Explaining Variation in SES-Health Gradients:

Evidence from Uganda

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INTRODUCTION

Poverty and Health

Repeated cross country studies of life expectancy and GDP/capita reveal a strong and complex correlation between health and economic performance that is stronger among low income countries and weaker among high income countries (Preston 1975). Indeed, it is possible to conclude that among the set of wealthy countries, higher GDP/capita is not correlated with better health. Yet among lower and middle income countries wealthier is healthier (Pritchett and Summers 1996).

The less steep health wealth gradient among high income countries that is seen in cross-country studies does not apply to individual health at an intra-country level. There appear to be socio-economic gradients in health between households that do not reliably weaken as countries gain in wealth or as the burden of disease shifts from acute infections to chronic disabilities (Marmot 2006). Social epidemiology has made great strides in exploring patterns in the socioeconomic gradient in health in high income countries and there are many excellent reviews of this literature(Marmot 2006; Starfield 2007).

Analogous studies in lower income countries have used household health surveys and data on household asset ownership to reveal a general pattern of intra-country health gradients by income (Victora, Wagstaff et al. 2003). The most common approach is to use household data representative of the entire country to produce one estimate of a nation's health gradient at a single point in time. There have been several studies which have tracked longitudinal progress of country level socioeconomic health gradients over time (Wagstaff 2002; Sastry 2004 ; Houweling, Kunst et al. 2006; Vapattanawong, Hogan et al. 2007). With notable exceptions, few have examined spatial patterns in socioeconomic gradients at the district or province level (Zere and McIntyre 2003; Hosseinpoor, Mohammad et al. 2005). Progress in understanding socioeconomic gradients in health will be slow as long as demographers persist in examining socioeconomic health gradients one country at a time and one year at a time (Starfield 2007).

Existing theoretical frameworks have been designed to explain patterns in high income countries and are ill suited to explain the genesis and evolution of a population's socioeconomic gradient over several generations. Our objectives in this paper are to develop and begin testing a general theory of how household level gradients evolve as an economy develops. Our theory will predict that steeper economic gradients in health are a byproduct of the earliest stage of the introduction of modern health interventions at the very start of economic development. The theory predicts an inverted U shaped relationship between the steepness of the health gradient and the level of economic achievement with poorest areas exhibiting greater uniformity of high death rates undifferentiated by household SES. With development, opportunities arise for richer households to purchase better health and the gradient increases. Finally the most developed areas are able to devise and sustain a socioeconomic safety net that lowers the steepness of the gradient.

Geographical heterogeneity in economic prosperity is a well known feature of development with the urban areas typically leading rural areas in development. Thus our theory suggests that spatial heterogeneity in economic performance should give rise to spatial heterogeneity in the socioeconomic gradient in health. Data from the Uganda National Household Survey of 2000 are used to measure socioeconomic differentials in household level measurements of childhood anthropometry. The Ugandan data show limited spatial heterogeneity in the economic gradient and no systematic geographic pattern.

BACKGROUND

Poverty and health: Within-country analysis

Household level studies confirm a socioeconomic gradient that is most visible when inspecting differences in child survival by household schooling attainments (Orubuloye and Caldwell 1975; Caldwell 1986; Lindenbaum 1990)and by asset scores (Gwatkin 2000). There is conflicting evidence about how urbanicity moderates the effects parental education on child health. Data from 7 African countries, 5 Asian, 3 Latin American data sets failed to support a consistent interaction effect between mothers' schooling and urbanicity on child survival

(Mensch, Lentzner et al. 1985). Other studies have found evidence of complementarity between urbanicity and maternal schooling in child survival in Brazil(Sastry 1997), but substitution in Colombia (Rosenzweig and Schultz 1982) In other words the protective effect of maternal schooling was accentuated by urbanicity in Brazil, but diminished by urbanicity in Colombia.

Studies of how place alters the protective effect of urbanicity have been more consistent in showing that urban areas have larger wealth related differentials in child health. This result was first demonstrated in a set of 2 South Asian, 4 Sub Saharan African, and 4 Latin American DHS datasets that measured household wealth using a principal components based asset index (Menon, Ruel et al. 2000). Later Fotso focused on 15 countries in sub-Saharan Africa, and using a multilevel modeling strategy again found the asset index–health gradient to be greater among children in urban areas in only a subset of countries studied, reflecting the impact of heterogeneity on the income-health relationship (Fotso 2006). One of the limitations of the prior research is that it is based on surveys that lack extensive data on household economic status and employ an asset score as a proxy, despite evidence that the choice of assets in the asset score can have important effects on the size of the health gradients attributed to it (Houweling, Kunst et al. 2003). Another limitation is that the analyses are typically carried out at the level of the country, with district or province level studies focusing only on showing difference in the overall level of health across the districts but not on showing differences in the health gradient across districts.

The Role of Economic Development in the Evolution of Health Gradients

Although the general pattern of the Preston curve makes one optimistic that average health will improve with economic growth in low income countries, what can one say about the prospects for lowering disparities in health? Wagstaff offers a theoretical prediction that health gradients will worsen with economic growth in either of the following circumstances: 1) Growth is accompanied by more income inequality; 2) The household demand for discretionary spending on health becomes more intense as household income rises (Wagstaff 2002). Many economists

believe that both of these conditions are typical of low and middle income countries[1]. Victora and colleauges theorize that a health program that introduces high quality curative services at fixed sites is likely to preferentially attract middle and higher income patients at the outset, because of their higher savvy and income related intensity in the demand for health services (Victora, Vaughan et al. 2000). This is cited as one possible explanation for the failure of one clinic-based intervention known as IMCI to reach the poor (Gwatkin 2006).

Wagstaff shows in cross sectional analysis of data from 42 developing countries that concentration indices of child health demonstrate greater inequality with higher GDP/Capita (Wagstaff 2002). An alternative measure of inequality in child health that combines both the between group variance and the across group variance derived from a beta-binomial regression of individual child survival in 50 countries shows a contrasting pattern such that higher GDP/Capita is associate with a reduction in the overall inequality (but a possible rise in across group inequality) as GDP/capita rises (Gakidou and King 2002). Since both papers only included developing countries, neither can reveal whether continued economic growth will inevitably widen socioeconomic gradients in health. Indeed, data from the Indonesia suggests that economic growth without widened gradients is possible (Houweling, Kunst et al. 2006).

