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Do Human Parents Face a Quantity-Quality Tradeoff?: Evidence From a Shuar Community

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ABSTRACT A number of evolutionary theories of human life history assume a quantity-quality tradeoff for offspring production: parents with fewer offspring can have higher biological fitness than those with more. Direct evidence for such a tradeoff, however, is mixed. We tested this assumption in a community of Ecuadorian Shuar hunter-horticulturalists, using child anthropometry as a proxy for fitness. We measured the impact of household consumer/producer (CP) ratio on height, weight, skinfold thicknesses, and arm and calf circumferences of 85 children and young adults. To control for possible "phenotypic" correlates that might mask the effect of CP ratio on anthropometry, we also measured household garden productivity, wealth, and social status. Regression models of the age-standardized variables indicated a significant negative impact of CP ratio on child growth and nutrition. The age-standardized height and weight of children in households with the largest CP ratio (10) were 1.38 and 1.44 standard deviations, respectively, below those of children in households with the smallest CP ratio (2). Surprisingly, garden productivity, wealth, and status had little to no effect on the fitness proxies. There was, however, an interesting and unexpected interaction between status and sex: for females, but not males, higher father status correlated significantly with higher values on the proxies. Am J Phys Anthropol 130:405–418, 2006.

Offspring can provide economic or biological fitness benefits. When these benefits increase with offspring number and quality, yet time and resources to invest in offspring are limited, parents face a tradeoff between having fewer "high-quality" vs. more "low-quality" offspring. This tradeoff can be in the form of economic returns, such as having fewer but better-educated children who can obtain higher-paying jobs, vs. having more, less well-educated children to work the family farm; or it can come in the form of biological fitness, such as having fewer but better-nourished children who are each more likely to survive and reproduce vs. having more, less well-nourished children who are each less likely to survive and reproduce (Lack, 1947; for theoretical details and evidence from nonhuman species, see Clutton-Brock, 1991; Roff, 1992; Stearns, 1992). The existence of such quantity-quality tradeoffs is a fundamental assumption of theoretical work in demography, economics, life history, and parental investment theory.

The demographic transition (dramatic reductions first in mortality and then in fertility in developing countries over the last two centuries) is rightly seen as a fundamental problem for evolutionary social scientists (Vining, 1986; Borgerhoff Mulder, 1998). Parents in populations with unprecedented access to food, resources, and effective medical care are having significantly *fewer* children (Coale and Treadway, 1986), not more, as life history and parental investment theory predict. Further, it is the wealthiest individuals in posttransition societies who have the fewest children (Livi-Bacci, 1986), whereas in "traditional" societies, the opposite pattern holds (Hill and Kaplan, 1999 and references therein).

Evolution-minded scholars proposed a number of theories to resolve the dilemma, most of which invoke quantity-quality tradeoffs.[1] These theories implicitly or explicitly assume a mismatch between the contemporary environment and a parenting psychology that evolved to create fitness-enhancing tradeoffs in family size in ancestral environments. Limiting family size, the arguments go, currently results in large increases in fitness proxies like status, cultural success, professional attainment, and wealth, but maladaptively decreases fitness itself (Borgerhoff Mulder, 1998; Boyd and Richerson, 1985; Irons, 1983; Kaplan et al., 1995; Kaplan, 1996; Kaplan and Lan-

[1]There are a number of nonevolutionary theories that we do not discuss.

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caster, 2000; Lancaster and Lancaster, 1987; Lancaster, 1997; Turke, 1989; Alexander, 1974; Chagnon, 1988). Some of these models bear close similarity to models developed by economists that assume that when increasing but costly investment in human capital (i.e., education) yields increasing economic payoffs, parents will reduce family sizes (e.g., Becker, 1993; Joshi et al., 1996 and references therein). Other researchers (Rogers, 1990, 1995; Borgerhoff Mulder, 1998; Luttbeg et al., 2000) argued that intergenerational transfers of wealth are diluted by large families, potentially reducing fitness, so parents can maximize fitness by limiting family size relative to transferable wealth.

Quantity-quality tradeoffs also form the foundation of a large and growing body of research by social scientists with an evolutionary perspective, investigating patterns of differential child investment. Proxy measures of offspring investment include rates of infanticide, homicide, and abuse (e.g., Daly and Wilson, 1984, 1988), emotional attitudes toward and interactions with infants (e.g., Hagen, 1999, 2002; Hagen and Barrett, n.d.; Mann, 1992), patterns of wealth inheritance, including bridewealth and dowry (e.g., Borgerhoff Mulder, 1995; Dickemann, 1981; Mace, 1998), direct care (e.g., Betzig and Turke, 1986; Hewlett, 1991), educational investment (e.g., Borgerhoff Mulder, 1998), and birth weight and lactation (e.g., Gaulin and Robbins, 1991; Margulis et al., 1993; San José et al., 1997).

EVIDENCE FOR A QUANTITY-QUALITY TRADEOFF IS MIXED

Despite its importance to many evolution-minded theories of demography, fertility, and parenting, evidence for a quantity-quality tradeoff in humans is mixed. For very young offspring, there is compelling, albeit indirect, evidence for a quantity-quality tradeoff. Twinning is rare in humans, with litter-sizes almost always equal to one. Further, the existence of fertility-limiting mechanisms like lactational amenorrhea (Wood, 1994) suggests the evolutionary importance of constraints or tradeoffs in simultaneous investment in multiple infants. The fact that women in all societies regularly care for multiple offspring at different life stages, however, tempers this conclusion, suggesting that if there is a tradeoff, it is not so extreme as to make care of multiple offspring impossible (Hill and Kaplan, 1999).

The few studies that attempted to directly detect a quantity-quality tradeoff in fitness, usually measured as number of children or grandchildren, had decidedly mixed results. Blurton-Jones (1986), for example, found that among the !Kung, offspring mortality increased sharply for shorter interbirth intervals, offsetting the higher number of live births. This study, however, was criticized (Harpending, 1994; Hill and Hurtado, 1996; for a response, see Blurton-Jones, 1994). Borgerhoff Mulder (2000) found that, controlling for wealth, Kipsigis women who produced intermediate numbers of offspring maximized their number of grandchildren. No such effect was found for men, however, who maximized their number of grandchildren by maximizing their number of offspring. The controlled study by Hill and Hurtado (1996) of the Ache found no detectable tradeoff from shorter interbirth intervals. Those with the shortest interbirth intervals had the most offspring. The controlled study by Kaplan et al. (1995) of New Mexico men similarly found maximizing number of offspring maximized number of grandchildren, although high fertility levels had a negative impact on offspring education and income.

As each of the above studies acknowledged, quantity-quality tradeoffs can be difficult to detect for a number of reasons. Individuals in "good condition" may be able to have more offspring without suffering a tradeoff, so studies must control for "phenotypic" correlates like wealth, genetic quality, and social resources. Although several studies did attempt to control for at least some such factors, it is conceivable that these controls were inadequate. Tradeoffs may be a threshold phenomenon (Jonsson and Tuomi, 1994), or they may simply be undetectable in populations such as New Mexico that are far below carrying capacity. It is possible that tradeoffs only appear during periods of extreme stress, or that the tradeoff is in fact suffered by relatives of high-fertility individuals, who, by helping to raise many nieces and nephews, reduce their own fertility (Hill and Hurtado, 1996). Finally, if food-sharing were extensive in the study population, large families might receive enough food from small families to offset any food shortages caused by having more mouths to feed.

FITNESS PROXIES

The studies that failed to find a quantity-quality effect for one or both sexes are often those that measured fitness (e.g., Borgerhoff Mulder, 2000; Hill and Hurtado, 1996; Kaplan et al., 1995). Fitness measures integrate *every* factor that has an impact on reproduction. This is an advantage because no relevant factor is omitted. It is a disadvantage because a single and perhaps population-unique factor could mask effects that would otherwise manifest themselves. The study by Kaplan et al. (1995), for example, was conducted in a population that had undergone a demographic transition, had extremely low rates of child mortality relative to nontransition populations, and was very likely far below carrying capacity: all factors that could mask the effects of a quantity-quality tradeoff. Even aspects of pretransition populations, such as access to vaccines, could mask quantity-quality tradeoffs in fitness.

