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The Effects of Foreign Aid on Government Policies: Theoretical and Empirical Analyses

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THE EFFECTS OF FOREIGN AID ON GOVERNMENT POLICIES: THEORETICAL AND EMPIRICAL ANALYSES

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TITLE: THE EFFECTS OF FOREIGN AID ON GOVERNMENT POLICIES: THEORETICAL AND EMPIRICAL ANALYSES

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Chapter 1 develops a two-period general equilibrium trade-theoretic model to examine if foreign aid discourages the recipient countries from pursuing trade liberalization. In the model, foreign aid is given to the recipient in period two and its amount is negatively related to the period-one real income. The recipient optimally chooses a tariff on imports. It can also choose domestic investment endogenously in period one, and this choice has an important bearing on our main result. We consider two variants of the model depending upon whether the recipient can or cannot have access to international borrowing. In the case without international borrowing, when domestic investment is exogenous, optimal tariff is zero. In contrast, when domestic investment is endogenous, optimal tariff is positive. This positive optimal tariff is induced by the link of aid negatively to the period-one real income. In the case with international borrowing, even though domestic investment is exogenous, optimal tariff is positive. But the reason for the positive tariff is its beneficial effect on an improvement in the terms-of-trade of international borrowing. When, in addition, domestic investment is endogenous, the tying of aid increases positive optimal tariffs further.

Chapter 2 develops a microeconomic model of health policies and the optimal allocation of health aid in a poor recipient country. In the model, each poor household in the country chooses the optimal number of sick children taken to hospitals to maximize its

lifetime utility. There are three policy options for policymakers to improve public health: raising the quality of health care, providing more preventive care and reducing the cost of health care. We examine how three policy options influence the optimal number of sick children who are medically treated. Also, the country's health authority allocates health aid for three policy options to support poor households' lifetime utility maximization. We find that more health aid should be allocated for cost reduction in health care so as to help poor households maximize their lifetime utility.

Chapter 3 primarily examines the hypothesis that there is heterogeneity in health aid, that is, different types of health aid work differently for health outcomes in aid-recipient countries. In order to test our hypothesis, we first disaggregate health aid per capita data into three policy options: health aid per capita for improving the quality of health care, health aid per capita for providing preventive care and health aid per capita for reducing the cost of health care. Then, we empirically examine the effects of disaggregated health aid on three different health indicators: child mortality, life expectancy and death rate. Using a panel data set of 119 aid-recipient countries from 1975 and 2010, we find supporting evidence for the hypothesis of heterogeneity in health aid. We find no empirical evidence of the beneficial effects of health aid on reducing child mortality. In contrast, we find that an improvement in life expectancy and a reduction in death rate are driven mostly by health aid for reducing the cost of health care. We also find that there is heterogeneity in the allocation of health aid. Health aid for preventive care and the cost reduction of health care is allocated by the needs of the recipients. However, more health aid for the quality of health care flows to countries with better health status.

DEDICATION

This dissertation is dedicated to my Lord and Savior, Jesus Christ, who pursued me relentlessly, even when I wandered far away from him in order to go my own way, and led me to study economics to help the world's poor for His sake. This study is also dedicated to my lovely wife, Yoo Kyung Lee, and adorable son, Jacob Yuncheol Shin for their endless patience, support and encouragement. I also dedicate this research to my family in South Korea: my father, Seok Gyun Shin, my mother, Young Bok Kwon, my father-in-law, In Sik Lee, my mother-in-law, Byung Eun Jeong, my brother, Hyeon Min Shin, and my sister-in-law, Eun Young Lee. Their prayers and petitions with tears kept me in God's hands.

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CHAPTER 1

DOES FOREIGN AID DISCOURAGE TRADE LIBERALIZATION? A THEORETICAL ANALYSIS

1.1 INTRODUCTION

Over the past few years Kenya has performed a curious mating ritual with its aid donors. The steps are: one, Kenya wins its yearly pledges of foreign aid. Two, the government begins to misbehave, backtracking on economic reform and behaving in an authoritarian manner. Three, a new meeting of donor countries looms with exasperated foreign governments preparing their sharp rebukes. Four, Kenya pulls a placatory rabbit out of the hat. Five, the donors are mollified and the aid is pledged. The whole dance then starts again. (The Economist, 1995)

The above describing the aid donor-recipient table between the Kenyan government and the donor countries has been quoted by the voluminous literature on foreign aid (see, for example, Dollar and Easterly, 1999; Kanbur, 2000; Svensson, 2000b) to show how foreign aid obstructs policy reform in aid-recipient countries. In the quotation above, the Kenyan government decided to undo their policy reform and receive foreign aid rather than to endure the hardships of reform and achieve sustainable economic growth. But this may not be an issue only in Kenya but in most recipient countries.

Foreign aid is originally designed for helping poor countries achieve sustainable economic growth and poverty reduction. Thus, it is no exaggeration to say that foreign aid is at least partly disbursed by the needs of the recipients.[1] The idea that more aid should be disbursed to poorer countries has been taken for granted. Quoting passages from Pearson Commission (1969) and Commission for Africa (2005), Easterly (2007) shows that

[1] In reality, of course, aid allocation is not only driven by the needs of the poor recipients but also by the self-interest motives of the donors. For example, aid is in part given as an incentive to reward political reforms. See Maizels and Nissanke (1984), Trumball and Wall (1994), Alesina and Dollar (2000), Burnside and Dollar (2000), Alesina and Weder (2002) and Bandyopadhyay and Wall (2007) for the detailed information of the determinants of aid.

this idea has been widely supported in the aid sector from the past to the present.[2] Also, Birdsall and Williamson (2002) advocate the reform of the current aid architecture and make sure that their "new aid architecture" implies that 'both fundamental reform in the way aid delivered and a substantial increase in the volume of aid to the world's poorest countries for the next decade and beyond (p. 101).' Easterly and Pfutze (2008) confirm that, in reality, more aid is being directed towards the poorest countries. They find that, since the 1960s, about 60% of total aid has been headed to low-income countries, that is, the least developed countries and other low-income countries, but, since in 1973 World Bank president Robert McNamara emphasized the effect of aid on poverty alleviation, the share of total aid towards least developed countries has been expanding at the expense of the share of aid towards other low-income countries.[3]

Interestingly, a strand of the theoretical literature has indicated that the foreign aid, if it is disbursed by the need of the recipients, has an adverse effect on the recipients' incentives to pursue good domestic policies. Svensson (2000b) argues that it is a moral hazard problem that adversely affects the recipients' incentive to undertake policy reforms. In his moral hazard model, anticipating that aid will be allocated to the recipients in most need, the recipient governments do not exert every effort in improving the welfare of the poor. Providing a game-theoretic rent-seeking model, Svensson (2000a) theoretically shows that the foreign aid intensifies rent-seeking activities and corruption among different interest groups to appropriate the government revenue, thereby inducing the government to reduce their productive public expenditure.[4]

[2] According to Easterly (2007), Pearson Commission (1969) presents this idea by saying *'IDA (International Development Association of the World Bank) has decided to make a special effort to assist the poorest members in project preparation so that they can benefit more fully from IDA financial assitance (p. 226).'* Commission for Africa (2005), which echoes Pearson Commission's (1969) idea, proposes the improvements of aid quality by *'allocating aid to countries where poverty is deepest and where aid can be best used (p. 99).'*

[3] Of course, the extent to which the distribution of foreign aid is directed towards the poorest countries differs across aid donors. Constructing aid concentration curves for monetary poverty, child malnutrition, primary school enrollments, and under-five mortality, Baulch (2006) examines whether aid is distributed to the poorest countries. He finds that the United Kingdom and World Bank distribute around two-thirds of their development assistance to the low income countries, whereas the United States and European Commission spend the majority of their aid budgets in middle income countries.

[4] Reinikka and Svensson (2004) confirms how serious rent-seeking activities and corruption are in aid-

Rodrik (1996) also claims that foreign aid reduces the costs both of reform and of doing nothing. As he says, foreign aid should help reforms get launched and sustained by reducing these costs. When aid is disbursed by the needs of the recipients, that is, more aid is directed towards poorer recipients, however, foreign aid also reduces the cost of doing nothing and can also be considered as the benefit of doing nothing or avoiding reforms by compensating for these costs. What will happen if the cost of doing nothing or delaying reforms is less than these benefits? Needless to say, the recipient governments will have little incentive to push through reforms, thereby worsening their welfare and getting more aid. This is what the Kenyan government actually did in the early 1990s. Collier (1997) points out that, in African countries where their policies have improved, the amount of aid received has rather decreased, whereas most aid-increasing African countries have made their policies worse.[5]

In this study, we theoretically examine if foreign aid discourage the recipients from pursuing good policy reforms, when more aid is allocated to poorer recipients. In particular, we focus on the adverse effect of foreign aid on trade liberalization. The imposition of a tariff has been considered to be a bad trade policy for a small open economy. In the partial equilibrium approach of a small open economy, a tariff cannot improve the commodity terms-of-trade but only causes deadweight loss by decreasing demand for imports and reallocating resources to inefficient production. Collier (2007) claims that high tariffs in poor countries protect their domestic firms from external competition, thereby making

recipient countries. Surveying primary school in Uganda, they find that from 1991 to 1995 only 13% of grant reached the primary schools, whereas most of the grant was captured by local officials and politicians. Svensson (2000a) also provides interesting empirical evidence that foreign aid worsens corruption in countries more likely to suffer from competing social groups. Two recent empirical studies offer the conflicting results on the effect of aid on corruption. Okada and Samreth (2012) investigate the effect of aid on corruption in 120 developing countries using a quantile regression method and conclude that foreign aid reduces corruption and its effect is bigger in less corrupt countries. On the other hand, using the data from 52 African countries, Asongu (2012) finds robust evidence that aid worsens corruption and points out that Okada and Samreth's (2012) finding may not be relevant for Africa.

[5]Dollar and Easterly (1999) introduce Zambia as an example showing that foreign aid is closely related to bad policies. From 1970 through 1993 period, Zambia, although they were getting poorer, had bad policies and delayed their reforms, whereas the amount of foreign aid flowed into Zambia increased continuously, thus reaching 11% of real GDP by the early 1990s.

them less productive and shifting the cost of protection to domestic consumers.

There is abundant theoretical and empirical literature showing that trade liberalization is growth-enhancing and poverty-alleviating in low-income aid recipients in the long run. The innovation-based endogenous growth literature has theoretically shown that trade openess generates technological progress, thereby speeding up productivity growth.[6] Empirical evidence also broadly supports the link between trade liberalization and growth. For example, Wacziarg and Welch (2008) reexamine Sachs and Warner's (1995) study with a new data over the 1950-98 period and find that countries that liberalized their trade regimes achieved about 1.5 percentage points higher average annual growth rates than those before liberalization. Onafowora and Owoye (1998) find empirical evidence that economic growth in Sub-Saharan African countries is driven mainly by trade liberalization. Surveying recent theoretical and empircal evidence of trade liberalization and poverty, Winters et al. (2004) conclude that trade liberalization is, in principle, beneficial for the poor, even though its impact on poverty depends upon the environment in which it is carried out.[7] Unfortunately, however, Benedek et al. (2012) find empirical evidence that foreign aid impedes trade liberalization in aid-recipient countries. They examine how foreign aid affects a variety of tax revenues such as VAT, exercise tax, income tax and tariff revenues and find that foreign aid increases only tariff revenues, whereas it decreases the other tax revenues. One of the reasons for this may be higher tariffs induced by foreign aid.

This paper is the first to theoretically investigate whether foreign aid discourages trade liberalization in aid-recipient countries. For this study, we develop a two-period general equilibrium trade-theoretic model of an aid recipient country with a small open economy. The general equilibrium trade-theorectic model is widely used and accepted in the literature

[6] See, for example, Grossman and Helpman (1991) and Rivera-Batiz and Romer (1991a, b) for the link between trade and growth.

[7] Winters et al. (2006) also point out that trade liberalization by its nature accompanies distributional adjustments, and poorer households may be less able to protect themselves against the adverse effects of the adjustments. Hence, they emphasize that trade reform should be accompanied by complementary social policies strengthening social protection for losers.

of international trade and foreign aid theory.[8] In our model, foreign aid is given to the recipient country in period two and the amount of aid is negatively related to the level of the period-one real income, which reflects the fact that more aid is disbursed to poorer recipients. The recipient country chooses optimally a tariff on imports, and may have access to international borrowing in period one. It can also choose domestic investment endogenously in period one, and this choice has an important bearing on our main finding.

We consider two variants of the model depending upon whether the recipient can or cannot have access to international borrowing. Only when international borrowing is not allowed and domestic investment is exogenous, optimal tariff is zero, which means that, in this case, foreign aid does not discourage trade liberalization. When domestic investment is chosen endogenously but international borrowing is still not allowed, we find that foreign aid discourages the recipient from pursuing trade liberalization because aid negatively linked to the period-one real income induces positive optimal tariffs. In the case with international borrowing, even though domestic investment is exogenous, optimal tariff is positive. However, it is not foreign aid but intertemporal terms of trade (interest rate) that causes this positive tariff. Finally, when, in addition, domestic investment is endogenous, the tying of aid to the period-one real income increases optimal tariffs further. We also find that aid-induced positive optimal tariffs are higher in poorer countries in period one.

This study is closely related to two strands of literature. Djajić et al. (1999, 2004) incorporate an intertemporal optimization problem into the traditional static model of international trade and foreign aid and thus, in their intertemporal framework, aid affects welfare not only via the international terms-of-trade of commodity but also via domestic savings and investment. In the same fashion as Djajić et al. (1999, 2004), our intertemporal model allows the recipient country to choose domestic investment endogenously in period one. In reality, endogenous domestic investment represents that it is market-determined, whereas exogenous domestic investment is considered to be fixed or

[8] See, for example, Dixit and Norman, 1980; Bhagwati et al., 1985; Lahiri et al., 2002

regulated by government. Our study adds value to Djajić et al. (1999, 2004) by also allowing international borrowing to be endogenously chosen, while they assume the absense of international borrowing. Also, our study focuses on the role of foreign aid in the effect of a tariff on welfare, whereas Djajić et al. (1999, 2004) examines the effect of foreign aid on welfare. The main idea of this paper comes from the the other strand of literature, for example, Lahiri and Raimondos-Møller (1995, 1997a, 1997b) and Lahiri et al. (2002), that focuses on the optimal tariff in aid-recipient countries. In this study, therefore, we develop a theoretical model which incorporates two strands of literature in order to examine whether foreign aid discourages trade liberalization.

The remainder of this paper is organized as follows. In section 2, the theoretical model is developed. In section 3, we commit the comparative statics to answer our main question. In particular, section 3 is divided into two subsections according by whether international borrowing is allowed. Section 4 makes some concluding remarks.

1.2 THE THEORETICAL MODEL

We consider a two-period general equilibrium trade-theoretic model of a small open economy. In the model, a poor country participates in international trade and receives foreign aid from the world. Without any loss of generality, we assume that the aid-recipient country is an exporter of the numeraire good and an importer of the non-numeraire good by the principle of comparative advantage. Foreign aid, T, is given to the recipient country in the second period. In order to reflect the distribution of aid towards the poorest countries, the amount of foreign aid received in period two is assumed to be negatively related to the recipient's real income in period one, U^1. Mathematically, it is assumed that T is decreasing in U^1 at decreasing rate, that is, $T^{'} < 0$ and $T^{''} > 0$. The recipient country also invests in period one, augmenting its capital stock in period two. The domestic investment in period one can be chosen endogenously. Capital stock is assumed to be accumulated in the form of the numeraire good. The recipient country chooses optimally a

tariff, τ, on imports in period one, and may have access to international borrowing in period one. The amount of international borrowing, B, is determined by the optimal borrowing decision in period one, and the whole amount of principal and interest is repaid in period two. In this model, relative import price, P, is given exogenously because the recipient country is a small open economy so that its import policies cannot affect world prices and the imposition of a tariff cannot improve the terms-of-trade of commodity. In contrast, the recipient's international borrowing can affect world interest rate, and an increase in world interest rate worsens the terms-of-trade of international borrowing.

Assuming that tariff revenue and foreign aid are distributed to the consumers in the recipient in a lump-sum fashion, the general equilibrium conditions in the recipient country are given by (1) for the first period and (2) for the second period as:

$$E^1(1, P^1 + \tau, U^1) + I = R^1(1, P^1 + \tau, K^1) + \tau(E_2^1 - R_2^1) + B, \quad (1)$$

$$E^2(1, P^2, U^2) = R^2(1, P^2, K^1 + I) + T(U^1) - (1 + r)B, \quad (2)$$

where the superscripts 1 and 2 represent the period one and period two, respectively, $E(\cdot)$ and $R(\cdot)$ are the expenditure and revenue functions, respectively, P is the relative price of the non-numeraire good when the price of the numeraire good is normalized to one, U is the utility or real income, K is the capital stock, I is the amount of investment in period one, T is the foreign aid tranferred from the world to the recipient country in period two, B is the amount of international borrowing in period one, τ is the tariff on the non-numeraire good in period one, r is the international interest rate, and E_2^1 and R_2^1 are the partial derivatives of $E^1$and R^1 with respect to $P^1 + \tau$.

