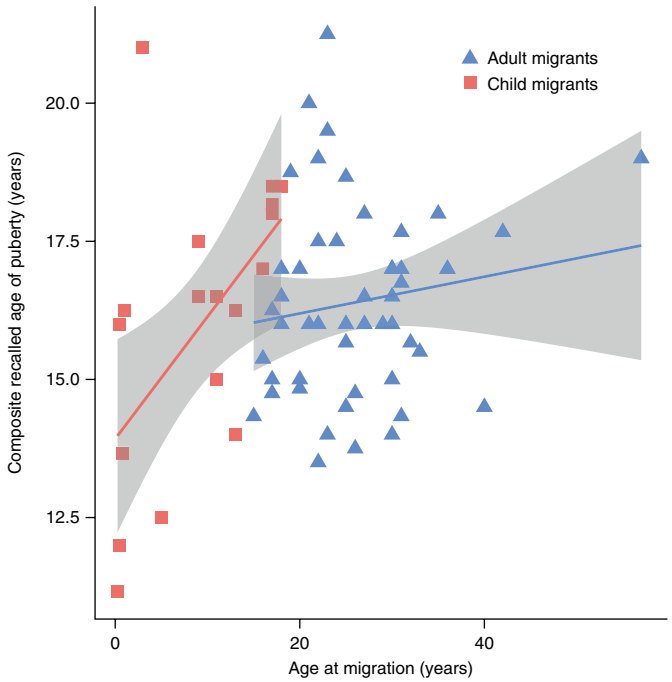

Fig. 3 | Composite recalled age at puberty by age at migration. Migrants were split into cohorts based on whether they migrated before ('child') or after ('adult') their composite recalled age at puberty, or aged ≤19 years if not recalled. The linear regression of z-transformed values in those ≤19 at migration was significant (β = 1.22 s.d., 95% CI = 0.311 to 2.13, $n = 19$, $P = 0.01$). For those ≤19 at migration with age at recruitment as a covariate (β = 1.10 s.d., 95% CI = −0.106 to 2.30, $n = 19$, $P = 0.071$) and for those >19 years at migration with age at recruitment as a covariate (β = 0.159 s.d., 95% CI = −0.209 to 0.527, $n = 49$, $P = 0.39$), the linear regression was not significant. Lines indicate linear regression ± s.e.m. (grey shaded area). Points indicate the average remembered age at four developmental milestones.

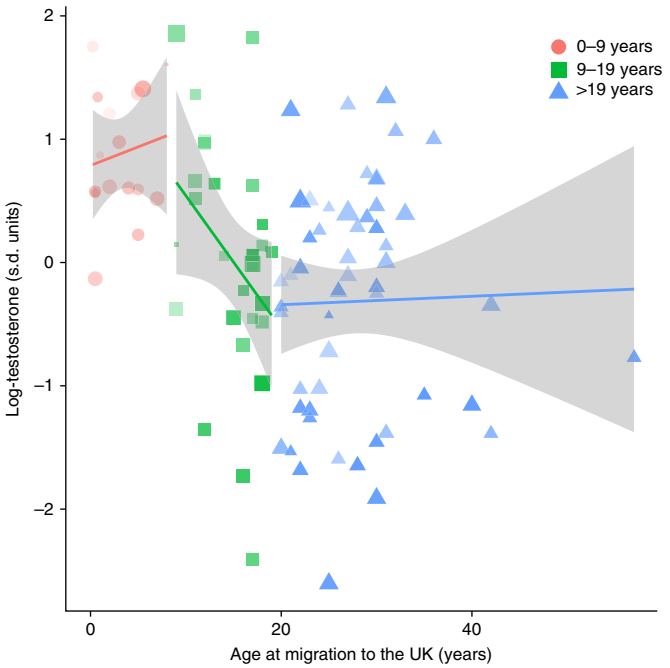

Fig. 4 | Daily mean salivary testosterone by age at migration. Salivary testosterone (log- and z-transformed, expressed in s.d. units) of migrants arriving in the United Kingdom in early childhood (0–9 years, $n = 26$), late childhood (9–19 years, $n = 33$) and adulthood (>19 years, $n = 55$). Darker points indicate older age at recruitment. A larger point size indicates a greater BMI. BMI imputed at the population mean (24.07) for men lacking anthropometric data $n = 6$, 8 and 7, respectively. Lines indicate linear regression ± s.e.m. The linear regression difference between migrants aged >19 and 0–9 years at migration, adjusted for age at recruitment and imputed BMI, was significant (s.d. = 0.529, 95% CI = 0.044 to 1.141, $P = 0.035$). The difference between those >19 and 9–19 years at migration was not (s.d. = 0.141, 95% CI = −0.253 to 0.536, $P = 0.48$). Each point indicates the mean salivary testosterone sampled on two non-consecutive days from a single individual at waking and before bed. Samples were analysed by radioimmunoassay in duplicate.

95% CI = −2.01 to −0.435, $n = 76$, $P = 0.002$) but not evening (−0.91 pg ml^{-1} y^{-1}, 95% CI = −1.839 to 0.020, $n = 77$, $P = 0.055$) salivary testosterone. Waking salivary testosterone remained 34.1 pg ml^{-1} higher (95% CI = 0.0769 to 68.21, $P = 0.049$) in second-generation migrants ($n = 25$) compared with Europeans after adjusting for this UK-born decline.

Reanalysis performed on men aged ≤40 years at recruitment and replication of the child migrant regressions applying multiple imputation methods for BMI yielded substantially similar findings to those performed with mean imputation and either supported or failed to contradict findings within the full cohort (Supplementary Section 5 details both reanalyses).

Discussion

Both childhood developmental hypotheses and predictions were supported: Bangladeshi men who migrated from Sylhet (with greater ecological risks, higher exposure to infectious diseases and poorer healthcare) to London during childhood had higher levels of adult salivary testosterone, an earlier age at puberty and taller stature compared with men who completed their childhood in Sylhet. Differences were particularly marked if individuals migrated in early childhood, aged 0–8 years, and most pronounced for second-generation British Bangladeshis. We conclude that variations in male reproductive phenotype are explained, in this case, by exposure to less constrained ecological conditions during childhood. Male reproductive function apparently remains plastic into late childhood and more plastic in early than late childhood.