An alternative research strategy is to explore quantity-quality tradeoffs, not in fitness, but in *proxies* for fitness like child nutrition and growth. Although this has the disadvantage that the relationship of proxies with fitness might be complex and difficult to ascertain, it has several advantages. Proxies can reflect short-term quantity-quality effects that might have had significant fitness consequences over evolutionary time, even if such consequences are not detectable in the study population. Kaplan et al. (1995), for example, *did* find a tradeoff in education, a potential fitness proxy. More importantly, they offer the possibility of isolating population-specific factors that might be masking quantity-quality effects, as well as identifying tradeoffs that are common across many populations.

A number of standard anthropometric indices are promising fitness proxies, at least in food-constrained populations. Height is an index of skeletal growth. Deficits in height-for-age generally indicate long-term, cumulative inadequacies of health or nutrition. Deficits in weight-for-age can indicate either acute or chronic inadequacies. Nutrition inadequacies largely involve deficiencies in energy and protein intake, but there is increasing evidence that deficiencies in micronutrients like vitamin A, iron, and zinc may also play an important role (WHO, 1995).

Skinfold thicknesses measure skin and adipose tissue, correlate well with overall body fat (Lohman, 1981; Sarría

et al., 1998), and are thus informative indices of nutritional status. Children's body fat reflects relatively short-term access to food provided by parents and other group members, food they forage for themselves, and the negative impact of disease (e.g., diarrhea). Arm and calf circumferences index fat, muscle, and bone development. Children's muscle and bone development reflects long-term access to resources, including protein, from parents and their own foraging efforts, as well as the negative impact of chronic disease.

Anthropometric deficits in height and weight for age appear to be risk factors for increased child morbidity, including acute lower respiratory infections and diarrhea (Zaman et al., 1996; Ballard and Neumann, 1995; Baqui et al., 1993a,b; el Samani et al., 1988). Poor growth is also associated with impaired cognitive development, poor performance in school, and a host of other deficits (Martorell and Haschke, 2001; Semba and Bloem, 2001). Importantly, although the predictive ability for death of anthropometric indicators is generally low (Pelletier, 1991), anthropometric deficits in height-for-age and weight-for-age are clearly associated with increased child mortality rates (Pelletier et al., 1993; Pelletier and Frongillo, 2003). With no clear threshold effect, this supports the use of anthropometric measurements as proxies for fitness.

The negative impact of anthropometric deficits can persist across generations. Childhood deficits often result in reduced adult size (Martorell et al., 1992), reducing work capacity (e.g., Spurr et al., 1977) and thus the ability to provide food for offspring. Additionally, short women are at greater risk for obstetric complications due to smaller pelvic size, and give birth to lower birth-weight babies (Prasad and Al-Taher, 2002). Low birth-weight babies, in turn, are more likely to suffer anthropometric deficits at later ages (Binkin et al., 1988).

Several previous studies, most with a public health rather than parental investment theory perspective, found a negative relationship between family size and child health and nutrition (e.g., Ballard and Neumann, 1995; Hagen et al., 2001; Nanda, 1996; Rao and Gopalan, 1969; Wolfe and Behrman, 1982). Some studies also failed to find such a relationship (e.g., Hesketh et al., 2003; Tada et al., 2002; Taha, 1979). Many of the studies were conducted in urban populations or rural populations practicing intensive agriculture. For those studies that found an effect, it could be argued that such populations do not practice the extensive food-sharing typical of small-scale societies, food-sharing that might mask quantity-quality tradeoffs. For those studies that did not find an effect, it could be argued that such populations either have ready access to food, or to healthcare programs that provide supplemental nutrition. In either case, it remains an open question to what extent family-size effects on child nutrition and growth exist in the small-scale societies that frequently inspire evolutionary theories of human parenting.

STUDY POPULATION

A focus on fitness proxies such as anthropometric indices of nutrition and growth might begin to clarify whether quantity-quality tradeoffs exist, at least in small-scale societies with constrained access to food. The present study was conducted in a village of Shuar hunter-horticulturists who had fundamental features in common with most small-scale societies, including a kin-based social organization, a subsistence economy, and food-sharing. The Shuar are a large subgroup of the Jivaro, a Native South American group which also includes the Huambisa, Aguaruna, Achuar, and Shiwiar. The village was located on the western edge of the Ecuadorian Amazon and the lower, eastern slopes of the Andes, at an altitude of approximately 1,000 m. Plantains (*Musa balbisiana*) and sweet manioc (*Manihot esculenta*) are the principle dietary staples, supplemented by shotgun and blowgun hunting and fishing, and purchases of food in a nearby town. Timber and cattle sales were an important source of cash, and cash-crops were of limited but increasing importance. Several decades of contact with Protestant missionaries had precipitated a decline in traditional practices such as polygyny and warfare. All residents regularly spoke Shuar, but most under the age of 60 also knew Spanish. The majority of residents were closely related descendents of two brothers who helped found the village several decades ago.

The village had 306 residents in 50 households during our study, with a sex ratio of 120:100. About half lived in or very near the village center, mostly in wood-plank dwellings. The rest lived within a several-kilometer radius. Most households consisted of a single nuclear family, and only two men were openly polygynous at the time of the study. This was a small, kin-based community with widespread food-sharing that might buffer quantity-quality tradeoffs. The average coefficient of relatedness between residents was 0.045, which was relatively high (for comparison, the coefficient of relatedness between second cousins, i.e., individuals with different grandparents but sharing a pair of great-grandparents, is 0.031). On average, each resident was related by blood to nearly half the village (mean number of consanguineal kin, 147; SD = 74.7). We regularly observed hunted and gathered foods being shared between households, and the sharing of prepared meals of cultivated foods was common. Quantifying food-sharing is a very challenging problem (e.g., Gurven, 2004) that we did not attempt to address, so we could not control for this important variable. A failure to detect a quantity-quality tradeoff could therefore possibly be attributed to food-sharing; success in detecting a quantity-quality tradeoff, on the other hand, would suggest that food-sharing was insufficient to offset the cost of a large family. We made no attempt to determine whether parents were producing an optimal number of offspring (i.e., whether completed family size or reproductive decision-making optimized fitness).

METHODS

Participants

We sampled 138 (45%) members of the village in 32 different households. This was an opportunity sample comprising almost all families living near the village center. The village president scheduled many of our visits to family homes, and he also arranged for us to interview and measure children attending the local school, contingent upon the permission of their parents. We will pay particular attention to a subsample of 85 individuals (62% of the sample), labeled *dependents*, between ages 3–20 years (M = 10.5; SD = 4.5), who were not parents, heads of household, or married. Dependents came from 27 different households, and included 48 males and 37 females. One child with an obvious neurological condition was omitted from the study. The study protocol was approved by the University of California, Santa Barbara (UCSB) Human Subjects Committee, as well as by village leaders, and all participants gave informed consent. Parents of participants under age 18 signed permission forms allowing

their children to participate. Participating families received a small package of gift items (e.g., a plastic flashlight with batteries).

Measures

Considerable effort was put into accurately determining participants' ages, particularly the ages of children and adolescents. Initial ages were obtained from a census conducted by the village president in the preceding year. These ages were then checked against an older genealogy assembled by one of us (H.C.B.) during an earlier field visit, and by checking ages against identity cards and birth certificates when these were available. Availability of an identity card or birth certificate, however, did not guarantee the accuracy of an individual's age, since these documents were sometimes obtained years after the fact (we know some were inaccurate). In the two cases when age discrepancies could not be resolved, we picked the age (census, genealogy, or documents) that minimized the deviance of that individual's age-predicted height from their actual height. Because our study aimed to model within-village variance in height-for-age and other measures, this procedure was conservative with respect to our hypotheses. That is, it would maximize the variance in height explained by age alone, minimizing the residual variance that could then be explained by our predictor variables.

