The Colonial Origins of Comparative Development: An Empirical Investigation

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We exploit differences in European mortality rates to estimate the effect of institutions on economic performance. Europeans adopted very different colonization policies in different colonies, with different associated institutions. In places where Europeans faced high mortality rates, they could not settle and were more likely to set up extractive institutions. These institutions persisted to the present. Exploiting differences in European mortality rates as an instrument for current institutions, we estimate large effects of institutions on income per capita. Once the effect of institutions is controlled for, countries in Africa or those closer to the equator do not have lower incomes. (*JEL* O11, P16, P51)

What are the fundamental causes of the large differences in income per capita across countries? Although there is still little consensus on the answer to this question, differences in institutions and property rights have received considerable attention in recent years. Countries with better "*institutions*," more secure property rights, and less distortionary policies will invest more in physical and human capital, and will use these factors more efficiently to achieve a greater level of income (e.g., Douglass C. North and Robert P. Thomas, 1973; Eric L. Jones, 1981; North, 1981). This view receives some support from cross-country correlations between measures of property rights and economic development (e.g., Stephen Knack and Philip Keefer, 1995; Paulo Mauro, 1995; Robert E. Hall and Charles I. Jones, 1999; Dani Rodrik, 1999), and from a few micro studies that investigate the relationship between property rights and investment or output (e.g., Timothy Besley, 1995; Christopher Mazingo, 1999; Johnson et al., 1999).

At some level it is obvious that institutions matter. Witness, for example, the divergent paths of North and South Korea, or East and West Germany, where one part of the country stagnated under central planning and collective ownership, while the other prospered with private property and a market economy. Nevertheless, we lack reliable estimates of the effect of institutions on economic performance. It is quite likely that rich economies choose or can afford better institutions. Perhaps more important, economies that are different for a variety of reasons will differ both

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in their institutions and in their income per capita.

To estimate the impact of institutions on economic performance, we need a source of exogenous variation in institutions. In this paper, we propose a theory of institutional differences among countries colonized by Europeans,[1] and exploit this theory to derive a possible source of exogenous variation. Our theory rests on three premises:

1. There were different types of colonization policies which created different sets of institutions. At one extreme, European powers set up "extractive states," exemplified by the Belgian colonization of the Congo. These institutions did not introduce much protection for private property, nor did they provide checks and balances against government expropriation. In fact, the main purpose of the extractive state was to transfer as much of the resources of the colony to the colonizer.
   At the other extreme, many Europeans migrated and settled in a number of colonies, creating what the historian Alfred Crosby (1986) calls "Neo-Europes." The settlers tried to replicate European institutions, with strong emphasis on private property and checks against government power. Primary examples of this include Australia, New Zealand, Canada, and the United States.
2. The colonization strategy was influenced by the feasibility of settlements. In places where the disease environment was not favorable to European settlement, the cards were stacked against the creation of Neo-Europes, and the formation of the extractive state was more likely.
3. The colonial state and institutions persisted even after independence.

Based on these three premises, we use the mortality rates expected by the first European settlers in the colonies as an instrument for current institutions in these countries.[2] More specifically, our theory can be schematically summarized as

$$\text{(potential) settler mortality} \Rightarrow \text{settlements}$$

$$\Rightarrow \text{early institutions} \Rightarrow \text{current institutions}$$

$$\Rightarrow \text{current performance.}$$

We use data on the mortality rates of soldiers, bishops, and sailors stationed in the colonies between the seventeenth and nineteenth centuries, largely based on the work of the historian Philip D. Curtin. These give a good indication of the mortality rates faced by settlers. Europeans were well informed about these mortality rates at the time, even though they did not know how to control the diseases that caused these high mortality rates.

Figure 1 plots the logarithm of GDP per capita today against the logarithm of the settler mortality rates per thousand for a sample of 75 countries (see below for data details). It shows a strong negative relationship. Colonies where Europeans faced higher mortality rates are today substantially poorer than colonies that were healthy for Europeans. Our theory is that this relationship reflects the effect of settler mortality working through the institutions brought by Europeans. To substantiate this, we regress current performance on current institutions, and instrument the latter by settler mortality rates. Since our focus is on property rights and checks against government power, we use the protection against "risk of expropriation" index from Political Risk Services as a proxy for institutions. This variable measures differences in institutions originating from different types of states and state policies.[3] There is a strong

[1] By "colonial experience" we do not only mean the direct control of the colonies by European powers, but more generally, European influence on the rest of the world. So according to this definition, Sub-Saharan Africa was strongly affected by "colonialism" between the sixteenth and nineteenth centuries because of the Atlantic slave trade.

[2] Note that although only some countries were colonized, there is no selection bias here. This is because the question we are interested in is the effect of colonization policy *conditional* on being colonized.

[3] Government expropriation is not the only institutional feature that matters. Our view is that there is a "cluster of

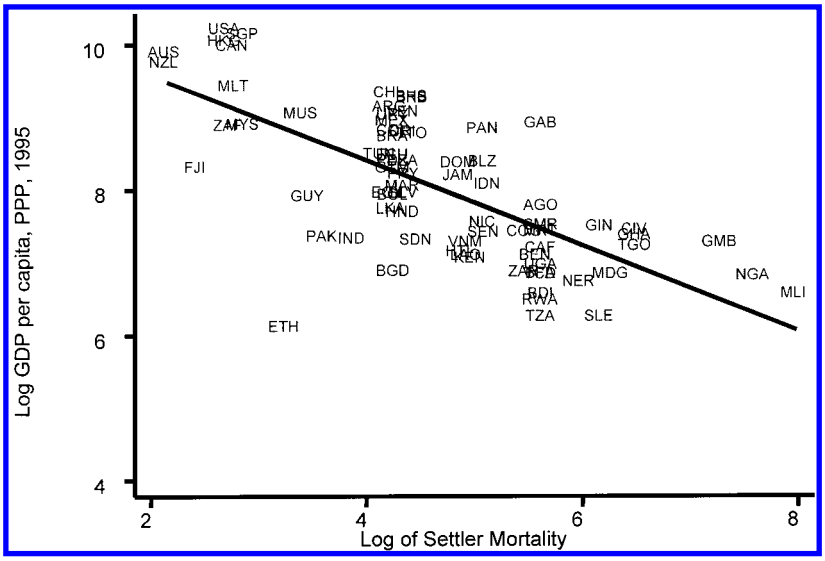

FIGURE 1. REDUCED-FORM RELATIONSHIP BETWEEN INCOME AND SETTLER MORTALITY

(first-stage) relationship between settler mortality rates and current institutions, which is interesting in its own right. The regression shows that mortality rates faced by the settlers more than 100 years ago explains over 25 percent of the variation in current institutions.[4] We also document that this relationship works through the channels we hypothesize: (potential) settler mortality rates were a major determinant of settlements; settlements were a major determinant of early institutions (in practice, institutions in 1900); and there is a strong correlation between early institutions and institutions today. Our two-stage least-squares estimate of the effect of institutions on performance is relatively precisely estimated and large. For example, it implies that improving Nigeria's institutions to the level of Chile could, in the long run, lead to as much as a 7-fold increase in Nigeria's income (in practice Chile is over 11 times as rich as Nigeria).

The exclusion restriction implied by our instrumental variable regression is that, conditional on the controls included in the regression, the mortality rates of European settlers more than 100 years ago have no effect on GDP per capita today, other than their effect through institutional development. The major concern with this exclusion restriction is that the mortality rates of settlers could be correlated with the current disease environment, which may have a direct effect on economic performance. In this case, our instrumental-variables estimates may be assigning the effect of diseases on income to institutions. We believe that this is unlikely to be the case and that our exclusion restriction is plausible. The great majority of European deaths in the colonies were caused by malaria and yellow fever. Although these diseases were fatal to Europeans who had no immunity, they had limited effect on indigenous adults who had developed various types of immunities. These diseases are therefore unlikely to be the reason why many countries in Africa and Asia are very poor today (see the discussion in Section III, subsection A). This notion is

institutions," including constraints on government expropriation, independent judiciary, property rights enforcement, and institutions providing equal access to education and ensuring civil liberties, that are important to encourage investment and growth. Expropriation risk is related to all these institutional features. In Acemoglu et al. (2000), we reported similar results with other institutions variables.

[4] Differences in mortality rates are *not* the only, or even the main, cause of variation in institutions. For our empirical approach to work, all we need is that they are *a source of* exogenous variation.

supported by the mortality rates of local people in these areas. For example, Curtin (1968 Table 2) reports that the annual mortality rates of local troops serving with the British army in Bengal and Madras were respectively 11 and 13 in 1,000. These numbers are quite comparable to, in fact lower than, the annual mortality rates of British troops serving in Britain, which were approximately 15 in 1,000. In contrast, the mortality rates of British troops serving in these colonies were much higher because of their lack of immunity. For example, mortality rates in Bengal and Madras for British troops were between 70 and 170 in 1,000. The view that the disease burden for indigenous adults was not unusual in places like Africa or India is also supported by the relatively high population densities in these places before Europeans arrived (Colin McEvedy and Richard Jones, 1975).

We document that our estimates of the effect of institutions on performance are not driven by outliers. For example, excluding Australia, New Zealand, Canada, and the United States does not change the results, nor does excluding Africa. Interestingly, we show that once the effect of institutions on economic performance is controlled for, neither distance from the equator nor the dummy for Africa is significant. These results suggest that Africa is poorer than the rest of the world not because of pure geographic or cultural factors, but because of worse institutions.

The validity of our approach—i.e., our exclusion restriction—is threatened if other factors correlated with the estimates of settler mortality affect income per capita. We adopt two strategies to substantiate that our results are not driven by omitted factors. First, we investigate whether institutions have a comparable effect on income once we control for a number of variables potentially correlated with settler mortality and economic outcomes. We find that none of these overturn our results; the estimates change remarkably little when we include controls for the identity of the main colonizer, legal origin, climate, religion, geography, natural resources, soil quality, and measures of ethnolinguistic fragmentation. Furthermore, the results are also robust to the inclusion of controls for the current disease environment (e.g., the prevalence of malaria, life expectancy, and infant mortality) and the current fraction of the population of European descent.

Naturally, it is impossible to control for all possible variables that might be correlated with settler mortality and economic outcomes. Furthermore, our empirical approach might capture the effect of settler mortality on economic performance, but working through other channels. We deal with these problems by using a simple overidentification test using measures of European migration to the colonies and early institutions as additional instruments. We then use overidentification tests to detect whether settler mortality has a direct effect on current performance. The results are encouraging for our approach; they generate no evidence for a direct effect of settler mortality on economic outcomes.

We are not aware of others who have pointed out the link between settler mortality and institutions, though scholars such as William H. McNeill (1976), Crosby (1986), and Jared M. Diamond (1997) have discussed the influence of diseases on human history. Diamond (1997), in particular, emphasizes comparative development, but his theory is based on the geographical determinants of the incidence of the neolithic revolution. He ignores both the importance of institutions and the potential causes of divergence in more recent development, which are the main focus of our paper. Work by Ronald E. Robinson and John Gallagher (1961), Lewis H. Gann and Peter Duignan (1962), Donald Denoon (1983), and Philip J. Cain and Anthony G. Hopkins (1993) emphasizes that settler colonies such as the United States and New Zealand are different from other colonies, and point out that these differences were important for their economic success. Nevertheless, this literature does not develop the link between mortality, settlements, and institutions.

Our argument is most closely related to work on the influence of colonial experience on institutions. Frederich A. von Hayek (1960) argued that the British common law tradition was superior to the French civil law, which was developed during the Napoleonic era to restrain judges' interference with state policies (see also Seymour M. Lipset, 1994). More recently, Rafael La Porta et al. (1998, 1999) emphasize the importance of colonial origin (the identity of

the colonizer) and legal origin on current institutions, and show that the common-law countries and former British colonies have better property rights and more developed financial markets. Similarly, David Landes (1998 Chapters 19 and 20) and North et al. (1998) argue that former British colonies prospered relative to former French, Spanish, and Portuguese colonies because of the good economic and political institutions and culture they inherited from Britain. In contrast to this approach which focuses on the identity of the colonizer, we emphasize *the conditions in the colonies*. Specifically, in our theory—and in the data—it is not the identity of the colonizer or legal origin that matters, but whether European colonialists could safely settle in a particular location: where they could not settle, they created worse institutions. In this respect, our argument is closely related to that of Stanley L. Engerman and Kenneth L. Sokoloff (1997) who also emphasize institutions, but link them to factor endowments and inequality.

