Economics 270B
Ph.D. Development Economics

Professor Ted Miguel
Department of Economics
University of California, Berkeley

Lecture 5 – March 2, 2015

I. Overview of International Economic Development
Lecture 1: Understanding economic growth and development (1/26)
Lecture 1B: Persistence of historical institutions and shocks
(read during holiday week of 2/16)

Lecture 2: The Psychology of Poverty (2/2)

II. Human Capital in Economic Development
Lectures 3-4: Education (2/9, 2/23)
Lectures 5-7: Health and nutrition (3/2, 3/9, 3/16)

III. Political economy
Lectures 8-9: Democracy, Corruption and Development (3/30, 4/6)
(guest lectures by Prof. Fred Finan)

Lecture 10: Ethnic and Social Divisions (4/13)
Lectures 11-12: The Political Economy of Conflict (4/20, 4/27)

I. Overview of International Economic Development
Lecture 1: Understanding economic growth and development (1/26)
Lecture 1B: Persistence of historical institutions and shocks
(read during holiday week of 2/16)

Lecture 2: The Psychology of Poverty (2/2)

II. Human Capital in Economic Development
Lectures 3-4: Education (2/9, 2/23)
Lectures 5-7: Health and nutrition (3/2, 3/9, 3/16)

III. Political economy
Lectures 8-9: Democracy, Corruption and Development (3/30, 4/6)
(guest lectures by Prof. Fred Finan)

Lecture 10: Ethnic and Social Divisions (4/13)
Lectures 11-12: The Political Economy of Conflict (4/20, 4/27)

- Prerequisites: Graduate economic theory, econometrics

- Grading:
  Four referee reports – 40%
  → Report #3 on Morjaria paper due next week (3/9)

  Two problem sets – 20%
  Research proposal – 30%
  Class participation – 10%
  No final exam

- All readings are available on bCourses

Any questions?

Lecture 5 outline

(1) Health, education and economic development

(2) Miguel and Kremer (2004) on deworming in children

(3) Acemoglu and Johnson (2007) on life expectancy and income growth across countries

(1) Health and economic development

- An observation: health and wealth are correlated both across countries and across people within societies. Why?

- Question #1: What impact of **income on health**? Do richer people consume more "health"?

- Question #2: What is the **impact of health/nutrition on labor productivity**? Does health cause wealth?

- Question #3: **Which policies/institutions** improve the delivery of public health services in poor countries?

(1) A simple model of health and education

(1) A simple model of health and education

- A production function for academic skills, as measured by test scores (or attendance) when the child is in primary school (time 2). Time period 1 is pre-primary:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- H_t is child health at t, EI_t is parents' provision of educational inputs (supplies, time spent teaching the child), α is the child's innate intelligence (ability), SC is school characteristics, YS years of schooling by time 2

(1) A simple model of health and education

- A production function for academic skills, as measured by test scores (or attendance) when the child is in primary school (time 2). Time period 1 is pre-primary:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- H_t is child health at t, EI_t is parents' provision of educational inputs (supplies, time spent teaching the child), α is the child's innate intelligence (ability), SC is school characteristics, YS years of schooling by time 2

(1) A simple model of health and education

- A production function for academic skills, as measured by test scores (or attendance) when the child is in primary school (time 2). Time period 1 is pre-primary:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- H_t is child health at *t*, EI_t is parents' provision of educational inputs (supplies, time spent teaching the child), α is the child's innate intelligence (ability), *SC* is school characteristics, *YS* years of schooling by time 2
- The production function shows how child health status in both time periods could affect learning. This is a *structural relationship* because all of the variables in the production function *directly* affect academic skills, and all variables with direct effects included (assumed)

(1) A simple model of health and education

- If one had accurate data on all the variables in equation (1), and knew the correct functional form, one could estimate it using relatively simple methods, such as OLS, to obtain unbiased estimates of the direct impacts of all variables on child academic skills

(1) A simple model of health and education

- If one had accurate data on all the variables in equation (1), and knew the correct functional form, one could estimate it using relatively simple methods, such as OLS, to obtain unbiased estimates of the direct impacts of all variables on child academic skills

- However, some of the variables are unobserved and the observable variables themselves are *chosen* in a household optimization problem. Correlations between unobserved child ability and healthiness complicate interpretation. For example, innately clever kids could naturally be healthier. Parents may also direct more educational investments towards healthier (or sicker) kids, depending on the household welfare function.

(1) A simple model of health and education

- Child health is also chosen (in part) by households:

  $H_1 = H(C_1, M_1, HE_1, \eta)$

  $H_2 = H(C_2, M_2, HE_2, \eta; H_1)$

- C_t is the child's consumption of the aggregate good (e.g., food) in period t, M_t is health inputs ("medicine") broadly defined, HE_t is the local health environment (prevalence of infectious diseases, air / water quality, etc.) and η is the innate healthiness of the child

(1) A simple model of health and education

- Child health is also chosen (in part) by households:

  $H_1 = H(C_1, M_1, HE_1, \eta)$

  $H_2 = H(C_2, M_2, HE_2, \eta; H_1)$

- C_t is the child's consumption of the aggregate good (e.g., food) in period t, M_t is health inputs ("medicine") broadly defined, HE_t is the local health environment (prevalence of infectious diseases, air / water quality, etc.) and η is the innate healthiness of the child

(1) A simple model of health and education

- Child health is also chosen (in part) by households:

  $H_1 = H(C_1, M_1, HE_1, \eta)$

  $H_2 = H(C_2, M_2, HE_2, \eta; H_1)$

- C_t is the child's consumption of the aggregate good (e.g., food) in period t, M_t is health inputs ("medicine") broadly defined, HE_t is the local health environment (prevalence of infectious diseases, air / water quality, etc.) and η is the innate healthiness of the child

  -- Households then maximize the utility function

  $U = U(C_1, C_2, H_1, H_2, T_2)$ subject to a budget constraint

- Variables are difficult to measure; investments respond to α and η; α and η are plausibly correlated

(1) Randomized evaluation methods

- One approach to addressing econometric identification concerns around health and income is the randomized evaluation approach
- Randomized provision of a health/nutrition intervention breaks the link between household characteristics, (unobserved) child innate ability and health, and prior investments in child health/education

(1) Randomized evaluation methods

- One approach to addressing econometric identification concerns around health and income is the randomized evaluation approach
- Randomized provision of a health/nutrition intervention breaks the link between household characteristics, (unobserved) child innate ability and health, and prior investments in child health/education

- There may be endogenous household behavioral response to an intervention. Thus the difference between the treatment / comparison groups should be thought of as the combined impact of the intervention *per se* together with any resulting behavioral changes (though these changes can also be measured)

(1) Randomized evaluation methods

- Imagine a public intervention that improves the health outcomes of young children, increasing H_1

- In the production function for academic skills:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- This exogenous change in young child health not only directly affects academic performance (potentially) but also affects later health outcomes H_2 as well as parent education investment levels EI_2 and "medicine" M_2

(1) Randomized evaluation methods

- Imagine a public intervention that improves the health outcomes of young children, increasing H_1

- In the production function for academic skills:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- This exogenous change in young child health not only directly affects academic performance (potentially) but also affects later health outcomes H_2 as well as parent education investment levels EI_2 and "medicine" M_2

(1) Randomized evaluation methods

- Imagine a public intervention that improves the health outcomes of young children, increasing H_1

- In the production function for academic skills:

  (1) $T_2 = T(H_1, H_2, EI_1, EI_2, \alpha, SC, YS)$

- This exogenous change in young child health not only directly affects academic performance (potentially) but also affects later health outcomes H_2 as well as parent education investment levels EI_2 and "medicine" M_2

- The overall program impact – directly, and indirectly through behavior – is of public policy interest; the extent of behavioral change is also very important

(2) Miguel and Kremer (2004)

(2) Miguel and Kremer (2004)

- To frame the discussion: why might there be scope for public intervention in the health sector? In other words, why don't households provide the necessary health investments themselves privately?

(2) Miguel and Kremer (2004)

- To frame the discussion: why might there be scope for public intervention in the health sector? In other words, why don't households provide the necessary health investments themselves privately?

  (1) Poor (or incorrect) knowledge of new health technologies among individuals

  (2) Credit constraints prevent good investments

(2) Miguel and Kremer (2004)

- To frame the discussion: why might there be scope for public intervention in the health sector? In other words, why don't households provide the necessary health investments themselves privately?

  (1) Poor (or incorrect) knowledge of new health technologies among individuals

  (2) Credit constraints prevent good investments

  (3) Within-household agency problems or imperfect parental altruism towards children

  (4) Positive treatment externalities

(2) Miguel and Kremer (2004)

- To frame the discussion: why might there be scope for public intervention in the health sector? In other words, why don't households provide the necessary health investments themselves privately?

