Impact of Development Assistance for Health on Maternal and Child Health:
Findings from the Global Burden of Disease 2016

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A dissertation
submitted in partial fulfillment of the
requirements for the degree of

Master of Public Health

University of Washington

2017

Reading Committee:

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Program Authorized to Offer Degree:

School of Public Health
Abstract


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Development assistance for health (DAH) has grown to US$36.4 billion annually, but its impact on population health – particularly nonfatal outcomes – remains unclear. I develop a cross-national longitudinal data analysis combining DAH disbursement records with maternal and child health outcomes from the Global Burden of Disease 2016. Controlling for development status and domestic health expenditure, I estimate the effect of DAH on health outcomes. The elasticity of aid on maternal and child mortality (-0.026 and -0.026) is similar, but both are greater than the elasticity for nonfatal maternal and child outcomes (-0.007 and -0.004). Based on these findings, if the United States enacted its proposed 24% DAH budget cut, I estimate that 8938 fewer child and 497 maternal deaths would be averted annually.
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I thank Nick Kassebaum and David Smith for their intellectual curiosity and unfailing patience. Theo Vos and Joe Dieleman have been invaluable assets without which the comprehensive data and analysis wouldn’t have been possible. Sincere gratitude goes to Emmanuela Gakidou, Sean Lassiter, Krista Clouser, and Bernardo Hernandez Prado for their tireless work to advance for the Post Bachelor Fellowship within IHME and UW.
1. Introduction

Development Assistance for Health (DAH) has increased from US$7.2 billion in 1990 to $36.4 billion in 2015, growing at 11.3% annually from 2000-2010 (Dieleman 2016). Concurrently, the world has improved key health indicators listed in the Millennium Development Goals, surpassing a 50% decrease in both child and maternal mortality rates (Naghavi, in press). However, substantial debate remains over the effects of DAH on economic and health development. Randomized trials have heralded the success of widely funded projects such as vitamin A supplementation and insecticide treated bed nets (Jones 2003), but critics debate the evidence that DAH improves population-level outcomes.

Table 1 summarizes cross-national studies evaluating the effect of DAH on national health outcomes. Past aid evaluations have focused outcomes ranging from insecticide-treated net coverage (Flaxman 2010) to national health indicators including infant and child mortality, adult mortality, life expectancy, and cause-specific mortality. As the population health outcome (e.g. national life expectancy) becomes increasingly separated from interventions (e.g. district-level ITN distribution) from a causal and geographic standpoint, effect sizes and statistical power diminish. Wilson (2011) cites the conflicting results of eight different models—albeit many of which he caveats as inappropriate—to demonstrate that aid evaluation is extremely sensitive to model specification. In attempts to estimate the effect of PEPFAR funding on adult mortality, Bendavid (2012) analyzed mortality rates from 2004-2008 and came to a different conclusion than Duber (2010) did using rates from 2000-2006. Reasons for null results extend beyond statistical concerns – common criticisms of DAH itself include poor program implementation and corruption (Easterly 2011), the negative effect of DAH on domestic health expenditure in the recipient country (Lu 2010), and the observation that “DAH appears to be following success,
rather than *causing it*” (Wilson 2011). Several authors have refuted the last critique using Granger causality tests to confirm directionality of causation from aid to health improvement (Bendavid 2014, Ventelou 2013).

<table>
<thead>
<tr>
<th>Citation</th>
<th>Exposure</th>
<th>Outcomes of interest</th>
<th>Result of primary analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Williamson 2008</td>
<td>Overall DAH</td>
<td>Life expectancy, U5MR, mortality, immunizations</td>
<td>Inconclusive</td>
</tr>
<tr>
<td>Bendavid 2009</td>
<td>PEPFAR focus</td>
<td>HIV deaths, HIV prevalence</td>
<td>Conclusive for HIV deaths, inconclusive for HIV prevalence</td>
</tr>
<tr>
<td>Mishra 2009</td>
<td>Overall DAH</td>
<td>Infant mortality</td>
<td>Conclusive</td>
</tr>
<tr>
<td>Duber 2010</td>
<td>PEPFAR focus</td>
<td>14 health indicators (including adult mortality rate, U5MR, immunization rates)</td>
<td>Inconclusive; no significant relationship in 13 of 14 indicators</td>
</tr>
<tr>
<td>Wilson 2011</td>
<td>Overall DAH</td>
<td>Life expectancy, U5MR, IMR</td>
<td>Inconclusive; results sensitive to model specification</td>
</tr>
<tr>
<td>Bendavid 2012</td>
<td>PEPFAR focus</td>
<td>Adult mortality</td>
<td>Conclusive</td>
</tr>
<tr>
<td>Feeny 2013</td>
<td>Overall DAH</td>
<td>Immunization</td>
<td>Conclusive</td>
</tr>
<tr>
<td>Bendavid 2014</td>
<td>Overall DAH</td>
<td>Life expectancy, U5MR</td>
<td>Conclusive</td>
</tr>
<tr>
<td>Hsiao 2015</td>
<td>DAH targeted to malaria, HIV, TB</td>
<td>Cause-specific mortality</td>
<td>Conclusive for HIV and malaria mortality, inconclusive for TB</td>
</tr>
<tr>
<td>Negeri 2016</td>
<td>Overall DAH</td>
<td>IMR in Sub-Saharan Africa</td>
<td>Conclusive</td>
</tr>
</tbody>
</table>


