

Hispanic Paradox in Biological Risk Profiles

Eileen M. Crimmins Jung Ki Kim Dawn Alley Arun Karlamangla Teresa Seeman

CCPR-029-07

December 2007

California Center for Population Research On-Line Working Paper Series

Hispanic Paradox in Biological Risk Profiles

Eileen M. Crimmins, PhD, Jung Ki Kim, PhD, Dawn E. Alley, PhD, Arun Karlamangla, MD, PhD, Teresa Seeman, PhD

Many studies report that Hispanics in the United States have better or similar health to that of non-Hispanic Whites (hereafter referred to as Whites), despite Hispanics having lower incomes and less education.^{1,2} Most studies that examine differences in adult mortality find that Hispanics have relatively lower mortality rates compared with Whites.^{2–6} This betterthan-expected health and mortality of Hispanics, given their lower socioeconomic status (SES), has been called the Hispanic paradox.^{6,7}

Not all empirical findings support the existence of a Hispanic paradox. Differentials depend on the domain of health and the population investigated. Evidence for a Hispanic mortality advantage is strongest among men, persons of advanced age, and those born in Mexico.⁶ However, some studies have found no difference in mortality between Hispanics and Whites,⁸ and others have questioned the data quality in estimates of mortality among Hispanics.^{9,10}

Ethnic differences are even less clear-cut in analyses of function, disability,^{11,12} and morbidity.³ Self-reports of health status may be influenced by cultural differences in reporting or differences in health knowledge acquired through interaction with the medical system.¹³ Analyses of self-reported health status usually find that Hispanics report worse health than Whites.^{1,14}

Researchers have argued recently that the migrants who immigrate are different from persons from the same country of origin who do not migrate may also play a large role in observed Hispanic health advantages, suggesting that the "paradox" may not be so paradoxical in a population that is heavily weighted with immigrants.^{15,16} The healthy migrant hypothesis provides 1 explanation for betterthan-expected health outcomes among Hispanics.^{17,18} It has been suggested that Hispanics who immigrate to the United States are healthier than Hispanics who remain in their country of origin; this selection of healthy persons from the sending population can improve the level of health in the receiving population. Another explanation of the Hispanic paradox is the

Objectives. We examined biological risk profiles by race, ethnicity, and nativity to evaluate evidence for a Hispanic paradox in measured health indicators.

Methods. We used data on adults aged 40 years and older (n = 4206) from the National Health and Nutrition Examination Surveys (1999–2002) to compare blood pressure, metabolic, and inflammatory risk profiles for Whites, Blacks, US-born and foreign-born Hispanics, and Hispanics of Mexican origin. We controlled for age, gender, and socioeconomic status.

Results. Hispanics have more risk factors above clinical risk levels than do Whites but fewer than Blacks. Differences between Hispanics and Whites disappeared after we controlled for socioeconomic status, but results differed by nativity. After we controlled for socioeconomic status, the differences between foreign-born Hispanics and Whites were eliminated, but US-born Mexican Americans still had higher biological risk scores than did both Whites and foreignborn Mexican Americans.

Conclusions. There is no Hispanic paradox in biological risk profiles. However, our finding that foreign-born Hispanics and Whites had similar biological risk profiles, but US-born Mexican Americans had higher risk, was consistent with hypothesized effects of migrant health selectivity (healthy people in-migrating and unhealthy people out-migrating) as well as some differences in health behaviors between US-born and foreign-born Hispanics. (*Am J Public Health.* 2007;97: 1305–1310. doi:10.2105/AJPH.2006.091892)

"salmon hypothesis," which suggests that sick persons return to their place of origin.^{16,17} Palloni and Arias¹⁶ emphasized this second explanation for their finding that the mortality advantage is limited to foreign-born Hispanics, particularly those who were born in Mexico. Both of these explanations for the Hispanic paradox imply that the Hispanic health advantage is a feature exclusive to foreign-born Hispanics, rather than US-born Hispanics.

Measurements of biological risk factors for poor health (e.g., blood pressure, blood glucose, and cholesterol) should provide objective indicators of health status that are related to subsequent onset of disease, loss of function, and mortality.¹⁹⁻²¹ We examined differences in 10 physiological indicators by race, ethnicity, and nativity.²¹ These indicators represent multiple physiological processes and have individually and cumulatively been linked to important agerelated health outcomes, including cardiovascular disease, cognitive decline, physical disability, and death.²²⁻²⁴ We examined both a summary indicator of risk as well as blood pressure, metabolism, and inflammation risk profiles to investigate whether it was possible to identify

which physiological systems accounted for overall differentials by race, ethnicity, and nativity. If differentials were concentrated among 1 or 2 sets of indicators rather than spread across categories, this would provide another indicator of how health differences arise.