All anthropometric measurements were obtained by a single researcher (E.H.H.) according to guidelines by WHO (1995). Participants were informed that they were under no obligation to participate in the study, and could refuse to answer any questions or refuse to have measurements taken. Participants were asked to remove their shoes and any heavy articles of clothing prior to measurement. Height was measured to the nearest millimeter using an aluminum anthropometer by having participants stand on a wooden floor, feet together with their heels, buttocks, and head against a vertical wall. Weight was measured to the nearest 50 g, using a digital field scale (Seca model 770) that had been leveled on a hard, flat surface. All participants were wearing lightweight cotton clothing, and no adjustments were made for clothing weight. Two consecutive readings of triceps and abdominal skinfolds, and mid-upper arm and calf circumferences, were taken using Lange calipers, with circumferences read to the nearest millimeter using a plastic tape measure. These measurements were then averaged.

Anthropometric status is influenced by increased rates of nutrient utilization (as in many infectious diseases like diarrhea), and/or impaired absorption or assimilation of, or access to, macro- and micronutrients (WHO, 1995). Skinfold thicknesses in particular fluctuate rapidly in response to changes in nutritional intake (e.g., Mascarenhas et al., 1998). Participants were therefore asked whether they had experienced any diarrhea, vomiting, fever, or other illness. Because we often interviewed younger children whose recall of more distant events might be questionable, we limited our inquires to illness in the last week. Almost all data collection took place in groups, so these questions could usually not be asked in private. Two women known to be pregnant were excluded from the study.

We were interested in the impact of the number of siblings on children's anthropometry. Although most of the households in our sample consisted of a single nuclear family, a few included grandparents, grandchildren, in-laws, or other relatives. Because the impact of the number of resident siblings on anthropometry is a special case of the impact of overall household size on anthropometry, because number of biological siblings in each household was highly correlated with household size ($r = 0.95$, $P << 0.001$), and because we did not want to unnecessarily complicate our analyses, we decided to operationalize family size as household size, which we will term *Consumers*. The number of *Consumers* was the size of each nuclear family (father, wife or wives, and biological offspring), plus the number of extended family members like grandparents, grandchildren, in-laws, stepchildren, or other relatives living in a household. The majority of calories consumed by village members came from family gardens, and older teenage and adult women do almost all of the gardening. Older teenagers and adult men primarily work in cash-producing enterprises like lumbering and cash-cropping, but also engage in limited hunting and fishing (which yield few calories), and in household activities like garden-clearing and house construction. Since the relationship between calories and nutrition and growth is much clearer than the relationship between cash or child nutrition and growth, we operationalized the number of *Producers* as the number of women aged 15 or older in each household.[2] The ratio of *Consumers* to *Producers* (*CPRatio*) in each household was our primary predictor variable of interest.

In order to determine whether the use of *CPRatio* was an acceptable approximation of an expanded model of *Consumers* and *Producers* that would include siblings and nonsiblings as separate predictor variables, we also analyzed this expanded model. Results are presented in the Appendix, and are very similar to those obtained using *CPRatio*. Due to our limited sample size, and the restriction on the total number of model variables which that imposes, we did not use the expanded model with our illness variables or the phenotypic control variables (described next).

Family wealth, an important control variable, was operationalized in three ways: family garden productivity (*Garden productivity*), father's wealth (*Wealth*), and father's social status (*Father status*) in the village (almost all heads of household were men). *Garden productivity* was measured by asking the head of each household the size (in square meters) and soil quality (1, poor; 2, medium; 3, good) of each of their gardens. The size of each garden was multiplied by its soil quality to obtain a garden productivity score; these scores were then summed for all gardens owned by the head of household to obtain their total *Garden productivity* score. Due to time constraints, we were only able to interview 21 heads of the 32 households in our sample.

Wealth and *Father status* were measured by asking four male informants to rank each adult male according to their wealth and the respect they were accorded by other village members (for rank, we used a binary comparison/sorting technique). *Wealth* and *Father status* scores for each adult male were then computed by averaging these rankings. Note that *Wealth* and *Father status* rankings were reverse-coded: lower values correspond to higher rankings, with a value of 1 being the highest rank. Informant rankings of *Wealth* and *Father status* were highly concordant, with a Cronbach's alpha of 0.95 and 0.92, respectively.

[2]An analysis (not reported) that included older teenage and adult men as producers did not increase the variance explained by our models, providing post hoc justification for this definition of *Producers*.

Compositing and transforming variables

HeightZ, *WeightZ*, and *BMIZ* were each individual's height, weight, and BMI Z-scores with respect to the 2000 National Center for Health Statistics/Centers for Disease Control (NCHS/CDC) growth curves (Kuczmarski et al., 2002). There are no NCHS/CDC curves for skinfold thicknesses and body circumferences; nor is there an appropriate reference population, so we constructed an internal "reference" for the latter variables as follows. We first composed a *Bodyfat* index as the sum of the Z-scores of triceps skinfold thickness and abdominal skinfold thickness for each individual (we used Z-scores to equally weight the variance of triceps and abdominal skinfold thicknesses; these Z-scores were *not* with respect to a reference population). We similarly composed a *Circumference* index as the sum of the Z-scores of calf circumference and mid-upper arm circumference for each individual. Using loess regression, we then fitted a smooth curve to *Bodyfat* and *Circumference* as a function of age, computing separate curves for each sex among *dependents* (Fig. 1). *BodyfatR* and *CircumferenceR* were the standardized residuals of *Bodyfat* and *Circumference* relative to the fitted age curve for each. We similarly computed *HeightR*, *WeightR*, and *BMIR*. Again, separate curves were computed for each sex. As we will see, the results for *HeightR*, *WeightR*, and *BMIR* were similar to those for the standard *HeightZ*, *WeightZ*, and *BMIZ*, which offers partial validation of this approach to variable construction. Results reported below were robust to the choice of smoothing parameter within the range 2/3–3/4.

A principle objective of our study was to examine the relationship of between-household variation in *CPRatio* vs. anthropometric variables. With respect to this relationship, dependents within a household did not represent independent cases. Further, our theoretical interest was in the average impact of *CPRatio* on child anthropometry. We therefore pursued two analytical strategies. First, we computed the average *BodyfatR*, *CircumferenceR*, *HeightZ*, *WeightZ*, *BMIZ*, *HeightR*, *WeightR*, and *BMIR* of all dependents in each household. This was possible because the latter variables had already been corrected for age and sex. These household averages (*HA*) were labeled *HABodyfatR*, *HACircumferenceR*, *HAHeightZ*, *HAWeightZ*, *HABMIZ*, *HAHeightR*, *HAWeightR*, and *HABMIR*. Second, we analyzed mixed-effects models of all dependents, with household as a grouping factor.

We predicted a negative impact of *CPRatio* on all anthropometric variables and their averages, except perhaps those involving BMI. If family size negatively impacted both weight and height, then since BMI is a ratio of weight to height (weight/height^2), these negative effects might at least partially cancel out. An effect of *CPRatio* on BMI would be particularly sensitive to the relative impact of family size on weight vs. height. Despite our concern that BMI might not be an appropriate measure, we included it in our analyses because it is widely used.

All variables were screened for outliers and conformance to conditions of the statistical tests in which they appear. Unless otherwise noted, a significance level of $\alpha = 0.05$ was used for all tests. Statistical analyses were computed using R 1.9.1 and SPSS 10.

RESULTS

Descriptive statistics

Descriptive statistics of dependents' variables are given in Table 1. It is possible that the very low Z-scores of some dependents' height and weight relative to the US reference population were due to overestimating their ages or measurement errors. However, their standardized residuals relative to their population (*HeightR*, *WeightR*) were much less extreme, with no value exceeding 3 Z-score units. Examining histograms of these variables did not reveal any clear outliers (Fig. 2), and adult Shuar are short: adult height Z-scores ranged from -3.4 to -0.7 ($M = -2.3$), similar to results of Orr et al. (2001), who found that mean adult Achuar height Z-scores were ≤ -2. Because we were interested in within-population rather than between-population comparisons, because eliminating these cases could bias the sample, and because age and measurement errors should be random with respect to our hypotheses, we did not remove them.

