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Foreign aid and HIV infections: evidence of causal effects from country-level panel data

Ranjeeta Thomas



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Abstract

This paper considers the impact of foreign aid for HIV on the number of new infections in low middle income countries . We provide causal estimates by improving on earlier studies by addressing the endogeneity of aid disbursements through the use of instrumental variables. In addition, we effectively control for unobserved country specific effects that might jointly influence aid allocation and new infections in a country. We provide evidence on the effectiveness of foreign aid in reducing new infections of HIV across age groups, as well as separately amongst adults and children. We find a statistically significant and important negative effect of foreign aid on the number of new infections for our overall measure - across age groups and amongst adults. We find no statistically significant evidence amongst children across low middle income countries, but find evidence of strong differential effects in sub-Saharan Africa.

Keywords: HIV, New infections, Foreign aid, Instrumental variables

JEL classification: F35 , I15, O53, O55

*Business School, Imperial College , Level 1 Sherfield Building, London SW7 2AZ, United Kingdom. Tel:+44 (0)20 7594 0923. Email:ranjeeta.thomas@imperial.ac.uk. Acknowledgments: The author is supported by funding from the HIV Prevention Trials Network 071 Study (HPTN 071). HPTN 071 is funded by the U.S. National Institutes of Health (NIH), including the National Institute of Allergy and Infectious Diseases (NIAID), the National Institute of mental Health (NIMH), the National Institute of Drug Abuse (NIDA), as well as the Office of the United Stated Global AIDS Coordinator (OGAC) and the Bill & Melinda Gates Foundation (BMGF). However, the views expressed in this paper are those of the author, and do not necessarily reflect those of the funders or the HPTN 071 team.

1 Introduction

Since the beginning of the Human Immunodeficiency Virus (HIV) epidemic in the 1980s nearly 30 million people have died from acquired immune deficiency syndrome (AIDS) and related diseases. As of 2012 there were around 35 million people living with HIV, and each year a further 2.5 million individuals become infected. The disease burden has been most severe in sub-Saharan Africa (SSA) where in some countries almost one in five people is infected with HIV. Despite the magnitude and severity of the epidemic significant progress has been made since the late 1990s when HIV came into the focus of the international development community. New infections have fallen across all age groups by 38% since 2001 and rapid scale-up of access to treatment has been possible.

The response of the international donor community to the epidemic has seen extraordinary amounts of funds channeled to prevention and treatment of HIV. In 2013 funding reached its peak with an estimated US$19.1 billion being committed to programs in low and middle income countries. Despite these vast amounts of resources the end of the epidemic is still not in sight and there remains a large financing gap in the amount needed to reach the 2015 targets of universal access to prevention, treatment and care for HIV worldwide. Schwartlander et al. (2011) have developed an investment framework for 139 low-middle income countries and estimate around US$22 billion being needed by 2015. On the other hand, there have been questions raised about whether foreign aid for HIV has been displacing funding for other important health concerns Shiffman (2008). For example between 1998 and 2007 funding for HIV control grew from 5.5% to almost 50% of all aid given for health (Shiffman, Berlan and Hafner, 2009). The same study finds that in contrast, during the same time period the amount of aid given to reproductive health declined by almost half. While reducing the impact of the epidemic on health is in its own right important, the epidemic has also led to negative consequences on the socio-economic environment of affected countries. Several studies have examined the economic and human capital consequences of the epidemic. Fortson (2011) finds that in sub-Saharan Africa areas with higher levels of HIV had larger reductions in schooling. In a paper studying the labor market effects of the epidemic in South Africa Chicoine (2012) finds that AIDS mortality led to reductions in employment in Africa and reduced wages amongst Black South Africans. But the importance and benefits of treatment and access to care is clearly highlighted

in a study by Thirumurthy, Graff Zivin and Goldstein (2008) who find six months after treatment initiation the probability of labor force participation amongst HIV positive individuals increased by 20 percent in Western Kenya. These statistics and studies highlight the importance of controlling the HIV epidemic but equally they highlight the importance of understanding the returns to the massive investments made. Our study is aimed at addressing this question with a specific focus on the reductions in the number of new infections of HIV.

There have been a few studies that have analyzed the direct relationship between foreign aid flows for HIV and health outcomes. The most comprehensive of these Bendavid et al. (2012) uses data from Demographic and Health Surveys in 27 sub-Saharan African countries to investigate the impact of the President's Emergency Plan for AIDS Relief (PEPFAR) on all cause mortality in 12 focus countries. The authors find that after introduction of PEPFAR (2004-2008) the odds ratio of all cause mortality in focus countries was 0.84 compared with non focus countries. Another study, and one that is closer in objective to ours is by Nunnenkamp and Ohler (2011). The authors use estimates from UNAIDS on AIDS related mortality and a constructed measure of new infections to study the impact of foreign aid (for HIV) commitments. The authors use a difference-in-differences approach and compare high prevalence countries that received more foreign aid (intervention) with those that received less (control). They find no statistically significant effect of aid on new infections of HIV. In the case of mortality, they find no overall effect, but reductions from aid given by bilateral donors only.

In contrast, our paper provides empirical evidence that foreign aid for HIV has reduced new infections of HIV and we do this by improving upon their empirical strategy in several ways. First, we recognize the endogenous properties of aid allocations. There is extensive literature particularly focusing on aid and economic growth , or aid and conflict (de Ree and Nillesen, 2009; Tavares, 2003; Collier and Hoeffler, 1998) that have highlighted the importance of controlling for the endogeneity of foreign aid. In our case, several time varying unobservable factors may influence the amount of aid a country receives, in addition donors my predict or anticipate changes in the number of new infections and adjust aid flows accordingly. Estimates of aid flows that do not account for this endogeneity will combine the causal impact from aid to new infections with correlations caused

by endogeneity. We use a strong instrument based on GDP per capita of international donors to separate and recover the casual effect. Second, rather than construct a variable of new infections as the difference between prevalence and mortality as done by Nunnenkamp and Ohler (2011) we use published UNAIDS estimates. These estimates are the most reliable source of information on new infections and are widely used to measure the state of the epidemic. Third, we look at a longer time frame as compared to the previous studies. Our data covers 1995 -2011. As far as we are aware this paper is the first to use an instrumental variables approach to recover the causal effect of foreign aid for HIV on new infections.

