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A Country-Level Panel Data Analysis

Rodrigo Moreno-Serra and Peter Smith
Business School & Centre for Health Policy,
Imperial College London, United Kingdom

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Abstract

The debate around universal health coverage (UHC) and the best ways to achieve it has come under the spotlight lately. The 2010 World Health Report reinforced calls for a push towards UHC across countries, broadly defined as providing all people with access to needed health services of sufficient quality to be effective, without financial hardship associated with their use. Although the potential link between health system coverage and population health has played a crucial role in that debate, the expected relationship between population outcomes and different proxies for system coverage is ambiguous in theory, with much of the available empirical evidence mirroring such indeterminacy.

The main goal of this study is to contribute to the UHC debate by providing robust empirical evidence on the causal link from national levels of health system coverage to population outcomes. We assemble a large panel dataset available at the cross-country level, with annual data for the period 1995-2008 encompassing 153 developing and developed countries. We measure the level of health system coverage through indicators of pre-paid (pooled) public and private health expenditure and immunization rates, to try to capture effective access to needed care and protection from financial hardship due to health payments. Population health is measured by the under-five mortality rate and female and male adult mortality rates. We use a two-step instrumental variables approach that directly estimates the reverse causal effects of under-five and adult mortality on coverage indicators, so as to explicitly adjust for these impacts when estimating the effects of health coverage on mortality outcomes. We subject this model to a battery of specification and robustness tests, and also examine differential effects of the coverage variables according to country income levels.

Taken together, our results strongly indicate that expansions in health system coverage lead, on average, to improved general population health. Higher government health spending per capita is consistently found to reduce both child and adult mortality rates. The estimated gains are the largest when under-five mortality is examined and are larger for low and middle income countries than in the full sample. Based on the results for under-five mortality and public health spending, the implied marginal cost of saving a year of life is just around US\$1,000 in the full sample of countries. For the average country, pre-paid public spending seems more effective in reducing mortality than pre-paid private insurance funds. Higher immunization coverage is also found to decrease mortality rates. Thus, our study offers hard evidence that investing in broader health coverage can generate significant gains in terms of population health.

1. Introduction

Adequate access to the highest attainable standard of health for every citizen has been recognized as a fundamental human right and a central component in reversing socio-economic and health system inequities (Backman et al. 2008). Most of a country's population stands to benefit from improved access to health care, encompassing aspects such as timely use of preventive and curative services, and reduced risk of impoverishment due to health costs (e.g., through a higher participation of pre-paid funds in health financing) (World Health Organization 2010a). Yet the gains from better access to health care are likely to be even larger for the poorer and less healthy individuals who are, in general, at a higher risk from being deterred from seeking timely medical care, and less able to cope with uncertainty about health care needs than the rich through insurance and credit mechanisms. Indeed, it has been estimated that most of the 1.3 billion poor citizens around the world have restricted access to health services due to inability to pay (Preker et al. 2003).

From the above, it has often been concluded that expansions in health system coverage must be linked to improvements in population health outcomes (cf. e.g., Commission on Macroeconomics and Health 2001; World Health Organization 2008, 2010a). While it is acknowledged that many of the potential channels to make individuals healthier are beyond the remit of health systems (formal education and housing being prime examples), enhanced access to affordable health services through a well-functioning health system is generally regarded as fundamental for achieving better population outcomes. In this context, there have been repeated calls in the international community for countries to take concrete steps towards achieving universal health coverage, broadly defined as providing all people with access to needed health services of sufficient quality to be effective, without financial hardship associated with their use (Shengelia et al. 2003, World Health Organization 2010a). Effective access to care, higher prominence of financial risk pooling, and higher levels and shares of pre-paid health spending are regarded as key dimensions of extended health system coverage. The challenges to reach universal health coverage have received great attention as of recently, with the WHO 64th World Health Assembly resolution recommending that the topic be further discussed at the United Nations General Assembly, in light of the push to achieve the Millennium Development Goals (World Health Organization 2011a).

Although the potential link between system coverage and population health status has played a crucial role in the aforementioned debate, the expected relationship between health outcomes and system coverage—measured either by pre-paid spending (total, public or private) or health service utilization—is ambiguous *a priori*. An increase in government health expenditure (which normally corresponds basically to pre-paid funds and makes up the majority of national health spending¹) may be accompanied by a commensurate reduction in private health expenditures, leading in turn to no changes in total spending and potentially no significant changes in health status. Even if government spending increases the total amount of resources devoted to health care,

¹ Government expenditure on health represented on average 60% of national health spending in 2008 (average for 192 countries; World Health Organization 2011b).

positive consequences in terms of population outcomes may not arise if the additional funds are spent mainly on low productivity inputs (e.g., better tertiary care when the real gains are in extended primary care) or services without complementary network (e.g., more hospitals and clinics when no roads are available) (Wagstaff and Claeson, 2004).

In a similar vein, an increase in the overall capacity to pay privately for health care may serve as an incentive for governments to implement or expand cost-sharing arrangements while public funds previously allocated to health are diverted to other sectors, with no real changes in service provision. More private spending may also mean that more households are at risk of falling into poverty due to medical payments, with resulting worse health outcomes in the future (for example, if individuals wait longer to be seen by a doctor when ill). But incremental private spending may also translate into improved access to care—or better quality care—through a more widespread use of insurance mechanisms, potentially leading to health gains (Levy and Meltzer 2004).

Therefore, the realization of any health benefits from higher expenditures is likely to depend, among other factors, on how well targeted the extra spending is in terms of enhanced access to care. Having more people covered for a broader set of services with lower cost-sharing should lead to improved access to and utilization of health services. If such services are effective, extended access and use should in principle generate aggregate health gains. Nonetheless, the magnitude of these aggregate gains will probably depend on aspects such as the identity of beneficiaries: for instance, if in poorer countries access increases only for smaller groups of richer people, there is no reason to expect substantial gains at the population level. Also, incremental coverage for lower productivity services (e.g., high-tech equipment in specialist hospitals) might result in small health gains measured at the aggregate level, if any.

The empirical literature on the issue mirrors the indeterminacies just described. Much of this research (especially in the early 1990s) has focused on identifying simple correlations between public health expenditures and health outcomes in cross-country data, finding no systematic evidence of an effect on mortality indicators such as child death rates (Musgrove 1996). The same is true of a more rigorous empirical study by Filmer and Pritchett (1999), which finds at best small public spending impacts on under-five and infant mortality.

More recently though, a few econometric studies using panel data have found evidence of increased public spending leading to better child and maternal mortality outcomes. Wagstaff and Claeson (2004), examining data for up to 120 countries, generally find statistically significant beneficial effects of government spending (as a proportion of GDP) on under-five and maternal mortality. For under-five deaths, the authors estimate reductions ranging between 0.8-1.5% in mortality for a 10% higher share of government health expenditure. Bokhari et al. (2007), for their sample of 127 countries, estimate that a 10% increase in government health expenditure per capita leads to average reductions of 3.3% and 5% in under-five and maternal mortality rates, respectively. It has also been found that the positive health effects from increased public spending tend to vary across population groups and countries, with poor people in poorer countries benefiting the most from additional public health spending (Bidani and Ravallion 1997). On the other hand, the limited empirical evidence on the links

between private spending and health outcomes (coming from individual country studies) normally indicates an association between higher out-of-pocket (OOP) health payments and worse health outcomes, whereas higher spending in private insurance has been linked with better self-perceived health status in a few countries like China (cf. e.g., Gotsadze et al. 2005, Wang et al. 2008).

There is a growing econometric literature examining the relationship between health outcomes and extended health services coverage through public and private insurance arrangements, again mostly in individual countries rather than from an international perspective. One general conclusion that emerges from this research is that the effects of expanded coverage on population outcomes depend largely on the group of countries analyzed. For richer countries, most of the empirical literature finds improvements in health status due to broader health insurance coverage. This is the case, for example, in the USA, where a number of studies (reviewed in Gruber 2009) have linked better adult and infant health outcomes to the implementation and expansion of the Medicare and Medicaid schemes. The evidence is less conclusive for poorer countries. Among the several studies reviewed in Escobar et al. (2010), almost all find that expansions in coverage of outpatient and inpatient services through public or private health insurance raise utilization and reduce the incidence of catastrophic health expenditures. Yet these do not seem to clearly result in improved mortality or morbidity, measured by a wide range of objective and self-assessed health indicators.²

Since *a priori* the link between health system coverage and population outcomes seems ambiguous, establishing the direction of these impacts in order to guide policy-making becomes essentially an empirical matter. To ascertain said relationship from an empirical perspective is not, however, a trivial task, and many of the studies mentioned above exhibit important methodological limitations. The first big concern is the potential endogeneity of the indicators used to measure health coverage, for instance health expenditure aggregates. It seems possible that countries with unobserved factors causing poor health outcomes might decide to broaden health system coverage via higher government spending to deal with the problem, potentially leading to a spurious correlation in the data between higher public spending and worse population health. Another issue is that public investment in health may take some time to yield benefits, particularly if health status is measured through indicators such as overall mortality. The contemporaneous impacts of extended coverage might then be smaller than the accumulated effects over time.

Most of the empirical literature previously described is not well equipped to address these points. Simple ordinary least squares (OLS) regressions will probably identify only correlations in the data. Even when more sophisticated methods have been used (such as instrumental variable estimators, e.g., Bokhari et al. 2007), the analyses have normally relied on cross-sectional data and focused on specific countries or insurance interventions. These aspects make it difficult for the analysis to rule out the influence of reverse causality and unobserved factors, as well as to account for the time frame of coverage effects or generalize the empirical findings to other settings.

² Wagstaff and Moreno-Serra (2009) also find little cross-country evidence that the introduction of social health insurance during the 1990s and early 2000s in the countries of Central and Eastern Europe and Central Asia led to population health improvements.

The main goal of this study is to contribute to the universal health coverage debate by providing econometric evidence on the direction and magnitude of coverage effects on mortality outcomes at the national level. We do so by using a large panel dataset at the country level, with annual data for a period of 14 years (1995-2008), and various instrumental variable specifications allowing for potential reverse causality in our data and unobserved country-specific characteristics. As discussed previously, health system coverage is a broad concept that has become commonly associated with two basic elements: effective access to needed care and protection from financial hardship due to health payments, through pooling and pre-payment mechanisms. We therefore employ a number of health care financing and access indicators as proxies for the level of coverage in a health system, and quantitatively examine their effects on three measures of mortality. Our results (summarized for convenience in Table 1) indicate that expansions in health system coverage through higher government spending per capita and immunization rates result in lower child and adult mortality rates, with the positive impact of pre-paid health funds on child mortality being larger in poorer countries.

This paper is organized as follows. Section 2 outlines our econometric methodology. Section 3 describes the dataset used in the analyses. Section 4 presents the results of our main estimations and a number of specification and robustness checks. Section 5 presents a discussion of our empirical results and concludes.