Empirically, our work is related to a number of other attempts to uncover the link between institutions and development, as well as to Graziella Bertocchi and Fabio Canova (1996) and Robin M. Grier (1999), who investigate the effect of being a colony on postwar growth. Two papers deal with the endogeneity of institutions by using an instrumental variables approach as we do here. Mauro (1995) instruments for corruption using ethnolinguistic fragmentation. Hall and Jones (1999), in turn, use distance from the equator as an instrument for social infrastructure because, they argue, latitude is correlated with "Western influence," which leads to good institutions. The theoretical reasoning for these instruments is not entirely convincing. It is not easy to argue that the Belgian influence in the Congo, or Western influence in the Gold Coast during the era of slavery promoted good institutions. Ethnolinguistic fragmentation, on the other hand, seems endogenous, especially since such fragmentation almost completely disappeared in Europe during the era of growth when a centralized state and market emerged (see, e.g., Eugen J. Weber, 1976; Benedict Anderson, 1983). Econometrically, the problem with both studies is that their instruments can plausibly have a direct effect on performance. For example, William Easterly and Ross Levine (1997) argue that ethnolinguistic fragmentation can affect performance by creating political instability, while Charles de Montesquieu [1748] (1989) and more recently David E. Bloom and Jeffrey D. Sachs (1998) and John Gallup et al. (1998) argue for a direct effect of climate on performance. If, indeed, these variables have a direct effect, they are invalid instruments and do not establish that it is institutions that matter. The advantage of our approach is that conditional on the variables we already control for, settler mortality more than 100 years ago should have no effect on output today, other than through its effect on institutions. Interestingly, our results show that distance from the equator does not have an independent effect on economic performance, validating the use of this variable as an instrument in the work by Hall and Jones (1999).

The next section outlines our hypothesis and provides supporting historical evidence. Section II presents OLS regressions of GDP per capita on our index of institutions. Section III describes our key instrument for institutions, the mortality rates faced by potential settlers at the time of colonization. Section IV presents our main results. Section V investigates the robustness of our results, and Section VI concludes.

I. The Hypothesis and Historical Background

We hypothesize that settler mortality affected settlements; settlements affected early institutions; and early institutions persisted and formed the basis of current institutions. In this section, we discuss and substantiate this hypothesis. The next subsection discusses the link between mortality rates of settlers and settlement decisions, then we discuss differences in colonization policies, and finally, we turn to the causes of institutional persistence.

A. *Mortality and Settlements*

There is little doubt that mortality rates were a key determinant of European settlements. Curtin (1964, 1998) documents how both the British and French press informed the public of mortality rates in the colonies. Curtin (1964)

also documents how early British expectations for settlement in West Africa were dashed by very high mortality among early settlers, about half of whom could be expected to die in the first year. In the "Province of Freedom" (Sierra Leone), European mortality in the first year was 46 percent, in Bulama (April 1792–April 1793) there was 61-percent mortality among Europeans. In the first year of the Sierra Leone Company (1792–1793), 72 percent of the European settlers died. On Mungo Park's Second Expedition (May–November 1805), 87 percent of Europeans died during the overland trip from Gambia to the Niger, and all the Europeans died before completing the expedition.

An interesting example of the awareness of the disease environment comes from the Pilgrim fathers. They decided to migrate to the United States rather than Guyana because of the high mortality rates in Guyana (see Crosby, 1986 pp. 143–44). Another example comes from the Beauchamp Committee in 1795, set up to decide where to send British convicts who had previously been sent to the United States. One of the leading proposals was the island of Lemane, up the Gambia River. The committee rejected this possibility because they decided mortality rates would be too high even for the convicts. Southwest Africa was also rejected for health reasons. The final decision was to send convicts to Australia.

The eventual expansion of many of the colonies was also related to the living conditions there. In places where the early settlers faced high mortality rates, there would be less incentive for new settlers to come.[5]

B. *Types of Colonization and Settlements*

The historical evidence supports both the notion that there was a wide range of different types of colonization and that the presence or absence of European settlers was a key determinant of the form colonialism took. Historians, including Robinson and Gallagher (1961), Gann and Duignan (1962), Denoon (1983), and Cain and Hopkins (1993), have documented the development of "settler colonies," where Europeans settled in large numbers, and life was modeled after the home country. Denoon (1983) emphasizes that settler colonies had representative institutions which promoted what the settlers wanted and that what they wanted was freedom and the ability to get rich by engaging in trade. He argues that "there was undeniably something capitalist in the structure of these colonies. Private ownership of land and livestock was well established very early ..." (p. 35).

When the establishment of European-like institutions did not arise naturally, the settlers were ready to fight for them against the wishes of the home country. Australia is an interesting example here. Most of the early settlers in Australia were ex-convicts, but the land was owned largely by ex-jailors, and there was no legal protection against the arbitrary power of landowners. The settlers wanted institutions and political rights like those prevailing in England at the time. They demanded jury trials, freedom from arbitrary arrest, and electoral representation. Although the British government resisted at first, the settlers argued that they were British and deserved the same rights as in the home country (see Robert Hughes, 1987). Cain and Hopkins write (1993 p. 237) "from the late 1840s the British bowed to local pressures and, in line with observed constitutional changes taking place in Britain herself, accepted the idea that, in mature colonies, governors should in future form ministries from the majority elements in elected legislatures." They also suggest that "the enormous boom in public investment after 1870 [in New Zealand] ... was an attempt to build up an infrastructure ... to maintain high living standards in a country where voters expected politicians actively to promote their economic welfare." (p. 225).[6]

[5] Naturally, other factors also influenced settlements. For example, despite the relatively high mortality rates, many Europeans migrated to the Caribbean because of the very high incomes there at the time (see, e.g., Richard S. Dunn, 1972; David W. Galenson, 1996; Engerman and Sokoloff, 1997; David Eltis, 2000).

[6] Robert H. Bates (1983 Chapter 3) gives a nice example of the influence of settlers on policy in Africa. The British colonial government pursued many policies that depressed the price of cocoa, the main produce of the farmers in Ghana. In contrast, the British government supported the prices faced by the commercial cereal farmers in Kenya.

This is in sharp contrast to the colonial experience in Latin America during the seventeenth and eighteenth centuries, and in Asia and Africa during the nineteenth and early twentieth centuries. The main objective of the Spanish and the Portuguese colonization was to obtain gold and other valuables from America. Soon after the conquest, the Spanish crown granted rights to land and labor (the *encomienda*) and set up a complex mercantilist system of monopolies and trade regulations to extract resources from the colonies.[7]

Europeans developed the slave trade in Africa for similar reasons. Before the mid-nineteenth century, colonial powers were mostly restricted to the African coast and concentrated on monopolizing trade in slaves, gold, and other valuable commodities—witness the names used to describe West African countries: the Gold Coast, the Ivory Coast. Thereafter, colonial policy was driven in part by an element of superpower rivalry, but mostly by economic motives. Michael Crowder (1968 p. 50), for example, notes "it is significant that Britain's largest colony on the West Coast [Nigeria] should have been the one where her traders were most active and bears out the contention that, for Britain ... flag followed trade."[8] Lance E. Davis and Robert A. Huttenback (1987 p. 307) conclude that "the colonial Empire provides strong evidence for the belief that government was attuned to the interests of business and willing to divert resources to ends that the business community would have found profitable." They find that before 1885 investment in the British empire had a return 25 percent higher than that on domestic investment, though afterwards the two converged. Andrew Roberts (1976 p. 193) writes: "[from] ... 1930 to 1940 Britain had kept for itself 2,400,000 pounds in taxes from the Copperbelt, while Northern Rhodesia received from Britain only 136,000 pounds in grants for development." Similarly, Patrick Manning (1982) estimates that between 1905 and 1914, 50 percent of GDP in Dahomey was extracted by the French, and Crawford Young (1994 p. 125) notes that tax rates in Tunisia were four times as high as in France.

Probably the most extreme case of extraction was that of King Leopold of Belgium in the Congo. Gann and Duignan (1979 p. 30) argue that following the example of the Dutch in Indonesia, Leopold's philosophy was that "the colonies should be exploited, not by the operation of a market economy, but by state intervention and compulsory cultivation of cash crops to be sold to and distributed by the state at controlled prices." Jean-Philippe Peemans (1975) calculates that tax rates on Africans in the Congo approached 60 percent of their income during the 1920's and 1930's. Bogumil Jewsiewicki (1983) writes that during the period when Leopold was directly in charge, policy was "based on the violent exploitation of natural and human resources," with a consequent "destruction of economic and social life ... [and] ... dismemberment of political structures."

Overall, there were few constraints on state power in the nonsettler colonies. The colonial powers set up authoritarian and absolutist states with the purpose of solidifying their control and facilitating the extraction of resources. Young (1994 p. 101) quotes a French official in Africa: "the European commandant is not posted to observe nature, ... He has a mission ... to impose regulations, to limit individual liberties ... , to collect taxes." Manning (1988 p. 84) summarizes this as: "In Europe the theories of representative democracy won out over the theorists of absolutism ... . But in Africa, the European conquerors set up absolutist governments, based on reasoning similar to that of Louis XIV."

Bates shows that this was mainly because in Kenya, but not in Ghana, there were a significant number of European settler farmers, who exerted considerable pressure on policy.

[7] See James Lang (1975) and James Lockhart and Stuart B. Schwartz (1983). Migration to Spanish America was limited by the Spanish Crown, in part because of a desire to keep control of the colonists and limit their independence (see, for example, John H. Coatsworth, 1982). This also gives further support to our notion that settlers were able to influence the type of institutions set up in the colonies, even against the wishes of the home country government.

[8] Although in almost all cases the main objective of colonial policies was to protect economic interests and obtain profits, the recipients of these profits varied. In the Portuguese case, it was the state; in the Belgian case, it was King Leopold; and in the British case, it was often private enterprises who obtained concessions or monopoly trading rights in Africa (Crowder, 1968 Part III).

C. *Institutional Persistence*

There is a variety of historical evidence, as well as our regressions in Table 3 below, suggesting that the control structures set up in the nonsettler colonies during the colonial era persisted, while there is little doubt that the institutions of law and order and private property established during the early phases of colonialism in Australia, Canada, New Zealand, the United States, Hong Kong, and Singapore have formed the basis of the current-day institutions of these countries.[9]

Young emphasizes that the extractive institutions set up by the colonialists persisted long after the colonial regime ended. He writes "although we commonly described the independent polities as 'new states,' in reality they were successors to the colonial regime, inheriting its structures, its quotidian routines and practices, and its more hidden normative theories of governance" (1994 p. 283). An example of the persistence of extractive state institutions into the independence era is provided by the persistence of the most prominent extractive policies. In Latin America, the full panoply of monopolies and regulations, which had been created by Spain, remained intact after independence, for most of the nineteenth century. Forced labor policies persisted and were even intensified or reintroduced with the expansion of export agriculture in the latter part of the nineteenth century. Slavery persisted in Brazil until 1886, and during the sisal boom in Mexico, forced labor was reintroduced and persisted up to the start of the revolution in 1910. Forced labor was also reintroduced in Guatemala and El Salvador to provide labor for coffee growing. In the Guatemalan case, forced labor lasted until the creation of democracy in 1945. Similarly, forced labor was reinstated in many independent African countries, for example, by Mobutu in Zaire.

There are a number of economic mechanisms that will lead to institutional persistence of this type. Here, we discuss three possibilities.

(1) Setting up institutions that place restrictions on government power and enforce property rights is costly (see, e.g., Acemoglu and Thierry Verdier, 1998). If the costs of creating these institutions have been sunk by the colonial powers, then it may not pay the elites at independence to switch to extractive institutions. In contrast, when the new elites inherit extractive institutions, they may not want to incur the costs of introducing better institutions, and may instead prefer to exploit the existing extractive institutions for their own benefits.

