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Barriers to Prosperity: Parasitic and Infectious Diseases, IQ, and Economic Development

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Summary. — IQ scores differ substantially across nations. This study argues that cross-country variations in IQ scores, to a large extent, reflect the burden of parasitic and infectious diseases (PIDs) and iron and iodine deficiency (IID) in infancy and *in utero*. Furthermore, it is shown that the prevalence of health insults, through the channel of cognitive ability, is influential for the level as well as the growth in productivity across the world. Using data for 181 countries and an instrumental variable approach, regressions reveal that the prevalence of PID–IIDs is influential for growth and income inequalities globally.
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Key words — cross-country income inequality, parasitic and infectious diseases, cognitive ability

1. INTRODUCTION

The relationship between health, growth, and development is controversial. Some studies argue that health is important for income (Andersen, Dalgaard, & Selay, 2011; Arora, 2001; Bloom, Canning, & Fink, 2009; Cervellati & Sunde, 2005; Chakraborty, 2004; Iyigun, 2005; Kalemli-Ozcan, 2002; Kalemli-Ozcan, Ryder, & Weil, 2000; Lorentzen, McMillan, & Wacziarg, 2008; Sachs, 2001; Soares, 2005; Zhang & Zhang, 2005), while others argue that the income effects of improved health are negative (Acemoglu & Johnson, 2007), small (Ashraf, Lester, & Weil, 2008; Weil, 2007) or non-linear (see e.g., Cervellati & Sunde, 2011; Hansen, 2012).

This paper suggests that health is a crucial determinant of cross-country income inequalities through the channel of cognitive skills. Referring to recent medical evidence showing that health *in utero* and early childhood is decisive for cognitive ability during adulthood Heckman (2007) argues that the marked cross-country income inequality is a result of different exposures to parasitic and infectious diseases (PIDs) and the prevalence of iron and iodine deficiencies (IIDs). A high burden of PID–IIDs severely impairs average cognitive ability, which in turn can significantly and permanently reduce the quality of human capital. Since most of the brain's development occurs from half way through pregnancy until a child reaches the age of two, this period is by far the most important for cognitive development (Niehaus *et al.*, 2002). Adequate development of the brain during infancy requires an adequate supply of energy and oxygen, bearing in mind that the brain in a newborn baby uses at least 87% of the body's energy budget compared to only 20% in adulthoods (Drubach, 2000; Holliday, 1986).

Most infants and young children in countries with a high prevalence of PID–IID carry one or more parasites in their body during most of their childhood, have frequent bouts of diarrhea, and often suffer from IID (Watkins & Pollitt, 1997). Furthermore, the burdens from these diseases are often compounded by generally poor nutrition and, in some countries, malnutrition due to famine etc. Under these conditions children's cognitive development is at risk of being impaired further by an inadequate energy supply to the brain while they are growing up. As shown in the next section, reduced cognitive ability due to PID–IID goes a long way in explaining why the average IQ in many tropical countries is often found to be below 70 (Lynn & Vanhanen, 2006).

Furthermore, it is suggested that the level and the growth in per capita income are strongly related to PID–IIDs through the channel of cognitive ability. Previous studies have demonstrated a positive relationship between cognitive ability and income or growth but have not investigated the reasons behind the differences in cognitive ability and have abstained from using external instruments. Using Bayesian averaging, Jones and Schneider (2006) find IQ to be the most robust predictor of cross-country growth differences; however, due to the nature of their study, they did not investigate the underlying cause of IQ variations. This study goes a step further by arguing that PID–IID impinges on cognitive development that in turn slows economic development through the following causal chain:

$$\left.\begin{array}{l}\text{Parasitic and infectious disease}\\ \text{and iron and iodine deficiency}\end{array}\right\} \rightarrow \text{impaired congitive skills} \rightarrow \text{lower income per capita.}$$

The hypothesis proposed in this paper extends the conventional explanation in which health insults have adverse economic outcomes due to high absence from work, reduced productivity, and low quantity and quality of educational attainment because of low focus at school, absence from

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school, and low returns from investment in schooling (see for instance Ashraf *et al.*, 2008; Gallup, Sachs, & Mellinger, 1999; Sachs, 2001; Sala-i-Martin, 2005; Weil, 2007). Probably the study that comes closest to this research is that of Bleakley (2007), who finds that the hookworm eradication program in the US resulted in a marked relative, as well as an absolute, improvement in school enrollment, school attendance, and literacy in counties that previously suffered from high rates of hookworm disease. Similarly, Brinkley (1997) finds that the increasing prevalence of hookworm infection goes a long way in explaining the marked decline in agricultural productivity in the Southern states of the US in the period 1860–80. Finally, Carstensen and Gundlach (2006) show that malaria has been a more important factor behind cross-country economic inequalities than institutions.

This paper makes two contributions to the literature on growth and development. First, using three different indicators of cognitive ability it is shown that the burden of PID–IIDs in infancy and *in utero* is influential for cognitive abilities across nations. This issue is important since the alternative hypothesis in which cross-country variations in cognitive abilities are often attributed to schooling or inherited ability, (see for example Lynn & Vanhanen, 2006), has completely different policy implications than the hypothesis of this paper. The influence of disease and other factors on IQ are discussed and tested in Sections 2 and 3. As a second contribution, the influence of PID–IIDs, through the channel of cognitive ability, on the level and the growth in per capita income is tested using various instruments (Section 4). It is shown that cognitive ability, through the channel of PID–IIDs, goes a long way in explaining cross-country income inequalities.