In Section 2 we discuss our empirical strategy and detail our identification strategy. Section 3 describes our data, its sources and presents stylized facts. In Section 4 we present our regression outcomes and discuss our results along with findings from several robustness checks. Section 5 concludes.

2 The empirical analysis of new HIV infections

The objective of this paper is to identify and to estimate the causal effects of aid flows on new infections of HIV in low middle income countries. Using non-experimental data to identify causal effects requires certain identifying restrictions. For example, Nunnenkamp and Ohler (2011) use a difference-in-differences approach. Their approach relies on a "parallel trend" assumption which requires that the time effect is common to both treatment and control groups and any group effects are driven by "time-invariant" differences (observable or unobservable). In studying the relationship between aid and new infections, there are two problems with this approach. The first, is that countries in their selected treatment and control groups are unlikely to have a parallel trend. The authors find this is the case and that the outcome variable (mortality or new infections) is correlated with the dynamic of the epidemic. In addition, Nunnenkamp and Ohler (2011) assume that aid allocation is exogenous with respect to future levels of new infections. We argue that ignoring this issue could potentially bias their estimates and may explain why they find no correlation between aid and new infections.

This paper focuses on two key aspects to recovering reliable causal estimates. *Time invariant*

effects: such as gender equality, governance, cultural and social beliefs or other partly unobserved factors (de Ree and Nillesen, 2009) are likely to determine the extent of new infections. These factors are also likely to be correlated to the extent of aid that countries receive. In fact Nunnenkamp and Ohler (2011) illustrate that despite their sample countries all having being classified as having an HIV prevalence over 1%, the amount of aid received differed greatly amongst these countries. They use this fact to divide their sample into treatment and control groups. To address this issue we include a country-specific effect that captures time-invariant unobservables that are potentially correlated with HIV aid flows and new infections in a country. If such a specification captured all potential sources of endogeneity then such a fixed-effects specification would suffice to recover causal effects.

Endogeneity in aid allocation: There could be two causes of endogeneity, firstly - time-varying unobservables not captured by fixed-effects such as failing health systems or governments, anticipated shifts in other disease burdens or the potential for conflict in the countries are likely to be correlated with aid-flows for HIV (omitted-variable bias) and are potentially likely to influence new HIV infections. Secondly, reverse causality. Our concern in this case is that it difficult to perceive aid allocations as being independent of the level of new infections in the recipient country. We thus adopt an instrumental variables (IV) approach to address the endogeniety issue.

Our instrumental variables model with country-specific effects is specified below, where inf_{it} represents new HIV infections in country i at time t. New infections depend on aid_{it} - the amount of aid received and a vector X_{it} containing covariates that potentially influence both the outcome (new infections) and the amount of aid received. The model can be written as:

$$inf_{it} = \beta aid_{it} + \delta X_{it} + \alpha_i + \epsilon_{it} \tag{1}$$

where α_i is a country-specific effect that captures the above mentioned *time invariant* effects that are likely to be correlated with both HIV aid allocations and new infections in a specific country. ϵ_{it} represents a random, idiosyncratic error term. The above model when estimated using a fixed-effects approach to eliminate correlations between βaid_{it} , δX_{it} and α_i will yield unbiased and consistent estimates of the parameters of interest under the following assumption:

$$E[\epsilon_{it}|\alpha_i, aid_{it}, X_{it}] = 0 \tag{2}$$

However we have argued that the endogeneity of aid flows is driven not only by correlations with the fixed effects but also by other time varying omitted variables and potential reverse causality. In the presence of potential endogeneity of aid_{it} the above will lead to biased estimates of the β coefficients.

To recover unbiased and consistent estimates an IV estimator requires that we identify one or more "instrumental" variables Z_{it} that create exogenous sources of variation in the endogenous aid variable aid_{it}. Specifically, a suitable instrument must have the following properties, with all other relevant variables controlled for, the instrument should be:

1. correlated with changes in the endogenous variable (aid_{it}) but should not lead to changes in new infections (except indirectly via aid_{it}) ie. good first stage explanatory power

2. uncorrelated with the error term of the regression model specified in equation (1) ie the exclusion restriction must not be violated:

$$E[\epsilon_{it}|\alpha_i, Z_{it}, X_{it}] = 0 \tag{3}$$

If the instruments have the above properties, we should be able to recover unbiased estimates (of β) of the effect of HIV aid allocations on new infections from equation (1) using two-stage least squares (IV-2SLS) (Cameron and Trivedi, 2005).

We follow the literature on aid-effectiveness in selecting our instrument (de Ree and Nillesen, 2009; Tavares, 2003; Collier and Hoeffler, 1998). We use GDP per capita of large western donor countries as an instrument for HIV aid flows. The rationale for this selection is based on the idea that aid flows to low middle income countries are linear functions of donor GDP where donor contributions are generally in proportion to their country income levels. The 2001 United Nations General Assembly Special Session (UNGASS) in their "Declaration of commitment on HIV/AIDS" reaffirmed their resolution (2626) that donor countries would contribute 0.7% of gross national

product (GNP) for overall overseas development assistance (ODA) with about 0.15% to 0.20% GNP aimed at least developed countries (Assembly, 2001). In particular reaffirming that those countries that had failed to thus far achieve this target would aim to do so. Although only a few countries have achieved this target, generally ODA commitments tend to be a specific fraction of GDP levels irrespective of the instabilities or potential conflicts within the recipient countries. Disbursements of ODA to countries are however often dependent on the strategic interests of donors (Lane and Glassman, 2007), political situation of the recipient country and other often unobservable factors, thus making aid flows endogenous.