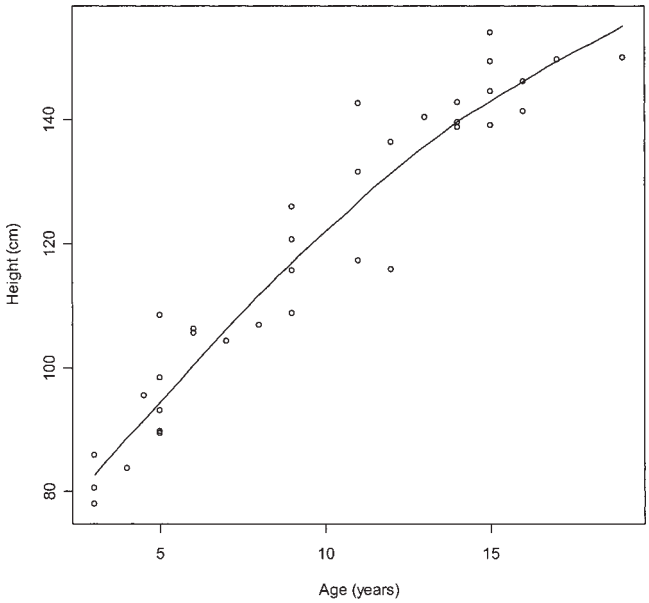

Fig. 1. Girls' height vs. age. Curve fit by loess regression (smoothing parameter = 0.75, degree = 2). *HeightR* is standardized residual of an individual's height relative to fitted curve. Same procedure was followed for each of other anthropometric variables.

Average anthropometry *vs.* consumer/producer ratio

We first examined the correlation of *CPRatio* with household averages of anthropometry. Shapiro-Wilk tests and inspection of histograms revealed that *CPRatio*, *HAWeightR*, and *HABMIR* deviated from a normal distribution. Further, a single household had exceptionally high average values on several anthropometric variables, even given its low *CPRatio* (the head of household was one of the highest-status males). This outlier had undue influence on some analyses. We therefore decided to use iterated reweighted least squares (IWLS) to fit a robust regression model with Tukey's bisquare M-estimator. Because the asymptotic approximations used by this procedure to estimate standard errors may not be trustworthy in such a small sample (Fox, 1997), we also used bootstrapping to estimate standard errors and confidence intervals. Household averages were weighted by household size. For comparison, we also computed the nonparametric Spearman's rank correlation, which imposes no constraints on variable

TABLE 1. Descriptive statistics for Dependents *subsample*[1]

	N	Range	Mean	SD
Individual response variables				
HeightR	84	−2.39–2.31	0.0	1.0
WeightR	85	−2.79–2.82	0.0	1.0
BMIR	84	−2.40–2.27	0.0	1.0
BodyfatR	77	−2.37–4.15	0.0	1.0
CircumferenceR	80	−2.43–3.79	0.0	1.0
HeightZ	84	−4.60–0.22	−2.60	1.06
WeightZ	85	−5.07–0.77	−1.51	1.22
BMIZ	84	−1.87–2.08	0.269	0.747
Household response variables				
HAWeightR	27	−1.24–2.90	*na*	*na*
HABMIR	27	−0.99–2.65	*na*	*na*
HABodyfatR	27	−1.12–2.11	*na*	*na*
HACircumferenceR	27	−1.44–2.57	*na*	*na*
HAHeightz	27	−3.85–0.13	*na*	*na*
HAWeightZ	27	−2.88–0.58	*na*	*na*
HABMIZ	27	−0.48–1.14	*na*	*na*
HAHeightR	27	−1.47–1.98	*na*	*na*
Predictor variables				
CPRatio	27	2–10	4.93	2.25
Parents	85	0–2	1.88	0.42
Garden productivity	27	3,600–97,200	40,681.0	26,246.0
Wealth	27		Average rank score	
Father status	27		Average rank score	
Vomiting	79	Present/absent	7 present (9%)	
Fever	79	Present/absent	13 present (16%)	
Diarrhea	79	Present/absent	6 present (8%)	
Other illness	79	Present/absent	33 present (42%)	

[1] Response variables are in standardized units. Means and SDs for household averages are not reported because in analyses, households were weighted by household size.

distributions and is robust to outliers. Standardized coefficients are reported for all models.

Results revealed a significant, negative impact of *CPRatio* on household averages of all anthropometric variables of dependents except *HABMIR* and *HABMIZ*, even without controlling for *Garden productivity*, *Wealth*, or *Father status*. Bootstrapped standard errors were larger in all analyses, but 95% CIs excluded zero for all but the BMI variables. There was little difference between robust regression coefficients and rank correlation coefficients. The rank correlation of *HABMIZ* was significant, even though its regression coefficient was not (Table 2).[3]

Mixed-effects models

Modeling household means ignores interindividual variability, but ordinary linear regression (OLR) models of the sample of dependents would ignore any intrahousehold correlation in anthropometric responses and would thus overestimate the significance of model parameters. So, using

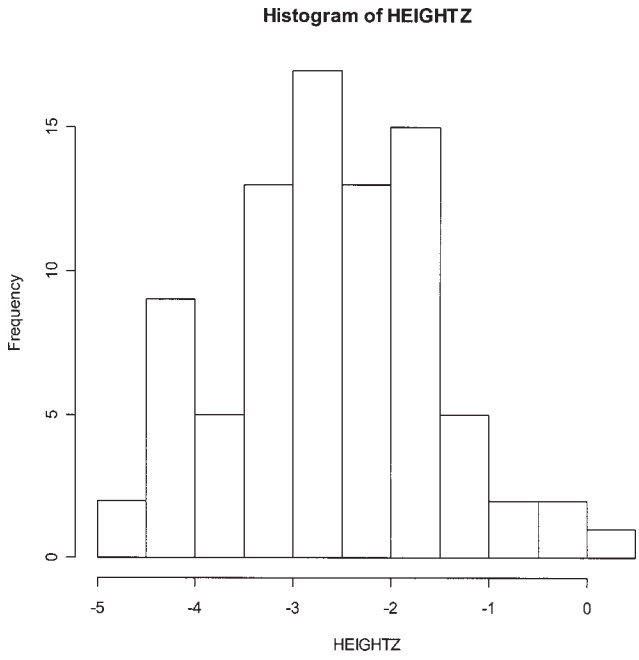

Fig. 2. Histogram of dependents' *HeightZ* (Z-scores of height relative to NCHS/CDC standards). Cases below −3 were not clearly outliers, so they were included in study.

restricted maximum likelihood estimation (REML), we then fitted a linear mixed-effects model (LME) of the form:

$$y_{ij} = b_i + \beta x_i + \varepsilon_{ij}\ i = 1, \ldots, M;\ j = 1, \ldots, n_i$$
$$b_i \sim N(0, \sigma_b^2),\ \varepsilon_{ij} \sim N(0, \sigma^2) \quad (1)$$

[3]The value of *CPRatio* pertains only to the present. Over the course of an individual's development, the *CPRatio* of their household will change as new siblings are born and females reach the age of 15 and begin producing. Although *BodyfatR*, *WeightR*, and *WeightZ* (and therefore possibly *BMIR* and *BMIZ*) are probably best predicted by current household conditions, it is possible that an individual's *HeightR* and *HeightZ*, and perhaps *CircumferenceR*, would be better predicted by a measure that averaged *CPRatio* over the life course. For each individual, we estimated the *CPRatio* in their family at each year of their life, and then averaged these ratios, resulting in a *MeanCPRatio* for each individual. Compared to *CPRatio*, *MeanCPRatio* was a slightly but not significantly better predictor of *HeightR* and *HeightZ*, and a worse predictor of all other growth and nutrition variables. We therefore restricted all analyses to *CPRatio*.

