Fasting during Ramadan and subsequent long term impact on health of children: Comparing the Foetal Origin and Predictive Adaptive Response Hypotheses

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1. Introduction

Despite her remarkable economic growth over the last decade, India has been slipping behind other South Asian countries with respect to health outcomes (Sen and Dreze, 2011). Studies report that the health status of India's children is worse off than even that in Sub-Saharan African countries (Nandi Trust, 2011). The issue of malnutrition is particularly severe among children from SC, ST and Muslim families and from low income households (Nandi Foundation, 2011).

The consequences of this nutrition crisis are enormous; in addition to being the cause of one third to one half of child deaths, malnutrition causes stunted physical growth and compromises cognitive development over the lifetime. The economic losses associated with malnutrition are estimated at 3 per cent of India's Gross Domestic Product annually (Horton, 1999). While such studies deservingly highlight the impact of child nutrition on adult health, some studies have argued that the health status of adults is programmed even earlier —*in utero* to be precise. This proposition, commonly known as the foetal origin hypothesis, states that if the mother is exposed to starvation and malnutrition during pregnancy, this will make it more likely that, on attaining adulthood, the child will suffer from cardio-vascular disease, diabetes, etc.

The hypothesis also has important implications for the modeling of human capital development. The Grossman model of health capital development (Grossman, 1972) models the motion of health stock as:

$$H_t = (1 - \delta)\, H_{t-1} + I_t,$$

where I_t represents investments in health capital H and δ represents depreciation of this stock. This formulation presumes that health stock depreciates over time, incorporating the assumption that impact of shocks to health capital fades over time. The foetal origin hypothesis suggests that we could have investments *in utero* and investments occurring during the rest of childhood:

$$H_{adult} = A\left[\, I_{prenatal}^{\alpha} + (1-\delta)\, I_{postnatal}^{\alpha} \,\right]^{1/\alpha}.$$

This formulation implies that the effects of prenatal investment shocks may be non-monotonic in age. Instead of a fadeout of early shocks (as in the Grossman model), the potential for complementarities between prenatal shocks and subsequent investments underscore the persistent importance of a 'good start'. Furthermore, 'dynamic complementarities' would imply that investments in period t are more productive when there is a high level of capability in period t-1 (Heckman, 2007). The economic losses of foetal starvation, although not measured, may comprise a substantial proportion of GDP. Moreover, in societies where out of pocket expenditure is high and where medical expenditure is one of the leading causes of households slipping into poverty and indebtedness, the economic impact of malnutrition of carrying women will have a spill over into future generations, creating a vicious cycle of malnutrition, poverty and poor health.

Attempts to test the foetal origin hypothesis are, however, hamstrung by the long interval between the cause (occurring *before* birth) and the effect (occurring *decades* after birth), making it difficult to identify women who have been exposed to starvation *in the past*. Consequently, recent studies have focused on the Muslim community, as Muslims fast for a month during the month of Ramadan every year. This makes this community particularly suitable to studies of long term health impact of foetal starvation. The present study proposes to examine the impact Ramadan fasting has on the anthropometric development of children whose gestation coincided with the fast observed by the pregnant women residing in the chronically underdeveloped Sundarban area of West Bengal, India.

The study contributes to the current body of knowledge in two ways. Firstly, while health of the mother is known to have an impact on the health of the offspring and has been documented adequately (Bharati & Basu, 1990), the *long run* impact of nutritional deficiency *during pregnancy* has possibly not been studied in India. Secondly, in contrast to the existing studies of the impact of Ramadan fasting, our study is based on primary data. This enables us to discard the assumption that all mothers fasted during Ramadan with its associated intent to treat approach inherent in these studies, and successfully distinguish between those actually fasting and those who are not. Thus, we are able to directly assess whether the mother was exposed to nutritional deficiency or not.

The study is structured as follows: Section 2 surveys the literature on the foetal origin hypothesis. This is followed by a description of the research methodology and survey area. Section 4 presents the findings, and is followed by a concluding section discussing the implications of the findings.