Risk profiles based on multiple factors are useful in the analyses of a variety of health outcomes.^{25–27} The total number of indicators of physiological status outside the normal operating range has been shown to be a better predictor of health outcomes than individual markers,^{21,24} but differences in risk by type of marker may be informative for the analysis of racial and ethnic differences. It is possible that differentials in risk factors by race, ethnicity, and nativity may be more concentrated in some physiological systems than in others. For instance, Blacks have been shown to have a higher prevalence of hypertension, a cardiovascular risk factor, than either Whites or Hispanics of Mexican origin (Mexican Americans).²⁸ Conversely, metabolic syndrome is more prevalent in Mexican Americans than it is in Blacks.²⁹ Blacks also have been shown to have higher levels of inflammatory markers such

as C-reactive protein^{30,31} and fibrinogen.³² Differences in Hispanics are more complicated and vary by subgroup. One study of all subgroups of Hispanic women found that C-reactive protein levels were similar to those in White women,³¹ but an analysis of Mexican American women found them to have higher levels of C-reactive protein than Whites.³⁰

We used measured indicators of physiological status to determine whether Hispanics had biological risk profiles similar to those of Whites. We further examined how biological risk profiles vary by nativity in the total Hispanic population and in Hispanics of Mexican origin. If there is a Hispanic paradox in biological profiles, Hispanics would be expected to have better risk profiles compared with Whites after control for SES.

METHODS

Data

We obtained data from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, a representative sample of the US civilian, noninstitutionalized population collected by the National Center for Health Statistics. NHANES included information from a questionnaire, laboratory analysis, and clinical exams. We used data for Whites, Blacks, and Hispanics aged 40 years and older (N=5912) who participated in the laboratory analysis and physical exams and who had information on 10 biological markers used in our study (n=4855) and additional independent variables (n=4206).

The analytic sample (n=4206) was ethnically diverse, consisting of Whites (n=2338), Blacks (n=717), and US-born (n=505) and foreign-born Hispanics (n=646). Hispanics were self-identified as born in or with ancestors from Spain or other Spanish-speaking countries in Central America, South America, or both, including the Caribbean basin. Hispanics of Mexican birth or descent who selfidentified as Mexican, Mexican American, Chicano, or Tex-Mex were classified as Mexican Americans. Because of the sampling design, the Hispanic population in NHANES 1999-2002 is primarily Mexican American (84.2%). The sample of 4206 persons who had complete data included 455 US-born participants who identified themselves as

being of Mexican origin and 508 persons who indicated that they were born in Mexico. For the analysis of differences between the total Hispanic population and others, the data were weighted to represent the total US Hispanic population, about half of which is individuals of Mexican origin (46.7 %).

Respondents excluded from the survey because of missing data in all race and ethnic groups were more likely to be women, older, and to have lower education levels than those cases included in the analysis. Because of missing biological data, 16% of Whites, 16% of Hispanics, and 27% of Blacks were excluded. Whites and Hispanics did not differ significantly from each other in the percentage missing data for any of the 10 individual indicators; nor did foreign-born and US-born Hispanics differ from each other in the percentage of missing data for any of the indicators. However, foreign-born Hispanics were more likely than were Whites to lack values for blood pressure (P=.003) and were less likely to be missing data for body mass index (weight in kilograms divided by height in meters squared; P=.005). Blacks were more likely than other groups to have missing data for all indicators except body mass index. Self-reported health status for individuals who were missing data and for cases used in the analysis, when compared within racial and ethnic groups, indicated no difference in self-reported health for Blacks and Mexican Americans, but Whites and all Hispanics who are missing from the analysis reported somewhat worse health.

Some respondents had missing data for 1 or more indicators of SES, health behaviors, or access to care (n=649). Information about income was most likely to be missing (n=449). We tested the sensitivity of our results to the inclusion of people who did not report income by coding them to the mean of the poverty ratio and including a dummy variable to indicate their missing status. This did not change results.

Measures

We created risk scores by summing the number of biological risk factors that met clinical high-risk criteria. Details about measures are provided in Table 1. Our measurements included 3 indicators related to blood pressure (systolic and diastolic blood pressure and pulse); 4 indicators of metabolic functioning

TABLE 1—Clinical High-Risk Criteria for Risk Factors: National Health and Nutrition Examination Survey, 1999–2002

Biological Risk Indicators	High-Risk Cutpoint
Blood pressure risk factors	
Systolic blood pressure ^a	\geq 140 mm Hg ³³
Diastolic blood pressure ^a	\geq 90 mm Hg ³³
Pulse rate at 60 s	≥90
Metabolic risk factors	
Total cholesterol ^b	\geq 240 mg/dL ³⁴
HDL cholesterol ^b	<40 mg/dL ³⁴
Body mass index ^c	\geq 30 kg/m ³⁵
Glycated hemoglobin ^d	$\geq 6.4 \%^{36}$
Inflammation risk factors	
C-reactive protein ^e	> 3.0 mg/L ³⁷
Fibrinogen	>400 mg/dL ³⁸
Albumin	< 3.8 g/dL ³⁹

Note. HDL = high-density lipoprotein.