*TABLE 2. Robust regression of household averages of dependents' anthropometric variables with consumer/producer ratio (*CPRatio*) in 27 households (cases weighted by household size)*[1]

Response variable	r_{robust}	SE	*t*	*P*	Bootstrap estimates			Spearman rank correlation
					Bias	SE	95% CI	
HAHeightR	−0.44	0.09	−4.95	<0.001	−0.007	0.22	−0.93–−0.06	r = −0.46, *P* = 0.009
HAWeightR	−0.41	0.06	−6.51	<0.001	0.051	0.17	−1.00–−0.16	r = −0.55, *P* = 0.002
HABodyfatR	−0.40	0.08	−5.03	<0.001	−0.017	0.19	−0.81–−0.06	r = −0.47, *P* = 0.007
HACircumferenceR	−0.46	0.08	−5.90	<0.001	0.060	0.23	−1.00–−0.14	r = −0.51, *P* = 0.003
HABMIR	−0.23	0.08	−2.72	0.011	−0.040	0.20	−0.99–0.05	r = −0.32, *P* = 0.054
HAHeightZ	−0.40	0.09	−4.44	<0.001	−0.023	0.16	−0.68–−0.04	r = −0.49, *P* = 0.005
HAWeightZ	−0.49	0.09	−5.52	<0.001	0.013	0.18	−0.82–−0.11	r = −0.45, *P* = 0.010
HABMIZ	−0.41	0.10	−4.21	<0.001	0.045	0.22	−0.75–0.091	r = −0.35, *P* = 0.039

[1] Bootstrapped estimates were computed using 5,000 bootstrap replications.

TABLE 3. Linear mixed-effects models for each of the anthropometric variables vs. CPRatio[1]

Response variables	N/groups	Fixed effects				Random effects				*CPRatio* impact (SE)
		β	SE	*t*	*P*	σ_b	σ	L.ratio	*P*	
HeightR	84/27	−0.40	0.13	−3.20	0.004	0.37	0.85	2.34	0.130	1.28 (0.27)
WeightR	85/27	−0.43	0.14	−3.13	0.004	0.45	0.79	6.21	0.013	1.33 (0.27)
BodyfatR	77/27	−0.39	0.13	−2.99	0.006	0.37	0.85	2.89	0.089	1.23 (0.29)
CircumferenceR	80/27	−0.44	0.13	−3.33	0.003	0.43	0.79	5.03	0.025	1.40 (0.27)
BMIR	84/27	−0.27	0.15	−1.78	0.086	0.56	0.79	10.85	0.001	0.87 (0.29)
HeightZ	84/27	−0.41	0.14	−2.96	0.007	0.45	0.82	3.58	0.058	1.38 (0.29)
WeightZ	85/27	−0.42	0.14	−2.95	0.007	0.48	0.78	8.75	0.013	1.44 (0.33)
BMIZ	84/27	−0.27	0.14	−1.98	0.059	0.43	0.86	4.79	0.029	0.64 (0.21)

[1] Likelihood ratio test was used to determine whether LME had significantly better fit than OLR. For heteroscedastic LME of *WeightZ*, exponential variance parameter δ = −0.31. *CPRatio Impact* was predicted population Z-score value of each anthropometric variable at *CPRatio* of 2 (lowest value among dependents) minus Z-score value at *CPRatio* of 10 (highest value among dependents). Thus, mean height of children in households with *CPRatio* of 10 was 1.28 standard deviations below those in households with *CPRatio* of 2.

for each anthropometric variable, where y_{ij} was the anthropometric response of the *jth* dependent in the ith household, x_i was the *CPRatio* of the ith household, β was the fixed-effect (population-level) slope, b_i represented the random, household variability in intercept, and the ε_{ij} were individual errors. Using the Akaike information criterion (AIC) and likelihood ratio tests, we compared each LME to a simpler OLR:

$$y_i = \beta x_i + \varepsilon_i \quad (2)$$

(where i indexes individuals) to determine whether including the random effects for household significantly improved the model (for consistency, we always report the LME along with the likelihood ratio and *P*-value). Standardized coefficients are reported for all models.

The LME models we used assume that 1) within-group errors are independent and normally distributed and independent of random effects, and 2) random effects are normally distributed and are independent for different groups (Pinheiro and Bates, 2000). For each model, we assessed the validity of these assumptions using a variety of tests, including Q-Q plots and plots of standardized residuals vs. fitted values. *WeightZ* was notably heteroscedastic. We therefore fit a heteroscedastic LME, with an exponential variance function of form:

$$\mathrm{Var}(\varepsilon_{ij}) = \sigma^2 \exp(2\delta v_{ij}) \quad (3)$$

where in our analysis the v_{ij} were fitted values. The heteroscedastic LME was significantly better than the OLR and marginally significantly better than the homoscedastic LME. The variance of standardized residuals vs. fitted values of the heterodcedastic LME was markedly improved (more homogeneous), so we report the heteroscedastic LME for *WeightZ*.

All variables showed a significant, negative correlation with *CPRatio* (*BMIR* and *BMIZ* were significantly correlated with *CPRatio* in the OLR; in the LMEs, the *βs* were not significant, but the overall models, including random effects, were significantly better than the OLRs). Including a random household effect significantly improved models for *WeightR*, *CircumferenceR*, *BMIR*, *WeightZ*, and *BMIZ*, indicating significant intrahousehold correlations for these variables. Adding a random household effect did not significantly improve models for *HeightR*, *BodyfatR*, and *HeightZ*. All models had similar slopes and random effects (Table 3). A scatter plot of *HeightZ* vs. *CPRatio*, illustrating their negative relationship, can be seen in Figure 3.

To test whether *Parents*, the number of biological parents residing with dependents, was a predictor of anthropometry, we included *CPRatio* and *Parents* in multivariate models of children's anthropometry. *Parents* was not a significant predictor in any model (*P*-values ranged from 0.23–0.91). This negative result might be due to the small number of dependents with no biological parents (3) or only one parent (4) in our sample.

"Phenotypic" control variables

Parents with large gardens, wealth, or high status might be able to have large families at lower or no "cost" relative to other families. Controlling for these variables

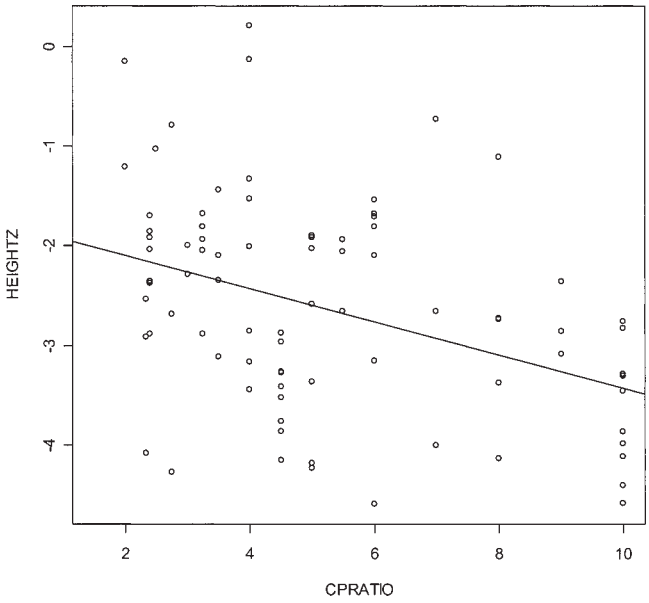

Fig. 3. Dependents' height Z-scores as function of household C/P ratio. Line was fit by robust regression.

could therefore increase the predictive ability of the consumer/producer ratio.

Garden sizes ranged from 1,200–32,400 m^2 (M = 15,964; SD = 9,475). Chayanov (1966) claimed that there should be a positive relationship between the number of consumers and household production, and most tests confirmed this relationship, with correlation coefficients ranging from approximately 0.30–0.90 (for a cross-cultural examination of Chayanov's theories, see Chibnik, 1984; see also Durrenberger, 1984; Hagen et al., 2001). We, too, found a strong, linear correlation between the number of household consumers and *Garden productivity* scores ($r = 0.71$, $P < 0.001$), a result that provided some post hoc validation of our *Garden productivity* measure.

As in the earlier analysis of household averages vs. *CPRatio* alone, a single household had exceptionally high average values of anthropometry, even given its low *CPRatio*. This outlier tended to inflate ordinary regression coefficients, so we again used IWLS robust regression models to explore the effect of phenotypic control variables, comparing robust coefficients with ordinary regression coefficients.