The studies in Table 1 focus on process indicators or mortality measures, but nonfatal conditions account for 34% of the total burden (Vos, in press). Additionally, non-fatal outcomes in children can pose a threat of long-term cognitive and motor impairment (Lozoff 2006). Causes of death and disability can differ – the leading causes of child mortality in 2015 are neonatal disorders and lower respiratory infections, whereas the leading causes of child morbidity are iron-deficiency anemia, skin diseases, and protein-energy malnutrition (Vos 2016). Hence,
improvements in child mortality and morbidity are logically correlated, but not directly proportional.

Many past studies have tried to address exogenous variation using different control variables, most frequently Gross Domestic Product (GDP) per capita. However, it is surprising that none have controlled for changes in the biggest component of spending, namely domestic (i.e., non-DAH) health expenditure (DHE). Even in the lowest income countries, DHE still accounted for 64% of 2014 healthcare spending, rising to 97% of spending in lower-middle income countries (Dieleman 2017).

In this analysis, I build a modelling framework drawing from the analytic structures of Bendavid (2014) and Hsiao (2015) to assess the impact of DAH on mortality and morbidity indicators from the Global Burden of Disease 2016. By incorporating estimates of DHE, I aim to mitigate the unobserved variable bias inherent in previous analyses. Additionally, I expand upon previous efforts by assessing the impact of DAH on both fatal and nonfatal health outcomes. Using a longitudinal panel dataset of health outcomes and health expenditure from 136 DAH recipient countries from 1990-2016, I assess whether child and maternal morbidity has the same response to DAH as child and maternal mortality.
2. Methods

2.1 Data Sources

Both economic and health outcome estimates were provided by the Institute for Health Metrics and Evaluation (IHME). The IHME DAH dataset aggregates disbursement records from various agencies, including the Organization for Economic Cooperation and Development (OECD), and categorizes it by source, channel, recipient, and health focus area from 1990-2016. Transfers between agencies are removed and aid is inflation-adjusted to 2015 USD. The Global Burden of Diseases, Injuries, and Risk Factors (GBD) is a comprehensive study estimating mortality and disability of all causes of death and disability from 1990-2016. As part of its Financing Global Health (FGH) efforts, IHME also estimates total health expenditure, from which I subtract DAH to calculate domestic health expenditure (DHE). I restrict all analyses to countries receiving at least 10 years of DAH disbursement. Palestine, Zimbabwe, and North Korea were excluded from the analysis because DHE is unavailable due to missing data on national health expenditure. I transform DHE and DAH to per capita estimates using GBD population figures.

The analysis uses comparable measures of mortality and morbidity: Years of Life Lost (YLL) measure the burden of premature death calculated as number of deaths multiplied by standard life expectancy at time of death and Years Lived with Disability (YLD) measure the burden of disability calculated as the summed prevalence of disabling sequelae multiplied by corresponding standard disability weights. Maternal mortality burden is defined as the YLL rate due to direct and indirect complications of pregnancy in women; for this analysis I have included only the age range of 15-49 years. Child mortality burden is defined as the YLL rate for all causes in children under 5 years of age. I choose to use the rate of YLL rather than the mortality
rate itself to maximize comparability to the nonfatal component of this analysis, which is measured in YLDs; interpretation of YLL rates should otherwise be identical to mortality rates. GBD also estimates Socio-Demographic Index (SDI) for each location-year. SDI is an indicator, scaled from 0-1, calculated as a geometric mean of income per capita, maternal education, and total fertility rate. SDI is intended to serve as a single comprehensive measure of societal development that incorporates more than just national income.

