Does foetal starvation matter in human capital formation?
Examining the foetal origin hypothesis in the chronically under developed areas of *Sunderban*, India

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Abstract

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. Some studies have argued that the health status of adults is programmed even earlier —*in utero* to be precise. The fetal origin hypothesis (Barker, 1990) 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 instead of a fadeout of early shocks (as in the Grossman model), the impact of prenatal shocks may persist till adulthood. Studies have shown that individuals exposed to foetal starvation are more likely to suffer from poor physical development, cardiac diseases, renal ailments, diabetes, etc. Thus, the potential for complementarities between prenatal shocks and subsequent investments underscore the persistent importance of a 'good start'.

Although it is now accepted that the traditional 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), empirical studies to verify the foetal origin hypothesis suffers from some methodological problems. Initial studies by David Barker and his colleagues at the MRC Environmental Epidemiological Unit in Southampton, UK were based on correlation analysis. Subsequent studies (Hoek et al., 1998; St. Clair et al., 2005) took weight at birth as

proxy for foetal health, despite evidence showing that it may not be a comprehensive or sensitive measure.

The problem with attempts to verify the foetal origin hypothesis is that such studies have to focus on two widely spread out time periods—pre natal stage and adulthood. Short of time traveling, it is not easy to obtain reliable data on *both* these time periods, creating an identification problem. 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).

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. Plotting birth data against occurrence of Ramadan in a particular year, researchers are able to distinguish between children who were in utero during Ramadan, and those who were not. These studies found that being exposed to Ramadan in early pregnancy has significant adverse health effects in middle age.

These studies 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 mothers fasted, resulting in the inclusion of control group members into the study group.

In the present study we undertook a survey of women who had been pregnant between 1993 and1998. The survey was undertaken in the chronically poor and under-developed 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.

After testing the reliability of their memory through a recall test, we ascertained how many of them had fasted during pregnancy. Using the date of birth of children born in this period we identified how many of them had been in utero during the fasting period. Combining the two

pieces of information provides a more reliable method of dividing the sample into study and control groups.

The primary hypothesis of the work was:

> Does exposure to *in utero* starvation produce long term health effects manifested in anthropometric measurements?

We tested whether such effects were conditioned by trimester of exposure.

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

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) Blood pressure and pulse.
6) Resting metabolism, Body density, Body Mass Index and Body age.

In all, 27 indicators of health status were used.

Econometric analysis—controlling for household variables like education of parents, (past and current) occupation of parents, (past and current) and standard of living index, along with age, gender, education and occupation of respondent—was undertaken for the entire sample and for male and female respondents respectively. Results show that foetal starvation, particularly during the first trimester, does impact some of these indicators of physical development.

We conclude by pointing out that the impact of foetal malnutrition—a common phenomenon in India—will reduce productivity of the workforce, divert resources from productive use and lower the potential growth rate of the economy.