In contrast, adult exposure to less constrained ecological conditions did not positively influence salivary testosterone. Instead, adult migrants to the United Kingdom had lower waking salivary testosterone while evening levels were not significantly different from non-migrant sedentees. Additionally, the number of adult years in the United Kingdom did not positively affect salivary testosterone.

We found partial and contradictory evidence relating male reproductive investment to biological and cultural traits specific to the two ethnicities studied. Neighbouring British Europeans with similar developmental histories and socioeconomic positions to resident Bangladeshis did not show greater investment in male reproductive traits compared with second-generation British-Bangladeshi men. Instead, salivary testosterone was not significantly higher in men of European origin compared with any Bengali group, and waking samples were marginally lower than in second-generation migrants. This unexpected result potentially relates to research linking male testosterone to dominance ranking in primates, as well as human status interactions, perceived social position, competition, and provisioning or caregiving[50–58]. While testing for such relationships falls outside the scope of the analyses here, further exploration of social hypotheses[59,60] forms the basis of future study (K.M., R.T.C and G.R.B., manuscript in preparation).

While the recalled age of puberty was earlier for British Europeans compared with groups born in Bangladesh, this did not differ from their UK-born, Bengali counterparts. European men

were taller than all ethnic Bengali groups, but this difference was smallest when comparing second-generation migrants, suggesting a generational trend towards matching local averages in height—a well-documented phenomenon following migration[61,62]. This supports the assumption that UK conditions are more conducive to childhood growth than Bangladesh conditions.

Patterns of male reproductive ageing varied with childhood and adult conditions. The relationship between age and testosterone was: (1) significantly different between men who shared childhood but not adult conditions (negative for adult migrants and positive for sedentees); (2) significantly different between men who shared adult but not childhood conditions (steeper for child than adult migrants); and (3) not different between men sharing both sets of conditions (no significant pattern in second-generation migrants and British Europeans). Such variability adds to evidence of the non-universality of male age-related testosterone decline, particularly in non-industrialized societies, possibly representing developmental responses to energetic conditions[2,8,33,34].

The lack of a robust age-related decline in salivary testosterone within British Europeans and comparatively shallow $-1.43\,pg\,ml^{-1}\,yr^{-1}$ decline of UK-born men remains unexpected considering the male ageing effects widely documented elsewhere[45,46]. The serum testosterone decline reported in a large, longitudinal study of middle-class Caucasian (87%) men in the USA[46] equates to $-2.13\,pg\,ml^{-1}\,yr^{-1}$ of salivary testosterone[63]. Characteristics of the relatively small, socioeconomically poor, urban population of UK-born men may explain differences between our findings and large-scale epidemiological studies.

Our findings support the conclusion that developmental reproductive responses to ecological conditions are most distinctly expressed in early adulthood[2,64] and diurnally at waking[65]. Differences in salivary testosterone between groups were greatest in early adulthood, but trended towards convergence around age 40 (Supplementary Section 2). Migrants experiencing decreased ecological constraints during childhood development had the steepest age-related decline, suggesting that early-life improvements lead to adjustment of male reproductive function in early adulthood, accompanied by rapid decline at later ages.

The above findings lend further support to the 'developmental hypothesis', whereby pre-birth, early infancy or childhood conditions influence reproductive development in later infancy[13,14], developmental transitions to adulthood[15–18,66], and adult reproductive and senescent traits in both women and men[18–21]. Migration during childhood to a less constrained ecology leads to increased investment in two proximate measures of male reproductive function: salivary testosterone and age at puberty. Ecological conditions in the United Kingdom during all or part of childhood also lead to greater childhood growth, as evidenced by taller stature (Fig. 1). Child migrants recruited at younger ages were taller, probably reflecting developmental effects on growth combined with cohort effects of peak migration, as discussed below.

The association of childhood development in the United Kingdom with increased male reproductive investment across the life course mirrors the results from migrant studies of Bangladeshi women that found higher salivary progesterone, higher rates of ovulation, an earlier age at adrenarche and menarche, later menopause, and slower reproductive ageing among women who migrated during childhood[19,23–25].

We interpret population differences in these reproductive traits as evolved strategies to balance lifetime investment in reproduction against the demands of growth, immunity and maintenance[67]. From this life-history theory perspective, ecological conditions during critical phases in the organization of hormonal axes and somatic tissues shape investment in reproductive effort at a population level in ways that are expressed throughout adulthood. While we selected age 8 as an important childhood biosocial threshold[68,69] when early male hormonal organization becomes set, sufficient plasticity persists into late childhood such that migration to the United Kingdom before sexual maturity apparently promotes greater investment in phenotypic measures of reproductive effort[3].

The differences seen here between child migrant cohorts do not argue against gradual linear transitional stages, as opposed to punctuated thresholds of sensitivity to ecology at middle childhood and adolescence[70,71]. We split early- and late-childhood migrants according to a chronological, not physiological marker, and ecology probably influences the timing of physiological transitions, as documented in migrant girls from Bangladesh to the United Kingdom[23], and as seen in self-reports of puberty in this population. Moreover, cohorts were separated at thresholds when we expected completion of pre-adrenarche or pre-pubertal development, meaning they probably contained individuals who were peri-adrenarcheal or peri-pubescent at the time of migration, with associated linear trends suggesting diminishing organizational effects by chronological age at migration.

