



SCHOOL OF  
ECONOMICS AND  
MANAGEMENT

Master's Programme in Economic Development and Growth

## Belgen<sup>i</sup> and the Parental Purse

### The Impact of the Karamoja Famine on Intra-Household Resource Allocations

by

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Early-life health shocks can have very serious long-term consequences for those exposed to them, a fact which must be considered when parents make decisions with regards to allocating resources across their children. Here, the 1980 famine in Karamoja, Uganda is used as a natural experiment in which to test whether parental investment responses reinforce or compensate the impacts of early-life shocks. Through a novel implementation of the Latent Engel Curve Approach, evidence is uncovered that boys, those exposed at younger ages and those living in a male-headed household were all more likely to see reinforcement of the famine's effects, whilst girls, older children and those living under a female head were more likely to see compensatory effects.

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# 1. Introduction

It has been well established in numerous fields that one's early years are vital in determining outcomes across several axes later in life. Within the economic lexicon, one topic in this area which has gained a great deal of attention has been the long-term impacts of exposure to major early-life health shocks on later health, educational and economic outcomes. It is common within this literature to use major events such as famines, epidemics and other such disasters to model exogenous shocks to the health of populations in order to uncover the causal links between early-life health and later-life outcomes (Almond and Currie 2011). Another somewhat less deeply explored part of the literature looks to how parents respond to differences in human capital endowments between their children, with papers tending to focus on whether parents use investment strategies which will reinforce these initial differences in or attempt compensate for them (Strauss et al 2000). This study builds upon these two strands of literature, providing evidence of how parents respond with regards to their intra-household resource allocations to the exposure of their children to health shocks during their critical stages of development. The health shock in this case comes in the form of the 1980 famine in Karamoja, Uganda. The famine itself acts as a quasi-exogenous shock to the health endowments of the exposed children, allowing for an identification of the causal links between health conditions in the early years and the allocation of resources within the household thereafter. In essence, the question being asked is; given that famine exposure is likely to have long term negative impacts on the health of exposed children in the household, how do parents respond to such a shock with the intra-household allocation of resources?

In investigating this topic, several existing challenges within the aforementioned strands of research can be overcome. When looking into the long-term impacts of major shocks such as famines and epidemics, most studies will approach the problem using data taken when those exposed in their early years are fully grown adults, identifying who was exposed, the likely intensity of their exposure and then seeing how adult outcomes vary between comparable exposed and unexposed individuals. If it is established that the shock in question is exogenous then conditional on any included controls, the differences between exposed and unexposed individuals reflect the causal impacts of the event. This approach, however, ignores the channels through which these effects transmit themselves, focussing only on the net impact of the event. The channel of health is well understood at this point. Results from the epidemiological literature have shown that exposure to nutrition shocks whilst either in-utero or in early childhood can generate maladaptive changes in the physiology of foetuses, leaving

individuals frailer and at higher risks of developing diabetes, heart disease and other conditions (Barker and Osmond 1986, Paul 2011). Concurrently, exposure to disease in these periods can lead to inflammatory responses which divert bodily resources away from the development of important tissues (Crimmins and Finch 2006). Very little effort, however, has been made with regards to understanding how parents respond to the exposure of their children to such events. With results from the literature on human capital formation highlighting the importance of parental investments in the generation of later-life outcomes, this is likely an important channel when it comes to understanding how exactly the long-term consequences of these events are generated.

The strand of literature regarding the interaction of the within-household distribution of human capital endowments and parental investment decisions faces a number of empirical problems. One major issue is the measurement of individual endowments. Given that a great deal of investment is made into children whilst they are in gestation, it is very difficult to identify variables which can capture variation in their endowments which is not influenced in some manner by parental investment decisions. In studying the relationship between individual endowments and the intra-household allocation of resources in the context of a famine, it is possible to identify variation in these endowments which is somewhat uncorrelated with previous parental investments.

Finally, this paper also offers a novel approach with regards to how one might measure the relationship between individual endowments and parental investment. Here, the Latent Engel Curve Approach, introduced by Maldonado (2019) is applied. While the technique was originally formulated as a means of measuring gender biases in intra-household resource allocations, here it is demonstrated that the approach may be applicable to a much broader range of questions. Like its predecessor, the Engel Curve Approach (Deaton et al 1989), the technique relies upon the use of household consumption data, a widely available resource, making the method very promising when it comes to further studies of this kind. However, in specifying the system of expenditure shares across different consumption categories as a factor model, Maldonado (2019)'s method is able to sidestep a number of the technical issues of Deaton et al (1989)'s Engel Curve Approach, an innovation made possible by the advances in the empirical analysis of consumption preferences by Barigozzi and Moneta (2016) and in factor analysis by Bai and Liao (2016).

Using data from Uganda's 1992 Integrated Household Survey (Khan 2019), the investigation uncovers evidence that the famine did elicit an investment response from the parents of those children who were exposed in their critical stages of development. Those children exposed below the age of 3 saw less investment relative to comparable children of a similar age, reinforcing the effects of the initial health shock. Those exposed between the ages of 3 and 4 tended to see slightly more investment than control groups, compensating them for the famine's impacts. Boys tended to see far more in the way of reinforcement, while exposed girls were relatively more likely to experience compensatory effects. Female household heads were also more likely to compensate for the effects of exposure than were male heads. We also see that those households with incomes higher than the median in the sample exhibited stronger responses in both the compensatory and reinforcing directions than those households with lower incomes. Results for households with larger numbers of children are similarly accentuated when compared to those households with fewer children.

The paper is structured as follows: first, a discussion of the Karamoja famine of 1980 is presented. Following this, a conceptual framework is given, setting out the present state of the literature regarding the impacts of early-life health shocks and the interaction of endowments and the allocation of resources within households. Here, a simple model is also discussed, providing a set of hypotheses to be tested within the empirical section. The fourth section of the paper is devoted to a discussion of the Integrated Household Survey, as well as some cursory analysis of the data. The next section presents the methodology, discussing the Latent Engel Curve Approach in relation to its predecessor, the Engel Curve Approach, as well as detailing the technical aspects of its application. Section 6 then discusses the results of the estimations, followed by section 7 which compares the implications of these results compared to the theory and predictions set out in section 2. Section 8 concludes.

## **2. Karamoja and the Famine**

Karamoja, as seen in figure 1 (overleaf), is located in the North East of Uganda, along the borders of Sudan and Kenya. The region is largely made up of a plateau, with the odd cluster of mountains of heights close to 10, 000 feet. It is known as being home to one of Uganda's harsher natural environments, with much of its vegetation being made up of thorn scrub, though denser coverings of small trees and grass may be found toward the west of the region (Biellik & Henderson 1981). With its semi-arid climate, droughts are expected to hit Karamoja every few years, an eventuality which has often been well prepared for by the

Karamojong, the region's main residents. However, an extended period of drought between 1979 and the end of 1980 sparked a major famine, which is estimated to have killed between 20,000 and 50,000 people, the majority of whom were children (Biellik and Henderson 1981, Alnwick 1985). Here, I will briefly describe the processes which contributed to this famine, as well as providing some background information regarding the region and its residents.

Traditionally, the residents of Karamoja's plateaus have been transhumants, residing around permanent base settlements where women would cultivate cereal crops such as sorghum, while young men would travel with their herds to take advantage of the seasonal variation in grazing opportunities. As a further source of food stuffs, hunting and gathering would often be undertaken (Lamphear 1976). The diversification of food sources across agricultural and pastoral means provides a natural degree of protection against droughts. During years of particularly low rainfall, the Karamojong would forcibly encroach onto more fertile lands to the South and West, as well as raiding other tribes' cattle herds to replenish their own (Gartrell 1985).



Figure 1: Map of Uganda

Under colonial rule (1921-1962), British authorities placed restrictions on the activities of the Karamojong. Firstly, the creation of hunting reserves, later to become national parks, meant that the movements of Karamojan herds were restricted. Second, the practice of cattle



raiding was all but stamped out for around 30 years. Using data from the Ugandan National Census, Baker (1975) finds that during the latter part of the colonial period, the population of Karamoja was increasing rapidly, expanding by 36% between 1948 and 1959 and by 65% between 1959 and 1969. At the same time, raids from the Turkana, a group from northern Kenya, began to affect much of eastern Karamoja, concentrating its population in already over-used grazing zones to the west (Gartrell 1985). These processes all worked to undermine the traditional coping mechanisms that the Karamojong had for dealing with crises, concentrating their population in ever smaller areas. In response to the worsening overgrazing in the region, British authorities adopted a marketing scheme aimed at de-stocking Karamojan cattle herds. Baker (1975) points out that this scheme was less used as a means of reducing herd sizes by the Karamojong, but more to adapt to drought risks. At times when rain was scarce, cattle were sold on the scheme, with the cash being put towards maize meal, though this led to deteriorations in the region's terms of trade.

Many of the events of the independence era worsened Karamoja's vulnerability to famine and drought. Under the rule of Idi Amin (1971-1979), Uganda's economy and social services took a dramatic turn for the worse. 1972 saw Amin expel the Asian residents of the country, who made up a huge share of healthcare workers. This and the migration of many other professionals to other countries in East Africa left many regions within Uganda without functioning hospitals or emergency services (Dodge 1986). In April 1979, the Ugandan National Liberation Army (UNLA) overthrew Amin. In the chaos that followed, 3 successive governments were formed before December 1980. Concurrently, sophisticated weaponry began to be brought into the Karamoja region by the UNLA, which was quickly taken up by many of the region's residents (Okudi 1992).

The liberation war coincided with a drought in the Karamoja region, the effects of which interacted with the dramatically reduced capacity of the Karamojong to cope with such conditions, leading to calamity (Umana-Aponte 2011). Some groups returned to cattle raiding, making use of the weapons provided by the UNLA. This led to the concentration of the more poorly armed Karamojans in ever smaller enclaves, worsening their ability to respond to the poor natural conditions and pushing food entitlements down even further (Stites et al 2007). At this time, Uganda's government was focussed on reinstating itself after the fall of Amin, so much so that the then president denied the existence of the crisis (Oloka-Onyango et al 1993). Thus, relief was slow to arrive to the region, with most aid distribution being run by the WFP and religious missions (Cisternino 1985). While the response was in full swing by around

August of 1980, the situation had already become critical in May of that year (Robinson et al 1980). Only toward December did the crisis begin to fully subside.

The effects of the famine appear to have been particularly severe. Table 1 shows estimated age-specific death rates during the time of the famine provided by Biellik and Henderson (1981). Firstly, we see exceedingly high rates of mortality for infants and children. The infant mortality rate of 607/1000 represents an incredible increase when compared to the same measure taken from the census of 1969; 129 per 1000 live births. This increase is in no way confined to infants, comparing the crude death rate during the famine, 212/1000, with that of the earlier census, 23/1000, we see that all age groups see a substantial increase in mortality. These numbers rates are comparable with those of some of the most severe famines of the 20<sup>th</sup> century, with the crude death rate in the 1974 Bengal famine standing at 123/1000, while infant mortality rates among the nomadic Issa stood at 615/1000 during Ethiopia’s 1974 famine (Alamgir 1980, Seaman et al 1979). The causes of death uncovered by the study also points to famine as the major cause, with 78% of those dying during the sample period succumbing to starvation, while another 20% died from disease. With such high death rates, the issue of selective mortality arises here. In such events, those at the bottom of the health distribution are most likely to die, leaving those with better pre-existing health overrepresented in the group of survivors. The effects of this process on the study’s results will be set out in more detail in the discussion section.

Table 1: Karamojan Death Rates

Age Group	Death Rate/1000
<1 year	607
1-4 years	305
5-17 years	171
≥18 years	140

Source: Biellik and Henderson (1981)

Here we have seen how the itself famine came about as a result of interactions between colonial policies imposed by the British, increasing land pressures, the deterioration of public services under Idi Amin, the chaos induced by the liberation war, as well as the drought of 1979. The severity of the famine has also been highlighted; while the area affected may be small compared to, say China’s Great Famine, the death rates are comparable to some of the worst famines of the 20<sup>th</sup> century. Clearly, even for those who survived the event, it would have

acted as a serious shock to their health. With all this in mind, it is now necessary to set out how exactly a major health shock might affect the allocation of resources within households.

### **3. Conceptual Framework**

In order to properly frame the research question at hand, it is necessary to delve into the literatures on the impacts of early-life health shocks upon human capital endowments, as well as that on the links between individual endowments and parental investment decisions. Once the two have been summarised, a simple model is presented in hopes of clarifying the links between famine exposure and investment decisions, as well as the covariates over which these decisions may vary.

#### **3.1 Early Life Health Shocks and Individual Endowments**

When discussing early life health, we are predominantly interested in the nutritional and disease environment an individual is exposed to whilst in-utero or in their neonatal phases of development. A seminal paper regarding the link between early-life health and later-life outcomes is that of Barker and Osmond (1986), who studied the link between neonatal and post-neonatal mortality rates with ischaemic heart disease across England and Wales. They conclude that those exposed to a poorer nutritional environment in early life were more vulnerable to chronic illnesses later in life. This study gave rise to what would be known as the foetal origins hypothesis. The hypothesis states that uterine rates of nutrition act to signal the environment the foetus will experience once born and that the foetus will adapt based upon these signals. If the signals are representative of the environment the foetus will encounter, then this process leaves them well-suited to the outside world. However, if there is a mismatch between the environment implied by these signals and the true environment, many in-utero developments may be maladaptive. Given that a shock is, by definition, a transitory phenomenon, then it is likely that the in-utero adaptations generated by it will be inappropriate for the outside world (Paul 2011).

Such programming is not just confined to the womb. The process of programming depends largely upon the plasticity of cells during development. While it is true that this is at its highest during a child's uterine stages of development, several cell types retain their plasticity well into early childhood (Langley-Evans 2015). This is of concern when it comes to the development of the brain. With neural pathways remaining in development well into

childhood, the potential for shocks in early childhood to affect an individual's neurological development remains high (Plagemann et al 2000).

Of course, nutrition is not the only important factor in an individual's early stages of development. Exposure to disease, something which often comes hand in hand with famine, can have devastating impacts. Infections and the body's inflammatory response to them are highly taxing on the metabolism, requiring resources to be redirected away from the development of new cells and tissues (Crimmins and Finch 2006). Further, many of the conditions common in times of famine such as diarrheal disease, are accompanied by anorexia, which can reduce already stretched energetic intakes (Martorell et al 1980, Butte et al 1989). Exposures to diarrhoea in early childhood have been linked to the impairment of cardiac muscle synthesis, which can dramatically increase the risk of cardiovascular disease later in life (Hunter et al 2001).

With economic and social outcomes being closely linked to an individual's health, it is no surprise that several studies have linked early life health shocks with factors such as education and income. For example, using longitudinal data from the US and UK, Case and Paxson (2008) find that those who were taller in childhood and had earlier growth spurts in adolescence, both indicators of good early-life nutrition, performed better on cognitive tests and earned higher incomes. Further, Currie and Hyson (1999) found children born in the UK with birthweights below 2.5kg were over 25% less likely to have passed their Maths or English O-Levels<sup>1</sup> than others.

Famines offer a natural testing ground for the study of the links between early life health and later life outcomes. They act as a quasi-exogenous shock to the health of those cohorts which are exposed in-utero or in early childhood, allowing for identification strategies which can sidestep the need for imperfect proxies of child health (Almond and Currie 2011). A particularly well-known group of studies has investigated the Dutch Hungerwinter of 1944-45, during which a German blockade cut off supplies to several towns in the western provinces of the Netherlands. Those children born immediately after the famine were 250g lighter on average than those born before (Lumey 1992). Looking to long term effects of the famine, we see that coronary heart disease, blood clotting factors and obesity were more common among those exposed in early gestation, whilst those exposed later in their development experienced

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<sup>1</sup> O-levels were the standardised secondary school leaving tests taken by students within England, Wales and Northern Ireland between 1951 and 1988.

greater difficulties with their renal functions (Ravelli et al 1976, 1999, 2000; Roseboom et al 2001; Painter et al 2005).

The Development Economics literature has produced a swathe of studies looking into the long-term effects of famines, providing important links between exposure and both later-life health and economic outcomes. For example, Dercon and Porter (2009) find that those exposed to the 1984 Ethiopian famine were 5cm shorter than control groups and that their earnings were likely reduced by 5% on average. Meng and Quian (2009) take up a study of China's Great Famine (1959-1961), finding that exposure reduced adult heights, weights, educational attainment and labour supply. Finally, in a study of the famine in question here, Umana-Aponte (2011) finds that exposure to the Karamoja famine in-utero or during infancy reduced individuals' likelihood of being literate as well as reducing the number of years they spent in school.

From the evidence presented by both the epidemiological and economic literature on early life health shocks, we see that there can be significant impacts on the endowments of exposed individuals across several axes. Holding constant the investment decisions of their parents, this leaves them at a significant disadvantage compared to the other children living in the same household who were not exposed during their critical stages of development.

