DOES RACE MATTER? CHILDREN'S HEIGHT IN BRAZIL AND SOUTH AFRICA*

SARAH BURGARD

I examine racial differences in child stunting in mid-1990s South Africa and Brazil, two multiracial societies with different histories of legal support for racial discrimination. Using data from nationally representative household samples linked to community-level measures, the analysis shows that racial inequality in the distribution of socioeconomic resources across households and communities explains much of the racial difference in stunting in both countries. Even after these factors are controlled, however, the results indicate that in South Africa, nonwhite children are still at greater risk of growth faltering than are white children. The nature of socioeconomic and racial differences in children's growth is examined, and major determinants are discussed. These findings suggest that although state-sanctioned racism may help to explain the greater racial inequality in stunting in South Africa than in Brazil, the eradication of a disadvantage for nonwhites will depend on changes in the same fundamental socioeconomic inequalities that characterize both nations.

Racial inequality has important implications for the health of individuals in multiracial societies. This analysis compares children's health status in mid-1990s South Africa and Brazil, two multiracial societies with different histories of race-related legislation. In South Africa between 1948 and 1994, the official apartheid system organized individual opportunities almost entirely on the basis of race, whereas in Brazil, no race-related legal institutions emerged after slavery was abolished in the late nineteenth century. The South African state instituted a program of racial inequality in individual socioeconomic status (SES) opportunities and the development of community-level infrastructures, whereas Brazil did not pursue overt policies that promoted racial inequality. To the degree that apartheid institutions did, in fact, create racial inequality in the provision of a health-promoting infrastructure and individual opportunity, racial differences in children's health should be sharper in South Africa than in Brazil.

Children's height is an indicator of health status and living standards that reflects cumulative exposure to low nutrition and infections, such as diarrhea (Martorell and Habicht 1986; Pelletier 1998; Waterlow et al. 1977). Height measurements can be compared to an age- and sex-standardized international reference value to establish whether a child has experienced substandard growth (Martorell and Habicht 1986). These deviations can be compared across groups to assess inequality in levels of living by race. Although genetically based racial differences in the heights of adults do exist, worldwide evidence indicates that well-nourished children of diverse ethnic backgrounds are comparable along age- and sex-standardized growth curves until age 10 (Habicht et al. 1974). Children who are severely height-for-age deficient are denoted as stunted, and these children are the focus of the present analysis. Stunting is an important sentinel indicator of ill health because growth faltering that is due to undernutrition and infection in infancy and early childhood can increase a child's risk of mortality in the short term (Pelletier 1991; Pelletier, Frongillo, and Habicht 1993) and have adverse effects on a child's cognitive

*Sarah Burgard, Departments of Sociology and Epidemiology, University of California at Los Angeles, 264 Haines Hall, 375 Portola Plaza, Los Angeles, CA 90095-1551; E-mail: sburgard@ucla.edu. This research was made possible by a Mellon Fellowship in Latin American Sociology. I am grateful to Donald Treiman, Narayan Sastry, Edward Telles, and three anonymous reviewers for their valuable input on this article.

and behavioral development in the long term. Severe deficits in nutrition have been linked to reduced educational achievement in later childhood (Mendez and Adair 1999) and could diminish other outcomes. Racial inequality in stunting among children may indicate or exacerbate existing patterns of disadvantage for nonwhites in South Africa and Brazil.

This analysis differs from and complements earlier work on racial inequality and children's health in several ways. First, previous studies of racial effects in both countries have focused on outcomes for adults, including educational, occupational, and income attainment (Hasenbalg 1985; Silva 1985; Telles 1992; Treiman, McKeever, and Fodor 1996), whereas this study's focus is on health, another important sentinel measure of inequality. Second, previous studies of children's height in Brazil have revealed important determinants of inequality but have not focused on racial effects. Finally, analyses of children's health in South Africa and Brazil that have considered race have been based on samples from geographically circumscribed areas. By contrast, this study was based on nationally representative samples from both countries to compare differences in children's health by race.

RACE AND INEQUALITY IN SOUTH AFRICA AND BRAZIL

For many years, South Africa and Brazil have been characterized by the world's highest levels of income inequality (Lam 1999), and they share colonial histories that created multiracial societies. When one studies the effects of racial inequality on children's health, it is challenging to find a better comparison because the United States is also a multiracial society but is much more developed than these two. South Africa has more in common with Brazil than with many of its African neighbors because few African societies have a significant white minority population and South Africa has had an independent government for much longer than other parts of Africa. Nonetheless, important differences in legal and social institutions and racial composition complicate the comparison of children's growth across the countries.

In South Africa, a history of segregation and separate development explicitly reinforced inequality on the basis of race. In 1948, the National Party came to power and implemented the principles of the apartheid system in a country that had long been immersed in colonial segregation based on race (Christopher 1994). Four official racial groups were defined that still persist today: whites, Asians, coloreds, and blacks. Afrikaaners, the descendants of Dutch settlers, make up more than two-thirds of the white population, with the remainder descended from English settlers. Generally less well educated and more rural than English settlers, Afrikaaners made efforts to increase their status by developing the apartheid system, at the expense of the nonwhite populations (Treiman et al. 1996). Asians in South Africa are mainly descended from agricultural indentured laborers who were brought from the Indian subcontinent late in the nineteenth century; the colored population is made up of the progeny of mixed couples, mostly Afrikaaners and the indigenous population of the Cape Town region. Blacks, also referred to as Africans, are descendants of the indigenous Bantu-speaking peoples. Most blacks live in rural areas, both because they are traditionally cattle-herding peoples and because the apartheid policies that created the territorial "homelands" relegated them to completely nonurban areas, separated from industrially developed or agriculturally rich territories.

Under apartheid, white privilege was maintained by a series of laws that established urban racial residential segregation. South Africa was divided geographically into four provinces within the white Republic of South Africa (RSA) that were inhabited by whites, Asians, coloreds, and properly documented blacks and 10 rural homelands for blacks who were divided on the basis of ethnic groupings. Of these homelands, four (Transkei, Boputhatswana, Venda, and Ciskei) eventually were designated "independent states" with sovereign rights, while other homelands remained part of the RSA but held self-governing rights. Black families were forcibly relocated to the homelands from the 1950s through the early 1980s (Kaufman 1998), although many settled on the fringes of "white areas" in the hopes of obtaining employment and services. The implementation of apartheid meant separate and unequal development by race in educational systems, hiring practices in the labor force, and the provision of health services and other infrastructures. The homeland areas that constituted 13% of the total land of South Africa were expected to support almost three-quarters of the country's population, but were almost totally reliant on the RSA government for financial survival. Under these conditions, a system of migrant labor became entrenched as black men with few employment opportunities took contract work outside the homelands and sent their wages to their families during long periods of absence. New types of households developed to accommodate migrant labor, including nuclear families, widow households, wives of migrants households, grandmother households, and never-married households (Siqwala-Ndulo 1993).[1] Conditions in the later apartheid period changed gradually as restrictions on geographic mobility weakened, but there were still many black households under great financial strain, a substantial proportion of which were headed by women.

Health profiles vary dramatically across the provinces and homelands of the RSA. The most urbanized areas (former "white areas") have mortality profiles like those of Brazil or Mexico, whereas rural areas (homelands) have mortality profiles like those of neighboring countries in southern Africa, with many infectious diseases and diseases associated with poverty (National Economic Research Associates 1996). Differences in cause-of-death structures for infants are also informative; although the decline in white mortality in the 1980s was the result of decreasing early neonatal mortality (because of such factors as low birth weight), the decline among nonwhite children was due to reductions in fatal gastroenteritis, pneumonia, and malnutrition, all of which reflect shortages of basic health care and adequate resources (Andersson and Marks 1988). Comparisons of governmental health expenditures in the same period showed dramatic regional differences: the per capita expenditure in the developed province of Natal was almost four times that for the adjoining black homeland of KwaZulu (Jinabhai, Coovadia, and Abdool-Karim 1986).

There has been only limited empirical study of children's growth, but it suggests differences by race. In a two-year longitudinal study of height and weight among Johannesburg children aged 3.5 to 18.5 years, white children had more rapid increases in growth and attained greater heights and weights than did black children (Chaning-Pearce and Solomon 1986). Racial inequality that was institutionalized in the apartheid system, racially distinctive family patterns, and separate development policies across provinces and homeland areas suggest that nonwhite children are at a greater risk of stunting than are white children in South Africa.

Brazil's regional and SES inequalities, as well as its racial makeup, are a product of colonialism and slavery. During the early colonial period, the northeastern region drew a large number of slaves to work the dominant sugar coast, whereas the southern states eventually became the more wealthy centers of trade and commerce. By 1850, after centuries of slavery and miscegenation, the majority of Brazil's population was nonwhite, made up of mixed-race people and black Africans who were brought as slaves.[2] The color scheme in Brazil is not easily characterized by discrete categories, but instead resembles

1. Siqwala-Ndulo (1993) noted that there may be important differences among black rural communities in different regions of South Africa in their forms of adaptation to migrant labor systems and apartheid policies. However, the common experiences of blacks during the apartheid era mean that blacks probably share features of family life unlike those of whites or others.

2. Throughout the colonial period, there were far more white men than white women in Brazil, and there was considerable miscegenation with indigenous women and African-origin slaves. Intermarriage is still common today, although marriages between blacks and browns are more common than between blacks and whites, for example (Degler [1971]1986; Silva 1985).

a continuum from white to black with many shades of brown in between. Despite the global racism of the period, Brazilian elite of the early 19th century chose to idealize the fluid color continuum and promoted an ideology of "racial democracy." Although largely discounted today, the common wisdom was that existing class inequalities exercised a more profound influence on everyday social relations than did skin color (Reichmann 1999). In stark contrast to South Africa, there was virtually no legislation dealing with race after the abolition of slavery in 1888.