We use GDP per capita of the USA as our instrument. The US government remains the largest donor to HIV efforts, with disbursements accounting for almost 64% of all contributions from donor governments. Good first stage explanatory power as described in property (1) above means that donor GDP per capita is partially correlated with our aid variable (after controlling for other factors affecting new infections). This means that variations in GDP per capita will induce variations in the amount of aid disbursed across countries and within countries over time. This exogenous variation in the amount of aid will in turn lead to changes in the number of new infections. It is reasonable to assume that changes in the number of new HIV infections in a country as a response to variations in GDP per capita of donors is channeled solely through the amount of HIV aid received by a country ie, property (2) holds. But in order to control for any potential indirect effects of donor GDP on the number of new HIV infections, in all the models we control for GDP *per capita* of the recipient country and public health expenditure as a share of total health expenditure. By including public health expenditure we also control for the effect of domestic spending on new infections. We also provide results of specification tests. Finally, to assess the relevance of our instrument we present F-tests of its significance in the IV regressions as well as results from underidentification tests (Angrist and Pischke, 2009).

The omitted variables that cause endogeneity problems in the fixed-effects least squares setting also limits are choice of instruments. Often instruments are chosen that represent specific characteristics of recipient countries that are correlated with aid disbursements. However, these country characteristics are also likely to be correlated with our outcome variable - new HIV infections.

Hence we choose not to interact our instrument with any country specific characteristics as done by Tavares (2003); Collier and Hoeffler (1998). For example cultural proxies, geographical distance or common language could be interacted with the US GDP per capita to give us a country specific instrument. However, we feel that geographical positioning, language or cultural factors are likely to be correlated with new infections and interacting them with US GDP per capita will give us a less reliable instrument. For example, Oster (2012) uses geographical positioning as an instrument for HIV prevalence by measuring distance of a country to the origin of the HIV epidemic.

Using US GDP per capita provides us with a strong instrument, but comes with a limitation in that because it does not vary by country we are unable to include time dummies for all years in the models. This would present a problem if for example a time shock was correlated with US GDP that also influences new HIV infections. As mentioned earlier, we include GDP per capita for each country in our model along with public health expenditure (as a share of total health expenditure). We would expect these variables to be able to capture most of these effects. But, we also include other variables such as HIV prevalence, the occurrence of conflict and political status to capture any additional effects not captured by GDP per capita or health expenditure as well as population structure to capture cross country differences. In addition, in our robustness checks we also allow for GDP growth and the number of refugees in a country. We also estimate models with time dummies for years that have witnessed global shocks.

3 Data Description

3.1 Data and Variables

Our estimation sample focuses on low income and low middle income countries as defined by the World Bank classification and covers the years 1995 to 2011. In total there are 82 countries in our sample for which we have estimates of new infections. Table 1 presents descriptive statistics of all the variables.

Table 1: Summary statistics - Low income and low middle income countries

Variable	Mean	Standard deviation	Minimum	Maximum
Ouctomes				
New infections - all age groups	33011.1	63301.74	100	360000
New infections - adults	26526.47	52602.79	100	350000
New infections - children	6185.13	12465.46	100	66000
Regressors				
Aid disbursements per capita	1.13	3.24	0	50.59
Polity2 index	1.45	5.77	-10	10
Conflict	0.22	0.42	0	1
HIV prevalence - adults (%)	2.69	5.01	0.1	28.7
Health expenditure, public (% of total health expenditure)	43.20	17.41	3.32	86.24
GDP per capita	979.04	853.54	50.04	4690.94
% Population 0 to 14	39.01	7.90	13.87	50.74
% Population 15 to 64	56.70	6.07	45.99	73.50
US GDP per capita	38936.38	7056.659	27776.43	48407.08

The time period is 1995 -2011

3.1.1 Outcome variable - new HIV infections

Our main variable for analysis is the number of new HIV infections. We use data on the annual number of new HIV infections (adults and children) estimated by UNAIDS. These data are available at the country level for the years 1990 to 2013. In addition, data are available by sub-groups - adults 15 years and older and children 0-14 years newly infected with HIV. For some countries, for years with very few new infections (estimate < 100), the number is rounded off to 100. These data are are widely used as progress measures of the fight against the HIV epidemic and presented annually in UNAIDS' report on the epidemic (UNAIDS, 2013*b*). We use all three variables and conduct analysis on the overall number of infections and on sub-groups.

3.1.2 Foreign aid for HIV

The primary explanatory variable of interest is foreign aid for HIV measured as development assistance for health (DAH) allocable to all programs relating to HIV/AIDS. These data are taken from the development assistance for health database developed by the Institute for Health Metrics

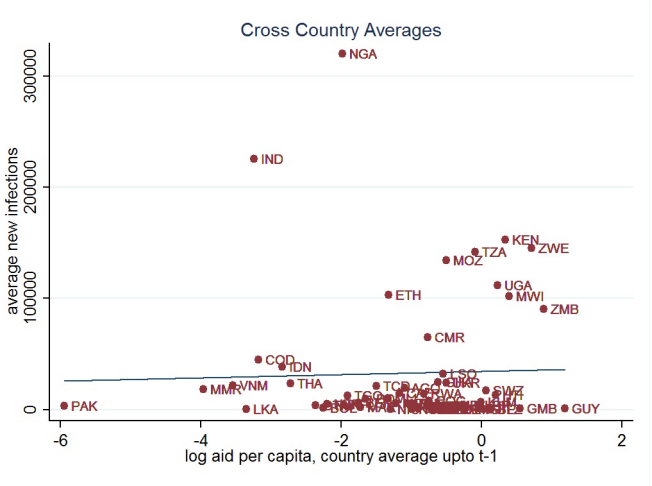
and Evaluation (IHME, 2009). The DAH database provides annual country level data on disbursements of HIV specific aid by key funding agencies. Data are available from 1995 - 2011. We calculate the foreign aid variable as HIV specific DAH flows to a country relative to its population (*per capita*) lagged one period (t-1). The variable is measured in US$. In all cases we construct *log's* of our foreign aid variable, thus the effect of a percentage change in foreign aid on the number of new infections can be estimated directly.