2. Literature survey

2.1 The Foetal Origin Hypothesis (FOH)

In the late 1950s epidemiologists believed that the placenta was a "perfect filter, protecting the fetus 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 colleagues at the MRC Environmental Epidemiological Unit in Southampton, UK. The latter put forward the proposition 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 aging may be programmed before birth, Barker (1990) 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 including "programming by the environment in foetal and infant life" (Barker, 1990: 1111).

[1] Thalidomide was licensed in 1957 (except in US) 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 (McBride, 1961; Von Lenz and Knapp, 1962).

Barker's work was based on correlation analysis, which tests for only (linear) association.[2] Subsequent studies applied more rigorous methodologies to find an increase in schizophrenia among those potentially affected by the Dutch famine of 1944 (Hoek et al., 1998) and Chinese famine of 1959-61 (St. Clair et al., 2005). Such studies, however, take weight at birth as proxy for foetal health, despite evidence showing that it may not be a comprehensive or sensitive measure.[3]

2.2 Verifying the hypothesis

There have been a large number of studies by development economists examining the foetal origins hypothesis (for citation and a brief review see Almond and Currie, 2011a: 160-167). The problem with such studies is that they focus on two widely spread out time periods—pregnancy and adulthood. Short of time travelling, it is not easy to obtain reliable data on *both* these time periods, creating an identification problem (Paneth and Susser, 1995).[4] As a result, economists have attempted to model starvation in terms of a national/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).

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[2] For instance, Barker and Osmond (1986) estimated correlation coefficients between adult mortality rates from 1968-1978 and infant mortality rates from 1921-1925 by geographical areas based on extracts from British's Office of Censuses and surveys of all death certificates from 1968 to 1978.

[3] A study by Stein et al. (1975) found that cohorts exposed to famines in the first half of pregnancy had relatively normal birth weight, but displayed health effects subsequently. In contrast, those affected in the latter half of pregnancy were healthier, despite having lower birth weight. In fact, Schultz (2010) observes that intrauterine exposures having long term health impacts do not alter birth weights.

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

For instance, the Influenza Pandemic of 1918 affected mothers of one third of male children born in early 1919. Comparing to children born in early 1918, Almond (2006) found that children of infected mothers were about 20 percent more likely to be disabled, experienced wage decreases of at least 5 percent and had lower educational attainments. Similar findings were reported for pandemic influenza in Brazil, Britain, Switzerland and Taiwan by Nelson (2010), Kelley (2011), Neelson and Stratman (2011) and Lin (2011). Using the 19th century blight to French vineyards caused by the phylloxera insect as a shock, Banerjee et al. (2010) found that prenatal exposure reduced height of army recruits aged 20 years by 0.5 to 0.9 cm.

Another method that has been used in some recent studies is to focus on the Muslim community. One of the duties of Muslims is to fast from sunrise to sunset during the lunar month of Ramadan. Although pregnant women may seek exemption from fasting, in practice they rarely do so—ignoring medical advice to the contrary.[5] We have been able to trace two studies of the long run impact of Ramadan fasting by the mother on long term health impact of the child. Almond and Mazumdar's study is based on secondary data for (2011). Plotting birth data against occurrence of Ramadan in a particular year, they are able to distinguish between children who were *in utero* during Ramadan, and those who were not. The study found that being exposed to Ramadan in early pregnancy has significant health effects, with the likelihood of adult disability increasing by over 20

[5] Ewijk (2011: 1248) reports that "A great majority of 70-90% of pregnant Muslim women do fast (during Ramadan), as is evidenced by research around the world, from Iran (Arab and Nasrollahi, 2001), to Singapore (Joosoph et al., 2004), rural West Africa (Prentice et al., 1983) and the UK (Malhotra et al., 1989)." Gynecologists practicing in Kolkata also reported that their patients fasted during pregnancy.

percent among Iraqis and Ugandan Muslims. Substantially larger effects are observed for mental and learning disabilities.

The second study (Ewjik, 2011) is based on Indonesian Family Life Survey data. Using a similar intent to treat approach, Ewjik found that Ramadan fast during pregnancy may cause considerable negative health effects on the offspring, irrespective of the stage of pregnancy in which Ramadan took place. Further, some effects (risk of anemia, coronary heart disease and Type 2 diabetes) may actually get stronger, or only show up, when the offspring gets older.

