Foetal Starvation, Economic Adversity and Health a Difference-in-Difference Approach

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Abstract The foetal origin hypothesis argues that starvation during the foetal stage increases the probability of the onset of non-communicable diseases in midlife. The theory, however, fails to identify the mechanisms underlying the outcome. Nor does it succeed in distinguishing between study and control groups. The predictive adaptive response theory addresses the former deficiency by hypothesising that nutrition supply at the foetal stage signals the future nutrition supply and leads to adaptation of the foetus to the future expected environment. Mismatch between expected and actual environment will increase the likelihood of non-communicable diseases. The study examines the long-term impact of foetal starvation on anthropometric indicators among residents in the Sundarban region in India. We hypothesise that nutrition deficiency in the foetal stage signals the future expected environment to the foetus. This leads to the growth of a thrifty phenotype ensuring optimal performance of the offspring in a nutrition-deficit environment. A primary survey, undertaken between May 2014 and April 2015, was used to collect the data. In the first stage of the survey, Muslim women who had offspring in the period 1993–1997 were listed. In the second phase, anthropometric measurements of their offspring were taken. Respondents are placed within the study group if their mothers had kept the Ramadan fast (provided it coincided with conception); remaining respondents were defined as controls. Differences in mean anthropometric outcomes are tested using Monte Carlo simulations. A Difference-in-difference method is also applied. Respondents exposed to foetal starvation had better outcomes than those in the control group if they remained in poverty or their economic status deteriorated. Results were reversed

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for children with a sustained high standard of living, or those whose economic conditions improve. Findings are interpreted to provide support for the predictive adaptive response theory. However, tests using larger samples are required before arriving at a firm conclusion.

Keywords Foetal origin hypothesis · Predictive adaptive response · Anthropometric outcomes · Monte Carlo simulations · Difference-in-difference · India

1 Background

In the late 1950s, epidemiologists believed that the placenta was a "perfect filter, protecting the foetus from harmful substances in the mother's body and letting through helpful ones" (Landro 2010). This belief was shaken by the Thalidomide episode in the 1950s and 1960s[1] and by the research of David Barker and his associates. The latter proposed that the nine months in utero was one of the most critical periods in a person's life, shaping future health trajectories, endowments and capabilities. Based on the finding that several chronic diseases associated with ageing may be programmed before birth, Barker proposed that the old model of adult degenerative disease, based on an interaction of genes and an adverse environment in adult life, should be replaced by a new approach based on "programming by the environment in foetal and infant life" (Barker 1990, p. 1111). This is called the foetal origin hypothesis (FOH).

Barker viewed the effects of foetal under-nutrition on adult diseases as side effects of foetal adjustments instigated to boost survival of the foetus. The permanent changes in the structure and the functioning of the body resulting from foetal starvation becomes disadvantageous later in adult life. Hales and Barker later published the "thrifty phenotype" hypothesis, proposing that adjustment of foetal growth rate might set nutritional expectations and thereby condition the organism's response to nutrition later in life (Hales and Barker 2001). The idea was subsequently expanded to argue that the mother provides signals about the future environment to the foetus who adapts accordingly (Bateson 2001; Bateson et al. 2004; Gluckman et al. 2005a, b, 2008; Gluckman and Hanson 2007). Bateson, for instance, argues that "… the pregnant woman in poor nutritional condition may unwittingly signal to her unborn baby that it is about to enter a harsh world." (Bateson et al. 2004: 420). This leads to the baby being born with adaptations that help it to cope in food-deficit environments. This is referred to as predictive adaptive response (PAR).

One important adaptation to the nutritional shortage is that the foetus will use most of the scarce energy for the most vital organs (particularly the brain) and their metabolism so that they will be protected against lack of fuel (Godfrey and Barker

[1] Thalidomide was licensed in 1957 (except in the USA) and widely prescribed to pregnant women for morning sickness, until 1961 when it was identified as the cause of an epidemic of severe birth defects such as missing arms and legs (Haymann 1962; Smithells 1962).

2001). This "brain sparing" may slow down cell division in organs undergoing their critical growth period. Other foetal adaptations consist of increased maternal concentration levels of the stress hormone CRH (corticotropin-releasing hormones). This prepares the foetus for an expected preterm delivery—foetal growth is reduced and tissue maturation accelerated (Hobel and Culhane 2003).

However, the signal received at the foetal stage may also fail to correctly anticipate the future environment. If individuals with a thrifty phenotype find themselves growing up in conditions of relative affluence, there will be an environmental mismatch. Changes that are adaptive under poor conditions, such as storing food as fat and maintaining high blood glucose levels, now become maladaptive under good conditions (Bateson et al. 2004; Gluckman et al. 2005a). In such cases, the costs of such adaptations emerge in the form of increased risks of coronary heart disease and its biological risk factors, such as hypertension and type II diabetes mellitus (Seckl et al. 1997; Godfrey and Barker 2001; Seckl and Holmes 2007; Van Abeelen et al. 2012).