Models of household averages including *CPRatio*, *Garden productivity*, and their interaction as predictors did not explain significantly greater variance in any of the household averages of anthropometric variables than that accounted for by *CPRatio* alone (all *P*-values for the increase, >0.40). Multiple regression and mixed-effects models of all dependents that incorporated *CPRatio*, *Garden productivity*, and their interaction also failed to provide a better fit of the data than did *CPRatio* alone (all *P*-values for the increase, >0.30). It must be noted, however, that our sample size (21 households) was smaller because we did not have *Garden productivity* scores for every household in the *dependents* subsample.

Including *Father status* in a multiple linear regression model with *CPRatio* as predictors of household averages significantly increased the explained variance over *CPRatio* alone for two anthropometric variables: *HACircumferenceR* and *HABMIR*, with marginal significance for *HABodyfatR* and *HAWeightR*. The effects for all but *HABodyfatR* were an artifact of the outlier. For the marginally signficant *HABodyfatR* effect, the ordinary regression coefficients were essentially identical to the robust coefficients, so we report the ordinary coefficients. As expected, increasing *Father status* was associated with higher levels of *HABodyfatR* (*Father status* was reverse-coded). There was no significant interaction between *CPRatio* and *Father status*. We similarly included *Father status* in linear regression and mixed-effects models for all dependents. *Father status* significantly increased explained variance over *CPRatio* only for *BodyfatR*. Random effects were not significant, so we report the OLR. There were no significant interactions between *CPRatio* and *Father status* (Table 4).

Linear regression models that included *CPRatio*, *Wealth*, and their interaction as predictors did not explain significantly greater variance in any of the household averages of anthropometric variables than that accounted for by *CPRatio* alone, nor did corresponding OLR or LME models including all dependents (*P*-values for the increase ranged from 0.09–0.93); but again, our sample size was smaller.

Illness symptoms

Large family sizes might negatively impact child anthropometry directly, e.g., by reducing per capita food allocations, or they might do so indirectly, e.g., by increasing susceptibility to disease, as found in some studies (e.g., Ballard and Neumann, 1995); increased disease could then cause anthropometric deficits. Diarrhea and vomiting, for example, are known to have a negative impact on child anthropometry (WHO, 1995). To estimate the impact of disease on our *dependents* subsample, we simply coded whether four illness symptoms, *diarrhea*, *vomiting*, *fever*, and *other illness*, were present or absent in each participant. To explore the validity of our variables as indicators of disease, we ran *t*-tests to determine whether *dependents'* age- and sex-corrected anthropometric variables were negatively impacted by the presence of any of the illness conditions. We treated each dependent as an independent case for the purposes of these tests. *WeightR*, *BodyfatR*, and *CircumferenceR* differed significantly from a normal distribution, so the nonparametric Wilcoxon rank sum test was used to test for differences involving those variables. Comparing dependents' eight anthropometric variables grouped by four dichotomous illness variables resulted in 32 tests. Mean levels of anthropometric variables were lower for dependents with an illness condition in 29 of 32 tests, a highly significant ($P \ll 0.001$, exact test) pattern in the predicted direction (exceptions were the *HeightR* and *WeightZ* of dependents with a *fever*, and the *BMIZ* of dependents with *vomiting*). Even without adjusting our significance level for the large number of *t*-tests, however, only two (one-tailed) tests were significant (about the number expected by chance, given $\alpha = 0.05$): The *CircumferenceR* of dependents with *diarrhea* was significantly lower, and the *BMIR* of dependents with a *fever* was significantly lower. A Bonferroni correction rendered all tests nonsignificant. Individual illness symptoms were not significant predictors of anthropometry.

We also formed a composite illness variable indicating whether at least one of *diarrhea*, *vomiting*, or *fever* was present. We omitted *other illness* because this category often included vague health complaints by participants. Testing each anthropometric variable grouped on this

TABLE 4. Relationship of HABodyfatR *and* BodyfatR *to* CPRatio *and* Father status[1]

Response variables	Predictor variable	Coefficient	SE	*t*	*P*	F-test and effect size
HABodyfatR	*CPRatio*	−0.48	0.14	−3.30	0.003	RSE(24) = 1.41
	Father status	−0.32	0.17	−1.84	0.08	F(2,24) = 7.08, $P = 0.004$ $F_{increase} = 3.40$, $P = 0.08$ Adjusted, $R^2 = 0.32$
BodyfatR	*CPRatio*	−0.36	0.11	−3.41	0.001	RSE(72) = 0.91
	Father status	−0.23	0.11	−2.31	0.036	F(2,72) = 8.19, $P < 0.001$ $F_{increase} = 4.55$, $P = 0.036$ Adjusted, $R^2 = 0.16$

[1] RSE, residual standard error.

composite variable found no significant differences between dependents with no illness condition present and those with one or more illness conditions present. All eight means were in the expected direction, however, with lower levels of anthropometry for those with at least one illness condition, a significant pattern ($P = 0.004$, exact test). Although our general impression was that our population was relatively healthy, and illness might therefore have had little impact on nutrition and growth, these results cast some doubt on the validity of our illness measures.

To test whether the apparent negative impact of large consumer/producer ratios on anthropometry might be due to higher levels of illness in families with large ratios, we looked for any association of illness with *CPRatio*. We first compared the mean *CPRatio* for those with and without diarrhea, vomiting, fever, or other illness. In each case, the mean *CPRatio* for individuals with an illness symptom was *lower* than for those without that symptom. We then compared the mean *CPRatio* for those with any illness symptom vs. those with no illness symptoms (our composite illness variable). Again, the mean *CPRatio* for those with any illness symptom was lower than for those with none. These results reduce the possibility that the association of higher *CPRatio* with poorer anthropometry is a consequence of a confound between illness and higher *CPRatio*, although we only inquired about illness symptoms in the week prior to our anthropometric measurements.

Sex differences

Males and females have physiologically different growth curves for height, weight, muscle development, and deposition of body fat. Because we age- and sex-corrected each dependent's anthropometric variables, there should be no main effect of sex on any of these variables. There could, however, be significant interactions between sex and any of the covariates: *CPRatio*, *Father status*, *Garden productivity*, or *Wealth*. Given eight anthropometric variables and four covariates, there were 32 tests for interactions. Since we had no a priori predictions regarding sex differences, these were post hoc tests; we therefore adopted an $\alpha_{post\ hoc} = 0.05/32 \sim 0.00156$. Although three interactions of sex with *CPRatio* reached a conventional level of significance (0.05), none reached the $\alpha_{post\ hoc}$ level of significance (interactions reaching a conventional level of significance involved *CircumferenceR*, *BodyfatR*, and *BMIR*, which showed a strong, negative correlation with *CPRatio* for females, and a much weaker, negative correlation for males). No interactions of sex with either *Garden productivity* or *Wealth* even reached a conventional level of significance.

We then explored the interaction of sex with *Father status* (after first controlling for *CPRatio*).[4] Leverage analyses revealed an outlier in both the *HeightR* and *HeightZ* analyses (the same case for each). These regressions were rerun, using a robust regression model as above. There was little change in the coefficients, so we report the (more conservative) ordinary multiple regression model. Inspection of residuals vs. fitted values in the initial *WeightZ* model revealed notable heteroscedasticity, violating assumptions of the model. We therefore fit a heteroscedastic LME, with an exponential variance function of form (3) above, where again the v_{ij} were the fitted values. The heteroscedastic LME was significantly better than either the OLR or the homoscedastic LME, and variance of the residuals vs. fitted values was adequately homogeneous, so we report the heteroscedastic LME.

We found a consistent impact of sex on the relationship of *Father status* to several of our anthropometric variables; the interaction was significant at $\alpha_{post\ hoc}$ for *WeightZ*, marginally significant at $\alpha_{post\ hoc}$ for *HeightR*, *HeightZ*, and *WeightR*, marginally significant only at $\alpha = 0.05$ for *CircumferenceR*, and not significant for *BodyfatR*, *BMIR*, and *BMIZ* (although for the latter three variables, the effect was of similar size and in the same direction as for the other variables). The anthropometry of female dependents was positively influenced by higher *Father status*, but anthropometry of male dependents was not. This pattern was similar to that found for interactions with *CPRatio*. The LME was significantly better than the OLR for three variables: *WeightR*, *WeightZ*, and *CircumferenceR* (Table 5).