Both studies, however, suffers from an identification problem, stemming from the reliance on secondary data. As Ewjik communicates:

> "I did not know whether mothers had fasted and therefore had to assume that all Muslim mothers whose pregnancies overlapped with a Ramadan did fast. (The control group is then children born to Muslim mothers whose pregnancies did not overlap with a Ramadan.) Of course, in reality, not all fasted. Therefore, the magnitude of all estimates would have to be multiplied by one over the share of fasting to get to the "true" effect of fasting. (So, if e.g. a random 50% of all mothers fasted, all my effect estimates would have to be multiplied by a factor 2.)" (Ewjik, personal communication, 30 November 2011).

To clearly distinguish between parents who fasted, and those who did not, we proposed to employ a study based on a primary data set.

3. Methodology

3.1 Hypothesis

The primary hypothesis of the present work was:

> Does exposure to *in utero* starvation produce long term health effects manifested in malnutrition as measured by selected anthropometric measurements?

The health impact of the foetal shock was examined for the following indicators:

1) *Anthropometric measures*: Height, Weight, Mid-upper arm circumference, Waist circumference and Hip circumference.
2) *Skinfold thickness*: Bicep, Tricep, Subscapular and suprailiac.
3) *Body fat composition*: Body fat, Visceral fat and Subcutaneous fat (whole body, trunk, arms and legs).
4) *Muscle composition*: Subcutaneous Muscle (whole body, trunk, arms and legs).
5) *Miscellaneous*: Blood pressure and pulse, Resting metabolism, Body density, Body Mass Index and Body age.

In all, 27 indicators of health status were used. The instruments used for measuring were Galaxy anthropometer, Harpenden Skinfold calliper, Omron Karada HBF 375 Body Fat Monitor and Omron HEM7200 Blood Pressure Monitors.

3.2 Survey site

The survey was undertaken in the chronically poor and underdeveloped Basanti block in the Sunderban 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.

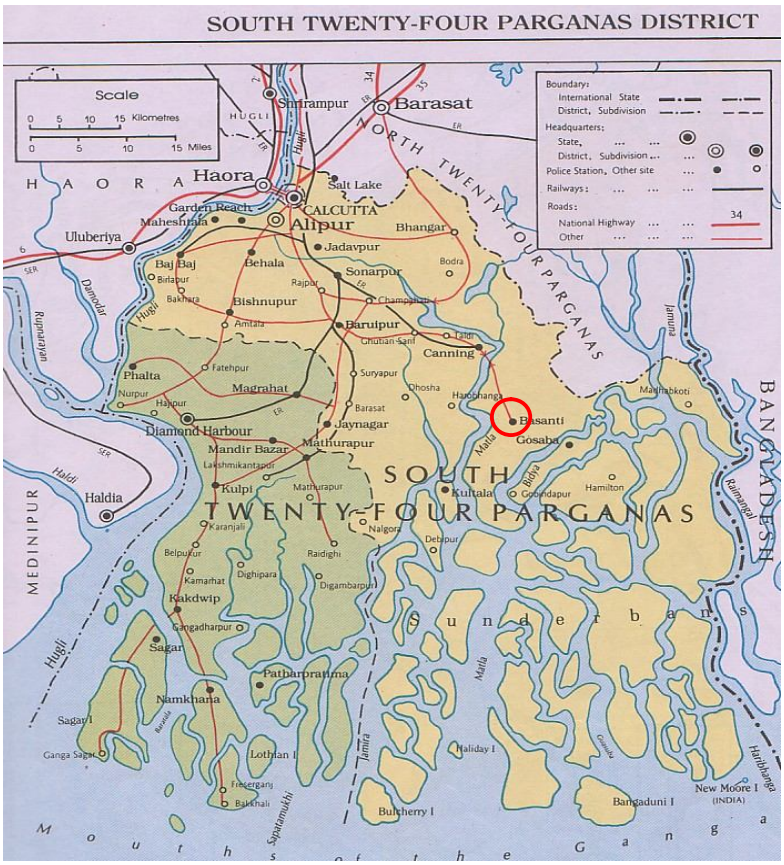

Figure 1: Map showing survey site

About 65 per cent of the population lies below the poverty line, while 37 per cent 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 (GWB, 2008). The main occupations in Basanti are farming and pisiculture. About 42 of the workers are daily labourers, while 36 per cent are cultivators.

