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Measuring the Child Mortality Impact of Official Aid for Fighting Infectious Diseases, 2000-2010

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Measuring the child mortality impact of official aid for fighting infectious diseases, 2000-2010^{*}

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Abstract

Aid for fighting infectious and parasitic diseases has had a statistically significant role in the under-five mortality reduction in the last decade. Point estimates indicate a country average reduction of 1.4 deaths per thousand under fives live-born attributable to aid at its average level in 2000-2010. The effect would be an average drop of 3.3 in the under-five mortality rate at the aid levels of 2010. By components, a dollar per capita spent in fighting malaria has caused the largest average impact, statistically higher than a dollar per capita spent in STD/HIV control. We do not find statistically significant effects of other infectious disease aid, including aid for the control of tuberculosis.

Keywords: ODA, child mortality, infectious diseases.

JEL Classification Codes: F35, J13, O15

1 Introduction

According to the WHO, 8.3 million children under five died in 2008, down from 10.5 million in 2002.¹ That represents a 20.5% reduction in the under five mortality rate worldwide. The death toll for the group of causes that includes

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¹We use the figures available from the WHO at http://apps.who.int/ghodata/?vid=10012 (consulted February 22, 2012) since they enable comparison with estimates in 2002 and 2008 using the same methodology. Alternatively, more inclusive estimates for 2008 can be found in Black et al. (2010), and for the 1990-2007 period, in Ravishankar et al. (2009). The declining tendency has continued: WHO estimates for 2012 are now at 7.6 million for 2010 (see http://www.who.int/mediacentre/factsheets/fs178/en/index.html, consulted February 22, 2012)

only communicable, perinatal, and nutritional conditions, which represent close to 90% of all under five deaths, decreased by approximately the same percentage. However, a significantly higher reduction has occurred in the number of deaths attributed to a small subgroup of these causes: HIV, malaria, and tuberculosis.² Since 2002, the reduction in the number of under-five deaths as a result of these three diseases has been 37.6%, almost twice the reduction in the total under-five deaths.

These patterns have been running parallel to official (and unofficial) donors' growing concern about malaria, HIV, and tuberculosis. A symptom of this focused attention was the inclusion of the fight against malaria and HIV as one of the eight Millennium Development Goals. Also in line with this enhanced interest was the creation in 2002 of the Global Fund to Fight AIDS, Tuberculosis and Malaria (GF), or the enactment since 2003 of the U.S. President's Emergency Plan for AIDS Relief (PEPFAR). As a result, official development assistance (ODA) disbursed for the purpose of fighting these three diseases increased from less than \$ 900 million in 2002 to close to \$ 8 bn in 2008. For reference, during the same time period, ODA disbursed for all health sectors increased from almost \$4.5 bn in 2002 to around \$15 bn in 2008.³

Despite some criticism of the excessive emphasis on malaria and HIV,⁴ one might be tempted to conclude that the focus on malaria, HIV and tuberculosis has been appropriate, and has helped to reduce child mortality rates.

Jumping to that conclusion is, of course, unwarranted. Indeed, the causal

 $^{^2\}mathrm{In}$ fact, tuberculosis killed 44% more children under five in 2008, compared with 2002. However, the tuberculosis death toll represented less than 0.5% of all under five deaths in 2002.

 $^{^{3}}$ An alternative estimation is offered by Pitt et al (2010), by completing data from the CRS. According to this estimate, total health ODA increased from around \$ 7.6 bn in 2003 to \$ 15.6 bn in 2008, whereas health ODA directly addressed at maternal, newborn, and child health increased from \$ 2.6 bn to \$ 5.4 bn over the same time span. The DAC IDA for the sample we will use in this analysis increased from \$ 2002 to \$ 6.3 bn in 2008. Of this, the aid for the purpose of fighting malaria, STD/HIV and tuberculosis represented less than a third in 2000, about 50% in 2002, but more than 90% in 2008.

⁴See for instance Liese and Schubert (2009), or England (2008).

relationship between increased aid for these purposes and the reduction in mortality attributable to the three causes cannot be concluded from the parallel developments of aid and mortality.⁵ The conclusion that the choice of focus was correct is even more problematic. This would imply establishing that ODA targeted at malaria, HIV, and tuberculosis had a bigger impact on the reduction in mortality than ODA targeted at fighting other causes of mortality. The goal of this paper is to investigate whether the data available offers evidence to support these two claims. To this end, we investigate the statistical relationship between aid for different purposes and child mortality across time and countries once endogeneity concerns have been taken into account, so that the relationship found can indeed be considered causal.