3.1.3 Instrument and other variables

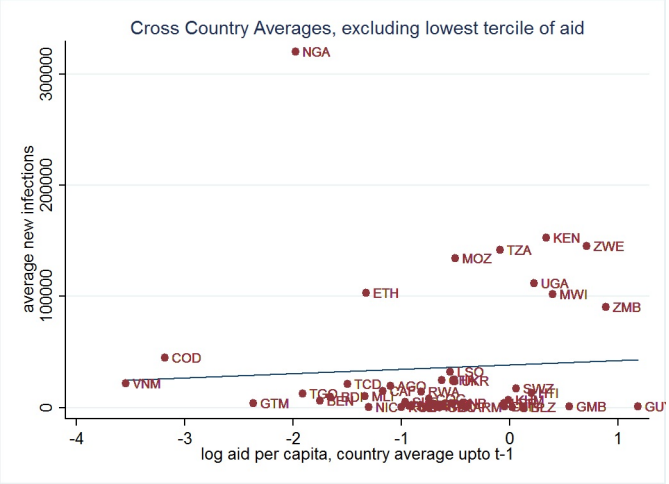
As discussed in the methodology section we use US GDP per capita as an instrument to address the endogeneity of the aid variable . We use GDP per capita data taken from the World Bank's World Development Indicators (WDI) database. Similar to our endogenous variable, the instrument is also lagged one period (t-1). In addition, to control potential indirect effects of foreign aid we include GDP per capita (t-1) of recipient countries, public health expenditure as a share of total health expenditure (t-1) and population structure (t-1). These data are taken from the WDI database and the WHO Global Health Observatory database. Several studies Brckner (2013); Alesina and Dollar (2000); Trumbull and Wall (1994) have highlighted the importance of political institutions and civil war on donor contributions of aid. For these two factors to be omitted variables, it must be the case that annual within-country changes in political institutions or occurrence of conflicts must lead to an immediate effect on the number of new infections. We include two variables in our model, the first a dummy variable for conflict (lagged one period, t-1) which takes the value 1 if armed conflict occurred in the recipient country in a specific year. These data are take from the PRIO/UPSALLA database on armed conflicts. This data set has been widely used since it was made available (Gleditsch et al., 2002). Our second variable represents the political set-up within a country. The variable (lagged one period, t-1) ranges from -10 to +10 (democratic) and is taken from the Polity IV database. In addition, we include lagged (t-1) HIV prevalence in each country taken from UNAIDS estimates to control for the extent of the epidemic with a country.This setup represents our main specification and results. We then carry out a series of robustness checks described later in the paper.

3.2 Stylized Facts

We first examine some basic correlations between new infections and foreign aid for HIV. Figure 1 summarizes the correlation between cross-country averages of the log of aid per capita and the number of new infections for all age groups. The data is constructed by averaging each variable over the sample time frame. The observations are labeled using their World Bank country codes. For brevity we do not present graphs for our two other outcome variables - new infections amongst adults and new infections amongst children, as our conclusions are the same.



(a) Aid and new HIV infections - LMICs



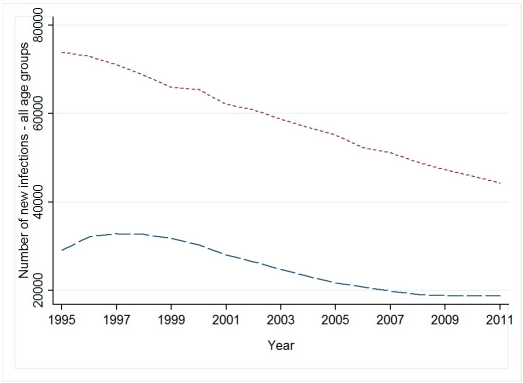
(b) Aid and new HIV infections - excluding lowest tercile of aid

Figure 1: Between country correlations between aid and new HIV infections

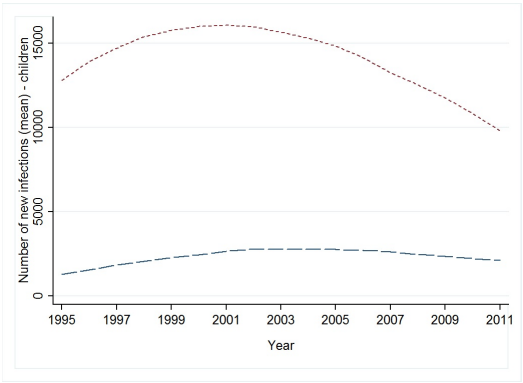
Figure 1(a) reveals no clear direction in the relationship between aid per capita and new infections. This lack of relationship is also reflected in the correlation coefficient which is very low (0.03) and statistically insignificant. In figure 1(b) we exclude countries that received low amounts of aid for HIV by constructing a variable that represents terciles of aid received, where the lowest tercile includes countries with the least increase in the amount of aid received and the top tercile includes countries with the greatest increase in aid over our sample period. Excluding countries in the lowest tercile still yields a very low correlation coefficient (0.05) which is also not statistically significant. This lack of correlation may reflect the complex relationship between foreign aid for HIV and new infections. Aid allocation mechanisms and new infections are influenced by a range of factors, many of which are unobservable - such as social, cultural and epidemiological factors. Ignoring these unobservable influences can cause bias in our estimates, particularly those that affect both aid allocations and new infections of HIV.

Figure 2 presents the correlation between number of new infections and aid over time, in this case our focus is to identify aggregate time series variation. We present graphs for all three outcome variables. The correlations are again presented by terciles of aid received. We focus on the top and bottom terciles for brevity.