Statement of the problem

Almost everything we know about health gradients is based on studies at the level of an entire country. This high degree of aggregation has the advantage of a more precise measurement of the gradient , but it inhibits attempts to account for the variation in the gradient. The few

1 The Kuznets hypothesis Kuznets, S. (1955). "Economic Growth and Income Inequality." American Economic Review **45**: 1-28 that income inequality initially rises as subgroups participate in the development process and are later joined by more widespread participation of all members of society has borne up to repeated analysis of country cross-sectional data Ahluwia, M. (1976). "Inequality, Poverty and Developoment." Journal of Development Economics **6**: 307-342, Deininger, K. and L. Squire (1996). "A New Data Set Measuring Income Inequality." World Bank Economic Review **10**: 565-591, but is not inevitable. The relationship between GDP/capita and health spending as a share of GDP on the national level has been explained as rooted in the complementarity of better health to the enjoyment of higher wealth. The richer people are, the more rational it is to spend ever increasing shares of that wealth on being healthy, because health makes spending all of the other money so much more enjoyable Hall, R. E. and C. I. Jones (2004). "The Value of Life and the Rise in Health Spending." NBER Working Papers **10737**.

within-country studies of the gradient have shown geographic heterogeneity, but we lack a coherent account of why this would be the case. Increasingly, there is a need to understand health gradients in a manner that is as localized as possible in order to determine how local policies impact gradients. Our study of Uganda will examine income-health gradient at district level. Our two hypotheses are

1) That there will be geographical heterogeneity in the income-health gradient as a result of political, administrative, socio-cultural and economic variations across the country.
2) That higher district level income will increase the size of health disparities.

CONCEPTUAL FRAMEWORK

Another Inverted U: Health Gradients over the Course of Economic Development

We hypothesize that in the multi-century process of economic development the economic-health gradient will show three phases. In the first phase, the rich and poor are similarly affected by intermittent crisis mortality, and socioeconomic gradients, though present, are small by today's standards. In this early phase, the most effective health interventions are non-rival, non-excludable interventions in public health that are of more benefit to the poor than the rich. As prosperity emerges, and medical care actually becomes effective, households with better incomes are able to access better services and achieve better health status, representing the phase with strong correlations between income and health. The last phase, that a few highly developed countries have attained, is one in which better health will mostly result from targeted social interventions intended to narrow the gradient.

In our conceptual model we partition the casual forces improving population health into 2 components: 1) Environmental and governmental factors 2) Household factors (Mokyr 1993). Environmental causes include physical environmental features like swamps, stagnant water and

animal reservoirs of zootoxic disease. The concept of "environment" can be expanded to include features of the social and cultural landscape that can be gradually altered by government policies and economic development. For example, social norms and cultural traditions can alter the frequency and spread of exposure epidemics by affecting the adoption rate of hygienic practices, the volume of foreign trade and migration. The physical and social environment responds to government decisions over the long run. Governmental decisions can impact public health thought the enactment of regulations improving sanitation, housing, eater, and food safety. Governments can also take actions like means tested health insurance that are intended to lower socioeconomic gradients in health. Finally households can determine their health through behavioral choices that alter personal risk and by acquiring resources that can be traded to lower mortality risks. This can be as simple as earning enough money to pay for a doctor visit or health insurance premiums. It can be as complex as relocating to a healthier neighborhood or using social connections to improve health knowledge.

These causal forces lie distal to the final causes of death and disease that epidemiological data disclose[2] and imply three different effects on the evolution of the socioeconomic gradient in health over time as economic development progresses. Figure 1a and b display schematically those general patterns. We now discuss why these general patterns are postulated to occur.

Environmental and Governmental Effects on Health

Crisis mortality in Europe took the pattern of epidemics that would have been sometimes rooted in a preceding famine or pestilence that lowered food availability. Indeed Galloway's study of parish mortality registers from Rouen reveals a strong correlation between grain prices and mortality 1 and 2 years later. However, Galloway's data contain very important clues about

[2] Tchaikovsky's death from cholera can be attributed in part to St. Petersburg's waterfront location and participation in global trade that led to the 1893 cholera epidemic as well as government ineptitude at public health surveillance and control. Tchaikovsky's controversial (some say suicidal) decision to drink tap water at the height of the epidemic also played a role. Tchaikovsky despite his privileges succumbed to epidemic cholera and pathophysiological

the nature of socioeconomic gradients in this era. Rouen was partitioned into arrondisments and data on the social class of the residents of each arrondisment permits class-specific estimation of the response of each group to a rise in grain prices(Galloway 1986). Galloway's remarkable finding was that the burden of crisis mortality was nearly equally distributed among upper and lower classes. The rate ratio for mortality between upper and lower class children was 1.4 overall. More importantly, the regression coefficient connecting grain prices to child mortality was not significantly different between the poorest compared to the wealthiest children in medieval Rouen (Galloway 1986). Social differentials in adult mortality were also relatively small with life expectancy at age 20 estimated at 41.2 for a noblewoman and 38.1 for the wife of a day labourer in Paris in the early 1860s and a six year life expectancy gap when comparing a nobleman to a day labourer(Blum, Houdaille et al. 1990). In Ipswich, English labourers aged 20-44 experienced a crude mortality rate of 7.9% while elites suffered a relatively similar 6.4% mortality in the mid 1800s (Razzell and Spence 2006).

Emerging evidence, thus suggests that socioeconomic gradients were present in Europe 200 years ago but appear to have intensified later during industrialization and economic development. Although the basic physiology of economic deprivation leading to nutritional differentials was surely active in medieval Europe, patterns of universal and repetitive contagious exposure may have diminished the protective effects of wealth. This pattern of social leveling in crisis mortality would be consistent with the interpretation of crisis mortality as caused by an intersection of highly contagious disease agents and a vertically integrated society with significant rates of direct contact between lower and upper classes. A substantial amount of this contact may have occurred in the form of domestic servitude.

In England, a long succession of public health legislation was initiated beginning around 1848 with the Public Health Act of 1848 and followed successively by the 1853 Metropolitan

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accounts of cholera cannot tell us the ultimate causes of his death. Environment, government, and personal factors all were complicit.

Smoke Nuisance Abatement Act, the 1875 Public Health Act, a series of Adulteration of Foods Acts in the 1870s, the 1890 Housing Act, the 1899 Dairies Cowsheds and Milkshop Order, and the 1907 Notification of Births Acts (Szreter 1988). England is not unique in enacting public health protection early in the process of economic development. Part of Preston's major finding in his seminal paper was that the entire cross-country relationship between GDP and Mortality had shifted between the 1930s and the 1960s (Preston 1975) providing evidence that public health technology had accounting for 75-90% of the uplift in how healthy a population could be with a given amount of GDP. Acemoglu and Johnson chronicle the introduction of public health technology in various developing countries subsequent to 1940 (Acemoglu and Johnson 2006).

Many public health measures have been shown stronger effects on the poor than the rich. For example the introduction of measles vaccination in Bangladesh lowered mortality among the lowest asset quintile more than the higher asset quintile and reduced the size of the socioeconomic gradient in mortality (Bishai, Suzuki et al. 2002; Bishai, Koenig et al. 2003). Similarly Vitamin A distribution in Nepal reduced the size of caste related differentials in mortality (Bishai, Kumar et al. 2003). Burstom et al. show that improved water and sanitation in Sweden lowered socioeconomic gradients in diarrhea related mortality between 1878 and 1925 (Burstrom, Macassa et al. 2005).