The geographic patterning of slavery and European immigration to Brazil led to marked variation in regional distributions of color groups. The uneven distribution of infrastructure and health services across the major geographic regions of Brazil is not unrelated to residential patterns by race. Whites are advantageously concentrated in southern metropolitan areas, whereas nonwhites are more likely to live in poorer northeastern cities (Silva 1999). Beyond the regional concentrations, there is moderate racial residential segregation in urban areas that cannot be explained by SES alone (Telles 1992). Although every Brazilian citizen has been entitled to medical treatment in public or contracted private facilities since 1987, the northern regions and rural areas throughout the country are drastically underserved (Buss and Gadelha 1996). The effects of the inequitable distribution of health care services can be seen in infant mortality rates, which are twice as high in the Northeast as in the Southeast (74 versus 37 per 1,000) (BEMFAM/DHS 1996). A comparison of Brazilian anthropometric surveys from the mid-1970s and late 1980s shows that large gains in average heights occurred during a period of exceptional growth in both household income and the provision of services and infrastructure in the 1970s (Monteiro, Benecio, and Gouveia 1994), but disparities among geographic regions and income groups persisted (Monteiro et al. 1992).

Because of the traditional research focus on regional disparities and class inequality, there have been relatively few studies of racial inequality in Brazil, although research interest is growing. Appropriate data are limited because censuses and other surveys often failed to include a question about race/color. In one of the few studies of race and children's health, child and infant mortality were estimated indirectly using the 1950 and 1980 demographic censuses of Brazil, which included information on mother's color (Wood and Carvalho 1988). Controlling for a set of standard socioeconomic indicators, including region, education, and income, reduces but does not fully explain differences in child mortality between whites and nonwhites (Wood and Carvalho 1988; Wood and Lovell 1992). A related analysis showed that the gap in life expectancy between nonwhites and whites did not narrow between 1950 and 1980, even within categories of household income and despite the dramatic development in Brazil over the period (Lovell and Wood 1998). In Brazil, historical patterns of prejudice, high levels of class inequality, and uneven development across regions and in rural areas may increase the risk of child stunting for nonwhites, despite the absence of officially sanctioned racial discrimination.

Comparing Racial Groups in South Africa and Brazil

The use of racial categories in research on health outcomes is not without controversy. One contemporary disagreement concerns the use of racial groups, which are socially, not scientifically, demarcated, to explain biological distinctiveness (see the exchange among Frank 2001; van den Oord and Rowe 2001; and Zuberi 2001). The research presented here emphasizes that racial categorizations, as well as racial inequality in children's health outcomes, are socially created. For comparative purposes, an ordinal scale of relative socioeconomic disadvantage is used, distinguishing whites, browns, and blacks. For South Africa, Asian and colored respondents are combined into a single brown category, a convention that sometimes occurs in the literature on South Africa, despite the fact that these categories are not strictly comparable. For Brazil, whites and a small number of Asians are combined to form a single white group.[3] This analysis compares the way that race affects children's growth in two multiracial settings; the implicit argument is that race and child stunting are associated differently in South Africa and Brazil because the two countries' legacies of discrimination vary. Although in both countries, patterns of racial disparity in household and community socioeconomic resources are responsible for some portion of the racial difference in growth faltering, the ability to make detailed cross-national comparisons of the effects of race or the sources of racial differences is still limited.

Conceptual Framework

The immediate determinants of child stunting are not socioeconomic characteristics of the household or community, but biological and behavioral factors that reflect exposure to malnutrition and infection; for example, the quality of feeding is one determinant of nutritional status. Another immediate determinant is protection from environmental contamination that can cause diarrhea, whether this contamination reflects poor parental cleanliness practices in the home or the lack of household sanitation and a clean water supply in the community. Mosley and Chen (1984) developed a framework for the proximate determinants of growth faltering and mortality in children, which include maternal factors, environmental contamination, nutritional deficiency, injury, and personal illness control (i.e., healthy individuals taking preventive measures to avoid disease). Socioeconomic and demographic characteristics, such as race or mother's education, can exert their effects only through their influence on these proximate determinants of stunting. Nonetheless, it is often socioeconomic and demographic characteristics that are included in analyses of children's health, both because the measurement of the immediate determinants themselves is difficult and because the proximate determinants are potentially endogenous to the child health outcome of interest. In other words, the same decisions that account for a mother's tactics for controlling illness in her child, for example, will account for her child's overall height for age through other channels, such as the provision of adequate nutrition. The correct estimation of the effect of the proximate determinants in health-production functions requires controls for simultaneity bias or instrumental variables techniques, but good instruments are unavailable in the cross-sectional data used here. Given the absence of a satisfactory solution to potential endogeneity, this analysis presents models both with and without measures of some of the proximate determinants of stunting.

The microeconomic model of the family (Becker 1981) is used to represent the way that families choose health-producing inputs. For example, parents can spend more time at work earning income to buy food and medical care, but this extra work time limits their direct control over food preparation or sanitary child care practices. Choices about parents' allocation of resources may be influenced by characteristics of the family, such as the mother's education, or characteristics of the community, such as the availability of sewage facilities. For example, well-educated women may be more likely to breast-feed their children for longer periods because they have greater contact with, and understanding of, medical advice to do so.

Given the set of health-producing inputs represented by the proximate determinants and a model of family decision making about these inputs, it is possible to generate reduced-form relationships between stunting and background characteristics of the child, the household, and the community, such that $z_i = h(C_i, C_h, C_c, \varepsilon_i)$, where z_i is the height-for-age outcome for child i, C_i are the individual characteristics of the child, C_h are household

3. In Brazil, the racial identity assigned by the interviewer may be influenced by the SES or appearance of the respondent and may not match the self-assessment of the individual. Because it is others' perceptions of an individual's race that often influence the opportunities open to that individual, interviewer-assessed racial identity of the mother is the most appropriate indicator of her and her child's race-related experiences (Telles and Lim 1998). Both the interviewer's assessment and self-identification of race are available for most women in the sample for Brazil used here, but the substantive results of the analysis do not depend on which is used.

characteristics, C_c are characteristics of the community, and ε_i is an individual-specific random disturbance associated with the height outcome of child i. Unmeasured inputs represented by ε_i could include parents' intrinsic ability to raise healthy children or unobservable environmental factors. In the reduced-form models used here, children's characteristics include sex and age, household characteristics include parental education and household wealth, and community characteristics include region and type of place of residence. As I noted earlier, proximate determinants are not normally included in reduced-form models of child health outcomes, but they are included in health-production functions or are explored as separate outcome variables. However, models that include some of the proximate determinants of stunting like breast-feeding or exposure to diarrhea can be used to examine the mechanisms through which background factors like race or mother's education exert their effects (Bollen, Glanville, and Stecklov 2001).

In addition, a comparison of reduced-form models with those that include measures of proximate determinants shows the sensitivity of the findings to the inclusion of these potentially endogenous variables. The proximate determinants of interest are maternal factors, here represented by mother's age; environmental contamination, represented the presence of a toilet and piped water in the home; nutritional deficiency, represented by the child's age at weaning; and personal illness control, represented by measures of health facilities in the community (Brazil only). Both background factors, such as parent's education, and proximate determinants like environmental contamination, here represented by the provision of piped water in communities, are open to policy intervention and should be explored as potential contributors to racial inequality in growth faltering.

DATA AND METHODS

The data come from the South Africa Integrated Household Survey (SAIHS), a nationally representative clustered sample that covered approximately 9,000 households in 1993–1994 (South African Labour Development Research Unit 1994).[4] Once weighted, the data represent about 95% of the noninstitutionalized population of South Africa.[5] After children are omitted for whom information is missing, the South African sample contains 3,156 observations.[6]

Household-level data for Brazil come from the 1996 Demographic and Health Survey (DHS-3), a nationally representative sample of women aged 15 to 49 (BEMFAM/DHS 1996). The DHS-3 is a multistage, clustered sample of 13,000 households, with anthropometry measurements available for 4,014 children after those with missing information are omitted. A brief examination of differences between the children with available height measures and the entire sample shows that those with valid height measurements had a slightly higher SES background than did the overall sample for both South Africa and Brazil (information is available from the author on request).

To capture differential infrastructure development across geographic areas, magisterial district-level information from the 1991 and 1996 censuses of South Africa was linked to the SAIHS households.[7] The 1991 South African census did not enumerate the

4. Under apartheid, the nominally independent black homelands were often excluded from census enumeration and other surveys, and vital-events data were poor for nonwhites (Botha and Bradshaw 1985). A recent DHS survey (BEMFAM/IRD 1998) did not collect anthropomtric measures, and those data were not available for public use at the time of writing.

5. Difficulties with data collection (including violence in some areas), the greater nonresponse of whites, and other sampling problems necessitated the use of weights based on the 1991 South African Census.

6. A significant fraction (about 10%) of children in the SAIHS sample were living with neither parent, although they would have been excluded from the analysis because anthropometric measurements were missing for many of these children. Child fosterage is an important issue in South Africa, but would unnecessarily complicate the present comparative analysis; thus these children were excluded.

7. The SAIHS included a community-level survey, but a significant amount of missing data precluded its use.

former black homelands, so information for these areas was obtained from the later census. Census data were collected in different years than were the SAIHS data, but broad trends in the inequitable distribution of infrastructure are likely to have remained stable over the period. No indicators of health services are available in the South African census, so magisterial district-level indicators of education and employment were used as proxy measures of basic infrastructure development. For Brazil, municipality-level information was obtained from the DATASUS (1996) data collection system of the Sistema Unico de Saude (the Brazilian Department of Health). Two general indicators of health services were used in the analysis, as well as an indicator of the basic educational infrastructure.[8] Admittedly, magisterial districts and municipalities are too large to represent "communities" in the sense of neighborhoods, but these indicators provide some information about the geographic dispersion of resources.