(2) The gains to an extractive strategy may depend on the size of the ruling elite. When this elite is small, each member would have a larger share of the revenues, so the elite may have a greater incentive to be extractive. In many cases where European powers set up authoritarian institutions, they delegated the day-to-day running of the state to a small domestic elite. This narrow group often was the one to control the state after independence and favored extractive institutions.[10]

(3) If agents make irreversible investments that are complementary to a particular set of institutions, they will be more willing to support them, making these institutions persist (see, e.g., Acemoglu, 1995). For example, agents who have invested in human and physical capital will be in favor of spending

[9] The thesis that institutions persist for a long time goes back at least to Karl A. Wittfogel (1957), who argued that the control structures set up by the large "hydraulic" empires such as China, Russia, and the Ottoman Empire persisted for more than 500 years to the twentieth century. Engerman and Sokoloff (1997), La Porta et al. (1998, 1999), North et al. (1998), and Coatsworth (1999) also argue that colonial institutions persisted. Engerman et al. (1998) provide further evidence supporting this view.

[10] William Reno (1995), for example, argues that the governments of postindependence Sierra Leone adopted the tactics and institutions of the British colonizers to cement their political power and extract resources from the rest of society. Catherine Boone (1992) provides a similar analysis of the evolution of the modern state in Senegal. Most scholars also view the roots of authoritarianism under Mobutu in the colonial state practices in the Belgian Congo (e.g., Thomas M. Callaghy, 1984, or Thomas Turner and Young, 1985, especially p. 43). The situation in Latin America is similar. Independence of most Latin American countries came in the early nineteenth century as domestic elites took advantage of the invasion of Spain by Napoleon to capture the control of the state. But, the only thing that changed was the identity of the recipients of the rents (see, for example, Coatsworth, 1978, or John Lynch, 1986).

TABLE 1—DESCRIPTIVE STATISTICS

			By quartiles of mortality			
	Whole world	Base sample	(1)	(2)	(3)	(4)
Log GDP per capita (PPP) in 1995	8.3	8.05	8.9	8.4	7.73	7.2
	(1.1)	(1.1)				
Log output per worker in 1988 (with level of United States normalized to 1)	−1.70	−1.93	−1.03	−1.46	−2.20	−3.03
	(1.1)	(1.0)				
Average protection against expropriation risk, 1985–1995	7	6.5	7.9	6.5	6	5.9
	(1.8)	(1.5)				
Constraint on executive in 1990	3.6	4	5.3	5.1	3.3	2.3
	(2.3)	(2.3)				
Constraint on executive in 1900	1.9	2.3	3.7	3.4	1.1	1
	(1.8)	(2.1)				
Constraint on executive in first year of independence	3.6	3.3	4.8	2.4	3.1	3.4
	(2.4)	(2.4)				
Democracy in 1900	1.1	1.6	3.9	2.8	0.19	0
	(2.6)	(3.0)				
European settlements in 1900	0.31	0.16	0.32	0.26	0.08	0.005
	(0.4)	(0.3)				
Log European settler mortality	n.a.	4.7	3.0	4.3	4.9	6.3
		(1.1)				
Number of observations	163	64	14	18	17	15

Notes: Standard deviations are in parentheses. Mortality is potential settler mortality, measured in terms of deaths per annum per 1,000 "mean strength" (raw mortality numbers are adjusted to what they would be if a force of 1,000 living people were kept in place for a whole year, e.g., it is possible for this number to exceed 1,000 in episodes of extreme mortality as those who die are replaced with new arrivals). Sources and methods for mortality are described in Section III, subsection B, and in the unpublished Appendix (available from the authors; or see Acemoglu et al., 2000). Quartiles of mortality are for our base sample of 64 observations. These are: (1) less than 65.4; (2) greater than or equal to 65.4 and less than 78.1; (3) greater than or equal to 78.1 and less than 280; (4) greater than or equal to 280. The number of observations differs by variable; see Appendix Table A1 for details.

money to enforce property rights, while those who have less to lose may not be.

II. Institutions and Performance: OLS Estimates

A. *Data and Descriptive Statistics*

Table 1 provides descriptive statistics for the key variables of interest. The first column is for the whole world, and column (2) is for our base sample, limited to the 64 countries that were ex-colonies and for which we have settler mortality, protection against expropriation risk, and GDP data (this is smaller than the sample in Figure 1). The GDP per capita in 1995 is PPP adjusted (a more detailed discussion of all data sources is provided in Appendix Table A1). Income (GDP) per capita will be our measure of economic outcome. There are large differences in income per capita in both the world sample and our basic sample, and the standard deviation of log income per capita in both cases is 1.1. In row 3, we also give output per worker in 1988 from Hall and Jones (1999) as an alternative measure of income today. Hall and Jones (1999) prefer this measure since it explicitly refers to worker productivity. On the other hand, given the difficulty of measuring the formal labor force, it may be a more noisy measure of economic performance than income per capita.

We use a variety of variables to capture institutional differences. Our main variable, reported in the second row, is an index of protection against expropriation. These data are from Political Risk Services (see, e.g., William D. Coplin et al., 1991), and were first used in the economics and political science literatures by Knack and Keefer (1995). Political Risk Services reports a value between 0 and 10 for each country and year, with 0 corresponding to the

lowest protection against expropriation. We use the average value for each country between 1985 and 1995 (values are missing for many countries before 1985). This measure is appropriate for our purposes since the focus here is on differences in institutions originating from different types of states and state policies. We expect our notion of extractive state to correspond to a low value of this index, while the tradition of rule of law and well-enforced property rights should correspond to high values.[11] The next row gives an alternative measure, constraints on the executive in 1990, coded from the Polity III data set of Ted Robert Gurr and associates (an update of Gurr, 1997). Results using the constraints on the executive and other measures are reported in Acemoglu et al. (2000) and are not repeated here.

The next three rows give measures of early institutions from the same Gurr data set. The first is a measure of constraints on the executive in 1900 and the second is an index of democracy in 1900. This information is not available for countries that were still colonies in 1900, so we assign these countries the lowest possible score. In the following row, we report the mean and standard deviation of constraints on the executive in the first year of independence (i.e., the first year a country enters the Gurr data set) as an alternative measure of institutions. The second-to-last row gives the fraction of the population of European descent in 1900, which is our measure of European settlement in the colonies, constructed from McEvedy and Jones (1975) and Curtin et al. (1995). The final row gives the logarithm of the baseline settler mortality estimates; the raw data are in Appendix Table A2.

The remaining columns give descriptive statistics for groups of countries at different quartiles of the settler mortality distribution. This is useful since settler mortality is our instrument for institutions (this variable is described in more detail in the next section).

[11] The protection against expropriation variable is specifically for foreign investment, since Political and Risk Services construct these data for foreign investors. However, as noted by Knack and Keefer (1995), risk of expropriation of foreign and domestic investments are very highly correlated, and risk of expropriation of foreign investment may be more comparable across countries. In any case, all our results hold also with a variety of other measures of institutions (see Tables 4a, b, c, d, and e in Acemoglu et al., 2000, available from the authors).

B. *Ordinary Least-Squares Regressions*

Table 2 reports ordinary least-squares (OLS) regressions of log per capita income on the protection against expropriation variable in a variety of samples. The linear regressions are for the equation

$$\log y_i = \mu + \alpha R_i + \mathbf{X}_i'\gamma + \varepsilon_i, \tag{1}$$

where y_i is income per capita in country i, R_i is the protection against expropriation measure, $\mathbf{X}_i$ is a vector of other covariates, and ε_i is a random error term. The coefficient of interest throughout the paper is α, the effect of institutions on income per capita.

Column (1) shows that in the whole world sample there is a strong correlation between our measure of institutions and income per capita. Column (2) shows that the impact of the institutions variable on income per capita in our base sample is quite similar to that in the whole world, and Figure 2 shows this relationship diagrammatically for our base sample consisting of 64 countries. The R^2 of the regression in column (1) indicates that over 50 percent of the variation in income per capita is associated with variation in this index of institutions. To get a sense of the magnitude of the effect of institutions on performance, let us compare two countries, Nigeria, which has approximately the 25th percentile of the institutional measure in this sample, 5.6, and Chile, which has approximately the 75th percentile of the institutions index, 7.8. The estimate in column (1), 0.52, indicates that there should be on average a 1.14-log-point difference between the log GDPs of the corresponding countries (or approximately a 2-fold difference—$e^{1.14} - 1 \approx 2.1$). In practice, this GDP gap is 253 log points (approximately 11-fold). Therefore, if the effect estimated in Table 2 were causal, it would imply a fairly large effect of institutions on performance, but still much less than the actual income gap between Nigeria and Chile.

Many social scientists, including Montesquieu [1784] (1989), Diamond (1997), and

TABLE 2—OLS REGRESSIONS

	Whole world (1)	Base sample (2)	Whole world (3)	Whole world (4)	Base sample (5)	Base sample (6)	Whole world (7)	Base sample (8)
	Dependent variable is log GDP per capita in 1995						Dependent variable is log output per worker in 1988	
Average protection against expropriation risk, 1985–1995	0.54 (0.04)	0.52 (0.06)	0.47 (0.06)	0.43 (0.05)	0.47 (0.06)	0.41 (0.06)	0.45 (0.04)	0.46 (0.06)
Latitude			0.89 (0.49)	0.37 (0.51)	1.60 (0.70)	0.92 (0.63)		
Asia dummy				−0.62 (0.19)		−0.60 (0.23)		
Africa dummy				−1.00 (0.15)		−0.90 (0.17)		
"Other" continent dummy				−0.25 (0.20)		−0.04 (0.32)		
R^2	0.62	0.54	0.63	0.73	0.56	0.69	0.55	0.49
Number of observations	110	64	110	110	64	64	108	61

Notes: Dependent variable: columns (1)–(6), log GDP per capita (PPP basis) in 1995, current prices (from the World Bank's World Development Indicators 1999); columns (7)–(8), log output per worker in 1988 from Hall and Jones (1999). Average protection against expropriation risk is measured on a scale from 0 to 10, where a higher score means more protection against expropriation, averaged over 1985 to 1995, from Political Risk Services. Standard errors are in parentheses. In regressions with continent dummies, the dummy for America is omitted. See Appendix Table A1 for more detailed variable definitions and sources. Of the countries in our base sample, Hall and Jones do not report output per worker in the Bahamas, Ethiopia, and Vietnam.

Sachs and coauthors, have argued for a direct effect of climate on performance, and Gallup et al. (1998) and Hall and Jones (1999) document the correlation between distance from the equator and economic performance. To control for this, in columns (3)–(6), we add latitude as a regressor (we follow the literature in using the absolute value measure of latitude, i.e., distance from the equator, scaled between 0 and 1). This changes the coefficient of the index of institutions little. Latitude itself is also significant and has the sign found by the previous studies. In columns (4) and (6), we also add dummies for Africa, Asia, and other continents, with America as the omitted group. Although protection against expropriation risk remains significant, the continent dummies are also statistically and quantitatively significant. The Africa dummy in column (6) indicates that in our sample African countries are 90 log points (approximately 145 percent) poorer even after taking the effect of institutions into account. Finally, in columns (7) and (8), we repeat our basic regressions using the log of output per worker from Hall and Jones (1999), with very similar results.