  (1) Poor (or incorrect) knowledge of new health technologies among individuals

  (2) Credit constraints prevent good investments

  (3) Within-household agency problems or imperfect parental altruism towards children

  (4) Positive treatment externalities (this paper)

Implications of treatment externalities

- Standard public finance theory: individual behaviors that generate positive externalities for other people are "under-provided", since people do not take into account the social benefits of their actions. Thus in the absence of a subsidy, there is too little deworming

  → a strong rationale for public deworming subsidies

- Previous randomized studies of deworming within schools showed positive but small impacts on child health, nutrition. Why? Is "deworming not worth it"?

  -- Simple T – C analysis may not give reliable estimates in the presence of externalities (e.g., infectious diseases)

(2) Miguel and Kremer (2004)

- Worms infections (e.g., hookworm, whipworm, roundworm, schistosomiasis) are among the world's most common infections

- We study school-based deworming treatment. In our sample of rural Kenyan school children, over 90% were infected at baseline. Between one third and one half had "serious" infections

- Worms do not reproduce within the body. They pass worm larvae out through human fecal matter and this can infect others. Treatment generates a positive externality by reducing this transmission to others

(2) Miguel and Kremer (2004)

- 75 primary schools, over 30,000 children (aged 6-18)

- Deworming treatment (drugs, health education) phased in experimentally across three treatment groups. These groups are similar along observables

(2) Miguel and Kremer (2004)

- 75 primary schools, over 30,000 children (aged 6-18)

- Deworming treatment (drugs, health education) phased in experimentally across three treatment groups. These groups are similar along observables
  - Listed school alphabetically (by zone), by pupil enrollment (by school), and counted off 1-2-3, 1-2-3, etc. Assignment is arbitrary and should be orthogonal to omitted variables

- Group 1: **treatment 1998 and 1999**
- Group 2: no treatment 1998, **treatment 1999**
- Group 3: no treatment in 1998 or 1999

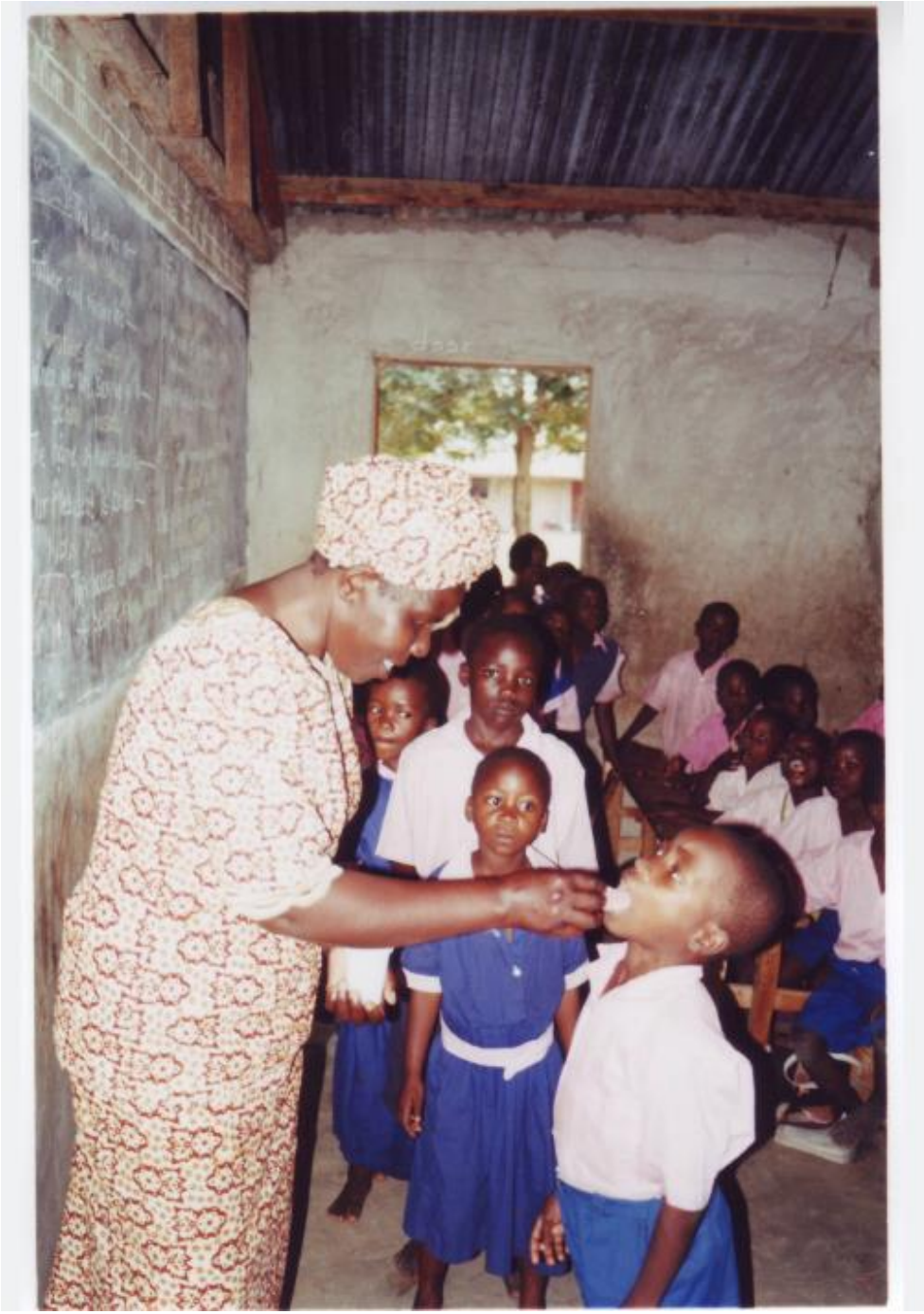


TABLE I

1998 AVERAGE PUPIL AND SCHOOL CHARACTERISTICS, PRE-TREATMENT[a]

	Group 1 (25 schools)	Group 2 (25 schools)	Group 3 (25 schools)	Group 1 – Group 3	Group 2 – Group 3
Panel A: Pre-school to Grade 8					
Male	0.53	0.51	0.52	0.01	−0.01
				(0.02)	(0.02)
Proportion girls <13 years, and all boys	0.89	0.89	0.88	0.00	0.01
				(0.01)	(0.01)
Grade progression (= Grade − (Age − 6))	−2.1	−1.9	−2.1	−0.0	0.1
				(0.1)	(0.1)
Year of birth	1986.2	1986.5	1985.8	0.4**	0.8***
				(0.2)	(0.2)
Panel B: Grades 3 to 8					
Attendance recorded in school registers (during the four weeks prior to the pupil survey)	0.973	0.963	0.969	0.003	−0.006
				(0.004)	(0.004)
Access to latrine at home	0.82	0.81	0.82	0.00	−0.01
				(0.03)	(0.03)
Have livestock (cows, goats, pigs, sheep) at home	0.66	0.67	0.66	−0.00	0.01
				(0.03)	(0.03)
Weight-for-age Z-score (low scores denote undernutrition)	−1.39	−1.40	−1.44	0.05	0.04
				(0.05)	(0.05)

TABLE II

JANUARY 1998 HELMINTH INFECTIONS, PRE-TREATMENT, GROUP 1 SCHOOLS[a]

	Prevalence of infection	Prevalence of moderate-heavy infection	Average infection intensity, in eggs per gram (s.e.)
Hookworm	0.77	0.15	426 (1055)
Roundworm	0.42	0.16	2337 (5156)
Schistosomiasis, all schools	0.22	0.07	91 (413)
Schistosomiasis, schools <5 km from Lake Victoria	0.80	0.39	487 (879)
Whipworm	0.55	0.10	161 (470)
At least one infection	0.92	0.37	–
Born since 1985	0.92	0.40	–
Born before 1985	0.91	0.34	–
Female	0.91	0.34	–
Male	0.93	0.38	–
At least two infections	0.31	0.10	–
At least three infections	0.28	0.01	–

TABLE III

PROPORTION OF PUPILS RECEIVING DEWORMING TREATMENT IN PSDP[a]

	Group 1		Group 2		Group 3	
	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years
	Treatment		*Comparison*		*Comparison*	
Any medical treatment in 1998 (For grades 1–8 in early 1998)	0.78	0.19	0	0	0	0
Round 1 (March–April 1998), Albendazole	0.69	0.11	0	0	0	0
Round 1 (March–April 1998), Praziquantel[b]	0.64	0.34	0	0	0	0
Round 2 (Oct.–Nov. 1998), Albendazole	0.56	0.07	0	0	0	0