2. Methodology

It is difficult to credibly estimate the effects of health system coverage on population outcomes through a cross-sectional econometric analysis at the country level. This is because there may be unobservable variables that are correlated with the level of coverage in a country *and* the outcomes of interest. This potential *endogeneity* of coverage indicators would lead to biased estimated coefficients if not taken into account in the regressions. One way to address the problem of omitted variables is to take advantage of longitudinal country-level data and look at *changes* over time in health coverage, so as to eliminate the effect of time-invariant omitted variables. Even in the latter situation, however, coverage measures may still be endogenous if (a) there are time-varying omitted country characteristics correlated with coverage levels and health outcomes; or (b) there is reverse causality or simultaneity in the relationship between coverage and outcomes (e.g., if governments respond to changes in population health by adjusting health coverage levels); or (c) there are larger systematic measurement errors in the reporting of coverage and health outcomes for particular countries, for example in lower income nations. In what follows, we describe the various strategies used to deal with these potential confounders of the relationships of interest.

2.1 *Basic fixed effects models and reverse causality*

Consider a simple model where the population health outcome of interest in country i at time t , y_{it} , depends on a vector C_{it} containing indicators of the level of health system coverage, and a vector X_{it} of covariates that might potentially influence both the outcome and the level of health coverage. As described in the next section, we measure

population health through mortality rates and coverage through health spending and immunization rates. This simple model can be written as:

$$(1) \quad y_{it} = \beta C_{it} + \delta X_{it} + e_{it}$$

where e_{it} is an error term capturing unobservable variables and random noise. We can decompose the error term in equation (1) into three components that account for different sources of endogeneity. Let the error term be denoted as:

$$(2) \quad e_{it} = \alpha_i + \theta_t + \varepsilon_{it}.$$

In the formulation above, α_i is a country-specific effect which captures time-invariant unobservables that are potentially correlated with the levels of coverage and population health observed in a given country. The term θ_t is a period-specific intercept that captures aggregate shocks affecting health outcomes in all countries at the same time. If all potential sources of endogeneity have been accounted for in the previous two components, ε_{it} represents a random, idiosyncratic error component. This formulation of the error term leads, by substituting equation (2) into (1), to the following model:

$$(3) \quad y_{it} = \beta C_{it} + \delta X_{it} + \alpha_i + \theta_t + \varepsilon_{it}.$$

This model can be estimated through a fixed effects approach: in this case, the within-country variation in outcomes and regressors over time is used to obtain the coefficients of interest and the estimating equation can be written as:

$$(4) \quad y_{it} - \bar{y}_i = \beta (C_{it} - \bar{C}_i) + \delta (X_{it} - \bar{X}_i) + \xi_t + (\varepsilon_{it} - \bar{\varepsilon}_i).$$

Changes in outcomes and regressors over time are computed with reference to the corresponding within-country means during the period of analysis ($\bar{C}$ and $\bar{X}$). Since the country-specific effect α_i is fixed over time, the effects of time-invariant unobservables captured by α_i are eliminated in the estimation. If the endogeneity of health coverage is entirely due to omitted time-invariant country factors, equation (4) can then be estimated by pooled OLS.³

Problems arise if our variables of interest, the coverage indicators contained in C_{it} , are correlated with the error term even after sweeping away unobserved time-invariant country factors. In this case, estimating equation (1) with fixed effects by pooled OLS would lead to biased estimates of the β coefficients. This would happen, for instance, if countries with unobserved characteristics that lead to higher-than-expected levels of mortality deliberately choose to extend system coverage in order to tackle such problem (say, through additional public spending). In our study, we adopt a two-step instrumental variables (IV) approach that seeks to estimate any reverse causal effects of mortality outcomes on coverage indicators, so as to directly adjust for these impacts when estimating the effects of health coverage on mortality. This methodology to deal with the simultaneous determination of dependent variable and regressors has been

³ Fixed effects estimations with longitudinal data require the estimated standard errors to be adjusted for arbitrary types of serial correlation and heteroskedasticity (Bertrand et al. 2004), so in the empirical analyses we use panel-robust standard errors clustered at the country level.

proposed and applied in a different context by Brückner (2011) but has not, to the best of our knowledge, been used to analyze the research questions in our paper.

2.2 *Dealing with reverse causality: an instrumental variables approach*

Step one: estimation of the (reverse) causal effect of health outcomes on coverage

Let the relationship between mortality and coverage be expressed as:

$$(5) \quad C_{it} = \lambda y_{it} + \phi X_{it} + \gamma_i + \omega_t + \mu_{it}.$$

As before, although country-specific and time-specific effects are included in the model, endogeneity due to time-varying omitted variables and measurement error may also be present when health coverage is examined as a function of mortality. So we use an IV approach that addresses these endogeneity concerns to consistently estimate the effects of our mortality outcomes on health coverage. An unbiased and consistent IV estimator requires that we identify one or more variables Z_{it} —the instruments—that are sources of exogenous variation in the mortality outcome y_{it} . In other words, the instruments must be: (i) *external* to mortality and coverage, that is, they must not be affected by our mortality and coverage measures; and (ii) *orthogonal* to coverage, that is, they must have an effect on health sector spending and immunization rates only through their effect on the health outcome (and not have by themselves a direct effect on coverage). If the identified instruments are valid as per the previous criteria, and *relevant* in the sense of being reasonably correlated with the instrumented mortality indicator (assumptions that can be statistically tested), they should allow us to obtain unbiased regression coefficients from an IV estimation of equation (5). This can be done through two-stage least squares (IV-2SLS) in the case of a single instrument, or using the more efficient two-step generalized method-of-moments (IV-GMM) estimator in the case of more than one instrumental variable (Cameron and Trivedi 2005).

We use two variables as instruments for the country's mortality level in a given year. The first is annual CO2 emissions per capita, given that higher outdoor air pollution has often been linked to a higher incidence of some diseases and higher levels of mortality (see, for example, Prüss-Üstün and Corvalán 2006). The second instrument is the annual number of battle-related deaths in internal or international conflicts for each country. Conflict deaths typically represent a small fraction of national mortality rates in our sample (see next section), but will tend to be more correlated with the latter in those regions where civil and international wars have been more frequent during our period of analysis, notably Sub-Saharan Africa and the Middle-East.

The assumption we make is that differences in both CO2 emissions and conflict deaths will induce variations in population health and measured mortality rates across countries and within countries over time, which will in turn—and only through health outcomes—trigger a response in terms of coverage indicators such as health care expenditure and immunization rates. Intuitively, it seems reasonable to think of CO2 emissions and battle-related deaths as uncorrelated with our coverage measures except by affecting health outcomes. But we also statistically test for the exogeneity and relevance of the two instruments we use. Their relevance is assessed through F tests of joint insignificance of the two instruments in the IV regressions, and through a χ^2 under-identification test proposed by Angrist and Pischke (2009) which accounts for

the clustered structure of the error terms in the estimated equations. A cluster-robust version of Hansen’s over-identification J test is used to check the exogeneity of the instruments in the estimated models (Cameron and Trivedi 2005).

For each of our mortality and coverage measures, we use IV-GMM on equation (5) to obtain unbiased estimates of the reverse causal effect of mortality on coverage, $\hat{\lambda}$. By inspecting the sign and statistical significance of $\hat{\lambda}$, we can infer the direction of the bias that would affect the coefficients of our coverage variables if we used a simple least squares fixed effects estimator on equation (3). A positive $\hat{\lambda}$ would mean that higher mortality causally leads to higher coverage, thus resulting in an estimated coverage coefficient β in equation (3) that is biased upwards, i.e. closer to zero or “more positive” than its true value. Conversely, a negative $\hat{\lambda}$ would imply a negative causal effect of mortality on coverage, leading to a fixed effects estimate of the effect of coverage in equation (3) that is biased downwards.

Step two: estimation of the causal effect of coverage on health outcomes

Our empirical strategy is to use the results from the consistent estimation of (5) to avoid the potential biases described above. This is done by estimating equation (3) through an IV procedure that expunges the reverse effect of mortality on coverage measures. We use the IV mortality coefficients estimated from equation (5) to construct adjusted series of coverage indicators C_{it}^* for each country, subtracting the effect of mortality on each coverage indicator:

$$(6) \quad C_{it}^* = C_{it} - \hat{\lambda} y_{it}.$$

We then use C_{it}^* as an instrument for the corresponding coverage indicator in equation (3). Since this procedure leads to only one constructed instrument for each coverage measure, model (3) is estimated through an IV-2SLS approach. We formally examine the relevance of the generated instruments through the LM version of the under-identification test proposed by Kleibergen and Paap (2006).⁴ The IV estimator will be free from any reverse causality bias, and the instruments valid, by construction. This IV procedure should in principle be better equipped than the fixed effects estimator to account for the confounding effects of omitted variables and measurement error in our context (all the IV models estimated in this paper include country and time fixed effects). Even if bias due to reverse causality is not present in some models, our IV estimator will still offer unbiased and consistent estimates of health coverage effects in these cases, provided the instruments are valid.⁵ Therefore, in the Results section, we present the estimates from the IV estimation of equation (3) as a preferred approach,

⁴ The Kleibergen-Paap LM test is a more appropriate alternative of joint under-identification test for the case of two or more instrumented regressors in each equation, as in the estimation of equation (3). Of course, since the model is exactly identified (only one instrument available per endogenous regressor), it is not possible to perform Hansen exogeneity tests of over-identifying restrictions.

⁵ This will, however, come at the cost of inflated standard errors compared to basic least squares fixed effects estimation.

and compare those with the results obtained using the basic fixed effects estimator on the same equation.

3. Data

3.1 *Period of analysis, definition of variables and sources*

The definitions and sources of all the variables used in our empirical study are given in Table 2. We use publicly available annual data at the country level from three databases: World Bank's *World Development Indicators* (World Bank 2011a), World Health Organization's *Global Health Observatory* (World Health Organization 2011b) and Institute of Health Metrics and Evaluation's *Global Health Data Exchange* (Institute of Health Metrics and Evaluation 2011). The period of analysis is 1995 to 2008, although for adult mortality rates the information for many countries is available for a slightly more restricted period (from 1998-99 onwards). In total, the dataset includes data for 153 different countries.

3.2 *Health outcomes*

We measure population health status through three mortality indicators. Our dataset contains yearly data on under-five mortality rates (deaths per 1,000 live births), and female and male adult mortality rates (deaths per 1,000). The indicators available can be regarded as measures of the overall performance of a health system, after controlling for other factors such as socio-economic and demographic characteristics. Under-five mortality tends to react relatively quickly to improvements in access to and quality of health care provision, having been selected by the international community as a key outcome within the Millennium Development Goals framework (United Nations 2011; World Health Organization 2008).

Table 3 presents averages, standard deviations, minima and maxima of the outcome variables for the countries in our estimation sample, during the period of study. The descriptive statistics are presented both for the full sample and only the sub-sample of country-year observations with GDP per capita up to \$12,195 (the World Bank's threshold below which countries are included in the low and middle income—LMIC—group; see World Bank 2011b).⁶ Unsurprisingly, mortality indicators tend to be worse in the LMIC group compared to the averages in the full sample that includes high income nations, with substantial data dispersion for all outcomes both in the full and LMIC samples.