^aAn average of 2 or 3 sets of seated blood pressure measurements.

 ^bMeasured with Boehringer Mannheim/Hitachi 737
 Analyzer (Roche, Basel, Switzerland).
 ^cWeight in kilograms divided by height in meters squared.
 ^dMeasured by boronate affinity chromotagraphy.
 ^eC-reactive protein analyzed by high-sensitivity latexenhanced nephelometry on a BNII nethelometer.

(total cholesterol, high-density lipoprotein cholesterol, glycated hemoglobin, and body mass index); and 3 indicators of inflammation (C-reactive protein, fibrinogen, and albumin). Risk was determined by levels measured by clinical and laboratory tests without consideration of prescription drug usage. Although drugs can be used to control hypertension and cholesterol levels, many people who take them do not achieve levels below the cutoff of what is considered high, particularly for hypertension.⁴⁰ We developed a summary measure that indicated the number of elevated risk factors present in total (range 0 to 10) and in each of the 3 systems: blood pressure (0-3), metabolic (0-4), and inflammatory (0-3).

We examined associations between race, ethnicity, nativity, and biological risk scores after we controlled for SES.^{41–44} We used 2 indicators of SES: (1) education (less than 12 years of schooling), which would reflect lifelong conditions, and (2) household income as a percentage of poverty level, which would reflect current conditions. The definition of education was limited by the detail on

education provided in NHANES 1999–2002 as a result of privacy concerns.

To better account for sources of differences in biological risk by race, ethnicity, and nativity, we controlled for some of the mechanisms by which demographic and social factors might be associated with biological risk profiles, including health behaviors and availability of medical care. Health behaviors included an indicator of lack of exercise (no vigorous or moderate activity during the last 30 days), current smoking, and poor diet (defined as more than 30% of energy consumption coming from fat).^{45–48} Because health care use may also affect biological risk profiles and be related to SES, we controlled for current health insurance availability, either public or private, which we used as a proxy for access to health care.

Analysis

We determined the mean number of biological risk factors at high-risk levels by race, ethnicity, and nativity groups and adjusted for covariates using Stata 8.2 (Stata Corp, College Station, Tex) to account for the complex sample design. We first examined differences by race, ethnicity, and nativity; we controlled for age and gender because they both vary across these population groups and both are related to these risk factors.^{49,50} We then controlled for SES to examine how the means and differences in the risk profile score by race, ethnicity, and nativity would change. Finally, we added a control for health behaviors and access to care to determine whether race, ethnicity, and nativity differences were independent of these factors. Because there is significant variation in health, SES, and migration history¹ within the Hispanic population, we also separately examined biological risk scores in the largest Hispanic group, Mexican Americans.

RESULTS

In health-related surveys, socioeconomic characteristics of ethnic groups have been shown to differ in different national samples⁴⁹; therefore, it was important to clarify how the race, ethnic, and nativity groups in our analysis differed in education and income (Table 2). Hispanics had the lowest SES of the 3 major groups in our sample. They had the largest percentage of individuals who had low levels of education (49.8%), the lowest income-to-poverty ratio (2.19), and the largest proportion of individuals living below or near the federal poverty level (37.8%), all significantly larger than those for Whites or Blacks. Foreign-born Hispanics had significantly lower levels of education and higher levels of poverty than US-born Hispanics. Among Mexican Americans, those who were born in Mexico had very low levels of education and income, lower than those of Whites, Blacks, and US-born Mexican Americans. USborn Mexican Americans were less likely than other Hispanic groups or Blacks to live below or near the federal poverty level.

TABLE 2—Socioeconomic Characteristics of Respondents (N=4206), by Race, Ethnicity, and Nativity: National Health and Nutrition Examination Survey, 1999–2002

			All Hispanic			Mexican American		
	White (n = 2338)	Black (n = 717)	All (n = 1151)	US born (n = 505)	Foreign born (n = 646)	All (n=963)	US born (n = 455)	Mexican born (n = 508)
Less than 12 years education (%)	15.4	39.3ª	49.8 ^{a,b}	36.6 ^{a,b}	58.1 ^{a,b,c}	59.1 ^{a,b}	40.0 ^{a,b}	75.9 ^{a,b,c}
Household income less than 125% of federal poverty level (%)	12.2	27.3ª	37.8 ^{a,b}	30.7 ^{a,b}	42.3 ^{a,b,c}	32.8 ^{a,b}	21.0 ^{a,b}	43.2 ^{a,b,c}
Poverty income ratio ^d (mean)	3.50	2.55ª	2.19 ^{a,b}	2.60 ^a	1.92 ^{a,b,c}	2.27 ^{a,b}	2.84 ^{a,b}	1.77 ^{a,b,c}

^aSignificantly different from White.