These considerations motivate the pattern shown in Figure 1a where governmental and environmental factors first lower the mortality of the poor during the process of development and then later trickle up to have significant effects on the rich. These trickle up effects are postulated to occur both through the direct benefit of the rich from the public health improvements, but also due to lower contagious exposure from the poor. The net effect of the differential timing in the response of rich and poor households is to first reduce the socioeconomic gradient as the health of the poor responds more to public health interventions than the health of the rich.

Household Effects on Equity in Health

Figure 1b shows the postulated effect of economic development on household mediated trends in population health. As noted by Victora and Vaughn, the introduction of personal health services may benefit the middle and upper class before trickling down to the poor (Victora, Vaughan et al. 2000). Household demand for health services will express itself through care-seeking and through the ability to purchase improved housing, water supplies and nutrients. Each of these effects is positively related to income so that wealthier households will benefit before poorer households. This leads to pattern shown by the blue dotted line in Figure 1b in which health gradients first rise and then fall.

Net Effects

Figure 1a and Figure 1b predict opposite effects of economic growth on the socioeconomic gradient in health. Environmental and government factors are posited to lead to a reduction and then a rise in the gradient, while household factors are posited to lead to the opposite. What is the net prediction? In the health transition, one might anticipate with Szreter that public health and environmental factors might be of greater importance early on when the principal burden of disease is infectious. Later, when the burden of disease shifts to chronic, behaviorally mediated diseases, household effects may predominate. The cross-country comparison of health gradients in low income countries by Wagstaff has clearly shown rising gradients with economic development, suggesting that most of the developing world today is being subjected to a burden of disease that is more strongly under the control of household level factors that widen the gradient as countries improve GDP/Capita.

The theory's prediction for a single country would be that gradients would be smaller in poor areas than in rich areas to the extent that household factors have higher importance in the determination of health. We now turn to a detailed consideration of the case of Uganda.

Uganda Context

Uganda is a country in East Africa with a population of 27 million. It has marked heterogeneity within regions and districts, not only because of high ethnic variations (over 50 tribes) or climatic and topological differences that favor different crop farming activities, but also because of differential economic successes and administrative management that resulted from decentralization. During the 1990s health sector reform efforts focused on improving access to services and reducing the average distance traveled to within 5 km, in part recognition of the need to reduce the burden of the population that mostly relied upon walking to services (Hutchinson, Habte et al. 1999). Renovation of previously existing infrastructure and rebuilding to expand and extend basic services all over the country were done (Hutchinson, Habte et al. 1999). There was also a shift in health investment away from the more expensive curative services to more preventive and primary health care, which saw the establishment of key programs. Disease-specific programs (with later introduction of more integrated programs) such as the malaria and tuberculosis (TB) control programs that targeted major causes of mortality were reactivated. New programs included the AIDS control Program (ACP) in 1986, the Uganda Essential Drugs Management Program (UEDMP) in 1986 to ensure distribution of basic drugs, and the Uganda National Expanded Program on Immunization (UNEPI) in 1987. Successes by then end of the 1990s included a 400% increase (from 1972) in infrastructure, including a 49% increase in health facilities within a 5 km radius (by 1993). Universal Primary Education had been introduced, and 49.6% of rural areas had safe water sources . However, by the late 1990s ,strong regional differences remained and they were reflected in the continued inter- and intra-regional health inequalities seen in the quality of services (Gidwani 2005).

In addition, while the government now owned two thirds of all health facilities, they provided less than two thirds of care, most being taken up by private services. This was mainly a result of lack of improvements in the quality of care. Health workers remained few and there had been no significant changes towards the use of modern health care practices. User fees had been introduced in 1989 to provide additional funding to improve the quality and enable the

government to direct the scanty health resources to more critical areas of health; however the revenues generated were insufficient, non-uniform and most importantly had not been used to improve the quality of care (Tashobya, Chao et al. 2001). Towards the end of the 1990s the decentralization of the health sector had began, partly to address the problems but also to improve social participation.

METHODS

Data

Our analysis was performed on data drawn from the 1999/2000 Uganda National Household Survey (UNHS). Our focus was on the socio-economic module of the survey which collected information on demographics, education, health and economic activity. The survey was a nationally representative sample of 10,696 households (22% in urban areas) with 57,529 household members of whom 9,524 (16.6%) were children aged under five years.

Survey methods

The survey was conducted over a period of one year to capture predictable risks of vulnerability related to seasonal or climate variations. The sampling frames were based on the 1991 population census and the 1992/93 Integrated Household Survey (IHS). Two- and three-stage (in areas with no enumeration area frame) sampling designs were used. In the two stage design, the first stage was at the district level and the second at the household level. In the three stage design, the first stage was at the parish level, the second at the village level and the third at the household level. 1,100 sampling units were selected both by simple random sampling from the IHS frame and probability proportional to number of households from the census frame. Ten households were selected from listed households within each sampling unit. To obtain homogeneous strata within these areas, stratification was done at the national, district and household levels. At the national level, stratification was done to yield four regions: central, eastern, northern and western. At the district level (with the exception of Kampala - the capital

city), stratification was done to yield three main areas: urban, other urban and rural areas. At the household level, stratification was done based on farming activity levels.

Data for analysis

We used the analytical software package STATA SE 9.2 supplemented by the GLLAMM commands for multilevel analysis (Rabe-Hesketh and Skrondal 2002; Skrondal and Rabe-Hesketh 2004). We initially stratified the households into 55 districts and further into whether the primary sampling unit (PSU) was classified as urban, semi-urban or rural. This generated a framework of 3 possible zones for each district, but many districts did not have urban or semi-urban zones and Kampala did not have a rural zone.

We excluded any zones where less than 20 observations on the height of a child under 5 were available. This led to a sample of 15 urban, 39 rural, and 3 semi-urban zones. For 2 districts we were able to study 3 zones (urban, rural, and semi-urban). For 10 districts we were able to study urban and rural zones. Three districts had only urban zones, and 27 had only rural zones.

We computed weight for age and height for age z-score using the reference standards from the WHO and the WHO's "igrowup" program (World Health Organization 2005). Children whose anthropometric information was missing or flagged as extreme (i.e. biologically implausible) z-scores (Weight-for-age z-score < -6 or > 5 and Length/height-for-age z-score < -6 or > 6) were denoted as missing. The selected sample was tested for comparability with the original data sample using the analysis of variance (ANOVA) chi square comparisons. Continuous variables were compared using the two sided t-test, while the z-test (prtesti) was used for comparison of the continuous variables, and p values were determined. The significance level was determined to be at alpha = 0.05.

