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Does Aid Decrease Child Mortality?

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The literature has traditionally found no or little aggregate impact of foreign aid on infant mortality. We suggest that exceptionally high persistence in infant mortality data and sector aid data incompleteness may have played some role in generating such disappointing results. Accounting for both issues, this paper estimates the aggregate impacts of total and sector aid on the neo-natal, infant and under-five mortality rates using fractional estimation techniques for panel data controlling for time-invariant country-specific effects, measurement errors and endogeneity. We confirm that total aid has no impact on child mortality rates. We find mixed evidence that health aid reduces child mortality but robust evidence that agricultural aid has large effects. Aid policies aimed at reducing child mortality in developing countries should recognise the increased importance of targeting the agriculture.

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1. Introduction

The conceptual and empirical debate on aid effectiveness has now long occupied the scientific community and policy makers (Sen, 1999; Easterly, 2001; Sachs, 2005). While foreign aid (or Official Development Assistance – ODA) aims to promote both economic and human development, most studies have mainly concentrated on economic growth (e.g. Burnside and Dollar, 2000; Easterly, 2006; Minoiu and Reddy, 2010). It is only in recent years that the literature has examined the impacts of foreign aid on indicators of human development (Masud and Yontcheva, 2005; Williamson, 2008; Alvi and Senbeta, 2012). Among those studies, several have especially focussed on infant mortality, one of the most imperative Millennium Development Goals (MDG). The attention given to this issue is particularly welcome as it directly affects the potential economic growth of low-income and developing countries. Infant mortality is also considered to be a good indirect indicator of poverty and development because it usually decreases as the benefits from economic development reach the poorest (Mishra and Newhouse, 2009).

Recent research on child mortality has coincided with the recognition in policy circles that aid must be increasingly targeted at improving health and development outcomes. As we approach the end of the MDG era there is a need to evaluate the progress towards human development. Also, credible and relevant action plans must be drawn to support the Sustainable Development Goals (SDG) agenda. While the latter will most likely imply a scaling up of aid inflows in the coming years, research is required to help shaping policies that maximise the impacts of aid on human development. The question of aid effectiveness thus remains essential in the current context.

From a theoretical perspective, foreign aid is expected to promote economic growth and to raise average incomes, especially for the poorest, resulting in higher access and consumption of health goods and services, which should in turn improve children's health outcomes and lower child mortality rates. Those theoretical links have been scrutinised and strongly questioned in the

empirical literature. First, a vast array of the literature has examined the link between aid and economic growth and has not yet established a clear positive relationship between aid and growth. Several authors have found that foreign aid has been effective in promoting economic growth (Hansen and Tarp, 2001; Karras, 2006), while others have shown that foreign aid has no effect, because aid is captured to benefit the wealthy elite of developing countries (Boone, 1996), or can even be detrimental to the recipients because it undermines the competitiveness of the labour-intensive or exporting sectors (Rajan and Subramanian, 2005). Another area of the literature has shown that aid is conditionally effective and usually highlights the importance of good institutions and economic policies (Svensson, 1999; Burnside and Dollar, 2000). Second, even if aid indeed promotes economic growth, the improvements in child mortality will likely not materialise quickly because economic growth has little impact on the nutritional status of children (Vollmer et al., 2014; Mary and Gomez y Paloma, 2014), the main cause of child mortality (Black et al., 2013).

Yet, while the evidence supporting the (economic) growth-based transmission channel between aid and child mortality appears quite tenuous, it is conceptually possible that foreign aid has a direct effect on child mortality (without any effect on economic growth) through policies and/or investments specifically targeted at populations at risk, and/or at specific areas, and/or the implementation of diverse interventions including the provision of vaccines, vitamin supplements or sanitation infrastructures. Indeed, at the micro-level, there are numerous reports and case-studies highlighting the effectiveness of such sectoral interventions, which seem to indicate that aid works to improving many children's daily lives and motivate the existence of development assistance (Levine, 2004; Riddell, 2007; Bhutta et al., 2008). However, at the macro-level, the empirical literature has typically found that total aid has no significant aggregate impact on infant mortality (Boone, 1996). One of the advanced explanations for this finding is that total aid may not be specifically dedicated to improving health outcomes and that the relationship between the two variables is therefore difficult to establish.

Recent studies have thus investigated sector-specific aid, with a quasi-exclusive focus on health, but similarly have found no or little impact. In particular, Williamson (2008) finds that health aid does not affect infant mortality using a sample of high-, medium- and low-income countries observed between 1973 and 2003. Her static analysis accounts for potential endogeneity biases using linear two-step least squares (2SLS) estimation and concludes that

health aid is an ineffective human development tool. In a similar vein, Wilson (2011) concludes that health aid does not affect infant mortality in poor and high mortality countries observed between 1975 and 2005 using static and dynamic models, estimated through a variety of techniques (Ordinary Least Squares – OLS, Fixed Effects, and Generalised Method of Moments – GMM). It is noteworthy to mention that, despite their conclusions, both papers also report a few statistically significant and positive estimates on the effects of aid on infant mortality (though of little economic importance) – in other words, foreign aid would increase child mortality (see Table 2 in Williamson, 2008; Table 1 in Wilson, 2011) – illustrating the practical difficulty in estimating the relationship between aid and infant mortality.

In an earlier paper Mishra and Newhouse (2009) use a dynamic modelling framework and system GMM estimation to test the effects of health aid on infant mortality between 1973 and 2004. Unlike Williamson (2008) and Wilson (2011), they find that health aid significantly reduces infant mortality but that the estimated effects are small. In sum, the existing evidence supports the view that the micro-macro paradox, the somewhat puzzling combination of evidence that aid works at the project level and the absence of aggregate effects, would empirically apply in the case of aid and infant mortality.

However, one must be cautious in hastily concluding that aid has not been effective because all the above mentioned studies share several common methodological characteristics that may have resulted in estimating practically inexistent aid effects. First, most studies, with the exception of Boone (1996), use the logarithm of infant mortality as dependent variable (often as a 3 or 5-year average) in their analysis so it assumes values on the real line. This is because infant mortality data are fractional (or proportional) data, bounded between 0 and 1, and are not by definition defined by the domain over which the normal distribution is defined (Figure 1). The problem is that this log-transformation exhibits exceptionally high persistence and may even contain a unit root[1]. For example, the existence of unit roots is implicitly acknowledged in Mishra and Newhouse (2009) as the estimated coefficient on the lagged dependent variable is greater than 1 (See Table 1 in their paper). The potential presence of unit roots implies that previous regression analyses may be spurious (Granger and Newbold, 1974) and as a result any evidence on the

[1] We provide evidence in the Appendix A1 that this transformed variable contains a unit root in our dataset.

