

Economics 270c
Graduate Development Economics

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Graduate Development Economics

Lecture 12 – April 14, 2009

Macroeconomic growth empirics

Lecture 1: Global patterns of economic growth and development (1/20)

Lecture 2: Inequality and growth (1/27)

The political economy of development

Lecture 3: History and institutions (2/3)

Lecture 4: Corruption (2/10)

Lecture 5: Patronage politics (2/17)

Lecture 6: Democracy and development (2/24)

Lecture 7: War and Economic Development (3/3)

Lecture 8: Economic Theories of Conflict (3/10) – Guest lecture by Gerard Padro

Human resources

Lecture 9: Human capital and income growth (3/17)

Lecture 10: Increasing human capital (3/31)

Lecture 11: Labor markets and migration (4/7)

Lecture 12: Health and nutrition (4/14)

Lecture 13: The demand for health (4/21)

Other topics

Lecture 14: Environment and development (4/28)

Lecture 15: Resource allocation and firm productivity (5/5)

Additional topics for the development economics field exam

-- Ethnic and social divisions

-- The Economics of HIV/AIDS

- Prerequisites: Graduate microeconomics, econometrics
- Grading:
Four referee reports – 40%

Two problem sets – 20%

→ Problem set 2 posted today, due Tuesday April 21

Research proposal – 30%

Class participation – 10%

Lecture 12 outline

- (1) Health, education and economic development
- (2) Acemoglu and Johnson (2007) on life expectancy and income growth across countries
- (3) Using randomized evaluations to estimate causal relationships
- (4) Miguel and Kremer (2004) on deworming in children

(1) Some leading questions

- An observation: health and wealth are correlated both across countries and across people within societies. Why?
- Question #1: What impact of income on health and nutrition? 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? Banerjee et al (2004) on Rajasthan - 50% absenteeism of rural clinic workers

(1) A simple model of health and education

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

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

- 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

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- H_t is child health at t , El_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) 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

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- If one had accurate data on all the variables in equation (1) 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 kids.

(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

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-- 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

(2) 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 the earlier Acemoglu, Johnson and Robinson 2001 piece, to rule out direct tropical disease effects on development

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-- An important piece of support for the earlier Acemoglu, Johnson and Robinson 2001 piece, 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)

(2) 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 in the medium-term (capital), then per capita income falls when population increases
-- Apparently any gains in productivity (from greater human capital or TFP) were outweighed by this effect