The selected Bangladeshi communities share dietary, physical and cultural practices, and the migrant populations are uniquely homogenous in socioeconomics and geography. While these attributes reduce potential sources of variation in male reproductive function, cohort differences between migrant groups also limit our findings. Demographically, migration of adult Bangladeshi men to the United Kingdom peaked in the 1970s, while wives and children of adult migrants typically followed in the 1980s[72]. The average recruitment age of adult migrants reflects the 1970s peak. The correlation between age at migration and recruitment in child migrants reflects the 1980s peak. The timing of UK family unification limits the maximum age of British-born offspring. While we included age as a covariate in all models testing for intergroup differences, we remain limited in our ability to contrast ecological influences on the reproductive function of older males. Moreover, despite screening for family members of migrants among sedentees, we cannot exclude the possibility of a selection bias in our migrant groups.

Retrospective measures of pubertal timing are open to recall error, which is likely to be exacerbated by ageing[73,74]; however, in cross-sectional life-course research, combined recall instruments of this kind provide limited but internally consistent estimates of relative maturational rates with low test–retest variation[44,75,76]. Older men recalled later ages at puberty, which may represent a secular trend independent of our cohort differences in childhood ecology or systematic recall/response bias. However, relative differences in developmental cohorts remained evident after including age at recruitment as a covariate and restricting our analysis to men aged <40 years (Supplementary Section 2). Finally, while we propose immunological aspects of ecology as a primary explanation for the interpopulation differences observed above, we cannot exclude the possibility that the recalled age of puberty, as well as biomarkers of reproductive investment, result exclusively from social components of acculturation, stress from discrimination, or perceived threats to status unique to growing up as a minority (such as identifying as part of an outgroup)[77] or social stimuli from interactions within peer groups.

Based on our study design, we conclude that variation in biomarkers of reproductive function within Bangladeshi men relate to inconsistencies in their ecologies. We consider childhood exposure to disease within Bangladesh, as well as the experience of migration itself, as the most plausible causes of the observed variation. Results from British Europeans suggest limits to the biological and cultural traits associated with ethnicity in predicting adult male reproductive function, and potential differences in the influence of social position on the testosterone levels of migrant and non-migrant men. These findings have implications for life-history interpretations of reproductive disease and aetiology[78], by relating early-life conditions to prostate cancer or disease[79], incorporating ecological variations to documented health outcomes of

age-related changes in testosterone[46,80], trends in pubertal timing[81,82], and global clinical definitions of 'normal' ranges in androgen supplementation[83] therapies.

Methods

Study population. Bangladeshis in London and Sylhet form a homogeneous ethnic group originating from an affluent socioeconomic position relative to other Bangladeshis, share consistency in dietary, religious and social practices, and are subject to limited physical work and nutritional stress in either country. Following migration to the United Kingdom, access to Bangladeshi foods and community cohesion within a geographically condensed region preserves much of this homogeneity[84]. In 2004–2010, we recruited 359 healthy male volunteers aged 17–78 at completion of the study, screened to exclude thyroid conditions or diabetes. First-order relatives were excluded from participation to avoid closely shared genetic or immediate environmental confounders. Participants were divided into the following groups: (1) Bangladeshi sedentees ($n=107$) born and still resident in the Sylhet City District, northeast Bangladesh; (2) first-generation migrants from Sylhet ($n=75$) who moved to the United Kingdom after reaching puberty, determined from our data to be >19 years (adult migrants); (3) first-generation migrants from Sylhet ($n=59$) who moved to the United Kingdom before completing puberty (aged ≤19 years) (child migrants); (4) second-generation British-Bangladeshi men ($n=56$) born to parents who had themselves migrated to the United Kingdom from Bangladesh; and (5) London residents of British-European ethnicity ($n=62$) recruited from similar neighbourhoods and of similar socioeconomic status to the migrant groups.

Migrants were classified as adults if they arrived in the United Kingdom post-puberty, based on a self-recalled, composite age at puberty (measures detailed below). Of 68 migrants who provided age at migration and recalled pubertal age, 19 reported arriving before, and 49 after puberty. The remaining first-generation men were classified as adult migrants if they arrived after the mean composite age of puberty +2 s.d.: $15.75+(2\times2.09)=19.93$, which was rounded to 20 years. To ensure that the sedentee population reflected an ethnic and socioeconomic group comparable to migrants, with sufficient means to emigrate, participants in Sylhet were screened for relatives who had migrated to the United Kingdom, mainland Europe or the North American continent, and were recruited using local networks and snowballing techniques. Participants in London were recruited from community centres, mosques, fitness centres/clubs, or internet and newspaper advertisements.

Questionnaires. We collected demographic, migration, reproductive and nutritional data, recalled pubertal markers, and health information using previous methods employed in a study of Bangladeshi migrant women[19,85]. Native English speakers were given the option of completing a slightly shortened questionnaire online via a protected portal.

Saliva sampling. A total of six saliva samples were collected over two non-consecutive days from each participant. To capture diurnal patterns of hormonal profiles that included later analyses of salivary cortisol, one sample was requested immediately upon waking, one approximately 30 min post-waking and one immediately before retiring to bed. For the purposes of salivary testosterone analyses, we only report here the first waking and evening samples. Participants were asked to record the exact times of sampling each day; all reported giving their first sample within 30 min of waking. Salivary testosterone was measured in duplicate by radioimmunoassay without extraction[86]. Antiserum was prepared, and all analyses were performed between 2006 and 2010 in the laboratory of R.T.C. at Northwestern University, Chicago, USA. Inter-assay coefficients of variation were within 15% for high (100 pg ml^{-1}), low (50 pg ml^{-1}) and internal (pooled saliva sample) quality controls, while recovery of spiked samples was 97.1% ± 18.2 s.d. Sensitivity was 0.028 nmol l^{-1} and the average intra-assay coefficient of variation was 2.01%. Duplicate readings of two samples were excluded as both exceeded the limits of detection of the high standard of the assay, four samples were based on single readings due to a limited sample or laboratory error of the second reading. Seven outlying samples with z-scores above 3.29 were recoded to +2 s.d. of the population mean of salivary testosterone for that time point.

Anthropometry. Standing height and weight measurements were collected according to standardized methods[87]. Eight child migrants lacked anthropometric data. To preserve sample sizes in analyses of migration effects, testosterone regressions within this group only were performed with BMI imputed at the population mean and also replicated with complete cases only and with multiple imputation methods (see Supplementary Section 2).