Four sets of instruments are used for identification: (1) per capita foreign health aid to recipient countries; (2) pathogen (biological agent causing disease to its host) prevalence circa 1900; (3) pathogen prevalence circa 1940; and (4) ecozone variables representing the variety and density of pathogens. Health aid is a good instrument because, as demonstrated below, it is directed toward countries with the highest pathogen prevalence and yet it is likely to be strictly exogenous because other foreign aid indicators, which would perhaps be inversely related to per capita income, are uncorrelated with indicators of health insults as shown below. Pathogen prevalence circa 1900 and 1940 are likely to be exogenous and, at the same time, give an indication of the historical prevalence of pathogen stress. The key here is that cross-country per capita income inequality was not nearly as pronounced around 1900 and 1940 as it is today, as shown in Section 3(b), ensuring that pathogen prevalence, at that time, was unlikely to have been driven by income. Particular ecozone variables are useful instruments because pathogen prevalence, as argued in Section 3(b), is related to ecozones.

The next section discusses the medical evidence on the relationship between PID–IID and cognitive development *in utero* and during childhood, Section 3 tests for the influence of PID–IID on cognitive development and Section 4 shows the influence of PID–IID on cross-country income and growth inequalities through the channel of cognitive development. Section 5 concludes.

2. PID–IIDS AND COGNITIVE DEVELOPMENT [1]

Referring to recent medical and microeconomic research, this section argues that the burden of PID–IIDs is highly influential for cognitive development, where widespread PIDs include helminthic parasites such as schistosomes and tapeworms, protozoan parasites such as malaria and giardia, and viral and bacterial infections especially when they cause diarrhea (WHO, 2014). It is argued that the cognitive development of young children is highly sensitive to the presence of PIDs as their brains are highly sensitive to damage and biochemical changes. PID's manifest these effects in a number of ways depending on the type of parasite, parasite load, and site of infection. Of major concern is the development or exacerbation of malnutrition. In particular, repeated bouts of enteric infection result in intestinal injury and consequently malabsorption during the critical first years of childhood (Ijaz & Rubino, 2012). This can have detrimental effects on growth and cognitive development that extend long beyond the infection (Guerrant, Oriá, Moore, Oriá, & Lima, 2008; Ijaz & Rubino, 2012). A number of PIDs, particularly worms, cause intestinal bleeding that results in the loss of iron. This reduces the formation of hemoglobin leading to anemia (Caulfield, Richard, & Black, 2004) and exacerbates cognitive decline (Jáuregui-Lobera, 2014). PIDs may also directly affect the central nervous system, for example some helminth infections release toxins leading to biochemical changes (Holding & Snow, 2001) while other organisms such as bacteria or viruses (that cause meningitis) give rise to direct brain injury in many children (Olness, 2003). These biochemical changes along with immune excitation can lead to behavioral changes that include appetite loss (Holding & Snow, 2001) and therefore affect nutritional status.

One outcome of immune activation during infection is the production of cytokines. These have a wide range of effects that, in excess, result in pathology including changes to brain function (Clark, Alleva, & Vissel, 2010). One consequence may be damage to mitochondria (Singer, 2014) resulting in bioenergetic dysfunction. Moreover, cytokines can also enhance glucose uptake by cells (Shikhman, Brinson, Valbracht, & Lotz, 2001) contributing to hypoglycemic conditions (Bach *et al.*, 2013) and, therefore, a reduction in the host's energy budget (Eppig, Fincher, & Thornhill, 2010; Watkins & Pollitt, 1997).

The direct effects of PID are aggravated through malnutrition since a substantial proportion of malnutrition across the world is caused by impaired intestinal absorptive function resulting from multiple and repeated enteric infections that render children susceptible to repeated bouts of enteric infections (Guerrant *et al.*, 2008; Ijaz & Rubino, 2012). In fact, Guerrant *et al.* (2008) view malnutrition as an infectious disease and argue that the effects of heavy diarrheal burdens and multiple enteric infections in the early years of childhood extend long beyond the infection itself and may result in stunting and impaired cognitive development in affected children. Several studies find that IQ can be reduced by approximately 10 points in children who have been exposed to long spells of malnutrition (see, for discussion Guerrant *et al.*, 2008; Scrimshaw, 1998). Malnutrition often results in a reduction in brain size, and a reduction in the number of brain cells; both of which are associated with irreversible impaired cognitive ability (Levitsky & Strupp, 1995).

Turning to the IID family, iodine deficiency causes hypothyroidism, symptoms of which are extreme fatigue, goiter, mental retardation, and depression and is the leading cause of preventable mental retardation (Felig & Frohman, 2001). Iron deficiency in infancy and early childhood is found to impair cognitive development and IID during infancy and, particularly, *in utero* are highly influential for a child's cognitive development because, as noted earlier, the brain is growing fastest during this time (Scrimshaw, 1998). Iron deficiency in infancy and in early childhood is considered to be a key deterrent to

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