(absence of) causality between aid and infant mortality found in these papers may be unreliable[2] (besides explaining the positive estimates between aid and mortality found in the literature).

[INSERT FIGURE 1]

Even if the unit root problem does not affect other studies in the literature, the high persistence in the (transformed) dependent variable has direct implications for the estimations of aid effects. For example, the weak instrument problem in the case of persistent data (Blundell and Bond, 2000) may explain why the GMM estimates in Table 1 in Wilson (2011), though of the expected negative sign, are not statistically significant[3].

Another key issue in the literature is the fact that previous studies have used fairly incomplete sector aid data for their estimation periods. The incompleteness of sector aid data may have potentially contributed to under-estimate the impacts of sector ODA as the pre-2002 annual coverage of sector disbursements is below 60 per cent. This problem is so acute that the OECD does not recommend using earlier data for sector analysis (OECD, 2014). Since 2002 the coverage of sector disbursements is around and over 90 per cent and reached nearly 100 per cent starting with 2007 flows. Despite the fact that the new database on sector disbursements has been recording information for several years, and it is now possible to use it for empirical analysis in a panel data context, no paper has yet focussed on this more reliable database in the empirical literature.

More fundamentally, child undernutrition has been found to be the main cause of child mortality, and as such, any intervention that has links with nutrition may be expected to lower child mortality rates. Yet, past studies have ignored other types of sector aid that have *de facto* such links with nutrition; one can logically expect that foreign aid specifically targeting agriculture through irrigation systems or soil improvements, could structurally address child undernutrition. In fact, Smith and Haddad (2015) show that improvements in the quantity and quality of food available have been key drivers in past child stunting reductions and that the

[2] The unit root problem typically results in inflated t-values, suggesting (increased) statistical significance.

[3] Additionally, as child mortality data typically display asymmetry, any inference based on traditional linear regression techniques is likely to be misleading (Kieschnick and McCullough, 2003; Ferrari and Cribari-Neto, 2004) and may mask the statistical significance of existing aid impacts. In particular, Kieschnick and McCullough (2003) imply that standard errors may be biased upwards in the case of proportional data.

benefits can be quick to materialise. This confirms that nutrition-sensitive aid, such as agriculture-specific aid inflows, may have substantial impacts on child mortality in the short run and deserve some attention in the empirical literature.

Given this background, this paper provides new evidence on the aggregate impacts of total and sector aid inflows on the infant mortality rate, as well as on the neo-natal and under-five mortality rates, and accounts for the data incompleteness problem by limiting the analysis to the new database on sector disbursements available from 2002 onwards. The focus on the aggregate impacts of aid aims, first, to sidestep concerns over the cumulative and long-lasting efficiency of aid at the micro-level, sometimes emphasised in the media coverage of the failure of individual projects (for instance, see The Economist, 2012) and, second, to answer the more substantial question whether aid makes a significant overall contribution to human development (Riddell, 2014); put simply, the paper answers the following question: does aid make a contribution to reducing the number of neonate, infant and under-five child deaths in developing countries?

From a methodological perspective, when the dependent variable is non-stationary, the traditional solution is to first-difference the dependent variable. Then, the estimation of the relationship between aid and infant mortality can typically be performed using GMM approaches to account for potential endogeneity. The problem is that the underlying assumptions of GMM approaches may not be always be satisfied and sometimes prevent the use of such estimators. Specifically, the difference GMM estimator suffers from the weak instrument problem in the case of persistent data and the system GMM estimator, preferable with persistent data, requires strong additional restrictions that may not always be applicable[4].

In the face of such practical issues, there is however a more natural and intuitive approach to solving this problem, which allows modelling the infant mortality rate without any prior transformation, therefore preventing the unit root problem and lessening estimation concerns resulting from exceptionally high persistence[5]. In this paper, we use the nonlinear model for fractional responses for panel data developed by Papke and Wooldridge (2008), based on the Generalised Estimating Equations (GEE) framework. Their approach allows for unobserved

[4] We originally estimated the model of Mishra and Newhouse (2009) with the first-differenced log-transformed dependent variable (Δlny) but the underlying assumptions of the system GMM estimator were not fulfilled (in particular, on the autocorrelation of residuals) therefore preventing its use. The use of deeper lags did not resolve the issue.

[5] Evidence in Appendix A1 indeed suggests that the mortality rate shows less persistence than its log-transformation.

heterogeneity (or fixed effects) and corrects for endogeneity, through a time-varying omitted factor that can be correlated with explanatory variables to account for country-specific factors that influence the dependent variable (e.g. measurement errors). We also control for multiple factors that are likely to affect child mortality, such as economic growth, fertility, or HIV, as we use the parsimonious empirical modelling framework proposed by Mishra and Newhouse (2009).

This paper extends the previous literature in several ways. First, we estimate the impacts of total and health aid inflows on the infant mortality rate, as well as on the neo-natal and under-five mortality rates. The estimations at different child ages allow us investigating the existence of aid impacts across time (if there are any). Second, we extend the analysis to sector-specific aid that has not yet received attention in the literature but may affect child mortality through nutrition channels, that is, aid inflows specific to agriculture. Third, we use a new identification strategy that has never been applied yet in the literature on aid effectiveness and human development and naturally fits the proportional nature of child mortality data. Fourth, this paper is the first paper to restrict the empirical analysis to the more reliable sector aid database available since 2002. While there is an obvious Cornelian dilemma between data quality and quantity, our empirical results will at least explicitly clarify whether the absence of substantial aid impacts in previous studies has been caused by sector data incompleteness. Fifth, we evaluate the robustness of our main findings to numerous alternative specifications, namely, several alternative aid variables and instrumentations, the inclusion of outliers, additional control variables in an extended empirical model, the use of three-year averaged variables in the empirical model and the use of alternative linear estimation techniques.