Pubertal measures. A composite age at puberty was adapted from the Adolescence Scale (AS-ICSM) retrospective self-assessment of puberty milestones[44]. Age at puberty was estimated by averaging when men recalled, where possible, four markers of male secondary sexual development: (1) voice breaking; (2) appearance of facial hair or start of shaving; (3) first appearance of pubic and underarm hair; and (4) first nocturnal emission. Questions were phrased, "Do you remember how old you were when…?" Participants were asked to respond yes or no, and following this, were asked: "If you remember, how old were you?". This response was open ended, and if the age in years was unknown, respondents were free to estimate by other measures such as year of school or other historical events. Men responding with estimates spanning two years; for example, "12–13", were coded at the midpoint, 12.5 years.

Statistical analyses. We tested all hypotheses by multiple linear regression analysis. A full description of all variables used and statistical tests performed can be found in Supplementary Section 3. All intergroup regressions included age at recruitment as a covariate to adjust for cohort differences, potential effects of male reproductive ageing[45,46], and demographic or secular trends unrelated to our hypotheses. Salivary testosterone analyses included BMI as a covariate with an established predictive relationship with adult testosterone[34,47–49].

To test for evidence that contrasting ecological conditions before puberty relate to dependent measures of adult reproductive function, we performed multiple linear regressions, with age at recruitment, BMI (in testosterone regressions only) and residence group included as covariates. To test for evidence that contrasting ecological conditions during early childhood relate to dependent measures of adult testosterone, we performed multiple linear regressions with covariates being either age at recruitment or number of years spent in the United Kingdom since migration, imputed BMI, and two cohorts of child migrants split by age of migration before and after reaching nine years of age. As inclusion of both age at recruitment and number of years in the United Kingdom in the same model exceeded limits of collinearity (variance inflation factor >10)[88], for salivary testosterone, number of years in the United Kingdom was considered a combined measure of the influences of exposure to adult and current ecological conditions, and the age at recruitment. Results including only complete cases for BMI and reanalysis applying multiple imputation techniques are included in the Supplementary Materials.

Second-generation men and child migrants exposed to UK conditions from before birth to age 8 were combined into a single cohort in a linear regression contrasting pubertal recall with cohorts of later childhood (9–19 years at migration) or adult migrants (aged >19 years at migration). Age of recruitment was also included as a control for secular demographic trends in puberty regressions where cumulative influences of environment were expected to become fixed at adulthood. In addition, we tested for linear relationships between dependent variables and age at migration within either the child migrant or adult migrant group only, and included age at migration and either age at recruitment or number of years in the United Kingdom as covariates additional to BMI or imputed BMI in the testosterone regressions. To limit confounding between the effects of ageing/senescence and exposure to ecological conditions in the United Kingdom in adult migrants, we ran the above regressions separately within two age cohorts (≤40 and >40 at recruitment), split at a conventional point of inflection for male life-course studies of sex hormones[89].

Post-hoc analysis of the regressions where 'group' or 'cohort' was an independent variable, with Tukey correction of all-pair multiple comparison using the R package *multicomp* tested for evidence for ethnic or developmental cohort differences.

To test for differences in age-related trends in salivary testosterone, we ran both linear regression and analysis of covariance, including an interaction effect between each residence group and age at recruitment on transformed and untransformed values. Between-group differences in the slope of age-related declines in testosterone were tested in post-hoc analysis, as described above. Within UK-born men, we performed an additional regression with measured salivary testosterone offset by multiplying the number of years after 22 (an established point of male age-related decline[46]) by the UK-born population trend as the dependent variable, and age at recruitment, BMI and ethnic group as covariates. Between-group differences in descriptive variables were tested using linear regressions and post-hoc analysis of differences between residence groups.

Before running the models, salivary testosterone measures were transformed by natural logarithm to correct for skewed normality of distribution, and all measures were z-transformed to a mean of zero and a standard deviation of 1, except calculations for age-related effects on salivary testosterone (Supplementary Table 9), which were left untransformed for comparison with published rates of decline. All analyses were performed using R statistical software version 3.3.1 (ref. [90]) with packages detailed in the analysis code provided in Supplementary Section 4.

Ethics. Ethical approval was granted by the University College London Research Ethics Committee (ID: 0144/002) and Osmani Medical College in Sylhet. All participants provided written consent and were compensated for their time upon completion of the study. Data were stored in accordance with the Data Protection Act (United Kingdom).

Reporting Summary. Further information on experimental design is available in the Nature Research Reporting Summary linked to this article.

Data availability. Code and source data for all analyses and figures generated during the current study are included within this published article and

Supplementary Sections 4 and 5, and are also available in the GitHub repository at https://github.com/kessonovitch/BHAI_Data/.

