Dynamic Complementarity in Child Growth

A Research Sketch

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Summary

Using Cunha and Heckman's (2007) capacity formation model, I will test for dynamic complementarity between nutrition *in-utero* and in childhood using data on child height from Indonesia. Testing dynamic complementarity requires two instruments, one for *in-utero* nutrition and another for childhood nutrition. My first instrument will be *in-utero* exposure to Ramadan, and my second, seasonal rainfall. I will also test for endogenous parental responses to *in-utero* shocks, allowing me to bound my estimate of dynamic complementarity from above or below.

Motivation

Dynamic Complementarity

Accumulation is no longer an acceptable model for human capital. The realization that *in-utero* shocks have large effects throughout the life cycle has revolutionized our understanding of capacity formation (Almond and Currie 2011). New evidence has required the development of new theory, such as Cunha and Heckman (2007), which can explicitly model critical periods of development. New theory has generated new questions, among which is the degree of complementarity of inputs between developmental stages. Under dynamic complementarity, the productivity of an investment in childhood is increasing in the level of investment *in-utero*.

The presence dynamic complementarity has implications for the optimal timing of policy interventions, as argued extensively by Heckman (2006, 2007), but also for the evolution of inequality. Under dynamic complementarity, differences in initial endowments, rather than washing out as in the Grossman model, will be reinforced over a lifetime. Despite its importance, the presence of dynamic complementarity has, thus far, suffered few direct tests. I will test the specific case of dynamic complementarity in child growth. Child height is a strong indicator of health, and we have strong a-priori reason to believe that nutritional inputs exhibit complementarity between gestation and early childhood.

The Thrifty Phenotype

Infant growth, with sufficient nutrition, follows a well-defined trajectory. Malnutrition causes departure from a given trajectory, slowing the velocity of growth. Following malnutrition, increased nutrition may result in accelerated growth. This is known as "catch-up" growth, as the child reaches a higher growth curve, closer to their biological potential (Tanner 1990). The biological potential has a genetic component, but also an epigenetic component (Furey and Sethupathy 2013). The thrifty phenotype hypothesis suggests that poor nutrition *in-utero* will result in a phenotype both shorter and with a slower metabolism (Hales and Barker 1992). Such a child will then be better suited for an environment with low nutrient availability.

Although epigenetic switches are not yet well understood, they are for the most part determined *in-utero*. Thus, gestation offers a unique period of programming, in which the child's potential growth path may be set.

¹For example, DNA methylation resets with each generation and the levels of methylation in somatic tissues is generally determined by *in-utero* conditions (Cedar and Bergman 2012).

To what degree this potential is reached is determined by net-nutrition during childhood. While oversimplified, this narrative can be easily represented within the capability-production framework, as complementarity of nutritional inputs between gestation and childhood.

Theory

Consider the following model of capacity formation, from Currie and Almond (2011):

$$h = A[\gamma I_1^{\theta} + (1 - \gamma)I_2^{\theta}]^{\frac{1}{\theta}}$$

where h is child height, I_1 is nutrition in the pre-natal period and I_2 is nutrition post-birth. Consider a positive shock to *in-utero* nutrition, μ_g :

$$h = A[\gamma(I_1 + \mu_g)^{\theta} + (1 - \gamma)I_2^{\theta}]^{\frac{1}{\theta}}$$

Dynamic complementarity is characterized by:

$$\frac{\partial^2 h}{\partial \mu_q \partial I_2} > 0$$

Parental Investments

Suppose now that that μ_g also affects I_2 , through parental investments. We obtain:

$$\frac{\partial^2 h}{\partial \mu_q \partial I_2} = \frac{\partial^2 h}{\partial I_1 \partial I_2} + \frac{\partial^2 h}{\partial^2 I_2} \frac{\partial I_2}{\partial \mu_q}.$$

We see that $\frac{\partial^2 h}{\partial \mu_g \partial I_2}$ is no longer equal to the cross-derivative of I_1 and I_2 , making it a biased test of dynamic complementarity. However, with knowledge of the direction of parental responses, we can determine the direction of this bias, assuming that $\frac{\partial^2 h}{\partial^2 I_2}$ is weakly negative from diminishing marginal returns to investment within a period.

