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Colonialism and genetics of comparative development☆

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ABSTRACT

This study argues that European colonial policies and former colonies' genetic variation (genetic distance to Europeans and genetic diversity) were interlinked. Over a prolonged period of time, populations that were genetically far from Europeans and had extreme levels of genetic diversity (e.g. in Sub-Saharan Africa and the Americas) adapted to environments that were significantly different from the climatic conditions of continental Europe. This resulted in a divergence in populations' resistance to infectious diseases and positive relationships between European settler mortality at the time of colonization, genetic distance to the technological frontier, and genetic diversity. I evaluate the consequences of the aforementioned relationships first, for the role of genetic distance and diversity in development (e.g. Spolaore and Wacziarg, 2009; Ashraf and Galor, 2013), and second, for studies that use European settler mortality as an instrument for institutions (e.g. Acemoglu et al., 2001). The results highlight a potential bias in the estimates of the effect of genetic distance and diversity on contemporary development in a sample of former colonies and suggest that the effect of these measures on current economic and institutional outcomes is indirect and works through Europeans' colonial policies.

1. Introduction

History matters. Empirical literature on comparative development provides two – perhaps complementary – explanations as to which aspects of human history are relevant for contemporary development. Some maintain that the relevance of history is mainly due to the persistence of institutions and emphasize the patterns of European colonialism as their major determinant (e.g. Acemoglu et al., 2001, 2014; Engerman and Sokoloff, 1997, 2011 La Porta et al., 1998). Others suggest that deep-rooted cultural and genetic traits – measured by genetic distance to the technological frontier and genetic diversity – explain poverty and prosperity, and, therefore, the roots of contemporary differences in economic performance can be found even in prehistoric times (e.g. Spolaore and Wacziarg, 2009, 2013; Ashraf and Galor, 2013).[1] This study connects and evaluates these explanations.

My argument begins with the observed associations between European settler mortality at the time of colonization and the genetic distance and diversity of former colonies' *indigenous* populations. Figs. 1 and 2 plot the relationships between European settler mortality rates, former colonies' genetic distance to Western Europe in 1500 (Fig. 1), and their genetic diversity in 1500 (Fig. 2).[2] Both figures show positive associations. This means

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[1] The general idea in this line of research is that populations' characteristics that are summarized by genetic distance and diversity have persisting effects on development. For example, Spolaore and Wacziarg (2009) document a negative cross-country relationship between genetic distance to the United States and contemporary income per capita (Fig. A.1 of Appendix A) and take this correlation as evidence of a *barrier* effect of cultural traits (e.g. beliefs, habits, customs) on the diffusion of development from Europe. Ashraf and Galor (2013) document a hump-shaped cross-country relationship between genetic diversity and income per capita (Fig. A.2 of Appendix A) and argue that very high and very low genetic diversity cause poverty. Section 2 reviews this literature.

[2] In a nutshell, genetic distance measures the extent of genetic variation between two currently separated populations, while genetic diversity measures the extent of genetic variation within a population. See Sections 3 and 4 for more detailed and technical definitions of these measures.

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that, on average, Europeans experienced higher mortality when they encountered populations that were genetically far from them, and when they confronted populations that had extreme levels of genetic diversity. Since settler mortality is among the key determinants of variation in Europeans' colonial policies (Acemoglu et al., 2001) Figs. 1 and 2 imply that Europeans' colonial policies and ex-colonies' genetic variation were interlinked.

The co-movement of European settler mortality and former colonies' aggregate genetic traits (distance and diversity) reflects the historical differences between the disease environments of Eurasia, Sub-Saharan Africa, and the New World (and the adaptation of the indigenous populations of these regions to their disease environments). A comparison of the experience of Europeans in Africa and the New World clarifies this point.

Since Homo sapiens originally evolved in East Africa and only a small fraction of them migrated out of this continent, sub-Saharan populations are genetically highly diverse and are genealogically least related to other populations (Henn et al., 2012). The disease environment of sub-Saharan Africa is significantly different from the rest of the Old World (Europe and Asia), which has a relatively more temperate climate. When Europeans came into contact with the populations of the west coast of Africa in the 15th century, their lack of immunity to African diseases such as malaria and tropical yellow fewer resulted in very high mortality among them. This provided Africa with a natural barrier against European penetration and settlement until the 19th century.

Europeans' experience in the New World was very different from their experience in Africa. The New World's indigenous populations were genetically closer to Europeans (compared to Africans) and were highly genetically homogeneous.[3] At the time of the arrival of conquistadors to South America, the disease environment of this region was generally favorable to Europeans. This was mainly due to the fact that Aztecs and Incas underwent their Neolithic transitions in environments that were devoid of animals suitable for domestication.[4] Thus, the "crowd epidemic diseases" of the Old World – which were mainly due to the close proximity of the Old World's animals and humans – were completely absent in this continent (Wolfe et al., 2009). However, the Old World's pathogens worked as colonists' *agents of conquest*, i.e., Amerindians' lack of immunity to diseases such as small pox and bubonic plague resulted in their eradication and ultimately the collapse of their civilizations (Diamond, 1997).[5] This relatively quick reduction in the size of the indigenous population resulted in shortage of labor for Europeans' colonial enterprise, which reinforced the incentives for the Atlantic slave trade. In fact, African pathogens that were brought to South America by slave ships were the major cause of mortality among Europeans in this continent as well.