Using annual data from 2002 to 2012, we find that total aid has no impact on child mortality. This is in line with the previous literature. Evidence also suggests sector aid can be effective, but not all sector-specific aid inflows are equally important. In particular, we find some sparse evidence that health aid inflows significantly decrease the infant and under-five mortality rates. We also find strongly robust evidence that aid specific to agriculture has significantly large impacts on infant and under-five mortality rates. A 1-dollar increase per year in agriculture aid per capita would result in a decrease in the infant mortality rate by almost 1 death. Overall, our results imply that aid policies aimed at reducing child mortality in developing countries should recognise the increased importance of targeting the agriculture. The remainder of the paper is as follows. Section 2 describes the modelling framework and discusses the causal identification of

aid impacts. Section 3 presents the data. Section 4 analyses the estimation results. Section 5 concludes.

2. Modelling framework

2.1 Empirical model

The empirical model builds upon the linear framework that has been traditionally used in the literature and can be expressed as:

$$y_{it} = x_{it}b_{it} + c_i + \mu_{it} \quad (1)$$

Where countries are indexed by i and year is indexed by t; y_{it} is the child (neo-natal, infant or under-five) mortality rate of country i in year t; x_{it} is a vector of independent variables (that is, aid, GDP per capita, fertility, population); $c_i$is the country-specific (time-constant unobserved) fixed effect.

As explained in the introduction, the use of linear models for fractional dependent variables may not result in an accurate assessment of the impacts of aid on child mortality. Following Papke and Wooldridge (2008) who suggest a nonlinear model for fractional dependent variables for panel data, and given the linear representation in Equation (1), we specify the nonlinear model for fractional data:

$$E(y_{it}|\, x_{i1}, x_{i2} \ldots x_{iT}, c_i) = \Phi(x_{it}b_{it} + c_i) \quad (2)$$

Where Φ is the standard normal cumulative distribution function. Let's note that this model also includes fixed effects that can be correlated with explanatory variables to account for time-invariant country-specific factors that influence child mortality.

This model assumes that all independent variables are exogenous. Yet the problem of aid endogeneity has generated much discussion in the literature and, if ignored, can lead to wrong conclusions. Let's now assume that one of the independent variables d_{it} may be (potentially) endogenous (for example, aid) while the remaining independent variables z_{it} are exogenous. We can reformulate the model in Equation (2) as:

$$E(y_{it}|d_{it}, z_i, c'_i, v_{it}) = \Phi(\alpha d_{it} + b'_{it}z_{it} + c'_i + v_{it}) \quad (3)$$

Where c'_i is the time-constant unobserved effect and v_{it} is a time-varying omitted factor (e.g. measurement error) that can be correlated with d_{it}.

For the sake of simplicity in expressing the modelling framework above, we only consider contemporaneous independent variables. In practice, we follow closely the empirical model from Mishra and Newhouse (2009) and therefore the list of independent variables includes one-period lagged values of GDP per capita, fertility and population, a dummy for the contemporaneous existence of wars and severe conflicts and a contemporaneous value of HIV/AIDS rate.

2.2 Identification and estimation

Several biases may complicate the causal identification between aid and infant mortality. For example, the existence of time-invariant omitted variables in the empirical model may result in simultaneous causation. This is because omitted variables may both cause aid and mortality. However, this can be easily accounted for using estimators with time-invariant country fixed effects. In a somewhat related manner, short time series can bias the identification of causal effects as they are often characterised by measurement errors. We account for such potential bias in our estimation strategy as the model allows for time-varying country-specific effects.

More importantly, the endogeneity bias may lead to reverse causality between aid and infant mortality, that is, mortality causes aid. If donors are motivated by the desire to help failing countries and behave like Good Samaritans, aid will flow to countries with high infant mortality rates; higher aid could be correlated with higher mortality but it does not mean that aid causes increases in infant mortality. If such endogeneity is not accounted for, the econometric estimation might incorrectly imply aid ineffectiveness, or worse, conclude that aid is detrimental to recipient countries. On the other side, if donors are more pragmatic and want to "make aid count", aid will flow to successful countries with (already) declining mortality rates; higher aid could then be correlated with lower mortality, but again, this would not mean that aid causes decreases in child mortality. In that case, this would wrongly prove aid effectiveness.

The problem of endogeneity is evidently not trivial and needs careful treatment. It is also important to mention that such endogeneity biases may not be homogenous throughout aid type. The rationale for the endogeneity of total aid is not obvious in the case of infant mortality, but it may be more relevant for health and agricultural aid as they have a much more direct relationship

with child mortality. The endogeneity of aid (as well as other independent variables) can be tested. Following Papke and Wooldridge (2008), in a first step, we assume a linear reduced form for the (potentially) endogenous variable that may be correlated with unobserved heterogeneity and the time-varying omitted factor and estimate the coefficients using pooled OLS estimation with robust standard errors. In a second step Equation (3), augmented of the first-step residuals, is estimated using the pooled Quasi-Maximum Likelihood Estimation (QMLE) developed by Papke and Wooldridge (1996). The approach allows testing the endogeneity of independent variables in a very similar way to a Hausman test.

If the test does not reject the exogeneity of all independent variables (typically, at 5 per cent), the empirical model can be estimated with either the pooled QMLE or a GEE approach as described in Papke and Wooldridge (2008). Here, we use the GEE approach with the exchangeable correlation structure. On the contrary, if the test rejects the exogeneity of aid (or other variables), we use several strategies to account for endogeneity and disentangle correlation from causation. Mainly, we rely on instrumentation and the two-step approach defined above, with the first-stage regression estimated with robust OLS estimation and the second-stage regression, augmented of the first-stage residuals, estimated with the pooled QMLE. Standard errors in the second step are adjusted using bootstrapping (500 replications). In the paper we provide average partial affects (APE) that represent the partial effects across the population and are computed on scaled coefficients. APE are directly comparable to linear estimates found in the literature.