From the standard properties of duality theory in microeconomics, E_2^1 is the compensated demand function for the non-numeraire good and R_2^1 is the supply function of the non-numeraire good. Therefore, $E_2^1 - R_2^1$ is the excess demand for the non-numeraire good (or import demand) and $\tau(E_2^1 - R_2^1)$ represents the tariff revenue of the recipient

government from importing the non-numeraire good from the world. Equation (1) and (2) represent the budget constraints in the first period and the second period, respectively, for the representative consumer in the recipient country. In the equilibrium of the first period, the sum of consumption expenditure, E^1, paid for achieving U^1 at $P^1 + \tau$ and investment expenditure, I , should be equal to the sum of revenue, R^1, generated from production using the factor K^1 at $P^1 + \tau$, tariff revenue, $\tau(E_2^1 - R_2^1)$, and international borrowing, B. In the second period equilibrium, the sum of consumption expenditure, E^2, paid for achieving U^2 at P^2 should be equal to the sum of revenue, R^2, generated from production using the factor $K^1 + I$ at P^2, foreign aid, $T(U^1)$, and reimbursement of international borrowing, $-(1 + r)B$.[9]

The intertemporal welfare function of the representative consumer in the recipient country is time separable and takes the following simple form:

$$W(U^1, U^2) = U^1 + \frac{1}{1+\delta}U^2, \tag{3}$$

where δ is the constant rate of time preference in the recipient country. If domestic investment is taken to be endogenous, the representative consumer in the recipient country chooses the optimal quantity of investment to maximize his/her lifetime utility $W(U^1, U^2)$ in period one. By setting $\partial W/\partial I = 0$ as the first order condition, the optimal investment condition is obtained as:

$$(1 + \delta)E_3^2 = R_3^2 E_3^1, \tag{4}$$

where E_3^1 and E_3^2 are the reciprocals of marginal utility of income in the first and second period, respectively, and R_3^2 is the marginal revenue of capital in the second period, also representing the shadow price of capital in the second period. The left hand side is the marginal benefit of investment, that is, the welfare gain from the investment-induced

[9]The other production factors except capital stock are assumed to be unchanged through two periods and thus are omitted from the revenue function without any loss of generality. Also, the scrap value of capital stock at the end of the second period is assumed to be zero due to total depreciation.

increase in the second period consumption by allocating the marginal unit of the numeraire good to investment rather than consumption, whereas the right hand side is the marginal cost of investment, namely, the welfare loss from lower consumption in period one. Given any price level, $E^1$and E^2 are clearly increasing in $U^1$and U^2, meaning that $E^1_3 > 0$ and $E^2_3 > 0$ and, by the law of diminishing marginal utility of income, $E^1_{33} > 0$ and $E^2_{33} > 0$. Then, from these properties and the first order condition, $\partial(\partial W/\partial I)/\partial I = -[E^1_{33}E^2_3 + (1+\delta)E^2_{33}E^1_3]/(E^1_3)^3E^2_3 < 0$. Therefore, the second order condition is satisfied and guarantees that the optimal investment yields a maximum of the welfare function. In this model, endogenous investment means that the quantity of investment is market-determined and thus varies with changing market conditions, in particular, occurred by the imposition of a tariff.

The quantity of international borrowing, B, is also optimally chosen by the representative consumer in the recipient country in period one. By setting $\partial W/\partial B = 0$, we can get the optimal borrowing condition as:

$$(1+\delta)E^2_3 = (1+r)E^1_3. \tag{5}$$

The marginal cost of international borrowing on the left hand side is the period-two welfare loss from less consumption incurred by reimbursements, while the marginal benefit on the right hand side represents the period-one welfare gain caused by more consumption and investment. Also, that $\partial(\partial W/\partial B)/\partial B = -[E^1_{33}E^2_3 + (1+\delta)E^2_{33}E^1_3]/(E^1_3)^3E^2_3 < 0$ gives us a sufficient condition for a maximum of welfare.

A system of five equations from (1) to (5) can be solved for the five endogenous variables (U^1, U^2, I, r and W) as functions of two exogenous variables (τ and B). In the five-equation system, the optimal quantity of international borrowing, B, is taken to be an exogenous variable and affects interest rate, r. This enables us to pay attention to the terms-of-trade of international borrowing, that is, interest rate. After solving the model

above, we perform comparative static analyses to examine (i) how total welfare responds to a change in a tariff in an equilibrium, (ii) whether zero tariff is optimal for the recipient country, and (iii) how foreign aid affects the optimum tariff decision in the recipient country. We consider two variants of the model depending upon whether the recipient country can or cannot borrow from the international capital markets.

1.3 THE COMPARATIVE STATICS

Totally differentiating (1) and (2), using (4) and (5), we get

$$dU^1 = \frac{\partial U^1}{\partial \tau} d\tau + \frac{\partial U^1}{\partial B} dB + \frac{\partial U^1}{\partial I} dI, \tag{6}$$

$$dU^2 = \frac{\partial U^2}{\partial \tau} d\tau + \frac{\partial U^2}{\partial B} dB + \frac{\partial U^2}{\partial I} dI + \frac{\partial U^2}{\partial r} dr, \tag{7}$$

where

$$\begin{aligned}
\frac{\partial U^1}{\partial \tau} &= \frac{\tau(E^1_{22} - R^1_{22})}{E^1_3 - \tau E^1_{23}} < 0, \\
\frac{\partial U^1}{\partial B} &= \frac{1}{E^1_3 - \tau E^1_{23}} > 0, \\
\frac{\partial U^1}{\partial I} &= -\frac{1}{E^1_3 - \tau E^1_{23}} < 0, \\
\frac{\partial U^2}{\partial \tau} &= \frac{\tau(E^1_{22} - R^1_{22})}{(E^1_3 - \tau E^1_{23})E^2_3} T' > 0, \\
\frac{\partial U^2}{\partial B} &= -\frac{1+\delta}{E^1_3} + \frac{1}{(E^1_3 - \tau E^1_{23})E^2_3} T' < 0, \\
\frac{\partial U^2}{\partial I} &= \frac{1+\delta}{E^1_3} - \frac{1}{(E^1_3 - \tau E^1_{23})E^2_3} T' > 0 \text{ and} \\
\frac{\partial U^2}{\partial r} &= -\frac{B}{E^2_3} < 0,
\end{aligned}$$

where T' is the negative partial derivative of T with respect to U^1, E^1_{22} is the slope of the compensated demand curve for the non-numeraire good in period one, R^1_{22} is the slope of the supply curve of the non-numeraire good in period one, and E^1_{23} is the partial derivative

of the compensated demand for the non-numeraire good with respect to U^1 in period one. We know that $E^1_{22} < 0$ and $R^1_{22} > 0$. Thus, the slope of the excess demand for the non-numeraire good in period one is also negative, that is, $E^1_{22} - R^1_{22} < 0$. Now both the numeraire good and non-numeraire good are assumed to be normal goods. Then the "Hatta normality condition" (see, Hatta, 1977) is also assumed to be satisfied, which means that $E^1_3 - \tau E^1_{23} = E^1_3[1 - \tau(E^1_{23}/E^1_3)] = E^1_3(1 - \tau C^1_Y) > 0$.[10]

Totally differentiating (4) and (5), and substituting (6) and (7) for dU^1 and dU^2, we get dI and dr as functions of two exogenous variables, τ and B, as:

$$dI = \frac{\partial I}{\partial \tau} d\tau + \frac{\partial I}{\partial B} dB, \tag{8}$$

$$dr = \frac{\partial r}{\partial \tau} d\tau + \frac{\partial r}{\partial B} dB, \tag{9}$$

where

$$\frac{\partial I}{\partial \tau} = \frac{1}{\Omega}\left[-\frac{\tau(1+\delta)E^2_3E^1_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})} - \frac{(1+\delta)E^2_3E^1_{32}}{(E^1_3)^2R^2_{33}} + \frac{\tau(1+\delta)E^2_{33}(E^1_{22}-R^1_{22})}{E^1_3R^2_{33}(E^1_3-\tau E^1_{23})E^2_3}T'\right],$$

$$\frac{\partial I}{\partial B} = \frac{1}{\Omega}\left[-\frac{(1+\delta)E^2_3E^1_{33}}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})} - \frac{(1+\delta)^2E^2_{33}}{(E^1_3)^2R^2_{33}} + \frac{(1+\delta)E^2_{33}}{E^1_3R^2_{33}(E^1_3-\tau E^1_{23})E^2_3}T'\right] > 0,$$

$$\frac{\partial r}{\partial \tau} = \frac{1}{\Omega}\left[-\frac{\tau(1+\delta)E^2_3E^1_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2(E^1_3-\tau E^1_{23})} - \frac{(1+\delta)E^2_3E^1_{32}}{(E^1_3)^2} + \frac{\tau(1+\delta)E^2_{33}(E^1_{22}-R^1_{22})}{E^1_3(E^1_3-\tau E^1_{23})E^2_3}T'\right] \text{ and}$$

$$\frac{\partial r}{\partial B} = \frac{1}{\Omega}\left[-\frac{(1+\delta)E^2_3E^1_{33}}{(E^1_3)^2(E^1_3-\tau E^1_{23})} - \frac{(1+\delta)^2E^2_{33}}{(E^1_3)^2} + \frac{(1+\delta)E^2_{33}}{E^1_3(E^1_3-\tau E^1_{23})E^2_3}T'\right] < 0,$$

where

$$\Omega = 1 - \frac{(1+\delta)E^2_3E^1_{33}}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})} - \frac{(1+\delta)^2E^2_{33}}{(E^1_3)^2R^2_{33}} + \frac{(1+\delta)BE^2_{33}}{E^1_3E^2_3} + \frac{(1+\delta)E^2_{33}}{E^1_3R^2_{33}(E^1_3-\tau E^1_{23})E^2_3}T' > 0,$$ [11]

and R^2_{33} represents the decreasing shadow price of capital (or investment) in the second

[10] The term $C^1_Y = E^1_{23}/E^1_3$ is the pure income effect of the Slutsky equation in terms of the expenditure function in period one representing how Marshallian (uncompensated) demand responds to changes in real income in period one. Given any level of τ, $(P^1 + \tau)C^1_Y$ is the period-one marginal propensity to consume (MPC) on the non-numeraire good in the recipient country.

[11] For convenience, we simply treat Ω just as a positive constant from now on.

period. Since $R_{33}^2 < 0$, it follows that unambiguously $\Omega > 0$, $\partial I/\partial B > 0$ and $\partial r/\partial B < 0$. In particular, the negative partial effect of B on r corresponds to the Walrasian stable condition for the international borrowing market.

Totally differentiating (3) and using (6)-(9), we obtain the effects of a tariff change on intertemporal welfare as:

$$\begin{aligned}
\frac{dW}{d\tau} &= \frac{dU^1}{d\tau} + \frac{1}{1+\delta}\frac{dU^2}{d\tau} && (10)\\
&= \left(\frac{\partial U^1}{\partial \tau} + \frac{1}{1+\delta}\frac{\partial U^2}{\partial \tau}\right) && (11)\\
&\quad + \left(\frac{\partial U^1}{\partial I} + \frac{1}{1+\delta}\frac{\partial U^2}{\partial I}\right)\frac{\partial I}{\partial \tau} && (12)\\
&\quad + \frac{1}{1+\delta}\frac{\partial U^2}{\partial r}\frac{\partial r}{\partial \tau}. && (13)
\end{aligned}$$

In (10), there are three distinct effects of a tariff change on intertemporal welfare: (i) the intertemporal direct income effect, (ii) the intertemporal indirect income effect via investment, and (iii) the period-two indirect income effect via interest rate.

The intertermporal direct income effect is shown in (11) and consists of two conflicting terms. The first term in (11), the period-one direct income effect, is negative. This represents that a tariff increase causes deadweight loss in period one incurred by reducing import demands for the non-numeraire good and increasing its ineffcient dometic production. In contrast, the second term in (11), which is the period-two direct income effect, is positive because foreign aid negatively tied to the period-one real income compensates for the period-one welfare loss from the tariff increase.

The intertemporal indirect income effect via investment, shown in (12), is the sum of two conflicting effects. Two partial effects of investment on intertemporal welfare are of opposite sign because they are the marginal cost of investment in period one and the marginal benefit of investment in period two, respectively. The partial effect of a tariff on investment in period one is ambiguous. A tariff increase yields more tariff revenue, thereby

encouraging more investment in period one. On the contrary, the tariff increase can discourage investment in period one in two indirect ways: it causes welfare loss in period one; and this period-one welfare loss invites more aid in period two, thereby reducing investment.

In (13), the indirect income effect via interest rate exists only in period two and is also ambiguous. The partial effect of interest rate on the period-two income is obviously negative because an increase in interest rate worsens the terms-of-trade of international borrowing. But, the partial effect of a tariff on interest rate is unclear. An increase in tariff revenue decreases demand for international borrowing, thereby lowering interest rates. In contrast, a tariff increase raises interest rates in two indirect ways: it causes welfare loss in period one, thus stimulating demand for international borrowing; and larger inflows of aid in period two induce to borrow more in period one.

1.3.1 The case without International Borrowing ($B = 0$)

1.3.1.1 Exogenous Domestic Investment

We now analyze the case when there is no international borrowing ($B = 0$), that is, the recipient country cannot have access to international credit markets. When $B = 0$, the terms-of-trade of international borrowing does not affect intertemporal welfare. Hence, the period-two indirect income effect via interest rate, (13), disappears. Also, domestic investment is not affected by a tariff change because it is exogeously given. Therefore, the investment-induced indirect income effect, (12), also does not exist. Therefore, *ceteris paribus*, we obtain $dW/d\tau$ only as the same as the intertemporal direct income effect of a

tariff in (11):

$$\begin{aligned}\frac{dW}{d\tau} &= \frac{\partial U^1}{\partial \tau} + \frac{1}{1+\delta}\frac{\partial U^2}{\partial \tau} \\ &= \frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}} + \frac{1}{1+\delta}\frac{\tau(E^1_{22}-R^1_{22})T'}{(E^1_3-\tau E^1_{23})E^2_3} \\ &= \frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right). \end{aligned} \tag{14}$$

Also, the second-order derivative of W with respect to τ is obtained as:

$$\begin{aligned}\frac{d^2W}{d\tau^2} &= \frac{E^1_{22}-R^1_{22}}{E^1_3-\tau E^1_{23}}\left[1-\frac{\tau^2(E^1_{22}-R^1_{22})E^1_{33}}{(E^1_3-\tau E^1_{23})^2}\right]\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right) \\ &\quad + \left[\frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}\right]^2 \frac{1}{(1+\delta)E^2_3}\left[T'' - (1+\delta)^2 E^2_{33}\right]. \end{aligned} \tag{15}$$

In (14), the period-one direct income effect has a negative sign because of the deadweight loss from imposing a tariff in period one. In contrast, the positive period-two direct income effect indicates that foreign aid negatively related to the period-one real income improves the period-two real income. Therefore, the intertemporal direct income effect of a tariff depends upon which one of two conflicting effects dominates.

