Reproductive Ecology And Life History Of Human Males:

A Migrant Study Of Bangladeshi Men

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Declaration

I, Kesson Shane Magid declare confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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Abstract

Developmental constraints influence individual energetic apportionment between growth, maintenance and reproduction with long-term implications for health and longevity. Such life-history trade-offs are hypothesised to explain the observed variability of human male and female reproductive steroid levels. Salivary testosterone (salT), anthropometric, and demographic data were collected from: 1) sedentees in Sylhet, Bangladesh (n=107; aged 20-78 years, mean 39); 2) Bangladeshi born men who migrated to London as adults aged ≥18 (n=61; aged 23-76, mean 49); 3) Bangladeshi born men who migrated to London as youths <18 (n=50; aged 18-69, mean 32); 4) British born Bengalis (n=48; aged 18-42, mean 25); and Londoners of white British or other white European parentage from 5) similar socioeconomic background compared to migrant groups (n=58; aged 18-75, mean 41); and 6) higher status socioeconomic background compared to migrant groups (n=30; aged 22-54, mean 37).

SalT and somatic markers of adult Bengalis is dependent upon the age at which they migrated from Bangladesh to the UK and suggests differences in male reproductive phenotype, health behaviours and diet due to changes in ecological conditions during development. These findings contribute to the growing body of evidence that salT, stature and apportionment of skeletal muscle vary in accordance with early life conditions and the strategic allocation of reproductive effort in the human male, with a corresponding increase in early symptoms of adult onset disease of the prostate and glucose metabolism, and low socioeconomic status (SES). Predicted blunting of diurnal salT profile in adult migrants was inconclusive. Contrary to the predictions of this study, Bengali men do not have lower salT in relation to reproductive status of paternity or marriage, while older British-born European men of low SES have higher salT in relation to number of offspring and marital status. British-born Bengalis and migrants who arrived as children under the age 12 years were revealed to be of significantly higher SES than migrants who arrived in London after the age 18, possibly reflecting a generational shift away from historical conditions of poverty within the London Bengali community.

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2NG:	Second-Generation migrant, British-born Bengali
ADM:	Adult Migrant, Bangladesh-born and migrated to the UK aged 18 years or older
AL:	Arm length
ANCOVA:	Analysis of Covariance General Linear Model
AR:	Androgen Receptor
BD:	Bangladesh
BDT	Bangladeshi Taka
BED:	Evening salivary sample
BPH:	Benign Prostatic Hyperplasia
BSF:	Biceps Skin Fold
DAYM:	Averaged daily salivary sample
DHT:	Dihydrotestosterone
DIR:	Diurnal ratio
E_2:	Oestradiol
EHI:	British-born European of high socioeconomic status
ELO:	British-born European of low socioeconomic status
FSH:	Follicular Stimulating Hormone
GBP:	Pound Sterling
GH:	Growth Hormone
GnRH:	Gonadotropin Releasing Hormone
HPT:	Hypothalamic Pituitary Testicular axis
IPSS(+QoL):	International Prostate Symptom Score (+ Quality of Life score)
LH:	Luteinising Hormone
LUTS:	Lower Urinary Tract Symptoms
MeanAM:	Averaged morning salivary samples
MLR:	Multiple Linear Regression
MUA:	Mid-Upper Arm
MUAC:	Mid-Upper Arm Circumference
ONS:	Office of National Statistics
salT:	Salivary testosterone
SES:	Socioeconomic status
T:	Testosterone
TSF:	Triceps Skin Fold
W+30:	Thirty minutes post-waking salivary sample
WAKE:	Waking salivary sample
YOM:	Youth migrant, Bangladesh-born and migrated to the UK before age 18.

A note on terminology as used in this document

Bengali refers to the ethno-linguistic group native to the regions of historic Bengal, encompassing the modern state Bangladesh as well as surrounding regions of the Indian subcontinent.

Bangla refers to the language (and sometimes culture) of the Bengali people.

Bangladeshi refers to the citizens and culture of the state of Bangladesh, though Sylheti refers to both a dialect of Bangla and to the residents of the Sylhet district of Northeast Bangladesh.

Londoni is the term Sylheti sedentees apply to Bengalis living in the UK (usually without regard to whether they actually live in London).

While in practice these terms are commonly used interchangeably by Bengalis living in the UK[1] within this thesis London-born children of Bangladeshi-born migrants will be referred to as "Bengali" or "British-born Bengalis" to distinguish the ethnic and political terminology.

[1] In an informal poll conducted on the British Bengali social networking website www.networkbangla.co.uk in November 2007, I received the following responses to the question, "How would you rank the following in terms of importance how you'd describe your own identity, i.e. no.1 being the most important and 7 being least important? For the poll, just pick the most important then post your full ranking!"

None of the 58 respondents numbered their responses, so if they specified multiple identities, each identity was given a weight of one.

Preferred Self Identity	**N**	**%**
Asian	1	1.3
Bangladeshi	6	7.9
Bengali	10	13.2
British	11	14.5
British-Bangladeshi	5	6.6
British-Bengali	13	17.1
Muslim	17	22.4
Other	13	17.1
Total	76	100

Though "Muslim" was preferred more than any other identity, there was not a single significantly preferred name for cultural identity, and the number of individuals selecting an identity versus those not choosing it was not significant, $X^2(12,6)$, p=.07.

Acknowledgements

Every thesis project is a team effort and this one is no exception, though I am sure there will be inadvertent exceptions from this list. I would like to thank all the research assistants who helped collect the data, in particular David Lawson, Farid Ahamed, Zhaved Selehin, and Raskin Ahmed as well as the rest of the team from SUST. I am grateful to Robert Chatterton and for opening his lab to me, and Shanthi Muttukrishna for her assistance in interpreting assay results. I thank Osul Chaudhury, Lynnette Leidy Sievert, Lauren Houghton, Taniya Sharmeen, Khurshida Begum and Ale Núñez de la Mora, for their support in Sylhet and London. I thank Ben Wright and my UCL colleagues in Reproduction Lunch for stimulating discussion.

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Dedication

Dedicated to the memory of my father Dr. Ken Magid.

Preface

Ecological conditions experienced during development influence adult reproductive function, according to a foundational hypothesis of Human Reproductive Ecology (Ellison 1996). This project contributes to the growing body of supporting evidence that the hormone testosterone (T) varies in accordance with early life conditions and the strategic allocation of reproductive effort in the human male (Bribiescas 2001a; Bribiescas 2006; Kuzawa et al. 2010). Specifically, the salivary T and somatic markers of adult Bengalis are dependent upon the age at which they migrated from Bangladesh to the UK.

Two observations provide the impetus for this project. Firstly, human males exhibit a wide variation in T, between individuals and across populations. Non-industrialised or subsistence populations show lower levels of free T when compared with populations in developed nations (Bribiescas 1996; Ellison et al. 2002). Inter-individual variation in T is greatest between young males, during the period of the lifecourse when reproductive competition is typically considered at its height. Over the whole of the adult lifecourse T is thought to modulate behaviour, immunity and somatic investment. These measures of T are presumed to reflect greater immunological, nutritional or other energetic challenges (Bribiescas 2001; Charnov 1993). Androgen levels of adult males, particularly free T, respond acutely to changes in nutrition, social conditions, physical activity, and immune challenges (Bribiescas 2001; Campbell et al. 2001; Muehlenbein and Bribiescas 2005).

Secondly, the reproductive function of women, as measured by salivary progesterone, appears to be influenced by conditions experienced prior to puberty, and remains unchanged despite improved conditions in adulthood (Núñez de la Mora et al. 2007a). It is unknown, however, whether adult male reproductive function is similarly constrained by childhood conditions, although recent evidence suggests that environmental stressors in the first six months of life influence hormonal and somatic characters in adult males (Kuzawa et al. 2010).

Design of the project

Migrants from Sylhet, Bangladesh to London, UK experience a discontinuous developmental environment, with fewer immune challenges or other presumed constraints on energy balance following migration. In order to understand further such influences on the reproductive hormones of adult men, this project compared salivary testosterone and anthropometric measures of a group of adult Bangladeshi migrants who relocated to London as children (aged <18 years) and as adults (aged ≥18 years) with men of Bengali or European ethnicity resident

all their lives in London, or with Sylheti sedentees. Age at migration acts as an experimental variable in this study in order to observe whether adult patterns of salT variation are influenced by ecological conditions experienced during development.

Structure of thesis

Chapter one introduces the project in three parts. I begin by describing the ecological conditions and background of the Bengalis living in the UK and Bangladesh. Next, I place the project in context with other research and the current state of our understanding of male reproductive development, adult function and behaviour. Finally, I present the theoretical basis for the project and propose hypotheses to test interactions of biological signals measured by hormones with developmental markers, health and behavioural change, and cultural conditions.

Chapter two describes the methods of the project, and presents results validating these methods for this project.

Chapters three through five present the results of the project. They fall into three general, overlapping categories of development, dietary and health behaviours, and social ecology. These three categories frame the hypotheses tested within the three results chapters presented in this thesis.

Chapter three tests developmental hypotheses: If developmental conditions influence investment in persistent structures and response thresholds of hormonal axes and somatic tissue, then the timing of a change in ecological conditions at a point in the lifecourse will measurably influence reproductive function in adult men as well as physical growth and developmental tempo and during childhood. The specific predictions are that Bengali men who spent all or part of their childhood in London will show higher salT, taller stature and will recall reaching sexual maturity at an earlier age than Bengalis who spent their childhood in Sylhet.

Chapter four tests hypotheses related to diet and health. Regarding dietary and health behaviours, if men live in Sylhet all their lives, do they report nutritional stress? Do men who migrated to London as adults consume similar diets as sedentees? Does childhood acculturation to life in London influence Bengali dietary and health behaviours? The predictions are that men from Sylhet are not nutritionally stressed, that the Bengali diet in London is similar to that of sedentee counterparts, with less consumption of fish and more consumption of other meat. Bengalis who spent all or part of their childhood in London will show greater similarity in their patterns of dietary and health behaviours to SES-matched British European men compared with Bengalis who did not migrate to London as children.

Regarding ecological conditions and health, if adult onset diseases are related to a mismatch between early life developmental conditions and adult ecology, does migration after key stages of development mean migrants are more prone to symptoms of prostatic disease and dysregulation of glucose metabolism? Proximately, if men have high salT, are they more likely to report more LUTS than men who have low salT? The prediction tested is that Bengalis who migrated London after childhood will report more lower urinary tract symptoms (LUTS) than British-born Bengalis, and Bengalis who migrated as children. Finally, do dietary and health behaviours adequately explain Bengali inter-population variation in measures of salT tested in chapter three?

Chapter five tests hypotheses based on social ecology and male reproduction. Regarding socioeconomic positioning, if a male is of high SES relative to current surrounding ecological conditions, does he divert more effort toward reproductive function than men of low SES, relative to current ecological conditions? Is current relative SES more influential on reproductive effort of men in the latter half of their reproductive stage of life, compared to men in the first half of this stage of life? The specific predictions are that high SES males have higher salT, and that relative SES is more highly associated with salT in men aged 40 years or older. Finally, do SES, dietary and health behaviours adequately explain Bengali inter-population variation in measures of salT tested in chapter three?

Regarding relationship and reproductive status, if different ecologies during childhood development determine coordination of male endocrine function and the social relationships of pair-bonds and offspring, do men who are exposed to Western influences of acculturation during childhood development exhibit greater reduction in reproductive function if they are married or married with children as compared to men who were less exposed to such influences? The specific prediction is that Bengalis who spent all or part of their childhood in London and British European men will show lower salT if they are married or have young children than Bengalis who spent all of their childhood in Sylhet.

Chapter six draws conclusions from the findings of the project as a whole at two levels of inquiry. Proximately, how does the functioning of reproductive organs and hormonal axes interact with developmental history and current surroundings? Ultimately, how do these results reflect the balancing of the competing biological functions of survivorship and reproductive effort has been shaped by natural selection? These principles extend to the field of evolutionary medicine, where trade-offs of investment between competing physiological requirements explain senescence and disease.

Chapter 1: Introduction

This project tests for evidence of adaptive allocation of reproductive effort in the adult human male across the life course, based on evolutionary hypotheses. The hypotheses are structured by contrasting conditions within three variables: ethnicity, ecology, and developmental phase. The first contrast is between two ethnic groups, the Bengalis and British-born Europeans. The second contrast is between the ecologies of Sylhet, Bangladesh and London, UK. The third contrast is between six key phases of development: pre-birth, infancy, childhood, adolescence and early and late adulthood.

In this chapter I review each of these conditions in turn. I begin with the history of Bengali migration to the UK and a brief summary of the community's current demographic and socioeconomic characteristics, and how these differ from their sedentee counterparts and British-born Europeans. Next, I describe features of the ecologies of Sylhet and London relevant to this project. Then I review the current understanding of human reproductive development and how ecological or genetic interactions with development determine adult reproductive function.

After reviewing the contrasts, I move to the outcome variable, adult reproductive function. Finally I review how variation in male reproductive function is assessed through hormonal, somatic, and lifestyle measures, and then propose hypotheses based on life history theory variables to test for predicted differences in male reproductive function.

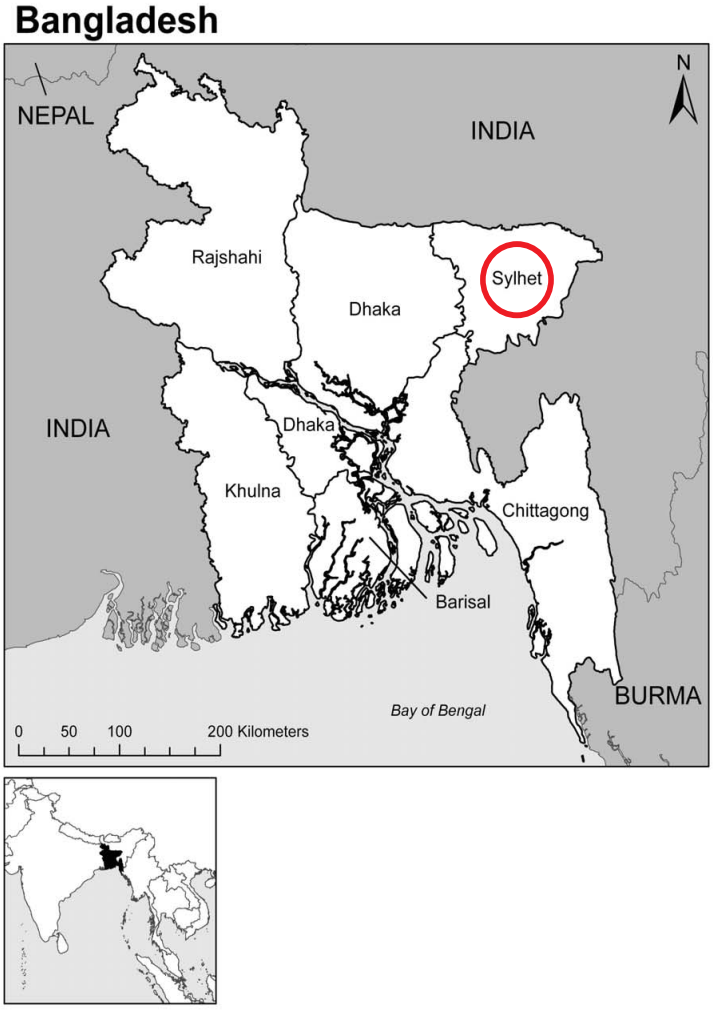
I propose three general categories of hypotheses to be tested in three results chapters. The first category tests if developmental conditions influence investment in persistent structures and response thresholds of hormonal axes and somatic tissue depending upon timing of a change in ecological conditions. The second category of hypotheses test whether Sylheti dietary and health behaviours are conserved in migrants, and whether exposure to the ecology of London during childhood influences conservation of these behaviours. In addition, if adult onset diseases are related to a mismatch between early life developmental conditions and adult ecology, does migration after key stages of development mean migrants are more prone to symptoms of prostatic disease and dysregulation of glucose metabolism? The final category of hypotheses concern social ecology and male reproduction. Regarding socioeconomic positioning, if a male is of high SES relative to current surrounding ecological conditions, does he divert more effort toward reproductive function than men of low SES, relative to current ecological conditions? Is current relative SES more influential on reproductive effort of men in the latter half of their reproductive stage of life, compared to men in the first half of this stage of life? If different ecologies during childhood development determine coordination of male endocrine function and the social relationships of pair-bonds

and offspring, do men who are exposed to Western influences of acculturation during childhood development exhibit greater reduction in reproductive function if they are married or married with children as compared to men who were less exposed to such influences?

1.1 The Bengalis

The Bengali community provides the ethnographic basis of this research due to their unique multi-generational history of migration to the UK, which allows for recruitment of participants across age categories and country of birth. Bengalis in the UK form a geographically-concentrated and culturally cohesive migrant community originating from a single, homogenous population, >95% of whom are descended from the land-holding middle-class of Sylhet, a distinct region of modern-day Northeast Bangladesh (See fig 1) (Eade et al. 1996; Siddiqui 2004).

Figure 1: Map of Bangladesh



Adapted from Bangladesh Demographic and Health Survey (NIPORT 2009)

On average, Bengalis in Britain are significantly younger and more ethnically segregated than other South Asian minorities or the British population as a whole (DCLG 2009), and together with Pakistanis are the poorest major ethnic minority in Britain (ONS 2002).

In this section I will review the history of Bengali migration to the UK, how this history contributed to their current demographic and socioeconomic characteristics. These characteristics form the basis of contrasts and assumed consistencies between the migrant and sedentee populations, and between migrants and their ethnically-European London neighbours.

The UK Bengali community has a long history of immigration to East London, with the earliest settlers arriving in the late 1940s and are now entering the third generation of regular migration (Eade et al. 1996). The UK Bengali community originates from the Sylheti middle class for a number of historical reasons.

Unlike the rest of present-day Bangladesh, Sylhet was included within the Assam province of British India, which allowed Sylheti landholders to acquire wealth by consolidating cultivation into lucrative export crops like tea, unlike the tenant farming of neighbouring Bengal province (Banglapedia 2010). This led to the formation of a socioeconomically distinct landholding class in Sylhet (Gardner and Shukur 1994). The male offspring of these families were not required to work the land and had the economic means to migrate and connections to the shipping industry, so many joined the steamship trade in Calcutta (Alexander et al. 2010; Gardner 2002).

The first Bengalis to arrive in significant numbers in London were “lascars”, teams of men employed on steamships serving the British Empire up to the 1960s (Al-mahmood 2008). Many of the contracts were one-way, so those who stayed behind remained in the docklands of East London, where they maintained close links to Sylhet and uncertain futures in London (Alexander et al. 2010; www.portcities.org 2010). Those that found work or started businesses in East London did so from relatively unskilled capacities as textile labourers in the “rag trade”, small shops or the first Indian restaurants, the latter remains an important source of income and employment within the Bengali community to this day (Carey 2004).

The 1962 passage of the first UK Commonwealth Immigrants Act restricted migration of unskilled labour from East Pakistan, which included Sylhet following the 1947 partition of India (a period of great social and political upheaval in the region, see ecology section below). Bengali migrants required vouchers, which were set to decrease in number each year following the Act. Despite these limitations, this was the period when the bulk of first generation male migrants arrived through networks of Sylheti relatives and friends based in the UK to work in the developing restaurant business or factories. The UK Bengali population

is estimated to have increased ten-fold in the period between 1960 and 1970s (see figure 2), and the vast majority of these arrivals were men (DCLG 2009). Many of these arrivals continued to support families in Sylhet, splitting their time between the two countries (Gardner 2002). Shifting economic cycles and the violent Bangladeshi War of Independence of 1971 prompted many of the Bengalis living in the UK to bring their female family members and children, and further restrictions of migration laws in the 1980s meant only family members could migrate to the UK (Alexander et al. 2010; Dench et al. 2006; Gardner and Shukur 1994).

This led to a period of "chain" migration when most of the migrants were wives and children of the original "voucher" migrants. The average age of a Bengali wife is about 10 years younger than her husband (Mitra et al. 1997). The age profile of the UK Bengali population shifted downward as a demographic consequence of these "chain" migrants. By 2001 there was a balanced sex ratio between the ages of 15-29, but 50% more men in the 30-44 age range (UK Census data, cited in Alexander, 2010). Almost 1/3 of Bengali migrants had arrived in the UK after 1985, and most of them were women and adult children of first generation migrants (Ahmed 2005).

The timing of this "chain" migration shapes the present day contrast in socioeconomic profile of Bengalis living in London and Sylhet.

Bengali migration peaked between 1980-1985, ten years later than peak migration flows from present-day India and Pakistan (1975-1984). As this Bengali migration was for family reunification purposes, it did not coincide with a period of economic prosperity, which had allowed for rapid economic integration and a higher level of employment for other South Asian migrants. This may explain the legacies of underemployment and high level of "blue collar" employment (Ansari 2004; Peach 1999).

Lack of opportunity and cultural resistance to women working outside the home means that Bengali families have the lowest participation rates for females in the labour force (22%), highest male unemployment rate (32%), highest average family size (4.7 persons) of any British ethnic minority (Dunnell 2008; UK Office for National Statistics 2005b). The young age structure, tendency to have only one wage-earner, high unemployment and large average family size has led to a dependence upon social housing. In the 1980s the largest concentration of housing was in blocks of flats in Tower Hamlets, where the dockland industry of the borough had been recently removed and economic activity had collapsed. This legacy of deprivation helps explain the present socioeconomic conditions of the UK Bengali community (Eade et al. 1996), which will be described in further detail in the Ecology section of this chapter.

Figure 2: Estimated UK Bengali population

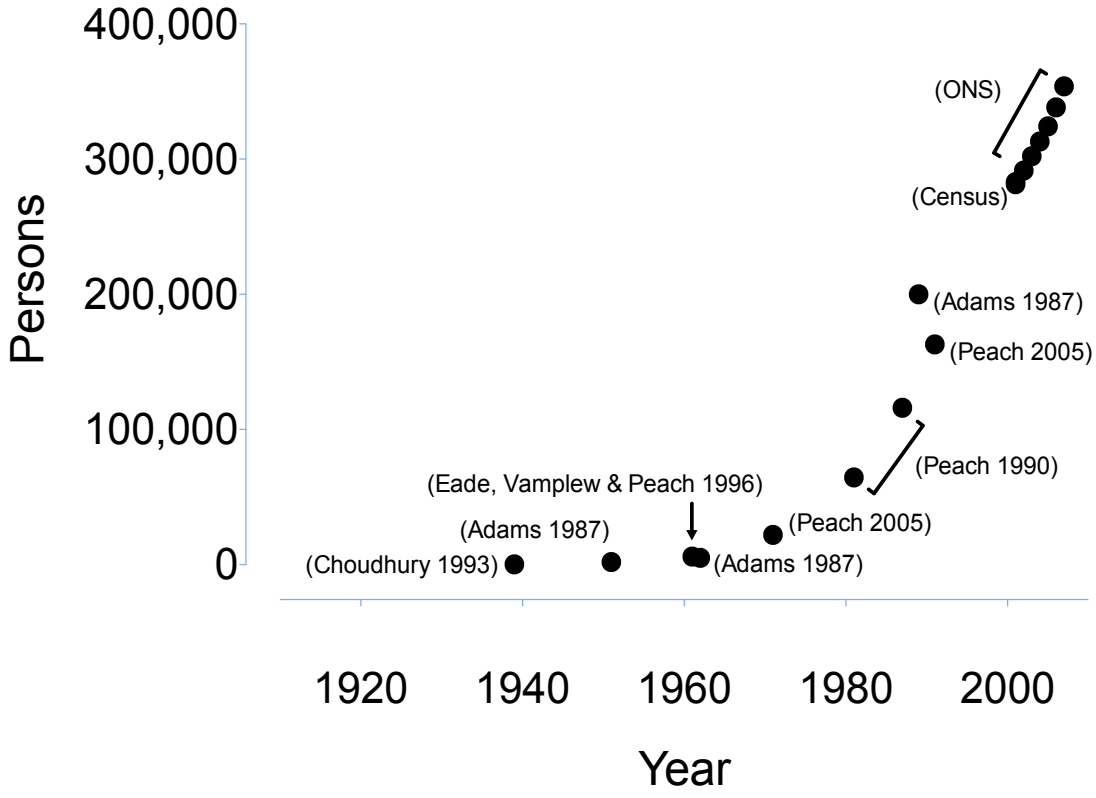


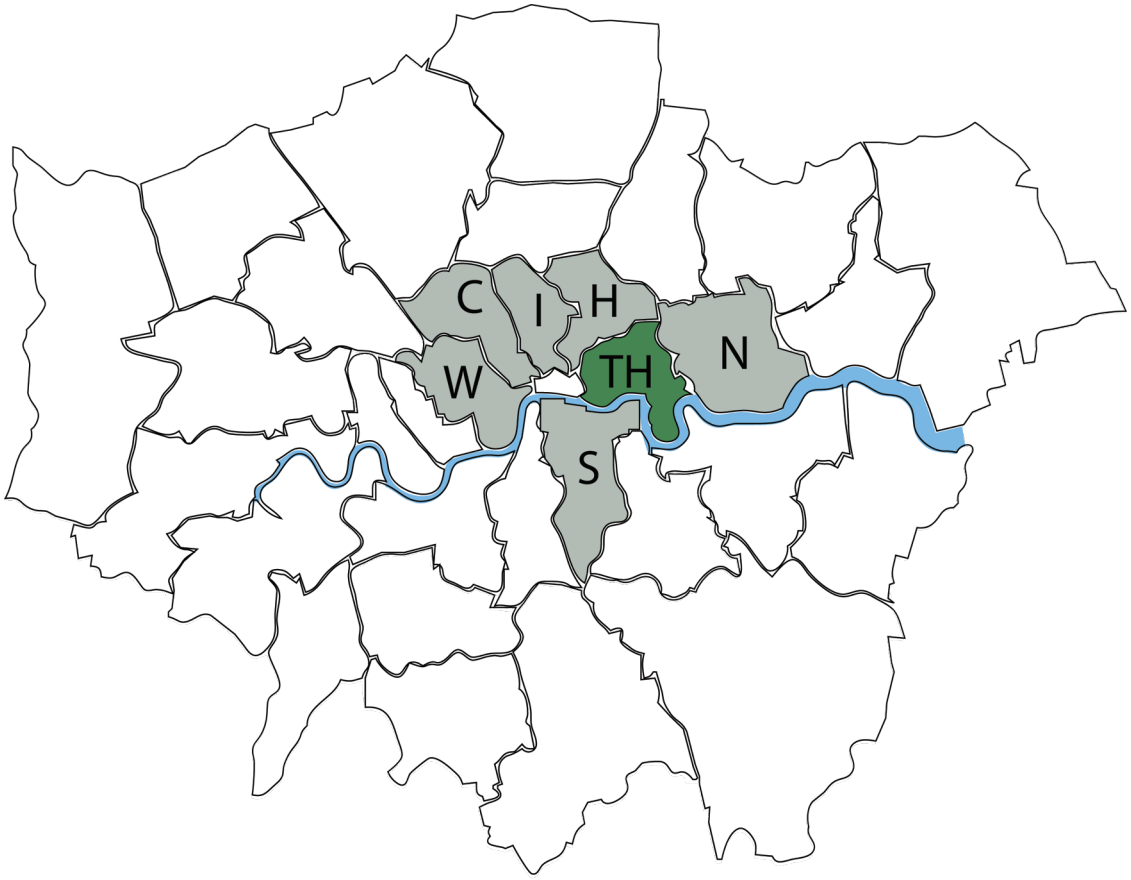
Table 1: Estimated UK Bengali population

Source	Year	Estimated population in UK
(Choudhury 1993)	1939	200
(Adams 1987)	1951	2000
(Eade, Vamplew & Peach 1996)	1961	6000
(Adams 1987)	1962	5000
(Peach 2005)	1971	22000
(Peach 1990)	1981	64561
(Peach 1990)	1987	116000
(Adams 1987)	1989	200000
(Peach 2005)	1991	163000
(Census)	2001	283063
(ONS)	2001	281500
(ONS)	2002	291600
(ONS)	2003	302100
(ONS)	2004	313100
(ONS)	2005	324300
(ONS)	2006	338300
(ONS)	2007	353900

The migration history detailed above has led to the UK Bengali community's present social geography of interconnected Sylheti families centred around Tower Hamlets and forming an inner ring of London boroughs running from Westminster, Islington and Camden on one side and Hackney, Newham and Southwark on the

other (see figure 3). The kin networks are so regionally-specific that many of these London neighbourhoods correspond to 11 sub-districts of the Sylhet region (Eade and Garbin 2006). In the 2001 census, approximately 42% (120,000) of the UK Bengali population lived within this inner London ring (Garbin 2005).

Figure 3: Areas of London with large Bengali populations



London Boroughs:

W - City of Westminster
C - Camden
I - Islington
TH - Tower Hamlets
H - Hackney
N - Newham
S - Southwark

While the position of Bengali migrants in London is historically one of economic deprivation relative to their surroundings, the wide economic differential between the UK and Bangladesh and a tradition of remittances has led to a highly localised "geography of prosperity" among the relatives of migrants living in Sylhet, and maintained a high standard of living compared to other Bangladeshis (Gardner 1995). Remittances from the migrant *Londoni* community support extended family networks and fund investment in apartment buildings, shopping malls, and other businesses in Sylhet (Buerk 2005). The practice of sending remittances appears to be in decline, however, especially among second and third generation migrants (Alexander et al. 2010; Eade and Garbin 2006).

The London neighbourhoods where the Bengali migrants arrived were traditionally white and working class areas of the East End. The housing needs of the new

immigrants created competition for housing and other resources, likely contributing to the rise of racial conflict in Tower Hamlets in the 1980s (Alexander et al. 2010; Dench et al. 2006). While much of the white population moved further east to suburban Essex or other regions of London, the white working-classes of East London remain well-matched as a control group of non-ethnic Bengalis living under similar ecological conditions.

The migration history of the London Bengalis supports an assumption of ethnic homogeneity and comparable genetic admixture when comparing the migrant and sedentee populations, key to building hypotheses for this project. This history has essentially maintained two intact branches of the Sylheti family tree, living within the contrasting ecologies of London and Sylhet. I now turn to the ecological differences and consistencies of these respective locations.

1.2 Ecological conditions

Human migration and settlement gives insight into how individual hormonal profiles respond to profound external changes in culture and environment, here referred to as “ecologies”. Migration between two ecologies will have a different effect depending on the point in the lifecourse when an individual relocates (Warnes 1992). In this section I describe salient features of both ecologies, the contrasting SES and consistencies of dietary and cultural conditions of Bengalis living in London and Sylhet, and the ecological risk factors facing males born and raised in either location.

Socioeconomics

Bengalis are the most recent, youngest, poorest, most underemployed and, by measures of education housing and health, the most disadvantaged Asian immigrant group in London (Eade et al. 1996; Garbin 2005; ONS 2002).

Households are traditionally married couple families with all relationships contained within the ethnic group (Peach 1999). Families are large, sometimes extended, and living conditions are cramped, with the highest levels of overcrowded housing (1.5 or more persons per room), of any ethnic group according to the UK ONS (2005) (Kempson 2000).

The Bengali community is highly dependant upon social housing, a rate 3 times that of the total population, and owner-occupation is 38%, (less than half the London average) and the majority of households live in social sector rented accommodation (Peach 2005).

As an ethnic group, Bengalis have the highest unemployment rates in Britain (20%); four times that of White British men. Bengali males have the lowest rates of

economic activity (61.7 %), and for two fifths of these men, it is due to being long-term sick or disabled (UK Office for National Statistics 2005a). More Bengalis fall into the category “never worked or long-term unemployed” than any other ethnic group (17.1% compared to 2.7% of all people) (Peach 2005).

Bengalis who are employed work mostly (65%) in the hotels and catering industry, representing the particular reliance upon the Indian restaurant trade (Carey 2004). Of Indian restaurants in the UK, an estimated 85% are owned by Bengalis (Carey 2004).

There is not a tradition of educational attainment in the community Forty percent of Bengali men do not possess any qualifications, the highest rate of any UK ethnic group are the most likely ethnic group to be unqualified (UK Office for National Statistics 2002; UK Office for National Statistics 2005a).

While the reliance upon low-skilled employment and limited educational attainment have historically contributed to poverty in the community, there are signs of generational shifts toward an improved educational and professional achievement in third generation Bengalis (Economist 2007; Sunder and Uddin 2007).

In contrast, the sedentee community is of a traditionally landholding class of high socioeconomic position relative to the surrounding population (Gardner and Shukur 1994; Siddiqui 2004).

Diet

There is no indication that Sylheti sedentees of the middle classes from which the migrants originate are currently nutritionally stressed. It is important to distinguish this population from the well-documented poor and nutritionally-stressed populations of Bangladesh (Brown et al. 1982; Koenig et al. 1990; NIPORT 2009).

Diet is highly conserved within the migrant community. Most meals are prepared and consumed in the home, with imported foods from Bangladesh widely available from specialist shops throughout East London, and the observation of halal dietary restrictions has buffered the adoption of new dietary practices in the UK (Núñez-de la Mora et al. 2004).

While previous work suggests there may be increased consumption of meat proteins in the migrant population, and reduced fish consumption among young migrants and British-born Bengalis, migrants report shopping at markets specialising in Bangladeshi produce and purchasing imported foods regularly. They report frequent consumption of Asian main meals and consuming ‘western’ foods only rarely or occasionally (Núñez-de la Mora et al. 2004). Western-style

fast food is also increasingly available in Sylhet and consumption patterns of fried foods and sweet snacks among younger sedentees and migrants may be contributing to increased diabetes and obesity risk in developing nations like Bangladesh (Yach et al. 2006).

Ethnic and cultural homogeneity

Islam and village tradition, combined with a compact social geography, extensive family networks and marginal economic integration keep Bengalis culturally distinct and socially closed off from other ethnic groups in London (Lieberson 1963). Peach (1999) parallels the "encapsulation" of the London Bengalis to the Hasidic community of Williamsburg, Brooklyn. The households are traditionally married couple families with all relationships contained within the ethnic group. Levels of intermarriage are extremely low, at 3%, they are lower than for any other ethnic group in Britain (Dunnell 2008).

In Britain, the ethnic and cultural homogeneity of the Bengalis appears to be continuing to be maintained trans-generationally (Eade 1994). In fact, disengagement of the community may be increasing among British born Bengalis as they embrace a more fundamentalist Islamic identity than their parents (DCLG 2009; Hussain 2007).

These observations are supported by Indices of Dissimilarity, as calculated from Local Base Statistics (ESRC 1991 census holding, University of Manchester Computer Centre). According to this measure, Bengalis are the most segregated ethnic group in Britain (Eade et al. 1996). Bengalis are segregated from fellow South Asian or other migrant ethnic groups, and are more segregated from Indians than they are from Whites, they are equally as segregated from Pakistanis as they are from Whites.

Ecological risk

Compared with migrants in London, sedentees are subject to greater ecological risk factors from infectious disease and environmental instability (i.e. political unrest, periodic flooding, poor public health and sanitation), Adult and childhood life expectancy is much lower in Bangladesh for all socioeconomic groups, with under age five mortality for those in the top quintile of income in Bangladesh still 12 times that of the UK average. For males born in Bangladesh, under-five mortality is 81 per 1000 live births and for those in the highest wealth quintiles, this figure was 72 (both sexes) (Kabir and Islam 2001). In the UK the male under-five mortality has national rate of six per 1000 (WHO 2006a).

For males resident in Bangladesh, immune factors place a considerable constraint upon growth and development. Disease burden, sanitation and public health

are areas of considerable contrast between the two ecologies of Sylhet and London (Howard and Bartram 2003; NIPORT 2009; Heitzman et al. 1989). These ecological factors are influential across social and economic boundaries.

Unsafe disposal of solid waste and poor municipal sanitation mean there is a very high exposure to water-borne pathogens in Sylhet (Alam et al. 2006a). A 2003 study of water quality in Sylhet detected unsafe levels of coliform bacteria in the two main sources of drinking water for residents, the Surma River and tube wells, as well as in 100% of the drinking water served in restaurants, indicating high risk of bacterial gastroenteritis (Alam et al. 2006b; Iqbal et al. 2006).

Drinking water in the Sylhet district also contains high levels of arsenic: where more than 50% of the tube wells exceed WHO safety guideline of 0.01 mg/litre, and 29.3% exceeded the 0.1 mg/litre level (Howard and Bartram 2003; WHO 2007).

Infectious diseases at high prevalence in Bangladesh include bacterial diarrhoea, hepatitis A and E, typhoid fever and leptospirosis.

In the case of diarrhoeal infection, susceptibility appears to cut across demographic categories in both children and adults (Mitra et al. 1997; Stanton and Clemens 1987).

By international standards both child and adult mortality risk from infectious disease in this region is high. Early life exposure to these ecological stresses leads to a high rate of infant mortality (WHO 2006; Ezzati et al. 2002).

Infectious diseases are the largest cause of childhood death in Bangladesh, with diarrhoeal diseases causing 20% of non-neonatal death in children under-5 years in Bangladesh, followed by pneumonia (18%)(Ahmed et al. 2009; WHO 2006).

While the national figures are likely skewed by children born into poverty, a study of infant mortality in Bangladesh found maternal education and other measures of economic status reduced infant mortality, but were still much higher than observed values for women living in inner-city London (Kabir and Islam 2001).

The mean years of life expectancy at birth for ethnic Bengali males living in England is 74.4, for white British males, it is 76.2. In comparison, the life expectancy at birth for men living in Bangladesh is 62. Adult mortality is 2.46 times higher for Bangladeshi men than for men in the UK (251 to 102 per 1000, respectively) (WHO 2006a; WHO 2006b).

Over the lifetimes of the men studied, those living in Bangladesh experienced acutely stressful events in ways that would not have been experienced by migrants living in the UK. Bangladesh has been the site of political and natural disasters over the last half century. Social upheaval following partition of India in 1947, a

period of conflict with West Pakistan leading to the War of Independence in 1971, numerous floods and cyclones have led to periodic food shortages, devastation, and hardship for the population. Even during periods of relative political stability, living in Bangladesh carries greater risks and is less predictable than in the UK, with the limited state infrastructure or access to health care, unreliable power supply, and high rates of accidental death by drowning and injury (Giashuddin et al. 2009; Linnan and Centre 2008; Rahman 2005). For instance, reported road traffic deaths and injuries in Bangladesh are, respectively, 30 and 50 times those in the UK and the figure is estimated to be vastly under-reported in Bangladesh (Rahman 2005; WHO 2009).

Men within the UK Bengali community are at high risk of adult onset diseases of non-insulin-dependent diabetes mellitus (NIDDM) and heart disease, and associations between lower SES and poor health likely contribute to the level of risk in this community (Balarajan and Raleigh 1997; Bhopal et al. 1999; Marmot 2006).

1.3 Development and reproductive function

Having introduced the characteristics of the ethnic Bengalis, and the ecological conditions where they live, the remaining variable of relevance to this life history analysis of their reproductive function is developmental timing. The project split the male life span into six key phases of development: pre-birth, infancy, childhood, adolescence and early and late adulthood.

The points in the life course at which ecological conditions modulate male reproductive function are unclear, in part due to the difficulties in determining whether adult steroid levels are a product of current or developmental conditions. Sex steroids are crucial to organisation as well as regulation of adult reproductive processes (Forest 1983). The former are relatively irreversible (e.g. sexual differentiation or pubertal timing), while the latter may fluctuate in response to current environment throughout life (e.g. spermatogenesis, fat deposition).

“Programming” refers to finite periods of development when the long-term organisation of a physiological system is sensitive to environmental stimuli (Lucas 1994). Sensitive periods of organisation preceding sexual maturity shape the adult reproductive phenotype of humans and other mammals (Davies and Norman 2002).

Migration between two contrasting ecologies, with age at migration as an experimental variable, allows for the testing of predictions of how ecological conditions experienced during key developmental stages lead to variations in adult physiological characteristics (Greulich 1958; Lasker 1995). In this project,

the physiological variable of interest is the organisation and regulation of the hypothalamic-pituitary-testicular (HPT) axis.

I begin with an outline of the components of the HPT axis, before moving on to discuss its role in the development of the human male at the key points of importance to the design of this project.

The HPT axis regulates male reproductive neuroendocrine activity, and consequently, adult patterns of androgen variation. All the components of the axis interact through agonistic and antagonistic feedback loops, in order to modulate sex steroid and gonadotropin production and spermatogenesis. Beyond the hormones directly regulating male reproductive physiology, the HPT axis is sensitive to other hormones such as cortisol, leptin, and thyroid hormone. As androgens, in particular T, influence somatic systems, this sensitivity to other hormonal factors is important for coordinating male reproductive and somatic functioning.

The HPT axis primarily communicates through 6 major hormones: the sex steroid testosterone, two gonadotropins, luteinising hormone (LH) and follicular stimulating hormone (FSH), two cytokines, inhibin and activin, and the small neurosecretory peptide gonadotropin releasing hormone (GnRH).

LH and FSH are manufactured by and released from the pituitary gland to stimulate the two major testicular functions: androgen production and spermatogenesis. These two functions are anatomically compartmentalised within the testes into intratubular and extratubular regions. These regions contain two specialised and separated cell-types, Leydig and Sertoli. LH stimulates Leydig cells to produce and secrete T (steroidogenesis). The extratubular Leydig cells produce the majority of the body's T (in males, 95% of circulating testosterone is of testicular origin) (van Houten and Gooren 2000). Because lipid-soluble steroids can easily pass through cellular boundaries, the T produced by the Leydig cells passes through the barrier between the extratubular and intratubular regions to bind with androgen receptors within Sertoli cells, where spermatogenesis occurs. FSH acts upon Sertoli cells to initiate spermatogenesis and to secrete inhibin or activin. These cytokines have a broad range of effects, influencing Leydig cell T production, and pituitary FSH secretion. Both FSH and LH are regulated by pulsatile (meaning secreted rhythmically) release from the hypothalamus of GnRH. In turn, circulating T exerts negative feedback upon GnRH and the pituitary gonadotrophins. Due to its wide number of targets and differential effects within the HPA axis, T functions as a primary messenger between the testes and the brain.

Figure 4: Diagram of the hypothalamic-pituitary-testicular axis

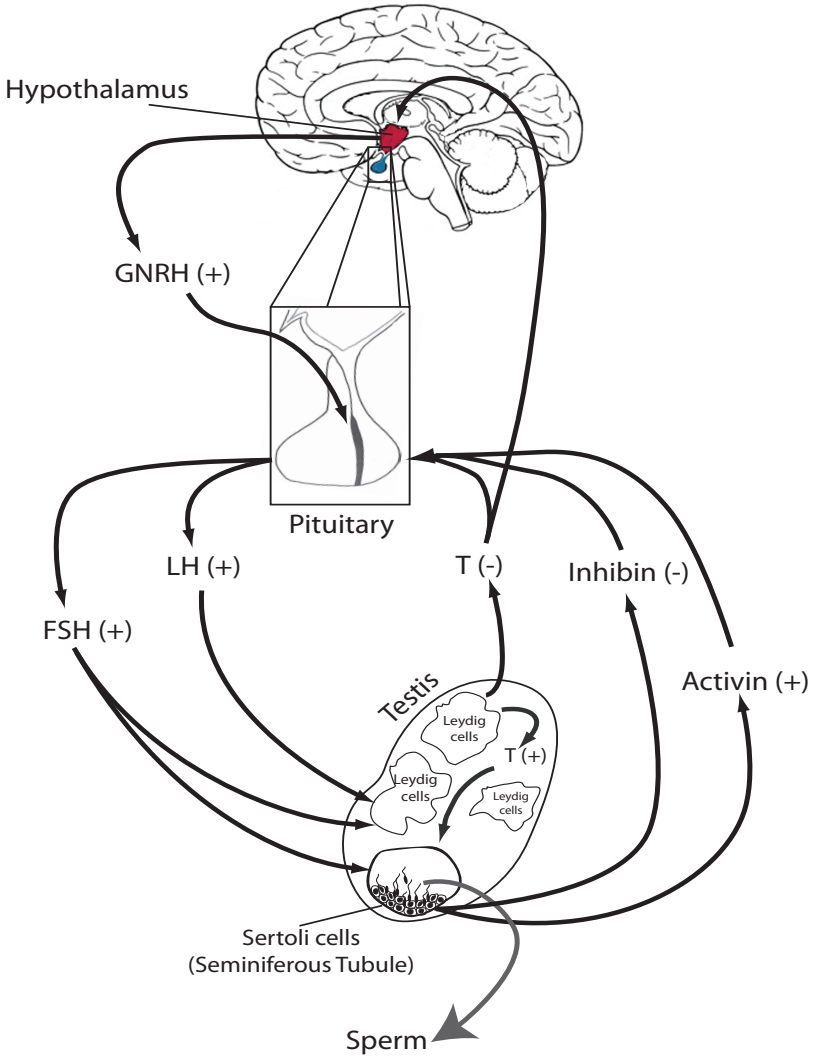


Figure 4: Diagram of the hypothalamic-pituitary-testicular axis illustrating sites of secretion of steroids and gonadotropins, the negative (-) and positive (+) feedback loops regulating other components of the axis and spermatogenesis. (Adapted from Griffin, 1996)

Key stages of development

This project divides the male life history into six developmental stages importance to the alignment of adult somatic and reproductive function. These stages are 1. Pre-birth 2. Early infancy (from birth to age 2 years) 3. Mid-childhood (age 3-12) 4. Adolescence/puberty (age 13-18) 5. Early adulthood (age 19-39) 6. Later adulthood (age 40 years and older). The periods of organisation critical to adult reproductive function within each of these stages will be described below.