### **3.2 Individual Endowments and Differential Investment**

The fundamental question being answered in the literature on the links between individual endowments and differential investment is whether the differential levels of investment across children reinforces or compensates for the initial inequalities in individual human capital endowments? It is worth noting that while this strand of literature is not nearly as well developed as that which considers the links between early-life health and later-life outcomes, it has produced several very useful pieces of information when it comes to answering the above question.

The majority of the relevant models treat households as unitary decision-making units. That is, they are based upon the constrained optimisation of a single household utility function. One of the earlier iterations of these models was introduced by Becker and Tomes (1976), in which the parental utility function is defined over own consumption and the quantity and quality of children. Constraints come in the form of the family's budget and the production function for child quality. The decision of whether to reinforce or compensate would of course

depend on how the marginal product of investment relates to individual endowments. In the context of their model, the authors show that when the cost of child quality is falling with the size of a given child's endowment, then parents will choose to reinforce the initial endowment distribution. This introduces the crucial idea of static complementarity between individual endowments and parental investments. That, holding all else equal, when investments and endowments are complementary to one another in the human capital production function, the marginal product of investment is increasing in the size of a given individual's endowment and thus reinforcement is more likely to arise.

The concept of static complementarity was later built upon by Cunha and Heckman (2007) who introduce a multiperiod human capital formation model. Within their context, investments during different stages of a child's development can be complementary to one another. In such a context, both the initial human capital endowment and earlier investments are complementary to subsequent investments, a feature dubbed dynamic complementarity (Aizer and Cunha 2012). Like static complementarity, the presence of dynamic complementarity is likely to generate reinforcement responses to the initial distribution of human capital endowments.

While the idea of complementarity is certainly useful, it says very little with regards to important aspects of child investment such as inequality aversion and discrimination. Behrman et al (1982) present several iterations of a general preference model wherein both market opportunities and preferences regarding the relative treatment of children jointly determine allocations. More specifically, the parental utility function includes arguments which capture the expected wages of all the children in the household. The assumption of convex preferences implies that in a household with for example 3 children, an expected wage vector of (500, 500, 500) would be preferred to one of (1000, 100, 400). That is, that parents are somewhat inequality averse. The choice of whether to reinforce or compensate is determined in part by the children's earnings functions; if there are higher marginal returns to investment for higher endowments, then reinforcement is more likely. Parental inequality aversion, however, will dampen the incentive to reinforce, making compensation more likely. In an extension to the model, using data on a sample of male fraternal twins, they find evidence of compensatory behaviour.

As noted by Yi et al (2015), the definition of reinforcing or compensating investment strategies does not and need not presuppose some specific model of intra-household resource

allocation. The question of whether or not either strategy is chosen is largely empirical. With regard to the pure empirics on the topic, roughly as many studies find evidence of reinforcement (Behrman et al 1994, Rosenzweig and Zhang 2009) as do compensation (Behrman et al 1982, Pitt et al 1990). Interestingly, in their study of the impact of childhood disease exposure on a set of Chinese twins, Yi et al (2015) find that parents reinforce the effects of these illnesses on educational outcomes, but compensates for them in terms of health investments, implying that different forms of human capital may be treated differently.

With all this in mind, there is certainly reason for us to expect differences in the individual endowments across children to affect resource allocation decisions. And if a health shock in the form of famine exposure either in-utero or in the critical stages of childhood development is able to affect individual health and human capital endowments, then there is certainly reason to believe that such shocks may impact on how resources are allocated within affected households. However, from the previous literature, it is not a-priori obvious which kinds of investment strategies may be taken or how investment strategies may vary across households in response to such shocks. The decision to reinforce or compensate appears to be largely determined by the competing forces of complementarities between endowments and investments and parental preferences regarding equity. As a means of gaining a little more clarity with regards to these issues in the context in question here, it will be useful to formally restate the trade-offs facing parents in the form of a simple model.

### **3.3 A Simple Model**

Here, I will present a simple model tying together the relationships between individual endowments, health shocks and parental investment decisions. The model will be at the level of the household, with investment and consumption decisions being made by the parents. Two periods will be considered. During the first, children are still living at home and largely dependent upon their parents. In the second, the children have grown up, earn their own incomes and are responsible for supporting their parents. Such a set-up is chosen in light of this study's context; Uganda, during the 1990s. Within the data sample, the median household has a daily income, far below the poverty line – around 87 cents. With such degrees of poverty in the sample, it is unlikely for a large proportion of them to be able to earn enough above the subsistence level to save for their retirement. Further, the country did not have a well-developed pension system at this time (Kuteesa et al 2007). Under such conditions, children are often treated partially as a form of old age insurance, wherein parents (and other relatives) invest

during their childhoods and reap returns in the form of transfers and other assistance as the children age (Bledsoe 1994). It is worth noting here that under a two-stage setup, the dynamic complementarities of Cunha and Heckman (2007) will not be captured, this limitation is generated for the sake of simplicity.

For simplicity, assume that the household has two children. Say that the parental utility function takes the form:

$$U = U(P_1, P_2, C_{11}, C_{12}, C_{21}, C_{22}) \quad (A1)$$

Where  $P_t$  is parental consumption in period  $t$  and  $C_{it}$  is the consumption of child  $i$  in period  $t$ . Assume that  $U$  is increasing at a diminishing marginal rate in all its arguments. Parents have the power to choose how much each individual consumes in the first period. With this in mind, first period expenditure is constrained according to:

$$P_1 + C_{11} + C_{21} \leq Y_1 \quad (A2)$$

Where  $Y_1$  is parental income in period 1. The price of all consumption is normalised to 1.

We can make the further assumption that the consumption of the children in period 2 will be determined by some increasing function of their human capital, which is in turn determined by their individual endowments and by how much they consumed in the first period:

$$C_{i2} = H(e_i, C_{i1}) \quad (A3)$$

Where  $e_i$  is the endowment of child  $i$ . Finally, we can assume that without the provision of a state pension fund and with severe capital market imperfections, parental consumption in period 2 can be characterised by an increasing function of their children's human capital:

$$P_2 = G(H(e_1, C_{11}), H(e_2, C_{21})) \quad (A4)$$

Thus, the consumption of the children in the first period plays a dual role for the parents; they value the immediate welfare of the child, but also know that the welfare of the child today has an implication for that child's future success and for the parents' future material wellbeing. Assuming that preferences are well behaved, the optimal choice of period 1 consumption for child  $i$  will then be given by a function of each child's individual endowment and the first-stage income:

$$C_{i1}^* = \varphi(e_i, e_j, Y_1) \quad (A5)$$



Exposure to famine during critical stages of development, say for child  $i$ , in this context will express itself through the  $e_i$  argument of the function. Thus, for a family with child  $i$  exposed to famine in their early years and child  $j$  not, we have:

$$C_{i1}^* = \varphi(e_i - F_i, e_j, Y_1) \quad (A6)$$

Where  $F_i$  captures the impact of exposure on child  $i$ 's human capital endowment.

Without making further assumptions regarding the form of the functions mentioned above, it is difficult to make firm predictions about the sign or magnitude of any of the differentials of this function. This is not necessarily problematic, after all the previous literature on differential investment uncovers both evidence of endowment reinforcement ( $\frac{\partial C_{i1}}{\partial(e_i - F_i)} > 0$ ) and endowment compensation ( $\frac{\partial C_{i1}}{\partial(e_i - F_i)} < 0$ ). However, much may be inferred from considering the likely impacts of changing magnitudes of the exogenous variables in the above equations, as well as the impacts of those factors which may affect the forms of the equations.

In the context of this study, it is likely that the chance of us seeing either reinforcement or compensation will depend upon household incomes ( $Y_1$ ) and the magnitude of the famine's effect on the exposed individual's endowment ( $F_i$ ). Let's make the somewhat loose assumptions that  $e_i$  and  $C_{i1}$  are complementary in the human capital function and that  $H(e_i, C_{i1})$  and  $H(e_j, C_{j1})$  are perfect substitutes in  $G(\cdot)$ . With the utility derived from each of  $U(\cdot)$ 's arguments falling at the margins, parents will be at least slightly averse to inequality between their children. This aversion will be more strongly expressed at higher levels of income. If  $Y_1$  is particularly low, then  $C_{i1}$  is tightly constrained for both children, which then constrains second period income and consumption for all those in the household. Under such circumstances, the marginal product of an extra unit of  $P_2$  is likely to be particularly high, regardless of which child generated it. When  $e_i$  and  $C_{i1}$  are complementary,  $\frac{\partial P_2}{\partial C_{i1}}$  is an increasing function of  $e_i$  and thus, at lower levels of income parents may choose to reinforce the initial endowment distribution as the benefit of satisfying their material needs may exceed those of satisfying their inequality aversion.

To the extent that  $e_i$  and  $C_{i1}$  are complementary in  $H(\cdot)$  and the greater is the magnitude of  $F_i$ , the lower will be  $\frac{\partial P_2}{\partial C_{i1}}$ . When the marginal utility of an extra unit of  $P_2$  is high, this may lead the parental proclivity to reinforce the impact of the shock to be increasing in the

magnitude of  $F_i$ . Given that most of the affected households in the sample exist below the poverty line, this may well be the case. Under such circumstances, we might expect reinforcement to be more likely or of a greater magnitude for those who were exposed in-utero, who according to Umana-Aponte (2011) appeared to show some of the worst scarring impacts of the famine. We might also expect boys to be more likely to experience reinforcement; it is well-established that young males are often more vulnerable to health shocks in their neonatal phases of development, with low birthweight males being much more likely to develop early-life morbidities such as pulmonary disease and intracranial haemorrhages, both of which can affect one's development (Stevenson et al 2000). It is worth noting that in order for the worsened health impacts for boys to generate greater degrees of reinforcement for them, it would have to be that girls in the sample are not being systematically discriminated against. This condition which may well be met; in an anthropological study of the Karamojong, Stites et al (2007) find no evidence of boy-girl discrimination.

Putting the magnitude of the exogenous components to one side, it may also be that the form of the model's functions vary from subsample to subsample. For example, the utility function itself is likely to vary from household to household depending upon the characteristics of the household head. With the choice to reinforce or compensate often being determined by a trade-off between inequality aversion and aggregate material wellbeing, the degree to which both these factors are weighted in a given household's utility function may have major impacts upon the results we see. Along this axis, the gender of household heads may be an important factor. The behavioural economics literature has uncovered evidence to suggest that women may be more inequality averse than men (My et al 2018). Thus, we may expect to see a greater degree of compensation in female headed households.

Up until this point, it has been assumed that the number of children within the home is fixed at 2, though this is obviously not the case in reality and family size may also generate different reactions among parents to famine exposure amongst their children. The topic of child quantity is very well established in the literature on parental investments. Models introduced by Becker and several co-authors discuss the trade-off between child quality and child quantity, implying that as family sizes increase, levels of investment, which are the source of quality, fall in each child (Becker 1960, Becker and Lewis 1973, Becker and Tomes 1976). A useful extension to this framework has been developed by Aizer and Cunha (2012), who model the interactions between human capital endowments, investment and family size. In their model, when endowments and investments are complementary in the production of human capital and

when parental inequality aversion is moderate, the degree of reinforcement of the initial endowment distribution increases in family size. To the extent that these conditions are met in the Ugandan sample, we may expect that exposed children with more siblings would be more likely to see the effects of the famine reinforced.

As a summary of these predictions, we would expect in the empirical analysis to see:

- $\frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Low Income Sample}) > \frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{High Income Sample})$
- $\frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Boys}) > \frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Girls})$
- $\frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Younger at Exposure}) > \frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Older at Exposure})$
- $\frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Male Headed Household}) > \frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Female Headed Household})$
- $\frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Larger Household}) > \frac{\partial C_{i1}}{\partial(e_i-F_i)} (\text{Smaller Household})$

More intuitively, it can be expected that those in lower income households will be more likely to see reinforcement of the famine's effects in comparison to those in higher income households. Boys, given that they tend to feel worse health effects from nutrition shocks, will be more likely to see reinforcement than girls. For similar reasons, those exposed at younger ages should also see more reinforcing effects. We might expect female household heads to be more likely to compensate for the famine's effects than male heads. Finally, we might expect to see more reinforcement in households with more children.

We have now formally determined some of the mechanisms through which famine exposure may affect the intra-household allocation of resources, as well as considering a few axes over which these effects may vary. It is now time to introduce the data used for the study and present some preliminary analyses, before going on to describe the central methodology of the paper and the results derived from it.

## 4. Data

### 4.1 1992 Integrated Household Survey

The data for this investigation comes from Uganda's 1992 Integrated Household Survey, provided by Khan (2019). The survey was conducted between January of 1992 and June 1993 by Uganda's Ministry of Planning and Economic Development. The dataset includes 9,924 individual households drawn from the 49 districts which comprised the country,<sup>2</sup> the households in each district are labelled as either being in the major town of the given district, in another urban area or a rural area. Of the 9,924 households in the full sample, 400 were located in Karamoja, our region of interest.

The survey contains detailed information regarding the demography of the households, including the age, gender and interrelations between all household members. Detailed accounts of the main activities, whether in the worlds of work, education or housekeeping are given for household members, in addition to further information regarding their health and migration histories.

Of particular interest to this investigation is data on the income and expenditure patterns across the households in the survey. It is using this data on household consumption that we can uncover the variation in resource allocations between children. With regard to expenditure patterns, the survey contains 4 main expenditure categories, namely food, household non-durables, other living expenses (e.g. clothing and power costs) and household durables. These four major categories are comprised of 185 more specific expenditure categories, listing consumption expenditure for everything from beans, to matches, to nights spent in hotels. Food and non-durable categories list weekly and monthly expenses, other living costs are given monthly and yearly, while household durable expenses are given as yearly values. The dataset also contains information regarding household incomes, whether cash or in-kind, as well as listing if it comes from the main economic activity of the household or secondary activities.

In order to effectively conduct the factor analysis, upon which the Latent Engel Curve Approach is based, it will be helpful to make use of as many separate expenditure categories as possible. The main analysis will focus on monthly expenditures, with food, non-durable and living expenditures being kept as they are in the original dataset, while all the yearly non-

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<sup>2</sup> Since 1992, several new districts have been created in the country, for example Nakapiripirit was classified as being a part of Moroto at the time the survey was taken, but has since become a separate district in its own right.

lasting expenditures will be divided by 12. Although doing this may introduce some distortions, lasting expenditures only account for 1.78% of total expenditure and thus should not pose to much of a problem when it comes to the reliability of estimates. Further, while in the interest of maximising the number of expenditure categories, it may seem sensible to conduct the analysis across all 185 categories, this is unlikely to be feasible. For many households, data on many of these very narrow categories is missing or listed as zero. It is likely that several of the cases of either missing or zero values across expenditure categories are symptomatic of recall issues, or an understandable unwillingness of those being surveyed to list their spending habits in minute detail. In the best-case scenario, these errors will be randomly distributed, meaning that only the efficiency of estimates will be undermined. It is, however, wise to assume that this best-case scenario has not materialised. As a result, instead of using all 185 expenditure categories, the analysis will focus on 36 aggregate categories. Put simply, instead of considering expenditure on bananas, apples and oranges separately, we will be looking at expenditure on fruit as a whole.

## **4.2 Preliminary Analysis**

It is worth spending some time looking at the characteristics of those in our sample, it may be especially valuable to compare the conditions faced by those in our treatment group with comparison groups. Noting the conclusions drawn from the model presented in section 3, it may be especially informative to look at how treatment and comparison groups size up with regards to household income, their gender balances, the characteristics of their household heads and the sizes of the families that they live in. To do this, it is of course vital to explicitly define who exactly exists within each group. Nutritional shocks tend to have lasting effects for those exposed while in-utero or below the age of five, thus our treatment group in the broad sense will be those born in Karamoja between 1975 and 1981. With the survey being taken between January 1992 and June 1993, these individuals were between 11 and 18 years old. In later estimations, this treatment group will be narrowed to include just those born between 1977 and 1981. Given that those born in 1975 and 1976 were transitioning into being young adults at the time of the survey, the dynamics of the intra-household allocation of resources will likely have worked in a different way for them than for the younger cohorts. For example, individuals in this age range will be far more likely to earn their own income than those born just a handful of years earlier. Thus, considering them with the idea of parental investment in mind is likely to lead to some confusion.

Now, it is worth noting that several other regions in Uganda did suffer some food shortages during the same period as Karamoja, though these shortages were significantly less severe. These regions were namely Mbale, Tororo, Lira, Gulu, Kitgum and Masindi (Okudi 1992). In order to maintain a clear definition of who is our true treatment group and true comparison group, those born in these regions will be omitted from most of the estimations.