Analysis

We conducted a multilevel analysis as shown in the equations below:

[1] Height Age_{ij} $=C+\beta_j Log\ Income_{ij}+\beta_1 Log\ Income + \beta_2 X_{ij}+\mu_j+\varepsilon_{ij}$

[2] $\beta_j= \varphi_j$

Where 'i' subscripts children
'j' subscripts zones defined as urban, semi-urban, or rural samples from a given district
β_j is a random coefficient that on log income differs for each PSU as shown in Eq [2]
β_1 is a universally shared coefficient on log income for the whole sample
β_2 is a vector of coefficients on household and child level covariates
X_{ij} is a vector of household and child level covariates
μ_j is the contribution of unobservables to child height from the j'th community
ε_{ij} is an individual specific error term
φ_j is the contribution of unobservables to the income-height slope term from the PSU j

Both the gllamm command and the xtmixed commands in Stata were used for estimation. We estimate 3 different versions of the above model. In the first version we impose the restriction that $\beta_1=0$, thereby fitting the random coefficient φ_j based on within cluster variation only. In the second version we estimate the system of [1] and [2] exactly as written. In the third version we impose the restriction that $\beta_j=0$. These successive restrictions were tested using likelihood ratio tests. The random coefficients derived from model 2 were obtained for further analysis to examine their spatial distribution in Uganda and to assess their correlation with district level averages of log income.

The dependent variable is height age z score among children under five years (age 0-59 months). The primary independent variable of interest was the natural logarithm of total household income. The total household income was determined as the total sum of income in cash and in kind from all household members engaged in economic activity. This included household enterprise and property incomes, and benefits, wages and salaries. We adjusted for potential confounding effects related to stunting, specifically child's gender, father's education level, number of co-residing female adults, number in the house, and marital status of parents.

RESULTS

We had available data on 10,461 (97.8%) households with 56, 232 (97.8%) household members and 9,059 (16.2%) children. There were no significant differences between the original and analytical samples as a result of our selection criteria. Table 2 shows the results of the regression analysis. All models showed a positive relationship between log income and height such that one log shilling would raise child height by 0.126 to 0.141 standard deviations. Father's years of schooling had positive effects that were statistically significant. In separate models we found evidence of collinearity between mother's years of schooling and father's years of schooling with stronger effects for fathers, so we retained only fathers schooling. There were no gender effects. Height for age was 0.36 to 0.41 standard deviations smaller in rural as opposed to urban areas.

The likelihood ratio tests rejected the first version of the model that was shown in column 1 of Table 2, but the test could not distinguish between model2 and model 3. This suggests that the estimates of income effects on height are improved when the between community variation is allowed to express itself. In model 2, the average community contribution to the slope connecting log income and height age was near 0 (at -0.0003) but the community contributions ranged from -0.078 to 0.080. This means that in the community with the shallowest economic gradient in health the effect of income was as small as 0.048 (=0.126 minus 0.078) and in the community with the steepest gradient, the effect of income on height age was as high as 0.206 (=0.126 plus 0.080). Thus where a child lived in Uganda could reduce or magnify the effects of income on their health by a factor of just over 50%. The intracluster correlation coefficient in the third column shows that which zone a child lived in accounted for only 3% of the overall variance in child height with most of the determination of child height being consigned to the individual and household factors.

Figure 2 displays a histogram showing that the range of slopes runs from 0.048 to 0.206 and shows little evidence of a central tendency. The heights of the bars in the histogram are

proportional to the number of children exposed to each health gradient and offer reassurance that the splay in the estimated gradients is not simply due to imprecision from zones with fewer children. Figure 3 offers the same histogram confined to urban zones only to show that the splay is not due solely to urban rural differences in the health gradient. The histogram is just as wide and uniform for the rural areas only (not shown).

In Figure 4 we offer a simple assessment of the hypothesis that there will be a systematic relationship between the health gradient and income. The scatter plot shows no relationship and the raw correlation is -0.03. Seeing this relationship we did not proceed to model the slopes as functionally related to cluster means of log income. Figure 5 is a diagnostic plot verifying the extent of heteroskedasticity in height age z scores.

Figure 6 verifies what prior poverty maps have shown about Uganda: that rural poverty is concentrated in the north, and higher income households are located more centrally. This pattern was originally seen in the 1991 census data and has been mapped in detail elsewhere (Okwi, Hoogeveen et al. 2005). Armed with the prior knowledge of the distribution of poverty, one can then examine the distribution of the health gradients in Figure 7. If our hypothesis 2 is correct one would expect northern zones to have smaller health gradients and central zones to have larger gradients. The map in Figure 7 shows no spatially meaningful patterns in the connection between poverty and health. The map is essentially haphazard.

Discussion

The analysis of health gradients from Uganda in 2000 showed a range of gradients among both urban and rural populations. Gradients vary by +/- 50% of the country average based on where a child resides. We found no areas where household income was uncorrelated or negatively correlated with child height.

Our first hypothesis was that there would be spatial heterogeneity in the child health gradient. This is partially confirmed. There is no absolute metric that can guide a determination of what constitutes low or high geographical variation in the size of a health gradient inside a

country. For those who expected complete uniformity, our results would indicate heterogeneity. For those who expected a 10-fold difference in gradients, our results would indicate homogeneity. Future studies of intra-country health gradients will be necessary before one can gauge how unusual the pattern of Uganda is.

Our second hypothesis was that there would be a systematic pattern in the health gradient such that steeper gradients would be associated with urbanicity and with higher area measures of income. Our analysis showed no such systematic relationships. The maps showed a haphazard scatter of gradients in space and there was no evidence of a rural-urban gap. Figure 4's scatter plot showed no systematic relationship between gradient and area-average of log income.

There are two possible explanations for the random patterns in the gradients. The simplest explanation is that the measurements remain imprecise because they are based on small samples. On average, the gradient in any given zone is being estimated based on a sample size of 106 children. If a zone lacks sufficient variation in household income--a phenomenon that is common in rural Uganda--then the gradient cannot be estimated precisely. The multilevel estimation procedure gains efficiency from using the entire sample's income-height covariance simultaneously with the covariance in a single zone, but small samples in each zone will remain a limitation. There is evidence that imprecision plays a role in our result in that the random coefficients model (model 2) and the random intercepts model (model 3) gave indistinguishable performance as judged by the LR test. This means that although there is a community specific contribution to child health, our restriction that this contribution act via household income did not improve the estimates. Enlarging the sample sizes in future studies will improve the estimate, but living standards surveys and/or surveys with objective health measures seldom have samples greater than 10,000.

Besides simple imprecision, it is possible that the determinants of the size of a local health gradient will have idiosyncratic contributions that would work against finding systematic geographical patterns. Health gradients are amenable to being intensified or reduced by local

governments and social norms. With Uganda's decentralized health system and the wide variety of ethnic backgrounds throughout the country, there may be idiosyncratic factors at the district level that interrupt the effects of geography or district economic performance. Any given district may underperform or overperform in implementing basic public health measures like vaccination and micro-nutrient distribution, and these differences may not be related to geography of economics.

