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Mines: The local wealth and health effects of mineral mining in developing countries☆

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ABSTRACT

We assess health and wealth impacts of mineral mining using micro-data from about 800 mines in 44 developing countries. Gains in asset wealth (0.3 standard deviations) coexist with a higher incidence of health conditions linked to heavy metal toxicity: anemia among women (ten percentage points), and stunting in young children (five percentage points). Consistent results emerge from a range of distinct identification strategies. Two difference-in-difference tests exploit information on mineral and pollutant characteristics to show that the observed health effects are due to pollution: impacts arise only near mines where metal contamination is to be expected, and the recovery of blood hemoglobin levels in women after childbirth shows a characteristic signature of lead toxicity. Our results add to the nascent literature on health-wealth tradeoffs near industrial operations in developing countries.

1. Introduction

This paper studies the local wealth and health impacts of mineral mining in developing countries. Using micro-level data from 44 developing countries, we ask whether, in order to enjoy greater wealth and amenities, residents in mining communities incur a price in terms of a specific health burden.

The decision to live near centers of industrial activity involves weighing the promise of economic opportunity against the risks posed by pollution. The resulting net impact on health is theoretically ambiguous: pollution may adversely affect health, but higher incomes may enable households to make investments in health care that could compensate. Nowhere is the choice starker than in developing countries. More often than not, opportunities for making a good living are few. At the same time, pollution tends to be poorly regulated, and information on health risks, scarce. Poor infrastructure and inflexible housing markets commonly make it impracticable to avoid pollution. Yet, while the literature on the health effects of pollution have advanced greatly in recent decades, "almost all of this research has been conducted in developed country settings" (Greenstone and Jack, 2015, p. 17). The mining and mineral processing industry is an attractive test case for the study of pollution in that it poses particularly sharp trade-offs. Single plants generate very high value – up to billions of dollars per year. At the same time, they are large polluters, and precisely because they are important sources of revenue, foreign exchange, and employment, there is a risk of weak environmental regulation and enforcement (Greenstone and Hanna, 2014). In consequence, mining projects habitually evoke strong passions among the communities affected (Bebbington et al., 2008; Nash, 1993). Some projects have been supported vociferously, and people have fought over the right to work in mines. Yet, elsewhere, mining has been desperately opposed, as citizens feared damage to their health and environment. This study shows that these political passions

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are grounded in a real trade-off. Across a broad range of settings, the local benefits of mining are real, but so are the costs.

We analyze the effect of mining activity on asset wealth, on general health, and on two specific health outcomes known to be linked to pollutants that may be present around mines in our sample, namely anemia in adults and children, and growth in young children. To study these effects across the developing world, we compile 104 waves of geo-coded Demographic and Health Surveys from 44 countries, spanning a time period from 1986 until 2012. We spatially overlay this data with the location of mining and smelting operations across the world, using data provided by Unites States Geological Survey (USGS), as well as two business intelligence datasets.

To study the effects of exposure to a mining environment, we then construct a range of statistical tests that rely on different control groups. In our preferred specification, we estimate the impact of exposure to mining activity in a pseudo-panel that compares the treatment group of households living close to the mine and the control group of households living farther afield across time, between years when the mine was operational, and when it was dormant.[1] Guided by prior evidence from the environmental sciences on the spatial extent of pollution, we define households within 5 km of a mine to be treated. The estimated spatial pattern of wealth impacts suggests that this is a conservative cut-off to detect health impacts. A mine-level panel compares households observed near the same mine in different years; a mother-level panel compares among siblings born in different years. The panels allow for common effects shared by all households observed in the same country and survey round. We also document cross-sectional evidence from the broader sample of mines for which production information is not available. To assess concerns about residential sorting,[2] we estimate the same specifications only with the sample of households who never moved.[3] To address possible endogeneity in production decisions, we check for robustness to instrumenting with mineral deposit locations for mine location, and with world mineral prices for operational status.

In addition, we develop two difference-in-differences tests that are tailored to prior knowledge on the toxic properties of mining pollution. Our purpose in designing these tests is two-fold. Firstly, they help bolster our claims to observing a causal impact of mining on health. In particular, they provide greater confidence against interference of residential sorting. Secondly, our tests provide evidence to suggest that the observed health effects are due to pollution, not other mechanisms. To devise the tests, we (i) leverage knowledge on the association of specific mine types with lead contamination – and by extension, health impacts specific to lead – to conduct falsification tests. We show both that we only observe those health impacts that are expected from exposure to lead pollution, and that we only observe them near mine types strongly associated with the release of lead. Furthermore, we (ii) exploit detailed information on the birth history of women to describe a pattern of impaired ability to recover from blood loss after pregnancy among women living in mining communities, as compared to those living in the general vicinity. This effect is consistent with a known pathophysiological pattern of lead toxicity in adults, but not with other mechanisms.

Our results show that, at the global mean, long-run asset wealth in mining communities rises by about 0.1 standard deviations of an asset index computed for the country where the community is located and the year in which the survey was taken. The medium-term wealth of households living in the vicinity of an operating mine rises by about 0.3 standard deviations. We illustrate that these are considerable effects, given the high variation in asset ownership within survey rounds. Wealth effects are strongly concentrated in the direct vicinity of the mine; there are benefits across the wealth distribution, although in the long run, the wealthiest households benefit the most.

We find evidence of two health impacts that are known consequences of exposure to lead and other heavy metals. Thus, women in mining communities show depressed blood hemoglobin, and increases in the incidence of anemia of three to ten percentage points. They also recover more slowly from blood loss during pregnancy and delivery, a pattern consistent with prior toxicological research. Children in mining communities suffer some important adverse growth outcomes from *in utero* exposure, with a five percentage point increase in the incidence of stunting – although there is very little evidence of lower birth weight. Growth impacts are weaker among older children, perhaps because of the greater wealth enjoyed by households in mining communities. We note that, while our data contains no good measure of cognitive ability, lead exposure has previously been shown to cause cognitive deficits in children at exposure levels below those associated with growth retardation, and far below those associated with overt anemia (Aizer et al., 2018; Ferrie et al., 2012). By way of contrast to these specific health impacts, we find no effects on health outcomes that are not linked to heavy metal pollution, nor are mining communities differentially affected by other known causes of anemia, or under-served by health care. Because our paper shows reduced form impacts, our health results should be interpreted as the *compensated* impact of mining. By implication, since living in mining communities goes hand in hand with economic benefits across the distribution, there is no indication that ill health is caused by deprivation. Rather, health impacts arise despite wealth gains.

This paper seeks to make two contributions. First, it shows a trade-off between real economic benefits and specific health costs that has not been reported before. Prior work has either considered economic or health effects in isolation, or shown consistently adverse or beneficial effects across both dimensions. In pioneering work, Aragón and Rud (2013) leverage a change in local hiring and procurement policies to report on economic gains and short term beneficial health effects near a large gold mine in Peru.[4] In another paper, Aragón and Rud (2016) find stark decreases in agricultural productivity, accompanied by dire welfare consequences for rural households, and evidence of some adverse health effect near twelve gold mining operations in Ghana. Wilson (2012) shows that asset ownership increased among residents of copper mining communities in Zambia during a boom in the 2000s, alongside a decrease in sexual risk-taking. Corno and De Walque (2012) report increased risk taking and HIV infection in mining communities in southern Africa among migrant miners, but not non-migrants. Kotsadam and Tolonen (2016) argue that mining activity in a comprehensive sample of African mines fosters sectoral shifts in employment out of agriculture and increases cash employment among women, but is also associated with women leaving the labor force altogether.[5] In recent papers, Benshaul-Tolonen, 2018 finds that gold mining expansion in Africa has reduced infant mortality, while Romero and Saavedra (2015) document evidence from Colombia that contamination from gold mines adversely affects cognitive ability and increases stunting in newborns, but only when they live downstream of the mine.

Secondly, we add to the limited evidence on the consequences of industrial pollution in developing countries. Most large and well-identified studies of the issue are set in developed countries (e.g.

[1] Mining and processing facilities are often located close to each other, including among facilities in our data. We therefore prefer pooling both types of facilities, but show robustness to excluding smelters from the sample.

[2] That is, the possibility that households with different potential health and wealth outcomes may be selectively moving in or out of a mining area.

[3] Households are categorized as 'never-mover' if they have always been living in their current location, as per their response recorded in the DHS data.

[4] While the empirical study of the local economic and health impacts of mining is recent, the importance of mining to development is reflected in a long tradition of research on the macroeconomic implications of mining and the optimal management of mineral resources. For a textbook-level overview survey of the former, see Frankel (2010); for a textbook-level overview of the latter, e.g., Hartwick et al. (1986).

[5] Other work has leveraged resource revenue at a disaggregated scale to study of other objects of interest (Acemoglu et al., 2013; Dell, 2010; Dube and Vargas, 2013; Monteiro and Ferraz, 2009).

Table 1
Mine types, associated pollutants, and health effects.

Mine type	Pollutants of concern	Health effects
Polymetallic mines	Heavy metals, especially lead	Neurodevelopmental damage, anemia, growth deficits (from Lead)
Small-scale gold and silver mining	Mercury	Renal disease, neurological conditions
Large-scale gold mining	Cyanide	Heart irregularities, thyroid problems
Bulk metal mines, gemstone mines	Particulates	Respiratory disease, GI problems associated with turbid water
Phosphate rock	Radionuclides	Lung cancer and non-malignant respiratory disease
Coal	Particulates, radionuclides	Respiratory disease, GI problems, lung cancer
Metal smelters	*Heavy metals, SO2*	*As shown for polymetallic mines, and respiratory disease*

Notes. Not all mine types are mutually exclusive. Mapping based on ATSDR Toxicological Profiles for the respective pollutants, Alloway (2013), Ripley and Redmann (1995), and Wright and Welbourn (2002). Health effects as reported from chronic low-level environmental exposure.