Received: 15 January 2018; Accepted: 1 May 2018;
Published online: 25 June 2018

References

1. Bentley, G. R., Harrigan, A. M., Campbell, B. & Ellison, P. T. Seasonal effects on salivary testosterone levels among lese males of the Ituri forest, Zaire. *Am. J. Hum. Biol.* **5**, 711–717 (1993).
2. Ellison, P. T. et al. Population variation in age-related decline in male salivary testosterone. *Hum. Reprod.* **17**, 3251–3253 (2002).
3. Bribiescas, R. G. Testosterone levels among Aché hunter-gatherer men : a functional interpretation of population variation among adult males. *Hum. Nat.* **7**, 163–188 (1996).
4. Richard, A. et al. Racial variation in sex steroid hormone concentration in black and white men: a meta-analysis. *Andrology* **2**, 428–435 (2014).
5. Panizzon, M. S. et al. Genetic and environmental influences of daily and intra-individual variation in testosterone levels in middle-aged men. *Psychoneuroendocrinology* **38**, 2163–2172 (2013).
6. Travison, T. G. et al. The heritability of circulating testosterone, oestradiol, oestrone and sex hormone binding globulin concentrations in men: the Framingham Heart Study. *Clin. Endocrinol.* **80**, 277–282 (2014).
7. Muehlenbein, M. P., Alger, J., Cogswell, F., James, M. & Krogstad, D. The reproductive endocrine response to *Plasmodium vivax* infection in Hondurans. *Am. J. Trop. Med. Hyg.* **73**, 178–187 (2005).
8. Trumble, B. C. et al. Age-independent increases in male salivary testosterone during horticultural activity among Tsimane forager-farmers. *Evol. Hum. Behav.* **34**, 350–357 (2013).
9. Priskorn, L. et al. Is sedentary lifestyle associated with testicular function? A cross-sectional study of 1,210 men. *Am. J. Epidemiol.* **184**, 284–294 (2016).
10. Cangemi, R., Friedmann, A. J., Holloszy, J. O. & Fontana, L. Long-term effects of calorie restriction on serum sex-hormone concentrations in men. *Aging Cell* **9**, 236–242 (2010).
11. Boonekamp, J. J., Ros, A. H. F. & Verhulst, S. Immune activation suppresses plasma testosterone level: a meta-analysis. *Biol. Lett.* **4**, 741–744 (2008).
12. Gettler, L. T., McDade, T. W., Agustin, S. S., Feranil, A. B. & Kuzawa, C. W. Testosterone, immune function, and life history transitions in Filipino males (*Homo sapiens*). *Int. J. Primatol.* **35**, 787–804 (2014).
13. Thompson, A. L. & Lampl, M. Prenatal and postnatal energetic conditions and sex steroids levels across the first year of life. *Am. J. Hum. Biol.* **25**, 643–654 (2013).
14. Xia, K. et al. Environmental and genetic contributors to salivary testosterone levels in infants. *Front. Endocrinol.* **5**, 1–15 (2014).
15. Jardim-Botelho, A. et al. Age patterns in undernutrition and helminth infection in a rural area of Brazil: associations with ascariasis and hookworm. *Trop. Med. Int. Heal.* **13**, 458–467 (2008).
16. Holmgren, A. et al. Pubertal height gain is inversely related to peak BMI in childhood. *Pediatr. Res.* **81**, 448–454 (2016).
17. Villamor, E. & Jansen, E. C. Nutritional determinants of the timing of puberty. *Annu. Rev. Public Health* **37**, 33–46 (2016).
18. Kuzawa, C. W., McDade, T. W., Adair, L. S. & Lee, N. Rapid weight gain after birth predicts life history and reproductive strategy in Filipino males. *Proc. Natl Acad. Sci. USA* **107**, 16800–16805 (2010).
19. Núñez-de la Mora, A., Chatterton, R. T., Choudhury, O. A., Napolitano, D. A. & Bentley, G. R. Childhood conditions influence adult progesterone levels. *PLoS Med.* **4**, e167 (2007).
20. Jasienska, G., Ziomkiewicz, A., Lipson, S. F., Thune, I. & Ellison, P. T. High ponderal index at birth predicts high estradiol levels in adult women. *Am. J. Hum. Biol.* **18**, 133–140 (2006).
21. Ellison, P. T. Developmental influences on adult ovarian hormonal function. *Am. J. Hum. Biol.* **8**, 725–734 (1996).
22. Parent, A. S. et al. Timing of normal puberty and the age limits of sexual precocity: variations around the world, secular trends, and changes after migration. *Endocr. Rev.* **24**, 668–693 (2003).
23. Houghton, L. C. et al. Childhood environment influences adrenarcheal timing among first-generation Bangladeshi migrant girls to the UK. *PLoS ONE* **9**, e109200 (2014).
24. Murphy, L. et al. Life course effects on age at menopause among Bangladeshi sedentees and migrants to the UK. *Am. J. Hum. Biol.* **25**, 83–93 (2013).
25. Begum, K. et al. Ethnicity or environment: effects of migration on ovarian reserve among Bangladeshi women in the United Kingdom. *Fertil. Steril.* **105**, 744.e1–754.e1 (2016).
26. Geary, D. C. Evolution of sex differences in trait- and age-specific vulnerabilities. *Perspect. Psychol. Sci.* **11**, 855–876 (2016).
27. Hamilton, W. D. & Zuk, M. Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384–387 (1982).
28. Kokko, H., Brooks, R., McNamara, J. M. & Houston, A. I. The sexual selection continuum. *Proc. R. Soc. B* **269**, 1331–1340 (2002).
29. Andersen, A. et al. Serum levels of testosterone do not provide evidence of selection bias in studies of male reproductive health. *Epidemiology* **11**, 232–234 (2000).
30. Bhasin, S. et al. Testosterone dose–response relationships in healthy young men. *Am. J. Physiol. Endocrinol. Metab.* **281**, E1172–E1181 (2001).
31. Bribiescas, R. G. Reproductive ecology and life history of the human male. *Am. J. Phys. Anthropol.* **44**, 148–176 (2001).
32. Alvergne, A., Faurie, C. & Raymond, M. Variation in testosterone levels and male reproductive effort: insight from a polygynous human population. *Horm. Behav.* **56**, 491–497 (2009).
33. Vitzthum, V. J. et al. Seasonal and circadian variation in salivary testosterone in rural Bolivian men. *Am. J. Hum. Biol.* **21**, 762–768 (2009).
34. Campbell, B., Leslie, P. & Campbell, K. Age-related changes in testosterone and SHBG among Turkana males. *Am. J. Hum. Biol.* **18**, 71–82 (2006).
35. Ahmed, T. et al. Nutrition of children and women in Bangladesh: trends and directions for the future. *J. Heal. Popul. Nutr.* **30**, 1–11 (2012).
36. *Human Development Report 2016*: *Human Development for Everyone* (United Nations Development Programme, New York, 2016).
37. *Bangladesh Demographic and Health Survey 2014* (National Institute of Population Research and Training (NIPORT), Dhaka, and Mitra and Associates, Rockville, 2014).
38. *The World Factbook* (Central Intelligence Agency, Washington DC, 2018); https://www.cia.gov/library/publications/the-world-factbook/geos/bg.html
39. Howard, G. & Bartram, J. *Domestic Water Quantity Service Level and Health* (World Health Organisation, Geneva, 2003).
40. Das, S. & Gulshan, J. Different forms of malnutrition among under five children in Bangladesh: a cross sectional study on prevalence and determinants. *BMC Nutr.* **3**, 1–12 (2017).
41. Stanton, B. F. & Clemens, J. D. Socioeconomic variables and rates of diarrhoeal disease in urban Bangladesh. *Trans. R. Soc. Trop. Med. Hyg.* **81**, 278–282 (1987).
42. Alam, M. J. B., Rahman, M. H., Khan, S. K. & Munna, G. M. Unplanned urbanization: assessment through calculation of environmental degradation index. *Int. J. Environ. Sci. Technol.* **3**, 119–130 (2006).
43. Kenway, P. & Palmer, G. *Poverty Among Ethnic Groups: How and Why Does it Differ?* (Joseph Rowntree Foundation, York, 2007).
44. Kaiser, J. & Gruzelier, J. H. The Adolescence Scale (AS-ICSM): a tool for the retrospective assessment of puberty milestones. *Acta Paediatr. Suppl.* **88**, 64–68 (1999).
45. Morley, J. E. et al. Longitudinal changes in testosterone, luteinizing hormone, and follicle-stimulating hormone in healthy older men. *Metabolism* **46**, 410–413 (1997).
46. Harman, M. S., Metter, J. E., Tobin, J. E., Pearson, J. & Blackman, M. R. Longitudinal effects of aging on serum total and free testosterone levels in healthy men. *J. Clin. Endocrinol. Metab.* **86**, 724–731 (2001).
47. Allen, N. E., Appleby, P. N., Davey, G. K. & Key, T. J. Lifestyle and nutritional determinants of bioavailable androgens and related hormones in British men. *Cancer Causes Control* **13**, 353–363 (2002).
48. Mantzoros, C. S. & Georgiadis, E. I. Body mass and physical activity are important predictors of serum androgen concentrations in young healthy men. *Epidemiology* **6**, 432–435 (1995).
49. Mazur, A. The age–testosterone relationship in black, white, and Mexican-American men, and reasons for ethnic differences. *Aging Male* **12**, 66–76 (2009).
50. Wingfield, J. C., Hegner, R. E., Dufty Jr, A. M. & Ball, G. F. The "challenge hypothesis": theoretical implications for patterns of testosterone secretion, mating system, and breeding strategies. *Am. Nat.* **136**, 829–846 (1990).
51. Archer, J. Testosterone and human aggression: an evaluation of the challenge hypothesis. *Neurosci. Biobehav. Rev.* **30**, 319–345 (2006).
52. Muehlenbein, M. P. & Watts, D. P. The costs of dominance: testosterone, cortisol and intestinal parasites in wild male chimpanzees. *Biopsychosoc. Med.* **4**, 21 (2010).
53. Sapolsky, R. M. Testicular function, social rank and personality among wild baboons. *Psychoneuroendocrinology* **16**, 281–293 (1991).
54. Mazur, A. & Booth, A. Testosterone and dominance in men. *Behav. Brain Sci.* **21**, 353–397 (1998).
55. Carré, J. M. & Olmstead, N. A. Social neuroendocrinology of human aggression: examining the role of competition-induced testosterone dynamics. *Neuroscience* **286**, 171–186 (2015).
56. Book, A. S., Starzyk, K. B. & Quinsey, V. L. The relationship between testosterone and aggression: a meta-analysis. *Aggress. Violent Behav.* **6**, 579–599 (2001).
57. Trumble, B. C. et al. Physical competition increases testosterone among Amazonian forager-horticulturalists: a test of the 'challenge hypothesis'. *Proc. Biol. Sci.* **279**, 2907–2912 (2012).