In summary, we have presented a theoretical framework that predicts intra country variation in the size of health gradients due to regional differences in economic attainment and capacity to implement public health measures. Our analysis offers a glimpse of what can be learned by studying health gradients on a sub-national scale. If nothing else, the inclusion of district level effects does improve the quality of the estimate of the national economic gradient. Imprecise estimates of the gradient because of small samples within each subnational unit and low interhousehold variation will remain an obstacle. Consequently inferences must be drawn with caution. Conducting similar studies in subsequent Ugandan datasets can help to assess change over time. Similar studies in other datasets can also help to assess whether the degree of variation in the gradients observed in Uganda is unusual.

Table 1a. Baseline characteristics comparison between original and final analysis datasets at the individual level

Characteristic	Overall		Final Data		
Children under 5 years (0-59mo):					
Gender: N	9,278		9,095		
% male	50.5		50.5		
Age (months):N	9,524		9,095		
Mean (SD)	28.1 (16.8)		28.2 (16.9)		
Age categories (months) No.(%)	9,524		9,095		
0 – 5	1019 (10.7)		984 (10.8)		
6 – 23	2,815 (29.6)		2,687 (29.5)		
24 – 35	1,864 (19.6)		1,768 (19.5)		
36 – 47	1,933 (20.3)		1,850 (20.3)		
48 – 59	1,893 (19.8)		1,806 (19.9)		
Education of mother: N	9,240		8,827		
Median - No. of years in school	4		4		
None - less than 4 years (%)	45.8		46.0		
Primary (p4-p6) (%)	29.2		29.2		
Post primary (p7-s3; cert) (%)	20.9		20.7		
Secondary/post (s4-s6; cert) (%)	4.0		4.0		
Degree level (%)	0.06		0.05		
Height-for-age (haz -WHO): N	7,366		7,236		
Mean (SD)	-1.48 (1.60)		-1.48 (1.60)		
Stunting rate (%)	37.2		37.4		
Weight-for-age (waz-WHO): N	7,754		7,611		
Mean (SD)	-0.76 (1.32)		-0.76 (1.33)		
Wasted rate (%)	15.8		15.8		
Immunization	N	%	N	%	
BCG	9,219	81.3	9,048	81.3	
DPT3 completion rate	8,510	75.8	8,102	71.0	
Up to date for DPT:					
All	9,210	83.9	8,786	84.0	
Card	5,812	74.4	5,562	74.5	
Up to date for measles (9-59)	9,054	66.7	8,636	67.0	
Breastfeeding					
Ever breastfed	9,434	99.3	9,001	98.7	
Still breastfeeding by age (%)	9,361	37.1	8,931	37.6	
0 – 5 mo	1,006	98.3	989	97.8	
6 – 23 mo	2,786	76.7	2,658	77.0	
Duration excl. b/feeding (mo):N	8,631		8,220		
Mean (SD)	4.7 (3.3)		4.7(3.3)		
Reasons stopping b/feeding: N	6,159		5,327		
Child old (%)	72.3		79.6		
Mother pregnant (%)	13.8		15.4		
Mother sick (breasts) (%)	2.2		2.4		
No mother (%)	1.6		1.6		
Major Illnesses last 30dy/6mo: N	9,524		9,095		
Malaria (%)	35.0		35.0		
Respiratory (%)	7.0		7.0		
Diarrhea (%)	5.4		5.3		
Measles (%)	3.8		3.8		
AIDS (%)	0.07		0.05		

Table 1b. Baseline characteristics comparison between the original and final analysis datasets at the household and community levels

Characteristic	Overall	Final Data	p value
Community level:			
Households: N (%)	10,696 (100)	10,461 (97.8)	
# HH with child U5: n(%)	5,820 (54.4)	5,557 (53.1)	
Overall # people: N	57,529 (100)	56,232 (97.8)	
Children U5: n (%)	9,524 (16.6)	9,095 (16.2)	
Gender* - % male	49.5	49.5	NS
Household Level:			
People /House	10,696	10,461	NS (overall
Mean No. (SD)	5.4 (3.2)	5.4 (3.2)	est is 5.2 in
Range	1-46	1-46	the cty)
Children U5: n(%)	9,524 (16.6)	9,095 (16.2)	NS
Mean No. (SD)/ House	1.6 (0.8)	1.6 (0.8)	
Median	1	1	
Range	0-9	0-9	
Allincomes (USD)**			
Median	1,019,500	1,013,000	
Mean (SD)	1,751,539 (3,211,247)	1,733,693 (3,186,452)	
Range	6,000 – 88,000,000	6,000 – 88,000,000	
Main water source* : N**	10,683	10,449	NS
Safe (%)	59.0	58.7	
Distance to Services (km)			NS
Nearest school – mean (SD)	1.6 (3.8)	1.6 (3.4)	
Nearest health care – mean (SD)	4.1 (9.2)	4.0 (9.2)	
HH head characteristics:			
Number	10,696	10,461	
Gender - % male	73.7	73.7	NS
Age (years): N	10,693	10,458	NS
Median	40	40	
Mean (SD)	43.3 (16.0)	43.4 (16.0)	
Range	12-99	12-99	
Marital status: N	10,693	10,458	NS
Married (%)	67.2	67.1	
Cohabiting (%)	5.0	5.0	
Widowed (%)	13.3	13.4	
Separated (%)	7.5	7.5	
Unmarried (%)	7.0	7.0	
Duration of stay (mo): N	10,690	10,455	NS
12 months stay (%)	98.3	98.3	
Mean (SD)	11.9 (0.9)	11.9 (0.9)	
Range	0 - 12	0 - 12	
Occupations (5 commonest) **:N**	10,687	10,453	NS
Crop farming (%)	62.3	62.9	
Trading (%)	8.3	8.	
Professional (%)	6.5		
Transport (%)	4.8		
Unskilled (%)	4.5		

Table 2. Multilevel models of the height for age Z score among children in Uganda

	Height Age Z-Score		
	Random Coefficients Model Version 1	Random Coefficients Model Version 2	Random Intercept Model Version 3
Log income (Slope for Full Sample)		0.126 (0.033)***	0.129 (0.002)***
Log income (Average Community Level Slope)	0.141 (0.040)***	-0.0003 (0.04)	
Father's Education (Continuous)	0.009 (0.002)***	0.007 (0.001)***	0.007 (0.002)***
Number of Females in Household	0.031 (0.032)	0.025 (0.032)	0.024 (0.315)
Number of People in Household	-0.004 (0.008)	-0.011 (0.008)	-0.011 (0.008)
Child is Boy	0.055 (0.069)	0.077 (0.069)	0.081 (0.069)
Parents Married	0.018 (0.071)	0.016 (0.07)	0.020 (0.07)
Rural	-0.410 (0.116)***	-0.362 (-0.119)***	-0.386 (0.113)***
Semi-Urban	0.212 (0.282)	0.172 (0.281)	0.133 (0.271)
Constant	-1.416 (0.169)	-3.13 (0.473)	-3.16 (0.432)
Variance at individual level (ε_{ij})			2.460
Variance at community level			0.074
Intra cluster correlation coefficient			0.029
Number of Observations	6070	6070	6070
Number of Clusters	57	57	57
Average Children per Cluster	106	106	106
Log Likelihood	-11405.925	-11399.505	-11400.261
LR Test Of Model vs. Model to its Left		12.84 ***	1.51