Mortality, and child mortality in particular, is not the only indicator to measure the impact of aid on health. Nevertheless, this indicator is widely used as the best proxy for needs and results. For instance, the MDG's include the reduction of under five mortality rates as a target in itself. Mortality is easier to measure and less subject to variation of definition than other measures of health.⁶ The reduction in mortality is a common output of health interventions that may be somewhat diverse in their immediate purposes, and so it offers grounds for comparing performance. Finally, child mortality is arguably more directly related to health interventions than adult mortality, and so should reflect the impact of those interventions more clearly. Thus, bearing in mind that child mortality is but one of the indicators of health, we investigate whether its reduction in developing countries is a (partial) consequence of aid for the control of infectious diseases.

⁵ The WHO World Malaria Report, 2010 recognizes the difficulties in establishing causality between measured interventions and malaria outcomes, although it then takes the less-than-

between measured interventions and malaria outcomes, although it then takes the less-thansatisfactory approach of considering a more or less constant health outcome as the right counterfactual.

 $^{^6{\}rm For}$ instance, malaria prevalence indexes are difficult to construct, and data on these indexes is only available for very recent times.

Our approach may be considered complementary to other exercises that attempt to directly measure the impact of certain interventions on related health outcomes. In most cases, these are better instruments for evaluating specific health programmes and testing alternative interventions. However, success in those exercises does not suffice to make a real change happen. As stressed by, for instance, the Task Force on Child Health and Maternal Health, we need to understand what it takes to deploy those proven interventions, an important component of which is the absorptive capacity of recipient health systems. The Task Force puts it nicely: "the transition from efficacy of interventions to effectiveness of delivery strategies is where we so often lose our way" (Freedman et al., 2005). Our exercise helps measure the extent to which the current set of interventions is attaining its goal. If real progress is being made in improving strategies and efficiency, then real progress should also be observed in aggregate health outcomes. Child mortality is perhaps one of the best indices of these aggregate outcomes in recipient countries.

Our task requires the identification of an exogenous variation of ODA for the purposes mentioned. To do that, we use a simple instrumentation strategy that we introduced in a previous article.⁷ This strategy exploits country-specific time trends in ODA in order to establish a causal relationship between ODA and child mortality. The identification strategy is one of the main differences between our exercise and previous ones. Other recent papers find that aid has no causal effect on child mortality.⁸ There are two main differences between that literature and this paper. First, while the previous literature uses data on aid commitments, we focus on aid disbursements. It is well known that aid commitments do not always translate into actual disbursements, which highlights the need to focus on the latter in order to evaluate aid effectiveness. The second is that

⁷See Burguet and Soto (2011).

 $^{^8\}mathrm{Chauvet}$ et al. (2009), Mishra and Newhouse (2009), and Wilson (2011).

we explicitly address the endogeneity of aid to account for the "feedback" from mortality to aid⁹. The estimation techniques used in the existing literature are not appropriate if the shocks to mortality are persistent¹⁰.

We find that infectious disease aid (IDA) has a statistically significant impact on under-five mortality over the 2000-2010 period, whereas other forms of health aid lack significance. We estimate that at the country mean per capita aid, IDA caused an average drop of 1.4 deaths per thousand under-five children born alive over the sample period. The effect is an average drop of 3.3 in the under-five mortality rate at IDA levels of 2010. For reference, the country average under-5 mortality rate in our sample for 2010 is about 54 per thousand.

When we consider the effects of the various targets of IDA, we find that aid for malaria control, which represents about 12 per cent of total IDA in the period, has significantly contributed to the reduction in child mortality. In fact, this type of aid accounts for around two thirds of the reduction in the average child mortality that can be attributed to IDA. Aid for STD/HIV control also had a statistically significant effect on child mortality, although the per capita dollar impact in this sector is statistically smaller than that of aid for malaria control. Finally, aid for the control of tuberculosis and other infectious diseases had no statistically significant impact on under-five mortality.¹¹

Thus, the data suggest that the focus on malaria and STD/HIV in particular may have been well founded. On the other hand, the smaller impact of aid for STD/HIV control in comparison with the impact of aid for malaria control may be interpreted as indication that, as some critics have suggested, there is an overemphasis on STD/HIV control. Note that almost 80% of total IDA disbursed in our sample period accrues to this sector.

