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Macro Shocks and Micro(scopic) Outcomes: Child Nutrition During Indonesia's Crisis

Steven A. Block
(corresponding author)
Fletcher School of Law & Diplomacy
Tufts University
Medford, MA 02155
e-mail: steven.block@tufts.edu

Lynnda Keiss
Helen Keller International Asia-Pacific Regional Office

Patrick Webb
Friedman School of Nutrition Science and Policy
Tufts University

S. Kosen
National Institute for Health Research and Development
Ministry of Health, Government of Indonesia

Regina Moench-Pfanner
Helen Keller International Asia-Pacific Regional Office

Martin W. Bloem
Helen Keller International Asia-Pacific Regional Office

C. Peter Timmer
School of International Relations and Pacific Studies
University of California at San Diego

BAPPENAS/DEPARTMEN PERTANIAN/USAID/DAI FOOD POLICY ADVISORY TEAM

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Abstract

This study uses a new survey of households in rural Central Java to assess the nutritional impact of Indonesia's drought and financial crisis of 1997/98. Implementing a time/age/cohort decomposition of high frequency survey data reveals significant nutritional impacts. While there was no meaningful decline in child weight-for-age measures, mean weight-for-height declined by over one-third of a standard deviation. The most severe nutritional impact of the crisis was on micronutrient status, indicated by sharp declines in blood hemoglobin concentration. We also demonstrate the efficacy of applying econometric decomposition of time, age, and cohort effects to high frequency nutrition surveillance data.

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I. Introduction

Indonesia's dramatic economic collapse that began in 1997, generally referred to as a "financial" crisis, is more accurately portrayed as an interaction among three separate processes: a financial crisis with regional ramifications, a national political upheaval leading to a change of government, and a series of agroclimatic shocks linked to global climate events. The Indonesian economy shrank by 14 percent in 1998 alone as the meteoric depreciation of the Rupiah beginning in January 1998 interacted with a banking crisis and loss of investor confidence, to generate severe inflation (Radelet, 1999; Radelet and Sachs, 1998). This sudden and widespread economic chaos undermined the regime of President Suharto, who resigned in May 1998 in the wake of serious street rioting, leaving a political vacuum in his wake. In addition, most rural areas were also suffering a severe drought (and wildfires) linked to the *El Niño* phenomenon, which preceded and exacerbated the financial crisis.

For Central Java – the focus of this study – the period of lowest rainfall ran from February 1997 to January 1998 causing a serious reduction in the subsequent harvest (Gilligan, Jacoby, and Quizon, 2000). The production shortfall generated food shortages, which drove up food prices thereby contributing further to aggregate inflation. Indeed, January 1998 saw the highest monthly inflation in 24 years (6.9 percent), half of which resulted from a greater than 10 percent monthly increase in food prices (with rice alone accounting for 15 percent of *total* inflation) in that month. (Government of Indonesia, 1998).

Inflation in general, and food price inflation in particular, was driven by a combination of a supply shock resulting from drought and the collapse of the Rupiah, which increased the price of tradables. Table 1 (Friedman and Levinsohn, 2001) summarizes price changes for selected foods (in urban markets across 27 provinces) between January 1997 and October 1998, the critical period for our analysis.

Drought and financial crisis combined, primarily through their effect on food prices, to impose a negative shock on food consumption. Micronutrient intake is of particular concern in this context, as food price shocks may lead poor consumers to buffer their caloric intake at the expense of the *quality* of their diets in terms of micronutrient content. Micronutrient deficiencies can cause learning disabilities, impair work capacity, and have been associated with heightened morbidity and mortality – particularly among pre-school children and pregnant women (World Bank, 1994; Commission on Macroeconomics and Health, 2002).

Our results confirm that these concerns are well founded. Despite the apparent ability of rural households in Central Java to cope with the impact of the crises on the gross nutritional status of their young children – weight-for-age changed little during the episode – more subtle measures of nutritional status clearly deteriorated significantly, in both biological and statistical terms. In particular, blood hemoglobin concentrations dropped sharply during the crises, and had not returned to pre-crisis levels (low as they were) by early 2001. Indeed, the incidence of child anemia increased from a baseline of nearly 50 percent to over 70 percent during the peak of the crisis. Iron deficiency is particularly dangerous for children 6 to 18 months old, for whom there is evidence of permanent damage to mental and motor development, and negative consequences for schooling achievement and adult wages (Gillespie and Haddad, 2001). The crises thus had a significant impact on the micro-nutritional status of children in rural Central Java.

These nutritional effects were driven entirely by changes in food consumption patterns at the household (and individual) level. These, in turn, are driven by changes in family incomes and prices for various foods in local markets. The impact of changes in these economic variables may well be mediated by how well the home decision maker, usually the mother, understands the health consequences of reducing intake of nutrient-rich foods. Most mothers seemed to understand the importance of maintaining protein-energy intake, especially from rice, and thus gross nutritional status was largely maintained. Yet, only a subset of mothers seemed aware of the importance of micronutrient-rich foods, and their children were better protected from the crisis than others.

This paper examines the impact of these multiple crises on child nutrition in rural Central Java. Although the macroeconomics of Indonesia's "implosion" have been widely documented and analyzed, its household-level consequences have received relatively less attention. Several recent studies have used household data to assess real price effects of the crisis to derive improved deflators for measuring poverty (Levinsohn, Berry and Friedman, 1999; Suryahadi, et. al., 2000; Friedman and Levinsohn, 2001). One study examines the effects of the crisis on agricultural households, emphasizing income, production, and input demand effects (Gilligan, Jacoby, and Quizon, 2000). They find that Indonesia's multiple crises had mixed effects. Many agricultural households on the outer island experienced an income boom. Yet, per capita agricultural income on Java stagnated, and landless Javanese households were among the worst off in relative terms (Gilligan, Jacoby, and Quizon, 2000).

The impact of the crisis on nutritional outcomes has also been addressed in several studies, albeit with sometimes conflicting results (Saadah, 1999; Frankenberg, et. al, 1999; Bloem, et. al. 2000; Atmarita, et. al, 2000; de Pee, et. al., 2001). Different conclusions about crisis impacts on nutrition arise for several reasons. On the one hand, studies have compared different time periods—indeed, a majority of studies compare only two or three points in time. On the other hand, different indicators of nutritional status are used, and in some cases results have been presented for differing age groups or using non-standard cutoffs.

The present study distinguishes itself in several key respects. The first is methodological. This paper, to the best of our knowledge, is the first to apply to high frequency nutritional data an econometric modeling technique that decomposes data trends into the specific impacts of time, age, and cohort effects (Deaton, 1997). This technique has two benefits in the present context. The first is that it enables us to isolate the time path (our proxy for the crisis) of particular nutritional indicators, disentangling the time effect from the potentially confounding effects of age and cohort. This technique has the added benefit of creating a framework that permits us to link maternal nutrition experience during shocks with the subsequent nutritional and health outcomes of particular cohorts of offspring. Two earlier papers (de Pee, et. al., (2001) and Bloem, et. al., (2000)), used the same nutrition surveillance data (repeated cross sectional surveys for Central Java), focusing on up to 6 waves of data collection.

By contrast, the present analysis is based on 14 waves of data which provide not only broader coverage of the crisis period, but also a more detailed picture of the dynamics of nutrition change during Indonesia's crises (thanks to the higher frequency of time observations used). What is more, the earlier studies relied on maternal wasting and childhood anemia as indicators of crisis impact while this study expands further into the realm of micronutrients, using biochemical measures of deficiency rather than relying only on physical parameters. Micronutrient intake is of particular concern as food price shocks may lead poorest consumers to buffer their caloric intake at the expense of the *quality* of their diets in terms of micronutrient content.

While this study concurs with the findings of Atmarita, et. al. (2000), namely, that there was no meaningful increase in underweight status in children, we do observe a negative impact on both weight-for-height and micronutrient status (hemoglobin concentration), with only partial recovery in both indicators as of January 2001. We also find suggestive evidence of linkages between maternal and child nutritional status relating to micronutrient status and child anemia.

The paper is organized as follows. Section II sets the context for the nutritional shock by tracing trends in per capita consumption of key micronutrient-rich foods. Section III describes the NSS data, its sampling and coverage, descriptive statistics, and defines the key indicators that we analyze. Section IV explains the methodology used to decompose the survey results into time, age, and cohort effects. Section V presents key results, and Section VI concludes.

II. Trends in Consumption of Micronutrient-Rich Foods

Figure 1 illustrates the impact of Indonesia's crises on per capita household consumption of three foods of high micronutrient and caloric quality – eggs, dark green leafy vegetables, and vegetable oil. As household size and composition may change over the course of the survey period, perhaps as a consequence of the crisis itself, we adjust household consumption to more closely approximate per capita intake.[1]

Eggs

Eggs are a relatively affordable and important source of micronutrients, as well as protein and calories, and are also likely to be a good proxy for high quality foods in the diet. In the absence of a close substitute, we would expect reduced household egg consumption to signify a decline in diet quality and to constitute an increased risk of child and maternal malnutrition.