2.2 Panel Data Analysis

I first conduct several exploratory analyses to examine the relationship between health aid and health outcomes. First, I assess the allocation of aid by comparing cumulative aid received from 1990-2016 to development status and child mortality rates in 1990. Second, I look at the overall association between DAH received and health outcomes to assess overall trends before the more nuanced regression analysis.

I perform a longitudinal analysis of the association between DAH and health outcomes using a country and time-period fixed effects model. Country fixed effects control for time-invariant unobserved determinants of health. Time-period fixed effects allow non-linear changes in health outcomes. I use the following equation for all health outcomes:

\[
\log (H_{it}) = \beta_1 \log (H_{it-1}) + \beta_2 \log (DAH_{it-1}) + \beta_3 \log (DHE_{it}) + \beta_4 SDI_{it} + \alpha_i + \mu_t + \varepsilon_{it}
\]

where \(H_{it}\) is the health outcome in in country \(i\) and time period \(t\), \(H_{it-1}\) is the lagged health outcome, \(DAH_{it}\) is the DAH per capita between time period \(t-1\) and \(t\), \(DHE_{it}\) is the DHE per capita in time period \(t\), \(\alpha_i\) and \(\mu_t\) are the country and time-period fixed effects, and \(\varepsilon_{it}\) is the error term. As both the exposure \((DAH_{it-1})\) and outcome \((H_{it})\) are log-transformed, I interpret the
coefficient of interest, $\beta_2$, as the DAH elasticity of health, or the percent change in health outcome resultant from a 1% change in health aid. I fit the model to 5-year time-periods, which reduces autocorrelation of outcome and annual variability of DAH (Mishra 2009). Since the model requires a lag of health outcomes and DAH, both of which begin in 1990, 1995 is the first estimation year (with lagged health outcomes from 1990 and DAH from 1990-1994).

2.3 Granger Causality

To examine the temporality of the association between DAH and health outcomes, and provide evidence for causal inference, I test for Granger causality. Granger causality is a common econometric concept used to assess the time directionality of an association between two or more variables (Bendavid 2014). If changes in DAH precede health improvements, I expect lagged DAH should help predict future health improvements beyond past health trends alone. In other words, the equation (1) above should be more predictive than if the DAH$_{it-1}$ term were removed. I interpret the F statistic estimated by the Wald method as evidence for a difference between the two linear models.

Conversely, we can assess if past health improvements precede DAH by comparing the following autoregressive equations for DAH:

2) $\log(\text{DAH}_{it}) = \beta_1 \log(\text{DAH}_{it-1}) + \beta_3 \log(\text{DHE}_{it}) + \beta_4 \text{SDI} + \alpha_i + \mu_t + \epsilon_{it}$

3) $\log(\text{DAH}_{it}) = \beta_1 \log(\text{DAH}_{it-1}) + \beta_2 \log(H_{it-1}) + \beta_3 \log(\text{DHE}_{it}) + \beta_4 \text{SDI} + \alpha_i + \mu_t + \epsilon_{it}$

If equation (3) is statistically different from equation (2), there is evidence that health improvement – which I consider the outcome in the primary analysis – precedes the DAH. No correction was made for multiple comparisons.
2.4 Sensitivity Analysis

In the primary analysis, I use all DAH disbursement as the exposure, as all health focus areas affect child health and several affect maternal health. As a sensitivity analysis, I parse DAH targeted to outcome and other DAH, and model both independent exposures simultaneously. Disbursements were categorized into 11 health focus areas by Dieleman (2016), including child health, maternal health, HIV malaria, tuberculosis, and health sector strengthening.

All analyses were performed using R version 3.3.2.

3. Results

From 1990-2016, India received the most DAH ($632 million per year) and the Federated States of Micronesia received the most DAH per capita ($98 per year). Sub-Saharan Africa received $127 billion in aid during the period, almost half of all disbursement. There is a positive but weak correlation between 1990 child mortality rates and cumulative aid received during the study period ($r^2 = 0.05$), and negative correlation between SDI and aid received ($r^2 = 0.11$) (Figure 1). Interestingly, the opposite trend is observed within Sub-Saharan Africa, in which aid per capita tends to flow to more developed countries and those with lower child mortality rates. The direction of these associations do not change whether evaluating the entire study period from 1990-2016 or focusing on each decade, using SDI or child mortality in e.g. 2000 and cumulative aid in the following decade.
Figure 1. Cumulative DAH per capita from 1990-2016 against 1990 child mortality rate. Panels A and B include all DAH recipient counties; C and D include only DAH recipient countries in Sub-Saharan Africa. The log values of 2, 4, 6, and 8 correspond to $7, $55, $403, and $3000 cumulative DAH per capita.