STATISTICAL METHODOLOGY

Modeling Approach

The determinants of child stunting were explored using the generalized estimating equations (GEE) approach of Liang and Zeger (1986). GEE models have several important advantages over standard maximum-likelihood methods: first, it is possible to take account of the correlation of unobservable factors across children in the same family. In the multistage, clustered design of the samples used here, children were grouped within mothers, and mothers were grouped within geographic clusters. The growth patterns of children born to the same mother are likely to be more homogenous than those of children who are chosen at random, and GEE models specify the marginal distribution for each mother. Second, GEE models do not require the complete specification of the joint distribution of all correlated observations, which can be difficult to implement when there are few children per mother, as there are in the data (Zenger 1993). Finally, GEE models can correct both parameter estimates and standard errors for clustering at the level of mothers by applying the Huber-White, or "sandwich," estimator as implemented in Stata (StataCorp 1999).

The baseline model estimated here contains only indicator variables for mother's race, to establish the magnitude of the unadjusted effect of race. The next model incorporates the exogenous background characteristics of the child, household, and community. Parameter estimates obtained from these reduced-form models can be interpreted as the full effects of exogenous covariates, which include the effects of the proximate determinants (DaVanzo and Gertler 1991). Thus, the estimated impact of mother's education includes the effects of her schooling that work through the quality of nutrition she provides for the child and her control of environmental contamination. In the final model, potentially endogenous predictor variables are added; here, the interpretation of the effects of background factors (such as mother's race) must be interpreted carefully because the effects represent the net effect after controlling for a limited set of the mechanisms through which race works. If the size of the racial effect is significantly diminished by the addition of these indicators of particular proximate determinants, we will have a better idea of the way in which racial inequality in growth faltering is generated.

Descriptive Analysis of Children's Height for Age and Stunting

To measure children's height and stunting, measures of height, age, and sex were used to calculate z scores with ANTHRO software, Version 1.02, provided by the Division of

8. Other indicators of community infrastructure were tested, including the provision of piped water and toilets and the racial composition of the municipality, but they were highly correlated with community literacy. Because literacy had the largest independent effect on child stunting, the remaining variables were dropped from the analysis.

Table 1. Means and Standard Deviations of Height-for-Age z Scores and Prevalence of Stunting, by Country and Key Variables: South Africa (N = 3,156) and Brazil (N = 4,014)

	South Africa			Brazil		
	Mean Height-for-Age z Score	*SD* of z Score	Percentage Stunted	Mean Height-for-Age z Score	*SD* of z Score	Percentage Stunted
Mother's Race						
White	0.00	1.28	5.7	−0.40	1.22	9.7
Brown	−0.85	1.26	17.6	−0.71	1.30	14.6
Black	−1.34	1.54	31.9	−0.71	1.60	17.6
Mother's Education						
Secondary +	−0.95	1.51	22.1	−0.31	1.21	7.6
Primary	−1.33	1.48	32.6	−0.90	1.32	18.3
None	−1.44	1.78	35.4	−1.13	1.28	23.4
Father's Education						
Secondary +	−0.67	1.38	15.4	−0.33	1.24	7.8
Primary	−1.39	1.57	34.5	−0.72	1.28	14.4
None	−1.22	1.82	31.9	−1.26	1.31	26.9
No father/Don't know	−1.33	1.50	31.2	−0.66	1.27	15.6
Place of Residence						
Urban	−0.86	1.52	21.6	−0.46	1.25	9.8
Rural	−1.39	1.52	32.9	−1.02	1.36	22.6
Overall	−1.18	1.54	28.3	−0.59	1.30	12.9

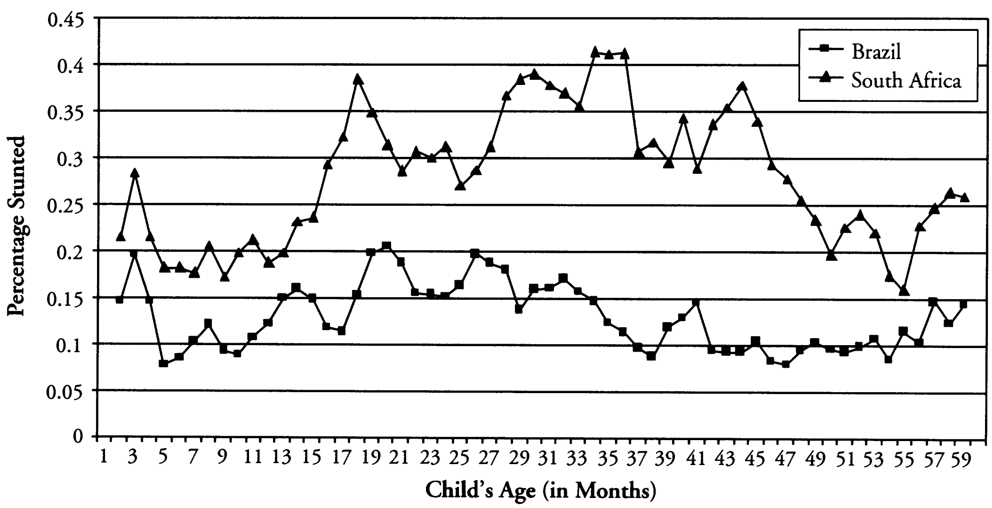
Source: From the author's calculations based on data from the SAIHS (South Africa) and DHS-3 (Brazil).

Nutrition of the Centers for Disease Control and Prevention and the Nutrition Unit of the World Health Organization (Sullivan and Gorstein 1999).[9] The z score represents the deviation from the expected stature for a child of that age and sex according to an international reference population, according to the formula $z_i = (Y_i s,a - H^{s,a}) / \sigma^{s,a}$, where z_i is the z score for child i, $Y_i s,a$ is the measured height in centimeters for child i of sex s and age a, $H^{s,a}$ is the median height in centimeters for children of sex s and age a in the international reference population, and $\sigma^{s,a}$ is the standard deviation in height in centimeters for children of sex s and age a in the reference population. A z score of zero means that a child has a height for age equal to the international reference median, which is normal.

According to the usual practice in nutritional and epidemiological research, a child is defined as stunted when his or her height for age is two or more standard deviations below the international reference median. Table 1 shows the mean height-for-age z scores, standard deviations, and the proportion stunted for each sample by categories of mother's race, mother's and father's education, and place of residence. Overall, South African children are about 1.2 standard deviations below the international reference population height

9. These cross-sectional samples include height measures only for surviving children. Survivor bias in the DHS has been identified as a problem when mortality differences between the birth cohorts are large (Boerma 1996), but this was not the case for the samples used here.

Figure 1. Proportion of Children Stunted, by Child's Age (in Months) and Country, Three-Month Moving Averages: South Africa (SAIHS Data, N = 3,156) and Brazil (DHS-3 Data, N = 4,014)[a]



[a]Data are weighted.

for age, and about 28% are stunted. In the Brazilian sample, children average about 0.6 standard deviation below the reference population, and about 13% are stunted. The height-for-age scores for children of white mothers (henceforth, white children) are closest to the international reference standard, whereas brown children are relatively shorter and black children are as short as brown children (in Brazil) or still shorter (in South Africa). Stunting is similarly much more common for nonwhite children.

The racial difference between whites and nonwhites is much greater in South Africa than in Brazil, as is the variation by level of mother's education. Children of parents with secondary schooling or more have a large height advantage in both countries, as do those who live in urban areas. Although Brazilian children are taller than South African children, on average, the pattern of stunting by age is similar in both countries. Figure 1 shows the proportion of stunted children by age in months and country, displayed as a three-month moving average to smooth month-by-month fluctuations owing to the sample size. In both Brazil and South Africa, the prevalence of stunting is low just after birth, rising to about 40% in South Africa between ages 18 and 36 months, and falling thereafter. The rise is more gradual for Brazil, with about 20% of children stunted at about 20 months of age, and South African children are more likely to be stunted than Brazilian children at all ages. After age 2 (in Brazil) or age 3 (in South Africa), the proportion stunted declines in both countries, but still 20%–25% of South African children are stunted at age 4, compared with less than 15% of Brazilian children.

Explanatory Variables

Summary statistics for exogenous background factors and potentially endogenous predictors of stunting are presented in Table 2 for South Africa and Table 3 for Brazil. *Child's*

Table 2. Summary Statistics for Variables in the South African Sample (N = 3,156)

	Full Sample	Whites	Browns	Blacks
Exogenous Background Factors				
Child's age (in months)	29.8 (16.5)	32.1 (17.6)	31.9 (15.8)	29.3 (16.4)
Child's sex				
Female	49.6	52.1	53.0	49.0
Male	50.4	47.9	47.0	51.0
Mother's race				
White	8.1	—	—	—
Brown	10.4	—	—	—
Black	81.5	—	—	—
Mother's education				
Secondary +	43.8	78.5	55.7	38.8
Primary	44.7	15.1	42.2	47.9
None	11.5	6.4	2.1	13.2
Father's education				
Secondary +	23.7	86.7	45.5	14.7
Primary	24.4	4.9	20.4	26.8
None	8.2	3.4	1.3	9.5
No father/Don't know	43.7	4.9	32.8	49.0
Household wealth (proxy measure)[a]	1.23 (0.782)	2.45 (0.297)	1.97 (0.583)	1.02 (0.660)
Place of residence				
Urban	40.5	91.2	94.6	28.6
Rural	59.5	8.8	5.4	71.4
Province or homeland				
Cape	11.9	21.9	67.9	3.8
Natal	4.2	2.7	22.1	2.1
Transvaal	20.8	70.1	8.3	17.5
Orange Free State	4.7	5.0	1.2	5.1
KwaZulu	16.1	0.0	0.0	19.8
KaNgwane	1.5	0.0	0.0	1.9
QwaQwa	0.6	0.0	0.0	0.8
Gazankulu	3.1	0.0	0.0	3.7
Lebowa	13.0	0.0	0.0	15.6
KwaNdebele	1.7	0.0	0.0	2.1
Transkei	13.5	0.0	0.3	16.5
Bophuthatswana	5.2	0.4	0.0	6.3
Venda	2.3	0.0	0.3	2.8
Ciskei	1.5	0.0	0.0	1.8
Potentially Endogenous Factors				
Age at weaning				
0 months	27.7	10.6	18.2	30.6
1–5 months	15.0	40.4	32.6	10.2