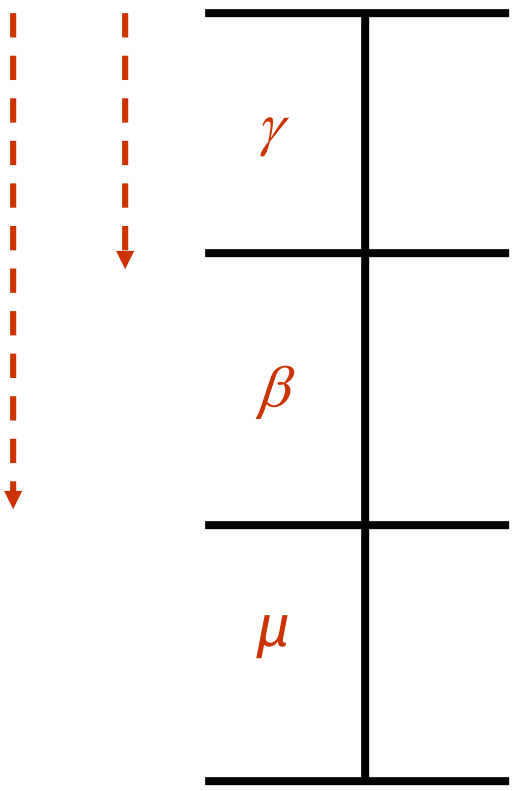
(2) Miguel and Kremer (2004)

- One of the goals of the paper is to compare the naïve treatment effect estimator, “Treatment minus control”, $E(Y_{ij} \mid T_{1i} =1) – E(Y_{ij} \mid T_{1i} =0)$, to estimators that take into account “contamination” of the experiment from externalities. This contamination may produce gains in the control group

(2) Miguel and Kremer (2004)

- One of the goals of the paper is to compare the naïve treatment effect estimator, "Treatment minus control", $E(Y_{ij} \mid T_{1i} =1) – E(Y_{ij} \mid T_{1i} =0)$, to estimators that take into account "contamination" of the experiment from externalities. This contamination may produce gains in the control group

- Externalities would lead us to doubly under-estimate treatment effects in a mass treatment program: (i) miss impacts in the comparison group, (ii) understate impacts in the treatment group
  - A real concern in existing studies that randomize within schools and often found small impacts



Infection level, Control

Infection level, Control benefiting from externalities

Infection level, Treatment benefiting from externalities

0

"Naïve" treatment effect is β.

Actual treatment effect for a mass program is $\beta + \gamma > \beta$

Externalities γ for the control group, too.

→ *If proportion ρ of schools in a region are treated, then the average effect is $\rho(\gamma + \beta) + (1- \rho)\gamma = \gamma + \rho\beta$*

(2) Miguel and Kremer (2004)

- The naïve program impact estimator (in existing studies, which often find small or insignificant effects):

  $E(Y_{ij} \mid T_{1i} = 1) - E(Y_{ij} \mid T_{1i} = 0)$, which can be re-written

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})$

TABLE V

JANUARY TO MARCH 1999, HEALTH AND HEALTH BEHAVIOR DIFFERENCES BETWEEN GROUP 1 (1998 TREATMENT) AND GROUP 2 (1998 COMPARISON) SCHOOLS[a]

	Group 1	Group 2	Group 1 – Group 2
Panel A: Helminth Infection Rates			
Any moderate-heavy infection, January–March 1998	0.38	–	–
Any moderate-heavy infection, 1999	0.27	0.52	−0.25***
			(0.06)
Hookworm moderate-heavy infection, 1999	0.06	0.22	−0.16***
			(0.03)
Roundworm moderate-heavy infection, 1999	0.09	0.24	−0.15***
			(0.04)
Schistosomiasis moderate-heavy infection, 1999	0.08	0.18	−0.10*
			(0.06)
Whipworm moderate-heavy infection, 1999	0.13	0.17	−0.04
			(0.05)
Panel B: Other Nutritional and Health Outcomes			
Sick in past week (self-reported), 1999	0.41	0.45	−0.04**
			(0.02)
Sick often (self-reported), 1999	0.12	0.15	−0.03**
			(0.01)
Height-for-age Z-score, 1999 (low scores denote undernutrition)	−1.13	−1.22	0.09*
			(0.05)
Weight-for-age Z-score, 1999 (low scores denote undernutrition)	−1.25	−1.25	−0.00
			(0.04)

(2) Miguel and Kremer (2004)

- The naïve program impact estimator (in existing studies, which often find small or insignificant effects):

  $E(Y_{ij} \mid T_{1i} = 1) - E(Y_{ij} \mid T_{1i} = 0)$, which can be re-written

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})$

- The program impact estimator, taking into account treatment externalities:

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)$

  which is equivalent to

  $\{E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})\}$
  $+ \{E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)\}$

(2) Miguel and Kremer (2004)

- The naïve program impact estimator (in existing studies, which often find small or insignificant effects):

  $E(Y_{ij} \mid T_{1i} = 1) - E(Y_{ij} \mid T_{1i} = 0)$, which can be re-written

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})$

- The program impact estimator, taking into account treatment externalities:

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)$

  which is equivalent to

  $\{E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})\}$
  $+ \{E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)\}$
  $=$ "β" + "γ"

(2) Miguel and Kremer (2004)

- The naïve program impact estimator (in existing studies, which often find small or insignificant effects):

  $E(Y_{ij} \mid T_{1i} = 1) - E(Y_{ij} \mid T_{1i} = 0)$, which can be re-written

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})$

- The program impact estimator, taking into account treatment externalities:

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)$

  which is equivalent to

  $\{E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG})\}$
  $+ \{E(Y_{ij} \mid T_{1i} = 0, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)\}$

- The estimator for a mass treatment program would be:

  $E(Y_{ij} \mid T_{1i} = 1, N^T = N) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)$

(2) Miguel and Kremer (2004)

- More generally, you can imagine the effect of program assignment is a function of the proportion of the local population that is also receiving treatment.

(2) Miguel and Kremer (2004)

- More generally, you can imagine the effect of program assignment is a function of the proportion of the local population that is also receiving treatment.

- Let the real-world treatment effect of interest be:

$$\pi(N^{AVG}) \equiv E(Y_{ij} \mid T_{1i}=1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i}=0, N^T = 0)$$

$$= \{E(Y_{ij} \mid T_{1i}=1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i}=0, N^T = N^{AVG})\} + \{E(Y_{ij} \mid T_{1i}=0, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i}=0, N^T = 0)\}$$

$$= \beta(N^{AVG}) + \gamma(N^{AVG})$$

= Direct effect of treatment assignment (at local treatment saturation, N^{AVG}) + Externality (at N^{AVG})

(2) Miguel and Kremer (2004)

- There are three relevant cases for $\beta(N^{AVG})$:

- Case 1: Treatment and local saturation are neither complements nor substitutes, i.e., $\beta(N^{AVG}) = \beta^* \ \forall \ N^{AVG}$
- Case 2: Treatment and local saturation are complements, i.e., $\beta(N^{AVG})$ is increasing in N^{AVG}
- Case 3: Treatment and local saturation are substitutes, i.e., $\beta(N^{AVG})$ is decreasing in N^{AVG}

(2) Miguel and Kremer (2004)

- There are three relevant cases for $\beta(N^{AVG})$:

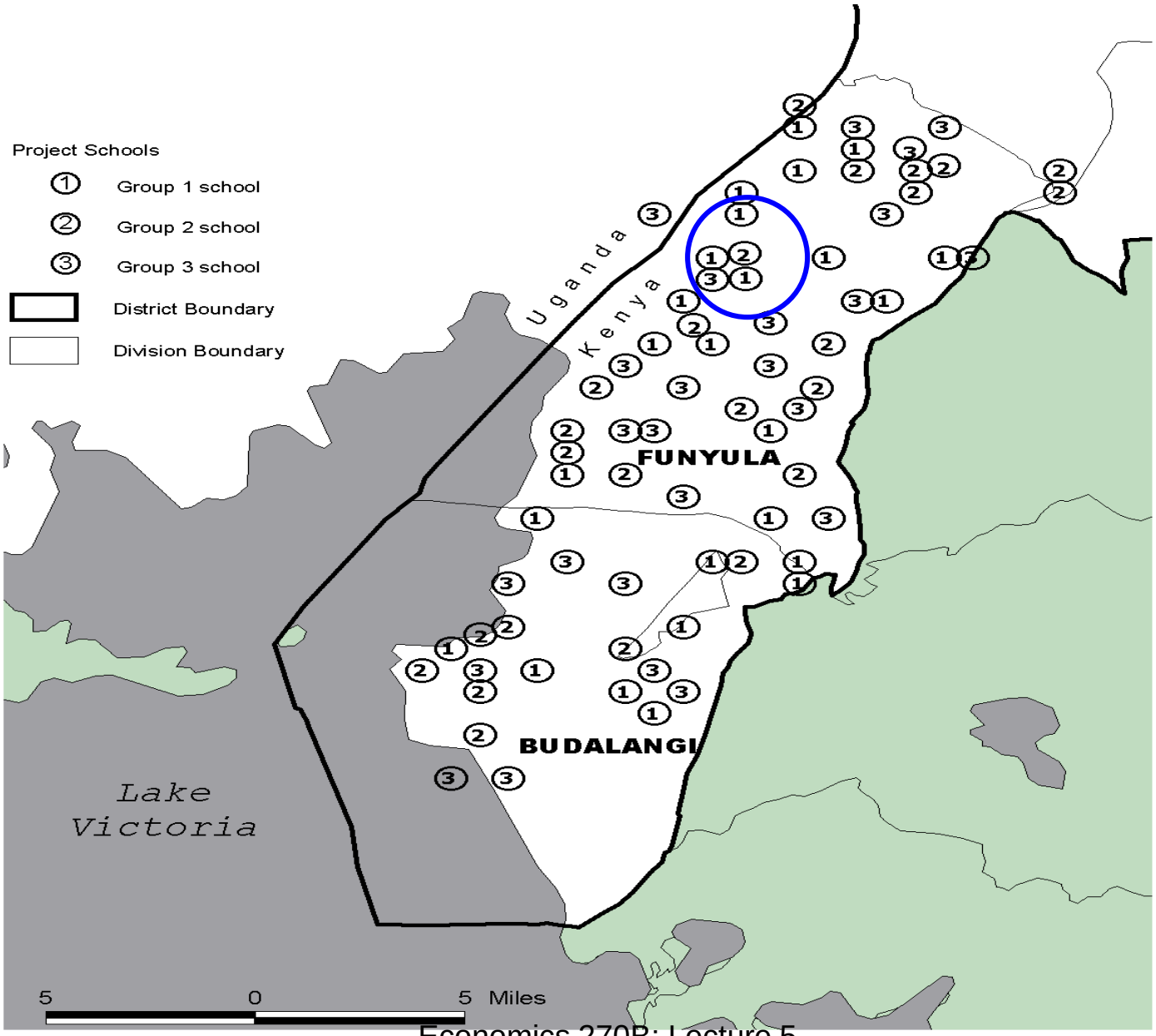
- Case 1: Treatment and local saturation are neither complements nor substitutes, i.e., $\beta(N^{AVG}) = \beta^* \ \forall \ N^{AVG}$
- Case 2: Treatment and local saturation are complements, i.e., $\beta(N^{AVG})$ is increasing in N^{AVG}
- Case 3: Treatment and local saturation are substitutes, i.e., $\beta(N^{AVG})$ is decreasing in N^{AVG}

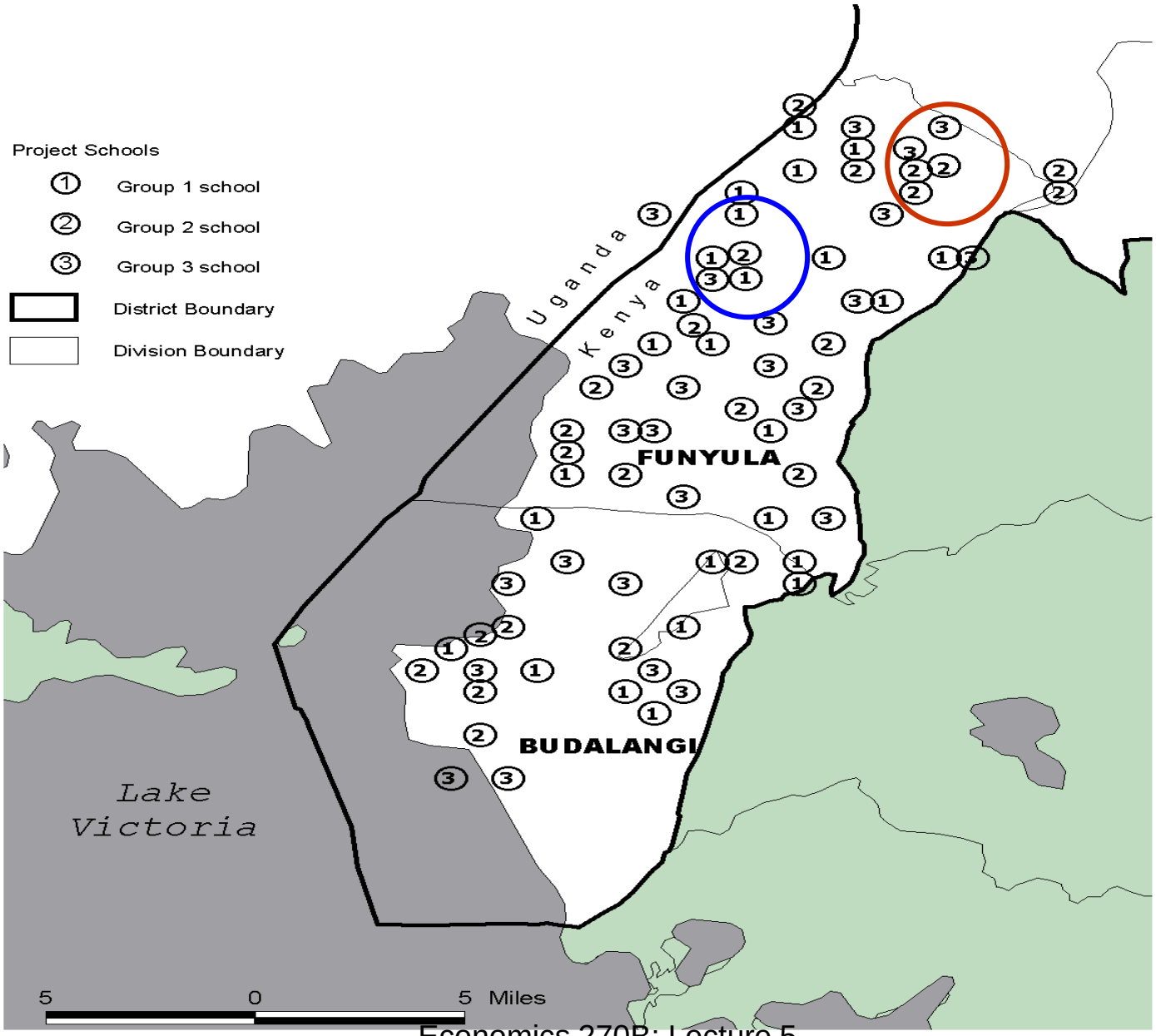
- It is easy to show that all estimates are "bounds" as long as externalities are always (weakly) positive.
- We find only weak evidence of interactions between treatment and externality exposure, or of non-linear externalities → additively separable, linear specification as the benchmark

We first estimate program impacts in treatment schools, as well as cross-school treatment externalities:[24]

$$(1) \qquad Y_{ijt} = a + \beta_1 \cdot T_{1it} + \beta_2 \cdot T_{2it} + X'_{ijt}\delta + \sum_d (\gamma_d \cdot N^T_{dit}) + \sum_d (\phi_d \cdot N_{dit}) + u_i + e_{ijt}.$$

Y_{ijt} is the individual health or education outcome, where i refers to the school, j to the student, and $t \in \{1, 2\}$ to the year of the program; T_{1it} and T_{2it} are indicator variables for school assignment to the first and second year of deworming treatment, respectively; and X_{ijt} are school and pupil characteristics. N_{dit} is the total number of pupils in primary schools at distance d from school i in year t, and N^T_{dit} is the number of these pupils in schools randomly assigned to deworming treatment. For example, in Sections 5 and 6, $d = 03$ denotes schools that are located within three kilometers of school i, and $d = 36$ denotes schools that are located between three to six kilometers away.[25] Individual disturbance terms are assumed to be independent across schools, but are allowed to be correlated for observations within the same school, where the school effect is captured in the u_i term.