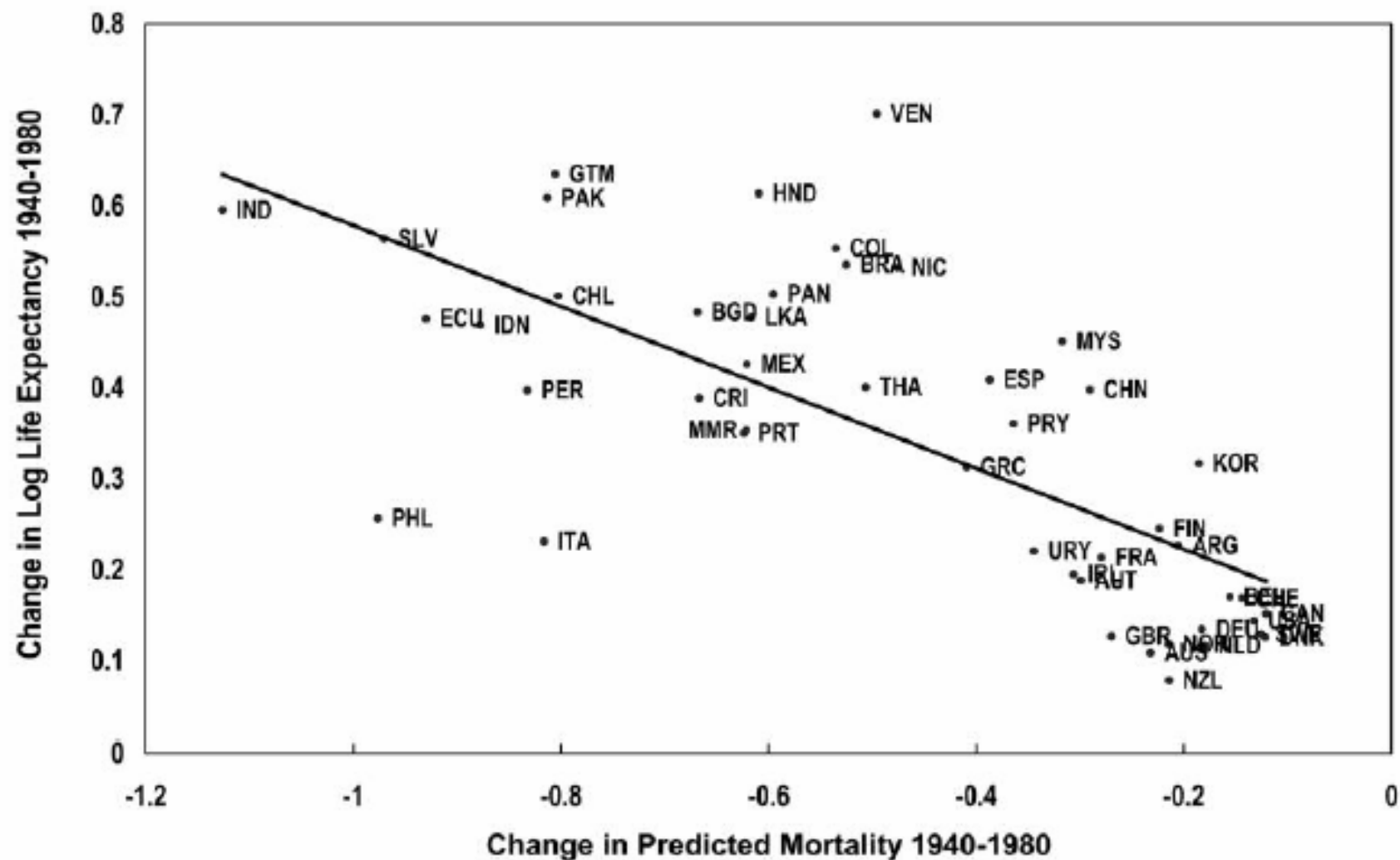


FIG. 3.—Change in log life expectancy and change in predicted mortality, 1940–80, base sample.