(continued)

(Table 2, continued)

	Full Sample	Whites	Browns	Blacks
Age at weaning (cont.)				
6–11 months	9.2	19.1	15.9	7.3
12–23 months	24.2	5.5	8.8	28.0
24–59 months	14.9	3.5	9.3	16.8
Unweaned	9.1	21.0	15.2	7.1
Mother's age				
< 20 years	5.1	0.0	2.2	6.0
20–29 years	50.6	46.6	47.3	51.4
30+ years	44.3	53.4	50.5	42.6
Toilet facilities in the home				
Modern toilet	33.7	99.0	89.5	20.1
No modern toilet	66.3	1.0	10.5	79.9
Piped water in the home				
Piped water	43.1	100.0	97.5	30.5
No piped water	56.9	0.0	2.5	69.5
MD[b] female (25+) educational attainment	6.07	8.85	7.99	5.54
	(2.24)	(1.31)	(1.19)	(2.08)
MD[b] level of male (25+) employment (%)	56.9	81.8	75.3	52.0
N	3,156	214	325	2,617

Source: From the author's calculations of data in the SAIHS, 1991 and 1996 census (South Africa).

Notes: For categorical variables, percentages are reported. For continuous variables, means and standard deviations (in parentheses) are reported.

[a]Range for household wealth proxy measure: 0 to 2.56.

[b]MD refers to the magisterial district.

age reflects a changing level of risk for nutritional deficit and infectious contact; the typical pattern in developing countries is for height-for-age scores to decrease until age 24 months and then to level off (Martorell and Habicht 1986). In the statistical analysis, child's age is entered as a piecewise linear spline function, so that the effect of an additional month of age can vary across three age groups (0–5, 6–23, and 24–59 months). *Child's sex* is included because weight for age among boys was shown to be lower than that among girls in six African countries (Madise, Matthews, and Margetts 1999), indicating differential care practices or greater biological frailty. There may be similar differences in frailty that are manifest in height for age. Overall, both samples have a similar mean age of about 30 months and are fairly evenly split by sex.

Background household characteristics include mother's race, education, and height (for Brazil only), father's education, and household wealth. *Mother's race* is captured with two dichotomous indicator variables for brown and black mothers; whites are the reference category.[10] One of the most obvious differences between these country samples is the dramatic variation in racial distribution. Blacks form a large majority of the sample for

10. Using father's or child's race in addition to mother's race would have been preferable for the Brazil model because miscegenation is common, but this information was unavailable. Father's and child's race were available for the South African sample, but because there is little intermarriage between the racial groups, and for comparative purposes, these variables were not used.

Table 3. Summary Statistics for Variables in the Brazilian Sample (N = 4,014)

	Full Sample	Whites	Browns	Blacks
Exogenous Background Factors				
Child's age (in months)	29.3 (17.3)	29.6 (17.4)	29.2 (17.3)	28.8 (17.1)
Child's sex				
Female	49.5	49.6	49.3	51.6
Male	50.5	50.4	50.7	48.4
Mother's race				
White	38.1	—	—	—
Brown	57.0	—	—	—
Black	5.0	—	—	—
Mother's education				
Secondary +	54.3	64.9	48.1	45.4
Primary	39.4	32.1	43.5	48.3
None	6.3	3.1	8.5	6.2
Mother's height (in cm.)	156.1 (6.38)	157.0 (6.35)	155.4 (6.30)	157.3 (6.58)
Mother's height missing	2.0	2.1	1.7	3.5
Father's education				
Secondary +	45.3	56.5	39.3	28.0
Primary	33.6	28.3	36.6	39.7
None	10.6	6.4	13.0	15.3
No father/Don't know	10.5	8.9	11.1	17.0
Household wealth (proxy measure)[a]	2.33 (0.929)	2.68 (0.932)	2.14 (0.864)	1.96 (0.827)
Place of residence				
Urban	76.1	79.9	73.7	74.3
Rural	24.0	20.1	26.3	25.7
Region of residence				
Rio de Janeiro	8.2	8.2	7.4	16.1
São Paulo	19.0	26.1	14.8	12.7
South	13.3	20.2	9.2	7.9
Center-East	12.6	12.9	12.0	16.6
Center-West	6.8	7.7	6.4	5.0
Northeast	34.6	22.5	42.5	36.7
North	5.6	2.4	7.7	5.0
Potentially Endogenous Factors				
Age at weaning				
0 months	11.0	12.0	10.6	8.3
1–5 months	34.7	37.4	33.2	31.3
6–11 months	15.6	16.8	15.0	12.5
12–23 months	11.6	9.5	12.6	17.0

(continued)

(Table 3, continued)

	Full Sample	Whites	Browns	Blacks
Age at weaning (cont.)				
24–59 months	5.8	5.8	5.8	6.0
Unweaned	21.3	18.4	22.9	25.0
Mother's age				
< 20 years	8.6	7.1	9.5	9.2
20–29 years	53.3	53.6	53.4	52.0
30+ years	38.1	39.3	37.1	38.8
Toilet facilities in the home				
Modern toilet	66.5	75.8	60.6	61.9
No modern toilet	33.5	24.2	39.4	38.1
Piped water in the home				
Piped water	71.6	75.4	68.7	75.2
No piped water	28.4	24.6	31.3	24.8
Community literacy (%)	64.4	68.6	61.5	65.1
Private hospital beds per 1,000 residents	2.09	2.25	1.99	1.94
	(1.94)	(1.93)	(1.96)	(1.80)
Any hospital beds in the municipality (%)	93.0	94.4	92.2	90.7
N	4,014	1,371	2,445	198

Source: From the author's calculations of data in the DHS-3 and DATASUS.

Notes: For categorical variables, percentages are reported. For continuous variables, means and standard deviations (in parentheses) are reported.

[a]Range for household wealth proxy measure: 0 to 4.82.

South Africa (82%), while the majority in Brazil are brown (57%) or white (38%). *Mother's education* denotes those with primary schooling or no schooling; mothers with secondary schooling or more are the reference category. Many studies have shown a large and significant positive effect of mothers' education on children's height, even after controls for other household and maternal characteristics are included in the models (Barrera 1990; Horton 1986; Strauss 1990; Thomas, Strauss, and Henriques 1990, 1991). Mothers' schooling may affect children's height because well-educated women have better access to information, and they may be more productive at translating resources and information into children's health (Barrera 1990; Rosenzweig and Schultz 1982). In addition, highly educated women may be more likely to hold jobs that have high-quality health care benefits, which could improve the health of their children. There are dramatic differences in the distribution of schooling across racial groups in South Africa; among white children, 79% have mothers with at least a secondary school education, compared with 56% of brown children and only 39% of black children. In Brazil, these differences are not as great; the figures are 65% for white children, 48% for brown children, and 45% for black children.

Mother's height, available only for Brazil, reflects the mother's genetic background and its interaction with nutrition and infection in childhood and adolescence.[11] Taller

11. This variable addresses the argument that observed effects of mother's education on children's health outcomes may be spurious if mother's education partially reflects unobserved background characteristics and human capital (Behrman and Wolf 1987).

mothers are also likely to have grown up in households with more resources and may have gained skills or expectations about child rearing that influence the growth of their own children. Alternatively, women who were well off when they were growing up are likely to marry men who have a relatively high earnings potential and SES themselves. An indicator variable denotes mothers who are missing height measurements (n = 65 out of 4,014) and for whom race-specific mean heights were imputed. Mother's height is similar across racial groups.

Father's education, often used in the literature on children's health to represent earning power or household SES, is measured in the same way as mother's education, with one additional category denoting children for whom information about their fathers' education was unknown or whose fathers were not part of the households.[12] The proportion of children for whom information on father's education is missing varies dramatically by race in South Africa, from 5% of whites to 33% of browns and 49% of blacks, indicating the large proportion of nonwhite mothers who are currently raising their children without fathers; these figures are not as large as in Brazil, where they are 9% for whites, 11% for browns, and 17% for blacks. In both countries, white children are the most likely to have fathers with secondary schooling or more, followed by brown children and black children.

A proxy indicator of *household economic resources,* based on an index of owned possessions, is used to control for the ability of households to purchase food and housing, prevent exposure to toxic or hazardous agents, and provide medical prenatal and childhood care (see Filmer and Pritchett 2001).[13] This indicator also controls for the fact that effects associated with parents' educational attainment could be indicating the higher earnings of better-educated people. Household economic resources are strongly associated with children's health, in general, and tend to be distributed unequally by race or ethnicity. In Guatemala, for example, ethnic differences in children's height are substantially reduced when social class and economic status are controlled in models of children's height (Glei and Goldman 2000; Pebley and Goldman 1995). White South African children live in households with an average score of 2.5, whereas brown and black children have average scores of 2.0 and 1.0, respectively. White children in Brazil also have the highest average score (2.7), followed by brown (2.1), and black (2.0) children.