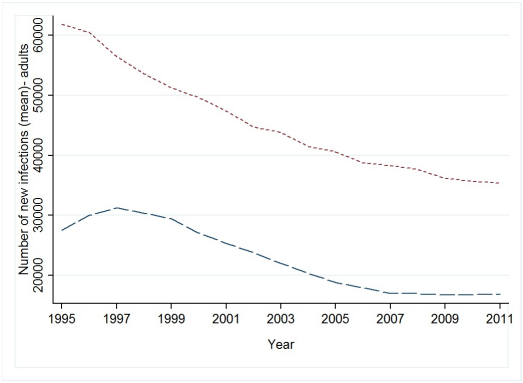
In the case of new infections across all age groups (figure 2(a)) we see a clear negative relationship with aid in both the upper and lower terciles. However, these unadjusted correlations show no clear direction in the case of new infections in children(2(b)), where in the top tercile we see a rise in the number of new infections followed by a downward trend. In the case of adults, countries in the top tercile of increase in HIV aid per capita exhibit a clear reduction in adult new infections over time, while countries in the bottom tercile also show reductions but primarily after 1997. Thus we observe a fair amount of aggregate time series variation in the relationship between aid and new infections of HIV. The top tercile in general consists of most of the "HIV focus" countries of sub-Saharan Africa which clearly start the sample with much higher levels of new infections, indicating that aid has been channeled in most cases to countries with large disease burdens. These raw correlations suggest that the returns to an additional dollar being spent (especially in high prevalence countries) could potentially yield higher marginal returns.



(a) New infections - all age groups



(b) New infections - children



(c) New infections - adults

Figure 2: Average new HIV infections over time across groups (terciles) of countries with the largest and smallest increases in aid. (- -, top tercile; — —, bottom tercile).

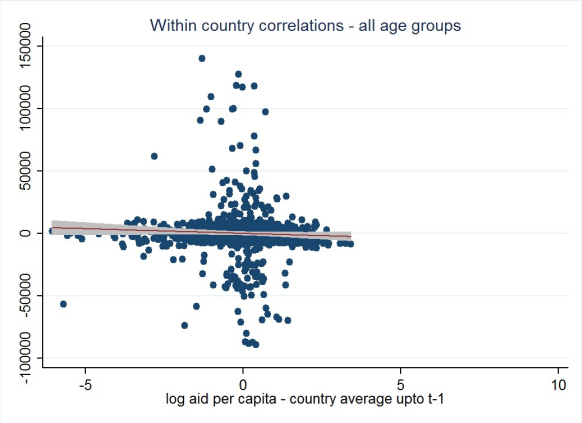
Thus far we have observed little correlation from cross-country variations and some from aggregate time-series variation. Finally, before we move on to our main estimates we look for evidence of within-country correlations overtime. Figure 3 shows that within countries, more aid per capita is associated with fewer new infections in the case of adults and across age groups. In the case of children no clear direction is visible. These correlations are generally difficult to interpret as they do not control for any observable factors and only remove the country and time specific effects.

In general the mixed outcomes from the raw correlations shows the complex nature of the relationship between aid and new infections and the potential risks associated with assuming a lack of causality due to lack of variation. In section 4 we present estimates of our models that address the concerns raised with these raw correlations and provide evidence of the robustness of our main results.

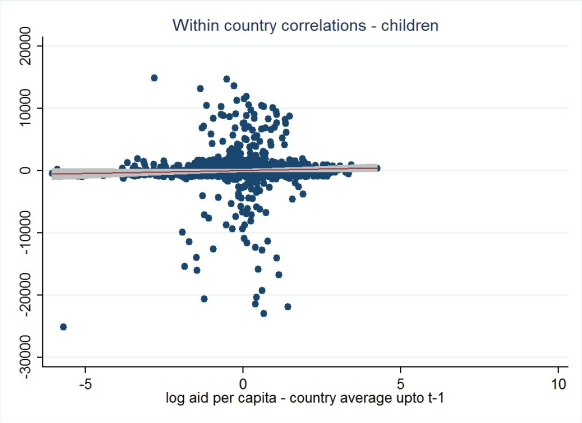
4 Results

4.1 The causal effect of aid on new HIV infections

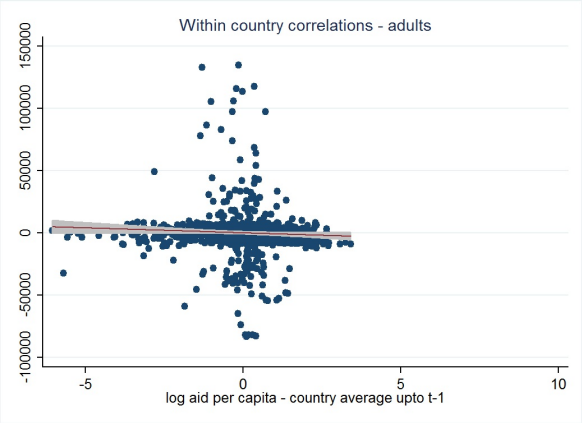
We use the instrumental variables approach outlined in section 2 to recover credible estimates of the impact of foreign aid on new infections. In tables 2 and 3 we present our main results for all three models -"all age groups", "adults" and "children". Before we begin our discussion of the results we analyse the performance of our instrument. First, we return to our methodology section where we suggested that a potential reason why Nunnenkamp and Ohler (2011) find no statistically significant effects could be because they fail to account for the endogeneity of the aid variable. In table 2 we present first stage estimates of our IV-2SLS models that link the instrument (US GDP per capita) to the endogenous variable (foriegn aid per capita). All three models show the instrument is statistically significant at least at the 1%. The point estimate is positive as we would expect and shows that aid disbursements increase with US GDP per capita. The table also presents results from the Lagrange Multiplier (LM) test for underidentification (Kleibergen and Paap, 2006). The results strongly reject underidentification in the first stage IV estimations. The instrument also yields large first-stage F-statistic indicating that bias due to weak instruments is



(a) Within country correlations - new infections - all age groups



(b) Within country correlations - new infections - children



(c) Within country correlations - new infections - adults

Figure 3: Within-country correlations are identified by regressing both aid per capita and new infections on a set of country and time dummies. The residuals from both regressions are plotted against each other

unlikely to be a problem in our model and there is good first stage explanatory power (property 1 discussed earlier). This means that donor's GDP is partially correlated with our measure of foreign aid (after controlling for other factors that might influence new infections).