Overall, the results in Table 2 show a strong correlation between institutions and economic performance. Nevertheless, there are a number of important reasons for not interpreting this relationship as causal. First, rich economies may be able to afford, or perhaps prefer, better institutions. Arguably more important than this reverse causality problem, there are many omitted determinants of income differences that will naturally be correlated with institutions. Finally, the measures of institutions are constructed *ex post,* and the analysts may have had a natural bias in seeing better institutions in richer places. As well as these problems introducing positive bias in the OLS estimates, the fact that the institutions variable is measured with considerable error and corresponds poorly to the "cluster of institutions" that matter in practice creates attenuation and may bias the OLS estimates

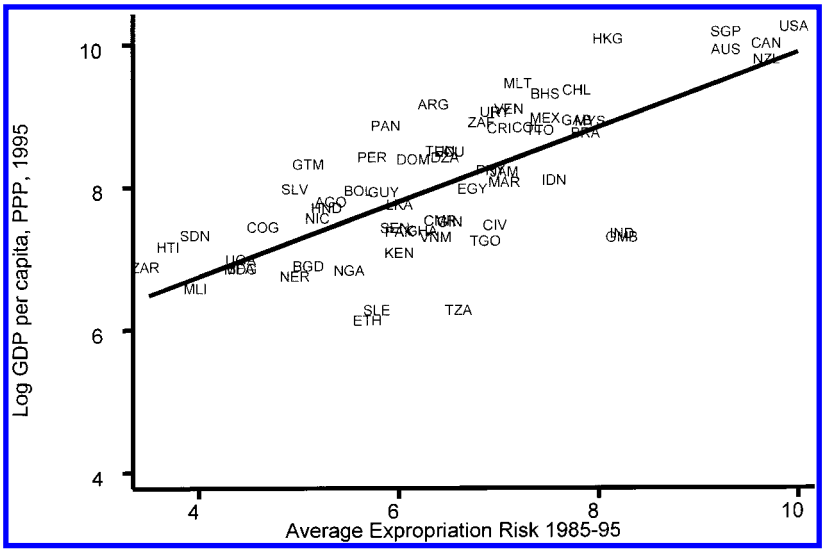

FIGURE 2. OLS RELATIONSHIP BETWEEN EXPROPRIATION RISK AND INCOME

downwards. All of these problems could be solved if we had an instrument for institutions. Such an instrument must be an important factor in accounting for the institutional variation that we observe, but have no direct effect on performance. Our discussion in Section I suggests that settler mortality during the time of colonization is a plausible instrument.

III. Mortality of Early Settlers

A. *Sources of European Mortality in the Colonies*

In this subsection, we give a brief overview of the sources of mortality facing potential settlers. Malaria (particularly Plasmodium falciporum) and yellow fever were the major sources of European mortality in the colonies. In the tropics, these two diseases accounted for 80 percent of European deaths, while gastrointestinal diseases accounted for another 15 percent (Curtin, 1989 p. 30). Throughout the nineteenth century, areas without malaria and yellow fever, such as New Zealand, were more healthy than Europe because the major causes of death in Europe—tuberculosis, pneumonia, and smallpox—were rare in these places (Curtin, 1989 p. 13).

Both malaria and yellow fever are transmitted by mosquito vectors. In the case of malaria, the main transmitter is the *Anopheles gambiae* complex and the mosquito *Anopheles funestus*, while the main carrier of yellow fever is *Aedes aegypti*. Both malaria and yellow fever vectors tend to live close to human habitation.

In places where the malaria vector is present, such as the West African savanna or forest, an individual can get as many as several hundred infectious mosquito bites a year. For a person without immunity, malaria (particularly Plasmodium falciporum) is often fatal, so Europeans in Africa, India, or the Caribbean faced very high death rates. In contrast, death rates for the adult local population were much lower (see Curtin [1964] and the discussion in our introduction above). Curtin (1998 pp. 7–8) describes this as follows:

> Children in West Africa ... would be infected with malaria parasites shortly after birth and were frequently reinfected afterwards; if they lived beyond the age of about five, they acquired an apparent immunity. The parasite remained with them, normally in the liver, but clinical symptoms were rare so long as they continued to be infected with the same species of P. falciporum.

The more recent books on malariology confirm this conclusion. For example, "In stable endemic areas a heavy toll of morbidity and mortality falls on young children but malaria is a relatively mild condition in adults" (Herbert M. Gilles and David A. Warrell, 1993 p. 64; see also the classic reference on this topic, Leonard J. Bruce-Chwatt, 1980 Chapter 4; Roy Porter, 1996).[12] Similarly, the World Health Organization (WHO) points out that in endemic malaria areas of Africa and the Western Pacific today "... the risk of malaria severity and death is almost exclusively limited to non-immunes, being most serious for young children over six months of age ... surviving children develop their own immunity between the age of 3–5 years" (Jose A. Najera and Joahim Hempel, 1996).

People in areas where malaria is endemic are also more likely to have genetic immunity against malaria. For example, they tend to have the sickle-cell trait, which discourages the multiplication of parasites in the blood, or deficiencies in *glucose-6-phosphate dehydrogenase* and *thalassaemia* traits, which also protect against malaria. Porter (1996 p. 34) writes: "In such a process, ... , close to 100 percent of Africans acquired a genetic trait that protects them against vivax malaria and probably against falciporum malaria as well." Overall, the WHO estimates that malaria kills about 1 million people per year, most of them children. It does not, however, generally kill adults who grew up in malaria-endemic areas (see Najera and Hempel, 1996).

Although yellow fever's epidemiology is quite different from malaria, it was also much more fatal to Europeans than to non-Europeans who grew up in areas where yellow fever commonly occurred.[13] Yellow fever leaves its surviving victims with a lifelong immunity, which also explains its epidemic pattern, relying on a concentrated nonimmune population. Curtin (1998 p. 10) writes: "Because most Africans had passed through a light case early in life, yellow fever in West Africa was a strangers' disease, attacking those who grew up elsewhere." Similarly, Michael B. A. Oldstone (1998 p. 49) writes:

> Most Black Africans and their descendants respond to yellow fever infection with mild to moderate symptoms such as headache, fever, nausea, and vomiting, and then recover in a few days. This outcome reflects the long relationship between the virus and its indigenous hosts, who through generations of exposure to the virus have evolved resistance.

In contrast, fatality rates among nonimmune adults, such as Europeans, could be as high as 90 percent.

Advances in medical science have reduced the danger posed by malaria and yellow fever. Yellow fever is mostly eradicated (Oldstone, 1998 Chapter 5), and malaria has been eradicated in many areas. Europeans developed methods of dealing with these diseases that gradually became more effective in the second half of the nineteenth century. For example, they came to understand that high doses of quinine, derived from the cinchona bark, acted as a prophylactic and prevented infection or reduced the severity of malaria. They also started to undertake serious mosquito eradication efforts and protect themselves against mosquito bites. Further, Europeans also learned that an often effective method of reducing mortality from yellow fever is flight from the area, since the transmitter mosquito, *Aedes aegypti,* has only a short range. Nevertheless, during much of the nineteenth century, there was almost a complete misunderstanding of the nature of both diseases. For example, the leading theory for malaria was that it was caused by "miasma" from swamps, and quinine was not used widely. The role of small collections of water to breed mosquitoes and transmit these diseases was not understood. It was only in the late nineteenth century that Europeans started to control these diseases.[14]

[12] Because malaria species are quite local, a person may have immunity to the local version of malaria, but be highly vulnerable to malaria a short distance away. This is probably the explanation for why Africans had such high mortality when they were forced to move by colonial powers. (Curtin et al., 1995 p. 463).

[13] Because yellow fever struck Europeans as an epidemic, many of the very high death rates we report below for European troops are from yellow fever.

[14] Even during the early twentieth century, there was much confusion about the causes of malaria and yellow

These considerations, together with the data we have on the mortality of local people and population densities before the arrival of Europeans, make us believe that settler mortality is a plausible instrument for institutional development: these diseases affected European settlement patterns and the type of institutions they set up, but had little effect on the health and economy of indigenous people.[15]

A final noteworthy feature, helpful in interpreting our results below, is that malaria prevalence depends as much on the microclimate of an area as on its temperature and humidity, or on whether it is in the tropics; high altitudes reduce the risk of infection, so in areas of high altitude, where "hill stations" could be set up, such as Bogota in Colombia, mortality rates were typically lower than in wet coastal areas. However, malaria could sometimes be more serious in high-altitude areas. For example, Curtin (1989 p. 47) points out that in Ceylon mortality was lower in the coast than the highlands because rains in the coast washed away the larvae of the transmitter mosquitoes. Similarly, in Madras many coastal regions were free of malaria, while northern India had high rates of infection. Curtin (1998 Chapter 7) also illustrates how there were marked differences in the prevalence of malaria within small regions of Madagascar. This suggests that mortality rates faced by Europeans are unlikely to be a proxy for some simple geographic or climactic feature of the country.

B. *Data on Potential Settler Mortality*

Our data on the mortality of European settlers come largely from the work of Philip Curtin. Systematic military medical record keeping began only after 1815, as an attempt to understand why so many soldiers were dying in some places. The first detailed studies were retrospective and dealt with British forces between 1817 and 1836. The United States and French governments quickly adopted similar methods (Curtin, 1989 pp. 3, 5). Some early data are also available for the Dutch East Indies. By the 1870's, most European countries published regular reports on the health of their soldiers.

The standard measure is annualized deaths per thousand mean strength. This measure reports the death rate among 1,000 soldiers where each death is replaced with a new soldier. Curtin (1989, 1998) reviews in detail the construction of these estimates for particular places and campaigns, and assesses which data should be considered reliable.

Curtin (1989), *Death by Migration,* deals primarily with the mortality of European troops from 1817 to 1848. At this time modern medicine was still in its infancy, and the European militaries did not yet understand how to control malaria and yellow fever. These mortality rates can therefore be interpreted as reasonable estimates of settler mortality. They are consistent with substantial evidence from other sources (see, for example, Curtin [1964, 1968]). Curtin (1998), *Disease and Empire,* adds similar data on the mortality of soldiers in the second half of the nineteenth century.[16] In all cases, we use the

fever. *The Washington Post* on Nov. 2, 1900 wrote: "Of all the silly and nonsensical rigmarole of yellow fever that has yet found its way into print ... the silliest beyond compare is to be found in the arguments and theories generated by a mosquito hypothesis" (quoted in Oldstone, 1998 pp. 64–65).

Many campaigns during the nineteenth century had very high mortality rates. For example, the French campaign in Madagascar during the 1890's and French attempts to build the Panama Canal during the 1880's were mortality disasters, the first due to malaria, the second due to yellow fever (see Curtin, 1998, and David McCullogh, 1977). In Panama, to stop ants the French used water pots under the legs of beds in barracks and hospitals. These pots provided an ideal milieu for the breeding of *Aedes aegypti,* causing very high rates of mortality (Oldstone, 1998 p. 66).

[15] In Acemoglu et al. (2001), we document that many of these areas in the tropical zone were richer and more densely settled in 1500 than the temperate areas later settled by the Europeans. This also supports the notion that the disease environment did not create an absolute disadvantage for these countries.

[16] These numbers have to be used with more care because there was a growing awareness of how to avoid epidemics of the worst tropical diseases, at least during short military campaigns. For example, the campaign in Ethiopia at the end of the nineteenth century had very low mortality rates because it was short and well managed (see Figure 1). Although the mortality rates from this successful campaign certainly underestimate the mortality rates faced

earliest available number for each country, reasoning that this is the best estimate of the mortality rates that settlers would have faced, at least until the twentieth century.

The main gap in the Curtin data is for South America since the Spanish and Portuguese militaries did not keep good records of mortality. Hector Gutierrez (1986) used Vatican records to construct estimates for the mortality rates of bishops in Latin America from 1604 to 1876. Because these data overlap with the Curtin estimates for several countries, we are able to construct a data series for South America.[17] Curtin (1964) also provides estimates of mortality in naval squadrons for different regions which we can use to generate alternative estimates of mortality in South America. Appendix B in Acemoglu et al. (2000), which is available from the authors, gives a detailed discussion of how these data are constructed, and Appendix Table A5 (available from the authors), shows that these alternative methods produce remarkably similar results. Appendix Table A2 lists our main estimates, and Table A1 gives information about sources.

IV. Institutions and Performance: IV Results

A. *Determinants of Current Institutions*

Equation (1) describes the relationship between current institutions and log GDP. In addition we have

$$R_i = \lambda_R + \beta_R C_i + \mathbf{X}_i'\gamma_R + \nu_{Ri}, \tag{2}$$

$$C_i = \lambda_C + \beta_C S_i + \mathbf{X}_i'\gamma_C + \nu_{Ci}, \tag{3}$$

$$S_i = \lambda_S + \beta_S \log M_i + \mathbf{X}_i'\gamma_S + \nu_{Si}, \tag{4}$$

where R is the measure of current institutions (protection against expropriation between 1985 and 1995), C is our measure of early (circa 1900) institutions, S is the measure of European settlements in the colony (fraction of the population with European descent in 1900), and M is mortality rates faced by settlers. $\mathbf{X}$ is a vector of covariates that affect all variables.