The search for valid instruments has been difficult and several instruments have been used in the literature (e.g. Burnside and Dollar, 2000; Masud and Yontcheva, 2005). In this paper we follow an approach that has been extensively used in the literature (e.g. Boone, 1996; Hansen and Tarp, 2001; Williamson, 2008) and in which aid is instrumented with two-period and three-period lags of aid[6]. In fact, our econometric approach mirrors the methodology used in Williamson (2008) though in a nonlinear setting. In line with Boone (1996), she argues that twice-lagged (and deeper) aid variables can be valid instruments because aid is given as a strategic and political move and represents the interest of donors. From a methodological perspective, the rationale for using lagged aid as instruments is also much stronger in our paper

[6] We use a slightly over-identified model but we also test a model that is just-identified as well as a model with deeper lags. Results are displayed in the robustness analysis.

than in the growth literature which typically uses dynamic models. In a dynamic model, the inclusion of a lagged dependent variable could make instruments dated t-2 invalid in the presence of serial correlation. Instead, we use a static model, and there is no risk of making the instruments invalid.

In addition, we test the relevance and validity of the instruments. We report F-statistics to assess the relevance of our instruments. In particular, if the F-statistic is above 10, we consider the instruments to be relevant. We use Hansen's J-test to investigate the exogeneity or validity of our instruments[7]. This test tests the null that the lagged aid instruments are uncorrelated with the error term. If the p-value is above 5 per cent, all instruments are exogenous and therefore valid.

Finally, we focus on sector aid that is expected to have an immediate or early impact on child mortality. This typically weakens the possibility of endogeneity as lagged values of sector aid are unlikely to have long-lasting impacts, especially considering their smaller size. Third, we reduce the risk of reverse causation by lagging aid one period; this introduces a short delay for aid to have an impact on child mortality. The last two steps, lagging aid and focussing on early impact aid, have already been used in the literature to eliminate potential reverse causality (Clemens et al., 2011).

2.3 Timing of aid effects

Following Boone (1996), most studies traditionally used averages over 3 or 5 years to minimise the risk that time-varying country-specific factors are correlated to foreign aid and then bias the estimation results, though there are also several studies using annual data in the empirical literature (e.g. Masud and Yontcheva, 2005; Karras, 2006). Given the difficult trade-off between data reliability and the quantity of information available (that forces us to consider a rather short dataset), we use annual data instead of three-year averages because it would otherwise result in a substantial loss of information. This is not the first study to rely on a short dataset. For example, Masud and Yontcheva (2005) estimate the impacts of foreign aid on several indicators of human development using a dataset covering 12 years. Any concerns about measurement errors (or

[7] We use Anscombe's residuals to calculate the J-statistic. We also test a version of the test using Pearson residuals. The implications of the different tests are the same in all cases.

other factors) are lessened because the econometric approach directly controls for time-varying omitted factors that can be correlated with explanatory variables.

From an empirical perspective, the use of annual data can also be justified by the fact that health and agricultural aid inflows are likely to have short-term effects on child nutrition and therefore could be expected to result in lower child mortality rates within the short run. In any case, we also test the robustness of our empirical results based on annual data against the use of three-year averages.

3. Data

We collect data for a sample of 86 low-income, lower-middle and upper-middle income countries between 2002 and 2012. The list of countries can be found in appendix A2. The rates of neo-natal mortality, infant mortality, under-five mortality are taken from the World Development Indicators (WDI) database. Infant mortality is the number of infants dying before reaching one year of age, per 1,000 live births in a given year. Neonatal mortality rate is the number of neonates dying before reaching 28 days of age, per 1,000 live births in a given year. Under-five mortality rate is the probability per 1,000 that a new-born baby will die before reaching age five, if subject to current age-specific mortality rates.

Aid data come from the Creditor Reporting System (CRS) of the Organisation for Economic Cooperation and Development (OECD). The database details ODA disbursements (in US$ constant 2012) for total aid and sector aid; in particular, we collect sector ODA specific to health, agriculture, education, water and sanitation. The OECD defines disbursement as the placement of resources at the disposal of a recipient country or agency, or in the case of internal development-related expenditures, the outlay of funds by the official sector. As explained earlier, the OECD does not recommend using pre-2002 sector data because the coverage is largely incomplete before 2002.

Several other variables are taken from the WDI database, such as GDP per capita (in constant US$ 2005), total population and fertility. The total fertility rate represents the number of children that would be born to a woman if she were to live to the end of her childbearing years and bear children in accordance with current age-specific fertility rates. The prevalence of HIV refers to the percentage of people aged 15-49 who are infected with HIV and is taken from the

World Health Organisation (WHO) database. The war dummy represents the existence of wars and severe conflicts in a country and relies directly on data from the Heidelberg Institute. Data for the additional control variables included in the extended model respectively come from the International Disaster Database EM-DAT for the occurrence of natural disasters, from the WDI for public health expenditure per capita in constant dollars and from the Polity IV project for the level of democracy (Polity2). Descriptive statistics are shown in Table 1[8].

[INSERT TABLE 1]

4. Results

The aim of this paper is to estimate the impacts of total and sector-specific aid inflows on the neo-natal, infant and under-five mortality rates, with a focus on health and agricultural aid. We include constant terms, year and region dummies in all regressions. The unbiased sandwich estimate of the variance is used in the calculation of clustered robust standard errors. We report the APE (calculated at the means of independent variables) that are directly comparable with coefficients obtained from linear estimations that can be found in the literature.

4.1 Total aid and child mortality

Table 2 displays the estimation results of the impacts of total aid on the neo-natal, infant and under-five mortality rates. Because aid variables are expressed in US dollars per capita, the APE is directly and easily interpretable as the result of a 1-dollar increase in (total or health) aid per capita in year t on the rate of infant (neo-natal or under-five) mortality in $t + 1$.

[INSERT TABLE 2]

[8] The Hadi procedure is used to remove outliers; in total 19 observations are removed.

The first result we can extract from Table 2 is that total aid is exogenous in all regressions according to the exogeneity tests. This seems to suggest that total aid inflows are not dependent on child mortality rates in recipient countries. Similarly, all other independent variables are exogenous. More importantly, foreign aid has no impact on any child mortality rates. All APE of interest are insignificant. This result is consistent with the previous literature (e.g. Boone, 1996) and has been explained by several factors. First, it is possible that aid generates both positive and negative effects that end up offsetting each other. For example, while aid provides a stream of additional income to the recipients' budgets, it may also deteriorate their competitiveness (Rajan and Subramanian, 2005). More importantly, aid may be fungible and the practical allocation of funds may not be related to the original objectives. In a somewhat related explanation, Boone (1996) claims that aid is captured to benefit the wealthy elite of developing countries. From a methodological perspective, the detection of any significant positive impacts might also have failed because of the complexity of transmission channels between aid and development (Mishra and Newhouse, 2009). Also, some have argued that overall aid may not be a good indicator as aid programs have multiple objectives and that analyses should be instead looking at the impacts of sector aid.