Table 2
Prior literature on blood lead levels in communities near smelters.

Study	Distance to smelter	Mean PbB
Fontúrbel et al. (2011)	0.5–1.8 km	n/a
Roels et al. (1980)	1–2.5 km	13–30 g/dL
Recio-Vega et al. (2012)	2 km	14–19 g/dL
Factor-Litvak et al. (1999)	2–4 km	28–39 g/dL
Benin et al. (1999)	3 km	20–40 g/dL
Landrigan and Baker (1981)	4 km	≥40 g/dL *in 87% of subjects*

Notes. The table summarizes prior studies of lead levels in communities near smelters. It shows the maximum distance between the smelter and the communities considered highly exposed, alongside mean blood lead in highly exposed communities. Ranges of mean PbB refer to means for population groups that differ in age, gender, and other characteristics. Incidence for Landrigan and Baker summarized by the authors. In the case of Benin et al. (1999), PbB was predicted from observed environmental pollution; in all other studies, PbB was measured directly.

Currie et al., 2013). Studies of developing countries – especially using large samples – remain rare, although the relationship between pollution and health outcomes likely differs pronouncedly among countries at different stages of development (Chen et al., 2013; Ebenstein, 2012; Hanna and Oliva, 2015). Most closely related to our setting is work by Rau et al. (2015), who show cognitive losses from lead exposure near an abandoned toxic waste site in Chile.[6] Further, this study complements evidence in the health sciences by showing that the health effects of mining pollution are salient in the general population near a large number of mines, and are robust to tests that require weak identifying assumptions.[7]

Two limitations of our study are worth noting. First, in our data, we do not observe households who decided to move away from the vicinity of mines. We attempt to address this sorting issue using multiple strategies as far as our data allows us. Secondly, the DHS data jitters geolocations, which attenuates results and limits our ability to study health effects extremely close to mines.

The remainder of the paper is organized as follows. Section 2 introduces results from environmental science and toxicology that guide the way we develop hypotheses, measure impacts, and interpret results. Section 3 discusses data, and Section 4 summarizes econometric methods. Section 5 presents results; Section 6 concludes.

2. Scientific background

2.1. Expected pollutants near mines in our sample

Table 1 provides a stylized summary of pollutants associated with common mine types in our sample, and their health impacts. We leverage this association to show that we find predicted health impacts only near mine types where pollutants specific to the health impact in question are found. Our focus is on 'polymetallic' mines, where any combination of copper, gold, lead, silver, and zinc are extracted. These are linked with a characteristic suite of highly toxic pollutants that includes most prominently lead, but also arsenic, cadmium, and chromium (henceforth, 'heavy metals'[8]). Polymetallic mines make up a large share of mines in our sample (40% of mines in the cross-section, and 70–90% in the panel), and our health data includes key variables required to measure the impact of exposure to lead pollution.

2.2. Spatial extent and intensity of lead contamination

We use distance to the nearest mine as a proxy for exposure to environmental pollution. The choice of a distance cutoff to define the treated group is therefore crucial. Table 2 reviews evidence on the distribution of lead pollution around smelters. High exposure is associated with small distances from the point source of emissions, of 0.5–4 km. In line with this evidence, we define our treatment group tightly, and

[6] Studies of overall urban pollution (Arceo et al., 2016; Greenstone and Hanna, 2014) are related, but not specific to industry, while studies of air pollution from urban traffic (e.g. Gallego et al., 2013) are less closely related.

[7] Evidence to date consists of important and thorough case studies that have shown local treatment effects on (incipient) anemia in communities near smelters. Factor-Litvak et al. (1999) and Roels et al. (1976) use comparisons to a matched control group in addition to dose-response relationships to demonstrate impacts on blood-cell formation (hematopoiesis). Dose-response relationships between blood lead (PbB) and lower blood hemoglobin (Hgb) have been reported from a lead smelter in Idaho, U.S. (Landrigan and Baker, 1981; Schwartz et al., 1990), and between PbB and IQ show near a lead smelter in Port Pirie, Australia (Baghurst et al., 1992). A range of papers by Hendryx and various co-authors (e.g. Hendryx and Ahern, 2008) shows associations between county-level health outcomes and Appalachian coal mining.

[8] The term is imprecise in that it does not refer to a well-defined group of chemical elements – and arsenic is actually a metalloid – but it has the advantage of being in everyday semantics associated with the pollutants in question.

Table 3
Sample size.

	A. Surveys with observations within 20 km of a mine		
Survey rounds	104		
Countries	44		
Interview years	25		
	B. Number of households		
	Full sample	Within 5 km of a mine	Within 5–20 km of a mine
Households	1,192,492	37,608	132,797
% of total		3.2%	11.1%
Children under five years of age	1,364,156	31,964	121,519
Women aged 15 and over	2,877,024	87,234	310,096
Men aged 15 and over	2,717,928	82,973	294,723
	C. Mines and smelters near DHS sampling clusters		
	USGS data	RMD data	Infomine data
DHS cluster within 20 km	838	508	7
DHS cluster within 0–5 km	339	225	4
DHS cluster within 5–20 km	687	455	6
DHS cluster in both distance categories	226	172	3

Notes. Sample size based on all types of mines, smelters and legacies, excluding quarries. Not all variables used in this study are available for the entire sample. The count of locations from Infomine includes only those mines not covered in the RMD data.

include only households within no more than 5 km of a mine.[9] This choice is also in line with the definition of high-exposure zones in van Geen et al. (2012). Further, the spatial distribution of wealth suggests that our treatment definition is conservative in that households within 5 km of the mine experience the clearest wealth increases – some of which might mitigate health impacts (see Section 5.1).

Mean blood lead (PbB) levels among children in the highly exposed communities reported in Table 2 ranged from 13 μg/dL to more than 40 μg/dL. These values far exceed the reference value of 5 μg/dL (the 97.5th percentile of blood lead levels found in the U.S.) set by the Centers for Disease Control to "trigger lead education, environmental investigations, and additional medical monitoring," (Centers for Disease Control and Prevention, 2012) as well as the more dated 'level of concern' of 10 μg/dL (Roper et al., 1991). Dose responses for the health conditions we purport to measure have been previously reported at 10–50 μg/dL (Appendix Table B.1). There is therefore a *prima facie* case to investigate whether the health conditions likely to be affected by mine-specific pollution, are prevalent near mines in our sample.

2.3. Detecting clinical effects of lead and other metal exposure in our data

The toxic properties of lead have long been studied, and are well understood. (See ATSDR (2007) for a comprehensive discussion.) Two key health impacts are observable in our data: reduced blood hemoglobin (Hgb) in adults and children, and growth retardation in children. Regrettably, we do not have a good measure of a third well-established impact, namely impaired cognitive performance and behavioral problems due to neurological damage in children. However, while the health impacts we can observe require high blood lead, "there is no evidence of a threshold for the adverse consequences of lead exposure" for cognitive development (Lanphear et al., 2005, p. 899). Hence, demonstrating overt anemia or growth deficits implies a strong likelihood that the affected individuals – and presumably others with lower PbB – also suffer some cognitive and behavioral impairment. (Appendix Table B.1 summarizes how the health consequences we observe affect the well-being of those exposed, and what their economic cost might be.) For falsification tests, we use all observed health outcomes that have not been linked to lead (cough, fever), or linked only weakly (all-cause mortality), or at very high exposure (gastro-intestinal problems).

Appendix B further discusses how lead affects blood Hgb levels among children and adults, explains how post-partum Hgb recovery in women can be used to detect lead exposure, and describes the impact of lead exposure on child growth.

3. Data

3.1. Socio-economic and health data

We obtain socio-economic and health data by pooling all 104 geocoded Demographic and Health Surveys (DHS) available at time of writing from countries for which we have mining data. This yields a dataset of repeated cross-sections covering 44 countries, with a total of 1.2 m households, and several million individual records. About 170,000 households are within no more than 20 km of a mine recorded in our data (Table 3). Their location is shown in Fig. A.1.

The DHS data is an obvious choice to study health and development at the micro level across multiple countries: it covers a very broad range of developing countries; surveys have been conducted for nearly 30 years; individual surveys are fairly comparable; sampling cluster geocodes are available for many survey rounds; and there is strong data on maternal and under-five health, including anthropometrics and specifically, Hgb.[10] However, the data also has important limitations with implications for our work. (i) There is relatively little data on socio-economic status and employment, and no information on wages. We therefore work with an asset index, rather than more direct measures of wealth or of income, and discuss employment outcomes only in passing. (ii) Because the surveys have kept changing and improving, very few of our indicators were collected in all surveys. Indeed, the set of observations for which all indicators are available is very small, and impractical to work with. (iii) Finally, the data are cross-sectional. Therefore, while our difference-in-difference tests

[9] As is appropriate to our focus on health impacts, this is considerably tighter than in other current studies of mining in economics. Wilson (2012) uses a cut-off of 10 km, while Aragón and Rud (2016) and Kotsadam and Tolonen (2016) use a baseline cutoff of 20 km, with sensitivity analysis for other choices. With perfect data, we might define closeness even more restrictively. However, in the context of available data, a tighter cut-off would risk introducing excessive noise, both because of the practice of jittering DHS cluster geolocations in our socio-economic data, and because of the fact that we work with (imperfectly recorded) mine point locations as provided by USGS and business intelligence sources, while mining operations can measure several kilometers across.

[10] Other data with high coverage that include both health and socio-economics are either less rich (IPUMS), or less harmonized (LSMS).

Table 4
Summary statistics.