The time path for (log) egg consumption is illustrated in Figure 1.[2] A generally positive trend in the pre-crisis year may reflect (among other factors) a successful social marketing campaign to increase egg and vegetable consumption in Central Java.[3] However, that initially successful campaign appears to have been swamped by the combination of drought and financial crisis. Household egg consumption declined steeply from December 1996 through October 1998, falling at an average rate of 2.5% per month over that period. The level of egg consumption over that period fell from 0.54 to 0.24 eggs per person per week. During 1999 and 2000 we observe a moderate recovery and stabilization of egg consumption, though at a level during 2000 that was only half the level during 1996. Friedman and Levinsohn (2001) report a 117 percent increase in dairy and egg prices in national markets over the same period (Table 1).

Dark Green Leafy Vegetables

Dark green leafy vegetables are an important and relatively inexpensive source of iron, vitamin A, calcium, folate, and other trace minerals. Figure 1 also presents the time path for per capita consumption of dark green leafy vegetables. In this case, we observe a 12% increase in per capita consumption during the pre-crisis year, which again, may reflect, again, the successful social marketing campaign. Between December 1996 and July 1998, however, per capita consumption of dark green leafy vegetables fell by 5.8% (a statistically significant difference). Though a relatively small decline in percentage terms, its nutritional impact could still be substantial for poor households. For instance, vegetables accounted for on the order of two-thirds of child vitamin A intake (adjusted retinol equivalent) in the sample.[4] While this decline still left per capita consumption of dark green leafy vegetables in July 1998 higher than during the pre-crisis year, consumption between July and December 1998 fell by 30%. Indeed, average per capita consumption of dark green leafy vegetables during 2000 was 20% lower than the average level for the pre-crisis year of 1996 (a statistically significant difference; t = 24.4).

Oil

The NSS data set for this period does not include consumption of rice – the primary source of calories in rural Java. However, oil used in cooking can be an important source of calories and fat. Data from the 1978 SUSENAS consumption module analyzed by Meesook and Chernichovsky (1984) indicated that in Java cooking oil, coconut, butter, and lard consumption accounted for 54 percent of total dietary fat.[5] As a relatively expensive source of calories, oil consumption may also serve as a rough proxy for the quality of household caloric intake.

Figure 1 presents the unconditional time path of oil consumption at the household level. The pre-crisis trend shows a slight decrease in per capita consumption of cooking oil, with per capita consumption in December 1996 being 3.7% below its level in January 1996. From December 1996 to October 1998, however, per capita oil consumption fell nearly 19 percent (approximately 1% per month). The time effects are jointly significant to a high degree of certainty. Moreover, the estimated level of per capita oil consumption was statistically significantly lower in July 1998 than in December 1996. To the extent that oil may proxy for household caloric intake, Figure 1 alone provides reason for concern about the nutritional impact of Indonesia's crisis. Over that same period, Friedman and Levinsohn (2001, and in Table 1) report a 122% increase in the price of cooking oil in urban markets.

Thus, the peak crisis period between December 1996 and July 1998 saw significant declines in all three of these micronutrient-rich foods; and, only in the case of cooking oil, was average consumption during late 2000 greater than during the pre-crisis year. The negative trends in consumption of eggs and dark green leafy vegetables in particular signal a decline in the quality of the diet, and suggest an increased risk of iron deficiency in crisis-affected children. Measuring these nutritional impacts is the central focus of the following sections.

III. Data

Nutrition surveillance activities were started in Central Java in December 1995 as part of a monitoring and evaluation system for a social marketing campaign focused on Vitamin A. Five rounds of data collection were completed through January 1997. NSS data collection was not reinitiated in Central Java until June 1998, since when data have been collected on a regular basis (approximately every three months). The present analysis uses all 14 rounds of data, covering the period from December 1995 through January 2001.

For each new round, a random sample of 7200 households was selected using a multi-stage cluster sampling design. A total of 30 villages were selected from each of Central Java's 6 agroecological zones by probability proportional to size sampling techniques. Each village provided a list of households containing at least one child under 36 months of age (the age eligibility criterion was expanded to 59 months in round 7 (August 1998)). From this list, 40 households were selected by fixed interval systematic sampling using a random start. The total sample size for the 14 rounds is 33,600 households, providing observations on 107,753 children. The number of children observed across the 14 rounds varied from 5,450 to 10,553.[6]

Table 2 provides descriptive statistics (over the entire sample) for variables included in the analysis. For household food intake, each respondent was asked to estimate the amount of oil used in cooking during the previous week.[7] Household intake of dark green leafy vegetables was obtained by asking the respondent if vegetables had been prepared in the last 3 days and if so, how much (in kg) from different sources (e.g. market purchase versus own production or gathering). This information was used to calculate the amount prepared in the household per day. Egg intake was estimated by recording the number eggs consumed from two sources – own production and purchased from the market. Child weight, height and mid upper arm circumference were measured on children below 59 months of age, as well as for their mothers. Weight was measured to the nearest 0.1 kg, length and height measurements to the nearest 0.1 cm. Blood samples were collected from a random sub-sample (approximately 18 percent) of children and mothers by fingerprick to measure hemoglobin concentration.

III. Methodology

The methodological challenge in assessing the effects of Indonesia's drought and financial crisis on child nutrition lies in the need to trace an inherently dynamic process in the absence of panel data in which individuals are followed over time. Rather, the NSS data present successive cross sections of clusters that are randomly re-sampled in each survey round[8]. While this data structure precludes tracing individuals' nutritional status over time, it does enable us to divide the sample population into homogeneous groups and to trace the average status of those groups over time. In the present setting, date of birth provides a natural grouping for individuals in the sample. Tracking the experience of the resulting cohorts thus provides a means of approximating the dynamics of the phenomenon of interest – in this case child nutrition. Towards this end, we adopt (with modifications described below) the average cohort techniques proposed in Browning, Deaton, and Irish (1985), Deaton and Paxson (1994), Attanasio (1998), and reviewed in Deaton (1997).

The underlying motivation for this approach is that the "snapshot" of a single cross section may distort the dynamics of interest. Our primary interest lies in the combined effects of drought and financial crisis on child nutrition in Indonesia (the political crisis being more remote to child outcomes in rural Central Java). Yet, if child nutrition is also a function of a child's age, variation in age can confound the interpretation of cross-sectional evidence[9]. If the sample's age composition changes over time (as does ours), the potential dynamic distortions become even greater in considering the sequence of "snapshots" provided by multiple survey rounds[10]. Moreover, if there are secular changes over time in nutritional status, *cohort* effects provide an additional confounding variable. For instance, post-crisis 'rebound' in economic growth may be relatively more beneficial to children born more recently (into the recovery period). The techniques developed by Deaton and others address these potentially confounding effects. Appendix 1 demonstrates the efficacy of this technique for measuring the dynamic nutritional impact of Indonesia's crises by juxtaposing the estimated time path for several indicators estimated with and without controls for age and cohort effects. In key instances, omitting these effects results in substantial underestimation of the crisis's nutritional impact.

As noted, prior applications of this decomposition methodology have been limited to lower frequency data.[11] In such applications, cohorts are typically identified by year of birth, and observed annually. As a result, tracking a given cohort involves observing those age 25 in the first survey, age 26 in the second survey, and so on. The subsequent cohort would begin with those aged 26 in the first survey, aged 27 in the second survey, and so on. By contrast, the present study, based on observations separated by as few as four months, requires a definition of cohorts based on the month of birth *relative to the final survey round*, with subsequent age effects also measured in months.

Our decomposition model of child nutrition outcomes builds from several related hypotheses. The first is that the real-time income and consumption shock imposed by Indonesia's crises affected child nutrition. The second hypothesis, supported by extensive biological evidence, is that child anthropometry and blood hemoglobin concentrations tend to follow known patterns as a function of child age. Finally, it follows from the first two hypotheses that the time shock may have shifted downward the age profile of particular cohorts that were at especially vulnerable ages at the time of the crises. Thus, for the health and nutrition indicators of interest, *N*, we begin with the simple model:

$$N = f(t, a, c) \tag{1}$$

where *t* indicates time, *a* indicates age, and *c*, indicates cohort.

For local changes in this function, we can express this model as an additive function in logarithms:

$$\ln N = \ln f(t) + \ln g(a) + \ln h(c) \tag{2}$$

where the specific functional forms of *f*, *g*, and *h* are unknown *a priori*.[12]

Cohorts are numbered to indicate when they were born relative to the NSS surveys. The 14 survey rounds used here were collected over a period spanning 61 months. The cohorts are numbered according to their age at the time of the final survey. Thus, the youngest cohort observed (born during Round 14, or survey month 61) is designated as cohort 1; that is, $c = a - \textit{survey month} + 61$. Given the age range of children in the sample (0 – 35 months in survey rounds 1 through 5, 0 – 59 months beginning in round 6) and 14 rounds of surveys, we have at least one observation of 95 monthly cohorts.[13] Table 3 illustrates the cohort structure of the sample, showing the specific survey rounds in which each cohort was observed as well as the number of children observed in each cohort-time cell.

The least restrictive (parametric) approach to estimating equation (2) is to allow the data to define the specific functions by representing each of the functions with dummy variables.