3.3 *Health system coverage variables*

We attempt to capture the different dimensions of health coverage (as defined in the Introduction) through measures of the level and proportion of pre-paid (pooled) funds in health care financing, and the actual provision of health services. The first indicator

⁶ The World Bank uses Gross National Income (GNI) per capita to classify countries. Here, we apply the same cut-off points to our sample of countries but using GDP per capita (purchasing power adjusted) instead.

is government health expenditure per capita.⁷ Public spending represents the majority of health resources in most countries and is basically made up of pooled funds paid by citizens before the need for medical care, through channels such as social health insurance contributions or taxes. All else equal, higher levels and shares of pooled health resources tend to make individuals more likely to seek care when needed and increase protection against financial hardship caused by medical payments (Preker et al. 2003, Wagstaff 2009). Hence, government spending serves as a proxy for the extent of health system coverage. We also include an indicator of privately pooled health resources, voluntary health insurance (VHI) spending per capita. Out-of pocket (OOP) private health spending has been shown to be positively and highly correlated with poorer financial protection, as measured by the incidence of catastrophic health expenditure across countries (Xu et al. 2007). Since financial protection is a key dimension of health system coverage, in our regressions we use either OOP health spending per capita (baseline models) or OOP health spending as a share of total health expenditure (robustness checks) as additional system coverage proxies.

We aim at further capturing the impact of effective access to care—another basic dimension of health coverage—through a constructed (aggregate) immunization indicator. In our dataset, annual cross-country information for the period of study is available for six immunization rates: diphtheria-pertussis-tetanus (DTP3), hepatitis type B, haemophilus influenzae type B, polio, BCG and measles. Since the coverage figures for these six vaccines are highly correlated in our sample (strongly significant correlation coefficients of about 0.60 on average), including each immunization variable separately in the regressions would introduce a high degree of multicollinearity and unnecessarily inflate standard errors. We therefore summarize the information provided by these six indicators by constructing an aggregate immunization rate variable, representing the median rate across the six immunization categories for a given country in a given year. Although more directly linked to the primary care sector, immunization rates are widely used as markers of the overall performance of health care systems in guaranteeing access to services (World Health Organization 2010a).⁸

The descriptive statistics for our health coverage variables (Table 3) show an average government health spending per capita of \$599 in the whole sample, around three times larger than the average OOP spending per capita (\$188). OOP spending represents over a third of national health financing on average in the full sample. On the other hand, VHI spending is not a large component of health financing in the vast majority of countries in the sample, totaling less than \$60 per capita on average (and only 4% of total health expenditure). Immunization coverage figures reach 86% on average in the full sample. Low and middle income countries exhibit lower immunization rates (83%) and substantially lower per capita health spending figures compared to the corresponding full sample averages, with a higher participation of OOP payments in total health spending (40%) and very small annual VHI expenditure (\$14 per capita).

⁷ All expenditure figures used in this paper are in constant 2005 international (purchasing power parity adjusted) dollars.

⁸ Ideally, we would have liked to include other access and utilization measures in our analyses, in particular indicators such as coverage of outpatient services at health centers and hospitals. Unfortunately, indicators usually employed in cross-sectional country comparisons (e.g., births attended by skilled personnel and outpatient visits) are not available as a usable time-series for most countries.

3.4 *Instruments and other covariates*

We use two variables as instruments for the observed mortality rates in each country. The first is annual CO2 emissions per capita in metric tons, whose average in the full sample (5.2 tons) is more than twice as higher than in the LMIC group. As expected, the second instrument used in the IV models, annual battle-related deaths in conflicts, exhibits a higher average in the LMIC sub-sample than in the full sample. The averages are still relatively low, however, and are particularly driven by the occurrence of conflicts in low income nations over the period of study.

The remaining covariates used in the econometric analyses attempt to control for observed cross-country heterogeneity in terms of national income (GDP per capita, purchasing power parity adjusted), levels of formal education (the primary education enrolment rate) and demographic profile (shares of population aged 0-14 and over 65). These variables act as constraints for what countries can achieve in terms of population health and should pick up the main effects of epidemiological and socio-economic differences across countries. Table 3 shows that countries have a GDP per capita of over \$12,100 in our sample, although there is huge variation in the data with a minimum value of \$280—the average in the LMIC group is \$4,237—and other countries with per capita income well above the sample average. As explained in the Methods section, all models estimated in the paper also include a full set of year-specific indicators (time dummies) to capture the effects of common aggregate shocks during the study period.

4. Results

4.1 *Preliminary analyses: what the raw data tell us*

We first examine the raw relationships between the levels of mortality outcomes and two indicators of health coverage, government health expenditure per capita and immunization coverage (Figure 1). The comparisons are made for the beginning and end of the study period. To increase the number of observations in the plots (there are about 115 countries with available data for most of the period, compared to 61 countries on average between 1995-98), we use five-year average values between 1995-99 as the initial data points, and average values between 2004-08 as the final data points. To save space, the graphs presented refer only to under-five mortality rates against government health expenditure (Panel A) and immunization coverage (Panel B). The red line in the graphs is for the non-parametric regression of under-five mortality on the corresponding health coverage indicator, with no other covariates.

The graphs in Figure 1 show that the expected unadjusted relationships hold for both the initial and final periods. For most of the distribution, broader health system coverage—i.e. higher government spending and immunization rates—is associated with lower under-five mortality. Similarly, from graphs not shown, observed adult mortality rates (female and male) tend to be lower for higher levels of government spending and immunization coverage. As for the private health spending indicators, higher out-of-pocket expenditure per capita is also associated with lower mortality in the raw data, while there is no clear bivariate relationship between mortality outcomes and VHI per capita spending arising from the graphical analyses.

Since the variation used to estimate coefficients in regressions with country-specific effects (such as fixed effects models) comes from within-country *changes* in health outcomes and coverage indicators over time, we also examine raw correlations between changes in our health coverage measures between 1995-2008 and changes in outcomes. We divide countries into terciles of increase in each coverage indicator, where the bottom tercile comprises those countries with the smallest increases in coverage during the period 1995-99 to 2004-08, whereas the top quartile contains those countries with the largest increases in coverage in the same period.

Figure 2 compares the evolution of under-five mortality rates across the bottom and top tercile groups of increase in government health spending (Panel A) and immunization coverage (Panel B), over the period 1995-2008. These unadjusted analyses show that countries in the bottom tercile of increase in government expenditure per capita actually exhibited *faster* decreases in under-five mortality. Moreover, female and male adult mortality rates do not seem to have followed different paths according to changes in health spending (graphs not shown). By contrast, under-five mortality—and adult mortality—improved faster over the period in countries with the largest expansions in immunization rates. From graphical analyses not shown, adult mortality rates seem to have followed roughly parallel trajectories over the period across countries in the top and bottom terciles of increase in private spending per capita, either OOP or VHI, whereas lower increases in OOP and VHI health expenditure per capita are associated with faster decreases in under-five mortality.

What to expect then from the econometric regressions in light of our examination of the raw changes in outcomes vis-à-vis health coverage? According to the above results, the relationship between health coverage and mortality seems somewhat ambiguous. Larger expansions in immunization coverage seem to be associated with better mortality outcomes in the unadjusted data, although of course it remains to be seen whether this relationship will persist once observable and unobservable differences between countries are taken into account in the regressions. Also, while in the raw data larger increases in government health expenditure per capita seem to have little to do with improvements in mortality, it is noteworthy that the group of countries with the largest increases in government spending is formed mainly by high income countries, which started the period at a much lower baseline in terms of mortality levels compared to countries in the bottom tercile of increase in spending. This might explain to some extent why countries with the smallest increases in government spending—basically lower income countries for which a dollar of extra public spending might yield higher marginal returns in terms of outcomes, compared to richer nations—seem to have achieved faster improvements in under-five mortality over the period. This result points therefore to the importance of adjusting the coverage-outcome relationships for differences in national income, as well as examining the possibility of different impacts of health coverage expansions according to national income levels. It also draws attention to the potential influence of time-invariant country-specific characteristics such as initial population health levels in analyses of this type. Our IV regression models attempt to formally deal with these observable—and other intrinsically unobservable—elements that may influence the relationships of interest.

4.2 Main regression results

The causal effect of health outcomes on coverage

The first step of the IV approach outlined in section 2.2 involves the estimation of the reverse causal effects of mortality outcomes on each of our health spending and immunization indicators. For each coverage measure as dependent variable, we estimate three separate versions of equation (5)—each having one of the mortality rates as regressor of interest, plus the remaining education, income and demographic covariates—using an IV-GMM procedure. In each equation, the corresponding mortality rate is instrumented by our two instruments, CO2 emissions and conflict deaths.

The IV models perform well in this first step. The full results are presented in Table A1 in the Appendix. According to the diagnostic tests, the instruments are relevant to predict mortality outcomes in the first stage of the IV procedure: for all models, the F and χ^2 tests reject both the joint statistical insignificance of the instruments and first stage model under-identification at conventional levels. Furthermore, Hansen over-identification tests cannot reject the null hypothesis of exogenous instruments by a large margin, providing statistical support to more intuitive arguments concerning the validity of our chosen instruments.

Overall, the response of our coverage indicators to changes in mortality rates does not appear to be substantial. The estimated mortality coefficients (the $\hat{\lambda}$ from equation (5)) generally fail to be statistically significant at conventional levels. However, in one instance—the effect of under-five mortality variations on government health spending—the point estimate comes close to statistical significance. It implies that an increase of one-standard deviation in under-five mortality (48.9 deaths per thousand) leads to a governmental response of around \$0.25 increase in health spending per capita. For comparison purposes, this spending effect is roughly twenty-five times larger than the measured governmental response to one-standard deviation increase in adult male mortality (around \$0.01 per capita). The positive sign and p-value of the estimated under-five mortality coefficient—as well as the remaining mortality coefficients across all models, with only two exceptions—implies the possibility of obtaining (non-IV) fixed effects estimates of the impact of coverage measures on mortality that are biased towards zero or positive values. We compare both sets of estimates below.

The causal effect of coverage on health outcomes

We now present in Table 4 the results from the estimation of equation (3) for each mortality outcome. For comparison, the first column for a given outcome shows the results of the basic fixed effects approach, whereas the second column shows the corresponding results for the preferred IV approach. The latter are obtained by instrumenting each of the coverage indicators in the regression by their generated, reverse causality-adjusted counterpart obtained from the first IV step above. So, for example, column 2 presents the IV-2SLS estimated effects of coverage measures on under-five mortality, where the instrument for government health spending is constructed as in (6) using the corresponding λ coefficient estimated in the previous

sub-section, and so forth for the other spending and immunization variables. In all instances, the statistical tests support the relevance of the generated instruments, strongly rejecting under-identification in the first stage of the IV estimations.

The focus here is on the estimated effects of our proxies for the various dimensions of health system coverage: government, OOP and VHI health spending per capita, as well as immunization coverage. In addition to the variables included in the tables and country-specific effects, all regressions control for GDP per capita, the primary education enrolment rate, the share of population aged 0-14, the share of population aged over 65 and a full set of year dummies.⁹

Public health spending. The IV results strongly indicate that higher levels of government health expenditure per capita lead to better population outcomes, measured either by under-five or adult mortality rates. The point estimates, all statistically significant at the 5% level, imply economically important effects: a \$100 increase in government spending per capita results in a reduction of 13.2 per 1,000 in the under-five mortality (Table 4, column 2), as well as decreases of around 2.6 and 2.2 per 1,000 in the adult female and male mortality rates, respectively (columns 4 and 6). In all cases, the IV point estimates are larger in size than the equivalent fixed effects estimates. The most noteworthy case is that of under-five mortality, the outcome for which there was somewhat stronger evidence of reverse causality arising from the first step of the IV modeling. While the fixed effects estimator suggests a counter-intuitive, positive and statistically significant impact of public health spending on under-five deaths, accounting for any reverse causality bias through our IV approach leads to a negative point estimate. This result is therefore in line with the expected upward bias of the fixed effects estimate in our context, as previously discussed. Overall, accounting for the possible endogeneity of government health spending through an IV estimator (beyond what is captured by country- and time-specific factors) leads to larger estimated effects of public spending on under-five and adult mortality.