In (14), we define a negative term, η, as follows:

$$\eta = \frac{1}{1+\delta}\frac{\partial U^2/\partial \tau}{\partial U^1/\partial \tau} = \frac{1}{1+\delta}\frac{T'}{E^2_3} < 0, \tag{16}$$

where the absolute value of η represents the extent to which foreign aid in period two compensates for the period-one welfare loss from the impostion of a tariff. From now on, we make an assumption that the negative term, η, is greater than negative one. This assumption implies that the period-one welfare loss from a positive tariff is more than offset by foreign aid in period two. It makes sense because, as mentioned in the first footnote, foreign aid is not determined only by the needs of the recipients but also by the donors' political and strategic considerations. If foreign aid is disbursed only by the needs

of the recipients, the period-one welfare loss incurred by a positive tariff would be exactly offset by foreign aid given in period two at any τ, and thus η would be negative one. Technically speaking, if η is negative one, the first-order condition would be fulfilled at any τ and thus there would be no significant choice to be made regarding the optimal τ for the maximization of W. Thus, any positve tariff is neither beneficial nor detrimental to the recipient country. If η is less than negative one, W would be strictly increasing in τ at a incresing rate, and thus there is no finite maximum if the set of nonnegative real numbers is taken to be its domain. Thus, our assumption also enables us to properly rule out the case when η is equal to or less than negative one. This assumption is formally expressed as

$$-1 < \eta = \frac{1}{1+\delta}\frac{T^{'}}{E_3^2} < 0. \tag{17}$$

Assumption 1 simply implies that (14) always has a negative value because the negative effect in period one dominates. We add another assumption to the model. We assume that η becomes closer to zero as τ increases. This assumption reflects that aid is conditional upon trade liberalization in recipient countries. In other words, this assumption means that higher tariff rates make the absolute value of η lower because donor countries are more reluctant to provide aid to recipient coutries with higher tariffs. This assumption takes the following form:

$$\frac{\partial \eta}{\partial \tau} = \frac{1}{1+\delta}\frac{\tau(E_{22}^1 - R_{22}^1)}{(E_3^1 - \tau E_{23}^1)E_3^2}\left[T^{''} - \left(\frac{T^{'}}{E_3^2}\right)^2 E_{33}^2\right] > 0. \tag{18}$$

Under Assumption 1 and 2, the first-order condition of W for relative extremum in (14) is satisfied at $\tau = 0$, and $d^2W/d\tau^2 < 0$ in (15)[12], meaning that the function W is strictly concave. Therefore, zero tariff is optimal and the stationary value of W at $\tau = 0$ is the unique global maximum. In this case, foreign aid does not discourage trade

[12]From Assumption 1, we obtain $\left(\frac{T^{'}}{E_3^2}\right)^2 < (1+\delta)^2$. When we substitute $(1+\delta)^2$ for $\left(\frac{T^{'}}{E_3^2}\right)^2$, we obtain the condition $T^{''} - (1+\delta)^2 E_{33}^2 < 0$. This condition guarantees that $d^2W/d\tau^2 < 0$.

liberalization. A positive tariff has only a negative effect on the recipient's intertemporal welfare, because the marginal cost of a tariff increase from zero is greater than the marginal benefit.

1.3.1.2 Endogenous Domestic Investment

We now turn to the case when domestic investment is endogenously chosen. In this case, the effect of a tariff change on intertemporal welfare is the sum of the intertemporal direct income effect and the investment-induced intertemporal indirect income effect. *Ceteris paribus*, the term $dW/d\tau$ can be simplified and reorganized as:

$$\begin{aligned}\Omega\frac{dW}{d\tau} &= \Omega\left[\left(\frac{\partial U^1}{\partial\tau}+\frac{1}{1+\delta}\frac{\partial U^2}{\partial\tau}\right)+\left(\frac{\partial U^1}{\partial I}+\frac{1}{1+\delta}\frac{\partial U^2}{\partial I}\right)\frac{\partial I}{\partial\tau}\right]\\ &= \tau\left[\frac{E_{22}^1-R_{22}^1}{E_3^1-\tau E_{23}^1}\left(1+\frac{1}{1+\delta}\frac{T'}{E_3^2}\right)+\Lambda\right]+\Pi T', \end{aligned} \tag{19}$$

where

$$\Lambda=-\frac{(1+\delta)E_3^2E_{33}^1(E_{22}^1-R_{22}^1)}{(E_3^1)^3R_{33}^2(E_3^1-\tau E_{23}^1)}+\frac{(1+\delta)E_3^2E_{23}^1E_{32}^1}{(E_3^1)^3R_{33}^2(E_3^1-\tau E_{23}^1)}-\frac{(1+\delta)^2E_{33}^2(E_{22}^1-R_{22}^1)}{(E_3^1)^2R_{33}^2(E_3^1-\tau E_{23}^1)}<0 \text{ and}$$

$$\Pi=\frac{E_{32}^1}{(E_3^1)^2R_{33}^2(E_3^1-\tau E_{23}^1)}<0.^{13}$$

Also, the second derivative of W with respect to τ is obtained as:

$$\begin{aligned}\Omega\frac{d^2W}{d\tau^2} &= \left[\frac{E_{22}^1-R_{22}^1}{E_3^1-\tau E_{23}^1}\left(1+\frac{1}{1+\delta}\frac{T'}{E_3^2}\right)+\Lambda\right]\\ &+\frac{\tau(E_{22}^1-R_{22}^1)}{E_3^1-\tau E_{23}^1}\left[1-\frac{\tau^2(E_{22}^1-R_{22}^1)E_{33}^1}{(E_3^1-\tau E_{23}^1)^2}\right]\left(1+\frac{1}{1+\delta}\frac{T'}{E_3^2}\right)\\ &+\tau\left[\frac{\tau(E_{22}^1-R_{22}^1)}{E_3^1-\tau E_{23}^1}\right]^2\frac{1}{(1+\delta)E_3^2}\left[T''-(1+\delta)^2E_{33}^2\right]\\ &+\Pi\frac{\tau(E_{22}^1-R_{22}^1)}{E_3^1-\tau E_{23}^1}T''. \end{aligned} \tag{20}$$

[13] For convenience, we treat Λ and Π as constants.

On the right hand side of (20), only the last term is positive while the other terms are negative. Assuming that the last term is negligible, the second order condition is satisfied, which gives us a sufficient condition for the unique global maximum of W.

Setting (19) equal to zero as the first-order condition, we obtain a positive optimal tariff

$$\tau^* = \frac{-\Pi T'}{\frac{E_{22}^1 - R_{22}^1}{E_3^1 - \tau E_{23}^1}\left(1 + \frac{1}{1+\delta}\frac{T'}{E_3^2}\right) + \Lambda} > 0. \tag{21}$$

The stationary value of W at the postive optimal tariff τ^* is the global maximum of W. In addition, when (19) and (20) are evaluated at $\tau = 0$, the first and second derivatives of W with respect to τ are obtained as:

$$\Omega \frac{dW}{d\tau}|_{\tau=0} = \Pi T' > 0 \text{ and} \tag{22}$$

$$\Omega \frac{d^2W}{d\tau^2}|_{\tau=0} = \frac{E_{22}^1 - R_{22}^1}{E_3^1}\left(1 + \frac{1}{1+\delta}\frac{T'}{E_3^2}\right) + \Lambda < 0. \tag{23}$$

In (22) and (23), W is increasing in τ at a decreasing rate at $\tau = 0$, which also shows that optimal tariff is not zero but positive.

Interestingly, if $T' = 0$ in (19)–(22), then optimal tariff is zero and it guarantees the unique global maximum of W. This obviously suggests that, when domestic investment is endogenous, the positive optimal tariff is induced by foreign aid which is negatively tied to period-one income. In this case, therefore, foreign aid does discourage trade liberalization. In (22) and (23), a tariff increase from zero leads to more investment in period one, thereby generating the period-two welfare gain. Of course, the cost of a tariff increase is the welfare loss in period one from more investment and less consumption. But the tying of foreign aid to the period-one income partly compensates for the period-one welfare loss. At a zero tariff, therefore, the marginal benefit of a tariff increase is greater than its marginal cost. The recipient country increases its tariff up until the marginal cost of a tariff increase becomes equal to the marginal benefit. Therefore, the recipient country has an incentive to

impose a positive tariff to increase investment in period one. If foreign aid is not negatively related to the period-one real income, that is, $T' = 0$, then the period-one welfare loss from a tariff increase from zero would always be more than offset by the period-two welfare gain, and thus the recipient would not be willing to raise its tariff from zero.

1.3.2 The case with international borrowing ($B > 0$)

1.3.2.1 Exogenous Domestic Investment

We consider the case when the recipient country can borrow from the international credit markets ($B > 0$). Intertemporal welfare is affected not only directly by a tariff change but also indirectly by a change in the terms-of-trade of international borrowing. When domestic investment is exogenous, we obtain $dW/d\tau$ as:

$$\begin{aligned}
\Omega\frac{dW}{d\tau} &= \Omega\left[\left(\frac{\partial U^1}{\partial \tau}+\frac{1}{1+\delta}\frac{\partial U^2}{\partial \tau}\right)+\frac{\partial U^2}{\partial r}\frac{\partial r}{\partial \tau}\right] \\
&= \tau\left[\frac{(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}-\frac{(1+\delta)E^2_3E^1_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})^2}\right]\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right) \\
&\quad -\frac{\tau(1+\delta)^2E^2_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})}+\frac{\tau^2(1+\delta)E^1_{23}E^2_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2R^2_{33}(E^1_3-\tau E^1_{23})^2E^2_3}T' \\
&\quad +\frac{\tau E^2_{33}(E^1_{22}-R^1_{22})}{E^1_3R^2_{33}(E^1_3-\tau E^1_{23})^2(E^2_3)^2}T'T' \\
&\quad +\left[\frac{\tau E^1_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2(E^1_3-\tau E^1_{23})}+\frac{E^1_{32}}{(E^1_3)^2}+\frac{\tau(1+\delta)E^2_{33}(E^1_{22}-R^1_{22})}{E^1_3(E^1_3-\tau E^1_{23})E^2_3}\right]B. \qquad (24)
\end{aligned}$$

Unfortunately, (24) is not easy to be solved for the optimal τ since it is a quadratic form in the variable τ. But we can basically check out if the optimal tariff is zero or not by evaluating the first and second derivatives of W with respect to τ at $\tau = 0$. Using (24), we obtain the first and second order condition evaluated at $\tau = 0$ as:

$$\Omega\frac{dW}{d\tau}|_{\tau=0}=\frac{E^1_{32}B}{(E^1_3)^2}>0 \text{ and} \qquad (25)$$

$$\begin{aligned}\Omega\frac{d^2W}{d\tau^2}|_{\tau=0} &= \left[\frac{E_{22}^1-R_{22}^1}{E_3^1}-\frac{(1+\delta)E_3^2E_{33}^1(E_{22}^1-R_{22}^1)}{(E_3^1)^4R_{33}^2}\right]\left(1+\frac{1}{1+\delta}\frac{T'}{E_3^2}\right)\\ &\quad -\frac{(1+\delta)^2E_{33}^2(E_{22}^1-R_{22}^1)}{(E_3^1)^3R_{33}^2}\left[1-\left(\frac{1}{1+\delta}\frac{T'}{E_3^2}\right)^2\right]\\ &\quad +\left[\frac{E_{33}^1(E_{22}^1-R_{22}^1)}{(E_3^1)^3}-\frac{2(E_{32}^1)^2}{(E_3^1)^3}+\frac{(1+\delta)E_{33}^2(E_{22}^1-R_{22}^1)}{(E_3^1)^2E_3^2}\right]B\\ &< 0 \qquad (26)\end{aligned}$$

In (25) and (26), the first-order condition for relative extremum is not satisfied at $\tau = 0$, and W is increasing in τ at a decreasing rate at $\tau = 0$. Therefore, it is revealed that optimal tariff is not zero but positive. We can also know that, when $B = 0$, zero tariff guarantees the unique global maximum of W. Hence, the positive optimal tariff in this case is not caused by foreign aid negatively tied to period-one real income but caused by the terms-of-trade of international borrowing, that is, interest rate. The imposition of a positive tariff causes the period-one welfare loss from less consumption but yields tariff revenue in period one. The tariff revenue decreases the demand for international borrowing, thereby improving the term-of-trade of international borrowing in period two. Obviously, this improvement reduces the burden of reinbursement in period two, thereby making the recipient country better off in period two. At a zero tariff, the marginal benefit of a tariff increase from zero is greater than its marginal cost. The recipient country increases it tariff up until the marginal cost of a tariff becomes equal to the marginal benefit. In this case, foreign aid does not discourage trade liberalization.

1.3.2.2 Endogenous Domestic Investment

In the case when, in addition, domestic investment is endogenous, the expression

$dW/d\tau$ is obtained as the sum of all three distinct effects:

$$\begin{aligned}
\frac{dW}{d\tau} &= \frac{dU^1}{d\tau} + \frac{1}{1+\delta}\frac{dU^2}{d\tau} \\
&= \left(\frac{\partial U^1}{\partial \tau} + \frac{1}{1+\delta}\frac{\partial U^2}{\partial \tau}\right) + \left[\left(\frac{\partial U^1}{\partial I} + \frac{1}{1+\delta}\frac{\partial U^2}{\partial I}\right)\frac{\partial I}{\partial \tau}\right] + \frac{1}{1+\delta}\frac{\partial U^2}{\partial r}\frac{\partial r}{\partial \tau} \\
&= \tau\left[\frac{E^1_{22}-R^1_{22}}{E^1_3-\tau E^1_{23}}\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right)+\Lambda+\Phi B\right]+\Pi T'+\Psi B, \qquad (27)
\end{aligned}$$

where

$$\Phi = \frac{E^1_{33}(E^1_{22}-R^1_{22})}{(E^1_3)^2(E^1_3-\tau E^1_{23})} + \frac{(1+\delta)E^2_{33}(E^1_{22}-R^1_{22})}{E^1_3(E^1_3-\tau E^1_{23})E^2_3} < 0 \text{ and}$$

$$\Psi = \frac{E^1_{32}}{(E^1_3)^2} > 0.^{14}$$

Also, the second derivative of W with respect to τ is obtained as:

$$\begin{aligned}
\Omega\frac{d^2W}{d\tau^2} &= \left[\frac{E^1_{22}-R^1_{22}}{E^1_3-\tau E^1_{23}}\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right)+\Lambda\right] \\
&\quad +\frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}\left[1-\frac{\tau^2(E^1_{22}-R^1_{22})E^1_{33}}{(E^1_3-\tau E^1_{23})^2}\right]\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right) \\
&\quad +\tau\left[\frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}\right]^2\frac{1}{(1+\delta)E^2_3}\left[T''-(1+\delta)^2E^2_{33}\right] \\
&\quad +\Pi\frac{\tau(E^1_{22}-R^1_{22})}{E^1_3-\tau E^1_{23}}T'' \\
&< 0 \qquad (28)
\end{aligned}$$

In (28), the function W is strictly concave, which means that the stationary value of W at a critical value τ is the unique global maximum.

Setting (27) equal to zero, the positive optimal tariff is obtained as:

$$\tau^* = \frac{-(\Pi T' + \Psi B)}{\frac{E^1_{22}-R^1_{22}}{E^1_3-\tau E^1_{23}}\left(1+\frac{1}{1+\delta}\frac{T'}{E^2_3}\right)+\Lambda+\Phi B} > 0. \qquad (29)$$

This positive optimal tariff guarantees the unique global maximum of W. Also evaluating

[14] For convenience, we treat Φ and Ψ as negative constants.

(27) and (28) at $\tau = 0$, we obtain

$$\Omega \frac{dW}{d\tau}|_{\tau=0} = \Pi T' + \Psi B > 0 \text{ and} \tag{30}$$

$$\Omega \frac{d^2W}{d\tau^2}|_{\tau=0} = \frac{E_{22}^1 - R_{22}^1}{E_3^1 - \tau E_{23}^1} \left(1 + \frac{1}{1+\delta} \frac{T'}{E_3^2}\right) + \Lambda < 0. \tag{31}$$

In (30) and (31), W is increasing in τ at a decreasing rate at $\tau = 0$, which makes sure that zero tariff is no longer optimal. The positive optimal tariff is induced not only by the endogenous terms-of-trade of international borrowing but also by foreign aid negatively tied to the period-one income.

In this case, foreign aid discourages trade liberalization. There are two beneficial effects of a tariff increase from zero on the intertemporal welfare: a positive tariff allows the recipient country to invest more in period one, thereby increasing the period-two consumption; and it also improves the terms-of-trade of international borrowing, thereby making the recipient country repay its external debt less in period two. The cost of a tariff increase from zero is the period-one real income loss from less consumption. But this cost is in part offset by foreign aid given in period two. At a zero tariff, the marginal benefit of a tariff increase is greater than its marginal cost. A positive tariff continues to increase until the marginal cost becomes equal to the marginal benefit. In this case, if foreign aid is not tied to the period-one real income, that is, $T' = 0$, optimal tariff is still positive because of the beneficial effect of a positive tariff on the terms-of-trade of international borrowing. But the tying of foreign aid to the period-one income increases its positive optimal tariff further.

1.4 CONCLUSION

This study theoretically examines if foreign aid discourages aid-recipient countries from pursuing trade liberalization policies, when more aid is disbursed to poorer countries, using a two-period general equilibrium trade-theoretic model of a small open economy. In

the traditional static model of international transfer of income, the commodity terms-of-trade is the main mechanism of an exogenous change for affecting welfare. But our intertemporal model has the virtue of allowing us to consider endogenous investment as another channel through which affects intertemporal welfare, which is closely linked to our key findings. Also, we consider international borrowing in our intertemporal settting, even though it has been usually ignored in the traditional static model. This enables us to find that endogenous term-of-trade of international borrowing in a small open economy, that is, interest rate, also plays an important role in intertemporal welfare in aid-recipient countries.