Table 2: First stage estimates - Effect of US GDP per capita on foreign aid for HIV

	All age groups	Adults	Children
	1	2	3
	IV- 2SLS	IV- 2SLS	IV-2SLS
Dependent variable: Log per capita foreign aid for HIV (t-1)			
US GDP per capita (t-1)	0.0002***	0.0002***	0.0002***
	(0.000)	(0.000)	(0.000)
Controls	Yes	Yes	Yes
Underid (LM test) p-val	0.000	0.000	0.000
First-stage F-stat	109.57	109.57	96.80
Observations	881	881	904

The method of estimation is two-stage least squares.
Regressions control for GDP per capita, public health exp (% of total health expenditure), conflict, polity2, share of population 0-14 years and 16 -64. Models include country fixed effects.
Robust standard errors clustered at the country level presented in parentheses.
* indicate significant difference from zero at *90% confidence, **95% confidence and *** 99% confidence

In table 3 we present two sets of results - a fixed-effects specification (FE-LS) and corresponding IV estimation (IV-2SLS). Columns (1) and (2) report results for the effect of aid on new infections across all age groups. Columns (3) and (4) for adults, (5) and (6) for children. In all models our foreign aid variable is in *log* form. This enables us to interpret a percentage change in aid per capita as a change in the number of new infections.

We begin our discussion of the results with the model in column (2) of table 3. The IV estimations strongly indicate that higher levels of aid disbursements *per capita* lead to lower number of new infections. The point estimate is statistically significant at the 5% level and shows that a 1% increase in aid disbursed *per capita* to a low-middle income country results in 4849 fewer new infections. The corresponding fixed-effects estimates presented in column (1) shows much smaller statistically significant effect of aid on reduced new infections. Using an IV estimator to control for endogeneity of the aid variable leads to a larger, negative, statistically significant effect. These results indicate an overall effect of aid on new HIV infections. This result can be corroborated

Table 3: The effect of foreign aid on new infections of HIV

	All age groups		Adults		Children	
	1	2	3	4	5	6
	FE-LS	IV-2SLS	FE - LS	IV-2SLS	FE - LS	IV-2SLS
Aid disbursements per capita (t-1) (log)	-2327**	-4849**	-2080**	-4411**	-221	-365
	(1079.35)	(2207.38)	(972.82)	(2041.26)	(200.13)	(401.61)
Polity2 (t-1)	654	882	547	758	97	110
	(615.27)	(627.69)	(526.53)	(529.72)	(145.17)	(142.79)
Conflict (t-1)	2515	2882	2231	2570	188	215
	(5660.41)	(5533.61)	(5446.59)	(5320.34)	(369.79)	(355.95)
HIV prevalence - adults (t-1)(%)	3952**	4175**	2266*	2473*	1687**	1706**
	(1820.54)	(1805.47)	(1306.45)	(1342.12)	(673.54)	(668.29)
Health expenditure, public (t-1) (% of total health expenditure)	-348	-187	-309	-160	-41*	-32
	(216.47)	(239.25)	(207.90)	(234.58)	(22.74)	(24.60)
GDP per capita (t-1)	2	1	0	-0	1	1
	(5.04)	(5.64)	(5.43)	(5.81)	(1.03)	(1.09)
% Population 0 to 14 (t-1)	3198	4452	3093	4252	76	101
	(3329.86)	(3645.01)	(3250.07)	(3481.98)	(493.61)	(507.40)
% Population 15 to 64 (t-1)	2859	4888	2933	4808	-76	-10
	(3346.28)	(3877.49)	(3181.32)	(3626.46)	(549.45)	(562.15)
Constant	-257014		-258522		2822	
	(321291.15)		(309820.20)		(50255.31)	
Underid (LM test) p-val		0.000		0.000		0.000
First-stage F-stat		109.57		109.57		96.80
Observations	881	881	881	881	904	904

All models include country fixed effects. Robust standard errors clustered at the country level presented in parentheses.

* indicate significant difference from zero at *90% confidence; **95% confidence and *** 99% confidence

by recent reports from UNAIDS (UNAIDS, 2013*a*) which have found that by 2012 there was 33% reduction in new infections amongst adults and children when compared with 2001.

Over the last decade prevention programs have focused on many different kinds of interventions. For adults, these have included behaviour change interventions, medical male circumcision, focus on high-risk groups such as injecting drug users, sex-workers and men-who-have-sex with men. But HIV is also a leading cause of death amongst women of reproductive age worldwide and a major cause of infant mortality. Mother-to-child transmission (MTCT) is when an HIV - positive mother passes the virus to her child during pregnancy, labour, delivery or breast-feeding. Since

2001 when the UN member states made a global commitment to the prevention of mother-to-child transmission (PMTCT), it has become a major focus area in the fight against the HIV epidemic. Therefore in addition to our model for combined age groups and adults, we also run a separate model to identify whether foreign aid has had an impact in reducing new infections in adults and children.

In columns (3) and (4) we present the impact on new infections amongst adults. The IV estimates are striking and show that foreign aid has a clear negative effect on new infections amongst adults. The estimate is statistically significant at the 5% level and shows that a 1% increase in per capita foreign aid reduces new infections in adults by about 4411. These impressive results are also reflected in the UNAIDS World AIDS Report (UNAIDS, 2012) which highlights that more than 25 LMICs achieved atleast a 50% reduction in the new infections amongst adults between 2001 and 2011. As expected the fixed-effects specification shows a small negative impact. In column (6) we present results for new HIV infections amongst children. In this case however, while we see a negative association between foreign aid and new infections, our result is not statistically significant. This result seems rather unusual to us given that many countries have declared successfully reducing new infections in children. We believe there are two possible reasons for the results we see. First, it is possible that these results are masking differential effects for regions such as sub-Saharan Africa, a region that has the most number of HIV positive children and receives a large proportion of aid. For example, of all newly infected children in 2011, more than 90% live in sub-Saharan Africa. We return to the question of differential effects in our section of robustness checks and investigate further. Second, recent reports from UNAIDS mention that most of the reductions amongst children have taken place in the last two years of our sample (2009-2011)(UNAIDS, 2012). In general we conclude that across all LMICs there have been significant reductions in new infections amongst adults but not amongst children.