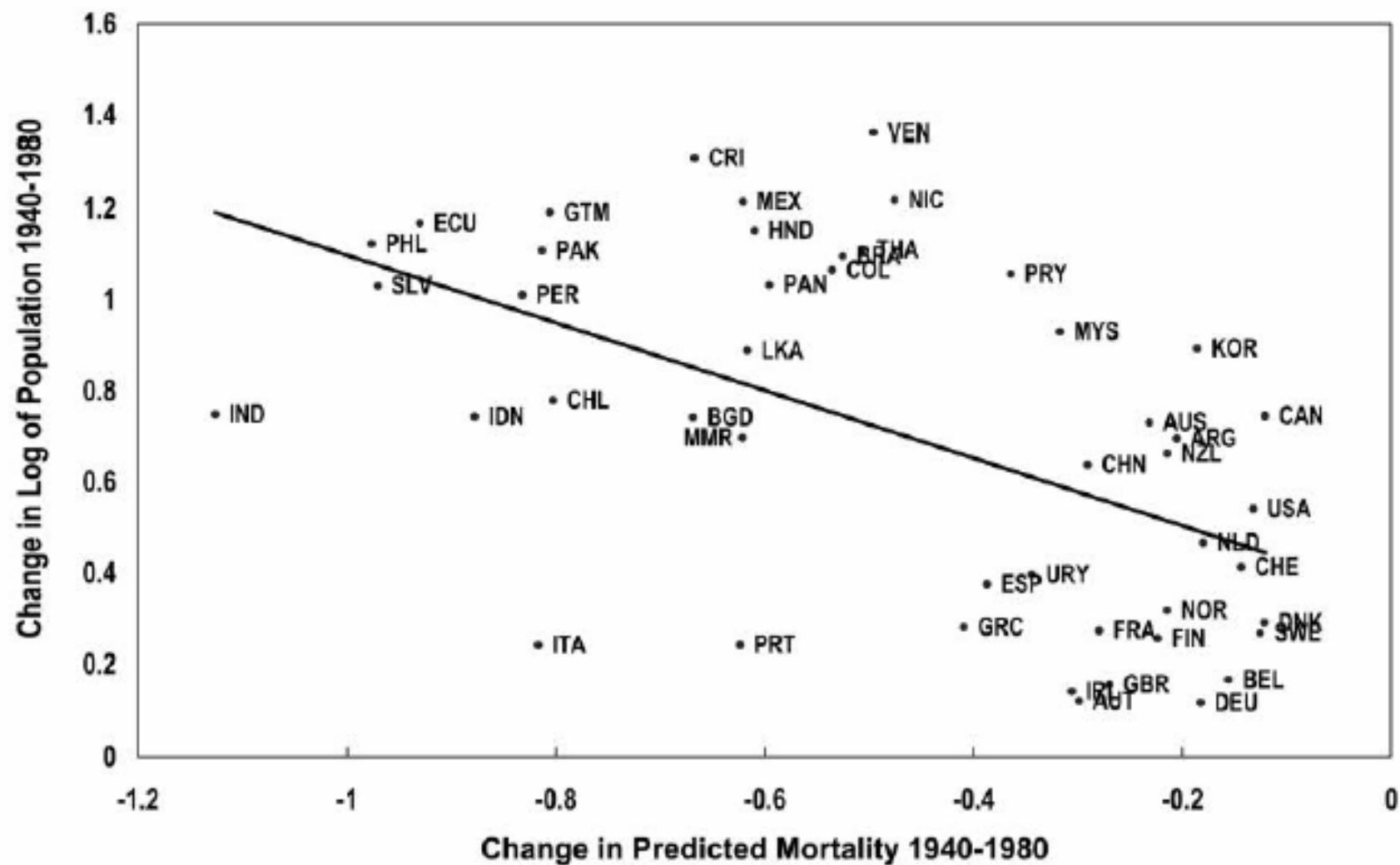


FIG. 7.—Change in log of population and change in predicted mortality, 1940–80, base sample.