	Observations (1)	Mean (2)	Std. Dev. (3)
A. Wealth outcomes			
Close (house within 5 km of a mine)	90,319	0.197	0.397
Asset index	90,319	0.206	0.976
Household head age (years)	90,319	43.15	13.29
Urban (=1 if urban, 0 if rural)	90,319	0.615	0.487
B. Woman health outcomes			
Woman age (years)	38,217	29.14	9.771
Woman anemia	36,225	0.349	0.477
Woman haemoglobin (g/dL)	38,217	12.34	1.724
C. Child health outcomes			
Child age (years)	44,073	1.974	1.408
Birth order	44,073	1.549	0.886
Child gender (=1 if male, 0 if female)	44,073	0.508	0.500
Mother's age at child birth (years)	44,073	27.12	6.637
Infancy	44,073	0.202	0.401
Child stunted	40,552	0.231	0.422
Child height (height for age z-score)	40,552	−1.002	1.459
Child anemia	18,339	0.554	0.497
Child haemoglobin (g/dL)	18,029	10.64	1.632

Notes. The table presents summary statistics of key variables used in the analysis. These statistics correspond to the maximum sample available to estimate the respective sets of results indicated above in the baseline specification of Equation (1), using mine-year fixed effects. Other specifications generate smaller estimating samples (e.g. Equation (5)), or larger estimating samples (e.g. state-year fixed effects).

are designed to yield evidence of causal effects, they always compare across different individuals.

Our core measure of wealth is a standard asset index computed over household durables and housing characteristics.[11] (Filmer and Pritchett (2001); see Appendix C for details.) We further obtain detailed data on health among children below five years of age, and among women aged 15–49 years. There is little information on other population groups. Our core health indicators are blood Hgb levels and an age-adjusted height index. Hgb is adjusted for altitude, and expressed either in units of grams of hemoglobin per deciliter of blood (g/dL), or as a binary indicator for the clinical condition of anemia, associated with blood Hgb below 12 g/dL in non-pregnant women and 11 g/dL in pregnant women and in children (World Health Organization, 2011). We use the anemia indicator as provided by the DHS. Following standard practice, height is expressed as the difference between a respondent's height and the age-group median, normalized to standard deviations.[12] We consider the continuous height measure, as well as the clinical outcome of stunting (severe stunting), defined as a height of at least two (three) standard deviations below the median. In addition to our core outcomes, we collect data on a range of general adult and child health outcomes, on health care, sexual risk taking, and nutrition. Finally, we construct infant and under-five mortality data for all children whose births were recorded in any survey module.[13] Table 4 provides the summary statistics for the main variables used in the analysis.

3.2. Mining data

We obtain data on the location and characteristics of mines and mineral deposits from four data sources. In total, we observe communities near 838 mines in the cross-section, and 515 mines in the panel, though the set of mines that enters our estimating samples is generally smaller (Table 3).[14]

In the cross-section, we work with the United States Geological Survey's Mineral Resource Database (United States Geological Survey, 2005). It contains the point locations of a very large set of mines, legacies, deposits, and smelters (about 25,000 locations in total) across developing countries. The data records geological information and a basic description of the mine for a substantial subset of entries. However, there is no data on production, and start dates and status of operation are only available for very few mines. In our baseline cross-sectional sample, we include all active mines, legacies (that is, former mines that are now dormant), and smelters. We include smelters because they are often located close to mines, and it is intuitive to think of a single mineral extraction and processing chain from mining to smelting.[15] We include legacies, because the cross-sectional data gives us little guidance in defining whether a mine was operational during a given survey round. The resulting treatment definition should be thought of as yielding 'the effect of living in a location ever exposed to mineral mining or processing'. We extensively parse information on the types of minerals present to sort mines by expected pollutants and hence, corresponding health effects (see Appendix A and B for details).

For a time-varying treatment, we draw additional information from two business intelligence firms: Infomine (2013), and IntierraRMG (2013) – for whose product we henceforth write 'RMD', for 'Raw Materials Data'. Both record dates of operation and annual production. Most mines included in the Infomine data are also available in the RMD data,

[11] We check robustness by constructing an alternative rural asset measure. Details are provided in Appendix D.

[12] We normalize using the median and standard deviation provided by DHS (alternative normalizations make no empirical difference).

[13] Because we construct these variables from birth records of all children ever born to the women in sample, the mortality variables must be interpreted as being conditional on the mother's survival until the survey time.

[14] Nearly all of those mines enter into our model when we use state-level effects. The number of mines near which we observe at least one community within 5 km (treatment) and one within 5–20 km (control) is lower, with 226 mines in the cross-section, and 175 in the panel. These are the mines that enter into our mine-effects models. (See Section 4.)

[15] As shown in Appendix Table H.2, our core results are nearly fully robust to excluding smelters. In one case (panel results on women's hemoglobin), the effect is not significant, although it is consistent in sign and approximate size.

but not vice versa. We therefore work with RMD as our basic data, and add those Infomine entries that are not also contained in the RMD data. RMD mines are more homogenous than those in the USGS sample: most are large mines, and most of those close to DHS clusters are metal mines. While fewer mines are included than in the USGS data, coverage of large mines is quite comprehensive, and the mines included account for a very large share of global metal production. For instance, they account for around 80% of global gold production and 80–90% of global iron ore production in the most recent decade for which data is available. Because there is some question as to the precision of geolocations recorded in the RMD data, we use mine geolocations from an additional dataset, Mining Atlas (2014), for three purposes. First, we add geolocations for RMD mines wherever location is missing in the original data. Secondly, we use company records and Google Earth images to investigate the small number of cases where there are very large discrepancies in location between the two sources; we discard a few records where location is plainly not recorded with any precision in either dataset. Thirdly, we use the two independent but noisy measures of location to check robustness of our results to measurement error in geolocation (see Appendix G).

4. Econometric specification

4.1. Baseline treatment definition

In the baseline model, we define exposure to mining as being geographically close to a mine interacted with the mine being active. This choice is immediate for the study of economic impacts: with transport and search cost, proximity is the treatment of interest. For the purpose of studying health impacts, proximity acts as a proxy for pollution – which we do not observe.

We define a cluster as being *close*, and hence, 'treated', when it is within 5 km of the nearest mine. We will also refer to this as the 'direct vicinity' of the mine. We define a cluster as being in the control group when it is within 5–20 km of the nearest mine. We will refer to this as the 'general vicinity' of the mine. As noted, we bound our treatment group tightly, to correspond to the region in which pollution is likely to occur, and considerably ease the stringency of identifying assumptions required for a causal interpretation of our results. The cost of doing so is that we can only achieve reasonable sample size by allowing our panels to be unbalanced. We argue that this is a reasonable price to pay for the sake of working with a treatment definition that is in line with prior scientific knowledge, and a control group definition that promises to provide a credible counterfactual.

In time, we define mining activity as a dummy variable taking value one when the mine had non-zero output, and value zero when the mine was known to have had zero output. (We conservatively impute inactivity – see Appendix A.) That is, we consider only extensive margin impacts of production. We do so because year-on-year variation in output is likely to be more weakly associated with health outcomes: extracting minerals, breaking them up, and processing them generates a flow of pollution, but the stock of tailings dumped after processing will in many cases continue to pollute. The exact time pattern of pollution is thus hard to predict, but bound to lie somewhere between a pure flow and a pure stock problem. We hope to do it justice by studying extensive margin variation alongside the cross-sectional 'once on, always on' measure. We note, however, that to the degree that impacts arise from cumulative pollution, the approach we take may lead to an underestimation of the treatment effect in the panel.

4.2. Cross section model

In our baseline specification, we consider wealth and health outcomes y for individuals or households i in sampling cluster j within no more than 20 km of a mine, conditional on whether the cluster is *close* (within 5 km) to a mine. Additional covariates X always include an indicator for whether the cluster is in an urban or rural setting, and some appropriate measure of the age of the respondent, the respondent's mother, or the household head. Because we pool repeated DHS cross-sections, our model allows for common effects γ for all observations near the same mine surveyed in the same year (mine-year effects). We cluster error terms at the mine level to account for residual correlations. Wherever the outcome of interest is binary, we use a linear probability model.

$$y_{ij} = \beta_1 close_j + \beta_2 X_i + \gamma_{mine-year} + \epsilon_{ij} \tag{1}$$

Identifying assumptions would be violated if mining towns differed from neighboring communities in geography, institutions or other characteristics in ways that correlate with potential outcomes. However, differences would have to arise even compared to locations very close by, because we restrict control locations to those within the general vicinity of mines. Identification is also only affected if differences are not in some way due to the presence of the mine in long-run equilibrium (for instance, through infrastructure construction, or the emergence of institutions).

We read our cross-sectional results as the long-run effect of mining on 'mining *communities*', analogously to district or county-level studies. To the degree that spatial disparities matter, they are of policy interest. However, they say little about mechanisms of treatment transmission; and due to the possibility of sorting, they do not allow us to make claims about the impact of mining on individuals.