Project Schools
Group 1 school
Group 2 school
Group 3 school
District Boundary
Division Boundary
Uganda
Kenya
FUNYULA
BUDALANGI
Lake Victoria
5 0 5 Miles


Project Schools
Group 1 school
Group 2 school
Group 3 school
District Boundary
Division Boundary
Uganda
Kenya
FUNYULA
BUDALANGI
Lake Victoria
5 0 5 Miles

Another form of externalities: within-school

Another form of externalities: within-school

$$(3) \qquad Y_{ijt} = a + \beta_1 \cdot T_{1it} + b_1 \cdot D_{1ij} + b_2 \cdot (T_{1it} * D_{1ij}) + X'_{ijt}\delta + \sum_d (\gamma_d \cdot N^T_{dit}) + \sum_d (\phi_d \cdot N_{dit}) + u_i + e_{ijt}.$$

where D_1 is an indicator for actually taking the treatment when offered it for the first time (i.e., 1998 for Group 1 and 1999 for Group 2 schools)

TABLE VI

DEWORMING HEALTH EXTERNALITIES WITHIN SCHOOLS, JANUARY TO MARCH 1999[a]

	Group 1, Treated in 1998	Group 1, Untreated in 1998	Group 2, Treated in 1999	Group 2, Untreated in 1999	(Group 1, Treated 1998) – (Group 2, Treated 1999)	(Group 1, Untreated 1998) – (Group 2, Untreated 1999)
Panel A: Selection into Treatment						
Any moderate-heavy infection, 1998	0.39	0.44	–	–	–	–
Proportion of 1998 parasitological sample tracked to 1999 sample[b]	0.36	0.36	–	–	–	–
Access to latrine at home, 1998	0.84	0.80	0.81	0.86	0.03	–0.06
					(0.04)	(0.05)
Grade progression (= Grade – (Age – 6)), 1998	–2.0	–1.8	–1.8	–1.8	–0.2**	–0.0
					(0.1)	(0.2)
Weight-for-age (Z-score), 1998 (low scores denote undernutrition)	–1.58	–1.52	–1.57	–1.46	–0.01	–0.06
					(0.06)	(0.11)
Malaria/fever in past week (self-reported), 1998	0.37	0.41	0.40	0.39	–0.03	–0.01
					(0.04)	(0.06)
Clean (observed by field worker), 1998	0.53	0.59	0.60	0.66	–0.07	–0.07
					(0.05)	(0.10)
Panel B: Health Outcomes						
Girls <13 years, and all boys						
Any moderate-heavy infection, 1999	0.24	0.34	0.51	0.55	–0.27***	–0.21**
					(0.06)	(0.10)

Cross-school infection externalities (1999)

- Large reductions in moderate-heavy infection levels within 3 km (2 miles) of treatment schools in 1999, smaller reductions up to 6 km
- An average reduction in moderate-heavy infections of >10 percentage points in the study area can be attributed to cross-school externalities

- Coding error in original paper only considered the **12 schools closest** to each school; correcting this does not affect the 0-3 km effect (since never more than 12 schools in 3 km) but leads the overall 3-6 km effect to be estimated less precisely (CEGA Working Paper 2014)

Figure B1. Original vs. updated "overall effect", with 95% confidence intervals

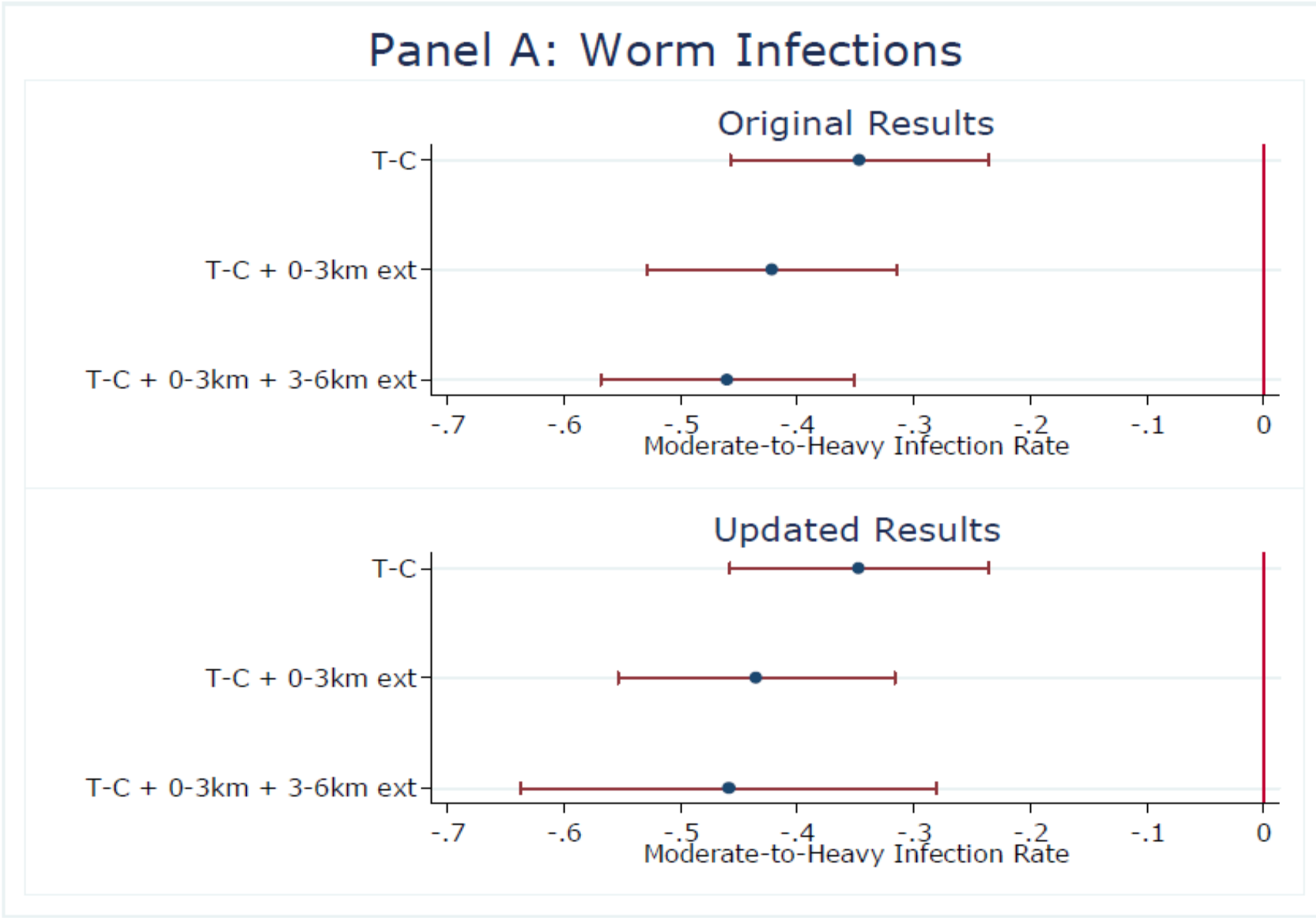


Table B1: Summary of any moderate-to-heavy helminth infection results, updated and original

	UPDATED			ORIGINAL		
	(1)	(2)	(3)	(4)	(5)	(6)
Treatment Indicator	-0.347***	-0.333***	-0.313***	-0.347***	-0.311***	-0.247***
	(0.052)	(0.052)	(0.057)	(0.052)	(0.052)	(0.053)
Treatment pupils w/in 3 km		-0.234**	-0.212**		-0.249***	-0.256***
(per 1000 pupils)		(0.097)	(0.104)		(0.085)	(0.087)
Treatment pupils w/in 3 - 6 km			-0.050			-0.140**
(per 1000 pupils)			(0.077)			(0.060)
Total PSDP 'eligible' students w/in 3 km		0.069*	0.046		0.074**	0.109***
(per 1000 pupils)		(0.037)	(0.036)		(0.033)	(0.040)
Total PSDP 'eligible' students w/in 3-6 km			-0.022			0.133**
(per 1000 pupils)			(0.039)			(0.056)
Calculated Effects						
Average 0-3 km externality effect		-0.102**	-0.090**		-0.111***	-0.106***
		(0.043)	(0.044)		(0.038)	(0.037)
Average 3-6 km externality effect			-0.052			-0.096**
			(0.079)			(0.042)
Average overall cross-school externality effect		-0.102**	-0.146		-0.111***	-0.212***
		(0.043)	(0.110)		(0.038)	(0.065)
Overall deworming effect	-0.347***	-0.435***	-0.459***	-0.347***	-0.421***	-0.460***
	(0.057)	(0.061)	(0.091)	(0.057)	(0.055)	(0.055)

(2) Miguel and Kremer (2004)

- Educational outcomes: school absenteeism (both from poor attendance and drop outs) fall by roughly 7 to 8 percentage points, or one quarter

  -- One of the most cost-effective ways to boost school participation estimated in less developed countries

Table B2: Summary of school participation results, updated and original

	UPDATED			ORIGINAL		
	(1)	(2)	(3)	(4)	(5)	(6)
Treatment Indicator	0.057***	0.058***	0.055***	0.051**	0.054**	0.055**
	(0.014)	(0.014)	(0.014)	(0.022)	(0.023)	(0.023)
Treatment pupils w/in 3 km		0.045**	0.038*		0.046**	0.048**
(per 1000 pupils)		(0.021)	(0.021)		(0.018)	(0.019)
Treatment pupils w/in 3 - 6 km			-0.024			-0.013
(per 1000 pupils)			(0.015)			(0.015)
Total PSDP 'eligible' students w/in 3 km		-0.030**	-0.030**		-0.031***	-0.037***
(per 1000 pupils)		(0.013)	(0.012)		(0.012)	(0.012)
Total PSDP 'eligible' students w/in 3-6 km			0.012			-0.014
(per 1000 pupils)			(0.009)			(0.012)
Calculated Effects						
Average 0-3 km externality effect		0.027**	0.023*		0.028**	0.029**
		(0.013)	(0.013)		(0.011)	(0.012)
Average 3-6 km externality effect			-0.040			-0.009
			(0.024)			(0.011)
Average overall cross-school externality effect		0.027**	-0.017		0.028**	0.020
		(0.013)	(0.030)		(0.011)	(0.013)
Overall deworming effect	0.057***	0.085***	0.039	0.051**	0.081***	0.075***
	(0.014)	(0.017)	(0.032)	(0.022)	(0.026)	(0.027)

(2) Miguel and Kremer (2004)

- Educational outcomes: school absenteeism (both from poor attendance and drop outs) fall by roughly 7 to 8 percentage points, or one quarter

  -- One of the most cost-effective ways to boost school participation estimated in less developed countries

- But test scores do not improve in either year 1 or year 2 (or in cognitive tests administered in year 3)

  -- The average test gain from deworming is **zero**. Why? increased congestion in the classroom; the quality of classroom learning is low; time lags; other explanations?