Table 1: Profile of Basanti block

Indicators	2001	2011
Households	50,751	70,818
Population	278,543	336,717
Urban population	-	6,625 (1.97%)
Percentage of literate population	56.98	68.32
Percentage of SC/ST population	47.84	41.49
Percentage of Muslim population[6]	41.18	44.87

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

The health sector is underdeveloped. This is a major problem, given the high levels of arsenic contamination of ground water. In 2009, however, there were only 63 Sub-centres, 1 Family Welfare Centre, 1 Block Primary Health Centre, 3 Primary Health Centres and 1 registered private nursing home. Not surprisingly, health indicators are very poor in the block. While block-wise mortality rates are not available, the early age of marriage (about half of the women marry before attaining 18 years) coupled with poor ante-natal coverage (about 44 per cent of pregnant women do not receive ante-natal check-ups), low rate of institutional or safe deliveries (39 and 70 percent, respectively) and poor immunization record (less than 80 per cent of children are fully immunized) makes it likely that maternal and infant mortality will be high. The infrastructural deficiencies reported in the District Human Development Report (GWB, 2008), along

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

with reliance on non-registered 'quack' doctors also contribute to high mortality rates. Education facilities, too, needs to be developed—there are only 3,683 recognized primary schools, out of a total of 4,490 recognized schools.

The selection of the survey site in an underdeveloped area with poor human development indicators has an important implication for the study. Earlier attempts to verify the FOH were situated in areas where the nutrition deficiency was a temporary shock. The environment in which the child subsequently grew up was, therefore, quite different from the foetal stage in terms of nutrient deficiency. In the present study, however, the signal to the foetus was compatible with the subsequent physical reality where the children grew up, facing nutrient shortage. This enables us to examine whether the FOH works in chronically deprived regions.

3.3 Data collection

The study was based on a primary survey of Muslim youth aged between 18-22 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 age of mother would also increase. This has the potential danger that her memory will fall.

Based on the birth date we calculated whether conception had coincided with Ramadan, and the trimester in which it had coincided.[7] The mother was asked if she had fasted

[7] 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.

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. 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. Details of the pregnancy (whether premature, whether mother had any illness during pregnancy) and breastfeeding of the child was also collected.

The field survey was undertaken with the help of a local NGO, Sundarban Unnayan Niketan (SUN). The NGO identified Muslim women with children borne between our reference period (1993-1997) and administered the first round of survey in which birth dates of the children borne in this period was 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, asset ownership, conceptions (whether there were any complications, whether delivery was premature, etc.) and relevant post-delivery issues (whether breast-fed). The questionnaire is attached in Appendix A. This part was undertaken between July-October 2013.

In the second stage of the survey, undertaken between December 2013-April 2014, a team of three members was formed. Two of these were Physiology Post Graduates from Presidency University, while the third was Under Graduate student and/or the Principal Investigator. This team, along with the representatives of SUN, set up periodic camps in different localities in the survey region and mobilised the children identified in the first

stage to appear for anthropometric measurements (Fig. 2). Those respondents who did not have a meal in the two hours preceding the survey was measured. In addition, some new persons borne in this period turned up for the camps. Measurements were taken for these persons, and the first round questionnaire administered to their mothers subsequently.

The members of SUN identified the household codes to which second round respondents belonged. If their mothers had not been surveyed they were assigned new household codes. After collecting basic information on education and income, the respondents were sent for anthropometric measurements. This was taken by the Physiology students, who had prior experience of handling the equipment used in the survey (Figure 3). The measurements were noted down by the additional members of the team.

3.4 Methodology

The data entry was undertaken using CS Pro version 5, and analysed using STATA version 13. The measure of anthropometric development was regressed on a dummy indicating whether respondent was exposed to *in utero* fasting. In addition we controlled for socio-demographic traits (age of child, age of mother when delivered, parity, gender of respondent, religion, caste, past and current economic status, education of parents, current and past occupation of parents). Three sets of regression models were estimated, based on the dummy measuring the shock of foetal malnutrition:

a) Binary exposure dummy indicating whether respondent was exposed to foetal starvation or not (reference category).

b) Exposure dummies indicating whether respondent was exposed to 30 days Ramadan fasting, whether respondent was exposed to less than 30 days Ramadan fasting[8] and respondents not exposed to foetal starvation (reference category).

c) Dummies indicating whether exposure to foetal origin occurred in the first, second or third semester (reference category was those not exposed to foetal starvation).