4.2 Sector aid and child mortality

Now, if we look at sector aid, we find in Table 3 that health aid may have sizeable impacts on the infant and under-five mortality rates. A doubling of health aid would result in 1.7 fewer infant deaths (or a decrease of 3.7 percentage points in the infant mortality rate). In comparison with Mishra and Newhouse (2009) who report that a doubling of health aid would reduce infant mortality by 1.5 deaths or 2 percentage points, our estimate implies much larger relative impacts. However, the estimate is somewhat imprecise (p-val.: 0.11).

[INSERT TABLE 3]

What's more, as the APE in Table 3 is twice larger for under-five mortality than for infant mortality, the corresponding positive effect of health aid is even larger. Increasing health aid per capita per year from US$3.36 to US$6.72 would result in a 5.6 percentage point decrease in the

under-five mortality rate. As the probability for health aid to be specifically spent on health-related projects is higher than for total aid (that has economic, political or heath objectives), it should logically have larger impacts on health outcomes than total aid. Results in Table 3 indeed appear to confirm this conjecture that health aid is indeed better at reducing child mortality than total aid and arguably support the view that the empirical degree of fungibility of health aid is relatively lower. F-tests and J-tests indicate, respectively, that the instruments are relevant and valid for both the infant and under-five mortality regressions. Finally, endogeneity tests reveal that health aid is endogenous while other independent variables are exogenous.

Furthermore, we examine the impacts of agricultural aid on child mortality. It is particularly relevant to look at the impact of such aid type because of the role of nutrition in causing child mortality. As for health aid, we confirm that agricultural aid is endogenous (while other independent variables are exogenous). The main finding in Table 4 is that agriculture aid decreases the infant and under-five mortality rates. The APE of the lagged agriculture aid for the infant mortality regression is -0.938 and significant at 1 per cent. This implies that a 1-dollar increase per year in agriculture aid per capita would result in a decrease in infant mortality by almost 1 death. This estimate is much higher than the one obtained in Table 3 for health aid (i.e. 0.5 deaths). Similarly, the impact of agricultural aid is also higher than for health aid on the under-five mortality rate. For example, increasing agricultural aid per capita by US$3.36 (that is, the amount necessary to double health aid per capita) would result in 3.1 fewer infant deaths; that is, a decrease in the infant mortality rate of about 6.8 percentage points, against *only* 3.7 percentage points with a similar absolute monetary increase in health aid. Again, F-tests and J-tests indicate, respectively, that the instruments are relevant and valid for both the infant and under-five mortality regressions.

Next, we turn to the other independent variables in Tables 3 and 4; across all regressions we find some sparse evidence that fertility decreases child mortality rates. This is somewhat in line with Mishra and Newhouse (2009) who report a negative relationship between fertility and infant mortality. Logically, the existence of war and severe conflicts affects positively child mortality as the sign of the war dummy is significant and positive in almost all regressions. Last, there is some evidence that the HIV/AIDS epidemic plays a role in child mortality as an increase of 5 per cent in HIV prevalence increases the infant mortality rate by 0.7 deaths, according to Table 3.

[INSERT TABLE 4]

The literature on aid effectiveness has previously shown that empirical results can be particularly model-driven as the inclusion of outliers and/or additional control variables in the modelling framework or the use of various estimation techniques can result in dramatically different empirical results (Easterly, Levine and Roodman, 2004). In the next section, we proceed to an extensive robustness analysis to test the sensitivity of our empirical results to numerous alternative specifications.

4.3 Robustness analysis

We re-examine our main results across 9 alternative specifications for health and agricultural aid inflows for the neo-natal, infant and under-five mortality rates[9]. We do not report robustness analysis for total aid because we find no impact across all alternative specifications[10]. Robustness analyses use various alternative specifications: a different sample where the 19 identified outliers are not excluded; models with different aid *explanatory* variables to investigate the possibility of different transmission channels (contemporaneous aid and two-period lagged aid, lagged aid in logarithmic form); models with different instrumentations (one model where aid is instrumented with two-period lags of aid, and the other where aid is instrumented with two-period, three-period and four-period lags of aid); a model with additional controls, namely the occurrence of natural disasters, public health expenditure per capita and the level of democracy; a model estimated with linear 2SLS and a model using three-year averages. Tables 5 and 6 summarise the estimation results from 54 regressions[11] and only report the APE for aid variables that are our main interest in this paper.

Table 5 provides some mixed evidence of health aid impacts on the infant and under-five mortality rates. In particular, we find estimates of the same order of amplitude to those reported

[9] Given the large number of tests in the paper, Bonferroni-corrected p-values would help comparing all specifications. While we do not explicitly report such p-values in our tables, an easy and practical way to account for such adjustment is to discard all results that are not strongly significant in the paper (that is, significant at 1 per cent).

[10] We also evaluate the exogeneity of independent variables at the 10 per cent (instead of 5 per cent) to see whether our estimation results would be dramatically different. All results hold when we do and the estimates are of the same order of amplitude.

[11] The full set of results is available upon request.

in Table 3 when using an alternative sample, the just-identified model, the model estimated with 2SLS or the three-year averages model. The impacts are statistically significant and relatively large. However, across other specifications, health aid is not found to have any statistically significant and substantial effects on the infant mortality rate. In the few instances when the effect is estimated with precision (Models 2 and 3), it is much smaller than those reported in Table 3[12]. For example, the coefficient in the model using the twice-lagged aid explanatory variable is -0.160 (against -0.519 in Table 3). In other words, we do not find robust evidence that health aid has significant impacts on infant mortality, as the results seem to be very dependent on model specification. However, alternative estimations are not as problematic in the under-five mortality regressions. We report relatively robust evidence that health aid is effective in reducing the under-five mortality rate; out of all alternative specifications, this result holds in 7 specifications. Such results indicate that health aid may be an effective development tool. This is in contrast with Williamson (2008), Wilson (2011) and to a lesser extent with Mishra and Newhouse (2009). Yet, the degree to which such sector aid is effective is not precisely estimated. The range of estimates is especially wide as the impact of a 1-dollar increase in health aid could result in between 0.3 and 1.6 fewer under-five deaths.