Private health spending. There is no evidence that variations in VHI private spending are related to variations in mortality outcomes: in all instances the estimated IV (or fixed effects) coefficients are nowhere near statistical significance. The IV estimations do, however, suggest an effect of private OOP health spending on adult mortality. The point estimates are statistically significant at the 5% level and indicate reductions of around 23.4 female and 15.6 male deaths per 1,000 in response to a \$100 higher per capita OOP expenditure (Table 4, columns 4 and 6). These effects are substantially larger than the ones implied by the (statistically insignificant) fixed effects estimates. On the other hand, higher OOP spending does not seem to systematically affect under-five mortality judged by the small and statistically insignificant point estimates.

Effective access to care: immunization coverage. As in the case of government spending, the results shown in Table 4 for immunization rates strongly indicate that expansions in health coverage *per se* lead to improvements in population health,

⁹ In order to make the interpretation of estimated coefficients and cross-country comparisons more intuitive, we use natural units of the variables and re-scale our rate and share regressors to be expressed as tenths (e.g., 10 percentage points of immunization coverage) and per capita variables to be expressed as hundreds (e.g., \$100 of government health spending per capita). For ease of reference, we also provide a summary of our main results, with impacts presented in terms of 10% increases in each health coverage indicator (Table 1).

measured by mortality outcomes. The statistically significant IV-2SLS coefficients suggest reductions of around 2.2 under-five deaths per 1,000 live births (column 2), and 9.8 and 7.9 fewer adult deaths per 1,000 (female and male, respectively; columns 4 and 6), in response to an increase of 10 percentage points in the immunization coverage rate. Once again, the IV estimation procedure generates larger point estimates than those from the basic fixed effects model that make no adjustment for reverse causality, in particular for adult mortality outcomes.

Other covariates in the regressions. For the sake of conciseness, we do not show in the tables estimated coefficients for the remaining covariates used in our regressions, but we discuss here their main implications. The results for national income, education and demographic controls are broadly in accordance with the relationships one would expect to find *a priori*. Higher primary education enrolment rates are found to be associated with better population health: for example, all else equal, the IV-2SLS estimates point to around 8 fewer under-five deaths per 1,000 on average in countries with a 10 percentage point higher primary school enrolment. Higher shares of population aged 0-14 years are found to be significantly associated with higher child and adult mortality rates (e.g., about 42 per 1,000 additional under-five deaths in countries with a 10 percentage point higher proportion of people aged 0-14, all else equal). By contrast, after including country-specific effects, time dummies and all other covariates in the IV (and fixed effects) models, higher GDP per capita is not found to be associated with lower mortality rates. Finally, the year dummies systematically suggest an overall downward trend in mortality rates during the study period, with negative and generally statistically significant coefficients.

4.3 Further specification and robustness checks

In this section, we undertake a battery of tests to check whether the health coverage effects identified previously are robust to changes in the econometric specification and estimation sample. Our focus is on the stability of the statistically significant coefficients of health coverage effects on mortality outcomes found in the preferred IV-2SLS estimations (Table 4). Therefore, in Table 5 we display the results of the specification and robustness checks for three coverage indicators: government health spending, OOP health spending and immunization coverage. For each of these measures, the first row shows the statistically significant baseline coefficients found in the IV-2SLS models from Table 4, followed by the corresponding coefficients and statistical significance levels estimated in each specification and robustness test. All models are estimated using our IV-2SLS procedure, with the same income, education and demographic controls as before, plus country-specific effects and year dummies.¹⁰

(a) Allowance for lagged coverage effects

It seems reasonable to expect the effect of current expansions in health coverage in a country to have different effects over time depending on the health outcome analyzed. For example, adult mortality indicators may take longer to react to policies of enhanced

¹⁰ For all the specification and robustness checks in Table 5, the estimated models pass the diagnostic tests discussed in the previous sections (results not shown): first stage IV under-identification is always rejected at least at the 2% level for under-five mortality models and 1% level for adult mortality models.

health system coverage than child mortality measures. Moreover, for some outcomes, the cumulative health effect over time of expanded coverage may be larger than any such impacts measured contemporaneously. Focusing only on contemporaneous impacts may therefore mask potentially important non-linearities in health impacts over time.

In order to deal with the above issues, we estimate a finite distributed lag specification as an extension of our basic model. Assuming up to two-year lagged coverage effects, equation (3) can be re-written as:

$$(7) \quad y_{it} = \beta_1 C_{it} + \beta_2 C_{i,t-1} + \beta_3 C_{i,t-2} + \delta X_{it} + \alpha_i + \theta_t + \varepsilon_{it}.$$

So we add to our baseline model the first and second lags of each coverage indicator. The cumulative effect of coverage on the health outcome y_{it} will then be given by the sum of the contemporaneous and lagged estimated impacts.¹¹ We estimate equation (7) through our preferred IV-2SLS approach, using the first and second lags of the generated instruments C_{it}^* as instruments for the observed lagged coverage values.

For each mortality outcome and health coverage variable in Table 5, row (a) shows the main estimated IV coefficients from model (7). The key message from these estimations is that the baseline mortality results are mostly robust to the inclusion of lagged coverage measures and, except in a couple of instances, the effects of coverage indicators on mortality seem to be adequately captured by the baseline model of contemporaneous impacts. As an exception, there appear to be delayed effects of variations in immunization coverage on adult mortality rates, captured by the first and second lags of the regressor, suggesting that the baseline model may provide underestimated immunization effects for these mortality outcomes. The sum of contemporaneous and lagged coefficients points to larger total reductions of about 13.4 and 9.9 adult deaths per 1,000 (female and male, respectively) for a 10 percentage point contemporaneous increase in the median immunization rate. This seems a sensible result as some of the beneficial impact of expansions in immunization coverage may be expected to take some years to be reflected in adult mortality outcomes, where these larger cumulative effects over time are being captured by the two lagged immunization variables in equation (7). On the other hand, the baseline estimated effect of immunization on the under-five mortality rate, and that of government health spending on adult female mortality, do not resist to the inclusion of lags becoming smaller and statistically insignificant at conventional levels.

¹¹ Of course, additional variables can be included to capture lagged effects beyond the two-year period and their statistical significance tested through conventional methods. In our case, we have also estimated alternative models using additional lags for each regressor of interest ($t - 3$; $t - 4$; and $t - 5$). In no case did we find statistically significant effects of lagged variables corresponding to years beyond $(t - 2)$ (results not shown). In order to avoid multicollinearity problems and inflated standard errors through the introduction of irrelevant variables in the model, we focus only on the specification with contemporaneous and two-year lagged coverage impacts.

(b) Exclusion of outliers

An examination of the raw data (see Figure 1, for example) indicates the presence of some data points that, given their noteworthy discrepancy from the values observed in the rest of the sample, might be driving the conclusions of our empirical exercise. We investigate this possibility by identifying those country-year observations that exhibit “too large” or “too small” values of any of the mortality outcomes and health coverage measures, and testing the robustness of the baseline estimation results to excluding such observations.

In addition to an informal examination of the dataset, we use a formal statistical procedure proposed by Billor et al. (2000) to identify the outliers.¹² The procedure leads to the nomination of 11 country-year observations as outliers in terms of mortality outcomes, mainly due to adult mortality rates in excess of 500 per 1,000: Botswana (years 2001 to 2006), Swaziland (2004-2007) and South Africa (2007). Another 30 observations for high income countries are considered outliers in terms of our spending indicators: Luxembourg (2002-2006), Netherlands (2003-2005), Switzerland (1995-1996, 1999-2007) and the USA (1995-1996, 1998-2006). Luxembourg exhibits figures well in excess of \$4,000 for government health expenditure per capita in the period (about seven times the sample average); Switzerland usually exhibits OOP per capita health expenditure figures of around \$1,200 (over six times the sample average), whereas the Netherlands and the USA have VHI expenditure per capita typically between \$600 and \$2,300 (10-40 times the sample average, respectively).

We then re-run our baseline IV-2SLS models excluding the 41 outlying observations. The estimates in Table 5 (row (b) for each coverage measure) show that our main IV results are largely robust to the exclusion of outliers. For example, a \$100 increase in government health expenditure per capita is estimated to result in a 16.3 per 1,000 lower under-five mortality rate, as well as 2.9 and 2.2 per 1,000 lower female and male death rates (respectively). These are very similar point estimates to the ones obtained in the corresponding baseline estimations. The new results for OOP spending effects on adult mortality are still statistically significant as well, although the point estimates are smaller than the corresponding baseline estimates. This suggests that some country observations with very high OOP spending values and very low mortality rates are partly driving the baseline estimation results for this particular health coverage indicator. Once these outliers are excluded, however, there still remain statistically significant negative effects of OOP spending on adult mortality. By contrast, the exclusion of outliers makes the baseline estimated effect of immunization coverage on under-five mortality to once again lose conventional statistical significance in the robustness checks, although its point estimate is not too dissimilar from the baseline estimate.

¹² The algorithm for formally identifying outliers in the data is based on the Mahalanobis distances (i.e. Euclidean distances weighted by the inverse of the sample variances) between (i) the observation's set of outcomes and coverage indicators, and (ii) the respective sample medians for a “core” subset of observations in the data. See Billor et al. (2000) for details. As for the cut-off significance level for the Billor et al. procedure, we use the rather conservative threshold (i.e. identifying a higher number of outliers) of 15%.

(c) Exclusion of the period prior to 1999

We have assembled a complete dataset of mortality outcomes and covariates for around 115 countries per year on average for most of the period 1995 to 2008, encompassing 153 different countries in total. However, the data are notably sparser for the years prior to 1999, in particular as far as adult mortality rates are concerned: there are usable estimation data for about 61 countries on average between 1995-98. To avoid concerns about bias in our estimations due to the over-representation of countries with better reporting systems in the sample before 1999 (which may be systematically correlated with mortality outcomes and coverage measures), we estimate our baseline IV specifications using a restricted sample containing data only for the period 1999-2008.

The results summarized in Table 5 (row (c) for each coverage indicator) are reassuring in terms of the robustness of our baseline results to using a shorter period of analysis. The beneficial effects of government health spending on population health, found in the baseline estimations, remain evident in the restricted sample. The new point estimates are very similar to the corresponding baseline results in the case of adult mortality outcomes (both in terms of size and statistical significance of coefficients), yet somewhat smaller for under-five mortality: the new point estimate indicates an average decrease of 8.3 child deaths per 1,000 in response to \$100 additional government health spending. For OOP health spending and immunization coverage, the new estimated effects on mortality outcomes are again very similar to the baseline results.