From 1990-2016, DAH recipient countries averaged a 3.7% annual reduction in child mortality rate, falling short of the 4.4% annual reduction necessary to meet Millennium Development Goal 4. However, mortality fell more quickly than child and maternal morbidity rates, which fell at 2.3% and 0.5%, respectively. There is a positive correlation between cumulative aid received from 1990-2016 and annualized rate of change of health outcomes (Figure 2), meaning that on average locations receiving more aid had lesser decreases in disease burden. However, the opposite trend is observed in Sub-Saharan Africa, in which countries receiving more cumulative aid per capita have more rapid improvements in all health outcomes. These conflicting patterns motivate a regression approach.
Figure 2. Cumulative DAH per capita from 1990-2016 and annual rates of change of health outcomes. The black dashed line represents the overall association in DAH recipient countries; the pink line represents the association within Sub-Saharan Africa recipients. The log values of 2, 4, 6, and 8 correspond to $7, $55, $403, and $3000 cumulative DAH per capita.

The regression model helps unpack some of the more nuanced relationships between DAH and outcomes in recipient countries. DAH was significantly associated with health improvements for all outcomes in the regression model (full regression results in Table 2). DAH and DHE elasticities of health are displayed in Figure 3. DAH elasticities were the same for both mortality outcomes (-0.026 [p<0.001]) and were greater than that of the morbidity outcomes (-0.004 [p=0.003] for child YLD rate and -0.007 [p=0.004] for maternal YLD rate). The mortality elasticities can be interpreted that, for every 10% increase in DAH per capita, there was a 0.26% decrease in maternal and child YLL rates. The implied improvement in YLD rates due to DAH were correspondingly much more modest. DHE elasticities were non-significant for maternal
YLL and YLD rates (-0.009 [p=0.771] and -0.019 [p=0.055], respectively), but were actually bigger than DAH elasticities for child YLL and YLD rates (-0.09 and -0.019 [both p<0.001], respectively).

Table 2. Country and period fixed effects regression of DAH and maternal and child health outcomes with lagged health outcome and control variables. Year 1995 is reference for the time period fixed effects.

<table>
<thead>
<tr>
<th></th>
<th>Under 5 YLL</th>
<th>Under 5 YLD</th>
<th>Maternal YLL</th>
<th>Maternal YLD</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Intercept)</td>
<td>0.876</td>
<td>-0.135</td>
<td>-0.707</td>
<td>-1.948</td>
</tr>
<tr>
<td>B</td>
<td>std. Error</td>
<td>p</td>
<td>std. Error</td>
<td>p</td>
</tr>
<tr>
<td>Year 2000</td>
<td>-0.019</td>
<td>0.02</td>
<td>0.26</td>
<td>0.00</td>
</tr>
<tr>
<td>Year 2005</td>
<td>-0.035</td>
<td>0.02</td>
<td>0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>Year 2010</td>
<td>-0.062</td>
<td>0.03</td>
<td>0.22</td>
<td>0.01</td>
</tr>
<tr>
<td>Year 2016</td>
<td>-0.162</td>
<td>0.04</td>
<td>&lt;.001</td>
<td>-0.005</td>
</tr>
<tr>
<td>log(DAH per capita)</td>
<td>-0.026</td>
<td>0.00</td>
<td>&lt;.001</td>
<td>-0.064</td>
</tr>
<tr>
<td>log(Lagged Outcome)</td>
<td>0.645</td>
<td>0.03</td>
<td>&lt;.001</td>
<td>0.494</td>
</tr>
<tr>
<td>SDI</td>
<td>-0.914</td>
<td>0.27</td>
<td>&lt;.001</td>
<td>-0.254</td>
</tr>
<tr>
<td>log(DHE per capita)</td>
<td>-0.090</td>
<td>0.02</td>
<td>&lt;.001</td>
<td>-0.019</td>
</tr>
</tbody>
</table>

Figure 3. Elasticities of maternal and child health outcomes, with 95% confidence intervals.
For all four outcomes, Granger causality analysis supports a path from DAH to improved YLL and YLD rates (Figure 4). However, the Wald tests are also significant (p < 0.05) in the reverse direction for both mortality outcomes, but not for the nonfatal outcomes, and not as strongly as the forward direction.

Figure 4. Wald tests with F statistics and p-values for Granger Causality tests for the directionality of DAH and health outcomes. The primary analysis treats DAH as the exposure.