Following Deaton and Paxson (1994) and Deaton (1997), we write C, T, and A as matrices of dummy variables for each cohort, survey month (time), and age. The numbers of columns, respectively, in these matrices are 81, 14, and 60. Rewriting equation (2) in terms of these dummy variable matrices and adding an error term yields our estimating equation:

$$\ln N = \iota\beta + T\psi + A\alpha + C\gamma + \varepsilon \tag{3}$$

where ι is a vector of ones, and *ψ, α*, and *γ* are parameters to be estimated for time, age, and cohort effects.

From each dummy variable matrix, we must eliminate one column. This specification, however, presents additional problems for identification arising from the complete determination of cohort by age and time. Identification thus requires dropping one more column from one of the dummy variable matrices.

Without imposing further structure, however, the linear relation between age, cohort, and time effects cannot be separately identified. As Attanasio (1998) notes, the differences between two individuals observed at the same age could be due to time or to cohort effects; the differences between two individuals observed at the same time could be due to age or to cohort effects. In either case, we can estimate only two linear combinations of the three coefficients. Addressing this problem requires a strong identifying assumption, making one of the three effects orthogonal to the others and zero on average (i.e., the residual).

The approach taken in previous studies was to construct the time effects to be orthogonal, with mean equal to zero (in effect, the residual in the decomposition). This is equivalent to assuming that all linear trends in the data can be interpreted as a combination of age and cohort effects. In the context of life cycle and technology vintage studies it was appropriate to treat the time effect as a zero-mean business cycle effect. However, this standard identifying assumption is *not* appropriate for present purposes. In this case, time effects represent the dynamic effects of the crisis, which continued throughout our period of observation. Forcing the time dummies to sum to zero would thus predetermine our results. Instead, we make the explicit assumption that all linear trends in these data can be interpreted as a combination of time and age effects, leaving the cohort effect as an orthogonal residual.

This identifying assumption is justified by a combination of biological and economic considerations. The methodology rests on the assumption that there is no trend or predictable pattern in the dimension chosen as residual. The present application concentrates on hypothesized negative impacts of a known (and ongoing) economic crisis, making time an inappropriate candidate for residual status. Biological evidence strongly supports the existence of predictable age patterns in the indicators of interest. It is known, for example, that blood hemoglobin concentration tends to increase with child age after 12 months, following a dramatic decline between 0-5 months. Thus age is also an inappropriate candidate for residual status.

The only remaining candidate is the cohort effect. In the present context this dimension is the best choice for residual, not only for the economic and biological reasons just noted, but also because in such high frequency data, in which the cohorts are separated in birth by only one month and observed only until age 5, it is reasonable to assume that there is no apparent trend in that dimension. If the crisis did have differential impacts across cohorts that result would not appear as a trend, but would still appear in the estimated cohort effects.

Following Deaton (1997), we impose the normalization that

$$s_c'\gamma = 0 \tag{4}$$

where s_c is a (vector) arithmetic sequence (0,1,2,3…) of the length given by the number of columns in the cohort dummy variable matrix. Like Deaton, we implement this normalization by constructing the cohort dummies (d_c equal to one for cohort c, zero otherwise) as:

(5) $$d_c^* = d_c - [(c-1)d_2 - (c-2)d_1]$$

Thus, estimation of equation (3) becomes a regression of the given health or nutrition indicator on dummies for each time period (excluding the first), for each age in months (excluding the first), and for each cohort (excluding the first two). These regressions are run on cell means for each of the cohort/round combinations in the sample.[14]

As a precaution against the disproportionate influence of outliers these regressions are run using a robust estimator, though in practice, the results are not sensitive to this precaution. The approach is first to estimate an OLS regression to screen for and eliminate gross outliers, based on a measure of residuals (Cook's distance >1). With the remaining observations, a weighted least squares regression is estimated in which the weights are calculated as the inverse of each observation's absolute residual.[15] The following section presents the key findings.

IV. Results

This section presents the results of applying time/age/cohort decompositions to both anthropometric and biochemical indicators of child nutritional status. In each case, we test the following null hypotheses:

1) H_0: $\Psi = 0$ (time effects are jointly zero);

2) H_0: $\alpha = 0$ (age effects are jointly zero);

3) H_0: $\gamma = 0$ (cohort effects are jointly zero);

4) H_0: $\Psi_{Jul98} - \Psi_{Dec96} = 0$ (time coefficients are equal before and after the peak of the crisis); and,

5) H_0: $1/19\ (\Psi_{Jul98} - \Psi_{Dec96}) - 1/11\ (\Psi_{Dec96} - \Psi_{Jan96}) = 0$ (the slope of the time path during the year before the crisis equals the slope of the time path during the first 19 months of the crisis).

Hypotheses 4 and 5 address the magnitude of the shock to child nutrition by testing differences in both the levels and the slopes of the time path at critical junctures. In particular, the timing of events in Indonesia suggests that the key survey rounds to observe are those conducted in December 1996 and the subsequent round in July 1998. Hypothesis 4 tests the difference in levels of those observations while hypothesis 5 compares the slopes of the time paths during that interval and the prior ("pre-crisis") year. In what follows, the only instance in which we fail to reject a null hypothesis is the case of hypothesis 4 for weight-for-age z-scores.

Anthropometric Indicators

Anthropometry is commonly used to follow changes in nutritional status (Pinstrup-Andersen et. al., 1995). Child weight-for-age (WAZ) reflects body mass relative to age. WAZ below two standard deviations of the reference mean has been widely adopted as a cut-off for underweight. Weight-for-height (WHZ) is an indicator of current nutritional welfare. WHZ below two standard deviations of the reference mean has been widely adopted as the cut-off for wasting. This section present results for child weight-for-age, as well as for child weight-for-height.

Child Weight-for-Age (WAZ)

Figures 2 presents the time effects (e.g., crisis impacts, conditional on age and cohort effects) on trends in mean weight-for-age. Consistent with previous findings by Atmarita, et. al. (2000), and Frankenberg, et. al. (1999), the time effect for WAZ shown in Figure 2 fails to show a substantial decline during Indonesia's crisis period. While a statistically significant decline is apparent during the pre-crisis year, the decrease of 0.16 standard deviations relative to international reference standards is arguably not *biologically* significant. Indeed, a decrease of this magnitude only increases the prevalence of underweight from 27 to 30 percent.

The age effect in WAZ presented in Figure 3 reflects a steep decline by 1.25 standard deviations during the first year of life, after which the sample mean stabilizes at approximately 1.5 below the international reference. This pattern is common in samples from developing countries (WHO, 1995), and thus provides an additional illustration of the justification for treating cohort effects as the trendless residual in our model. The cohort effect (graph omitted), while jointly different from zero (we fail to reject hypothesis 3), indicates that the crisis did not differentially affect different cohorts of children in rural Central Java.

Earlier studies, based on fewer survey rounds (and with no statistical distinction made between time and age effects), reached broadly similar conclusions about the lack of impact of Indonesia's crisis on the incidence of underweight children. A more nuanced picture of nutritional impact, however, emerges when the time/age/cohort decomposition is applied to more responsive indicators of nutritional status. Child weight-for-height, while closely related to WAZ ($r^2 = 0.62$), may respond more quickly to recent shocks (WHO, 1995).

Child Weight-for-Height (WHZ)

Figure 2 also presents changes over time in mean child weight-for-height (conditional on age and cohort effects), which is revealed to have declined during the crisis. Between December 1996 and July 1998 – the period that includes both the most severe impact of the *El Niño* drought and the collapse of the Rupiah – mean WHZ declined by over one-third of a standard deviation. The decline is statistically significant and potentially biologically significant, as well.[16] The initial crisis impact on WHZ was followed by a partial recovery during the second half of 1998, before stabilizing throughout 1999 and 2000 at a level approximately one-fourth of a standard deviation below the level observed in January 1996. Thus, the crises had a substantial impact on WHZ. Prevalence of wasting over that period doubled from 6 to 12 percent, and the subsequent recovery (at least until 2001) was limited. Severe wasting in young children may have lasting effects, including higher morbidity rates, later entrance to school, and impaired learning (Gillespie and Haddad, 2001). Indeed, Martorell (1996) cites evidence of reduced IQ resulting from severe undernutrition in early childhood.

It is noteworthy that ignoring the potentially confounding effects of age and cohort on trends in mean WHZ leads to a 50 percent *under*estimate of the decline over this period (Appendix 1). Figure 3 juxtaposes the age effect for WHZ with that of WAZ, demonstrating a similar pattern though with a less dramatic decline in the case of WHZ. As with WAZ, there is no discernable pattern in the cohort effect on WHZ (graph omitted).

By bringing to bear both a richer and more frequently observed set of nutritional indicators, and a methodology that distinguishes time from age and cohort effects, this analysis begins to uncover previously undocumented crisis impacts. Note that we exclude height-for-age from this assessment of crisis impacts. While height-for-age is widely accepted as an indicator of child nutrition status, it is explicitly a long-run indicator for which a longer time series would be required. Moreover, it is unlikely that a short-term crisis would be reflected in this least responsive anthropometric indicator – a result that would be consistent with our finding that the crisis had no discernable impact on the average time path of child weight-for-age. Indeed, we find the most severe crisis impact on child nutrition, not in the anthropometric evidence, but at the microscopic level; that is, in the highly responsive blood chemistry measure of hemoglobin concentration.