The simplest identification strategy might be to use S_i (or C_i) as an instrument for R_i in equation (1), and we report some of these regressions in Table 8. However, to the extent that settlers are more likely to migrate to richer areas and early institutions reflect other characteristics that are important for income today, this identification strategy would be invalid (i.e., C_i and S_i could be correlated with ε_i). Instead, we use the mortality rates faced by the settlers, log M_i, as an instrument for R_i. This identification strategy will be valid as long as log M_i is uncorrelated with ε_i—that is, if mortality rates of settlers between the seventeenth and nineteenth centuries have no effect on income today other than through their influence on institutional development. We argued above that this exclusion restriction is plausible.

Figure 3 illustrates the relationship between the (potential) settler mortality rates and the index of institutions. We use the logarithm of the settler mortality rates, since there are no theoretical reasons to prefer the level as a determinant of institutions rather than the log, and using the log ensures that the extreme African mortality rates do not play a disproportionate role. As it happens, there is an almost linear relationship between the log settler mortality and our measure of institutions. This relationship shows that ex-colonies where Europeans faced higher mortality rates have substantially worse institutions today.

In Table 3, we document that this relationship works through the channels hypothesized in Section I. In particular, we present OLS regressions of equations (2), (3), and (4). In the top panel, we regress the protection against expropriation variable on the other variables. Column (1) uses constraints faced by the executive in 1900 as the regressor, and shows a close association between early institutions and institutions today. For example, past institutions alone explain 20 percent of the variation in the index of current institutions. The second column adds the latitude variable,

by potential settlers in Ethiopia, we did not exclude this country because excluding it would have helped our hypothesis.

[17] Combining data from a variety of sources will introduce measurement error in our estimates of settler mortality. Nevertheless, since we are using settler mortality as an instrument, this measurement error does not lead to inconsistent estimates of the effect of institutions on performance.

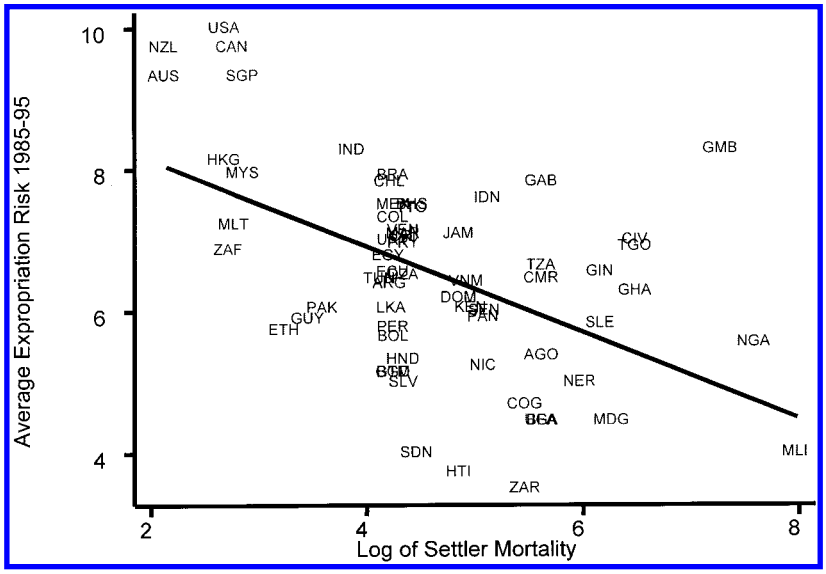

FIGURE 3. FIRST-STAGE RELATIONSHIP BETWEEN SETTLER MORTALITY AND EXPROPRIATION RISK

with little effect on the estimate. Columns (3) and (4) use the democracy index, and confirm the results in columns (1) and (2).

Both constraints on the executive and democracy indices assign low scores to countries that were colonies in 1900, and do not use the earliest postindependence information for Latin American countries and the Neo-Europes. In columns (5) and (6), we adopt an alternative approach and use the constraints on the executive in the first year of independence and also control separately for time since independence. The results are similar, and indicate that early institutions tend to persist.

Columns (7) and (8) show the association between protection against expropriation and European settlements. The fraction of Europeans in 1900 alone explains approximately 30 percent of the variation in our institutions variable today. Columns (9) and (10) show the relationship between the protection against expropriation variable and the mortality rates faced by settlers. This specification will be the first stage for our main two-stage least-squares estimates (2SLS). It shows that settler mortality alone explains 27 percent of the differences in institutions we observe today.

Panel B of Table 3 provides evidence in support of the hypothesis that early institutions were shaped, at least in part, by settlements, and that settlements were affected by mortality. Columns (1)–(2) and (5)–(6) relate our measure of constraint on the executive and democracy in 1900 to the measure of European settlements in 1900 (fraction of the population of European decent). Columns (3)–(4) and (7)–(8) relate the same variables to settler mortality. These regressions show that settlement patterns explain around 50 percent of the variation in early institutions. Finally, columns (9) and (10) show the relationship between settlements and mortality rates.

B. *Institutions and Economic Performance*

Two-stage least-squares estimates of equation (1) are presented in Table 4. Protection against expropriation variable, R_i, is treated as endogenous, and modeled as

$$R_i = \zeta + \beta \log M_i + \mathbf{X}_i'\delta + v_i, \tag{5}$$

where M_i is the settler mortality rate in 1,000 mean strength. The exclusion restriction is that this variable does not appear in (1).

TABLE 3—DETERMINANTS OF INSTITUTIONS

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A	Dependent Variable Is Average Protection Against Expropriation Risk in 1985–1995									
Constraint on executive in 1900	0.32	0.26								
	(0.08)	(0.09)								
Democracy in 1900			0.24	0.21						
			(0.06)	(0.07)						
Constraint on executive in first year of independence					0.25	0.22				
					(0.08)	(0.08)				
European settlements in 1900							3.20	3.00		
							(0.61)	(0.78)		
Log European settler mortality									−0.61	−0.51
									(0.13)	(0.14)
Latitude		2.20		1.60		2.70		0.58		2.00
		(1.40)		(1.50)		(1.40)		(1.51)		(1.34)
R^2	0.2	0.23	0.24	0.25	0.19	0.24	0.3	0.3	0.27	0.3
Number of observations	63	63	62	62	63	63	66	66	64	64

Panel B	Dependent Variable Is Constraint on Executive in 1900				Dependent Variable Is Democracy in 1900				Dependent Variable Is European Settlements in 1900	
European settlements in 1900	5.50	5.40			8.60	8.10				
	(0.73)	(0.93)			(0.90)	(1.20)				
Log European settler mortality			−0.82	−0.65			−1.22	−0.88	−0.11	−0.07
			(0.17)	(0.18)			(0.24)	(0.25)	(0.02)	(0.02)
Latitude		0.33		3.60		1.60		7.60		0.87
		(1.80)		(1.70)		(2.30)		(2.40)		(0.19)
R^2	0.46	0.46	0.25	0.29	0.57	0.57	0.28	0.37	0.31	0.47
Number of observations	70	70	75	75	67	67	68	68	73	73

Notes: All regressions are OLS. Standard errors are in parentheses. Regressions with constraint on executive in first year of independence also include years since independence as a regressor. Average protection against expropriation risk is on a scale from 0 to 10, where a higher score means more protection against expropriation of private investment by government, averaged over 1985 to 1995. Constraint on executive in 1900 is on a scale from 1 to 7, with a higher score indicating more constraints. Democracy in 1900 is on a scale from 0 to 10, with a higher score indicating more democracy. European settlements is percent of population that was European or of European descent in 1900. See Appendix Table A1 for more detailed variable definitions and sources.

Panel A of Table 4 reports 2SLS estimates of the coefficient of interest, α from equation (1) and Panel B gives the corresponding first stages.[18] Column (1) displays the strong first-stage relationship between (log) settler mortality and current institutions in our base sample, also shown in Table 3. The corresponding 2SLS estimate of the impact of institutions on income per capita is 0.94. This estimate is highly significant with a standard error of 0.16, and in fact larger than the OLS estimates reported in Table 2. This suggests that measurement error in the institutions variables that creates attenuation bias is likely to be more important than reverse causality and omitted variables biases. Here we are referring to "measurement error" broadly construed. In reality the set of institutions that matter for economic performance is very complex, and any single measure is bound to capture only part of the "true institutions,"

[18] We have also run these regressions with standard errors corrected for possible clustering of the mortality rates assigned to countries in the same disease environment. This clustering has little effect on the standard errors, and does not change our results.

TABLE 4—IV REGRESSIONS OF LOG GDP PER CAPITA

	Base sample (1)	Base sample (2)	Base sample without Neo-Europes (3)	Base sample without Neo-Europes (4)	Base sample without Africa (5)	Base sample without Africa (6)	Base sample with continent dummies (7)	Base sample with continent dummies (8)	Base sample, dependent variable is log output per worker (9)
Panel A: Two-Stage Least Squares									
Average protection against expropriation risk 1985–1995	0.94	1.00	1.28	1.21	0.58	0.58	0.98	1.10	0.98
	(0.16)	(0.22)	(0.36)	(0.35)	(0.10)	(0.12)	(0.30)	(0.46)	(0.17)
Latitude		−0.65		0.94		0.04		−1.20	
		(1.34)		(1.46)		(0.84)		(1.8)	
Asia dummy							−0.92	−1.10	
							(0.40)	(0.52)	
Africa dummy							−0.46	−0.44	
							(0.36)	(0.42)	
"Other" continent dummy							−0.94	−0.99	
							(0.85)	(1.0)	
Panel B: First Stage for Average Protection Against Expropriation Risk in 1985–1995									
Log European settler mortality	−0.61	−0.51	−0.39	−0.39	−1.20	−1.10	−0.43	−0.34	−0.63
	(0.13)	(0.14)	(0.13)	(0.14)	(0.22)	(0.24)	(0.17)	(0.18)	(0.13)
Latitude		2.00		−0.11		0.99		2.00	
		(1.34)		(1.50)		(1.43)		(1.40)	
Asia dummy							0.33	0.47	
							(0.49)	(0.50)	
Africa dummy							−0.27	−0.26	
							(0.41)	(0.41)	
"Other" continent dummy							1.24	1.1	
							(0.84)	(0.84)	
R^2	0.27	0.30	0.13	0.13	0.47	0.47	0.30	0.33	0.28
Panel C: Ordinary Least Squares									
Average protection against expropriation risk 1985–1995	0.52	0.47	0.49	0.47	0.48	0.47	0.42	0.40	0.46
	(0.06)	(0.06)	(0.08)	(0.07)	(0.07)	(0.07)	(0.06)	(0.06)	(0.06)
Number of observations	64	64	60	60	37	37	64	64	61

Notes: The dependent variable in columns (1)–(8) is log GDP per capita in 1995, PPP basis. The dependent variable in column (9) is log output per worker, from Hall and Jones (1999). "Average protection against expropriation risk 1985–1995" is measured on a scale from 0 to 10, where a higher score means more protection against risk of expropriation of investment by the government, from Political Risk Services. Panel A reports the two-stage least-squares estimates, instrumenting for protection against expropriation risk using log settler mortality; Panel B reports the corresponding first stage. Panel C reports the coefficient from an OLS regression of the dependent variable against average protection against expropriation risk. Standard errors are in parentheses. In regressions with continent dummies, the dummy for America is omitted. See Appendix Table A1 for more detailed variable descriptions and sources.

creating a typical measurement error problem. Moreover, what matters for current income is presumably not only institutions today, but also institutions in the past. Our measure of institutions which refers to 1985–1995 will not be perfectly correlated with these.[19]

Does the 2SLS estimate make quantitative sense? Does it imply that institutional differences can explain a significant fraction of income dif-

[19] We can ascertain, to some degree, whether the difference between OLS and 2SLS estimates could be due to measurement error in the institutions variable by making use of an alternative measure of institutions, for example, the constraints on the executive measure. Using this measure as an instrument for the protection against expropriation index would solve the measurement error, but not the endogeneity problem. This exercise leads to an estimate of the effect of protection against expropriation equal to 0.87 (with standard error 0.16). This suggests that "measurement error" in the institutions variables (or the "signal-to-noise ratio" in the institutions variable) is of the right order of magnitude to explain the difference between the OLS and 2SLS estimates.

ferences across countries? Let us once again compare two "typical" countries with high and low expropriation risk, Nigeria and Chile (these countries are typical for the IV regression in the sense that they are practically on the regression line). Our 2SLS estimate, 0.94, implies that the 2.24 differences in expropriation risk between these two countries should translate into 206 log point (approximately 7-fold) difference. In practice, the presence of measurement error complicates this interpretation, because some of the difference between Nigeria and Chile's expropriation index may reflect measurement error. Therefore, the 7-fold difference is an upper bound. In any case, the estimates in Table 4 imply a substantial, but not implausibly large, effect of institutional differences on income per capita.