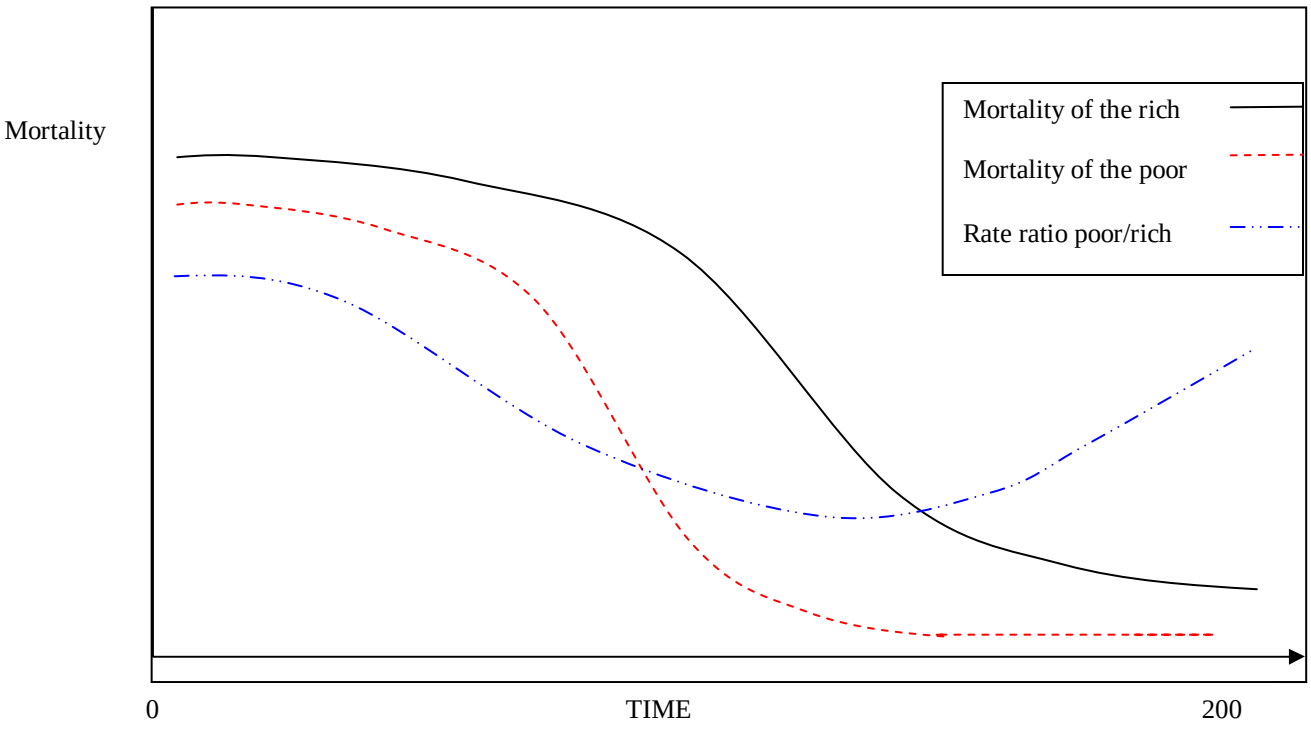

Figure 1a. Schematic time course of mortality reductions attributable to governmental and social improvements in the environment. As the environment improves the poor derive greater benefit first, followed later by the rich, in part due to their ability to benefit from the elimination of the contagious reservoir among the poor. The blue dotted line denotes the rate ratio and reflects a biphasic relationship. In which inequality first decreases and then increases. Not drawn to scale.

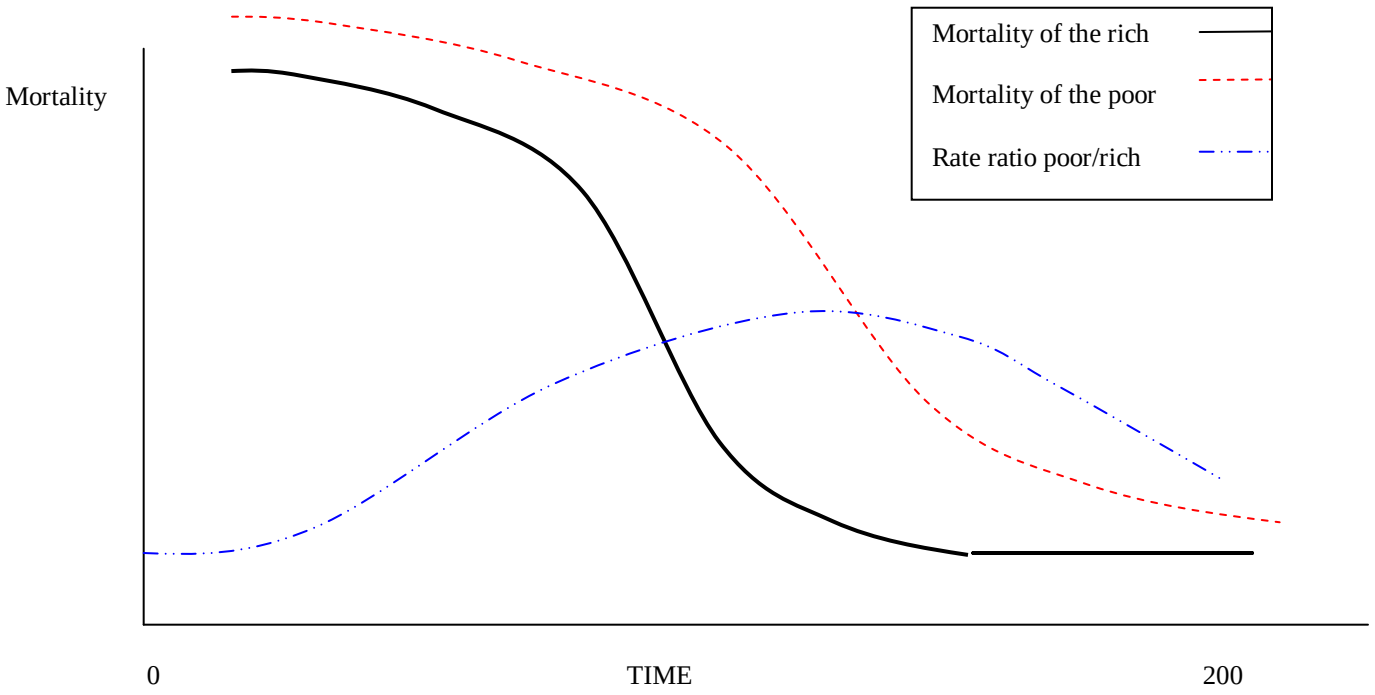

Figure 1b. Schematic time course of mortality reductions attributable to improvements in household choices. The black curve denotes the earlier response of the rich, followed later by reductions among the poor. The blue dotted line denotes the rate ratio and reflects a biphasic relationship. In which inequality first increases and then decreases.