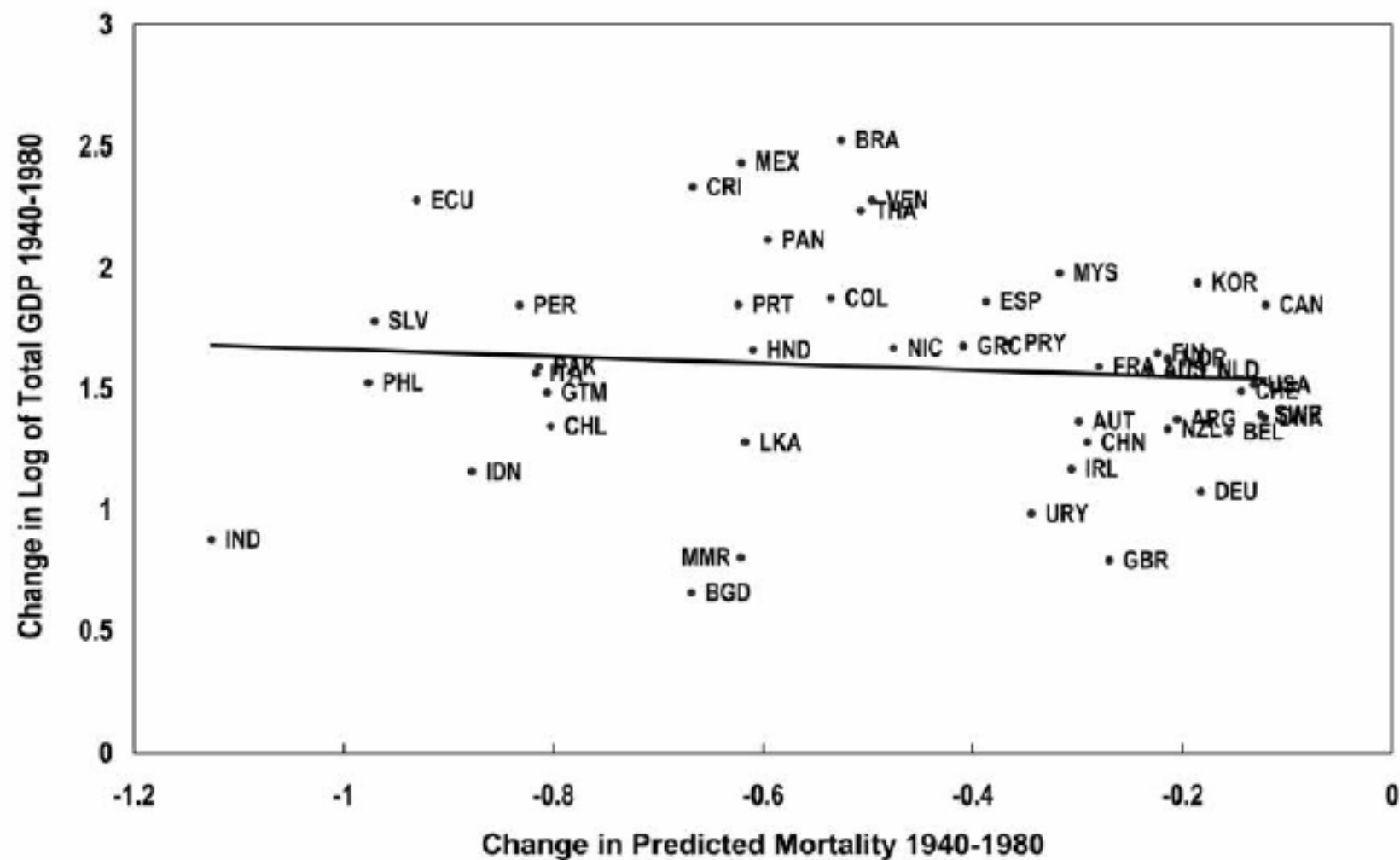


FIG. 8.—Change in log of total GDP and change in predicted mortality, 1940–80, base sample.

(2) 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?

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 - (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?
 - (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?

(2) 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

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- 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

(3) 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

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- 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

(3) 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) $T_2 = T(H_1, H_2, El_1, El_2, \alpha, SC, YS)$
- 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 El_2 and “medicine” M_2

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

(3) Randomized evaluation methods

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 - (1) Randomization helps address an array of well-known biases, e.g., it can resolve the selection problem that often plagues treatment effect estimates
 - (2) As a result, randomized research designs can allow the researchers to identify behavioral parameters that are of theoretical interest, and that are difficult or impossible to estimate using other methods (e.g., estimating social effects)
 - (3) The results of randomized evaluations are typically transparent and highly credible to policymakers

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 - (1) External validity – estimated impacts are “local”
But true for all micro-empirical work (e.g., ICRISAT)
 - (2) They cannot address all problems (e.g., in macro)
That is setting the bar too high for any method

(3) Randomized evaluation methods

- What are the limitations of randomized methods?
 - (1) External validity – estimated impacts are “local”
 - (2) They cannot address all problems (e.g., in macro)
 - (3) They are “too easy”, anyone can use them
 - (4) These methods are inherently atheoretical
 - (5) They cannot estimate general equilibrium effects

(3) Randomized evaluation methods

- What are the limitations of randomized methods?

(1) External validity – estimated impacts are “local”

(2) They cannot address all problems (e.g., in macro)

(3) They are “too easy”, anyone can use them

This is arguably a strength rather than a weakness

(4) These methods are inherently atheoretical

Not true: many recent papers use these methods to tackle fundamental theory issues (e.g., Karlan and Zinman 2006 on moral hazard and adverse selection in credit markets, Todd and Wolpin 2005 on schooling)

(5) They cannot estimate general equilibrium effects

Large-scale experiments properly designed (cluster randomizations) can estimate spillovers, price effects

(3) Broader critiques

- The trend towards empirical work in development economics has been criticized by some senior leaders in the field (i.e., the Economic and Political Weekly “debate” in 2005 pitting Bardhan, Basu and Mookherjee vs. Banerjee; Deaton 2008 vs. Banerjee/Duflo and Imbens)
- Development is part of a broader intellectual trend towards applied micro empirical work throughout economics (e.g., labor economics) as micro-data has improved, computing power has become cheaper, and better applied econometric tools have been developed

(4) Miguel and Kremer (2004)

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(2) Positive treatment externalities

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

(2) Positive treatment externalities

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

(4) Credit constraints prevent good investments

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)

(4) 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

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- 75 primary schools, over 30,000 children (aged 6-18)
- Deworming treatment (drugs, health education) phased in randomly across three treatment groups. These groups are similar along observables

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 - Listed school alphabetically (by zone), 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
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- Group 3: no treatment in 1998 or 1999