4.2 Specification and robustness checks

In this section we undertake a series of specification and robustness checks to verify that the results we observe above are reliable estimates of the causal effect of foreign aid on new infections

of HIV.

4.2.1 Robustness to specification

Table 4: Effect of aid on new infections - including additional controls and time effects

	All age groups		Adults		Children	
	1	2	3	4	5	6
	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS
Aid disbursements per capita (t-1) (log)	-4938**	-5070**	-4546**	-4637**	-307	-387
	(2354.12)	(2361.01)	(2184.31)	(2189.20)	(371.99)	(433.61)
Additional variables	Yes		Yes		Yes	
Time dummies		Yes		Yes		Yes
Underid (LM test) p-val	0.000	0.000	0.000	0.000	0.000	0.000
First-stage F-stat	117.91	96.17	117.91	96.17	109.72	94.33
Observations	838	881	838	881	861	904

The method of estimation is two-stage least squares. Columns (1), (3) and (5) include GDP growth rate and number of refugees
Columns (2), (4) and (6) include time dummies for years 2000-2003 and 2007-2010.
Regressions control for GDP per capita, public health exp (% of total health expenditure), conflict,
polity2, share of population 0-14 years and 16 -64. Models include country fixed effects.
Robust standard errors clustered at the country level presented in parentheses.
* indicate significant difference from zero at *90% confidence, **95% confidence and *** 99% confidence

We described in our methodology section that while we have a strong instrument, it has limitations in that we are unable to include time dummies for all years to capture shocks to the number of new infections. Instead we have included several variables we believe will capture any indirect effects of US GDP per capita on new infections of HIV. In this section we provide two alternate specifications. First, we include two further variables - growth rate of GDP in each country and the number of refugees in a country. We expect these variables to capture the influence of external economic influences as well as any regional uncertainty leading to migration. Second, we include time dummies for years in which there were global shocks such as recessions or global financial crises $2000 - 2003$ and $2007 - 2010$. In addition, the 2000-2003 dummies will also capture any shocks from the setting up of the Global Fund for AIDS, TB and Malaria (GF) and The US President's Emergency Plan for Aids Relief (PEPFAR). Table 4 presents these results. Columns (1) , (3) and (5) include the additional variables mentioned above and columns (2) , (4) and (6) include time

dummies. In both sets of specifications the results are similar to our main findings in table 3 in terms of magnitude as well as statistical significance. We also estimate the models with time dummies for the years 2000-2010. These results are very similar to our baseline estimates and hence we do not present them.

4.2.2 Allowing for differential effects in sub-Saharan Africa

Table 5: Effect of aid on new infections - differential effects in sub-Saharan Africa

	All age groups	Adults	Children
	1	2	3
	IV- 2SLS	IV- 2SLS	IV-2SLS
	Baseline estimates		
Aid disbursements per capita (t-1) (log)	-4849**	-4411**	-365
	(2207.38)	(2041.26)	(401.61)
	Sum of coefficients of main effect and interaction term		
Aid disbursements per capita (t-1) (log)	-4441	-3611	-604**
	(971.26)	(936.73)	(118.28)
Underid (LM test) p-val	0.000	0.000	0.000
First-stage F-stat	93.90	93.90	58.40
Observations	881	881	904

The method of estimation is two-stage least squares.
Regressions control for GDP per capita, public health exp (% of total health expenditure), conflict, polity2, share of population 0-14 years and 16 -64. Models include country fixed effects.
Robust standard errors clustered at the country level presented in parentheses.
* indicate significant difference from zero at *90% confidence, **95% confidence and *** 99% confidence

The raw correlations examined in section 2 indicate that aid was primarily targeted at countries with high initial levels of new infections. This is particularly the case for HIV where sub-Saharan Africa has the greatest disease burden. The majority of these countries are also low-income countries. It is thus possible that high-prevalence countries in sub-Saharan Africa (SSA) may have experienced different results to aid flows when compared to other regions. Our specifications have included country-specific effects which should capture the impact of initial levels of new infections that may confound our results. In addition, the time varying variables we include and the use of an instrument should account for any unobservables that are likely to confound our results. Also

as discussed earlier in our results for the model for children, we suspect that the reason we see no statistically significant results across all LMICs is because differential effects are being masked. In this section we explore further if the returns to international aid is different in sub-Saharan Africa - countries that are mainly low income but also high prevalence, we allow for differential effects in our model by adding an interaction term between the foreign aid variable and a dummy variable representing SSA (=1). We use our full sample of LMICs and IV-2SLS where our instrument for the interaction term is the interaction between US GDP per capita and SSA. Table 5 presents these results. The coefficients presented are the sum of the aid variable (main effect) and the respective interaction term which gives the total estimated effect of aid disbursements on new infections of HIV for SSA. The first and second columns of table 5 shows no statistically significant differential effects for our model for all age groups and adults. In column (3) we present our estimate for the model for children. Unsurprisingly we find a clear statistically significant reduction in new infections with a 1% increase in foreign aid resulting in almost 600 fewer infections amongst children in SSA. The magnitude of our coefficient is more than 1.5 times larger than our estimate for all LMICs. These results indicate that our findings for children in table 3 were indeed masking regional differences.