Inadequate community-level health inputs may also increase the risk of stunting. In both countries, nonwhites not only have fewer household economic resources but may also live in racially segregated areas where it is difficult to obtain health-producing goods and services, such as education or health care (LaVeist 1996). Background characteristics of the community include the type of place and region of residence. *Type of place of residence* distinguishes urban from rural households (as designated in each survey) because urban households are likely to be better served by developed infrastructures and services. In South Africa, the apartheid system ensured great inequality in the level of urbanization by race: only 29% of black children, but 91% of white children and 95% of brown children, live in urban areas. By contrast, in Brazil, about three-quarters of the entire sample live in urban areas. The characteristics of the major geographic regions of each country may differ because of specific racially targeted policies or other reasons. In South Africa, the four provinces and 10 homelands created under the apartheid system

12. Father's educational and occupational statuses are not perfectly correlated, but there is a considerable problem of missing data for father's occupation, especially in the South African sample. In the Brazilian survey, information was collected from the child's mother about her current partner, who may have been the child's father or a more recent partner. In the South African survey, information about the father is specific to each child, but information is available only if the father is considered a current member of the household.

13. For South Africa, the index of household wealth was based on ownership of an automobile, television, radio, and refrigerator. For Brazil, the index included ownership of these items, as well as a vacuum cleaner, washing machine, and video cassette recorder; the number of rooms in the dwelling; the presence of electricity in the dwelling; materials used in constructing the dwelling; and the number of maids employed.

were the geographic boundaries officially recognized at the time of the SAIHS survey. The homelands are represented almost solely by black children, with white and brown children concentrated in the more developed provinces. Children are also distributed unequally by race across the seven regions of Brazil. Notably, the greatest proportion of brown children live in the Northeast region, the least developed populated part of the country; a substantial proportion of white children live in the more developed São Paulo or South regions. The largest fraction of black children live in the Northeast, but there are also large numbers in the Center-East and Rio de Janeiro regions.

Potentially endogenous variables representing the proximate determinants of a child's growth include the child's age at weaning, mother's age, household toilet and water facilities, and community-level indicators of infrastructure or health services specific to each country. *Age at weaning*, which captures the child's current breast-feeding status and duration of breast-feeding, measures nutritional intake. As a mother weans her child, the nutrition and antibodies provided by breast milk are lost, and the introduction to poor-quality weaning foods may increase the likelihood of malnutrition and more frequent infections. Although a longer duration of breast-feeding is generally considered protective, in six African countries (not including South Africa), breast-feeding beyond 12 months of age was associated with low weight-for-age scores, suggesting that mothers of weaker children may wait longer to wean them or that the quality of complementary feeding is lower for children who are still breast-feeding (Madise et al. 1999).

Mother's age, a maternal characteristic in the proximate-determinants framework, may reflect the mother's greater experience with child care and/or a choice to delay childbearing, both of which can be expected to improve a child's potential for adequate growth. In addition, women who are younger than age 20 or older than age 30 are at a greater risk of adverse pregnancy outcomes, and those who are younger than age 20 may have less access to economic resources than may older mothers. There were relatively few mothers under age 20 in either sample, although there were more in Brazil (9%); children in the South African sample were more likely than those in Brazil to have mothers who were older than 30 years old (44%).

Modern toilets and *piped water* are a marker of household economic resources, but also represent the potential control of environmental contamination. The use of modern sanitation can reduce the frequency of infections, especially diarrhea, that can increase a child's risk of stunting. While 99% of white children have flush toilets in South Africa, only 20% of black children have these facilities. Slightly more children have access to piped water in the home, although again, the proportion for white children (100%) far exceeds that for black children (31%). In Brazil, there are not dramatic racial differences in household sanitation; about two-thirds of children have access to flush toilets, and slightly less than three-quarters have access to piped water in the home.

Community characteristics, such as a developed infrastructure or the availability of health services, may be endogenous if they are purposively placed in areas with particularly poor health or where there is higher demand and more lobbying power.[14] In South Africa, overt apartheid measures ensured the purposive withholding of high-quality services from homeland and rural areas, whereas in Brazil, there is some indication that maternal and child health services and basic sanitation activities have been targeted at municipalities with the highest levels of poverty (Pan American Health Organization 2001). South African indicators of community infrastructure are the *magisterial district-level*

14. Another way that infrastructure could be endogenous to children's health outcomes is if parents moved to communities with better educational opportunities or health services with the intent of obtaining better health-producing inputs for their children. Parents with more economic resources, however, are likely to be better able to make voluntary moves for such a purpose, and these families are less likely to have children with deficient growth.

average years of schooling among women over age 25 and the *magisterial district-level of full-time employment among men over age 25*. These indicators are proxy measures for the provision of socioeconomic opportunities and are used to represent the level of state investment in a particular district. The average level of schooling among women in the district is almost 9 years for white children, compared with 5.5 years for black children. Similarly, about 82% of men are employed in communities where white children are raised, compared with only 52% employed in communities where black children are raised.

For Brazil, community indicators include the proportion of *literate* residents, the *number of private hospital beds per 1,000 residents*, and a dichotomous indicator of the *availability of any hospital beds* in the municipality. About 64% of the residents in the average municipality are literate, with little variation by race. The number of private beds per 1,000 residents indicates the extent of the availability of private services, which tend to be of a higher quality than public health services. If private facilities are the only services available, however, families with few resources may be relatively less likely to use health services. Whites live in communities with about 2.3 private beds per 1,000 residents; the figures for browns and blacks are 2.0 and 1.9, respectively. The criterion of *any* hospital beds marks those municipalities with the fewest available services; between 91% and 94% of all municipalities have beds available.

MULTIVARIATE RESULTS

A series of multivariate models was estimated to assess the relative contributions of factors that are linked to child stunting. The dependent variable was a dichotomous indicator coded 1 if the child was stunted and 0 otherwise. In all the models, the Huber-White correction for robust standard errors was used to account for the clustering of children of the same mother. As a formality, a pooled model with the data from both South Africa and Brazil was estimated to test whether the determinants of growth faltering worked similarly in both settings. The pooled model, which is not meaningful conceptually because of the dramatic differences in racial composition and the distribution of key background characteristics in the two countries, is only summarized here. It is not surprising that there were several statistically significant interactions with country, including father's education, household wealth, and mother's race. The results of the pooled model suggest that independent variables have significantly different effects across countries, so the remainder of the analysis was conducted separately for South Africa and Brazil.

Next, a series of country-specific models were considered, comparing the effects of race alone; race plus exogenous background factors; and, finally, race, background factors, and potentially endogenous determinants. The estimated odds ratios and their associated p values are presented in Table 4 for South Africa and Table 5 for Brazil, with Wald tests of significance for the addition of new sets of covariates at the bottom of each table. Possible interaction effects between race and key independent variables were also examined (results not shown here).[15] The effect of mother's education and household economic resources did not vary by race in South Africa or Brazil, suggesting that it is the deficit in resources that accounts for poorer outcomes for children in nonwhite households, not racial differences in the value of resources. Especially for South Africa, the dramatic differences in the distribution of many household characteristics precluded the use of interaction terms with race. Because none added significantly to the fit of the models, and for ease of interpretation, no interaction terms are presented here. For both South Africa and

15. A preferable strategy for each country would have been to compare a pooled model without indicators of race to three duplicate models estimated separately by racial group. Doing so would have allowed for a more formal test of the hypothesis that determinants of stunting work in the same way for each racial group. However, the small size of the black group in Brazil and the white and brown groups in South Africa precluded stratifying the analysis by race.

Table 4. Estimated Odds Ratios and p Values (in Parentheses) for the GEE Logistic Model of Stunting, South Africa (N = 3,156)

	Model 1	Model 2	Model 3
Exogenous Background Factors			
Mother's race [White]			
Brown	3.54*** (< .001)	2.01* (.047)	2.05* (.043)
Black	7.75*** (< .001)	3.22*** (< .001)	3.31*** (< .001)
Child's age[a]			
0–5 months		0.918 (.192)	0.918 (.193)
6–23 months		1.07*** (< .001)	1.08*** (< .001)
24–59 months		0.979*** (< .001)	0.982*** (< .001)
Child's sex [Female]			
Male		1.39*** (< .001)	1.41*** (< .001)
Mother's education [Secondary +]			
Primary		1.23* (.033)	1.23* (.037)
None		1.37* (.041)	1.39* (.039)
Father's education [Secondary +]			
Primary		1.50** (.007)	1.51** (.006)
None		1.17 (.421)	1.18 (.407)
No father/Don't know		1.43** (.009)	1.40* (.015)
Household wealth (proxy measure)		0.675*** (< .001)	0.711*** (< .001)
Place of residence [Urban]			
Rural		0.856 (.270)	0.766 (.135)
Province or homeland [Transvaal]			
Cape		1.34 (.182)	1.33 (.200)
Natal		0.756 (.352)	0.849 (.590)
Orange Free State		1.07 (.733)	1.05 (.813)
KwaZulu		1.10 (.567)	1.08 (.725)
KaNgwane		0.813 (.572)	0.891 (.770)

(continued)

(Table 4, continued)

	Model 1	Model 2	Model 3
Province or homeland [Transvaal] (cont.)			
QwaQwa		0.718 (.610)	0.730 (.634)
Gazankulu		0.606 (.083)	0.663 (.217)
Lebowa		1.48* (.030)	1.47 (.091)
KwaNdebele		0.893 (.749)	1.11 (.790)
Transkei		1.35 (.103)	1.09 (.775)
Bophuthatswana		1.35 (.201)	1.19 (.526)
Venda		1.88* (.043)	1.86 (.068)
Ciskei		0.481 (.090)	0.415 (.062)
Potentially Endogenous Factors			
Age at weaning [0 months]			
1–5 months			0.947 (.741)
6–11 months			0.723 (.098)
12–23 months			0.733* (.037)
24–59 months			0.714* (.047)
Unweaned			0.894 (.546)
Mother's age [20–29 years]			
< 20 years			1.27 (.221)
30+ years			0.983 (.856)
No modern toilet in the home			1.25 (.268)
No piped water in the home			1.15 (.413)
MD[b] female (25+) educational attainment			1.06 (.183)
MD[b] level of male (25+) employment (%)			0.995 (.330)
Wald chi-square	72.0*** (< .001)	228.1*** (< .001)	236.6*** (< .001)

(continued)

(Table 4, continued)

	Model 1	Model 2	Model 3
Wald Test Contrasts	Chi-square	*df*	*p* value
Model 2 – Model 1	98.7	20	< .001
Model 3 – Model 2	14.4	11	.213

Source: From the author's calculations of data from the SAIHS (South Africa).