(d) Share of out-of-pocket health payments as a proxy for financial risk protection

The main objective of this study is to investigate the effects of health coverage expansions on population outcomes. As we argue throughout the paper, health coverage is a broad concept, encompassing various dimensions of access to health care and financial risk protection. We attempt to capture the various dimensions related to coverage by including, in our main econometric models, a range of indicators of health spending levels—public and private—in addition to immunization rates. Specifically on the financial protection domain, we use OOP health spending per capita as a proxy for the degree of risk protection, and we also include in the models the components of pooled health expenditure (government and VHI).

It has been argued, however, that the incidence of catastrophic health payments in a given country—the most commonly used measure of financial risk protection in the health system—is better predicted by the *proportion* of national health spending funded privately through OOP payments, instead of the level of OOP health payments (cf. e.g., Xu et al. 2007). In fact, if anything, our statistically significant baseline IV estimations actually suggest *beneficial* effects of the level of OOP health spending on adult mortality. We thus explore the sensitivity of the OOP spending conclusions to replacing OOP expenditure per capita by OOP payments as a share of total health expenditure in the analyses. Given that private health spending is virtually equivalent to OOP health spending in several countries in the sample, we seek to avoid multicollinearity problems by estimating specifications including as coverage measures only total health expenditure, OOP health expenditure as share of total and the immunization rate (in addition to the income, education, demographic and time

dummy controls).¹³ We generate instruments for these spending measures and perform IV-2SLS estimations as explained in section 2.2.

The estimation results for this specification test are presented in Table 5 (row (d) for the OOP per capita spending variable).¹⁴ Keeping the level of national health spending constant, the share of OOP health spending over total has a deleterious effect on both adult mortality outcomes. The IV point estimates, statistically significant at the 5% level, suggest that an increase of 10 percentage points in the share of OOP health financing leads to increases of 34.2 and 38.9 adult deaths per 1,000 (female and male, respectively).¹⁵ Therefore, there is evidence that health coverage expansions aimed to improve financial protection—proxied by a lower share of OOP payments in health care financing—have a beneficial impact on adult health.¹⁶

(e) Allowance for differential coverage effects in low and middle income countries

The examination of the raw data in section 4.1 suggested the possibility of non-linear effects of health coverage expansions according to national income levels. For example, it might be the case that a given increase in the level of public health spending has a different health impact in low and middle income countries (LMIC) compared to high income countries, also because countries in the former group started the study period with worse mortality conditions and remained so throughout.¹⁷ The addition of country-specific effects to our IV estimator should capture any confounding effects of initial mortality levels for the estimation of average coverage effects. But it seems important from a policy perspective to investigate whether the average relationship between population health outcomes and coverage measures varies across countries depending on their income levels (and, therefore, baseline health status). For a given expansion in health system coverage, countries characterized by much higher mortality rates may obtain larger marginal health gains than countries with already low mortality levels.

¹³ OOP expenditure represents about 80% of private health spending on average in the full sample (75th percentile = 95%) and 81% for the subset of low and middle income countries (75th percentile = 96%).

¹⁴ As in the case of the remaining specification and robustness checks in Table 5, the new IV models pass all the under-identification and instruments' exogeneity tests previously discussed (results not shown). In particular, first stage IV under-identification is strongly rejected for the estimation of reverse causal effects of female and male mortality on the OOP share (*F* statistics for excluded instruments of 7.19 and 11.60, respectively), resulting in negative and statistically significant estimated mortality coefficients (-0.023 and -0.033, respectively). For both mortality outcomes, the results in row (d) of Table 5 come from models where under-identification is rejected with *p*-values lower than 0.001.

¹⁵ An increase of 10 percentage points in the share of OOP payments over total health expenditure has a particularly appealing interpretation in our context: for a given country in the sample, this increase is close to moving from the sample average (share of OOP = 34%) to the average among low income countries (share of OOP = 46%).

¹⁶ The remaining estimates from these models (not shown) indicate that increases in total health spending per capita lead to lower female mortality (coefficient = -2.712; *p*-value = 0.027) but do not significantly affect male mortality (coefficient = -0.896; *p*-value = 0.174).

¹⁷ For instance, the average under-five mortality rate among high income countries is 9.1 per 1,000 for the whole period in our sample, compared to 63.3 per 1,000 in the LMIC group. In 1995, the start of the study period, the equivalent figures are 8.4 and 77.6 per 1,000 (respectively).

We can investigate this possibility by estimating an extension of equation (3), adding interaction terms between a LMIC indicator (equal to one if the country-year observation has a GDP per capita up to \$12,195; zero otherwise) and each of the health spending and immunization variables, i.e. adding one $C_{it} \times LMIC_i$ term for each coverage measure.¹⁸ This allows us to get at the issue of whether the estimated average impacts of health coverage expansions discussed previously vary according to levels of national income and, in particular, if being a poorer country means that increased coverage leads to differential health gains compared to the average country in the full sample.

The results from the interacted specifications, estimated by IV-2SLS using the full sample of countries, are presented in Table 6. For each mortality outcome, the first column presents the results from model (3) expanded by the inclusion of the four interacted coverage terms; as before, the focus is on the statistically significant coefficients found in the baseline IV estimations (Table 4), so we only present estimates for government and OOP health spending per capita, and immunization coverage. The second column for adult mortality outcomes displays the results from a model where the coverage indicators are the share of OOP health spending over total, total health spending per capita and median immunization rate (as in the robustness test (d) above), plus their interactions with $LMIC_i$. For conciseness, we only show the estimated sum of the coefficients of main effects and interaction terms (and their standard errors and significance levels), which give the total estimated health coverage effects for low and middle income countries.

The first conclusion from Table 6 is that the average beneficial effect of higher public health spending on under-five mortality, identified in the baseline estimations, seems to be substantially larger for low and middle income countries. Column 1 shows an estimated reduction of about 90.8 under-five deaths per 1,000 for a \$100 increase in government health expenditures, an incremental effect that is over six times larger than the average effect estimated for the full sample of countries. Also, there is some evidence—albeit weak—that additional public health spending has larger adult female and male mortality impacts in the LMIC group (columns 2 and 4). Despite being statistically insignificant at the 10% level, the two point estimates are not far away from that conventional significance threshold (p-values of 0.135 and 0.120, respectively), have a negative sign and are at least five times larger than the corresponding full sample estimates. They suggest reductions of 18.4 and 12 deaths per 1,000 in female and male adult mortality, respectively, in response to \$100 higher government health spending.

For the two adult mortality outcomes, there is evidence that higher levels of OOP health spending per capita are linked to improved health in the LMIC group as well. The coefficients in Table 6 (columns 2 and 4) are statistically significant and indicate that female and male mortality rates tend to decrease, respectively, by 49 and 37.9 deaths per 1,000 for an extra \$100 of OOP spending per capita, about twice as much as the estimated effect in the full sample. Once again, those beneficial effects seem to be restricted to the *level* of OOP spending and do not extend to the *share* of OOP payments in the total financing of the health system. Controlling for the level of total

¹⁸ This procedure results in the LMIC indicator taking on the value of one for 116 different countries (943 observations) in the under-five mortality regressions, and 113 different countries (830 observations) in the adult mortality regressions.

health spending, a 10 percentage point higher share of OOP payments over total leads to an estimated additional 34.4 female deaths per 1,000 in low and middle income countries (column 3), with a statistically insignificant positive coefficient for male mortality (column 5). The latter results should be treated with some caution, nonetheless, since the IV models in columns 3 and 5 are the only ones in our paper for which the null hypothesis of first stage under-identification fails to be statistically rejected.

Finally, in our baseline IV estimations, higher immunization coverage was found to reduce both child and adult mortality. For the former outcome, the coefficient was found to be less robust according to the tests performed above, and this is the case also for the LMIC group in particular. The result in Table 6 (column 1) shows a statistically insignificant coefficient for the immunization coverage rate in the under-five mortality model. By contrast, the beneficial average effects of expanded immunization on adult mortality seem to be present also for the LMIC group of countries. The estimated coefficients in columns 2 and 4 indicate reductions of 9.4 and 7.2 deaths per 1,000 in female and male mortality rates (respectively) in response to a 10 percentage point higher median immunization rate. These figures are very similar to the estimated reductions of 9.8 and 7.9 deaths per 1,000 from the baseline models for the full sample.

5. Discussion and conclusions

The debate around universal health coverage and the best ways to achieve it across countries has come under the spotlight lately. Much of the justification for the push towards universal coverage is based on the associated improvements in population health, ultimately a fundamental goal of any health system. The main purpose of this study is to contribute to the debate by providing sound empirical evidence on the causal relationship between national levels of health system coverage and mortality outcomes.

We assemble a large panel dataset available at the cross-country level, with annual data for the period 1995-2008 encompassing 153 countries. Subject to the limitations imposed by the available cross-country data, we measure the level of health system coverage by a range of indicators, including measures of pre-paid public and private health expenditure and immunization rates, to try to capture the main elements commonly advanced to define adequate coverage: effective access to needed care and protection from financial hardship due to health payments. In order to answer our research question reliably, we use a two-step instrumental variables (IV) approach that directly estimates the reverse causal effects of under-five and adult mortality on coverage indicators, so as to explicitly adjust for these impacts when estimating the effects of health coverage on mortality outcomes. Even if bias due to reverse causality is not substantial in the models, our IV estimator offers estimates of health coverage effects that are likely to be unbiased and consistent in the presence of relevant omitted variables and measurement error. We subject this model to a battery of specification and robustness tests, including examinations of the influence of delayed coverage effects, outlying observations, changes in the period of analysis, and using a different proxy for financial risk protection within the health system.

Our results strongly indicate that expansions in health system coverage through higher government spending per capita lead, on average, to improved general population health measured by lower child and adult mortality rates. The magnitude of the estimated effects varies of course according to the specific mortality outcome in question. The estimated gains are the largest when under-five mortality is examined (see Table 1 for a comparative summary of results): on average for the whole sample of countries, a 10% increase in government expenditure per capita results in approximate reductions of 7.9 under-five deaths per 1,000, and at least 1.3 deaths per 1,000 in adult mortality rates.¹⁹ If we focus on the group of low and middle income countries (and take into account their lower average public health spending per capita than in the full sample), the estimated effect of a 10% increase in public spending in reducing under-five mortality is around 12.3 deaths per 1,000, or 1.5 times larger than in the full sample.

The positive impact of incremental public expenditure on mortality identified here should not be interpreted as an inevitable outcome regardless of how the additional money is spent. Recent studies have suggested that the quality of national institutions (stability of the political system, degree of public sector accountability and so forth) can influence the effectiveness of public spending (Wagstaff and Claeson 2004). Yet our empirical results do offer support to the claim that broader health system coverage—through higher levels of pre-paid health funds, which is essentially the case of public expenditures—tends to lead to better population health. Moreover, the economic relevance of these health effects is not trivial, as a simple back of the envelope calculation of saved life years can illustrate. According to the coefficients from the baseline IV specification for under-five mortality and keeping other factors constant, the average country would experience a reduction of 0.132 under-five deaths per 1,000 for an extra dollar of government health spending per capita.²⁰ Based on figures for average country population aged 0-4, average under-five mortality rate and life expectancy at age 5, this would lead to a total of 451 lives and 30,443 years of life saved in the typical country.²¹ For the average low and middle income country, the point estimates would imply even larger totals of 3,707 lives and 240,061 life years saved per country, for an extra dollar of government spending per capita.²² The

¹⁹ Our result that public health spending reduces under-five mortality is in accordance with more recent literature in the area (see the introductory section). For example, Bokhari et al. (2007) also find a statistically significant effect of government health expenditures on under-five mortality, although their estimate and sample averages imply a lower reduction of 2.4 child deaths per 1,000 in response to a 10% increase in government spending per capita.