Table 2 shows some summary statistics regarding the yearly household incomes and household sizes faced by those born in Karamoja between 1975 and 1981, as well as a comparison group of children born in the same timespan but residing in other parts of Uganda, excluding the food shortage regions. We see that on average, those children in the treatment group live in households with slightly fewer people; with the median household size for the treatment group being 6, compared with 7 in the comparison group. Household incomes and incomes per person are significantly lower in the treatment group. Were we to calculate the median daily income per person, those in the treatment group would be living on 32 cents per day, compared with 77 cents in the comparison group. While the median individual in both groups clearly reside below the poverty line, it is clear that the degree of poverty is far more acute for our Karamojan children.

Table 2: Household Income and Size

		Mean	Median	Std. Dev.
Household Income (\$)	Treatment Group	1069.73	688.64	1177.91
	Comparison Group	3548.98	1978.02	5286.8
Household Size	Treatment Group	6.285	6	2.38
	Comparison Group	8.06	7	4.03
Income Per Person (\$)	Treatment Group	163.75	117.96	146.7
	Comparison Group	431.49	281.66	551.45

Source: 1992 Ugandan Integrated Household Survey (Khan 2019)

Table 3 shows the share of girls, the share of those living in rural areas and the shares of those living in female-headed households in the two groups. We see that the Karamojan children are 12.6 percentage points more likely to live in a rural environment than are those in the comparison group. Further, the share of girls in the treatment group is also somewhat higher than in the comparison. The difference is not particularly significant, but this finding is in line

with that of Umana-Aponte (2011) who saw a greater share of women in Karamojan cohorts affected by the famine using data from Uganda’s 2002 census. These patterns may be reflective of the mortality patterns of the famine. Stevenson et al (2000) note that boys tend to be more vulnerable from health shocks in their early years than girls. Thus, this slight discrepancy may reflect a higher mortality rate among male rather than female children. We can also see that those in the treatment group are almost twice as likely to live in households with a female household head than those in the control group.

Table 3: Rural and Female Shares

	Treatment Group	Comparison Group
Female Share (%)	53.5	51.04
Rural Share (%)	75	62.4
Female-Headed (%)	59.5	30

Source: see Table 2

The two groups also differ somewhat with regards to their educational status. While 70% of those in the comparison group list studying as their primary activity, this figure is only 26.5% amongst the Karamojan children. Further, rates of illiteracy are far higher amongst the Karamojans, with their rate standing at 80% compared with just 21.5% among comparison groups.

While certainly interesting, these figures are not the primary focus of this study. We are of course interested on the impact of famine exposure on the intra-household allocation of resources. Very conveniently, the integrated household survey does contain data regarding how much parents spend on each child with regard to their education. With this data, it is possible to make some early and simple attempts to look at the impacts of famine exposure on child investment. This can be done with the following spline model:

$$\ln (School\ Spend)_{idt} = \rho_d + \gamma_t + \sum_{t=1977}^{1981} \beta_t(K_{id} * \gamma_t) + Urban_i + \varepsilon_{idt} \quad (B1)$$

Where  $\ln (School\ Spend)_{idt}$  is the natural logarithm of the total school expenditure directed towards individual  $i$ , from district  $d$ , who was born in year  $t$ .  $\rho_d$  and  $\gamma_t$  are district and birthyear fixed effects respectively.  $K_{id}$  is a variable taking a value of 1 if individual  $i$  was born in Karamoja and zero otherwise.  $Urban_i$  is a dummy variable taking the value 1 if individual  $i$  lives in an urban area and zero otherwise. The equation is estimated for all individuals not born in a food shortage region between the years 1977 and 1986. Those born before 1977 are

excluded given that the number of school attendees in these older cohorts begins to drop quite dramatically. With this attrition likely being non-random, the degree of comparability of these older individuals and the others in the sample is likely to be limited. The equation is estimated separately for boys and girls.

Figure 2 gives a visual representation of the results of the estimated equation. The first panel shows the results for boys, the second shows results for girls. The red solid line shows the fitted values for school expenditure for the treatment group, while the blue line represents a counterfactual in which the treatment effects are zero. Table B11 in the appendix provides a more formal restatement of the results.

Figure 2a: Effects of Famine Exposure on Educational Investment, Boys.

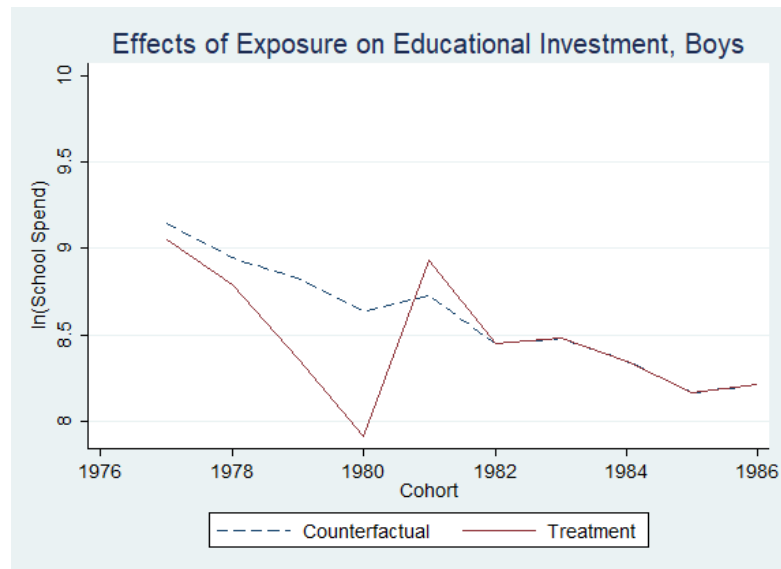
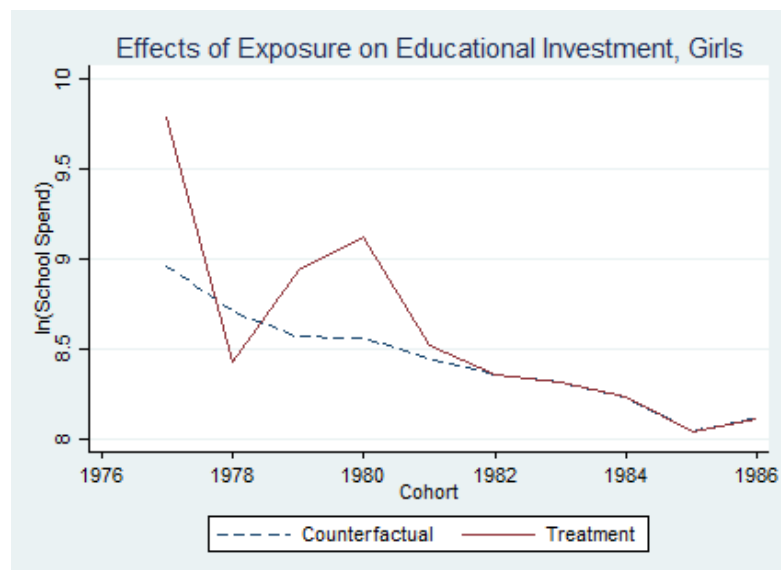


Figure 2b: Effects of Famine Exposure on Educational Investment, Girls.



Source: author's own calculations



Very different patterns appear for boys and girls. Boys in the treatment group see largely negative impacts implied by famine exposure, with this effect becoming gradually stronger from the 1977 cohort up until the 1980 cohort, after which a slight positive exposure impact is seen for the 1981 cohort. For girls, the only cohort seeing a negative impact implied by famine exposure is the 1978 cohort, for all others the fitted series for school expenditure lies above what would be expected under the counterfactual. To note, effects appear to be statistically significant for the 1977 and 1980 male cohorts and for all female cohorts bar that from 1977. These results imply that there has been some change in the allocation of resources directed to those children who were exposed to the famine in their critical development stages. Further, the fact that these effects appear to be more reinforcing for boys and more compensatory for girls is in line with the theory presented in section 3.

From all this, we have drawn some potentially useful information. The median household in the sample most certainly exists below the poverty line. According to the discussion in section 3, this may imply that there is a higher likelihood of there being reinforcement responses to famine exposure. We have also seen some preliminary evidence that parents may, in some cases, change their educational investments in children who were exposed to the health shock embodied in the famine. The responses are compensatory with regards to girls but reinforcing with regards to boys, both of which are consistent with the predictions of the model presented earlier. With all this in mind, it is time to move to formally set out the central empirical model of this investigation, before going on to discuss the results.

## **5. Methodology**

### **5.1 The Engel Curve Approach**

The Engel Curve Approach, presented in Deaton et al (1989) was introduced as a means of detecting gender discrimination in intra-household resource allocations. The approach involves the estimation of Engel Curves with the aim of inspecting the variation in expenditures on certain goods in response to variations in the demographic structure of households. From these variations, it is possible to determine whether people of a particular demographic group are being treated differently to others when it comes to intra-household resource allocations. While this investigation will make use of the Latent Engel Curve Approach, an innovation on Deaton et al (1989)'s work presented in Maldonado (2019), a good understanding of the original method will be very useful in understanding how this new method functions.

Deaton et al (1989)'s method stems from the concept of demographic separability in consumption, that only some demographic groups within a household will consume certain types of goods. From this, we can derive the idea of adult goods – those goods which are consumed by adults and not children. With regards to expenditure per person, the addition of a child to a household will elicit both income and substitution effects across all categories of goods. For all goods, income effects will be negative given that a fixed income will now be spent across a greater number of people. Substitution effects will vary across different types of goods. If we have goods which are consumed by children, then it may be that the addition of an extra child to the household will elicit a positive substitution effect. However, if we are inspecting an adult good, substitution effects will be negative. From this we should be able to see that the addition of an extra child must lead to a reduction in expenditure per person on any adult good. The authors exploit this fact to find a novel way of measuring gender biases in household expenditures; if a child of one gender elicits *less* of a reduction in expenditure per person on adult goods than those of another gender, then it is likely that children of that gender are being discriminated against in some manner.

For the purposes of operationalising this idea, the following Engel Curve is specified at the level of the household:

$$y_i = \frac{p_i q_i}{Y} = \alpha_i + \beta_i \ln\left(\frac{Y}{N}\right) + \eta_i \ln(N) + \sum_{j=1}^{J-1} \gamma_{ij} \left(\frac{n_j}{N}\right) + Z' \delta_i + u_i \quad (C1)$$

Where  $p_i$  is the price of adult good  $i$ ,  $q_i$  is the quantity purchased of adult good  $i$ ,  $Y$  is total expenditure,  $N$  is the number of people in the household,  $n_j$  is the number of people of demographic group  $j$  in the household and  $Z$  is a vector of control variables capturing the relevant characteristics of the household.

In order to compare the relative reductions in expenditure on adult goods in response to the addition of an extra child to the household, the authors suggest the use of the outlay equivalence ratio (OER). Formally speaking, for adult good  $i$ , it is the derivative of the expenditure share of adult good  $i$ , with respect to the number of people in group  $j$  within the household, divided by the derivative of good  $i$ 's expenditure share with respect to total expenditure:

$$\pi_{ij} = \frac{\partial y_i / \partial n_j N}{\partial y_i / \partial Y Y} \quad (C2)$$

More intuitively, the ratio shows by how much total expenditure would have to fall to elicit the same percentage reduction in expenditure on good  $i$  as would the addition of an extra individual from group  $j$ . Put in terms of the parameters of equation C1, the ratio can be given by:

$$\pi_{ij} = \frac{\eta_i - \beta_i + \gamma_{ij} - \sum_{j=1}^{J-1} \gamma_{ij} \frac{n_j}{N}}{\beta_r + \gamma_i} \quad (C3)$$

Where all parameters are replaced by their OLS estimates and all variables replaced by their sample means.

When group  $j$  is made up of children and good  $i$  is an adult good, the OER will be negative. If it is the case that the outlay equivalence ratio is more negative for one group than another, then it implies that the group with the more strongly negative OER has relatively more resources devoted to them across the households in the sample.

It is easy to see how such a method could be extended to looking at how those exposed to famine may be treated differently to those who were unexposed. Here we would need to find some subset of goods which were demographically separable from those who were exposed at critical ages to the famine and then compare the changes in expenditure on these goods or the variation in OERs elicited by the addition of a member of our treatment or of our control groups.

One burning question is how exactly adult goods are identified in the data. While the authors suggest two different tests, only one is regularly applied in the wider literature. In this test, the expenditure on a potential adult good is regressed upon a set of variables capturing the numbers (rather than shares) of those in each demographic group, whilst controlling for the vector  $Z$  and the total expenditure across all potential adult goods. If the coefficients associated with all child groups return as being jointly insignificant, then that good is deemed an adult good.

## **5.2 Why move to the Latent Engel Curve approach?**

While the Engel Curve Approach may be a good tool under certain conditions, it has generally struggled to perform well in its empirical applications. For example, Zimmermann (2012) finds that in India while individual level discrimination does exist, when extending the level of analysis to the household level using the Engel Curve Approach, this discrimination is no longer detectable. There are numerous other studies which have had similar difficulties

when it comes to uncovering evidence of discrimination (see for example Subramanian 1993, Horrel and Oxley 1999, Kingdon 2005, Haddad and Reardon 1993 amongst others). While we should not a-priori assume that discrimination is taking place while conducting any kind of study, the fact that the Engel Curve Approach fails to uncover reliable evidence of it across numerous different domains is potentially problematic.

Several authors have commented on the weaknesses of the approach, pointing out where these empirical failings may stem from. A major issue comes from the technique for the initial detection of adult goods, wherein we compare consumption patterns across goods within a set of potential adult goods. Here, the set of potential adult goods is selected by the researcher and thus, the goods included in the set could vary arbitrarily based on different researchers' conceptions of what constitutes an adult good. The F-test used to determine whether the consumption given good is demographically separable is very much sensitive to the set of adult goods used. As a result, it is possible that even in the same dataset, different goods can be classified as separable and not, simply based on the preferences of different investigators (Kebede 2008).

Maldonado (2019) points out that the Engel Curve Approach implicitly assumes that all goods can either be defined as adult or non-adult goods. In reality, lines may be far more blurred. If we think about food, the consumption of dietary staples or calories up to a given threshold cannot be a demographically separable form of consumption, given that all members of a household must engage in such behaviour in order to survive. However, the consumption of food past a given calorie threshold may be associated with a far wider number of behaviours, some of which may be separable from certain groups in the household. Under the original approach, we are unlikely to be able to differentiate between these two types of food consumption, with the likely result being a great deal of under-utilised variation in adult consumption habits, leaving us with far less to play with when it comes to detecting variations in household resource allocations.

When using the Engel Curve Approach, we must estimate separate Engel Curves for each adult good. This necessarily discards any variation stemming from substitution effects. This is problematic for two reasons. First, we are discarding potentially useful variation to be used in our estimation procedures. Second, we are making the implicit assumption that substitution effects are either unimportant or do not exist, which is not particularly realistic and may lead to strange readings when it comes to our results. Say that a family initially had fairly

high levels of consumption when it came to expensive beef tenderloins, but then a child was born into the household. In such circumstances, it may be rational to substitute towards a cheaper cut e.g. rump steaks. In estimating OERs without any of these substitution effects, a misleading reading of an increase in the OERs for rump steaks could easily be detected in response to an extra child being born.

### 5.3 The Latent Engel Curve Approach - Intuition

Here, I will set out the intuition behind the Latent Engel Curve Approach, before going on to explain the technical aspects of its application.

The Latent Engel Curve Approach is founded on the idea of demographic separability in preferences, as opposed to demographic separability in consumption. This idea goes back as far as Engel (1857) himself, who stated that the latent drivers of consumption determine the shape of Engel Curves and that goods should be classified according to the final purpose which they are meant to satisfy. These purposes could be the meeting of basic consumption needs, recreation, social signalling, amongst others. There may therefore exist a number of underlying consumption preferences which are associated only with certain groups within the household. That is, that they are demographically separable.

The question now arises as to how we might approach exploiting the concept of demographic separability in preferences to look for variation in intra-household resource allocations. The answer again comes in the form of Engel Curves, but instead of looking at variations in the expenditure on particular goods, we can look at variations in the levels of expenditure being put toward satisfying these underlying motives. The equations used to model these interactions are known as Latent Engel Curves (Barigozzi and Moneta 2016).

Say that we were able to identify the latent drivers of consumption, denoting them as  $f_r$ . Then it would be possible to derive Latent Engel Curves of the following form:

$$f_r = \alpha_r + \beta_r \ln\left(\frac{Y}{N}\right) + \eta_r \ln(N) + \sum_{j=1}^{J-1} \gamma_{rj} \frac{n_j}{N} + Z' \delta_r + u_r \quad (C4)$$

A useful parameter for our purposes here is  $\gamma_{rj}$ . This is a measure of the average effect of the addition of 1 person from demographic group  $j$  to a household on the amount of resources being directed to latent factor  $r$ . As an extension, we can also derive Latent Outlay Equivalence Ratios (LOERs) of the form:

$$\Omega_{rj} = \frac{\partial f_r / \partial n_j}{\partial f_r / \partial Y} \frac{N}{Y} = \frac{\eta_r - \beta_r + \gamma_{rj} - \sum_{j=1}^{J-1} \gamma_{rj} \frac{n_j}{N}}{\beta_r + f_r} \quad (C5)$$

To the extent that  $f_r$  is interpretable as the percentage share of income being allocated to factor  $r$ , then the ratio shows by how much total expenditure would have to fall to elicit the same percentage reduction in expenditure on the latent factor  $r$  as would the addition of an extra individual from group  $j$ .