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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
<i>Panel A: Pre-school to Grade 8</i>					
Male	0.53	0.51	0.52	0.01 (0.02)	–0.01 (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** (0.2)	0.8*** (0.2)
<i>Panel B: Grades 3 to 8</i>					
Attendance recorded in school registers (during the four weeks prior to the pupil survey)	0.973	0.963	0.969	0.003 (0.004)	–0.006 (0.004)
Access to latrine at home	0.82	0.81	0.82	0.00 (0.03)	–0.01 (0.03)
Have livestock (cows, goats, pigs, sheep) at home	0.66	0.67	0.66	–0.00 (0.03)	0.01 (0.03)
Weight-for-age Z-score (low scores denote undernutrition)	–1.39	–1.40	–1.44	0.05 (0.05)	0.04 (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

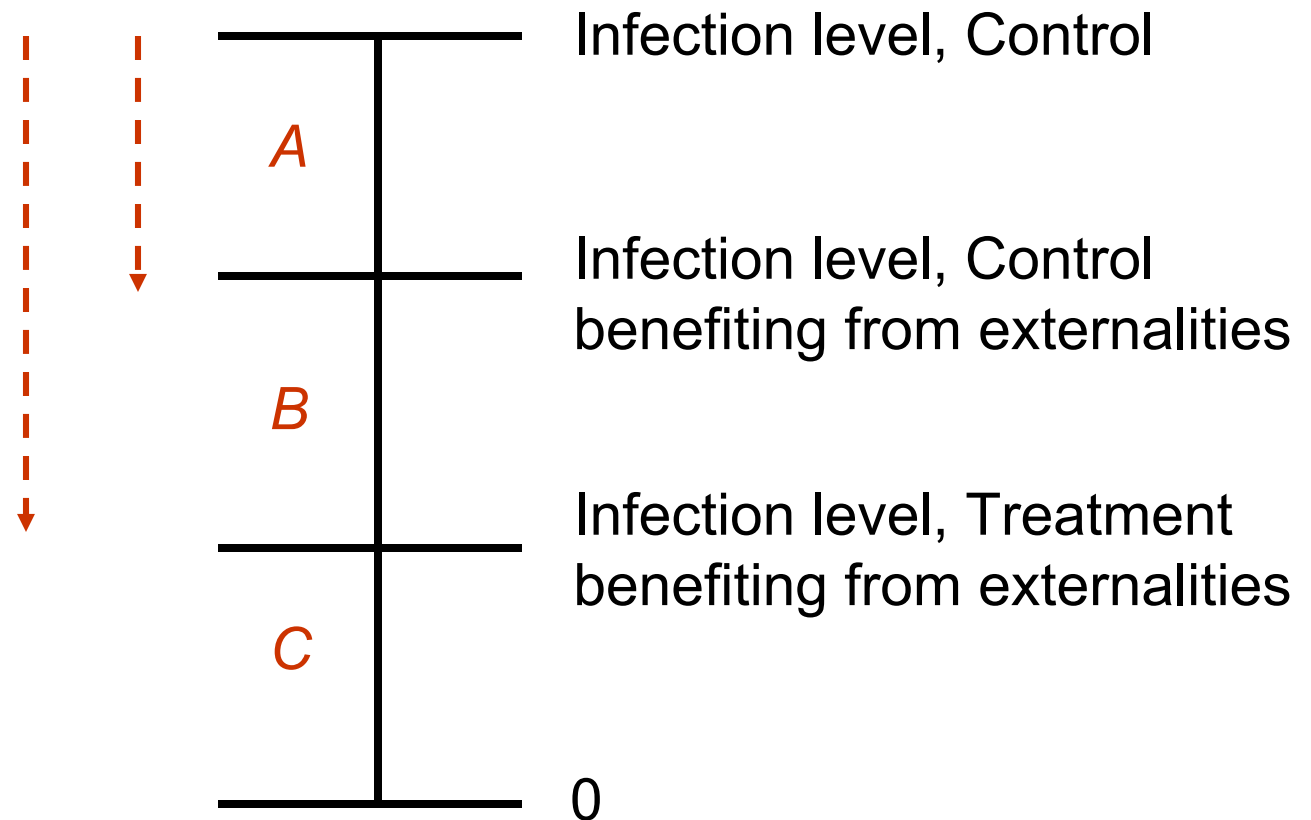
	Group 1		Group 2		Group 3	
	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years	Girls <13 years, and all boys	Girls ≥ 13 years
	<i>Treatment</i>		<i>Comparison</i>		<i>Comparison</i>	
Any medical treatment in 1998 (For grades 1–8 in early 1998)	0.78	0.19	0	0	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

(4) Miguel and Kremer (2004)

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

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- Externalities would lead us to doubly under-estimate treatment effects: (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 no significant impact



Naïve treatment effect is B .