²⁰ This is equivalent to a per capita spending increase of 0.2% in the average country in the sample and 0.7% in the average low and middle income country (or total public health expenditure increases of around \$32.5 million and \$34.8 million, respectively).

²¹ The calculations have been performed as follows. We make the conservative assumption that all children die at age 5. From WHO life tables, 5-year olds are expected to live an extra 67.5 years (world figures, World Health Organization 2010b). Using the average observed under-five mortality rate in the sample and our estimates of deaths averted among the population aged 0-4, this results in 451 lives saved in a given country. Combined with the expected extra years of life this leads to an estimated $451 \times 67.5 = 30,443$ life years saved in total. The data on under-five population by country refer to year 2008 and come from UNICEF (2011).

²² As before, the calculations for low and middle income countries are based on the assumption that all child deaths occur at the age of 5. WHO life tables have separate information on life expectancy at age 5 for low, lower-middle, upper-middle and high income countries (World Health Organization 2010b); we use an average of the life expectancy figures for the first three income categories, weighted by the average under-five population in the sample for each

calculations above amount to average public health spending figures per life saved of around \$72,000 for the average country and only \$9,400 per life saved for the typical lower income country. The values above also indicate that the marginal cost of saving a year of life is on average around \$1,000 in the whole sample of countries, whilst the analogous figure for a low or middle income country is just \$145. These figures can be very favorably compared with a widely cited benchmark of \$100,000 used in high income countries as the implicit value of a life year (Cutler and McClellan 2001).²³

In addition to looking at pre-paid health funds channelled through the public sector, the data allow us to disaggregate private health spending into voluntary health insurance (VHI) and out-of-pocket (OOP) payments. For the average country, pre-paid public spending seems far more effective in reducing mortality than pre-paid private VHI funds, although it should be noted that there is a high frequency of zero VHI values at the country level (particularly in lower income countries) and generally very small non-zero figures, thus making it difficult to identify VHI effects from variations at the aggregate level.²⁴

The conclusions regarding the aggregate health effects of private OOP spending are mixed. Higher overall levels of OOP expenditures per capita may indicate that individuals in such countries have improved capacity to pay to obtain more health care and/or better quality care (than available at public facilities, for example). This provides a potential explanation for the beneficial average effects of higher OOP spending levels on adult mortality which we identify here; given the data limitations, we are unable to further examine this possibility. But the latter result is also consistent with a different explanation. The coefficients from the baseline IV regressions may be simply picking up the effects of increased national (overall) capacity and willingness to pay for health care in countries where the public budget for health is more severely limited. Two pieces of evidence from our data provide support to this hypothesis. First, the estimated beneficial effects of OOP spending per capita levels on adult mortality are twice as large for low and middle income countries as in the full sample. Second, and perhaps more importantly, we find strong detrimental effects of a higher share of OOP health payments over total on adult mortality, controlling for total health spending. This offers evidence that a higher participation of pre-paid funds in total health financing, and thus broader health coverage in terms of financial protection, leads to aggregate health gains in addition to any welfare benefits of risk protection *per se* (Xu et al. 2007).²⁵

income group. From these calculations, children aged 0-4 in low and middle income countries are expected to live an extra 64.8 years. Combined with the average under-five mortality rate in that group of countries, the relevant estimate of averted under-five deaths (a reduction of 0.908 deaths per 1,000 for an extra dollar of public health spending per capita) results then in 3,707 lives saved in the average low and middle income country, and 240,061 extra life years saved.

²³ This is a mere illustrative exercise that should be interpreted with due caution and does not intend to serve as a full computation of welfare gains, as it does not compare the costs and benefits of public investments in health vis-à-vis other sectors (such as education, for example).

²⁴ More than half of our country-year observations have VHI expenditure per capita lower than \$0.05; in the low and middle income sub-sample, that figure reaches two-thirds of the observations.

²⁵ It is difficult to compare our results concerning OOP health payments to the existing literature, since the latter has tended to concentrate on individual country cases (as outlined in the Introduction).

At first glance, our finding that immunization coverage on average improves adult health but not (robustly at least) child health may seem odd. After all, the immunization variable we use refers to interventions targeting children in their first or second year of life. These findings may be at least partly explained by the fact that broader immunization coverage is usually achieved through public health campaigns undertaken by governments, instead of privately funded efforts. Therefore, most of the potentially beneficial immunization effects on under-five mortality may end up being captured by the government health spending variable included in the models (i.e. through higher public spending). This would explain why the estimated baseline coefficient of immunization rate in the under-five mortality equation does not retain statistical significance across all the robustness tests undertaken, despite exhibiting always a negative sign indicating reductions in child mortality. In the adult mortality models, it seems more reasonable to think of our immunization rate variable as a proxy for the overall conditions of effective access to the health care system, not least because immunization rates show a significant degree of correlation with other frequently used indicators of care access.²⁶ In this case, our immunization coverage results corroborate the idea that improved access to health care is an important instrument for countries to achieve better population health outcomes.

Some limitations imposed by the available data to our study must be acknowledged, pointing also to areas where further investigation seems warranted. First, although we use some widely cited health coverage measures in our empirical work, the analysis would have been improved if panel data on other coverage indicators—such as measures of outpatient and inpatient visits, and barriers to health system access—were available for a reasonable number of countries during the study period. International agencies may be able to make related data systematically available by centrally collating the growing amount of information provided by national-level annual surveys, which often contain questions on aspects such as effective access to care (International Household Survey Network 2011). Also, for this study we concentrate on population health measured by mortality indicators. Additional gains from broader health system coverage may include elements such as improvements in quality of life and more equitable health financing and access to health care (other explicit objectives in many health systems; see e.g., World Health Organization 2010a).

Second, as most of the previous literature in the area, our research is unable to exploit a natural experiment to help identify the impacts of changes in health coverage on population health. We have attempted to mimic a natural experiment using instrumental variable techniques that seem appropriate to our context—and that we subject to several formal specification and robustness checks—since exogenous events triggering variations in system coverage (unrelated to population outcomes) and encompassing several health systems at once rarely occur.²⁷ Future studies should in this context carefully deal with the confounding influence of observable and

²⁶ For example, the pairwise correlation coefficient between our immunization variable and the share of births attended by skilled personnel is 0.7 in our sample (significant at the 1% level). The correlation coefficients are also statistically significant between the immunization rate and the proportion of pregnant women receiving prenatal care (0.6), number of physicians per 1,000 (0.6), hospital beds per 1,000 (0.4) and outpatient visits per capita (0.4). These correlation coefficients refer to the estimation sample in year 2005, which has the higher number of available country observations for these indicators (as previously noted, we unfortunately do not have usable time series for any of these additional access measures).

²⁷ As an exception, see Wagstaff and Moreno-Serra (2009).

unobservable factors, in addition to reverse causal effects, to go beyond the identification of mere associations in the data that are likely to arise from simple least squares regression models.

Finally, it is also worth stressing that our estimates reflect what would happen *on average* to population outcomes if countries experienced variations in health coverage.²⁸ Of course, there are likely to be particular country stories underneath these averages. An examination of the regression residuals shows that our econometric models do a good job in predicting the observed mortality outcomes across countries, yet there are some countries for which relatively large residuals indicate the presence of other factors influencing observed mortality, beyond those accounted for in our models.²⁹ As examples among low and middle income countries, Burundi and Malawi consistently exhibit observed under-five mortality rates lower than the levels predicted by the models, while the opposite is true for countries like Trinidad and Tobago, Equatorial Guinea and some Middle-Eastern nations. Case-studies focused on the specific stories of these countries would be better suited to explain their relatively better- or poorer-than-predicted health system performance.

The 2010 World Health Report reinforced calls for a push towards universal health coverage across countries. It suggested that many countries are still lacking the necessary investment in the health sector so as to improve population outcomes in line with the Millennium Development Goals. Our study offers hard evidence that investing in broader health coverage can generate significant gains in terms of population health. Therefore, it seems important for countries with sufficient resources to regard enhancements in health system coverage as a key investment target, as well as for the international community to ensure that the poorest countries have the ability to invest adequate amount of funds in the area over the coming years.

²⁸ Moreover, the results indicate the expected consequences if incremental government funds for health care (for example) are spent in line with the existing allocation of resources in the sector, i.e. if current health programs are proportionally scaled up.

²⁹ Importantly, these omitted factors should not bias our estimated coverage impacts if they are not simultaneous determinants of system coverage and mortality levels.

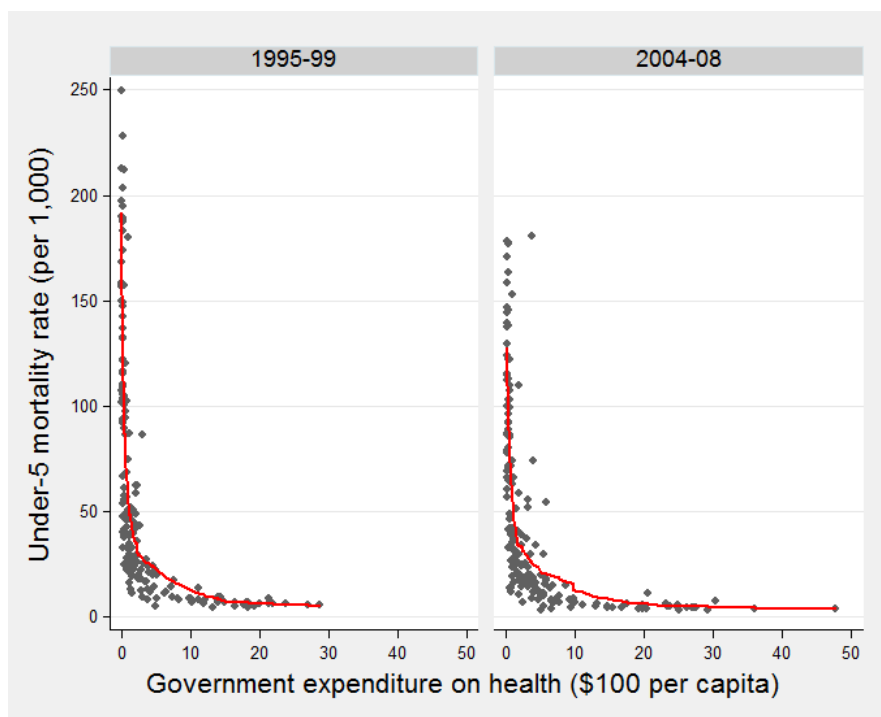
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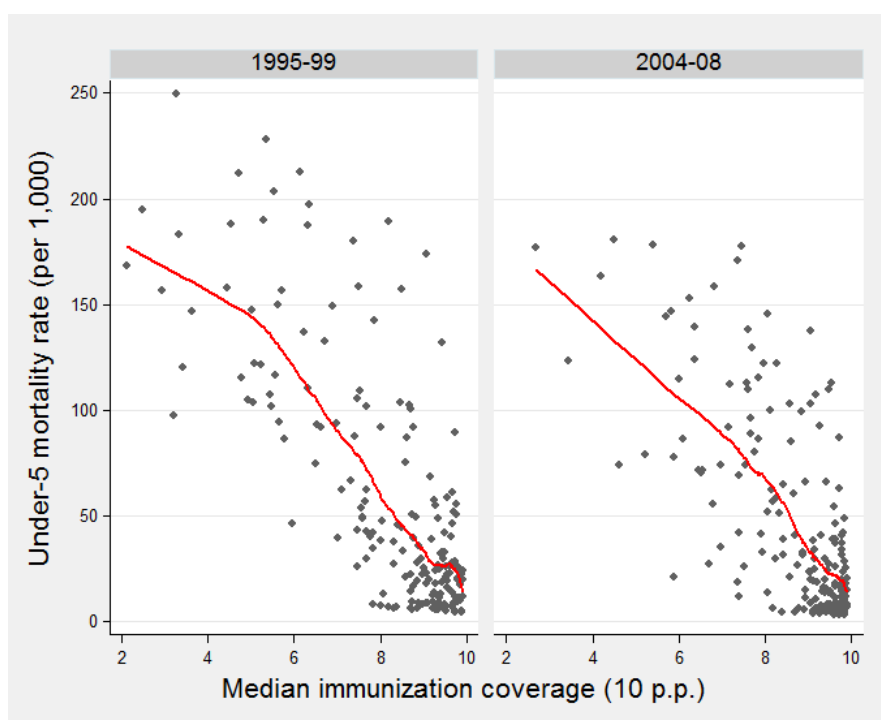
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Figure 1: The relationship between measures of health coverage and under-five mortality (five year averages, 1995-99 and 2004-08)