 $^{^{9}}$ Deaton (2010).

¹⁰Arellano and Bond (1991) and Blundell and Bond (1998).

 $^{^{11}}$ We find that in Sub-Sahara Africa TB control aid has a significant effect at a 90% confidence level. However non African countries, and Asia in particular, have attracted relatively more aid for this purpose.

We should be quick to point out that there may be reasons to expect a modest impact on child mortality in particular during the very first years of application of the most effective strategies to fight HIV/AIDS, even if the overall effect on the population is sizable. Indeed, most children with HIV acquire the virus from their mothers (MTCT) during pregnancy, delivery, or through breastfeeding. The prevention of this MTCT requires strategies that are either unaffordable for the population most affected or only recently available (c-section, alternative to or prophylactic breastfeeding, etc.).¹² More than in other cases, success in this area may depend on strengthening the general health system, and in particular maternal, neonatal and child health services.¹³ Progress in that area in particular may then be expected to be slower than in the fight against HIV in the population as a whole. Thus, perhaps by only measuring the effect of STD/HIV control on child mortality we are underestimating the effect on total mortality and health outcomes in general. For instance, according to Druce and Nolan (2007), only 8% of all infants exposed to HIV in that year were receiving antiretroviral prophylaxis, whereas for adults the figure was 28%. That said, the data suggests the need to discuss whether the relative emphasis on each purpose is well founded.

2 Data

We use data from the OECD's Creditor Reporting System on aid. This data is classified according to the purpose of aid, country of destination and the year in which disbursements take place. The focus here is on aid to prevent and control infectious diseases. The OECD distinguishes four different types

 $^{^{12}}$ See Newell et al., (2004).

¹³ Antiretroviral therapy for pregnant women seems to reduce the risk of transmission. However, according to the UNAIDS' report, in 2008 only an estimated 18% of pregnant HIV-positive women received testing in antenatal care clinics worldwide, with wide variations across countries. The report explicitly expresses to donors and countries the need for improvement in this aspect.

of aid in this area. These are "Malaria control", "Tuberculosis control" "STD control including HIV/AIDS" and "Infectious disease control" (which includes immunization, prevention and control of any infectious disease not included in the other categories). As is well known, there has been a considerable increase in IDA (Figure 1). Total official aid disbursements rose from about \$527 million in 2000 to more than \$10.5 billion in 2010 (all figures are in constant 2010 prices; aid commitments increased from \$1.8 billion to nearly \$10.8 billion over the same period). This massive increase in IDA is mainly due to a surge in aid to fight STD/HIV, which accounts for more than 2/3 of total IDA in 2010.

It is important to note that the increase in IDA is not homogeneously distributed across countries. In particular, being a high mortality country does not imply that the country receives relatively larger amounts of IDA (Figure 2; in this and following figures we represent the 30 countries with the highest under-five mortality rates in the sample). For instance, the Central African Republic has higher than average mortality and HIV prevalence rates but receives lower than average per capita IDA. More importantly, some countries, like Chad, Guinea and Niger, display a fairly flat evolution of IDA over the sample period, whereas in other countries the evolution of IDA has a markedly positive trend. The heterogeneity in the distribution and evolution of aid also applies to individual IDA categories. Some countries with historically high malaria death rates like Nigeria and Sierra Leone¹⁴ have received relatively modest and constant amounts of aid for malaria control, while others have benefited from large aid increases. A similar situation occurs in the case of aid for STD/HIV control, tuberculosis control and, to a lesser degree, other infectious disease control (figures 4 to 6, respectively). From a statistical point of view, this diversity in the evolution of aid is important because it produces variation in the data which can be used to identify the effect of aid on mortality. As discussed below, this

 $^{^{14}}$ Murray et al (2012).

is the key for our identification strategy.