This close relationship between the *colonists*' mortality and the aggregate genetic traits of the *colonized* populations poses a challenge to the both strands of literature mentioned before. Those studies that examine the role of genetic distance and diversity in development (e.g. Spolaore and Wacziarg, 2009; Ashraf and Galor, 2013) neglect an important aspect of historical differences between populations: their adaptation to continent-specific diseases, which became a relevant factor to development with the start of European expansion. This could bias the estimates of the effect of genetic distance and diversity on economic performance.[6] As an example, Europeans' colonial policies in Sub-Saharan Africa (which, among other factors, were determined by this region's hostile disease environment) affect sub-Saharan Africa's institutions and economic performance even in the contemporary era (Acemoglu et al., 2001; Nunn, 2009). Thus, not accounting for this fact, one might attribute the persisting effect of colonial policies on Sub-Saharan Africa's development to its populations' high genetic distance and diversity.

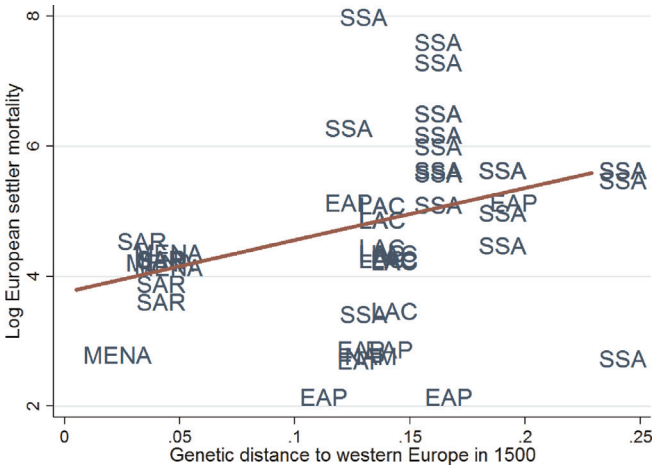

Fig. 1. Settler mortality and genetic distance to Western Europe in 1500.

In their seminal study, Acemoglu et al. use (log) settler mortality as an instrument for contemporary institutions, and argue for a first-order effect of institutions on economic performance. As noted by the authors, the validity of this empirical strategy "is threatened if other factors correlated with the estimates of settler mortality affect income per capita" (p.1372). Therefore, the correlations depicted in Figs. 1 and 2 also raise concerns regarding the validity of Acemoglu et al.'s (2001) exclusion restriction.

I examine the consequences of the relationships depicted in Figs. 1 and 2 for statistical analyses using genetic distance to the technological frontier, genetic diversity, and settler mortality as determinants of contemporary economic performance in a sample of 68 former European colonies. The results suggest that in OLS regressions of income per capita on historical determinants of development, controlling for settler mortality results in statistical insignificance of genetic distance to the technological frontier and

[3] The latter is due to the fact that the Americas and Australia are further away from Africa (compared to Eurasia) along Homo sapiens' out-of-Africa migratory path and human populations lost their genetic diversity with each step of their migration out of Africa (Henn et al., 2012).

[4] The only domesticable animals in South America was llama and North America was not endowed with any species of domesticable animals. While, Eurasian populations were, on average, endowed with 8 species of domesticable mammals (Diamond, 1997).

[5] Since infectious pathogens can easily spread through homogeneous populations (e.g. Cook, 2015), the high degree of genetic homogeneity of the New World's populations contributed to their inability to establish any effective resistance against European colonists. See footnote 7 and Section 5.2 for a discussion of this issue and its possible consequences of the argument of this paper.

[6] The virulence of infectious pathogens depends (among other things) on the extent of genetic homogeneity of the host population, i.e., infectious pathogens can more easily spread and survive in genetically homogeneous populations. Thus, a concern here is that, the inclusion of (log) settler mortality in, for example, an OLS regression of income per capita on genetic diversity (or genetic distance) is an example of a bad control (Angrist and Pischke, 2008), i.e., if settler mortality is an outcome of aggregate genetic traits, then its inclusion as a control variable in the aforementioned regression is inappropriate. However, this is unlikely to be a valid concern. The main reason for this is that it is not the level of overall diversity within a population that matters for population-level resistance to pathogens, it is the extent of heterozygosity within the Major Histocompatibility Complex (MHC) region (these are genes on chromosome 6 that are tasked with the recognition of self from non-self and play a vital role in the immune response of vertebrates). Indeed, it is shown that pathogen-induced selection affects the level of diversity of the MHC region and, therefore, the disease environment influence the diversity of the MHC region independent of out-of-Africa migratory distance. See Section 4 and 5.2 for a comprehensive discussion of this issue.