4.3. Panel model

We construct panels in two ways. Firstly, we compare observations from households surveyed at different times, but near the same mine ('mine-level panel'). Secondly, we compare children born to the same mother at different times ('mother-level panel'). Equations (2) and (3) describe the mine-level and mother-level models, for individuals i in cluster j at time t.

$$\begin{aligned} y_{ijt} = {} & \beta_1 close_j + \beta_2 operating_{j(t-\tau)} + \beta_3 close_j * operating_{j(t-\tau)} \\ & + \beta_4 X_{i(t-\tau)} + \gamma_{mine} + f(t) + \epsilon_{ijt} \end{aligned} \tag{2}$$

$$\begin{aligned} y_{ijt} = {} & \beta_1 operating_{j(t-\tau)} + \beta_3 close_j * operating_{j(t-\tau)} \\ & + \beta_4 X_{i(t-\tau)} + \gamma_{mother} + f(t) + \epsilon_{ijt} \end{aligned} \tag{3}$$

In Equation (2), we allow for time-invariant effects γ_{mine} for each mine, and model outcomes at time t as being conditional on whether the respondent lived in a community *close* to a mine during the time period relevant for treatment, $t - \tau$, and whether the mine was *operating* at time $t - \tau$. $\tau \in \{0, age, age + 1\}$.[16] The time periods of interest t and $t - \tau$ depend on the outcome being investigated. For instance, where we analyze height-for-age in children, the outcome is measured in the survey year t, and may be modeled conditional on exposure to mining operations during the survey year ($\tau = 0$), the birth year ($\tau = $ age), or while the child was *in utero* ($\tau = $ age + 1). Our preferred specification includes country-year dummies as time-specific effects $f(t)$. Modifications in the mother-level model are immediate (Equation (3)); because of the much smaller sample sizes, we include country linear trends $f(t)$ in our baseline model.[17]

[16] For each respondent in our sample, we only observe current residence, and how long the household has been resident there. We have no information on previous residence. Therefore, the panel is inherently restricted to respondents who have lived for at least τ years in the location where they were surveyed.

[17] Because we do not observe location of prior residence for migrants, no coefficient on *close* can be estimated in the mother-level panel.

Table 5
Effects on mean asset wealth and wealth disparities.

	Mean asset wealth			
	All HHs (1)	Never-movers (2)	All HHs (3)	Never-movers (4)
HH close to mine	0.105***	0.0784*	−0.113	−0.0352
	(0.035)	(0.0423)	(0.089)	(0.0892)
Mine operating			−0.0296	−0.0585*
			(0.0348)	(0.0354)
Mine operating ∗ HH close (DiD)			0.262***	0.173*
			(0.0958)	(0.0897)
Number of households	90,319	31,079	22,579	9,459
Number of groups	1,562	1,371	218	205
R-squared	0.094	0.081	0.13	0.141

Notes. The table reports estimates of Eqs. (1) and (2). Cross-sectional estimates in columns (1–2) use indicator variables for each mine-year pair as fixed effects. Panel estimates in columns (3–4) use mine-level indicators for area fixed effects, and country-year indicators for time effects. The dependent variable in Columns (1–4) is the asset factor index, with units expressed in standard deviations. Controls include a quadratic in the household head's age and an indicator for urban/rural status. Columns marked 'Never-movers' restrict the sample to households with at least one respondent who had always been resident in the current location at survey time. Standard errors are clustered at the mine level. Significant at * 10%, ** 5%, *** 1%.

4.4. Difference-in-differences tests tailored to the health conditions studied

We leverage the scientific understanding of the health conditions of interest to construct additional difference in differences (DiD) tests. Like the pseudo-panel, they compare the impact of mining across groups that are and are not expected to be affected. Yet, because they each build upon a different insight into the likely nature of exposure and the organism's reaction to it, they generate distinct control groups, and hence, further "reduce the importance of biases or random variation in a single comparison group" (Meyer, 1995, p.157).

Mine types: Distinct mine types are associated with specific pollutants and health effects. This allows us to contrast differences across distance groups near mines where an effect is expected, and near mines where none is expected, as in Equation (4).

$$y_{ij} = \beta_1 close_j + \beta_2 heavy\ metal\ mine_j + \beta_3 close_j * heavy\ metal\ mine_j + \beta_4 X_i + \gamma_{mine-year} + \epsilon_{ij} \quad (4)$$

Because the test does not rely on time-varying production data, it preserves sample size particularly well, and generates a balanced estimating sample. Identification rests on the assumption that potential outcomes vary among those close and not close to the mine in similar ways near mines of different types. Most obviously, if wealth effects varied systematically among mine types, health results might be confounded. With respect to preference-based sorting, the assumption would be violated if respondents were aware of how mine types differ in health outcomes, and sorted accordingly. We address the issue in two ways. Firstly, we show that differences arise for health outcomes, but not wealth. Secondly, we show that there are DiD effects only on specific expected health outcomes, not general health.

Maternal Hgb recovery: We develop a DiD test based on the observation that in lead-exposed adults, the recovery of Hgb after blood loss is even more readily affected than the steady-state level of Hgb. In the toxicological literature, this result was previously proven by studying Hgb recovery after donating blood (see Appendix B). In our data, we study another population group that experiences dramatic drops in Hgb: women who are pregnant, or have recently given birth. We ask whether differences in Hgb between women i in mining and control communities j are particularly stark during pregnancy and postpartum. As we discuss in Section 5.2, the model (Equation (5)) requires particularly weak identifying assumptions, and we can devise a range of tests to exclude any interference from sorting. Because the sample of women we observe within the time period of interest is small, in our preferred specification, we (i) pool observations near heavy metal mines from all of our data sets, and (ii) estimate the model with state-year effects.

$$y_{ij} = \beta_1 close_j + \beta_2 pregnant\ or\ postpartum_i + \beta_3 close_j * pregnant\ or\ postpartum_i + \beta_4 X_i + \gamma_{state-year} + \epsilon_{ij} \quad (5)$$

5. Results

5.1. Effect on wealth

5.1.1. Mining towns are wealthier than control communities, both in the medium and long term

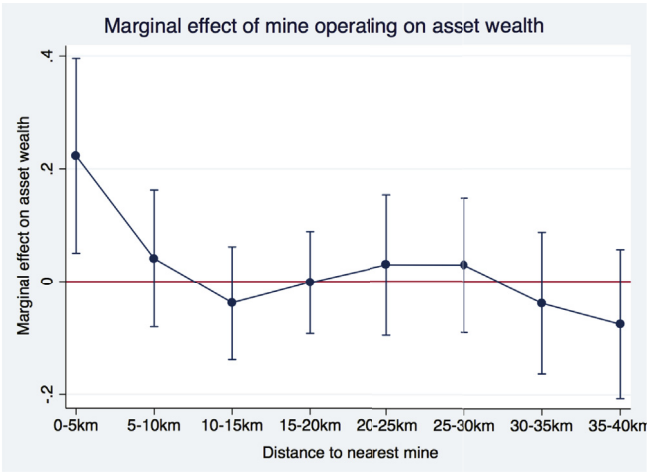
Households in mining communities are at the mean considerably wealthier in terms of asset ownership than control households. The magnitude of the effect at the global average is on the order of 0.11 standard deviations of the asset index in the cross-section, and 0.26 standard deviations in the panel (Table 5, Columns 1 and 3). Since survey rounds are typically about five years apart in the panel, we interpret the latter as a medium-term effect.

The effect size is appreciable, given that in our sample, there is generally great within-country variation in asset ownership. In the linear index, the magnitude of the cross-sectional effect is comparable to that of owning a car or motorbike in the case of Peru in the year 2000, and to the effect of owning a radio or a watch in the case of Burkina Faso in the year 2010. The panel effect is comparable to having an electricity connection or living in a dwelling with finished flooring in the case of Peru in the year 2000, and to the effect of owning a motorbike or mobile phone in the case of Burkina Faso, in the year 2010. (See Appendix C for further detail.)[18]

Effects among households that report never having moved from their current location are somewhat smaller and weaker (though not significantly different) in both the cross-section and the panel (Table 5, Columns 2 and 4). We interpret this as limited evidence of residential sorting of migrants with better potential socio-economic outcomes into mining communities, or sorting of previous residents with better poten-

[18] An in-depth analysis of effects on employment is not possible. In the cross-section, unemployment among men is virtually unaffected, consistent with long-run general equilibrium. As is intuitive, the sectoral share of agriculture decreases alongside ownership of agricultural land. In the panel, employment effects tend to be adverse in sign – consistent with queuing – but estimates are noisy and not stable. (Results available upon request.) See Kotsadam and Tolonen (2016) for a detailed discussion of effects on women and sectoral shifts in sub-Saharan Africa.

(a) Effect of closeness: cross-section



(b) Marginal effect of mine operating: panel

Fig. 1. Effect of closeness to mines on asset wealth.
Notes. The figure shows the spatial pattern in wealth effects. Plot (a) shows predicted asset wealth given distance to the nearest mine, from the cross-sectional regression in Equation (1), using mine-year fixed effects. Plot (b) shows the estimated effect of the nearest mine being active, conditional on distance, from the mine-level panel regression in Equation (2), using mine fixed effects and country-year dummies. Controls include a quadratic in the household head's age and an indicator for urban/rural status. Standard errors are clustered at the mine level. 95% confidence intervals are shown.

tial outcomes out of mining communities.[19]

Wealth effects decay steeply with distance to the nearest mine. In the panel, effects are limited to those living with 5 km; in the cross-section, there is a gradient up to a distance of 20 km, perhaps reflecting diffusion of gains over time (Fig. 1). It is notable that even in the cross-section, the estimated spatial extent of wealth gains is smaller than in the case study analyzed in Aragón and Rud (2013, p. 13), who find "positive and significant [income effects] for households located within 100 km of Cajamarca city," the community closest to the mine studied. The discrepancy could be due to the fact that Aragón and Rud study a policy change that can be presumed to be very favorable for local welfare; or the fact that they consider the case of a very large mine in a region with reportedly high transport cost. In addition, Aragón and Rud have income data available; presumably, a more sensitive measure of well-being than our asset index.

Quantile regression shows that wealth benefits arise across the distribution, with progressive impacts in the panel, but higher gains for the top quantiles in the long run (Fig. 3).[20]

5.1.2. Robustness checks for effects on wealth

In the appendices, we show that our results are robust and conservative. We obtain larger and significant estimates when instrumenting for mine locations and operating times with deposit location and world mineral prices (Appendix D). Our unweighted baseline estimates are smaller than estimates obtained by alternative weighted regressions (Appendix F). In addition, we use two independent measures of the geolocation of mines to show that our baseline results likely carry substantial attenuation bias – in our preferred specification, some 18% of the estimate (Appendix G). Finally, Appendix Table D.2 shows that in a simple meta-analysis of mine-level estimates, long-run wealth gains are greater in a weak overall economic environment. While these correlations cannot be interpreted as causal relationships, they raise the question whether the local economic effect of mining might be driven not by the interaction of mining with other economic activity, but by the opportunities mining provides in areas where there is a paucity of other options.

