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Transgenerational effects of childhood conditions on third generation health and education outcomes

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ABSTRACT

This paper examines the extent to which pre-puberty nutritional conditions in one generation affect productivity-related outcomes in later generations. Recent findings from the biological literature suggest that the so-called slow growth period around age 9 is a sensitive period for male germ cell development. We build on this evidence and investigate whether undernutrition at those ages transmits to children and grandchildren. Our findings indicate that third generation males (females) tend to have higher mental health scores if their paternal grandfather (maternal grandmother) was exposed to a famine during the slow growth period. These effects appear to reflect biological responses to adaptive expectations about scarcity in the environment, and as such they can be seen as an economic correctional mechanism in evolution, with marked socio-economic implications for the offspring.

1. Introduction

Inequalities in health and social status tend to persist across generations, but the extent to which this is a causal process is still an open question. Do strong intergenerational correlations of health and social status variables imply that human capital shocks transmit from parents to children and grandchildren? To find an answer to this question, several recent studies have examined the intergenerational effects of policy changes (see e.g. Oreopoulos et al., 2006; Dahl and Lochner, 2012; Black and Devereux, 2011), while others have focused the effects of health-related shocks on child outcomes (Almond et al., 2012; Andreella et al., 2014). Yet, multigenerational studies spanning more than two generations are rare in social sciences and the existing studies mostly investigate the persistence of socio-economic variables (Lindahl et al., 2014; Sacerdote, 2005; Behrman and Taubman, 1985; Clark, 2014). However, as shown in this paper, studies with more than two generations can be useful to separate the effects of biological and social processes. From a policy perspective it is essential to understand whether human capital shocks and investments causally affect later generations. Knowing that there are intergenerational returns would imply that the costs and benefits of any policy measure had to be reevaluated to take their long-term effects into account.[1] In this paper, we take a step into

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[1] See also Mare (2011) for a discussion on the importance of multigenerational studies.

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this direction by providing evidence of environmentally induced transgenerational biological effects of adolescent undernutrition on children and grandchildren.

While transgenerational effects of nutritional deprivation during adolescence may seem far-fetched from the point of view of a social scientist, there exists ample evidence from mice models (and some on humans) showing that the effects of nutritional shocks can indeed persist for several generations. For example, Zamenhof et al. (1971) and Cowley and Griesel (1966) have shown that if rats are malnourished before or during gestation, brain sizes, maturation and cognitive performance of two subsequent generations are reduced even if all descendants are fed a normal diet. More recent evidence shows that the offspring of male mice, who consumed a low protein diet during late childhood (from weaning until sexual maturity), have elevated expression of many genes in liver tissue and exhibit changes in cytosine methylation (Carone et al., 2010). Similarly, it has been shown that the offspring of Holocaust survivors had altered gene expression levels related to cortisol (Yehuda et al., 2015). Many studies argue that the most likely mechanism behind such phenomena are environmentally induced but heritable changes in the epigenome (Rakyan et al., 2003; Ng et al., 2010). Such heritable epigenetic modifications may depend on the sex of the parent who transmits it and can lead to transgenerational non-genetic inheritance of lifetime experiences (Hochberg et al., 2011).[2]

Evidence on whether the above findings on mice translate to human probands is rare. Studies focusing on later-life health and cognitive outcomes among offspring have to rely on non-experimental data, which leads to identification problems if parental conditions are endogenously related to unobserved characteristics that also influence outcomes in another way. To identify causal biological effects of food deprivation on subsequent generations, one needs to observe an exogenous shock in nutrition in the first generation as well as the relevant outcomes in subsequent generations. By now, a consensus has emerged that the study of transgenerational epigenetic inheritance that is induced by environmental shocks early in life requires the observation of at least three generations (see Grossniklaus et al., 2013). After all, effects of parental exposure to environmental shocks on their children's health may not only result from inheritance but can have many other biological and behavioral explanations. Moreover, environmental shocks during pregnancy affect the mother, the fetus, *and* the fetus's primordial germ cells that will produce the grandchildren of the mother.[3] Along these lines, it is particularly interesting to consider environmental shocks that may occur before reproductive ages, and to simultaneously analyze any effects of conditions faced by each grandparent.

The transmission of a nutritional shock over three generations of humans has only been studied in a single line of research papers using historical harvest data and church registers from the remote Överkalix region and connected regions in Northern Sweden (Bygren et al., 2001; Kaati et al., 2002, 2007; Pembrey, 2002, 2010; Pembrey et al., 2006; Kaati, 2010). Due to its pathbreaking nature, the sequence of studies based on the Överkalix data has evoked great interest in the biological literature (see e.g. Zeisel, 2007; Gräff and Mansuy, 2008; Masterpasqua, 2009; Francis, 2011; Low et al., 2012, and Grossniklaus et al., 2013). The authors find that low paternal grandfather's food supply in the years just before adolescence is associated with a lower mortality risk of grandsons, while low paternal grandmother's food supply is linked to a lower mortality risk of their granddaughters. Low food supply during the paternal grandfather's pre-puberty phase is also associated with lower third generation mortality from cardiovascular diseases, and higher diabetes mortality with a surfeit of food. The authors postulate that these effects are triggered by methylation of epigenetic marks during the ancestor's slow growth period (SGP) which takes place at ages 8–10 for girls and at ages 9–12 for boys. The SGP is a sensitive period for the methylation of male sperm, and the authors hypothesize that the resulting methyl tags are transmitted to subsequent generations via epigenetic imprinting (Pembrey et al., 2006). This mechanism could be an evolved transgenerational response to developmental conditions. Adverse grandparental SGP conditions may then cause an improvement in the offspring's capability to face certain living conditions. Note that the sign of the effect within the first generation members' lives (as typically found in single-generation studies of long-run effects of early-life conditions) is then opposite to the sign of effects on certain later generations.[4]

In this paper we build on the above finding that the slow growth period might be a critical period for sperm development implying that shocks during that age period can have a biological effect on the descendants. Our goal is hence to investigate whether a nutritional shock at that age causally affects health, schooling and mental health outcomes of later generations. Specifically, we examine

[2] Epigenetic inheritance is an increasingly accepted explanation for why nutritional shocks may persist across generations and one which has become a focal point in biological and epidemiological research on the long run effects of nutrition, stress and other early life circumstances (see Gräff and Mansuy, 2008; Masterpasqua, 2009; Lundborg and Stenberg, 2010; Hochberg et al., 2011; Kuzawa and Thayer, 2011, and Low et al., 2012, for overview articles).

[3] For a study on the intergenerational effects of in-utero exposure to undernutrition see Lumey (1992).

[4] While the studies have evoked great interest, it is fair to state that from a statistical point of view the analysis of the data has some limitations. The studies consider up to three different degrees of food availability, among six ancestors (4 grandparents and 2 parents), during several parental and grandparental pre-adult age periods, and they examine their associations with several outcomes among grandchildren distinguished by sex. In the absence of strong theoretical priors, this amounts to the detection of a large number of associations. Even if no transgenerational transmission exists, a statistical analysis would typically result in a few numbers of false positives. Simply put, under the null hypothesis of no effects, and with a 5% test size, one finds an effect in 5% of the cases. Furthermore, samples of third generation members are rather small, ranging from ca. 100 to 300 individuals. For such a large number of parameters and given the small sample size, it is possible that the authors found effects that prove unimportant in other samples. Hence, in a sense this paper also aims to assess the external validity of these findings.