Dynamic Complementarity in Child Growth

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# Summary

Using Heckman and Cunha’s (2007) capacity formation model, in conjunction with the bio-medical literature on the thrifty phenotype hypothesis, I test for dynamic complementarity between nutrition *in-utero* and nutrition 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 is *in-utero* exposure to Ramadan, and the second is seasonal rainfall. I 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 provides an explicit framework for critical periods of development. New theory has generated new questions, among which is the degree of complementarity of inputs between developmental stages. We know that having a good *in-utero* experience improves adult health, but how does having a good *in-utero* experience affect the response to health shocks in later periods. 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. 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 as explained below, we have strong *a-priori* reason to believe that nutritional inputs exhibit dynamic 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 of a slower metabolism and less growth in height (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* .[[1]](#footnote-25) Thus, gestation offers a unique period of programming, in which the child’s potential growth path is determined. To what degree this potential is reached is determined by net-nutrition during. While oversimplified, this narrative can be easily represented within the capability-production framework, as dynamic complementarity of nutritional inputs between gestation and childhood.

# Theory

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

where is the adult height of an individual, is nutrition in the pre-natal period and is nutrition post-birth. Assume that , and . Consider a positive shock to nutrition, :

Dynamic complementarity is characterized by:

## Parental Investments

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

We see that is no longer equal to the cross-derivative of and , making it a biased test of dynamic complementarity. However, with knowledge on the direction of parental responses, we can determine the direction of this bias, assuming that is weakly negative from diminishing marginal returns to investment within a period.

Using this decomposition, we see that if parental responses are reinforcing, then constitutes a severe test for 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 a quasi-random shock to childhood nutrition. As outcome variables, we need both child height and parental responses. I take a pragmatic approach to this challenge.

I appropriate identification strategies from previous studies and overlap them in one population. My setting is Indonesia from 1991-2015. My instrument for *in-utero* nutrition is exposure to Ramadan fasting *in-utero*.[[2]](#footnote-29) My instrument for nutrition in childhood is seasonal rainfall, which has a positive effect local incomes and food availability across Indonesia.[[3]](#footnote-30) 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 women choose to fast during pregnancy.[[4]](#footnote-31)

# 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 responses, 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. The Indonesian DHS has this information for 1991, 1994, 2002 and 2007. 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:

where is the hemoglobin of infant , is exposure to Ramadan *in-utero*, and and are year and month fixed-effects.

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

where is duration of breastfeeding. If , then parental responses are compensating, and if , they are reinforcing.

As my main specification, I would estimate the effect of rainfall during childhood on height as mediated by whether the child was exposed to Ramadan *in-utero*:

where is child height-for-age and is a statistic of rainfall during childhood. Dynamic complementarity would imply .

# Future Steps

In order to operationalize this framework, I need to determine how I will measure exposure to Ramadan, as the trimester of exposure to Ramadan may affect its effect. I also need to consider the effect of extreme rainfall events, like drought and flood.

# References

Almond, Douglas, and Janet Currie. 2011. “Killing Me Softly: The Fetal Origins Hypothesis.” *Journal of Economic Perspectives* 25 (3): 153–72.

Almond, Douglas, and Bhashkar Mazumder. 2011. “Health Capital and the Prenatal Environment: The Effect of Ramadan Observance During Pregnancy.” *American Economic Journal: Applied Economics* 3 (4): 56–85.

Cedar, Howard, and Yehudit Bergman. 2012. “Programming of DNA Methylation Patterns.” *Annual Review of Biochemistry* 81. Annual Reviews: 97–117.

Cornwell, Katy, and Brett Inder. 2015. “Child Health and Rainfall in Early Life.” *The Journal of Development Studies* 51 (7). Taylor & Francis: 865–80.

Cunha, Flavio, and James Heckman. 2007. “The Technology of Skill Formation.” *American Economic Review* 97 (2): 31–47.

Currie, Janet, and Douglas Almond. 2011. “Human Capital Development Before Age Five.” In *Handbook of Labor Economics*, 4:1315–1486. Elsevier.

Furey, Terrence S., and Praveen Sethupathy. 2013. “Genetics Driving Epigenetics.” *Science* 342 (6159). American Association for the Advancement of Science: 705–6.

Hales, C. Nicholas, and David JP Barker. 1992. “Type 2 (Non-Insulin-Dependent) Diabetes Mellitus: The Thrifty Phenotype Hypothesis.” *Diabetologia* 35 (7). Springer: 595–601.

Heckman, James J. 2006. “Skill Formation and the Economics of Investing in Disadvantaged Children.” *Science* 312 (5782): 1900–1902.

Heckman, James J., and Dimitriy V. Masterov. 2007. “The Productivity Argument for Investing in Young Children.” *Applied Economic Perspectives and Policy* 29 (3). Oxford University Press: 446–93.

Joosoph, J., J. Abu, and S. L. Yu. 2004. “A Survey of Fasting During Pregnancy.” *Singapore Med J* 45 (12). Citeseer: 583–86.

Kunto, Yohanes Sondang, and Jornt J. Mandemakers. 2016. “The Effect of Prenatal Ramadan Exposure on Child Health in Indonesia: A Longitudinal Perspective.” PhD Thesis, Petra Christian University.

Levine, David I., and Dean Yang. 2014. “The Impact of Rainfall on Rice Output in Indonesia.” National Bureau of Economic Research.

Maccini, Sharon, and Dean Yang. 2009. “Under the Weather: Health, Schooling, and Economic Consequences of Early-Life Rainfall.” *American Economic Review* 99 (3): 1006–26.

Majid, Muhammad Farhan. 2015. “The Persistent Effects of in Utero Nutrition Shocks over the Life Cycle: Evidence from Ramadan Fasting.” *Journal of Development Economics* 117. Elsevier: 48–57.

Savitri, Ary I., Nasim Yadegari, Julia Bakker, Reyn JG van Ewijk, Diederick E. Grobbee, Rebecca C. Painter, Cuno SPM Uiterwaal, and Tessa J. Roseboom. 2014. “Ramadan Fasting and Newborn’s Birth Weight in Pregnant Muslim Women in the Netherlands.” *British Journal of Nutrition* 112 (9). Cambridge University Press: 1503–9.

Tanner, James M. 1990. *Foetus into Man: Physical Growth from Conception to Maturity*. Harvard University Press.

Van Ewijk, Reyn. 2011. “Long-Term Health Effects on the Next Generation of Ramadan Fasting During Pregnancy.” *Journal of Health Economics* 30 (6). Elsevier: 1246–60.

Van Ewijk, Reyn JG, Rebecca C. Painter, and Tessa J. Roseboom. 2013. “Associations of Prenatal Exposure to Ramadan with Small Stature and Thinness in Adulthood: Results from a Large Indonesian PopulationBased Study.” *American Journal of Epidemiology* 177 (8). Oxford University Press: 729–36.

1. For 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). [↑](#footnote-ref-25)
2. The 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). [↑](#footnote-ref-29)
3. The 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). [↑](#footnote-ref-30)
4. For context, Joosoph et. al. (2004) found that from a sample of pregnant Muslim women in Singapore only 13% percent did not fast during Ramadan. [↑](#footnote-ref-31)