The approach is able to sidestep the issues with the original Engel Curve Approach. Firstly, the identification of the latent factors,  $f_r$ , can be entirely data-driven, thus cutting out the problem of the arbitrary selection of goods into the set of possible adult goods. Second, we can represent the system of budget shares and underlying preferences as a factor model, meaning that consumption of a given good can be written as a linear combination of underlying factors. As a result, it is possible for the consumption of a given good to meet more than one fundamental aim, relaxing the unrealistic assumption that goods must either be ‘separable’ or ‘non-separable.’ Third, if it is the case that two goods are consumed to meet the same fundamental aim, then their consumption patterns will be explained by the same latent factor. Thus, variations in the latent factors are capable of reflecting substitution effects, unlike the budget shares of individual goods.

To summarise, the Engel Curve Approach and its variants can be adapted to looking into the effects of famine exposure on the intra-household allocation of resources by simply reorienting the approach to compare those in a treatment group with those in a control group, rather than just comparing boys and girls. The Engel Curve itself comes with several issues, namely that the definition of which goods are demographically separable from a given group may vary arbitrarily, the implicit assumption that all goods must either be separable or not is unrealistic and that the approach is not capable of capturing important substitution effects. The Latent Engel Curve Approach can be adapted in a similar way to the case of famine exposure, but is capable of sidestepping the aforementioned problems.

#### **5.4 The Latent Engel Curve Approach in Practice**

The application of the Latent Engel Curve Approach can be split into three steps. The first is the identification of the number of latent factors, the second is the estimation and extraction of the latent factors, the third is the estimation of the Latent Engel Curves.

### 5.4.1 Factor Identification

In order to identify the number of latent factors and later find estimates of them, we must first express the system of household budget shares and latent preferences in terms of a factor model. This model is summarised in equation C6 below:

$$Y = PF' + \varepsilon \quad (C6)$$

Where Y is a H by G matrix of budget shares. P is a G by R matrix of factor loadings, where R is the number of factors to be estimated. F is the H by R matrix of latent factors and  $\varepsilon$  is a G by H matrix of disturbance terms. In the present case, we have 9929 households (H) and 36 budget share categories (G); R is of course the number of latent factors, which will now be estimated.

To estimate R, I apply the tests recommended by Ahn and Horenstein (2013). These tests are used given their superior performance to other alternatives (see for example Bai and Ng 2002) when, as is the case here, dealing with factor models with one large and one small dimension. The authors recommend the use of two test statistics, the first being an eigenvalue ratio test (ER) and the other being a growth ratio test (GR). Details of the specific construction of these tests can be found in the appendix, however in the context of this discussion, the only relevant piece of information is that R is chosen such that it maximises the value of the ER and GR test statistics.

Table 4 presents the results from these tests. It should be noted that the test for r=1 is omitted, given that having one latent factor would not be compatible with the idea of demographic separability in preferences. Tests above r=5 are also omitted given that the explanatory power of each extra factor with regard to expenditure shares falls rapidly with r. This makes the interpretation of the properties factors past r=5 particularly difficult. As can be seen, r=4 appears to satisfy the conditions of both the ER and GR tests and thus, the rest of the analysis will be carried out using 4 factors.

Table 4: Results from Ahn and Horenstein (2013) tests.

	r=2	r=3	r=4	r=5
ER(r)	1.15	1.34	1.40	1.12
GR(r)	0.93	1.09	1.16	1.00

Source: author's own calculations

### 5.4.2 Factor Extraction and Identification

With the optimal number of latent factors identified, the next step is to estimate and extract the latent factors within the dataset. The process of generating consistent and efficient estimates in this setting is somewhat complex. Bai (2003) established that if both the H and G dimension of Y were large, then consistent estimation of both the factor loadings and the space spanned by the matrix F could be delivered via principal component analysis (PCA), even in the presence of heteroskedasticity and orthogonality. The efficiency of these estimates could be improved via the application of Feasible Generalised Principal Component Analysis (FGPCA), proposed in Choi (2012). The method is essentially the factor analysis equivalent of FGLS estimation and hinges on finding the solution to:

$$\min_{F,P} tr[(Y - PF')\Sigma_{\varepsilon}^{-1}(Y - PF')'] \quad (C7)$$

Where  $\Sigma_{\varepsilon}$  is the G by G covariance matrix of the disturbance terms. Being akin to FGLS,  $\Sigma_{\varepsilon}$  and P are first estimated via Principal Component Analysis, before F is chosen such that the above objective function is minimised. However, if either G or H were somewhat small, as is the case here with G = 36, then consistent estimates could only be delivered in the absence of heteroskedasticity and orthogonality. This condition is unlikely to be met in any practical setting. Thus, given that FGPCA hinges on the estimation of both its loadings and disturbance terms via PCA, the results delivered here would be far from consistent.

Maldonado (2019) proposes a solution to this problem, wherein P and  $\Sigma_{\varepsilon}$  are first estimated via penalised maximum likelihood, before FGPCA is then applied to retrieve F. He applies an algorithm developed by Bai and Liao (2016) which can produce consistent estimates of both P and  $\Sigma_{\varepsilon}$  in finite samples, even in the presence of heteroskedasticity and orthogonality. A formal statement of the algorithm can be seen in Appendix A.

Factor models are well known for their indeterminacy problem, that even when well estimated,  $\hat{F}$  only gives consistent estimates of the true F up to an orthogonal transformation (Barigozzi and Moneta 2016). Put plainly, this means that we are unlikely to be able to uniquely identify each column of the F matrix unless some very strict conditions are met (Bai and Ng 2013). Even with these conditions met, our estimates would likely show the latent factors to be highly dependent upon one another, something which would leave their interpretation as *distinctive* consumption drivers very much unclear.

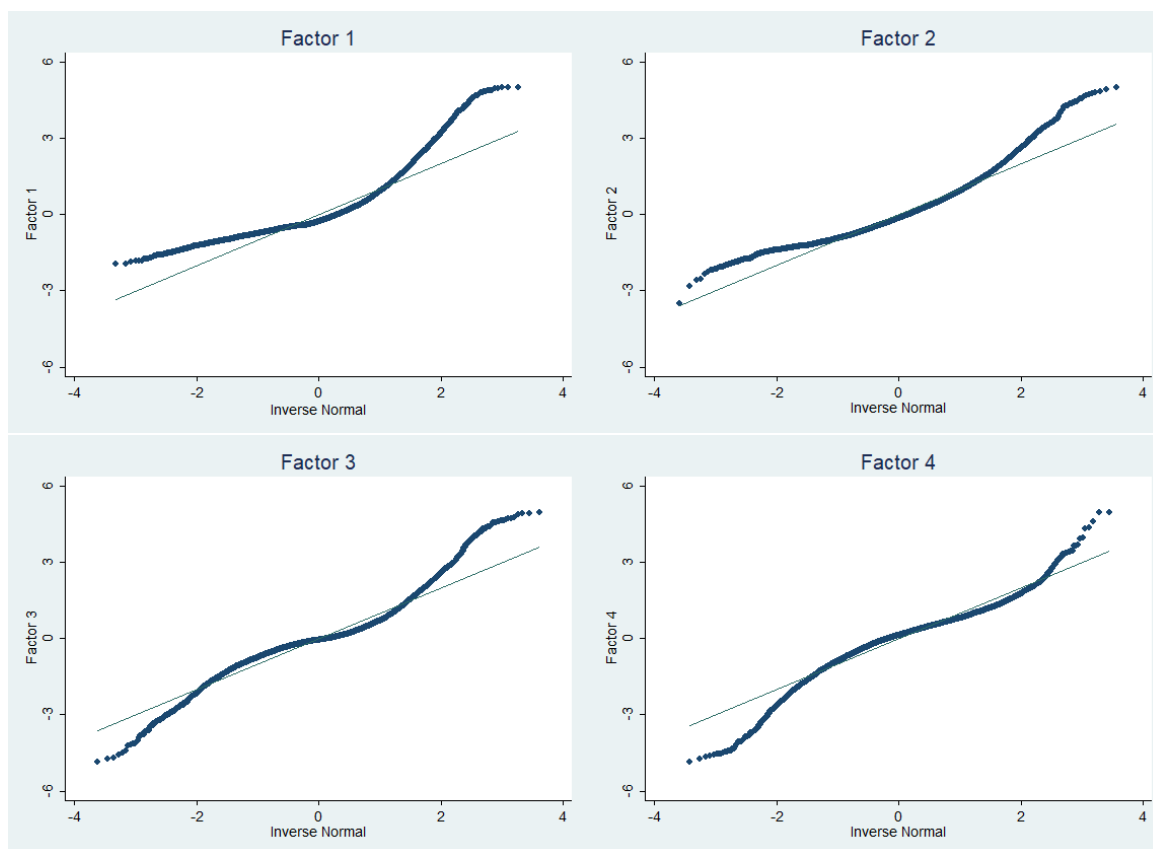


To overcome these difficulties, Maldonado (2019) recommends the application of Independent Component Analysis (ICA) to the estimated F matrix. ICA algorithms minimise statistical dependencies between factors such that rotated factors are unique up to a permutation, a sign and scaling factor. As in Barigozzi and Montena (2016), Maldonado applies the JADE (Joint Approximate Diagonalisation of Eigen-Matrices) Algorithm, as proposed by Cardoso and Souloumiac (1993). The algorithm re-estimates the matrix F, eliminating the dependencies between each of its columns, as well as determining the orthogonal rotation of it which maximises the non-Gaussianity of the extracted factors. Again, a discussion of the technical aspects of the JADE algorithm's construction can be found in section A of the appendix.

For the algorithm to provide consistent estimates of the latent factors, it must be that the latent factors are mutually independent and that the extracted factors have non-Gaussian distributions. By definition, the factors that are being estimated correspond to independent consumption motives, and thus the first condition is likely to be met. The second can be very easily tested. Figure 3 (overleaf) plots the quantiles of all the extracted factors against the quantiles of an inverse normal distribution. As can be seen from the non-linearity of the factor plots, none of the estimated factors follow Gaussian distributions.

While this method will have delivered consistent estimates of the latent factors, a caveat appears with regards to their units of measurement. In a perfect world, the estimates would precisely show the shares of household incomes being allotted to each latent factor. Although the extracted patterns will certainly be reflective of these patterns, the units they are given in are not percentage-points. While there may exist some permutation of the factors in these units, there exist a plethora of other possible ways in which they could be expressed. To my knowledge, the above methods cannot be extended to select a particular permutation of the factors and thus expressing them in percentage terms is not possible. Resultantly, the precise interpretation of the cardinal magnitude of a given Latent Outlay Equivalence Ratio is unclear, however the interpretations of their *relative* magnitudes and signs remains the same. With the interpretation of the magnitudes of the LOERs now unclear, the majority of the analysis will focus on the values of the  $\gamma_{rj}$  parameters. In doing so, the magnitude of treatment effects can be expressed in comparison to the distribution of  $f_r$  itself, which does have a somewhat intuitive interpretation. Essentially, if we see a one standard deviation change in  $f_r$ , this is still equivalent to a one standard deviation change in the percentage of expenditure being directed to latent factor r.

Figure 3: inverse normal quantile plots of the identified factors



Source: author's own calculations

As a summary of the method so far, the system of budget shares has been expressed in terms of a factor model. The loadings and covariance matrix of the model were consistently estimated via penalised maximum likelihood, which provided an estimate of the space spanned by the factor matrix  $F$ . The individual columns of this matrix, representing the latent drivers of consumption were then estimated via the application of the JADE algorithm. With these drivers now identified, the next step is to determine which of the latent consumption drivers are demographically separable from those of the same age range as our treatment group, before going on to the final analysis.

### 5.4.3 Factor Selection

When it comes to the identification of which factors are suitable for the final analysis, it is worth noting that it is not necessary in this case to establish that a given factor is driving adult consumption and not child consumption, just that it does not drive the consumption of

those born around the same time as the broad treatment group i.e. born around 1975 to 1981.<sup>3</sup> For a given factor to be demographically separable from a group, it should be that the addition of an individual from that group to the household does not have a statistically significant impact on the amount of resources being directed to the factor. Now, the extracted factors here have been derived from expenditure shares data rather than expenditure levels. To run such a test, it is therefore important to hold constant the levels of expenditure across households. As a preliminary test for demographic separability, I estimate the following equation at the household level:

$$f_r = \alpha_{0r} + \alpha_{1r}Y + \sum \lambda_{rj}n_j + Z' d_r + v_r \quad (C10)$$

Where  $f_r$  is extracted factor  $r$ ,  $Y$  is total monthly expenditure,  $n_j$  is the number of people from demographic group  $j$  residing in the household and  $Z$  is a vector of district fixed effects and a dummy capturing whether or not the household is in an urban location. Standard errors are clustered at the district level. A statistically insignificant reading of  $\lambda_{rj}$  would provide some evidence to suggest that factor  $r$  is demographically separable from group  $j$ .

Results from the estimation of equation C10 are given in table 5 on the next page. Of interest to us is the coefficient for the group born between 1975 to 1981. As can be seen, coefficients across all factors bar that of factor 2 turn up statistically insignificant. To further whittle down our choices, we can look to the latent outlay equivalence ratios associated with each group and factor. Theoretically, it should be that the ratios are negative for our group of interest, were they positive it would imply that the addition of an individual born between 1975 and 1981 would lead to an increase in the resources being allocated toward the given factor. Such a reading would be incompatible with the theory presented earlier.

Table 6 shows the latent outlay equivalence ratios associated with each factor for a number of age groups. These ratios were estimated from an iteration of equation C4, where  $Z$  again contains a vector of district fixed effects and an urban/rural dummy. Again, factors 1, 3 and 4 show readings compatible with demographic separability for the age group of interest. However, while for the fourth factor each adjacent group to the group of interest also display

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<sup>3</sup> The broad treatment group is used in this case such that we can be sure that the factor is separable for groups born during adjoining to the narrow treatment group. Were a factor accepted which appeared separable for only the narrow treatment group and not those born in adjoining years, the chance that the acceptance would be based upon statistical error could be somewhat high.

negative LOERs, the readings are much more mixed for factors 1 and 3, possibly implying that the fourth factor is safest for use in later estimations.

Table 5: Demographic Separability Tests, Equation C11

VARIABLES	F1	F2	F3	F4
Born 1989/later	-0.0195 (0.0140)	0.0321* (0.0183)	-0.00182 (0.0129)	0.0413*** (0.0149)
Born 1982-88	0.000997 (0.0148)	0.0411*** (0.00832)	-0.0164 (0.0101)	-0.00165 (0.00639)
Born 1975-81	-0.0160 (0.0144)	0.0493*** (0.0139)	-0.0117 (0.0130)	-0.00418 (0.00928)
Born 1968-74	-0.0255* (0.0148)	0.00786 (0.00994)	-0.0441*** (0.0140)	0.00306 (0.0110)
Born 1955-67	-0.0230 (0.0176)	-6.37e-05 (0.0131)	-0.0229* (0.0132)	0.0332** (0.0160)
Born 1942-54	-0.00295 (0.0257)	-0.0344 (0.0215)	0.00773 (0.0218)	0.0441** (0.0206)
Born 1941/earlier	-0.00110 (0.0239)	0.00191 (0.0241)	-0.00433 (0.0244)	-0.0446 (0.0263)
Total Expenditure	-5.18e-07*** (8.15e-08)	-9.19e-07*** (2.32e-07)	-3.19e-07** (1.48e-07)	2.10e-06*** (4.05e-07)
Urban Dummy	0.149** (0.0546)	0.339*** (0.0469)	0.0339 (0.0613)	-0.190*** (0.0541)
Constant	-0.380*** (0.0275)	0.485*** (0.0253)	1.116*** (0.0254)	0.0932*** (0.0323)
District FE	Y	Y	Y	Y
Observations	7,289	7,289	7,289	7,289
R-squared	0.060	0.135	0.149	0.057

Robust standard errors in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table 6: Demographic Separability Tests, Latent Outlay Equivalence Ratios

Birthyear	F1	F2	F3	F4
>1989	-5.36619	1.330449	-0.46275	0.478174
1982-1988	4.087142	1.505357	1.195383	-0.24669
1975-1981	-5.59657	0.797875	-1.05944	-0.57806
1968-1974	-6.81572	-1.21896	2.973562	-0.37245
1955-1967	-7.05728	-1.6154	1.68671	-0.36495
1942-1954	-5.55946	-1.28269	2.128862	0.031222

As a final check to determine which factor is most suited to the later analysis, we can inspect the factor loadings. In seeing how the consumption of certain goods correlates with each factor, a more intuitive sense of which factor is separable from those in the treatment group may be reached. Table B21 in the appendix contains the factor loadings. The first factor appears most strongly correlated with food consumption, with almost all its loadings being positively correlated to food expenditure categories, while it gives negative readings for almost all other expenditure categories. It is unlikely that a factor so closely correlated with food expenditure could be truly demographically separable from any age group and thus it is unlikely to be suitable for the later analysis. The third factor is more positively correlated to staple foods such as cereals, potatoes and legumes, as well as to durable consumption goods like furniture pieces. The fourth factor appears to be most strongly correlated to luxurious consumption items such as meats, alcohol and tobacco, as well as jewellery and recreation activities. With those born between 1975 and 1981 being children and teenagers at the time of the survey, it is likely that the luxury consumption associated with the fourth factor is at least somewhat separable from them. Given this, and the factor's strong performance across the other tests, the rest of the analysis shall be carried out using the fourth factor.