Column (2) shows that adding latitude does not change the relationship; the institutions coefficient is now 1.00 with a standard error of 0.22.[20] Remarkably, the latitude variable now has the "wrong" sign and is insignificant. This result suggests that many previous studies may have found latitude to be a significant determinant of economic performance because it is correlated with institutions (or with the exogenous component of institutions caused by early colonial experience).

Columns (3) and (4) document that our results are not driven by the Neo-Europes. When we exclude the United States, Canada, Australia, and New Zealand, the estimates remain highly significant, and in fact increase a little. For example, the coefficient for institutions is now 1.28 (s.e. = 0.36) without the latitude control, and 1.21 (s.e. = 0.35) when we control for latitude. Columns (5) and (6) show that our results are also robust to dropping all the African countries from our sample. The estimates without Africa are somewhat smaller, but also more precise. For example, the coefficient for institutions is 0.58 (s.e. = 0.1) without the latitude control, and still 0.58 (s.e. = 0.12) when we control for latitude.[21]

In columns (7) and (8), we add continent dummies to the regressions (for Africa, Asia, and other, with America as the omitted group). The addition of these dummies does not change the estimated effect of institutions, and the dummies are jointly insignificant at the 5-percent level, though the dummy for Asia is significantly different from that of America. The fact that the African dummy is insignificant suggests that the reason why African countries are poorer is not due to cultural or geographic factors, but mostly accounted for by the existence of worse institutions in Africa. Finally, in column (9) we repeat our basic regression using log of output per worker as calculated by Hall and Jones (1999). The result is very close to our baseline result. The 2SLS coefficient is 0.98 instead of 0.94 as in column (1).[22] This shows that whether we use income per capita or output per worker has little effect on our results. Overall, the results in Table 4 show a large effect of institutions on economic performance. In the rest of the paper, we investigate the robustness of these results.[23]

[20] In 2SLS estimation, all covariates that are included in the second stage, such as latitude, are also included in the first stage. When these first-stage effects are of no major significance for our argument, we do not report them in the tables to save space.

[21] We should note at this point that if we limit the sample to African countries only, the first-stage relationship using the protection against expropriation variable becomes considerably weaker, and the 2SLS effect of institutions is no longer significant. The 2SLS effect of institutions continue to be significant when we use some (but not all) measures of institutions. Therefore, we conclude that the relationship between settler mortality and institutions is weaker within Africa.

[22] The results with other covariates are also very similar. We repeated the same regressions using a variety of alternative measures of institutions, including constraints on the executive from the Polity III data set, an index of law and order tradition from Political Risk Services, a measure of property rights from the Heritage Foundation, a measure of rule of law from the Fraser Institute, and the efficiency of the judiciary from Business International. The results and the magnitudes are very similar to those reported in Table 4. We also obtained very similar results with the 1970 values for the constraints on the executive and income per capita in 1970, which show that the relationship between institutional measures and income per capita holds across time periods. These results are reported in the Appendix of the working paper version, and are also available from the authors.

[23] In the working paper version, we also investigated the robustness of our results in different subsamples with varying degrees of data quality and different methods of constructing the mortality estimates. The results change very little, for example, when we use data only from Curtin (1989), *Death by Migration,* when we do not assign mortality rates from neighboring disease environments, when

V. Robustness

A. *Additional Controls*

The validity of our 2SLS results in Table 4 depends on the assumption that settler mortality in the past has no direct effect on current economic performance. Although this presumption appears reasonable (at least to us), here we substantiate it further by directly controlling for many of the variables that could plausibly be correlated with both settler mortality and economic outcomes, and checking whether the addition of these variables affects our estimates.[24] Overall, we find that our results change remarkably little with the inclusion of these variables, and many variables emphasized in previous work become insignificant once the effect of institutions is controlled for.

La Porta et al. (1999) argue for the importance of colonial origin (identity of the main colonizing country) as a determinant of current institutions. The identity of the colonial power could also matter because it might have an effect through culture, as argued by David S. Landes (1998). In columns (1) and (2) of Table 5, we add dummies for British and French colonies (colonies of other nations are the omitted group). This has little affect on our results. Moreover, the French dummy in the first stage is estimated to be zero, while the British dummy is positive, and marginally significant. Therefore, as suggested by La Porta et al. (1998), British colonies appear to have better institutions, but this effect is much smaller and weaker than in a specification that does not control for the effect of settler mortality on institutional development.[25] Therefore, it appears that British colonies are found to perform substantially better in other studies in large part because Britain colonized places where settlements were possible, and this made British colonies inherit better institutions. To further investigate this issue, columns (3) and (4) estimate our basic regression for British colonies only. They show that both the relationship between settler mortality and institutions and that between institutions and income in this sample of 25 British colonies are very similar to those in our base sample. For example, the 2SLS estimate of the effect of institutions on income is now 1.07 (s.e. = 0.24) without controlling for latitude and 1.00 (s.e. = 0.22) with latitude. These results suggest that the identity of the colonizer is not an important determinant of colonization patterns and subsequent institutional development.

von Hayek (1960) and La Porta et al. (1999) also emphasize the importance of legal origin. In columns (5) and (6), we control for legal origin. In our sample, all countries have either French or British legal origins, so we simply add a dummy for French legal origin (many countries that are not French colonies nonetheless have French legal origin). Our estimate of the effect of institutions on income per capita is unaffected.[26]

An argument dating back to Max Weber views religion as a key determinant of economic performance. To control for this, in columns (7) and (8), we add the fraction of the populations that are Catholic, Muslim, and of other religions, with Protestants as the omitted group. In the table we report the joint significance level (p-value) of the corresponding F-statistic for these dummies as well as the 2SLS estimate of

the use data for Latin America from naval stations instead of bishops, and when we do not use data from small African samples. These results are available in Appendix Table A5 available from the authors, or in Acemoglu et al. (2000).

[24] Joseph N. Altonji et al. (2000) develop an econometric methodology to assess the importance of omitted variable bias. The basic idea is that if the estimate of the coefficient of interest does not change as additional covariates are included in the regression, it is less likely to change if we were able to add some of the missing omitted variables. Our methodology here is an informal version of this approach.

[25] Moreover, the British colonial dummy is negative and significant in the second stage. The net effect of being a British colony on income per capita is in fact *negative*. More specifically, British colonies have, on average, an index of institution that is 0.63 points lower. Given the 2SLS estimate of 1.10, this translates into 69 log points higher income per capita for British colonies ($1.10 \times 63 \approx 69$). The second-stage effect of being a British colony is -78 log points, implying -9 log point (approximately 10 percent) negative net effect of being a British colony. A possible explanation for this pattern is that (Anglo-Saxon?) researchers are overestimating how "bad" French institutions are, and the second-stage regression is correcting for this.

[26] The first stage shows that French legal origin is associated with worse institutions, but similarly, the net effect of having French legal origin is actually *positive:* $-67 \times 1.1 + 89 = 15$ log points (approximately 15 percent).

TABLE 5—IV REGRESSIONS OF LOG GDP PER CAPITA WITH ADDITIONAL CONTROLS

	Base sample (1)	Base sample (2)	British colonies only (3)	British colonies only (4)	Base sample (5)	Base sample (6)	Base sample (7)	Base sample (8)	Base sample (9)
Panel A: Two-Stage Least Squares									
Average protection against expropriation risk, 1985–1995	1.10 (0.22)	1.16 (0.34)	1.07 (0.24)	1.00 (0.22)	1.10 (0.19)	1.20 (0.29)	0.92 (0.15)	1.00 (0.25)	1.10 (0.29)
Latitude		−0.75 (1.70)				−1.10 (1.56)		−0.94 (1.50)	−1.70 (1.6)
British colonial dummy	−0.78 (0.35)	−0.80 (0.39)							
French colonial dummy	−0.12 (0.35)	−0.06 (0.42)							0.02 (0.69)
French legal origin dummy					0.89 (0.32)	0.96 (0.39)			0.51 (0.69)
p-value for religion variables							[0.001]	[0.004]	[0.42]
Panel B: First Stage for Average Protection Against Expropriation Risk in 1985–1995									
Log European settler mortality	−0.53 (0.14)	−0.43 (0.16)	−0.59 (0.19)	−0.51 (0.14)	−0.54 (0.13)	−0.44 (0.14)	−0.58 (0.13)	−0.44 (0.15)	−0.48 (0.18)
Latitude		1.97 (1.40)				2.10 (1.30)		2.50 (1.50)	2.30 (1.60)
British colonial dummy	0.63 (0.37)	0.55 (0.37)							
French colonial dummy	0.05 (0.43)	−0.12 (0.44)							−0.25 (0.89)
French legal origin					−0.67 (0.33)	−0.7 (0.32)			−0.05 (0.91)
R^2	0.31	0.33	0.30	0.30	0.32	0.35	0.32	0.35	0.45
Panel C: Ordinary Least Squares									
Average protection against expropriation risk, 1985–1995	0.53 (0.19)	0.47 (0.07)	0.61 (0.09)	0.47 (0.06)	0.56 (0.06)	0.56 (0.06)	0.53 (0.06)	0.47 (0.06)	0.47 (0.06)
Number of observations	64	64	25	25	64	64	64	64	64

Notes: Panel A reports the two-stage least-squares estimates with log GDP per capita (PPP basis) in 1995 as dependent variable, and Panel B reports the corresponding first stage. The base case in columns (1) and (2) is all colonies that were neither French nor British. The religion variables are included in the first stage of columns (7) and (8) but not reported here (to save space). Panel C reports the OLS coefficient from regressing log GDP per capita on average protection against expropriation risk, with the other control variables indicated in that column (full results not reported to save space). Standard errors are in parentheses and *p*-values for joint significance tests are in brackets. The religion variables are percentage of population that are Catholics, Muslims, and "other" religions; Protestant is the base case. Our sample is all either French or British legal origin (as defined by La Porta et al., 1999).

the effect of institutions.[27] Finally, column (9) adds all the variables in this table simultaneously. Again, these controls have very little effect on our main estimate.

Another concern is that settler mortality is correlated with climate and other geographic characteristics. Our instrument may therefore be picking up the direct effect of these variables. We investigate this issue in Table 6. In columns (1) and (2), we add a set of temperature and humidity variables (all data from Philip M. Parker, 1997). In the table we report joint significance levels for these variables. Again, they have little effect on our estimates.

[27] The religion dummies are significant in the first stage, but once again they are estimated to have offsetting effects in the second stage, implying little net effect of religion on income.