[INSERT TABLE 5]

Table 6 report results on sector aid inflows specific to agriculture. First, there is some evidence that agriculture aid decreases neo-natal mortality. Using the logarithmic model, a doubling of aid would reduce the neo-natal mortality rate by 3.1 percentage points. Similarly, this type of aid has significantly large impacts on the infant and under-five mortality rates for all alternative specifications. For instance, results from the logarithmic model imply that a doubling of aid would result in 4 under-five fewer deaths (or a decrease in the under-five mortality rate of 5.9 percentage points). Based on the model using two-period lags of agriculture aid as regressor (specification 3 in Table 6), results suggest an even stronger impact on child mortality. A 1-dollar increase in this type of aid per capita generates 2.5 fewer under-five deaths. This implies

[12] One of the explanations for this is that in those alternative specifications, the endogeneity tests support health aid exogeneity.

that increasing yearly agriculture aid per capita by US$3.17 would decrease under-five mortality by about 12 percentage points. Even if we use the model based on three-year averages or the linear 2SLS estimation, the same pattern of results remains though estimates are slightly lower. Overall, we find strongly robust evidence that agricultural aid decreases infant and under-five mortality rates and weak evidence that it causes decreases in the neo-natal mortality rate.

[INSERT TABLE 6]

5. Conclusions

Many non-governmental organisations and policy makers have called for a massive scaling up of aid under the SDG agenda. This is despite much of the scientific literature that has struggled to find any sizeable aggregate impact of aid on human development, especially infant mortality. The general absence of consensus between policy practitioners (whose beliefs are clearly established by the recommended scaling up of aid) and the scientific community on the existence of aid impacts and on their potential amplitude has generated much criticism and doubt in the public eye on the importance of aid programs and has led some to dispute the reason of their existence. While many explanations have been proposed to justify the absence of impacts, we suggest that the previous literature on infant mortality has potentially been affected by, first, inappropriate modelling choices for fractional and inherently persistent data and, second, data incompleteness, and as a result that previous evidence on aid effectiveness may be unreliable. Instead, our paper accounts for both issues and relies on a new approach to estimating the impacts of aid on child mortality that embraces the specific nature of child mortality data.

Using the fractional modelling framework developed by Papke and Wooldridge (2008) and data between 2002 and 2012 for 86 low-income and middle-income countries, we estimate the impacts of total and sector aid inflows on the neo-natal, infant and under-five mortality rates. We find that total aid has no impact on any child mortality measures. This is consistent with the previous literature. More fundamentally, we find that sector aid can be effective, but not all sector aid inflows are equally important. We find mixed evidence that health aid inflows are effective in reducing infant mortality. Given the lack of robustness of health aid impacts on infant mortality, our results would somewhat be in line with the existing literature. However, we

find relatively robust evidence that health aid is effective in reducing under-five mortality. The estimated effects are not trivial and much larger than those reported in Mishra and Newhouse (2009) in the case of infant mortality. Increasing health aid per capita per year from US$3.36 to US$6.72 would result in a 5.6 percentage point decrease in the under-five mortality rate. Such findings seem to depict a key role for health aid in promoting human development, and as such, should support the allocation of aid towards health interventions.

Furthermore, we find strongly robust evidence that agriculture aid has large reducing impacts on infant and under-five mortality rates. For example, a 1-dollar increase per year in agriculture aid per capita would result in a decrease in infant mortality by almost 1 death and a decrease in under-five mortality by approximately 1.6 deaths. Why is agricultural aid particularly effective at reducing child mortality? Perhaps, the answer lies in the nature of agricultural aid. A quick look at the sub-components of the latter indicates that a large proportion of this aid is arguably dedicated to addressing market failures, increasing farmer's productivity and developing and strengthening existing agricultural markets. In other words, it is conceivable that agricultural aid is effective because of the incentives it generates and/or alters for recipients. In turn, such incentives may improve farming practices, agricultural outputs and nutrition outcomes in the regions that are especially affected by child mortality. Such transmission channels are probably reinforced by the existence of strong linkages in low-income economies that relate farming performance and household welfare. Additional empirical research, using data on sub-sectoral aid, is needed in order to confirm this conjecture. Regardless, our results portray agricultural aid as an effective tool to fight child mortality and promote human development. This is somewhat in line with Kaya et al. (2013) who find that agricultural aid reduces poverty.

Given the current context, in which action plans are currently designed for the SDG agenda, our results have clear implications, and as such they may be of prime interest to policy makers and economists. First, our results contradict the public choice perspective in that they support the use of foreign aid as an effective development tool to reduce child mortality. In other words, aid *can* work and the micro-macro paradox does not empirically apply in the case of child mortality. In fact, given the estimated effects, the proposed scaling up of aid within the SDG agenda will likely contribute to decrease child mortality rates across developing countries (assuming constant shares of health and agricultural aid). Second, our results suggest that aid policies must be sector-sensitive, especially if they relate to a specific objective. While donors have been invited to

increase their share of aid to agriculture over the last two decades (OECD, 2005), and despite some re-allocation between aid sectors in the mid-2000s, the sector composition of aid inflows still implicitly favours other sectors (e.g. education) over agriculture (Table 1). Our results explicitly remind us that aid policies aiming at reducing child mortality should recognise the importance of targeting the agriculture (as well as the health sector) and call for continued sectoral re-allocation of aid inflows.

A few caveats should be mentioned. First, some caution is required in taking our main estimates at face value. Given the novelty of our approach and the use of a time-constrained database, our estimates should be considered upper bound estimates. One might want to consider the average estimate of aid effects over all alternative specifications as a more realistic measure of aid effectiveness. For example, the average measurement of a 1-dollar increase in agricultural aid per capita would respectively be 0.9 and 1.5 fewer infant and under-five deaths. Despite the fact they are somewhat lower than the main estimates reported in Section 4.2, they still provide strong evidence for aid effectiveness. Second, our policy recommendations *strictly* apply to the fight against child mortality; any extension to other dimensions of human development should be avoided as the relationships between aid and human development may be multiple and particularly complex. There is thus a need for further research looking at the impacts of aid on literacy, poverty or undernourishment. We suggest that empirical researchers interested in these related issues pay special attention to the nature of the data since many development and health indicators are often reported as prevalences, rates or proportions, with high levels of persistence.