We investigate whether the tying of foreign aid in period two negatively to the period-one real income influences the imposition of a positive optimal tariff in the recipient country. Two variants of the model are considered depending upon whether there is international borrowing or not. We find that optimal tariff is zero only when the recipient country cannot borrow from the world and domestic investment is exogenously given. In this case, foreign aid does not discourage trade liberalization. In the other cases, in contrast, optimal tariff is positive. When there is international borrowing but investment is exogenous, the reason for the positive optimal tariff is not the tying of aid negatively to the period-one real income but the beneficial effect of a positive tariff on the improvement in the terms-of-trade of international borrowing. Most importantly, when domestic investment is endogenous, foreign aid negatively linked to the period-one real income induces a positive optimal tariff. In this case, the reason for the positive optimal tariff is that foreign aid in period two partly compensates for the period-one real income loss and this enables the recipient country to invest more in period one. We also find that aid-induced positive optimal tariff gets higher as the recipient gets poorer in period one.

Intuitively, when domestic investment is endogenous, that is, it is market-determined, foreign aid discourages trade liberalization in aid-recipient countries. As the benefit of not pursuing trade liberalization, foreign aid enables the recipients to impose its optimal

positive taiff so as to invest more in period one.

CHAPTER 2

HEALTH POLICIES AND THE OPTIMAL ALLOCATION OF HEALTH AID: A THEORY

2.1 INTRODUCTION

Let hunger be ranked first, because if you are hungry, you cannot work! No, health is number one, because if you are ill, you cannot work! – Discussion Group, Musanya Village, Zambia (WHO & World Bank, 2002)

There is little doubt about the magnitude of good health in low income countries. Good health is a determinant of economic growth and a component of the well-being of the population. Illness can reduce household savings, lower learning ability, reduce productivity and lead to a low quality of life, thereby creating or perpetuating poverty. Thus, ill health in low income countries does not only represents the humanitarian tragedy but also puts a drag on economic growth. The Millennium Summit of the United Nations in 2000 established total eight international development goals, which is also called the Millennium Development Goals (MDGs), and five out of the eight goals are the health-related goals.[15] Children are more vulnerable than adults to illness. Poor families, if their children get sick, often lose their precious income and assets to pay for their sick children's medical costs, thereby impoverishing them even further and exacerbating the vicious circle of poverty and ill health. Therefore, Millennium Development Goal 4 (MDG 4) calls for reducing the under-five mortality rate by two thirds between 1990 and 2015. The world has made substantial progress, thereby reducing the under-five mortality rate 47 percent from 1990 to 2012. But still, in 2012, approximately 6.6 million children worldwide

[15] The representative health indicators for the health-related MDGs are as follows: underweight prevalence of under-5 children (MDG 1: to eradicate extreme poverty and hunger), under-5 mortality rate (MDG 4: to reduce child mortality rates), maternal mortality ratio (MDG 5: to improve maternal health), AIDS prevalence, use of insecticide-treated bed nets (MDG 6: to combat HIV/AIDS, malaria, and other diseases), use of improved water source, and use of improved sanitation facilities (MDG 7: to ensure environmental sustainability)

– 18,000 children per day – died before reaching their fifth birthday, mostly from preventable causes and treatable diseases (UN-IGME, 2013).

Nevertheless, the stark imbalances of health care provision between rich and poor countries have been reported. As of 2009, low-income developing countries have 92% of the global disease burden, whereas they account for only 16% of global health spending (Moon and Omole, 2013). Therefore, the international community has paid attention to health challenges in developing countries. The past decade has witnessed extraordinary growth of international financing in global health and a compelling move of foreign aid from financing physical infrastructure to improving public health.[16] The logic behind this move is that if people are healthier, then they will work better and longer, thus achieving sustainable economic growth. In reality, development assistance for health has increased from US $5.7 billion in 1990 to a record-high of US $28.2 billion in 2010 (IHME, 2012). Approximately 10% of Africa's health care spending is financed by foreign aid (IFC, 2007). As a result, it is widely accepted that foreign aid for health works relatively better than for other sectors.[17] For example, Levine's surveys (2004, 2007) on successful cases in public health in the developing world show how health aid made a contribution to their success. Keeping pace with health aid's tangible success, foreign aid has undergone a considerable change both in the quantities given and in the way it is spent.

The remarkable increases in health aid over the past years have made examining the effectiveness of health aid all the more important. It is the optimal allocation of health aid that ensure health aid effectiveness in the aggregate. There are two different perspective on

[16] Traditionally, foreign aid has been justfied by filling the gap between the amount of investment necessary to achieve a certain level of economic growth and the available domestic saving (Chenery and Strout, 1966). However, Empirical evidence has not favored the link from aid to growth via investment. Boone (1996) claims that aid has been used to increase consumption rather than investment. Easterly (2001) tests both whether foreign aid has increased investment and whether investment has increased economic growth. But he finds no evidence that foreign aid has financed the gap effectively. Actually, the voluminous literature has attempted to examine the aid-growth relationship using a variety of methods and frameworks. Yet, it does not provide robust evidence of either positive or negative relationship between them.

[17] The empirical literature, however, have not reached a robust consensus on the positive effect of health aid on public health outcomes. For the details, see Williamson (2008), Mishra and Newhouse (2009) and Wilson (2011).

the optimal allocation of health aid. The first one is a macro perspective on whether relatively more health aid goes to countries with lower levels of public health. On the other hand, the second one is a micro perspective on whether health aid in a recipient country is allocated in a way that helps the poor effectively. This study provides a theoretical explanation for the latter one. Our theoretical model takes heterogeneity within health aid into account. In other words, we argue that various types of health aid are given to a recipient country on their own purposes and thus their effectiveness varies considerably in quality. For example, some of them are used to raise the quality of health care (e.g. health aid for basic health infrastructure), while others are given to provide preventive care (e.g. health aid for basic nutrition) or to reduce the cost of health care (e.g. heath aid for supply of basic health care). Among them, there are types of health aid that work better than the others.

In this study, we develop a microeconomics model addressing two issues. The first issue is the effectiveness of health policies in the context of the poor household's utility maximization. What will happen to poor households if their children get sick? Poverty denies sick children access to reliable health services and affordable medicines. The costs of health care are too high for poor parents to take their sick children to health care providers. Also, not all sick children taken to hospitals are guaranteed to survive, because of the bad quality of medical services. One of the feasible ways to improve children's health is to provide them with relevant preventive care. It can be effective in preventing children from getting severe illness. But it does not guarantee severely sick children to survive. In the model, therefore, poor households have no choice but to choose the optimal number of their sick children taken to hospitals to maximize their lifetime utility, given their intertemporal budget constraints. There are three policy options that policymakers use to improve public health: raising the quality of health care, providing preventive care and reducing the costs of health care. We show how these three policy options influence the optimal number of sick children who are medically treated.

The second issue relates to the optimal allocation of health aid supporting the poor household's utility maximization in a poor country. The country's health authority maximizes the representive household's lifetime utility by allocating health aid for the three policy options. We find that allocating more health aid for reducing the cost of health care is the most effective way to maximize each poor household's lifetime utility. The main idea of this study comes from Lahiri and Self (2008) where they examine gender bias problem in health care provision. For this study, we use a similar theoretical framework as Lahiri and Self's (2008) but focus on the optimal allocation of health aid.

The organization of this chapter is as follows. In section 2, we present a theoretical model of the effectiveness of public health policies. Section 2 is divided into two parts. The first part deals with the each poor household's utility maximization to choose the optimal number of sick children who are medically treated. In the other part, we theoretically examine how three policy options influence the optimal number of sick children taken to hospitals. In section 3, we theoretically find the health authority's optimal allocation of health aid in order to maximize each household's lifetime utility. Section 4 concludes and draws policy implications.

2.2 THE EFFECTIVENESS OF HEALTH POLICIES

2.2.1 The Household's Utility Maximization

People live for only two periods in a poor country. In period one, when people are children, some of them become sick. Each household has N childrens. The proportion of the sick children, i, is known to the families. Thus, total Ni children are sick in each household. Part of the sick children is severe. But there is uncertainty of how many sick children are severe. Assume that the number of children who are severely sick is a random variable X and it follows the binomial distribution with the probability that a child is

severely sick, p, denoted by

$$\Pr(X = x) = \binom{Ni}{x} p^x (1-p)^{Ni-x},\ p \in (0,1), \tag{32}$$

where p is an index of the provision of preventive care. Thus, the more provides the preventive care, the lower becomes the likelihood of severely sickness, p. Severely sick children die unless they don't receive health care treatment.

A household takes q proportion of sick children randomly to health care providers. Thus, qNi children receive the medical treatment. Among qNi children who receive the medical treatment, severely sick qX children are in need of the medical treatment to survive, whereas $q(Ni - X)$ children don't have to receive the medical treatment to survive since they are not severely ill. The medical treatment from health care providers costs the family the fixed positive amount of c per child. A proportion of δ among qX children do not survive although they receive the medical treatment. In this context, δ represents the quality of health care. The higher becomes the quality of health care, the lower goes δ. Severely sick children who don't receive the medical treatment, $(1-q)X$, also die. Thus, the number of children who die, D, is

$$D = \delta q X + (1-q)X = (\delta q + 1 - q)X,\ \delta q + 1 - q \in (\delta, 1) \tag{33}$$

where $\delta q + 1 - q$ is the probability that severely sick children die. We also know that

$$\frac{\partial D}{\partial q} = -(1-\delta)X < 0 \tag{34}$$

for $\delta \in (0,1)$. That is, the more sick children taken to hospitals, the less sick children who die of their disease.

The children who do not die become adults and earn wage w in the second period.

Thus, a household's net total income in terms of the period-two price, y, is

$$\begin{aligned} y &= w(N-D) - (1+r)cqNi \\ &= wN - w(\delta q + 1 - q)X - (1+r)cqNi \end{aligned} \tag{35}$$

where r is a discount factor. The net utility of the household, u, is

$$u = U(y - \phi D), u' > 0, u'' < 0, \tag{36}$$

where ϕ is a constant parameter representing marginal disutility from bereavement and thus ϕD denotes disutility from bereavement of childrens' deaths. We use the concept of certainty equivalence to treat uncertainty. According to the Markowitz's (1952) model of mean-variance analysis of portfolio selection, the certainty equivalence of the household's utility, u^c, is written as

$$u^c = E(y - \phi D) - \gamma var(y - \phi D), \tag{37}$$

where γ is the measure of relative risk preference. We assume that the economic agents are risk averse, which means that $\gamma > 0$.

Again, the number of severely sick children, X, is assumed to follow a binomial distribution. Thus, the expected value and variance of X can be written as

$$E(X) = Nip, \tag{38}$$

$$Var(X) = Nip(1-p). \tag{39}$$

From (33) and (35),

$$y - \phi D = wN - (\delta q + 1 - q)(w + \phi)X - (1+r)cqNi. \tag{40}$$

Therefore, using (38) and (39), we get the expected value and variance of $y - \phi D$ as:

$$E(y - \phi D) = wN - (\delta q + 1 - q)(w + \phi)Nip - (1 + r)cqNi, \tag{41}$$

$$Var(y - \phi D) = (\delta q + 1 - q)^2(w + \phi)^2 Nip(1 - p). \tag{42}$$

Substituting (41) and (42) into (37), we also get

$$\begin{aligned} u^c &= E(y - \phi D) - \gamma var(y - \phi D) \\ &= wN - (\delta q + 1 - q)(w + \phi)Nip - (1 + r)cqNi \\ &\quad -\gamma(\delta q + 1 - q)^2(w + \phi)^2 Nip(1 - p). \end{aligned} \tag{43}$$

Each household chooses the optimal q by maximizing u^c with respect to q. Taking a partial derivative of (43) with respect to q, we obtain the first order condition as

$$\frac{\partial u^c}{\partial q} = (1 - \delta)(w + \phi)Nip - (1 + r)cNi + 2\gamma(1 - \delta)(\delta q + 1 - q)(w + \phi)^2 Nip(1 - p) = 0, \tag{44}$$

which can be simplified as

$$(1 - \delta)(w + \phi)p + 2\gamma(1 - \delta)(\delta q + 1 - q)(w + \phi)^2 p(1 - p) = (1 + r)c. \tag{45}$$

Also, the second order condition is satisfied as in the following:

$$\frac{\partial^2 u^c}{\partial q^2} = -2\gamma(1 - \delta)^2(w + \phi)^2 Nip(1 - p) < 0. \tag{46}$$

This gives us a sufficient condition for a global maximum of the certainty equivalence of the household's utility at the optimal q. The left hand side of (45) represents the marginal benefit of taking an additional sick child to health care. On the left hand side, the first

positive term comes from the expected value of $E(y - \phi D)$, representing that, if an additional sick child is taken to hospital and she survives, the household would get more wage and feel less pain of bereavement. The second positive term originates in disutility from variance, $-\gamma Var(y - \phi D)$, thereby meaning that taking another sick children to hospitals lowers the disutility from variance. It is also worth noticing that the marginal benefit varies with δ and p which are interdependent on each other. The marginal cost, shown on the right hand side of (45), is the opportunity cost of taking an additional sick child to health care in terms of the period-two price. Given r, the marginal cost depends only on c in the model.

From (45), the closed-form solution for the optimal proportion of sick chilren receiving health care, q^*, is expressed as a function of exogenous variables including three health care policy variables δ, p and c:

$$q^* = q(\delta, p, c) = \frac{(1-\delta)(w+\phi)p + 2\gamma(1-\delta)(w+\phi)^2 p(1-p) - (1+r)c}{\Lambda}, \tag{47}$$

where

$$\Lambda = 2\gamma(1-\delta)^2(w+\phi)^2 p(1-p) > 0, \tag{48}$$

and, for convenience' sake, is reasonably assumed to be a constant.

2.2.2 The Comparative Statics

In this model, there are three different policy options for health authorities to improve their public health: raising the quality of health care (lowering δ), providing preventive care (lowering p) and reducing the cost of health care (lowering c). The equation (34) obviously shows that the number of children who die decreases as the optimal number of sick children taken to hospitals increases. Policymakers can use three health care policy options to encourage poor parents to take more sick children to hospitals. Therefore, we now pay attention to the effects of three policy options, δ, p and c, on the optimal number

of sick children who receive medical treatments, q^*. Totally differentiating (47), we obtain

$$dq^* = \frac{\partial q^*}{\partial \delta} d\delta + \frac{\partial q^*}{\partial p} dp + \frac{\partial q^*}{\partial c} dc, \tag{49}$$

where

$$\frac{\partial q^*}{\partial \delta} = \frac{-(w+\phi)p - 2\gamma(w+\phi)^2 p(1-p)}{\Lambda} < 0, \tag{50}$$

$$\frac{\partial q^*}{\partial p} = \frac{(1-\delta)(w+\phi) + 2\gamma(1-\delta)(w+\phi)^2 - 4\gamma(1-\delta)(w+\phi)^2 p}{\Lambda} \text{ and} \tag{51}$$

$$\frac{\partial q^*}{\partial c} = -\frac{1+r}{\Lambda} < 0. \tag{52}$$

2.2.2.1 Improving the quality of health care

Based upon (50), we can examine the effect of a change in δ on q^*. In (50), *ceteris paribus*, q^* is basically a decreasing linear function of δ. In other words, as the quality of health care improves, more sick children are taken to hospitals. As δ decreases, there is greater likelihood that sick children who are medically treated will survive, and thus $E(y - \phi D)$ increases and $Var(y - \phi D)$ decreases. From (47), we derive

$$\lim_{\delta \to 0} q^* = q^*_{\max}(\delta) = \frac{(w+\phi)p + 2\gamma(w+\phi)^2 p(1-p) - (1+r)c}{\Lambda} \text{ and} \tag{53}$$

$$\lim_{\delta \to 1} q^* = q^*_{\min}(\delta) = -\frac{(1+r)c}{\Lambda} < 0. \tag{54}$$

When δ is sufficiently close to zero, q^* can be made as close to a maximum $q^*_{\max}(\delta)$. In contrast, as δ is closer to one, q^* theoretically approaches its negative minimum value, $q^*_{\min}(\delta)$. Also, q^* is equal to zero at a value $\widehat{\delta}$, which is definitely less than one from (54) and is called the threshold level of δ, where

$$\widehat{\delta} = 1 + \frac{(w+\phi)p - (1+r)c}{2\gamma(w+\phi)^2 p(1-p)} < 1. \tag{55}$$

For $\delta \in [\widehat{\delta}, 1)$, that is, until the quality of health care improves over the threshold level, $\widehat{\delta}$, no sick child is taken to hospitals, because the cost of taking a sick child to health care providers exceeds its benefit. As δ decreases from $\widehat{\delta}$ to zero, q^* turns to be positive and continues to increase at the same rate.