While there is a substantial body of the empirical literature on the FOH, most of the works suffer from an identification problem. The lengthy time gap between the nutrition shock/signal and the manifestation of the outcome of interest creates an identification problem. Short of time travelling, it is not easy to obtain reliable data for *both* these time periods. This creates an identification problem (Paneth and Susser 1995).[2]

To solve this problem, early works either studied animals which mature rapidly or relied on proxies of foetal starvation. Both methods are flawed. Wells points out that human beings have longer periods of growth before attaining maturity the random component in the "future" environment increases. It is doubtful, therefore, whether signals received at either the foetal or other stages produce "a developmental trajectory (that) … is genuinely predictive of the environment experienced as an adult" (Wells 2006: 424). The use of birth weights, a commonly used proxy for foetal starvation, has also been criticised. In developing countries, mothers are short in general and produce stunted or underweight babies. Focus on simply the weight or height at birth, however, ignores the *thin-fat* syndrome, as such babies may have low skeletal muscle mass but high central adiposity (Yajnik 2004).

In response, economists have attempted to model starvation in terms of a national or regional shock (generally famines) occurring over a single period and affecting a single cohort. All persons in the cohort are assumed to be exposed to the in utero shock, and their health outcomes compared to preceding or subsequent birth cohorts (who are *all* assumed to be sheltered from any nutritional deprivation). Using the nineteenth-century blight to French vineyards caused by the phylloxera insect as a shock, a study found that prenatal exposure reduced the height of army recruits aged 20 years by 0.5–0.9 cm (Banerjee et al. 2010). Studies on pandemic influenza in Brazil (Nelson 2010) and Britain (Kelly 2009) also reported long-term health

[2]Douglas Miller refers to this as the science fiction problem in analysing the foetal origins hypothesis—sans time travelling, we have to wait for a generation for the effects of a prenatal intervention of interest to be observed in adulthood (cited in Almond and Currie 2011: 157).

impacts on children born to women during the pandemic; an adverse labour outcome was reported for American (Almond 2006) and Danish (Schwandt 2017) children. Increased risk of diabetes and cardiovascular disease was observed for children born during the Dutch famine at the time of German occupation (Ravelli et al. 1998) and in the course of the Leningrad siege (Stanner et al. 1997), respectively.

Another method that has been used in recent studies is to use fasting during the lunar month of Ramadan as a proxy for foetal shock (Almond and Mazumder 2011; Van Ewijk 2011). A study found that Muslim children exposed to Ramadan in early pregnancy have significant health effects, with the likelihood of adult disability increasing by over 20% among Iraqis and Ugandan Muslims; substantially larger effects are observed for mental and learning disabilities (Almond and Mazumder 2011). The second study is based on the Indonesian Family Life Survey data. Using a similar intent to treat approach, the study found that Ramadan fast during pregnancy may increase risk of anaemia, coronary heart disease and type II diabetes in midlife, irrespective of the stage of pregnancy in which Ramadan took place (Van Ewijk 2011). Another study using the Indonesian Family Life Survey data reported a difference in height (Kunto and Mandemakers 2019). A recent meta-analysis of 22 studies using the Ramadan approach reported the need for further studies using a more carefully design approach before coming to any conclusion (Glazier et al. 2018).

This approach, however, suffers from the identification problem because all pregnant Muslim women need not fast. The second problem with using Ramadan as a proxy for foetal starvation is that the approach focuses on the signals received in one month, ignoring the signals received by the foetus during the remaining eight months of pregnancy. It is quite possible that the nutrition intake of the mother is high before and after Ramadan. In that case, the question is whether the signal received by the foetus during the month-long Ramadan is sufficiently strong enough to override the signal received during the remaining 8-month period becomes important.

Finally, such studies tend to view the onset of adulthood disease as a cost of the adaptation triggered by foetal starvation. Literature on FOH is marked by a failure to consider the causal pathways through which the foetal origin hypothesis operates—the mismatch of the thrifty phenotype to the adult environment. A close examination of FOH reveals that four key predictions relating to environmental mismatch emerge from the PAR theory:

(i) "For those born in good conditions, fitness is highest under good adult conditions and progressively decreases as the adult environment deteriorates.
(ii) In good adult conditions, those individuals that developed under good conditions have higher fitness than those that developed under poor conditions.
(iii) For those born in poor conditions, fitness is highest when the environment matches that of development, that is poor, and progressively decreases as the adult environment departs from this, i.e. as the conditions in the adult environment improve.
(iv) In poor adult conditions, those individuals that developed under poor conditions have higher fitness than those that developed under good conditions" (27: 1638).

Rather than examining these hypotheses linked to the causal pathways linked to the impact of foetal starvation, studies have generally assumed that early signals of nutrition deficient environment induce adaptations, irrespective of whether the signals correctly predict the future environment, costly in the long term. Attempts to test PAR have been rare, limited to animals and produced mixed evidence among rodents (McCay and Crowell 1934; Mccay et al. 1935; Taborsky 2006).

In this study, we examine whether signals about the future environment received in the form of nutritional supply at the foetus stage enable the respondent to utilise its development plasticity and adapt accordingly. The distinctive features of the study are as follows:

(1) The study uses data from a primary survey of Muslim youths aged 18–22 years, along with their mothers. We have collected information on the current and past standard of living, as well as on Ramadan fasting during conception. This provides better proxies of the signals received at the foetal stage.
(2) Unlike studies that focus only on verification of the FOH, this study also takes into consideration present conditions. This enables us to verify whether the thrifty phenotype is optimal designed only if the signals accurately predict the future environment. In brief, we are able to link the FOH with PAR and test the latter.
(3) We are able to assess the impact of a short-term nutritional shock (the Ramadan effect), overlapping the average nutritional supply during conception (proxied by the past standard of living).
(4) Finally, most of the studies have been undertaken in developed societies, where food shortage is generally not a problem.[3] The present study examines the FOH in a nutrition-deficit context, a situation more relevant in Afro-Asian developing countries.