Impact of CP ratio on parents

Large families could also have a negative impact on parental nutrition, negatively affecting their ability to invest in current and/or future children. We therefore examined the effect of *CPRatio* on mothers' and fathers' *Weight*, *Circumference*, *Bodyfat*, and *BMI*. Descriptive statistics for mothers' and fathers' variables are listed in Table 6.

Unlike our analyses of dependents, we did not use age-corrected anthropometric variables because adult age-related differences in weight, body fat, etc. are much less likely due to developmental trajectories and more likely due to age-related differences in work levels, consumption of nutrients, or disease. Older parents, for example, were

[4] We investigated *Sex* as a within-household grouping factor, but no model grouping on *Sex* outperformed models using only a household level of grouping structure or no grouping structure.

TABLE 5. LME models of impact of CPRatio *and* Father status *on dependents' anthropometry that include interactions between* Father status *and* sex[1]

Response variables	Predictor variables	Coefficient	SE	*t*	*P*	Random effects
HeightR	Intercept	−0.01	0.16	−0.09	0.932	$\sigma_b = 0.31$
N = 81	*CPRatio*	−0.35	0.12	−2.92	0.008	$\sigma = 0.82$
Groups = 26	*Fstatus* (female)	−0.47	0.17	−2.81	0.010	Likelihood ratio = 1.52
	Sex (male)	0.08	0.20	0.39	0.699	$P = 0.22$
	*Fstatus*Sex* (male)	0.61	0.21	2.96	0.005	
WeightR	Intercept	0.02	0.16	0.11	0.912	$\sigma_b = 0.40$
N = 82	*CPRatio*	−0.39	0.13	−2.95	0.007	$\sigma = 0.77$
Groups = 26	*Fstatus* (female)	−0.52	0.17	−3.13	0.005	Likelihood ratio = 5.66
	Sex (male)	0.05	0.19	0.26	0.798	$P = 0.017$
	*Fstatus*Sex* (male)	0.54	0.20	2.77	0.008	
CircumferenceR	Intercept	−0.05	0.17	−0.28	0.784	$\sigma_b = 041$
N = 77	*CPRatio*	−0.42	0.14	−3.12	0.005	$\sigma = 0.77$
Groups = 26	*Fstatus* (female)	−0.43	0.17	−2.51	0.019	Likelihood ratio = 5.33
	Sex (male)	0.17	0.20	0.81	0.423	$P = 0.021$
	*Fstatus*Sex* (male)	0.39	0.20	1.91	0.062	
HeightZ	Intercept	−0.03	0.17	−0.15	0.878	$\sigma_b = 0.39$
N = 81	*CPRatio*	−0.36	0.13	−2.78	0.010	$\sigma = 0.79$
Groups = 26	*Fstatus* (female)	−0.50	0.17	−2.96	0.007	Likelihood ratio = 2.63
	Sex (male)	0.14	0.20	0.72	0.473	$P = 0.11$
	*Fstatus*Sex* (male)	0.58	0.20	2.87	0.006	
WeightZ	Intercept	−0.08	0.17	−0.45	0.654	$\delta = -0.46$
N = 82	*CPRatio*	−0.29	0.12	−2.51	0.019	$\sigma_b = 0.28$
Groups = 26	*Fstatus* (female)	−0.80	0.18	−4.57	<0.001	$\sigma = 0.76$
	Sex (male)	0.20	0.20	1.01	0.317	Likelihood ratio = 13.3
	*Fstatus*Sex* (male)	0.81	0.20	4.12	<0.001	$P = 0.001$

[1] Reported are treatment contrasts, which indicate change from base level (here, females). Hence, "*Fstatus* (female)" is slope of *Father status* on anthropometric variables for females (controlling for *CPRatio*), and "*Fstatus*Sex* (male)" is *change* in slope for males. Model of *WeightZ* was heteroscedastic LME.

more likely to have larger families, so age-related changes in nutrition could be attributed to increases in family size. If age were first factored out, the effect of interest (family size-related changes in nutrition) would then be difficult, and perhaps impossible, to detect.

Outliers caused correlation coefficients to be unreliable. We therefore used IWLS to fit a robust regression model as above. Results of the robust estimation occasionally differed dramatically from the ordinary coefficients, especially for fathers, so we report the robust regression coefficients (which were usually but not always more conservative). Due to small sample sizes, we again used bootstrapping to estimate standard errors and confidence intervals. No effects for mothers were significant, but all effects were of similar size and in the same direction, showing a negative impact of increasing *CPRatio* on nutrition. Results for fathers showed only an insignificant negative trend for *Bodyfat* with increasing *CPRatio* (Table 7).

DISCUSSION

Parental investment theory assumes a tradeoff between the quantity of offspring and their quality, an assumption we tested in a Native South American hunter-horticulturist village. We operationalized "quantity" as the number of household dependents, and "quality" as child growth and development. Our indices of nutrition and growth were several standard anthropometric measurements. Despite widespread food-sharing, we found a consistent, negative impact of consumer/producer ratio on each of our anthropometric indices (with the possible exception of BMI, perhaps because BMI is a ratio of weight-to-height^2, both of which were negatively impacted by *CPRatio*). Given the high correlation between number of household dependents and number of offspring (r = 0.95), we believe it is reasonable to infer that, for a given number of producers, increasing family size will have a modest but significant negative impact on several aspects of child growth and development. These results are bolstered by results presented in the Appendix. We cannot say whether this imposes a net fitness cost. Comparing households with the largest *CPRatio* in our sample, 10, to households with the smallest *CPRatio*, 2, that negative impact ranges from a 1.23 Z-score unit reduction for *BodyfatR* to a 1.38 Z-score unit reduction for *HeightZ* and a 1.44 Z-score unit reduction for *WeightZ*. Given the wide range of ages in our sample, *CPRatio* may impact differing aspects of growth in very young vs. older children and young adults, a hypothesis we cannot test with our limited sample size.

Results regressing *CPRatio* on the Z-scores of height, weight, and BMI with respect to the NCHS/CDC growth curves were very similar to results regressing *CPRatio* on residuals of these variables relative to our internal "standards." This suggests that our method of computing residuals relative to internal "standards" for our other variables (e.g., skinfold thicknesses) is probably acceptable.

The negative impact of increasing family size is offset by the increasing number of producers once daughters are old enough to work. Young girls also appear to do a considerable amount of childcare. Given that the calorie production benefit of female offspring will only be realized 15 years after their birth, a benefit that is often soon lost once

TABLE 6. Descriptive statistics for mothers' and fathers' nutrition variables

	N	Range	Mean	SD
Mothers				
Age (years)	21	18–61	34.0	10.91
Weight (kg)	21	46.45–76.60	57.25	8.14
BMI (g/m^2)	21	20.31–34.36	25.90	3.01
Body fat index Z-score	21	0.23–6.55	2.12	1.57
Circumference index Z-score	20	0.74–2.97	1.73	0.62
Fathers				
Age (years)	19	19–57	37.5	10.12
Weight (kg)	19	50.85–78.55	61.69	6.91
BMIR (g/m^2)	19	20.96–28.17	24.13	1.82
Body fat index Z-score	19	−2.09–6.34	0.35	1.78
Circumference index Z-score	19	−1.05–3.12	2.07	0.62

TABLE 7. Robust regression of mothers' and fathers' anthropometry on CPRatio[1]

			Bootstrap estimates		
Response variable	N	r_{robust}	Bias	SE	95% CI (BCa)
Mothers					
Weight	21	−0.24	0.029	0.22	−0.61–0.30
Body fat	21	−0.20	−0.015	0.20	−0.70–0.13
Circumference	20	−0.41	0.042	0.24	−0.81–0.17
BMI	21	−0.30	0.0005	0.20	−0.80–0.066
Fathers					
Weight	19	0.08	−0.045	0.29	−0.54–0.50
Body fat	19	−0.23	0.012	0.16	−0.44–0.19
Circumference	19	0.04	−0.18	0.54	−1.0–0.51
BMI	19	−0.10	−0.23	0.42	−0.78–0.88

[1] Bootstrap estimates based on 5,000 bootstrap resamples.

daughters marry and start new households with their husbands, and given that the hazards of lower nutrition and growth are experienced across childhood in the form, for instance, of increased morbidity, we do not believe that young daughters completely offset their measurable cost on siblings in the short and intermediate term (future studies in similar populations should attempt to quantify childcare of siblings by girls, however). Our study cannot determine whether daughters offset their cost over the long term.