However, the effect of a change in δ on q^* varies with p. In other words, the slope of q^* with respect to δ changes according to p. We know that (50) is a function of p and thus

$$\frac{\partial}{\partial p}\left(\frac{\partial q^*}{\partial \delta}\right) = \frac{-(w+\phi) - 2\gamma(w+\phi)^2 + 4\gamma(w+\phi)^2 p}{\Lambda}, \tag{56}$$

where the beneficial effect of an improvement in δ on q^* achieves the maximum level at a critical point

$$\widetilde{p} = \frac{1}{2} + \frac{1}{4\gamma(w+\phi)}, \ \frac{1}{2} < \widetilde{p} < 1. \tag{57}$$

As p diverges from $\widetilde{p}$, the beneficial effect of an improvement in δ on q^* dies away. Obviously, an improvement in δ is the most beneficial to poor households when they take as many sick children as possible to hospital. If $p = 0$, there would be no marginal benefit of taking a sick child to hospital, because her illness would obviously not be severe and thus she would definitely survive without going to hospital. If $p = 1$, parents would know that she is, for sure, severely sick. Thus, there would be no marginal benefit coming from less variance. Intuitively, we find that extremely good or bad provision of preventive care undermines the beneficial effect of an improvement in the quality of health care.

2.2.2.2 Providing more preventive care

In (51), we can see how a change in p affects q^*. The function q^* is a quadratic function of p. The second derivative of q^* with respect to p is obtained as

$$\frac{\partial^2 q^*}{\partial p^2} = \frac{-4\gamma(1-\delta)(w+\phi)^2}{\Lambda} < 0, \tag{58}$$

which gives us a sufficient condition for a global maximum, $q^*_{\max}(p)$. From (47), we get

$$\lim_{p\to 0} q^* = q^*_{\min}(p) = -\frac{(1+r)c}{\Lambda} < 0 \text{ and} \tag{59}$$

$$\lim_{p\to 1} q^* = \frac{(1-\delta)(w+\phi)-(1+r)c}{\Lambda}. \tag{60}$$

Also, q^* attains its global maximum,

$$q^*_{\max}(p) = \frac{(1-\delta)\left[2(w+\phi)+1\right]^2 - 8\gamma(1+r)c}{\Lambda}, \tag{61}$$

at a critical point $\widetilde{p}$ in (57). The function q^* is an inverse U-shaped curve in p. If we assume that $q^*_{\max}(p)$ is positive, then $\widetilde{p}$ is located between $\widehat{p}_L$ and $\widehat{p}_H$, where $0 < \widehat{p}_L < \widehat{p}_H < 1$, making q^* equal to zero. Intuitively, when p is very low (less than $\widehat{p}_L$) or high (greater than $\widehat{p}_H$), that is, the probability that a child is severely sick is very low or high, poor families don't take any sick child to health care providers because, in both cases, the cost of taking a sick child to hospital is greater than its benefit. When p is very low and close to zero, poor parents believe that their sick children will survive because their illness is not expected to be severe. In this case, there is no reason for them to see a doctor. Also, when p is very high and close to one, poor parents are confident that their sick children are highly likely to be severely sick and thus they are highly likely to die. In both cases, given the fixed marginal cost, the marginal benefit coming from less variance disappears. Therefore, poor parents are likely to choose to save their money rather than to pay for hospital bills.

In (51), the effect of a change in p on q^* varies with δ. From (20), we know that

$$\frac{\partial}{\partial\delta}\left(\frac{\partial q^*}{\partial p}\right) = \frac{-(w+\phi) - 2\gamma(w+\phi)^2 + 4\gamma(w+\phi)^2 p}{\Lambda}, \tag{62}$$

which is the same as (56) and is equal to zero at $\widetilde{p}$ such as in (57). In this case, for example, as δ decreases, $\widehat{p}_L$ and $\widehat{p}_H$ converge towards $\widetilde{p}$. This can be simply interpreted as that, as the quality of health care improves, the range of p where it can be effective in q^*

becomes narrower.

2.2.2.3 Reducing the cost of health care

The effect of a change in c on q^* is shown in (52). The function q^* is a linear function of c with a negative slope, thereby meaning that, as the cost of health care is reduced, more sick children receive medical treatments. As mentioned above, the effects of δ and p on q^* are interdependent on each other. Unlike this, the effect of c on q^* does not depend on δ or p. The optimal q^* is equal to zero when c is equal to its threshold level, $\widehat{c}$, where

$$\widehat{c} = \frac{(1-\delta)(w+\phi)p + 2\gamma(1-\delta)(w+\phi)^2 p(1-p)}{1+r} > 0. \tag{63}$$

For any $c > \widehat{c}$, poor parents don't pay for hospitals because the marginal cost of going to hospital exceeds the marginal benefit. As c is reduced from $\widehat{c}$ to zero, q^* increases at the same rate, which means that more sick children have a chance to see a doctor. When c approaches zero, q^* can be made as close to as a maximum level, $q^*_{\max}(c)$ as in the following:

$$\lim_{c\to 0} q^* = q^*_{\max}(c) = \frac{(1-\delta)(w+\phi)p + 2\gamma(1-\delta)(w+\phi)^2 p(1-p)}{\Lambda} > 0 \tag{64}$$

for p and $c \in (0,1)$.

2.3 THE OPTIMAL ALLOCATION OF HEALTH AID

2.3.1 The Health Authority's Utility Maximization

From the previous section, we know that three health policies improves public health in a way that increases the optimal number of sick children taken to hospital. We now suppose that a poor country receives health aid, T, from the world. The country's health authority allocates the funding for public health in a way that helps each household to maximize its lifetime utility by choosing the optimal number of sick children taken to

hospital. We still assume that there are three different policy options to improve health care in the recipient country: raising the quality of health care (lowering δ), providing preventive care (lowering p) and reducing the cost of health care (lowering c). Let λ_δ, λ_p, and λ_c be the proportion of health aid allocated to lowering three health care policy variables δ, p, and c, respectively. Then, the total amounts of health aid allocated for three policy options are $\lambda_\delta T$, $\lambda_p T$, and $(1-\lambda_\delta-\lambda_p)T$, respectively.[18] We now assume that each policy variable is a decreasing linear function of the amount of aid allocated for it. It then means that

$$\delta = \delta(\lambda_\delta T),\ p = p(\lambda_p T),\ \ c = c((1-\lambda_\delta-\lambda_p)T);\ \ \delta_T\ , p_T\ , c_T\ < 0 \tag{65}$$

where δ_T , p_T and c_T are the first partial derivatives of δ, p, and c, respectively, and are given exogenously.

So far, the certainty equivalence of each household's lifetime utility, u^c, has been considered as a function of three policy variable δ, p, and c. But from now on, δ, p and c are no longer exogenous policy variables but endogenous variables determined by their amounts of aid allocated. Therefore, the function u^c can be expressed as:

$$u^c = u^c(q, \delta(\lambda_\delta T), p(\lambda_p T), c((1-\lambda_\delta-\lambda_p)T)), \tag{66}$$

where

$$q = q(\delta(\lambda_\delta T), p(\lambda_p T), c((1-\lambda_\delta-\lambda_p)T)). \tag{67}$$

Given the non-zero amount of health aid, T, the health authority chooses the optimal allocation of health aid for three different policy options, λ_δ, λ_p and $\lambda_c = 1-\lambda_\delta-\lambda_p$ to maximize u^c. Thus, we now have two choice variables, λ_δ and λ_p. Totally differentiating

[18] The sum of the proportions of health aid should be equal to one. Thus, $\lambda_c = 1-\lambda_\delta-\lambda_p$.

(66) and (67), we obtain

$$du^c = \frac{\partial u^c}{\partial q}dq + \left(\frac{\partial u^c}{\partial \delta}\delta_T T - \frac{\partial u^c}{\partial c}c_T T\right)d\lambda_\delta + \left(\frac{\partial u^c}{\partial p}p_T T - \frac{\partial u^c}{\partial c}c_T T\right)d\lambda_p. \tag{68}$$

Dividing both sides of (68) by $d\lambda_\delta$ and $d\lambda_p$, respectively, and using $(\partial u^c/\partial q) = 0$ from the household's utility maximization, we can get two first order conditions as

$$\frac{du^c}{d\lambda_\delta} = \left(\frac{\partial u^c}{\partial \delta}\delta_T - \frac{\partial u^c}{\partial c}c_T\right)T = g_\delta(\lambda_\delta, \lambda_p, T) = 0 \text{ and} \tag{69}$$

$$\frac{du^c}{d\lambda_p} = \left(\frac{\partial u^c}{\partial p}p_T - \frac{\partial u^c}{\partial c}c_T\right)T = g_p(\lambda_\delta, \lambda_p, T) = 0, \tag{70}$$

where

$$\frac{\partial u^c}{\partial \delta} = -q(w+\phi)Nip - 2\gamma(\delta q + 1 - q)q(w+\phi)^2 Nip(1-p) < 0, \tag{71}$$

$$\frac{\partial u^c}{\partial p} = -(\delta q + 1 - q)(w+\phi)Ni - \gamma(\delta q + 1 - q)^2(w+\phi)^2 Ni(1-2p) < 0 \text{ and} \tag{72}$$

$$\frac{\partial u^c}{\partial c} = -(1+r)qNi < 0. \tag{73}$$

In other words, for non-zero T, the optimal λ_δ^* and λ_p^* must always satisfy (69) and (70). The second order conditions are assumed to be satisfied, that is, $g_{\delta\delta} < 0$, $g_{pp} < 0$ and $g_{\delta\delta}g_{pp} - g_{\delta p}g_{p\delta} > 0$ and this guarantees that λ_δ^* and λ_p^* yield a maximum of the household's lifetime utility. If the functional forms of δ, p, and c are given, we can derive the closed-form solutions for λ_δ^*, λ_p^* and λ_c^* as functions of non-zero T.

2.3.2 The Comparative Statics

Given each household maximizes its lifetime utility by choosing the optimal number of sick children taken to hospitals, and the health authority allocates health aid for three policy options in a way that help each household maximize its lifetime utility, we wonder how health aid should be reallocated as it increases. From (69)–(73), λ_δ^* and λ_p^* are a

function of an exogenous variable T. Therefore, all other things being equal or held constant, a system of two equations from (69) and (70) can be solved for the two endogenous variables, λ_δ^* and λ_p^*, as functions of T. Having set up the model above, we perform comparative static analyses to examine how a change in the amount of health aid affects its optimal allocation for three health care policy options.

Totally differentiating (69) and (70) with respect to λ_δ^*, λ_p^* and T and doing algebraic manipulations, we obtain a system of two equations as

$$g_{\delta\delta}d\lambda_\delta^* + g_{\delta p}d\lambda_p^* + g_{\delta T}dT = 0 \text{ and} \tag{74}$$

$$g_{p\delta}d\lambda_\delta^* + g_{pp}d\lambda_p^* + g_{pT}dT = 0, \tag{75}$$

where

$$g_{\delta T} = g_{\delta\delta}\frac{\lambda_\delta^*}{T} + g_{\delta p}\frac{\lambda_p^*}{T} \text{ and} \tag{76}$$

$$g_{pT} = g_{p\delta}\frac{\lambda_\delta^*}{T} + g_{pp}\frac{\lambda_p^*}{T}. \tag{77}$$

Therefore, using (76) and (77), we can express the system of equations (74) and (75) in matrix form as

$$\begin{bmatrix} g_{\delta\delta} & g_{\delta p} \\ g_{p\delta} & g_{pp} \end{bmatrix} \begin{bmatrix} d\lambda_\delta^* \\ d\lambda_p^* \end{bmatrix} = - \begin{bmatrix} g_{\delta\delta}\frac{\lambda_\delta^*}{T} + g_{\delta p}\frac{\lambda_p^*}{T} \\ g_{p\delta}\frac{\lambda_\delta^*}{T} + g_{pp}\frac{\lambda_p^*}{T} \end{bmatrix} dT. \tag{78}$$

In order for u^c to attain a local maximum at λ_δ^* and λ_p^*, the second-derivative Hessian matrix

$$H(\lambda_\delta, \lambda_p) = \begin{bmatrix} g_{\delta\delta}(\lambda_\delta, \lambda_p) & g_{\delta p}(\lambda_\delta, \lambda_p) \\ g_{p\delta}(\lambda_\delta, \lambda_p) & g_{pp}(\lambda_\delta, \lambda_p) \end{bmatrix} \tag{79}$$

must be negative definite at λ_δ^* and λ_p^*. We now assume that

$$D_1(\lambda_\delta, \lambda_p) = g_{\delta\delta}(\lambda_\delta, \lambda_p) < 0 \text{ at } (\lambda_\delta^*, \lambda_p^*) \tag{80}$$

and

$$D_2(\lambda_\delta, \lambda_p) = \ g_{\delta\delta}(\lambda_\delta, \lambda_p) g_{pp}(\lambda_\delta, \lambda_p) - [g_{\delta p}(\lambda_\delta, \lambda_p)]^2 = \Omega > 0 \text{ at } (\lambda_\delta^*, \lambda_p^*), \tag{81}$$

thereby satisfying the second partial derivative criterion determining if $u^c(\lambda_\delta^*, \lambda_p^*)$ is a local maximum.

Solving the system of two equations in matrix form (78), we obtain

$$\begin{aligned}
\frac{d\lambda_\delta^*}{dT} &= \frac{g_{\delta\delta}(g_{p\delta}\frac{\lambda_\delta^*}{T} + g_{pp}\frac{\lambda_p^*}{T}) - g_{pp}(g_{\delta\delta}\frac{\lambda_\delta^*}{T} + g_{\delta p}\frac{\lambda_p^*}{T})}{\Omega} \\
&= -\frac{g_{\delta\delta}g_{pp} - g_{\delta p}g_{p\delta}}{\Omega}\frac{\lambda_\delta^*}{T} \\
&= -\frac{\lambda_\delta^*}{T} \text{ and} \qquad (82) \\
\frac{d\lambda_p^*}{dT} &= \frac{g_{p\delta}(g_{\delta\delta}\frac{\lambda_\delta^*}{T} + g_{\delta p}\frac{\lambda_p^*}{T}) - g_{\delta\delta}(g_{p\delta}\frac{\lambda_\delta^*}{T} + g_{pp}\frac{\lambda_p^*}{T})}{\Omega} \\
&= -\frac{g_{\delta\delta}g_{pp} - g_{\delta p}g_{p\delta}}{\Omega}\frac{\lambda_p^*}{T} \\
&= -\frac{\lambda_p^*}{T}. \qquad (83)
\end{aligned}$$

Therefore,

$$\frac{d\lambda_c^*}{dT} = -\left(\frac{d\lambda_\delta^*}{dT} + \frac{d\lambda_p^*}{dT}\right) = \frac{\lambda_\delta^* + \lambda_p^*}{T}. \tag{84}$$

Thes results show that, as total amount of health aid grows, λ_δ^* and λ_p^* decrease and, in contrast, λ_c^* increases. Intuitively, it can be interpreted as that an additional health aid should be allocated for reducing the cost of health care so as to help each poor household maximize its lifetime utility in the most effective way. As health aid continues to flow into the country, the proportion of health aid for cost reduction will be increasing at a diminishing rate, while the others decreasing at a diminishing rate. At the end, λ_δ^*, λ_p^* and λ_c^* will be stationary when λ_δ^* and λ_p^* are equal to zero.

The previous section can provide us with the reason for these findings. The effects of improvements in δ and p, respectively, on q^* are interdependent on each other. An improvement in δ is positively associated with an increase in q^* but it undermines the

beneficial effect of an improvement in p. Also, an improvement in p has a positive effect on q^* but it weaknes the beneficial effect of an improvement in δ. In contrast, the positive effect of a reduction in c on q^* is independent of the improvements in δ and p. For this reason, reducing the cost of health care is supposed to be the most effective in increasing the optimal number of sick children who see a doctor. Therefore, allocating health aid more for reducing the cost of health care is the most effectively way to help poor households choose the optimal number of sick children taken to hospital by maximizing their lifetime utility given their budgen constraints.

2.4 CONCLUSION

In this study, we develop a theoretical model of public health and the optimal allocation of health aid in a poor country. In our model, poor households choose the optimal number of their sick children taken to hospitals to maximize their lifetime utility. Policymakers are supposed to use three policy options to improve public health: raising the quality of health care, providing more preventive care and reducing the costs of health care.

We show how these three policy options influence the optimal number of sick children who are medically treated. The optimal number of sick children who see a doctor obviously increases at a steady rate as the quality of health care improves or the cost of health care reduces. The effect of a change in preventive care provision on the optimal number of sick children who are medically treated is an inverse U-shaped curve. As more preventive care is provided out of nothing, the optimal number of sick children taken to hospital increase to begin with, and at a critical point of the preventive care, it achieves the maximum level. As the provision of preventive care increases above the critical point, however, the optimal number of children seeing a doctor rather starts to decrease and quickly reaches zero. Also, two policy options, quality improvement and preventive care provision, are interdependent on each other. In other words, an improvement in one undermines the beneficial effect of the other. In contrast, the beneficial effect of cost reduction in health care does not depend

on the other policy options. Therefore, reducing the cost of health care is the most obvious way for policymakers to implement to increase the optimal number of sick children who are medically treated.

Also, the country's health authority allocates its health aid for three policy options so as to maximize each household's lifetime utility. Solving the model and performing the comparative static analyses, we find that the proportion of health aid for reducing the cost of health care should increase, until all amounts of health are used for cost reduction in health care. This finding also means that health aid used to reduce the cost of health care is the most effective in helping poor household maximize their lifetime utility.