Figure 2. Histogram of slope connecting log income to child height in 57 different clusters in Uganda from model 2.

Urban only

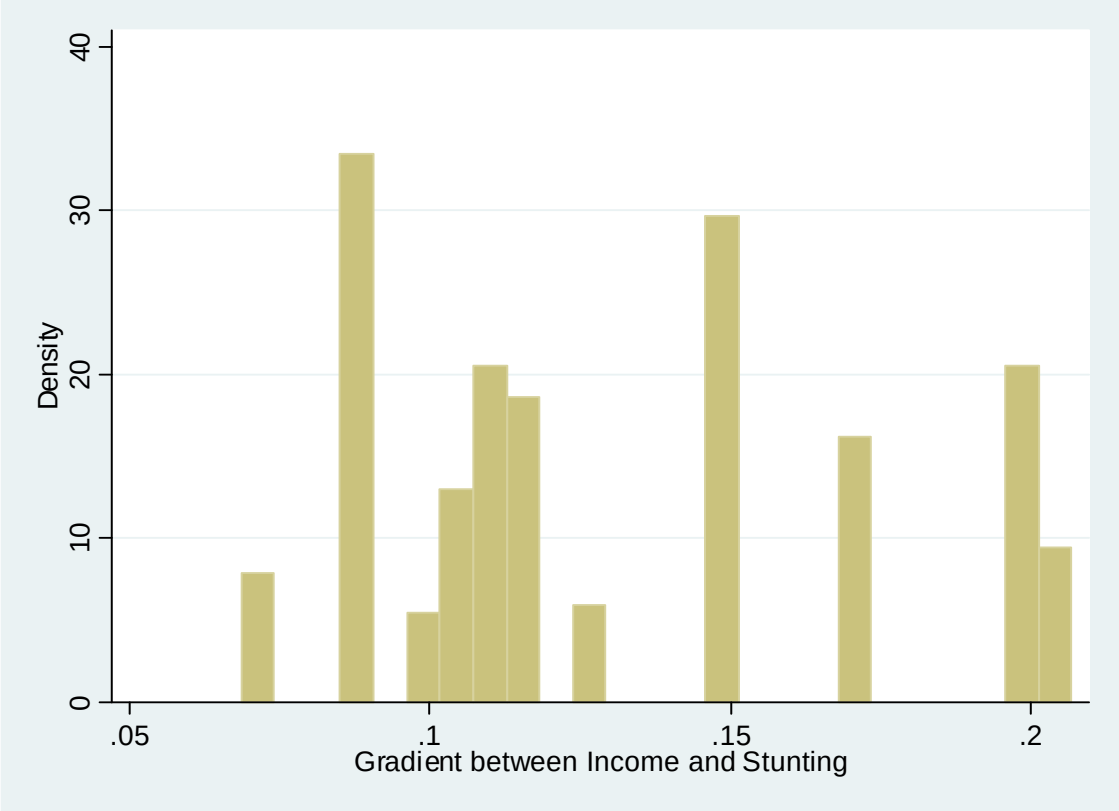

Figure 3. Histogram of slopes connecting log income to child height in 15 different urban clusters in Uganda. Height of each bar is proportional to the number of children in the sample exposed to that regime.

Figure 4. Scatter plot by cluster of the income-height gradient vs. cluster mean of household income.

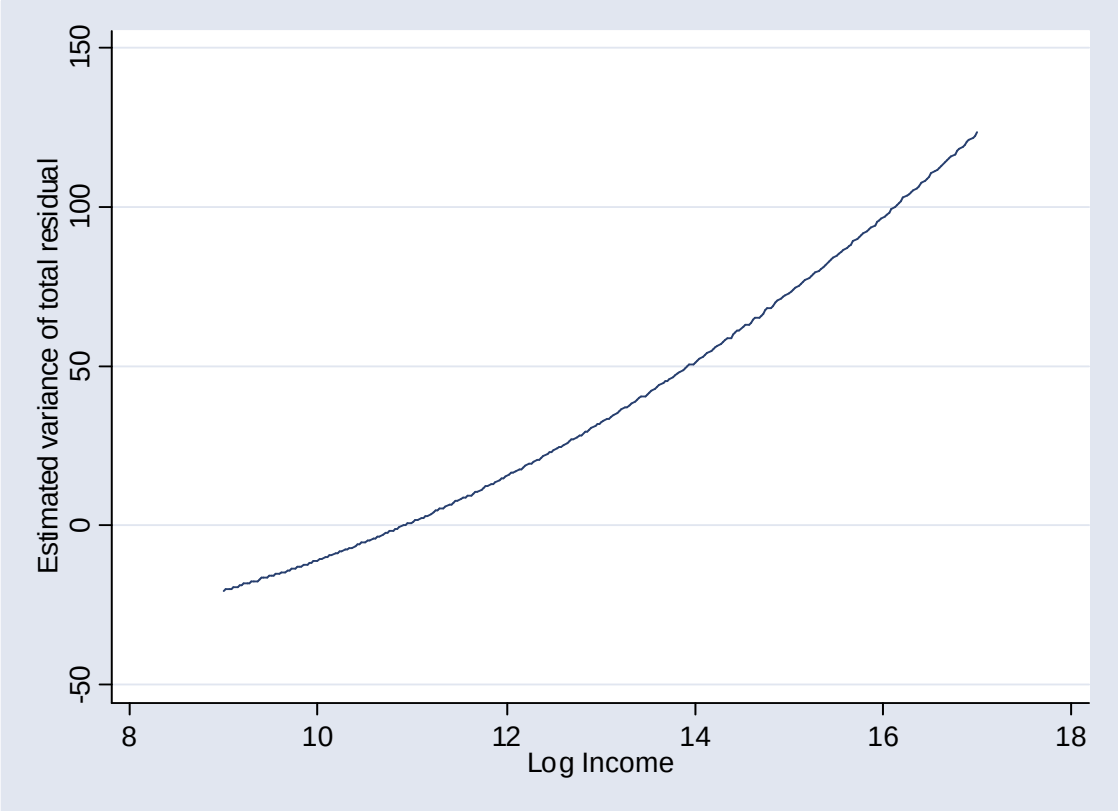

Figure 5. Graph showing how the variance of height age increases with log income based on estimated parameters from model 2. This heteroskedasticity mandates appropriate models to adjust the standard errors