TABLE 6—ROBUSTNESS CHECKS FOR IV REGRESSIONS OF LOG GDP PER CAPITA

	Base sample (1)	Base sample (2)	Base sample (3)	Base sample (4)	Base sample (5)	Base sample (6)	Base sample (7)	Base sample (8)	Base sample (9)
Panel A: Two-Stage Least Squares									
Average protection against expropriation risk, 1985–1995	0.84 (0.19)	0.83 (0.21)	0.96 (0.28)	0.99 (0.30)	1.10 (0.33)	1.30 (0.51)	0.74 (0.13)	0.79 (0.17)	0.71 (0.20)
Latitude		0.07 (1.60)		−0.67 (1.30)		−1.30 (2.30)		−0.89 (1.00)	−2.5 (1.60)
p-value for temperature variables	[0.96]	[0.97]							[0.77]
p-value for humidity variables	[0.54]	[0.54]							[0.62]
Percent of European descent in 1975			−0.08 (0.82)	0.03 (0.84)					0.3 (0.7)
p-value for soil quality					[0.79]	[0.85]			[0.46]
p-value for natural resources					[0.82]	[0.87]			[0.82]
Dummy for being landlocked					0.64 (0.63)	0.79 (0.83)			0.75 (0.47)
Ethnolinguistic fragmentation							−1.00 (0.32)	−1.10 (0.34)	−1.60 (0.47)
Panel B: First Stage for Average Protection Against Expropriation Risk in 1985–1995									
Log European settler mortality	−0.64 (0.17)	−0.59 (0.17)	−0.41 (0.14)	−0.4 (0.15)	−0.44 (0.16)	−0.34 (0.17)	−0.64 (0.15)	−0.56 (0.15)	−0.59 (0.21)
Latitude		2.70 (2.00)		0.48 (1.50)		2.20 (1.50)		2.30 (1.40)	4.20 (2.60)
R^2	0.39	0.41	0.34	0.34	0.41	0.43	0.27	0.30	0.59
Panel C: Ordinary Least Squares									
Average protection against expropriation risk, 1985–1995	0.41 (0.06)	0.38 (0.06)	0.39 (0.06)	0.38 (0.06)	0.46 (0.07)	0.42 (0.07)	0.46 (0.05)	0.45 (0.06)	0.38 (0.06)

Notes: Panel A reports the two-stage least-squares estimates with log GDP per capita (PPP basis) in 1995, and Panel B reports the corresponding first stages. Panel C reports the OLS coefficient from regressing log GDP per capita on average protection against expropriation risk, with the other control variables indicated in that column (full results not reported to save space). Standard errors are in parentheses and *p*-values for joint significance tests are in brackets. All regressions have 64 observations, except those including natural resources, which have 63 observations. The temperature and humidity variables are: average, minimum, and maximum monthly high temperatures, and minimum and maximum monthly low temperatures, and morning minimum and maximum humidity, and afternoon minimum and maximum humidity (from Parker, 1997). Measures of natural resources are: percent of world gold reserves today, percent of world iron reserves today, percent of world zinc reserves today, number of minerals present in country, and oil resources (thousands of barrels per capita). Measures of soil quality/climate are steppe (low latitude), desert (low latitude), steppe (middle latitude), desert (middle latitude), dry steppe wasteland, desert dry winter, and highland. See Appendix Table A1 for more detailed variable definitions and sources.

A related concern is that in colonies where Europeans settled, the current population consists of a higher fraction of Europeans. One might be worried that we are capturing the direct effect of having more Europeans (who perhaps brought a "European culture" or special relations with Europe). To control for this, we add the fraction of the population of European descent in columns (3) and (4) of Table 6. This variable is insignificant, while the effect of institutions remains highly significant, with a coefficient of 0.96 (s.e. = 0.28). In columns (5) and (6), we control for measures of natural resources, soil quality (in practice soil types), and for whether the country is landlocked. All these controls are insignificant, and have little effect on our 2SLS estimate of the effect of institutions on income per capita.

In columns (7) and (8), we include ethnolinguistic fragmentation as another control and treat it as exogenous. Now the coefficient

of protection against expropriation is 0.74 (s.e. = 0.13), which is only slightly smaller than our baseline estimate. In Appendix A, we show that the inclusion of an endogenous variable positively correlated with income or institutions will bias the coefficient on institutions downwards. Since ethnolinguistic fragmentation is likely to be endogenous with respect to development (i.e., ethnolinguistic fragmentation tends to disappear after the formation of centralized markets; see Weber [1976] or Andersen [1983]) and is correlated with settler mortality, the estimate of 0.74 likely understates the effect of institutions on income. In column (9) of Table 6, we include all these variables together. Despite the large number of controls, protection against expropriation on income per capita is still highly significant, with a somewhat smaller coefficient of 0.71 (s.e. = 0.20), which is again likely to understate the effect of institutions on income because ethnolinguistic fragmentation is treated as exogenous.

Finally, in Table 7, we investigate whether our instrument could be capturing the general effect of disease on development. Sachs and a series of coauthors have argued for the importance of malaria and other diseases in explaining African poverty (see, for example, Bloom and Sachs, 1998; Gallup and Sachs, 1998; Gallup et al., 1998). Since malaria was one of the main causes of settler mortality, our estimate may be capturing the direct effect of malaria on economic performance. We are skeptical of this argument since malaria prevalence is highly endogenous; it is the poorer countries with worse institutions that have been unable to eradicate malaria.[28] While Sachs and coauthors argue that malaria reduces output through poor health, high mortality, and absenteeism, most people who live in high malaria areas have developed some immunity to the disease (see the discussion in Section III, subsection A). Malaria should therefore have little direct effect on economic performance (though, obviously, it will have very high social costs). In contrast, for Europeans, or anyone else who has not been exposed to malaria as a young child, malaria is usually fatal, making malaria prevalence a key determinant of European settlements and institutional development.

In any case, controlling for malaria does not change our results. We do this in columns (1) and (2) by controlling for the fraction of the population who live in an area where falciporum malaria is endemic in 1994 (as constructed and used by Gallup et al., 1998). Since malaria prevalence in 1994 is highly endogenous, the argument in Appendix A implies that controlling for it directly will underestimate the effect of institutions on performance. In fact, the coefficient on protection against expropriation is now estimated to be somewhat smaller, 0.69 instead of 0.94 as in Table 4. Nevertheless, the effect remains highly significant with a standard error of 0.25, while malaria itself is insignificant.

In a comment on the working paper version of our study, John W. McArthur and Sachs (2001) discuss the role of geography and institutions in determining economic performance. They accept our case for the importance of institutions, but argue that more general specifications show that the disease environment and health characteristics of countries (their "geography") matter for economic performance. In particular, they extend our work by controlling for life expectancy and infant mortality, and they also instrument for these health variables using geographic variables such as latitude and mean temperature. Table 7 also expands upon the specifications that McArthur and Sachs suggest. Columns (3)–(6) include life expectancy and infant mortality as exogenous controls. The estimates show a significant effect of institutions on income, similar to, but smaller than, our baseline estimates. Infant mortality is also marginally significant. Since health is highly endogenous, the coefficient on these variables will be biased up, while the coefficient of institutions will be biased down (see Appendix A). These estimates are therefore consistent with

[28] For example, the United States eliminated malaria from the Panama Canal Zone, and Australia eliminated it from Queensland (see Crosby, 1986 pp. 141–42). Even in Africa, there have been very successful campaigns against malaria, including those in Algeria and that conducted by the Rio-Tinto Zinc mining company in Zambia (then Northern Rhodesia). The WHO's Roll Back Malaria program contains a number of effective recommendations for controlling malaria that are relatively straightforward to implement if families have enough money (e.g., insecticide-treated bed nets).

TABLE 7—GEOGRAPHY AND HEALTH VARIABLES

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
	Instrumenting only for average protection against expropriation risk						Instrumenting for all right-hand-side variables			Yellow fever instrument for average protection against expropriation risk	
Panel A: Two-Stage Least Squares											
Average protection against expropriation risk, 1985–1995	0.69 (0.25)	0.72 (0.30)	0.63 (0.28)	0.68 (0.34)	0.55 (0.24)	0.56 (0.31)	0.69 (0.26)	0.74 (0.24)	0.68 (0.23)	0.91 (0.24)	0.90 (0.32)
Latitude		−0.57 (1.04)		−0.53 (0.97)		−0.1 (0.95)					
Malaria in 1994	−0.57 (0.47)	−0.60 (0.47)					−0.62 (0.68)				
Life expectancy			0.03 (0.02)	0.03 (0.02)				0.02 (0.02)			
Infant mortality					−0.01 (0.005)	−0.01 (0.006)			−0.01 (0.01)		
Panel B: First Stage for Average Protection Against Expropriation Risk in 1985–1995											
Log European settler mortality	−0.42 (0.19)	−0.38 (0.19)	−0.34 (0.17)	−0.30 (0.18)	−0.36 (0.18)	−0.29 (0.19)	−0.41 (0.17)	−0.40 (0.17)	−0.40 (0.17)		
Latitude		1.70 (1.40)		1.10 (1.40)		1.60 (1.40)	−0.81 (1.80)	−0.84 (1.80)	−0.84 (1.80)		
Malaria in 1994	−0.79 (0.54)	−0.65 (0.55)									
Life expectancy			0.05 (0.02)	0.04 (0.02)							
Infant mortality					−0.01 (0.01)	−0.01 (0.01)					
Mean temperature							−0.12 (0.05)	−0.12 (0.05)	−0.12 (0.05)		
Distance from coast							0.57 (0.51)	0.55 (0.52)	0.55 (0.52)		
Yellow fever dummy										−1.10 (0.41)	−0.81 (0.38)
R^2	0.3	0.31	0.34	0.35	0.32	0.34	0.37	0.36	0.36	0.10	0.32
Panel C: Ordinary Least Squares											
Average protection against expropriation risk, 1985–1995	0.35 (0.06)	0.35 (0.06)	0.28 (0.05)	0.28 (0.05)	0.29 (0.05)	0.28 (0.05)	0.35 (0.06)	0.29 (0.05)	0.29 (0.05)	0.48 (0.06)	0.39 (0.06)
Number of observations	62	62	60	60	60	60	60	59	59	64	64

Notes: Panel A reports the two-stage least-squares estimates with log GDP per capita (PPP basis) in 1995, and Panel B reports the corresponding first stages. Panel C reports the coefficient from an OLS regression with log GDP per capita as the dependent variable and average protection against expropriation risk and the other control variables indicated in each column as independent variables (full results not reported to save space). Standard errors are in parentheses. Columns (1)–(6) instrument for average protection against expropriation risk using log mortality and assume that the other regressors are exogenous. Columns (7)–(9) include as instruments average temperature, amount of territory within 100 km of the coast, and latitude (from McArthur and Sachs, 2001). Columns (10) and (11) use a dummy variable for whether or not a country was subject to yellow fever epidemics before 1900 as an instrument for average protection against expropriation. See Appendix Table A1 for more detailed variable definitions and sources.

institutions being the major determinant of income per capita differences, with little effect from geography/health variables.

Columns (7)–(9) report estimates from models that treat both health and institutions as endogenous, and following McArthur and Sachs, instrument for them using latitude, mean temperature, and distance from the coast as instruments in addition to our instrument, settler mortality. McArthur and Sachs (2001) report that in these regressions the institution variable is still significant, but geography/health are also significant. In contrast to McArthur and Sachs' results, we find that only institutions are signif-

icant. This difference is due to the fact that McArthur and Sachs include Britain and France in their sample. Britain and France are not in our sample, which consists of only ex-colonies (there is no reason for variation in the mortality rates of British and French troops at home to be related to their institutional development). It turns out that once Britain and France are left out, the McArthur and Sachs' specification generates no evidence that geography/health variables have an important effect on economic performance.[29]

As a final strategy to see whether settler mortality could be proxying for the current disease environment, we estimated models using a yellow fever instrument. This is a dummy variable indicating whether the area was ever affected by yellow fever (from Oldstone, 1998; see Appendix Table A1). This is an attractive alternative strategy because yellow fever is mostly eradicated today, so this dummy should not be correlated with the current disease environment. The disadvantage of this approach is that there is less variation in this instrument than our settler mortality variable. Despite this, the yellow fever results, reported in columns (10) and (11) of Table 7, are encouraging. The estimate in our base sample is 0.91 (s.e. = 0.24) comparable to our baseline estimate of 0.95 reported in Table 4. Adding continent dummies in column (11) reduces this estimate slightly to 0.90 (s.e. = 0.32).[30]

[29] McArthur and Sachs (2001) also report specifications with more instruments. However, using six or seven instruments with only 64 observations leads to the "too-many-instruments" problem, typically biasing the IV estimate towards the OLS estimate (see John Bound et al., 1995). We therefore did not pursue these estimates further.

Finally, McArthur and Sachs also argue that our ex-colonies sample may not have enough geographic variation. In their view, this may be why we do not find a role for geographic variables. Nonetheless, there is substantial variation in the geography variables in our sample which includes countries such as Canada, the United States, New Zealand, and Australia. The standard deviation of distance from the equator in the world is 1.89, greater than 1.33 in our sample. This is mainly because there are a large number of European countries with high latitudes in the world sample, but not in our sample.

[30] If we drop the Neo-Europes (not reported here), the estimate is still similar and highly significant, 1.05 (s.e. = 0.35).