#### 5.4.4 Testing for the Effects of Famine Exposure

Having identified the correct latent factor to use, it is now possible to test for the effects of famine exposure on household resource allocations. This will be done using equation C11 below.

$$f_4 = \alpha_4 + \beta_4 \ln(y) + \eta_4 \ln(N) + \sum_{j=1}^{J-1} \gamma_{4j} S_j + \sum_{j=2}^{J-2} \varphi_{4j} K_j + Z' \delta_4 + u_4 \quad (C11)$$

For simplicity,  $y = Y/N$  and  $S_j = n_j/N$ . The term  $K_j$  represents the share of people in the household who are of demographic group  $j$  and were listed in the data as having always lived in Karamoja. The demographic groups defined under  $J$  will be those with birthyears corresponding to {pre-1932, 1933-1974, 1975-1976, 1977, 1978, 1979, 1980, 1981, post-1981}. In many specifications, these categories will be split by gender. Those born before 1932 are omitted in the first summation, those in the 1933-1976 and post-1981 ranges are also omitted in the second summation. All households with children born between 1975 and 1981 in regions which suffered food shortages in 1980 are omitted from the sample, to clearly delineate between who was exposed to a health shock and who was not.  $Z$  is again a set of

district fixed effects and an urban/rural dummy. Standard errors will all be clustered at the district level.

To note, it was mentioned earlier that while those born in 1975 and 1976 were exposed to the famine during their critical stages of development, their age at the time of the survey means that the dynamics of the intra-household allocation of resources was likely different for them than for the other members of the treatment group. The parameter  $\varphi_{4j}$  is simply being included for them such that they are not mistakenly interpreted as being in the control group.

A simple measure of the effect of the famine for those in demographic group  $j$  will be given by  $\varphi_{4j}$ . This parameter captures the impact on  $f_4$  of the addition of a member of the treatment group to a household, compared with the addition of a member of the control group. In this case, a *negative* coefficient implies that *more* resources are being allocated to those in the treatment group (compensation), while a *positive* coefficient would imply that *fewer* resources were being allocated towards them (reinforcement). Though they will not be widely used in the analysis, it is also easy to calculate LOERs for both treatment and control groups according to:

$$\Omega_j (\textit{treatment}) = \frac{\eta_4 - \beta_4 + (\gamma_{4j} + \varphi_{4j}) - (\sum_{j=1}^{J-1} \gamma_{4j} S_j + \sum_{j=2}^{J-2} \varphi_{4j} K_j)}{\beta_4 + f_4} \quad (C12)$$

$$\Omega_j (\textit{control}) = \frac{\eta_4 - \beta_4 + \gamma_{4j} - (\sum_{j=1}^{J-1} \gamma_{4j} S_j + \sum_{j=2}^{J-2} \varphi_{4j} K_j)}{\beta_4 + f_4} \quad (C13)$$

To summarise the method, it has been noted that in comparing the changes in expenditures on consumption categories which are deemed demographically separable for those in our treatment group, we can determine whether parents' resource allocation decisions respond to the exposure of their children to the famine. In order to do this, the system of budget shares across different goods was estimated as a factor model. Once estimated, the underlying factors which drive consumption in the sample were compared to find out which are demographically separable from our groups of interest. Once factor 4 was identified as being suitable for the final analysis, Latent Engel Curves were specified and estimated in relation to this factor. The next section will discuss the results of these estimations and their implications regarding how intra-household resource allocations responded to famine exposure.

## 6. Results

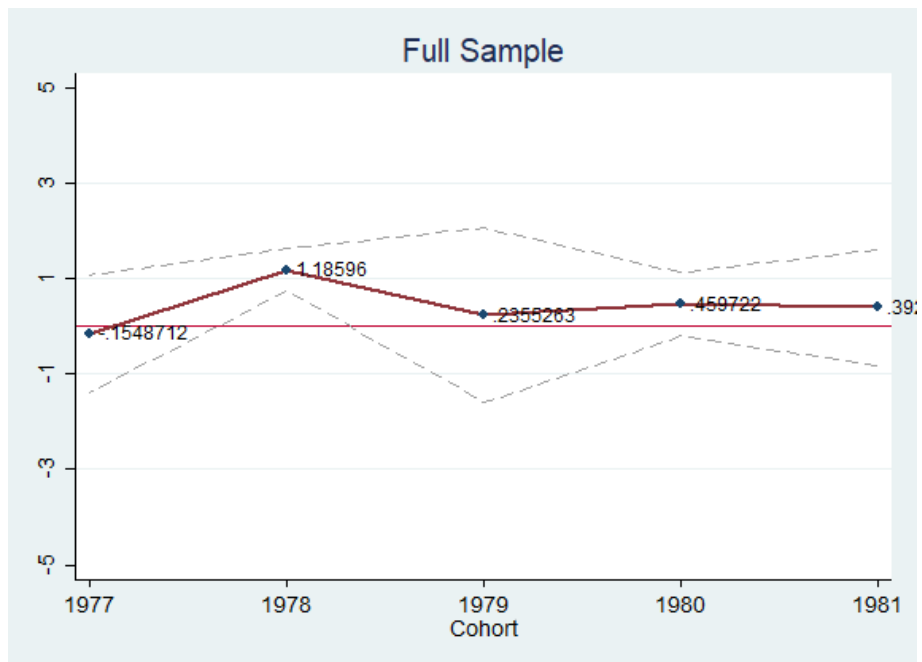
Visual representations of the results are provided in figures 4 to 8, which contain plots of the treatment effects,  $\varphi_{4j}$ , for each cohort in the treatment group, as well as their associated 95% confidence intervals. So as not to provide the reader with too great a preponderance of graphs, gendered treatment effects will only be illustrated in this manner when discussing the full sample, with the rest of the gendered effects being confined to the output tables. The output tables (tables B31a, B31b, B32a and B32b) for the estimates are located in the appendix. Table B32 shows results for when all demographic groups are split by gender, while table B31 shows results with boys and girls taken together. The first column in each table gives results for the full sample, while the other columns show results from different sub-samples of the dataset, corresponding to some of the predictions given in section 3. Namely, columns 2 and 3 show results for households below and above the median yearly income; columns 4 and 5 show results for female and male headed households; columns 6 and 7 show results for households with fewer than 4 children and 4 children or more.

From a visual inspection of the figures it becomes apparent that for the most part, coefficients show up as being positive, indicating something of a tendency towards reinforcement in the sample, though the magnitude and statistical significance of these effects differs from cohort to cohort and subsample to subsample.

Looking first to the full sample results shown in figures 4 and 5, treatment effects appear positive but insignificant for all those born between 1979 and 1981. We also see a negative but very small effect for the 1977 cohort. There does, however, appear to be a large treatment effect for the 1978 cohort. To put the coefficient's magnitude of 1.19 into context, the standard deviation of the latent factor's distribution is almost exactly one. Thus, the coefficient for the 1978 cohort implies that the addition of an exposed individual from that cohort leads to just over a 1 standard deviation increase in the share of income being allotted to the fourth factor as compared to unexposed individuals of a similar age. If the coefficients are allowed to vary by gender, we see different patterns emerge. In the female sample, the treatment effects for the 1977, 1979 and 1980 cohorts all show up as negative, with that of the 1977 cohort being statistically significant at the 10% level. Like in the un-gendered iteration of the model, girls born in 1978 appear to experience a fairly large reinforcement (positive) effect. Boys see a very different pattern; all coefficients apart from that of the 1981 cohort are

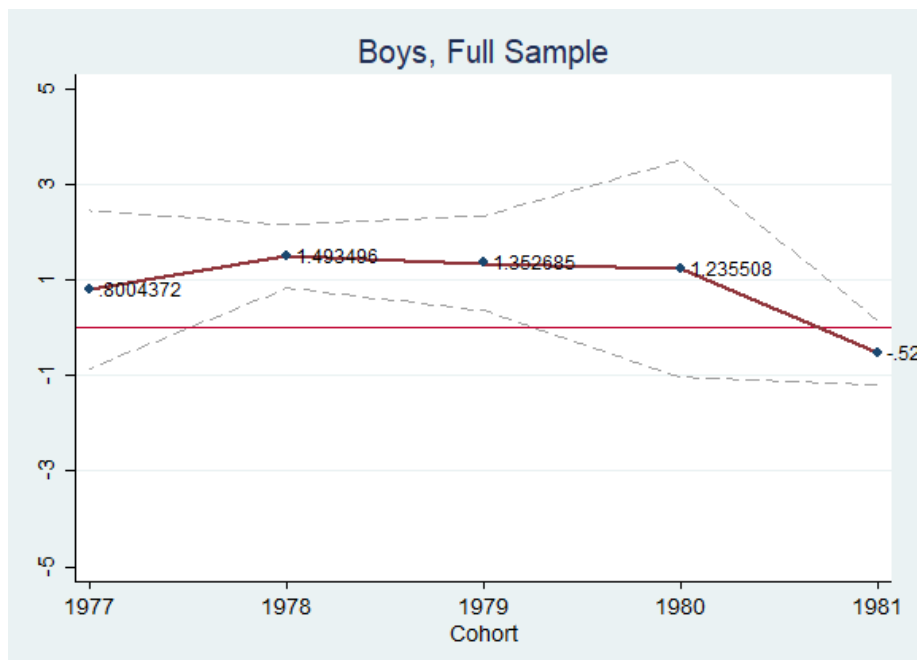
positive and around the equivalent of 1 standard deviation of the latent factor's in magnitude. The effects for the 1978 and 1979 cohorts are significant at the 1% level.

Figure 4: the impacts of famine exposure on latent factor 4, full sample.



Source: author's own calculations

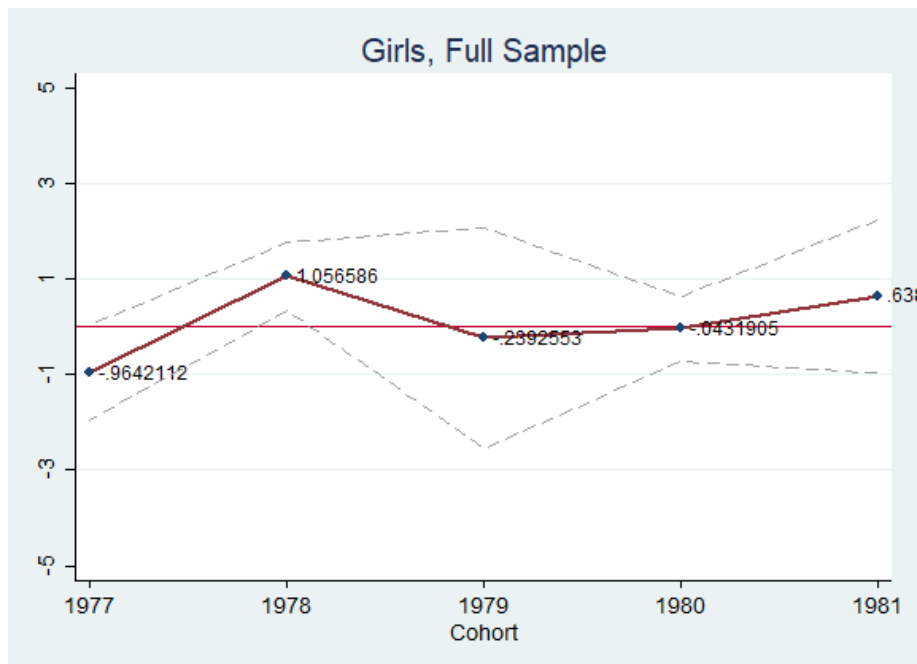
Figure 5a: the impacts of famine exposure on latent factor 4, boys in the full sample.



Source: see figure 4



Figure 5b: the impacts of famine exposure on latent factor 4, girls in the full sample.

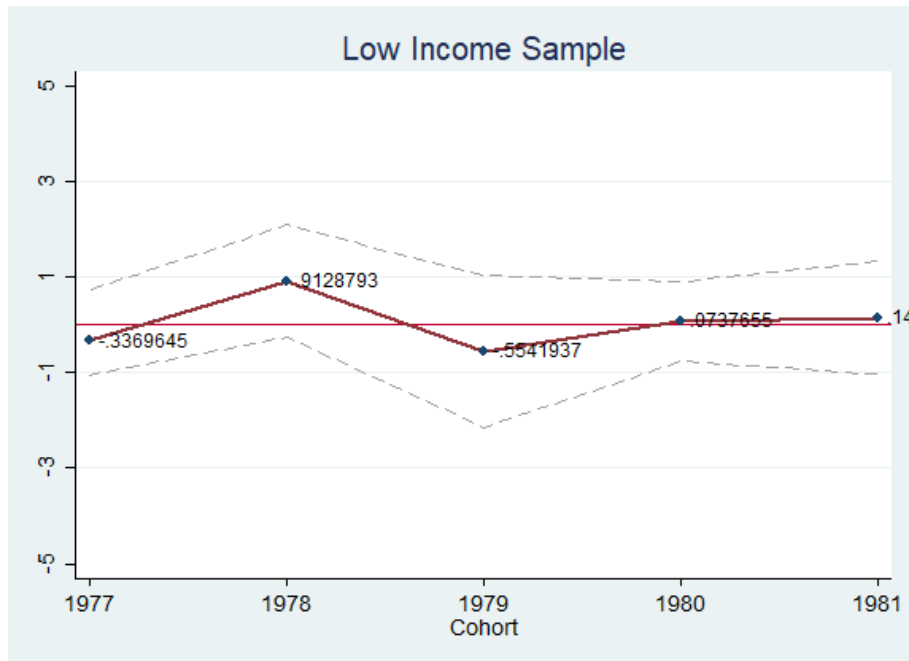


Source: see figure 4

If we compare the un-gendered results for those households with above and below-median income, shown in figure 6, it appears that low income samples appear to be notably muted in comparison to those in the high-income sample. Among low-income households no pattern across cohorts appears with regards to the pattern of coefficients and no effects are distinguishable from zero. In the high-income sample, all coefficients bar that for the 1981 cohort are significant at least at the 5% level. Looking from the 1977 cohort onwards, we see a strong compensatory effect for the oldest cohort, with the coefficients rising in value to 0.81 for the 1978 cohort, reaching a peak of 2.7 for the 1979 cohort, before falling toward the 1981 cohort. With separate male and female treatment effects (shown in table 32a), we see that impacts for girls in the low income sample are either below or close to zero, while a similar pattern to the full sample is seen for boys, with coefficients being positive and somewhat large for all cohorts except that from 1981. The major difference with the full sample male results is that these coefficients are significantly less efficiently estimated; despite being reasonably large in magnitude, none but that of the 1978 cohort are statistically significant. It appears that the heavily muted results in the un-gendered model stem from competing effects for girls and boys. Boys in the high-income sample appear to show a very different pattern to those in other subsamples. We see particularly strong reinforcement effects for the 1979 and 1980 cohorts and to a lesser extent the 1981 cohort, in contrast to elsewhere the 1978 cohort appears to see a compensation effect which is over 1 standard deviation of the latent factor's distribution in

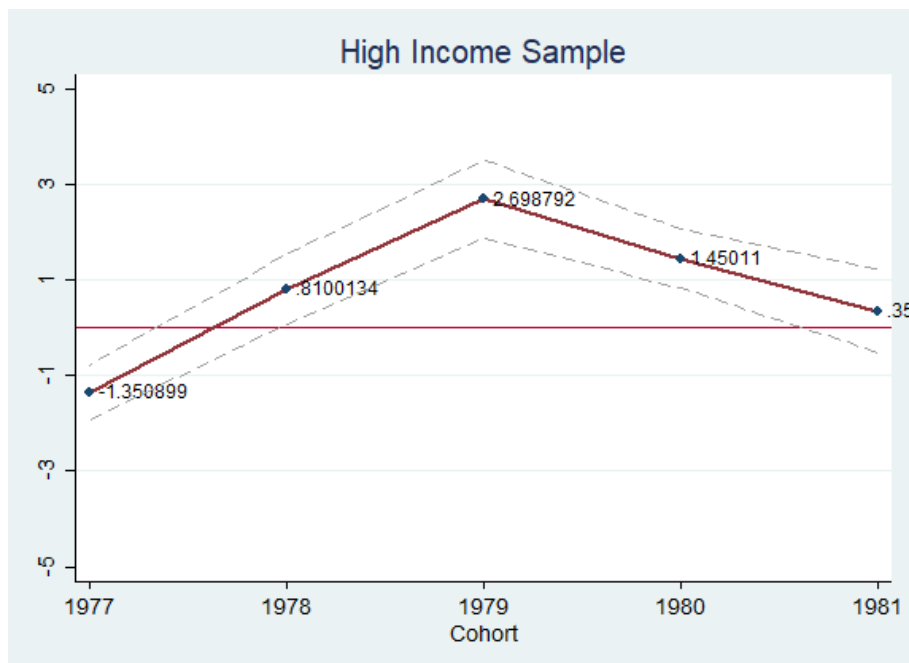
magnitude. Girls in high income households also appear to have been treated differently to elsewhere, with them seeing large reinforcement effects among the 1978, 1979 and 1981 cohorts. It should be noted again that these effects are not particularly efficiently estimated.