2 Hypotheses and Method of Data Collection

2.1 Research Hypotheses

The basic premise of this study is that an organism's phenotype is influenced by its own environment. But past environmental effects on its parent(s) are also transmitted to their developing offspring. The nutritional intake received by the mother during her pregnancy is an example; it indicates the level of nutrition in the future expected environment. Accordingly, such signals interact with the genetic make-up to prepare the offspring to perform optimally in the expected environment. If there is a mismatch between actual and expected environment, however, performance may be adversely affected.

[3]There are only a few studies of FOH in India (Stein et al. 1996, 1997; Fall et al. 1998; Kumaran et al. 2000; Yajnik 2004). Instances of studies using the Ramadan approach in other developing societies are (Kunto and Mandemakers 2018; Savitri et al. 2018).

Three aspects of this hypothesis are tested in our study:

(1) The level of nutritional intake at the foetal stage signals a particular type of future environment. Depending upon the signal, the offspring is prepared for optimal survival in a nutrition-deficit or nutrition-rich environment. If the actual environment matches the expected environment, the outcome will be better than if there is a mismatch. This consists of testing the four propositions derived by Monaghan (2008), stated earlier.
(2) Ramadan fasting provides a short-term intensive nutritional shock lasting for a month. Given its short duration, can it override the signal provided by nutritional intake during the eight months comprising the rest of the foetal stage?
(3) Finally, we examine whether foetal starvation genetically programs the offspring to face latter life economic adversity more efficiently than an offspring anticipating a nutrition-plenty future.

2.2 Survey Site

The survey was undertaken in the chronically poor and underdeveloped Basanti block in the Sundarban areas. Basanti is an administrative division in Canning subdivision of South 24 Parganas district in the Indian state of West Bengal. The headquarters of this block is at Sonakhali. A brief profile of the district is given in Table 1.

About 65% of the population lies below the poverty line, while 37% do not have two square meals per day. Basanti ranks as the worst block in South 24 Parganas according to the Standard of Living Index calculated by the 2005 Rural Household Survey (Government of West Bengal 2008). The main occupations in Basanti are farming and pisciculture. About 42% of the workers are daily labourers, while 36% are cultivators.

Table 1 Profile of Basanti block

Indicators	2001	2011
Households	50,751	70,818
Population	278,543	336,717
Urban population	–	6625 (1.97%)
Percentage of literate population	56.98	68.32
Percentage of SC/ST population	47.84	41.49
Percentage of Muslim population[a]	41.18	44.87

[a]In 2011, the share of Muslim male and female population was 44.38 and 45.38%, respectively. Corresponding percentages for 2001 were 41.18 and 40.63%, respectively

Source http://www.censusindia.gov.in/2011census/population_enumeration.html

2.3 Sampling Strategy

The study was based on a primary survey of Muslim youth aged between 18 and 22 years (born between 1993 and 1997). This age group was selected keeping in mind two factors:

(a) Lowering the age below 18 years might have included respondents whose anthropometric development is not fully complete; and
(b) Increasing the age above 22 years would mean that time gap between birth of respondent and time of survey would also increase, increasing risk of recall error.

Based on the birth date of the child, we calculated whether the foetal stage had coincided with Ramadan.[4] The mother was asked if she had fasted during the pregnancy. The mother's memory was tested by asking her questions on incidents that had occurred in the 1990s. Only those who passed the recall test were included in the survey. Thus, we are able to clearly distinguish between children exposed to foetal starvation and those who were not. In addition, information on the asset holding and economic status of the family and occupation of parents (at the time of pregnancy and current), along with education of parents, was collected.

The field survey was undertaken by a local NGO, Sundarban Unnayan Niketan (SUN), under the supervision of the Investigators. The NGO identified Muslim women with children born between our reference period (1993–1997) and administered the first round of survey, in which birth dates of the children born in this period were noted, recall tests performed to assess reliability of the respondent's memory and information collected on the educational level of the respondent and her husband, past and current occupation of the couple and asset ownership. This part of the survey was undertaken from July to October 2013.

In the second stage of the survey, undertaken between December 2013 and April 2014, camps were held in different localities within the survey region, and the following measurements of children listed in the first stage were taken:

(1) *Body mass index* (BMI), kilograms/metre^2: BMI is a measure of one's height in relation to one's weight and used to categorise a person as underweight, normal weight, overweight or obese.
(2) *Mid-upper arm circumference* (MUAC), centimetre: the circumference of the left upper arm, measured at the mid-point between the tip of the shoulder and the tip of the elbow (olecranon process and the acromion). MUAC is used for the assessment of nutritional status. It is a good predictor of mortality, and in many studies, MUAC predicted death in children better than any other anthropometric indicator.

[4]We used the Muslim calendar available in the webpage http://www.al-islam.com, cross-checking the dates with http://www.islamicfinder.org/dateConversion.php. Birth dates were cross-checked with official certificates and documents.