Regression models were estimated for male and female respondents separately.[9] Since the dependent variables were continuous, the Ordinary Least Square (OLS) method was used for estimation. The statistical significance of the dummy variables (given by its associated t-statistic) indicated whether foetal starvation had any significant impact, while the sign of the corresponding regression coefficient (β) indicated the direction of impact—whether positive, or negative.

4. Findings

4.1 Sample profile

A total of 684 women and 447 children (of which 57.40 percent were males) were surveyed. 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. The sample profile is given in Table 2.

[8] Either because mother had not fasted, or because gestation did not fully coincide with Ramadan.

[9] We did not estimate models aggregating male and female respondents as the standard for the anthropometric measures used varies across genders.

Table 2: Sample profile—by control/study group

Education of mother	No exposure to foetal starvation	Exposure to foetal starvation	Education of husband	No exposure to foetal starvation	Exposure to foetal starvation
Illiterate	39.29	60.71	Illiterate	40.86	59.14
Can read & write	20.83	79.17	Can read & write	30.38	69.62
Class 1 to 4	22.95	77.05	Class 1 to 4	38.98	61.02
Class 5 to 8	30.65	69.35	Class 5 to 8	25	75
Class 9 to 10	28.57	71.43	Class 9 to 10	19.4	80.6
Others	0	100	Class 11 to 12	25	75
			Graduate & above	33.33	66.67
			Others	0	100
Past occupation of mother	**No exposure to foetal starvation**	**Exposure to foetal starvation**	**Current occupation**	**No exposure to foetal starvation**	**Exposure to foetal starvation**

Housewife/unemployed	31.31	68.69	Housewife/unemployed	31.22	68.78
Labourer	60	40	Labourer	50	50
Handicrafts	4.76	95.24	Handicrafts	19.23	80.77
			Service (salaried)	66.67	33.33
Primary sector	50	50	Others	66.67	33.33
Respondent's gender	**No exposure to foetal starvation**	**Exposure to foetal starvation**	**Respondent's occupation**	**No exposure to foetal starvation**	**Exposure to foetal starvation**
Male	35.11	64.89	Housewife/unemployed	30.15	69.85
Female	22.84	77.16	Labourer	41.57	58.43
Respondent's education	**No exposure to foetal starvation**	**Exposure to foetal starvation**			
Illiterate	31.25	68.75	Agriculture (-related	33.33	66.67
			Pisiculture	75	25

Can read/write	33.33	66.67	Transport	12.5	87.5
Class 1-4	35.82	64.18	Handicrafts	20.22	79.78
Class 5-8	27.66	72.34	Other trade	40.74	59.26
Class 9-10	27.94	72.06	Salaried	66.67	33.33
Class 11-12	31.37	68.63	Student	24	76
Graduate	27.78	72.22	Others	11.11	88.89
Other	100	0			

Previous financial status	**No exposure to foetal starvation**	**Exposure to foetal starvation**	**Current financial status (5 quantiles)**	**No exposure to foetal starvation**	**Exposure to foetal starvation**
Poor	25.73	74.27	Poorest	43.02	56.98
Middle	30.36	69.64	Poor	25.81	74.19
Rich	39.24	60.76	Middle	28.21	71.79
Richest	31.17	68.83	Rich	24.49	75.51
			Richest	29.11	70.89

4.2 Regression results

In all we have estimated 27 (for each indicator) X 2 (for each gender) X 3 (for each formulation of the starvation dummy) = 108 regression models. Instead of presenting all these results, or even a summary of all these results, we have presented results for only those regressions where the difference is statistically significant at 10% level. All the models have control variables described in Section 3.