Micronutrient Status

Blood hemoglobin concentration provides the most revealing picture of crisis impacts--one that reveals effects on dietary *quality* in addition to quantity, and serious shortfalls have been associated with heightened mortality and reduced learning capacity. The peak crisis period in Indonesia was accompanied by substantial declines in household consumption of eggs and dark green leafy vegetables -- foods that are important sources of iron and other micronutrients. Decomposing trends in blood iron levels (measured by blood hemoglobin concentration) in children reflects the expected consequence for micronutrient status. Indeed, the share of children in rural Java vulnerable to iron deficiency anemia is substantial. Anemia is characterized by a hemoglobin concentration (Hb) of less than 11 g/dL of blood (WHO, UNICEF, UNU,98). Mean Hb in children in the NSS data set is 11.02 g/dL, and the prevalence of anemia among children over the entire sample period is 47 percent.[17]

Disaggregating Hb results by cohort reveals a moderate degree of heterogeneity not observed in the anthropometric indicators. Figure 4 traces the age paths (from age 0-36 months) for individual cohorts. This allows a comparison of mean levels of hemoglobin of these age paths for different cohorts when each cohort is between 6-18 months of age (when deficiencies have severe effects on child growth).[18] Note that each cohort is a different age at any given point in time (and thus close groups of cohorts are in a particular age range at any given point in time), such as at the onset of the crisis. The panels in Figure 4 group cohorts based on time of birth relative to the onset of the crisis, and allow us to compare Hb across groups of cohorts during their most vulnerable ages. Systematic vertical shifts in cohort-specific age profiles would indicate that the crisis differentially affected various cohorts. Figure 4 demonstrates that cohorts in that vulnerable age range during and just after the crisis did, in fact, suffer higher rates of anemia (as indicated by the horizontal cut-off at 10.5 g/dL hemoglobin concentration). It is more intuitive to consider the panels of Figure 4 in reverse order.

Panel D groups together cohorts ($C_{67} – C_{79}$) that were already 18 months or older in the last pre-crisis survey (December 1996). The age traces for those cohorts lie exclusively above the severity level indicated by the horizontal line at 10.5 g/dL (indeed, the earliest cohorts were too old to have been observed at all below age 18 months). Panel C considers those cohorts (C_{55} – C_{66}) who were aged 6-18 months during the initial phase of the crisis. Nearly all of the age profiles for those cohorts fall between 11 and 10.5 g/dL during their most vulnerable age window of 6 – 18 months. Panel B shows the next youngest group of cohorts (C_{36} – C_{51}), who were *born* during the crisis. Panel B reflects a continued downward trend in the mean age profiles, compared with the older cohorts in the previous panels. Cohorts born during the crisis all fell below 10.5g/dL hemoglobin concentration at some point during this vulnerable period in their life. Indeed, the decline is most evident in Panel A, among the youngest cohorts considered; namely, those *conceived* during the crisis (C_{18} – C_{33}). Nearly all of those youngest cohorts displayed hemoglobin concentrations of less than 10.5 g/dL (during the same critical age range when older cohorts did not). While it appears that there was some recovery among the latest cohorts, there may also have been longer-term developmental consequences. A key question for future exploration is whether micronutrient deficiency in utero during a crisis period (fetal insult) has different child growth consequences than micronutrient deficiency in the early period of infant development.

In addition to the impact on diet and nutritional status of children, the crisis period may also have reduced mothers' hemoglobin concentrations. It has been shown that the risk of anemia is greater among the young offspring of anemic mothers.[19] An important added virtue of cohort analysis is that it permits explicit linkages to be made between maternal nutritional status during pregnancy and the subsequent status of children (identifiable by cohort) conceived and born to nutritionally stressed mothers.

Figure 5 aggregates the time effects of Hb (in logarithms). The decline in mean child hemoglobin concentration from December 1996 to July 1998 was 6.1% (or 0.32% per month). In absolute terms, this corresponds to a decline of 0.68 g/dL over the entire period, which is greater than one standard deviation for the full sample of those cohorts. The time effects are statistically significant, as are the differences in levels between December 1996 and July 1998 (we fail to reject hypotheses 1 and 4). While it is difficult to explain the peak in December 1998, average child Hb tended to stabilize at a post-April 1999 average that was 0.5 g/dL lower than the level in the initial survey round (joint F-score = 16.89). As illustrated in Appendix 1, ignoring age and cohort effects can lead to a substantial underestimation of the decreases in mean child Hb over time.

Figure 6 presents the age effect on child Hb, controlling for time and cohort effects. The typical age profile for child Hb indicates an initial rapid drop during the first 3 months of life. However, over the entire span of 60 months covered in the sample Hb is a positive function of age, increasing on average by 0.22% per month of age.

The conditional cohort effects presented in Figure 7 show the lifetime averages by cohort illustrated in the cohort-specific age profiles shown in Figure 4. As in the more disaggregated format, average hemoglobin concentrations were decreasing among progressively younger cohorts at the time of the crisis (cohort 54 back through cohort 8).[20] The cohort effect levels off in the older cohorts, consistent with Figure 4.

Explaining this deterioration in nutrient status in progressively more recent cohorts requires recognition that nutrition outcomes are determined not only by exogenous shocks that translate into food consumption shortfalls, but also from endogenous maternal responses in care and feeding practices. The economic crisis lowered real income and purchasing power of households in Central Java, as elsewhere in the region (Levinsohn, et. al. 1999; Friedman and Levinsohn, 2001). Rice intake, the major food and calorie source, was relatively stable during this period (Frankenberg, et. al.,1999), but because of the increase in rice price and share of household expenditure on rice, fewer resources were available for other foods, mainly those foods which are better sources of micronutrients. Figure 1 reflects this decline in consumption of “high quality“ foods. The larger impact on hemoglobin concentration of children born (or conceived) during the crisis thus appears to reflect maternal malnutrition during pregnancy as well as changes in diet during the crisis. As noted above, iron deficiency during early childhood can result in permanently impaired cognitive and motor development, as well as lower wages and reduced labor productivity in later life (Ross and Horton, 1998).

V. Conclusions

This study introduces econometric methodologies for decomposing trends into time, age, and cohort effects to the analysis of high frequency nutritional surveillance data. This approach has two potential advantages over more typical cross-sectional approaches. Most directly, the decomposition applied here allows us to disentangle the potentially confounding effects of time, age, and cohort. Indeed, failing to do so in the present context would result in substantial *underestimation* of the nutritional impact of Indonesia's multiple crises, and *overestimation* of the nutritional recovery. Several previous studies had concluded that there were no major impacts on child nutritional outcomes. By contrast, the present study reveals significant nutritional impacts. While there was no meaningful decline in child weight-for-age measures, mean weight-for-height declined by over one-third of a standard deviation. Furthermore, blood hemoglobin concentration – an even more responsive indicator, and one that provides insight into the quality, as well as the quantity of the diet – also declined sharply during the crisis. While both indicators improved again during late 1998, neither had recovered to its pre-crisis level by January 2001. The crisis thus significantly reversed what had previously been a ten-year period of improving nutritional status in Indonesia.

In addition, applying cohort decomposition opens greater possibilities for linking the outcomes of maternal malnutrition with the subsequent nutrition of identifiable offspring. This important aspect of cohort analysis provides at least suggestive evidence that the cohorts conceived and born to increasingly anemic mothers also experienced a higher incidence of anemia.

These findings invite further investigation along several dimensions. Having established some of the basic nutritional impacts of Indonesia's crises a next step will be to differentiate these impacts by types of household in policy-relevant ways that can better inform the design of interventions to mitigate the most damaging nutritional impacts of such crises. Were some household types more vulnerable to the crisis than others? Were there identifiable biases in terms of intrahousehold distribution of food or other resources by age or gender? Potential explanations to be explored in differentiating households include maternal education, nutrition knowledge, occupation group, and other household characteristics.

Our finding that the nutritional consequences of Indonesia's crisis were particularly concentrated at the micronutrient level invites further investigation of the determinants of child micronutrient status. In this context, we expect maternal nutrition knowledge to be critical, given the hidden nature of foods' micronutrient context. Indeed, preliminary results (Block, 2002) suggest that maternal nutrition knowledge is critical, more so even than formal schooling, in determining child micronutrient outcomes. Geographic and environmental distinctions across Central Java's six agroecological zones may also yield policy-relevant insights into what makes certain population groups more or less vulnerable to nutritional shocks.[21]

More generally, future research should also investigate the efficacy of applying cohort decomposition to longer-term nutritional questions than can be addressed with the NSS data. Given a longer time series of cross sectional surveys, it may be possible to use cohort techniques to link maternal nutritional conditions to the long term health and cognitive development of offspring. This would be particularly informative in the context of other transitory shocks, in which declining maternal nutritional status might affect the incidence of low birth weights, which are thought to undermine children's subsequent mental and physical development.

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Appendix 1
The Effect of Ignoring Age and Cohort Effects

This appendix briefly demonstrates the effect of excluding age and cohort effects from the estimation of time paths for child weight-for-height and child blood hemoglobin concentrations. To illustrate the difference, we simply juxtapose the original results (the solid lines, repeated from Figures 2 and 5) with the analogous time path estimated unconditionally (e.g., not controlling for age and cohort effects). While it would generally be inappropriate, in the unconditional case, to include children above and below 36 months of age in the same estimation, the point here is simply to demonstrate the difference made by methodology by comparing similar time paths.