Actual treatment effect is $A + B > B$

(4) Miguel and Kremer (2004)

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

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

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
<i>Panel A: Helminth Infection Rates</i>			
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)
<i>Panel B: Other Nutritional and Health Outcomes</i>			
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)
Hemoglobin concentration (g/L), 1999	124.8	123.2	1.6 (1.4)
Proportion anemic (Hb < 100g/L), 1999	0.02	0.04	–0.02** (0.01)
<i>Panel C: Worm Prevention Behaviors</i>			
Clean (observed by field worker), 1999	0.59	0.60	–0.01 (0.02)
Wears shoes (observed by field worker), 1999	0.24	0.26	–0.02 (0.03)
Days contact with fresh water in past week (self-reported), 1999	2.4	2.2	0.2 (0.3)

(4) 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 ideal program impact estimator, taking into account treatment externalities:
- $E(Y_{ij} \mid T_{1i} = 1, N^T = N^{AVG}) - E(Y_{ij} \mid T_{1i} = 0, N^T = 0)$,

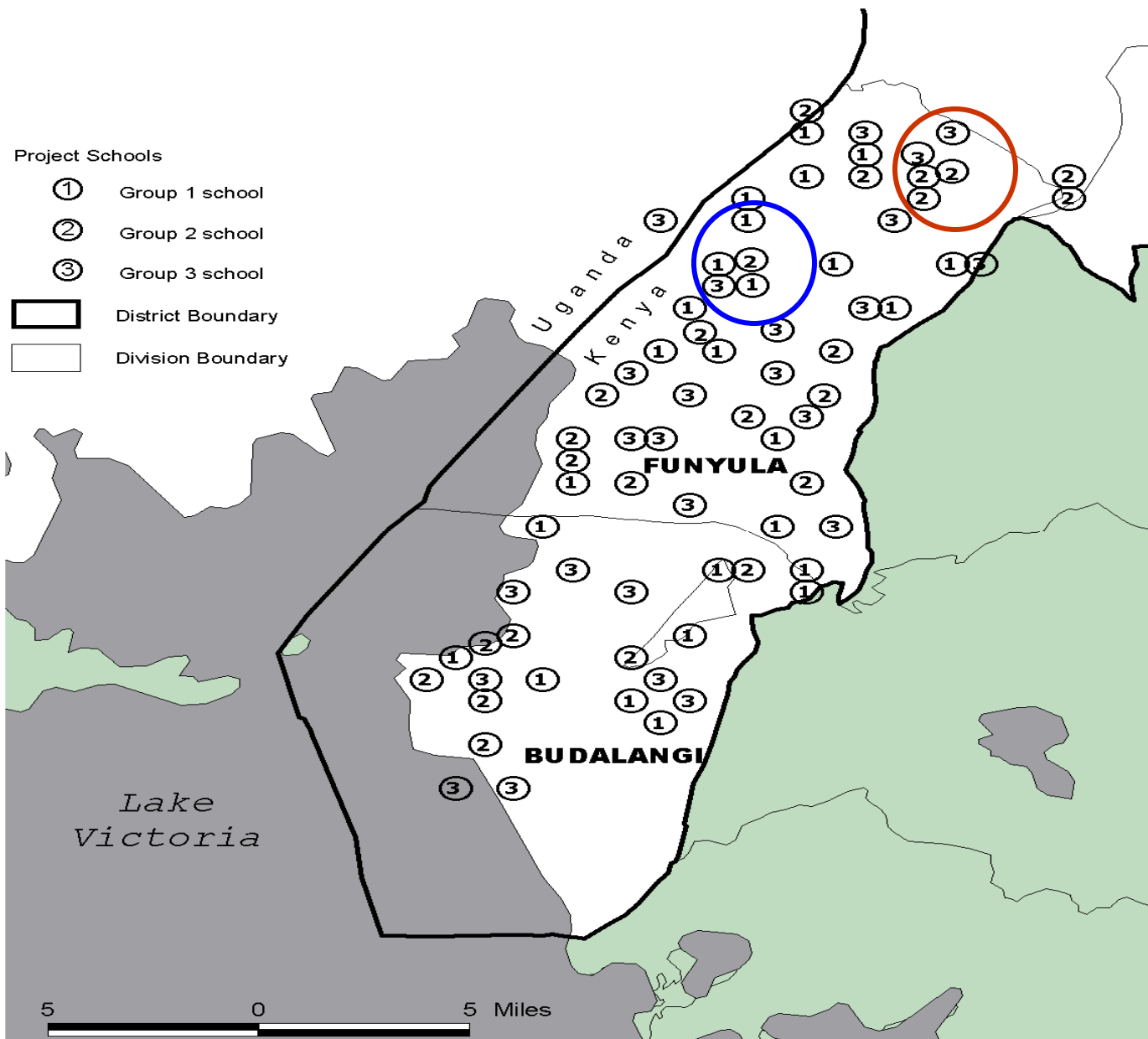
which is equivalent to

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

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

$$(1) \quad Y_{ijt} = a + \beta_1 \cdot T_{1it} + \beta_2 \cdot T_{2it} + X'_{ijt} \delta + \sum_d (\gamma_d \cdot N_{dit}^T) + \sum_d (\phi_d \cdot N_{dit}) \\ + u_i + e_{ijt}.$$

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_{dit}^T 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.



$$\begin{aligned}