TABLE X

ACADEMIC EXAMINATIONS, INDIVIDUAL-LEVEL DATA[a]

	Dependent variable: ICS Exam Score (normalized by standard)		
	(1)	(2)	(3) Among those who filled in the 1998 pupil survey
Average school participation (during the year of the exam)	0.63***		
	(0.07)		
First year as treatment school (T1)		−0.032	−0.030
		(0.046)	(0.049)
Second year as treatment school (T2)		0.001	0.009
		(0.073)	(0.081)
1996 District exam score, school average	0.74***	0.71***	0.75***
	(0.07)	(0.07)	(0.07)
Grade indicators, school assistance controls, and local pupil density controls	Yes	Yes	Yes
R^2	0.14	0.13	0.15
Root MSE	0.919	0.923	0.916
Number of observations	24958	24958	19072
Mean of dependent variable	0.020	0.020	0.039

Cost-benefit calculations

- Cost of this program: US$1.46 per pupil per year
- Cost of a larger-scale program in neighboring Tanzania: only US$0.49 per pupil per year

- Cost of health education component (classroom lessons, teacher training) was US$0.44 per pupil per year

- How do these costs compare to the later labor market effects? Discussed hypothetically in Miguel and Kremer (2004) and using follow-up data in Baird et al (2015).

(3) Acemoglu and Johnson (2007)

(3) Acemoglu and Johnson (2007)

- One approach to the difficulties of micro-analysis: exploit policy changes in cross-country data

- They argue that the large increases in life expectancy – and presumably gains in other dimensions other health, i.e., morbidity – across countries since the "international epidemiological transition" in the 1950s has not translated into faster economic growth

  -- An important piece of support for Acemoglu, Johnson and Robinson (2001, *AER*), to rule out direct tropical disease effects on development

(3) Acemoglu and Johnson (2007)

- One approach to the difficulties of micro-analysis: exploit policy changes in cross-country data

- They argue that the large increases in life expectancy – and presumably gains in other dimensions other health, i.e., morbidity – across countries since the "international epidemiological transition" in the 1950s has not translated into faster economic growth

  -- An important piece of support for Acemoglu, Johnson and Robinson (2001, *AER*), to rule out direct tropical disease effects on development

- Instrumental variable approach: use country levels of mortality from 15 leading (and later "eradicated") diseases circa 1940 as an IV for the increase in life expectancy from 1940-1980 (and 2000)

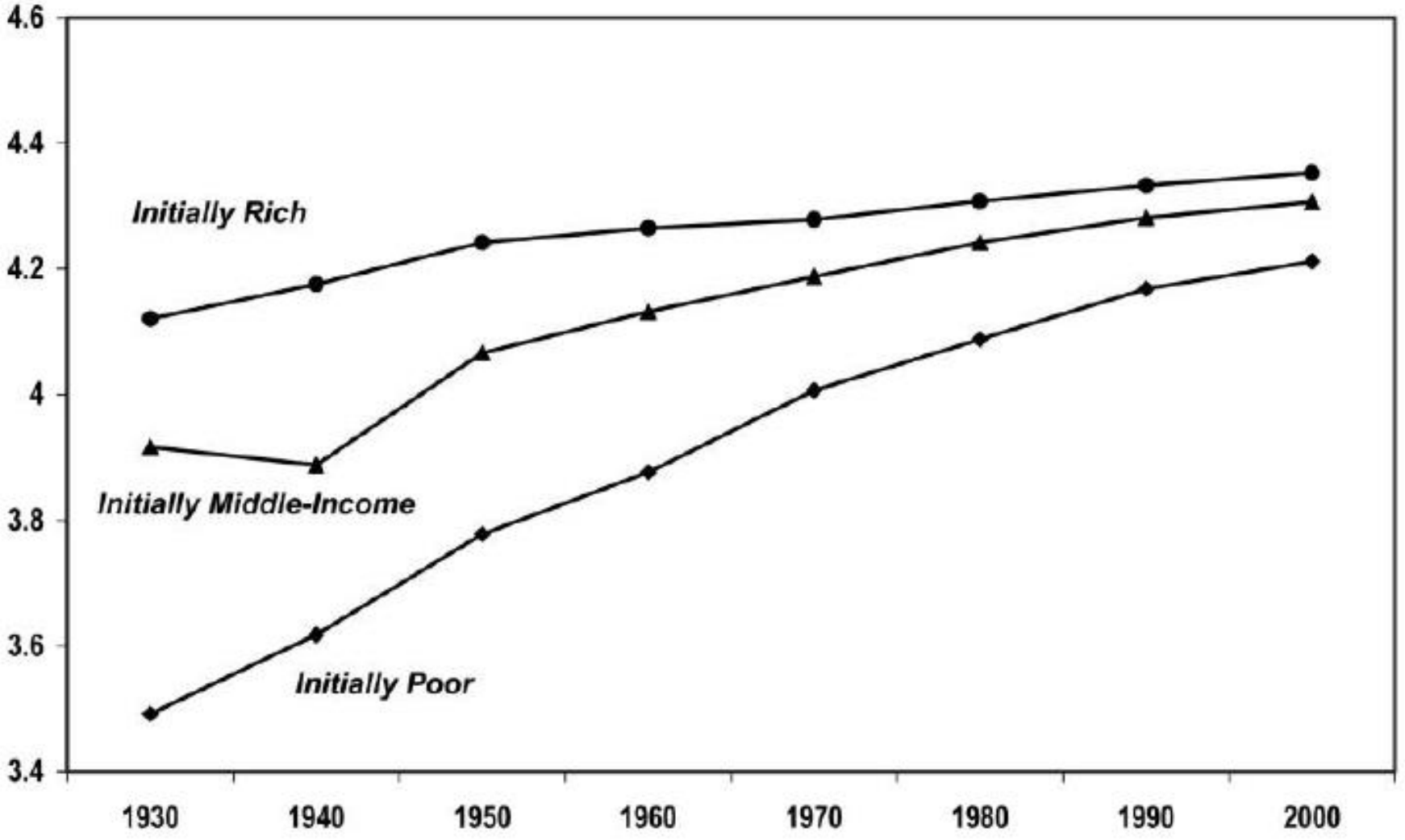


FIG. 1.—Log life expectancy at birth for initially rich, middle-income, and poor countries in the base sample.

(3) Acemoglu and Johnson (2007)

- The discovery of new vaccines (e.g., yellow fever), antiobiotics (e.g., streptomycin for TB) and chemicals (DDT for mosquito spraying), together with better hygiene and sanitation knowledge, all contributed to greatly improved life expectancy after roughly 1940

(3) Acemoglu and Johnson (2007)

- Punch line: increased life expectancy led to large increases in population (1.7% for each 1% increase in life expectancy, due to fertility effects) and no increase in aggregate income. Thus per capita income fell substantially in these countries relative to wealthy countries that experienced smaller mortality reductions

(3) Acemoglu and Johnson (2007)

- Punch line: increased life expectancy led to large increases in population (1.7% for each 1% increase in life expectancy, due to fertility effects) and no increase in aggregate income. Thus per capita income fell substantially in these countries relative to wealthy countries that experienced smaller mortality reductions

- Why? If certain factors of production are fixed in the long-run (e.g., land) or medium-term (capital), then per capita income may fall when population increases

  -- Apparently any gains in productivity (better human capital or TFP) due to health gains were outweighed by this population effect. If key factors are fixed, then rising population lowers output per capita.