58. Gray, P. B., Campbell, B. C., Marlowe, F. W., Lipson, S. F. & Ellison, P. T. Social variables predict between-subject but not day-to-day variation in the testosterone of US men. *Psychoneuroendocrinology* **29**, 1153–1162 (2004).
59. Gray, P. B., McHale, T. S. & Carré, J. M. A review of human male field studies of hormones and behavioral reproductive effort. *Horm. Behav.* **91**, 52–67 (2017).
60. Gettler, L. T. Becoming DADS: considering the role of cultural context and developmental plasticity for paternal socioendocrinology. *Curr. Anthropol.* **57**, S38–S51 (2016).
61. Bogin, B., Smith, P., Orden, A. B., Silva, M. I. V. & Loucky, J. Rapid change in height and body proportions of Maya American children. *Am. J. Hum. Biol.* **14**, 753–761 (2002).
62. Mascie-Taylor, C. G. N. & Little, M. A. History of migration studies in biological anthropology. *Am. J. Hum. Biol.* **16**, 365–378 (2004).
63. Wang, C., Plymate, S., Nieschlag, E. & Paulsen, C. A. Salivary testosterone in men: further evidence of a direct correlation with free serum testosterone. *J. Clin. Endocrinol. Metab.* **53**, 1021–1024 (1981).
64. Ellison, P. T. Endocrinology, energetics, and human life history: a synthetic model. *Horm. Behav.* **91**, 97–106 (2017).
65. Kuzawa, C. W., Georgiev, A. V., McDade, T. W., Bechayda, S. A. & Gettler, L. T. Is there a testosterone awakening response in humans? *Adapt. Hum. Behav. Physiol.* **2**, 166–183 (2016).
66. Zemel, B. S., Kawchak, D. A., Ohene-Frempong, K., Schall, J. I. & Stallings, V. A. Effects of delayed pubertal development, nutritional status, and disease severity on longitudinal patterns of growth failure in children with sickle cell disease. *Pediatr. Res.* **61**, 607–613 (2007).
67. Chisholm, J. S. Death, hope, and sex: life-history theory and the development of reproductive strategies. *Curr. Anthropol.* **34**, 1–24 (1993).
68. Del Giudice, M., Angeleri, R. & Manera, V. The juvenile transition: a developmental switch point in human life history. *Dev. Rev.* **29**, 1–31 (2009).
69. Herdt, G. & Mcclintock, M. The magical age of 10. *Arch. Sex. Behav.* **29**, 587–606 (2000).
70. Hochberg, Z. Evo-devo of child growth II: human life history and transition between its phases. *Eur. J. Endocrinol.* **160**, 135–141 (2009).
71. Palmert, M. R. et al. The longitudinal study of adrenal maturation during gonadal suppression: evidence that adrenarche is a gradual process. *J. Clin. Endocrinol. Metab.* **86**, 4536–4542 (2001).
72. Peach, C. South Asian migration and settlement in Great Britain, 1951–2001. *Contemp. S. Asia* **15**, 133–146 (2006).
73. Koo, M. M. & Rohan, T. E. Accuracy of short-term recall of age at menarche. *Ann. Hum. Biol.* **24**, 61–64 (1997).
74. Cooper, R. et al. Validity of age at menarche self-reported in adulthood. *J. Epidemiol. Community Health* **60**, 993–997 (2006).
75. Casey, V. A. et al. Accuracy of recall by middle-aged participants in a longitudinal study of their body size and indices of maturation earlier in life. *Ann. Hum. Biol.* **18**, 155–166 (1991).
76. Gilger, J. W., Geary, D. C. & Eisele, L. M. Reliability and validity of retrospective self-reports of the age of pubertal onset using twin, sibling, and college student data. *Adolescence* **26**, 41–53 (1991).
77. Flinn, M. V., Ponzi, D. & Muehlenbein, M. P. Hormonal mechanisms for regulation of aggression in human coalitions. *Hum. Nat.* **23**, 68–88 (2012).
78. Jasienska, G., Bribiescas, R. G., Furberg, A. S., Helle, S. & Núñez-de la Mora, A. Human reproduction and health: an evolutionary perspective. *Lancet* **390**, 510–520 (2017).
79. Alvarado, L. C. Do evolutionary life-history trade-offs influence prostate cancer risk? A review of population variation in testosterone levels and prostate cancer disparities. *Evol. Appl.* **6**, 117–133 (2013).
80. Kaufman, J. M. & Vermeulen, A. The decline of androgen levels in elderly men and its clinical and therapeutic implications. *Endocr. Rev.* **26**, 833–876 (2005).
81. Worthman, C. M. & Kuzara, J. Life history and the early origins of health differentials. *Am. J. Hum. Biol.* **17**, 95–112 (2005).
82. Walvoord, E. C. The timing of puberty: is it changing? Does it matter? *J. Adolesc. Health* **47**, 433–439 (2010).
83. Handelsman, D. J. Global trends in testosterone prescribing, 2000–2011: expanding the spectrum of prescription drug misuse. *Med. J. Aust.* **199**, 548–551 (2013).
84. *The Bangladeshi Muslim Community in England*: *Understanding Muslim Ethnic Communities* (Change Institute, London, 2009).
85. Núñez-De La Mora, A., Bentley, G. R., Choudhury, O. A. & Napolitano, D. A. The impact of developmental conditions on adult salivary estradiol levels: why this differs from progesterone? *Am. J. Hum. Biol.* **20**, 2–14 (2008).
86. Vittek, J., L'Hommedieu, D. G., Gordon, G. G., Rappaport, S. C. & Southren, A. L. Direct radioimmunoassay (RIA) of salivary testosterone: correlation with free and total serum testosterone. *Life Sci.* **37**, 711–716 (1985).
87. Frisancho, A. R. *Anthropometric Standards for the Assessment of Growth and Nutritional Status* (Univ. Michigan Press, Ann Arbor, 1990).
88. Hair, J. F., Anderson, R. E., Tatham, R. L. & Black, W. C. *Multivariate Data Analysis* (Pearson Education International, Uppersaddle River, 2010).
89. Feldman, H. A. et al. Age trends in the level of serum testosterone and other hormones in middle-aged men: longitudinal results from the Massachusetts Male Aging Study. *J. Clin. Endocrinol. Metab.* **87**, 589–598 (2002).
90. R Development Core Team *R: A Language and Environment for Statistical Computing* (R Foundation for Statistical Computing, Vienna, 2017).

