ACEMOGLU, JOHNSON, ROBINSON

Lots of papers show correlations between low Y/L & bad instns. But causality?

IF bad instns → low Y/L, what causes bad instns? Why would a society adopt bad instns?

AJR's big ideas:

(1) Good instns: property rights for everyone

Bad instns: "extractive". Elite gets stuff, others face expropriation risk. Elites act as rent seeking predators.

(2) In European colonies, Euros established (or preserved existing) extractive instns. in places where Euros wanted to extract, not settle. established good instns in places they wanted to settle.

(3) Instns persist to today. How? Not explained in these papers; see later work.

AJR

AJR's big ideas (cont.)

(4) Euro choice to settle/extract in response to conditions they found in 17th–19th c.

Some of the conditions that discouraged settlement / promoted extraction are otherwise unrelated to current Y/L:

— local disease environment then, measured by "settler mortality"

— density/sophistication of native popn.

  — disease vector

  — local L is extractable resource

  — native civ. has extractive instns, Euros can take over at low cost.

These can be <u>instruments</u> to see effect of instns. on Y/L! in samples of former colonies.

AJR (cont.)

"Colonial Origins" (AER, 2001)

Local disease environment

Tricky bit: "exclusion restriction."

Lots of people find "tropics," bad disease environment associated with low Y/L in cross-country regs, have stories for it.

AJR must convince us that's not true for stuff that deterred white settlers.

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

AJR

"Colonial Origins" (cont.)

(4)

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The Colonial Origins of Comparative Development: An Empirical Investigation: Comment†

By DAVID Y. ALBOUY*

This comment argues that there are several reasons to doubt the reliability and comparability of their European settler mortality rates and the conclusions that depend on them. First, out of 64 countries in the sample, only 28 countries have mortality rates that originate from within their own borders. The other 36 countries in the sample are assigned rates based on conjectures the authors make as to which countries have similar disease environments. These assignments are generally unfounded and potentially contradictory. Six assignments are based on an incorrect interpretation of former colonial names for Mali. Another 16 assignments are extrapolated from thin bishop mortality data in Latin America from Gutierrez (1986), using a "benchmarking" procedure that can produce highly contradictory rates, depending on how the data are benchmarked. At a minimum, the sharing of mortality rates across countries requires that statistics be corrected for clustering (Moulton 1990). This correction alone noticeably reduces the significance of the results. If, in the hope of reducing measurement error, the 36 conjectured mortality rates are dropped from the sample, the point estimates relating mortality rates with expropriation risk become substantially smaller, particularly in the presence of covariates, which often gain significance.

Second, the mortality rates never come from actual European settlers, although some settler rates are available in the authors' sources. Instead, the data come primarily from European and American soldiers in the nineteenth century. In some countries, rates apply to soldiers at peace in barracks, while in others the rates apply to soldiers on campaign. As is well known, soldiers on campaign typically have higher mortality from disease. This causes problems as AJR uses rates campaigns more often in countries with greater expropriation risk and lower GDP, artificially favoring the article's hypothesis. In a few countries, the data include the peak mortality rates of African laborers, but these are not comparable with average soldier mortality rates. Controlling for the source of the mortality rates weakens the empirical relationship between expropriation risk and mortality rates substantially. Furthermore, if these controls are added and the conjectured data are removed, the relationship virtually disappears, suggesting that it is largely an artifact of the data's construction.

AJR (cont.)

"Reversal of Fortune" (QJE, 2002)

Natives' urbanization rates, popn. density

It is not needed for their main point, but they additionally argue that civilization meant high T/L, so "reversal."

(maybe not true: think Malthusian mod.)

Again, main weakness of argument is "exclusion" restriction,

AJR can do a test because they have another instrument: settler mortality.

AJR

"Reversal" (cont.)

V.A. *Institutions and the Reversal*

We next provide evidence suggesting that institutional differences statistically account for the reversal in relative incomes. If the institutional reversal is the reason why there was a reversal in income levels among the former colonies, then once we account for the role of institutions appropriately, the reversal should disappear. That is, according to this view, the reversal documented in Figures I and II and Tables III, IV, V, and VI reflects the correlation between economic prosperity in 1500 and income today working through the intervening variable, institutions.

How do we establish that an intervening variable X is responsible for the correlation between Z and Y? Suppose that the true relationship between Y, and X, and Z is

(1) $$Y = \alpha \cdot X + \beta \cdot Z + \epsilon,$$

income — instns. — conditions in 1500

and $X = \lambda Z + \nu$ and $\beta = 0$

there is a simple way of testing this hypothesis, which is to run an OLS regression of Y on Z and X:

(2) $$Y = a \cdot X + b \cdot Z + u_2$$

to obtain the estimates $\hat{a}$ and $\hat{b}$. The fact that ϵ in (1) is independent of both X and Z rules out omitted variable bias, so $\text{plim}\hat{a} = \alpha$ and $\text{plim}\hat{b} = \beta$. Hence, a simple test of whether $\hat{b} = 0$ is all that is required to test our hypothesis that the effect of Z is through X alone.

In practice, there are likely to be problems due to omitted variables, endogeneity bias because Y has an effect on X, and attenuation bias because X is measured with error or corresponds poorly to the real concept that is relevant to development (which is likely to be a broad range of institutions, whereas we only have an index for a particular type of institutions). So the above procedure is not possible.