Wealth gains at the mean and across the distribution, and *prima facie* evidence of sorting of households with better potential economic outcomes all provide confidence that any health effects we report are unlikely to be caused by poverty. They also underline that the estimated health effects must be viewed as *net* effects: households have higher income, but are unable or unwilling to avoid losses in health.

5.2. Evidence of hematologic toxic effects

We have argued above that exposure to lead among residents of mining communities may be reflected in the DHS data in depressed Hgb concentration, because lead affects the hematopoietic system and reduces red blood cell survival (see Appendix B).

5.2.1. Hemoglobin levels in adult women are strongly depressed in mining communities

In the cross-section, blood hemoglobin (Hgb) levels are depressed among women living in mining communities by about 0.09 g/dL (Table 6, Column 1). The effect among never-movers is larger (0.13 g/dL), consistent with longer exposure to environmental lead, although (on this smaller sub-sample) it is below significance (t = 1.56) (Column 2). The prevalence of the clinical outcome of anemia is significantly elevated by three percentage points among all households, and by five percentage points among never-movers (Columns 4–5).

Panel results confirm these patterns. Point estimates are larger, with DiD coefficients of a 0.33 g/dL decrease in blood Hgb, and a ten percentage point increase in the incidence of anemia in our preferred specification. (Table 6, Columns 3 and 6) A number of causes could account for the larger point estimate in the panel; notably, the share of metal mines associated with lead pollution is high in the panel sample (as we show below, the treatment effect is concentrated near such mines). In the long-run, there might also be more adaptation to avoid pollution.

[19] There is only weakly more migration in mining communities than in neighboring communities. However, in both mining and control communities, the share of migrant households is very high: around 60% of households migrated at some time, and about 23% migrated within the five years preceding the survey. Sorting could therefore explain some of the cross-sectional differences, if the characteristics of migrants (including those unobserved households who left the communities) are sufficiently different.

[20] Long-term effects could be accounted for by slow sorting of high-income households into mining communities, or the gradual emergence of some economic opportunities that are open only to a select few. The medium-term pattern is consistent with distributional effects reported in Aragón and Rud (2013). See Appendix D for further discussion.

Table 6
Hematologic toxic effects on women.

	Altitude-adjusted hemoglobin (g/dL)			Anemia		
	All HHs (1)	Never-movers (2)	All HHs (3)	All HHs (4)	Never-movers (5)	All HHs (6)
HH close to mine	−0.0863**	−0.131	0.396***	0.0262**	0.0495*	−0.107***
	(0.0438)	(0.0838)	(0.146)	(0.0126)	(0.0268)	(0.0292)
Mine operating			0.0852			−0.0277
			(0.136)			(0.0309)
Mine operating * HH close (DiD)			−0.330*			0.0966**
			(0.173)			(0.0390)
Number of women	38,217	13,506	9,845	36,225	13,204	9,845
Number of groups	934	785	122	934	784	122
R-squared	0.0001	0.001	0.007	0.0003	0.001	0.006

Notes. The table reports estimates of Equations (1) and (2). Cross-sectional estimates in columns (1–2) and (4–5) use mine-year fixed effects, while panel estimates in columns (3) and (6) use mine-level indicators as area fixed effects, and country-year indicators as time effects. Columns (1–3) show effects on hemoglobin levels (in grams per deciliter of blood) at survey time among adult women; columns (4–6) show results for the incidence of anemia (defined as Hgb below 12 g/dL in non-pregnant women, and Hgb below 11 g/dL in pregnant women). Controls include a quadratic in the respondent's age, and an indicator for urban/rural status. Columns (2) and (5) restrict the sample to respondents who had always been resident in the current location at survey time. Standard errors are clustered at the mine level. Significant at * 10%, ** 5%, *** 1%.

The size of the effect on Hgb levels can be compared, for instance, to changes in Hgb on the order of 1 g/dL associated with treating anemic pregnant women with a course of iron supplementation (Sloan et al., 2002). That is, we obtain a general population effect estimate on the order of one-tenth to one-third of the effect of a targeted intervention in a highly susceptible population. The increase in the incidence of anemia is a large effect in absolute terms, though it must be seen in the context of a baseline proportion of anemic women of 36% in control locations. The cross-sectional effect thus amounts to a 7% relative increase in incidence, and the panel effect, to a 27% relative increase. The contemporaneous productivity loss due to anemia in adults has been estimated to be on the order of 5–17% (Horton and Ross, 2003). Appendix E shows spatial and distributional patterns, and Appendix I shows the distribution of mine-level effects across countries.

We adduce two additional difference-in-differences (DiD) tests, both to further bolster identification, and to help establish that pollution is the likely cause of depressed blood hemoglobin.

5.2.2. *Hemoglobin levels are only affected near mines where we expect heavy metal pollution*

Table 7 (Panel A) shows that the effect on Hgb levels of living in mining communities are statistically zero (and mildly negative) in women living near mines where there is less reason to expect heavy metal contamination (Column 1). However, in mines where there is a high likelihood of such contamination, Hgb levels are strongly and significantly depressed – by about 0.22 g/dL relative to women living farther away from the same mines, and by 0.19 g/dL compared to women living near non-heavy metal mines. Correspondingly, the incidence of anemia is five percentage points higher compared to women living near non-heavy metal mines (compared to women living further away from the same mines, it is six percentage points higher) (Column 2). The size of the cross-sectional effect near heavy metal mines is far closer to the panel effect than the average effect in the cross-section.[21] As noted (in Appendix B), our definition of heavy metal mines is best thought of as a meaningful but imperfect proxy of the presence of lead and other toxic metals. In consequence, DiD estimates are likely attenuated.

The DiD effect is robust to including interactions of the treatment dummy with region indicators (hence allaying concerns over geographical clustering of heavy metal mines), as well as to including an interaction of the treatment with a pregnancy dummy (see Appendix Table H.8, Columns 5–6). We note that there is a significant negative effect of living near *any* mine in Latin America (the base category for the region interaction), perhaps due to the imperfect nature of our definition of heavy metal mines. The effect near any mine is statistically zero for the other regions.[22] We further estimate the DiD model for the asset index, and confirm that there is no differential wealth impact of living close to a heavy metal mine, as opposed to any mine (Table 7, Panel A, Column 3). Finally, we do not observe similar differential effects of living near a mine associated with heavy metal contamination on two general indicators of ill health among women, namely miscarriage, and grave sickness (Columns 4–5).

5.2.3. *The trajectory of maternal Hgb recovery after birth in mining communities corresponds with a known pathophysiological pattern*

The left panel in Fig. 2 shows the pattern of recovery from blood loss during pregnancy and delivery among women living close to heavy metal mines, and those living in adjacent areas. Hgb levels conspicuously diverge during pregnancy, and stay apart during the first one and one-half years of the child's life. However, thereafter, they converge to an apparent noise pattern about a common mean. (The right panel shows the same data, with effects smoothed out for the nine months from conception to birth, and each year of the newborn's life, thereafter.) The pattern is characteristic of a pollution-induced decrease in the ability to recover Hgb after blood loss, as described in Grandjean et al. (1989) (and discussed in Appendix B), but not of other causes of anemia.

While the pattern is visually striking, given limited sample size, we cannot test the difference between coefficients for the two distance groups in each individual trimester. Instead, we test for the difference in differences between the groups across two time periods: pregnancy and the first year of the infant's life (when there is the clear impression of divergence), and the second and third years of the child's life (when there is not). The results presented in Table 7 (Panel B) show that the DiD coefficient is negative, large (0.25 g/dL), and significant (Column 1). That is, the difference in Hgb levels between women exposed to mining and other women is far greater during and after blood loss due to pregnancy and delivery, than after some time has passed since delivery. The single difference in distance is negative, but not stable on the small sub-sample of women in the model. As expected, Hgb is dramatically

[21] A similar test is hard to construct for the panel, since mines that are potentially associated with heavy metal contamination make up a large part of the sample.

[22] As a further robustness check, Appendix I demonstrates that the median difference between heavy metal mines and non-heavy metal mines is always at least weakly negative in each individual country for which sufficient mine-level estimates can be computed.

Table 7
Difference-in-differences tests of hematologic toxic effects on women.