^bSignificantly different from Black.

^cSignificantly different from US-born at the P = .05 level.

^dThe poverty income ratio was the ratio of the family income in a given year to the federally defined poverty level.

Biological Risk Including All Hispanics

Table 3 shows mean numbers of high-risk biological factors that have been adjusted for age and gender. Differences in risk profiles by race and ethnicity, and when controlled for age and gender, indicated that Hispanics had a higher average biological risk score than did Whites. This was true for total risk as well as for the metabolic and inflammatory subcategories of risk factors, which indicated that increased risk was spread over multiple physiological systems (Table 3, model 1). Hispanics also had significantly fewer biological risk factors at high-risk levels in total and for blood pressure and inflammatory scales than did Blacks. Hispanics and Blacks had similar numbers of metabolic factors at high-risk levels even though previous research has emphasized high metabolic risk among Hispanics compared with Blacks.

Both US-born and foreign-born Hispanics had higher numbers of high-risk biological factors than did Whites. In addition, US-born Hispanics had more inflammatory factors at high-risk levels than did Whites. A comparison of biological risk factors among all Hispanics who were US born and those who were foreign born indicated that the number of biological risk factors at high-risk levels was not significantly different between all US-born and foreign-born Hispanics.

The question of the Hispanic paradox deals with differences relative to SES, so it was appropriate to examine differences in biological risk factors while controlling for education and income level. After we controlled for SES (Table 3, model 2), we found no significant difference in the number of risk factors at high-risk levels between Whites and all Hispanics in any of the measured biological systems, which indicated that if the distribution of low education and income or poverty were the same, biologically estimated risk would not be significantly different between these 2 groups. Another interpretation is that the differences are "explained" by SES. Results were the same for foreign-born and US-born Hispanics.

Blacks had a higher number of biological risk factors at high-risk levels than did Hispanics or Whites, even after we controlled for SES. Although Blacks no longer differed in metabolic risk from the other groups, the

TABLE 3—Mean Biological Risk Score, by Race, Ethnicity, and Nativity: National Health and Nutrition Examination Survey, 1999–2002

			All Hispanic			Mexican American		
	White (n = 2338)	Black (n = 717)	All (n = 1151)	US born (n = 505)	Foreign born (n = 646)	All (n = 963)	US born (n = 455)	Mexican born (n = 508)
Model 1								
Total risk (0-10)	1.87	2.58ª	2.19 ^{a,b}	2.23 ^{a,b}	2.16 ^{a,b}	2.26 ^{a,b}	2.41 ^a	2.14 ^{a,b,c}
Blood pressure risk (0-3)	0.36	0.61 ^ª	0.43 ^b	0.41 ^b	0.45 ^b	0.45 ^{a,b}	0.51 ^ª	0.40 ^b
Metabolic risk (0-4)	0.82	0.96 ^a	0.96 ^a	0.99	0.94	1.00 ^a	1.03 ª	0.98 ^a
Inflammation risk (0-3)	0.69	1.01 ^a	0.79 ^{a,b}	0.83 ^{a,b}	0.77 ^b	0.81 ^{a,b}	0.87 ^{a,b}	0.76 ^b
Model 2								
Total risk (0–10)	1.92	2.40 ª	1.98 ^b	2.08	1.85 ^b	2.00 ^b	2.28 ª	$1.72^{b,c}$
Blood pressure risk (0-3)	0.37	0.58 ª	0.39 ^b	0.38 ^b	0.40 ^b	0.41 ^b	0.49	0.33 ^{b,c}
Metabolic risk (0-4)	0.85	0.89	0.85	0.92	0.80	0.87	0.97	0.78 ^c
Inflammation risk (0-3)	0.70	0.95 ^a	0.71 ^b	0.78 ^b	0.66 ^b	0.72 ^b	0.82 ^b	$0.62^{b,c}$
Model 3 ^{d,e}								
Total Risk (0–10)	1.92	2.40 ª	1.98 ^b	2.08	1.90 ^b	2.03 ^b	2.28 ª	$1.79^{b,c}$
Blood pressure risk (0-3)	0.37	0.59 ^a	0.39 ^b	0.38 ^b	0.40 ^b	0.37 ^b	0.49 ^b	0.33 ^{b,c}
Metabolic risk (0-4)	0.85	0.88	0.86	0.92	0.81	0.88	0.96	0.80 ^c
Inflammation risk (0-3)	0.70	0.93ª	0.73 ^b	0.78 ^b	0.69 ^b	0.74 ^b	0.83	0.65 ^{b,c}

Note. Model 1 adjusted for age and gender. Model 2 adjusted for age, gender, low education, and poverty score. Model 3 adjusted for age, gender, low education, poverty score, health behaviors, and access to health care.