(3) *Skinfold thickness (in the bicep, tricep, subscapular and suprailiac regions)*, millimetre: measures of subcutaneous fat at different regions of the human body.
(4) *Body density*, grams/millilitre: measure of body fat, based on measurements of subcutaneous fat in the bicep, tricep, subscapular and suprailiac regions.[5]
(5) *Body fat*, percentage: amount of body fat mass with regard to total body weight, expressed as a percentage. This was measured using the bio-electrical impedance method.

The instruments used for measuring were Galaxy anthropometer, Harpenden skinfold calliper and Omron Karada HBF 375 Body Fat Monitor. Only those respondents who did not have a meal in the two hours preceding the survey were measured as the metabolic activity may interfere with readings of body fat monitor.

2.4 Sample Profile

A total of 684 women and 447 youths who were offspring of the women respondents (of which 57.40% were males) were surveyed using questionnaires approved by the Ethical Committee of Indian Statistical Institute, Kolkata. At the time of the survey, informed consent of the respondent (both mother and child) was obtained. After entering the data, inconsistent or incomplete entries were discarded, leaving a sample of 424 respondents. Out of them, 262 were males and 162 females. The percentage exposed to foetal starvation was 64.89 and 77.16 for males and females, respectively.[6] The sample profile is given in Table 2.

2.5 Statistical Methodology

Our study assumes that consumption is positively related to the standard of living (SLI).[7] Therefore, information on past SLI provides cues about the consumption standard of the family and, more importantly, the mother. To test the first objective, we divided the sample into four groups based on past (during pregnancy) and current

[5]Estimated using the formula given in Durnin and Womersley (Durnin and Womersley 1974).

[6]It implies that 120 and 75 male and females formed the treatment group; and 92 and 37 male and female respondents formed the control group.

[7]Standard of living indices were constructed based on information elicited on asset ownership (radio and cycle for past SLI; TV, motorbike and electricity for current SLI) quality of housing (whether *pucca* and roof type) and ownership of land. Principal component analysis was used to construct the index. The values of KMO and χ^2 (for Bartlett's test for Sphericity) were 0.63 and 191.84, respectively, for the current SLI index. For the past SLI index, these values were 0.52 and 274.72, respectively.

Respondents were divided into three groups based on values of SLI index—low, medium and high. To sharpen the contrast, only individuals with high and low SLI were taken in the study.

Table 2 Sample profile—by control/study group (percentages)

	No exposure to foetal starvation	Exposure to foetal starvation
Education of mother		
Illiterate	39.29	60.71
Can read and write	20.83	79.17
Class 1–4	22.95	77.05
Class 5–8	30.65	69.35
Class 9–10	28.57	71.43
Others	0	100
Education of father		
Illiterate	40.86	59.14
Can read and write	30.38	69.62
Class 1–4	38.98	61.02
Class 5–8	25	75
Class 9–10	19.4	80.6
Class 11–12	25	75
Graduate and above	33.33	66.67
Others	0	100
Past occupation of mother		
Housewife/unemployed	31.31	68.69
Labourer	60	40
Artisans	4.76	95.24
Primary sector	50	50
Current occupation		
Housewife/unemployed	31.22	68.78
Labourer	50	50
Artisans	19.23	80.77
Service (salaried)	66.67	33.33
Others	66.67	33.33
Respondent's gender		
Male	35.11	64.89
Female	22.84	77.16
Respondent's occupation		
Housewife/unemployed	30.15	69.85
Labourer	41.57	58.43
Agriculture (-related)	33.33	66.67
Pisciculture	75	25
Transport	12.5	87.5

(continued)

Table 2 (continued)

	No exposure to foetal starvation	Exposure to foetal starvation
Handicrafts	20.22	79.78
Other trade	40.74	59.26
Salaried	66.67	33.33
Student	24	76
Others	11.11	88.89
Respondent's education		
Illiterate	31.25	68.75
Can read/write	33.33	66.67
Class 1–4	35.82	64.18
Class 5–8	27.66	72.34
Class 9–10	27.94	72.06
Class 11–12	31.37	68.63
Graduate	27.78	72.22
Other	100	0
Previous financial status		
Poorest	42.40	52.22
Middle	13.60	13.31
Richest	44.00	34.47
Current financial status (5 quintiles)		
Poorest	47.29	4.00
Middle	25.58	28.47
Richest	27.13	44.00

SLI—HH (SLI was high in both periods), HL (past SLI was high, while current SLI is low), LL (SLI was low in both periods) and LH (individuals had low SLI in the past, but this improved subsequently). High past SLI implies a high nutritional intake during pregnancy. Based on this signal, the foetus anticipates a nutrition-plenty environment. The reverse holds for respondents with low past SLI. Predictions are correct for the two groups, HH and LL; LH and HL, on the other hand, represent mismatches.

Monaghan's four propositions, stated in the introduction, can be restated as:

(1) **H11**: Outcome of respondents with high past SLI worsens if SLI decreases ($x^{HL} < x^{HH}$);
(2) **H12**: Outcome of respondents with low past SLI worsens if SLI increases ($x^{LH} < x^{LL}$);
(3) **H13**: Outcome of respondents with high current SLI will be better if their past SLI was also high ($x^{LH} < x^{HH}$); and,

(4) **H14**: Outcome of respondents with low current SLI will be better if their past SLI was also low ($x^{HL} < x^{LL}$).

when x is the outcome of interest described earlier.