Figure A1 compares the time paths for WHZ. While comparable in the early and late rounds, the magnitude of the estimated crisis impact excluding age and cohort effects is only *half* the effect estimated with controls for age and cohort. The difference between the estimated coefficients for July 1998 between these two paths is statistically significant (F = 5.72, P = 0.017).

In Figure A2, the paths for child hemoglobin concentration also differ substantially in the direction of underestimate when age and cohort effects are excluded. In this case, too, these exclusions result in a substantially more optimistic picture, possibly underestimating the crisis impact (the point estimates for the July 1998 survey differ only with P = 0.279), and almost certainly overestimating the subsequent recovery. When included, both age and cohort effects are jointly significant. In point-wise tests of statistical difference between the two paths depicted in Figure A2, F-tests reject the null hypothesis of equality between these estimated paths at each survey beginning with December 1998. F-scores for these tests range between 4.93 (P = 0.027) for the October 2000 survey estimates to 10.09 (P = .002) for the October 1999 estimates.

The explanation for the increasing underestimate of the crisis impact when age and cohort are excluded is clear. Given the cohort structure of the sample and the age cut-offs for inclusion in each round, the average age of the sample increases (doubles) between and first and seventh rounds (after which the average age is roughly constant. Given the strongly increasing age effect in child Hb (Figure 6), adding increasingly older children in each round biases the estimated time upward when that effect is excluded.

Table 1. Price Changes for Selected Foods, January 1997 to October 1998

	Mean Price Increase	Standard Deviation
Rice	195.2%	29.2
Other cereals & tubers	137.5%	101.8
Fish	89.1%	67.4
Meat	97.0%	49.3
Dairy & eggs	117.1%	31.9
Vegetables	200.3%	129.5
Pulses, tofu, & tempeh	95.2%	76.0
Fruit	103.7%	61.3
Oils	122.0%	74.8
Sugar, coffee, & tea	142.9%	28.3
Prepared food & beverages	81.4%	51.7

Source: Friedman and Levinsohn (2001) based on their analysis of SUSENAS and BPS surveys of urban markets in 27 provinces.

Table 2. Descriptive Statistics

Variable	Obs[b]	Mean	Std. Dev.	Min	Max
Oil (kg)	540	0.828	0.155	0.500	1.250
Oil_adj[a]	540	0.199	0.040	0.131	0.307
DGLV (kg)	540	1.366	0.372	0.848	8.000
DGLV_adj[a]	540	0.333	0.102	0.223	2.286
Eggs_tot (units)	540	1.364	0.509	0.000	3.000
Eggs_adj[a]	540	0.343	0.121	0.000	0.694
CWHZ	540	-0.507	0.362	-2.350	0.848
CWAZ	540	-1.431	0.487	-3.180	0.563
CHB	476	11.256	0.530	9.850	14.740

a. "Adjusted" quantities are total household consumption divided by adult equivalents (defined in text).

b. The number of observations refers to cohort-round cell means. Table 3 shows the number of observations in each cell.

Table 3. Sample Cohort Structure by Survey Round (Date)

					Date of Survey Round									
cohort	Ja96	Ap96	Jl96	Oc96	Dc96	Jl98	Oc98	Dc98	Ap99	Oc99	My00	Ag00	Oc00	Ja01
0														66
1														154
2														195
3													80	199
4													166	226
5												110	204	186
6												179	198	260
7												211	236	255
8											84	215	241	237
9											178	244	250	242
10											218	207	218	189
11											240	215	215	202
12											235	230	212	212
13											232	216	208	237
14											248	214	188	197
15										94	236	210	236	196
16										194	233	234	211	168
17										203	225	206	183	198
18										184	241	221	195	198
19										255	201	187	162	149
20										216	203	163	194	164
21									87	193	207	157	173	119
22									141	175	158	161	125	126
23									172	208	172	160	128	131
24									161	199	168	131	132	129
25							112		202	167	157	129	121	122
26							193		159	156	122	110	112	117
27							91	206	185	166	124	117	132	94
28							184	225	202	188	131	139	119	116
29							237	230	205	170	147	177	122	136
30						90	226	262	232	196	160	162	129	146
31						185	235	265	197	189	153	124	119	111
32						210	223	279	225	168	147	120	124	116
33						182	211	228	178	163	139	98	129	100
34						195	191	186	165	130	120	115	88	103
35						203	207	199	186	142	127	136	112	91
36						192	184	189	171	120	145	116	102	81
37						174	182	240	184	150	111	106	119	113
38						176	209	215	180	149	125	101	117	98
39						201	182	198	184	160	110	113	109	103
40						178	176	178	178	139	95	94	109	101
41						214	183	206	180	171	107	128	94	88
42						206	147	188	164	152	118	113	96	91
43						164	175	209	163	133	125	111	84	91
44						181	129	181	143	150	130	93	106	100
45						167	160	173	174	113	116	97	90	100
46						151	139	193	158	115	101	106	93	87
47						153	128	164	160	97	100	109	82	83
48						129	139	169	131	91	99	81	75	101
49					138	148	122	199	108	82	83	75	71	77

50					228	126	113	164	125	103	103	89	84	78
51				157	253	129	152	155	127	121	83	69	87	73
52				265	244	125	154	163	117	119	71	88	97	70
53				284	253	150	148	159	128	114	92	84	85	57
54			159	277	247	157	152	182	128	99	90	72	71	67
55			263	276	303	158	159	175	125	101	93	86	73	59
56			274	282	276	120	131	194	126	106	106	80	66	58
57		152	260	257	248	125	123	125	137	98	95	72	59	60
58		214	285	270	205	112	127	157	107	82	72	83	65	43
59		248	286	241	235	97	123	145	102	103	66	67	66	35
60	156	278	280	226	227	99	120	142	87	97	76	59	55	1
61	223	297	255	251	247	103	115	230	100	68	58	41	43	
62	233	280	242	214	222	96	129	158	110	80	54	39	38	
63	254	271	269	257	198	98	207	158	106	94	48	50		
64	293	287	255	239	227	100	159	141	102	98	54	48		
65	278	293	254	247	229	119	176	196	103	90	56			
66	283	253	233	238	216	11	161	147	100	76	55			
67	308	266	250	206	236	1	160	162	96	76	35			
68	300	273	264	212	212	3	141	182	96	77				
69	260	263	229	183	191	1	154	166	121	78				
70	265	252	199	188	196	1	142	152	98	71				
71	246	194	184	170	176		129	152	72	60				
72	285	229	194	186	159		125	142	64	58				
73	212	211	175	145	203		145	164	67	40				
74	208	200	181	189	135		120	138	57	48				
75	203	184	181	192	158		150	143	60					
76	213	176	169	138	160		136	144	57					
77	216	206	170	165	160		145	158	72					
78	221	190	197	157	115		142	142	67					
79	226	195	174	168	157		162	152	49					
80	202	185	166	164	151		133	123	62					
81	171	171	149	142	125		116	117						
82	144	134	139	133	127		113	104						
83	173	130	127	104	125		76	117						
84	158	142	120	116	137		81	142						
85	141	140	113	89	13		81							
86	112	108	102	127			89							
87	129	104	125	12										
88	136	152	106											
89	135	123	151											
90	122	120	7											
91	111	104												
92	107	131												
93	100	15												
94	124													
95	102													