Panel A – Government expenditure on health



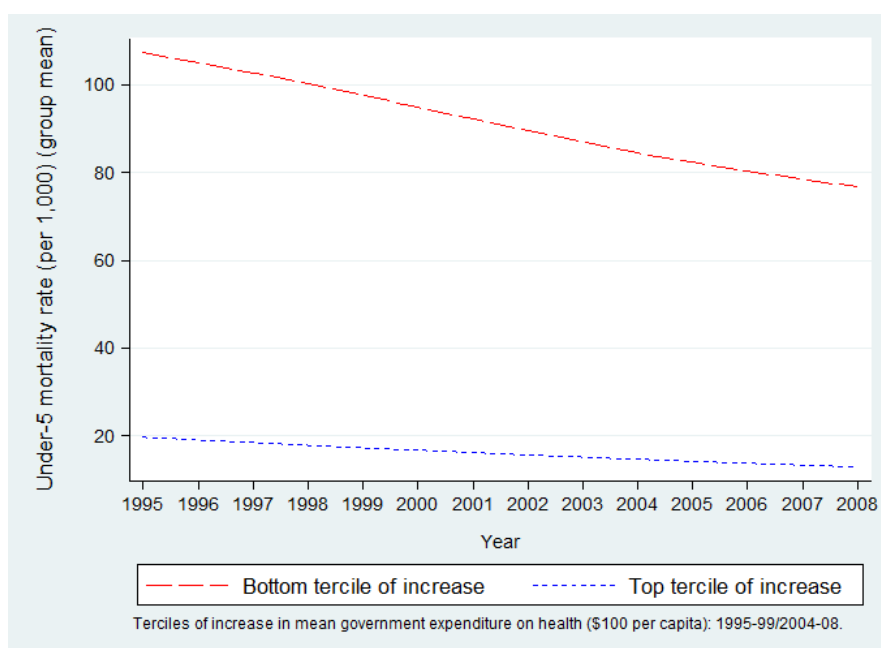
Panel B – Immunization coverage



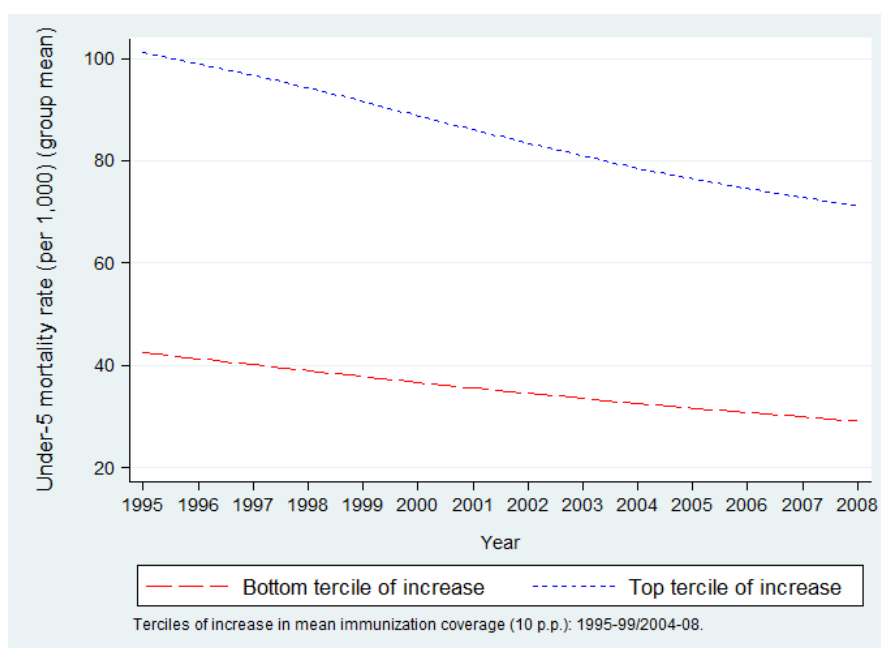
Notes: Each point in the figure represents a country. The line is the prediction from a locally weighted non-parametric regression of under-five mortality rate on the corresponding coverage indicator. Coverage indicators are government expenditure per capita (hundreds of constant 2005 international dollars) and immunization coverage rate (median value, in 10 percentage points, across six immunization rates: diphtheria-pertussis-tetanus, hepatitis B, haemophilus influenzae B, polio, BCG and measles). The plots refer to five-year average values of under-five mortality and coverage indicators between 1995-99 and 2004-08.

Figure 2: Average under-five mortality over time across groups (terciles) of countries with the largest and smallest increases in health coverage measures (1995-2008)

Panel A – Government expenditure on health



Panel B – Immunization coverage



Notes: The plots compare the evolution of average under-five mortality rates across the bottom and top tercile groups of increase in the corresponding coverage indicator, over the period 1995-2008. The bottom (top) tercile group comprises those countries with the smallest (largest) increases in the coverage indicator during the period 1995-99 to 2004-08. Coverage indicators are government expenditure per capita (constant 2005 international dollars) and immunization coverage rate (median value across six immunization rates: diphtheria-pertussis-tetanus, hepatitis B, haemophilus influenzae B, polio, BCG and measles).

Table 1: Summary of results

	<i>For a 10% increase in:</i>				
	Government health spending per capita	OOP health spending per capita	OOP health spending (share of total)	VHI health spending per capita	Immunization coverage rate
Under-five mortality rate	(-) 7.9 per 1,000	No effect.	No effect.	No effect.	Negative significant effect not robust.
Female mortality rate (adult)	(-) 1.6 per 1,000	(-) 4.4 per 1,000	(+) 11.6 per 1,000	No effect.	(-) 8.5 per 1,000
Male mortality rate (adult)	(-) 1.3 per 1,000	(-) 2.9 per 1,000	(+) 13.6 per 1,000	No effect.	(-) 6.8 per 1,000

Notes: The table presents the baseline estimated incremental effect, on each health outcome, for a 10% increase in the corresponding coverage indicator (relative to the observed average in the data). OOP = private out-of-pocket; VHI = private voluntary health insurance. Incremental effects expressed in deaths per 1,000. (+) denotes increase (positive regression coefficient) and (-) denotes decrease (negative regression coefficient). No effect = no statistically significant effect is found in the baseline model. Significant effect not robust = a statistically significant effect is found in the baseline model but not across robustness tests.

Table 2: Variable definitions and sources

Variable	Description	Source
<u>Outcomes</u>		
Under-five mortality rate	Mortality rate, children under five years old (per 1,000 live births)	Institute of Health Metrics and Evaluation
Female mortality rate (adult)	Adult mortality rate, ages 15-60, female (per 1,000 female adults)	World Bank (World Development Indicators)
Male mortality rate (adult)	Adult mortality rate, ages 15-60, male (per 1,000 male adults)	World Bank (World Development Indicators)
<u>Regressors</u>		
Government health spending per capita	Health expenditure per capita: general government (constant 2005 international dollars)	WHO (Global Health Observatory)
OOP health spending per capita	Health expenditure per capita: private out-of-pocket (constant 2005 international dollars)	WHO (Global Health Observatory)
OOP health spending (share of total)	Private out-of-pocket health expenditure as share of total health expenditure (%)	WHO (Global Health Observatory)
VHI health spending per capita	Health expenditure per capita: private prepaid plans (constant 2005 international dollars)	WHO (Global Health Observatory)
Total health spending per capita	Health expenditure per capita: total (constant 2005 international dollars)	WHO (Global Health Observatory)
Immunization coverage	Median immunization coverage: DTP3, HepB, Hib, polio, BCG and measles (% of children aged 1, or 12-23 months for measles)	WHO, World Bank
GDP per capita	GDP per capita (constant 2005 international dollars)	World Bank (World Development Indicators)
Primary education enrolment rate	Primary school enrolment (% of relevant age group)	World Bank (World Development Indicators)
Population 0-14	Population ages 0-14 (% of total population)	World Bank (World Development Indicators)
Population 65+	Population ages 65 and above (% of total population)	World Bank (World Development Indicators)
CO2 emissions per capita	Carbon dioxide (CO2) emissions (metric tons per capita)	World Bank (World Development Indicators)
Conflict deaths	Battle-related deaths in conflicts	Uppsala Conflict Data Program, Uppsala University

Table 3: Descriptive statistics

	Full sample					Low and middle income countries				
	Mean	Std. Dev.	Min	Max	Countries	Mean	Std. Dev.	Min	Max	Countries
<i><u>Outcomes</u></i>										
Under-five mortality rate (per 1,000)	45.7	48.9	2.9	250.1	153	63.3	49.6	7.0	250.1	116
Female mortality rate (adult, per 1,000)	155.8	114.9	39.2	630.5	151	196.1	116.6	54.8	630.5	113
Male mortality rate (adult, per 1,000)	226.4	116.9	66.9	628.5	151	271.7	109.8	104.6	628.5	113
<i><u>Regressors</u></i>										
Government health spending per capita (\$100)	5.99	8.43	0.00	48.03	153	1.36	1.35	0.00	7.35	116
OOP health spending per capita (\$100)	1.88	2.04	0.03	12.63	153	0.85	0.77	0.03	5.57	116
OOP health spending (share of total, 10 p.p.)	3.4	1.8	0.3	9.4	153	4.0	1.8	0.3	9.4	116
VHI health spending per capita (\$100)	0.58	2.10	0.00	23.72	153	0.14	0.40	0.00	3.22	116
Total health spending per capita (\$100)	8.71	11.43	0.09	69.22	153	2.45	2.15	0.09	12.06	116
Immunization coverage (10 p.p.)	8.6	1.4	2.1	9.9	153	8.3	1.6	2.1	9.9	116
GDP per capita (\$100)	121.15	131.67	2.80	744.22	153	42.37	32.18	2.80	121.37	116
Primary education enrolment rate (10 p.p.)	8.6	1.6	2.3	10.0	153	8.2	1.7	2.3	10.0	116
Population 0-14 (10 p.p.)	3.1	1.0	1.3	5.0	153	3.6	0.8	1.3	5.0	116
Population 65+ (10 p.p.)	0.8	0.5	0.1	2.1	153	0.5	0.3	0.2	1.7	116
CO2 emissions per capita (metric tons)	5.2	6.5	0.0	56.3	153	2.0	2.4	0.0	17.1	116
Conflict deaths (per 100,000 population)	1.4	25.8	0.0	683.6	153	2.1	31.3	0.0	683.6	116

Notes: Time period is 1995-2008. The table presents the mean, standard deviation (Std. Dev.), minimum (Min) and maximum (Max) values, and number of countries, for the corresponding variable in the full sample and separately for the sub-sample of low/middle income countries (GDP per capita up to \$12,195). 10 p.p. = variable measured in 10 percentage points.