We see that if parental responses are reinforcing, then $\frac{\partial^2 h}{\partial \mu_g \partial I_2} > 0$ constitutes a severe test of dynamic complementarity, as the bias goes against our hypothesis. If parental responses are compensating, the opposite is true.

Identification

In order to test for dynamic complementarity, then, we need a quasi-random shock to *in-utero* nutrition and to childhood nutrition, as well as information on both child height and parental responses. Given this challenge, I take a pragmatic approach, appropriating identification strategies from previous studies and overlapping them in one population.

My setting is Indonesia from 1991-2015. My instrument for *in-utero* nutrition is exposure to Ramadan fasting *in-utero*. My instrument for nutrition in childhood is seasonal rainfall, which has a positive effect

²The effect of Ramadan *in-utero* on later life outcomes has been well explored, particularly in Indonesia. See Almond and Mazumder (2011); Savitri et al. (2014); Kunto and Mandemakers (2016); R. Van Ewijk (2011); R. J. Van Ewijk, Painter, and Roseboom (2013); Majid (2015).

on local incomes and food availability across rural Indonesia.³ By using a panel data-set, I will be able to account for one of the main concerns of rainfall shocks: selective migration.

Exposure to Ramadan depends on exact day of birth and can be disentangled from seasonal effects because the timing of Ramadan change each year, cycling through the solar calendar over the course of 30 years. Because the timing of Ramadan is known in advance, there could be selection out of pregnancy as women choose not to be pregnant during a time of fasting. Previous studies have found little evidence of selective pregnancy (Almond and Mazumder 2011; R. Van Ewijk 2011), and I will test this possibility by checking whether *in-utero* exposure to Ramadan is balanced across observables. Ramadan creates an intent-to-treat effect, as we cannot observe whether a woman chooses to fast during pregnancy.⁴

Data

For child health and household characteristics, I have the Indonesian Family Life survey, a longitudinal household survey with waves in 1993,1997, 2000, 2007 and 2014. For parental investments, I have the Indonesian Demographic and Health survey (DHS), a repeated cross-section which includes data on the duration of breastfeeding and child immunization rates over the same time period. The Global Historical Climatology Network Monthly provides monthly rainfall data from local meteorological stations.

Estimation

I will first confirm that *in-utero* exposure to Ramadan has the expected, negative effect:

$$hemo_i = \alpha + \beta z_i + \phi_t + \psi_m + \epsilon_i \quad (1)$$

where $hemo_i$ is the hemoglobin of infant i, 5 z_i is exposure to Ramadan in-utero, and ϕ_t and ψ_m are year and month fixed-effects.

Then I will estimate the parental responses to the Ramadan shock:

$$y_i = \alpha + \lambda z_i + \phi_t + \psi_m + \epsilon_i \quad (2)$$

where y_i is duration of breastfeeding. If $\lambda > 0$, then parental responses are compensating, and if $\lambda < 0$, they are reinforcing.

As my main specification, I would estimate the effect of rainfall during childhood on height-for-age as mediated by *in-utero* exposure to Ramadan:

$$h_i = \alpha + \beta z_i + \gamma r_i + \delta r_i * z_i + \phi_t + \psi_m + \epsilon_i \quad (3)$$

where h_i is child height-for-age and r_i is a statistic of rainfall during childhood. Dynamic complementarity would imply $\delta < 0$. As discussed above, we can determine the severity of this test by the sign and magnitude of λ in equation (2).

Future Steps

In order to operationalize this framework, I need to determine how I will measure exposure to Ramadan, as the effect of Ramadan may depend on the trimester of exposure. I also need to consider the effect of extreme rainfall events, like drought and flood. Rainfall exhibits a non-linear relationship with crop yields, and also affects the disease environment.

³The effect of local rainfall on food production, adult height and child health has been established in previous studies. See Levine and Yang (2014); Maccini and Yang (2009); Cornwell and Inder (2015).

⁴For context, Joosoph et. al. (2004) found that from a sample of pregnant Muslim women in Singapore only 13% percent did not fast during Ramadan.

⁵The advantages of using infant hemoglobin to measure *in-utero* nutrition is explained in section 2.3 my MA thesis. See https://jrmcirwin.github.io/nut-trade/write-up/write-up.pdf.

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