3 Methodology

To estimate the effect of aid on mortality we follow the approach implemented in Burguet and Soto (2011). In particular, we estimate the following equation

$$m_{it} = \mu_i + \mu_t + \delta a_{it-1} + \beta \boldsymbol{X}_{it-1} + e_{it} \tag{1}$$

where m_{it} is the under-five mortality rate (per thousand children born alive)¹⁵ in country *i* and year *t* (t = 2000, ..., 2010), a_i is the amount of aid (in 2010 dollars) divided by total population in country *i*, X_i is a set of other variables that potentially determine mortality, μ_i and μ_t are country and year specific effects, and e_{it} is a mortality shock. In the regressions that we will discuss, X_i will be an aggregate of other types of aid, different from a_i . The term μ_t captures common time trends in mortality across countries. We seek to obtain reliable estimates of the coefficient δ . If aid effectiveness is small then δ would not be significantly different from zero.

Equation (1) implies that aid received in a given year only affects mortality the following year. This may be unrealistic if some aid programmes (for instance, a vaccination campaign) have long-term effects on mortality. A second concern about the estimation of (1) refers to the potential feedback from mortality to aid. Countries that undergo a mortality surge may attract more aid to try to counteract the increase in mortality. This implies that a simple ordinary least-square (OLS) regression for equation (1), would produce positively biased estimates for the coefficient δ .

We address both problems with two-stage least square (2SLS) estimation.

 $^{^{15}}$ Mortality data is from IGME (2011)

To do so we use two different instrumental variables¹⁶. In most developing countries, IDA displays a positive trend over the sample period. This is the result of the increased global awareness of the need to fight infectious diseases and not the consequence of increases in mortality due to those diseases. For instance, recent estimates for most developing countries find that the probability of death from malaria was smaller in 2010 than in 2000.¹⁷ We exploit this exogenous trend in aid to build an instrument from the predicted values of a regression of per capita aid on a country-specific time trend. These fitted values are strongly correlated with aid itself and thus fulfill the identification condition. We formally test the exogeneity of the trend instrument by performing standard overidentification restriction tests, which do not reject the exogeneity hypothesis. An important additional feature of this instrument is that the estimated parameter δ captures the cumulative effect that aid given in the past might have on current mortality. Thus, our concern for the potential long-term effects of aid on mortality is also addressed by this instrumentation strategy.

In order to perform overidentification restriction tests we require a second (exogenous) instrument. We draw on the strong negative correlation between the size of a country and the amount of per capita aid it receives.¹⁸ The literature on aid effectiveness has used total population as an instrument for aid.¹⁹ Here we use total GDP as an instrument rather than population in order to dissipate concerns about a potential correlation between population size and mortality shocks. Total GDP is also significantly and negatively correlated with per capita aid flows. Moreover there is no evidence that total GDP is correlated with mortality shocks, which validates its use as an exogenous instrument.

 $^{^{16}}$ In Burguet and Soto (2011) we provide a detailed description of the estimation technique. 17 Murray et al. (2012).

 $^{^{18}}$ Easterly (2009).

 $^{^{19}}$ See for example Boone (1996), Burnside and Dollar (2000), Hansen and Tarp (2001) and Clemens et al. (2004).

4 Results

We estimate the effects of IDA and other types of aid (table 1; in this and all subsequent tables we report OLS and 2SLS estimates). That is, in (1) we let X_i be total ODA net of IDA. We find that the coefficient of IDA is negative and highly significant while the coefficient of total non-IDA is not statistically different from zero. We repeat the exercise defining X_i as health aid different from IDA and obtain the same results for IDA and again there is a lack of significance of other health aid. As expected, point estimates from 2SLS are larger (in absolute value) than OLS coefficients, although differences are not statistically significant.

Next, we split IDA into four different purposes: fight against malaria, tuberculosis, STD/HIV, and other infectious diseases. In table 2 we report how these four different components of IDA impact under-five mortality. In each case, we let X_i in (1) be total IDA net of aid for the purpose considered. All coefficients in the 2SLS are negative and larger in absolute value (except in the regression for TB) than the corresponding coefficients in the OLS estimations, although they are not statistically different in the two sets of regressions. That is an indication that OLS coefficients suffer from an upward bias. In other words, aid flows to high mortality countries rather than the other way round.

The most robust conclusion that we can obtain from the results reported in table 2 is that malaria control and STD/HIV control had a statistically significant effect in reducing child mortality. Indeed, in any grouping, the coefficients of aid categories containing these types of aid are significant at a 99% confidence level.²⁰ Moreover, aid groupings not containing aid for malaria control or STD/HIV control are never significant at a 90% (or higher) confidence level. All the coefficients are negative, as we would expect, but the coefficients on "Other

 $^{^{20}}$ The sizes of the coefficients across regressions are coherent, taking into account the relative sizes of the four categories of aid considered.

infectious disease control" and "Tuberculosis control" are not statistically different from zero in either of the regressions.