Acknowledgements

The authors thank K. Begum, O. Choudhury, A. Chowdhury, D. Lawson, Z. Salehin, T. Sharmeen and students of Shahjalal University of Science and Technology for assistance with recruitment, translation and data collection, and the Bengali Workers Association, Chadswell Community Centre and Bengali Football Association for providing facilities and promotion. We thank L. Houghton, R. Mace and A. Núñez-de la Mora for advice on study implementation, and H. Colleran and A. Alvergne for comments on previous drafts. This work was supported by the Economic and Social Research Council (PTA-030-2005-00706), Prostate Research Campaign UK (G2003-07), and a Royal Society University Research Fellowship (to G.R.B., UF951006).

Author contributions

K.M. and G.R.B. designed the study and drafted the manuscript. K.M. carried out all data and laboratory analysis. K.M. and F.U.A. supervised and performed the data collection. R.T.C. designed, advised and assisted with laboratory analysis.

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information is available for this paper at https://doi.org/10.1038/s41559-018-0567-6.

Reprints and permissions information is available at www.nature.com/reprints.

Correspondence and requests for materials should be addressed to K.M.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

nature research

Corresponding author(s): Kesson Magid

Reporting Summary

Nature Research wishes to improve the reproducibility of the work that we publish. This form provides structure for consistency and transparency in reporting. For further information on Nature Research policies, see Authors & Referees and the Editorial Policy Checklist.