Notes: Reference categories are shown in brackets. Model 1 contains only indicators of mother's race; Model 2 contains racial effects and exogenous background factors; Model 3 contains racial effects, exogenous background characteristics, and potentially endogenous predictors. The models are adjusted for clustering on mother's identification number and use robust standard errors.

[a]Child's age is included as a spline function with nodes at 6 and 24 months.

[b]MD refers to the magisterial district.

$^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$

Brazil, the addition of background characteristics in Model 2 explained a large proportion of the impact of race on stunting, and for Brazil, there was no longer any statistically significant difference between the racial groups. The inclusion of potentially endogenous predictor variables in Model 3 did not improve the fit of the models, so the results for Model 2 are discussed here.[16]

South Africa

Even after background household and community resources were controlled, brown and black children have odds of stunting that are still 2.0 to 3.2 times that of white children, a large and statistically significant effect. Nonetheless, racial effects are greatly reduced compared with those in the race-only baseline model, suggesting that the unequal distribution of socioeconomic and other background resources explains a large part of the variation in stunting. Individual child-level factors predict stunting; from 6 to 23 months of age, the risk of stunting is the greatest, and each additional month in this period increases the odds of stunting by 7%. Between the ages of 24 and 59 months, the odds decline by about 2% per month, reflecting the period of catch-up growth. Boys have odds of stunting about 1.4 times as great as those of girls.

Parents' characteristics also strongly influence children's stature. Children of mothers with primary schooling have odds of stunting 1.2 times as great as those of mothers with secondary schooling or more, and for children of uneducated mothers, the odds are 1.4 times as great. Father's education also has a protective effect; children whose fathers have primary schooling have odds of stunting about 1.5 times as great as those for children with fathers with secondary schooling or more, and children who are missing information on their fathers have odds of stunting about 1.4 times as great. Household wealth is protective, with each unit of increase in the wealth index reducing the odds of stunting by 32%. The type of place of residence does not have a statistically significant impact on growth faltering in South Africa, but children in the homelands of Lebowa (odds ratio = 1.5) or Venda (odds ratio = 1.9) have odds of stunting that are significantly larger than those for children in Transvaal (reference). Because differences in the major socioeconomic characteristics of households are already controlled in the model, the remaining

16. To test whether stunting is determined by different factors than height for age, regression models with the same predictors were estimated using height-for-age *z* scores as a continuous outcome measure. Overall, the substantive results were the same.

Table 5. Estimated Odds Ratios and *p* Values (in Parentheses) for the GEE Logistic Model of Stunting, Brazil (*N* = 4,014)

	Model 1	Model 2	Model 3
Exogenous Background Factors			
Mother's race [White]			
Brown	1.60*** (< .001)	0.900 (.423)	0.901 (.429)
Black	2.01** (.004)	1.37 (.180)	1.38 (.170)
Child's age[a]			
0–5 months		0.898 (.129)	0.909 (.178)
6–23 months		1.04** (.002)	1.04** (.003)
24–59 months		0.975*** (< .001)	0.976*** (< .001)
Child's sex [Female]			
Male		1.47*** (< .001)	1.49*** (< .001)
Mother's education [Secondary +]			
Primary		1.40* (.014)	1.41* (.013)
None		1.11 (.662)	1.11 (.634)
Mother's height (in cm.)		0.904*** (< .001)	0.904*** (< .001)
Mother's height missing		0.718 (.511)	0.711 (.496)
Father's education [Secondary +]			
Primary		0.920 (.606)	0.892 (.489)
None		1.18 (.424)	1.13 (.565)
No father/Don't know		1.26 (.229)	1.22 (.308)
Household wealth (proxy measure)		0.596*** (< .001)	0.642*** (< .001)
Place of residence [Urban]			
Rural		1.45** (.004)	1.17 (.318)
Region of residence [Northeast]			
Rio de Janeiro		0.457* (.014)	0.547 (.076)
São Paulo		0.726 (.133)	0.896 (.654)
South		0.419*** (.001)	0.467** (.009)

(continued)

(Table 5, continued)

	Model 1	Model 2	Model 3
Region of residence [Northeast] (cont.)			
Center-East		0.567** (.009)	0.622* (.041)
Center-West		0.678 (.098)	0.698 (.177)
North		1.16 (.362)	1.15 (.432)
Potentially Endogenous Factors			
Age at weaning [0 months]			
1–5 months			0.740 (.134)
6–11 months			0.769 (.260)
12–23 months			0.969 (.889)
24–59 months			0.724 (.233)
Unweaned			0.904 (.657)
Mother's age [20–29 years]			
< 20 years			1.14 (.464)
30+ years			0.899 (.405)
No modern toilet in the home			1.32 (.051)
No piped water in the home			1.29 (.076)
Municipality literacy (%)			0.997 (.532)
Private hospital beds (per 1,000 residents)			1.05 (.103)
Any hospital beds in the municipality (%)			0.940 (.749)
Wald chi-square	17.4*** (< .001)	354.7*** (< .001)	381.8*** (< .001)
Wald Test Contrasts	Chi-square	*df*	*p* value
Model 2 – Model 1	346.3	19	< .001
Model 3 – Model 2	14.9	12	.245

Source: From the author's calculations of data from the DHS-3 (Brazil).

Notes: Reference categories are shown in brackets. Model 1 contains only indicators of mother's race; Model 2 contains racial effects and exogenous background factors; Model 3 contains racial effects, exogenous background characteristics, and potentially endogenous predictors. The models are adjusted for clustering on mother's identification number and use robust standard errors.

[a]Child's age is included as a spline function with nodes at 6 and 24 months.

*$p < .05$; **$p < .01$; ***$p < .001$

differences in regional effects may be attributable to differences in health inputs provided by unmeasured characteristics of the community.

As a group, the potentially endogenous factors in Model 3 do not help to explain stunting, but a child's age at weaning has a notable effect, with children who were weaned after age 1 year experiencing a reduced risk (odds ratio = 0.71–0.73). The measures of average magisterial district-level education and employment do not further explain differences in stunting, nor do household-level sanitation, probably because nearly every white or brown child has modern facilities, whereas few black children do.

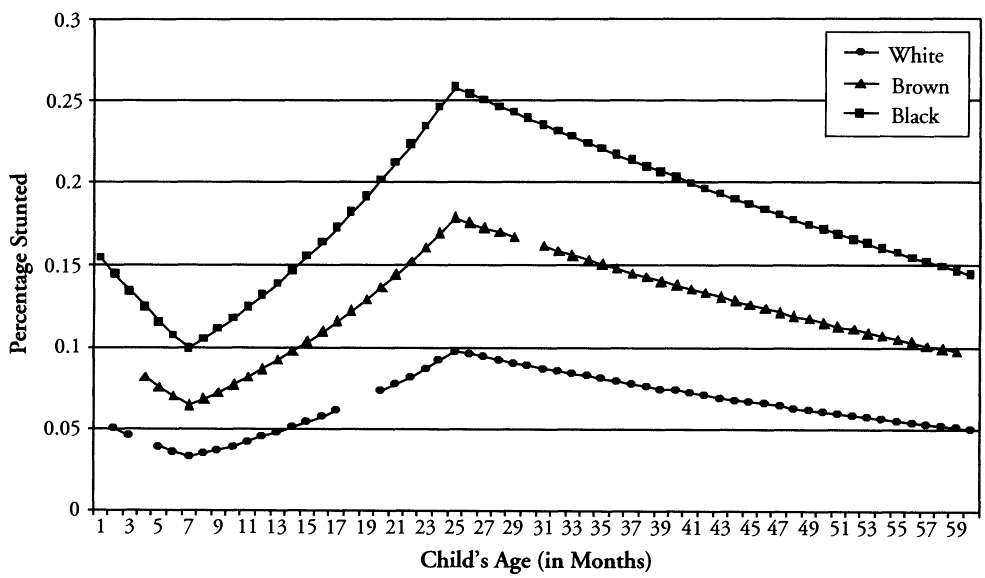
Brazil

Contrary to the results for South Africa, race does not have a statistically significant independent effect on child stunting in Brazil once exogenous child, household, and community characteristics are taken into account in Model 2. The race-only Model 1 in Table 5 shows that browns (odds ratio = 1.6) and blacks (odds ratio = 2.0) have statistically significantly higher odds of stunting, suggesting that inequality in household socioeconomic resources is largely to blame for racial inequality in growth faltering. Children's characteristics have similar effects in Brazil as in South Africa; the odds of stunting increase by 4% per month for children aged 6–23 months and decline by 2% per month for those aged 24–59 months. Boys have odds of stunting that are 1.5 times as great as those of girls.