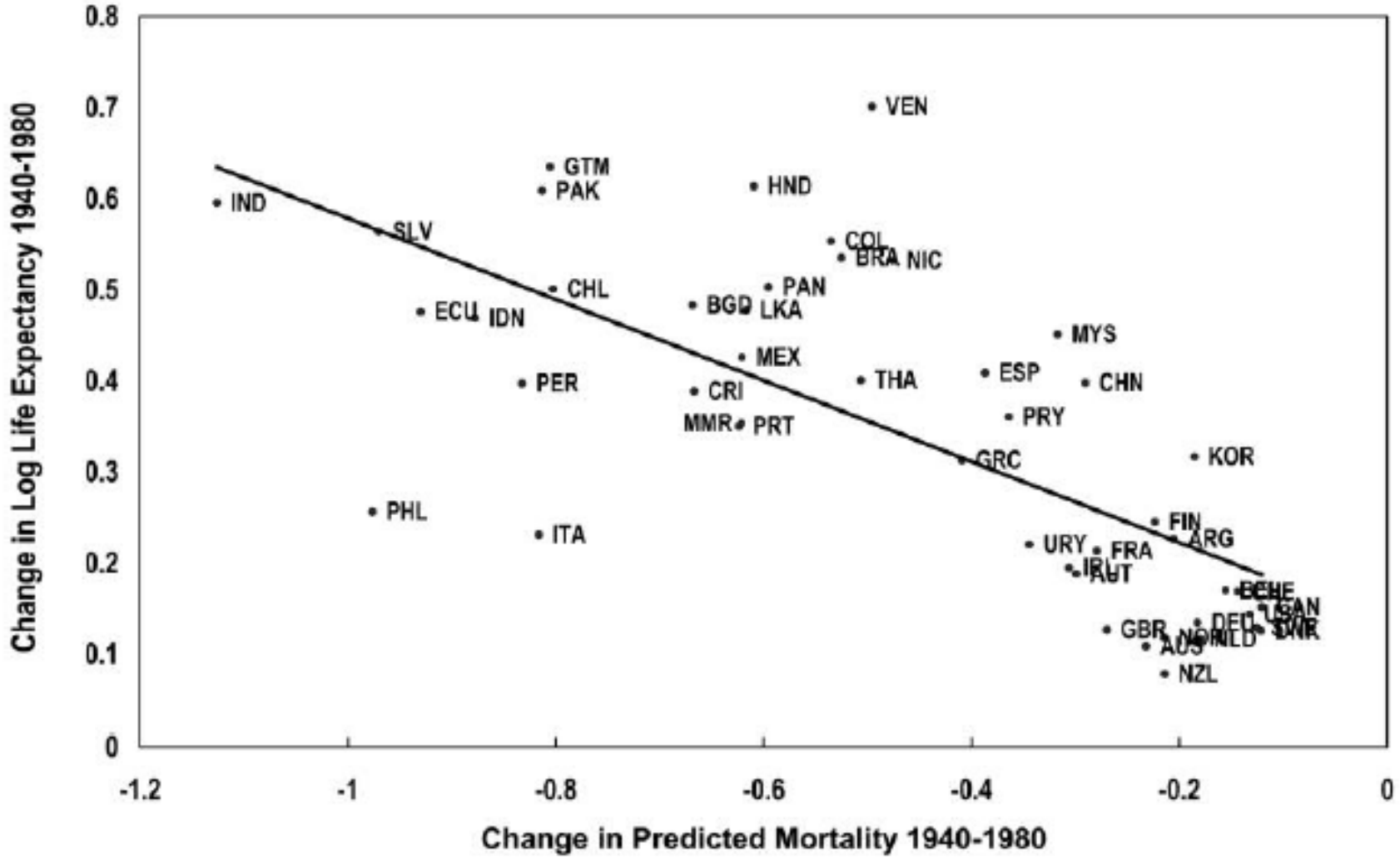


FIG. 3.—Change in log life expectancy and change in predicted mortality, 1940–80, base sample.

TABLE 5
FIRST-STAGE ESTIMATES: PREDICTED MORTALITY AND LIFE EXPECTANCY
Dependent Variable: Log Life Expectancy

	BASELINE PREDICTED MORTALITY						
	All Countries (1)	Base Sample (2)	Base Sample (3)	Low- and Middle-Income Countries Only (4)	Base Sample: Interaction with Institutions (5)	Base Sample: Interaction with Initial (1930) log GDP per Capita (6)	Base Sample: Interaction with Continent Dummies (7)
	A. Long Differences						
	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 2000	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980	Just 1940 and 1980
Predicted mortality	−.39	−.45	−.56	−.31	−.35	−.25	−.30
	(.07)	(.06)	(.07)	(.08)	(.07)	(.09)	(.07)
R^2	.93	.95	.95	.95	.96	.96	.96
Number of observations	150	94	94	72	94	94	94
Number of countries	75	47	47	36	47	47	47

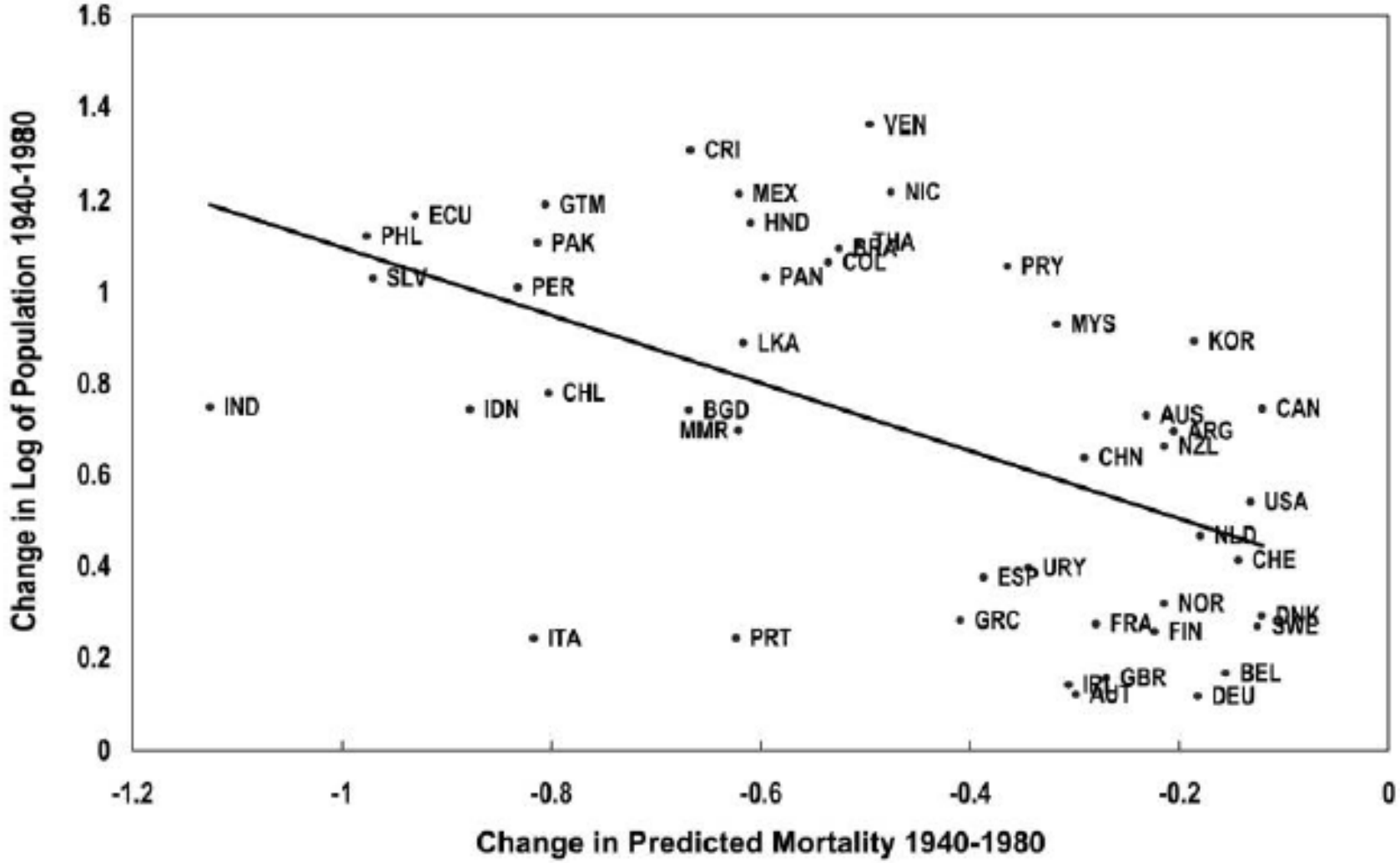


FIG. 7.—Change in log of population and change in predicted mortality, 1940–80, base sample.