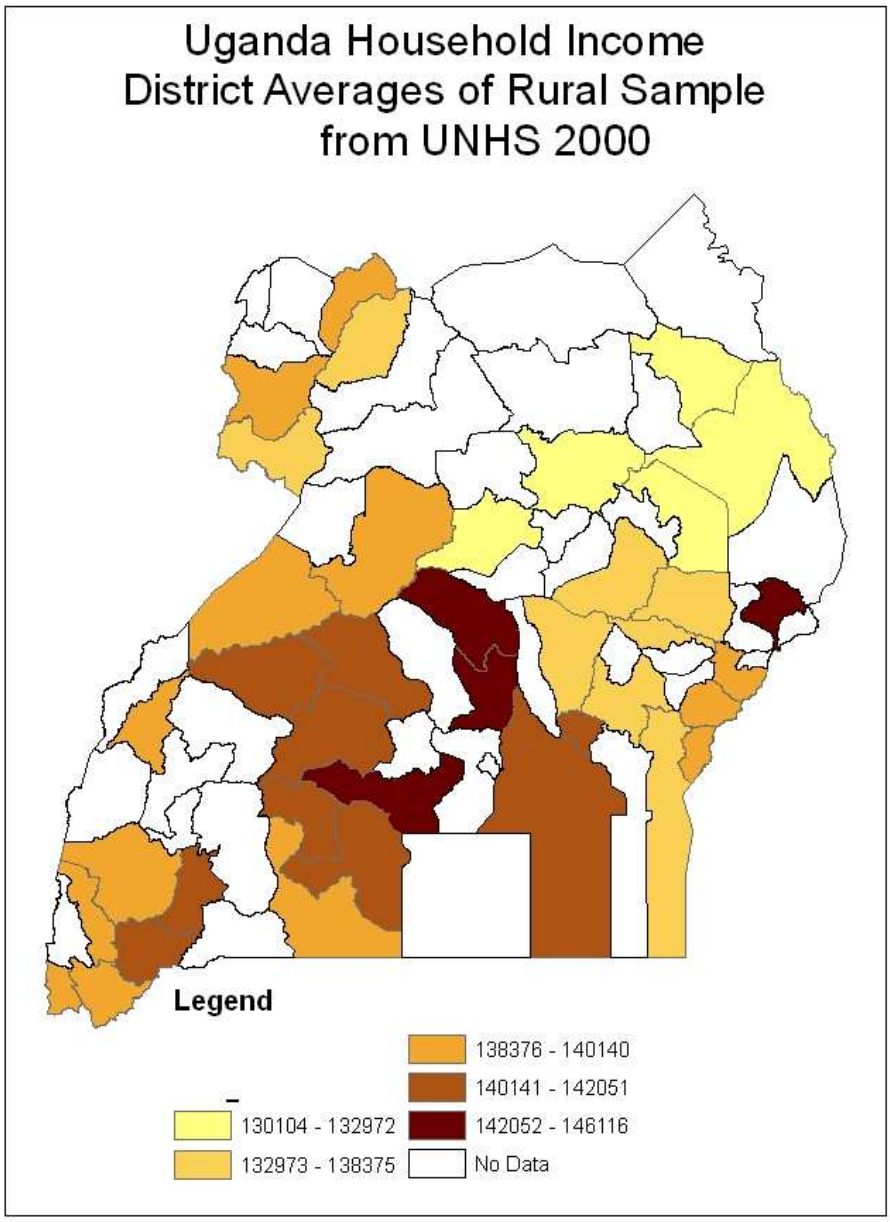

Figure 6. Map showing the spatial distribution of income in Uganda. Units are log shillings x 10^5

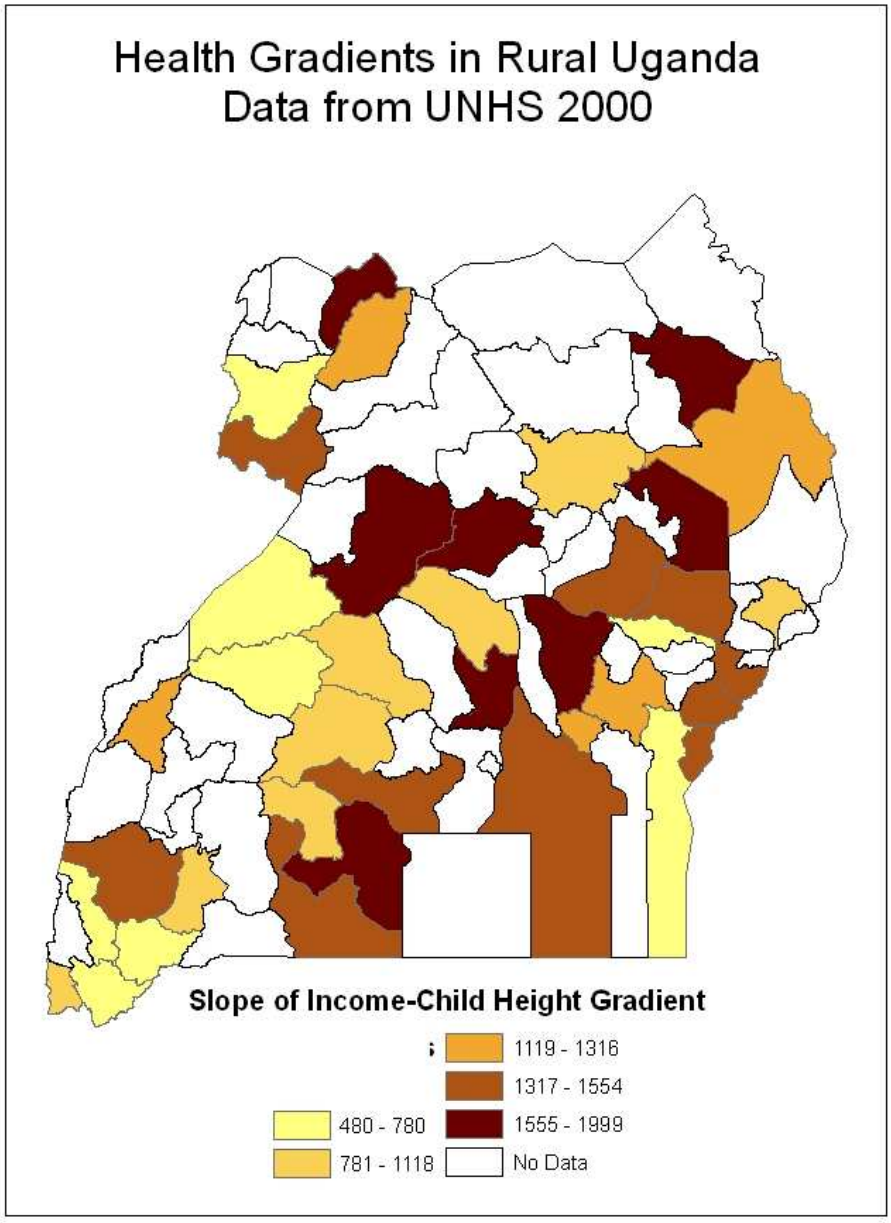

Figure 7. Spatial distribution of rural coefficients relating log income to child height. Units are slopes multiplied by 10^3.

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