^aSignificantly different from White.

^bSignificantly different from Black.

^cSignificantly different from US born at the .05 level.

^dHealth behaviors included current smoking status, poor diet (defined as more than 30% of energy coming from fat; from self-reports of all foods and beverages consumed in a 24-h period before interview); and no exercise (i.e., no vigorous or moderate exercise for at least 10 minutes over the past 30 days; vigorous activities defined as those that cause heavy sweating or large increases in breathing or heart rate [e.g., running, lap swimming, aerobics classes, or fast bicycling], and moderate activities defined as those that cause only light sweating or a slight to moderate increase in breathing or heart rate [e.g., brisk walking, bicycling for pleasure, golf, and dancing]).

^eMeasured as currently having insurance (private or public, Medicare, Medicaid/Children's Health Insurance Program, or other government insurance).

differences between Blacks and Hispanics and between Blacks and Whites hardly changed after SES was controlled, which indicated that Blacks had a higher risk for poor health than predicted by their level of SES. The disappearance of the difference between Blacks and others in metabolic risk indicates that the difference was related more to SES disparities than to disparities in other areas of biological risk.

Biological Risk Among Only Mexican Americans

When we limited our analysis of Hispanics to Mexican Americans, the results were generally similar to the results for all Hispanics. When we controlled for age and gender, the number of risk factors at high-risk levels in total and within the 3 subcategories was higher among Mexican Americans than among Whites. When we examined Mexican Americans by nativity, US-born Hispanics of Mexican origin had poorer biological risk profiles in total and within all systems than did Whites; Mexican-born Hispanics had higher total and metabolic risk levels than did Whites. The US-born population of Mexican origin did not differ from Blacks in their total biological and metabolic risk profiles, but they did have lower inflammatory risk than did Blacks. The Mexican-born population had lower mean numbers of total risk factors as well as blood pressure and inflammatory risk factors than did Blacks.

When we controlled for SES, US-born Mexican Americans still had worse total biological risk profiles than did Whites but not Blacks; they also had significantly higher total biological risk than did Mexican-born Mexican Americans. Also after we controlled for SES, those born in Mexico had levels of biological risk factors similar to Whites, in total and in the 3 subsystems.

Differences in Biological Risk and Health Behavior and Access to Care

To determine whether differences in health behaviors or access to health care explained the lower numbers of high-risk biological factors for foreign-born Hispanics and the higher numbers for Blacks and US-born Mexican Americans, we estimated mean differences and controlled for lack of exercise, current smoking status, percentage of diet from fat, and health insurance status (Table 3, model 3). Results did not change much relative to the model that controlled only for SES, suggesting that differences in health behaviors and access to health care cannot explain the differences in biological risk. Mexican-born persons were much less likely to have health insurance than were US-born Mexican Americans (17% vs 45%) and were less likely to exercise (38% vs 58%); by contrast, they were less likely to have a high-fat diet (52% vs 73%) and did not differ in the likelihood of smoking. Our control for the combination of these factors did not affect differences between US-born and foreign-born Mexican Americans.

DISCUSSION

Our goal was to determine whether objective biological measures of health risk profiles would provide evidence of the existence of a Hispanic paradox. However, we did not find a Hispanic paradox in biological risk profiles. The Hispanic population had higher biological risk than did the White population on 3 of 4 scores (total, metabolic, and inflammation risk profiles), which indicated worse biological profiles across a range of physiological systems. The differences existed between Whites and all Hispanics and specifically for those of Mexican heritage. This finding is in line with numerous studies in the literature that have not found lower levels of risk in individual risk factors for Hispanics.30,51-53

Our control for the low SES of the Hispanic population eliminated the differences in

biological risk between the White, total Hispanic, Mexican-origin, and foreign-born (Hispanic and Mexican) populations. When we controlled for low SES, we found that Mexican immigrants had a total biological risk profile similar to Whites and better than US-born Mexican Americans. This is not a Hispanic paradox but rather an indication that the high levels of risk in the foreign-born Mexican population relative to Whites are related to low SES. However, our control for SES did not eliminate differences between Whites and US-born Hispanics of Mexican origin. We were not able to "explain" this higher level of risk among US-born Mexican Americans relative to the White population. It may have arisen from the same social inequities that are at the basis of the differences between Blacks and others. This higher level of risk among US-born Mexican Americans relative to Whites, although not significant in the subcategories, appears to be spread across all 3 categories. When we controlled for SES, health behaviors, and access to care, metabolic differences between Hispanics and Whites disappeared, which suggests that SES factors played a role in metabolic risk differences. Our findings of higher metabolic risk for Mexican Americans are consistent with other studies that found this group to have a higher prevalence of diabetes mellitus. Because diabetes is primarily related to abdominal obesity, public health measures to improve diet and exercise in this group would mean reduction in the morbidity associated with diabetes.