Maternal investment and pre-birth factors: Prior to conception, maternal condition dictates the environment of foetal development (Ounsted et al. 2008). A developing male will be subject to environmental constraints or stressors as filtered by maternal intra-uterine conditions, and phenotype will be responsive to early genetic and developmental organisation (Grjibovski et al. 2004). Intra-

uterine investment will in turn reflect maternal ecological and biogenetic history, for example the persistence of lower birth weight of British-born South Asian mothers (Leon and Moser 2010). Maternal age, as well as the spacing and number of any previous births may also influence maternal investment (Fessler et al. 2005; Jasienska 2009).

In recent years, mounting data indicate paternal genomic imprinting or other as yet unknown mechanisms of transgenerational transmission may influence male development. Fathers subjected to environmental stress during critical windows in their development appear to influence the phenotype of their offspring (Pembrey 2010; Pembrey et al. 2005).

Hormonal adjustments in response to intra-uterine conditions are thought to condition the foetal hypothalamic pituitary gonadal (HPG) axis in ways that endure into later life. Inter-uterine adjustments of the human HPG axis explain correlations between low birthweight and the timing or duration of pubertal development (Delemarre-van de Waal et al. 2002; Hernández and Mericq 2008) or variations in levels of salivary oestradiol in adult women (Jasienska et al. 2006) and serum LH and T in adult men (Cicognani et al. 2002; but see also Meas et al. 2010). Experimental evidence from other mammals links restricted foetal nutrition and endocrine disruptions to enduring alterations of HPG functioning (Rhind et al. 2001).

While there is supportive evidence that foetal conditioning influences female fecundity in animals and humans, the lifelong alterations of androgen profiles due to foetal conditioning do not appear to affect male fecundability, that is, male gonads produce adequate numbers of spermatocytes for reproduction in spite of wide variations in androgens (Davies and Norman 2002). While adult reproductive functioning of the male gonads does not appear significantly affected by inter-uterine conditions, early developmental energetic conditions have been hypothesised to relate to T-dependent somatic apportionment in adult life (Bribiescas 2001a; Ellison 2003).

Beyond the development of foetal reproductive organs, another androgen-sensitive tissue, skeletal muscle plays an important role in developmental energetic allocation strategies. The ratio of muscle tissue to fat is acutely reflective of foetal metabolic constraints. Under conditions of adequate maternal nutrition, the developing foetus absorbs and sequesters glucose through foetal insulin. The glucose required for growth is deposited in the liver and muscle tissues in the form of glycogen, all additional glucose is diverted by foetal insulin to into fat stores (Johnson and Everitt, 2000). This trade–off holds lifelong implications for metabolic function and somatic investment (Bribiescas 2001a).

Under conditions of maternal nutritional stress, glucose is diverted preferentially toward growth at the expense of adipose storage. Muscle tissue both consumes and stores glycogen. The energetically expensive developing brain lacks glycogen stores, thus it is wholly reliant upon circulating glucose levels (Johnson and Everitt 2000). Considerable evidence suggests muscle tissue under such conditions develops insulin resistance in order to shunt glucose and sustain the growth of the brain (Campbell and Cajigal 2001; Ozanne and Hales 1999; Reaven 1998). Such conditions would arise more commonly in humans than in other species, as the human brain requires an extraordinary amount of developmental resources. This muscle insulin resistance may lead to systemic decreased insulin sensitivity in later life, with health consequences (Gluckman et al. 2005).

Androgens play a crucial organisational role in foetal masculinisation. The sex-determining region of the Y-chromosome prompts differentiation of the embryonic Sertoli and Leydig cells, forming the essential endocrinological and physical structure of the testis by the 8th gestational week (GW) (O'Shaughnessy and Fowler 2011). Leydig cells in the human testis actively secrete androgens from at least 8-10 GW onwards. In most mammals, including humans, this initial testicular organisation and functioning is independent of the HPG axis, which does not develop until approximately 26 GW (Beck-Peccoz et al. 1991). This pituitary-independent phase of Leydig cellular function coincides with the most critical period of foetal masculinisation (O'Shaughnessy and Fowler 2011). During the “masculinisation programming window” of 8 to 12 GW in the human (Welsh et al. 2008, Scott et al. 2009), testicular hormones actively divert anatomical development of the embryo from female to male line, without which the precursors to male reproductive organs atrophy and spontaneously regress (Johnson and Everitt 2000).

These HPG axis-independent foetal Leydig cells form a distinct population from those of the mature male testis (“biphasic”), and possibly those of the neonatal testis (“triphasic”) (Prince 2001), both of which are reliant upon pituitary gonadotropins to function. This means the cellular source of the foetal peak in testosterone is distinct from the two later neonatal and pubertal peaks. There is limited evidence as to the sensitivity of the foetal Leydig cells to ecological stressors or inter-uterine conditions during this masculinisation window. But it represents a critical period when the early formation, density and number of foetal Leydig cells potentially alter lifetime reproductive functional capacities and regulation through priming the developing components of the HPT axis and migration of stromal cells of the prostate (Bierhoff et al. 1997).The influences of pre-birth conditions (as measured by birth weight) upon adult male metabolism are well-documented, and appear linked to reproductive function (Hales and Barker 2001; Kuzawa 2007). Debate surrounds whether these phenotypic adjustments to intrauterine conditions result

from evolved predictive cues to match foetal phenotype to future environment, constraints from the mother to maximise maternal fitness, or are merely making the "best of a bad start" (Gluckman et al. 2005; Jones 2005; Kuzawa 2005; Wells 2010a).

Genetic developmental effects will include those factors that define limitations of plasticity or adaptations to environment shaped over evolutionary time, presumably including adaptations to famine (Neel 1962). Though the actual identity of "thrifty genes" remains somewhat elusive (Prentice et al. 2005), conditions of frequent famine in the Indian subcontinent (Maharatna 1996; Sen 1977) have been proposed as selective for the so-called "thin-fat baby" phenotype (Yajnik et al. 2002). This describes similarities among South Asian infants of low birth weight, greater central adiposity, and metabolic efficiency during rapid postnatal growth (Yajnik 2004).

While assuming Bangladeshi migrants to London and those living in Sylhet share a common biogenetic history, early interactions between genes, maternal cues and ecological variables may be especially finely tuned in these populations, with rapid responsiveness to changes in environment that may have lifelong repercussions for adult reproductive health.

Early infancy (0-2 years)

The extreme dependency of the human infant means the early post-natal environment remains highly buffered by maternal investment. This investment may be modulated by stresses upon the mother, disease load, number and demands of siblings, and cultural influences upon care or duration of breastfeeding (Mace and Sear 1997; Núñez-de la Mora et al. 2005). Contrasts in the biocultural environments of Sylhet and London will influence some, if not all of the above conditions (Núñez de la Mora 2005).

As the pre-weaning period carries the greatest risk of mortality over the human life-course (Jones 2009), there has been enormous selection pressure on the modulation of neonatal growth to maximise survival and accurately interpret signals of maternal condition and environmental risk (Kuzawa 2005; Wells 2006).

This period of dependency coincides with rapid growth of the infant brain and deposition of fat reserves. In addition, earlier investment in foetal growth (or lack thereof) means this is the period of rapid "catch up" growth in low birth weight babies, which has been associated with "thrifty" adaptations with pleiotropic or predictive effects, and health consequences in later life (Eriksson et al. 1999; Forsén et al. 2000; Gluckman et al. 2007).

Postnatal T peak (0-6mos): Within the first 6 months following birth, humans and some primates experience a characteristic second peak in Leydig cell activity and circulating T. At this stage of development the Leydig cells are no longer functionally independent of pituitary control via LH (Chen et al. 2009). A LH surge prompts Leydig cells to produce T until plasma concentrations approach the low end of normal adult levels (2-3 ng/mL) by around 2 months postpartum, after which the concentrations decline to .5 ng/mL. Following this peak, the neonatal population of Leydig cells regress or appear dormant, and testicular testosterone remains low until puberty (Bergadá et al. 2006).

While the function of this peak is not entirely clear, it is proposed as a critical window of male reproductive development when the feedback between components of the HPT axis are coordinated to thresholds of activity (Mann, 1996). In the brain, masculinisation and adult patterns of social and sexual behaviour are sensitive to disruption during this period in primate and other animal models.

In the testis this surge coincides with a second phase of Leydig and Sertoli cellular differentiation and apoptosis (Berensztein et al. 2002). The number of Sertoli cells differentiated at this period appears to be especially important for adult sperm count and testicular descent according to rat and primate models (Mann and Fraser 1996; Sharpe et al. 2003).

Recently, developmental stresses as estimated by neonatal growth rates have been associated with adult levels of LH, T, and somatic factors such as muscle mass and grip strength (Gettler et al. 2010; Kuzawa et al. 2010).

Juvenile period (3-12 years)

This post-weaning period of development has been described as a “phenotypic limbo” in which offspring are nutritionally independent from direct nourishment from the mother, but are not yet reproductively functional (Bogin 1999; Pagel and Harvey 1993).

The juvenile stage is referred to as reproductively quiescent as measures of sex hormones and activity of the reproductive glands are extremely low. While a period of quiescence is observed in every major taxonomic group, it is comparatively prolonged in most mammals, and among mammals, human juvenile periods are particularly long (Pereira and Fairbanks 1993). This suggests the juvenile stage has been prolonged due to selective pressure (Bogin 2009).

Growth velocities slow down during this stage, as compared to the rapid rate of neonatal growth (Tanner 1978). This coincides with a period of socialisation, acquaintance with cultural practices and sources of nutrition, and a period of frequent immune challenge.

Observations relate environmental stresses upon male children during this period to the phenotype of their male offspring (Bygren et al. 2001; Pembrey et al. 2005). Though the mechanism of this effect is not yet known, it has led to speculation that this period is a critical window for the organisation of trans-generational epigenetic information, which is particularly relevant to males (Pembrey 2010).

Adrenarche occurs at the end of the juvenile period (at approximately 7 years old in males), with a rise of adrenally-derived androgens (Worthman 1999). There is a subsequent reduction in hypothalamic sensitivity to androgens, with potential to adjust the HPT axis. This stage of development is postulated as important to the coordination of the male reproductive axis function and maturation with stress, risk taking and socialisation signals relayed by the adrenal corticosteroid, cortisol (Campbell 2003).

Adolescence (13-17 years)

Puberty begins with GnRH pulses that precipitate adult-like HPT function. In response to these pulses the pituitary gland begins producing peptide gonadotrophins FSH and luteinising hormone LH. Clinical and animal studies suggest developmental canalisation of male reproductive function. Hormonal activity during this period is thought to "set" many of the thresholds of activation and function for the remainder of a male's lifetime. Pubertal hormone–level variation precipitate long-term changes in pituitary and gonadal hormone receptor sensitivity, which may influence HPT irreversibly throughout the lifetime of the individual. This pubertal establishment of adult reproductive hormone function is an important step in characterising the evolution of male reproductive strategies (Bribiescas 2000).

Prior to maturity, children's body composition is relatively similar. But dimorphism appears at puberty with consequent changes in fat or muscle deposition and strength (Bribiescas 2006). Male growth rates suddenly accelerate to double or more those of childhood. This period of growth appears more highly heritable than childhood growth, and less subject to ecological influences (Tanner 1962). Despite this, there is likely an ecological component, particularly to the timing of the start of puberty and the duration and rate of the growth spurt. This is a critical period in which androgens play a determinative role in growth. Bone growth coincides with the increase of androgens, and they are thought to counter-balance the effects of oestrogens, which precipitates the ossification of the epiphysial plates of the long bones, thereby ending their further growth (Knussmann and Sperwien 1988). The interaction between somatic development and T may be revealed by the positive correlation between morning salivary T and stature in Nepalese men, but only during seasons of energetic stress (Ellison and Panter-Brick 1996).

Post-maturity

Inter-population differences in salivary testosterone are most pronounced in early reproductive years, with a trend toward convergence in later years.

Androgen levels of Western males decline with age, and many symptoms characteristic of hypogonadism in younger males are similar to normal age-related changes in older males, with losses in skeletal muscle mass and increased fat deposition (Campbell and Cajigal 2001; Ellison et al. 2002; Morley et al. 2005).

Unlike females, males lack a distinct cessation of their reproductive stage of life but there is increasing evidence of a pronounced shift in male reproductive function and fertility in mid-life, around the ages 35-45. In longitudinal studies of American populations, there is a clear age-related decline in male T of 1% per year after the age of 40 (Gray et al. 1991; Harman et al. 2001), while in Japanese populations T flattens and remains stable after age 40(Bribiescas 2006; Uchida et al. 2006). Cross-cultural analysis shows an age-related decrease in the mean difference in T between populations living in developed and developing nations, where the greatest differences are between young males and they are non significant after age 45(Ellison et al. 2002).

While the mechanisms of this decline are still unclear, early developmental events like the LH-dependent differentiation of Leydig cell number and senescence into adulthood may relate to exposure to oxidative stress (Chen et al. 2009). The mechanisms regulating “normal” reproductive function in the adult male will be covered in the next section.

1.4 Variations in adult male reproductive function

As a largely medical science, endocrinology focuses primarily on mechanistic explanations of androgens and how they proximately influence biological processes like reproduction, digestion, growth and development and the diseases they cause when these processes are disrupted. But much of what defines normal variation in male metabolism, morphology and behaviour is due to the actions of androgens. Understanding the interaction between androgens and human ecological variation gives insight into how evolution has shaped the human male to optimise the balance between survivorship and reproductive success. The balance of somatic expenses and energetic resources is regulated in part by androgens, as are aspects of male behaviour, and the androgen of most widespread influence is T. The interactions between androgens, the environment, and the body are important in explaining the proximate mechanisms by which evolved life history strategies are coordinated.

Application of life history theory allows for the interpretation of inter-individual variation in T, and associated clinical conditions from an evolutionary medical perspective. For men living under conditions of high nutritional intake and low physical exertion that characterise life in "developed" nations like the UK, energetic resources are unconstrained and stable, relative to Bangladesh. For men living under conditions of high pathogenic stress and ecological disruption that characterise life in "developing" nations like Bangladesh, energetic resources are constrained and less predictable, relative to the UK. If reproductive effort requires costly or risky somatic, metabolic and behavioural investment, then men under relatively affluent and stable conditions are expected to invest more into reproductive effort than men living in less affluent and unstable conditions. This difference in ecologies is predicted to lead to higher levels of T in men living in the UK than men living in Bangladesh. Over the course of a lifetime, the investment into the costly tissues, metabolic processes and behaviours linked with reproductive effort may have pleiotropic effects (Bribiescas and Ellison 2007), in which case men living in the UK will have higher incidence of disease associated with reproductive function.

For males, the primary constraints upon net lifetime reproductive opportunities are competitive interactions with other males for access to females and female choice. T corresponds to competitive and mate-seeking behaviour, as has been widely observed in birds (Ketterson et al. 1992; Wingfield et al. 1990) and in mammals including primates (reviewed in Fairbanks 2009)).

In social species, both of the above constraints are expected to limit reproductive opportunities for males of lower rank more than those of higher rank. Therefore, if T influences reproductive effort, it is expected to be higher in males of high rank compared to subordinates. This pattern has been observed in group living primates (Muller and Wrangham 2004; Rose et al. 1971), though the association is not always consistent and appears strongest at times of social instability (Sapolsky 1991). Data also support an association between social status and T in humans (Book et al. 2001; Dabbs and Morris 1990; Mazur and Booth 1998).

Spermatogenesis and peripheral function of androgens

Despite inter-individual or inter-population T variation, there is little indication of a high sensitivity of the primary reproductive process regulated by androgens, spermatogenesis. Testosterone shows 10-fold inter-individual variability between males, but only under extreme pathological conditions does testosterone production drop below levels presumed necessary to support adequate spermatogenesis for male fertility (Bribiescas 2001a; Ellison and Panter-Brick 1996; Lamb and Bennett 1994). The overall energetic investment in spermatogenesis by mammals is fairly negligible—especially in contrast to the energetic investment in reproduction by

females. In contrast to female physiology, direct metabolic investment from males in conception and development of offspring is relatively limited (Trivers 1972). Perhaps for this reason, ecological constraints and fluctuations rarely impinge upon male fecundability and basal reproductive function appears fairly robust to hormonal variation in males.

Although initiation of the spermatogenesis process requires a certain amount of stimulation by FSH, LH and T, the continuation of optimal spermatogenesis relies mainly upon the ongoing presence of FSH. Though T and LH are seen as playing a "permissive" role in spermatogenesis, they do not modulate the process (Johnson and Everitt 2000). But spermatogenesis appears to be such a robust metabolic process that human males with FSH transcription/receptor mutations and mice in which FSH production is absent still exhibit enough spermatogenesis to allow limited fecundity (Kumar et al. 1997; Tapanainen et al. 1997). Thus, spermatogenesis is an extremely resilient process which functions under even severely compromised hormonal conditions. In contrast, human female and mouse oogenesis and fecundability are highly sensitive to perturbations of reproductive axis hormones (Kumar et al. 1997; Layman et al. 1997).

There is significant variation in human sperm count (from 1-120million/ml), with a degree of controversy over the absolute threshold that defines full-fecundity (a general consensus of 20million/ml). Non-Western populations seem to show a tendency toward lower sperm counts compared to American males, but this does not seem to lead to subfecundity.

Efe and Lese men show reduced salivary T compared to males born and raised in Boston, USA. (Bentley et al. 1993; Ellison et al. 1989). African populations such as !Kung and Namibian men presented significantly lower T levels than American males. Turkana men (Campbell and Leslie 1995) showed lowered gonadotrophin levels (FSH and LH) compared to Western controls, perhaps reflective of reduced GnRH levels or pulsatility., decreased pituitary sensitivity to GnRH stimulation or greater inhibin suppression of GSH production.

Male reproductive capability also relies upon a suite of accessory glands of the urogenital tract to support and protect the spermatozoa on their journey between generations. Androgens also regulate these accessory glands including the prostate (which supplies nutrients, optimal pH, ionic milieu, etc.) for the travelling spermatozoa.

While the HPT axis forms the central hormonal network regulating male reproductive function, the components of the axis are also sensitive to other hormonal messengers such as cortisol, leptin, and thyroid hormone. Just as the HPT can be affected by these "non-reproductive" hormones, androgens, (particularly testosterone and its metabolites) exert influence peripherally, beyond

the glands of the HPT axis. This sensitivity to outside hormonal factors is important, as it hints to wider roles of androgens in somatic and energetic regulation.

As the primary adrenocorticoid, cortisol influences acute stress responses as well as patterns of activity and awakening (Tsigos and Chrousos 2002). Leptin, "the voice of the adipose tissue" (Blum 1997) regulates fat deposition. Balancing the proportion of somatic adipose and anabolic tissue has important consequences for development and lifelong metabolic adaptation to energetic conditions, as both tissues are potential sources and consumers of glucose (Campbell and Cajigal 2001; Reaven 1998). These adaptations of basal metabolic rates and balancing energetic costs are further regulated by insulin, which controls the concentration of circulating blood glucose.

Beyond the HPT axis, androgen receptors (AR) are found on the accessory glands of the male urogenital system such as the prostate and the pilosebaceous glands of hair follicles (Johnson and Everitt 2000). The prostate is the major site of non-testicular DHT (Hsing et al. 2002). These receptors are activated by the approximately 4% of unbound circulating T and thus able to enter prostatic cells through diffusion. However 95% of T entering the cell is converted to 5α-dihydrotestosterone (DHT) by the enzyme 5α-reductase (van Houten and Gooren 2000). Unlike T, DHT seems to be more effective upon organ receptors and has less regulatory effect upon LH. DHT is considered the major effector hormone in certain androgen dependant tissues, particularly in promoting hair growth, secretion of sebum and prompting the smooth muscle cells of the prostate to divide.

The division of muscle cells within the prostate continues throughout life, and as they become more numerous, the amount of 5α-reductase also increases. The prostate also responds to circulating E_2, which reduces factors that inhibit smooth muscle cell division. The process becomes a "vicious circle" although T decreases with age, the amount of T being converted to DHT accelerates in an age-dependant manner (perhaps due to E_2 levels) promoting further growth (Wick et al. 2003). Eventually, the prostate enlarges to sizes with pathological implications, these pathologies will be discussed in further detail below.

1.5 Clinical conditions associated with androgens

Benign prostatic hyperplasia

As discussed above, AR within the prostate are responsive to the androgens T and DHT, which leads to proliferative growth over the lifetime of a human male. Growth of the prostate leads to one of the most common diseases of male ageing, benign prostatic hyperplasia (BPH) and the associated lower urinary tract

symptoms (LUTS). The development of BPH requires testicular androgens, as evidenced by the reduction of prostate cellular growth by androgen inhibition. Men with reduced exposure of the prostate to androgens through physical (i.e. castration), pharmacological, or genetic means do not develop BPH (Bartsch et al. 2000; Bribiescas 2010). Below, I will discuss how populations vary widely in prostate disease rates, which has led to the hypothesis that these variations are due to influence of androgens during early development (vom Saal et al. 1997) or over the adult lifespan (Kehinde et al. 2006; Slater and Oliver 2000). The reproductive hormone hypothesis of prostate disease mirrors one proposed to explain the associations between mean salivary progesterone and the incidence of breast cancer within populations (Bribiescas and Ellison 2007; Jasienska and Thune 2001).

The incidence and severity of LUTS increases with age and is affected by diet, ethnicity and health behaviours (Hsing et al. 2002; Platz and De Marzo 2004). LUTS are also described as “prostatism” in men, and correlate with BPH. The causal link between LUTS and BPH is ambiguous, but appears to be a consequence of the enlargement of internal smooth muscle, duct epithelium, and connective tissue within the prostate gland. As the urethra passes through this gland, the enlarged prostate restricts the flow of urine and causes pain or an inability to completely empty the bladder. Impaired, painful or irregular urination ranks as a significant negative factor in quality of life indices (Morley et al. 2005). In North America and Western Europe these conditions are highly prevalent in older males, estimated to be 50% of all men over 50 and to increase by 10% of the population for every decade. However, a limited number of studies suggest a greater variability in BPH prevalence in non-Western indigenous and migratory populations (Campbell et al. 2005). Investigations of prostate cell neoplasia have focused on a genetic/environmental role for activity of 5α-reductase (Hsing et al. 2002).

African-American males have highest rate of BPH in world, while Japanese and Chinese have the lowest, by factor of 30-50 fold in risk (Jin et al. 1999; van Houten and Gooren 2000).

When Southeast Asians migrate to a Western country, prevalence of prostatic neoplasm increases but still remains low, even in second and third generations (Cook et al. 1999). Environmental factors such as diet and health behaviours affect rates of neoplasm, but there is also a likely genetic component. Asian males exhibit a lower incidence of CAG repeat length of androgen receptors and variation in this repeat length is associated with risk of prostate neoplasm. There also may be differences in peripheral androgen metabolism due to variations in the transcription of prostatic 5α-reductase. Lower levels of 5α found in Asian men

theoretically slow progression from neoplasm to clinical cancer (Ross et al. 1992; van Houten and Gooren 2000).

In comparative populations from a similar ethnic background, African-American males residing in Washington D.C. showed much greater levels of T than indigenous Nigerians (Ahluwalia et al. 1981). Certainly, it suggests higher T levels in American populations may be an example of physiologic release due to greater energetic availability rather than suppression among non-Western populations. Environmental factors such as diet and activity patterns play a more central role in disease aetiology (Meikle et al. 1997).

Sex steroids, particularly E_2 and DHT are implicated in prostate enlargement and are also suggested to foster cancerous growth. But a direct link between androgen-derived prostatic neoplasm and cancer risk is still far from agreed (Hsing et al. 2002). Steroid sensitive disease incidence varies across populations greatly (Jin et al. 1999).

In the past, the prevailing aetiologic significance has been couched in terms of ethnicity, and looked primarily at genetic differences in these populations. A limited number of studies on non-industrialised people suggest symptoms of prostate hyperplasia among subsistence–living peoples are particularly high (Campbell et al. 2005).

From this perspective, clinical research on Western populations should be viewed as representative of the extreme range of human variability and not the most common or healthiest representation of human populations (Ellison 1994).

Androgens, insulin and type II diabetes

Insulin plays an important role as a hormonal signal of the fed state in response to elevated blood glucose levels and amino acids. An inability to produce sufficient insulin or failure of insulin receptors leads to impaired carbohydrate, fat and protein metabolism. This condition is called diabetes mellitus. When diabetes occurs gradually with few signs of acute metabolic disturbance it is referred to as Non Insulin Dependant (or Type II) Diabetes Mellitus (NIDDM).

While there is a heritable component, the disease is associated with “miscalibrated” energy balance and is linked to obesity. A diet low in fat and high in fibre, combined with daily exercise has been suggested to reduce insulin levels and grant some epidemiological protection against irregular insulin levels leading to NIDDM. In addition, decreased insulin level might reduce proliferation of SMC within the prostate (Hales and Barker 2001). Measures of visceral body fat, insulin, E_2 and T all correlate in men with so-called “metabolic syndrome”.

Androgens and dietary restriction

In comparison to females, dietary factors are less significant in altering male hormonal profiles. Modest negative energy balances fail to effect significant changes in important sex steroid levels. More severe fasting or famine conditions appear to downregulate GnRH availability, subsequently reducing LH and T, but not FSH. Based on studies of animals and humans, increases in peripheral hormones under periods of caloric restriction such as leptin and cortisol are a possible route of action upon the HPT axis (Bribiescas 2000).

Diurnal patterns

The pulsatility of GnRH release is reflected by rhythmic fluctuations in circulating gonadotrophin and T concentrations, peaking at 1 to 3 hour intervals. Circulating testosterone generally exhibits a diurnal pattern, with peak levels early in the morning and a gradual decline throughout the day (Johnson and Everitt 2000). The generally observed pattern of male testosterone is diurnal, with a peak in the morning. These observations are based upon clinical and epidemiological samplings from populations resident in high-income, developed nations. There is less clear evidence of such an AM peak in populations of men residing in low-income nations or living under subsistence farming or foraging conditions. The limited evidence from these populations suggests less consistent pattern of an AM peak. (Bribiescas and Hill 2010; Vitzthum et al. 2009).

Birth seasonality in females appears associated with fluctuations in ovarian function (Ellison et al. 1993; Rojansky et al. 1992), evidence for seasonal fluctuations in T is inconsistent for human males.

T and the gonadotropins exhibit pulsatile secretion patterns, and these patterns are less ordered in older men than in young men (Keenan et al. 2006; Veldhuis et al. 2009). According to the “ensemble” model of the HPT axis, the degree to which these pulses are ordered reflects the degree of integration of functional thresholds of each component of the axis (Veldhuis et al. 2009). Conversely, irregularity of pulses suggests a less well-integrated HPT axis, and young men return to regular pulsatile functioning after experimental disruption more quickly than older men (Liu et al. 2006).

Androgens and Reproductive Behaviour

Humans form long-term pair bonds with mates, a reproductive strategy uncommon in ~95% of mammals. Across species, socially monogamous behaviour is influenced by magnitude of paternal care, mode of resource access, and mate choice (Reichard and Boesch 2003).

In monogamous birds, T increases in situations of high reproductive competition, and declines when males are required to care for offspring. This association between male reproductive challenges and T has been observed in a variety of other vertebrates, including primates. These observations led to the hypothesis that T modulates male-male competition, affiliative bonding, and direct paternal care amongst pair-bonded species (Archer 2006; Wingfield et al. 1990).

The length and types of human pair bonds vary significantly across cultures based on socioecological conditions. The degree of paternal investment in offspring and mate-seeking competition between human males is also widely variable reflecting the compromise between reproductive interests and offspring investment strategies (Ellison 2009).

A growing body of data suggests an association between pair bonding and/or parenthood and salivary testosterone in human males (Burnham et al. 2003; Gray et al. 2006). While cross-cultural research in this area is expanding (Gray et al. 2007), the bulk of these studies investigated North American men, with limited cultural variation in mate choice, extramarital sex and direct paternal investment in offspring.

1.6 Body composition.

Beyond the reproductive axis, T levels regulate somatic tissues, particularly anabolic muscle. Modulation of skeletal muscle mass may represent a mechanism by which T regulates the balance of male energetic expenditure upon reproductive effort.

Muscle tissue

T is a key hormone in the regulation of muscle anabolism and metabolism. T administration to humans stimulates fat catabolism and adipose redistribution while stimulating muscular protein synthesis and glucose uptake (Johnson and Everitt 2000). T even increases the metabolic rates of isolated muscle cells in vitro (Tsai and Sapolsky 1996). The importance of mediating muscle mass to human male reproductive ecology will be investigated in further detail in a developmental and comparative context in the corresponding sections.

Adipose tissue

Within the HPT axis, T is thought to inhibit the frequency of GnRH pulsatility and thereby suppress LH production (Crowley et al. 1991). However, the T metabolite E_2 also inhibits the release of hypothalamic GnRH production and decreases pituitary sensitivity to GnRH.

In a process called aromatisation, the enzyme aromatase converts T within adipocytes (fat cells) to E_2. This process has been suggested as explanation of lower T and hypogonadism in obese men (Kley et al. 1981). Aromatisation might function as a mechanism of androgenic modulation of somatic composition between adipose and muscle. T converted to E_2 by adipose tissue has the similar inhibitory effects as if it were still in circulation, but without the ameliorating effects upon muscle tissue. Men investing in greater adiposity may exhibit greater aromatisation of circulating T and also exhibit hypothalamic and pituitary inhibition as a result of higher E_2 levels.

The fact that both of these hormones elicit similar responses from the hypothalamus might reflect the process by which the brain monitors bodily states of tissue distribution through differential sensitivity to the two sex steroids. Studies of caloric restriction in rats show dramatic reductions in FSH and LH, followed by reduced T production and release (Bergendahl et al. 1998). Administration of GnRH eliminated this effect, and suggests hypothalamic receptors are monitoring energetic status, and modulate pituitary GnRH receptors. The mechanism of this monitoring is unknown, but most likely to be through direct signalling molecules of energetic status: particularly insulin, cortisol, and leptin.

Leptin, a protein hormone synthesised primarily by adipose tissue serves as a circulating signal of nutritional status and reflects fat stores and body weight. While leptin was once thought as the direct link between adipocytes and metabolism, immune function and reproduction, this association now appears more complex. The linkage between leptin and adiposity appears more relevant in populations with high caloric intake and low energetic demands (Chan et al. 2006), and may not reflect energetic status or body mass in foraging or subsistance-living people (Lindeberg et al. 2001). In populations where energy intake and output are equal, leptin reflects the amount of stored triglycerides in adipose tissue (Barb 1999). Studies of chimpanzees and humans show sexual dimorphism in leptin levels is a commonality, with females showing higher levels than males after sexual maturity (Barb 1999; Bribiescas and Anestis 2010). During adolescence, leptin levels in males fall or remain flat as T levels increase during puberty, while females show a pronounced rise (Rutters et al. 2009). It was once thought that a prepubertal pulse in leptin might represent a “permissive factor” by signalling the brain that the body is metabolically ready to go through puberty (Kiess et al. 1998). Now, however, the degree to which maturity is reliant on such a signal is not as clear in humans as it is in other species, and is more applicable to females than males (Chan et al. 2006; Rutters et al. 2009).

Immune system

Recently, the role of T in the regulation of energetic resources has been postulated as a modulating factor in balancing costs of immune system challenges and maintenance (Campbell et al. 2001; McDade 2005; Muehlenbein and Bribiescas 2005). T appears to regulate energy allocation by altering anabolically sensitive tissue, including skeletal muscle mass. However, the regulation of energetic resources also extends to the immune system, and immune activity appears suppressed by T (Muehlenbein and Bribiescas 2005). The maintenance of immunocompetence is energetically expensive and potentially crucial to the advertisement of genetic fitness to mates (Hamilton and Zuk 1982). Energetic investment into high reproductive effort and costly anabolic muscle competes with investment into immunocompetence, and males under significant immune stress would be expected to be unable to invest as heavily into reproductive effort, as measured by T (Folstad and Karter 1992). Assessing changes in testosterone and immune factors during infection may yield insight into male physiological ecology (Muehlenbein and Bribiescas 2005). These ideas have received some support from observations of greater numbers of chest infections among Turkana males of Kenya exhibiting higher testosterone, though the direction of causation is still unclear (Campbell et al. 2001).

1.7 Human male life history theory

Life history theory applies Darwinian principles to the study of human demography and epidemiology (Hill and Hurtado 1996). Namely, these principles assume biological evolution occurs through the mechanism of natural selection and adaptation is the result (Ellison 2001b). Adaptations are not abstract, they are specific to ecological conditions. Biological evolution is a cumulative response to the selective forces of previous environments. Therefore, inquiry into the nature of adaptation must include functional aspects of an organism's contemporary environment, but also the evolutionary history and selective forces that have shaped it over time.

J.T. Bonner said "the ultimate description of an organism is not just a description of its adult phase, but that of its whole life cycle." ((1965), quoted in Hill and Hurtado, 1996 p.19). Extending this logic, perhaps the ultimate description of a species is not just the description of a typical life-cycle, but all the collective life-cycles preceding the present. As most of the morphological evolution of our species presumably took place in a band-level foraging context, most of the collective life-cycles occurred under these conditions and much of life history theory is premised upon the demography of foraging populations.

Humans have evolved fertility and mortality patterns that lead to the highest contribution to the future gene pool, constrained within general human morphological, physiological, and social limitations, and the environments in which our species lives. This contribution can be direct in the form of reproduction or through helping kin likely to share the same genes (Hamilton 1964; Hill 1993).

General model of life history

Life history theory predicts that selection will favour physiological mechanisms that efficiently regulate the allocation of energy and time between four general competing functions: reproduction, maintenance, storage and growth (Hill 1993; Kaplan et al. 2000).

An organism's physiology can be thought of in economic terms, with energy a currency that must be divided and portioned. Energy can be spent only once and its rate of accumulation is dependent upon ecological conditions, so investments must weigh costs, risks, benefits, and future expectations. All of the consumers of metabolic energy produce measurable, somatic characteristics. Figure 5 illustrates a schematic of these competing interests as they apply to the life history of the human male. Broadly, investment in preadult growth is reflected by skeletal measures of stature, male investment in reproduction by somatic muscle, investment in maintenance by immunocompetence, and investment in storage by adipose tissue.

Figure 5: Male life history allocations

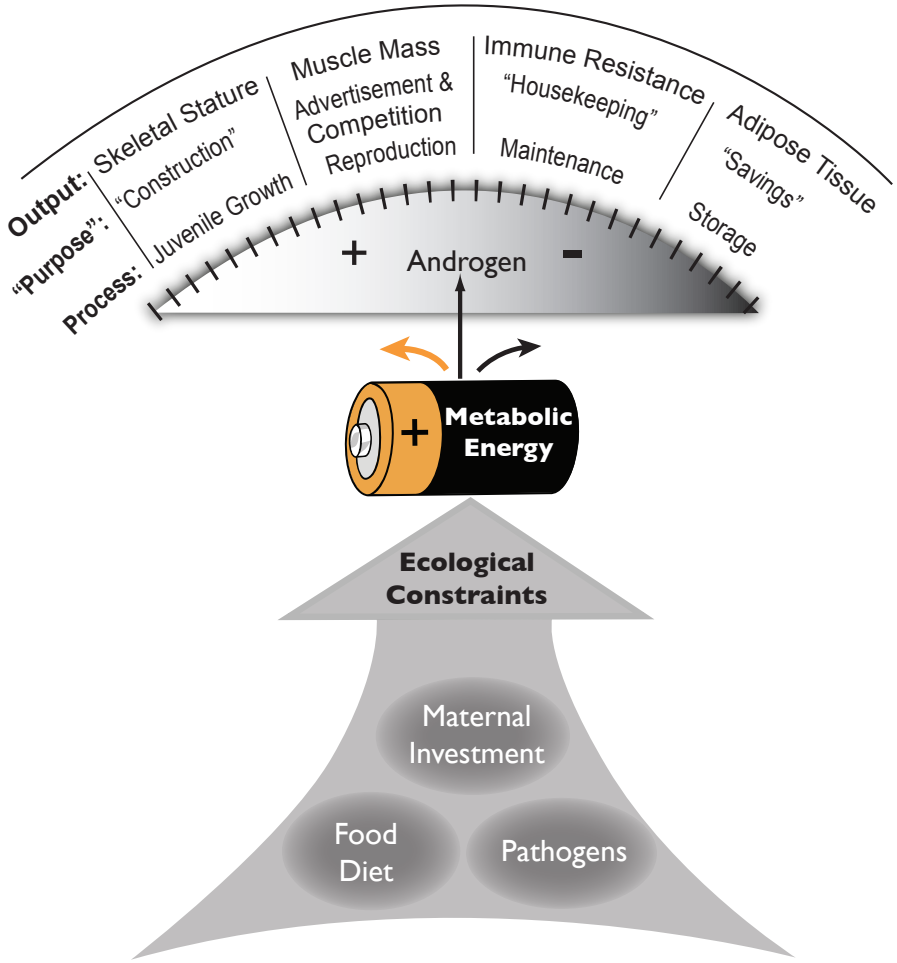


Figure 5: Simplified schematic of male life history variables: At any given time, available energy must be allocated between competing costs. Life history theory predicts such distribution will be based on optimal reproductive benefit as shaped by natural selection.

Life history theory and growth

Life history theory's foundations lie in investigations of growth rates by population ecologist Eric Charnov, who proposed measures of analysis of characters that are invariant across species or populations over time (Charnov 1993). This search for biological "laws" governing growth, maintenance or timing was premised on the idea that natural selection favours individuals that maximise their reproductive success by optimising trade-offs between growth and maintenance. This requires a view of the collective instances over the entirety of a lifespan, as potential rewards are balanced by risk and investment at any given moment.

When there are invariants within and across species, they are presumed to be constrained by universal, unchanging and predictable principles such as molecular limitations on cellular size (West 2006). Invariance in growth patterns is described as "canalisation". The continued diversion of energetic resources

to canalised growth despite fluctuations in ecological conditions is known as homeorhesis (Ellison 2001a).

When patterns of growth are variable they are predicted as optimal solutions to changeable conditions (Charnov 1993). Physiological adaptation must deal with degrees of certainty and variability in environmental conditions. Over evolutionary history the range of environmental variation, its pattern, and its predictability have selected for physiological mechanisms to track that variation and continuously adjust to it through phenotypic plasticity. For this reason, flexible adaptive phenotypic responses to predictably uncertain components of the environment are likely to be very advantageous and should be observable in humans. Such flexible adaptive phenotypic responses in individuals are known as reaction norms (Stearns 2000).

Much of human growth is highly canalised, indicating homeorhesis (Ellison 2001a). The basic pattern of human growth appears universal, and for the most part do not require population-specific standards (Ellison 2001a). Overall growth trajectories parallel standard growth centile lines across populations. Children of developing nations who are in the highest socioeconomic classes develop at same median rates as those in developed countries (Eveleth and Tanner 1990; Haas and Campirano 2006).

While migrants from poor to affluent conditions show a convergence from lower toward higher growth trajectories (Eveleth and Tanner 1990; Stinson 2000), ethnic patterns of growth remain distinct among migrants into the second and third generation (Chinn et al. 1996; Rona and Chinn 1986). Compared to the host population, trans-generational persistence of lower birthweight, patterns of “catch up” growth and adult obesity in migrants (Leon and Moser 2010; Wells) may be in part due to maternal constraints, somatic limitations and historical trends as discussed in the developmental section above ((Pollard et al. 2011; Wells 2010b)

The degree to which genetic, trans-generational and ecological factors contribute to observed ethnic variations in patterns of growth remains unclear, but a review of worldwide variations in growth by Haas and Campirano (2006) suggested that, across populations, the experience of favourable growing environments leads to comparable average linear growth prior to puberty.

Human skeletal growth is more tightly canalised than growth in weight. Soft tissues serve as reservoirs for carbohydrates, lipids and amino acids. This dynamic allocation strategy ensures the availability of important metabolic substrates is buffered from variation in intake and utilisation. Based on these observations, homeorhesis and the robust nature of human growth trajectories means slower

growth is adaptive for those who grow up under conditions of chronically low energy (Eveleth and Tanner 1990; Stinson 2000).

Bone age, the point at which epiphysial plates are fused is more accurately predictive of stage of reproductive maturity as compared to chronological age (Eveleth and Tanner 1990). This is thought to occur because skeletal maturation is influenced by the production of gonadal steroids (Tanner 1962). Androgens have a stimulatory effect on cellular proliferation and growth that are responsible for the elongation of long bones. In contrast, oestrogens stimulate the process of mineral deposition that results in ossification and eventual fusion of the epiphyses to the shaft of the bone (Johnson and Everitt 2000). In addition, androgen levels correspond to prolonged absolute period of skeletal growth prior to sexual maturity (Tanner 1962).

Beyond the age of 17.5 years, growth in male stature virtually ceases among British men (Tanner 1962). It should be noted that cessation of growth is subject to inter-population variation. A longitudinal study of boys living in rural Hyderabad, India under conditions of nutritional stress showed a comparatively prolonged pubertal growth period, with a mean age of completed growth of 19.2 years, though boys in the same region receiving adequate nutrition did not significantly differ (17.8 years) from British populations (Satyanarayana et al. 1989). When sampling from men who were not nutritionally stressed during childhood, one would expect the effects of ecological change post-maturity will not be expressed in measures of skeletal stature.

Life history theory and human reproductive ecology

Reproductive ecology focuses on the interaction between physiology of human reproduction and adaptation to environmental conditions. As natural selection operates through differential reproductive success due to heritable biological variation over evolutionary time, reproductive ecology plays a critical role in the shaping of human adaptations. The essential question of reproductive ecology is how an organism balances between the often-competing interests of reproductive effort and survivorship. The question is answered by estimating the overall allocation of resources to reproduction rather than to other competing biological functions such as growth or maintenance. In evolutionary studies, reproductive effort is a variable subject to strategic alterations in response to current constraints of individual constitutional and ecological circumstances. This is in contrast to the prevalent medical perspective, which often assumes that there is one optimum of reproductive function which is disrupted by pathological influences. Thus the primary assumption of human reproductive ecology is that the strategically variable allocation of reproductive effort has been shaped by natural selection (Ellison 2001b).

Aging

Human males lack the relatively clearly marked start and cessation of reproductive stage that occurs with female menarche and menopause. This makes a life-history analysis of male reproduction less conveniently demarcated and any division of the male reproductive stage is somewhat arbitrary, but these are not reasons to expect a change in reproductive strategies for aging human males. Along with the shift in male hormonal activity described in the development section, male fertility also appears to peak in the fourth decade of life in populations living in modern Germany and in hunter-gatherer populations (Hill and Hurtado 1996; Plas et al. 2000).

There is increasing evidence of a pronounced shift in male reproductive function and fertility in mid-life, around the ages 35-45. In longitudinal studies of American populations, there is a clear age-related decline in male T of 1% per year after the age of 40 (Gray et al. 1991; Harman et al. 2001), while in Japanese populations T flattens and remains stable after age 40(Bribiescas 2006; Uchida et al. 2006). Cross-cultural analysis shows an age-related decrease in the mean difference in T between populations living in developed and developing nations, where the greatest differences are between young males, and they are non significant after age 45 (Ellison et al. 2002).

Together, the above observations suggest a shift in reproductive life history of the human male around the age of 40 (Bribiescas 2006; Bribiescas 2010). While males appear to retain fecundability late into life, the likelihood of further reproduction declines for most men, excepting those with higher status or wealth (Cronk 1991; Kaplan et al. 2000; Marlowe 2000).