Compared with children whose mothers have secondary schooling or more, children of women with primary schooling have odds of stunting that are 1.4 times as great. Mother's height has a strong and significant effect on the probability that a child is stunted; for example, if two mothers are identical except for their heights, the odds of a child of a mother who is 10 centimeters taller being stunted are 36% lower than that of the child of the other mother (the coefficient associated with mother's height is –0.101, so $e^{-0.1014 \times 10} = 0.36$). Because mother's height is a measure of her own socioeconomic background (among other things), the independent effects of her education and height provide some evidence that mother's education is not simply a proxy measure for her socioeconomic background, but has important independent effects on her child's health. Although father's education is not a statistically significant predictor, household wealth has a strong and significant protective effect, with a 40% reduction in the odds of stunting for each unit of wealth. This effect of wealth may capture some of the consequences of father's schooling, through the father's relative income. In accordance with expectations, children who live in rural areas have odds of stunting 1.5 times as large as their urban counterparts, a significant effect. Region of residence also has a strong impact, and children in the South and Center regions have odds of stunting that are 43%–58% less than those in the Northeast; the effect is statistically significant for Rio de Janeiro, the South, and the Center-East. Although the potentially endogenous determinants were not jointly significant in Model 3, the lack of modern sanitation in the household increases the likelihood of stunting by 30% (no piped water) to 32% (no modern toilet); the effect, however, is significant only at the 10% level.

Predicted probabilities obtained from Model 2 are presented in Figure 2 for South Africa and Figure 3 for Brazil, which show the predicted proportion of children stunted by age in months, separately by mother's race. Mother's and father's education are set at secondary schooling or above, mother's height (Brazil only) is set to the Brazil-specific mean value, child's sex is set to female, household wealth is set to the country-specific mean value, place of residence is set to urban, province in South Africa is set to Transvaal, and region in Brazil is set to São Paulo. Reference values were chosen to reflect either the most protective levels (mother's and father's education, region) or the countrywide means (household wealth, mother's height). These reference values provide a conservative estimate of the effect of race because children who are raised in more favorable conditions will have scores closer, on average, to the international reference median height for age, and there is less room for racial variation among these better-off children.

Figure 2. Predicted Proportion of Children Stunted, by Child's Age (in Months) and Mother's Race, Weighted Values for South Africa (SAIHS Data, $N = 3{,}156$)[a]



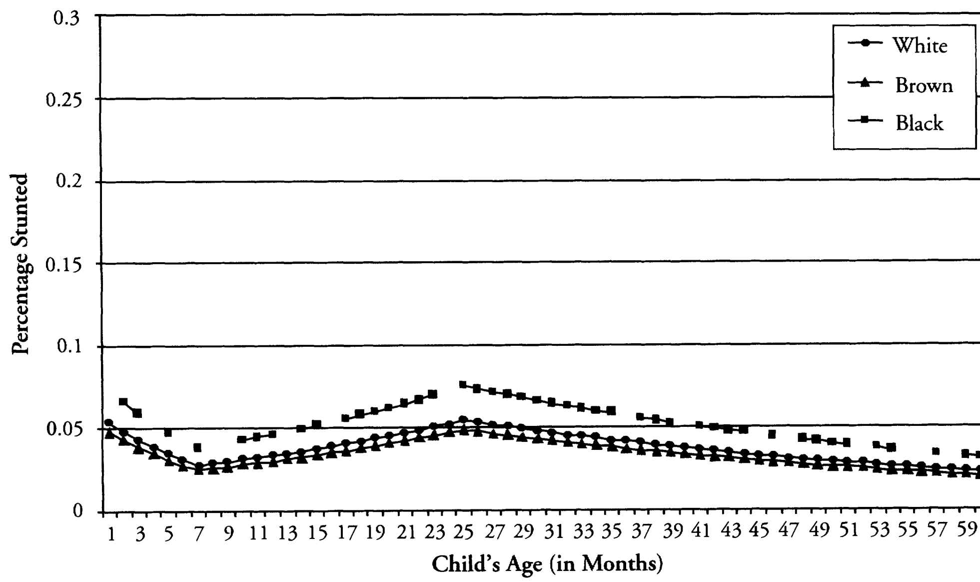
[a]Predicted values were obtained from Model 2, with mother's and father's education set at secondary schooling or above, child's sex set to female, household wealth set to the South Africa–wide mean value, place of residence set to urban, and province set to Transvaal.

The first obvious difference between Figures 2 and 3 is in the overall magnitude of stunting; even after differences in the distribution of children's and household characteristics are controlled, the predicted proportion of stunted children is much higher in South Africa. The peak predicted value of stunting, around age 24 months, is about 25% among black children in South Africa and less than 10% for black children in Brazil. Racial differences in stunting are greater in South Africa, with browns and blacks more likely than whites to be stunted at all ages. The comparisons of predicted values for all pairs of racial groups are statistically significant at the 5% level in South Africa. Although the racial differences in predicted values are not as large in Brazil, the difference between browns and blacks is significant at the 5% level. The variation in predicted levels of stunting over children's ages is much more dramatic in the South African sample, ranging from about 5% to 25%, whereas the range in the Brazilian sample is between about 2% and 8%.

DISCUSSION

These results suggest that racial differences in child stunting in both South Africa and Brazil are determined, in large part, by differences across racial groups in average levels of household socioeconomic resources. Regardless of state-sanctioned racism, both countries are characterized by racial inequality in the distribution of important health inputs like parent's education and household wealth, and these inputs are strong determinants of stunting. Nonetheless, even after these household background factors are controlled, brown and black children in South Africa are still significantly more likely to be stunted than are white children.

Figure 3. Predicted Proportion of Children Stunted, by Child's Age (in Months) and Mother's Race, Weighted Values for Brazil (DHS-3 Data, N = 4,014)[a]



[a]Predicted values were obtained from Model 2, with mother's and father's education set at secondary schooling or above, child's sex set to female, household wealth and mother's height set to the Brazil-wide mean value, place of residence set to urban, and region set to São Paulo.

There are at least two potential explanations for the persisting South African racial gap in stunting under apartheid. First, SES may have been poorly measured at the household and/or community level; second, state institutions that legitimated racial discrimination may have made it difficult to raise healthy children in ways that are not fully captured by models of background household and community determinants. Commonly used indicators of SES may not reflect the full extent of the differences in economic resources among racial groups (Williams and Collins 1995); for example, the simple household wealth index used here is not a good indicator of differences between whites and nonwhites in the volatility of income or the long-term accumulation of wealth. In addition, dramatic differences by race in average levels of education and wealth may make comparisons of whites and nonwhites difficult because of the insufficient overlap in circumstances (Krieger et al. 1993). SES and race do not act independently to influence health; future research will need to explore more deeply the way that race modifies the effect of household resources or community-level health inputs. Such work will require multiracial samples that are sufficiently large to allow for comparative analyses within different levels of household economic resources (Lillie-Blanton et al. 1996).

On the other hand, the racial gap in South Africa may provide some indication that state-sanctioned racism generated greater racial inequality in South Africa than exists in Brazil. Without controls for specific legal institutions in this analysis, however, it is difficult to reach conclusions about the causal role of apartheid. Nonetheless, a growing body of research based in developed countries suggests that racism is a central determinant of health status (Williams 1995). Racism against mothers could influence the use of medical services and medical information or restrict the residential options for nonwhite families to living in unhealthy communities. The simple controls used here to represent community-level health services and other conditions could not capture these aspects of racism, but future work should consider more sensitive measures of perceived discrimination against nonwhites and of the quality of health care services that are provided to mothers of different racial groups. It is challenging to collect accurate, detailed, and timely community-level information, especially for developing countries like South Africa, where each homeland had its own underfunded statistical collection system under apartheid. Nonetheless, future analyses of the effects of racism on children's health would be strengthened by indicators of social context that are directly influenced by state decision making.

Although it is clear that measures to reduce socioeconomic inequality would go a long way toward reducing racial differences in children's growth, the biosocial pathways through which these factors work are not obvious. Measures of the proximate determinants of stunting that were used in this analysis did not elucidate observable pathways between a mother's education, for example, and her child's health. Modern toilets and piped water in both countries and the duration of breast-feeding in South Africa may be relevant, but the results presented here are not conclusive, and further studies that focus specifically on these pathways are needed. The dearth of direct pathways is not surprising, given that stunting is the product of a cumulative series of insults to health that are related to socioeconomic deprivation on several levels. Although state-sanctioned racism may help to explain the greater magnitude of stunting among nonwhite children relative to white children in South Africa than in Brazil, the reduction of racial inequality in stunting will require both countries to address not only the immediate determinants, such as nutritional intake and morbidity, but the broad social causes of socioeconomic inequality that underlie them.

REFERENCES

Andersson, N. and S. Marks. 1988. "Apartheid and Health in the 1980s." *Social Science and Medicine* 27:667–81.

Barrera, A. 1990. "The Role of Maternal Schooling and Its Interaction With Public Health Programs in Child Health Production." *Journal of Development Economics* 32:69–91.

Becker, G. 1981. *A Treatise on the Family.* Cambridge, MA: Harvard University Press.

Behrman, J.R. and B.L. Wolf. 1987. "Women's Schooling and Children's Health: Are the Effects Robust With Adult Sibling Control for the Woman's Childhood Background?" *Journal of Health Economics* 6:239–54.

BEMFAM/DHS. 1996. *Preliminary Results: 1996 DHS Survey of Brazil.* Columbia, MD: Macro International.

BEMFAM/IRD. 1998. *South Africa—Demographic and Health Survey. Preliminary Report.* Calverton, MD: Macro International.

Boerma, J.T. 1996. *Child Survival in Developing Countries: Can Demographic and Health Surveys Help to Understand the Determinants?* Amsterdam: Royal Tropical Institute.

Bollen, K.A., J.L. Glanville, and G. Stecklov. 2001. "Socioeconomic Status and Class in Studies of Fertility and Health in Developing Countries." *Annual Review of Sociology* 27:153–85.

Botha, J.L. and D. Bradshaw. 1985. "African Vital Statistics—A Black Hole?" *South African Medical Journal* 67:977–81.

Buss, P. and P. Gadelha. 1996. "Health Care Systems in Transition: Brazil Part I: An Outline of Brazil's Health Care System Reforms." *Journal of Public Health Medicine* 18:289–95.

Chaning-Pearce, S.M. and L. Solomon. 1986. "A Longitudinal Study of Height and Weight in Black and White Johannesburg Children." *South African Medical Journal* 70:743–46.