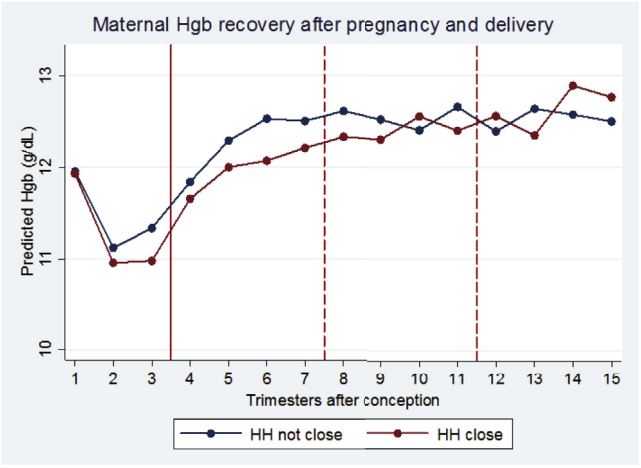
Panel A: Effects on women near different mine types					
	Hgb (g/dL)	Anemia	Falsification tests		
			Asset index	Miscarriage	Grave illness
	(1)	(2)	(3)	(4)	(5)
HH close to mine	−0.0317	0.0123	0.140*	0.00347	0.00366
	(0.0533)	(0.0155)	(0.0756)	(0.00519)	(0.00595)
HHs close ∗ 'heavy metal' mine (DiD)	−0.192**	0.0466*	0.0176	−0.00377	−0.00286
	(0.0944)	(0.0247)	(0.101)	(0.0109)	(0.00898)
Number of women	38,217	36,225	25,695	117,118	11,022
Number of groups	934	934	932	1,469	151
R-squared	0.001	0.0004	0.111	0.061	0.011
Panel B: Recovery of maternal Hgb after birth near heavy metal mines					
	Hgb (g/dL)		Falsification tests		
			Asset index	Hgb (g/dL)	Hgb (g/dL)
	(1)	(2)	(3)	(4)	(5)
Pregnancy and infancy	−0.558***	−0.581***	−0.0644**	−0.470***	−0.652***
	(0.0421)	(0.0375)	(0.0255)	(0.0771)	(0.0621)
HH close to mine	−0.0277	−0.00274	0.0304		
	(0.0791)	(0.101)	(0.0523)		
Pregnancy and infancy ∗ HH close (DiD)	−0.253**	−0.185*	−0.0122		
	(0.0982)	(0.107)	(0.0562)		
HH in lowest wealth quintile				−0.169*	−0.232***
				(0.0862)	(0.0551)
Pregnancy and infancy ∗ HH in lowest quintile				−0.0231	0.0783
				(0.0825)	(0.0538)
Number of women	5,004	5,004	4,892	6,851	14,857
Number of groups	167	521	167	139	269
R-squared	0.045	0.044	0.125	0.028	0.031

Notes. The table shows DiD estimates of the effect of living close to a 'heavy metal' mine on hemoglobin levels in women. Panel A shows estimates of Equation (4), using indicators for each mine-year pair as fixed effects. Panel B shows estimates of Equation (5), using indicators for state-year pairs as fixed effects in all columns except Column (2), where mine-year indicators are used. Columns (1–2) in Panel A and Columns (1–2 and 4–5) in Panel B show effects on hemoglobin levels and the incidence of anemia at survey time among adult women. Hgb is given in g/dL of blood, and anemia is defined as Hgb below 12 g/dL in non-pregnant women, and Hgb below 11 g/dL in pregnant women. Columns (3) in each panel show effects on asset wealth in households, and Columns (4–5) in Panel A show additional falsification tests with the incidence of two health conditions not specific to lead exposure – miscarriage and grave illness. 'Miscarriage' is an indicator for whether a woman of reproductive age has ever suffered a miscarriage; 'grave illness' it is an indicator for whether the respondent was gravely ill for three months or more in the year preceding the survey. Columns (4–5) in Panel B construct a falsification test in which the treatment variable is replaced with an indicator that takes value one if the respondent's household is in the bottom wealth quintile, and value zero if it is in the top wealth quintile. In Column (4), the falsification sample is restricted to women who are pregnant or have given birth in the past three years, but live in households at least 20 km from the nearest mine, and are observed in state-year pairs also represented in the sample in Column (1). In Column (5), it is restricted to women who are pregnant or have given birth in the past three years, live at least 20 km from the nearest mine, and are observed in country-year pairs also represented in the sample in Column (1). Where the dependent variable is a health condition, controls include a quadratic in the respondent's age at survey time and an indicator for urban/rural status. Where the dependent variable is the asset index, a quadratic in the household head's age replaces the respondent's age. Standard errors are clustered at the mine level in Panel A and Column 2 in Panel B, and at the state level in the remaining Columns in Panel B. Significant at * 10%, ** 5%, *** 1%.

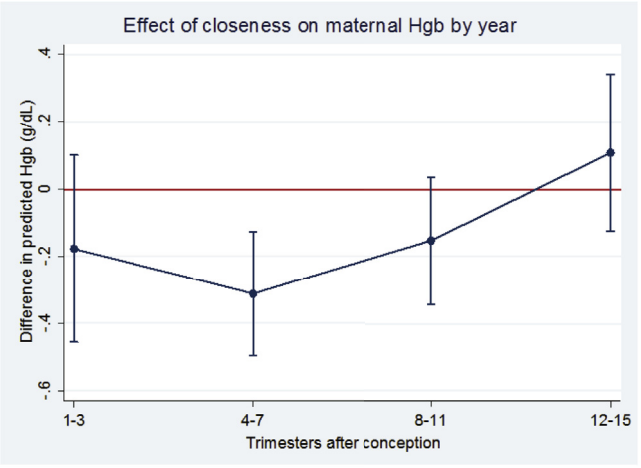
lower in all women during pregnancy and in the first year post-partum. To maximize sample size, in Panel B, we use a combined sample of metal mines from all datasets. The estimates are qualitatively similar but statistically insignificant when obtained from the USGS or RMD samples separately (Appendix Table H.13).

The pattern is similar when we estimate the model with mine-level fixed effects, as shown in Column (2) (Table 7, Panel B). Mine-level results do not always reach significance, but are as stable as the state-level results when we include controls, vary the treatment definition, or conduct falsification tests. Because of the small sample size and strong identification from the DiD setup, we prefer the state-level model. In our baseline model, we consider a postpartum period of three years. This seems more appropriate than shorter periods because the detailed time pattern of Hgb recovery shown in Fig. 2 suggests that differences even out only in the second year of the child's life. It seems more appropriate than longer periods because the more we extend the time window, the stronger are the identifying assumptions required. Results are robust to extending the post-partum control period to four or five years; they are directionally consistent but insignificant when we shorten it to just two years. (Results not shown.)

Because the test uses as a counterfactual women whose most recent birth lies at most three years in the past, identification requires only that the precise timing of pregnancies is ignorable within a limited time window. However, somewhat complex behavior patterns could generate the observed effect. Perhaps most simply, wealth could be associated with different child bearing choices in mining communities and control locations. For instance, it might be that wealthier women (with higher baseline Hgb levels) tend to have fewer children or space out births more in mining communities than in communities farther afield – perhaps because of better earnings opportunities. The DiD effect could then be due to comparing (relatively) poorer women in mining towns to richer controls in the pregnancy and post-partum group, and (relatively) wealthier women in mining towns to poorer controls for the following years.

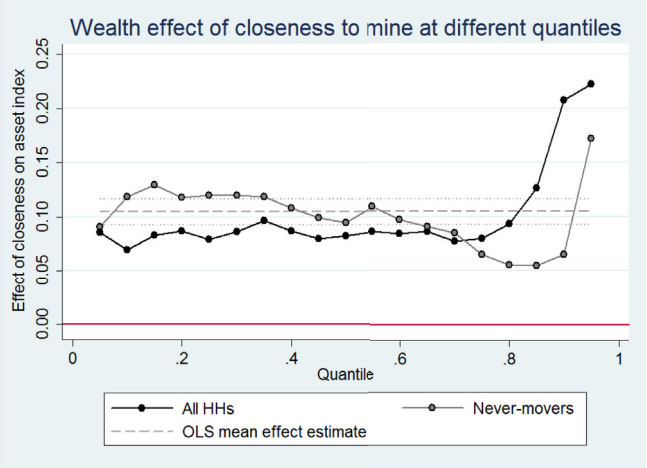


(a) Hgb recovery pattern: close vs. far

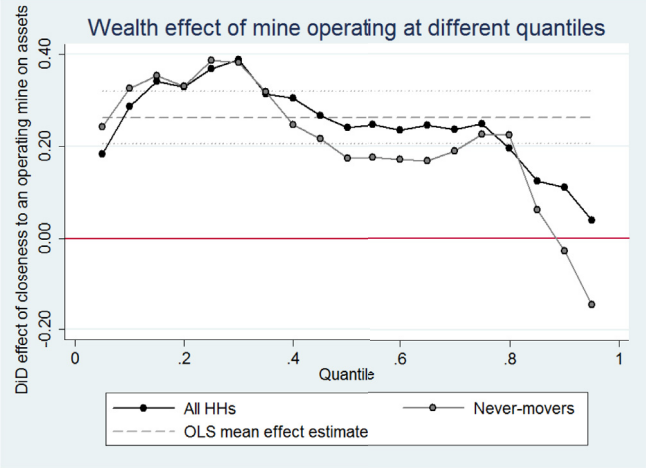


(b) Hgb difference between close and far

Fig. 2. Effect of closeness to heavy metal mines on maternal Hgb recovery.
Notes. The figure shows the pattern in women's Hgb loss and recovery during pregnancy and post-partum. Pregnancy and delivery are known to cause a significant blood loss in women, which recovers gradually following child birth. Plot (a) compares the Hgb recovery pattern by trimester after conception among women living close to heavy metal mines and those living farther away. Plot (b) shows the estimated differences between these two groups, smoothed out for the nine months from conception to birth, and each year of the newborn's life, thereafter. As in Equation (5), state-year fixed effects are used. Controls include a quadratic in the respondent's age at birth and an indicator for urban/rural status. Standard errors are clustered at the state level. 95% confidence intervals are shown.



(a) Cross-section: effect of closeness



(b) Panel: effect of mine operating

Fig. 3. Effect of mining on asset wealth at different quantiles of the wealth distribution.
Notes. The figure shows quantile regression estimates of effects on wealth, obtained at every 5 ppt of the distribution, using the two-step procedure described in Canay (2011). Plot (a) shows the wealth effect of living close to a mine in the cross-section, while Plot (b) shows the effect of living close to an operating mine in the panel, as in Equations (1) and (2). Both plots separately show the results for the full sample and for a sub-sample consisting of never-mover households. Mine-year fixed effects are used in the cross-section, and mine-level fixed effects and country-year dummies in the panel. Mean OLS effects are shown with a dashed line, with their 95% confidence intervals. Confidence intervals for the quantile regressions are very wide, and are omitted for readability.