The regression results, when exposure to foetal starvation—irrespective of the trimester or duration of exposure—is taken as the study variable, is summed up in Table 3. We can see that males exposed to foetal starvation are taller and have more skeletal muscle development in the legs; their body fat content is also significantly lower than males without exposure to foetal starvation. The regression coefficient, capturing the differences, is statistically significant at 10%, 5% and 5% level, respectively. Females exposed to foetal starvation, on the other hand, have lower skinfold thickness in the biceps region. In this case the coefficient is significant at 10% level.

Table 4 summarises the results of the models for full or partial exposure to foetal starvation. We find that males with exposure to foetal starvation are taller than those in the control group—the regression coefficients are significantly different from zero at 10% level (for those with partial exposure) and at 1% level (for those with full 30 days exposure). Males with partial exposure to foetal starvation are also observed to have lower body fat content (at 10% level) than males in the control group or with full exposure. Among females, we find partial exposure to foetal starvation results in reduced

skinfold thickness in the biceps and suprailiac region, higher systolic blood pressure and lower body density. The coefficients for partial exposure are statistically significant at 5%, 1%, 10% and 5% level, respectively.

Table 3: Impact of exposure to foetal starvation—Mean and regression results

	Male	**Female**		
	Height	**Skeletal muscle: Fat**	**Body fat**	**Skinfold thickness: Biceps**
Mean: Exposure to foetal starvation	161.2	52.0	16.0	8.0
Mean: No exposure to foetal starvation	163.4	53.1	14.2	6.84
$\beta_{exposure}$	3.03	1.09	-1.96	-1.64
T	3.21***	2.02**	-1.96**	-1.88*
R^2	0.30	0.18	0.27	0.27
N	255	254	252	252

Table 4: Impact of length of exposure to foetal starvation—Mean and regression results

	Male		Female			
	Height	**Body fat**	**Skinfold thickness: Biceps**	**Skinfold thickness: Suprailiac**	**Blood pressure: Systolic**	**Body density**
Mean: No exposure	161.2	15.9	8.0	8.0	115.4	3.8
Mean: Exposure during part of conception	162.9	13.9	7.0	6.6	118.4	3.7
Mean: Exposure during entire conception	163.7	14.4	6.8	8.5	115.5	3.8
β_{pe}	2.14	-2.36	-2.04	-2.41	6.45	-0.22
t	1.80*	-1.86*	-1.97**	-1.67*	1.74*	-2.21**
β_{fe}	3.58	-1.72	-1.43	-0.08	2.79	0.01
t	3.43***	-1.55	-1.56	-0.06	0.86	0.11
R^2	0.3	0.27	0.28	0.43	0.34	0.43
N	255	252	156	156	157	156

Table 5: Impact of exposure to foetal starvation by whether exposure occurred in first trimester: Mean and regression results

Gender (Sample size)	Indicator	Mean: Control	Mean: Study	β	t	R^2
Male (254)	Mid-upper arm circumference	23.3	25.2	3.14	2.72***	0.23
Female (157)	Mid-upper arm circumference	21.3	21.6	1.59	1.68*	0.36
	Waist circumference	66.9	65.1	7.10	2.08**	0.52
	Hip circumference	84.0	79.6	11.72	1.93*	0.28
	Skeletal muscle: Arm	30.3	33.3	-2.90	-1.96*	0.40

Results of the regression models reveal (Table 5) that exposure to foetal starvation in the first trimester actually increases mid-arm circumference for both males and females, with the difference being significant at 10% level. Females exposed to foetal starvation in the first trimester are also observed to be broader in the waist and hip, but have lesser skeletal muscle development in the arm. The differences are significant at 5%, 1% and 1% level, respectively.

4.3 Discussion: Programmed for starvation?

Overall, we find little evidence in support of the FOH. In fact, the results show that nutritional deprivation at the foetal stage actually leads to better anthropometric indicators in the case of both males and females. How do we explain this observation that contrary to the expectation on support of FOH? When the hypothesis was put forward by Barker and his associates, the common belief was that chronic diseases derived from an interaction between genotype and adult lifestyle. This led Barker to focus on the negative aspects of foetal starvation. Subsequent studies of the FOH undertook the survey in situations and areas where the food deficit was an aberration, rather than a permanent feature of the economy.