Christopher, A.J. 1994. *The Atlas of Apartheid.* New York: Routledge.

DATASUS. 1996. Informaes de Saude. Available on-line at http://tabnet.datasus.gov.br/tabnet/tabnet.htm

DaVanzo, J. and P. Gertler. 1991. "Household Production of Health: A Micro-economic Perspective on Health Transitions." Pp. 85–101 in *The Health Transition: Methods and Measures,* edited by J. Cleland and A.G. Hill. Canberra: Australian National University Press.

Degler, C.N. [1971] 1986. *Neither Black nor White: Slavery and Race Relations in Brazil and the United States*. Madison: University of Wisconsin Press.

Filmer, D. and L.H. Pritchett. 2001. "Estimating Wealth Effects Without Expenditure Data—or Tears: An Application to Educational Enrollments in States of India." *Demography* 38:115–32.

Frank, R. 2001. "The Misuse of Biology in Demographic Research on Racial/Ethnic Differences: A Reply to van den Oord and Rowe." *Demography* 38:563–68.

Glei, D. and N. Goldman. 2000. "Understanding Ethnic Variation in Pregnancy-Related Behaviors, Health Care and Birth Outcomes in Rural Guatemala." *Ethnicity and Health* 5:5–22.

Habicht J-P., R. Martorell, C. Yarbrough, R. Malina, and R.E. Klein. 1974. "Height and Weight Standards for Pre-school Children: How Relevant Are Ethnic Differences in Growth Potential?" *Lancet* 1:611–15.

Hasenbalg, C.A. 1985. "Race and Socioeconomic Inequalities in Brazil." Chap. 3 in *Race, Class, and Power in Brazil,* edited by P.M. Fontaine. Los Angeles: UCLA Center for Afro-American Studies.

Horton, S. 1986. "Child Nutrition and Family Size in the Philippines." *Journal of Development Economics* 23:161–76.

Jinabhai, C.C., H.M. Coovadia, and S.S. Abdool-Karim. 1986. "Socio-medical Indicators of Health in South Africa." *International Journal of Health Services* 16:163–76.

Kaufman, C.E. 1998. "Contraceptive Use in South Africa Under Apartheid." *Demography* 35:421–34.

Krieger, N., D.L. Rowley, A.A. Herman, B. Avery, and M.T. Phillips. 1993. "Racism, Sexism, and Social Class: Implications for Studies of Health, Disease, and Well-being." *American Journal of Preventive Medicine* 9(Suppl.):82–122.

Lam, D. 1999. "Generating Extreme Inequality: Schooling, Earnings, and Intergenerational Transmission of Human Capital in South Africa and Brazil." Unpublished manuscript, Department of Economics and Population Studies Center, University of Michigan.

LaVeist, T. 1996. "Why We Should Continue to Study Race . . . But Do a Better Job: An Essay on Race, Racism, and Health." *Ethnicity and Health* 6:21–29.

Liang, K.-Y. and S.L. Zeger. 1986. "Longitudinal Data Analysis Using Generalized Linear Models." *Biometrika* 73:13–22.

Lillie-Blanton, M., P.E. Parsons, H. Gayle, and A. Dievler. 1996. "Racial Differences in Health: Not Just Black and White, but Shades of Gray." *Annual Review of Public Health* 17:411–48.

Lovell, P.A. and C.H. Wood. 1998. "Skin Color, Racial Identity, and Life Chances in Brazil." *Latin American Perspectives* 25:90–110.

Madise, N.J., Z. Matthews, and B. Margetts. 1999. "Heterogeneity of Child Nutritional Status Between Households: A Comparison of Six Sub-Saharan African Countries." *Population Studies* 53:331–43.

Martorell, R. and J.-P. Habicht. 1986. "Growth in Early Childhood in Developing Countries." Pp. 241–62 in *Human Growth: A Comprehensive Treatise: Volume 3,* edited by F. Falkner and J.M. Tanner. New York: Plenum Press.

Mendez, M.A. and L.S. Adair. 1999. "Severity and Timing of Stunting in the First Two Years of Life Affect Performance on Cognitive Tests in Late Childhood." *Journal of Nutrition* 129:1555–62.

Monteiro, C.A., M.H. D'A. Benicio, and N.C. Gouveia. 1994. "Secular Growth Trends in Brazil Over Three Decades." *Annals of Human Biology* 21:381–90.

Monteiro, C.A., M.H. D'A. Benicio, R. Iunes, N.C. Gouveia, J.A.A.C. Taddei, and M.A.A. Cardoso. 1992. "Nutritional Status of Brazilian Children: Trends From 1975 to 1989." *Bulletin of the World Health Organization* 70:657–66.

Mosley, W.H. and L.C. Chen. 1984. "An Analytical Framework for the Study of Child Survival in Developing Countries." In *Child Survival: Strategies for Research,* edited by W.H. Mosley and L.C. Chen. *Population and Development Review* 10(Suppl.):25–45.

National Economic Research Associates. 1996. *The Health Care System in South Africa. Financing Health Care With Particular Reference to Medicines, Volume 19.* Basle: Pharmaceutical partners; London: National Economic Research Associates.

Pan American Health Organization. 2001. "Brazil: Basic Country Health Profiles." Available online at http://www.paho.org/english/SHA/prflbra.htm

Pebley, A.R. and N. Goldman. 1995. "Social Inequality and Children's Growth in Guatemala." *Health Transition Review* 5:1–20.

Pelletier, D.L. 1991. "Relationships Between Child Anthropometry and Mortality in Developing Countries: Implications for Policy, Programs, and Future Research." Monograph 12. Ithaca, NY: Food and Nutrition Policy Program, Cornell University.

———. 1998. "Malnutrition, Morbidity, and Child Mortality in Developing Countries." Pp. 109–32 in *Too Young to Die: Genes or Gender?* New York: United Nations.

Pelletier, D.L., E. Frongillo, and J-P. Habicht. 1993. "Epidemiological Evidence for a Potentiating Effect of Malnutrition on Child Mortality." *American Journal of Public Health* 83:1130–33.

Reichmann, R., ed. 1999. *Race in Contemporary Brazil: From Indifference to Inequality.* University Park: University of Pennsylvania Press.

Rosenzweig, M.R. and T.P. Schultz. 1982. "Child Mortality and Fertility in Columbia: Individual and Community Effects." *Health Policy and Education* 2:305–48.

Silva, N. 1985. "Updating the Cost of Not Being White in Brazil." Chap. 4 in *Race, Class, and Power in Brazil,* edited by P.M. Fontaine. Los Angeles: UCLA Center for Afro-American Studies.

———. 1999. "Racial Differences in Income: Brazil, 1988." Pp. 68–82 in *Race in Contemporary Brazil: From Indifference to Inequality,* edited by R. Reichmann. University Park: Pennsylvania State University Press.

Siqwala-Ndulo, N.W. 1993. *Rural African Family Structure in the Transkei, South Africa.* Unpublished doctoral dissertation, University of California at Los Angeles.

South African Labour Development Research Unit. 1994. "South Africans Rich and Poor: Baseline Household Statistics." Cape Town, South Africa: University of Cape Town.

StataCorp. 1999. *Stata User's Guide. Stata Statistical Software: Release 6.0.* College Station, TX: Stata Corporation.

Strauss, J. 1990. "Households, Communities and Preschool Child Nutrition Outcomes: Evidence From Rural Côte d'Ivoire." *Economic Development and Cultural Change* 38:231–61.

Sullivan, K.M. and J. Gorstein. 1999. *ANTHRO: Software for Calculating Anthropometry.* Version 1.02. Atlanta: Centers for Disease Control and Prevention; Geneva: World Health Organization.

Telles, E.E. 1992. "Residential Segregation by Skin Color in Brazil." *American Sociological Review* 57:186–97.

Telles, E.E. and N. Lim. 1998. "Does It Matter Who Answers the Race Question? Racial Classification and Income Inequality in Brazil." *Demography* 35:465–74.

Thomas, D., J. Strauss, and M.-H. Henriques. 1990. "Child Survival, Height for Age, and Household Characteristics in Brazil." *Journal of Development Economics* 33:197–234.

———. 1991. "How Does Mother's Education Affect Child Height?" *Journal of Human Resources* 26:183–211.

Treiman, D.J., M. McKeever, and E. Fodor. 1996. "Racial Differences in Occupational Status and Income in South Africa, 1980 and 1991." *Demography* 33:111–32.

van den Oord, E.J.C.G. and D.C. Rowe. 2001. "A Step in Another Direction: Looking for Maternal Genetic and Environmental Effects on Racial Differences in Birth Weight." *Demography* 38:573–76.

Waterlow, J., R. Buzina, W. Keller, J. Lane, M. Nichaman, and J. Tanner. 1977. "The Presentation and Use of Height and Weight Data for Comparing the Nutritional Status of Groups of Children Under the Age of Ten Years." *Bulletin of the World Health Organization* 55:489–98.

Williams, D.R. 1995. "Racism and Health: A Research Agenda." *Ethnicity and Disease* 6:1–6.
Williams, D.R. and C. Collins. 1995. "U.S. Socioeconomic and Racial Differences in Health: Patterns and Explanations." *Annual Review of Sociology* 21:349–86.
Wood, C.H. and J.A.M.D. Carvalho. 1988. *The Demography of Inequality in Brazil.* New York: Cambridge University Press.
Wood, C.H. and P. Lovell. 1992. "Racial Inequality and Child Mortality in Brazil." *Social Forces* 70:703–24.
Zenger, E. 1993. "Siblings' Neonatal Mortality Risks and Birth Spacing in Bangladesh." *Demography* 30:477–72.
Zuberi, T. 2001. "One Step Back in Understanding Racial Differences in Birth Weight." *Demography* 38:569–72.