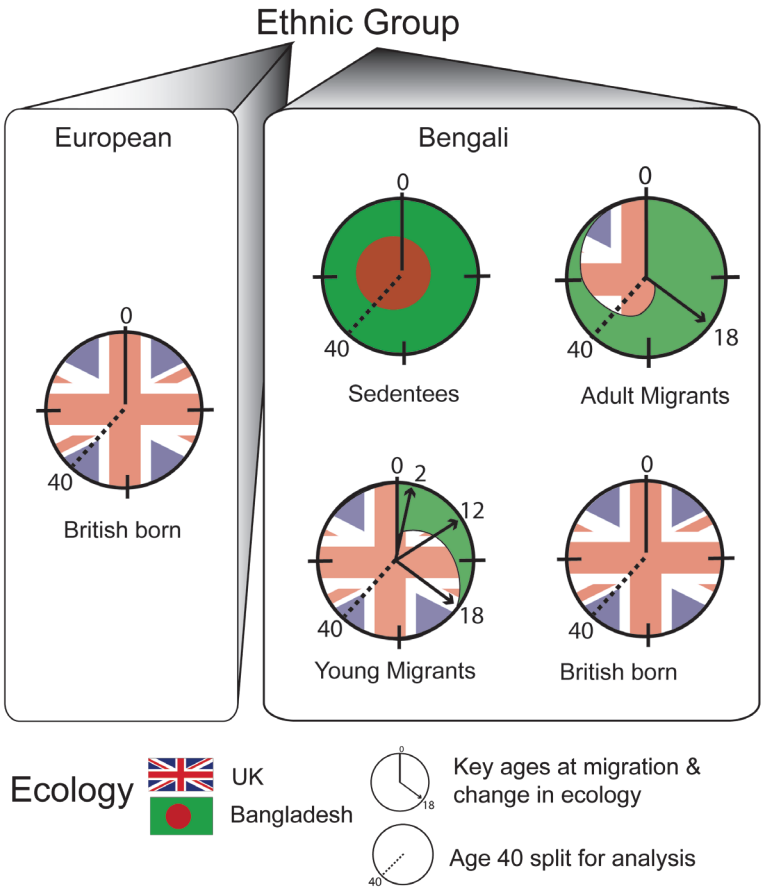
Predictions of this study

As reviewed above, ecological cues and constraints on metabolic energy mediate levels of androgens, which coordinate early development and adult reproductive function. The relevant ecological conditions of life experienced during key developmental stages in London and Sylhet are hypothesised to influence salT levels of adult Bengali men.

This project applies life history theory to predict male reproductive function across three key variables 1. Ethnicity 2. Ecology 3. Development. The design of the project is illustrated in figure 6, where the timing of an ecological change on the developmental “clock” separates Bengalis by time of migration, between men who migrated before or during maturity “young migrants” and men who migrated after maturity “adult migrants”. Young migrants are subdivided by whether they migrated as infants (before age 2 years), juveniles (age 2 to 12) and adolescents (age 13 to 18). Sedentees and British-born Bengalis serve to reference a constant

ecology throughout development and adulthood. The British-born Europeans serve as an ethnic outgroup, living under similar ecological conditions as the Bengalis in London. All men are subdivided by whether they are in the first or second part of their reproductive stage of life, at age 40.

Figure 6: Project design



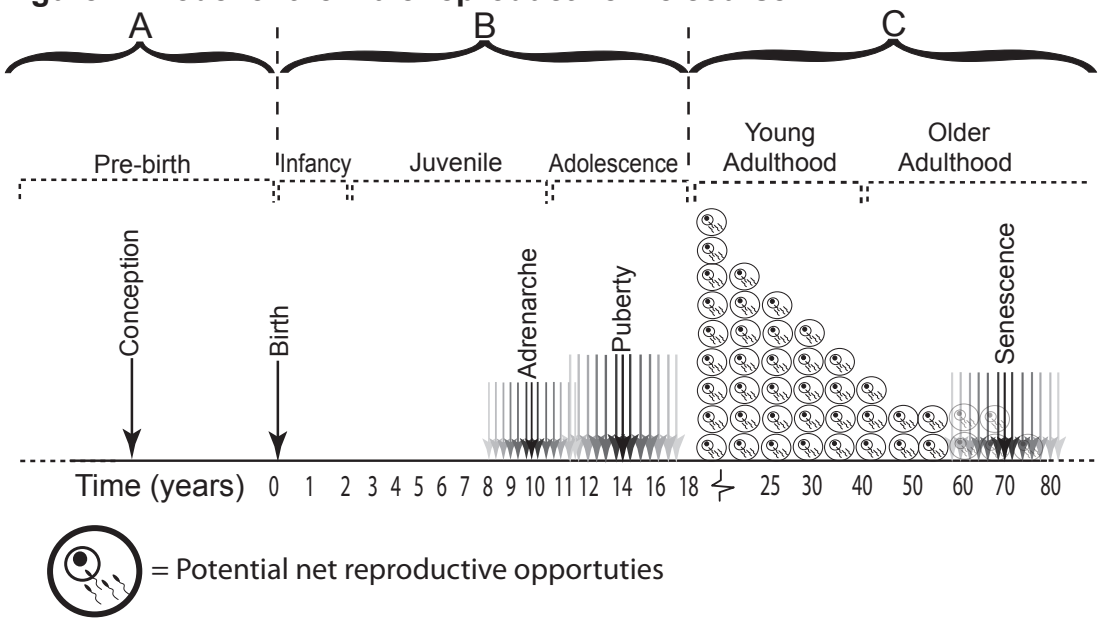
This project design allows for the following assumptions:

1. Ethnic variation: While subject to gene-environment interactions, influences upon reproductive function due to genetic variations in the migrant Bengalis is assumed to remain a constant, when compared to sedentees as a consequence of the migration history and geography of what is effectively two branches of one Sylheti family.

2. Ecology: The ecological influences of living in London upon the Bengalis operate on several levels:

Influence of low relative SES upon reproductive function should be similar in British-born Bengalis to those of SES matched British-born Europeans. Acculturation should also mean the health behaviours and dietary patterns of men who were born in London should be more similar than those of men born in Bangladesh. However, the high levels of segregation and preserved cultural practices of the London Bengalis are expected to limit the differences in diet and health behaviours between this population and Sylheti sedentees.

Figure 7: Model of the male reproductive life course



				Predicted Adult Condition									
Group	Ecological condition			salT					Anthropometric Measures			Health measures	
	Poor	Affluent	Phenotypic matching g	Daily mean	Diurnal variation (AM/PM)	Ontological variation (Old/Young Adult)	Paternity/ Marital Status	SES	Stature (Height, AL)	Skeletal Muscle	Axial Body Fat	Prostate symptoms	FBG
SED	A, B, C	N/A	matched	low	low	low	no effect		short	low	low	low	low
ADM	A, B, C	C	mismatch	chronically raised	low	low	no effect		short	high	high	high	high
YOM (9-18yr)	A, ~B	~B, C	partial match	dynamic, raised	low/ medium	low/ medium	partial effect		taller*	high	high	lower*	lower*
YOM (3-8yr)	A, ~B	~B, C	partial match+	dynamic, raised	higher	higher	partial effect		taller*	high	high	lower*	lower*
YOM (0-2yr)	A, ~B	~B, C	partial match++	dynamic, raised	highest	highest	reduced		taller*	high	high	higher*	lower*
2NG	~A	~A, B, C	~matched	dynamic, raised	high	high	reduced	>all MG <EHI	tall	high	high	low	low?
ELO	N/A	A, B, C	matched	dynamic, raised	high	high	reduced	>all MG <EHI	tall	high	high	low	low
EHI	N/A	A, B, C	matched	dynamic, raised	high	high	reduced	highest	tall	high	high	low	low

* = compared to ADU
\+ = indicates higher level of matching
MG = migrant groups

Figure 7: model of the male reproductive life course: key stages of development are of importance to adult condition, and are split into three broad categories, A: Pre-birth; B: Childhood and adolescence, which is subdivided between infancy (birth to aged two years), juvenile (aged two through 12 years) and adolescent stages (aged 12 through 18); C: Adulthood, which is subdivided between young and older adulthood at aged 40 years. Ecological cues and constraints during stages A and B are hypothesised to influence repro-

ductive function of men during stage C. If energetic conditions in Sylhet are interpreted by a developing male as “poor” and conditions in London are classified as “affluent”, then males are predicted to respond to a change from poor to affluent conditions by maximising potential net lifetime reproductive opportunities. If this change occurs in early childhood, age of adrenarche and puberty will be earlier, and stature will be taller than for men who experience the same change in conditions later in life. The later this change occurs in life, there will be a greater mismatch between adult ecological conditions and hormonal thresholds of reproductive activity, as measured by salT and prostate symptoms, and males will discount maximal investment in reproductive function in early life with earlier senescence in later life.

Life in Sylhet is more constrained by immune challenges and environmental instability, as compared to London. While stress due to pathogens and instability is not directly measured in this project, the assumption that childhood growth and development includes exposure to these stresses is based on epidemiological and historical evidence presented above. Hypothesis: Men who reached maturity in Sylhet will be phenotypically “matched” to this ecology in measurable ways in adulthood. Predictions: Bengalis who migrated prior to puberty will be taller than those migrating after puberty, and the age in childhood when a male migrates will predict adult salT, height, skeletal muscle and age at maturity.

3. Development: The model of the male lifecourse applied to this project is illustrated in figure 7. During the first developmental phase (A) pre-birth and trans-generational factors such maternal condition and inter-uterine development are important for establishing metabolic requirements in early life. The prereproductive phase (B) contains three subdivisions: the first two years of life are important for structural organisation of the HPT axis, the peripheral organs of the male reproductive system and possibly thresholds of function during the postnatal peak in T. The reproductively quiescent juvenile period is devoted to socialisation, growth and timing of maturity. Adolescence is important to the establishment of adult reproductive function and the cessation of growth. Males entering the reproductive phase (C) may employ different strategies, depending on whether they are maximising early reproductive opportunities at the expense of accumulation of resources, status or skills over the long term. While individual profiles of reproductive opportunities may actually be more curved in their distribution, potential opportunities for males may be highly “stacked” in a short period of their reproductive phase, or they may be distributed in a more “spaced” distribution. This phase contains two subdivisions: Early adulthood is important to the maximisation of reproductive opportunities based on balancing physical competition for immediate gain and the accumulation of skills, status and other capital over the long term, and this is expected to be the time when male physical competitive interactions are at their highest levels. Late adulthood is a period of shifting from highly physical competition of young males to the investment of paternal resources into offspring and the maintenance of survivorship, which would be expected to coincide with reduction in androgens, as illustrated in

figure 5. Alternatively, late adulthood allows for continued seeking of further reproductive opportunities for males able to capitalise on accumulated resources (see section on aging, above), which would be expected to coincide with retention of higher androgens from young adulthood. The timing and degree of this shift in reproductive effort will be subject to the likelihood of future reproductive opportunities. In evolutionary history, older males were unlikely to succeed in direct physical competition with younger males for reproductive opportunities, but older males will have had the opportunity to acquire status in early adulthood. The diversion of metabolic energy toward reproductive effort is more likely to be maintained in older males of high status than in men of the same age of lower status.

In summary, the migrants in this study transition from one set of ecological conditions to another during one of the developmental phases outlined above. Men are expected to match their reproductive phenotypes to ecological conditions experienced during development in ways that balance survivorship and competition that have been shaped by selective pressures over evolutionary time.

This project builds upon earlier findings, summarised in Appendix 3 that adult Bengali migrants had higher salT than sedentees (Cronk 1991) and that salT differences were reflected in apportionment of muscle mass and height in migrants who spent all or part of their childhood in London (Magid 2005; Magid et al. 2007). Hypotheses based upon the life-history model illustrated in figure 7 will be tested according to methodology detailed in chapter two, and presented in three results chapters, framed around developmental history, dietary and health behaviours, and social ecology. I begin by testing in chapter three whether developmental conditions influence investment in persistent structures and response thresholds of hormonal axes and somatic tissue depending upon timing of a change in ecological conditions. These will be measured by salT, anthropometric measures, and recalled age of maturity. The specific predictions are that Bengali men who spent all or part of their childhood in London will show higher salT, taller stature, and will recall reaching sexual maturity at an earlier age than Bengalis who spent their childhood in Sylhet.

In chapter four I test hypotheses related to dietary and health behaviours. I begin by testing the assumption that Bengali populations in both Sylhet and London are not nutritionally stressed, and that Sylheti dietary and health behaviours are conserved in migrants. I will also test whether exposure to the ecology of London during childhood influences conservation of these behaviours. The predictions are that men from Sylhet are not nutritionally stressed, that the Bengali diet in London is similar to that of sedentee counterparts, with less consumption of fish and more consumption of other meat. Bengalis who spent all or part of their

childhood in London will show greater similarity in their patterns of dietary and health behaviours to SES-matched British European men compared with Bengalis who did not migrate to London as children.

The second part of chapter four moves to the testing of evolutionary medical hypotheses of health. If adult onset diseases are related to a mismatch between early life developmental conditions and adult ecology, does migration after key stages of development mean migrants are more prone to symptoms of prostatic disease and dysregulation of glucose metabolism? Proximately, if men have high salT, and T stimulates proliferative growth of the prostate over the male life-span, are they more likely to report more LUTS than men who have low salT? The prediction tested is that Bengalis who migrated London after childhood will report more LUTS than British-born Bengalis, and Bengalis who migrated as children. Finally, do dietary and health behaviours adequately explain Bengali inter-population variation in measures of salT tested in chapter three?

Chapter five tests hypotheses of social ecology and male reproduction. Regarding socioeconomic positioning, if a male is of high SES relative to current surrounding ecological conditions, does he divert more effort toward reproductive function than men of low SES, relative to current ecological conditions? Is current relative SES more influential on reproductive effort of men in the latter half of their reproductive stage of life, compared to men in the first half of this stage of life? The specific predictions are that high SES males have higher salT, and that relative SES is more highly associated with salT in men aged 40 years or older. Finally, do SES, dietary and health behaviours adequately explain Bengali inter-population variation in measures of salT tested in chapter three?

The second part of chapter five tests hypotheses concerning relationship and reproductive status: If different ecologies during childhood development determine coordination of male endocrine function and the social relationships of pair-bonds and offspring, do men who are exposed to Western influences of acculturation during childhood development exhibit greater reduction in reproductive function if they are married or married with children as compared to men who were less exposed to such influences? The specific prediction is that Bengalis who spent all or part of their childhood in London and British European men will show lower salT if they are married or have young children than Bengalis who spent all of their childhood in Sylhet.

Chapter six draws conclusions from the findings of the project as a whole at two levels of inquiry. Proximately, how does the functioning of reproductive organs and hormonal axes interact with developmental history and current surroundings? Ultimately, how do these results reflect the balancing of the competing biological functions of survivorship and reproductive effort that has been shaped by natural

selection? These principles extend to the field of evolutionary medicine, where trade-offs of investment between competing physiological requirements explain senescence and disease.

Chapter 2: Methods and validation studies

In this chapter I describe participant recruitment, methods of data collection, sample storage and laboratory analysis. In the second half of this chapter I describe the results of experiments to validate salT measures of individual inter- and intra-daily variation and the influence of chewing betel nut.

2.1 Recruitment and interviews

The study recruited healthy males over 18 years of age. All were screened for non-insulin dependent diabetes (NIDDM) and thyroid disorders. Other exclusion criteria were first-order relatedness to another participant (e.g. brothers, fathers). Ethical approval and Data Protection approval were granted by the UCL Research Ethics Committee (ID: 0144/002), and Osmani Medical College in Sylhet. Participants were given an honorarium (£10 in UK, Tk500 in BD) upon completion of the study.

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 ($\alpha = 0.05$), power ($1-ß = 0.95$), and a conventional "medium" effect size (Cohen's "f" = 0.25) (Magid 2006).

Bangladeshi Sedentees (SED)

Data were collected on two separate field visits to Sylhet. Participants recruited in 2007 were asked to list any third-order (e.g. first cousins) or more closely related migrants living in the UK/Europe, Australia/New Zealand, or USA/Canada. Of 32 recruits, 2 (6%) did not list any relatives in these countries.

2005

Fifty-five men aged 20-78 (mean 39) born and resident in Sylhet, NE Bangladesh all their lives were recruited within the boundaries of Sylhet City Corporation. All measures were collected by a field team led by Dr. Farid Ahamed, University of Chittagong, Department of Anthropology. Dr. Ahamed assisted with pilot studies in London and trained in anthropometric techniques.

2007

A further 32 males aged 25-78 (mean 54) were recruited in the same region, in order to match the age range of adult migrants in the UK. All physical measures were collected by the author. Four postgraduate research assistants from Shah Jilal University of Science and Technology (Sylhet),

Department of Anthropology, assisted in word of mouth recruitment, interviews and translation.

London

Bengali participants were interviewed and saliva samples were collected within the neighbourhoods of East London with the highest density of Bengali migrants. Over 90% of the men were recruited from the London boroughs of Camden, Hackney, and Tower Hamlets (see figure 3 for map). Participants were recruited at community centres/events, mosques, and fitness centres or organisations such as Sunday league football organisations, or from Internet or newspaper advertising (see table 2). After correcting for age at recruitment and residence group effects, place of recruitment was not a significant predictor of salT (MLR: $t(3,275)=0.34$, $p=.7$). Recruitment, measurement, and collection were performed in London by the author or four research assistants/translators. All high status British-born Europeans were recruited and their data collected in a separate masters project by Robert deVries in 2008, using a simplified questionnaire that did not include dietary measures.

Table 2: Recruitment data

Recruitment Place Category	Residence Group					
	Bengali Sedentees		Adult Migrants		Youth Migrants	
	n	%	n	%	n	%
Domestic networks/snowball	97	91.51%	6	9.38%	5	8.77%
Community organisation	0	0.00%	53	82.81%	20	35.09%
Sports organisation	0	0.00%	1	1.56%	28	49.12%
Workplace	4	3.77%	0	0.00%	1	1.75%
Religious organisation	4	3.77%	3	4.69%	2	3.51%
Educational organisation	1	0.94%	1	1.56%	1	1.75%
Health organisation	0	0.00%	0	0.00%	0	0.00%
Advertisement	0	0.00%	0	0.00%	0	0.00%
	106		64		57	

	British-born Bengalis		Low SES European		High SES European	
	n	%	n	%	n	%
Domestic networks/snowball	4	7.84%	4	6.56%	30	100.00%
Community organisation	2	3.92%	2	3.28%	0	0.00%
Sports organisation	29	56.86%	20	32.79%	0	0.00%
Workplace	0	0.00%	14	22.95%	0	0.00%
Religious organisation	7	13.73%	15	24.59%	0	0.00%
Educational organisation	4	7.84%	0	0.00%	0	0.00%
Health organisation	4	7.84%	1	1.64%	0	0.00%
Advertisement	1	1.96%	5	8.20%	0	0.00%
	51		61		30	

Adult Migrants (ADM)

Sixty-one men aged 23-76 (mean 49) who were born in Bangladesh and migrated to the UK to settle permanently after an assumed age of sexual maturity (≥ age 18)

Young Migrants (YOM)

Fifty men aged 18-69 years (mean 32) born in the District of Sylhet, Bangladesh and who migrated to the UK to settle permanently at an age prior to sexual maturity (< age 18, mean age at migration 8 years)

British-born Bengalis (2NG)

Forty-eight men aged 18-42 years (mean 25) born in the UK with both parents born in Bangladesh.

British-born European, low SES (ELO)

Fifty-eight men aged 18-75 (mean 41) born in the UK or Ireland, with both parents of European descent, all resident in London and of similar neighbourhoods and socioeconomic background, as measured by education, income and employment as the migrant groups.

British-born European, high SES (EHI)

Thirty men aged 22-54 (mean 37) born in the UK or Ireland, with both parents of European descent, resident in London and of high socioeconomic background, as measured by education, income and employment.

2.2 Anthropometry:

All measurements were taken according to standardized methods (Lohman et al. 1988). Standing height, weight, arm length and mid-upper arm circumference, triceps, and biceps skinfold measures were collected by KSM or one of four other trained research assistants. Inter- and intra-measurer errors were within recommended technical errors of measurement (Ulijaszek and Kerr 1999)See table 2.2. Estimated mid-upper arm muscle and bone (MUAMus+Bone) was calculated according to the method proposed by Muller and Martorell (1988). Estimated axial fat and skin (MUAFat+Skin) was calculated by subtracting MUAMus+Bone from the total cross sectional area of the mid-upper arm.

Height

Height was assessed using a Seca 214 stadiometer (Seca Corp. Colombia, MD. USA) according to the following method (Gordon et al. 1988). Participants were

either barefoot or in thin socks and asked to stand with feet positioned with heels against the back of the footplate and medial borders of the feet at an angle of about 60°. The vertical plane was positioned along the line of the spine with heels, buttocks, scapulae and base of the cranium in contact with the vertical measure. All head coverings were requested to be removed and the head was positioned in the Frankfort Horizontal Plane where the lower orbital socket was placed in horizontal plane with the external auditory meatus (Henderson and Gregory 2002). Arms were held freely at the sides with palms facing the trunk of the body. Participants were asked to stand as fully erect as possible and take a deep breath. The movable headboard was positioned on the uppermost point of the head with sufficient pressure to compress the hair. Measurement was recorded to the nearest 0.1 cm as indicated on the scale. Intermeasurer differences were found to be within 0.1 cm.

Weight and Body Mass Index

Weight was assessed using a Tanita HD180 digital electronic scale (Tanita Corporation, Arlington Heights IL. USA). Participants asked to remove shoes, all heavy clothing, and any objects from pockets before stepping on the scale.

Anthropometric indices of body mass index (BMI) were categorised into US Dept of Health and Human Services divisions where BMI <18.5:Underweight, 18.5-24.9: Normal, 25-29.9: Overweight, >30: Obese (www.cdc.gov/nccdphp/dnpa/bmi/faq.htm).

2.3 Arm anthropometric measures

Arm length (AL) and arm circumference (AC) were determined using a flexible measuring tape provided by Chasmor Ltd, (London UK.) according to the following method (Callaway et al. 1988). All arm measures were taken from the right arm with the arm muscles in a relaxed state and with the participant standing. Arm circumference and skinfolds were taken with the participant's arm hanging loosely at the side of the trunk. All arm measurements were taken to the nearest 0.1 cm and repeated three times to reduce inter-measurement error (all CV<1.5%).

Arm Length

Participants were asked to remove any clothing obstructing access to the shoulder area and the elbow bent at 90° in order to palpate anatomical landmarks. The measurement was taken from the lateral projection of the acromion process of the scapula to the inferior margin of the olecranon process of the ulna.

Arm Circumference

Upon determining AL, the mean of three measures was divided by two and the mid-point of the upper arm was measured from the lateral projection of the acromion process. A small mark was made with a pen at this point. Using the mark, the zero mark of the tape was positioned just below the mid-point using the left hand, and the remaining part of the tape was aligned along the top of the tape using the right hand. Measurements were taken to the nearest 0.1 cm and repeated three times. All intra- and intermeasurer variations were within recommended limits of 0.2 cm (Callaway et al. 1988) (See table 3).

Skinfolds

Skinfold, or sometimes "fatfold" measurements gauge the thickness of a double layer of skin and subcutaneous adipose tissue at specific sites. A standardized pressure is applied at specific points on the body using calibrated spring-loaded callipers. These measures are useful as 40-60% of total body fat is deposited subcutaneously (Wang et al. 1997). Alone, skinfolds provide a simple and noninvasive method of estimating general fatness (Harrison et al. 1988). During adulthood, circumferences of the limbs, when combined with skinfold measures of subcutaneous adipose tissue at the corresponding level can provide cross-sectional areas of adipose tissue or the underlying area of muscle plus bone. When computed from appropriate formulae, such measures can provide a rough index of the relative somatic apportionment of adipose and muscle tissue (Callaway et al. 1988).

Anthropometric muscle + bone area estimations are limited by several assumptions: 1) the mid-arm circumference is circular 2) skinfold measures are twice the average adipose tissue diameter and this subcutaneous adipose tissue mantle is uniform in thickness 3) the muscle and bone compartments of the mid arm are also assumed to be circular 4) inter- and intra-muscle adipose tissue is absent. 5) the neurovascular bundle of the medial aspect of the arm is not accounted separately, and is included in muscle + bone estimation (Forbes 1987; Martine et al. 1997). These assumptions generally lead to an overestimation of total muscle + bone as the circumference of the arm is actually an ellipse, there is variability in the thickness of subcutaneous deposits of adipose tissue, muscle tissue has an irregular cross-sectional outline which contains minute amounts of inter- and intramuscular adipose tissue, and the medial neurovascular bundle contributes approximately 2-3 cm^2 area to muscle + bone estimations (Forbes 1987; Heymsfield et al. 1982; Martine et al. 1997).

Skinfolds were measured according to the methods outlined by Harrison, et al. (1988) using a Holtain/Whitehouse Skinfold Calliper (Holtain Ltd., Pembrokeshire

UK). Approximately 1 cm above the midpoint mark of AL, the measurer's thumb and forefinger of the left hand was used to elevate a double fold of skin and subcutaneous adipose tissue. Upon elevating the skinfold, callipers were held with the measurer's right hand at the same horizontal plane and level with the midpoint mark. The pressure was released from the callipers gently. Upon full release of calliper pressure, the measurer counted 3 seconds and took the reading in 0.1 cm. The 3-second count was standardised to reduce error effects of fluids being forced from skinfold tissues. The callipers were then removed, and the entire procedure repeated two more times to obtain three independent measures. Mean values were reported, those with CVs over 5% for length and circumference, or 15% for skinfolds were remeasured for a fourth reading or discarded. Triceps skinfold measurements were discarded from one participant due to high CV. Technical error of measurement (see table 3) was calculated using the following formulae (Muller and Martorell 1988; Ulijaszek and Kerr 1999).

$$TEM = \sqrt{(\sum^{N}((\sum^{K}M^{2})-((\sum^{K}M)^{2}/K)))/N(K-1)} \qquad (1)$$

$$\text{Total TEM} = \sqrt{((TEM(intra_{1})^{2} + TEM\ (intra_{2})^{2}) + TEM\ (inter)^{2})/2} \quad (2)$$

Where M, measurement; K number of times measured (In this study K=3 for all arm anthropometric measures); N number of subjects

Triceps skinfold (TSF)

The skinfold was elevated by placing the thumb and forefinger approximately 8 cm apart along a line perpendicular to the long axis of the arm along the posterior ridge of the arm over the triceps muscle.

Biceps skinfold (BSF)

The skinfold was elevated from the anterior aspect of the arm, over the belly of the biceps muscle at the same horizontal plane as the triceps skinfold.

Estimates of Mid-Upper Arm tissue apportionment

Prediction of the mid-upper arm cross-sectional area and muscle + bone area from anthropometric data was calculated using the following formulae (de Koning et al. 1986; Forbes 1987):

Total cross sectional area:

$$\text{Cross Sectional Area} = (AC)^{2}/4\pi \quad (3)$$

Where AC = Arm Circumference

Total muscle + bone area:

$$\text{Muscle + Bone } (cm^2) = [AC - \pi/2(BSF + TSF)]^2/4\pi \qquad (4)$$

These calculations assume the total arm, muscle and bone compartments are all circular. A comparative study of the accuracy of muscle cross-sectional area estimates by Martine et al. (1997), found this method returned the smallest discrepancy (1-5%) between anthropometric measures and cross-sectional CT scanning.

Table 3: Anthropometric technical errors of measurement (TEM)

	Intra-Measurer	N	K	TEM (cm/kg)	% TEM	Intrer-Measurer	N	K	TEM (cm/kg)	% TEM	Total TEM
Arm Length											
	KSM	248	3	1.03	3.14	KSM/RDV	2	3	0.36	1.04	0.6
	RDV	7	3	0.64	1.9						
Arm Circumference											
	KSM	250	3	0.5	1.76	KSM/RDV	2	3	0.09	0.3	0.44
	RDV	7	3	0.12	0.4						
Biceps Skin Fold											
	KSM	291	3	1.05	8.06	KSM/RDV	2	3	0.35	8.24	0.61
	RDV	7	3	0.68	11.1						
Triceps Skin Fold											
	KSM	295	3	1.05	12.91	KSM/RDV	2	3	1.54	13	1.71
	RDV	7	3	0.8	6.12						
Height											
	RDV	6	2	0.06	0.04	KSM/RDV	2	3	0.46	0.25	----
Weight											
	RDV	2	2	0.07	0.1		----	----	----	----	----

2.4 Questionnaires

Demographic and health data was collected using a general questionnaire adapted for migrants and Bangladeshi sedentees (see Appendix 1). The questionnaires were adapted from a similar study on female Bangladesh reproductive health (Núñez de la Mora 2005; Núñez de la Mora et al. 2007a).

Interviews were conducted in Bangla for non-English speakers. Semi-quantitative food frequency questionnaires were also filled in via interview, for future analysis.

After initial piloting (n=6), minor alterations were made to certain socioeconomic questions upon discovering that many of the men were uncomfortable with

socioeconomic questions regarding number and relation of people living in their home (question III.4, appendix 1). The question was altered and "rather not say" was included as an option. The only socioeconomic indicator used for analysis in this study was total household income. Additional questions were added regarding number and age of siblings and household members during the participant's childhood (see questions 1.15-1.17, appendix 1).

Native English speakers were given the option of completing the questionnaire online using the UCL Opinio web-based survey portal (http://www.ucl.ac.uk/isd/students/e-learning/tools/opinio). To encourage completion of the questionnaire, a number of questions were dropped from the web-based version. These questions were: from the Demographic and Health Questionnaire: IV.1-2, which distinguished paid work from home; VIII.8-IX.2, which asked whether practiced contraception, details of birthweight, and open-ended questions regarding leisure activities and social networks. From the Dietary Questionnaire: I.6-14 regarding places eaten outside the home and children's eating habits; III.1-10 regarding frozen, preserved and ready-made meals; and III.24, 33, 37, 41-44 which ask frequency of consumption of squash, seafood, nuts, fizzy drinks, and alternatives to cow's milk (see appendices 1 & 2 for complete printouts of questionnaires).

Prostate symptoms

The occurrence and severity of LUTS were assessed using the International Prostate Symptom Score (IPSS) (Barry et al. 1992). The IPSS combines reported symptoms of frequency, bother, and interference of daily urinary activities. The self-assessed symptoms were combined with perceived quality of life using 6-point Likert scales, summed to score 1-35.

IPSS scores are a World Health Organisation adopted international measure of prostate health used successfully in industrialized countries with a variety of cultures (Barry et al. 1992). Scores were broken into three categories of severity: 0-7 asymptomatic to mild, 8-19 moderate, and 20-23 severe.

Relationship status

Questions on marital status (Questions I.12-I.14, Appendix A) and number of offspring (question VIII, appendix 1) were asked to obtain relationship and fatherhood measures.

Socioeconomic status

To estimate wealth, an index was created of 11 household possessions in the UK, and 8 in Bangladesh (see appendix 1, question III.5). This method of estimating SES is frequently used in cross-cultural contexts as an indicator of the level

of wealth that is consistent with expenditure and income measures (Gwatkin et al. 2007; Montgomery et al. 2000). Television ownership was added to the Bangladeshi questionnaire and assumed nearly ubiquitous in the UK households, while “washing machine”, “tumble dryer”, “dishwasher” and “central heating” were omitted, as these are not common household possessions in Bangladesh where servants usually perform domestic chores.

2.5 Fasting blood glucose and dried blood spots

Fasting blood glucose (FBG) levels were measured from finger-prick blood samples using a disposable lancet and Bayer Asencia blood glucose monitor and detection strips (Bayer Diagnostics, Berkshire UK). Participants were asked not to eat any food or drink aside from water or tea without milk or sugar for 6 hours prior to testing, in practice this meant most (>90%) of the FBG sampling occurred in the morning, before breakfast

2.6 Salivary sampling

A total of six salivary samples were collected from each participant, one upon waking (WAKE), one approximately 30 minutes following waking (W+30), and one right before bed (BED). Participants were asked to record exact times of waking, sampling and going to sleep. The samples were collected over two nonconsecutive days at all three time points. For diurnal ratio (DIR), the two morning samples from both days were averaged (MeanAM) and divided by the mean of BED. Daily mean (DAYM) is an average of MeanAM and BED.

2.7 Salivary Testosterone and Cortisol

Cortisol and Testosterone were assayed from saliva samples to measure the potential influence of a changed environment upon daily variation in hormone levels in migrants. Steroid salivary assays are noninvasive, stable at room temperature, and an accurate measure of bioavailable free T (Dabbs 1990). All participants were asked to not brush their teeth, consume food or chew betel nut for at least 1 hour prior to providing a sample. Sticks of Carefree sugarless chewing gum provided as a stimulant for saliva flow, as this has been validated as not interfering with the assay (Dabbs 1990).

Measurements of salivary hormones should involve multiple samples in order to account for pulsatile and diurnal variation (Dabbs 1990), so samples were collected over two nonconsecutive days. Tubes contained either sodium azide or methionine as preservative and were stored at room temperature for 3-6 months, then refrigerated at 5°c until analysis.

Salivary assay protocol

Salivary testosterone assays were measured by radioimmunoassay without extraction. Antiserum was prepared in the laboratory of Dr. Robert Chatterton at Northwestern University, Chicago USA, where all analyses were performed. The interassay CVs were all within 15% for high (100pg/ml), low (50pg/ml), and internal (pooled saliva sample) quality controls. The sensitivity was 0.028 nmol/L.

Samples of salivary T were analysed according to the following criteria:

Outlying points with readings over 500pg/ml were excluded as they fell outside the standard curve (highest standard=480pg/ml) and estimates beyond this concentration could not be accurately estimated. Readings were made in duplicate and all with a CV greater than 15% were re-analysed or excluded. Of 2018 samples, a total of 39 (1.9%) were excluded for high CV. In addition, outlying readings were identified with absolute salT values showing Z-scores greater than 2.58 (40 samples, 2%) and 3.29 (17 samples, 0.8%). Of these outlying readings, a further 3 samples were dropped from further analysis, 10 were recoded as the daily mean concentration of salT for that participant, 7 were recoded as a singulate reading due to spillage or other laboratory error for the duplicate reading, and 9 were analysed without applying a correction factor.

2.8 Salivary validation studies

As salT was analysed over different years, a number of samples and QCs were measured repeatedly. These repeat measures were used to adjust readings between years of analysis. A total of 191 samples first collected and analysed in 2005, 66 samples first collected and analysed in 2006, and 17 samples first collected and analysed in 2008 were measured a second time in 2010. From these repeat measures, a correction factor of 3.06 was applied to all other readings from 2005, and 0.84 was applied to all other readings from 2006. Repeat measures from 2008 assays did not require a correction factor to match 2010 readings.

Betel nut study

As the chewing of betel nut (*paan*) is endemic practice among Bangladeshi males, (especially the older generation), a sub-study on potential interaction effects of betel nut chewing upon the salivary hormone assay was run on a sample of 20 male participants according to the following method: men were instructed not to chew *paan* for at least 12 hours prior to the study, and they were asked not to consume any food or drink aside from water for at least 30 minutes prior to the study. One saliva sample was taken as a baseline, following which the participant prepared and chewed their paan in their usual manner, typically a combination of betel nut, calcium oxide, and tobacco wrapped in a betel leaf to form a “quid”

which is chewed for approximately 20 minutes and spat out. A saliva sample was collected immediately following the chewing of the betel quid, and the time noted down. Further saliva samples were collected at 30, 60, 120, and 240 minutes following the chewing of the quid.

These saliva samples were analysed for levels of free testosterone and cortisol according to the same laboratory method outlined above to determine if there is a significant interaction between the chewing of paan and the salT. This experiment mirrors a similar investigation on the salivary influences of betel nut chewing on salivary hormone samples from Bangladeshi women (Núñez de la Mora et al. 2007b).

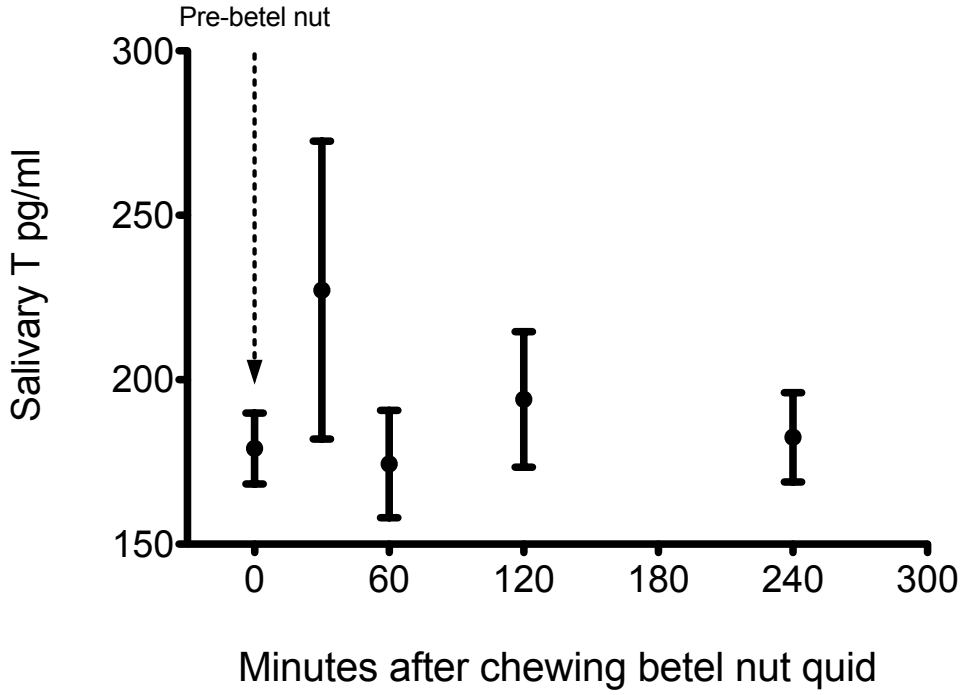
Betel nut results

Samples from 20 participants were analysed by a repeated measures ANOVA, which did not show significant differences between sample times ($F(4,19)=0.94$, $p=.44$). Correlation between mean salT at all sampling times indicated the pairing was significantly effective ($R^2=.35$, $p=.006$), and Bonferroni's post hoc tests did not show any significant differences between mean sample times (see table 4)

Table 4: Betel Nut Repeated Measures ANOVA

Bonferroni's Multiple Comparison Test	Mean Diff.	t	Summary	95% CI of diff
Pre-BN vs 30-Min Post-BN	-48.16	1.55	ns	-138.0 to 41.68
Pre-BN vs 1 hr post-BN	4.71	0.1516	ns	-85.13 to 94.55
Pre-BN vs 2 hrs post-BN	-14.92	0.4802	ns	-104.8 to 74.92
Pre-BN vs 4 hrs post-BN	-3.397	0.1093	ns	-93.24 to 86.44
30-Min Post-BN vs 1 hr post-BN	52.87	1.702	ns	-36.97 to 142.7
30-Min Post-BN vs 2 hrs post-BN	33.24	1.07	ns	-56.60 to 123.1
30-Min Post-BN vs 4 hrs post-BN	44.76	1.441	ns	-45.07 to 134.6
1 hr post-BN vs 2 hrs post-BN	-19.63	0.6318	ns	-109.5 to 70.21
1 hr post-BN vs 4 hrs post-BN	-8.107	0.2609	ns	-97.95 to 81.73
2 hrs post-BN vs 4 hrs post-BN	11.53	0.3709	ns	-78.31 to 101.4
			R^2	0.3548
			F	2.308
			P value	0.0055

Figure 8: Salivary T and recent betel nut use



Error bars ± SEM.

Matched saliva/serum samples

A smaller subgroup of 20 individuals provided a time-matched blood sample and saliva sample to validate the reliability of the salivary assay in determining serum concentrations of testosterone in this study population. The blood samples were collected at Sylhet Osmani Medical College by Dr. Kurshida Begum according to the methodology described above.

Salivary T assays were measured by radioimmunoassay without extraction see appendix 4 for laboratory protocol. Antiserum was prepared in the laboratory of Dr. Robert Chatterton at Northwestern University, Chicago USA, where all analyses were performed. The interassay CVs were all within 15% for high (100pg/ml), low (50pg/ml), and internal (pooled saliva sample) quality controls. The sensitivity was 0.028 nmol/L.

Samples of salT were analysed according to the following criteria:

The overall degree of diurnal change in salivary T was expressed as a mean value at each time point over two nonconsecutive days, with the evening sample value subtracted from the morning value and expressed as a percentage. The morning sample was considered the peak level of T, as has been observed in numerous other studies (Campbell et al. 2005; Ellison et al. 2002) and can be considered a “top” baseline for the day.

All statistical analysis was conducted using SPSS for Macintosh OSX 11.0.

ANCOVA was used to model the relationship between anthropometric measures (as dependent variable) and residence group after correcting for age differences. ANCOVA was also used to model the relationship between muscle + bone area with the number of years spent in the UK, where AI were subdivided into three groups indicating years since migrating: (<10 years, 11-40 and >40) and BR inserted as the dummy variable. ANCOVA was also used to model the relationship between salivary T (as dependent variable) and marital or fatherhood status after correcting for age and BMI differences.

2.9 Statistical analysis

All statistical analysis was conducted using SPSS 18.0 or GraphPad Prism 5 for Macintosh OSX.

Missing values analysis

All missing anthropometric, salT, age at maturity, dietary and SES, measures were analysed by expectation-maximization methods to estimate means, correlations, and covariances and found to be random in respect to residence group, whether respondents were older or younger than 40 years old and relationship/parental status (See table 5 for Little's MCAR test results).

Table 5: Missing values analysis : Little's MCAR test

	X^2	df	p
Anthropometrics	24.2	15	.6
salT	17.4	17	.4
Age at maturity	34.1	26	.1
SES measures	20.3	17	.3
Dietary frequencies	44.9	48	.6

Categorical variables: Residence group, Age 40 split, marital and offspring status

Residential variation in salivary T

To assess the relationship between ecological conditions and reproductive function, salivary T concentrations at the time points WAKE, W+30 and BED were each averaged between D1 and D2 readings, and analysed as outcome variables in separate multiple linear regressions. To measure group differences the categorical variable "residence" was entered into the model as a dummy variable, with the Sylheti sedentee group (SED) serving as reference group. Measures of participant age and BMI were entered into the model as covariates.

To isolate somatic and hormonal variation due to senescence, the sample groups were split at age 39 years and younger and age 40 years and older. To reduce the

influence of acute variations in salT in order to assess the relationship between ecological conditions and reproductive function, salT concentrations at the time points WAKE, W+30 and BED were each averaged between D1 and D2 readings, and analysed as outcome variables in separate multiple linear regressions.

Analyses of salT or anthropometric variables by residence group were performed with two separate multiple linear regressions (MLR), to test two hypotheses, after correcting for age and/or BMI where appropriate.

The first model, MLRI measured group differences from SED. The categorical variable “residence” was entered into the model as a dummy variable, with SED serving as reference group. Measures of participant age and BMI were entered into the model as covariates where appropriate.

The second model, MLRII applied planned orthogonal contrasts to test for differences between groups with contrasting developmental histories. Contrast 1 compared the men with shared childhood under Bangladeshi conditions (SED, ADM) to men with shared (all or part) childhood in the UK (YOM, 2NG, ELO, EHI). Contrast 2 subdivided the men who lived in the UK as children according to ethnicity (YOM, 2NG) versus (ELO, EHI). Contrast 3 compared Bengalis with differing childhood conditions: YOM versus 2NG. Contrast 4 compared European males with differing socioeconomic status, EHI versus ELO. The final planned contrast compared Bengali men experiencing different conditions in adulthood: SED versus ADM. Post hoc Sidak comparisons were run of all remaining group differences.

For age at migration analysis, MLRI, the groups YOM and ADU were split into developmental period of migration. YOM were split into three periods; infancy: aged <2 years (n=6); childhood: aged 2-12 (n=12); adolescence: aged 12-18 (n=15). ADU were split between early adulthood: age 18-30 (n=36) and late adulthood (n=12). A second model, MLRII included 2NG (n=26) as a reference group, and MLRIII combined 2NG with infant YOM into a single pre-birth to age 2 group (n=32) with late adult ADM serving as reference group.

Validation study results

Repeated measures and correction factors:

As salT was analysed over several years, a number of samples and QCs were measured repeatedly. These repeat measures were used to adjust readings between years of analysis. A total of 191 samples collected and analysed in 2005, 66 samples collected and analysed in 2006, and 17 samples collected and analysed in 2008 were measured again in 2010. From these repeat measures, a correction factor of 3.06 was applied to all other readings from 2005, and 0.84

was applied to all other readings from 2006. Repeat measures from 2008 assays did not require a correction factor to match 2010 readings.

Serum to salivary T comparisons

Method: a total of 22 participants were asked to provide a saliva sample according to the same collection procedure detailed for all samples collected in the field. Immediately following the provision of a saliva sample, a serum sample was collected. Two sample tubes were mislabelled and were therefore dropped from the final analysis.

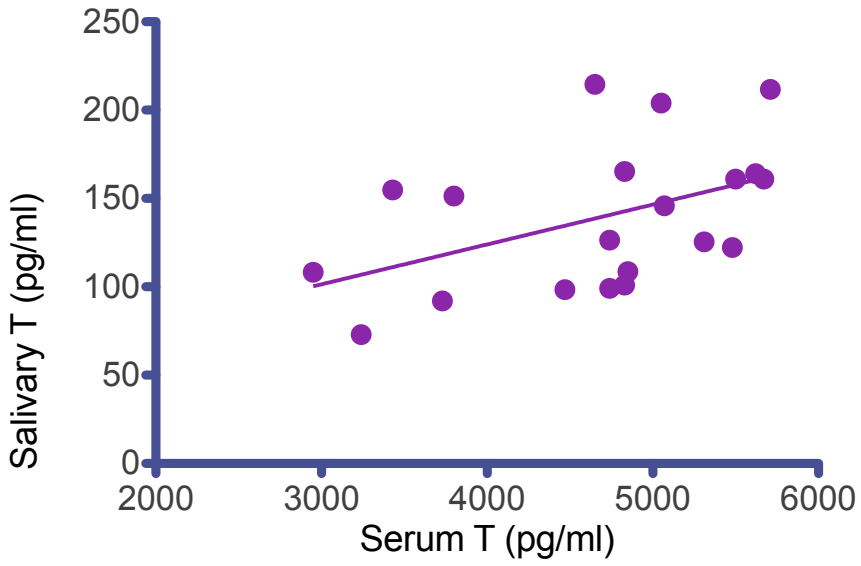
Serum concentrations of total T were determined using a commercially available radioimmunoassay (Diagnostic System Laboratories, Webster, Texas USA) at the Chatterton laboratory at Northwestern University. High and low concentration QCs were within recommended limits (see table 6)

Table 6: Serum T assay QCs

QC	Expected	Read
I	0.65 ± 0.2	0.62
II	5.7 ± 1.7	5.02

Result: A linear regression of salT by serum T concentrations was significant ($F(1,18)=4.9$, $p=.04$) indicating the correlation between matched salT and serum T samples, with Pearson's $R^2=.21$ (see figure 9).