To test whether such differences are statistically significant is not an easy task given that the samples are small (and will decrease as we progress to further tests) and unequal. The reliability of the Student's t-statistic has been questioned as it assumes that sample sizes are equal, have equal variances and are normally distributed (Ruxton 2006). Although the use of the Mann–Whitney U-test has sometimes been suggested, its inability to effectively substitute for the Student's t has been demonstrated (Kasuya 2001; Neuhäuser 2002). In such a situation, a modification of the degrees of freedom (Welch 1938, 1947) to increase the power of the t-test for unequal samples with unequal variances has been found to be useful (Ruxton 2006), particularly as it ensures better control of Type 1 error rates (Delacre et al. 2017). Resampling is another technique that has been suggested (Edgington 1964, 1969). This consists of taking repeated permutations of the original sample and estimating in how many cases the hypothesis is proved. In this study, we have taken 1000 simulations of the sample based on the Monte Carlo method. Welch's t-test was performed for all the permutations, and the proportion of times, our hypothesis was accepted, is reported.

The second set of hypotheses comprises of checking whether the short-term intensive nutritional shock of Ramadan fasting overrides the signal provided by nutritional intake during the eight months comprising the rest of the foetal stage.

To test this, we divided the sample into four groups based on their past and current SLI. We examined the impact of a short-term nutritional shock, in the form of Ramadan fasting, for respondents with high past SLI scores (HH and HL). Respondents belonging to the LH and HL groups are not considered as Ramadan fasting merely accentuates the low levels of nutritional intake of the mother. Our hypotheses are:

(1) **H21**: For families whose economic status has deteriorated (HL), children exposed to foetal starvation have better outcomes than those children not exposed to any such nutritional shock $\left(x_{FS}^{HL} > x_{NE}^{HL}\right)$, and
(2) **H22**: For families whose economic status has remained about the same (HH), children exposed to foetal starvation have worse outcomes than those children not exposed to any such nutritional shock $\left(x_{FS}^{HH} < x_{NE}^{HH}\right)$

when

FS indicates that the respondent has been exposed to foetal starvation, and
NE indicates that respondent has not been exposed to foetal starvation.

In the third step of our analysis, we test whether foetal starvation genetically programs the offspring to face latter life economic adversity more effectively than an offspring anticipating a nutrition-rich future. It is tested using the double-difference method. The first differences are:

$$D_{FE} = (x_{HH} - x_{HL})_{FE} \text{ for the study group}$$

$$D_{\mathrm{NE}} = (x_{\mathrm{HH}} - x_{\mathrm{HL}})_{\mathrm{NE}} \text{ for the control group}$$

It represents the decline in outcome due to deterioration in SLI. We then take the second difference:

$$\mathrm{DIDM} = D^{i}_{\mathrm{NE}} - D^{i}_{\mathrm{FE}}$$

Given that foetal starvation prepares respondents to face a nutrition deficient environment, it should be the respondents exposed to foetal starvation who are better able to face the adversity relative to respondents not exposed to foetal starvation. Although the anthropometric outcome of both groups will fall, the decline will be less for the control group vis-à-vis for the study group. The hypothesis is:

(1) **H31**: For families whose economic status has deteriorated (HL), the mean decline in outcome for children exposed to foetal starvation is less than the mean decline in outcome for those children not exposed to any such nutritional shock, i.e. DIDM > 0.

Since this is a difference-in-difference, a regression of the outcomes studied should be regressed on an EXPOSURE dummy (=1 if exposed to Ramadan fasting in the foetal stage, =0 otherwise), an indicator labelled TREATMENT (indicating whether respondent belongs to HH or HL group) and the interaction of these two dummies. The coefficient of the interaction is an estimate of the average treatment effect (ATE). As control variables, we have used birth order, and dummies indicating whether respondents were delivered prematurely, were breastfed and are currently engaged in any manual work. Past and current economic status is taken care of in the TREATMENT dummy. For households belonging to the HH group (high SLI in both periods), TREATMENT takes a value of unity; in case of households whose economic status has fallen (i.e. HL), TREATMENT equals 0.[8]

3 Findings and Discussion

3.1 Mismatch Between Actual and Expected Environment

Results are reported in Table 3. For male respondents, the percentage of indicators confirming the hypotheses are 100 (HH > HL), 88 (HH > LH), 63 (LL > LH) and 75 (LL > HL). Welch's test supported our hypothesis in only 23% of the simulations for suprailiac skinfold thickness (males, HH vs. HL) and in 32 and 38% cases for BMI and skinfold thickness at bicep region (male: LL vs. HL). On the other hand, the proportion of simulations supporting our hypothesis was high for all skinfold thicknesses (except bicep), body density and body fat per cent (Male: LL vs. LH).

[8]LH and LL households are not considered in these hypotheses.