Our study supports other studies that have found that the US-born Mexican American population has higher levels of cardiovascular risk than the foreign-born population.⁵⁴ In our analysis, the difference persisted after we controlled for SES and health behaviors and access to care. The higher level of risk in the US-born population relative to the foreignborn population appears to be unexplained by behavioral and care differences and may be related to initially healthy immigrants or the return migration of the unhealthy. The fact that differences in biological risk profiles by nativity are not as strong as differences in mortality may mean that return migration is more strongly related to serious illness than to the earlier-occurring changes in biological risk factors.¹⁶

Our study and others^{51,55,56} have found that Blacks have higher levels of biologically estimated risk than do Whites, even after we controlled for SES and health behaviors. We also found this to be true for the US-born Mexican American population. The disproportionate burden of biological risk in the Black and USborn Hispanic population may arise from generations of social inequality, which was not captured in our measure of SES.

Overall, our results demonstrate the usefulness of examining objective measures of risk for mortality and other poor outcomes and the importance of addressing differences between subgroups of the Hispanic population.^{1,57} Objective measures of risk for mortality and other poor outcomes show that ethnicity, nativity, and SES contribute to differences in clinically relevant risk factor profiles, which point to the potential for appropriate interventions. In addition, these biological measures may help researchers avoid some of the interpretation issues they face with selfreported and mortality data.^{9,10,13}

Our study had limitations. First, although this analysis adds to the literature by providing information about racial, ethnic, and nativity differences in risk factors for major diseases, disability, and death, we do not know that these risk factors are equally important when it comes to producing these outcomes in all groups. This is an important area for future research. Second, only respondents who had complete data for all 10 biomarkers were included in the study. Those missing data were more likely to be women, older, Black, have lower SES, and have somewhat worse self-reported health than those respondents with complete data. If the sickest individuals were less likely to provide complete data, it is probable that we underestimated the poorer health status of the Black and low-SES populations; therefore, nonresponse is unlikely to change our conclusions. Third, educational information in this sample was limited to 2 categories, rather than a continuous measure of years of education. Having limited categories may have obscured differences in education levels, particularly at low levels of education. Hispanics, particularly foreign-born Hispanics, have lower levels of education relative to both White and Black populations. Finally, because the sample was predominantly Mexican

American, our ability to look at other Hispanic subgroups was limited. Despite these limitations, this study provides evidence that Hispanics are not advantaged in their measured biological risk profiles.

About the Authors

Eileen Crimmins and Jung Ki Kim are with the Andrus Gerontology Center, University of Southern California, Los Angeles. Dawn Alley is with the Robert Wood Johnson Health and Society Scholars Program, University of Pennsylvania, Philadelphia. Arun Karlamangla and Teresa Seeman are with the Division of Geriatrics, School of Medicine, University of California, Los Angeles.

Requests for reprints should be sent to Eileen Crimmins, Andrus Gerontology Center, University of Southern California, Los Angeles, CA 90089-0191 (e-mail: crimmin@usc.edu).

Contributors

E. Crimmins designed the overall study and supervised all aspects of its implementation. J.K. Kim and D. Alley completed the analyses, interpretted findings, and reviewed drafts of the article. A. Karlamangla and T. Seeman interpretted findings and reviewed drafts of the article.

Human Participant Protection

This study was approved by the University of Southern California's University Park Institutional Review Board.

Acknowledgments

This research was supported by the National Institute on Aging (grants R01 AG023347, T32 AG00037, K12AG01004, and P30 AG17265).

References

1. Cho Y, Frisbie WP, Hummer RA, Rogers R. Nativity, duration of residence, and the health of Hispanic adults in the United States. *Int Migration Rev.* 2004; 38:184–211.

2. Elo IT, Turra CM, Kestenbaum B, Ferguson BR. Mortality among elderly Hispanics in the United States: past evidence and new results. *Demography*. 2004;41:109–128.