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Tables

Table 1. Descriptive statistics

	Mean	Standard deviation		Min	Max	Source
		within	overall			
Neo-natal mortality (per 1000 live births)	23.82	2.04	12.48	2.50	54.80	WDI
Infant mortality (per 1000 live births)	46.49	5.63	28.27	3.90	137.40	WDI
Under-five mortality (per 1000 live births)	68.17	10.87	48.61	5.20	223.40	WDI
Total aid per capita (in US dollars, constant 2012)	58.17	34.96	52.76	1.35	373.89	CRS
Health aid per capita (in US dollars, constant 2012)	3.36	1.85	3.52	0.00	27.52	CRS
Agriculture aid per capita (in US dollars, constant 2012)	3.17	2.8	4.09	0.00	59.83	CRS
Water and sanitation aid per capita (in US dollars, constant 2012)	2.58	2.6	3.68	0.00	39.91	CRS
Education aid per capita (in US dollars, constant 2012)	4.95	2.08	4.83	0.13	38.39	CRS
Fertility (births per woman)	3.81	0.18	1.62	1.20	7.68	WDI
Population (in millions)	26.70	2.44	40.20	0.48	244.10	WDI
GDP per capita (in US dollars, constant 2005)	1783	270	1819	133	8307	WDI
War (dummy)	0.04	0.15	0.18	0	1	Heidelberg Institute
HIV/AIDS prevalence (% of population aged 15-49)	3.00	0.39	5.50	0.10	26.90	WHO
Natural disasters (per year)	2.57	1.69	3.22	0	36	EM-DAT
Public health expenditure per capita (in US dollars, constant 2005)	253.5	80.1	249.4	10.9	1562.2	WDI
Level of democracy (polity2)	2.3	1.6	5.6	-9	10	Polity IV

Table 2. Impacts of total aid on child mortality

	Neo-natal mortality	Infant mortality	Under-five mortality
Lagged total aid per capita	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
Lagged GDP per capita	-0.000	-0.000	0.000
	(0.000)	(0.000)	(0.000)
Lagged fertility	0.149	1.585	1.130
	(0.836)	(1.814)	(3.430)
Lagged population	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
War dummy	0.286	0.658	0.866
	(0.393)	(1.078)	(1.835)
HIV/AIDS	-0.923	-0.566	-0.638
	(0.586)	(0.705)	(1.215)
Constant	23.817***	46.397***	67.716***
	(0.797)	(1.894)	(2.803)
Exogeneity test			
Aid (p-value)	0.26	0.25	0.10
Other variables	-	-	-
F-test of instruments	-	-	-
J-test (p-value)	-	-	-

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses. If no p-value is reported for the exogeneity tests of other independent variables, it means that they all are exogenous.

Table 3. Impacts of health aid on child mortality

	Neo-natal mortality	Infant mortality	Under-five mortality
Lagged health aid per capita	-0.018	-0.519	-1.134**
	(0.053)	(0.328)	(0.519)
Lagged GDP per capita	0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
Lagged fertility	0.132	-11.813**	-15.601*
	(0.837)	(5.811)	(9.231)
Lagged population	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
War dummy	0.286	6.921*	10.976**
	(0388)	(4.058)	(5.409)
HIV/AIDS	-0.927	4.723**	5.824
	(0.594)	(2.270)	(3.555)
Constant	23.855***	44.228***	64.228***
	(0.807)	(0.625)	(0.976)
Exogeneity test			
Aid (p-value)	0.136	0.03	0.02
Other variables	-	-	-
F-test of instruments	-	138.63	138.63
J-test (p-value)	-	1.73 (0.81)	2.05 (0.84)

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses. If no p-value is reported for the exogeneity tests of other independent variables, it means that they all are exogenous.

Table 4. Impacts of agriculture aid on child mortality

	Neo-natal mortality	Infant mortality	Under-five mortality
Lagged agricultural aid per capita	-0.148	-0.938***	-1.661***
	(0.118)	(0.289)	(0.449)
Lagged GDP per capita	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
Lagged fertility	-6.080**	-7.737	-8.698
	(0.837)	(5.476)	(8.466)
Lagged population	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)
War dummy	3.324*	6.487*	10.098**
	(1.842)	(3.723)	(4.918)
HIV/AIDS	0.497	5.064**	6.299*
	(1.232)	(2.363)	(3.555)
Constant	22.917***	43.956***	69.799***
	(0.271)	(0.575)	(0.898)
Exogeneity test			
Aid (p-value)	0.00	0.00	0.00
Other variables	-	-	-
F-test of instruments	19.06	19.06	19.06
J-test (p-value)	2.30 (0.87)	4.06 (0.95)	4.92 (0.97)

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses. If no p-value is reported for the exogeneity tests of other independent variables, it means that they all are exogenous.

Table 5. Impacts of health aid on child mortality: alternative specifications

Specification	Neo-natal mortality	Infant mortality	Under-five mortality
1. Alternative sample (no outlier)	-0.209	-0.849***	-1.593***
	(0.141)	(0.288)	(0.444)
2. Alternative lags in aid (t)	0.025	-0.169*	-0.349**
	(0.170)	(0.102)	(0.176)
3. Alternative lags in aid (t-2)	-0.033	-0.160*	-0.212
	(0.039)	(0.087)	(0.179)
4. Alternative instruments	0.129	-0.414	-1.013*
(2/3/4-period lags)	(0.169)	(0.348)	(0.587)
5. Alternative instruments	0.130	-0.628**	-1.360***
(2-period lags)	(0.168)	(0.319)	(0.516)
6. Alternative aid variable (in logs)	0.522	0.973	0.644
	(0.470)	(1.007)	(1.253)
7. Additional controls	0.125	-0.590	-1.313**
	(0.177)	(0.395)	(0.624)
8.2SLS	0.110	-0.564**	-1.085**
	(0.084)	(0.271)	(0.440)
9. 3-year average	0.081	-0.814*	-1.649**
	(0.145)	(0.486)	(0.701)

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses.