Figure 6a: the impacts of famine exposure on latent factor 4, low income sample.



Source: see figure 4

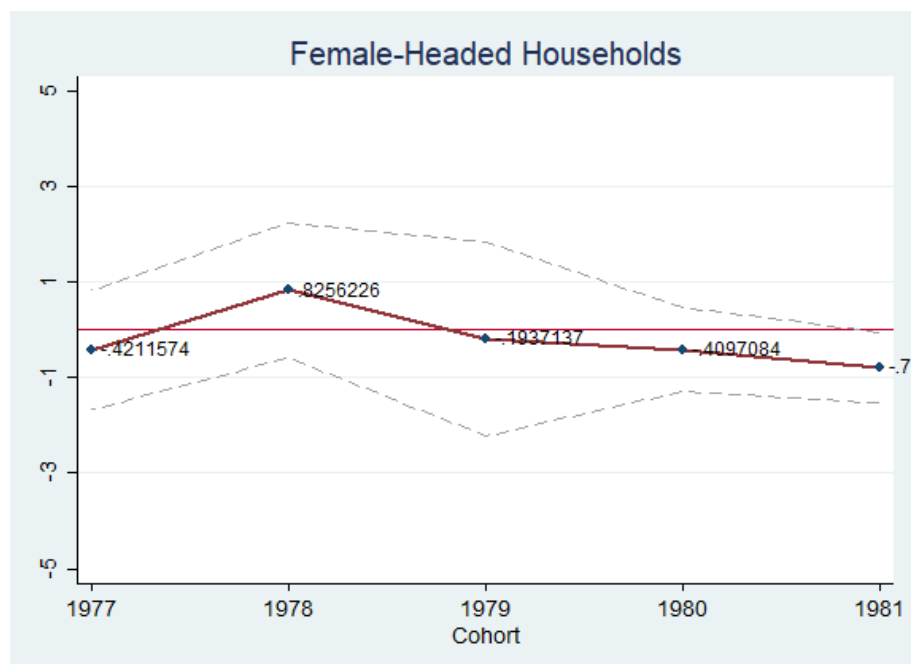
Figure 6b: the impacts of famine exposure on latent factor 4, high income sample.



Source: see figure 4

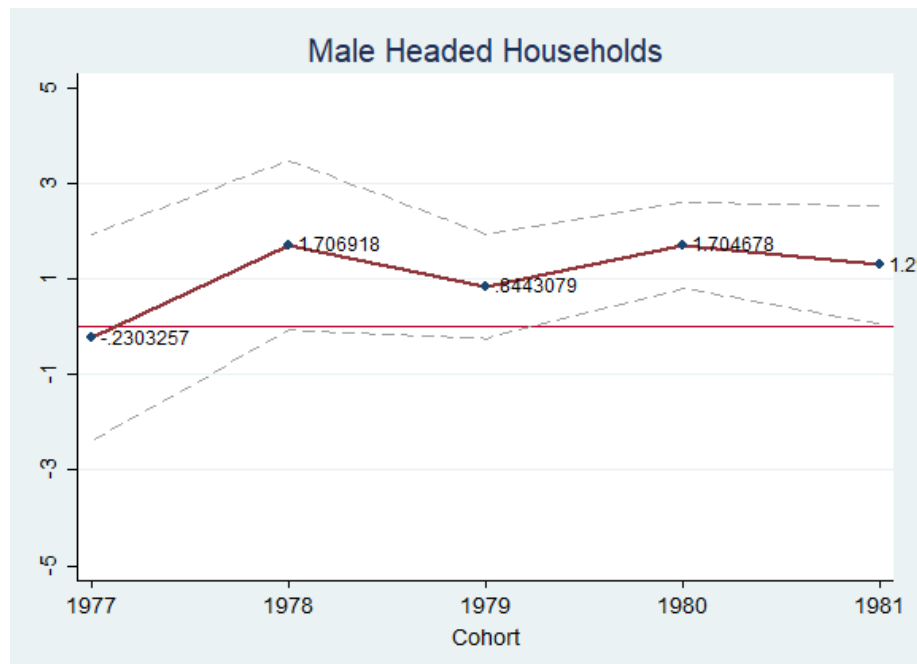
Major differences also appear between male and female-headed households (figure 7). Taking boys and girls together, all but one treatment effect for children in female headed homes are negative and largely insignificant, bar the coefficient for the 1981 cohort. In male headed households, however, we see reinforcement effects for all cohorts born after 1977, with these effects being statistically significant and greater than one standard deviation of the latent factor in magnitude for the 1978, 1980 and 1981 cohorts. In allowing effects to be different for boys and girls, it can be seen that in female headed households, female coefficients tend to be more strongly negative in value compared to the results shown in figure 7, though these compensatory effects are only statistically significant for the 1981 and 1977 cohort. If any effects are felt for girls in male headed households, they are more likely to be reinforcement effects, with all coefficients bar that for the 1977 cohort turning up positive, though it should be noted that none but the coefficient for the 1980 cohort are estimated efficiently enough to be differentiable from zero. For boys in female headed households, effects are largely statistically insignificant. The point estimates are somewhat indicative of reinforcement for the older parts of the treatment group and then turn compensatory for the 1980 and 1981 cohorts. In male headed households, the effects for the oldest and youngest cohorts are very close to zero. However, for those born between 1978 and 1980, the point estimates are positive and quite large, with that for 1980 coming in at around 3 standard deviations of the distribution of the latent factor's distribution.

Figure 7a: the impacts of famine exposure on latent factor 4, female-headed households.



Source: see figure 4

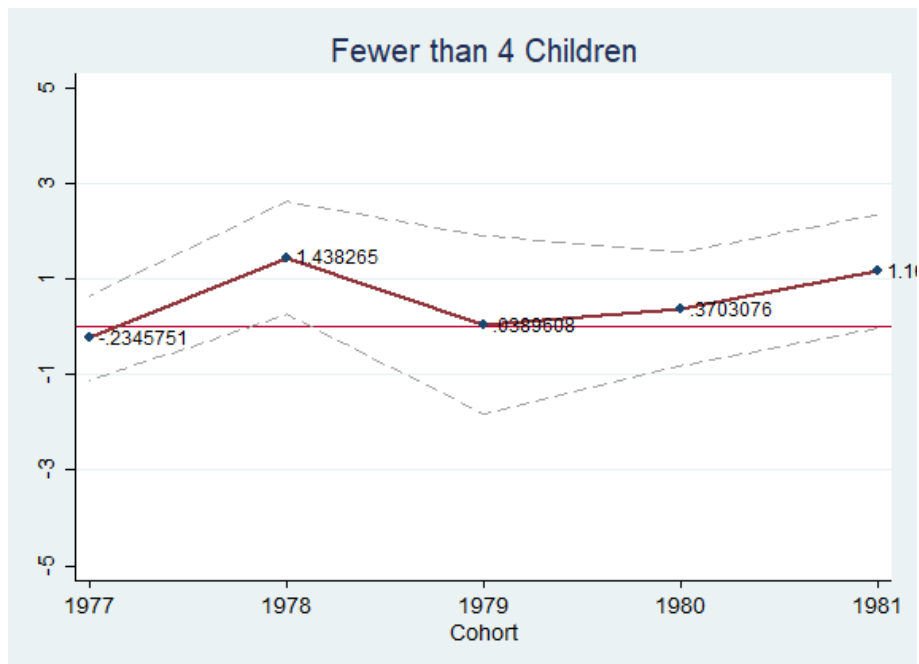
Figure 7b: the impacts of famine exposure on latent factor 4, male-headed households.



Source: see figure 4

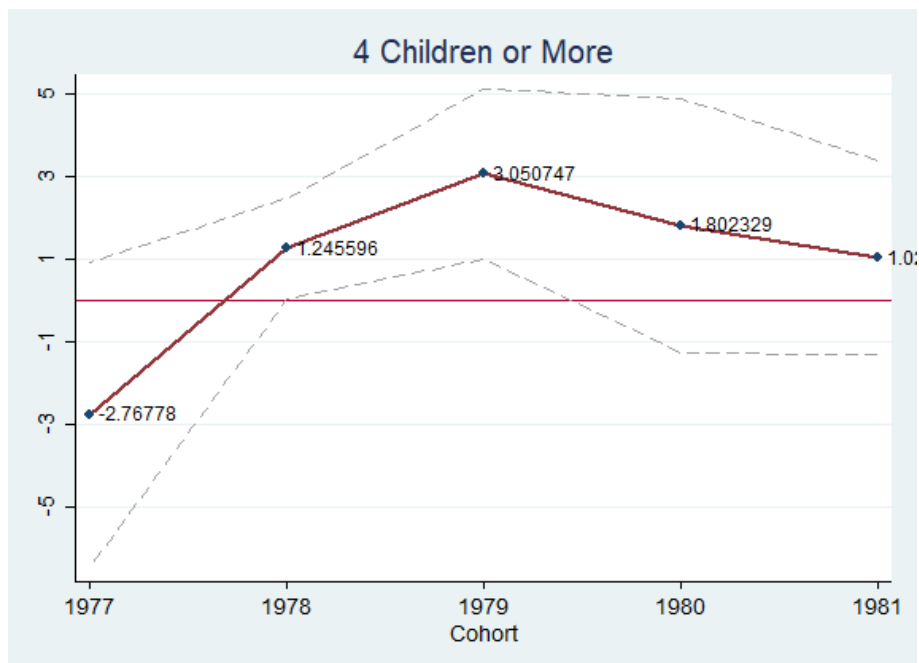
Finally, moving towards the comparison of households with fewer than 4 children and those with 4 or more children, illustrated in figure 8, it can be seen that treatment effects are much more varied in the large family sample. Among households with more than 4 children, a large but inefficiently estimated negative effect can be seen for the 1977 cohort, while all other effects are positive and at least 1 standard deviation of the distribution of  $f_4$  in magnitude, though they are only significant for the 1978 and 1979 cohorts. In smaller families, effects are largely closer to zero with the exceptions of the 1978 and 1981 treatment effects, both of which indicate reinforcement. These patterns are largely the same when the sample is split by gender; it can essentially be seen that in small families, male treatment effects veer somewhat closer towards reinforcement, while female effects fluctuate around zero. In larger households, the pattern of a stronger compensatory effect for the 1977 cohort and then reinforcement effects for the younger cohorts is born out for both boys and girls, the main difference being that the female treatment effects are smaller and statistically insignificant, while the male effects are larger in magnitude and more readily distinguishable from zero.

Figure 8a: the impacts of famine exposure on latent factor 4, smaller households.



Source: see figure 4

Figure 8b: the impacts of famine exposure on latent factor 4, larger households.



Source: see figure 4

## 7. Discussion

The results will now be discussed in relation to the theory presented in section 3. In that section, a number of predictions were made with regard to how parents may respond to the exposure of their children to the famine. Namely, that those households with higher incomes would be more likely to compensate for the effects of famine; that those children who experienced more severe shocks would be more likely to see reinforcement; that female household heads would be more likely to compensate and that larger households would be more likely to reinforce the famine's effects.

The evidence presented by the empirical model is not particularly in line with the first prediction. When taking boys and girls together, we see that the tendency to reinforce is much stronger for households with incomes above the median of the sample. When the effects for boys and girls are done separately, we see that boys and girls from both high and low income households appear to see both compensation and reinforcement, but that the impacts in either direction appear to be somewhat stronger and more reliably estimated in households with above-median income. It thus appears that the division between high and low income households is not along the lines of whether they compensate or reinforce, but more that when higher income households in the sample did respond, they did it more consistently and to a greater degree than lower income households. It should be kept in mind, however, that the definition of 'high' and 'low' income in this sample is somewhat different to what would be considered high and low income in other contexts. In the high income sample, the mean yearly income is the equivalent of around \$3470, while the median is \$2228, implying that the mean high income household would have to survive on \$9.50 per day and that the median would have to make do with \$6.10 per day. This would certainly imply that even relatively well-off households here are often still living on very little and thus what is a relatively high-income household here would likely be a low-income household in many other contexts. With this in mind, it could well be that the reason for the lesser preponderance of effects in either direction in the low-income sample stems from an inability to reallocate any resources whatsoever. The median daily income in this subsample lies at \$1.74 per day. At such low levels, the reallocation of resources from one child to another could well push the child who saw resources moved away from them have their consumption fall below the levels required for survival.

These predictions are not so clearly born out in the data. The fact that the only cohort to see compensatory effects was that from 1977, the oldest in the treatment group, is consistent

with the prediction, however, the patterns among younger cohorts are not. For the most part, the cohorts which appear to see the greatest reinforcement effects are the 1978 and 1979 cohorts, while the effects seen for the 1980 or 1981 cohorts, those exposed in-utero, see fairly small impacts if any at all. If it were the case that those in these younger groups did indeed suffer the worst effects of the famine, then lesser reinforcement effects for them as compared with older groups could indicate that endowments and investments are not necessarily complementary to one another. It may also be that the assumption that those from these cohorts would display the most severe effects of the famine is incorrect. While it is well established that, when we think on the level of the individual, those exposed to a health-shock in-utero tend to be worst affected, this is not necessarily going to be reflected at the level of the cohort. Given that the Karamoja famine was a particularly high mortality event, a degree of survivor selection is likely at work in the sample. This would mean that amongst the worst affected groups, we would see larger numbers of those from the bottom of the health distribution missing in this sample, given that they were more likely to succumb to the effects of the famine. With this in mind, it may be that those in the 1980 and 1981 are more strongly selected from the upper parts of the endowment distribution than are those from older cohorts. If this selection effect is strong enough, even if those exposed in-utero did see worse health impacts from the famine we would not necessarily see stronger reinforcement effects at the current level of analysis.

The work of Umana-Aponte (2011) uncovers some evidence to suggest that survivor selection is likely to be at work here. The author presents some estimations where children exposed in-utero, between the ages of 0 and 2 or between 3 and 5 are compared with unexposed children either in the same region or elsewhere. Other estimates are calculated wherein those in the treatment group are only compared with their unexposed siblings, such that genetic and economic heterogeneity across households, factors which might explain selective mortality patterns, can be accounted for. In the second set of estimates, it appears that the long-term effects of the Karamoja famine were much stronger than those implied by the first, hinting to the effects of selective mortality. Given that the Latent Engel Curve Approach is a household-level technique, it is not possible to control for survivor selection in a similar way here. However, the results of Umana-Aponte do give some credence to the idea that the survivors from the 1981 and 1980 cohorts are more strongly positively selected than those from older cohorts.

While the patterns of coefficients across cohorts may not have been entirely consistent with the prediction that the most severely affected groups would see stronger reinforcement

effects, the differences between male and female coefficients are somewhat in line with this narrative. In every subsample of the data, boys saw stronger and more regular evidence of the reinforcement of the famine's effects on their endowments than did girls. In fact, while girls only saw 4 statistically significant instances of reinforcement across all cohorts and subsamples, boys saw 13 such effects. Given that boys are on average more severely affected than girls by early-life health shocks, the greater preponderance of reinforcement for them may be suggestive of health endowments being complementary to investments in the formation of human capital. As was discussed earlier, the model predicts that so long as parental inequality aversion is weakly expressed, as would likely be the case in a sample with such low average incomes (Griliches 1979), worsened health endowments should be associated with lesser parental investments only when endowments and investments are complementary to one another.