Table 3 Mean outcomes for respondents belonging to HH, HL, LH and LL households—male and female respondents

Anthropometric indicators: male	HH (33)	HL (35)	Welch's *t*	Prob.	*c/n*	HH (33)	LH (30)	Welch's *t*	Prob.	*c/n*
BMI	21.51	19.42	1.83	0.04	0.9980	21.51	20.08	1.24	0.11	1.0000
Mid-upper arm circumference	24.28	22.48	2.01	0.02	0.9800	24.28	23.47	1.16	0.13	0.9750
Biceps: skinfold thickness	5.2	4.36	1.15	0.13	0.8650	5.2	4.71	0.76	0.23	0.9380
Triceps: skinfold thickness	7.85	5.94	1.57	0.06	0.9630	7.85	5.54	2.02	0.02	0.7950
Subscapular skinfold thickness	12.47	8.59	2.89	0.00	0.9980	12.47	10.45	1.40	0.08	0.8850
Suprailiac skinfold thickness	6.64	5.51	1.11	0.14	0.8180	6.64	6.04	0.54	0.30	0.2350
Body density	8.94	8.58	2.49	0.01	0.9870	8.94	7.56	1.40	0.08	0.8380
Percentage of body fat	15.57	13.13	1.84	0.04	0.8980	15.57	13.34	1.61	0.06	0.5590
Anthropometric indicators: male	LL (59)	LH (30)	Welch's *t*	Prob.	*c/n*	LL (59)	HL (35)	Welch's *t*	Prob.	*c/n*
BMI	19.09	20.08	−1.93	0.97	0.0800	19.09	19.42	−0.69	0.76	0.3170
Mid-upper arm circumference	22.66	23.47	−1.30	0.90	0.1460	22.66	22.48	0.22	0.41	0.5650
Biceps: skinfold thickness	4.2	4.71	−1.46	0.93	0.1990	4.2	4.36	−0.34	0.63	0.3820
Triceps: skinfold thickness	7.02	5.54	2.01	0.02	0.9040	7.02	5.94	1.28	0.10	0.8550
Subscapular skinfold thickness	11.07	10.45	0.54	0.29	0.7070	11.07	8.59	2.46	0.01	0.9820
Suprailiac skinfold thickness	7.48	6.04	1.35	0.09	0.8820	7.48	5.51	2.03	0.02	0.9580
Body density	7.91	7.56	0.60	0.27	0.7150	7.91	8.58	1.85	0.03	0.9530
Percentage of body fat	15.27	13.34	1.65	0.05	0.8550	15.27	13.13	1.95	0.03	0.8970

(continued)

Table 3 (continued)

Anthropometric indicators: female	HH (26)	HL (19)	Welch's *t*	Prob.	*c/n*	HH (26)	LH (16)	Welch's *t*	Prob.	*c/n*
BMI	19.25	17.72	2.07	0.02	0.9470	19.25	19.99	−0.91	0.82	0.7160
Mid-upper arm circumference	21.3	20.56	0.97	0.17	0.7900	21.3	22.16	−1.21	0.88	0.3940
Biceps: skinfold thickness	6.57	6.84	−0.26	0.60	0.3790	6.57	6.37	0.22	0.41	0.0380
Triceps: skinfold thickness	11.07	11.03	0.02	0.49	0.5190	11.07	10.15	0.54	0.30	0.8520
Subscapular skinfold thickness	12.77	10.63	1.14	0.13	0.9040	12.77	11.64	0.77	0.22	0.8810
Suprailiac skinfold thickness	3.63	3.38	0.19	0.43	0.5930	3.63	3.48	0.85	0.20	0.7640
Body density	3.86	3.78	−0.55	0.29	0.7450	3.86	3.79	0.50	0.31	0.6510
Percentage of body fat	25.08	22.75	1.51	0.07	0.8280	25.08	25.26	−0.13	0.55	0.1580
Anthropometric indicators: female	LL (41)	LH (16)	Welch's *t*	Prob.	*c/n*	LL (41)	HL (19)	Welch's *t*	Prob.	*c/n*
BMI	18.83	19.99	−1.49	0.93	0.0840	18.83	17.72	1.60	0.06	0.9210
Mid-upper arm circumference	21.58	22.16	−0.91	0.82	0.2750	21.58	20.56	1.44	0.08	0.8800
Biceps: skinfold thickness	7.81	6.37	1.71	0.05	0.9580	7.81	6.84	0.96	0.17	0.8980
Triceps: skinfold thickness	9.71	10.15	−0.32	0.62	0.3590	9.71	11.03	−0.81	0.79	0.1630
Subscapular skinfold thickness	11.21	11.64	−0.37	0.64	0.3720	11.21	10.63	0.35	0.36	0.6680
Suprailiac skinfold thickness	3.54	3.48	0.24	0.41	0.0573	3.54	3.38	−0.40	0.65	0.2910
Body density	3.82	3.79	0.22	0.41	0.5710	3.82	3.78	0.31	0.38	0.6210
Percentage of body fat	27.12	25.26	1.43	0.08	0.7900	27.12	22.75	2.87	0.00	0.9790

Note Figures in parentheses below HH, HL, LH and LL are sample size

Among females, three of our hypotheses (HH > HL, HH > LH and LL > HL) receive support from results of more than half the indicators—for seven, five and six out of the eight indicators, respectively. In these three cases, a low proportion of simulations support our hypothesis for biceps (HH vs. HL), mid-upper arm circumference, skinfold thickness in bicep and body fat per cent (HH vs. LH) and skinfold thickness in bicep and suprailiac regions (LL vs. HL). Therefore, there seems considerable evidence to support the starting group of hypotheses (H11–H14), stating that outcomes will be better if the signal received at the foetal stage matches with the actual future environment, vis-à-vis when there is a mismatch.