4.2.3 Further checks: other variables as instruments and excluding outliers

In this section we conduct two further tests of robustness. Our first robustness check uses GDP *per capita* of other large donors such as the United Kingdom (UK), Japan, France or Germany as an instrument for aid flows instead of US GDP *per capita*. Our results are similar irrespective of which country we substitute for the USA as our IV. For brevity we do not present these results here. However, in table 6 we present results of a model where we include the GDP per capita of France in addition to US GDP per capita as instruments. Our results remain the same if we substitute Germany instead of France. However we find that GDP per capita of UK or Japan - do not work well when included in addition to US GDP per capita. Their strong correlation with US GDP per capita eliminates their relevance in the first stage results. We therefore present results including France (the results for Germany are similar). Including the additional instrument allows

Table 6: Effect of aid on new infections - other specification and robustness checks

	All	Adults	Children
	1	2	3
	IV- 2SLS	IV- 2SLS	IV- 2SLS
	Baseline estimates		
Aid disbursements per capita (t-1) (log)	-4849**	-4411**	-365
	(2207.38)	(2041.26)	(401.61)
	Including GDP per capita of France as additional IV		
Aid disbursements per capita (t-1) (log)	-4437**	-3937**	-428
	(1960.36)	(1800.57)	(374.89)
Underid (LM test) p-val	0.000	0.000	0.000
First-stage F-stat	72.09	72.09	63.08
Hansen J-Statistic p-value	0.20	0.14	0.21
Observations	881	881	904
	Excluding outliers		
Aid disbursements per capita (t-1) (log)	-4901**	-4464**	-363
	(2240.19)	(2072.41)	(402.53)
Underid (LM test) p-val	0.000	0.000	0.000
First-stage F-stat	105.91	105.91	96.02
Observations	877	877	898

The method of estimation is two-stage least squares.

Regressions control for GDP per capita, public health exp (% of total health expenditure), conflict, polity2, share of population 0-14 years and 16 -64. Models include country fixed effects.

Robust standard errors clustered at the country level presented in parentheses.

* indicate significant difference from zero at *90% confidence, **95% confidence and *** 99% confidence

us to carry out tests for over-identification. This test refers to our second property for a valid instrument - ie. that it must be uncorrelated with the error term. The Hansen J-statistic p-values indicate our instruments are not likely to be correlated with the error term. The LM statistics and the first stage F-statistics indicate that weak identification is not a problem in our model. Overall, our results continue to show a statistically significant negative impact of foreign aid on new infections for our combined model and amongst adults, once again in the case of children we find no significant impact.

Our second test eliminates outliers that might be driving our main results in table 3. We use a formal statistical procedure for the elimination of outliers in multivariate cases (Weber, 2010). We

exclude outliers for both the foreign aid variable and each of our dependent variables. We eliminate 6 country-year observations that are identified as being outliers in our model across age groups (Lesotho 2011, Swaziland 2006-2011 and Zambia 2009). For our model of adults and children we eliminate Guyana 2007-2010 and Swaziland 2011. We estimate our IV models again excluding these observations. The results are presented in the third part of table 6. Once again we find that are results are close to the baseline estimates.

5 Conclusion

Our analysis shows that once we control for the endogeneity of foreign aid, it has a negative impact (reduction) on the number of new infections. The results show that increasing foreign aid for HIV results in a statistically significant decrease in the number of new infections amongst adults in LMICs. We do not however find such an effect amongst children. Further checks reveal that in the case of children there is a reduction once we allow for differential effects in sub-Saharan Africa, these effects are statistically significant. In terms of magnitude our results are also economically relevant with a marginal effect of -4411 fewer HIV infections amongst adults across LMICs and -604 amongst children in SSA. In our sample a 1% increase in foreign aid is on average equivalent to around $161,000 in the case of LMICs and $243,000 in SSA.

Through this study we add to the literature in several ways. As far as we know this is the first paper to explicitly account for the endogeneity of foreign aid (for HIV) in studying its impact on new infections of HIV. In fact it is one of the few papers to focus on this crucial indicator. Earlier studies have either ignored the endogeneity issue, used a roughly constructed measure of new infections or not focused on our outcome. We are also able to study the effects separately for adults and children. We eliminate any correlation with time-invariant unobservables through a fixed effects specification and use donor GDP as a powerful instrument to recover causal estimates. The other study that also considers the same question as us is that of Nunnenkamp and Ohler (2011) who find negative coefficients in their various specifications but none of which are statistically significant. Given this result the majority of their paper focuses on mortality as an outcome. We are thus able to provide crucial evidence on the effectiveness of aid for HIV in controlling the spread of the

epidemic, a question that is particularly relevant given the calls to increase the amount of funding channeled to the disease and concerns of displacement of other diseases. Our results are reassuring in that the vast of amounts of aid channeled towards the treatment, prevention and control of HIV do not seem to have been entirely misplaced.

Our study however has some limitations. The epidemiological and medical literature on HIV has recently shifted focus from behavior change and prevention programs to the idea of "treatment as prevention" (Cohen et al., 2011), recognizing that recent medical developments mean HIV positive individuals who adhere to their treatment are virally suppressed and therefore less likely to infect other individuals. An important question is whether the reductions we see are channeled through investments in prevention and behavior change programs or through increases in treatment. We are unable to shed light on this question as we lack data on foreign aid expenditure categories. As WHO extends its treatment threshold and more individuals become eligible for treatment, future research into the channels through which foreign aid influences new infections will become more relevant. This crucially also depends on how transparent donors are in their aid allocation decisions and the extent to which data on expenditure categories is made available. However, there still remains the larger question of whether disease specific "vertical" programs are the most appropriate methods of allocating development assistance for health. Recent years have seen a gradual shift towards more horizontal approaches to health care financing with health system strengthening becoming a core component of aid allocations which will hopefully limit displacement of funding from other diseases.

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Imperial College Business School
Tanaka Building
South Kensington Campus
London SW7 2AZ
United Kingdom

T: +44 (0)20 7589 5111
F: +44 (0)20 7594 9184

www.imperial.ac.uk/business-school