 (3) \quad Y_{ijt} = & a + \beta_1 \cdot T_{1it} + b_1 \cdot D_{1ij} + b_2 \cdot (T_{1it} * D_{1ij}) + X'_{ijt} \delta \\
 & + \sum_d (\gamma_d \cdot N_{dit}^T) + \sum_d (\phi_d \cdot N_{dit}) + u_i + e_{ijt}.
 \end{aligned}$$

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)
<i>Panel A: Selection into Treatment</i>						
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 (0.04)	–0.06 (0.05)
Grade progression (= Grade – (Age – 6)), 1998	–2.0	–1.8	–1.8	–1.8	–0.2 ^{**} (0.1)	–0.0 (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 (0.04)	–0.01 (0.06)
Clean (observed by field worker), 1998	0.53	0.59	0.60	0.66	–0.07 (0.05)	–0.07 (0.10)
<i>Panel B: Health Outcomes</i>						
<i>Girls <13 years, and all boys</i>						
Any moderate-heavy infection, 1999	0.24	0.34	0.51	0.55	–0.27 ^{***} (0.06)	–0.21 ^{**} (0.10)
Hookworm moderate-heavy infection, 1999	0.04	0.11	0.22	0.20	–0.19 ^{***} (0.03)	–0.09 [*] (0.05)
Roundworm moderate-heavy infection, 1999	0.08	0.12	0.22	0.30	–0.14 ^{***} (0.04)	–0.18 ^{**} (0.07)
Schistosomiasis moderate-heavy infection, 1999	0.09	0.08	0.20	0.13	–0.11 [*] (0.06)	–0.05 (0.06)
Whipworm moderate-heavy infection, 1999	0.12	0.16	0.16	0.20	–0.04 (0.16)	–0.05 (0.09)

Cross-school infection externalities (1999)

- Large reductions in moderate-heavy infection levels within 3 km (2 miles) of treatment schools in 1999, smaller positive reductions up to 6 km
- An average reduction in moderate-heavy infections of approximately 20 percentage points in the study area can be attributed to cross-school externalities