Point estimates of the 2SLS estimations suggest that, at the average composition in the sample period, a dollar per capita IDA disbursement is generally associated with a country average decline in under-five mortality rate by 0.72 (table 1, confidence interval (0.3, 1.1)).²¹ The sample country-average IDA is \$1.98 per capita, which means that IDA disbursed in the period has reduced under-five mortality by about 1.4 children per thousand children born alive (0.6, 2.2). In 2010, mean IDA disbursements were \$4.64, which indicates that later levels of IDA may contribute to an average reduction of about 3.4 points in the under-five mortality rate.

The regressions by IDA components offer a consistent picture, although the point estimates change. The most informative exercises are those presented in columns 4 and 8, where we regress under-five mortality respectively on malaria control and STD/HIV control using the remaining IDA as an additional co-variate. Column 4 indicates that malaria control disbursements, which amount to 0.22 on average in the sample, have reduced child mortality by almost 1.8 on average (0.9, 2.7). On the other hand, column 8 indicates that STD/HIV control disbursements, averaging 1.43 per capita in the sample, have reduced child mortality by 0.6 on average (0.2, 1.1).

As a robustness check, we replicate the regressions by including other controls that may have some impact on under-five mortality. The results are reported in table 3. Again, we estimate equation (1) using both OLS and 2SLS regressions. This time X_i is a vector that also includes a measure of violent conflict, the proportion of urban population, a measure of trade openness (exports plus imports as a share of GDP), the (log of) per capita income and

 $^{^{21}}$ The maximum 2SLS point estimate corresponds to the regression in column 8 in table 2, where we obtain an average effect of each per capita dollar of a 1.85 reduction in child mortality.

income growth.²² The urban population rate and violent conflict have statistically significant coefficients in all regressions. Income level is significant in some regressions, while trade openness and income growth are not statistically different from zero. All the coefficients have the expected sign. With respect to aid, the sizes and significance levels of the variables are virtually unaffected.²³

To summarize, the evidence suggests that IDA has contributed to the reduction in child mortality in developing countries experienced during the 2000-2010 period. Also, the emphasis on malaria in particular seems to have been well founded. Indeed, in comparison with other health interventions, malaria control seems to have had a clearly higher effect in reducing child mortality. STD/HIV control has also had a statistically significant effect on reducing child mortality, although the effect of a dollar per capita being spent in the average country has had a smaller impact than a dollar being spent for malaria control. Finally, the impact of tuberculosis control on child mortality is tenuous from a statistical point of view.

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 $^{^{22}}$ Data for violent conflict is from Banks (2009). The rest of variables are from the World Bank.

 $^{^{23}}$ We have also split the sample into SSA countries and the rest of the world. The effect in SSA is virtually unaffected with only a slight drop in significance for STD/HIV control. However, in the rest of the world the coefficients lose virtually all their significance. Only tuberculosis control gains some significance. Thus, it seems that IDA has had a statistically significant effect mainly in the African continent.