Table 6. Impacts of agricultural aid on child mortality: alternative specifications

Specification	Neo-natal mortality	Infant mortality	Under-five mortality
1. Alternative sample (no outlier)	-0.326***	-0.898***	-1.624***
	(0.121)	(0.284)	(0.429)
2. Alternative lags in aid (t)	-0.132	-0.885***	-1.557***
	(0.126)	(0.302)	(0.463)
3. Alternative lags in aid (t-2)	-0.367*	-1.453***	-2.591***
	(0.214)	(0.483)	(0.747)
4. Alternative instruments	-0.199	-0.980***	-1.718***
(2/3/4-period lags)	(0.150)	(0.357)	(0.560)
5. Alternative instruments	0.029	-0.953***	-1.771***
(2-period lags)	(0.070)	(0.337)	(0.552)
6. Alternative aid variable (in logs)	-1.071***	-2.745***	-5.835***
	(0.331)	(0.679)	(1.305)
7. Additional controls	-0.203	-0.956***	-1.677***
	(0.145)	(0.299)	(0.469)
8. 2SLS	-0.213*	-0.794**	-1.297**
	(0.127)	(0.353)	(0.571)
9. 3-year average	-0.056	-0.752*	-1.486**
	(0.170)	(0.429)	(0.700)

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses.

Figures

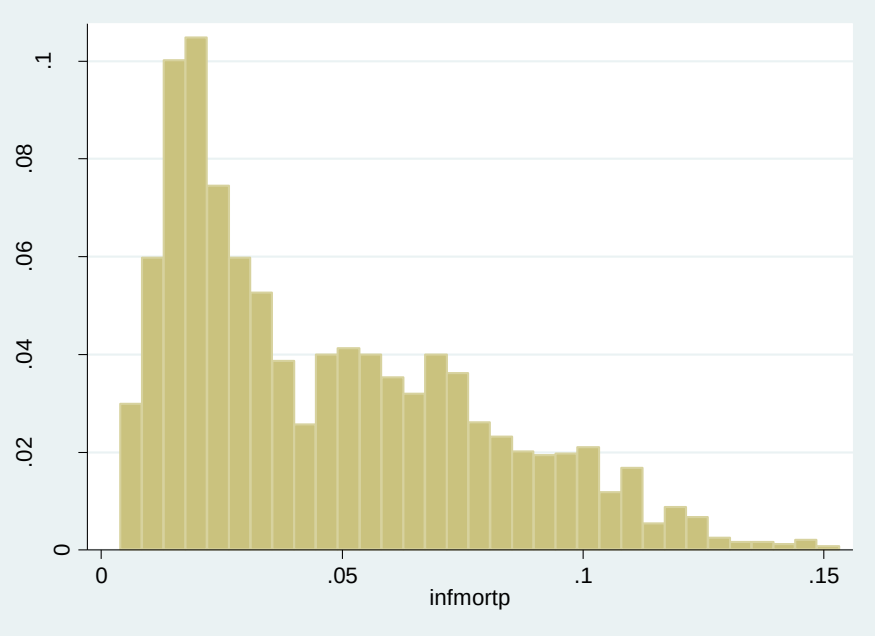

Figure 1. Histogram of infant mortality rate

Appendix

A1. Persistence in child mortality rates

If we estimate an Auto-Regressive of order 1 model using the logarithm of infant mortality as dependent variable, we can check the stationarity of the variable using the coefficient of the lagged dependent variableα. Let's use the following model:

$$lny_{it} = \alpha lny_{it-1} + \delta_i + \mu_{it}$$

Where y_{it} is the infant (or neo-natal/under-five) mortality rate, δ_i is a time-invariant country-specific effect, μ_{it} a i.i.d. shock. The model is estimated with OLS and system GMM approaches. Estimates are based on one-step system robust GMM estimators. Region and year dummies are included.

Table A1. AR(1) models for child mortality

Dependent: lny_{it}	Neo-natal		Infant		Under-five	
	Levels	Logs	Levels	Logs	Levels	Logs
α_{GMM}	0.850***	1.087***	0.877***	1.070***	0.846***	1.116***
	(0.061)	(0.025)	(0.041)	(0.038)	(0.052)	(0.113)
α_{OLS}	0.988***	1.003***	0.983***	1.001***	0.984***	1.002***
	(0.004)	(0.004)	(0.005)	(0.004)	(0.010)	(0.007)

Notes: *, **, *** significant at 10, 5 and 1 per cent levels. Robust standard errors in parentheses.

Results are displayed in Table A1. A quick look at Table A1 confirms that the coefficient on the lagged logarithm of infant mortality estimated with system GMM estimation clearly indicates non-stationarity. We obtain similar results using OLS. Using y_{it} instead, results confirm that the level of infant mortality does not have a unit root though it shows relatively high persistence. Results for the neo-natal and under-five mortality rates are similar.

A2. List of countries

Table A2. List of countries included in estimations

Angola	Ethiopia	Mauritius	Tanzania
Argentina	Fiji	Mexico	Thailand
Armenia	Gabon	Moldova	Togo
Azerbaijan	Gambia	Mongolia	Tunisia
Bangladesh	Georgia	Morocco	Uganda
Belarus	Ghana	Mozambique	Ukraine
Benin	Guatemala	Namibia	Uzbekistan
Bolivia	Guinea	Nepal	Venezuela
Botswana	Guinea Bissau	Nicaragua	Vietnam
Burkina Faso	Guyana	Niger	Yemen
Burundi	Haiti	Nigeria	Zambia
Cambodia	Honduras	Pakistan	Zimbabwe
Cameroon	Indonesia	Panama	
Chad	Iran	Papuasia	
Colombia	Jamaica	Paraguay	
Congo Democratic Republic	Kenya	Peru	
Congo Republic	Kyrgyz Republic	Philippines	
Costa Rica	Laos	Rwanda	
Cote d'Ivoire	Lesotho	Senegal	
Cuba	Liberia	Sierra Leone	
Dominican Republic	Madagascar	South Africa	
Ecuador	Malawi	Sri Lanka	
Egypt	Malaysia	Suriname	
El Salvador	Mali	Swaziland	
Eritrea	Mauritania	Tajikistan	