TABLE VII

DEWORMING HEALTH EXTERNALITIES WITHIN AND ACROSS SCHOOLS, JANUARY TO MARCH 1999^a

	Any moderate-heavy helminth infection, 1999			Moderate-heavy schistosomiasis infection, 1999			Moderate-heavy geohelminth infection, 1999		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Indicator for Group 1 (1998 Treatment) School	-0.25*** (0.05)	-0.12* (0.07)	-0.09 (0.11)	-0.03 (0.03)	-0.02 (0.04)	-0.07 (0.06)	-0.20*** (0.04)	-0.11** (0.05)	-0.03 (0.09)
Group 1 pupils within 3 km (per 1000 pupils)	-0.26*** (0.09)	-0.26*** (0.09)	-0.11 (0.13)	-0.12*** (0.04)	-0.12*** (0.04)	-0.11** (0.05)	-0.12* (0.06)	-0.12* (0.07)	-0.01 (0.07)
Group 1 pupils within 3–6 km (per 1000 pupils)	-0.14** (0.06)	-0.13** (0.06)	-0.07 (0.14)	-0.18*** (0.03)	-0.18*** (0.03)	-0.27*** (0.06)	0.04 (0.06)	0.04 (0.06)	0.16 (0.10)
Total pupils within 3 km (per 1000 pupils)	0.11*** (0.04)	0.11*** (0.04)	0.10** (0.04)	0.11*** (0.02)	0.11*** (0.02)	0.13*** (0.02)	0.03 (0.03)	0.04 (0.03)	0.02 (0.03)
Total pupils within 3–6 km (per 1000 pupils)	0.13** (0.06)	0.13** (0.06)	0.12* (0.07)	0.12*** (0.03)	0.12*** (0.03)	0.16*** (0.03)	0.04 (0.04)	0.04 (0.04)	0.01 (0.04)
Received first year of deworming treatment, when offered (1998 for Group 1, 1999 for Group 2)		-0.06* (0.03)			0.03** (0.02)			-0.04** (0.02)	
(Group 1 Indicator) * Received treatment, when offered		-0.14* (0.07)			-0.02 (0.04)			-0.10*** (0.04)	
(Group 1 Indicator) * Group 1 pupils within 3 km (per 1000 pupils)			-0.25* (0.14)			-0.04 (0.07)			-0.18** (0.08)
(Group 1 Indicator) * Group 1 pupils within 3–6 km (per 1000 pupils)			-0.09 (0.13)			0.11 (0.07)			-0.15 (0.10)
Grade indicators, school assistance controls, district exam score control	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations	2328	2328	2328	2328	2328	2328	2328	2328	2328
Mean of dependent variable	0.41	0.41	0.41	0.16	0.16	0.16	0.32	0.32	0.32

(4) Miguel and Kremer (2004)

- Educational outcomes: school absenteeism (both from poor attendance and drop outs) fall by roughly 7 percentage points, or one quarter
 - One of the most cost-effective ways to boost school participation found 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)
 - But 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 IX
SCHOOL PARTICIPATION, DIRECT EFFECTS AND EXTERNALITIES^a
DEPENDENT VARIABLE: AVERAGE INDIVIDUAL SCHOOL PARTICIPATION, BY YEAR

	OLS (1)	OLS (2)	OLS (3)	OLS (4) May 98– March 99	OLS (5) May 98– March 99	OLS (6) May 98– March 99	IV-2SLS (7) May 98– March 99
Moderate-heavy infection, early 1999						–0.028*** (0.010)	–0.203* (0.094)
Treatment school (T)	0.051*** (0.022)						
First year as treatment school (T1)		0.062*** (0.015)	0.060*** (0.015)	0.062* (0.022)	0.056*** (0.020)		
Second year as treatment school (T2)		0.040* (0.021)	0.034* (0.021)				
Treatment school pupils within 3 km (per 1000 pupils)			0.044** (0.022)		0.023 (0.036)		
Treatment school pupils within 3–6 km (per 1000 pupils)			–0.014 (0.015)		–0.041 (0.027)		
Total pupils within 3 km (per 1000 pupils)			–0.033** (0.013)		–0.035* (0.019)	0.018 (0.021)	0.021 (0.019)
Total pupils within 3–6 km (per 1000 pupils)			–0.010 (0.012)		0.022 (0.027)	–0.010 (0.012)	–0.021 (0.015)
Indicator received first year of deworming treatment, when offered (1998 for Group 1, 1999 for Group 2)					0.100*** (0.014)		
(First year as treatment school Indicator) * (Received treatment, when offered)					–0.012 (0.020)		
1996 district exam score, school average	0.063*** (0.021)	0.071*** (0.020)	0.063*** (0.020)	0.058 (0.032)	0.091** (0.038)	0.021 (0.026)	0.003 (0.023)

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 (0.046)	−0.030 (0.049)
Second year as treatment school (T2)		0.001 (0.073)	0.009 (0.081)
1996 District exam score, school average	0.74 ^{***} (0.07)	0.71 ^{***} (0.07)	0.75 ^{***} (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

Cost-benefit calculations

- Deworming as a human capital investment:
Health gains → More schooling → Higher adult wages
 - Deworming led to 7% gain in school participation
 - Previous study: each year of school → 7% higher wages
 - Take these gains in wages (7% x 7%) over 40 years in the workforce, discounted at 5% per year
- Deworming benefits are at least three times (3x) as large as treatment costs (using the Tanzania costs)

Whiteboard #1

Whiteboard #2

Whiteboard #3

Whiteboard #4

Whiteboard #5