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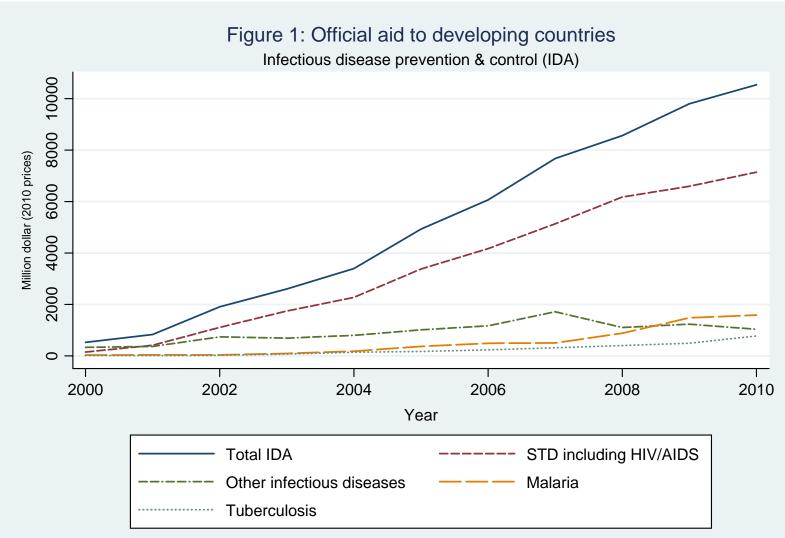
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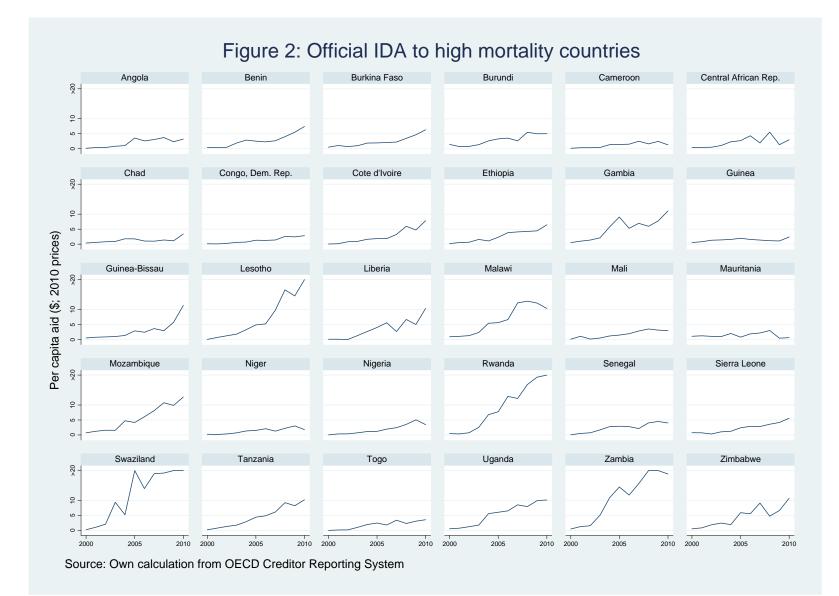
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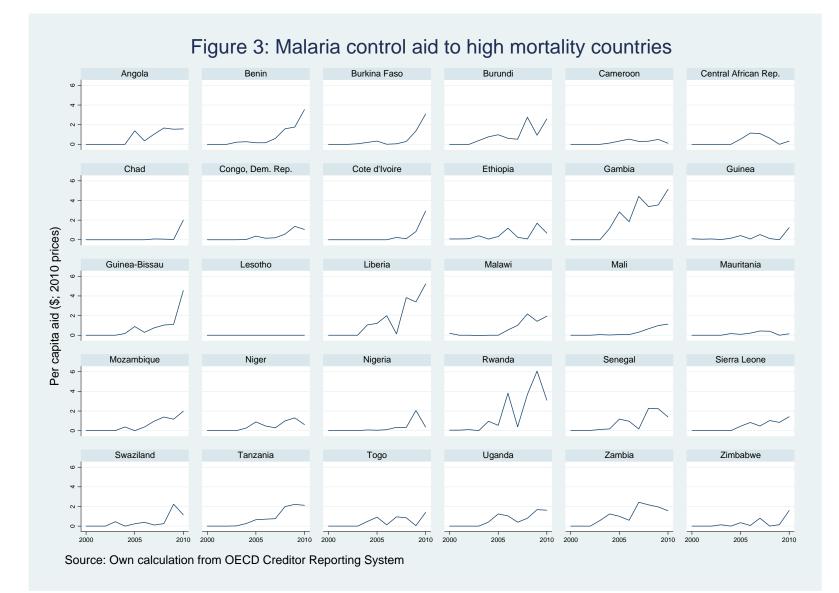
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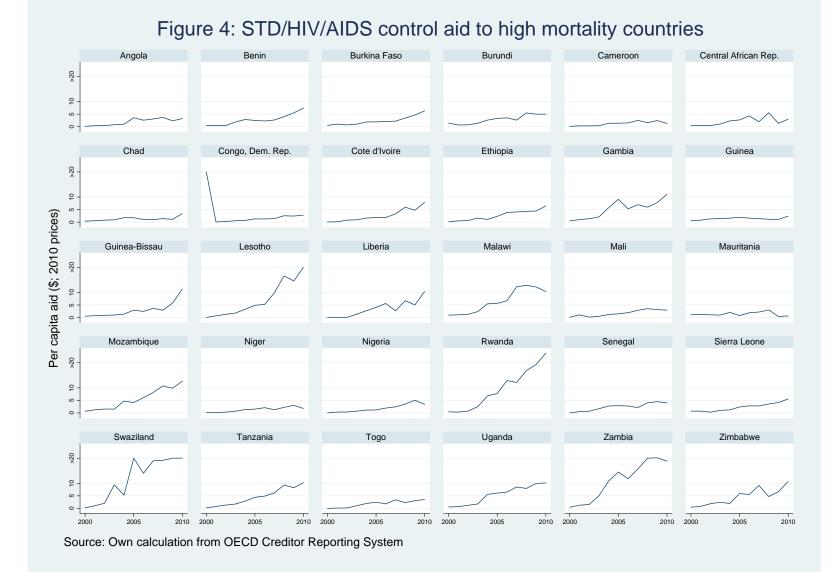
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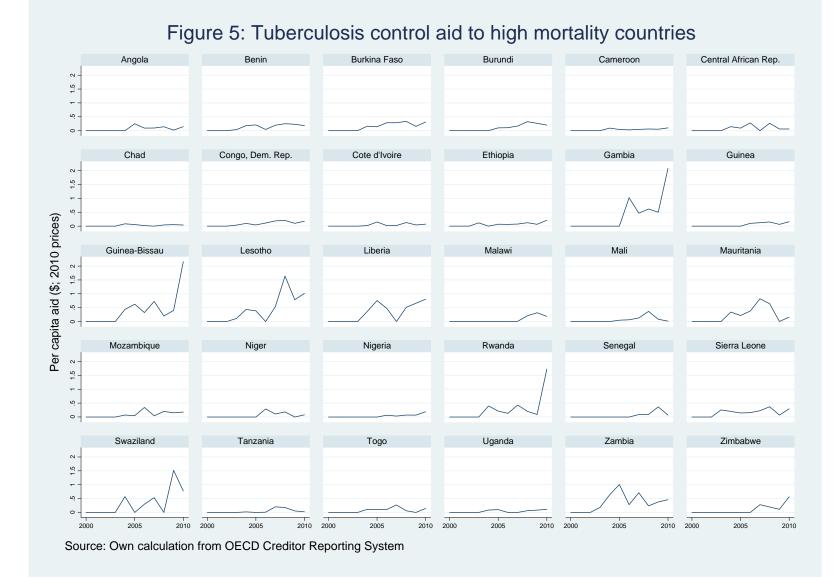