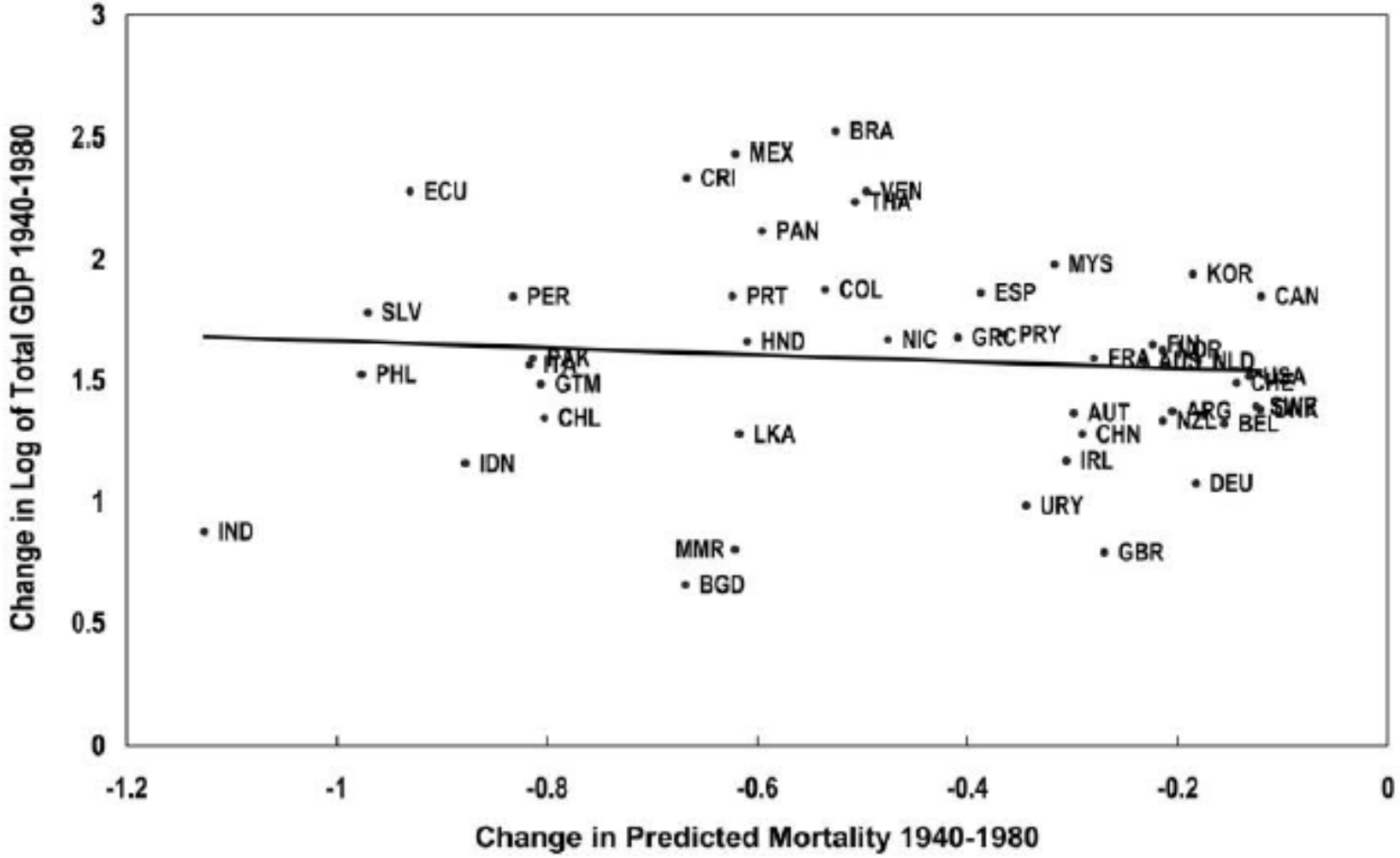


FIG. 8.—Change in log of total GDP and change in predicted mortality, 1940–80, base sample.

TABLE 9

EFFECT OF LIFE EXPECTANCY ON GDP, PER CAPITA GDP, AND GDP PER WORKING AGE POPULATION: 2SLS ESTIMATES

	BASELINE PREDICTED MORTALITY INSTRUMENT					
	Base Sample: Just 1940 and 1980 (1)	Base Sample: Just 1940 and 2000 (2)	Low- and Middle-Income Countries Only: Just 1940 and 1980 (3)	Low- and Middle-Income Countries Only: Just 1940 and 2000 (4)	Base Sample: Interaction with Institutions: Just 1940 and 1980 (5)	Base Sample: Interaction with Initial (1930) Value of Dependent Variable: Just 1940 and 1980 (6)
	A. Dependent Variable: Log GDP					
Log life expectancy	.32	.42	−.39	−.58	−.11	−.069
	(.84)	(.52)	(1.44)	(1.09)	(.99)	(.73)
Postyear dummy × institutions or initial log GDP					−.063	−.109
					(.055)	(.059)
Number of countries	47	47	36	36	47	47
	B. Dependent Variable: Log per Capita GDP					
Log life expectancy	−1.32	−1.51	−2.35	−2.70	−1.64	−1.59
	(.56)	(.57)	(1.13)	(1.40)	(.77)	(1.22)
Postyear dummy × institutions or initial log per capita GDP					−.049	−.073
					(.060)	(.278)
Number of countries	47	47	36	36	47	47

(3) Acemoglu and Johnson (2007)

- Some issues:

  (1) The main analysis excludes Africa

  (2) How reliable is the data on by-cause mortality from 1940? There is a long data appendix listing sources and assumptions, but fundamentally how good are League of Nations health reports from the late 1930s, or WHO reports from the 1940s/1950s, when much of rural Asia, Africa, and Latin America lacked health clinics then?

(3) Acemoglu and Johnson (2007)

- Some issues:

  (1) The main analysis excludes Africa

  (2) How reliable is the data on by-cause mortality from 1940? There is a long data appendix listing sources and assumptions, but fundamentally how good are League of Nations health reports from the late 1930s, or WHO reports from the 1940s/1950s, when much of rural Asia, Africa, and Latin America lacked health clinics then?

  (3) What does poor health in 1940 proxy for? They show results are robust to including 1940 income levels and institutions as controls. Is this enough? (Mismeasured?)

  (4) Stepping back, if these investments saved millions of lives, aren't they well worth investing in even without a positive effect on income per capita?

(3) Acemoglu and Johnson (2007)

- Placing this paper in the growth literature: this result is and flip-side of Young's (2005) paper on the growth impacts of HIV/AIDS in Sub-Saharan Africa (i.e., that drops in population due to the epidemic will raise the capital to labor ratio and wages/per capita income)

(3) Acemoglu and Johnson (2007)

- Hoyt Bleakley discussion (for a 2006 NBER meeting):

  (1) Morbidity vs. mortality. Many health investments reduce morbidity but not mortality so the negative population mechanism does not apply

  E.g., Bleakley 2007 *QJE* on hookworm in the US: investments in hookworm eradication boost schooling attainment, and income decades later

  -- Bleakley 2006 on malaria eradication in Colombia: regions with the high morbidity (but low mortality) *P. Vivax* strain show human capital and income gains, regions with the fatal *P. Falciparum* strain do not

(3) Acemoglu and Johnson (2007)

- Hoyt Bleakley discussion (for a 2006 NBER meeting):

  (1) Morbidity vs. mortality. Many health investments reduce morbidity but not mortality so the negative population mechanism does not apply

  (2) Mortality reductions affect children most, so interventions may take decades to be fully reflected in income. Consider a 1950s drop in infant mortality. By 1980, only a small share of working adults will have been affected.

(3) Acemoglu and Johnson (2007)

- Hoyt Bleakley discussion (for a 2006 NBER meeting):

  (1) Morbidity vs. mortality. Many health investments reduce morbidity but not mortality so the negative population mechanism does not apply

  (2) Mortality reductions affect children most, so interventions may take decades to be fully reflected in income. Consider a 1950s drop in infant mortality. By 1980, only a small share of working adults will have been affected.

  (3) Land and capital may have been relatively fixed in the 1950s, but not today: global capital flows, rapid urbanization, new agricultural technologies, cheap birth control. Thus the adverse income per capita effects of longer life expectancy could be smaller today


CANADA
U.S.A.
RUSSIA
KAZAKSTAN
MONGOLIA
CHINA
INDIA
BRAZIL
AUSTRALIA
INDONESIA
ALGERIA
LIBYA
EGYPT
SUDAN
NIGER
MALI
SAUDI ARABIA
IRAN
TURKEY
ETHIOPIA
SOMALIA
ANGOLA
ZAMBIA
NAMIBIA
SOUTH AFRICA
COLOMBIA
BOLIVIA
PARAGUAY
URUGUAY
ARGENTINA
PACIFIC OCEAN
ATLANTIC OCEAN
INDIAN OCEAN
SOUTHERN OCEAN
ANTARCTICA
The Hobo-Dyer Equal Area Projection
Cities

Next week

- For next week's lecture, please focus on the "Worms" follow-up articles, Kremer and Miguel (2007) and Baird et al. (2015).

- The third referee report is due next week (March 9th), on the Morjaria article.