To conclusively assess this concern, we first note that there are no significant DiD effects on wealth (Table 7, Panel B, Column 3). Secondly, we show a regression to test whether a similar recovery pattern emerges when we compare mothers in households in the bottom wealth quintile ('treatment' sample) to those in the top quintile ('control' sample). We generate two samples: a small sample designed to match the baseline sample particularly tightly, and a larger sample designed to allow for more power. Both samples include women who are pregnant or have given birth within the past three years, and reside at least 20 km away from the nearest mine. The small sample is restricted to observations in the same state-year pairs as those observed in the main model, and the large sample, to observations within the same survey rounds. As expected, Columns (4–5) in Table 7 (Panel B) show that women in poor households always have lower Hgb levels than those in wealthy households – but there is no indication of an adverse time pattern around pregnancy and postpartum, with DiD coefficients either near zero, or with an opposite sign. Appendix Table H.9 shows that results are also robust to controlling directly for the woman's height as a slow-moving wealth proxy, or for whether she gave birth in an 'improved' setting (Columns 3 and 4).

We argue that both DiD test results are instructive regarding mechanisms of treatment transmission and regarding identification. In terms of mechanisms, they offer strong evidence that the observed health effect is caused by pollution, not other facets of life near mines. For instance, if the observed effects on Hgb were due to iron deficiency or malaria infection, then nutritional behavior and infection rates

Table 8
Hematologic toxic effects on children.

Panel A: Cross-section		
	Hgb (g/dL)	
	Children (*age* ≤ 5 years)	Infants
	(1)	(2)
HH close to mine	−0.0653	−0.0487
	(0.0460)	(0.0505)
HH close to mine, and child in infancy		−0.0823
		(0.102)
Number of children	18,029	18,029
Number of mines	907	907
R-squared	0.068	0.021
Panel B: Mine-type DiD		
	Children (*age* ≤ 5 years)	Infants
	(1)	(2)
HHs close to a 'heavy metal' mine (DiD)	−0.109	−0.0184
	(0.108)	(0.119)
HH close to mine, nearest mine is a 'heavy metal' mine, and child in infancy (triple difference)		−0.597***
		(0.199)
Number of children	18,029	18,029
Number of mines	907	907
R-squared	0.068	0.022

Notes. Panel A shows estimates of Equation (1), and Panel B of Equation (4). Mine-year fixed effects are used. The dependent variable is hemoglobin at survey time (g/dL). In each panel, the treatment variables have been interacted with an indicator for whether the child was in her first year of life at survey time. The regressions also include a dummy for infancy, and all other appropriate interactions. For readability, only coefficients on treatment effects are shown; full results are available in Appendix Table H.10. Controls include an indicator for urban/rural status in all columns, a quadratic in the mother's age at birth, an indicator for gender, birth-order indicators, as well as indicator variables for the child's age. Standard errors are clustered at the mine level. Significant at * 10%, ** 5%, *** 1%.

would have to vary across distance groups in systematically different ways near metal and non-metal mines, and among pregnant and non-pregnant women – despite the fact that socio-economic outcomes do not vary in such ways. The results also provide reassurance on identification, most importantly because they are very hard to explain with sorting. Because mine types differ in health impacts, but not in wealth and non-specific health impacts, one would have to hypothesize that in their migration decisions, people not only take mine type into account, but also differentially sort on their potential health and wealth outcomes. This would require an extraordinary level of sophistication.

5.2.4. *Residents of mining communities are not differentially affected by causes of anemia other than lead exposure, do not bear a higher burden of disease unrelated to pollution, and are not under-served by health care*

The high dimensionality of the DHS data allows for diverse falsification tests that could yield evidence against our contention that the observed hematologic effects are due to pollution, not other mechanisms. Across a range of tests, we find no such evidence.

Firstly, we show in Appendix Table H.1 that here is no conclusive pattern in mining communities in the leading causes of anemia other than lead toxicity (nutritional iron deficiency, malaria, and intestinal worm infections). Secondly, we test whether residents of mining communities suffer ill health that is unlikely to be attributable to pollution. Significance patterns are very sparse in the cross-section, and there are no significant adverse health impacts at all in the panel (Appendix Tables H.4 and H.5).[23] Finally, we note that residents of mining communities are at least as well off in terms of health care as those living farther afield. As Appendix Table H.6 demonstrates, in the long run, women are more likely to have health insurance coverage, and to give birth with some level of skilled assistance. Panel results suggest that such benefits, along with access to health care, may extend beyond the immediate vicinity of the mine. The one potential exception to this pattern is that in our mother-level panel, we find significant decreases in the share of women who gave birth in an improved setting in mining communities when the mine was operational. The cross-sectional and mine-level panel evidence contradicts this finding. However, it is at odds with our otherwise consistent evidence on wealth. We note that our discussion of maternal Hgb recovery explicitly sought to exclude the potential effect of differences in maternal health care.

5.2.5. *Patterns of anemia among children mirror those among women, but are less conclusive*

Our data shows patterns of anemia among children in mining communities that resemble those found among adult women. However, significant results are hard to come by. This may be because the true treatment effect is weaker – we note in the Appendix B that children can effectively compensate for the hematologic toxicity of lead by increasing production of EPO and red blood cells. It may also be due to small sample size (for children, we only have about half the number of observations in the women's sample). In the cross-section, we observe insignificant decreases in Hgb on the order of 0.07 g/dL (Table 8, Panel A, Column 1); the effect is strongly concentrated near heavy metal mines, but the DiD coefficient is again not significant (Column 2). The panel shows statistically insignificant losses from current exposure to mining, but is highly sensitive to changes in the treatment definition. (Results omitted.)

Next, we ask whether infants might be more strongly affected by pollution than older children. There are two reasons to expect this pattern. Firstly, we have attributed anemia among women – and particularly, pregnant women – in mining communities to lead exposure, and it is known that children are born with a lead burden mirroring that of their mothers. Secondly, it has been previously shown that compensatory over-production of EPO and Hgb in lead-exposed children does not quite start at birth, but at some point during infancy (Wasserman et al., 1992).

The differential impact on infants near heavy metal mines is both significant and large (Table 8, Panel A, Column 2). The triple-difference coefficient shows a 0.60 g/dL difference in Hgb levels. The difference

[23] It is worth noting that residents of mining communities may obviously suffer health impacts – or enjoy health benefits – that we do not observe.

in differences between the effect on infants near heavy metal mines and infants near other mines is very similar in size and significance. Detailed results are provided in Appendix Table H.10, where we find a larger but insignificant effect of living near a mine on infant Hgb, relative to infants living farther away from the mine (Column 3). Falsification results show that infants near these mines are not indiscriminately less healthy (Columns 6–8). However, we caution that infants born in the direct vicinity of heavy metal mines tend to live in poorer households (Column 5). This makes it somewhat less compelling to interpret the effect as evidence of pollution – by way of contrast with results among women, where there is no correlation with wealth.

5.3. Evidence of adverse growth outcomes

As noted, exposure to environmental lead has previously been linked to decreased growth early in life, though the evidence is mixed (Appendix B). We consider impacts on height for age and the incidence of stunting and severe stunting.

5.3.1. Without regard to time patterns of exposure, children in mining communities grow taller than their peers

In the simple cross-section, we observe *better* outcomes for height among children of less than five years of age in mining communities than in the controls (Table 9, Panel A, Column 1). This may not be surprising: growth is strongly linked with nutrition (both the mother's and the child's), and with greater wealth in mining communities, there may also be better diets. There is also no differential impact near 'heavy metal' mines (Column 3).

However, the evidence is somewhat more subtle. Firstly, as Column 2 (Table 9, Panel A) makes obvious, there is no indication of a positive effect among never-movers. Secondly, although infants are not more affected than older children when we consider *all* types of mines, there is at least some indication of an adverse effect on infants of living near a 'heavy metal' mine. The triple-difference effects are adverse, and significant for stunting (Panel B, Columns 4–6). There are no significant differences between mine types in the economic status of families with infants (Appendix Table H.11).

The cross-sectional evidence alone is thus not easy to interpret. There clearly are growth benefits to be had for children in mining communities, and it seems obvious to connect these to the wealth increases enjoyed by residents. However, not all children appear to benefit. The question is whether this is because some children are simply left out from economic gains, or whether they suffer countervailing direct health damage. The absence of a DiD effect between mine types and of a differential effect on infants near all mines may suggest the former. Yet, the appreciable effect on infants near mines associated with heavy metals may point toward the latter. Similarly, the difference between never-movers and the general population is consistent with lower economic benefits among never-movers. (See Table 5.) However, since the differential in wealth effects is not very large, it is reasonable to note that children born to never-movers are also more likely to have been exposed to pollution, particularly *in utero*, through the maternal body burden of lead. We look to the panel for more conclusive evidence of the impacts of different exposure patterns.

5.3.2. Panel evidence shows that in utero exposure to mining increases the incidence of stunting

Results from the mine-level panel suggest that there is an effect of mining activity on height, that the effect is chiefly due to exposure *in utero*, and that it attenuates with age. It also allows us to at least suggest that there are genuinely positive effects of life in mining communities on growth in older children, so that children do not simply 'out-grow' *in utero* effects without further exposure, as earlier reported by Shukla et al. (1991).

The DiD effect of *in utero* exposure among all children under five years of age shows a loss of 0.14 standard deviations in the height index (Table 10, Column 1), and a five percentage point increase in the incidence of stunting and severe stunting (Columns 4–5). The effect on the discrete outcomes is significant; the one on the continuous measure is not significant but stable. With a baseline incidence of 23% and 8%, respectively, the impact on stunting is appreciable, and the impact on severe stunting very large. Associated economic losses are potentially important: stunting that persisted into adulthood has been estimated to have caused wage losses as large as an annual 53% drop

Table 9
Growth effects on children in the cross-section.