Figure 9: Matched Salivary T to SerumT



Discussion: The modest correlation between total serum T and free T measured in saliva by RIA is lower than correlations R^2=.62-.79 reported for human males elsewhere (Lewis 2006; Shirtcliff et al. 2002), but comparable to the correlation resulting from a salivary enzyme immunoassay (R^2=.29) (Shirtcliff et al. 2002). The disparity between readings is partly attributable to the binding of free T in serum. Approximately 60% of serum total T concentration is inhibited by high-affinity binding to sex hormone-binding globulin (SHBG), and this portion is generally considered non-bioactive. Another 38-39% is loosely bound to albumin, while the remaining 1-2% of "free" circulating T is lipid-soluble and freely passes through the membranes of the salivary gland acinar cells to permeate the saliva. In this way, the highly bioactive, unbound portion of the steroid diffuses freely through the salivary gland and independently of the salivary flow rate (Gröschl 2008; Lewis 2006).

In light of the above, the discrepancy between a total serum T and salT measure can lead to an underestimation between 8-32% of a correlation between T and a known behavioural effect, thus reducing analytical power and increasing the potential occurrence of type II error (Granger et al. 2004). This potential underestimation will be reduced by taking multiple measures (3) from each participant over 2 separate days, but should be kept in mind when interpreting null findings from smaller subgroups.

Inter-daily variation:

In order to detect systematic repeated measure effects, salivary T values of waking (WAKE), 30 minutes post-waking (W+30) and bedtime (BED) samples were compared between sampling day 1 (D1) and day 2 (D2).

Table 7: Paired *t*-test of inter-daily sample waking (WAKE), 30 Minutes Post-Waking (W+30) and Bedtime (BED) salivary T (pg/mL)

	Paired Correlations			Paired Differences			Paired Samples Test		
Pair	N	r	Sig.	Mean	Std. Dev.	S.E.M	t	df	Sig. (2-tail)
D1WAKE-D2WAKE	266	.58	.000	6.52	58.15	3.57	1.83	265	.068
D1W+30-D2W+30	253	.67	.000	-3.04	48.15	3.03	-1.01	252	.316
D1BED-D2BED	267	.55	.000	-0.20	47.78	2.92	-0.07	266	.945

Each pair was strongly correlated and highly significant (Pearson's $r \geq .55$ $p<.001$). None of the pairs showed a repeated measure effect, with non-significant differences between day 1 and day 2 of sampling (all $p \leq .07$, paired t-test, 2-tailed) (see table 7 for coefficients).

Intra-daily variation

To determine whether there were significant diurnal variations in salivary T measures taken at different times within the same day, salivary T values of WAKE, W+30 and BED samples were paired and tested for significance within D1 and D2.

Table 8: Paired *t*-test of intra-daily sample Waking (WAKE), 30 Minutes Post-Waking (W+30) and Bedtime (BED) salivary T (pg/mL)

	Paired Correlations			Paired Differences			Paired Samples Test		
Pair	N	r	Sig.	Mean	Std. Dev.	S.E.M	t	Df	Sig. (2-tail)
D1WAKE-D1W+30	278	.67	.000	9.26	48.55	2.91	3.18	277	.002
D1WAKE-D1BED	289	.50	.000	29.84	57.24	3.37	8.86	288	.000
D1W+30-D1BED	280	.55	.000	20.74	51.78	3.09	6.70	279	.000
D2WAKE-D2W+30	268	.62	.000	-0.38	53.17	3.25	-0.12	267	.907
D2WAKE-D2BED	269	.60	.000	25.91	52.14	3.18	8.15	268	.000
D2W+30-D2BED	262	.60	.000	27.54	50.43	3.12	8.84	261	.000

Each within-day pair was strongly correlated and highly significant (Pearson's $r \geq 5$ $p<.001$). All sample pairs except D2WAKE-D2W+30 were significantly different (all $p \leq .005$, paired t-test, 2-tailed). Means for the two morning samples were higher than the mean for bed, with diurnal declines in salivary T averaging between 20.7-29.8 pg/mL. The first 30 minutes following waking did not show a consistent pattern, with WAKE-W+30 pairs showing a mean difference of 9.26 pg/mL for D1, but the D2 pair were extremely similar with a mean difference of -0.38 pg/mL, $p=.9$ (see table 8).

2.10 Conclusions

There was no indication of a systematic effect depending on the day of sampling. Combining these values will reduce the amount of variation due to proximate daily events, thus all D1 and D2 samples were averaged according to sample time for subsequent analysis.

Samples collected at waking and 30 minutes post waking were significantly higher in salivary T than samples collected before bed, thus the average daily pattern was consistent with the general diurnal pattern of a morning peak in T, and gradual decline throughout the day. The two morning samples were significantly different for D1, but not D2, suggesting that any changes in salivary T in the first 30 minutes of the day were inconsistent or beyond methodological limits of detection. As they are closely spaced, the differences in these morning samples may be more reflective of pulsatile variation in salivary T. For the purposes of

measuring diurnal decline, WAKE and W+30 samples may be averaged to obtain a value for the morning peak, from which the averaged BED samples may be subtracted to obtain a value for diurnal decline.

Chapter 3: Testing developmental hypotheses

3.1 Introduction

In this chapter I test the hypothesis that improvements in developmental conditions increase reproductive investment in migrant males as measured by adult somatic and hormonal characteristics. Age at migration serves as a predictive variable for daily patterns of salivary testosterone, two measures of stature (standing height and humeral length), estimates of skeletal muscle and axial body fat.

In applying a life history analysis to anthropometric and hormonal measures, this chapter makes the following two assumptions. Firstly, the shift in environment from Sylhet, Bangladesh to London, UK, during childhood will influence patterns of growth and reproductive strategies in ways that will be detectable in adult males. Secondly, phenotypic matching to ecological conditions will be based on cues received during growth and, when the environment improves or becomes less stochastic, plastic characteristics will correspond to adult environment with a more matched phenotype. Phenotypic characters that are more canalised or have undergone irreversible development will be mismatched to adult environment (Kuzawa 2007; Stearns 2000; West-Eberhard 1989).

Life-history trade-offs are evident in observed variability in human male and female reproductive steroid hormone levels. Non-industrialised or subsistence populations show lower levels of free testosterone when compared with populations in developed nations (Bribiescas 1996; Ellison et al. 2002). Measures of testosterone (T) are presumed to reflect greater immunological, nutritional or other energetic challenges (Bribiescas 2001a; Charnov 1993). Androgen levels of adult males, particularly free T, respond acutely to changes in nutrition, social conditions, physical activity, and immune challenges (Muehlenbein and Bribiescas 2005) (Campbell et al. 2001) (Bribiescas 2001a).

For women, adult progesterone levels appear to be influenced by conditions experienced prior to puberty, and remain unchanged despite improved conditions in adulthood (Núñez de la Mora et al. 2007a). It is unknown, however, whether adult male reproductive function is similarly constrained by childhood conditions, although recent evidence suggests that environmental stressors in the first six months of life influence hormonal and somatic characters in adult males (Kuzawa et al. 2010).

Migrants from Sylhet, Bangladesh to London, UK experience a discontinuous developmental environment, with fewer immune challenges or other presumed constraints on energy balance following migration. In order to understand further such influences on the reproductive hormones of adult men, this project

compared salivary testosterone and anthropometric measures of a group of adult Bangladeshi migrants who relocated to London as children (aged <18 years) and as adults (aged >18 years) with men of Bengali or European ethnicity resident all their lives in London, or with Sylheti sedentees. Age at migration acts as an experimental variable in this study in order to observe whether adult patterns of salT variation are influenced by ecological conditions experienced during development.

Hypotheses

This chapter tests three main hypotheses:

The experience of more limited or stressful conditions during development prior to maturity constrains investment in reproductive effort, and primes males to respond to improvement in conditions during early adulthood with an immediate increase in reproductive function. This is a discounting future survivorship by increasing investment into reproductive competition, as measured by salT when young adults, if childhood conditions were poor and they have improved. In contrast, men divert more resources toward reproductive competition at older ages if they have attained high status, irrespective of preadult developmental conditions. In men aged under 40, investment in reproductive effort will be influenced more by immediate conditions than by those experienced prior to maturity, if the immediate conditions are an improvement from childhood conditions.

1. Men in a less constrained environment are free to invest greater resources into reproductive effort. If men migrate to the UK before the age of maturity, they will show greater investment in reproductive effort than sedentees and adult migrants as measured by salT and into growth, as measured by skeletal stature, muscle and body fat.

   Predictions:

   a. All migrant and British-born Bengalis will show higher salT, as well as greater BMI and skeletal muscle compared to sedentees.

   b. Stature of young migrants will be greater than sedentees and adult migrants.

   c. Young migrants and British-born Bengalis will have sexually matured at a younger age than adult migrants or sedentees.

2. If developmental environments of energetic affluence permit the adoption of a more diverse set of strategies in male reproductive effort, (in effect a wider bandwidth of potential reproductive strategies), this will be reflected in wider daily variation in reproductive hormones. Therefore, young men who

experienced an environment of fewer constraints will have greater inter- and intra-individual variability in salT.

Predictions:

British-born Bengalis will show greater diurnal variation in salT compared to young migrants, and both young migrants and British Bengalis will show greater diurnal variation compared to adult migrants and sedentees. European males will show similar diurnal variation to young migrants and British-born Bengalis, and greater measures of salT than sedentees and adult migrants.

3. In evolutionary history, older males were unlikely to succeed in direct physical competition with younger males for reproductive opportunities, but older males will have had the opportunity to acquire status in early adulthood. The diversion of metabolic energy toward reproductive effort is more likely to be maintained in older males of high status than in men of the same age of lower status. If older males are of high social status, relative to surroundings, they will invest more in reproductive effort. If they are lower, they will invest less into reproductive effort.

Predictions:

Adult migrants over the age of 40 will have greater salivary T than sedentees, high status European males will have greater salivary T than low status Europeans.

3.2 Methods

Recruitment

Salivary testosterone, anthropometric, and demographic data were collected from the following populations according to methods described in chapter two.

1. ADM: Bengali migrants who arrived in London at <18 years old (pre/peri-puberty) (N=61)
2. YOM: Bangladeshi migrants who arrived in London at >18 years old (post-puberty) (N=50)
3. 2NG: Second generation ethnic Bengalis born in the UK (N=48)
4. SED: Native Bengalis resident in Bangladesh (N=107)
5. ELO: Native Londoners of white British or other white European parentage, of similar socioeconomic background compared to migrant groups (N=58)

6. EHI: Native Londoners of white British or other white European parentage, of higher status socioeconomic background compared to migrant groups (N=30)

Statistical analysis:

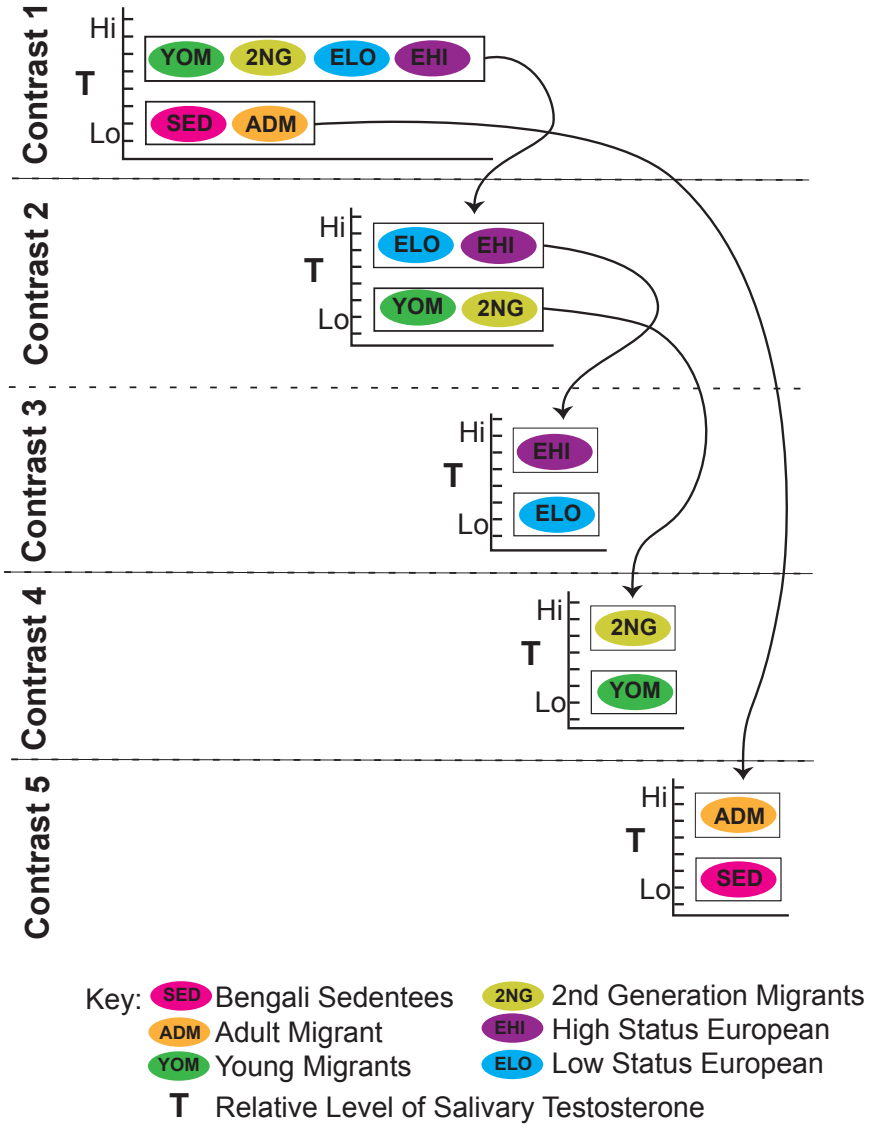
To isolate somatic and hormonal variation due to senescence, the sample groups were split at age 39 years and younger (<40) and age 40 years and older (≥40). In order to assess the relationship between ecological conditions and reproductive function and to reduce the influence of acute variations in salT, concentrations at the time points WAKE, W+30 and BED were each averaged between D1 and D2 readings, and analysed as outcome variables in separate multiple linear regressions.

Analyses of salT or anthropometric variables by residence group were performed with four separate multiple linear regressions (MLR), after correcting for age and/or BMI where appropriate. The addition of height as a separate covariate did not significantly improve the model, so was not included.

The first regression (MLR-I) tested for differences from the sedentee reference group. The categorical variable “residence” was entered into the model as a dummy variable. Measures of participant age and BMI were entered into the model as covariates where appropriate. The second model tested for differences between groups depending on age of migration or ethnic differences.

The second regression (MLR-II) applied planned orthogonal contrasts to test for differences between groups with contrasting developmental histories, with the hypothesis that men who spent more of their childhood in the UK would have greater reproductive investment, expressed as higher salT (see figure 10).

Figure 10: Sequence of hypothesis statements



- Contrast 1 compared the men with shared childhood under Bangladeshi conditions (sedentees and adult migrants) to men with shared (all or part) childhood in the UK (young migrants, British Bengalis and Europeans).
- Contrast 2 subdivided the men who lived in the UK as children according to ethnicity, comparing young migrant and British-born Bengalis with European men.
- Contrast 3 compared Bengalis with differing childhood conditions: Young migrants versus British-born Bengalis.
- Contrast 4 compared European males of high versus low socioeconomic status.
- Contrast 5 compared Bengali men who experienced different conditions in adulthood: sedentees versus adult migrants.

- Post hoc Sidak comparisons were run of all remaining group differences.

For age at migration analysis, (MLRIII), migrants were split according to stages of development when relocated. Young migrants were split into three periods; infancy: aged ≤2 years (n=6); childhood: aged 3-12 (n=12); adolescence: aged 13-18 (n=15); early adulthood: age 19-30 (n=36) and late adulthood ≥30 years (n=12).

A final analysis of age at migration (MLR-IV) included 2NG (n=26) as a reference group, and combined British-born with infant migrants into a single pre-birth to age 2 group (n=32) with adult migrants who arrived ≥30 serving as reference group.

3.3 Results

Descriptives

The adult migrants were significantly older than all other groups with an average of 48.8 years (15.8 SD), and the British-born Bengalis were the youngest group, averaging 24.5 years (5.7 SD), the average of all populations was 37.9 (15 SD). This cohort effect reflects the demography of the UK Bengali community, with most adult male migration having occurred in the 1970s, which was followed by wives and children in the 1980s (see chapter 1).

All migrant groups under the age of 40 have more upper arm muscle, and more axial body fat, compared to sedentees. All migrants over the age of 40 have more muscle, but not more axial fat than sedentees. Youth migrants and British Bengalis are both taller than adult migrants and sedentees, and British Bengalis were taller than youth migrants. Upper arm length was consistent within ethnic groups, with no difference between residence categories, and both measures of stature were higher in the European groups than the Bengali groups.

Residence groups were significantly different from one another in all anthropometric variables, except between a) youth migrants and British-born Bengalis for weight, AL and both MUA tissue estimates; b) sedentees and adult migrants for both MUA tissue estimates; c) sedentees and all three migrant groups for arm length; and d) high and low status European men for MUAFat+Skin (tables 9,10 and 11).

Regressions of anthropometric variables by age were significantly different (all $p \leq .05$) in the SED group for weight (slope=.15), AC (slope=.06), and both MUA estimates (M+B slope=.46; F+S slope=-.20) and was significant in the YOM group for height (slope=-.28). Analyses of these measures were therefore corrected for age effects.

Differences in intercepts between residence groups for all measures were highly significant (all $p \leq .001$), while the differences in slopes between groups was not significant, except for both MUA measures (both $p \leq .005$). Arm length and MUA fat+skin were log transformed to correct for negative skew (Levene's test, $p < .05$).

Linear regressions of each anthropometric variable by age, within age category <40, indicated significant relationships between age and weight, arm length, MUA muscle+bone, and arm circumference in sedentees, and for MUA fat+skin in high SES Europeans. Within age category ≥40, slopes were significant for weight in low SES Europeans, and for MUA muscle+bone and arm circumference in high SES Europeans (see figure 13). Analyses of the above measures were therefore corrected for age effects.

After splitting by age, the height difference between sedentees and youth migrants was not significant in the older age category, while adult migrants were not taller in either age category. All other residence groups were significantly taller than sedentees (see table 12).

All migrant groups < 40 have more upper arm muscle, and more axial body fat, compared to sedentees. All migrants ≥ 40 have more muscle, but not more axial fat than sedentees. Youth migrants and British Bengalis are both taller than adult migrants and sedentnees, and British Bengalis were taller than youth migrants. Upper arm length was consistent within ethnic groups, with no difference between residence categories, and both measures of stature were higher in the European groups than the Bengali groups.

Table 9: Descriptive measures by residence group, all ages, mean (std dev)

	Residence Group	SED	ADM	YOM	2NG	ELO	EHI	Total
		N=107	N=61	N=50	N=48	N=58	N=30	N=349
Somatic	**Age (years)**	38.6 (14.2)	48.8 (15.8)	31.5 (10.2)	24.5 (5.7)	41 (16.2)	37.1 (10.3)	37.9 (15)
	Height (cm)	162.8 (5.6)	164.1 (6.7)	167.5 (6.0)	171.5 (5.5)	177.3 (6.3)	178.9 (7.3)	168.5 (8.6)
	Weight (kg)	60.0 (9.2)	67.3 (9.1)	69.8 (12.0)	71.1 (12.7)	76.6 (10.8)	81.4 (10.7)	68.6 (12.5)
	BMI (kg/cm2)	22.6 (3.2)	25 (2.9)	24.8 (3.6)	24.3 (3.7)	24.3 (3.2)	25.3 (2.4)	24.1 (3.4)
	Arm Length (cm)	32.6 (1.6)	32.2 (2.0)	32.1 (1.9)	33.0 (1.7)	34.4 (2.8)	34.9 (1.9)	33 (2.2)
	Arm Circumference (cm)	27.7 (2.8)	30 (2.5)	30 (3.6)	29.2 (3.4)	31.2 (3.2)	31.6 (2.3)	29.5 (3.2)
	MUA Muscle+Bone (cm2)	44.2 (11.5)	58.1 (9.1)	59.4 (13.7)	56.6 (12.3)	63.4 (13.9)	68.5 (9.8)	55.1 (14.3)
	MUA Adipose+Skin (cm2)	17.3 (8.1)	13.7 (4.7)	12.7 (5.1)	12.3 (5.1)	12.3 (4.2)	11.3 (2.8)	14.2 (6.4)
Hormonal	**WAKE mean salT (pg/mL)**	100.9 (49.0)	93.48 (42.38)	137.32 (69.57)	151.18 (54.24)	114.54 (52.62)	150.9 (42.6)	115.68 (55.32)
	W+30 mean salT (pg/mL)	95.67 (47.83)	93.15 (38.49)	134.62 (56.65)	154.84 (66.87)	97.26 (53.96)	148.68 (48)	111.39 (55.71)
	BED mean salT (pg/mL)	76.22 (40.75)	73.76 (33.95)	102.59 (47)	118.82 (59.52)	92.07 (64.93)	109.62 (29.88)	88.93 (48.58)
	DAY mean salT (pg/mL)	87.85 (40.56)	84.25 (34.8)	119.65 (50.16)	135.82 (53.23)	98.94 (48.21)	129.71 (32.29)	101.71 (46.61)
	Diurnal Ratio (AM/BED)	1.46 (0.66)	1.34 (0.53)	1.6 (1.08)	1.63 (1.24)	1.71 (1.46)	1.41 (0.32)	1.5 (0.91)

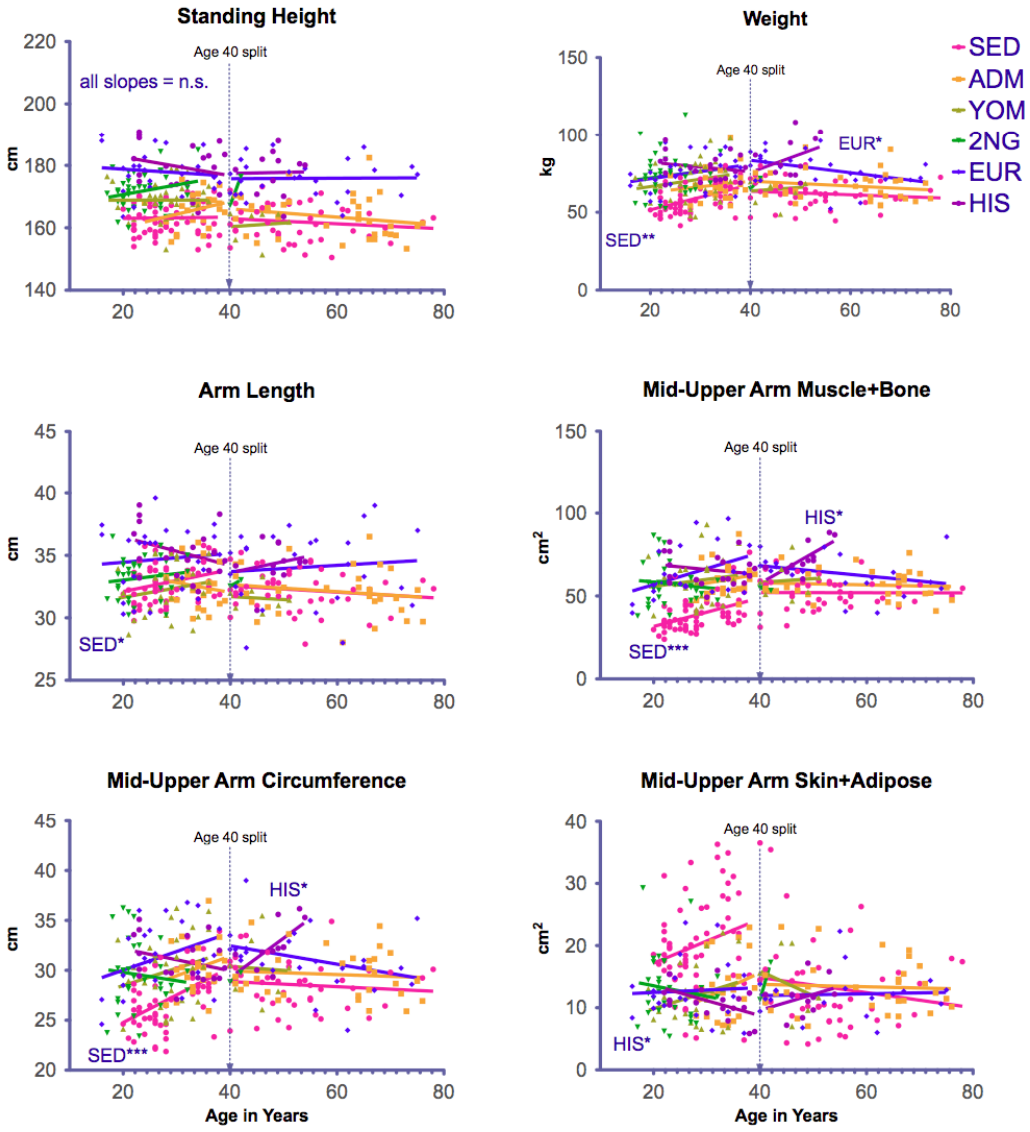
Table 10: Descriptive measures by residence group, split at age ≤39, mean (std dev)

	Residence Group	SED	ADM	YOM	2NG	ELO	EHI	Total
		Age ≤39 years						
		N=60	N=23	N=33	N=37	N=25	N=19	N=213
Somatic	Age (years)	28.1 (4.9)	33.2 (4.2)	27.5 (5.7)	23.7 (4.4)	27.7 (6.7)	30.5 (6.1)	28 (6)
	Height (cm)	163.2 (5.4)	165.8 (5.7)	168.9 (5.6)	171.7 (5.8)	178.0 (6.7)	179.6 (7.6)	169.5 (8.3)
	Weight (kg)	58.1 (9.3)	67.6 (10.2)	70.2 (12.6)	73.5 (12.8)	74.8 (11.4)	79.0 (8.4)	68.3 (13)
	BMI (kg/cm2)	21.8 (3.1)	24.6 (3.1)	24.5 (3.8)	24.9 (3.8)	23.6 (3.1)	24.5 (1.6)	23.6 (3.5)
	Arm Length (cm)	32.8 (1.6)	32.5 (1.7)	32.3 (2.1)	33.2 (1.6)	34.7 (2.6)	35.3 (2)	33.3 (2.1)
	Arm Circumference (cm)	27.0 (2.9)	30.2 (2.6)	29.9 (3.6)	29.5 (3.4)	31.3 (3.5)	31.0 (2.2)	29.2 (3.5)
	MUA Muscle+Bone (cm2)	38.6 (9.9)	59.9 (10.2)	59.3 (14.6)	57.3 (12.5)	63.7 (15.9)	65.8 (9.4)	53.3 (15.9)
	MUA Adipose+Skin (cm2)	20.1 (7.4)	13.2 (5)	12.1 (4.5)	13.0 (5.4)	12.7 (4.7)	11.0 (2.8)	15 (6.8)
Hormonal	WAKE mean salT (pg/mL)	85.5 (37.8)	96.6 (33.4)	157.3 (58.1)	156.7 (53.5)	109.6 (62.9)	154.7 (34.2)	119.0 (56.0)
	W+30 mean salT (pg/mL)	80.5 (35.9)	93 (31.4)	153.4 (52.4)	156.8 (63)	91.4 (57.1)	163.3 (49.9)	115.0 (58.6)
	BED mean salT (pg/mL)	63 (29.8)	77.7 (31.9)	112.1 (46.4)	119.9 (56.9)	73.9 (58.1)	114.2 (30.6)	88.0 (47.6)
	DAY mean salT (pg/mL)	73 (27.9)	87.2 (26.9)	133.8 (44.4)	137.1 (51.6)	86.6 (47.5)	136.6 (32.6)	102.7 (47.1)
	Diurnal Ratio (AM/BED)	1.5 (0.7)	1.4 (0.7)	1.7 (1.2)	1.5 (0.8)	2 (1.6)	1.4 (0.3)	1.6 (1.0)

Table 11: Descriptive measures by residence group, split at age ≥40, mean (std dev)

	Residence Group	SED	ADM	YOM	2NG	ELO	EHI	Total
		Age ≥40 years						
		N=45	N=34	N=7	N=2	N=27	N=11	N=133
Somatic	**Age (years)**	52.6 (9.6)	59.6 (11.1)	47.4 (8.5)	41 (1.4)	54.3 (11)	48.5 (3.6)	54 (10.6)
	Height (cm)	161.9 (5.8)	163.6 (6.8)	161 (5.2)	171.8 (6.5)	176.1 (5.9)	177.7 (7.2)	166.9 (8.9)
	Weight (kg)	62.1 (8.6)	67 (9.3)	64.9 (10.5)	68.4 (4.8)	78.4 (10.5)	85.6 (13.1)	69.2 (12.4)
	BMI (kg/cm2)	23.6 (3.1)	25 (2.8)	24.9 (2.9)	23.2 (0.1)	25.2 (3.2)	26.7 (3)	24.7 (3.1)
	Arm Length (cm)	32.2 (1.6)	32.1 (1.9)	31.6 (1)	32.8 (0.4)	34.1 (3)	34.4 (1.6)	32.8 (2.2)
	Arm Circumference (cm)	28.6 (2.5)	29.6 (2.4)	30.2 (3.2)	29.3 (0.7)	31.2 (3)	32.6 (2.2)	29.8 (2.9)
	MUA Muscle+Bone (cm2)	52 (8.9)	56.7 (8.7)	59.4 (11.1)	54 (1.2)	63.7 (12)	73.2 (9.2)	57.8 (11.4)
	MUA Adipose+Skin (cm2)	13.3 (7.3)	13.4 (4.2)	13.8 (5.2)	14.3 (4.2)	12.2 (3.6)	11.8 (2.9)	13 (5.5)
Hormonal	**WAKE mean salT (pg/mL)**	121.1 (54.9)	84.1 (41)	73.7 (52.1)	151.4 (12.5)	112.2 (44.4)	144.4 (55.5)	109.0 (52.4)
	W+30 mean salT (pg/mL)	115.8 (54.1)	85.5 (40.1)	89.8 (44.4)	106.9 (45.6)	100.1 (53.1)	123.5 (33.1)	104.3 (49.5)
	BED mean salT (pg/mL)	93.8 (46.4)	65.6 (33.3)	70.9 (45.1)	76.1 (40.3)	104.5 (68.4)	101.8 (28.3)	87.5 (49.1)
	DAY mean salT (pg/mL)	107.3 (46)	76.5 (36.9)	74.3 (35.6)	102.6 (34.7)	104.9 (48.9)	117.9 (29.4)	97.2 (44.4)
	Diurnal Ratio (AM/BED)	1.4 (0.6)	1.3 (0.4)	1.5 (0.6)	1.9 (0.6)	1.5 (1.4)	1.4 (0.4)	1.4 (0.8)

Figure 11: Regression of anthropometric variables by participant age



Linear regressions of resident group anthropometric variables by age, with analysis split at age 40 years.
Significance values are for slopes significantly deviating from 0 *=p≤.05 **=p≤.005, ***p≤.001.

Figure 12: General anthropometric measures

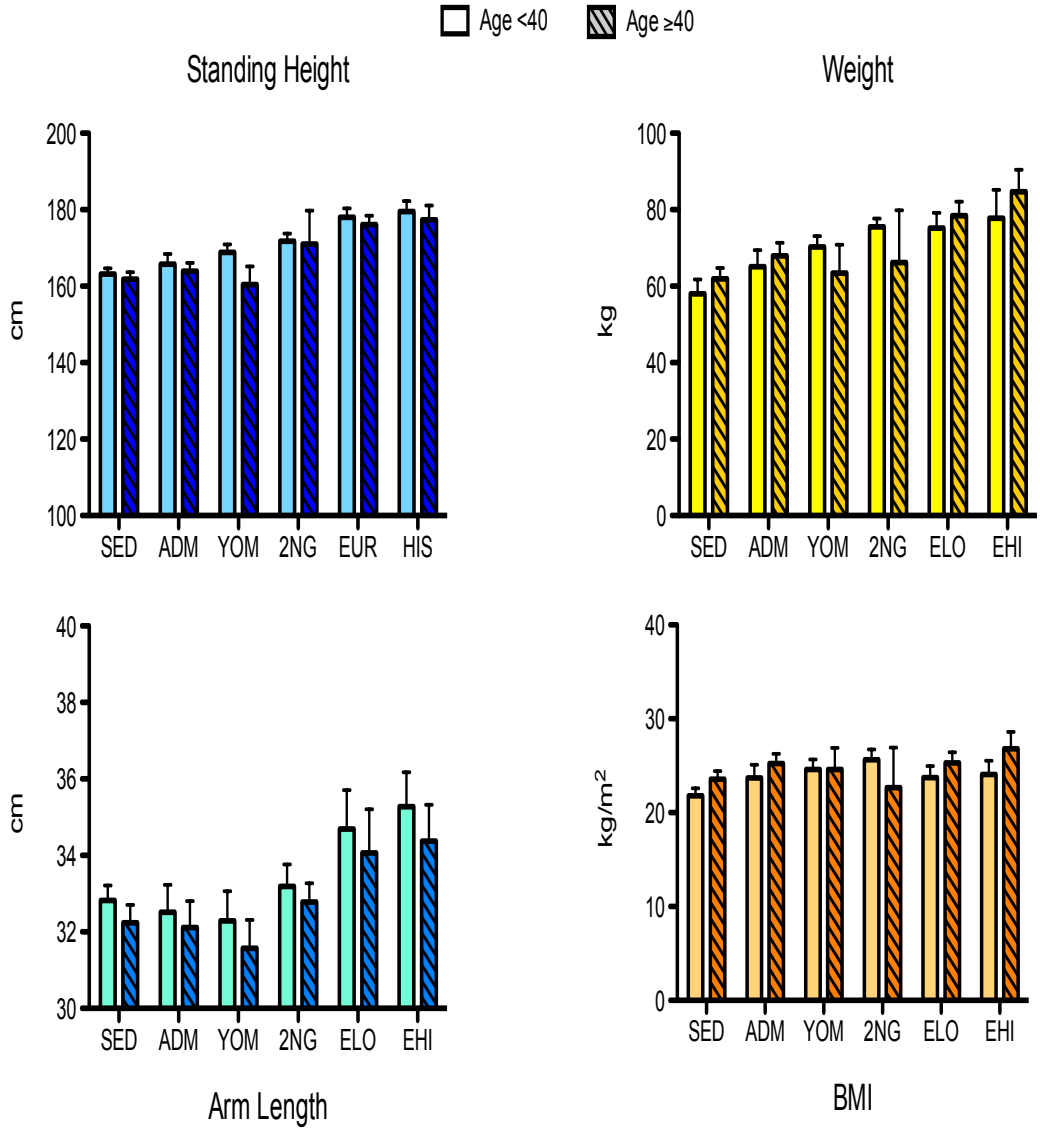


Figure 13: Mid-upper arm anthropometric measures

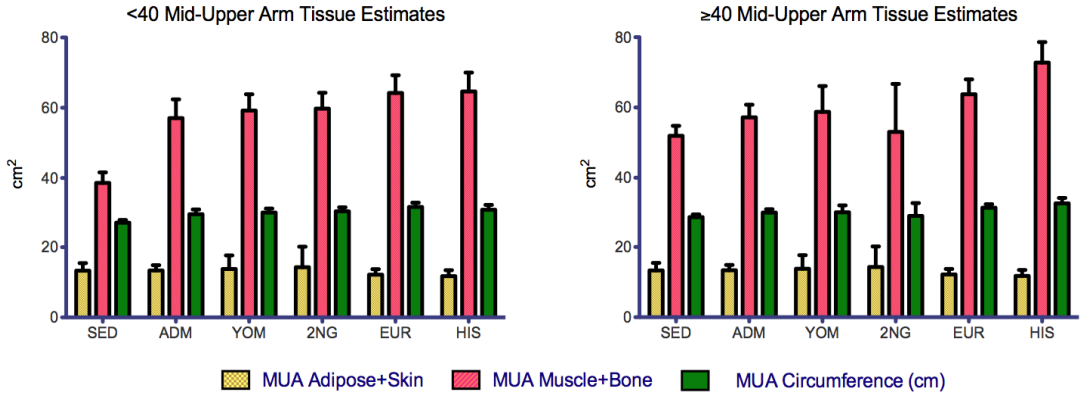


Figure 12 and 13: Resident group anthropometric variables by age, with analysis split at age 40 years. Error: ± 95% CI.

The covariate, participant age, was significantly related to resident group, $F(1,257) = 4.08$, $p < .005$, *partial* $\eta^2 = .07$, so an interaction effect was included in the model. After correcting for variance due to age and BMI, residence was a significant predictor of salT at all three sample times WAKE: $F(6,257) = 10.80$, $p < .005$, *partial* $\eta^2 = .17$; W+30: $F(6,263) = 11.67$, $p < .005$, *partial* $\eta^2 = .18$; BED: $F(6,269) = 6.22$, $p < .005$, *partial* $\eta^2 = .10$.

Planned contrasts (MLR-II) revealed that levels of salT in youth migrants (127.7 ±8.1 pg/mL) and British-born Bengalis (136.6 ±8.9 pg/mL), but not adult migrants (78.2 ±6.0 pg/mL) were significantly higher than sedentees (90.7 ±4.3 pg/mL). Among the European males, salT of low SES men (95.4 ±6.5 pg/mL) was not significantly different from either sedentees or adult migrants, while high SES men (127.5 ±7.7 pg/mL) had significantly greater salT concentrations than low status men, sedentees, or adult migrants, but were not different from youth migrants or British-born Bengalis (table 16, 17, 18 and 19).

Residence group differences in salT were greater in younger men than in older men. After splitting the older from younger group, the interaction effect between residence group and age was no longer significant.

Salivary T levels were not different between adult migrants and sedentees <40. In contrast, sedentee morning and evening salT is greater than that of adult migrants ≤40 (see fig. 14). Youth migrants and British-born Bengalis under 40 show no significant difference in salT, and have greater salT at all time points than sedentees, and greater morning, but not evening salT compared to adult migrants. Under 40 year olds in all Bengali groups resident in London have greater salT compared to low SES Europeans, but youth migrants and British-born Bengalis show no difference from high SES Europeans. High SES European salT is greater than sedentees, low SES Europeans, and morning samples from adult migrants.

Differences in salT were largest between morning samples from the younger men in all groups (tables 16 and 17). Youth migrants, British-born Bengalis and high status Europeans had significantly greater salT than sedentees at all three sample times ($p < .001$). High SES European salT was higher than low SES Europeans ($p < .001$). Post hoc Sidak comparisons within age <40 indicated significant differences between adult migrants and youth migrants, British-born Bengalis and high SES Europeans for both AM samples ($p < .005$), and between low SES Europeans and both youth migrants and British-born Bengalis for all three daily samples (AM samples $p < .005$; BED: $p < .05$) (see tables16,17 and 18).

Age at migration and salivary T

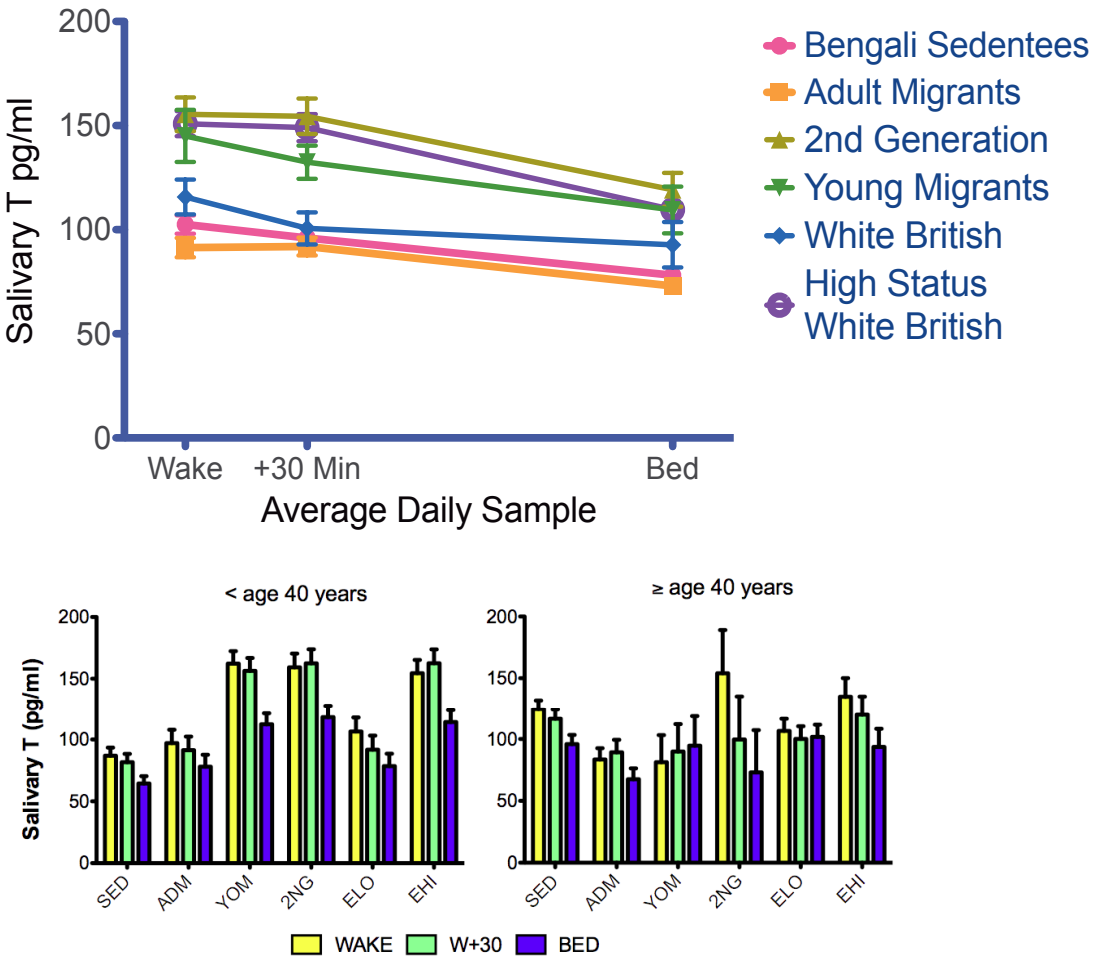
In MLR-I, age at migration significantly predicted salT, $F(4,74)=3.2$ $p=.017$, partial $\eta 2 = .14$. Planned contrasts showed the difference between childhood migrants (aged 3-12) and adult migrants was significant ($p=.049$), while the contrasts between all other stages of migration and older adult migrants were not significant. Inclusion of British-born Bengalis (MLR-III) or combining them with infant migrants into one group (MLR-IV) failed to improve the model, MLR-III: $F(5,99)=2.37$ $p=.045$, MLR-IV: $F(4,100)=2.99$. None of the planned contrasts in MLR-II or III were significant.

Thus, child migrants who arrived aged 3-12 years have significantly greater levels of salT than adult migrants who arrived at ages 18-30, but infants (aged 2 or under) and adolescent migrants (aged 13-17) were not measurably different from the adult migrant group. However, the small sample size of infant migrants (n=6) limits the likelihood of detecting a difference in this group.

Diurnal salT ratio and residence group

Residence group showed no significant relationship with diurnal ratio of AM/PM salT, within either age group <40: $F(5,153)=0.35$ $p=.88$ or age group ≥40: $F(5,106)=.45$ $p=.82$. MLR of residential differences in AM variance showed the variance in sedentees to be greater than adult migrants <40 years ($p=.035$) and ≥40 to be greater than adult migrants ($p<.001$), and both low and high SES European groups ($p=.011$ and .031, respectively).

Figure 14: Mean diurnal salT by residence



Estimated marginal means corrected for age and BMI. Error ± SEM.