In recent years, however, the perspective on such type of 'genetic programming' has changed. Bateson and Gluckman (2011), for instance, stress on the long term advantages deriving from foetal energy deficits. Their theory of "Predictive Adaptive Response" (PAR) argues that developing organisms receive information about the quality of the external environment and, in response, formulate predictions as to future ecological

conditions. In case of human foetus, this may comprise prioritized growth of specific organs (like heart and brain) vis-a-vis other tissues (like pancreas and muscles). This prepares the organisms optimally for its latter life (Gluckman et al. 2007). Environmental cues suggesting that the subsequent post natal environment will have certain characteristics, may induce genes to express themselves to the phenotype that best fits this expected environment (Gluckman and Hanson 2005). An example is that poor prenatal nutrition may suggest to the foetal body that it will encounter periods of malnutrition in later life as well. Resulting epigenetic adaptations allow the body to acquire a heightened tendency to store fat. This is beneficial if the person suffers from a regular deficiency of nutrition.

The implication for subsequent health of adults is as follows: if the prediction of the future proves correct, the organism will be healthy, while an incorrect prediction is likely to result in ill health. It is of course true that for PAR to work for human conditions, the anticipatory predictions generated during the brief foetal stage must hold successfully for several decades. Consequently, the PAR theory has not succeeded in passing in what its proponents call its acid test—"whether the small baby will be better suited to the poor environment predicted by the mother's low nutritional level than a big baby" (Bateson and Gluckman 2011: 74).

> "Studies based on animals have shown that those malnourished in early life do worst in all adult environments, but particularly do so in tough conditions. Contradictory evidence is also available in the reverse. In the 1930s, classic studies of rodents showed that calorific restriction increased longevity. Recent studies confirmed that slower postnatal growth following adequate foetal growth

likewise increased longevity. Contrary to the PAR theory, this mismatch is 'beneficial' for health (Wells, 2012: 233)."

4.4 Evidence for PAR

But, if the signals of nutritional deficiency received at the foetal stage *does* match the future environment to which the adult organism is exposed to, the nutritional deficiency at the foetal stage can be said to prepare the child for the nutritional deprivation that it will be subsequently exposed to during his/her subsequent life history. This hypothesis was tested using our data.

The data on Standard of Living (SLI) was used to sub-divide the sample into three groups—High, Medium and Low. Since we had collected data on nine SLI, this means that we had used nine categories. Using the first letter for past SLI and the second letter for current SLI these categories are HH, HM, HL, MH, MM, ML, LH, LM and LL. To sharpen the focus we concentrated all medium SLi cases (that is all, iM and Mj cases) so that we had only fur categories left—HH (households with high SLI throughout reference period), HL (households with high SLI initially, but whose economic status declined subsequently), LH (households with low SLI initially, but whose economic status improved subsequently) and LL (households with low SLI throughout reference period). The signal received at the foetal stage about nutrition scarcity is correct in two cases (LL and HL) and wrong in the remaining two cases (HH and LH). So respondents exposed to foetal starvation will be better off than the control group in the LL and HL groups, while the reverse will hold for the two groups HH and HL. After checking that the measures

were below WHO and other norms—so that higher values imply worse outcomes—we compared mean values for the different measures to see whether our hypothesis was valid or not. The results are summarized in table 6. The letters Y and N indicate whether the hypothesis holds or not, respectively. The last two columns sum up the evidence—in terms of number of cases and the percentage of cases when our hypothesis holds.

We can see that the hypotheses holds in quite a few cases. The acid test is, of course, HL. This represents a situation when economic status of an affluent household deteriorates. While we would expect, a priori, that such households have worse outcomes, Par implies that respondents to foetal starvation should be more suited to cope with declining fortunes. This is observed in one third of the measurements for males; in case off female respondents, the proportion of cases supporting PAR is even higher (73 per cent). PAR is found to hold among 60 (male) and 53 (female) percentage of respondents from LL households.

On the contrary, respondents *not* exposed to foetal starvation are better off with respect to the study group if they belong to HH and LH households, possibly because the signal of nutrition deficit environment does not hold in their latter life. This is observed in 73 and 40 per cent (male and female, among LH households) and 67 per cent (both male and female, among HH households) of measures. This provides some evidence that PAR may hold for human beings.