Overall, when signals of nutrition deficiency at the foetal stage match with the subsequent environment, male respondents were found to have higher MUAC, skinfold thickness at the triceps and subscapular region, body density and body fat; among female respondents, a similar finding was observed for skinfold thickness in the subscapular region, body density and body fat. In addition, BMI was also higher in general. Further, mean values of such respondents were in the normal range. The control group, in contrast, had lower values for these outcomes, bordering on the low-normal boundary. It indicates that mismatch in signals of expected environment and the ensuing environment may reduce body fat levels. This may have long-term health consequences (Blackburn and Phillips 2001)—affecting the ability to store energy (leading to fatigue), affect the functioning of the brain and cardiovascular system, reduce thyroid levels, affect the absorption of calcium and weaken muscles.

3.2 Impact of Ramadan Fasting

The second question that we turn to is the impact of Ramadan fasting. Past SLI provides information about consumption levels over the entire gestation period. But are short-term nutritional shocks also important as indications of the future environment? In particular, do short-run nutritional shocks, like Ramadan fasting, override the longer run signals provided by past SLI (and consumption) to the foetus?

Results are given in Table 4. Here, the evidence seems to be against the hypothesis. This confirms studies of the impact of Ramadan fasting among Muslims in diverse countries (Kavehmanesh and Abolghasemi 2004; Ziaee et al. 2010; Alwasel et al. 2013a, b; Savitri et al. 2014, 2018; Glazier et al. 2018). Results—whether percentage difference, Welch's *t*-test or *t*-tests after Monte Carlo simulations—supports our hypothesis to some extent only for male respondents in the HH group. For this group, *t*-tests confirm our hypothesis for skinfold thickness in tricep, subscapular and suprailiac regions and percentage of body fat. After Monte Carlo simulations, t-tests support our hypothesis for six out of eight indicators. The exceptions are mid-upper arm circumference and bicep skinfold thickness.

The failure to find evidence in support of the impact of Ramadan fasting is in contrast to the favourable results obtained for the impact of a sustained nutritional deficit. The present study is not directly comparable with existing studies of Ramadan fasting that uses large-scale survey data and relate coincidence of Ramadan with ges-

Table 4 Mean outcomes for male respondents belonging to HH and HL households—by exposure to foetal starvation

Anthropometric indicators: male	SLI has deteriorated: HL (17, 18)					SLI has always remained high: HH (12, 21)				
	No exposure	Exposure to foetal starvation	Welch's *t*	Probability	*c/n*	No exposure	Exposure to foetal starvation	Welch's *t*	Probability	*c/n*
BMI	19.64	18.66	0.54	0.70	0.0270	23.01	20.34	0.82	0.21	0.6450
Mid-upper arm circumference	21.46	23.08	−1.38	0.09	0.3800	24.48	23.78	0.25	0.40	0.0570
Biceps: skinfold thickness	5.52	6.47	−0.51	0.31	0.2720	8.4	6.36	0.43	0.34	0.3430
Triceps skinfold thickness	7.56	12.28	0.94	0.82	0.0150	14.65	11.1	1.37	0.09	0.7400
Subscapular skinfold thickness	9.65	6.98	1.59	0.93	0.0030	16.38	10.49	2.36	0.02	0.8520
Suprailiac skinfold thickness	6.18	4.95	1.14	0.87	0.0640	8.6	5.46	1.43	0.09	0.7650
Body density	3.45	3.32	1.17	0.87	0.0160	3.85	3.52	1.98	0.03	0.8190
Percentage of body fat	9.7	8.32	1.10	0.86	0.2690	12.18	10.3	0.78	0.22	0.7500

(continued)

Table 4 (continued)

Anthropometric indicators: female	SLI has deteriorated: HL (5, 10)					SLI has always remained high: HH (4, 18)				
	No exposure	Exposure to foetal starvation	Welch's *t*	Probability	*c/n*	No exposure	Exposure to foetal starvation	Welch's *t*	Probability	*c/n*
BMI	17.12	17.69	−0.97	0.83	0.6540	18.78	19.58	−0.50	0.70	0.2980
Mid-upper arm circumference	20.84	20.41	0.26	0.60	0.6990	20.53	21.66	−0.87	0.70	0.6050
Biceps: skinfold thickness	4.14	5.82	−1.10	0.14	0.0940	5.61	5.53	0.70	0.26	0.0800
Triceps skinfold thickness	6.47	5.13	−1.90	0.04	0.0620	10.3	6.6	0.09	0.20	0.0110
Subscapular skinfold thickness	8.16	10.72	−1.62	0.06	0.3560	13.55	13.49	0.94	0.20	0.0970
Suprailiac skinfold thickness	8.66	8.08	0.03	0.51	0.3290	9.9	9.44	0.27	0.40	0.5450
Body density	3.63	3.79	−1.05	0.16	0.2320	4.01	3.90	0.31	0.39	0.1740
Percentage of body fat	16.68	18.25	−0.25	0.41	0.4210	18.88	21.67	−1.34	0.91	0.4320

Note Figures besides HH and HL, in parentheses, are sample sizes for treatment and control groups, respectively

tation period (irrespective of whether mother had fasted or not) to onset of diseases at midlife. The sample size is much larger in the case of the latter, the proxy for foetal starvation is different and the phenomenon being studied is different. Nevertheless, the results of this subsection question the advisability of relying on short-term nutritional shocks in the form of Ramadan fasting as a proxy for foetal starvation, ignoring long-term nutritional intakes. It is a finding that is in line with meta-reviews suggesting that Ramadan fasting does not adversely affect birth weight and that there is insufficient evidence regarding potential effects on other perinatal outcomes (Glazier et al. 2018).