Table 12: Body anthropometrics (height & weight): MLR contrast coefficients

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	**Height (cm)**	1	(Constant)	163.4	.79		.000	161.9	.94		.000
			SED vs. ADM	2.3	1.53	.09	.137	1.9	1.45	.09	.198
			SED vs. YOM	5.3	1.50	.22	**.001**	-0.7	2.94	-.02	.817
			SED vs. 2NG	8.7	1.53	.35	**.000**	9.9	4.50	.14	**.030**
			SED vs. ELO	14.4	1.66	.53	**<.001**	14.7	1.56	.67	**<.001**
			SED vs. EHI	16.2	1.59	.63	**<.001**	15.8	2.10	.51	**0.001**
				R^2 = .51 (p=<.001)				R^2 = .55 (p=<.001)			
MLRII	**Height (cm)**	1	(Constant)	171.2	.45		.000	168.7	.94		.000
			SED ADM vs. YOM 2NG ELO EHI	1.7	.15	.60	**<.001**	1.5	.26	.49	**<.001**
			YOM 2NG vs. ELO EHI	2.1	.29	.39	**<.001**	2.6	.68	.34	**<.000**
			YOM vs. 2NG	1.4	.71	.10	**.047**	5.4	2.48	.16	**.031**
			ELO vs. EHI	0.8	.90	.04	.391	0.8	1.11	.05	.462
			SED vs. ADM	1.3	.73	.10	.073	0.8	.71	.07	.238
				R^2= .50 (p=<.001)				R^2 = .54 (p<.001)			

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	**Weight (kg)**	1	(Constant)	57.7	5.3		.000	81.9	6.0		.000
			Participant Age in Years	0.4	0.2	.16	.052	-0.2	0.1	-.19	.036
		2	(Constant)	45.2	4.9		.000	73.0	5.1		.000
			Participant Age in Years	0.5	0.2	.20	.006	-0.2	0.1	-.17	.027
			SED vs. ADM	7.4	2.8	.19	**.009**	6.7	2.3	.24	**.005**
			SED vs. YOM	12.8	2.6	.34	**<.001**	2.5	4.6	.04	.586
			SED vs. 2NG	18.2	2.8	.47	**<.001**	3.9	7.1	.04	.584
			SED vs. ELO	18.2	2.9	.43	**<.001**	17.1	2.4	.55	**<.001**
			SED vs. EHI	19.6	2.8	.49	**<.001**	22.6	3.3	.52	**<.001**
				Step 1 R^2 = .02 Step 2 ΔR^2 =.38 (p=<.001)				Step 1 R^2= .04 Step 2 ΔR^2 =.40 (p=<.001)			
MLRII	**Weight (kg)**	1	(Constant)	61.7	4.5		.000	79.8	5.7		.000
			Participant Age in Years	0.2	0.2	.11	.137	-0.2	0.1	-.17	.061
		2	(Constant)	57.8	4.3		0000	79.9	4.7		.000
			Participant Age in Years	0.5	0.1	.20	.003	-0.2	0.1	-.15	.051
			SED ADM vs. YOM 2NG ELO EHI	2.2	0.3	.50	**<.001**	1.4	0.4	.32	**.001**
			YOM 2NG vs. ELO EHI	0.9	0.5	.10	.093	4.2	1.1	.38	**<.000**
			YOM vs. 2NG	2.6	1.3	.12	**.047**	1.4	3.9	.03	.726
			ELO vs. EHI	1.3	1.6	.05	.429	3.1	1.7	.13	.076
			SED vs. ADM	3.5	1.4	.17	**.011**	3.0	1.1	.19	**.008**
				Step 1 R^2 = .01 Step 2 ΔR^2 =.34 (p=<.001)				Step 1 R^2 = .03 Step 2 ΔR^2 =.40 (p=<.001)			

Table 13: Body anthropometrics (BMI): MLR contrast coefficient

Model	Outcome	Step	Predictors	Age ≤39 years				Age ≥40 years			
				B	S.E. *B*	β	*p*	*B*	S.E. *B*	β	*p*
MLR I	**BMI (kg/ cm2)**	1	(Constant)	20.1	1.4		.000	26.7	1.5		.000
			Participant Age in Years	0.1	0.0	.21	.010	-0.0	0.0	-.12	.199
		2	(Constant)	17.3	1.5		.000	25.9	1.6		.000
			Participant Age in Years	0.2	0.0	.26	.002	-0.0	0.0	-.14	.135
			SED vs. ADM	2.1	0.8	.21	**.011**	1.8	0.7	.25	**.017**
			SED vs. YOM	3.1	0.8	.31	**<.001**	1.3	1.5	.08	.369
			SED vs. 2NG	4.0	0.8	.39	**<.001**	-1.0	2.2	-.04	.671
			SED vs. ELO	2.4	0.9	.21	**.007**	1.8	0.8	.23	**.024**
			SED vs. EHI	2.3	0.8	.22	**.007**	3.2	1.0	.29	**.003**
				Step 1 R^2 = .04 Step 2 ΔR^2=.18 (p =<.001)				Step 1 R^2 = .01 Step 2 ΔR^2=.11 (p=.017)			
MLRII	**BMI (kg/ cm2)**	1	(Constant)	20.6	1.2		.000	26.4	1.5		.000
			Participant Age in Years	0.1	0.0	.18	.011	-0.0	0.0	-.11	.231
		2	(Constant)	19.5	1.3		.000	26.7	1.5		.000
			Participant Age in Years	0.2	0.0	.26	.001	-0.0	0.0	-.13	.178
			SED ADM vs. YOM 2NG ELO EHI	0.3	0.1	.25	**.001**	0.1	0.1	.07	.562
			YOM 2NG vs. ELO EHI	-0.3	0.2	-.13	.058	0.6	0.3	.22	.074
			YOM vs. 2NG	0.5	0.4	.09	.192	-1.0	1.2	-.08	.424
			ELO vs. EHI	0.2	0.5	.02	.719	0.7	0.5	.13	.169
			SED vs. ADM	1.0	0.4	.17	**.020**	0.8	0.4	.21	**.019**
				Step 1 R^2 = .03 Step 2 ΔR^2 =.16 (p =<.001)				Step 1 R^2= .01 Step 2 ΔR^2 =.11 (p =.015)			

Table 14: Arm anthropometrics (LogAL & MUAC) : MLR contrast coefficients

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	LogAL (cm)	1	(Constant)	1.52	.003		.000	1.51	.004		.000
			SED vs. ADM	-0.00	.006	-.034	.663	-0.00	.007	-.018	.854
			SED vs. YOM	0.00	.006	.007	.925	-0.01	.013	-.054	.552
			SED vs. 2NG	0.01	.006	.090	**.250**	0.01	.020	.035	.699
			SED vs. ELO	0.03	.006	.364	**<.001**	0.02	.007	.324	**.001**
			SED vs. EHI	0.03	.006	.398	**<.001**	0.03	.009	.281	**.003**
				R^2 = .24 (p<.001)				R^2 = .16 (p<.001)			
MLRII	LogAL (cm)	1	(Constant)	1.52	.002		.000	1.52	.004		.000
			SED ADM vs. YOM 2NG ELO EHI	0.00	.001	.275	**<.001**	0.00	.001	.224	**.050**
			YOM 2NG vs. ELO EHI	0.01	.001	.390	**<.001**	0.01	.003	.254	**.032**
			YOM vs. 2NG	0.01	.003	.128	.057	0.01	.011	.076	.450
			ELO vs. EHI	0.00	.004	.070	.298	0.00	.005	.048	.595
			SED vs. ADM	-0.00	.003	-.048	.499	-0.00	.003	-.026	.759
				R^2 = .21 (p<.001)				R^2 = Age ≥40 years (p<.001)			

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	MUAC (cm)	1	(Constant)	25.17	1.505		.000	31.95	1.422		.000
			Participant Age in Years	0.14	.052	.221	.007	-0.04	.026	-.145	.128
		2	(Constant)	23.83	1.497		.000	30.42	1.410		.000
			Participant Age in Years	0.11	.051	.178	.028	-0.04	.026	-.129	.170
			SED vs. ADM	2.68	.822	.257	**.001**	1.35	.666	.201	**.045**
			SED vs. YOM	2.79	.806	.256	**.001**	1.79	1.244	.128	.153
			SED vs. 2NG	2.70	.834	.253	**.002**	0.34	1.918	.015	.861
			SED vs. ELO	5.73	.844	.499	**<.001**	2.56	.684	.352	**<.001**
			SED vs. EHI	3.69	.799	.346	**<.001**	3.91	.888	.403	**<.001**
				Step 1 R^2 = 0.04 Step 2 ΔR^2 =.28 (p<.001)				Step 1 R^2 = .02 Step 2 ΔR^2 =.20 (p=.918)			
MLRII	MUAC (cm)	1	(Constant)	25.90	1.252		.000	32.00	1.343		.000
			Participant Age in Years	0.12	.044	.198	.007	-0.04	.025	-.149	.103
		2	(Constant)	25.87	1.308		.000	32.04	1.285		.000
			Participant Age in Years	0.14	.045	.232	.002	-0.04	.024	-.134	.141
			SED ADM vs. YOM 2NG ELO EHI	0.39	.085	.340	**<.001**	0.24	.113	.242	**.038**
			YOM 2NG vs. ELO EHI	0.25	.155	.109	.103	0.61	.288	.243	**.037**
			YOM vs. 2NG	0.17	.413	.027	.687	-0.52	1.036	-.049	.617
			ELO vs. EHI	-0.40	.468	-.056	.395	0.62	.472	.117	.191
			SED vs. ADM	1.20	.396	.222	**.003**	0.64	.315	.176	**.044**
				Step 1 R^2 = .03 Step 2 ΔR^2 =.22 (p=<.001)				Step 1 R^2 = .022 Step 2 ΔR^2 =.204 (p=.869)			

Table 15: Arm anthropometrics (MUA & LogMUA): MLR contrast coefficients

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	MUA	1	(Constant)	34.73	7.167		.000	63.60	5.844		.000
	Musc+Bone (cm2)		Participant Age in Years	0.64	.246	.212	.011	-0.11	.106	-.101	.298
		2	(Constant)	27.62	6.069		.000	55.66	5.360		.000
			Participant Age in Years	0.41	.208	.135	.054	-0.07	.098	-.063	.482
			SED vs. ADM	19.46	3.302	.401	**<.001**	5.31	2.485	.200	**.035**
			SED vs. YOM	20.69	3.303	.398	**<.001**	8.89	4.638	.162	**.058**
			SED vs. 2NG	18.78	3.348	.379	**<.001**	1.22	7.154	.014	.865
			SED vs. ELO	30.68	3.690	.524	**<.001**	11.10	2.796	.351	**<.001**
			SED vs. EHI	25.86	3.204	.522	**<.001**	20.89	3.312	.549	**<.001**
				Step 1 R^2 = .04 Step 2 ΔR^2 =.46 (p=.011)				Step 1 R^2 = .010 Step 2 ΔR^2 =.320 (p=.128)			
MLRII	MUA	1	(Constant)	40.37	5.875		.000	63.91	5.489		.000
	Musc+Bone (cm2)		Participant Age in Years	0.46	.205	.167	.026	-0.11	.100	-.106	.258
		2	(Constant)	43.22	5.158		.000	63.71	4.870		0.000
			Participant Age in Years	0.50	.178	.181	.006	-0.08	.092	-.072	.407
			SED ADM vs. YOM 2NG ELO EHI	2.35	.335	.442	**<.001**	1.26	.422	.316	**.004**
			YOM 2NG vs. ELO EHI	1.23	.622	.113	**.049**	3.09	1.079	.303	**.005**
			YOM vs. 2NG	0.28	1.637	.010	.866	-2.85	3.849	-.069	.461
			ELO vs. EHI	0.22	1.878	.007	.907	4.52	1.824	.204	**.015**
			SED vs. ADM	9.22	1.545	.374	**<.001**	2.62	1.173	.184	**.027**
				Step 1 R^2 = .02 Step 2 ΔR^2 =.45 (p=<.001)				Step 1 R^2 = .011 Step 2 ΔR^2 =.321 (p=<.001)			

Model	Outcome	Step	Predictors	Age ≤39 years B	S.E. B	β	p	Age ≥40 years B	S.E. B	β	p
MLR I	Log MUA	1	(Constant)	1.16	.084		.000	1.09	.088		.000
	Fat+Skin (cm2)		Participant Age in Years	-0.00	.003	-.009	.916	-0.00	.002	-.010	.918
		2	(Constant)	1.22	.086		.000	1.10	.097		.000
			Participant Age in Years	0.00	.003	.042	.627	-0.00	.002	-.039	.723
			SED vs. ADM	-0.18	.047	-.329	**<.001**	0.05	.045	.121	.286
			SED vs. YOM	-0.21	.047	-.350	**<.001**	0.03	.084	.037	.717
			SED vs. 2NG	-0.17	.048	-.303	**<.001**	0.07	.129	.054	.596
			SED vs. ELO	-0.15	.052	-.221	**.006**	-0.01	.051	-.016	.884
			SED vs. EHI	-0.24	.046	-.417	**<.001**	-0.01	.060	-.024	.821
				Step 1 R^2 = <.001 Step 2 ΔR^2 =.25 (p=.916)				Step 1 R^2 = <.001 Step 2 ΔR^2 =.019 (p=.298)			
MLRII	Log MUA	1	(Constant)	1.12	.072		.000	1.11	.082		.000
	Fat+Skin (cm2)		Participant Age in Years	0.00	.002	.016	.832	-0.00	.002	-.030	.748
		2	(Constant)	1.07	.074		.000	1.13	.088		.000
			Participant Age in Years	0.00	.003	.030	.697	-0.00	.002	-.052	.621
			SED ADM vs. YOM 2NG ELO EHI	-0.02	.005	-.299	**<.001**	0.00	.008	.002	.990
			YOM 2NG vs. ELO EHI	-0.00	.009	-.033	.631	-0.01	.020	-.096	.455
			YOM vs. 2NG	0.02	.024	.048	.489	0.01	.070	.024	.832
			ELO vs. EHI	-0.03	.027	-.065	.335	-0.01	.033	-.023	.817
			SED vs. ADM	-0.09	.022	-.308	**<.001**	0.02	.021	.103	.304
				Step 1 R^2 = <.001 Step 2 ΔR^2 =.25 (p=<001)				Step 1 R^2 = <.001 Step 2 ΔR^2 =.016 (p=<001)			

Table 16: Untransformed (waking) salivary T MLR contrast coefficients

				Age ≤39 years				Age ≥40 years			
Model	**Outcome**	**Step**	**Predictors**	***B***	**S.E. *B***	**β**	***p***	***B***	**S.E. *B***	**β**	***p***
MLR I	WAKE salT	1	(Constant)	71.51	33.45		.034	67.43	47.25		.156
			Participant Age in Years	-1.82	.75	-.191	.016	-0.70	.45	-.141	.123
			Body Mass Index	4.16	1.26	.258	.001	3.20	1.53	.191	.038
		2	(Constant)	78.58	30.05		.010	31.54	46.33		.498
			Participant Age in Years	-0.48	.73	-.050	.512	-0.13	.46	-.026	.783
			Body Mass Index	0.93	1.22	.058	.446	4.08	1.51	.242	.008
			SED vs. ADM	10.53	12.85	.065	.414	-42.06	12.11	-.355	**.001**
			SED vs. YOM	68.37	12.05	.448	**<.001**	-53.80	19.07	-.252	**.006**
			SED vs. 2NG	65.99	13.15	.412	**<.001**	30.63	36.01	.074	.397
			SED vs. ELO	22.19	13.04	.128	.091	-15.30	12.45	-.119	.221
			SED vs. EHI	67.69	13.36	.382	**<.001**	9.90	17.27	.054	.568
				Step 1 R^2 = 0.08 Step 2 ΔR^2 =0.26 (p<.001)				Step 1 R^2 = 0.03 Step 2 ΔR^2 =0.18 (p<.001)			
MLRII	WAKE salT	1	(Constant)	69.35	34.36		.045	80.07	47.03		.091
			Participant Age in Years	-1.82	.79	-.182	.022	-0.86	.46	-.171	.062
			Body Mass Index	4.28	1.30	.260	.001	3.07	1.50	.185	.043
		2	(Constant)	114.56	32.23		.001	31.54	47.71		.510
			Participant Age in Years	-0.21	.77	-.021	.785	-0.25	.47	-.051	.592
			Body Mass Index	0.81	1.22	.049	.507	3.90	1.50	.236	**.011**
			SED ADM vs. YOM 2NG ELO EHI	8.94	1.43	.472	<**.001**	2.55	2.18	.140	.245
			YOM 2NG vs. ELO EHI	-7.56	2.80	-.188	**.008**	0.78	5.65	.016	.890
			YOM vs. 2NG	-1.49	7.24	-.014	.837	36.39	20.42	.168	**.078***
			ELO vs. EHI	23.97	7.79	.202	**.003**	14.22	8.97	.145	.116
			SED vs. ADM	5.10	6.38	.060	.426	-20.40	6.02	-.310	**.001**
				Step 1 R^2 = 0.08 Step 2 ΔR^2 =0.28 (p <.001)				Step 1 R^2 = 0.05 Step 2 ΔR^2 =0.14 (p<.005)			

*For WAKE≥40 YOM vs. 2NG, transformed values were significant (p=.027).

Note: for ease of interpretation, these are the non-transformed MLR results. Log transformation did not decrease significance or effect size.

Table 17: Untransformed (wake +30) salivary T MLR contrast coefficients

				Age ≤39 years				Age ≥40 years			
Model	**Outcome**	**Step**	**Predictors**	***B***	**S.E. *B***	**β**	***p***	***B***	**S.E. *B***	**β**	***p***
MLR I	W+30 salT	1	(Constant)	61.96	35.46		.083	141.97	47.14		.003
			Participant Age in Years	-1.40	.79	-.141	.078	-0.66	.45	-.141	.145
			Body Mass Index	3.90	1.34	.231	.004	-0.09	1.52	-.006	.953
		2	(Constant)	70.14	30.40		.022	122.31	48.93		.014
			Participant Age in Years	0.03	.74	.003	.964	-0.33	.48	-.070	.502
			Body Mass Index	0.42	1.23	.025	.736	0.51	1.60	.032	.752
			SED vs. ADM	10.09	13.00	.059	.439	-28.30	12.79	-.253	**.029**
			SED vs. YOM	75.00	12.19	.469	**<.001**	-29.71	20.14	-.147	.143
			SED vs. 2NG	70.94	13.30	.423	**<.001**	-13.82	38.03	-.035	.717
			SED vs. ELO	12.15	13.19	.067	.358	-17.30	13.14	-.142	.191
			SED vs. EHI	84.20	13.52	.454	**<.001**	3.99	18.24	.023	.827
				Step 1 R^2 = 0.06 Step 2 ΔR^2 =0.3 (p<.001)				Step 1 R^2= 0.01 Step 2 ΔR^2 =0.06 (p=.173)			
MLRII	W+30 salT	1	(Constant)	65.01	36.39		.076	158.76	46.83		.001
			Participant Age in Years	-1.64	.84	-.154	.053	-0.88	.46	-.185	.056
			Body Mass Index	4.11	1.37	.237	.003	-0.24	1.48	-.015	.872
		2	(Constant)	107.34	33.29		.002	127.89	49.68		.011
			Participant Age in Years	0.08	.80	.008	.920	-0.59	.49	-.124	.233
			Body Mass Index	0.62	1.24	.036	.615	0.26	1.55	.017	.866
			SED ADM vs. YOM 2NG ELO EHI	9.46	1.45	.472	**<.001**	-0.09	2.24	-.005	.969
			YOM 2NG vs. ELO EHI	-8.07	2.85	-.191	**.005**	3.79	5.74	.085	.511
			YOM vs. 2NG	3.06	7.49	.027	.683	4.88	20.68	.025	.814
			ELO vs. EHI	35.42	7.97	.283	**<.001**	9.84	9.16	.108	.285
			SED vs. ADM	4.89	6.56	.054	.458	-13.61	6.45	-.213	**.037**
				Step 1 R^2 = 0.06 Step 2 ΔR^2 =0.31 (p<.001)				Step 1 R^2 = 0.03 Step 2 ΔR^2 =0.05 (p=.25)			

Note: for ease of interpretation, these are the non-transformed MLR results. Log transformation did not decrease significance or effect size.

Table 18: Untransformed (bed) salivary T MLR contrast coefficients

Model	Outcome	Step	Predictors	Age ≤39 years *B*	S.E. *B*	β	*p*	Age ≥40 years *B*	S.E. *B*	β	*p*
MLR I	BED salT	1	(Constant)	53.63	28.59		.063	67.03	45.08		.140
			Participant Age in Years	-1.51	.64	-.187	.019	-0.72	.43	-.155	.097
			Body Mass Index	3.24	1.08	.236	.003	2.40	1.46	.152	.103
		2	(Constant)	61.77	26.59		.022	49.00	45.90		.288
			Participant Age in Years	-0.50	.64	-.061	.442	-0.41	.45	-.089	.367
			Body Mass Index	0.63	1.08	.046	.561	2.83	1.50	.180	.062
			SED vs. ADM	17.72	11.37	.128	.121	-30.97	11.99	-.279	**.011**
			SED vs. YOM	47.82	10.67	.368	**<.001**	-27.83	18.89	-.139	.144
			SED vs. 2NG	58.70	11.64	.431	**<.001**	-21.59	35.68	-.055	.546
			SED vs. ELO	10.80	11.53	.073	.351	6.55	12.33	.054	.596
			SED vs. EHI	50.95	11.82	.338	**<.001**	-2.62	17.11	-.015	.879
				Step 1 R^2 = 0.05 Step 2 ΔR^2 =0.21 (p<.001)				Step 1 R^2 = 0.04 Step 2 ΔR^2=0.07 (p=.10)			
MLRII	BED salT	1	(Constant)	50.51	28.79		.081	79.10	44.83		.080
			Participant Age in Years	-1.39	.66	-.165	.037	-0.85	.43	-.181	.053
			Body Mass Index	3.27	1.09	.236	.003	2.24	1.42	.145	.118
		2	(Constant)	83.49	28.95		.004	53.60	46.78		.254
			Participant Age in Years	-0.35	.69	-.042	.613	-0.53	.46	-.113	.255
			Body Mass Index	0.88	1.08	.063	.419	2.54	1.47	.165	.088
			SED ADM vs. YOM 2NG ELO EHI	5.76	1.27	.361	**<.001**	1.50	2.18	.087	.492
			YOM 2NG vs. ELO EHI	-4.73	2.48	-.141	**.058**	3.46	5.70	.076	.545
			YOM vs. 2NG	2.91	6.38	.032	.649	-10.79	20.61	-.050	.602
			ELO vs. EHI	17.82	6.98	.178	**.012**	-4.07	8.83	-.044	.646
			SED vs. ADM	6.79	5.74	.094	.238	-14.19	5.85	-.232	.017
				Step 1 R^2 = 0.04 Step 2 ΔR^2 =0.18 (p<.001)				Step 1 R^2 = 0.05 Step 2 ΔR^2 =0.05 (p=.24)			

Note: for ease of interpretation, these are the non-transformed MLR results. Log transformation did not decrease significance or effect size.

Table 19: Untransformed (day mean) salivary T MLR contrast coefficients

Model	Outcome	Step	Predictors	Age ≤39 years				Age ≥40 years			
				B	**S.E. *B***	**β**	***p***	***B***	**S.E. *B***	**β**	***p***
MLR I	DAYM salT	1	(Constant)	62.85	28.04		.026	85.24	40.22		.036
			Participant Age in Years	-1.61	.62	-.201	.011	-0.70	.38	-.168	.068
			Body Mass Index	3.58	1.06	.264	.001	2.02	1.30	.142	.123
		2	(Constant)	71.11	24.22		.004	62.60	40.49		.125
			Participant Age in Years	-0.42	.59	-.053	.475	-0.34	.40	-.081	.401
			Body Mass Index	0.60	.98	.044	.544	2.65	1.32	.186	**.047**
			SED vs. ADM	14.69	10.35	.107	.158	-33.24	10.58	-.331	**.002**
			SED vs. YOM	60.11	9.71	.468	**<.001**	-37.97	16.67	-.210	**.025**
			SED vs. 2NG	63.21	10.60	.469	**<.001**	-7.45	31.47	-.021	.813
			SED vs. ELO	14.00	10.50	.096	.185	-6.10	10.88	-.056	.576
			SED vs. EHI	63.27	10.77	.425	**<.001**	0.75	15.09	.005	.961
				Step 1 R^2 = 0.07 Step 2 ΔR2 =0.29 (p<.001)				Step 1R^2 = 0.04 Step 2 ΔR2 =0.12 (p=.007)			
MLRII	DAYM salT	1	(Constant)	61.24	28.57		.034	97.47	39.88		.016
			Participant Age in Years	-1.62	.66	-.192	.014	-0.86	.38	-.203	.027
			Body Mass Index	3.71	1.08	.266	.001	1.91	1.27	.136	.135
		2	(Constant)	98.55	26.46		.000	62.65	41.39		.133
			Participant Age in Years	-0.30	.63	-.036	.632	-0.50	.41	-.118	.221
			Body Mass Index	0.85	.99	.061	.395	2.50	1.31	.177	.059
			SED ADM vs. YOM 2NG ELO EHI	7.31	1.16	.457	**<.001**	0.84	1.89	.054	.660
			YOM 2NG vs. ELO EHI	-6.08	2.27	-.181	**.008**	3.53	4.93	.087	.476
			YOM vs. 2NG	0.99	5.83	.011	.866	8.09	17.82	.044	.651
			ELO vs. EHI	24.13	6.38	.240	**<.001**	4.08	7.82	.049	.603
			SED vs. ADM	6.26	5.24	.087	.235	-15.67	5.18	-.282	**.003**
				Step 1 R^2 = 0.07 Step 2 ΔR^2 =0.26 (p<.001)				Step 1 R^2 = 0.06 Step 2 ΔR^2 =0.09 (p=.039)			

Note: for ease of interpretation, these are the non-transformed MLR results. Log transformation did not decrease significance or effect size.

3.4 Age of maturity

Age of puberty onset was estimated by asking participants to recall and estimate their age at four developmental milestones: 1) when their voice first broke; 2) the first appearance of pubic hair; 3) the first appearance of facial hair or start of shaving; 4) and first nocturnal emission (See appendix 1 section VIII, questions 2-5). Combined recall questions of this type have been shown as reliable estimates of relative maturational rates with low test-retest variation (Gilger et al. 1991; Kaiser and Gruzelier 1999). Recalled age of each milestone was analysed by ANCOVA predicted by residence group membership, after correcting for age at recruitment in order to reduce potential recall bias.

A composite age of maturity for each participant was calculated by averaging all valid responses. Recalled age of voice breaking and of composite age of maturity were log-transformed prior to regression to correct for heterogeneity of variance (Levene's test: 4.46(4,122) p=.002 and 3.19(4,232), p=.01, respectively).

Missing data was high for all age of maturity variables, as might be expected with retrospective data, and with questions of a personal and sensitive nature (and in the case of nocturnal emissions, a non-universal milestone of puberty). Within residence groups, participants responding "do not remember" ranged from 21-34%, while "rather not say" ranged from 0.5-12%. Patterns of missing data were analysed by expectation-maximization methods to estimate means, correlations, and covariances and found to be random in respect to residence group and whether respondents were older or younger than 40 years old (Little's MCAR test: X^2=34.05, df=26, p=.13).

Men who spent the entirety of their childhood in Bangladesh reported the latest average age of maturity for all measures; adult migrants had the latest composite age of maturity, at 16.6±0.2 years and reported the latest average age of maturity for all measures except for appearance of facial hair/shaving, for which sedentees, with a composite age of 16.2±0.2 years were later (see table 20 for detailed descriptives). Men who spent the entirety of their childhood in the UK reported the earliest average age of maturity; British-born Bengalis and Europeans both had a composite age of maturity of 14.2±0.2 years, with British-born Bengalis reporting the earliest age of maturity for all measures except for appearance of pubic hair, for which Europeans were earlier. Youth migrants fell between the two extremes, with a composite age of maturity of 15.5±0.5 years.

Composite age of maturity was significantly predicted by residence group, F(1,235)=15.32, p<.001, after correcting for participant age at recruitment. Planned contrasts revealed that British-born Bengalis and Europeans matured earlier than sedentees (t(235)=-3.93 and -6.42, both p<.001), while age of maturity was not

significantly different between youth or adult migrants and sedentees (t(235)=-1.45 and 0.21, both p>.1). Residence group was a significant predictor of each individual measure of age of maturity, after correcting for age at recruitment.

Among youth migrants, age at migration significantly predicted composite age of maturity, and planned contrasts of developmental stages showed that migrants arriving in the UK before age 2 matured significantly earlier than those arriving after age 2 (Estimated marginal means correcting for age at recruitment: 13.5±1.0 years, n=6 versus 16.3±0.5, n=15, F(1,18)=5.48, p=.03).

Table 20: Estimated marginal means, composite age at maturity (years)

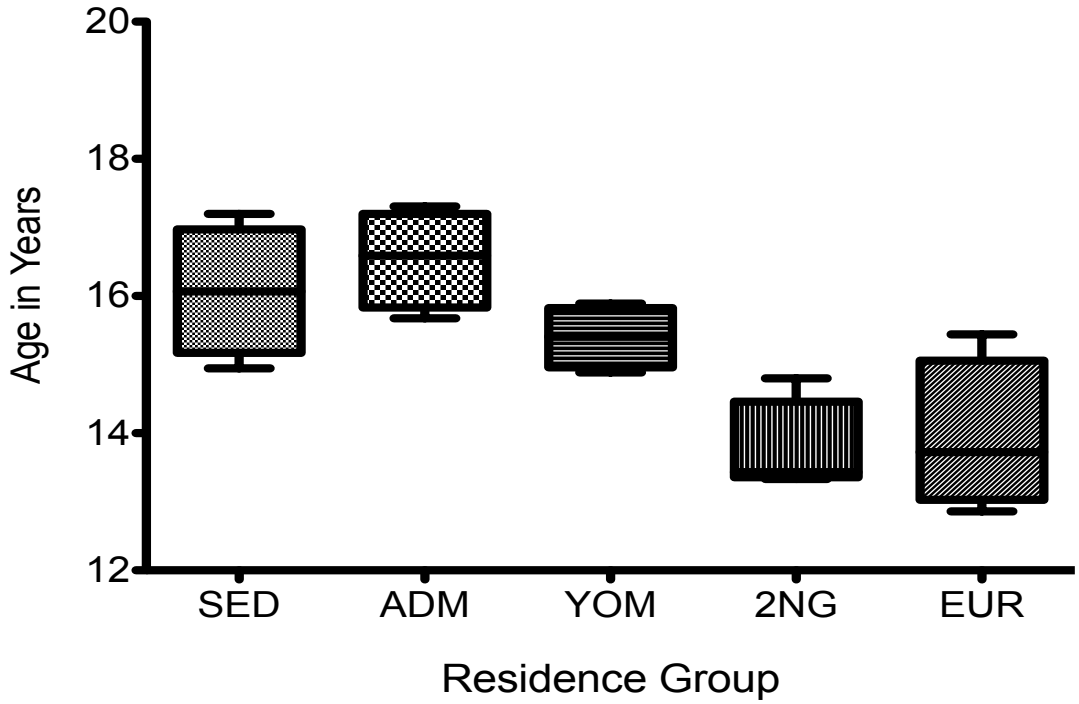
	Bengali Sedentees	Adult Migrants	Youth migrants	Second Generation Migrants	European
N	107	74	60	56	62
	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)
Voice Broke Age Recall	15 (2)	16 (3)	15 (3)	13 (1)	14 (1)
Shave/Beard Age Recall	17 (3)	17 (2)	16 (2)	15 (2)	15 (2)
Pubic Hair Age Recall	16 (2)	16 (3)	15 (2)	13 (1)	13 (2)
Nocturnal Emiss Age Recall	16 (2)	17 (2)	16 (4)	13 (1)	14 (1)
Composite age of puberty recall	16.3 (2)	16.3 (1.8)	15.7 (2.5)	14.5 (1.4)	14.2 (1.4)

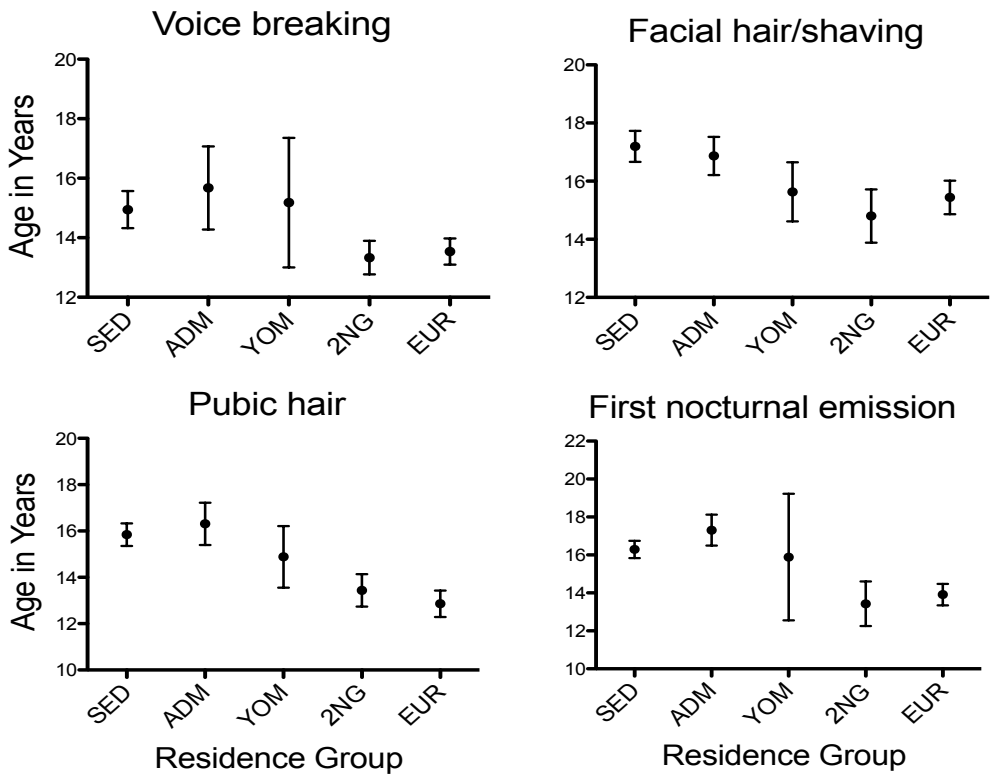
a. Covariates appearing in the model are evaluated at the following values: Age Participant Recruited = 39.4631.

After correcting for participant age at recruitment, an earlier composite age of maturity significantly correlated to increased morning and evening salT. Age of shaving and pubic hair both correlated with morning salT, and nocturnal emission correlated with all three daily samples, after correcting for age at recruitment. After also correcting for variation due to residence group, these correlations were no longer significant, except in the case of nocturnal emission, which remained significantly correlated to all daily samples. Within residence groups, composite age of maturity of British-born Bengalis aged <40 years significantly correlated to WAKE+30 and BED salT, after correcting for age at recruitment (r=-.68, -.61; df=12, 13; both p<.02). A negative correlation between age of maturity and WAKE salT of sedentees and WAKE+30 of adult migrants approached significance (r=-.17, -.28 respectively, both p=.08), but when split by age 40, the relationship was no longer significant. For all other residence groups, composite age of maturity was not significantly correlated to either morning or evening salT, after correcting for age at recruitment.

Partial correlations between composite age of maturity and anthropometric variables of standing height, weight, and MUA muscle+bone (but not axial fat) were all significant after correcting for age at recruitment (all r=-.28 or -.29, df=220-223, p<.01) but were no longer significant after also correcting for residence group.

Figure 15: Composite age of maturity





Mean age of recalled maturity by residence group. Error: ± 95% CI.

3.5 Conclusions

The above data support hypothesis 1: The experience of more constrained or stressful conditions during development constrains investment in reproductive effort, while priming males to respond to improvement in conditions during young adulthood with an immediate increase in reproductive functioning.

Predictions: If men migrate to the UK before the age of maturity, they will show greater investment into

a. reproductive effort than sedentees and adult migrants, as measured by higher concentrations of salT and an earlier age at maturity

b. growth, as measured by skeletal stature, muscle and body fat.

Findings: All migrant and British-born Bengalis show greater salT, greater BMI and skeletal muscle compared to sedentees. The stature of young migrants is taller, and age of maturity earlier than sedentees and adult migrants. Compared to non-nutritionally stressed sedentee counterparts, migration to the UK prior to sexual maturity promotes greater investment into reproductive effort as measured by somatic and hormonal characteristics of migrant men in early adulthood.

There are no measured salT differences between younger (< age 40) migrants and sedentees if migration occurs after the age of maturity. However, older (≥ age 40) sedentees appear to invest more into reproductive function as measured by salT than adult migrant counterparts.

The influences of these environmental stresses upon salT and age at maturity are both subject to critical periods of development: younger men under age 40 set their adult reproductive *function* according to their childhood environment, particularly during the juvenile period, which includes the “slow growth” between ages 9-12 years. But adult reproductive *tempo* appears to be set at an earlier age, as migration after age 2 years did not predict an earlier age of maturity than men who migrate after maturity, while men who were born in the UK, regardless of their ethnicity, mature at an earlier age than those who spent all or the latter part of their childhood in Bangladesh.

The juvenile period is generally considered a quiescent stage of male reproductive development, yet men who migrated between ages 2 and 12 have higher salT than sedentees, while men migrating earlier or later than this do not. However, the limited sample size of infant migrants in particular (≤ age 2 years, n=6) does not allow a rejection of the hypothesis that migration during infancy relates to adult salT.

The juvenile period of apparent dormancy of the testis and low levels of circulating sex hormones falls between the postnatal peak of activity in the first year of life and

the surge of activity accompanying puberty (see chapter 1). Recently, however an intriguing line of research suggests this is a sensitive period for male responses to environmental cues with implications for adult reproduction (Pembrey 2010; Pembrey et al. 2005), though admittedly, these are trans-generational effects.

The influence of present or early adult experience appears more important to reproductive functioning in older men, with higher socioeconomic status relative to current surroundings associated with greater testosterone.

The height of youth and second-generation migrants supports the hypothesis that conditions in the UK are more conducive to childhood growth than Bangladesh. Migrating prior to age of maturity leads to taller adult standing height, but this plasticity is not great enough to allow the young migrants to grow as tall as Bengalis born in the UK. While actual measurement of leg length is lacking from these data, the lack of differences in arm length indicates this is a more canalised trait, suggesting femoral length or other long bones of the legs are more responsive to improvements in childhood environment.

Hypothesis 2 was only partly supported: Young men who experienced an environment of fewer constraints will have greater inter- and intra-individual variability in salT.

Predictions: British-born Bengalis will show greater diurnal variation in salT compared to young migrants, and both young migrants and British Bengalis will show greater diurnal variation compared to adult migrants and sedentees. European males will show similar diurnal variation to young migrants and British-born Bengalis, and greater measures of salT than sedentees and adult migrants.

Findings: The difference between groups was greatest for the morning salT of young men, which fits with the expectation that these conditions produce the greatest variance in samples. But there was not a consistent pattern of the overall degree of daily decline. The lack of a consistent pattern between diurnal change in salT and residence group fits with findings which suggest the morning peak and the daily decline frequently observed in clinical studies e.g. (Diver et al. 2003), is less predictable or regular in non-Western populations (Bribiescas and Hill 2010; Vitzthum et al. 2009).

There has been speculation that morning testosterone peaks are more reflective of physiological “set points” while evening levels are more reflective of daily social and ecological interactions. However, the residence groups with the highest morning salT also had the highest evening levels, so are either subject to daily environmental influences which promote high salT, or the rate of decline is not more flexible in men with higher salT.

Hypothesis 3 was supported: If older males are of high social status, relative to surroundings, they will invest more in reproductive effort. If they are lower, they will invest less into reproductive effort.

Findings: Older males base their reproductive effort upon current more than upon early life conditions.

The lower salT of adult migrants compared to sedentees over the age of 40 hints at the importance of relative social positioning for men in the latter half of their reproductive career. The influence of social position on testosterone will be explored in further detail in chapter 5.

Overall conclusions:

Early life developmental conditions appear to influence both the developmental tempo and adult reproductive function of the human male. Men moving to an ecology presumed of less energetic stress than that of their early to mid childhood grow more skeletal muscle and higher salT than sedentee counterparts in the first half of the male reproductive stage of life, but this difference is reversed in the second half of adulthood. If they are still children when ecological conditions change, they also mature earlier, grow taller and maintain a higher daily salT profile than their sedentee counterparts. Taken together this suggests a flexibility in male reproductive strategy that switches at some point in the latter portion of a male's life, possibly from one dependent upon early life conditions, to one more reliant on current social status.

Chapter 4: Diet and health

In this chapter I test dietary and health behavioural hypotheses, focussing on developmental influences of a change in ecology upon these behaviours, and health outcomes as measured by prostate symptoms and fasting blood glucose. I then test whether dietary and health behaviours adequately explain Bengali inter-population variation in measures of salT observed in chapter three.

The specific dietary hypotheses are that current conditions of the Bangladeshi sedentee population means they are not nutritionally stressed, and that acculturation to life in the UK (as measured by age at migration or place of birth for Bengalis) leads to the adoption of more Western diet, in terms of food frequencies and eating patterns, as measured by number of meals and snacks per day. Previous work in this community, using the same measures of diet, leads to the prediction is that migrants will consume more meat and less fish than sedentees (Núñez-de la Mora et al. 2004). If dietary patterns shift with acculturation, there should be a significant relationship between the age at migration and the frequency of consumption of foods typical of the sedentee diet. This diet includes reduction in the amount of rice and fish consumed per week, but an increase in the amount of meat.

I then test whether period of residence in the UK, and presumed acculturation, influences health behaviours of smoking, betel nut use, alcohol consumption, and activity patterns. Finally, I moves to the testing of evolutionary medical hypotheses of health. If adult onset diseases relate to a mismatch between early life developmental conditions and adult ecology, does migration after key stages of development mean migrants are more prone to symptoms of prostatic disease and dysregulation of glucose metabolism? Proximately, if men have high salT, and T stimulates proliferative growth of the prostate over the male life-span, are they more likely to report more LUTS than men who have low salT? If this is the case one would predict youth migrants, British-born Bengalis, and European men should have higher incidence of LUTS than adult migrants or sedentees, after correcting for age at recruitment. Alternatively, men whose childhood development is “mismatched” to their adult ecology should show greater symptoms of disease than those who have remained in the same conditions all their lives. In this case, adult migrants and youth migrants should show a greater incidence of LUTS and dysregulation of glucose metabolism than British born Bengalis or European men. Finally, adult dietary or health behavioural differences will be applied as explanatory variables for the apparent differences in salT between migrant groups.

4.1 Diet

Food frequencies

Participants were asked to estimate their regular weekly consumption of 22-24 food items from a closed number of 5 optional frequencies ranging from “Rarely or never” to “Daily” (see Appendix 2 for full questionnaire). Staple foods like rice or vegetables were given additional options of “Once daily”, “Twice daily”, or “With every meal”. The number of items included on the list was adapted to resident/ethnic group (i.e. pork was omitted from Bengali questionnaires). For responses to individual foods, chi-square tests were run on categorical responses, and two-tailed t-tests were run on combined weekly averages and estimates of weekly consumption of foods.

Average weekly consumption of foods was estimated by converting the responses “Rarely or never” to 0, “< 1 time weekly” to 0.5, “1-2 times weekly” to 1.5, “3-4 times weekly” to 3.5, “Daily” or “Once daily” to 7, “Twice daily” to 14, and “With every meal” was 7 multiplied by the number meals per day reported by the participant, or if not reported, 7 times the average meals per day for their residence group.

The semi-quantitative food frequency questionnaire was adapted from a previous study of females from the same ethnic and geographic populations (Núñez-de la Mora, 2004). The intention of this dietary analysis is to establish consumption patterns and proportional variations in diet between residence groups, not to establish a precise nutritional profile.

Of all groups, sedentees reported the highest average weekly consumption of rice (15.8±0.5), of total fruit and vegetables (19.5±1.1), and together with adult migrants, fish (sedentees: 2.5±0.1, adult migrants: 2.6±0.2, $t(166)= -0.31$, $p=.7$). With the exception of the non-significant difference from adult migrants in fish consumption, sedentees ate more of these foods than all other residence groups (ANOVA: rice: $F(4,281)=126.16$, $p<.001$; fruit & vegetable: $F(4,280)=20.65$, $p<.001$; fish: $F(4,270)=32.78$, $p<.001$; see table 21 for descriptives).

Rice and fish consumption show a pronounced inverse relationship with age at migration to the UK (see fig. 16). In the case of rice, only 10% of British-born Bengalis (n=3) consume rice at least once daily, while 95% of adult migrants (n=59) and 93% of sedentees (n=100) said they eat rice at least once a day. Youth migrants fall between these two extremes, with 54% (n=19) eating rice on a daily basis. Sedentees were 124 times more likely than British-born Bengalis, and 12 times more likely than youth migrants to say they consume rice on at least a daily basis ($X^2=85.76$ and 29.81 respectively, both df=1 and $p<.001$). Half of all sedentees (n=54) said they consume rice with every meal, while only 6% of adult

migrants (n=4) reported eating rice this frequently. Instead, 82% of adult migrants (n=51) said they eat rice twice daily, compared with 39% of sedentees (n=42).