Table 4: Baseline results for the effects of coverage on health outcomes

	Under-five mortality rate		Female mortality rate (adult)		Male mortality rate (adult)	
	FE-LS	IV-2SLS	FE-LS	IV-2SLS	FE-LS	IV-2SLS
	(1)	(2)	(3)	(4)	(5)	(6)
<i>Public spending</i>						
Government health spending per capita	0.581*** (0.221)	-13.193** (5.516)	-1.218 (0.741)	-2.583** (1.306)	-1.019* (0.559)	-2.210** (0.975)
<i>Private spending</i>						
OOP health spending per capita	0.856 (0.634)	2.685 (5.033)	-0.754 (2.391)	-23.385** (11.259)	-1.487 (2.360)	-15.545** (6.396)
VHI health spending per capita	0.556 (0.388)	-6.143 (9.241)	0.680 (1.112)	5.153 (3.655)	0.595 (0.849)	8.731 (6.368)
<i>Effective access to health care</i>						
Immunization coverage	-1.962*** (0.544)	-2.203* (1.220)	-1.957 (1.666)	-9.841** (4.494)	-1.123 (1.482)	-7.858** (3.333)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
First stage under-identification LM test (statistic)		8.50		46.81		31.75
First stage under-identification LM test (p-value)		0.004		0.000		0.000
F statistic	17.75	3.95	11.52	4.40	23.68	9.89
F statistic (p-value)	0.000	0.000	0.000	0.000	0.000	0.000
Number of countries	153	153	148	148	148	148
Observations	1,397	1,397	1,222	1,222	1,222	1,222

Notes: Time period is 1995-2008. Models estimated by standard least squares fixed effects (FE-LS) or instrumental variables through a two-stage least squares approach (IV-2SLS), using as instruments the reverse causality-adjusted coverage indicators (see text). All regressions also control for GDP per capita, the primary education enrolment rate, the share of population aged 0-14 and the share of population aged over 65. Standard errors (in parentheses under coefficients) robust to arbitrary heteroskedasticity and autocorrelation. * Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 5: Specification and robustness checks for the baseline effects of coverage on health outcomes

		Under-five mortality rate	Female mortality rate (adult)	Male mortality rate (adult)
		IV-2SLS	IV-2SLS	IV-2SLS
		(1)	(2)	(3)
<i>Government health spending</i>				
	Baseline	-13.193** (5.516)	-2.583** (1.306)	-2.210** (0.975)
(a)	With first and second lags (sum of all coeffs.)	-7.860** (3.669)	-1.377 (1.098)	-1.966** (0.961)
	(b) Excluding outliers	-16.291** (7.042)	-2.877** (1.244)	-2.166** (1.076)
	(c) Excluding years before 1999	-8.341** (3.706)	-2.163** (1.094)	-2.303** (0.936)
<i>OOP health spending</i>				
	Baseline		-23.385** (11.259)	-15.545** (6.396)
(a)	With first and second lags (sum of all coeffs.)		-25.693* (14.789)	-14.928** (7.491)
	(b) Excluding outliers		-14.022** (5.544)	-12.475*** (4.480)
	(c) Excluding years before 1999		-22.731** (11.227)	-15.066** (6.310)
	(d) OOP as share of total health spending		34.196** (15.703)	38.934** (15.307)
<i>Immunization coverage</i>				
	Baseline	-2.203* (1.220)	-9.841** (4.494)	-7.858** (3.333)
(a)	With first and second lags (sum of all coeffs.)	-0.807 (1.202)	-13.419* (6.851)	-9.870** (4.821)
	(b) Excluding outliers	-1.807 (1.442)	-6.447** (2.880)	-5.947** (2.433)
	(c) Excluding years before 1999	-1.808* (0.932)	-7.968** (3.894)	-5.993** (2.853)

Notes: For each of health coverage indicator, the first row shows the statistically significant two-stage least squares (IV-2SLS) coefficients found in the baseline models (presented in Table 4), followed by the corresponding coefficients and statistical significance levels estimated in each specification and robustness test. Test (a) adds the first and second lags of the coverage indicators and the estimates refer to the sum of the contemporaneous and lagged effects. Test (b) excludes 41 outlying observations. Test (c) excludes the period 1995-98 from the estimations. Test (d) includes as coverage measures only OOP health expenditure as share of total (instead of OOP spending per capita), total health expenditure and the immunization rate. All regressions also control for GDP per capita, the primary education enrolment rate, the share of population aged 0-14, the share of population aged over 65, country and year fixed effects. Standard errors (in parentheses under coefficients) robust to arbitrary heteroskedasticity and autocorrelation. * Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 6: Estimates of coverage effects in low and middle income countries

	Under-five mortality rate	Female mortality rate (adult)		Male mortality rate (adult)	
	IV-2SLS	IV-2SLS		IV-2SLS	
	(1)	(2)	(3)	(4)	(5)
<i>Sum of coefficients of main effect and interaction terms</i>					
Government health spending per capita	-90.772* (49.631)	-18.414 (12.254)		-12.004 (7.674)	
OOP health spending per capita		-49.014* (24.921)		-37.914** (16.079)	
Immunization coverage	-0.011 (2.858)	-9.406** (4.446)		-7.185** (3.089)	
OOP health spending (share of total)			34.439** (16.280)		15.902 (206.820)
Country fixed effects	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes
First stage underidentification LM test (statistic)	4.96	17.58	1.06	5.39	0.01
First stage underidentification LM test (p-value)	0.026	0.000	0.303	0.020	0.911
F statistic: second stage	1.98	2.99	2.05	7.84	2.45
F statistic: second stage (p-value)	0.007	0.000	0.006	0.000	0.001
Number of countries	153	148	148	148	148
Observations	1,397	1,222	1,222	1,222	1,222

Notes: For each of health coverage indicator, the table shows the two-stage least squares (IV-2SLS) estimates from models where the baseline specification is expanded through the inclusion of interaction terms between an indicator for low/middle income country (equal to one if the country-year observation has a GDP per capita up to \$12,195; zero otherwise) and each of the health spending and immunization variables. All regressions also control for GDP per capita, the primary education enrolment rate, the share of population aged 0-14 and the share of population aged over 65. Standard errors (in parentheses under coefficients) robust to arbitrary heteroskedasticity and autocorrelation. * Significant at 10%; ** significant at 5%; *** significant at 1%.

Appendix

Table A1: Results for the effects of health outcomes on coverage

	Dependent variable			
	Government health spending	OOP health spending	VHI health spending	Immunization coverage
	IV-GMM (1)	IV-GMM (2)	IV-GMM (3)	IV-GMM (4)
Under-five mortality rate	0.504 (0.397) [0.145]	0.019 (0.045) [0.320]	0.044 (0.054) [0.454]	0.003 (0.045) [0.660]
Country fixed effects	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes
Excluded instruments: first stage <i>F</i> test (statistic)	3.07	3.16	3.16	3.07
Excluded instruments: first stage <i>F</i> test (p-value)	0.049	0.045	0.045	0.049
First stage under-identification χ^2 test (statistic)	6.26	6.44	6.44	6.26
First stage under-identification χ^2 test (p-value)	0.044	0.040	0.040	0.044
Over-identification Hansen J test (p-value)	0.400	0.328	0.574	0.352
<i>F</i> statistic: second stage	5.75	10.85	1.34	5.39
<i>F</i> statistic: second stage (p-value)	0.000	0.000	0.170	0.000
Number of countries	153	153	153	153
Observations	1,398	1,397	1,397	1,398

	Government health spending	OOP health spending	VHI health spending	Immunization coverage
	IV-GMM (1)	IV-GMM (2)	IV-GMM (3)	IV-GMM (4)
Female mortality rate (adult)	0.010 (0.015) [0.588]	0.007 (0.005) [0.301]	-0.001 (0.002) [0.329]	0.008 (0.006) [0.820]
Country fixed effects	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes
Excluded instruments: first stage <i>F</i> test (statistic)	7.34	7.19	7.19	7.34
Excluded instruments: first stage <i>F</i> test (p-value)	0.001	0.001	0.001	0.001
First stage under-identification χ^2 test (statistic)	15.00	14.70	14.70	15.00
First stage under-identification χ^2 test (p-value)	0.001	0.001	0.001	0.001
Over-identification Hansen J test (p-value)	0.598	0.281	0.237	0.889
<i>F</i> statistic: second stage	26.84	8.62	1.93	5.35
<i>F</i> statistic: second stage (p-value)	0.000	0.000	0.017	0.000
Number of countries	148	148	148	148
Observations	1,223	1,222	1,222	1,223

	Government health spending	OOP health spending	VHI health spending	Immunization coverage
	IV-GMM (1)	IV-GMM (2)	IV-GMM (3)	IV-GMM (4)
Male mortality rate (adult)	0.009 (0.027) [0.588]	0.005 (0.007) [0.301]	-0.004 (0.010) [0.329]	0.008 (0.007) [0.820]
Country fixed effects	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes
Excluded instruments: first stage <i>F</i> test (statistic)	12.02	11.60	11.60	12.02
Excluded instruments: first stage <i>F</i> test (p-value)	0.000	0.000	0.000	0.000
First stage under-identification χ^2 test (statistic)	24.57	23.71	23.71	24.57
First stage under-identification χ^2 test (p-value)	0.000	0.000	0.000	0.000
Over-identification Hansen J test (p-value)	0.553	0.210	0.273	0.884
<i>F</i> statistic: second stage	25.48	9.23	1.21	5.16
<i>F</i> statistic: second stage (p-value)	0.000	0.000	0.263	0.000
Number of countries	148	148	148	148
Observations	1,223	1,222	1,222	1,223

Notes: Time period is 1995-2008. Models estimated by instrumental variables through a two-step generalized method-of-moments approach (IV-GMM), using as instruments CO2 emissions per capita and the number of battle-related deaths in internal or international conflicts. All regressions also control for GDP per capita, the primary education enrolment rate, the share of population aged 0-14 and the share of population aged over 65. Standard errors (in parentheses under coefficients) robust to arbitrary heteroskedasticity and autocorrelation. P-values in square brackets correspond to the statistical significance test of the coefficients of endogenous regressors, robust to the presence of weak instruments, proposed by Stock and Wright (2000). * Significant at 10%; ** significant at 5%; *** significant at 1%.