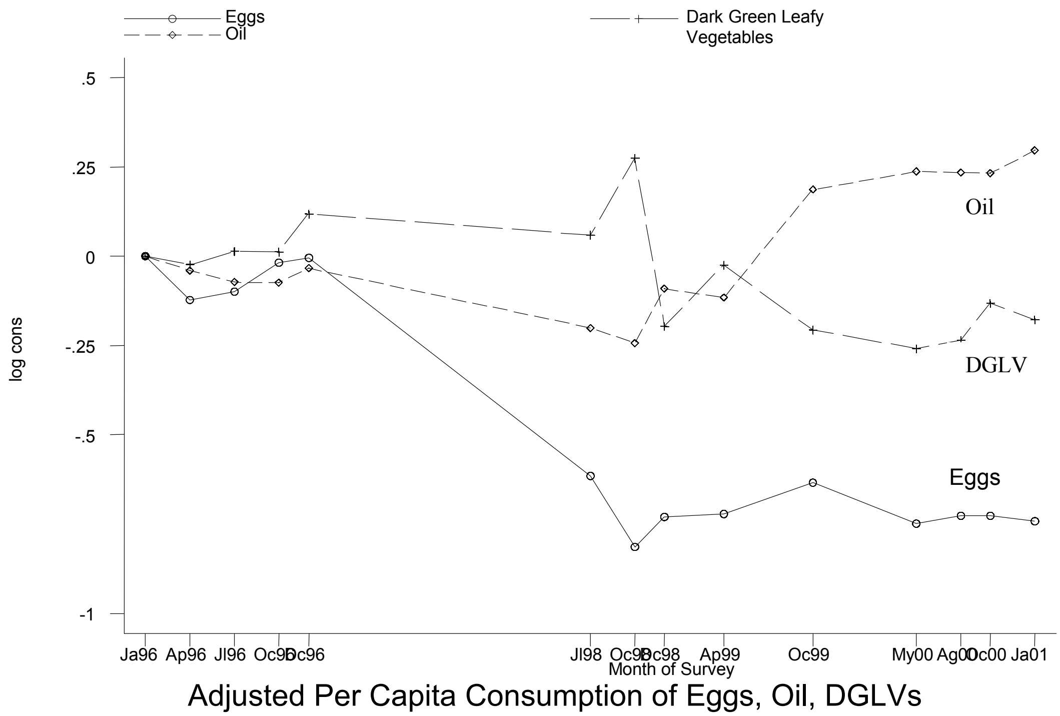
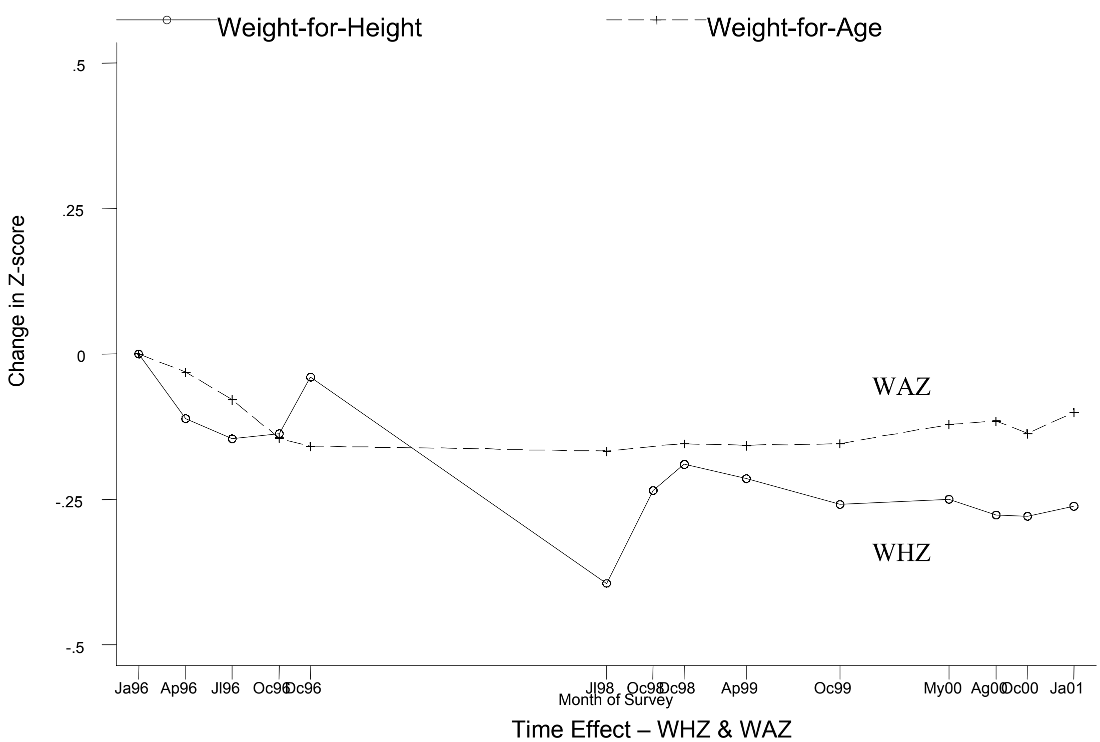


Figure 1



Initial (Jan. 1996) levels: WAZ = -0.86; WHZ = -0.01

Figure 2

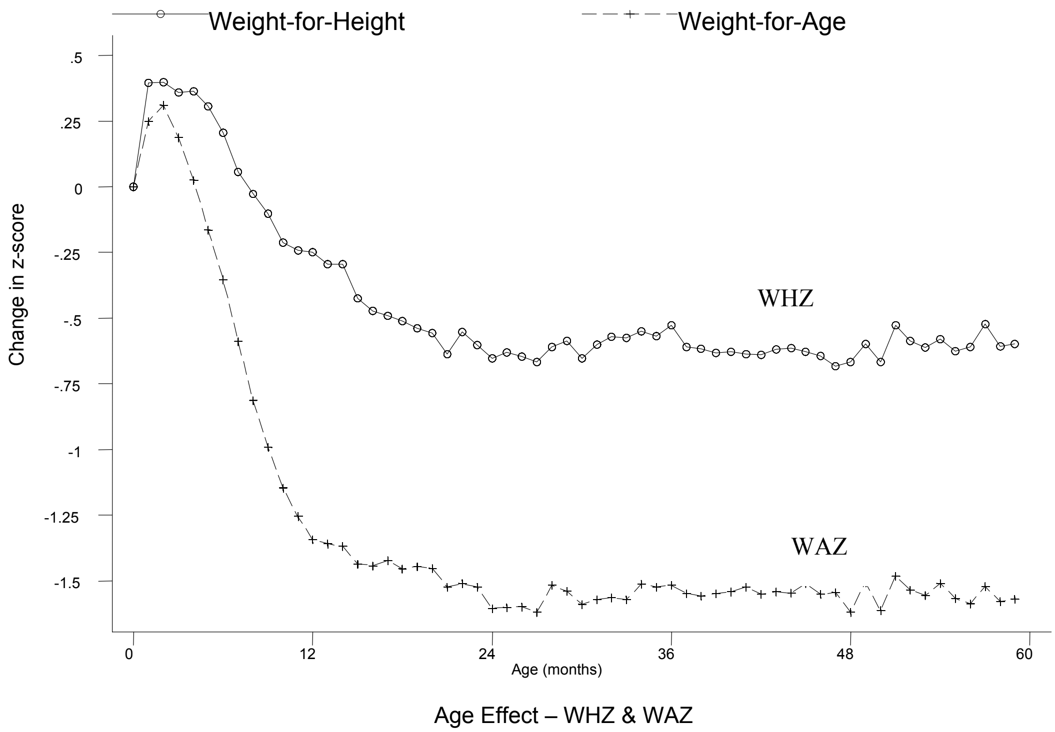

Weight-for-Height
Weight-for-Age
.5
.25
0
-.25
-.5
-.75
-1
-1.25
-1.5
Change in z-score
WHZ
WAZ
0
12
24
36
48
60
Age (months)
Age Effect – WHZ & WAZ