CHAPTER 3

HETEROGENEITY IN HEALTH AID: A PANEL DATA ANALYSIS OF THE EFFECTIVENESS OF HEALTH AID

3.1 INTRODUCTION

In poor countries, illness can decrease household savings, take away education opportunities and reduce productivity, thereby perpetuating poverty. WHO and World Bank (2002) analyze 127 case studies to examine why families have fallen into poverty and ill-health turns out to be the biggest single factor in poverty. They show that one of the most pressing agendas for poor countries is to improve the health of their people. We can see how seriously the global community takes health issues in less developed countries by looking at the eight Millennium Development Goals (MDGs) out of which five are the health-related goals.[19] The aid donor community has also paid attention to health challenges in developing countries. As a result, recent years have witnessed explosive growth of foreign aid for improving public health.[20]

Health aid has been widely credited with successfully financing the improvements of health care in poor countries. Levine (2004, 2007) surveys over 20 success cases in improving public health in developing countries and finds that most of the cases succeeded due to health aid. In contrast, however, the empirical literature has not reached a robust consensus on the positive effect of health aid on public health outcomes in the recipient countries. To the best of our knowledge, there are three past studies on the relationship between health aid and health outcomes, which are Williamson (2008), Mishra and

[19] The representative health indicators for the health-related MDGs are as follows: underweight prevalence of under-5 children (MDG 1: to eradicate extreme poverty and hunger), under-5 mortality rate (MDG 4: to reduce child mortality rates), maternal mortality ratio (MDG 5: to improve maternal health), AIDS prevalence, use of insecticide-treated bed nets (MDG 6: to combat HIV/AIDS, malaria, and other diseases), use of improved water source, and use of improved sanitation facilities (MDG 7: to ensure environmental sustainability)

[20] In reality, development assistance for health has increased from US $5.7 billion in 1990 to a record-high of US $28.2 billion in 2010 (IHME, 2012)

Newhouse (2010) and Wilson (2011). But they drew conflicting conclusions. Williamson (2008) is the first to empirically test the effectiveness of health aid to public health, using development aid for health (DAH) data from OECD's Credit Reporting System and a variety of health indicators such as infant mortality rate, life expectancy, death rate, two immunizations (DPT and measles). She found no evidence that international aid to the health sector is effective in improving public health outcomes. Wilson (2011) extends Williamson's (2008) study by using a new data set on health aid from AidData[21] and found no significant evidence of a positive effect of DAH on mortality. On the other hand, using the data on health aid from OECD, Mishra and Newhouse (2009) empirically found that health aid has a significantly positive effect on a reduction in infant mortality.

Why have we not achieved the empirical consensus on either positive or negative effects of health aid on health improvements in the country-level panel data studies, even though there are many obvious success stories on them? Of course, one of the reason is that a variety of panel data regression models often yield completely different results. In this study, we argue that another important reason for that is that past studies have ignored heterogeneity within health aid, which simply means that there are different types of health aid and each of them operates differently on health outcomes. A separate strand of the aid literature has been viewing aid effectiveness from another angle. The literature on, so called, the "aid heterogeneity" criticizes that the majority of the empirical literature on the aid-growth nexus have ignored the heterogeneous character of foreign aid (see, for example, Mavrotas, 2005; Mavrotas and Ouattara, 2003, 2006). In other words, the existing literature has not found no robust evidence of the positive effect of foreign aid on economic growth because it has evaluated aid effectiveness in the aggregate. It has not taken into account that aid is heterogenous and each component of aid yields different effect on the aid-recipient economy. By the same token, the reason that there is no

[21] AidData has been developed jointly by the College of William and Mary and Brigham Young University as a new database on foreign aid which combines the OECD's Creditor Reporting System (CRS) database with a range of data directly from both bilateral and multilateral donor agency sources. For the details, see Tierney et al. (2011).

empirical consensus on the effectiveness of health aid is also that the existing literature have used aggregate health aid data.

In this study, we rely on a panel data set of 119 aid-recipient countries from 1975 and 2010. We disaggregate health aid data from AidData and empirically reexamine if health aid either positively or negatively affects health outcomes and if there is heterogeneity in health aid, that is, more specifically, if there is a particular type of health aid that excels at improving a particular type of health indicator. We disaggregate health aid data into three health policy options: health aid for the quality of health care, health aid for the provision of preventive care, and health aid for the cost reduction of health care. Wilson (2011) also subdivides his health aid variable into nine categories for a robustness check. But the author uses the nine sub-sector variables only to estimate their effects only on infant mortality. While Mishra and Newhouse (2010) use infant mortality only as their primary health indicator, we use three different health indicators, which are child mortality, life expectancy and death rate, as proxies for health outcomes. Williamson (2008) also used three health indicators as the same as we do. But the author did not consider heterogeneity in health aid and used aggregate health aid variable as the main variable of interest.

Williamson (2008) uses current aid variable as the main aid regressor in her benchmark models. But we follow Mishra and Newhouse (2009) and use one-period lagged aid variables rather than current ones. The use of one-period lagged aid regressors basically enable us to partly tackle their possible endogeneity issues from reverse causality. It is not easy to come up with good instruments for health aid since many macroeconomic and political variables are all interrelated and affect both health aid and health indicators, but one possible solution is to use lags of the aid variables. The idea is that, while health aid may affect health indicators and vice versa, it is less likely that health indicators can influence past values of health aid and so they might be used as instruments for current health aid variables. Another important reason for the use of one-lagged aid regressors is that it allows health aid enough time to operate on health outcomes. In other words, we

consider the fact that it takes time for health aid to bear good fruit. By the same token, the other controls are also instrumented by their one-period lagged values.

For this study, four panel regression models are employed to estimate the effects of health aid on health outcomes. The baseline model is the least-squares dummy variable (LSDV) model with country- and time-specific effects. In this model, we find that the error terms are serially correlated. We basically compute cluster-robust standard errors that are robust to heteroskedasticity and serial correlation. We also use two altenative methods to correct for the serial correlation. The first one is the fixed-effects model with a lagged dependent variable. Under the assumption that the error terms follow the AR(1) model, the use of a lagged dependent variable as a regressor is expected to mitigate serial correlation and reduce omitted variable bias. It also enable us to remove omitted variable bias because the lagged dependent variable is the best proxy for all omitted variables. The second one is the fixed effects model with an AR(1) disturbance suggested by Baltagi and Wu (1999).

The fixed effects model with a lagged dependent variable ignores the correlation between the lagged dependent variable and the error term. This endogeneity of the lagged dependent variable make the estimated coefficients downward-biased (Nickell, 1981). This "Nickell bias" is even bigger with smaller T and more severe autocorrelation. We suspect one-period lagged aid regressors to be still endogenous because there may be omitted important variables that affect both yesteryear's health aid and this year's health indicators. Moreover, the other control variables are not free from endogeneity. Therefore, we employ the system GMM model to correct simultaneously for the Nickell bias and all the possible endogeneity of regressors.

Our main findings support the main hypothesis that there is heterogeneity in health aid. Unfortunately we find no statistically significant evidence of the beneficial effects of health aid on a reduction in child mortality. In contrast, we find empirical evidence that

health aid for the cost reduction of health care is the most effective in raising life expectancy and reducing death rate. We also find there is heterogeneity in the allocation of health aid. Countries with worse health status receive more Aid-P and Aid-C but less Aid-Q. In other words, Aid-P and Aid-C are allocated by the needs of the recipients, while more Aid-Q goes to countries where it will be more effective.

The outline of this chapter is as follows. Section 2 discusses the data, the empirical specifications and the estimation methods. In Section 3, we present the empirical results. Section 4 conclude and draw a couple of policy implications.

3.2 THE EMPIRICAL FRAMEWORK

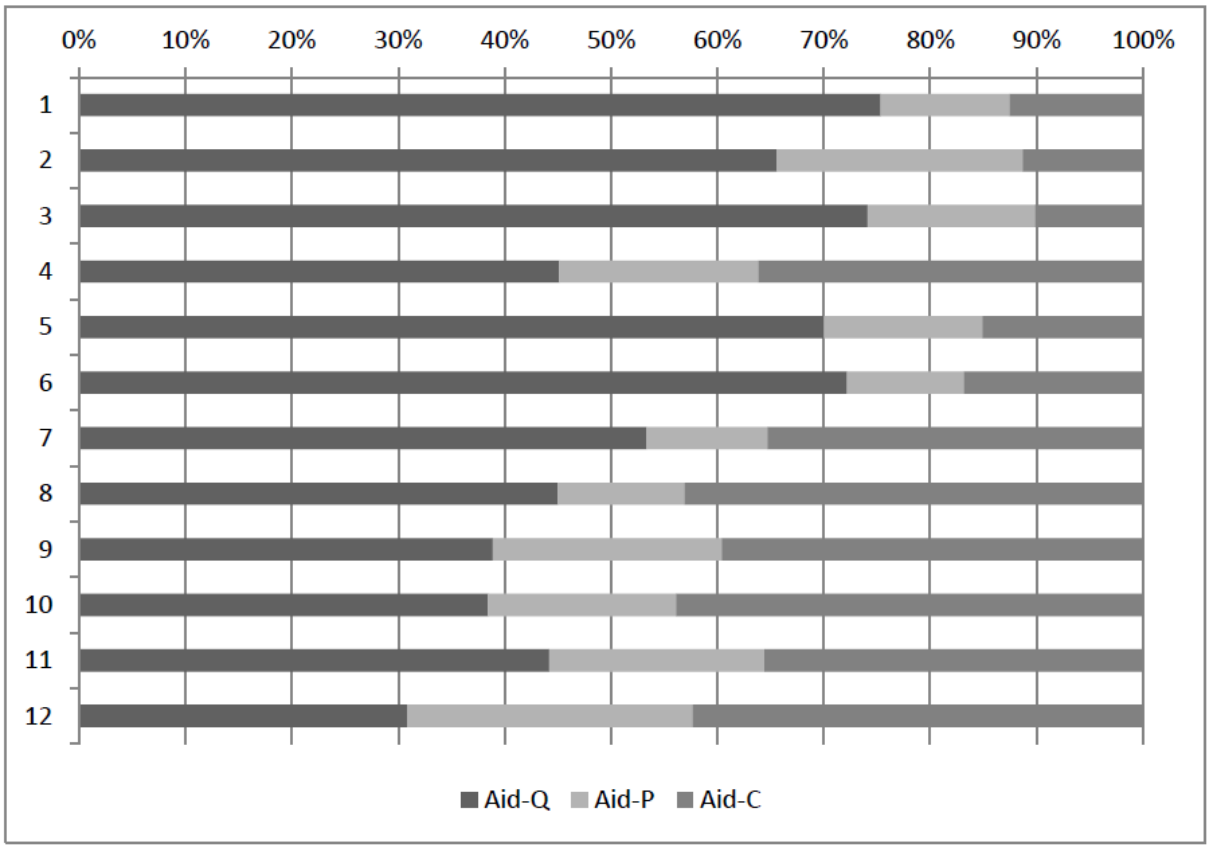
3.2.1 The Data

For this analysis, we use a three-year averaged panel data set of 119 aid-recipient countries ($N = 119$)[22] for 12 periods ($T = 12$) from 1975 through 2010. The data on health aid are collected from the online database AidData version 2.0.[23] In AidData, each health aid project, if its purpose is specified, is assigned one of ten five-digit purpose codes starting with 12000 to 12281, according by OECD CRS purpose code. In AidData, there are two more health-related aid classifications: polulation policies/programs and reproductive health (starting with 13000 to 13081); and water supply and sanitation (starting with 14000 to 14082). But we do not add these two types of aid to our data set because, for example, not all amount of aid for water and sanitation is focused on improving health issues. We rather choose the way of controlling for the effects of the two types of health-related aid on health outcomes.

For all i and t, total health aid per capita (hereafter, Aid-T) in country i at time t is categorized into three types by policy options so as to construct three heterogeneous health aid variables. They are i) health aid per capita for improving the quality of health care

[22] The list of 119 aid-recipient countries for this analysis is provided in Table 3.5 in the appendices.
[23] The updated AidData 2.1 full version is now available at http://aiddata.org.

(hereafter, Aid-Q), ii) health aid per capita for providing more preventive care (hereafter, Aid-P) and iii) health aid per capita for reducing the cost of health care (hearafter, Aid-C). Aid-Q contains aid for health administration, medical education and research and basic health infrastructure. Aid-P consists of aid for basic nutrition, infectious and parasitic disease control and public health education. Aid-C is composed of aid for supply of medical services and basic health care.[24] Figure 3.1 shows time trends in the ratios of three types of health aid to total health aid for twelve time periods. In 1970s and 1980s, Aid-Q took the largest proportion of Aid-T. Since 1990s, the proportions of Aid-P and Aid-C have been increasing, while Aid-Q has been representing an decreasing share of total health aid.



Note: The Y-axis represents three-year averaged twelve time periods

Figure 3.1: Trends of Three Types of Health Aid (Unit: % of Total)

[24]For more detailed information of the sub-division of health aid, see Table 3.6 in the appendices.

AidData contains both aid commitments and disbursements data. All data we use in this analysis are in the form of commitments for two reasons. First, while disbursements are theoretically more appropriate for the empirical study, the data on disbursements are missing in many cases. The OECD CRS data, one of AidData's largest sources, contain many disbursement records that cannot be reliably linked to commitment records for the same project. For this reason, AidData data exclude the records that only contain disbursements. This means that AidData's disbursement sums for OECD donors do not necessarily reflect the values reported by the donors (AidData, 2011). Second, in AidData data set, "year" is commitment year. This indicates that health aid disbursed years later after its commitment is considered to be occurred in its commitment year. This may distort our empirical results. One reasonable way to make up the weakness of using aid commitment data is to take the time lag between commitments and disbursements. Unfortunately, the time lag between them varies across projects and donors. Following the previous literature, therefore, we average our annual data over three-year periods.[25] Futher, we use one-period lagged aid varibles as our main variables of interest.[26] This method also enables us to reduce annual fluctuations and measurement errors and allow health aid enough time to operate longer on health outcomes. Further, it allows us to avoid endogeneity to some degree.

Our dependent variables are three health indicators: child mortality rate (under-5 mortality rate), life expectancy and death rate.[27] While we employ child mortality rate as one of our health indicators, Williamson (2008) and Mishra and Newhouse (2010) used infant mortality rate as the primary health variable.[28] Infant mortality covers only infant

[25] In general, the previous empirical literature on foreign aid has used five-averaged aid data. But we rather use three-averaged aid data to make our sample size bigger so that we can obtain more accurate estimation results. WHO, OECD and World Bank (2008) find that disbursements of health ODA are equivalent to more than 80% of the average annual commitment over the previous three to five years.

[26] Wilson (2011) used the same aid commitments data as we use. However, instead of using one-period lagged aid variables, he assumed a one-year offset between the time a commitment is made and the money is spent.

[27] Fore more detailed information on the definitions of health indicators, see Table A-3 in the appendix.

[28] For the reasons that infant mortality is a good health indicator, see Mishra and Newhouse (2010).

deaths less than one year of age and mainly occurs in the neonatal period. Leading causes of infant mortality are preterm birth, birth asphyxia (lack of breathing at birth), Sudden Infant Dealth Syndrome (SIDS) and various infections.[29] Therefore, infant mortality cannot cover children's death occured after their first birthday mainly by malnutrition and infectious and parasitic disease at which our Aid-P variable is targeted. Thus, child mortality rate is more appropriate than infant mortality as a dependent variable that is expected to be affected by health aid.[30] As one of the primary health indicators, life expectancy tells how long a person on average is likely to live. Actually, it is also highly correlated with infant and child mortality rates.[31] Changes in income, health conditions, and education are constantly occurring and will affect life expectancy in a country. Death rate (crude) is also a good indicator of the general health status of a geographic area or population. Unlike age-specific mortality rates, however, it reflects the overall mortality level of a population and summarizes the mortality pattern that prevails across all age groups in a given year. Interestingly, higher death rate can be found in some developed countries with high life expectancy and low age-specific mortality rates, because these countries have a higher proportion of older people due to lower birth rates. Therefore, using death rate as a dependent variable enables us to capture the effects of health aid on health status in old age groups.

Except for health aid variables, the other variables come from the World Development Indicators published by the World Bank.[32] All the variables used in this study are averaged over three-year intervals and are specified in logarithmic form. Therefore, our data set contains twelve-period time panel for 119 cross-sections. Descriptive statistics for all the variables for this study appear in Table 3.1.

[29] For more details, see Global Health Observatory on the website of WHO, available at http://www.who.int/gho/urban_health/outcomes/infant_mortality_text/en/

[30] Of course, infant mortality rate and child mortality rate are highly correlated. In our data set, correlation between them is 0.9924.