Not one British-born Bengali reported consuming fish 3-4 times a week (which was the highest category for this variable), compared with 63% of both sedentees (n=67) and adult migrants (n=39), and 46% of youth migrants (n=16). Differences in proportion of sedentees or adult migrants and youth migrants eating fish 3-4 times a week was not quite significant (X^2=3.33, df=1, *p*=.07), but on average youth migrants consume significantly less fish than their Bangladeshi-born counterparts (1.9±0.3 versus 2.6±0.1 times/week; *t*(201)=2.56, *p*=.01), and significantly more than British-born Bengalis (5.0±0.1; *t*(61)=4.78, *p*<.001).

While fish and rice consumption is roughly similar between sedentees and adult migrants, these two groups show the largest difference in average total fruit and vegetable consumption per week (19.5±1.1 versus 6.0±1.0 times/ week; *t*(165)=8.20, *p*<.001). This is mainly due to reported frequency of eating vegetables in curry: 82% of sedentees (n=87) said they eat vegetables in curry at least daily, compared to 22% of adult migrants (n=13) and 26% of youth migrants (n=9; X^2=43.31 and 86.75 respectively, both df=6 and *p*<.001). Only 2, or 7% of British-born Bengalis said they eat vegetable curry on a daily basis, but average weekly fruit and vegetable consumption was slightly higher than migrant Bengalis (9.1±2.2 versus 7.0±0.8 for migrants), though this difference was not significant (*t*(124)=-1.11, *p*=.3).

Excluding fish, British-born Bengalis and youth migrants report the highest meat consumption of all groups, averaging 2.9±0.4 and 2.7±0.4 times per week (*t*(61)=-0.362, *p*=.7), significantly more than adult migrants (1.6±0.2), sedentees (0.7±0.1) and Europeans (1.1±0.2). Sedentees report eating eggs more frequently than both migrant groups X^2=12.54, df=3, *p*=.006 and British-born Bengalis X^2=15.85, df=3, *p*=.001. All Bengalis living in London eat meat more frequently than sedentees, and this difference remains significant if including fish consumption, but the difference is no longer significant if egg consumption is also included.

Dairy consumption was lowest among adult migrants, where all but 3 (95%, n=59) said they rarely or never consume dairy products, a significantly greater proportion (odds ratio range: 4.4-17.1) than all other Bengali groups (X^2=12.24, df=3, *p*=.007). Average weekly dairy consumption was not significantly different between any of the other Bengali groups, but all consume dairy less frequently than Europeans (X^2=32.80, df=3, *p*<.001).

British-born Bengalis and youth migrants consume fried food, sweet snacks and chips more frequently than adult migrants but not more than Europeans. British-born Bengalis consume fried foods and sweet snacks more frequently than sedentees (X^2=12.24 and 23.84 respectively, both df=3, *p*≤.001), Adult migrants

did not report eating fried foods or sweet snacks more frequently than sedentees, but youth migrants eat sweets more frequently than sedentees (X^2=9.04, df=3, p<.03). Age at migration has a significant positive relationship with weekly consumption of fried food, sweet snacks and chips (see fig. 16).

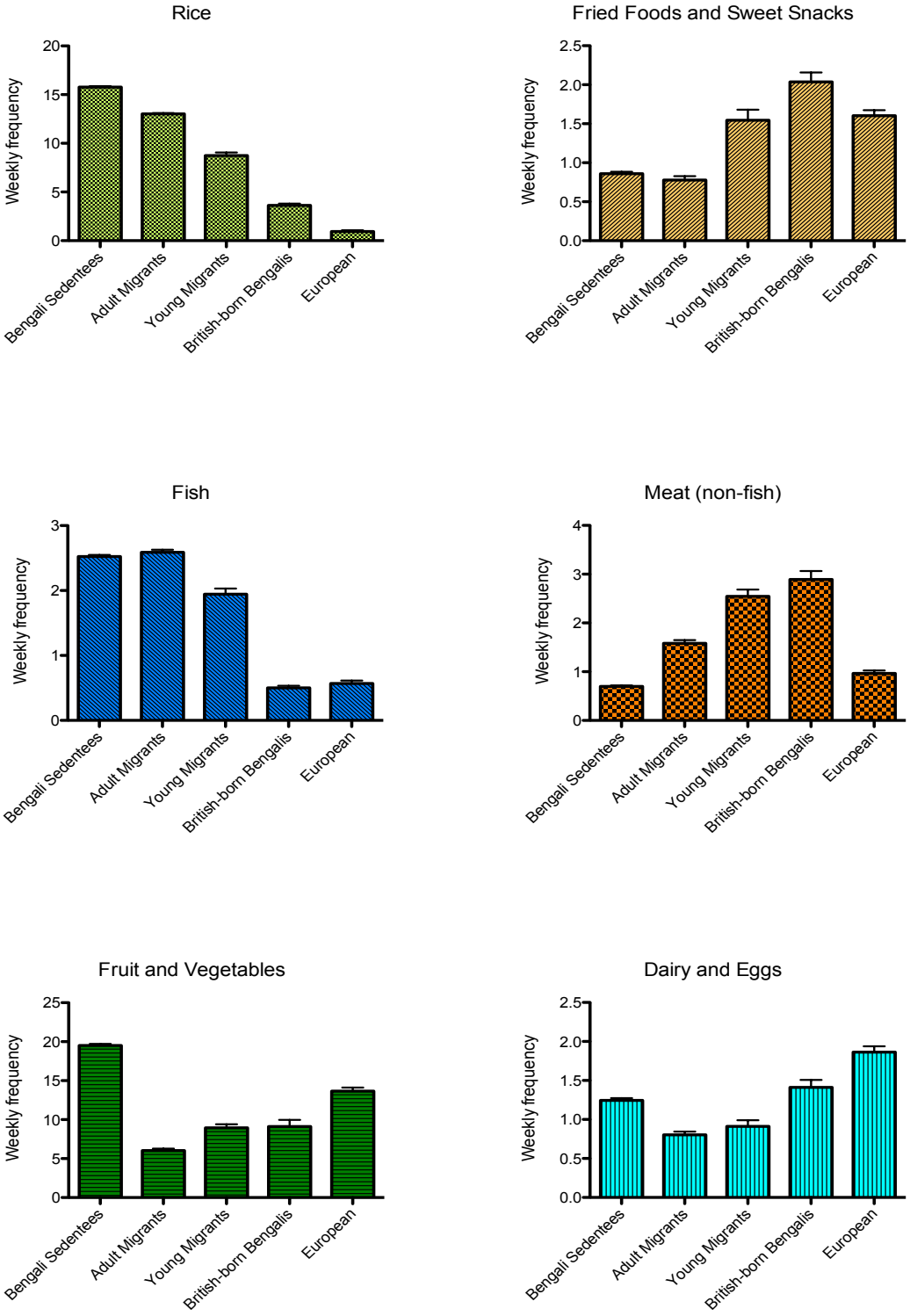
Dietary patterns

There is no difference in the average of 2.2±0.1 meals per day reported by sedentees and migrants (t(235)= 0.35, p=.73). Migrants consume 1.7±0.1 *nasta* or snacks per day, which is more than the 1.4±0.1 reported by sedentees (t(235)= -2.34, p=.02). British-born Bengalis eat 2.7±0.2 meals a day, significantly more than Bangladeshi-born Bengalis (t(235)= -2.93, p=.004), but report the same number of *nasta* per day as migrants.

European men average 2.9±0.1 meals and 2.0±0.2 snacks daily, significantly more than migrant Bengalis (meals: t(259)=-5.40, p<.001, snacks: t(259)=-2.99, p=.003), but the eating patterns of British-born Bengalis and European men, as measured in number of meals and snacks, are not significantly different.

Of Bengalis resident in London, 93% reported always eating Halal. Five youth migrants, 3 British-born Bengalis, and 1 adult migrant said they do not always eat Halal (or 4.4%, 2.8%, 1.2% of each group, respectively). Sedentees were not asked to avoid offence, and on the assumption of ubiquity of the Halal diet in the 92.6% majority Muslim region of Sylhet (Bangladesh Bureau of Statistics, 2001).

Figure 16: Average reported food frequencies



Recalled food frequencies by residence group. Error ±95% CI.

Table 21: Dietary food frequencies

	Sedentees		Adult Migrants		Youth migrants		British-born Bengalis		British-born Europeans	
Food Type	**Mode (%)**	**Avg times/ wk (SD)**	**Mode (%)**	**Avg times/wk (SD)**	**Mode (%)**	**Avg times/wk (SD)**	**Mode (%)**	**Avg times/wk (SD)**	**Mode (%)**	**Avg times/wk (SD)**
Rice	Every meal (50.4%)	15.8 (5.4)	Twice daily (82.2%)	13 (2.8)	Twice daily (42.8%)	5.5 (1.7)	3-4 times weekly (68.9%)	3.6 (2.5)	Rarely/never (62.2%)	1.0 (3.1)
Pasta	Rarely/never (77.5%)	0.3 (0.7)	Rarely/never (93.4%)	0.1 (0.3)	Rarely/never (82.3%)	0.3 (0.4)	<1 time weekly (51.7%)	0.4 (0.6)	Rarely/never (81.1%)	0.3 (0.8)
Potato (not chips)	1-2 times weekly (39.2%)	1.5 (2)	Rarely/never (80.6%)	0.4 (0.9)	Rarely/never (54.2%)	0.9 (1.0)	1-2 times weekly (37%)	1.1 (1)	Rarely/never (54%)	0.7 (1.1)
Lentils/dhal	<1 time weekly (36.7%)	4.4 (6.1)	<1 time weekly (66.1%)	0.5 (0.9)	Rarely/never (48.5%)	2.6 (1.5)	Rarely/never (48.2%)	0.5 (0.7)	Rarely/never (97.8%)	0.01 (0.1)
Chickpeas	Rarely/never (67.6%)	0.3 (0.6)	Rarely/never (100%)	0	Rarely/never (97.1%)	0.3 (0.3)	Rarely/never (62%)	0.2 (0.2)	Rarely/never (60.8%)	0.6 (1)
Fruit	Rarely/never (45.7%)	1.0 (1.6)	<1 time weekly (42.3%)	2.5 (4)	<1 time weekly (30.3%)	3.6 (1.9)	3-4 times weekly (41.3%)	3.2 (4.3)	Twice daily (26.4%)	7.9 (7.3)
Fruit juice	Rarely/never (78.3%)	0.3 (0.6)	Rarely/never (80%)	0.5 (1.2)	Rarely/never (62.8%)	1.3 (1.2)	3-4 times weekly (32.1%)	1.6 (1.4)	Rarely/never (62.2%)	1.0 (1.5)
Salad	Rarely/never (43.8%)	1.8 (3.6)	Rarely/never (46.6%)	0.6 (1.3)	Rarely/never (42.8%)	1.5 (1.2)	Rarely/never (55.1%)	1.7 (4.1)	<1 time weekly (39.1%)	1.6 (2.8)
Vegetables (in curry)	Every meal (53.7%)	13.6 (7.3)	Rarely/never (46.6%)	2.5 (4.5)	Rarely/never (23.5%)	5.4 (2.2)	1-2 times weekly (35.7%)	2.5 (4)	n/a	n/a
Vegetables (steamed/ boiled)	Rarely/never (66.9%)	2.9 (6.1)	Rarely/never (90.7%)	0.1 (0.2)	Rarely/never (78.7%)	1.8 (1.4)	Rarely/never (64.2%)	0.3 (0.5)	<1 time weekly (32.6%)	3.2 (4.7)
Eggs	Rarely/never (47.6%)	1.0 (1.3)	Rarely/never (67.7%)	0.7 (1.1)	Rarely/never (65.7%)	0.9 (0.9)	<1 time weekly (42.8%)	0.6 (0.8)	Rarely/never (74.5%)	0.4 (0.9)
Cheese	Rarely/never (97.1%)	0.03 (0.1)	Rarely/never (95.1%)	0.1 (0.5)	Rarely/never (81.8%)	0.7 (0.6)	Rarely/never (53.5%)	0.4 (0.8)	Rarely/never (73.5%)	0.4 (0.9)
Other dairy	Rarely/never (73.5%)	0.2 (0.5)	Rarely/never (95.1%)	0.04 (0.2)	Rarely/never (82.8%)	0.5 (0.7)	Rarely/never (64.2%)	0.4 (0.6)	Rarely/never (61.5%)	0.9 (1.4)
Fish	3-4 times weekly (63.2%)	2.5 (1.4)	3-4 times weekly (62.9%)	2.6 (1.3)	3-4 times weekly (45.7%)	1.5 (1.2)	<1 time weekly (57.1%)	0.5 (0.5)	Rarely/never (63.6%)	0.6 (1)
Chicken	Rarely/never (67.9%)	0.4 (0.7)	Rarely/never (56.4%)	0.9 (1.3)	1-2 times weekly (48.5%)	1.2 (1.0)	1-2 times weekly (46.4%)	1.5 (1.2)	Rarely/never (60%)	0.5 (0.9)
Beef	Rarely/never (72.3%)	0.3 (0.6)	Rarely/never (96.7%)	0.05 (0.3)	Rarely/never (79.4%)	0.7 (0.7)	Rarely/never (75%)	0.3 (0.7)	Rarely/never (75%)	0.2 (0.4)
Lamb/Mutton/ Goat	Rarely/never (93.3%)	0.04 (0.2)	Rarely/never (69.3%)	0.6 (1)	Rarely/never (47%)	1.3 (1.1)	1-2 times weekly (37%)	1.1 (1.1)	Rarely/never (90.9%)	0.1 (0.3)
Pork	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Rarely/never (82.5%)	0.2 (0.6)
Turkey	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Rarely/never (93.9%)	0.2 (0.7)
Chips	Rarely/never (88.8%)	n/a	Rarely/never (90.1%)	0.1 (0.4)	Rarely/never (74.2%)	0.8 (0.8)	<1 time weekly (51.7%)	0.6 (0.7)	Rarely/never (91.3%)	0.1 (0.2)
Other fried foods	Rarely/never (65.7%)	0.5 (0.9)	Rarely/never (86%)	0.3 (0.9)	Rarely/never (58.8%)	1.1 (1.0)	<1 time weekly (44.4%)	0.8 (0.9)	Rarely/never (78.8%)	0.4 (0.8)
Sweet snacks/ desserts	Rarely/never (75.2%)	0.4 (0.9)	Rarely/never (80.6%)	0.5 (1.1)	Rarely/never (55.8%)	1.4 (1.2)	1-2 times weekly (37%)	1.3 (1.2)	Rarely/never (46.1%)	1.2 (1.4)

4.2 Health behaviours

Analyses were split by participants aged above and below 40 years, to reduce cohort differences in smoking and betel nut chewing, and to reference other comparisons between younger male health behaviours and reproductive functioning.

Alcohol

Sedentees were not asked whether they consumed alcohol. As a majority Muslim nation, alcohol is not widely available, its consumption is prohibited under the Bangladeshi Narcotics Control Act (1990), and drinking carries a severe social and religious stigma.

Total units consumed per week were estimated based on reported frequency and number of beers, wine and spirits, and were multiplied by 0.5 for “On rare occasions”, 1 for “Once a week or less”, and 2 for “More than once a week”.

Most (95%) of migrant and British-born Bengalis said they never consumed alcohol. Bengalis who consume alcohol (n=6) were relatively evenly distributed between British-born (n=2, 6.9%), adult (n=3, 3.5%) and youth migrants (n=2, 6.1%).

Most (94%) of European men said they consume alcohol. Average units of alcohol consumed per week were not different between low and high status Europeans (9.0±1.2 and 10.1±2 units, respectively; $t(71)=-.452$, $p=.65$). While number of units consumed per week negatively correlated with age, it was not quite significant ($r=-.20$, $p=.07$), and older men >40 did not drink less than those <40 (8.0±1.3 and 9.8±1.8 units, respectively; $t(78)=.768$, $p=.45$).

Smoking

A 3-way loglinear analysis of residence group x age 40 split x current smoking produced a final model that retained all effects. The likelihood ratio of this model was $X^2=170.16$, df=23, $p<.001$, indicating the highest-order interaction was significant, but the partial association suggested that the interaction between residence and smoking was not significant ($X^2=9.03$, df=5, $p=.11$). To break down this effect, separate chi-square tests were performed on smoking rates within each residential group.

Overall rates of current smoking were not significantly different between sedentees, adult, or youth migrants (38.7%, 41.0%, 38.2% respectively; $X^2=0.11$, df=2, $p=.95$). Within these three Bangladeshi-born groups, older men >40 were 2.6 times more likely to currently smoke than those <40 ($X^2=10.11$, df=1, $p=.001$).

British-born Bengalis were much less likely to currently smoke (13.80%, odds ratio: 0.25) than the Bangladesh-born groups (X^2=10.11, df=1, *p*=.007), and were not significantly different from British-born Europeans (X^2=1.36, df=1, *p*=.24).

Rates of smoking within low and high status Europeans were not different from one another (24.5% and 23.3%, respectively; X^2=0.02, df=1, *p*=.90), and older European men >40 were no more likely to currently smoke than those <40 (19.4% and 28.3% respectively; X^2=.85, df=1, *p*=.36).

Betel Nut

European males were not asked about betel nut habits, as within the UK the practice is almost exclusive to Asian communities. Only 2 of 29 British-born Bengalis (7%) were betel nut users, and both said they only used it on special occasions, so were not included in further analysis.

Of Bangladeshi-born men, a 3-way loglinear analysis of residence group x age 40 split x current betel nut usage produced a final model that retained all effects. The likelihood ratio of this model was X^2=111.58, df=11, *p*<.001, indicating the highest-order interaction was significant, and the partial association suggested that the interaction between the age 40 split and betel nut usage was not significant (X^2=.19, df=1, *p*=.7). To break down this effect, separate chi-square tests were performed on smoking rates within each Bangladeshi-born residential group.

Sedentees had the highest prevalence of current betel nut usage at 84.1%, and were 4.6 times more likely to use betel nut than adult migrants (53.2%; X^2=18.90, df=1, *p*<.001), and 8.9 times more than youth migrants (37.1%; X^2=29.21, df=1, *p*<.001). The difference between adult and youth migrants was not significant (X^2=2.32, df=1, *p*<.13). Betel nut usage was not different between men aged >40 (69.3%) and men <40 (65.5%; X^2=.33, df=1, *p*<.57).

While migrants were generally less likely to chew betel nut than sedentees, neither age nor overall number of years spent in the UK, nor an interaction between the two predicted betel nut usage (Logistic Regression: X^2=0.299, df=1, *p*=.58).

Frequency of betel nut usage differed between sedentees and migrants (Logistic Regression: X^2=9.33, df=4, *p*=.05), with 51% of sedentees using it more than 3 times a day, compared with 30% of migrants. Migrants were significantly more likely to say they used betel nut only on special occasions compared to sedentees (21% and 10%, respectively; odds ratio: 3.8, p=.007). Frequency was not different between men split at age 40 (X^2=5.47, df=4, *p*=.24).

Activity: sport and exercise per week

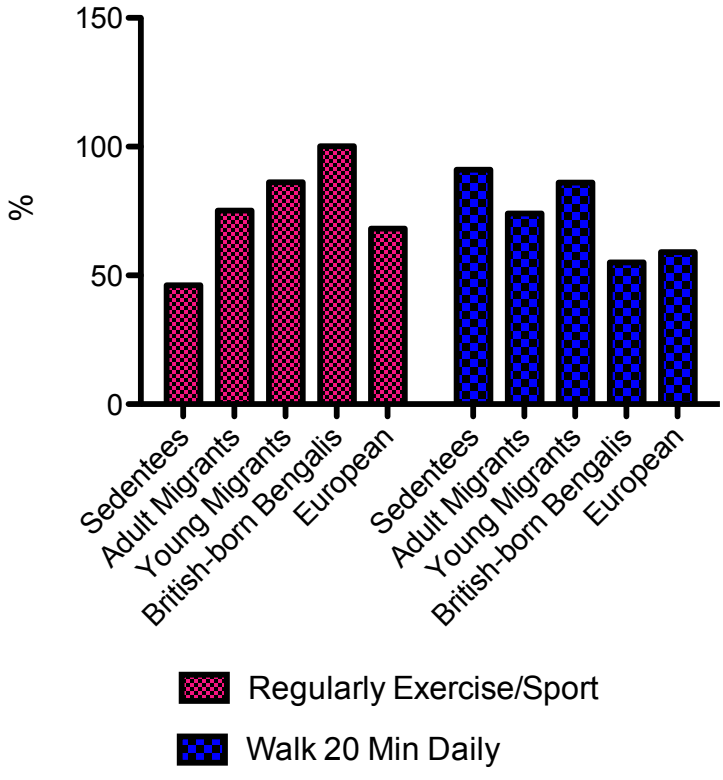
High SES European males were not asked if they regularly walked for at least 20 minutes every day as they were interviewed using a truncated version of the questionnaire.

Sedentees (91.5%) were the most likely residence group to say they walk for at least 20 minutes every day and were 3.7-8.7 times more likely to walk regularly than all other residence groups (all $p \leq .005$), aside from young migrants ($p = .35$). Sedentees were also the least likely group to say they regularly practiced sport or exercise (46.7%).

In contrast, British-born Bengalis were least likely to say they walked regularly (55.2%), but 100% of the 29 respondents said they regularly exercised or practiced sport. Young migrants were the residence group with the second highest rates of regular exercise and walking (86.1% for both). Walking rates were not significantly different between British-born Bengalis and European men (59.3%) or adult migrants (74.6%), though in the latter case this difference was nearly significant ($X^2 = 3.37$, df=1, $p = .07$).

Frequency of exercise per week negatively correlated with age ($r = -.16$, $p = .04$), but on average, younger men <40 did not report exercising significantly more than those >40 (2.7±0.2 and 2.4±0.3 times/week, respectively; $t(157) = .745$, $p = .46$). After correcting for age effects, residence groups did not differ in exercise frequency (MLR: $F(6,157) = 1.52$, $p = .18$).

Figure 17: Reported physical activity



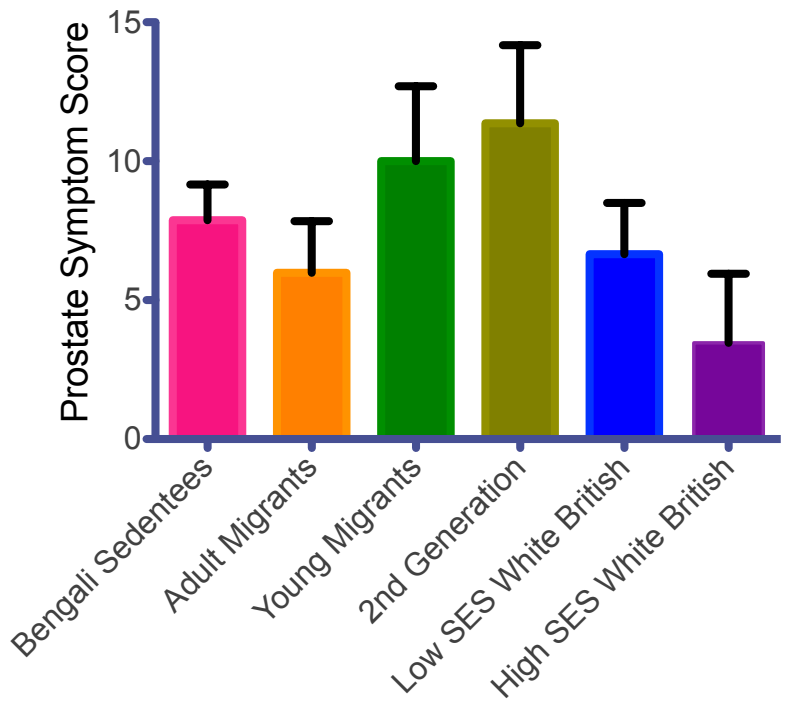
4.3 Prostate symptoms

The occurrence and severity of LUTS were assessed using the International Prostate Symptom Score (IPSS) (Barry et al. 1992). The IPSS combines reported symptoms of frequency, bother, and interference of daily urinary activities. The self-assessed symptoms were combined with perceived quality of life using 6-point Likert scales, summed to score 1-35.

IPSS scores are a World Health Organisation adopted international measure of prostate health used successfully in industrialized countries with a variety of cultures (Barry et al. 1992). Scores were broken into three categories of severity: 0-7 asymptomatic to mild, 8-19 moderate, and 20-23 severe.

Pearson's (two-way) tests for zero-order correlations to the IPSS scores were applied to salT, anthropometric, dietary and health behaviour variables. Age and residence group significantly correlated to the two IPSS scores. Partial correlations correcting for participant age at recruitment and residence group were used to select variables for inclusion in an exploratory stepwise (backward method) MLR, from which variables were selected for a final hierarchical MLR to test the hypothesis that salT predicts prostate symptoms after correcting for the influence of the selected health behaviours and age.

Figure 18: Mean prostate symptoms



Mean reported prostate symptoms by residence group. Error bars ±95% CI.

Hormonal and physical measures

Both IPSS measures significantly correlated to participant age at recruitment, height and residence group, but not to other anthropometric variables or salT in zero-order Pearson's correlations. MUA muscle+bone (but not axial fat) and weight both negatively correlated to IPSS measures after correcting for age at recruitment, but not after also correcting for residence group, so this association between axial muscle and prostate symptoms is explained by residence group. After correcting for age at recruitment and residence, salT was significantly correlated to both IPSS measures, though the effect was greater without the QoL measure. In exploratory MLR, mean evening salT, but not morning salT was a significant predictor variable for IPSSTotal (without QoL).

In exploratory MLR, weekly consumption of rice, fruit and vegetables, and fish all significantly and negatively correlate to both IPSS scores. Weekly consumption of meat (excluding fish) and of fried foods and sweet snacks correlate positively to both IPSS scores.

Health behaviours: Correcting for age at recruitment and residence group, IPSS scores do not correlate with whether men said they drink alcohol, use betel nut, exercise regularly or walk 20 minutes a day. But the exercise frequency and number of alcoholic drinks per week negatively correlate to IPSS score, while smoking positively correlates to IPSS scores. Reported frequency of betel nut use significantly positively correlates to IPSS scores.

To test whether residence group predicts prostate symptoms independent of dietary or health behaviours, a hierarchical entry-method MLR was run on IPSS and IPSS+QoL scores with the following three steps:

1) Age at recruitment and BMI

2) Smoking frequency, Betel nut frequency, Weekly units of alcohol consumed, Weekly exercise frequency, Number of daily meals and Estimated weekly consumption of the following foods: Rice, Meat (non-fish), Fish, Fruit and Vegetables, Fried foods and snacks

3) Residence groups (dummy variable: Sedentees), mean of MeanAM salT and BED salT.

Results: The physical measures and health behaviours (Step 2) of the model significantly predicted both IPSS scores (IPSS: $F(13,108)=3.18$, IPSS+QoL: =3.26, both $p<.001$). Inclusion of residence group and salT variables (step 3) slightly improved the overall fit of the model r^2 from .28 to .31, but this change was not significant (change statistics for IPSS: $F(6,102)=0.77$). The strongest

predictors in the final model were age and weekly rice consumption (standardised *b*=.24 and -.33, respectively, both *p*<.05).

After splitting men at age 40 and re-running the same regressions, the scores of adult migrants were significantly lower than sedentees, while all other residence groups were not significantly different from sedentees (standardised *b*=-.44 and -.47, IPSS, IPSS+QoL respectively, both *p*<.005).

4.4 Fasting blood glucose

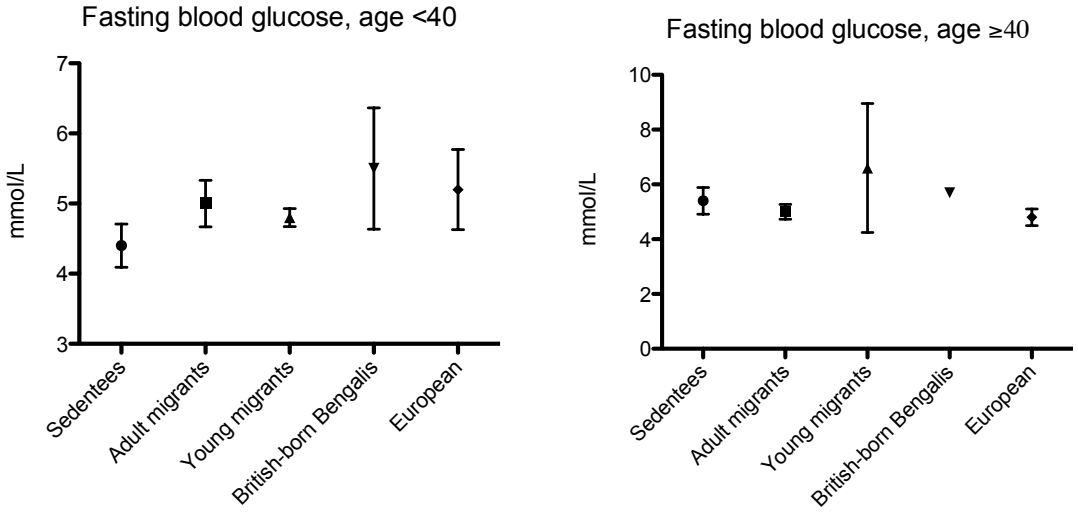
To test whether residence group predicts fasting blood glucose, independent of dietary or health behaviours, a hierarchical entry-method MLR was run on fasting blood glucose with the following three steps:

1) Age at recruitment and BMI

2) Smoking frequency, Betel nut frequency, Weekly units of alcohol consumed, Weekly exercise frequency, Number of daily meals and Estimated weekly consumption of the following foods: Rice, Meat (non-fish), Fish, Fruit and Vegetables, Fried foods and snacks

3) Residence groups (dummy variable: Sedentees), mean of MeanAM salT and BED salT.

Figure 19: Fasting blood glucose



Average fasting blood glucose by residence group. Error ±95% CI.

Results: The physical measures and health behaviours (Step 2) of the model were marginally significant predictors of fasting blood glucose ($F(12,81)=1.18$, $p=.06$). Inclusion of residence group and salT variables (step 3) did not improve the overall fit of the model r^2 change .05, model $p=.13$.

However, after splitting men at age 40 and re-running the same regressions the final model was highly significant and a strong predictor of fasting blood glucose for men <40, and the differences between sedentees and all residence groups were significant contributors to the model. The standardized *b* for residence groups ranged from .67 for adult migrants to 2.27 for British-born Bengalis (all $p<.001$, final model $r^2=.71$, $F(18,35)=4.74$, $p=<.001$). Other positive predictors of fasting blood glucose in the final model were; age; betel nut and alcohol use; fish, rice, fried foods and snack consumption; and times exercised per week, the only significant negative predictor was meat consumption (largest significant standardized $b=1.85$ was for alcohol consumption, smallest was and 0.27 for fried foods and snacks, all $p<.05$).

For men >40, the model was not significant at any step, (all $p=.5$-$.9$). Salivary T did not predict fasting blood glucose in any of the models.

4.5 Dietary or health behaviours and salT

To test whether adult dietary or health behaviours explain differences in salT between Bengalis resident in London, a GLM was run to predict daily mean salT including in the model: age at recruitment, BMI, SES, smoking, betel nut chewing, regular exercise or walking 20 minutes daily, number of daily meals, and consumption of rice, fruit and vegetables, meat, fried foods, and fish. This model was significant ($F(13,40)=2.98$, $p=.004$), after which neither residence group, nor age at migration significantly improved the model ($t(14,39)=1.22$, $p=.2$ and $t(14,38)=0.06$, $p=.9$, respectively).

To test whether adult dietary or health behaviours explain differences in salT between migrants and sedentees a GLM was run to predict daily mean salT as outcome and residence group (selecting ethnic Bengalis only) as predictor with all dietary, smoking, betel nut usage, exercise, age and BMI, as covariates. In the final model, sedentee salT was significantly higher than adult migrants (respective estimated marginal means: 96±5 versus 79±7 pg/mL, $p=.05$) while there is no significant difference between sedentees and young migrants or British-born Bengalis (respective estimated marginal means: 110±9 versus 104±12 pg/mL).

4.6 Conclusions

Adult migrants share many of the dietary characteristics of sedentees, notably in consumption of rice and fish. While fish and rice consumption don't appear to decline after migration, adult migrants partly follow the documented pattern of more frequent consumption of meat, a "special menu" Bengali food, but they don't report consuming more sweet snacks or fried foods as found elsewhere (Chowdhury et al. 2000). However the adult migrants do report eating more

snacks than the sedentees, and the customary nasta in both sedentee and migrant Bengali households is sweetened tea and biscuits, so these two results appear somewhat contradictory.

The sedentees appear to make up for a lower consumption of protein from meats other than fish by consuming more eggs, and the lack of a difference between sedentees and migrants when frequency of meat and egg consumption are combined indicates that the sedentees aren't necessarily consuming substantially less protein overall. Sedentees stand apart from all other groups in their reported consumption of fruit and vegetables, which indicates a maintenance of the traditional Bengali diet in Sylhet. In comparison, all Bengalis living in London eat fruit and vegetables much less frequently. This may be due to the expense of purchasing traditional Bengali vegetables, a concern expressed in focus groups of British Bengalis (Lawrence et al. 2007). The other striking feature of food frequencies was the clear acculturation trend away from rice and toward meat and fried foods among young migrants and British-born Bengalis.

Fasting blood glucose:

While the food frequencies indicate an increased adoption of a diabetogenic diet, the results of the fasting blood glucose regressions indicated that even after correcting for dietary differences, younger London Bengalis, and British-born Bengalis in particular were likely to have high fasting blood glucose compared to sedentees. The fact that these differences were not seen between older men >40 may be due to the relatively small number of young migrants and British-born Bengalis in this age range (n=4 and 1, respectively). But the fact that the model was not significant when comparing only the adult migrants and sedentees >40 suggests that residence group or health behaviours are not explanatory of fasting blood glucose levels. When looking at the data of the older men, it is critical to remember that diagnosed diabetes was an exclusion criterion for recruitment in this project, and it is likely that this selective sub-clinical group represents a different cohort than those <40 who currently have high fasting blood glucose and may develop the disease at a later date. The lack of an explanatory effect of salT in any of the models is not necessarily surprising, as previous associations between insulin and T were strongest when measured as a ratio to E_2 or longitudinally (Laaksonen et al. 2004; Phillips et al. 2002).

Dietary patterns:

The lack of a significant difference between sedentees and migrants in number of meals per day supports the assumption that the sedentee population is not currently under nutritional stress. Of course a lack of a difference does not confirm a hypothesis, and the lack of portion or meal size means there may be a lower

number of calories consumed per meal, though in light of the lack of significant differences in BMI, one can conclude that the sedentee population is not subjected to high dietary restriction. Migrants eat more snacks than sedentees though, suggesting a change in consumption to more of a modern British "grazing" pattern throughout the day and fewer family meals eaten together (Pollard et al. 2002). The nearly universal observation of Halal indicates that the migrant community maintains dietary patterns with cultural and religious considerations in mind, this fits with other studies of the British Bengali population (Chowdhury et al. 2000).

Prostate symptoms

The proximate hypothesis that prostate symptoms relate directly to salT, after correcting for dietary and health behaviours, was not supported except in the case of adult migrants aged ≥40 years. If lifetime exposure of the prostate to high T were responsible for LUTS, men with the highest T would have the highest incidence of symptoms. Instead, only Bengalis, but not Europeans who spent all or part of their childhood in London show high rates of symptoms. This does not fully support the hypothesis that men whose childhood development occurred under more constrained energetic conditions than those of their adulthood will exhibit a greater number of prostate symptoms. If this were the case, after correcting for age, both adult migrants and youth migrants would exhibit the highest incidence of LUTS. Instead, it would appear that men who spent all of their childhood in Bangladesh have the lowest rates of LUTS. This may be due to the poorer diet amongst Bengalis who migrated as children or were born in Britain. Keeping in mind small sample size for British-born Bengalis and youth migrants over age 40, incidence of prostate symptoms increase at a roughly linear rate.

Overall, these results suggest that youth migrants and British-born Bengalis adopt a poorer diet than other Bengali groups or Europeans. These health behaviours do not fully explain the difference in fasting blood glucose between the young men of these groups and their sedentee counterparts, and combined with the well-documented risk factors of South Asian ethnicity and low SES for NIDDM (Bhopal et al. 1999; McKeigue et al. 1991) these results suggest younger men of the migrant community are at a very high risk of adult-onset diseases in the future.

After correcting for adult dietary and health behaviours, differences in salT between Bengalis resident in London due to differences in developmental timing of migration were no longer evident. Bengalis who migrated at ages before maturity or were British-born adopt a diet that includes less rice, less fish, and more fried foods and meat compared with adult migrants. These behaviours, possibly combined with improvements in SES for Bengalis who spent all or part of their childhood in London, contribute to greater investment in reproductive function than Bengalis

who spent all of their childhood in Sylhet, as measured by salT. This change in behaviours, investment in reproductive function and growth is also reflected in increased stature, skeletal muscle mass, fasting glucose and prostate symptoms, compared to sedentees, adult and adolescent migrants.

The lack of a significant difference between sedentees and Bengalis who spent all or part of their childhood in London, once current dietary and health behaviours are taken into account, suggests the ecological differences experienced during childhood in London or Sylhet are not as influential upon adult salT as present surroundings and behaviours, or that current conditions are masking the influence of earlier developmental conditions. In addition, the unexpected finding of significantly higher salT among sedentees as compared to adult migrants, once current dietary and health behaviours are taken into account suggests that current conditions for sedentees in Sylhet, including presumed exposure to parasites or other ecological stressors are either not affecting salT at all, or other factors are counteracting such stressors to lead to higher salT levels in the sedentees when compared to men who shared the same childhood but are no longer subject to the stressors in adulthood. A potential distinguishing factor to the observed differences in salT between sedentees and adult migrants may be perceived status, and their SES relative to their surroundings. This will be explored further in the next chapter.

Chapter 5: Social factors and reproductive function

In this chapter I test hypotheses relating two measures of current social ecology: male status and reproductive relationships, to investment in reproductive effort as measured by salT of Bengali and European men. The first part of the chapter concerns socioeconomic hypotheses: if men are of low SES, relative to surroundings, then they will invest less into reproductive effort, as measured by salT than men who are of high SES, relative to surroundings. The influence of relative SES on male reproductive effort is hypothesised to be greater following a shift in male reproductive function and fertility in mid-life, at age 40 years. All males appear to retain fecundability late into life, but the likelihood of further reproduction declines for most men after age 40, excepting those with higher status or wealth (Cronk 1991; Kaplan et al. 2000; Marlowe 2000).

The second half of the chapter concerns two variables of male reproductive status, current marriage and number of offspring. The length and types of human pair bonds vary significantly across cultures based on socioecological conditions. The degree of paternal investment in offspring and mate-seeking competition between human males is also widely variable reflecting the compromise between reproductive interests and offspring investment strategies (Ellison 2009).

Socioeconomics

Dominance and rank are critically important to reproductive investment in males (Archer 2006; Dabbs and Dabbs 2000), and SES is arguably the closest approximation to social rank in humans (Sapolsky 2004). Psychosocial stress, as mediated by glucocorticoids, appears to suppress male gonadal function (Hardy et al. 2005). Health biomarkers of stress and SES correlate to relative positioning in a society more than absolute measures of wealth, in such a way that low-status individuals in wealthy countries with high inequality have poorer health outcomes than those resident in countries with low inequality (Marmot 2006; Wilkinson and Pickett 2009). In the context of this project, low-status men in the UK are expected to experience greater SES-related stress than high-status men living in Bangladesh, despite the great disparity in absolute measures of wealth between the two countries.

A fundamental transition for the male life history is the allocation of reproductive effort to the competing requirements between competition for mating opportunities and investment in maintaining current pair-bonds and paternal care. Younger males are more likely to compete physically for mating opportunities, regardless of SES as they will not have offspring in which to invest paternal care, and low status may mean all of a male's reproductive opportunities will occur in early adulthood. If men survive to older adulthood, they are unlikely to succeed in direct

physical competition with younger males, but they are likely to have further mating opportunities if they are of high status.

The contrasting ecologies of the UK and Bangladesh allow for the testing of two hypotheses:

1. If a male is of high SES relative to current surrounding ecological conditions, he is expected to divert more effort toward reproductive function than men of low SES, relative to current ecological surroundings.

Prediction: High SES males, relative to current surroundings will have higher salT than low SES males.

2. If salT modulates male allocation of reproductive effort between competition for mating opportunities and investment in maintaining current pair-bonds and paternal care, this allocation will be of greater importance in the later portion of adulthood. Current relative SES is hypothesised to be more influential on reproductive effort of men in the latter half of their reproductive stage of life, compared to men in the first half of this stage of life. Prediction: High SES males have higher salT, and that relative SES is more highly associated with salT in men aged 40 years or older.

Methods

To estimate wealth, an index was created of 11 household possessions in the UK, and 8 in Bangladesh (see Appendix 1: III.5). This method of estimating SES is frequently used in cross-cultural contexts as an indicator of the level of wealth that is consistent with expenditure and income measures (Gwatkin et al. 2007; Montgomery et al. 2000). Television ownership was added to the Bangladeshi questionnaire and assumed nearly ubiquitous in the UK households, while “washing machine”, “tumble dryer”, “dishwasher” and “central heating” were omitted as these are not common household possessions in Bangladesh where servants usually perform domestic chores.

The wealth index for Bengalis living in Sylhet is relative. As described in chapter 1, this population falls within a middle to high SES population by the standards of urban Bangladesh (Islam 2005). As this group is from the middle class, 89% of sedentees reported monthly household income at or above the national average for urban populations of Tk 9878 Bangladeshi taka (or approximately £82 GBP).

Housing type was categorised in the UK as “bedsit/hostel”, “flat/maisonette”, or “house” and in Bangladesh as “katcha”: corrugated iron or mud brick construction, “semi-pakka”: concrete walls with corrugated iron roofing or “pakka”: a permanent structure with concrete walls and roof (UN-HABITAT 1996). Number of rooms and total persons (adults and children) permanently resident were divided by number

of rooms to create an index of household crowding. All participants were asked if they owned or rented their accommodation and if renting, whether they were private or local authority tenants.

Participants were also asked to select a band estimating their total monthly income after tax.

Results

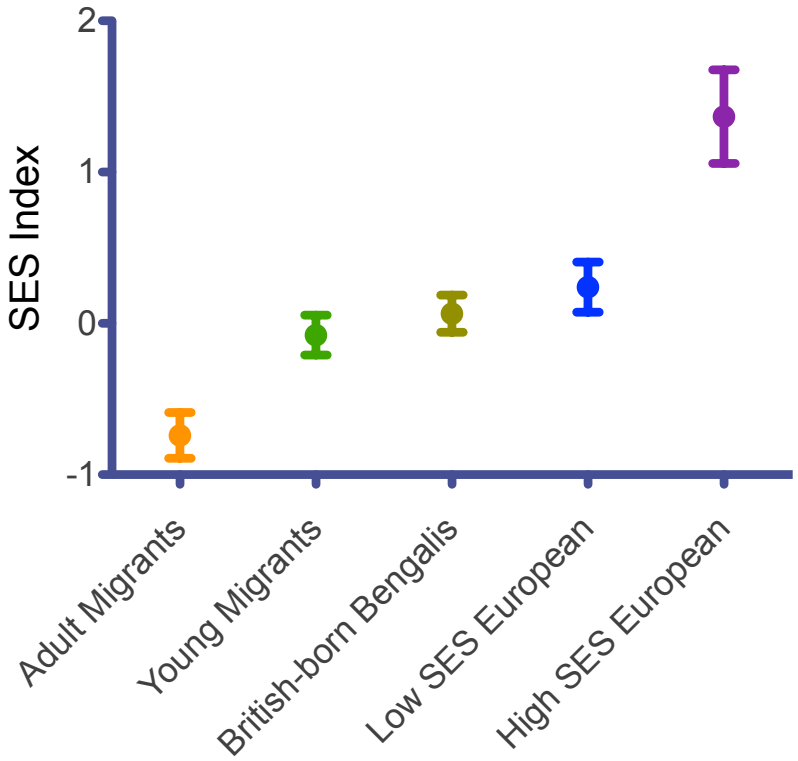
A principal component analysis (PCA) was conducted on 16 items for UK residents, and separately on 12 items for sedentees (see table 22 for descriptives). As income was frequently omitted, PCA excluded this variable pairwise and a factor weight was assigned to each participant. All variables were significantly correlated to income, aside from central heating and owning a freezer (both p>.2).

The London Bengali community was not socioeconomically homogenous. Residence group significantly predicts SES (ANOVA F(4,277)=67.0, r^2=.49 p<.0001; see figure 20). Age at recruitment is not a significant covariate for SES, except for migrants who arrived in London before adulthood. Within youth migrants, older males are significantly more likely to have a lower SES, as do those who migrated as an adolescent between the ages 12-18 years versus those who migrated as an infant or child, after correcting for age at recruitment. Youth migrant and British-born Bengalis are of significantly higher SES (0.06±0.5 SD) than adult migrants (adult migrants: −0.74±0.7; youth migrants: −0.07±0.5). Youth migrants were of significantly lower SES than low SES Europeans (0.24±0.8), while for British-born Bengalis this difference was not significant. High SES Europeans were significantly higher than all other groups (1.4±0.8).