Surprisingly, we found a negative impact of *CPRatio* on anthropometry, even without controlling for "phenotypic" qualities like wealth, status, and garden productivity. Even more surprisingly, controlling for these variables resulted in little or no improvement in our models. The lack of effect of garden productivity is perhaps the least surprising. Large tracks of uncultivated land were readily available. As family size grows, more hectares can be brought under cultivation. Thus, garden productivity may simply increase to accommodate family size. Clearing land for gardens requires considerable labor, which might be more easily recruited by fathers of high status and wealth. We did find a modest but significant correlation between higher *Father status* and larger families ($r = -0.32$, $P = 0.04$, $n = 32$), and a similar but not quite significant correlation between higher *Wealth* and larger families ($r = -0.31$, $P = 0.08$, $n = 22$) (*Father status* and *Wealth* rankings were both reverse-coded). Including *Father status* as a predictor of anthropometry produced modest improvements in a model containing the household average of body fat, as well as an OLR model of body fat of all *dependents*. But including *Wealth* as a predictor did not improve any model. The lack of effect of the phenotypic variables may indicate that there were few important differences on these dimensions among individuals in this small-scale, egalitarian society (yet village residents readily ranked members along them), or it may indicate that these dimensions simply had little impact on child anthropometry.

TABLE 8. Descriptive statistics for variables used in models reported in Appendix

Variable	N	Range	Mean	SD
Siblings	82	1–10	5.8	2.62
Nonsiblings	82	1–7	2.4	0.78
Producers	82	1–5	2.0	1.32
SCPRatio	82	0.5–8	3.8	2.21
NCPRatio	82	0.6–3.5	1.6	0.66

The most plausible interpretation of the negative impact of higher consumer/producer ratios on dependents' anthropometry is that it is more difficult to feed large families. Because disease can also negatively impact nutrition and growth, and might be confounded with higher consumer/producer ratios, we attempted to assess common illness symptoms in each dependent. Dependents with an illness symptom had *lower CPRatios*, reducing the probability of a confound. This suggests that the negative impact of higher consumer/producer ratios on child anthropometry may not be due to increased levels of disease, at least in this population. The lower mean indices on most of our anthropometric variables for dependents with an illness symptom suggest that our illness measures had some degree of validity, but these differences were small and not significant. It is probable that our self-report measure of illness symptoms like diarrhea had large errors because we could not interview most participants in private; we also only asked about illness in the week prior to our anthropometric measurements. A more

TABLE 9. Multivariate LME models of anthropometry for children with at least one biological parent in household[1]

Response variables	Predictor variables	Coefficient	SE	*t*	*P*	Random effects
HeightR	*SCPRatio*	−0.36	0.14	−2.70	0.012	$\sigma_b = 0.37$, $\sigma = 0.86$
N/groups: 81/27	*NCPRatio*	−0.02	0.13	−0.19	0.852	L.ratio = 1.86, $P = 0.17$
WeightR	*SCPRatio*	−0.30	0.15	−2.01	0.056	$\sigma_b = 0.52$, $\sigma = 0.78$
N/groups: 82/27	*NCPRatio*	−0.15	0.14	−1.11	0.276	L.ratio = 8.62, $P = 0.003$
BodyfatR	*SCPRatio*	−0.33	0.15	−2.24	0.034	$\sigma_b = 0.42$, $\sigma = 0.84$
N/groups: 74/27	*NCPRatio*	−0.11	0.14	−0.78	0.442	L.ratio = 3.97, $P = 0.046$
CircumferenceR	*SCPRatio*	−0.32	0.14	−2.25	0.034	$\sigma_b = 0.46$, $\sigma = 0.78$
N/groups: 77/27	*NCPRatio*	−0.18	0.14	−1.27	0.216	L.ratio = 6.07, $P = 0.014$
HeightZ	*SCPRatio*	−0.40	0.14	−2.78	0.010	$\sigma_b = 0.46$, $\sigma = 0.82$
N/groups: 81/27	*NCPRatio*	0.00	0.14	0.01	0.994	L.ratio = 3.14, $P = 0.076$
WeightZ	*SCPRatio*	−0.36	0.16	−2.27	0.032	$\sigma_b = 0.56$, $\sigma = 0.76$
N/groups: 82/27	*NCPRatio*	−0.10	0.14	−0.67	0.509	L.ratio = 3.47, $P = 0.062$, $\delta = -0.33$

[1] *SCPRatio* was ratio of siblings to producers, and *NCRatio* was ratio of nonsiblings to producers. Model of *WeightZ* was heteroscedastic LME.

potent effect of illness on our results therefore remains a distinct possibility, and is a limitation of our study.

Although we did not predict sex differences, the anthropometry of female, but not male, dependents appeared to depend positively on father status, with one significant effect and several nearly significant effects. One speculative interpretation of these results is that in this society, most boys received high levels of investment, whereas most girls received high levels of investment only if their fathers were of high status and could therefore afford to invest equally in both sexes.[5] Another similar interpretation is that girls were expected to work more than boys despite receiving similar amounts of food, but the amount of work was less in high-status households. The unexpected interaction of sex with father's status requires further investigation.

Finally, the negative impact of large families may fall not only on children but also on parents, limiting their ability to invest in current or future offspring. We found a consistent, albeit not significant, trend for higher *CPRatios* to negatively impact mothers' anthropometry, but only a slight and nonsignificant trend for higher *CPRatios* to negatively impact father's *Bodyfat* index. A possible negative impact of *CPRatio* on mothers' anthropometry would not be surprising, as maternal depletion was documented in many populations (e.g., Bongaarts and Delgado, 1979; Miller and Huss-Ashmore, 1989).

CONCLUSIONS

As found in several previous studies in a diverse range of communities, parents in this hunter-horticultural village paid a price for larger families in terms of the reduced nutrition and growth of their children, despite the presence of widespread food-sharing. Building and testing models of parental investment using a variety of fitness proxies can provide an important complement to previous empirical work on parental investment that emphasized direct measures of fitness. The increasing evidence for the negative impact of family size on child growth and nutrition in many populations suggests that these variables are promising candidates for inclusion in such models.

Future studies of quantity-quality tradeoffs in similar populations should attempt to quantify food-sharing in order to assess what, if any, buffering effect it has on quantity-quality tradeoffs; they should include more robust measures of illness and a wider range of potential "phenotypic" correlates; and they should also investigate the surprising interaction of female anthropometry with father's status (and to a lesser extent, with *CPRatio*).

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APPENDIX

In addition to investigating the impact of the overall consumer/producer ratio on child nutrition and growth, we also investigated a similar model in which the ratio of number of siblings (full and half) to producers (*SCPRatio*) and the ratio of nonsiblings to producers (*NCPRatio*) were entered as separate predictor variables in models of child nutrition and growth. In these models, we only included children who had at least one biological parent in the household. Nonsiblings equaled family size minus the number of siblings, and thus might include parents, grandparents, stepparents, step-siblings, or any other children living in the household. See Table 8 for descriptive statistics.

We computed OLRs and LMEs, including both response variables. Although some LMEs were not significantly better than the corresponding OLRs, for consistency we report the LME (Table 9).

SCPRatio had a significant, or marginally significant, negative impact on all child nutrition and growth variables except *BMIR* and *BMIZ*. *NCPRatio* was not significant in any model. Thus, nonsiblings did not appear to

[5]Trivers and Willard (1973) proposed an evolutionary theory of sex-biased parental investment. Testing this theory requires knowledge of the mating pool. We had little information on the potential mating pool of our dependents, so we could not test this theory.

have an impact on the anthropometry of children with at least one parent in the household. These results are similar to those reported for *CPRatio* in Table 3.

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