Figure 3

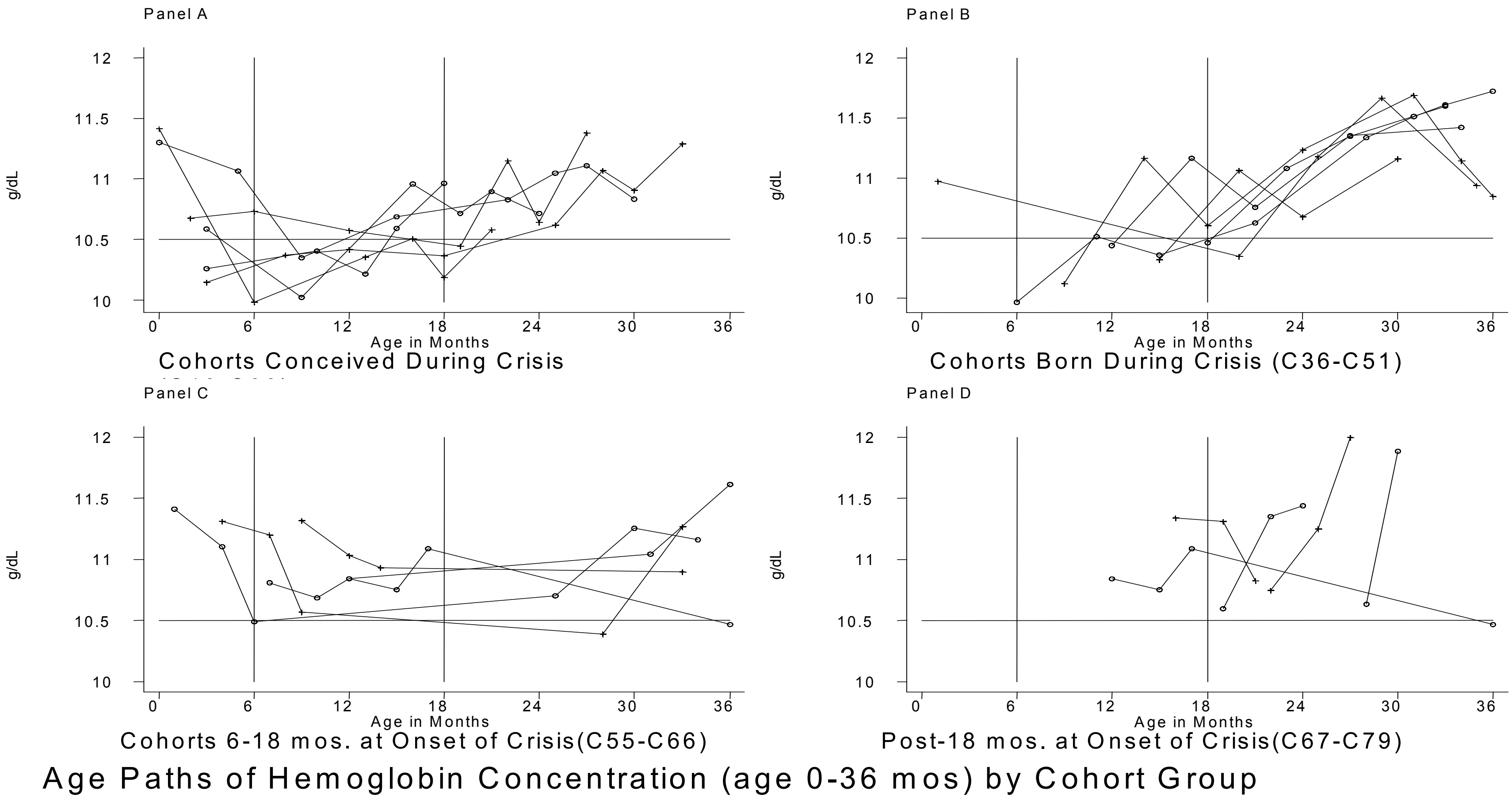
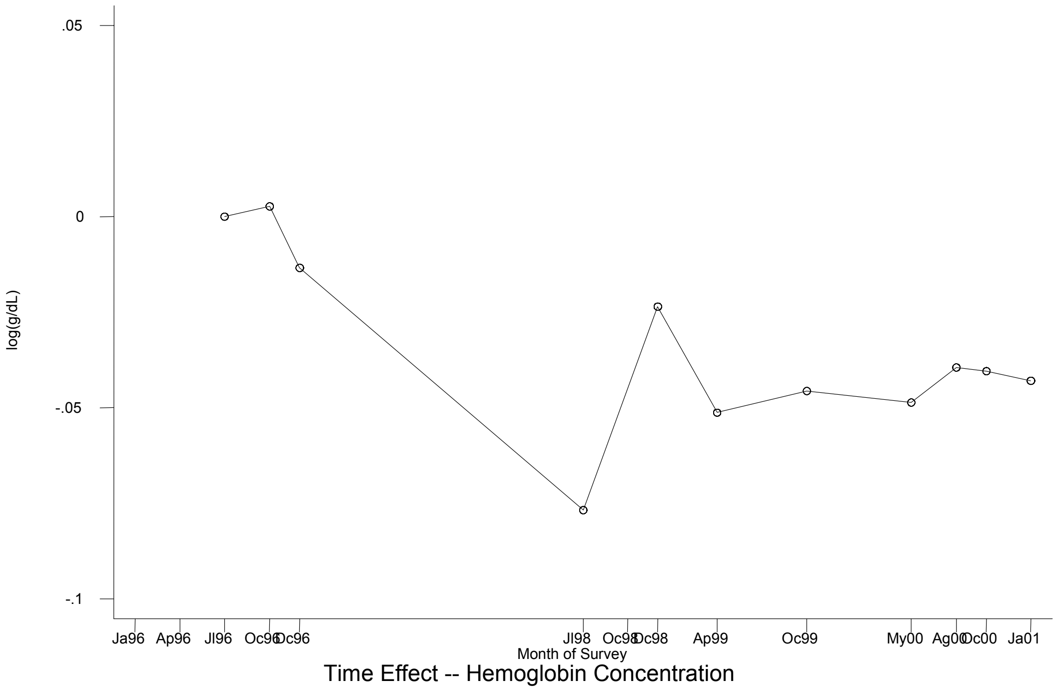

Panel A
Panel B
Panel C
Panel D
g/dL
12
11.5
11
10.5
10
0
6
12
18
24
30
36
Age in Months
Cohorts Conceived During Crisis
Cohorts Born During Crisis (C36-C51)
Cohorts 6-18 mos. at Onset of Crisis(C55-C66)
Post-18 mos. at Onset of Crisis(C67-C79)
Age Paths of Hemoglobin Concentration (age 0-36 mos) by Cohort Group

Figure 4



Initial (Jul. 1996) absolute mean Hb = 10.98g/dL

Figure 5

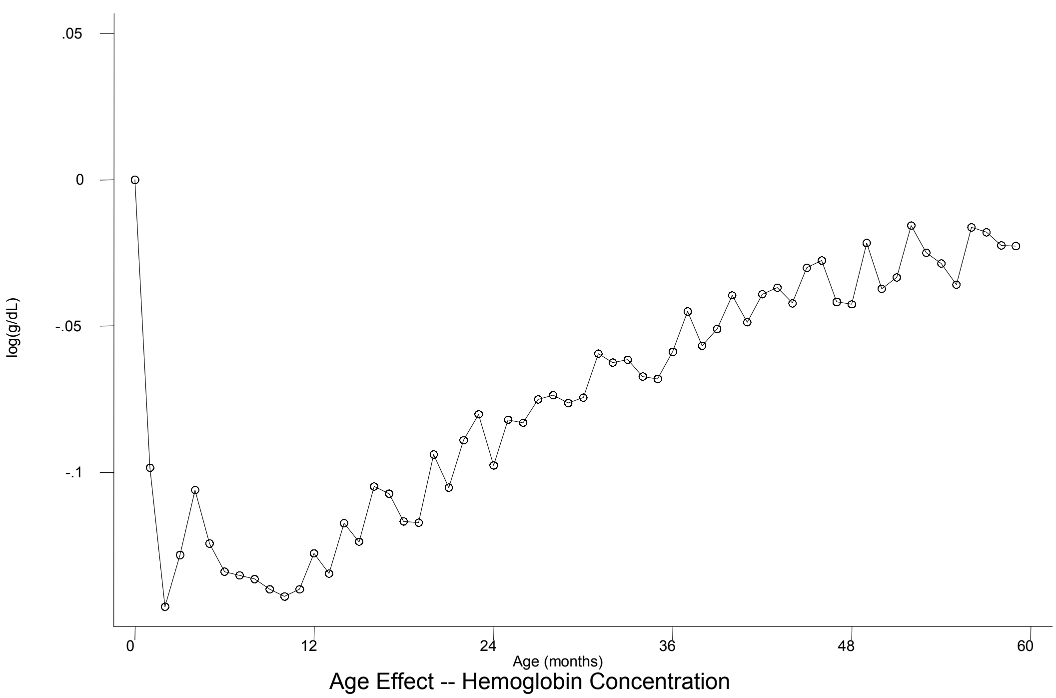
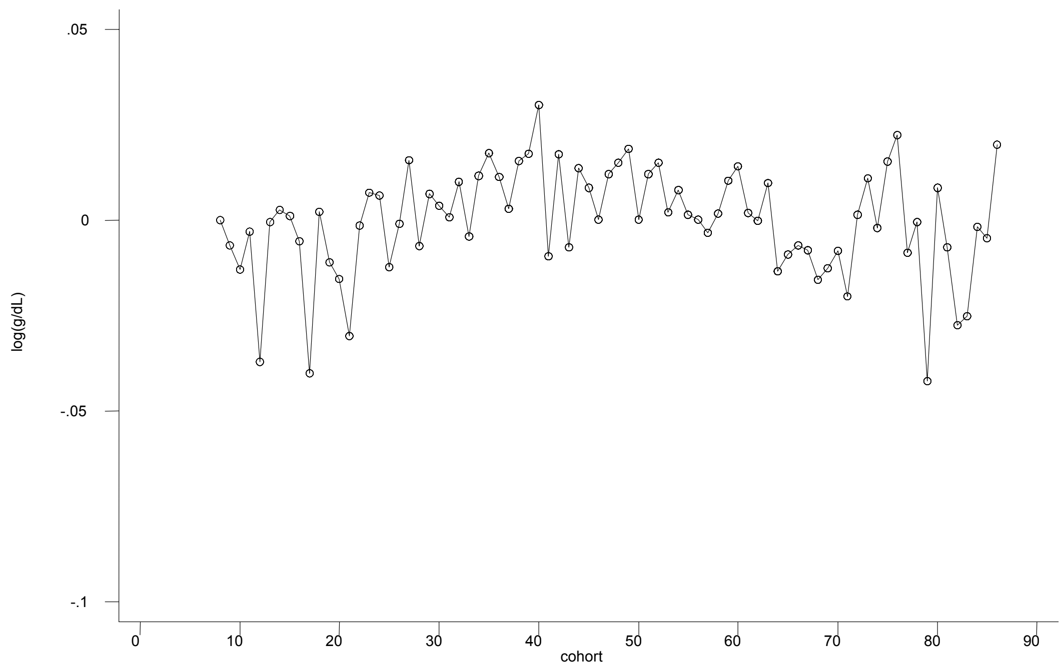