3. Franzini L, Ribble J, Keddie A. Understanding the Hispanic paradox. *Ethn Dis.* 2001;11:496–518.

4. Hummer RA, Benjamins MR, Rogers RG. Racial and ethnic disparities in health and mortality among the US elderly population. In: Anderson N, Bulato R, Cohen B, eds. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: The National Academies Press; 2004:53–94.

5. Liao Y, Cooper RS, Cao G, et al. Mortality patterns among adult Hispanics: findings from the NHIS, 1986 to 1990. *Am J Public Health*. 1998;88:227–232.

6. Markides KS, Eschbach K. Aging, migration, and mortality: current status of research on the Hispanic paradox. J Gerontol B Psychol Sci Soc Sci. 2005;60:68–75.

7. Adler NE, Boyce T, Chesney MA, et al. Socioeconomic status and health: the challenge of the gradient. *Am Psychol.* 1994;49:15–24.

8. Hunt KJ, Resendez RG, Williams K, Haffner SM, Stern MP, Hazuda HP. All-cause and cardiovascular

mortality among Mexican American and non-Hispanic White older participants in the San Antonio Heart Study–evidence against the "Hispanic paradox." *Am J Epidemiol.* 2003;158:1048–1057.

9. Patel KV, Eschbach K, Ray LA, Markides KS. Evaluation of mortality data for older Mexican Americans: implications for the Hispanic paradox. *Am J Epidemiol.* 2004;159:707–715.

10. Smith DP, Bradshaw BS. Rethinking the Hispanic paradox: death rates and life expectancy for US non-Hispanic White and Hispanic populations. *Am J Public Health.* 2005;96:1686–1692.

 Hajat A, Lucas JB, Kington R. Health outcomes among Hispanic subgroups: data from the National Health Interview Survey, 1992–1995. *Adv Data*. 2000;310:1–14.

12. Markides KS, Coreil J. The health of Hispanics in the southwestern United States: an epidemiologic paradox. *Public Health Rep.* 1986;101:253–265.

 Angel R, Thoits P. The impact of culture on the cognitive structure of illness. *Cult Med Psychiatry*. 1987;11:465–494.

14. Ren X, Amick B. Racial and ethnic disparities in self-assessed health status: evidence from the National Survey of Families and Households. *Ethn Health.* 1996;1:293–303.

 Jasso G, Massey DS, Rosenzweig MR, Smith JP. Immigrant health: selectivity and acculturation. In: Anderson N, Bulato R, Cohen B, eds. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: The National Academies Press; 2004:227–268.

 Palloni A, Arias E. Paradox lost: explaining the Hispanic adult mortality advantage. *Demography*. 2004;41:385–415.

17. Abraido-Lanza AF, Dohrenwend BP, Ng-Mak DS, Turner JB. The Latino mortality paradox: a test of the "salmon bias" and healthy migrant hypotheses. *Am J Public Health.* 1999;89:1543–1548.

18. Sorlie PD, Backlund E, Johnson NJ, Rogot E. Mortality by Hispanic status in the United States. *JAMA*. 1993;270:2464–2468.

 Brunner EJ. Toward a new social biology. In: Berkman LF, Kawachi I, eds. *Social Biology*. New York, NY: Oxford University Press; 2000:306–311.

 Crimmins EM, Seeman T. Integrating biology into the study of health disparities. *Popul Dev Rev.* 2004;30:89–107.

21. Seeman TE, Crimmins E, Huang M, et al. Cumulative biological risk and socio-economic differences in mortality: MacArthur Studies of Successful Aging. *Soc Sci Med.* 2004;58:1965–1997.

22. Fried LP, Kronmal RA, Newman AB, et al. Risk factors for 5-year mortality in older adults: the Cardiovascular Health Study. *JAMA*. 1998;279:585–592.

 Ridker PM, Rifai N, Rose L, Buring JE, Cook NR. Comparison of C-Reactive Protein and low-density lipoprotein cholesterol levels in the prediction of first cardiovascular events. *N Engl J Med.* 2002;347:1557–1565.

24. Seeman TE, Singer B, Rowe J. Exploring a new concept of cumulative biological risk–allostatic load and its health consequences: MacArthur Studies of Successful Aging. *Proc Natl Acad Sci USA*. 2001;98:4770–4775.

25. Reaven GM. Role of insulin resistance in human disease. *Diabetes*. 1998;37:1595–1607.

26. Reynes JF, Lasater TM, Feldman H, Assaf AR, Carleton RA. Education and risk factors for coronary heart disease: results from a New England community. *Am J Prev Med.* 1993;9:365–371.

27. Winkleby MA, Fortmann SP, Barrett DC. Social class disparities in risk factors for disease: eight-year prevalence patterns by level of education. *Prev Med.* 1990;19:1–12.

28. Burt VL, Welton P, Roccella EJ, et al. Prevalence of hypertension in the US adult population: results from the Third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension*. 1995;25:305–313.

29. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988–1994. *Arch Intern Med.* 2003;163:427–436.

 Ford ES, Giles WH, Mokdad AH, Myers GL. Distribution and correlates of C-reactive protein concentrations among adult US women. *Clin Chem.* 2004;50:574–581.

31. Albert MA, Glynn RJ, Buring J, Ridker PM. C-reactive protein levels among women of various ethnic groups living in the United States (from the Women's Health Study). *Am J Cardiol.* 2004;93:1238–1242.

32. Tracy RP, Bovill EG, Fried LP, et al. The distribution of coagulation factors VII and VIII and fibrinogen in adults over 65 years: results from the Cardiovascular Health Study. *Ann Epidem*. 1992;2:509–519.

33. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206–1252.

34. National Cholesterol Education Program (NCEP). Detection, evaluation, and treatment of high blood cholesterol in adults. Available at: http://www.nhlbi. nih.gov/guidelines/cholesterol/atp3xsum.pdf. Accessed March 4, 2004.

35. World Health Organization (WHO) expert consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet.* 2004;363:157–163.

36. United States Preventive Services Task Force (USPSTF). Use of glycated hemoglobin and microalbuminuria in the monitoring of diabetes mellitus. Available at: http://www.ahrq.gov/clinic/epcsums/glycasum. pdf. Accessed March 4, 2004.

37. Ridker PM. C-reactive protein: a simple test to help predict risk of heart attack and stroke. *Circulation*. 2003;108:e81–e85.

38. Palmieri V, Celentano A, Roman M, et al. Relation of fibrinogen to cardiovascular events is independent of preclinical cardiovascular disease: the strong heart study. *Am Heart J.* 2003;145:467–474.

39. Corti M, Guralnik JM, Salive ME, Sorkin JD. Serum albumin level and physical disability as predictors of mortality in older persons. *JAMA*. 1994;272:1036–1042.

40. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *JAMA*. 2003;290:199–206.

41. Brunner EJ, Marmot MG, Nanchahal K, et al. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II Study. *Diabetologia*. 1997;40:1341–1349. 42. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health.* 1992;82:816–820.

43. Winkleby MA, Cubbin C, Ahn DK, Kraemer HC. Pathways by which SES and ethnicity influence cardio-vascular disease risk factors. *Ann NY Acad Sci.* 1999; 896:191–209.

44. Mirowsky J, Ross CE. Education, personal control, lifestyle and health: a human capital hypothesis. *Res Aging*. 1998;20:415–449.

45. Hellenius ML, de Faire U, Berglund B, Hamsten A, Krakau I. Diet and exercise are equally effective in reducing risk for cardiovascular disease. Results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. *Atherosclerosis.* 1993;103:81–91.

46. Koster A, Bosma H, Penninx B, et al. for the Health ABC Study. Association of inflammatory markers with socioeconomic status. *J Gerontol A Bio Sci Med Sci.* 2006;61:284–290.

47. Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. *Soc Sci Med.* 1997;44:809–819.

48. Ockene IS, Miller NH for the American Heart Association Task Force on Risk Reduction. Cigarette smoking, cardiovascular disease, and stroke. *Circulation*. 1997;96:3243–3247.

49. Crimmins EM, Hayward MD, Seeman TE. Race/ ethnicity, socioeconomic status and health. In: Anderson N, Bulato R, Cohen B, eds. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: The National Academies Press; 2004: 310–352.

50. Seeman TE, Singer BH, Ryff CD, Love GD, Levy-Storms L. Social relationships, gender, and allostatic load across two age cohorts. *Psychosom Med.* 2002;64:395–406.

51. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005;15:1233–1241.

52. Mitchell B, Stern M, Haffner S, Hazuda H, Patterson J. Risk factors for cardiovascular mortality in Mexican Americans and non-Hispanic Whites. San Antonio Heart Study. *Am J Epidemiol.* 1990;131:423–433.

53. Ogden C, Carroll M, Curtin L, McDowell M, Tabak C, Flegal K. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006; 295:1549–1555.

54. Sundquist J, Winkleby M. Cardiovascular risk factors in Mexican American adults: a transcultural analysis of NHANES III, 1988–1994. *Am J Public Health*. 1999;89:723–730.

55. Geronimus AT, Hicken M, Keen D, Bound J. "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United States. *Am J Public Health.* 2006;96:826–833.

 Winkleby M, Kraemer H, Ahn D, Varady A. Ethnic and socioeconomic differences in cardiovascular disease risk factors. *JAMA*. 1998;280:356–362.

57. Borrell L. Racial identity among Hispanics: implications for health and well-being. *Am J Public Health*. 2005;3:379–381.