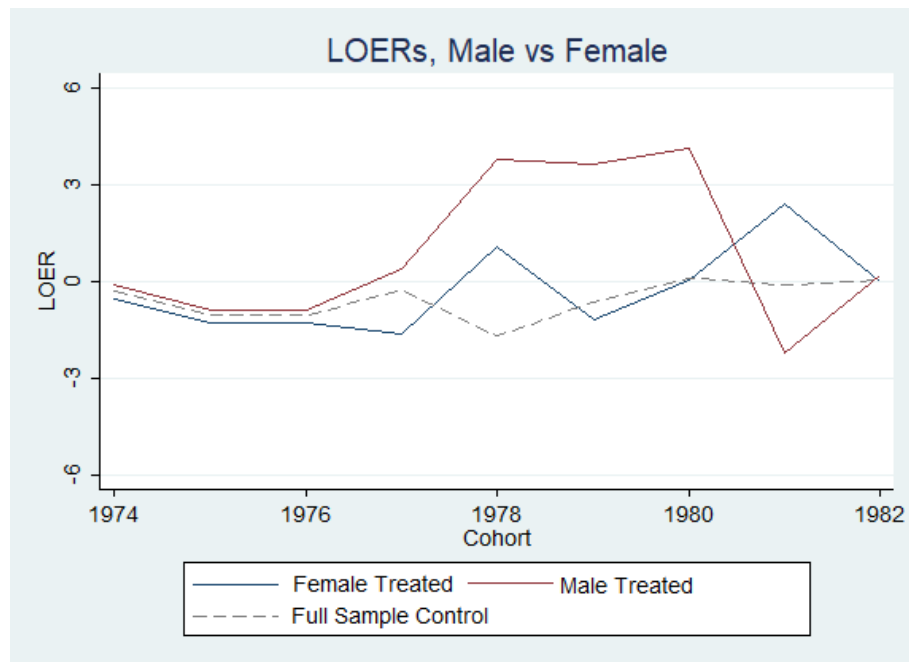
The topic of gender inequality arises from this discussion. The Latent Engel Curve approach and its predecessor of course arose as means of measuring boy-girl discrimination in intra-household resource allocations. With boys and girls seeing largely different investment responses to the famine, exposure will no doubt have had some effect on the relative quantities of resources being allocated to boys and girls. Figure 9 shows series of Latent Outlay Equivalent Ratios, one for the male treatment group, one for the female and one showing the ratios for an ungendered control group. The higher is the line, the fewer resources are devoted to the group associated with the line. Thus, when the boys' series is higher than that for girls, boys are receiving a lesser share of household resources. As can be seen from the figure, the boys and girls from the cohorts older than the treatment group see very little difference with regards to resource allocations. This is in line with the findings of Stites et al (2007) who, in a study of the Karamojong, found no evidence of gender discrimination with regards to food, medical care or access to education. The estimated LOERs for boys begin to take decidedly higher values when we look to those cohorts who were exposed at critical ages, the only exception being the 1981 cohort, for whom the opposite is true. These estimates would imply that in the case of the Karamoja famine, exposed cohorts see something of a bias in favour of girls with regards to the intra-household allocation of resources.

It is worth noting that this does not mean in any way that girls were made better off in the absolute sense, any extra investment they receive was of course came as a means of compensating for the welfare-loss generated by famine exposure. Further, the likelihood of similar boy-girl differences being found elsewhere is dependent upon boys not being favoured



in household resource allocations in the absence of famine exposure, which may not necessarily be the case.

Figure 9: Latent Outlay Equivalence Ratios, Boys and Girls



Source: author's own calculations

Continuing along the axis of gender, it was also predicted that female household heads would be more likely to compensate for the famine's impacts than male ones. This prediction was largely supported by the estimated results. When looking at the female-headed sample and taking both genders together, only the 1978 cohort has a treatment effect which could be interpreted as implying reinforcement. The opposite is true in the male-headed sample, with only one statistically significant instance of compensation for the 1977 cohort. Estimating differing treatment effects for the two genders shows that girls in female headed households certainly experienced some degree of compensation. No statistically significant compensation effects are seen for boys, however the positive coefficients for them are far smaller compared with those in the male-headed sample, implying that if they did experience reinforcement, it was to a lesser degree if they resided in a female-headed household. Assuming that it is true that female household heads on average exhibit greater inequality aversion than male heads, these results may suggest that, as we would expect, inequality aversion incentivises parents to compensate for, rather than reinforce the distribution of individual endowments.

The assumption that the differing results between male and female headed households is driven by differences in inequality aversion between the two groups should be taken with a

pinch of salt. There does exist experimental evidence to suggest that in resource allocation games, women exhibit a greater degree of altruism than men (Eckel and Grossman 1998). Extensions to these experiments have shown that women are more likely to be altruistic when altruism is costly, but that men are more likely to display the trait when its cost is low (Andreoni and Vesterlund 2001). If endowments and investments are in fact complementary to one another and parents in the sample expect to rely on their children in their old age, the decision to compensate a child could be characterised as a costly altruistic decision; the parent would be potentially giving up expected welfare in their old age in favour of the welfare of the child. Thus, in this circumstance greater inequality aversion amongst women would be observationally equivalent to them showing greater altruism when making costly resource allocation decisions.

Finally, it was predicted that families with more children would reinforce the effects of the famine to a greater degree than would those families with fewer children. This prediction stems from the work of Aizer and Cunha (2012) who present a model wherein the initial endowment distribution within a household will be increasingly reinforced in larger families, so long as inequality aversion is moderate and endowments and investments are complements. When looking to the results here, it is not clear that this prediction was entirely born out in the data. While coefficients for those born between 1978 and 1981 were often more strongly indicative of reinforcement in large families than they were in small families, the large family sample also shows consistent evidence that the 1977 cohort sees compensatory effects of the famine. Although this cohort did see compensation across a number of subsamples in the data, if the prediction set out in section 3 were correct, we would expect to see this larger compensation effect in the sample of households with fewer than 4 children, rather than that with 4 or more children. So, if we examine the younger parts of the treatment group, it would appear that large families were more likely to reinforce the effects of famine than smaller ones. However, when taking all cohorts together, it seems as though larger families exhibited more varied responses in comparison to those with fewer children, a pattern which is also reflected in the larger standard errors associated with their estimated coefficients.

From the results presented, we do see a pattern emerging which is consistent with both the idea that famines may affect individual endowments and that these impacts elicit reactions from parents with regards to the allocation of resources between children. More specifically, the comparison of effects across boys and girls implies that there may be some complementarity between health endowments and parental investment. In looking to

differences between male and female-headed households, we see some signs that inequality aversion can increase the likelihood of the compensation of differences in endowments. While these implications are in line with findings in previous studies (see Behrman et al 1982, Aizer and Cunha 2012), they should not be taken as being wholly conclusive. After all, the results of comparing coefficients across cohorts and between high and low-income households were not entirely in line with what would be expected.

The most important implication of the above findings is that previous studies looking to uncover the long-term impacts of early-life health conditions are often measuring the net-impact of multiple effects. The pure health channel will certainly be important with regards to understanding these impacts, but these results provide evidence to suggest that some of the long-term effects of such shocks may be generated by parental responses. Applied to this case, Umana-Aponte (2011) finds, using surveys taken roughly 10 years after that used in this study, that school attendance rates, literacy rates and years of completed education were reduced for affected cohorts, with these impacts often being more severe for boys than for girls. It is also uncovered that exposed individuals have lower weight-for-height scores and are more likely to be anaemic, especially those exposed in-utero. It is entirely possible that some portion of these impacts may be accounted for in part by parental responses as opposed to the pure health effects of the famine. The fact that boys tended to perform worse with regard to education measures is consistent with the evidence presented here showing that this group saw relatively more reinforcement with regard to the famine's effects.

While the evidence here is certainly useful in forming a fuller picture of how differences in early-life health may lead to different outcomes later in life, it does come with a conceptual caveat; that parental investment or the intra-household allocation of resources has largely been treated as a singular object. That a child can only receive more or fewer resources, rather than having parents able to invest in children across a number of different axes. Some effort was made to specifically show the impacts of famine on educational investments in section 4 and these results did appear largely in-line with those stemming from the main empirical section, but it is unclear as to whether parental investment decisions will differ over different axes. Developments in the literature on human capital have begun to emphasise the importance of the interactions between different forms of ability and attributes, with human capital being increasingly understood as an emergent property of cognitive and non-cognitive abilities and health, all of which are influenced by both genetic and environmental factors, as well as investment decisions across the lifespan (see Cunha and Heckman 2007; Heckman 2007;

Cunha et al 2010 ). It is clear that the information provided here will be of limited explanatory power in the context of these more sophisticated frameworks. The estimates here are essentially just measures of the net impact of famine exposure across all dimensions of child investment, as opposed to containing information regarding any particular investment path. For this channel of the impact of health shocks to be fully grasped, further investigation into the effect of health shocks on differing streams of parental investment will be required.

## **8. Conclusion**

This paper aims to estimate the impact of the Karamoja famine of 1980 on the allocation of resources toward exposed children, theorising that the famine's impacts on these children's health endowments would force parents to change their child-investment decisions in the interest of maximising household welfare. We see that there certainly were investment responses to the famine, with children exposed below the age of 3 being more likely to see the effects of the famine reinforced, while those exposed between the ages of 3 and 4 saw some of the impacts compensated for. Boys appeared to experience more strongly reinforcing investment responses, while girls were somewhat more likely to see compensatory responses, if any at all. Resource allocations tended to see more accentuated responses in those households with incomes above the median in the sample, as well as those households with greater numbers of children. It is also noted that female household heads were more likely than males to compensate for the effects for the famine. Many of these conclusions appear to be somewhat in line with the theory presented in the literature on the interaction of individual endowments and the intra-household allocation of resources. The patterns uncovered here, especially in the comparison of male and female headed households are consistent with the idea that parental inequality aversion dampens reinforcing investment responses. Further, the patterns of coefficients across genders and to a slightly lesser extent, across ages imply that those who likely felt more severe health impacts from the famine also saw more reinforcing responses from their parents, which is consistent with the idea that individual endowments and parental investment are complementary in the production of human capital.

The results presented here provide useful information to the literature regarding the long-term consequences of major shocks such as famines and epidemics. They imply that while the pure health channel will certainly be important in the generation of these long-term impacts, the household-level responses to the exposure of children to these shocks will also have some explanatory power. With studies of the long-term effects of shocks often uncovering

differences across genders and age groups, the differences in parental responses across these axes may be particularly salient. In a study of the impacts of the famine in question, Umana-Aponte (2011) finds that female outcomes were less responsive to exposure than male ones. With girls in this study seeing more compensatory responses to the famine, it may be that some of these patterns can be explained by parental investment decisions. With the findings presented here being in line with these results, it can be seen that the household has the potential to play an important dampening role when it comes to the consequences of major shocks.

The paper also overcomes several empirical challenges with regard to the measurement of the relationship between individual endowments and the intra-household allocation of resources. Here, famine exposure provides quasi-exogenous variation in the endowment distribution, whilst the application of the Latent Engel Curve Approach highlights a means of measuring variations in household resource allocations using data from household surveys.

In order to build upon these findings, means of measuring the impact of shock exposure on different channels of parental investment will bring results more in line with the modern theory of human capital formation presented by authors such as Cunha and Heckman (2007), Heckman (2007) Cunha et al (2010), as well as in-line with the empirical results of Yi et al (2015). Additionally, further innovations to the Latent Engel Curve Approach may provide more intuitively interpreted results, allowing for a greater understanding of what proportion of the long-term effects of famines such as this are generated by the shock itself and what proportion is accounted for by the responses of households.

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## Appendix

### Appendix A: Technical Appendix

#### A.1. Ahn and Horenstein (2013) Tests:

The eigenvalue-ratio test compares the  $r$  largest eigenvalues of the matrix  $YY'/(HG)$ , recommending the choice of  $r$  which satisfies:

$$\operatorname{argmax}_r[ER(r)] \equiv \operatorname{argmax}_r \left( \frac{\tilde{\mu}_{HG,r}}{\tilde{\mu}_{HG,r+1}} \right), \quad r \in \{r_{min}, \dots, r_{max}\}$$

Where  $\tilde{\mu}_{HG,r}$  is the  $r$ th largest eigenvalue of the matrix  $YY'/(HG)$ .

The growth-ratio test examines the growth rates of residual variances as one fewer principal component is used:

$$\operatorname{argmax}_r[GR(r)] \equiv \operatorname{argmax}_r \frac{\ln[V(r-1)/V(r)]}{\ln[V(r)]/\ln[V(r+1)]} = \operatorname{argmax}_r \frac{\ln(1 + \tilde{\mu}_{HG,r}^*)}{\ln(1 + \tilde{\mu}_{HG,r+1}^*)}$$

Where  $V(r) = \sum_{j=r+1}^m \tilde{\mu}_{HG,j}$ ,  $\tilde{\mu}_{HG,r}^* = \tilde{\mu}_{HG,j}/V(r)$  and  $m = \min(G, H)$ . Here,  $V(r)$  is equivalent to the mean of the squared residuals from the *within-household* regressions of expenditure shares on the first  $r$  principal components.

#### A.2. Algorithm for the consistent estimation of factors and loadings:

In order to estimate the factor model consistently and efficiently, the loadings,  $P$ , and covariance matrix,  $\Sigma_\varepsilon$ , must first be estimated by penalised maximum likelihood. This relies on the maximisation of the following quasi-likelihood function:

$$L(P, \Sigma_\varepsilon, S_f) = \frac{1}{G} \ln |Det(PS_f P' + \Sigma_\varepsilon)| + \frac{1}{G} \operatorname{tr}(S_y(PS_f P' + \Sigma_\varepsilon)^{-1})$$

Where  $S_f$  and  $S_y$  are the sample variances of the latent factors and observed expenditure shares. Maldonado's procedure for maximising  $L(\cdot)$  is as follows:<sup>4</sup>

1. At iteration  $i = 0$ , we need some starting values for  $P$  and  $\Sigma_\varepsilon$  which can be denoted as  $\hat{P}_0$  and  $\hat{\Sigma}_{\varepsilon,0}$ . Their PCA estimates are used.

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<sup>4</sup> The algorithm in Maldonado (2019) is very slightly different to that in Bai and Liao (2016). During the third step, Bai and Liao (2016) recommend the use of a penalty function for the off-diagonal values of  $\Sigma_\varepsilon$ . This is not included in Maldonado's algorithm, given that with smaller values of  $G$ , it is not necessary to assume that  $\Sigma_\varepsilon$  is sparse.

2. At iteration  $i + 1$ ,  $\hat{\Sigma}_{y,i} = \hat{P}_i \hat{P}_i' + \hat{\Sigma}_{\varepsilon,0}$ ,  $\hat{P}_{i+1} = AM^{-1}$ , with  $M = \hat{P}_i' \hat{\Sigma}_{y,i}^{-1} S_y \hat{\Sigma}_{y,i}^{-1} \hat{P}_i + I_4 - \hat{P}_i' \hat{\Sigma}_{y,i}^{-1} \hat{P}_i$  and  $A = S_y \hat{\Sigma}_{y,i}^{-1} \hat{P}_i$ .
3. At iteration  $i + 1$ ,  $\hat{\Sigma}_{\varepsilon,i+1} = \hat{\Sigma}_{\varepsilon,i} - t(\hat{\Sigma}_{y,i}^{-1} S_y \hat{\Sigma}_{y,i}^{-1})$  where  $t > 0$ .<sup>5</sup>

Repeat steps 2 and 3 until convergence.

Once consistent estimates of  $P$  and  $\Sigma_\varepsilon$  have been delivered, the FGPCA estimator of the factors is given by:

$$\hat{f}_r = (\hat{P}' \hat{\Sigma}_\varepsilon^{-1} \hat{P})^{-1} \hat{P}' \hat{\Sigma}_\varepsilon^{-1} (y_h - \bar{y})$$

Where  $\bar{y} = \frac{1}{H} \sum_{h=1}^H y_h$ .

### A.3. JADE Algorithm

In order to uniquely identify the individual columns of the factor matrix  $F$ , the JADE algorithm first takes the estimates from the Penalised-MLE/FGPCA algorithm. At the level of the household we will have a 1 by four vector, which will be denoted  $\hat{f}_h$ . The JADE algorithm finds the orthogonal  $R$  by  $R$  (4 by 4) matrix  $\hat{U}$  which generates the  $\tilde{f}_h = \hat{U}' \hat{f}_h$  which is maximally non-gaussian. To ensure that the columns are independent of one another, the algorithm exploits the fact that a set of random vectors are independent of one another when the coefficients of the Taylor expansion of the logarithm of the moment-generating function (the cross-cumulants) of order 2 or higher are equal to zero (Barigozzi and Moneta 2016).