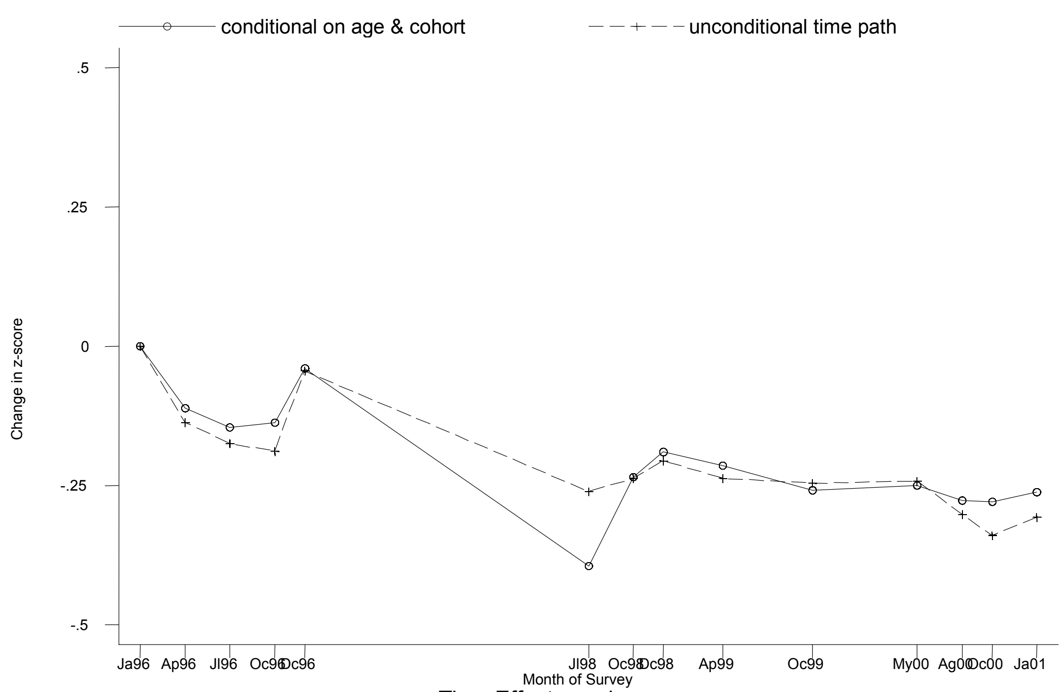
Figure 6



Cohort Effect -- Hemoglobin Concentration

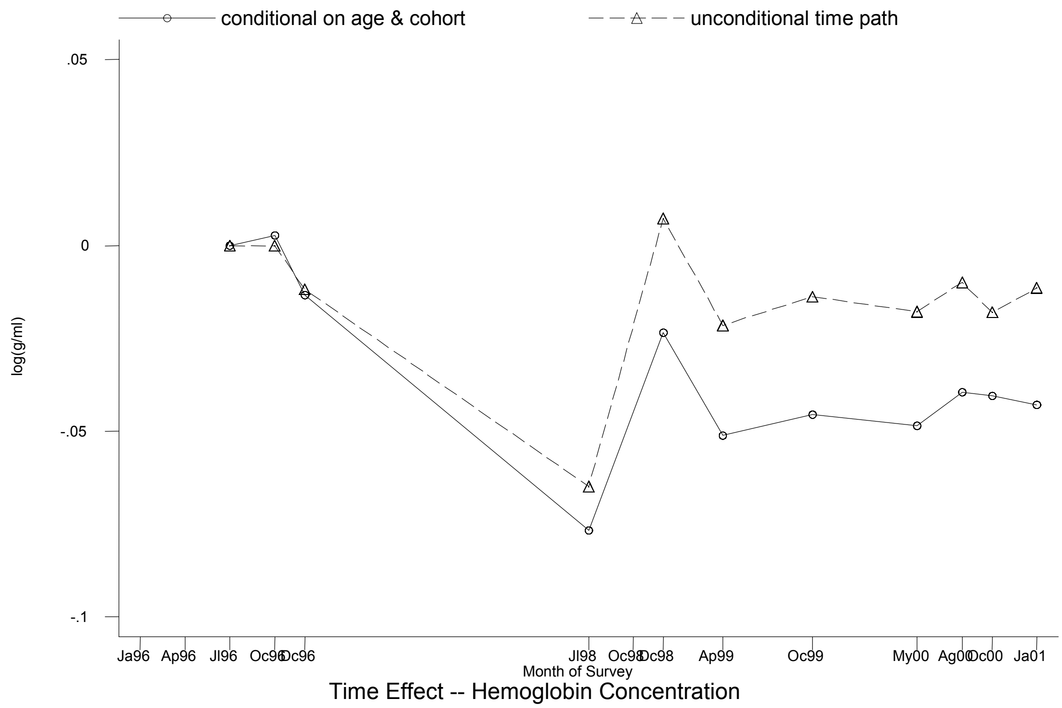
(Initial absolute mean Hb (Jan. 1996) = 10.98 g/dL)

Figure 7



Time Effect -- cwhz

Figure A1



conditional on age & cohort
unconditional time path
.05
0
-.05
-.1
log(g/ml)
Ja96
Ap96
Jl96
Oc96
Oc96
Jl98
Oc98
Oc98
Ap99
Oc99
My00
Ag00
Oc00
Ja01
Month of Survey
Time Effect -- Hemoglobin Concentration

Figure A2

ENDNOTES

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[1] Our approach is to divide total household consumption of a given commodity by a rough approximation of adult equivalents, in which each child under six years of age is counted as one-half an adult. Our data include total family size (*fs*) and the number of children under 6 (*u6*). We calculate the number of adult equivalents for a given household as $.5*u6 + (fs - u6)$.

[2] In this (and all other) graph of time effects, the observation points indicated on the x-axis are the dates of the 14 NSS survey rounds.

[3] De Pee, et. al. (1998) describes this campaign, implemented jointly by the Government of Indonesia, UNICEF, and Helen Keller International.

[4] In Bangladesh, Bouis and Novenario-Reese (1997) found vegetables to account for nearly 95% of vitamin A, 75% of vitamin C, and 25% of iron intake.

[5] In a study of micronutrient demand in Bangladesh, Bouis and Novenario-Reese (1997) found that cooking oil alone accounted for 43 percent of dietary fat intake.

[6] Greater detail on nutritional surveillance methods is available in de Pee, et. al. (2000), and in annual reports of Helen Keller International.

[7] Total household oil intake is probably underestimated because this variable/question does not capture the oil intake that would have been part of purchased foods (common street foods in Indonesia).

[8] Cluster sampling is perhaps the most widely accepted method of rapidly assessing large population groups, particularly in the context of emergencies. The aim is to secure sufficient information that is representative of the total population as well as for any subgroup that may be distinguished with the total (WHO 2000; WFP 2001).

[9] For example, Sahn and Alderman (1997) showed that for Mozambique increases in household incomes only affects the nutritional status of children older than 2 years, while increases in mother's education only affects the nutritional status of children under 2 years of

age. In other words, since the determinants of anthropometry can be different for different ages then age differences within a cross-section need to be controlled for.

[10] Growth faltering tends to increase from weaning onwards (between 1 and 2 years), and stunting compounds itself over time. Thus, there is greater likelihood of observing a stunted child in any household with several children over 2 years of age than in a household with several children under 3.

[11][11] Previous applications of this approach include Deaton and Paxson (1994) and Attanasio (1997), who examine life cycle models of savings and consumption, Hall (1971) who examines technical change for vintages of pickup trucks, and Weiss and Lillard (1978), who examine age, cohort, and time effects on earnings of scientists.

[12] Note that where *N* indicates a z-score (such as in the case of weight-for-height, etc.), we use the z-score directly in place of a logarithm.

[13] In implementing this approach cohorts numbered 87 and above are omitted since they "graduated" from the sample at age 36 months and were thus not observed in any post-crisis survey rounds. The first 5 cohorts are also omitted for this specification since they did not reach 6 months of age before the final round (and so were observed only 1-3 times).

[14] These combinations correspond to the cells in Table 3. The cell entries in Table 3 are thus the numbers of observations over which each cell mean is calculated.

[15] This estimator is available as "robust regression" (*rreg*) in Stata.

[16] The changes in weight-for-height Z-score are more complicated to interpret for three reasons. First, as an indicator of nutrition, changes in weight-for-height can reflect alterations in height, both height and weight, or in weight alone and thus at the individual level, the physiological implication of a change in weight-for-height may be positive or negative. Second, as an indicator, wasting reflects acute malnutrition. In Central Java, the prevalence of wasting is low, therefore changes in weight-for-height Z-score shift the mean and distribution

of the curve, but do not necessarily translate into major changes in malnutrition prevalence for the population as a whole. It is not known whether there are functional and developmental consequences of these curve shifts to the left, in absence of a major increase in the prevalence of wasting. Finally, wasting in children is influenced as much by acute illness and/or lack of medical and other care, as by an ongoing deficiency in food intake. As a result, the observed decline in weight-for-height Z-score is not easily attributed to an immediate cause.

[17] In the absence of other causes of anemia (hookworm, malaria), the main cause of anemia is iron deficiency. Anemia is the final stage in the development of iron deficiency and the prevalence of anemia suggests a much larger proportion of children, as many as twice, are likely to suffer from iron deficiency (though not with sufficient severity to qualify as anemia).

[18] Yip and Dallman (1996) show that iron deficiency peaks in this age range as a result of rapid growth, depleted iron stores, and low iron content of the diet.

[19] de Pee, et. al., 2002 show that maternal anemia is closely associated with anemia among infants 3-5 months, controlling for other factors. NSS data show an increase in anemia among non pregnant women during the crisis, although the increase was smaller than for children (Bloem and Darnton-Hill, 2000). The requirements for iron during pregnancy are higher. Therefore it is quite likely that rates of anemia during pregnancy also increased during this period (sample sizes of pregnant women are inadequate to explore this change).

[20] Note that smaller numbered cohorts are younger at any given point in time (born more recently), so looking at increasingly recent cohorts requires reading the graph from right to left.

[21] Block and Webb (2001) examine related questions regarding response to shocks in Ethiopia.