[31] In general, improvements in infant and child mortality rates result in increases in life expectancy. In our data set, life expectancy is negatively correlated with infant mortality (-0.8544) and child mortality (-0.8848).

[32] Data on the World Development Indicators are available at http://data.worldbank.org/products/wdi.

Table 3.1: Descriptive Statistics (in Logarithmic Form)

Variable	Obsevations	Mean	Standard Deviation	Minimum	Maximum
Child Mortality Rate	1396	4.278	0.859	1.723	5.808
Life Expectancy	1422	4.079	0.180	3.347	4.370
Death Rate	1423	2.312	0.458	1.091	3.569
Aid-T	1182	-0.234	2.148	-13.428	5.063
Aid-Q	1136	-1.023	2.212	-13.428	4.511
Aid-P	868	-2.088	2.280	-8.818	3.981
Aid-C	918	-1.392	2.360	-10.994	4.496
GDP Per Capita (PPP)	1114	7.593	0.997	4.533	10.281
GDP Per Capita Growth	877	0.846	1.133	-5.704	3.811
Population, Total	1423	15.891	1.564	12.209	20.986
Population Growth	1339	0.624	0.679	-4.669	2.312
Improved Water Source	815	4.276	0.347	1.579	4.605
Improved Sanitation Facilities	804	3.790	0.758	0.861	4.605
Birth Rate	1423	3.414	0.412	2.050	4.056
Fertility Rate	1424	1.398	0.465	0.125	2.217
Political Rights Index	1319	1.403	0.532	0	1.946
Civil Rights Index	1319	1.429	0.420	0	1.946

3.2.2 The Estimation Model

It is not easy for researchers to search for the best specification and model. Often, many alternative approaches yield different results in panel data analyses. For example, Williamson (2008) does not include a lagged dependent variable in the model, whereas Mishra and Newhouse (2010) and Wilson (2011) do. Also, Willamson (2008) and Wilson (2011) use current values of health aid as a variable of interest, while Mishra and Newhouse (2010) introduce one-period lagged aid to their model as we do. Wilson (2011) employs nine different models in his analysis and concludes that the overall results are very sensitive to specification and estimation method.

We totally agree with Wilson's (2011) comment on model selection that the choice of a model should be driven by theory and by the features of the data. By theory and by the features of the data, therefore, we also estimate a variety of panel data regression models from the fixed effects model without a lagged dependent variable to the system GMM model. For comparison purposes, we show all their estimation results.

3.2.2.1 The Baseline Model

Our baseline regression model is the least-squares dummy variable (LSDV) fixed-effects model with both country- and time-fixed effects,[33] which is as follows:

$$Y_{it} = \alpha + \mathbf{T}'_{it-1}\boldsymbol{\beta} + \mathbf{X}'_{it-1}\boldsymbol{\delta} + \nu_i + \xi_t + u_{it}, \tag{85}$$

where the subscript i denotes countries whereas and t denotes time, Y_{it} is a health indicator (child mortality rate, life expectancy or crude death rate), $\mathbf{T}_{it-1}$ is a column vector of three one-period lagged health aid variables allowing health aid to operate on improvements in health outcomes over a longer time-period, $\mathbf{X}_{it-1}$ is a column vector of the other lagged control variables, ν_i captures time-invariant country-specific effects, ξ_t captures country-invariant time-specific effects, and u_{it} is the remainder stochastic error terms that represents the omitted variables. We prefer the LSDV model to the within-groups model because we can explicitly obtain the estimated unobserved country-specific effects.

GDP per capita (PPP), GDP per capita growth, total population and population growth are introduced to capture the recipients' initial economic and population status. We include fertility rate and birth rate to control for the effects of aid for polulation policies/programs and reproductive health on health outcomes. In the same way, we also control for the effects of aid for water supply and sanitation by adding improved sanitation facilities (hearafter, sanitation) and improved water source (hereafter, clean water). All the variables for this study are specified in logarithmic form because the log-log specification smoothes our data and allows us to interpret the estimated coefficients as elasticities. As we mentioned in the introduction, we also use one-period lagged aid regressors both to tackle potential endogeneity issues and to allow health aid and other controls enough time to work for health improvements.

[33] We run the Hausman tests to decide between fixed or random effects. We obtain three test statistics as follows: $\chi^2_{(15)} = 43.60$ (child mortality), $\chi^2_{(15)} = 100.49$ (life expectancy) and $\chi^2_{(15)} = 54.89$ (death rate). They obviously exceeds the 1% critical value in the $\chi^2_{(15)}$ distribution. Thus, we reject the null hypothesis that the preferred model is random effects.

Using the Wooldridge test for autocorrelation in panel data, we test panel autocorrelation and find that the error terms are serially correlated.[34] The modified Bhargava et al. Durbin-Watson statistic (Bhargava et al., 1982) also tells that we reject the null hypothesis of no first-order autoregressive process in all three regressions. We suppose that it is caused by persistence of the effects of omitted variables. Under serial correlation, the regression coefficients remain unbiased but the high variation in the estimates causes the OLS estimator inefficient. Therefore, we calculate cluster-robust standard errors that make inference fully robust to heteroskedasticity and serial correlation for large N and small T panel data.

3.2.2.2 Correcting for Serial Correlation and Omitted Variable Bias

We now reasonably assume that in (85) u_{it} follows the AR(1) model because our data are three-year averaged annual data as in the following:

$$u_{it} = \rho u_{it-1} + \varepsilon_{it}; \;\; |\rho| < 1; \; \varepsilon_{it} \sim N \; (0, \; \sigma_{\varepsilon}^2). \tag{86}$$

We use two alternative models to deal with AR(1) autocorrelation. First, following Mishra and Newhouse (2010) and Wilson (2011), we consider the fixed effects model with a lagged dependent variable. The inclusion of a lagged dependent variable as a regressor mitigate serial correlation because the lagged dependent variables implicitly include lagged error terms into the specification. Moreover, adding the lagged dependent variable to the model significantly reduce omitted variable bias. Our fixed effects model with a lagged dependent variable (LDV) is as follows:

$$Y_{it} = \alpha + \gamma Y_{it-1} + \mathbf{T}_{it-1}^{'}\boldsymbol{\beta} + \mathbf{X}_{it-1}^{'}\boldsymbol{\delta} + \nu_i + \xi_t + u_{it}. \tag{87}$$

[34]The Wooldridge test results for the regressions of child mortality, life expectance and death rate are F(1, 61) = 114.946, F(1, 61) = 543.009 and F(1, 61) = 259.581, respectively. They tell us that we reject the null hypothesis of no first-order autocorrelation at the 1% level of significance.

We also the cluster-robust sandwich estimator of variance robust to groupwise heteroskedasticity and serial correlation.

Second, we estimate equation (85) again using another panel regression model specializing in a AR(1) disturbance, which is the within-group model with a AR(1) disturbance suggested by Baltagi and Wu (1999). The within-transformed equation can be used to estimate ρ. Given ρ, the panel-by-panel Cochrane-Orcutt is performed and the within-panel means are removed. After that, OLS on the within-transformed data is used to obtain the within estimator. We use a Stata routine, xtregar, to estimate this model. For the within transformation to remove the fixed effects, the first observation of each country is lost.

3.2.2.3 Dealing with Endogeneity and the Nickell Bias

In our panel regression models, however, our one-period lagged aid regressors are still suspected to be endogenous because, even though the possibility of reverse causality is very low, there may be important omitted variables influencing both one-period lagged aid regressors and current health indicators. If they are shown to be endogenous, their estimates from our OLS regression models would be biased. Also, other eight controls are not free from endogeneity. They should be instrumented with valid instrumental variables that are relevantly correlated with suspected endogenous regressors but are not correlated with health indicators. But it is not easy for us to find valid instruments for total 11 right hand side regressors. In this case, the difference and system GMM estmators can be good alternatives for tackle endogeneity of the aid regressors because these estimators embody the assumption that the only available instruments are internal (Roodman, 2009).

Nickell (1981) shows that the use of dummy variables to estimate individual-specific effects in a panel data model with a lagged dependent variable results in downward-biased estimates when T is small. Therefore, the fixed effects estimator is no longer consistent when N goes to infinity with fixed T. The "Nickell bias" approaches zero as T approaches

infinity. This bias is not directly caused by autocorrelation of the error terms. But if the error terms are autocorrelated, it is even more severe. The reason for this is, even though $\text{cov}(Y_{it-1}, u_{it}) = 0$, $\text{cov}(Y_{it-1}, u_{it-1}) \neq 0$ in (87) because $\text{cov}(u_{it}, u_{it-1}) \neq 0$ in (86). In other words, Y_{it-1} is endogenous. The instrumental variable methods correct for the correlation between Y_{it-1} and u_{it} by replacing with $\widehat{Y_{it-1}}$. Using a Monte Carlo approach, Judison and Owen (1999) find that the Nickell bias for dynamic panel data models can be sizeable, even when $T = 20$. Considering that our panel data set consists of 12 periods ($T = 12$), our fixed effects model with a lagged dependent variable is not free from the Nickell bias.

In order to remedy the Nickell bias and the endogeneity of regressors, the econometric literature has suggested a number of consistent instrumental variable (IV) and generalized method of moments (GMM) estimators, such as the Anderson-Hsiao IV estimator (Anderson-Hsiao, 1982), the first-differenced GMM estimator (Arellano and Bond, 1991) and the system GMM estimator (Blundell and Bond, 1998) and the bias-correction of least-squares dummy variable (LSDV) estimator (Bruno, 2005a, 2005b; Bun and Carree, 2005a, 2005b). Buddelmeyer et al. (2008) show that, when N is very large, the GMM estimators outperform the others. Flannery and Hankins (2013) confirm that the GMM estimators perform well in the presence of endogenous regressors and, in particular, the system GMM estimates are more consistent and accurate across a range of endogeneity in the presence of serial correlation.

Another advantage of the GMM estimators is to allow us to explore the nature of the allocation of health aid by shedding light on the direction of the bias and the sign of the feedback effect. We expect that there is also heterogeneity in the allocation of health aid, that is, different types of health aid is allocated to the recipients in different ways.

For this study, all things considered, the Blundell and Bond's (1998) system GMM estimator is chosen to tackle the Nickell bias and the endogeneity of health aid regressors. In the system GMM specification, the level equation is the same as (87) and the

first-differenced equation is as follows:

$$\Delta Y_{it} = \gamma\left(\Delta Y_{it-1}\right) + \Delta\left(\mathbf{T}'_{it-1}\right)\boldsymbol{\beta} + \Delta\left(\mathbf{X}'_{it-1}\right)\boldsymbol{\delta} + \Delta\xi_t + \Delta u_{it}. \tag{88}$$

Actually, three aid regressor are policy variables that are used systematically by the donor community to achieve certain health outcomes and thus they change to reflect the feedback from health indicators. In this model, therefore, they are treated as endogenous variables that has the property that $E(u_{it}|\ \mathbf{T}_{1,\ldots,}\mathbf{T}_{it-1}) \neq \mathbf{0}$. We also treat the other control variables in a vector of $\mathbf{X}_{it-1}$ as endogenous variables whose property is that $E(u_{it}|\ \mathbf{X}_{1,\ldots,}\mathbf{X}_{it-1}) = \mathbf{0}$ for all t. We treat the time-fixed effects as strictly exogenous.

We use a popular user-written routine, `xtabond2`, suggested by Roodman (2009) to estimate our system GMM models. We employ two-step GMM rather than one-step GMM to obtain more asymptotically efficient estimates. To correct for severe downward bias of the efficient two-step GMM estimators in small samples, we also apply the Windmeijer finite-sample correction (Windmeijer, 2005) to these standard errors.

Lagged differences of the endogenous variables in (88) are used as instruments in the level equation (87), while their lagged levels in (87) are introduced as instruments in the first-differenced equation (88). Roodman (2009) warns that adding too many instruments increases finite-sample bias and weaken the Hansen J test's power by producing nonsensically high p-values of 1.000. Interestingly, his stata routine, xtabond2, is designed to warn when instruments outnumber panel units, as a minimally arbitrary rule of thumb (Roodman, 2009). Thus, we try to minimize the number of instruments used in the system GMM model. In our system GMM estimation, therefore, suspected endogenous regressors are instrumented with only lag two of the levels for the first-differenced data and with only lag one of the differences for the level data. The time fixed-effects are assumed to be exogenous and enters instrument matrix on its own.

3.3 THE EMPIRICAL RESULTS

3.3.1 The Effects of Health Aid on Child Mortality

The effects of different types of per capita health aid on child mortality are shown in Table 3.2. As expected, four alternative models yields different results. In specification (1b), the Bhargava et al. Durbin-Watson statistics tells that we cannot reject the null hypothesis that the regression of child mortality has a AR(1) disturbance. In specification (2), Hansen's J-statitic indicates that all instruments in the system GMM model are exogenous.

Unfortunately, we cannot find any significant evidence of health aid on reducing child mortality, which is consistent with Williamson's (2008) and Wilson's (2011) conclusions. But we find that three types of aid are allocated differently to the recipients. The estimated cofficient on Aid-Q in specification (1) is negative and statistically significant at the 10% level of significance. In specification (1a) and (1b), it is still negative but no longer significant. But, this negative coefficient on Aid-Q may be biased from endogeneity. After tackling the Nickell bias and potential endogeneity, however, it turns to be positive in specification (2). This indicates that, in specification (1)–(1b), it may be biased downwards due to a negative correlation between the unexplained components of child mortality with Aid-Q. Intuitively, this suggests that more Aid-Q is distributed toward countries with lower child mortality. This is consistent with Wilson's (2011) health aid allocation story that donor countries put in place policies that are designed to give more health aid to countries where child mortality declined more steeply.

If we pay attention to Aid-P and Aid-C, their estimated signs in specification (1) and (1b) are positive. But after adding the lagged dependent variable and further avoiding the Nickell bias and endogeneity, they turn to be negative. This results from the removal of positive reverse causality. This suggests that more Aid-P and Aid-C go to countries with higher child mortality.

Table 3.2: The Estimated Effects of Health Aid on Child Mortality

	(1)	(1a)	(1b)	(2)
Model	FE-LSDV	LDV	FE: AR(1) Error	System GMM
Child Mortality (L1)		0.842*** (9.97)		1.053*** (34.97)
Aid-Q (L1)	-0.0165* (-1.86)	-0.00319 (-0.87)	-0.0414 (-1.10)	0.00371 (0.63)
Aid-P (L1)	0.00692 (1.00)	-0.00126 (-0.43)	0.000269 (0.09)	-0.000168 (-0.03)
Aid-C (L1)	0.00538 (1.00)	-0.00214 (-0.81)	0.000919 (0.29)	-0.000568 (-0.11)
GDP per capita (L1)	-0.0707 (-0.89)	-0.0316 (-0.97)	-0.0260 (-0.37)	0.0241 (0.97)
GDP Growth (L1)	0.00488 (0.71)	-0.00631* (-1.45)	0.00518 (1.27)	-0.0201*** (-2.68)
Total Pop. (L1)	0.244 (0.71)	0.0972 (0.74)	-0.0707 (-0.82)	-0.0139 (-1.52)
Pop. Growth (L1)	0.00236 (0.08)	0.00763 (0.55)	-0.00820 (-0.91)	-0.00949 (-0.45)
Fertility Rate (L1)	0.0954 (0.33)	0.135 (0.83)	0.338 (1.29)	-0.00885 (-0.06)
Birth Rate (L1)	0.413 (1.11)	-0.0654 (-0.34)	0.0739 (0.30)	-0.0213 (-0.18)
Clean Water (L1)	-0.110 (-0.51)	-0.0662 (-0.70)	-0.566 (-1.65)	0.0127 (0.31)
Sanitation (L1)	-0.0890 (-0.86)	-0.0233 (-0.61)	0.0126 (0.06)	-0.0186 (-0.66)
Constant	0.201 (0.03)	-0.410 (-0.19)	-0.103** (-2.40)	-0.164 (-0.44)
Observation	358	358	255	358
Adj. R^2	0.989	0.997		
Bhargava et al. DW			0.717	
# of IVs				132
AB-AR(1) (P-value)				0.545
AB-AR(2) (P-value)				0.152
Hansen's J (P-value)				0.975

Note: i) Both country and time fixed effects are included.
ii) * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$, iii) T-statistics in parenthesis

We have another example supporting that there is endogeneity in specification (1)–(1b). The coefficient on GDP per capita growth has the unexpected positive sign in specification (1) and (1b). After adding the lagged dependent variable, it turns to be negative and statistically significant at the 10% level of significance. In specification (2), it is statistically significant at the 5% level and its magnitude in absolute terms gets much bigger than that in specification (1a). This indicates that it may be upward-biased because of a positive correlation between the unobserved components of child mortality and GDP per capita growth. This positive correlation makes sense because developing countries that have higher child mortality than developed countries also have higher growth rates.