Table 6: Summary of evidence testing Predictive Adaptive Response

Outcome	MALE				FEMALE			
	HL	LH	HH	LL	HL	LH	HH	LL
Body Mass Index	N	**Y**	**Y**	N	**Y**	**N**	**N**	**N**
Dystolic Blood Pressure	N	N	N	**Y**	**Y**	**Y**	**Y**	**Y**
Systolic Blood Pressure	**Y**	**Y**	N	**Y**	**Y**	**Y**	**N**	**Y**
Triceps	N	**Y**	**Y**	N	**Y**	**N**	**Y**	**Y**
Biceps	**Y**	N	**Y**	**Y**	**Y**	**Y**	**Y**	**N**
Height	**Y**	**Y**	N	**Y**	**N**	**Y**	**Y**	**Y**
Mid-Upper Arm Circumference	**Y**	**Y**	**Y**	N	**N**	**Y**	**N**	**Y**
Body Density	N	**Y**	**Y**	**Y**	**Y**	**N**	**Y**	**N**
Body Age	N	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**N**
Weight	N	**Y**	N	**Y**	**N**	**N**	**N**	**Y**

Outcome	MALE				FEMALE			
	HL	**LH**	**HH**	**LL**	**HL**	**LH**	**HH**	**LL**
Pulse Rate	**-**	**Y**	**-**	**-**	**-**	**-**	**-**	**-**
Suprialiac skinfold thickness	N	**Y**	**Y**	N	**N**	**N**	**Y**	**N**
Subscapular skinfold thickness	N	**Y**	**Y**	**Y**	**Y**	**N**	**Y**	**N**
Waist-Hip Ratio	N	N	N	N	**Y**	**N**	**Y**	**N**
Waist Circumference	N	N	**Y**	N	**Y**	**N**	**Y**	**Y**
Supporting PAR: Number	**5**	**11**	**10**	**9**	**11**	**6**	**10**	**8**
Supporting PAR: %	**33**	**73**	**67**	**60**	**73**	**40**	**67**	**53**

5. Conclusions: From FOH to PAR

"Over time, (the backbone of the (foetal origins) theory has graduated from hypothesis to accepted biology even though the details remain controversial" (Adair and Prentice, 2004: 191). The two studies analysing the health impact of Ramadan fasting are very important in revealing that even a mild nutritional shock can have serious long term health impacts. While most researchers have examined the impact of severe and rare effects like famines, Almond and Mazumdar (2011) and Ewjik (2011) shows that the foetal origin effect may operate for milder effects as well. This implies that common disruptions to nutrition during pregnancy that is quite common among different economic classes in developing countries (though for different reasons) – like meal skipping – may cause serious health impacts that should be studied. This implies that the FOH has important policy implications for reproductive health and its long term effect on the economy.

The initial studies of the FOH, however, are carried out in UK (by Barker and his associates) and supported by studies in developed countries—like Banerjee's study of army conscripts in France (Banerjee et al., 2010) and the Dutch famine (Hoek et al., 1998). Subsequent studies shifted their attention to developing countries. But the shock was generally a temporary aberration in the normal course of events. For instance, St. Clair et al.'s attempt to verify the FOH in China was based on an extreme shock in the form of a famine (St. Clair et al., 2005). Similarly, in studies of the impact of Ramadan starvation (Almond and Mazumdar, 2011; Ewijk, 2011), the food deficit was only for a month. In other words, *the signal received during the foetal period was a maladjusted*

signal, triggering the starvation gene to function in a context of food availability. The subsequent impact resulted from the subsequent environmental reality not matching with the signal received as a foetus. Consequently, the plasticity of the organism led to the foetus *maladapting*; it is this maladaptation that leads to cardiac, metabolic, nephrological and other diseases at the mid-life stage (or even to poor anthropometric development).

Our study, however, sheds light, on what happens when the signal of food deficit is consistent with the future environment, correctly predicting that the organism will face chronic nutrition deficiency after birth. The PAR hypothesis argues that, in such cases, the FOH will break down and those programmed for starvation before birth will be fitter individuals—and this is what we find in our study. Our study, therefore, neither proves nor disapproves the FOH, but it does provide some evidence for the PAR.

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