In the sensitivity analysis, the elasticities of non-targeted DAH remained significant for all outcomes, but those of targeted DAH were not significant (Figure 5).
Figure 5. Elasticities of health outcomes when targeted DAH and non-targeted DAH are treated as independent exposures, displayed as point estimate, standard error, and 95% confidence interval.
4. Discussion

These results illustrate that at a national level, health aid is associated with child and maternal health improvements after controlling for development status and domestic health expenditure. Surprisingly, the elasticities of targeted aid are weaker than those of non-targeted aid. DAH affects non-target health outcomes by both direct and indirect means. In the case of child health, DAH directed toward malaria or HIV (particularly prevention of mother-to-child transmission) directly affects child mortality. Meanwhile, many donor organizations have begun shifting from vertical (disease-specific) interventions to horizontal (e.g. infrastructure or systems-based) interventions (Barnighausen 2012), which suggests targeted DAH may increasingly have spillover effects for other health outcomes.

For both maternal and child health, the elasticity of DAH is greater for mortality than morbidity, likely due to a combination of disease and programmatic factors. Millennium Development Goals (MDG) 4 and 5 explicitly target child and maternal mortality rates, and common interventions such as skilled birth attendance (an indicator for MDG 5) improve infant and maternal mortality without directly improving child malnutrition, a leading cause of child morbidity. This disconnect between mortality and morbidity gains is not unique to child and maternal health. About half of PEPFAR funding supports ART treatment (Bendavid 2009), which improves individual health outcomes but can increase population morbidity rates due to increased survival time. Conversely, improved survival of those with HIV can increase cohesiveness of families and communities, which can be associated with improvements in child health outcomes.
For all four outcomes, aid precedes health improvement, supporting Granger-causality and Hill’s causal criterion of temporality (Hill 1965). However, for both fatal outcomes, there was evidence this directionality flows both ways, as suggested by Wilson (2009). These two phenomena (aid driving health improvement, and health improvement attracting aid) are not mutually exclusive. Bidirectional temporality is not a critique on the effectiveness of health interventions (indeed, I emphasize that there is consensus on the efficacy of many core public health interventions), but provides another angle with which to question the rationality and equity of aid allocation. Equitable allocation is a problem at both the national and subnational level – an analysis of georeferenced aid data in Malawi did not detect a relationship between malaria burden and DAH, and found that greater existing infrastructure predisposed areas to aid receipt (Marty 2017). Such allocation patterns are not just a moral issue; a 2017 UNICEF report concluded that aid to the most deprived saved almost twice as many lives as equivalent aid to less deprived groups (“Narrowing the Gaps”).

A cross-national approach has key strengths and limitations. Results must be interpreted as a snapshot of the past system of development aid with respect to allocation and implementation. While Bendavid (2014) suggests aid effectiveness has increased over time, this analysis treats elasticity of DAH as static and estimates an average impact of DAH over the exposure period. Aid disbursement not directly applied to programs (whether due to foreign overhead or domestic corruption) reduces the aid elasticity in this analysis. While this provides a less optimistic impact assessment than a non-generalizable cost-effectiveness analysis (Hutubessy 2002), it is a valuable perspective for international agencies and donors wishing to estimate overall impact. Past analyses estimate that a dollar of DAH to a government displaces between $0.43 and $1.14 of government expenditure (Lu 2010). The present analysis uses DHE
as an independent control and therefore may exempt DAH elasticity estimates from the undesirable effects of fungibility.

A cross-national assessment of aid impact can be leveraged to predict the effects of various aid scenarios. Presently, the US government is considering a $2.5 billion cut in aid that amounts to 24% of its 2018 DAH budget. If each recipient country lost 24% of its American DAH, my elasticity estimates predict 8938 fewer child and 497 fewer maternal deaths would be averted annually. This estimate is within the range of an analysis using cost-per-life-saved estimates to predict between 7,000 and 31,300 consequent child and maternal deaths as well as additional HIV and TB infections (Kates 2017). These estimates come with many caveats, including the assumptions that a decrease in funding has the reciprocal effect of past increases and that all other aid is unaffected.

The Global Burden of Disease 2015 launched with the refrain “development is not destiny.” We conclude that while health aid improves health outcomes, particularly mortality, marked effects of development and domestic health expenditure remind us that development aid is not destiny either. The diminished elasticity of aid on nonfatal outcomes merits further assessments of interventions on nonfatal conditions, which have not experienced the improvements of mortality. Meanwhile, aid programs must strive to achieve equity in aid allocation and maintain rigorous implementation and evaluation of projects to ensure that future impacts of aid outpace those of the past.


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