Panel A: Effects on all children under five years of age (cross-section and mine-type DiD)			
	Height for age		
	(1)	(2)	(3)
HH close to mine	0.0828**	−0.00153	0.0754*
	(0.0389)	(0.0497)	(0.0430)
HHs close to a 'heavy metal' mine (DiD)			0.0378
			(0.0988)
Sample restriction	All Households	Never-movers only	All Households
Number of children	40,552	16,927	40,552
Number of groups	1,244	1,041	1,244
R-squared	0.064	0.054	0.064
Panel B: Effects on infants near 'heavy metal' mines			
	Height for age	Stunting	Severe stunting
	(4)	(5)	(6)
HH close to a mine, closest mine is a 'heavy metal' mine, and child in infancy (triple difference)	−0.170	0.0696**	0.0280*
	(0.110)	(0.0327)	(0.0166)
Number of children	40,552	40,552	40,552
Number of groups	1,244	1,244	1,244
R-squared	0.062	0.036	0.015

Notes. In Panel A, Columns (1–2) show estimates of Equation (1), and Columns (3), estimates of Equation (4). Mine-year fixed effects are used. In Panel B, the treatment variable and its interaction in Equation (4) are interacted with the indicator variable for infancy. For readability, only coefficients on treatment effects are shown; full results are available in Appendix Table H.11. Columns (1–4) show effects on height for age z-scores. The dependent variable in Column (5) is the prevalence of stunting, defined as a height of two standard deviations or more below the median; in Column (6), it is the prevalence of severe stunting, defined as a height of more than three standard deviations below the median. Controls include an indicator for urban/rural status in all columns, a quadratic in the mother's age at birth, an indicator for gender, birth-order indicators, as well as indicator variables for the child's age. Standard errors are clustered at the mine level. Significant at * 10%, ** 5%, *** 1%.

Table 10
Comparative growth effect of *in utero* and birth-year exposure in the panel.

	In utero exposure					In utero vs. birth year exposure			
	Height		Height (infants only)	Stunting	Severe stunting	Height			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Mine operating during pregnancy * HH close	−0.136	−0.460	−0.371*	0.0534*	0.0496***			−0.494***	−0.422
	(0.0981)	(0.361)	(0.217)	(0.0274)	(0.0140)			(0.139)	(0.359)
Mine operating in birth year * HH close						−0.00860	−0.163	0.423***	−0.0540
						(0.110)	(0.424)	(0.149)	(0.418)
Mine FE and Country-Year FE	Y		Y	Y	Y	Y		Y	
Mother FE and Country-Year trend		Y					Y		Y
Number of children	11,629	11,629	2426	11,629	11,629	11,629	11,629	11,629	11,629
Number of fixed effects	200	9,408	186	200	200	200	9,408	200	9,408
R-squared	0.113	0.204	0.091	0.072	0.055	0.113	0.204	0.114	0.205

Notes. The table shows the effect of mining exposure on child growth outcomes in the panel. Columns (1, 3–6 and 8) report estimates of Equation (2) using mine fixed effects, and Columns (2,7 and 9), estimates of Equation (3) using mother-level fixed effects. In Columns (1–5), treatment is defined as exposure to mining *in utero*. Columns (6–9) compare this to the effect of exposure in the birth year. For readability, only coefficients on treatment effects are shown; full results are available in Appendix Table H.12. The dependent variable in Columns (1–3) and (6–9) is the height-for-age z-score. In Column (4), it is the prevalence of stunting, defined as a height of two standard deviations or more below the median; in Columns (5) it is the prevalence of severe stunting, defined as a height of more than three standard deviations below the median. Controls include an indicator for urban/rural status in all columns, a quadratic in the mother's age at birth, an indicator for gender, birth-order indicators, as well as indicator variables for the child's age. For consistency across models, the sample is restricted to those observations where the operating status of the mine is known both in the birth year and during gestation (this removes about 3% of observations). Standard errors are clustered at the mine level. Significant at * 10%, ** 5%, *** 1%.

(Hoddinott et al., 2011).[24]

We next note that, in the case of the continuous index and of stunting, the effect of *in utero* exposure is larger and stronger when we estimate it for infants only (Table 10, Column (singular) 3; See Appendix Table H.12, Columns 5–6 for stunting results). This points either to a balancing effect – perhaps due to household wealth – in older children, or a spontaneous attenuation of *in utero* impacts with time. We shed some further light on this question by studying the effect of different exposure patterns. Thus, the estimated effect of exposure during the first year of life alone is centered near zero (Column 6). Results when estimating *in utero* and birth-year effects jointly are more instructive. We find robust and large adverse effects of *in utero* exposure on the continuous index (0.5σ), alongside beneficial effects of birth-year exposure (Column 8). This is at least consistent with exposure to maternal lead loads *in utero*, alongside positive effects from the socio-economic benefits of mining, once the child is born. It points less toward a mere attenuation of impacts. While it is attractive to allow *in utero* and birth-year effects to jointly enter into the model, the sample of children born just before and just after a mine opened or closed is small (conversely, operational status is highly serially correlated).[25] To further solidify the result, we therefore show in Appendix H that a similar pattern emerges when we first estimate separately the effect of the mine operating during the survey year (Appendix Table H.12, Column 9), and then compare this estimate to the one obtained when we include also the effect of the mine operating during gestation (Column 10).

Finally, when we estimate the effects of *in utero* and birth-year exposure with mother-level effects, the results match the pattern in the mine-level panel, but do not reach significance (Columns 2, 7 and 9 in Table 10). This is perhaps to be expected: although we observe more than 2,000 women near mines in our sample for whom our data records child growth outcomes for at least two children born within five years of each other, there are few mothers with recorded births both while the mine was operational and while it was not operational.

6. Conclusion

We present a systematic empirical assessment of the health-wealth trade-off facing mining communities, using micro-data from 44 developing countries. Communities exposed to mining enjoy important economic benefits in the medium and long term. Benefits are strongly concentrated within the immediate vicinity (5 km) of mines, and we find no effects beyond 20 km. Wealth rises quite evenly across the distribution, with modest increases in inequality in the long run. Yet, the evidence reveals that the real economic benefits generated in mining communities go hand in hand with serious health impacts. The incidence of anemia increases by three to ten percentage points in adult women. The ability to recover hemoglobin levels after blood loss due to pregnancy and delivery is particularly impaired. There is weaker but consistent evidence of hematologic toxic effects in children. Young children exposed to a mining environment *in utero* are more likely to be stunted or severely stunted than those born in control groups, with an increase in incidence of five percentage points. However, there may be a compensatory positive growth effect of living in mining communities after birth. These health impacts are consistent with exposure to lead contamination, and have previously been observed at body burdens of lead that are known also to cause cognitive deficits in children. By way of contrast, there is no general pattern of ill health in mining communi-

[24] Appendix H reports that we observe an effect on birth weight in the mother-level panel, but lack corroborating evidence from our other models. Prior studies have observed that adverse conditions *in utero* can impair long-run well-being without being reflected in birth weight (Schulz, 2010).

[25] The DHS surveys record only health data from children born no more than five years before the survey time. This helps identification, but limits sample size, in particular where we use mother-level effects.

ties. DiD tests leveraging pollutant characteristics and pathophysiological patterns show that the results are likely to be due to pollution, and unlikely to be influenced by residential sorting.

Our estimates of health cost and wealth benefits are not directly comparable without strong assumptions. However, we have argued above that both the observed wealth gains and the health costs are certainly sizeable. Whether costs or benefits dominate is likely to depend on the persistence of economic benefits, as well as on whether there are legacy effects of pollution after operations cease. Because there are wealth gains across the distribution, while relatively small population groups are affected by overt health conditions, the decision to live in mining communities is a risky choice.

Regardless of the exact balance of the trade-off, it is remarkable that adverse *compensated* health impacts persist among a generally wealthier population. One potential explanation would point to a high cost of avoiding exposure to pollution, perhaps due to the structure of settlements or the quality of public transport. The decision on living in mining towns in developing countries might resemble less the choice of an optimal distance along a continuum, and more a discrete choice between living in relatively unpolluted communities outside of a reasonable commuting distance to the mine, or in a highly polluted but bustling community adjacent to the mine. This conjecture is consistent with the high spatial concentration of medium-term economic benefits, and the fact that we observe the greatest wealth effects near mines in environments that are economically less active. An alternative explanation might point to limited information. Pollutant levels near mines vary greatly over small distances (van Geen et al., 2012), and contamination is not always easily observed. In addition, the health impacts of pollution may not be widely known. Despite their higher wealth, residents of mining communities are not making very decisive health investments to compensate for exposure to pollution: health care is only weakly better. We also find no differences in wealth across mine types, and hence, no *prima facie* indication of the kind of compensating wage differential one might expect if residents were widely aware of health risks.

Certainly, from a policy perspective, the presence of adverse compensated health effects suggests that the regulation and management of mining pollution deserves renewed scrutiny. We offer three leads for effective interventions. One, health concerns are most acute in the immediate vicinity of mines. At least for some countries in our sample, there may be a case for experimentation with programs to improve public transport, road infrastructure, flexibility in local housing markets, and engineering solutions to contain or remedy pollution, all with the goal of allowing residents to live away from the worst pollution. Secondly, residents may not have full information on pollution and health risks; interventions could remedy this. It is tempting to posit that the uneven distribution of health costs and spatial variation in pollution around mines might be causally related. If so, then *testing* of pollution levels in residential areas might enable residents to avoid the most dangerous sites, at a comparatively low cost. Finally, life in mining communities appears to offer substantial benefits in the expectation – with a considerable downside risk. This is a good fit for insurance, however formalized.

Appendix A-I. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jdeveco.2018.05.005.

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