Statistical parameters

When statistical analyses are reported, confirm that the following items are present in the relevant location (e.g. figure legend, table legend, main text, or Methods section).

n/a	Confirmed	
☐	☒	The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement
☐	☒	An indication of whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☐	☒	The statistical test(s) used AND whether they are one- or two-sided *Only common tests should be described solely by name; describe more complex techniques in the Methods section.*
☐	☒	A description of all covariates tested
☐	☒	A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons
☐	☒	A full description of the statistics including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
☐	☒	For null hypothesis testing, the test statistic (e.g. *F*, *t*, *r*) with confidence intervals, effect sizes, degrees of freedom and *P* value noted *Give P values as exact values whenever suitable.*
☒	☐	For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
☒	☐	For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
☐	☒	Estimates of effect sizes (e.g. Cohen's *d*, Pearson's *r*), indicating how they were calculated
☐	☒	Clearly defined error bars *State explicitly what error bars represent (e.g. SD, SE, CI)*

Our web collection on statistics for biologists may be useful.

Software and code

Policy information about availability of computer code

Data collection	Salivary assay standard curve results were analysed using GraphPad Prism 5.
Data analysis	All statistical analysis was performed on R Statistical Software v. 3.4.3 using the packages detailed in the analysis script (Supplemental section 4)

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors/reviewers upon request. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Research guidelines for submitting code & software for further information.

Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A list of figures that have associated raw data
- A description of any restrictions on data availability

Code and source data for all analysis and figures generated during the current study are included in this published article (supplemental sections 4 and 5) and are also available in the GitHub repository at: https://github.com/kessonovitch/BHAI_Data/

Field-specific reporting

Please select the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☐ Life sciences ☒ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/authors/policies/ReportingSummary-flat.pdf

Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	Quantitative endocrine, anthropometric and questionnaire data
Research sample	All participants were recruited from outwardly healthy, non-clinical populations of adult males (aged 16.5-78 years) resident in London, UK or Sylhet, Bangladesh. Men who responded to advertisements or presentations at recruitment sites were screened for diagnosed diabetes, thyroid disorder, or use of steroid medication. Sedentees were additionally screened for relatives who migrated to the UK/ Europe/Canada/USA to ensure they represented a family connection to the migrant community. British European men were screened for both parents born in Europe.
Sampling strategy	Initial target sample size comprising 70 per group was determined using an a priori power analysis for ANOVA (using G*Power) with a specified significance value (α = 0.05), power (1-ß =0.95), and a conventional "medium" effect size (Cohen's "f" = 0.25). Men were recruited through community networks/snowball sampling.
Data collection	Men were interviewed in their native language by trained research assistants or completed questionnaires on their own, depending on their literacy and language abilities. Interviews were conducted in semi-private conditions, typically community centres or private homes with men given the option to discuss sensitive topics in private. All interviewers and researchers were aware of the men's migration status at the time of data collection due to screening requirements. Salivary samples were collected at home by the participants in tubes containing sodium azide or methionine preservative and returned by post or collected by researchers. All laboratory analysis was performed using laboratory ID numbers, with ordering of analysis randomised and no indication of residence group.
Timing	Data were collected between July 2004 and November 2010 in London, with data collected in Bangladesh in 2005 and 2007.
Data exclusions	Prior to recruitment, participants were screened to exclude thyroid conditions or diabetes. First-order relatives were excluded from participation to avoid closely shared genetic or immediate environmental confounders. Participants in Sylhet were screened for relatives who had migrated to the UK, mainland Europe, Australia/New Zealand or the North American continent. Salivary data were excluded according to pre-determined laboratory protocols: duplicate salivary testosterone readings with coefficient of variation greater than 15% were re-analysed or excluded. Of 2018 samples, 39 or 1.9% were excluded for high CV. Duplicate readings of two samples were excluded as both exceeded the limits of detection of the high standard of the assay, four samples were based on single readings due to limited sample or laboratory error of the second reading. Seven outlying samples with z-scores above 3.29 were recoded to +2 SD of the population mean of salivary testosterone for that time point.
Non-participation	Two participants withdrew from the study without specifying the reason and their data and records were excluded from any further analysis. All other missing data resulted from failure to complete the study. A total of 68 (18%) of recruited men did not return salivary testosterone samples, 32 (9%) did not complete the demographic questionnaire, and 39 (10%) did not provide anthropometric data.
Randomization	Groups were assigned based upon developmental exposure to contrasting ecologies, and therefore were not random. Men were allocated into cohorts based on place of birth and age of migration, if applicable. Covariates relevant to our hypotheses were included in all statistical analyses.

Reporting for specific materials, systems and methods

Materials & experimental systems

n/a	Involved in the study
☒	☐ Unique biological materials
☐	☒ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology
☒	☐ Animals and other organisms
☐	☒ Human research participants

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging

Antibodies

Antibodies used	Antiserum to testosterone were raised from a rabbit source in the laboratory of coauthor RTC.
Validation	In-house validation studies indicated cross-reaction of 100% with testosterone, 13% with dihydro testosterone, 0.2% with androstenedione, and <0.1% with estradiol, androsterone and etiocholanoalone. Antiserum was used at dilution of 1:6000

Human research participants

Policy information about studies involving human research participants

Population characteristics	All men signed informed consent declarations and ethical approval was granted by the UCL Research Ethics Committee (ID: 0144/002), and the Osmani Medical College in Sylhet. Participants were compensated for their time (£10 in UK, Tk500 in BD) upon completion of the study. Data were stored in accordance with the Data Protection Act (UK).
Recruitment	Recruitment was in community centres, religious, sporting, educational and business establishments. While a self-selective group may have responded to the advertisements and presentations by participating in and completing the study, recruitment was not targeted to a single subpopulation other than members of the Bangladeshi community in the UK or men of European background living in neighbouring communities. Migrants in general may represent a self-selective group, however there were no indications that men who migrated in adulthood differed from sedentees in the reproductive measures examined here. Age differences in migration cohorts are likely representative of demographic results of peak migration patterns as opposed to self-selection, but demography limits our interpretation of ageing and senescent effects in child and second generation migrants over the age of 40 years.