B. *Overidentification Tests*

We can also investigate the validity of our approach by using overidentification tests. According to our theory, settler mortality (M) affected settlements (S); settlements affected early institutions (C); and early institutions affected current institutions (R)—cf., equations (2), (3), and (4). We can test whether any of these variables, C, S, and M, has a direct effect on income per capita, log y, by using measures of C and S as additional instruments. The overidentification test presumes that one of these instruments, say S, is truly exogenous, and tests for the exogeneity of the others, such as settler mortality. This approach is useful since it is a direct test of our exclusion restriction. However, such tests may not lead to a rejection if all instruments are invalid, but still highly correlated with each other. Therefore, the results have to be interpreted with caution.

Overall, the overidentification test will reject the validity of our approach if either (i) the equation of interest, (1), does not have a constant coefficient, i.e., $\log y_i = \mu + \alpha_i R_i + \varepsilon_i$, where i denotes country, or (ii) C or S has a direct effect on income per capita, log y_i (i.e., either S or C is correlated with ε_i), or (iii) settler mortality, M, has an effect on log y_i that works through another variable, such as culture.

The data support the overidentifying restrictions implied by our approach.[31] This implies that, subject to the usual problems of power associated with overidentification tests, we can rule out all three of the above possibilities. This gives us additional confidence that settler mortality is a valid instrument and that we are estimating the effect of institutions on current performance with our instrumental-variable strategy (i.e., not capturing the effect of omitted variables).

[31] In some specifications, the overidentification tests using measures of early institutions reject at that 10-percent level (but not at the 5-percent level). There are in fact good reasons to expect institutions circa 1900 to have a direct effect on income today (and hence the overidentifying tests to reject our restrictions): these institutions should affect physical and human capital investments at the beginning of the century, and have some effect on current income levels through this channel.

TABLE 8—OVERIDENTIFICATION TESTS

	Base sample (1)	Base sample (2)	Base sample (3)	Base sample (4)	Base sample (5)	Base sample (6)	Base sample (7)	Base sample (8)	Base sample (9)	Base sample (10)
Panel A: Two-Stage Least Squares										
Average protection against expropriation risk, 1985–1995	0.87	0.92	0.71	0.68	0.72	0.69	0.60	0.61	0.55	0.56
	(0.14)	(0.20)	(0.15)	(0.20)	(0.14)	(0.19)	(0.14)	(0.17)	(0.12)	(0.14)
Latitude		−0.47		−0.34		0.31		−0.41		−0.16
		(1.20)		(1.10)		(1.05)		(0.92)		(0.81)
Panel B: First Stage for Average Protection Against Expropriation Risk										
European settlements in 1900	3.20	2.90								
	(0.62)	(0.83)								
Constraint on executive in 1900			0.32	0.26						
			(0.08)	(0.09)						
Democracy in 1900					0.24	0.20				
					(0.06)	(0.07)				
Constraint on executive in first year of independence							0.25	0.22		
							(0.08)	(0.08)		
Democracy in first year of independence									0.19	0.17
									(0.05)	(0.05)
R^2	0.30	0.30	0.20	0.24	0.24	0.26	0.19	0.25	0.26	0.30
Panel C: Results from Overidentification Test										
p-value (from chi-squared test)	[0.67]	[0.96]	[0.09]	[0.20]	[0.11]	[0.28]	[0.67]	[0.79]	[0.22]	[0.26]
Panel D: Second Stage with Log Mortality as Exogenous Variable										
Average protection against expropriation risk, 1985–1995	0.81	0.88	0.45	0.42	0.52	0.48	0.49	0.49	0.4	0.41
	(0.23)	(0.30)	(0.25)	(0.30)	(0.23)	(0.28)	(0.23)	(0.25)	(0.18)	(0.19)
Log European settler mortality	−0.07	−0.05	−0.25	−0.26	−0.21	−0.22	−0.14	−0.14	−0.19	−0.19
	(0.17)	(0.18)	(0.16)	(0.17)	(0.15)	(0.16)	(0.16)	(0.15)	(0.13)	(0.12)
Latitude		−0.52		0.38		0.28		−0.38		−0.17
		(1.15)		(0.89)		(0.86)		(0.84)		(0.73)

Notes: Panel A reports the two-stage least-squares estimates with log GDP per capita (PPP basis) in 1995 as the dependent variable, and Panel B reports the corresponding first stage (latitude is included in even-numbered columns but is never significant and not reported here to save space). Panel C reports the p-value for the null hypothesis that the coefficient on average protection against expropriation risk in the second-stage regression (i.e., Panel A) is the same as when instrumented using log mortality of settlers in addition to the indicated instruments. Panel D reports results from the regression in which log mortality is included as an exogenous variable and current institutions are instrumented using the alternative instrument indicated. Standard errors are in parentheses. All regressions with constraint on executive and democracy in first year of independence also include years since independence as a regressor. All regressions have 60 observations, except those with democracy in 1900 which have 59 observations and those with European settlements in 1900 which have 63 observations.

The results of the overidentification tests, and related results, are reported in Table 8. In the top panel, Panel A, we report the 2SLS estimates of the effect of protection against expropriation on GDP per capita using a variety of instruments other than mortality rates, while Panel B gives the corresponding first stages. These estimates are always quite close to those reported in Table 4. For example, in column (1), we use European settlements in 1900 as the *only* instrument for institutions. This results in an estimated effect of 0.87 (with standard error 0.14), as compared to our baseline estimate of 0.94. The other columns add latitude, and use other instruments such as constraint on the executive in 1900 and in the first year of independence, and democracy in 1900.

Panel D reports an easy-to-interpret version of the overidentification test. It adds the log of mortality as an exogenous regressor. If mortality rates faced by settlers had a direct effect on income per capita, we would expect this variable to come in negative and significant. In all cases, it is small and statistically insignificant. For example, in column (1), log mortality has a coefficient of −0.07 (with standard error 0.17). This confirms that the

impact of mortality rates faced by settlers likely works through their effect on institutions.

Finally, for completeness, in Panel C we report the p-value from the appropriate χ^2 overidentification test. This tests whether the 2SLS coefficients estimated with the instruments indicated in Panels A and B versus the coefficients estimated using (log) settler mortality in addition to the "true" instruments are significantly different (e.g., in the first column, the coefficient using European settlements alone is compared to the estimate using European settlements and log mortality as instruments). We never reject the hypothesis that they are equal at the 5-percent significance level. So these results also show no evidence that mortality rates faced by settlers have a direct effect—or an effect working through a variable other than institutions—on income per capita.

VI. Concluding Remarks

Many economists and social scientists believe that differences in institutions and state policies are at the root of large differences in income per capita across countries. There is little agreement, however, about what determines institutions and government attitudes towards economic progress, making it difficult to isolate exogenous sources of variation in institutions to estimate their effect on performance. In this paper we argued that differences in colonial experience could be a source of exogenous differences in institutions.

Our argument rests on the following premises: (1) Europeans adopted very different colonization strategies, with different associated institutions. In one extreme, as in the case of the United States, Australia, and New Zealand, they went and settled in the colonies and set up institutions that enforced the rule of law and encouraged investment. In the other extreme, as in the Congo or the Gold Coast, they set up extractive states with the intention of transferring resources rapidly to the metropole. These institutions were detrimental to investment and economic progress. (2) The colonization strategy was in part determined by the feasibility of European settlement. In places where Europeans faced very high mortality rates, they could not go and settle, and they were more likely to set up extractive states. (3) Finally, we argue that these early institutions persisted to the present. Determinants of whether Europeans could go and settle in the colonies, therefore, have an important effect on institutions today. We exploit these differences as a source of exogenous variation to estimate the impact of institutions on economic performance.

There is a high correlation between mortality rates faced by soldiers, bishops, and sailors in the colonies and European settlements; between European settlements and early measures of institutions; and between early institutions and institutions today. We estimate large effects of institutions on income per capita using this source of variation. We also document that this relationship is not driven by outliers, and is robust to controlling for latitude, climate, current disease environment, religion, natural resources, soil quality, ethnolinguistic fragmentation, and current racial composition.

It is useful to point out that our findings do not imply that institutions today are predetermined by colonial policies and cannot be changed. We emphasize colonial experience as one of the many factors affecting institutions. Since mortality rates faced by settlers are arguably exogenous, they are useful as an instrument to isolate the effect of institutions on performance. In fact, our reading is that these results suggest substantial economic gains from improving institutions, for example as in the case of Japan during the Meiji Restoration or South Korea during the 1960's.

There are many questions that our analysis does not address. Institutions are treated largely as a "black box": The results indicate that reducing expropriation risk (or improving other aspects of the "cluster of institutions") would result in significant gains in income per capita, but do not point out what concrete steps would lead to an improvement in these institutions. Institutional features, such as expropriation risk, property rights enforcement, or rule of law, should probably be interpreted as an equilibrium outcome, related to some more fundamental "institutions," e.g., presidential versus parliamentary system, which can be changed directly. A more detailed analysis of the effect of more fundamental institutions on property

rights and expropriation risk is an important area for future study.

Appendix A: Bias in the Effect of Institutions When Other Endogenous Variables Are Included

To simplify notation, suppose that R_i is exogenous, and another variable that is endogenous, z_i, such as prevalence of malaria or ethnolinguistic fragmentation, is added to the regression. Then, the simultaneous equations model becomes

$$Y_i = \mu_0 + \alpha R_i + \pi z_i + \varepsilon_i$$

$$z_i = \mu_1 + \phi Y_i + \eta_i,$$

where $Y_i = \log y_i$. We presume that $\alpha \geq 0$, $\phi < 0$, and $\pi < 0$, which implies that we interpret z_i as a negative influence on income. Moreover, this naturally implies that $\text{cov}(\eta_i, \varepsilon_i) < 0$ and $\text{cov}(z_i, R_i) < 0$, that is, the factor z_i is likely to be negatively correlated with positive influences on income.

Standard arguments imply that

$$\text{plim } \hat{\alpha} = \alpha + \frac{\text{cov}(\tilde{R}_i, \varepsilon_i)}{\text{var}(\tilde{R}_i)}$$

$$= \alpha - \kappa \cdot \frac{\text{cov}(z_i, \varepsilon_i)}{\text{var}(\tilde{R}_i)},$$

where κ and $\tilde{R}_i$ are the coefficient and the residual from the auxiliary equation, $R_i = \kappa_0 + \kappa z_i + \tilde{R}_i$, and so $\kappa = \text{cov}(z_i, R_i)/\text{var}(z_i) < 0$, which is negative due to the fact that $\text{cov}(R_i, z_i) < 0$. The reduced form for z_i is:

$$z_i = \frac{1}{1 - \phi\pi}((\mu + \phi\pi) + \phi\alpha R_i + \phi\varepsilon_i + \eta_i). \tag{A1}$$

We impose the regularity condition $\phi \cdot \pi < 1$, so that an increase in the disturbance to the z-equation, η_i, actually increases z_i. Now using this reduced form, we can write

$$\text{plim } \hat{\alpha} = \alpha - \kappa \cdot \frac{\text{cov}(z_i, \varepsilon_i)}{\text{var}(\tilde{R}_i)} = \alpha - \kappa \cdot \frac{(\sigma_{\varepsilon\eta} + \phi\sigma_\varepsilon^2)}{(1 - \phi\pi) \cdot \text{var}(\tilde{R}_i)} \tag{A2}$$

where σ_ε^2 is the variance of ε, and $\sigma_{\varepsilon\eta}$ is the covariance of ε and η.

Substituting for κ in (A2), we obtain:

$$\text{plim } \hat{\alpha} = \alpha - \frac{(\sigma_{\varepsilon\eta} + \phi\sigma_\varepsilon^2)}{(1 - \phi\pi) \cdot \text{var}(\tilde{R}_i)} \cdot \frac{\text{cov}(z_i, R_i)}{\text{var}(z_i)}.$$

Recall that $\phi < 0$, $\sigma_{\varepsilon\eta} < 0$, and $\text{cov}(z_i, R_i) < 0$. Therefore, plim $\hat{\alpha} < \alpha$, and when we control for the endogenous variable z_i, the coefficient on our institution variable will be biased downwards.