Socioeconomic measures

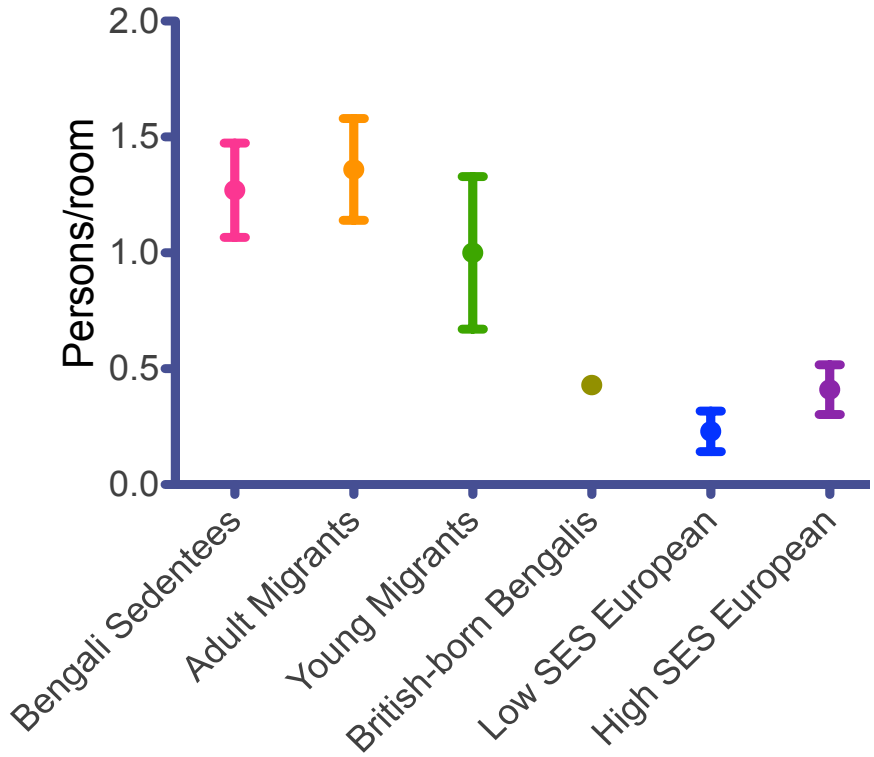
Within the UK, SES quintile with participant age at recruitment as a covariate does not significantly predict daily salT, but the model becomes significant if including residence group (ANCOVA $F(5,201)=10.31$, $p<.001$). Both SES and residence significantly contribute to the model ($F(4,184)=2.47$ and 3.82, both $p\leq.05$), with a nearly significant interaction effect as well ($F(13,184)=1.67$, $p=.07$). Parameter estimates indicate the interaction between residence group and SES is significant for all three Bengali groups in the UK, but not for Europeans.

Figure 20: UK SES by residence group



Reported housing crowding by residence group. Error ±95% CI.

Figure 21: House crowding



Composite SES index by residence group. Error ±95% CI. .

Table 22: SES variables, by residence group split age 40

		Residence Group											
		Bengali Sedentees		Adult Migrants		Youth migrants		Second Generation Migrants		Low Status White British		High Status White British	
Age 40 Split Ages 40 to oldest		Count	N %	Count	N %	Count	N %	Count	N %	Count	N %	Count	N %
Marital Status of Participant	Single, never married	0	0.00%	0	0.00%	0	0.00%	0	0.00%	13	48.10%	1	9.10%
	Married	43	97.70%	34	97.10%	9	100.00%	1	50.00%	10	37.00%	10	90.90%
	Cohabiting with Partner	0	0.00%	0	0.00%	0	0.00%	0	0.00%	2	7.40%	0	0.00%
	Divorced or Separated	0	0.00%	1	2.90%	0	0.00%	1	50.00%	2	7.40%	0	0.00%
	Widowed	1	2.30%	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%
Offspring Details	Yes, children	42	95.50%	31	88.60%	8	88.90%	2	100.00%	9	32.10%	1	9.10%
	No children	2	4.50%	4	11.40%	1	11.10%	0	0.00%	19	67.90%	10	90.90%
Housing Category	Bedsit/hostel	0	n/a	1	3.20%	0	0.00%	0	0.00%	1	3.80%	0	0.00%
	Flat/maisonette	0	n/a	26	83.90%	7	87.50%	1	50.00%	17	65.40%	0	0.00%
	House	0	n/a	4	12.90%	1	12.50%	1	50.00%	8	30.80%	0	0.00%
	Katcha	9	20.50%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
	Semi-Pakka	17	38.60%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
	Pakka	18	40.90%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
Own or rent home	Rents	6	13.60%	19	95.00%	1	33.30%	1	50.00%	9	34.60%	0	0.00%
	Owns	38	86.40%	1	5.00%	2	66.70%	1	50.00%	17	65.40%	11	100.00%
Age 40 Split Ages youngest to 39 years													
Marital Status of Participant	Single, never married	45	75.00%	3	12.00%	13	50.00%	19	82.60%	17	63.00%	11	57.90%
	Married	15	25.00%	22	88.00%	13	50.00%	4	17.40%	3	11.10%	8	42.10%
	Cohabiting with Partner	0	0.00%	0	0.00%	0	0.00%	0	0.00%	3	11.10%	0	0.00%
	Divorced or Separated	0	0.00%	0	0.00%	0	0.00%	0	0.00%	4	14.80%	0	0.00%
	Widowed	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%
Offspring Details	Yes, children	11	18.30%	21	84.00%	10	38.50%	1	4.30%	6	22.20%	11	57.90%
	No children	49	81.70%	4	16.00%	16	61.50%	22	95.70%	21	77.80%	8	42.10%
Housing Category	Bedsit/hostel	n/a	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%
	Flat/maisonette	n/a	0.00%	24	96.00%	13	50.00%	13	59.10%	15	55.60%	0	0.00%
	House	n/a	0.00%	1	4.00%	13	50.00%	9	40.90%	12	44.40%	0	0.00%
	Katcha	18	30.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
	Semi-Pakka	25	41.70%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
	Pakka	17	28.30%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%	n/a	0.00%
Own or rent home	Rents	0	0.00%	17	89.50%	13	54.20%	9	45.00%	16	66.70%	8	42.10%
	Owns	59	100.00%	2	10.50%	11	45.80%	11	55.00%	8	33.30%	11	57.90%

Average housing overcrowding was greatest for adult migrants, at 1.4±.09 persons/per room (see figure 21). In independent 2-tailed *t* test this was not significantly different from overcrowding rates for sedentees 1.3±.07 (: $t(126)=-1.0$, $p=.3$. All other groups living in London are less crowded than adult migrants, (ANOVA: $F(5,230)=22.3$, $p<.001$), while rates of crowding between youth migrants and British-born Bengalis do not differ from one another (0.98±.1 and 1.14±.2, respectively; $t(34)=.47$, $p=.5$), but were significantly more crowded than either European group (0.4±.05 for low SES, 0.5±.03 for high SES; difference from combined British-born and youth migrant Bengalis $t(64)=4.2$ $p<.001$).

For sedentees, the score on the Bangladeshi SES index does not predict salT, after correcting for age and BMI ($F(4,63)=1.97$, $p=.11$). After splitting at age 40, SES still had no predictive effect upon salT within the sedentee population.

To determine whether SES explained salT differences between Bengalis resident in London, a GLM was run predicting daily mean salT after selecting out migrants and British-born Bengalis, correcting for age, BMI and SES quintiles. The interaction effect between age at recruitment and SES significantly predicted salT ($F(5,63)=2.79$, $p=.03$), as did residence group alone ($F(1,63)=6.21$, $p=.02$), no other variables or interactions were significant. To check for a cohort effect, year of migration was added as a covariate, but did not significantly improve the model after correcting for age at recruitment ($t(5,75)=0.07$, $p=.9$).

5.1 Reproductive strategies

A growing body of data suggests an association between pairbonding and/or parenthood and testosterone of human males (Burnham et al. 2003; Gray et al. 2006). This association is thought to reflect the trade off between paternal investment in current offspring and effort directed toward obtaining further reproductive opportunities. While cross-cultural research in this area is expanding (Gray et al. 2007; Kuzawa et al. 2009), the bulk of these studies investigated North American men, with limited cultural variation in mate choice, extramarital sex and direct paternal investment in offspring. Previous analysis of a subsection of the Bengali sedentee population suggests the influence of marital or paternity status are not reflected by measures of salT (Magid et al. 2006).

Pair bonds in Bangladesh are traditionally arranged by guardians, usually older male relatives through a marriage broker, with a dowry paid to the groom at marriage (Aziz and Research 1979). The contrasts between Western and non-Western populations in the degree to which pairbonded men or fathers show lower reproductive investment than non-pairbonded or childless men are hypothetically due, at least in part, to differences in the social ecology of male relationships in the contrasting cultures. If the influence of different ecologies during childhood

development determine coordination of male endocrine function and the social relationships of pair-bonds and offspring, men who are exposed to Western influences of acculturation during childhood development are expected to exhibit greater reduction in reproductive function if they are married or married with children as compared to men who were less exposed to such influences.

The cultural differences in relationship and reproductive patterns between Bangladesh and the UK allow for the testing of the following hypothesis: If different ecologies during childhood development determine coordination of male endocrine function and the social relationships of pair-bonds and offspring, do men who are exposed to Western influences of acculturation during childhood development exhibit greater reduction in reproductive function if they are married or married with children as compared to men who were less exposed to such influences. The specific prediction is that Bengalis who spent all or part of their childhood in London and British European men will show lower salT if they are married or have young children than Bengalis who spent all of their childhood in Sylhet.

Methods

Questions on marital status (Questions I.12-I.14, Appendix 1) and offspring (Question VIII, Appendix 1) were asked to establish relationship and paternity status. SalT measures were collected and analysed as detailed in chapter 2.

Results:

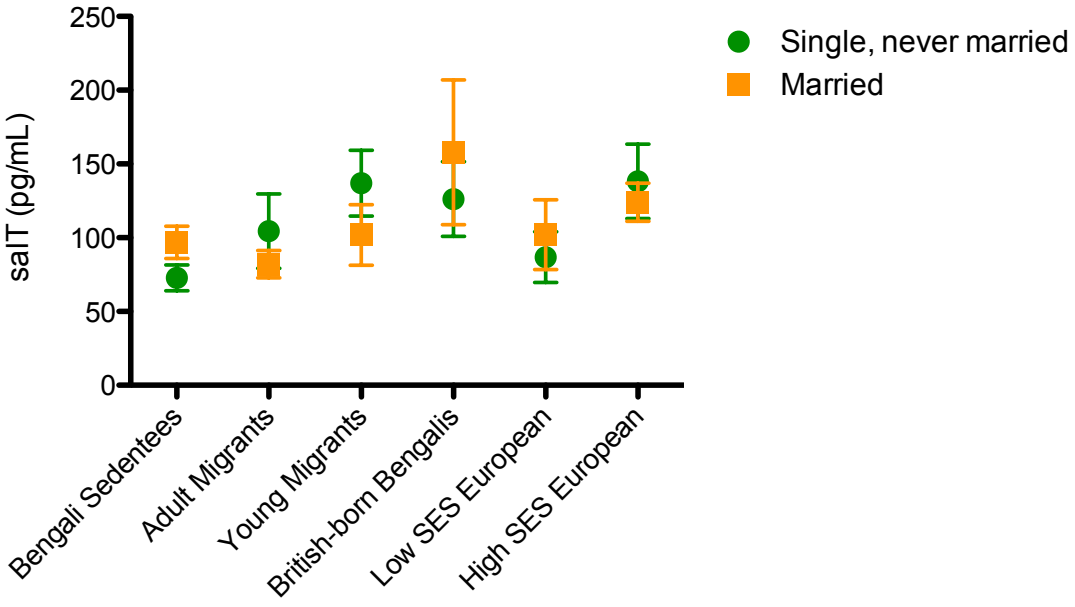
Overall, men who matured at a later age have more offspring in total, after correcting for age at recruitment (pearson's r= .16, df=234, p=.02), also, men who come from large families have more offspring themselves (r= .16, df=353, p=.003). Morning, evening or mean daily salT do not predict number of offspring, age of youngest offspring, or having offspring under age 5 (see table 23). When split by residence group or by age 40, salT does not predict either age of youngest offspring, or having offspring under age 5 for any grouping.

Mean daily salT correlates to total number of offspring, after correcting for age at recruitment, BMI, SES, and residence group differences (MLR corrected model: F(12,64)=6.845 p<.001). After splitting by age 40 and residence groups, salT predicts total offspring number in European men >40 (MLR: std$B$=0.513, t=2.7, df=(4,21) p=0.01), but not in Europeans <40 or any Bengali group, though there were not sufficient numbers of young migrants or British-born Bengalis >40 to perform any tests on those groups.

Marital status does not corollate with daily mean salT overall (see figure 22), after correcting for age at recruitment, BMI, and SES (F(4,171)=15.81, p<.2). After

splitting by age 40 and residence groups, there is not an effect of marital status upon salT for any residence group <40, though there is a non-significant trend toward higher salT among married British-born Bengalis ($t(4,16)=1.89$, $p=.04$. In men >40, married low SES Europeans have higher salT than non married counterparts within their residence group ($t(4,20)=2.21$, $p=.04$). For all other groupings, marriage does not predict salT.

Figure 22: Salivary T by marital status



Marital status by residence group, salT unadjusted for age. Error ±95% CI.

Table 23: Regression coefficients: total number of offspring

		Unstandardised Coefficients		Standardised Coefficients		
Model		*B*	Std. Error	β	*t*	*p.*
1	(Constant)	-0.134	0.846		-0.159	0.874
	Age Participant Recruited	0.048	0.007	0.428	6.702	**<.001**
	BMI (kg/m^2)	-0.024	0.033	-0.046	-0.731	0.466
	UK SES Index	-0.496	0.109	-0.289	-4.567	**<.001**
2	(Constant)	0.496	0.89		0.558	0.578
	Age Participant Recruited	0.041	0.008	0.364	5.174	**<.001**
	BMI (kg/m^2)	-0.016	0.033	-0.029	-0.472	0.638
	UK SES Index	-0.475	0.108	-0.276	-4.388	**<.001**
	Combined Daily Avg salT (pg/ml)	-0.005	0.002	-0.144	-2.089	**0.038**

Dependent Variable: Total number of offspring

Table 24: MLR Regression coefficients for men <40, resident in London

			Unstandardised Coefficients		Standardised Coefficients		
Residence Group	Model		*B*	Std. Error	β	*t*	*p.*
Adult Migrants	1	(Constant)	5.442	3.74		1.455	0.156
		Age Participant Recruited	-0.003	0.031	-0.014	-0.084	0.933
		BMI (kg/m^2)	-0.02	0.121	-0.029	-0.168	0.867
		UK SES Index	1.5	0.624	0.412	2.404	**0.023**
	2	(Constant)	5.697	3.927		1.451	0.158
		Age Participant Recruited	-0.007	0.036	-0.04	-0.199	0.844
		BMI (kg/m^2)	-0.012	0.126	-0.017	-0.098	0.922
		UK SES Index	1.422	0.702	0.39	2.024	**0.053**
		Combined Daily Avg salT (pg/ml)	-0.003	0.012	-0.059	-0.259	0.797
Low Status White British	1	(Constant)	-0.287	1.24		-0.231	0.819
		Age Participant Recruited	0.01	0.011	0.207	0.973	0.341
		BMI (kg/m^2)	0.006	0.045	0.028	0.139	0.891
		UK SES Index	0.383	0.202	0.403	1.901	**0.071**
	2	(Constant)	-1.551	1.195		-1.297	0.209
		Age Participant Recruited	0.022	0.01	0.434	2.102	**0.048**
		BMI (kg/m^2)	0	0.04	0.001	0.008	0.994
		UK SES Index	0.504	0.184	0.53	2.737	**0.012**
		Combined Daily Avg salT (pg/ml)	0.008	0.003	0.513	2.667	**0.014**

Dependent Variable: Total number of offspring

5.2 Conclusions

The predictive effect of residence group on salT was still significant after correcting for differences in SES, supporting the hypothesis that differences in ecology in early life leads to increased investment in adult reproductive function, and the observed salT differences are not due to differences in the relative social positioning of infant/child migrants and British-born Bengalis versus migrants who arrived as adolescents or adults. The interaction between age at recruitment and SES reflects the significant upward trend in SES among the younger Bengalis resident in London (Pearson's *r*=-.301, *p*=.003), but does not fully explain salT differences between London Bengalis.

Only low SES European males show an effect of reproductive status upon salT, as measured by marriage or number of children. In the case of marriage the effect is in the opposite direction from the prediction of this study; men in a married relationship who are in the second portion of their reproductive stage of life have

higher levels of salT than non-married men. For total number of offspring, the prediction that men would have lower salT if they had young children, or that age of youngest child would have a significant negative relationship with salT, is not supported. European men of low SES with more children overall have higher levels of salT. High SES Europeans do not show a systematic relationship between salT and offspring number or age, so the different patterns in paternity or relationship appear related to SES. The difference between older European men and any of the Bengali groups, with regard to reproductive status suggests a different reproductive strategy for men who grew to maturity in Bangladesh.

Unfortunately, a comparison that cannot be drawn from these results whether paternity or relationship status will have an effect upon older Bengali men who were born in Britain or came here as children because of the lack of men over 40 in these groups. The total number of offspring is much lower in the European group, as compared to either adult migrants or sedentees in the same latter portion of their reproductive stage of life. It may be that the effects of paternity upon reproductive function in older men is subject to a threshold dependent on the difference between having one child or two.

Overall, the lack of a paternity or relationship status effect upon salT in the Bengali population suggests the pattern of reduced salT among fathers or married men observed in Western populations may reflect aspects of paternal investment or mate-seeking that do not apply within Bengali culture.

Chapter 6: Conclusions

This project applies life history theory to predict male reproductive function across the three variables of ethnicity, ecology, and development. The preceding three results chapters present variations in Bengali and European male reproductive phenotype, health behaviours and diet due to changes in ecological conditions during development. These findings contribute to the growing body evidence that salT, stature and apportionment of skeletal muscle vary in accordance with early life conditions and the strategic allocation of reproductive effort in the human male (chapter three), with a corresponding increase in early symptoms of adult onset disease of the prostate and glucose metabolism (chapter four), and in accordance with perceived status and relative SES (chapter five). Predicted blunting of diurnal salT profile in adult migrants (chapter three) were inconclusive. Contrary to the predictions of this study, sedentees do not show lower levels of salT than adult migrant counterparts, despite greater exposure to presumed ecological stressors such as exposure to pathogens and environmental risks (chapter three). The presumption that the London Bengali community is socioeconomically homogeneous was not supported (chapter five). Bengali men do not have lower salT in relation to reproductive status of paternity or marriage, regardless of any acculturation due to developmental exposure to life in London, while older British-born European men of low SES have higher salT in relation to number of offspring and marital status (chapter five).

The measures used in this study were subject to differential environmental sensitivity, depending on the point in development when conditions improved. Five key points in the life history were considered: 1. Pre-birth 2. Infancy (0-2 yo) 3. Mid-childhood (3-8 yo) 4. Late-childhood/adolescence (9-18 yo) 4. Adulthood (18 years+). Each period contains critical developmental phases that influence adult male reproductive effort, as measured by salivary testosterone and somatic indices. This chapter draws conclusions from these findings at two levels of inquiry. At the proximate level, how does the functioning of reproductive organs and hormonal axes interact with developmental history and current surroundings? At the ultimate level, how do these results reflect the balancing of the competing biological functions of survivorship and reproductive effort has been shaped by natural selection? These principles extend to the field of evolutionary medicine, where trade-offs of investment between competing physiological requirements explain senescence and disease.

Proximately, exposure to the ecological conditions in London, if experienced during childhood lead to males growing taller, more muscular, maturing earlier and with higher testosterone compared to ethnically matched sedentees.

The influences of these contrasting environments are subject to critical periods of development: younger men set their reproductive effort according to their childhood environment, particularly during the juvenile period of “slow growth” between ages 3-12, while older migrants show decreased reproductive effort due to either current or previous adult experience, with higher SES relative to current surroundings associated with greater salT in sedentee counterparts. If migrating after the age of maturity, there are no measured differences in salT between migrants and sedentees in early adulthood before age 40, while older sedentees appear to invest more into reproductive function as measured by salT than their adult migrant counterparts. This contrasts with the somatic data from men under age 40, which show greater amounts of both skeletal muscle and axial fat in all migrant groups compared to sedentees, regardless of age of migration. The high levels of T in the older sedentees in the absence of corresponding raised skeletal muscle may indicate another modulating influence on muscular growth or apportionment, or reduced sensitivity of muscle cells to testosterone in older men living under the environmental stresses of life in Bangladesh.

The juvenile period is commonly considered a quiescent stage of male reproductive development, but in this study it appears sensitive to ecological conditions. This period of apparent dormancy of the testes and low levels of circulating sex hormones falls between the postnatal peak of activity in the first year of life and the surge of activity accompanying puberty. This leaves open the possibility that sensitivity during adrenarche is important in the coordination of the early initiation of puberty (Campbell 2003). The limited sample size of youth migrants who experienced a change in ecological conditions before and after adrenarche (approximately aged 7 years in males) was not large enough to definitively establish whether adult salT levels were “set” during this developmental period. The potential role of adrenarche may be pursued in future work applying a similar research design with a larger sample size.

Demographic observations relate environmental stresses upon male children during this period to the phenotype of their male offspring (Bygren et al. 2001; Pembrey et al. 2005). Though the mechanism of this effect is not yet known, it has led to speculation that this period is a critical window for the organisation of trans-generational epigenetic information, which is particularly relevant to males (Pembrey 2010).

The finding of an earlier timing of puberty for infant migrants and British-born Bengalis also supports the hypothesis that childhood development in the UK is less constrained than in Bangladesh. As age of migration only predicted earlier maturity for migrants who arrived before age 2, a change in environment during these years could conceivably alter both the tempo and hormonal parameters of

sexual maturity. This early stage of infancy may be important to the establishment of HPT axis thresholds of activity, after which the tempo of maturity is no longer sensitive to conditions conducive to growth.

Of course, behavioural differences between men living in London and Sylhet may prompt greater acquisition of muscle at younger ages and retention into late adulthood. Any potential behavioural difference is not accompanied by an increase in salT implies the opposite conclusion: that environmental conditions in the UK somehow increase the sensitivity of muscle cells to testosterone. In either case, the strength of the link between axial muscle and testosterone appears to be subject to the environment in which an older male lives.

Measures of current dietary and health behaviours appear to explain much of this variation, as salT differences between sedentees and youth migrants or British-born Bengalis are no longer significant, once these factors are taken into account. This does not necessarily mean the current diet and health behaviours of these groups is the cause of variation in reproductive function in these men, as presumably these adult behavioural patterns reflect developmental experience. The change in diet between migrants and sedentees reflects developmental experiences, with a departure from consumption of vegetables among all Bengalis resident in London. One of the most significant explanatory dietary variables for salT was consumption of rice. Vegetarians and Asian men have higher plasma levels of SHBG and slightly lower levels of free T than men on Western diet (Key et al. 1990; van Houten and Gooren 2000; Zitzmann and Nieschlag 2001). The adoption of a Western diet high in fat and protein, compared to Asian diet higher in vegetables may influence reproductive physiology as plant compounds are reduced by gut microflora to phytoestrogens, chemicals resembling steroidal oestrogens in structure or function and able to bind to estrogen receptors (van Houten and Gooren 2000).

Dietary effects upon free T are typically very small though, and a direct causal link between rice consumption and salT seems unlikely to explain all the variance attributed to early life conditions. Rice consumption as a variable more plausibly represents correlated acculturation or ecological exposure over the lifespan in that it mirrors developmental age at migration for youth migrants and was much lower in British-born Bengalis, while rice consumption was only slightly lower for adult migrants compared to sedentees.

The measures above were analysed before taking SES into full consideration in chapter five. The creation of an SES index revealed youth migrant and British-born Bengalis to be of significantly higher SES than adult migrants, though they were matched to low SES British-born Europeans. From these findings, the SES of the men sampled in the London Bengali community were not homogeneous. A

re-examination of the developmental hypotheses tested in chapter three with the composite SES index included in the analysis suggests that ecological conditions aside from SES also determine adult reproductive function in the London Bengali population.

The differences in SES between British-born and migrant Bengalis who arrived before the age of 12 and all migrants arriving after age 12 may reflect a cohort effect of the previously documented changing SES of London Bengalis (Sunder and Uddin 2007).

The height of youth migrants and British-born Bengalis supports the general assumption of this study: that conditions in the UK are more conducive to childhood growth than Bangladesh. Migrating prior to age of maturity leads to taller adult standing height, but the limits of this plasticity is shown by the failure of youth migrants to grow as tall as Bengalis born in the UK. While actual measurement of leg length was not collected in this project, the lack of differences in arm length indicates this is a more canalised trait, suggesting femoral length or other long bones of the legs are more responsive to improvements in childhood environment than humeral length. Whatever the conditions conducive to growth in childhood, they do not appear to allow for increased reproductive function in males if they are experienced after maturity. Presumed conditions of ecological stress from pathogens or other environmental risks do not appear to constrain adult sedentee reproductive function when comparing measures of salT or (in men over 40) skeletal muscle to adult migrants.

The difference between the older males hints at the importance of relative social positioning for men in the latter half of their adult lives. While much has been made of the potential links between relative status and health outcomes at older ages (Marmot 2006) (Wilkinson and Pickett 2009), this is the first study to my knowledge to contrast testosterone levels of men of living at high SES in a poor nation with men of the same heritage living at a low SES in a wealthy nation. The link between T and dominance is well-established in primate and other mammalian research, (Muller and Wrangham 2004; Rose et al. 1971), but is less clear in human hierarchies (Book et al. 2001; Dabbs and Morris 1990; Mazur and Booth 1998).

One major confounding feature of the research on human hierarchies is the difficulty in determining which social positioning is of greatest importance (Sapolsky 2004). In the case of migrants moving to the UK from Sylhet, the Londoni are considered wealthy and they form a powerful economic class. Money from London has led to a proliferation of shopping malls, hotels and apartment buildings in Sylhet. The Sylheti middle classes from which this sedentee population was recruited is broadly reliant upon money from London either directly in the form of remittances,

or indirectly through the management and rental income of properties owned by their Londoni relations. The findings of this study suggest the influence of SES upon adult male reproduction may correspond to relative positioning in a society, much like other measures of health outcomes. It would appear that the influence of status at older ages is not reliant upon the gauge of success or affluence in early life, or by one's extended kin group, but is reflected by immediate relative social positioning.

Low SES relative to surroundings appears related to low salT in older males over the age of 40, while the salT and muscle mass measures of older men of high relative SES are not lower in the second half of the reproductive phase. This has some resemblance to the well documented "victory effect" in humans (Booth et al. 1989; Mazur and Booth 1998), where after a competitive interaction, the victor retains a high level of testosterone, while that of the loser declines. While there was not a direct link between being in the high or low SES group among the Sylheti sedentees, this was a within-population variable, and by not by indices of wealth in Bangladesh. The sedentees are an affluent group in a very poor nation, and this may influence perceptions of status. In contrast, migrant neighbourhoods abut the financial district of London and the Bengalis historically have lived in conditions of poverty within one of the wealthiest cities in Europe. The SES indices of the migrants included those of their rich London neighbours, and thus give a more accurate measure of their positioning relative to the surrounding population. This means the comparisons between the salT of low SES adult migrants >40 with sedentees is likely to accurately reflect the influence of SES.

The lack of a paternity or marital effect among Bengalis in London or Sylhet suggests the mechanisms of reduction in reproductive function observed in relation to paternity or marital status do not apply to Bengalis. This is in contrast to the significant difference observed in European males, though the direction of the effect was the opposite of predictions. This may indicate the success of older males with high salT within a low SES group to attract and retain partners, as opposed to younger males with high salT. The tradition of marital arrangement in the Bengali community may not allow for such variations in salT to correlate to partnership status and mate-seeking competition to the same degree as observed in Western populations.

While the differences in morning salT were greatest in young men, there was not a consistent pattern of the overall degree of daily decline. The lack of a consistent pattern between diurnal change in salT and residence group does fit with findings which suggest the morning peak and daily decline frequently observed in clinical studies (Diver et al. 2003) is less predictable or regular in non-Western populations (Vitzthum et al. 2009) (Bribiescas and Hill 2010).

There has been speculation that morning testosterone peaks are more reflective of physiological “set points” and evening levels more reflective of daily social and ecological interactions, but the residence groups with the greatest morning salT also had the greatest evening levels, so are either subject to daily environmental influences which promote high salT, or the rate of decline is not more flexible in men with higher salT.

Ultimately, the adjustments to human male reproductive function presented in this thesis are developmental responses to environmental perturbations, and are ultimately the product of evolutionary processes. The males in this study respond to contrasting ecological conditions in order to balance the competing biological functions of survivorship and reproductive effort. Natural selection will have favoured phenotypic matching to ecological conditions based on cues received during growth (within maternal constraints), and when the environment improves or becomes less stochastic, phenotypic characters with plasticity will correspond with a more matched phenotype.

From the perspective of life history theory, a developing male relies upon: a) inherited information on historical conditions to: b) interpret indicators of current ecological conditions in order to: c) adopt a fitness-maximising reproductive strategy between stability and responsiveness to changes in ecology. In a sense, the HPT axis (and measures of reproductive function) could be thought of like a thermostat, a reaction norm (Stearns 2000) where the range of calibrations on the dial are set by conditions during development.

Without wishing to caricature human prehistory (Lee 1968), the Hobbesian description of a life “nasty, brutish and short” (Hobbes 1651) neatly cleaves how a developing male assesses current conditions, based on evolutionary history to optimally interact with future conditions. A male must make life-history allocations based on estimating whether ecological conditions are likely to be nasty or nice, i.e. will the future ecology be limited in resources and high in pathogens or will resources be relatively abundant and stable? A male must estimate whether life will be brutish or hospitable, based on the levels of male-male cooperation or competition and an individual’s position in social hierarchies. Finally, a male must make allocations based on forecasting whether his life will be short or long, as male lifetime reproductive opportunities may be spaced over the entirety of a long adulthood or only immediately following maturity (see figure 7).

Ecological cues and constraints experienced prior to birth and during infancy and childhood development were hypothesised to influence reproductive function of men during adulthood. If males have a limited period of time when the basic construction of a body must be completed, they will be subject to the constraints of this construction. Thus a male who is born under circumstances of environmental

depletion will not be able to modulate his physiology to the same degree as male who was born in environmental affluence, if both men are in an environment of affluence, post maturity. Organisational effects of foetal sexual differentiation, such as formation, density and number of Leydig cells, or migration of stromal cells of the prostate (Bierhoff et al. 1997) will also determine lifetime reproductive functional capacities and regulation.

Within genetically homogeneous populations living in two ecologies, energetic conditions in Sylhet are interpreted by a developing male as “poor” and conditions in London are classified as “affluent”, thus males were predicted to respond to a change from poor to affluent conditions by maximising potential net lifetime reproductive opportunities.

The findings support this assumption that migrants respond to an improvement in conditions with increased growth, earlier maturity, and increased investment into adult reproductive function. The degree to which men respond to such changes is limited by developmental timing. The later this change occurs in life, there greater the mismatch between adult ecological conditions and hormonal thresholds of reproductive activity, as measured by salT and prostate symptoms. Males will discount maximal investment in reproductive function in early life with earlier senescence during late adulthood. A male who is under circumstances of plenty, relative to childhood condition, will show more persistently raised levels of T throughout the day, though the evidence for males who migrate at later ages showing signs of a maximised reproductive effort with a blunted diurnal salT profile was not found.

The early signs of poor prostate health and glucose metabolism among the youth migrant and British-born Bengalis may represent a negative outcome of the shift to more affluent conditions as compared to men who reached adulthood in Bangladesh, and the adoption of a lifestyle of maximal competition.

In the allocating of reproductive effort toward competition versus investment in current pair-bonds or offspring there will be a point where the risk of death, injury or loss of status associated with failed competitive encounters will outweigh the value of investing into current offspring (Kaplan and Lancaster 2003; Low 2001; Trivers 1972). While there was not clear evidence of such a trade-off in Bengali men, the nature of pair-bonds and paternal investment in a culture where older male relatives arrange marriages may mean the trade-off does not occur at marriage or with small children. Potential future research might investigate whether the trade-off is actually one generation removed, with the hypothesis that Bengali men modulate T in relation to whether their offspring have married or not, as competition for a desirable marital arrangement for offspring may be of

greater importance to male reproductive fitness in a society where men have little influence over their own marriage.

The relationship between SES and older males fits with a pattern where relative positioning is of greatest importance in later life. Older males of high SES have advantages, particularly if there have been high rates of male mortality, then it is about timing of male reproduction. Males must make a forecast of the best strategy of timing any challenging until they have amassed the appropriate amount of resources.

Limitations and future directions

The interpretation of these findings should be considered in light of potential limitations due to a cross-sectional design which cannot fully correct for cohort or demographic factors. The first generation of migrants from Bangladesh may originate from men selectively more or less likely to invest in reproductive effort and growth. Longitudinal studies suggest that migrants have alternatively been identified as more or less healthy than sedentees, as measured by height, weight, blood pressure and immune function, though Polish migrants from a high SES home community are less likely to show selective bias than those from low SES groups (Zielińska 1991). All sedentees and migrants were of a relatively homogenous, and high SES group within Bangladesh. Potential selection bias due to genetic factors was reduced by recruiting from men with immediate relations who had migrated to Western nations (UK, Europe, USA, Canada, Australia or New Zealand). However, within-family variation in motivations or traits that lead to migration cannot be ruled out completely when interpreting the results of this study.

If there were a selection bias in the migration from Bangladesh to the UK, one would hypothesise that men with higher T or men who had more investment in childhood growth (as measured by taller stature) would be more motivated toward risk-taking behaviour like migration than their other family members. However, there was no difference in salT between adult migrants and sedentees <40 years of age, and for men ≥40 years, the difference was in the opposite direction of the prediction, with sedentees showing higher salT than their migrant counterparts. Also, taller men did not migrate, as the adult migrants were not significantly taller than sedentees.

Youth migrants who arrived in the UK since 1980s usually arrived for the purpose of family reunification, which reduces the degree of potential selection bias. Of course, parents may have chosen only to bring the healthiest or most outgoing boys (presumably those with higher T) with them, but any selection within this group did not lead to different measures of salT from the British-born Bengalis.

In a comparative population, an analysis of South Asians living in Glasgow did not find evidence of a positive selection bias for height among migrant children (Shams and Williams 1997).

Height differences between migrant groups might be due to a secular trend in increasing stature, in such a way where recent migrants might be taller than those who migrated 20 or more years ago. However, any recent trends in height would be expected to have a negative relationship with age within residential groups, and there was not a significant trend between height and age within any of the groups aside from youth migrants (figure 11). In the case of youth migrants, there was not a significant relationship between height and age when separating the older men (≥40 years) from the younger men, so differences within these cohorts would not appear to be subject to a secular trend. Men would have arrived before 1983 if they were under 18 at the time of migration and be over 40 at the time of recruitment (2005 or later). Potentially, conditions for children who migrated prior to the early 1980s were significantly less conducive to growth in stature, but this trend is not observed in Bengalis born in the UK, and growing up during a similar historical period and ecological conditions.

The skinfold calculations are not without limitations, in comparison to more objective clinical measures such as CT scanning, there is a 1-5% error in tissue estimates (Martine et al. 1997). As comparison, bioelectrical impedance estimated error of total fat free muscle in individuals with normal electrolyte balance is 3.5-6% (Kyle et al. 2004). Also, the estimates of muscularity were only based upon one region of axial apportionment, and could be subject to variation due to active training or exertion.

Sample size limits the analysis and conclusions based upon age at migration in youth migrants, particularly those who arrived before age 2 (N=6). The lack of a correlation between infant migration and adult salT or other reproductive parameters is inconclusive with a sample at this size. A post hoc calculation for ANCOVA with a specified significance value ($\alpha = 0.05$), and a conventional "medium" effect size (Cohen's "f" = 0.25) specifying N=33, 3 groups (infant, child, adolescent) and two covariates (age, BMI) returns a power of $1-ß = 0.10$, indicating the limits of any conclusions to be drawn from null results within this residence group (Faul et al. 2007). An obvious expansion of this project or further research would be to collect more data from men who arrived in the UK as infants and children, in order to increase the power of any analysis and potentially further discriminate between developmental milestones by dividing childhood age at migration into smaller subgroups.

A MIGRATION STUDY OF DEVELOPMENTAL EFFECTS ON REPRODUCTIVE HORMONE LEVELS

Department of Anthropology, University College London

DEMOGRAPHIC AND HEALTH QUESTIONNAIRE (MALES)

CONFIDENTIAL
NOTES TO INTERVIEWER:
- Please use the words or similar phrases to the patient when you see this mark ♦ at the start of some of the questions.
- Tick in the boxes if appropriate like this (√)
- Please do not write beyond the margin on the right.

ID NUMBER -

Date of interview: / /
Language of interview:_____
Recruitment place:_____
Refered by:_____
Interviewer's initials:_____
Coded by:_____
First entry by:_____

♦ *Thank you for taking part in this study. We would like to stress that your answers will be treated in strict confidence. All the information you provide will be only for statistical analysis. It will not be possible to identify individuals in any results.*

♦ *We would like to ask some general questions about yourself first.*

I. PERSONAL INFORMATION

I.1 What is your full name?……

I.2 What is your home address?……

……

……

I.3 What is your home telephone number?
……

Work Tel No (if applicable?) …… Mobile……..

Email (if applicable?)
……

I.4 Can you give us a permanent address/ telephone where we can locate you if future follow-up is necessary?
……

……

I.5 Do you know your exact date of birth?
( ) Yes
( ) No
If YES, what is it?: ……Day/Month/Year
IF YES, go to question 1.7

I.6 If not, were you born before, on or after Shadinata Judu (1971)?
( ) before
( ) the year of Shadinata Judu
( ) after
If before, what school class were you in during Shadinata Judu?……..……..

If after, during which of the following periods were you born?
( ) Mujib Period (1971-1975)
( ) Zia Period (1976-1981)
( ) Ershad Period (1982-1990)

I.7 Where were you born? (Country, Village/City/Town/District)

……

I.8 Is your birthplace:
( ) rural
( ) urban

I.9 Did you grow up in a different place than where you were born?
( ) yes
( ) no
If yes, please indicate the place(s) where you grew up (Country, Village/City/Town/District)

……

……

I.10 Where was your mother born? (Country, City/Village/Town/District)

……

I.11 Where was your father born? (Country, City/Village/Town/District)

……

I.12 What is your marital status?
( ) single, unmarried *(if never married skip to section II)*
( ) married
( ) separated
( ) divorced
( ) widowed

I.13 If ever married, how old were you at first marriage? ……..

Please list the dates and your age at all previous marriages, if applicable.

I.14 Date of marriage: ……dd/m/y Age at marriage:……

b. Date of marriage: ……dd/m/y Age at marriage:……

c. Date of marriage: ……dd/m/y Age at marriage:……

d. Date of marriage: ……dd/m/y Age at marriage:……

1.15 Could you please tell us who was regularly living in your household during the first 5 years of your childhood?
Please specify who (*If necessary, tick more than one)*

( ) not sure
( ) father ( ) mother
( ) brother ( ) sister
( ) grandmother ( ) grandfather
( ) aunt ( ) uncle
( ) other family members……

……

1.16 Could you please tell us who was regularly living in your household when you were 6-13 years of age?
Please specify who (*If necessary, tick more than one)*

( ) father ( ) mother
( ) brother ( ) sister
( ) grandmother ( ) grandfather
( ) aunt ( ) uncle
( ) other family members……

……

1.17 Do you have any siblings (brothers or sisters)?
( ) Yes
( ) No

If yes, please give their sex, and year they were born. (*If you are not sure what year they were born, please list how many years they are older or younger than you*)

	Sex	Year born
1.	( ) Brother ( ) Sister	
2.	( ) Brother ( ) Sister	
3.	( ) Brother ( ) Sister	
4.	( ) Brother ( ) Sister	
5.	( ) Brother ( ) Sister	
6.	( ) Brother ( ) Sister	
7.	( ) Brother ( ) Sister	
8.	( ) Brother ( ) Sister	
9.	( ) Brother ( ) Sister	
10	( ) Brother ( ) Sister	

II. MIGRATION

♦ *The questions in the following section concern the history of migration of your family and yourself. This will give us an idea of the type of environment you lived in at different stages of your life. If you were born in the UK, skip to question II.11*

II.1 If you were not born in the UK, in what year did you arrive in the UK?……..

II.2 How old were you when you arrived in the UK? ……..

II.3 Where did you live **before** coming to the UK? (Country, City/Village/Town/District)

……

II.4 Is that place:
( ) urban
( ) rural

II.5 What was your household's head's occupation before you came to the UK?

……

II.6 Who moved together with you at the time of migration? (*please tick all that apply)*

( ) wife ( ) father in law
( ) father ( ) mother in law
( ) mother ( ) sister in law
( ) sister ( ) brother in law
( ) brother ( ) son in law
( ) son ( ) daughter in law
( ) daughter
( ) other family members……..
( ) migrated without any family members

II.7 What was the purpose of your migration from Bangladesh? (*please tick all that apply)*
( ) economic
( ) education
( ) accompany family
( ) marriage
( ) other specify ……

II.8 Is/are there any other member(s) of your family living in Bangladesh?
( ) yes
( ) no

If yes, please specify who (*If necessary, tick more than one)*

( ) wife ( ) father in law
( ) father ( ) mother in law
( ) mother ( ) sister in law
( ) sister ( ) brother in law
( ) brother ( ) son in law
( ) son ( ) daughter in law
( ) daughter
( ) other family members……..

Where in Bangladesh do they live? (town/village/town/district)

……

II.9 Where did you live when first arriving in the UK? (city/town) ……..

II.10 What was your household's head's occupation when first arriving in the UK?

……

II.11 If you were born in the UK to Bangladeshi parents, in which year did **they** arrive in the UK?
Father …….. ( ) Don't know
Mother …… ( ) Don't know ( ) Not Applicable

III. SOCIOECONOMIC INFORMATION

♦ *The following section asks questions about yourself and your family. We only use this information to categorise health conditions. You will not be identified personally, and only members of the research team will have access to these papers. If you prefer not to answer a question, please tick the box "I'd rather not say" and skip that question.*

III.1 What type of accommodation does your household occupy?
( ) house
( ) flat
( ) other, specify……

III.2 Does your household own or rent accommodation?
( ) owns
( ) rents
If you rent, who is your landlord? ( ) I'd rather not say
( ) Council / Local authority
( ) Private landlord or letting agency

III.3 How many rooms do you have for use by your household?
Do not count bathrooms, toilets, halls or landings, or rooms that can only be used for storage such as cupboards.
Do count all other rooms, for example kitchens, living rooms, bedrooms, utility rooms and studies. If two rooms have been converted into one, count them as one room.

Number of rooms……..…… ( ) I'd rather not say

III.4 How many members are there *permanently* living in your household?
Please indicate their relationship to you and their ages: ( ) I'd rather not say

Relationship to you	Age

III.5 In your household, do you have a:

	Yes	No
a. Car		
b. CD player		
c. Washing machine		
d. Freezer		
e. Tumble dryer		
f. Central heating		
g. Video/DVD player		
h. Dishwasher		
i. Home computer		
j. Microwave		
k. Satellite/cable television		

III.6 Please tick your total household income in a month after tax? *(Please include the monies earned by all the members of your household, please include monies from benefits, salaries and pensions)*

( ) I'd rather not say
( ) less than £500 ( ) £1,500 – 2,000 ( ) £3,000 – 3,500
( ) £500-£1,000 ( ) £2,000 – 2,500 ( ) £3,500 – 4,000
( ) £1,000 – 1,500 ( ) £2,500 – 3000 ( ) £4,000 or more

III.8 Could you please specify the sources of your household income? *(Please tick all that apply)*

( )I'd rather not say
( ) Earnings from employment or self-employment
( ) State retirement pension
( ) Pension from former employer
( ) Child benefit
( ) Job-seekers allowance
( ) Income support
( ) Family credit
( ) Housing benefit
( ) Other state benefits
( ) Interest from savings and investments (eg. stocks and shares)
( ) Other kinds of regular allowance from outside your household (eg. maintenance, student grants, rent)

IV. EMPLOYMENT INFORMATION

♦ *We ask this information because there are often relationships between health issues and whether people are employed in or outside of the home.*

IV.1 Have you ever worked outside the home?
( ) yes
( ) no

IV.2 Have you ever worked from home (e.g. computing, book-keeping, etc.)?
( ) yes, please specify ……
( ) no

IV.3 What is your current occupation? *Include self-employment, volunteer work, retired)*

……

IV.4 If presently married, what is your wife's most recent occupation?