We now turn to the effects of other controls on child mortality. The estimated cofficients on the lagged dependent variable, which is close to one in specification (1a) and

a little bit over one in specification (2), show us that child mortality is highly persistent. This result is consistent with Mishra and Newhouse's (2010) and suggests that a virtuous or vicious circle of child mortality exists in each country (Mishra and Newhouse, 2010). In specification (2), as mentioned above, the coefficient on GDP per capita growth is negative and statistically highly significant, thereby indicating that higher economic growth leads to lower child mortality.

3.3.2 The Effects of Health Aid on Life Expectancy

We now move to the regressions of life expectancy. They appear in Table 3.3. In specification (1b), the Bhargava et al. Durbin-Watson statistics tells that we cannot reject the null hypothesis that the regression of life expectancy has a AR(1) disturbance. In specification (2), Hansen's J-statitic indicates that all instruments in this model are exogenous.

We find that specification (1) and (1b) suffer from omitted variable bias and endogeneity. In specification (1), the coefficient on Aid-Q is positive and those on Aid-P and Aid-C are negative. But after adding the lagged dependent variable as a proxy for all omitted variables and avoiding the Nickell bias and endogeneity, each of them turns to be the opposite sign. Intuitively, these results suggest that more Aid-Q is allocated to countries with higher life expectancy, while more Aid-P and Aid-C go to countries with lower life expectancy.

We now pay more attention to the coefficients on Aid-C. In specification (1a), the estimated cofficient on Aid-C is positive and statistically significant at the 5% level of significance. The estimated coefficient, 0.00134, means that, *ceteris paribus,* a 1% increase in Aid-C is significantly associated with a 0.00134% increase in life expectancy. In order to interpret this result, we take a specific example of Sierra Leone which has the lowest overall life expectancy in the world in 2013 by the WHO. From the period between 2002 and 2004, Sierra Leone received health aid per capita of only 12 cents for reducing the cost of health

care. For the next three years, Sierra Leone's overall life expectancy is 45.04. If Aid-C increased up to a dollar by 743.3% during the period from 2002 to 2004 in Sierra Leone, then its life expectancy would have been 45.5 for the next period.

Table 3.3: The Estimated Effects of Health Aid on Life Expectancy

	(1)	(1a)	(1b)	(2)
Model	FE-LSDV	LDV	FE: AR(1) Error	System GMM
Life Expectancy (L1)		0.591*** (10.63)		0.975*** (22.35)
Aid-Q (L1)	0.00265 (1.14)	-0.000195 (-0.22)	0.0000719 (0.08)	-0.00224 (-1.61)
Aid-P (L1)	-0.000338 (-0.26)	0.000904 (1.31)	0.000408 (0.58)	0.000133 (0.13)
Aid-C (L1)	-0.000855 (-0.72)	0.00134** (2.24)	-0.000571 (-0.80)	0.00217 (1.64)
GDP per capita (L1)	-0.00241 (-0.26)	-0.00430 (-0.80)	-0.00709 (-0.44)	-0.0173*** (-3.10)
GDP Growth (L1)	-0.00267 (-1.12)	0.000165 (0.19)	-0.00201** (-2.18)	0.00696*** (3.48)
Total Pop. (L1)	0.182*** (2.73)	0.0298 (0.82)	0.0429** (2.18)	-0.00113 (-0.44)
Pop. Growth (L1)	0.00230 (0.41)	0.000689 (0.21)	-0.000498 (-0.24)	0.00647 (0.75)
Fertility Rate (L1)	-0.0221 (-0.37)	-0.00839 (-0.42)	-0.151** (-2.52)	0.00659* (1.96)
Birth Rate (L1)	0.0432 (0.52)	0.0494** (2.01)	0.0769 (1.39)	-0.0843 (-1.66)
Clean Water (L1)	0.0535 (1.20)	0.0445 (1.21)	0.192** (2.49)	0.0252 (1.41)
Sanitation (L1)	0.0424* (1.68)	0.0129 (0.78)	-0.0668 (-1.43)	0.0364 (0.40)
Constant	0.329 (0.31)	0.715 (1.01)	-0.00177 (-0.18)	0.341 (1.34)
Observation	358	358	255	358
Adj. R^2	0.983	0.996		
Bhargava et al. DW			0.681	
# of IVs				132
AB-AR(1) (P-value)				0.300
AB-AR(2) (P-value)				0.776
Hansen's J (P-value)				0.993

Note: i) Both country and time fixed effects are included.
ii) * p < 0.1; ** p < 0.05; *** p < 0.01, iii) T-statistics in parenthesis

In specification (2), after the system GMM estimator removes the downward bias, the magnitude of the effect of Aid-C on an improvement in life expectancy is bigger than that in specification (1a), even though the estimated cofficient does not quite reach the limit of statistical significance ($p = 0.103$). In sum, therefore, Aid-C is supposed to be the most effective in increasing life expectancy.

We turn to the estimated coefficients on the other controls. The estimated coefficient

on GDP per capita growth has a negative sign in specification (1) and (1b). But this negative coefficient may be biased downwards because of a negative correlation between the unobserved components of life expectancy and growth. This negative correlation reflects that developed countries with higher life expectancy have lower growth rates. After adding the lagged dependent variable as a proxy for omitted variables in specification (1a), the coefficient on GDP per capita growth turns to be positive. In the system GMM model (2), it becomes highly statistically significant and its magnitude gets much bigger than that in specification (1a). In specification (2), we find that GDP per capita growth rate is positively associated with life expectancy at the 1% level of significace and an increase in fertility rate also makes a contribution to an improvement in life expectancy. But interestingly, GDP per capita is negatively correlated with life expectancy at the same significance level. This result is not what we would expect.

3.3.3 The Effects of Health Aid on Death Rate

Table 3.4 summarizes the estimated effects of health aid on dealth rate. In specification (1b), the Bhargava et al. Durbin-Watson statistic shows that our baseline model of dealth rate has an AR(1) disturbance. In specification (2), Hansen's J-statistic tells that all the instruments used to estimate the system GMM model are exogenous.

In the regressions of death rate, we find that in specification (1) and (1b) the estimate on Aid-Q may be biased downwards and those on Aid-P and Aid-C may be biased upwards from important omitted variables and endogeneity. In specification (1a) and (2), after adding the lagged dependent variable as a proxy for omitted variables and controlling for endogeneity and the Nickell bias, we find that countries with higher death rate receive more Aid-P and Aid-C but less Aid-Q, which is consistent with our findings in the regressions of child mortality and life expectancy.

Table 3.4: The Estimated Effects of Health Aid on Death Rate

	(1)	(1a)	(1b)	(2)
Model	FE-LSDV	LDV	FE: AR(1) Error	System GMM
Death Rate (L1)		0.676*** (9.91)		1.016*** (38.63)
Aid-Q (L1)	-0.00620 (-0.90)	0.00206 (0.72)	0.00245 (1.05)	0.00580 (1.37)
Aid-P (L1)	-0.000713 (-0.19)	-0.00300 (-1.27)	-0.00135 (-0.70)	-0.000209 (-0.07)
Aid-C (L1)	0.000387 (0.14)	-0.00519** (-2.62)	0.000330 (0.17)	-0.00935** (-2.09)
GDP per capita (L1)	0.0149 (0.39)	0.0165 (0.78)	0.0492 (1.10)	0.0440*** (3.77)
GDP Growth (L1)	0.00823 (1.16)	0.00000954 (0.00)	0.00382 (1.51)	-0.0203*** (-3.32)
Total Pop. (L1)	-0.727*** (-4.02)	-0.152 (-1.09)	-0.105* (-1.88)	0.000580 (0.11)
Pop. Growth (L1)	-0.0189 (-0.99)	-0.00138 (-0.09)	0.00378 (0.67)	0.000164 (0.01)
Fertility Rate (L1)	0.378* (1.67)	0.100 (0.78)	0.562*** (3.40)	-0.142* (-1.94)
Birth Rate (L1)	-0.456* (-1.84)	-0.331*** (-3.18)	-0.435*** (-2.86)	0.0825 (0.93)
Clean Water (L1)	0.00212 (0.02)	-0.0107 (-0.11)	-0.377* (-1.72)	-0.0718 (-1.31)
Sanitation (L1)	-0.203** (-2.39)	-0.0880* (-1.92)	0.0868 (0.66)	-0.00354 (-0.26)
Constant	16.70*** (5.63)	4.792* (1.94)	0.152*** (5.72)	-0.184 (-0.56)
Observation	358	358	255	358
Adj. R^2	0.979	0.995		
Bhargava et al. DW			0.645	
# of IVs				132
AB-AR(1) (P-value)				0.720
AB-AR(1) (P-value)				0.617
Hansen's J (P-value)				0.986

Note: i) Both country and time fixed effects are included.
ii) * p < 0.1; ** p < 0.05; *** p < 0.01, iii) T-statistics in parenthesis

We take a careful look at the estimated coefficient on Aid-C. In specification (1a), it is negative and statistically significant at the 1% level of significance. In specification (2), it is still negative and statistically significant at the 1% level of significance. Its magnitude, -0.00935, is almost twice as much as that in specification (1a). This shows that the estimates on Aid-C in other specifications may be biased upwards because the unexplained components of death rate is positively correlated with Aid-C. This positive endogeneity suggests that countries with higher death rate receive more Aid-C. In specification (2), therefore, the system GMM estimator eliminates the upward bias from the endogeneity of Aid-C.

In specification (2), the estimated coefficient, -0.00935, can be interpreted as that, *ceteris paribus,* a 1% increase in Aid-C is significantly reduces death rate by 0.00935%.

According to the CIA World Fact Book, South Africa has the highest crude death rate. In our data, their death rate was, on average, 15.1 per 1,000 persons for the period from 2005 to 2007. During the previous period, they received only 12 cents of health aid per capital for the cost reduction of health care. If it had increased up to a dollar by 746.57%, then their crude death rate would have decreased from 15.1 to 14.0 by 6.98%.

The estimated coefficient on GDP per capita is positive but statistically insignificant in specification (1). But this coefficient may be downward-biased because of a negative correlation between the unobserved components of death rate and GDP per capita. This negative correlation reflects that countries with higher death rate are likely to have lower GDP per capita. In specification (2), the coefficient on GDP per capita is still positive and turns to be highly statistically significant at the 1% level of significance. Moreover, the magnitude of the effect of GDP per capita on death rate is more than three times bigger than that in specification (1). In this case, the system GMM estimator removes the downward bias from the endogeneity of GDP per capita. This may reflect the fact that countries with higher income typically have a much higher proportion of older people who are more likely to die. Other control variables generally have their expected sign. In specification (1a), in particular, sanitation is shown to be beneficial in significantly reducing death rate. Its magnitude is bigger than that of Aid-C, thereby showing that sanitation is the most beneficial factor on reducing death rate in this analysis.

3.4 CONCLUSION

Most empirical studies on foreign aid have focused on the relationship between aid and growth. But they have failed to find robust evidence that foreign aid promotes economic growth. Meanwhile, the donor communty has paid more attention to global health challenges and has increased their funding for global health. Keeping up with the trend in foreign aid, empirical economists strive to find evidence of the relationship between health aid and public health. Unfortunately, past studies has not reached systematic evidence on

the effects of health aid on health outcomes. It is too soon for policymakers and scholars to decide that health aid has no effect on health outcomes. We believe that heterogeneity in health aid is the key to solving this problem.

In this paper, we primarily examine our hypothesis that there is heterogeneity in health aid, that is, different types of health aid work differently for public health in aid-recipient countries. In order to test our hypothesis, we first disaggregate health aid per capita data into three policy options: health aid per capita for improving the quality of health care, health aid per capita for providing preventive care and health aid per capita for reducing the cost of health care. Then, we empirically examine the effects of disaggregated health aid on three different health indicators: child mortality, life expectancy and death rate. Based on our hypothesis, we expect that three types of health aid operate differently on three health indicators and there is a particular type of health aid that excels at improving a particular type of health indicators.

A variety of panel data regression models are estimated in this analysis because alternative models often yield different regression results. In a sample of 119 aid-recipient countries from 1975 and 2010, we find supporting evidence for our hypothesis of heterogeneity in health aid. We basically ascertain that three types of health aid work differently for each health indicator. We find no statistically significant evidence supporting the beneficial effects of health aid on reducing child mortality. In contrast, we find that an improvement in life expectancy and a reduction in death rate are driven mostly by Aid-C. These results provide obvious policy implications to policymakers. If the main health target is to increase life expectancy or to reduce death rate, more Aid-C should be provided to achieve the target.

Also, we find empirical evidence showing that there is heterogeneity in the allocation of health aid. More Aid-Q is allocated to countries with better health status, whereas countries with worse health status receive more Aid-P and Aid-C but less Aid-Q. In other

words, Aid-P and Aid-C are allocated by the needs of the recipients, while Aid-Q goes to countries where it can be more effective.

3.5 APPENDICES

Table 3.5: List of Aid-Recipient Countries in Analysis

Total 119 Aid-Recipient Countries				
Afghanistan	Comoros	India	Mauritania	Suriname
Angola	Costa Rica	Iran	Mauritius	Swaziland
Albania	Cuba	Iraq	Malawi	Syrian Arab Rep.
Argentina	Djibouti	Jamaica	Malaysia	Chad
Armenia	Dominican Rep.	Jordan	Namibia	Togo
Azerbaijan	Algeria	Kazakhstan	Niger	Thailand
Burundi	Ecuador	Kenya	Nigeria	Tajikistan
Benin	Egypt	Kyrgyzstan	Nicaragua	Turkmenistan
Burkina Faso	Eritrea	Cambodia	Nepal	Timor-Leste
Banglasdesh	Ethiopia	Laos	Pakistan	Trinidad & Tobago
Bosnia & Herzegovina	Fiji	Lebanon	Panama	Tunisia
Belarus	Gabon	Liberia	Peru	Turkey
Bolivia	Georgia	Sri Lanka	Philippines	Tanzania
Brazil	Ghana	Lesotho	Papua New Guinea	Uganda
Bhutan	Guinea	Morocco	North Korea	Ukraine
Botswana	Gambia	Moldova	Paraguay	Uruguay
Central African Rep.	Guinea-Bissau	Madagascar	Rwanda	Uzbekistan
Chile	Equatorial Guinea	Mexico	Sudan	Venezuela
China	Guatemala	Macedonia	Senegal	Viet Nam
Côte d'lvoire	Guyana	Mali	Soloman Islands	Yemen
Cameroon	Honduras	Myanmar	Sierra Leone	South Africa
DR Congo	Croatia	Montenegro	El Salvador	Zambia
Congo	Haiti	Mongolia	Somalia	Zimbabwe
Columbia	Indonesia	Mozambique	Serbia	

Table 3.6: Disaggregation of Health Aid According to the AidData Sector Coding Scheme

Purpose Code	Description	Policy Option
121 Health, General		
12110	Health Policy & Administrative Management	
01	Health Policy & Administrative Management	
02	Health Sector Policy, Planning and Programs	
03	Institution Capacity Building, Health General	Aid-Q
04	Aid to Health Ministries, Public Health Administration	
05	Medical Insurance Programs	
12181	Medical Education/Training	
01	All Medical Education/Traning Activities	Aid-Q
12182	Medical Research	
01	All General Medical Research Activities	Aid-Q
12191	Medical Services	
01	Medical Services, Activitiy Unspecified	
02	Laboratories	
03	Specialized Clinics and Hospitals	
04	Specialized Medical Equipment and Supplies	
05	Ambulances	Aid-C
06	Dental Services	
07	Mental Health Care	
08	Control of Non-infectious Diseases	
09	Drug and Substance Abuse Control and Counseling	
122 Basic Health		
12220	Basic Health Care	
01	Basic Health Care, Activity Unspecified	
02	Basic/Primary Health Care Programs	Aid-C
03	Paramedical and Nursing Care Programs	
04	Supply of Drugs, Medicines and Vaccines (Basic)	
12230	Basic Health Infrastructure	
01	Basic Health Infrastructure, Activity Unspecified	
02	Basic Hospitals, Clinics and Dispensaries	Aid-Q
03	Basic Health Medical Equipment and Supplies	
12240	Basic Nutrition	
01	Basic Nutrition, Activity Unspecified	
02	Direct Feeding Programs	
03	Monitoring of Nutritional Status	
04	Provision of Nutrients	Aid-P
05	Nutrition and Food Hygiene Education	
06	Household Food Security	
12250	Infectious & Parasitic Disease Control	
01	All Prevention and Control Activities	
02	Other Infectious and Parasitic Disease Control	
03	Malaria Control	
04	Tuberculosis Control	Aid-P
05	Helminthiasis	
06	Polio	
07	Acute Respiratory Infections	
12261	Health Education	
01	Health Education, Activity Unspecified	
02	Information, Education and Training	Aid-P
03	Public Health and Awareness Campaigns	
12281	Health Personnel Development	
01	All Staff Traning, Basic Health Care Services	Aid-Q

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