Take the matrix  $\hat{F}$ , which was estimated by Maldonado (2019)'s penalized maximum likelihood/FGPCA algorithm, its cumulant-generating function is given by:

$$K(\zeta) = \log E[\exp(\zeta' F)]$$

Cardoso and Souloumiac (1993) focus on the coefficients of the fourth-order terms of the Taylor expansion of  $K(\zeta)$  in the neighbourhood of  $\zeta = 0$ . When  $E[F] = 0$ :

$$k_{ijkl} = E[f_i f_j f_k f_l]$$

There are  $R^4$  of these 4<sup>th</sup> order cumulants – 256 in our case. These objects make up the entries of an  $R^2$  by  $R^2$  matrix (16 by 16), from which the  $R^2$  eigenvectors are extracted and transformed

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<sup>5</sup> In both Bai and Liao (2016) and Maldonado (2019),  $t=0.1$ . It is kept to that same value here.

into the matrix  $V_i$  which will be of the smaller dimensions of R by R (4 by 4). The algorithm then finds the R by R matrix  $\hat{U}$  which satisfies:

$$\hat{U} = \underset{V}{\operatorname{argmin}} \sum_{i=1}^{R^2} \operatorname{off}(V'V_iV)$$

Where  $\operatorname{off}(X)$  takes the off-diagonal elements of the matrix X.

## Appendix B: Figures and Tables

### B.1. Attachments from Section 4.

Table B11: Impacts of Famine Exposure on Education Expenditures

VARIABLES	(1) Male	(2) Female
K <sub>1981</sub>	0.172 (0.201)	0.0313 (0.517)
K <sub>1980</sub>	-0.736*** (0.109)	0.536*** (0.147)
K <sub>1979</sub>	-0.473 (0.310)	0.346*** (0.0593)
K <sub>1978</sub>	-0.204 (0.251)	-0.315*** (0.0618)
K <sub>1977</sub>	-0.292*** (0.0931)	0.741*** (0.0997)
Birthyear FE	Y	Y
District FE	Y	Y
Observations	3,695	3,270
R-squared	0.988	0.986

Robust standard errors in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

*B.2. Attachments from Section 5.*

Table B21: Factor Loadings

	F1	F2	F3	F4
Potatoes	-1.54E-09	-1.26E-08	9.19E-09	-1.89E-08
Cereals	0.000187	1.39E-05	2.83E-05	-3.40E-05
Meat	5.08E-06	6.01E-06	-4.66E-06	9.27E-06
Fish	1.05E-05	7.07E-07	-4.66E-06	2.57E-06
Dairy	2.18E-06	2.44E-06	-2.00E-07	5.43E-06
Oil	2.75E-06	1.59E-06	-5.14E-07	2.77E-06
Vegetables	5.76E-06	1.04E-06	-6.13E-07	1.63E-06
Legumes	1.91E-05	-2.88E-06	1.68E-06	-6.86E-06
Tea & Coffee	6.45E-06	4.27E-06	-4.44E-06	6.97E-06
Other Foods	-8.60E-06	-4.06E-06	-1.20E-05	-7.80E-06
Non-Alcoholic Drinks	4.09E-08	3.63E-07	1.22E-07	9.30E-07
Alcoholic Drinks	1.78E-05	1.14E-06	3.24E-06	6.83E-06
Tobacco	2.09E-06	1.62E-07	1.25E-07	1.98E-06
Restaurants	-5.65E-06	-3.29E-06	2.80E-06	-3.04E-06
Non-Durables	-1.15E-05	-2.31E-06	-1.77E-05	-5.11E-06
Men's Clothes	-4.27E-06	1.38E-06	-1.76E-06	4.35E-06
Women's Clothes	-6.27E-06	1.84E-06	-5.18E-06	5.09E-06
Children's Clothes	-2.31E-06	8.92E-07	-2.59E-06	1.88E-06
Tailoring	-2.47E-07	1.44E-07	-2.80E-08	4.16E-07
Shoes	-7.67E-07	1.26E-06	-2.04E-08	3.08E-06
Small Furniture	-1.56E-06	1.22E-06	-1.11E-06	3.35E-06
Personal Goods	6.23E-08	1.94E-07	2.06E-07	6.06E-07
Electricity	-8.88E-05	-1.89E-05	-0.00013	-5.66E-05
Fuel	-4.51E-06	-1.09E-06	-9.90E-06	-2.01E-06
Kitchen Utensils	-8.74E-07	6.24E-07	-1.14E-06	1.48E-06
Vehicle Parts	-6.85E-07	6.67E-07	6.73E-08	2.02E-06
Communications	-1.20E-06	5.53E-06	9.03E-07	-2.94E-06
Recreation	-6.07E-06	-5.10E-08	-1.60E-06	9.47E-07
Domestic Servants	-3.72E-07	7.51E-07	-1.15E-07	-2.22E-07
Personal Care	-1.57E-06	2.25E-07	-4.58E-07	3.06E-07
Accommodation	-2.47E-07	5.05E-07	1.37E-07	1.11E-07
Large Furniture	-1.02E-06	1.03E-06	4.26E-07	2.79E-06
Vehicles	-4.12E-07	6.57E-08	1.20E-06	3.84E-06
Recreation Equipment	-1.08E-06	1.57E-06	5.98E-07	1.52E-06
Jewellery	-1.11E-06	-1.95E-08	-2.07E-07	5.24E-07
Misc. Expenditure	-0.00012	3.97E-05	0.000111	0.000128

Appendix B.3. Attachments from Section 6.

Table B31a: Ungendered Results, Treatment Effects

VARIABLES	(1) Full Sample	(2) Lower Income	(3) Higher Income	(4) Female Headed	(5) Male Headed	(6) <4 Children	(7) >3 Children
Treat. Effect 1981	0.393 (0.592)	0.145 (0.577)	0.352 (0.425)	-0.791** (0.363)	1.293** (0.603)	1.161* (0.580)	1.027 (1.148)
Treat. Effect 1980	0.460 (0.326)	0.0738 (0.406)	1.450*** (0.304)	-0.410 (0.427)	1.705*** (0.439)	0.370 (0.580)	1.802 (1.499)
Treat. Effect 1979	0.236 (0.893)	-0.554 (0.776)	2.699*** (0.397)	-0.194 (0.991)	0.844 (0.532)	0.0390 (0.910)	3.051*** (1.003)
Treat. Effect 1978	1.186*** (0.215)	0.913 (0.573)	0.810** (0.356)	0.826 (0.689)	1.707* (0.867)	1.438** (0.577)	1.246** (0.593)
Treat. Effect 1977	-0.155 (0.599)	-0.337 (0.522)	-1.351*** (0.280)	-0.421 (0.610)	-0.230 (1.059)	-0.235 (0.432)	-2.768 (1.791)
District FE	Y	Y	Y	Y	Y	Y	Y
Observations	7,063	3,477	3,586	1,976	5,087	4,934	2,129
R-squared	0.108	0.107	0.128	0.115	0.112	0.108	0.122

Robust standard errors in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1



Table B31b: Ungendered Results, Other Parameters

VARIABLES	(1) Full Sample	(2) Lower Income	(3) Higher Income	(4) Female Headed	(5) Male Headed	(6) <4 Children	(7) >3 Children
ln(y)	0.311*** (0.0356)	0.276*** (0.0456)	0.378*** (0.0418)	0.290*** (0.0423)	0.313*** (0.0393)	0.298*** (0.0351)	0.336*** (0.0479)
ln(N)	0.236*** (0.0262)	0.209*** (0.0519)	0.310*** (0.0356)	0.157*** (0.0558)	0.252*** (0.0342)	0.213*** (0.0403)	0.290*** (0.0698)
$\gamma(>1981)$	0.0579 (0.0802)	0.0489 (0.116)	0.0510 (0.228)	0.271 (0.160)	-0.0661 (0.112)	0.0717 (0.0963)	0.412 (0.392)
$\gamma(1981)$	0.0130 (0.199)	0.373 (0.301)	-0.344 (0.411)	0.311 (0.351)	-0.0748 (0.202)	0.0298 (0.240)	0.316 (0.435)
$\gamma(1980)$	0.0823 (0.240)	0.188 (0.336)	-0.106 (0.333)	0.410 (0.450)	-0.0651 (0.223)	-0.131 (0.342)	0.850 (0.587)
$\gamma(1979)$	-0.149 (0.245)	0.182 (0.308)	-0.400 (0.340)	0.263 (0.307)	-0.298 (0.346)	-0.134 (0.311)	0.285 (0.438)
$\gamma(1978)$	-0.479** (0.227)	-0.150 (0.212)	-0.817* (0.435)	-0.265 (0.586)	-0.487** (0.193)	-0.335* (0.179)	-0.341 (0.784)
$\gamma(1977)$	-0.0376 (0.251)	-0.0776 (0.348)	-0.0426 (0.385)	0.591 (0.462)	-0.358* (0.177)	0.0121 (0.275)	0.164 (0.567)
$\gamma(1975-1976)$	-0.280 (0.209)	0.0783 (0.288)	-0.647 (0.432)	0.0314 (0.273)	-0.352 (0.244)	-0.249 (0.258)	-0.0701 (0.422)
$\gamma(1933-1974)$	-0.0441 (0.0570)	-0.0102 (0.0676)	-0.0785 (0.189)	-0.00807 (0.165)	-0.111* (0.0587)	-0.0236 (0.0581)	0.245 (0.427)
$\phi(1975-1976)$	-0.180 (0.293)	-0.196 (0.417)	-0.155 (0.462)	0.123 (0.554)	-0.361 (0.380)	-0.0912 (0.335)	-0.442 (0.541)
Urban	-0.427*** (0.0551)	-0.420*** (0.0682)	-0.425*** (0.0678)	-0.401*** (0.0870)	-0.423*** (0.0559)	-0.454*** (0.0548)	-0.350*** (0.0843)

Robust standard errors in parentheses  
\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table B32a: Gendered Results, Treatment Effects

VARIABLES	(1) Full Sample	(2) Lower Income	(3) High Income	(4) Female Headed	(5) Male Headed	(6) <4 Children	(7) >3 Children
Female 1981	0.639 (0.787)	0.423 (0.723)	1.429 (1.411)	-1.913*** (0.535)	1.411 (0.853)	1.024 (0.743)	1.197 (1.366)
Female 1980	-0.0432 (0.331)	-0.267 (0.443)	0.286 (0.812)	-0.423 (0.545)	0.703** (0.297)	-0.0471 (0.323)	0.694 (1.450)
Female 1979	-0.239 (1.127)	-1.157 (0.799)	3.839*** (0.502)	-0.652 (1.168)	0.408 (0.950)	-0.653 (1.025)	2.729 (1.652)
Female 1978	1.057*** (0.348)	0.0760 (0.631)	4.253 (2.581)	0.547 (0.650)	0.807 (0.516)	1.344*** (0.426)	0.997 (1.031)
Female 1977	-0.964* (0.487)	-1.173* (0.607)	-2.364** (1.033)	-1.334* (0.691)	-0.642 (0.869)	-0.922 (0.549)	-1.512 (1.011)
Male 1981	-0.529 (0.327)	-1.254** (0.529)	0.928 (0.917)	-1.059 (0.987)	-0.112 (0.979)	2.131*** (0.702)	1.442 (1.202)
Male 1980	1.236 (1.108)	0.712 (1.272)	4.283*** (1.396)	-0.277 (1.098)	3.210*** (0.508)	1.019 (1.496)	4.892*** (0.621)
Male 1979	1.353*** (0.485)	1.107 (0.784)	1.624** (0.606)	0.990* (0.523)	1.473** (0.555)	1.689*** (0.543)	2.349** (0.852)
Male 1978	1.493*** (0.325)	2.212* (1.122)	-1.284*** (0.404)	1.234 (1.114)	2.643 (1.664)	1.731 (1.179)	0.741 (0.894)
Male 1977	0.800 (0.806)	0.817 (0.797)	-0.173 (0.376)	0.410 (0.990)	0.398 (1.388)	0.533 (0.673)	-1.752 (2.577)
District FE	Y	Y	Y	Y	Y	Y	Y
Observations	7,063	3,477	3,586	1,976	5,087	4,934	2,129
R-squared	0.111	0.110	0.133	0.117	0.114	0.110	0.129

Robust standard errors in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table B32b: Gendered Results, Other Parameters

VARIABLES	Full Sample	Lower Income	High Income	Female Headed	Male Headed	<4 Children	>3 Children
ln(y)	0.310*** (0.0355)	0.276*** (0.0457)	0.376*** (0.0415)	0.291*** (0.0427)	0.311*** (0.0393)	0.297*** (0.0352)	0.338*** (0.0483)
ln(N)	0.240*** (0.0260)	0.210*** (0.0498)	0.319*** (0.0336)	0.191*** (0.0519)	0.272*** (0.0340)	0.221*** (0.0387)	0.296*** (0.0737)
$\gamma(>1981, F)$	0.0349 (0.0949)	0.0220 (0.129)	-0.00541 (0.245)	0.195 (0.193)	-0.130 (0.124)	0.0941 (0.119)	0.273 (0.379)
$\gamma(1981, F)$	0.152 (0.230)	0.432 (0.307)	-0.169 (0.441)	0.177 (0.378)	0.135 (0.264)	0.0318 (0.305)	0.671 (0.429)
$\gamma(1980, F)$	0.0921 (0.228)	0.270 (0.384)	-0.169 (0.466)	0.234 (0.514)	-0.0334 (0.308)	-0.0107 (0.308)	0.705 (0.629)
$\gamma(1979, F)$	-0.0871 (0.255)	0.220 (0.335)	-0.299 (0.413)	0.174 (0.274)	-0.191 (0.458)	-0.00536 (0.263)	0.247 (0.491)
$\gamma(1978, F)$	-0.690** (0.304)	-0.364 (0.362)	-1.119** (0.521)	-0.454 (0.668)	-0.757** (0.286)	-0.690** (0.272)	-0.335 (0.962)
$\gamma(1977, F)$	0.501 (0.438)	0.477 (0.598)	0.438 (0.488)	0.850 (0.603)	0.238 (0.394)	0.380 (0.521)	1.095 (0.707)
$\gamma(1975-1976, F)$	-0.361* (0.201)	-0.117 (0.322)	-0.615* (0.308)	-0.00698 (0.429)	-0.480** (0.214)	-0.393 (0.255)	-0.0540 (0.449)
$\gamma(1933-1974, F)$	-0.130* (0.0764)	-0.0429 (0.100)	-0.305 (0.206)	0.00321 (0.165)	-0.227*** (0.0821)	-0.114 (0.0704)	0.153 (0.548)

Robust standard errors in parentheses

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table B32b cont.

VARIABLES	Full Sample	Lower Income	High Income	Female Headed	Male Headed	<4 Children	>3 Children
$\gamma(>1981, M)$	0.0952 (0.0842)	0.105 (0.122)	0.0388 (0.205)	0.193 (0.201)	-0.0427 (0.123)	0.0746 (0.0850)	0.534 (0.448)
$\gamma(1981, M)$	-0.127 (0.265)	0.348 (0.470)	-0.625 (0.443)	0.325 (0.490)	-0.371 (0.328)	0.0316 (0.281)	-0.102 (0.553)
$\gamma(1980, M)$	0.0755 (0.375)	0.122 (0.460)	-0.124 (0.481)	0.450 (0.503)	-0.216 (0.409)	-0.248 (0.474)	1.066 (0.690)
$\gamma(1979, M)$	-0.186 (0.400)	0.166 (0.432)	-0.533 (0.512)	0.224 (0.541)	-0.460 (0.459)	-0.230 (0.513)	0.382 (0.586)
$\gamma(1978, M)$	-0.287 (0.304)	0.0444 (0.357)	-0.627 (0.493)	-0.253 (0.750)	-0.300 (0.261)	0.000906 (0.277)	-0.393 (0.779)
$\gamma(1977, M)$	-0.637** (0.261)	-0.743 (0.485)	-0.625 (0.400)	0.0214 (0.652)	-0.980*** (0.216)	-0.412 (0.300)	-0.968 (0.634)
$\gamma(1975-1976, M)$	-0.238 (0.266)	0.232 (0.364)	-0.871 (0.595)	0.0169 (0.360)	-0.333 (0.312)	-0.150 (0.332)	-0.319 (0.607)
$\gamma(1933-1974, M)$	0.00983 (0.0621)	0.0170 (0.0802)	-0.00120 (0.175)	-0.166 (0.214)	-0.0710 (0.0656)	0.0294 (0.0655)	0.276 (0.395)
$\varphi(1975-1976, F)$	-0.306 (0.374)	-0.398 (0.473)	-0.145 (0.451)	-0.384 (0.656)	-0.366 (0.478)	-0.266 (0.436)	0.00915 (0.714)
$\varphi(1975-1976, M)$	-0.134 (0.403)	-0.171 (0.505)	-0.0846 (0.697)	0.301 (0.923)	-0.325 (0.498)	-0.0135 (0.462)	-0.323 (0.696)
Urban	-0.425*** (0.0550)	-0.419*** (0.0677)	-0.420*** (0.0679)	-0.405*** (0.0879)	-0.424*** (0.0559)	-0.452*** (0.0551)	-0.355*** (0.0829)

Robust standard errors in parentheses

\*\*\* p&lt;0.01, \*\* p&lt;0.05, \* p&lt;0.1

<sup>i</sup> Belgen is the god of the Tepes or Sor, whose traditional home is close to Moroto, Karamoja. It is thought that when under attack, they can be imbued with powers which can bring famine upon their assailants (Knighton 2000).