3.3 Facing Economic Adversity

In the third step of our analysis, we test whether persons exposed to foetal starvation are able to adapt better to conditions of scarcity and economic hardship than those who are not exposed to foetal starvation, using a double-difference approach. Results are given in Table 5.

Among male respondents, ATE is statistically significant for BMI (10% level), mid-upper arm circumference (10% level) and skinfold thickness in the subscapular region (5% level). Among females, ATE is high for skinfold thickness in tricep region (5% level). There seems some evidence indicating that exposure to foetal starvation may better prepare respondents to face a decline in economic status.

Table 5 Average treatment effects for male and female respondents—by exposure

Anthropometric outcomes	Male ($n_T = n_C = 17$)			Female ($n_T = 15$; $n_C = 5$)		
	ATE	*t*-ratio	Prob.	ATE	*t*-ratio	Prob.
BMI	4.04	1.77	0.08	0.90	0.41	0.69
Mid-upper arm circumference	3.53	1.90	0.06	−0.12	−0.06	0.95
Biceps: skinfold thickness	1.61	1.00	0.32	3.03	1.05	0.30
Triceps: skinfold thickness	3.95	1.54	0.13	10.41	2.00	0.05
Subscapular skinfold thickness	5.74	2.14	0.04	7.43	1.49	0.15
Suprailiac skinfold thickness	1.14	0.53	0.60	1.65	0.32	0.75
Body density	0.28	1.30	0.20	0.45	1.09	0.28
Percentage of body fat	2.02	0.72	0.48	1.34	−0.30	0.76

Note Figures in parentheses, besides male and female, are treatment and control group sizes

4 Conclusion

To sum up, the FOH argues that exposure to foetal starvation may increase disposition to develop diabetes mellitus, hypertension, cardiac ailments, nephrological diseases, etc. in midlife. The underlying mechanism is the growth of a "thrifty" phenotype that prepares the organism to survive in nutrition-deficit environments, but results in maladaptation if the organism is subsequently exposed to prosperous conditions. The PAR theory extends this idea to suggest that the foetus receives signals of the expected environment in which it will grow up. If the signals predict the future environment correctly, then the organism is suited to its local environment and will have optimal outcomes. If, however, signals are faulty and wrongly predict the future environment, then outcomes will be adversely affected as the organism is maladjusted to its local environment.

The present study attempts to contribute to the literature by testing for PAR in a chronically underdeveloped locality. Our study finds some evidence in support of PAR, particularly among male respondents. Mismatching of the predicted and actual environment appears to affect body fat levels, reducing measures to values slightly above the low levels. This may have serious long-term health consequences in midlife as the chances of suffering from non-communicable diseases will increase. However, there is scope to extend the study by undertaking a study with a larger sample size, which would enable controlling for more variables.

Even the limited evidence produced by this pilot study has important implications for maternal and child health—an important component of Sustainable Development Goals adopted by the United Nations in 2015. The current study shows that individuals exposed to foetal starvation may become programmed to function *only in nutrition-deficit conditions*. Subsequent prosperity affects their anthropometric outcomes adversely, which may increase the probability of developing lifestyle diseases in midlife. A sharp rise in non-communicable diseases in developing societies has been reported in various studies (World Health Organization 2018). It implies that, unless we ensure proper nutrition to pregnant women, their offsprings are likely to be programmed in such a manner that they will become susceptible to non-communicable diseases and unfit to reap the benefits of growth and development. This calls for concerted attempts to guarantee adequate nutrition during pregnancy to ensure that growth leads to an inclusive society.

Designing such strategies, however, is not easy. Nutritional interventions designed to increase birth weight have had only modest success (Kramer 1993; Rasmussen 1999). Even if one assumes that association between birth weight (BW) and cardiovascular diseases (CVD) is causal, increasing birth weights will improve cardiovascular outcomes marginally (Joseph and Kramer 1997). Such findings imply that "although studying factors in early life has offered some new ways of thinking about the origins of CVD, increasing BW is likely to be much less effective in reducing mortality than modifying the traditional risk factors observed during adulthood" (Rasmussen 2018: 87). Public health policies to increase birth weight has costs; they may expose infants to the risk of being macrosomic at birth or developing cancer

(Rasmussen 2018). Further, recent studies have argued that birth weight is only a poor proxy for a process or processes that affect the foetus. Improving our understanding of how and when genetic programming occurs, therefore, is an important precondition to design intervention strategies during pregnancy to reduce the development of the chronic disease.

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Consent for Publication Respondents had given their consent to the data being used for academic purposes after concealing their individual identities.

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Availability of Data and Material The data sets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate The study is based on a primary survey eliciting history, socio-economic characteristics and anthropometric measures of respondents and their children. There was no intervention. Hence, clearance was not required from the ethical committee. However, informed written consent, witnessed by an independent person, was taken before administering the questionnaires.

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