Source: Own calculation from OECD Creditor Reporting System









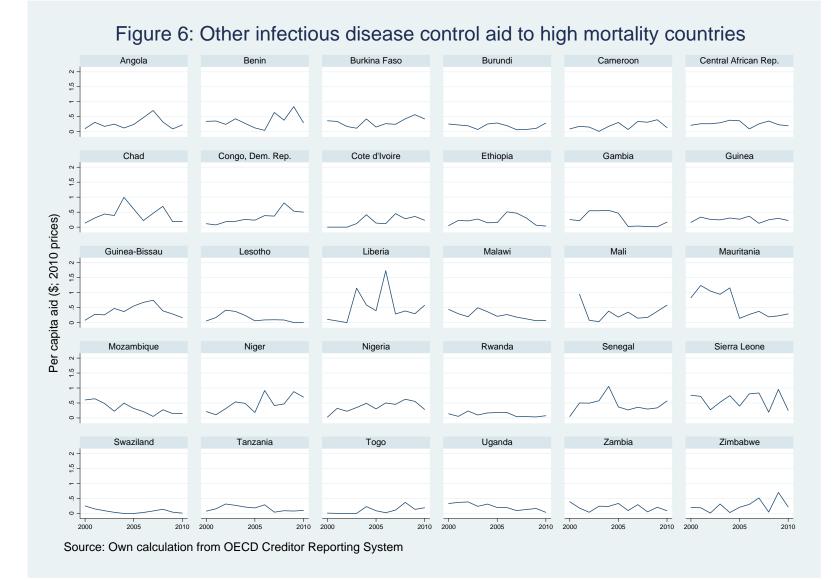


TABLE 1

	(1)	(2)	(3)	(4)
	OLS	2SLS	OLS	2SLS
Infectious disease aid (IDA)	-0.516***	-0.739***	-0.478**	-0.703***
	(0.171)	(0.181)	(0.198)	(0.216)
All other aid	0.00358	0.0170	. ,	. ,
	(0.00549)	(0.0258)		
Other health aid			-0.173	-0.741
			(0.129)	(0.494)
Observations	1,380	1,380	1,214	1,214
R-squared	0.625	0.611	0.604	0.573
Number of countries	130	130	129	129
F-test for instruments in first-stage regression		3844		138.7
Hansen J test p-value		0.259		0.547

Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1 Time and country effects included in all regressions

TABLE 2

	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) OLS	(6) 2SLS	(7) OLS	(8) 2SLS
Aid purpose:								
Other Infectious disease control	-1.281	-4.790						
	(1.346)	(6.043)						
Aalaria control			-3.991***	-8.141***				
			(1.175)	(2.207)				
Tuberculosis control					-2.206	-1.962		
					(2.046)	(4.838)		
STD control including HIV/AIDS							-0.370***	-0.440***
							(0.117)	(0.159)
let IDA	-0.476***	-0.690***	-0.390***	-0.510***	-0.480***	-0.706***	-3.103***	-6.702***
	(0.165)	(0.179)	(0.112)	(0.143)	(0.183)	(0.202)	(0.796)	(1.612)
Dbservations	1,277	1,277	1,376	1,376	1,373	1,373	1,266	1,266
R-squared	0.609	0.592	0.657	0.604	0.625	0.616	0.636	0.571
lumber of countries	130	130	130	130	130	130	130	130
-test for instruments in first-stage regression		116.0		4389		604.4		885.5
Hansen J test p-value		0.579		0.260		0.435		0.162

Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1 Time and country effects included in all regressions All explanatory variables are one-year lagged Net IDA is IDA net of the aid purpose considered

TABLE 3

	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) OLS	(6) 2SLS	(7) OLS	(8) 2SLS
<u>Aid purpose</u>								
Other Infectious disease control	0.376 (1.118)	3.146 (4.844)						
Malaria control	, , , , , , , , , , , , , , , , , , ,	· · ·	-3.686*** (1.145)	-8.110*** (2.140)				
Tuberculosis control					-2.803 (2.384)	-2.189 (5.349)		
STD control including HIV/AIDS							-0.418*** (0.155)	-0.520*** (0.145)
Net IDA	-0.530** (0.209)	-0.798*** (0.210)	-0.436*** (0.145)	-0.577*** (0.123)	-0.505** (0.221)	-0.765*** (0.240)	-2.893*** (0.834)	-6.352*** (1.639)
Other explanatory variables:								
Log(Per capita income)	-6.105* (3.461)	-6.996** (3.232)	-4.648 (3.298)	-7.607** (3.124)	-2.406 (3.432)	-3.039 (3.597)	-5.538 (3.785)	-6.957* (3.815)
Income growth rate	-1.379 (5.524)	-2.695 (5.383)	6.285 (4.876)	9.291* (4.869)	2.464 (5.099)	1.873 (5.018)	2.394 (5.123)	4.846 (4.919)
Trade Openess	-1.256 (2.186)	-1.102 (2.092)	-2.279 (2.432)	-2.058 (2.261)	-2.592 (2.613)	-2.646 (2.628)	-3.038 (2.357)	-3.042 (2.266)
Urbanization rate	-0.868*** (0.311)	-0.815*** (0.294)	-0.861*** (0.283)	-0.597** (0.260)	-1.044*** (0.311)	-0.948*** (0.298)	-0.956*** (0.301)	-0.781*** (0.290)
Conflict	4.860** (1.990)	4.898** (1.960)	4.898*** (1.793)	4.151** (1.677)	5.327*** (1.949)	5.227*** (1.918)	5.289** (2.177)	4.560** (2.022)
Observations	1,188	1,188	1,280	1,280	1,280	1,280	1,174	1,174
R-squared	0.646	0.627	0.690	0.628	0.665	0.654	0.670	0.609
Number of countries	126	126	126	126	126	126	125	125
F-test for instruments in first-stage regression Hansen J test p-value	•	272.7 0.268	•	2073 0.459	•	236.4 0.242	•	707.0 0.532

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1 Time and country effects included in all regressions All variables are one-year lagged Net IDA is total IDA net of the aid purpose considered