……

IV.5 What is/was your father's occupation?……

IV.6 What is/was your mother's occupation?……

V. EDUCATION INFORMATION

♦ *We ask this information because there are often relationships between the amount of education people have and their health in later life.*

V.1 Have you ever attended /are you attending school?
( ) yes If yes, please specify in which country ……..
( ) no
(if no, please skip to Question V.4)

V.2 What is the highest class in school/ college you have completed? Please also list qualifications (i.e. GCSE, A-Levels, university degrees, professional qualifications)

……

V.3 If presently married, has your wife ever attended school?

( ) yes If yes, please specify in which country ……..

( ) no *(if no, please skip to Question V.7)*

V.4 What is the highest class in school/ college your wife/partner has completed? Please also list qualifications (i.e. GCSE, A-Levels, university degrees, professional qualifications)

……

V.5 What is/was your parents first language?
( ) Sylheti
( ) Bengali
( ) English
( ) Other (please specify) ……

V.8 How proficient are you in your parents first language?

	Not at all	A little	Quite good	Fluent
Speaking				
Reading				
Writing				

V.9 How proficient are you in English?

	Not at all	A little	Quite good	Fluent
Speaking				
Reading				
Writing				

V.10 What language do you usually speak at home with your spouse or other adults in the house?
( ) mainly English
( ) mainly mother tongue, please specify ……
( ) English and mother tongue equally
( ) other language, please specify ……..

V.11 What language do you speak at home with your children?
( ) mainly English
( ) mainly mother tongue, please specify……
( ) English and mother tongue equally
( ) other language, please specify ……

VI. PHYSICAL ACTIVITY

♦ *In this section we ask some questions about physical activity, since this may have important effects on your hormone levels and your health.*

VI.1 Do you practice any sport/ do any exercise?
( ) yes If yes, how often (times/week)? ……
( ) no

VI.2 If yes, which type? (*If necessary, tick more than one)*
( ) swimming
( ) cycling
( ) work at a gym/exercise bike/ weight training
( ) aerobics/ keep fit/ gymnastics/ dance for fitness
( ) any other type of dancing
( ) running/ jogging
( ) badminton/ tennis
( ) squash
( ) exercises (e.g. press-ups, sit-ups)
( ) any other, please specify ……..

VI.3 Do you walk continuously for more than 20 min on a daily basis?
( ) yes
( ) no

VI.4 Do you have household help?
( ) yes, please specify ……
( ) no

VII. GENERAL HEALTH

♦ *The questions in the following section relate to your general health and the clinical history of your family. This information will provide a context in which to interpret the results about your hormone levels.*

VII.1 Do you have any long-term illness/ health problem/disability which limits your daily activities or the work you can do?
( ) yes If yes, please specify ……
( ) no

VII.2 When was your last visit to a GP/doctor?

……

VII.3 Have you had any illnesses in the last 6 months?
( ) yes
( ) no

If yes, what kind?

……

II.4 Have you been diagnosed with any intestinal parasitic infection in the past 6 months (e.g., amoebas, giardia, schisosomiasis/bilharzias)?
( ) yes
( ) no
If yes, what kind? ……..

VII.5 Do you suffer from any of these diseases?

Diabetes	( ) yes ( ) no ( ) don't know
Heart disease	( ) yes ( ) no ( ) don't know
Hyperlipidemias (High cholesterol)	( ) yes ( ) no ( ) don't know
Hypertension (High blood pressure)	( ) yes ( ) no ( ) don't know
Anaemias	( ) yes ( ) no ( ) don't know
Arthritis or rheumatism	( ) yes ( ) no ( ) don't know

Other (please specify)

……

VII.6 Is there any history of diabetes in your family?
( ) yes, if yes please specify who ……
( ) no
( ) don't know

VII.7 Is there any history of hypertension (high blood pressure) in your family?
( ) yes, if yes please specify who ……
( ) no
( ) don't know

VII.8 Is there any history of cardiovascular disease (e.g., heart attack, stroke) in your family?
( ) yes, if yes please specify who ……
( ) no
( ) don't know

VII.9 Is there any history of thyroid diseases in your family?
( ) yes
( ) no
( ) don't know
If yes, please specify which member of your close family has been affected and by which kind

……

VII.10 Is there any history of cancer in your family?
( ) yes
( ) no
( ) don't know
If yes, please specify which member of your close family has been affected and by which kind

……

VII.11 Have you had any operations?
( ) yes, please specify ……
( ) no

VII.12 Are you a vegetarian?
( ) yes
( ) no

If yes, please specify what type:
( ) vegan (no animal products)
( ) lacto-ovo vegetarian (no meat, fish, chicken, etc. but do eat dairy and eggs)
( ) other, please specify……

VII.13 Have you been on a special diet in the last 3 months?
( ) no
( ) yes
If yes, what were the reasons?
( ) to lose weight
( ) to gain weight
( ) low fat
( ) high fibre
( ) other, please specify the kind of diet and the reason……

VII.14 Have you lost or gained weight in the past 3 months?
( ) yes, please specify amount ……
( ) no

VII.15 Do you regularly take any nutritional supplements, vitamins or minerals?
( ) yes,
( ) no
If yes, please specify what kind ……

VII.16 Have you taken any medications during the past month?
( ) yes
( ) no
If yes, how many kinds (including homeopathy/kabiraji)

Name of medicine	Have you taken this medicine in the last 2 weeks? (Yes/No)
1	
2	
3	
4	

VII.17 Prostate Symptoms: *The following questions concern the health of your prostate gland. Please circle a number for each question.*

	Not at all	Less than 1 time in 5	Less than half the time	About half the time	More than half the time	Almost always
Incomplete Emptying Over the past month, how often have you had a sensation of not emptying your bladder completely after you finish urinating?	0	1	2	3	4	5
Frequency Over the past month, how often have you had to urinate again less than two hours after you finished urinating?	0	1	2	3	4	5
Intermittency Over the past month, how often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5
Urgency Over the last month, how difficult have you found it to postpone urination?	0	1	2	3	4	5
Weak stream Over the past month, how often have you had a weak urinary stream?	0	1	2	3	4	5
Straining Over the past month, how often have you had to push or strain to begin urination?	0	1	2	3	4	5

Night time	None	1 time	2 times	3 times	4 times	5 times or more
Over the past month, how many times did you most typically get up to urinate from the time you went to bed until the time you got up in the morning?	0	1	2	3	4	5

Quality of life due to urinary symptoms	Delighted	Pleased	Mostly satisfied	Mixed – about equally satisfied and dissatisfied	Mostly dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition the way it is now, how would you feel about that?	0	1	2	3	4	5	6

VIII. REPRODUCTIVE HISTORY INFORMATION

♦ *Some of the following questions will allow us to estimate how old you were when you went through puberty. We are interested in making comparisons between different generations and those boys who now go through puberty in the UK and not in Bangladesh, to see if this age might be earlier or later.*

VIII.1 Please list the sex and age of all your children:

1. Sex: …… Age……..
2. Sex: …… Age……..
3. Sex: …… Age……..
4. Sex: …… Age……..
5. Sex: …… Age……..
6. Sex: …… Age……..

……
……
……
……

VIII.2 Do you remember how old you were when your voice broke?
( ) Yes
( ) No
If you remember, how old were you?……

VIII.3 Do you remember how old you were when you first started to shave/developed facial hair?
( ) Yes
( ) No
If you remember, how old were you?……

VIII.4 Do you remember how old you were when you first started to develop pubic hair?
( ) Yes
( ) No
If you remember, how old were you?……

VIII.5 When you were going through puberty, did you have wet dreams (nocturnal emissions)?
( ) Yes
( ) No
If you remember, how old were you?……

VIII.6 Have you any fertility problems of which you are aware?
( ) yes If yes, specify ……
( ) no

VIII.7 Have you ever been diagnosed with any diseases that may impair your fertility (e.g. syphilis)?
( ) yes If yes, specify ……
( ) no

VIII.8 Have you **ever** used any contraceptive method?
( ) yes
( ) no
If yes, what type?
( ) condom
( ) rhythm
( ) withdrawal
( ) herbs
( ) abstinence
( ) other……

VIII.9 Were **you** born:
( ) at home
( ) at a hospital/clinic
If born at a hospital/clinic, please state the name and place (city/town) ……

VIII.10 Were you born prematurely?
( ) yes If yes, how many weeks ……
( ) no
( ) do not know

VIII.11 Do you know your birth weight?
( ) yes If yes, please indicate ……
( ) no

VIII.12 The information about your birthweight is very important for the interpretation of the results of this project. We would like to ask your permission to track your birth records from NHS and/or Hospital archives. We will be specifically requesting information on birthweight and if possible, birthlength and head circumference from your records. Would you agree to this?
( ) yes
( ) no

If yes, we would need your mother's and father's full name to be able to access your records. Can you give us:
your mother's full name?
……..
And your father's full name?
……..

IX. SOCIAL AND LEISURE ACTIVITIES
♦*This section will ask some general questions on your social activities which will be used for comparative purposes between different groups.*

IX.1 What ethnic background do **most** of your friends have?
( ) Bangladeshis
( ) Pakistanis
( ) Indians
( ) English
( ) Black caribbean
( ) Black african
( ) Chinese
( ) other, please specify ……

IX.2 How often do you have tea/ meals over at a relative's/friend's homes?

……..

IX.3. Do you ever eat meals in an English (white) home?
( ) yes If yes, how often? ……..
( ) no

IX.4 What do you commonly do for recreation in your free time?

……..

……..

♦ *Thank you very much for your participation and support in helping us with this study. We greatly appreciate your help and hope you have felt able to answer the questions without too much trouble. The information you have given will help us study what factors may influence hormone levels and how these may affect women's health.*
If there is anything further you would like to add or comment upon please let us know.

Comments/Observations:
……

……..

……..

……..

……..

A MIGRATION STUDY OF DEVELOPMENTAL EFFECTS ON REPRODUCTIVE HORMONE LEVELS
Department of Anthropology, University College London

FOOD HABITS QUESTIONNAIRE (MALE MIG)

CONFIDENTIAL

NOTES TO INTERVIEWER:
- Please use the words or similar phrases to the patient when you see this mark ♦ at the start of some of the questions.
- Tick in the boxes if appropriate like this (√) If the question is not applicable, please cross the number off.

ID NUMBER .

Date of interview: / /
Language of interview:_____
Recruitment place:_____
Refered by:_____
Interviewer's initials:_____
Coded by:_____
First entry by:_____

♦ *Thank you for agreeing to take part in this survey. The following questions relate to your usual eating habits. Please answer all the questions as fully as you can. The information will be held in strict confidence.*

I. FOODWAYS

I.1 How many meals do you normally eat a day……..

I.2 How many nasta/snack breaks do you normally have a day……

I.3 Do you always eat Halal?
( ) yes
( ) no

I.4 Which type of home-cooked food do you eat at home?

Food type	Daily	Monthly	Only on special occasions	Never
ASIAN Main meals				
ENGLISH main meals				

I.5 What type of English (i.e. non-Bangladeshi, "Western") foods do you eat the most?

……

……

I.6 Do your children (or children in your household) eat different meals or food from yours?
( ) yes
( ) no
If yes, what type of food do they eat?
……

……

I.7 Do you ever eat outside the home? If so, where, what type of food, and how often?

			How Frequently?				
Type of place	Type of food	Which meal? (Lunch, Dinner, etc.)	Daily	Weekly/ Fortnightly	Monthly	Less than once a month	Never

I.8 Who does the shopping for food at home? ……

I.9 Where is the shopping is normally done?……

I.10 How far do you normally travel when shopping for food?
……

I.13 Do you buy food in bulk?
( ) yes
( ) no
If yes, what type and how often?

Type of food	Weekly	Fortnightly	Monthly	Less than once a month

I.14 What is the most expensive food item on your shopping list that you buy regularly?

……

I.15 Do you buy any imported foods from Bangladesh?
( ) yes
( ) no
If yes, what types?

……

……

……

If you migrated from Bangladesh:
( ) not applicable (*if born in the UK, please skip to section II*)

III.1 Which foods do you eat **more** of in England than when you lived in Bangladesh?

……
……
……
……
……

III.2 Which foods do you eat **less** of in England than when you lived in Bangladesh?

……
……
……
……
……

II. NON-FOOD HABITS

II.1 Do you ***currently*** chew betel nut (paan)?
( ) yes
( ) no (*if no, please skip to question II.5*)

If yes, how often do you chew?

More than 3 times/day	3 times/day (after each meal)	Less than 3 times/day	On weekends	On special occasions (e.g., weddings)

II.2 How old were you when you started chewing betel nut?……

II.3 Why did you start chewing betel nut?

……

……

II.4 What ingredients do you put into your betel quid (e.g., lime, spices, etc)?

……

……

II.5 Have you ever chewed betel nut (paan), and now given up?
( ) yes
( ) no (*if no, please skip to question II.11*)

If yes, how often did you chew?

More than 3 times/day	3 times/day (after each meal)	Less than 3 times/day	On weekends	On special occasions (e.g., weddings)

II.6 How old were you when you started chewing betel nut?……

II.7 How old were you when you stopped?……

II.8 Why did you start chewing betel nut?

……

……

II.9 If you no longer chew betel nut, why did you stop?

……

II.10 What ingredients did you put into your betel quid?

……

……

****

II.11 Do you ***currently*** smoke cigarettes?
( ) yes
( ) no (*if no, please skip to question II.15*)

If yes, how often do you smoke?

More than 3 times/day	3 times/day (after each meal)	Less than 3 times/day	On weekends	On special occasions (e.g., weddings)

II.12 How many packets of cigarettes per week (20 in pack)? ……

II.13 How old were you when you started smoking? ……

II.14 Why did you start smoking?

……

……

II.15 Have you ever smoked cigarettes, and now given up?
( ) yes
( ) no (*if no, please skip to question II.21*)

If yes, how often did you smoke?

More than 3 times/day	3 times/day (after each meal)	Less than 3 times/day	On weekends	On special occasions (e.g., weddings)

II.16 How many packets of cigarettes did you smoke per week (20 in pack)? ……

II.17 How old were you when you started smoking? ……

II.18 Why did you start smoking?

……

……

II.19 How old were you when you stopped?……

II.20 Why did you stop smoking?

……

II.21 Do you ever drink alcohol?
( ) yes
( ) no (*if no, please skip to Section III*)
If yes, which type of alcohol and how often?

		How frequently?		
Type of drink	Number of drinks	More than once a week	Less than once a week	On rare occasions

III. SEMI- QUANTITATIVE FOOD FREQUENCY QUESTIONNAIRE

♦ *We would now like to ask you how often you eat certain kinds of foods.*

III.1 Do you ever eat Bangladeshi foods that you buy frozen? (e.g., imported fish, meat, etc)
( ) yes
( ) no

If yes, what type and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.2 Do you ever eat English foods that you buy frozen? (e.g., vegetables, fish, meat, fruit)
( ) yes
( ) no

If yes, what type and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.3 Do you ever eat tinned food? (e.g., baked beans, mango, sardines, or tuna?)
( ) yes
( ) no

If yes, what type and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.4 Do you ever eat cold meats (e.g., turkey or chicken slices, sausages)?
( ) yes
( ) no

If yes, what type of cold meats and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.5 Do you ever eat packaged, prepared foods (e.g., quiche, pizza, ready-meals like chicken Tandoori from supermarkets, etc.)?
( ) yes
( ) no

If yes, what type and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.6 Do you eat fish fingers, burgers, chicken nuggets (bought ready-made and just heated up at home)?
( ) yes
( ) no

If yes, what type and how often?

Type of food	Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.7 Do you ever eat *roti*?
( ) yes
( ) no

If so, how often?

Daily	Weekly/Fortnightly	Monthly	Less than once a month	Never

III.8 Do you eat bread?
( ) yes
( ) no

If yes, which type of bread do you eat?
( ) white
( ) brown
( ) wholemeal
( ) other ……

III.9 About how many pieces of bread/roti/chapatti/naan do you eat on a usual day?
( ) 5 or more a day
( ) 1 to 2 a day
( ) none

III.10 What do you normally eat for breakfast?

……

……

III.11 How often, on average, do you eat rice?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

III.12 How often, on average, do you eat noodles/pasta?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) less than once/week
( ) Rarely or never

III.13 How often on average do you eat potatoes?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.14 How often, on average, do you eat chips (french fries)?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.15 How often, on average, do you eat lentils (dhal)?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

III.16 How often, on average, do you eat other pulses (e.g., beans, baked beans, chickpeas)?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.17 How often on average, do you eat vegetables in curry?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

If yes, what type of vegetables?

……

……

III.18 How often do you eat boiled or steamed vegetables?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

If yes, what type of vegetables?

……

……

III.19 How often do you eat green salad?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

III.20 How often on average do you eat tomato, cucumber, and onion salad?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

III.21 How often do you eat fruit?
( ) with every meal
( ) twice daily
( ) once a day
( ) 2-3 times/week
( ) less than once/week
( ) Rarely or never

If yes, what type of fruit?

……

……

III.22 How often do you drink freshly squeezed fruit juice?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

If yes, what type?

……

III.23 How often do you drink packaged fruit juice (concentrated)?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

If yes, what type?

.....

III.24 Do you drink squash (e.g., Ribena or similar)?
( ) yes
( ) no

If yes, what type?

.....

III.25 How often, on average, do you eat cheese
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

If yes, what type(s)?

.....

.....

III.26 How often do you eat yoghurt/lassi?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.27 Do you ever eat cream or other dairy products (e.g., cream cheese, crème fraiche, fromage frais, etc)?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

If yes, what type(s)?

.....

.....

III.28 How often do you eat chicken
( ) daily

III.29 How often do you eat turkey?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.30 How often do you eat beef?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.31 How often do you eat lamb?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.32 How often do you eat fish?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

If yes, what type?

.....

.....

III.33 How often do you eat seafood?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.34 How often do you eat eggs?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.35 How often do you eat fried food?
( ) daily
( ) 4 to 3 times/week

III.36 How often, on average, do you eat savoury snacks (e.g.,crisps, savoury biscuits such as cream crackers, muri, chanachur, Bombai mix, etc).
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.37 How often do you eat peanuts, pistachios or other kinds of nuts?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.38 How often do you eat sweet snacks or desserts?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.39 What type of oil/fat do you use for cooking?

……..

III.40 During mealtimes, what do you normally drink?

……..
……..

III.41 How often do you drink fizzy sweetened drinks (eg. Coca cola, pepsi lemonade)?
( ) daily
( ) 4 to 3 times/week
( ) 1 to 2 times/week
( ) Less than once/week
( ) Rarely or never

III.42 Do you drink milk (as a beverage separately from cooking etc)?
( ) yes
( ) no

III.43 Do you eat cereal with milk?
( ) Yes
( ) No

III.44 What kind of milk do you drink?
( ) cow's milk
( ) goat's milk
( ) soya milk
( ) other, please specify……

III.45 Is the milk:
( ) whole
( ) semi-skimmed
( ) skimmed

III.46 Hot Drinks

Do you drink?	Do you add sugar?	Do you add milk?
tea yes ( ) no ( )	yes ( ) no ( ) *if yes, number of spoonfuls……..*	yes ( ) no ( )
coffee yes ( ) no ( )	yes ( ) no ( ) *if yes, number of spoonfuls……..*	yes ( ) no ( )
Other hot drink ( ), please specify……	yes ( ) no ( ) *if yes, number of spoonfuls……..*	yes ( ) no ( )

III.47 Do you add salt to your meal at the table?
( ) yes
( ) no

III.48 Do you often eat the same food for dinner as for lunch (leftovers)?
( ) yes
( ) no

Observations/Comments:

……..
……..
……..

♦ *Thank you very much for your participation. We greatly appreciate your help and hope you have felt able to answer the questions without too much trouble.*

Appendix 3: Salivary testosterone assays: methods and results from Magid 2005

Methods

Salivary T assays were measured by radioimmunoassay without extraction. Antiserum was prepared in the Ob/Gyn Laboratory of Dr. Robert Chatterton at the Feinberg School of Medicine, Northwestern University, Chicago USA, where all analysis was performed. The interassay CVs were all within 15% for high (100pg/ml), low (50pg/ml), and internal (pooled saliva sample) quality controls. The sensitivity was 0.028 nmol/L. Salivary cortisol will be analysed using a similar radioimmunoassay technique, validated and used in the Chatterton lab.

Samples of salivary T measured so far have been analysed according to the following criteria:

Outlying points with readings over 500pg/ml were excluded as they fell outside the standard curve (highest standard=480pg/ml) and estimates beyond this concentration could not be accurately estimated. Readings were made in duplicate and all with a CV greater than 15% were re-analysed or excluded. Of 676 samples, a total of 36 of were excluded.

The overall degree of diurnal change in salivary T was expressed as a mean value at each time point over two nonconsecutive days, with the evening sample value subtracted from the morning value and expressed as a percentage. The morning sample was considered the peak level of T, as has been observed in numerous other studies (Campbell et al., 2005;Ellison et al., 2002) and can be considered a “top” baseline for the day.

All statistical analysis was conducted using SPSS for Macintosh OSX 11.0.

ANCOVA was used to model the relationship between anthropometric measures (as dependent variable) and residence group after correcting for age differences. ANCOVA was also used to model the relationship between muscle + bone area with of the number of years spent in the UK, where AI were subdivided into three groups indicating years since migrating: (<10 years, 11-40 and >40) and BR inserted as the dummy variable.

Results

Mean ages of the adult immigrants and Bangladeshi residents were significantly different. Controlling for age, salivary testosterone levels of adult immigrants

(AI) to the UK were significantly greater than those of Bangladeshi residents (BR) for morning (BR=201, AI=245 pg/ml) and evening (BR=154, AI=210 pg/ml) samples.

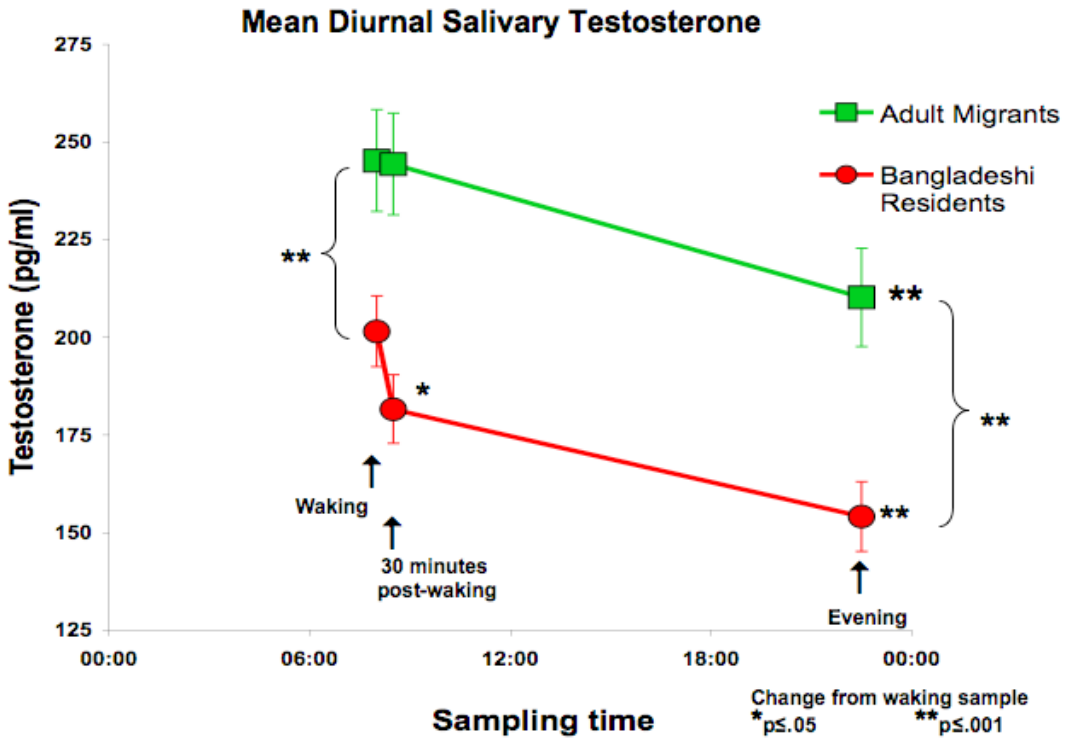


Figure 1: Mean Diurnal Salivary Testosterone Diurnal salivary T profiles for both residence groups showing significant difference between group mean waking (BR: 201.5 ±9.1, AI: 245.2 ±13.1 pg/ml) 30 minutes post-waking (BR: 181.5 ±8.8, AI: 244.3 ±13.1) and evening (BR: 154.1 ±8.9, AI: 210.2 ±12.6) samples and significant differences between morning and evening samples for both groups.

The immigrant group, but not the sedentee group showed a significant negative linear correlation between age and testosterone. The two groups did not significantly differ in BMI, but an ANCOVA correcting for both age and BMI effects still showed a significant difference.

Paired t-tests between residence groups revealed a diurnal rhythm in both AI and BR, with significant differences between group mean waking (BR: 201.5 ±9.1, AI: 245.2 ±13.1) 30 minutes post-waking (BR: 181.5 ±8.8, AI: 244.3 ±13.1) and evening (BR: 154.1 ±8.9, AI: 210.2 ±12.6) samples and significant differences between morning and evening samples for both groups (all $p<0.05$). BR, but not AI showed significant variation between waking and 30-minutes post-waking samples, suggesting a steeper decline in salivary T levels. The

degree of diurnal change in salivary T was not significantly different between the two resident groups, indicating a similar pattern of declining salivary T throughout the day, but from different baseline concentrations (see figure. 3).

In order to further assess the influence of age, participants were broken down into age brackets by decade. Immigrants at younger, but not older ages showed significantly higher levels of salivary testosterone when compared to sedentees (fig. 2). The diurnal profiles, when broken down by age categories showed a similar degree of daily decline across age groups, but from different baseline levels in the youngest category (20-29 years) (fig 2).

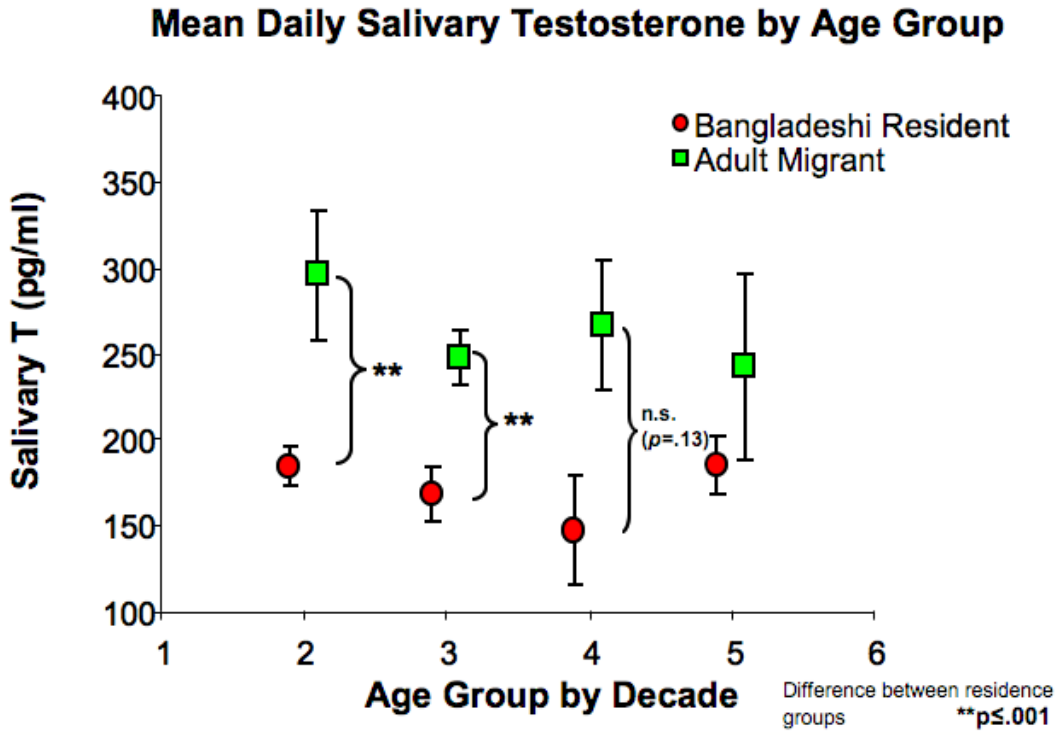


Figure 2: Age-related differences in salivary T between residence groups are significant in younger (ages 20-29: BR=184.1 ± 11.1 AI=295.6 ± 37.8; ages 30-39: BR=168.1 ± 15.3 AI=247.5 ± 73.2) but not older men (ages 40-49: BR=147.1 ± 55.4 AI=266.5 ± 37.5; ages 50-59: BR=184.8 ± 17.0 AI=242.4 ± 54.0).

An ANCOVA controlling for age showed a significant positive relationship between years since immigrating and pooled T in the AI group with years in the UK categorised as three subgroups (<10years, 11-40years, >40years, n=24, 19, 16 respectively) ($F(3, 59)=4.7$. $p<0.01$) the BR group was included as a dummy variable (indicating 0 years in UK). In a separate ANCOVA also controlling for age, years in the UK was predictive of total salivary T concentrations $F(5,109)=7.68$, $p<0.001$. A similar regression model with a selection of age subgroup of 20-49 year olds showed an even stronger association between

years spent in the UK and salivary T F(1,75)=15.02, p<0.001. The same modelling based on older men (age 50+) was not significant.

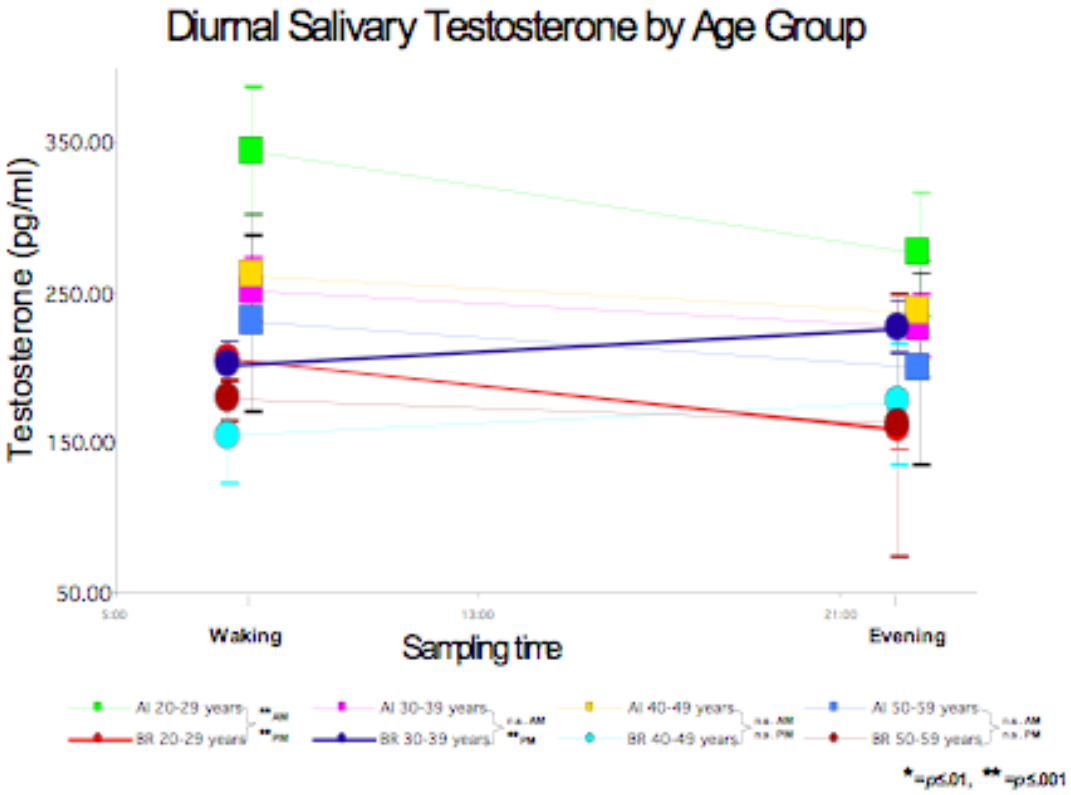


Figure 3: Diurnal Salivary Testosterone by Age Group: Comparison of daily testosterone profiles between residence groups, subdivided into 10-year age categories. (All comparisons independent t-test between AI and BR age subgroups). Both residence groups showed significant diurnal change in paired t-tests of mean waking (BR= 201.5 ± 9.1, AI=244.5 ± 13.0) and evening (BR= 154.1 ± 8.9, AI=210.2 ± 10.6) samples.

Appendix 4: Laboratory assay validation

Assay Method:

A competitive equilibrium for specific antibody sites is established at room temperature with either a testosterone standard or sample and 3H-T tracer. All standards and samples are run in duplicate, marked "**a**" and "**b**". The unbound steroid is removed from solution by addition of dextran coated charcoal and centrifugation at 4C. Bound radioactivity in the supernatant is measured by liquid scintillation. The amount of radioactivity is inversely related to the amount of T in the standard sample. A logit-log plot of percent bound radioactivity versus concentration of standards will give a linear dose response curve. Saliva concentrations are then calculated from the curve. If the duplicate samples have a CV over 15%, they must be re-assayed or not reported.

Quality Controls:

Pooled saliva samples, immediately aliquoted, stored unpreserved at -20C. A variation of 15% from the running mean of the previous ten assays is allowed. If any two QC parameters are outside of the acceptable limits, the assay will be re-run.

Codes for QCs used throughout:

Count values for the following QC references:
NSB: Non Specific Binding (raw count)
B0: Percent Binding

Dose values for the following QC references:
QC1: Stripped saliva, dosed to 100pg/ml T
QC2: Stripped saliva, dosed to 50pg/ml T
IQC: Internal QC made from a pool aliquot from numerous participant saliva samples
XQC: External QC made from a pool of male lab workers at Chatterton Lab
KQC 0: Saliva samples from male researcher (KSM) pooled over several days and frozen as aliquots
KQC Hi: Spiked KQC, diluted ½ with 480pg/ml T (Std7)
KQC Lo: Spiked KQC, diluted ½ with 7.5pg/ml T (Std1)
QC "**A**" were prepared and run at the beginning of the assay, immediately following stds
QC "**B**" were prepared and run at end of assay, last sample

Collected CV values for all assays, 2008

CVs for all Salivary T assays 2008

Date	XQC0 A	XQC0 B	XQC0 A&B	KQC0 A	KQC0 B	KQC0 A&B	KQC Hi	KQC Lo	>2 outside 15%?
25-Jun	13.42		13.42						
25-Jun									
27-Jun	14.59								
27-Jun	11.68	4.55	14.68	**18.94**	**27.24**	**19.15**	**19.24**	7.53	*
3-Jul	0.91	5.80	10.55	**16.19**	1.65	**38.98**	**15.86**	3.64	*
9-Jul	2.75	2.19	**39.79**	12.68	7.72	**45.35**	1.19	**22.62**	*
11-Jul	1.43	1.46	**33.54**	1.90	3.70	**30.13**	7.59	6.54	*
16-Jul	7.14	1.12	5.03	0.79	**20.23**	**18.48**	5.19	0.99	*
18-Jul	14.99	5.13	9.50	2.16	**21.27**	14.32	11.41	2.23	
21-Jul	1.87	3.24	2.18	3.73	**18.89**	**20.61**	3.77	**16.69**	*
22-Jul	**15.16**	0.86	8.78	16.08	1.79	13.78	0.88	11.30	
27-Aug	2.32		**15.35**	18.63	6.95	13.73	1.19	12.00	
27-Aug	2.01		13.32	16.02	6.02	11.90	1.08	9.88	
28-Aug				0.93	5.56	6.71	4.83	3.21	
29-Aug				5.48	6.81	8.10	1.72	**16.70**	
1-Sep				11.32	**16.91**	13.21	6.41	14.93	
24-Sep				31.01	5.57	7.63	**19.32**	**16.25**	*

6 Jun 05:
Aim: To measure effects of preparing standards at start of assay versus the end, to account for levels of reabsorbing of bound tracer from charcoal after spinning.

Methods: Standards and QCs A were the first prepared, bound to DCC and spun down. Stds and QCs B were the last to be prepared, bound to DCC and spun down.

All concentrations were calculated using a number of std curves:
Avg of A&B: unedited std curve of all avg counts of both A&B
Edited A&B: outlying points and Std1 were dropped from curve
Stds A: calculated values from curve based on Stds A
Stds B: calculated values from curve based on Stds A

Assay 4 Std Curves

Dose	Avg of A&B	Stds A	Stds B	Edited A&B
NSB	353.0	353.0	353.0	353.0
B0	2043.0	2043.0	2043.0	2077.5
T	3782.5	3782.5	3782.5	3782.5
7.50	1916.0	1878.5	1897.3	dropped
15.00	1905.7	1795.0	1850.4	2016.5
30.00	1885.7	1877.5	1881.6	1885.7
60.00	1788.0	1693.5	1740.8	1788.0
120.00	1645.8	1622.0	1633.9	1645.8
240.00	1365.0	1335.0	1350.0	1365.0
480.00	1017.8	994.5	1006.1	1017.8

Values returned

QC1a	23.25	13.81	42.05	45.92
QC1b	14.16	7.36	29.95	34.23
QC2a	22.55	13.30	41.15	45.08
QC2b	4.97	1.60	16.10	19.73
IQC A	97.16	74.91	124.86	115.50
IQC B	69.49	50.94	95.69	92.37

QC1

	QC1 A read a	QC1 A read b	avg A	cv A	QC1 B read a	QC1 B read b	avg B	cv B	intra assay avg A&B	intra assay CV A&B
Avg of A&B	30.84	16.61	23.72	**42.40**	14.74	13.60	14.17	5.71	18.95	**42.35**
Stds A	19.51	9.04	14.28	**51.86**	7.75	6.97	7.36	7.47	10.82	**54.15**
Stds B	51.55	33.31	42.43	**30.40**	30.75	29.15	29.95	3.76	36.19	**28.69**
Edited A&B	54.68	37.55	46.11	**26.27**	35.03	33.44	34.23	3.28	40.17	**24.44**

QC2

	QC2 A reading a	QC2 A reading b	avg A	cv A	QC2 B reading a	QC2 B reading b	avg B	cv B	intra assay avg A&B	intra assay CV A&B
Avg of A&B	25.19	20.05	22.62	**16.07**	8.83	1.93	5.38	**90.71**	14.00	**75.38**
Stds A	15.25	11.48	13.36	**19.92**	3.87	0.19	2.03	**128.09**	7.70	**89.48**
Stds B	44.51	37.89	41.20	11.37	22.24	10.54	16.39	**50.46**	28.80	**53.28**
Edited A&B	48.23	41.98	45.10	9.79	26.35	13.33	19.84	**46.42**	32.47	**48.45**

IQC

	IQC A reading a	IQC A reading b	avg A	cv A	IQC B reading a	IQC B reading b	avg B	cv B	intra assay avg A&B	intra assay CV A&B
Avg of A&B	86.91	108.16	97.54	**15.41**	68.11	70.88	69.50	2.82	83.52	**22.03**
Stds A	65.92	84.68	75.30	**17.61**	49.77	52.12	50.95	3.26	63.12	**25.41**
Stds B	114.22	136.10	125.16	12.36	94.20	97.20	95.70	2.21	110.43	**17.43**
Edited A&B	107.18	124.15	115.66	10.37	91.15	93.59	92.37	1.86	104.02	14.57

Results:
All intra-assay CVs were outside acceptable limits for QC1 and QC2 for all curves
Values returned for all curves were considerably below expected spiked values for QC1 and QC2
IQC was within acceptable limits for combined edited curve.

26-31 Oct 06
Aim: To establish working std curve from 2005 stock and newly prepared 2006 stock.

Methods: A small internal QC drawn from 15 samples was measured (SA06 QC

0), as well as a spiked QC (50% Std7). Concentrations were calculated from both curves.

Results:

	SA06 QC	SA06 QC Hi (50% Std7)	SA06 QC Lo (50% Std3)
Std assay 26Oct06	186.4817037	273.1397269	
Std assay 31Oct06	162.6423677	309.69	121.23

9-11 Nov 06
Aim: The first assay run with a new set of T standards on 8Nov06 seemed to show lower than expected QC values. This could be due to decay of T-As, so an old standard dosed at 60pg/ml was run and calculated according to the new curve. In addition, the old IQC used in 2005 was calculated using the new standards.

11 Nov 06: Three of the old standards (S1, S4, S7) were run at the end of the assay. In addition, the old IQC was run again.

Results: IQC was within acceptable CV from running avg for 2005 assays on both days

9 Nov: Old Std 4 had good recovery using the new standard curve: 63.12 pg/ml
11 Nov: Old Std 1 fell below limit of detection of assay
Old Std 4 had considerably lower recovery (approximately. 50% of expected value)
Old Std 7 recovery was within acceptable limits

Results:

9-Nov-06	reading a	reading b	avg	cv	running avg, 2005	cv from running avg
IQC	97.10	86.50	91.80	8.16	112.51	13.02
Old Std 4 (60pg/ml)	56.83	69.41	63.12	14.09		
11-Nov-06						
IQC	137.53	113.93	125.73	13.27	112.51	8.31
Old Std 1 (7.5pg/ml)	Below low std	Below low std				
Old Std 4 (60pg/ml)	26.15	33.32	29.73	**17.06**	60.00	**35.67**
Old Std 7 (480pg/ml)	411.33	438.37	424.85	4.50	480.00	8.13

23 Jun 08: Old Standards from November 2006 were run alongside newly made-up ones.

Results:
Both IQC & XQC were not significantly different when calculated using old or new standards:

	23/6 new stds	23/6 old stds	avg	cv
IQC	150.78	152.93	151.86	1.0
XQC	176.90	190.94	183.93	5.4

25 Jun 08
Aim: to determine if freezing and thawing significantly influences recovery of salivary T.

Method: Repeated freeze-thaws of XQC were run.

Results:
No significant difference in T for repeated freeze thaws (1-8 F/T cycles)

25-Jun						
	1 F/T	2 F/T	4 F/T	8 F/T	avg	cv
T (pg/mL)	89.92	105.08	111.90	107.43	103.6	9.2

Sample stability, 2005-2008
Aim: to measure stability of measured free T in multiple saliva samples from a single individual stored for 3 years at RT and +5C

Results:
Though some of the samples were outside the 15% CV, T levels were similar and did not appear directionally affected by period of storage.

Lab ID	Comparison of samples run in 2005 & 2008									
	Assay 2	3-Jun-05			Assay 19	3-Jul-08			Avg of 2005 & 2008	CV of 08&05
	reading a	reading b	avg	CV	reading a	reading b	avg	CV		
114	119.27	152.59	135.93	**17.33**	95.43	104.64	100.04	6.51	117.98	**21.25**
115					133.25	135.17	134.21	1.02		
116	134.50	133.29	133.90	0.64	127.39	117.11	122.25	5.95	128.07	6.20
117	131.29	111.86	121.58	11.30	87.12	89.43	88.27	1.85	104.92	**19.84**
118	135.72	155.67	145.69	9.68	174.12	157.62	165.87	7.03	155.78	10.10
119	120.03	108.97	114.50	6.83	94.69	84.25	89.47	8.25	101.99	**15.42**

Effects of different T-As, source stock, and std dilution
T10 Assay10

Objective:
To measure if there is significant variation due to age of standard preparation (2006 Lu stds, Dec 2009 Stds from 06 stock, Mar 2010 Stds from 06 stock, 2010 Lu standards from Lu 2006 5ug/ml stock)
To determine if inclusion of a different top (240 vs 480) and lower standard (7.5 vs. 3.25) significantly shifted recovery for a number of QCs.
To validate all previous assay assumptions of standard curve values and error by cross checking them on a number of other different curves.

Method: Newly made up standards Mar10 (from 1ug/ml NSB prepped Dec09, original stock of 1mg/ml prepped Oct06). Newly made up standards from Lu 5ug/ml stock prepped Oct06, compared with standards stored at 5c for 4 years.
Results:
All concentrations for standards made from 2006 stock gave readings by a factor of 0.7 pg/mL lower than expected values. All standards stored at 5c for 4 years gave readings a factor of 1.3 pg/mL higher than expected values.

Repeated measures and correction factors:
As salT was analysed over different years, a number of samples and QCs were measured repeatedly. These repeat measures were used to adjust readings between years of analysis. A total of 191 samples collected and analysed in 2005, 66 samples collected and analysed in 2006, and 17 samples collected and analysed in 2008 were measured again in 2010. From these repeat measures, a correction factor of 3.06 was applied to all other readings from 2005, and 0.84 was applied to all other readings from 2006. Repeat measures from 2008 assays did not require a correction factor to match 2010 readings.

Serum to salivary T comparisons
Method: a total of 22 participants were asked to provide a saliva sample according to the same collection procedure detailed for all samples collected in the field. Immediately following the provision of a saliva sample, a serum sample was collected. Two sample tubes were mislabelled and were therefore dropped from the final analysis.
Serum T was analysed by RIA using a kit from

Serum T assay QCs

QC	Expected	Read
I	0.65 ± 0.2	0.62
II	5.7 ± 1.7	5.02

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