Hispanic Mortality Paradox: A Systematic Review and Meta-Analysis of the Longitudinal Literature

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To investigate the possibility of a Hispanic mortality advantage, we conducted a systematic review and meta-analysis of the published longitudinal literature reporting Hispanic individuals' mortality from any cause compared with any other race/ethnicity. We searched MEDLINE, PubMed, EMBASE, HealthSTAR, and PsycINFO for published literature from January 1990 to July 2010.

Across 58 studies (4 615 747 participants), Hispanic populations had a 17.5% lower risk of mortality compared with other racial groups (odds ratio = 0.825; $P < .001$; 95% confidence interval = 0.75, 0.91). The difference in mortality risk was greater among older populations and varied by preexisting health conditions, with effects apparent for initially healthy samples and those with cardiovascular diseases. The results also differed by racial group: Hispanics had lower overall risk of mortality than did non-Hispanic Whites and non-Hispanic Blacks, but overall higher risk of mortality than did Asian Americans.

These findings provided strong evidence of a Hispanic mortality advantage, with implications for conceptualizing and addressing racial/ethnic health disparities. (*Am J Public Health.* 2013;103:e52–e60. doi:10.2105/AJPH.2012.301103)

DESPITE A SIGNIFICANTLY more disadvantaged risk factor profile, Hispanics in the United States often experience similar or better health outcomes across a range of health and disease contexts compared with non-Hispanic Whites (NHWs), an epidemiological phenomenon commonly referred to as the "Hispanic paradox." Among the most salient features of this advantage is evidence that Hispanics appear to live longer than NHWs.[1–3] These findings are largely based on national cohort data, with mortality data from the US Vital Statistics System used in the numerator and population counts from the US Census used in the denominator, yielding a death rate statistic. The classic explanations for these paradoxical findings suggest that either the denominator is artificially low because of Hispanics returning to their countries of origin before death (the "salmon bias hypotheses") or that the numerator is not representative due to the healthiest Hispanics migrating to the United States (the "healthy migrant hypothesis"). These hypotheses have been largely refuted.[4] The contemporary overarching concern is that the statistical estimation approach remains flawed because of underreporting of ethnicity on death certificates. Despite recent data suggesting that the associated error is negligible,[5,6] the validity of the paradox remains in question due to its strong ties to this methodology.

One solution to these issues is to examine longitudinal studies in which race and ethnicity are assessed at study entry and participants are followed longitudinally to mortality. This literature has added a wealth of data for and against a Hispanic mortality advantage, but has failed to clarify the overall relationship. A number of factors impede consensus, including differences in sample size, selection criteria, methodologies, follow-up time, statistical reporting, and outcomes (i.e., morbidity, specific-cause mortality, all-cause mortality). In addition, at least 5 narrative literature reviews of the associated data[7–11] were published in the last decade, asserting the level of interest but failing to provide an empirical test (e.g., meta-analysis) to clarify the discrepancy. Hence, the current status of the Hispanic mortality paradox can best be described as one of great interest with significant logistical confusion.

We systematically reviewed the longitudinal literature, comparing Hispanic mortality rates with those of other racial/ethnic groups and conducted a meta-analysis of the available data as a definitive test of whether there is a relative Hispanic mortality advantage. Resolving the validity of the phenomenon would facilitate future research efforts to identify contributing resilience factors that might lead to targeted interventions. In the present study, we focused on all-cause mortality (death from any cause) as the primary dependent variable and evaluated mortality within specific disease contexts to the extent that sufficient data were available. We improved on previous reviews by using meta-analytic procedures that took into account the differences in available studies regarding sample size, participant characteristics, selection criteria, and outcomes.

METHODS

Studies were identified through 2 techniques. First, we conducted extensive electronic database searches from January 1990 to July 2010, using MEDLINE, PubMed, EMBASE, HealthSTAR, and PsycINFO. January 1990 was used as the beginning search date because of methodological changes in the use of the terms such as Hispanic in race and ethnicity data collection and publication efforts.[12,13] To capture the broadest possible sample of relevant articles, 3 search term categories were used: (1) Hispanic (Hispanic, Latino, Mexican, Puerto Rican, Cuban), (2) mortality (mortality, death, longevity, survival, life span), and (3) design (prospective, longitudinal). Second, we manually examined the reference sections of past reviews and of studies meeting the inclusion criteria to locate articles not identified in the database searches.

Inclusion Criteria

We included only published studies meeting the following criteria in the meta-analysis: (1) written in English or Spanish, (2) used a longitudinal design, and (3) provided quantitative data regarding Hispanic mortality at the

individual level compared with that of other racial/ethnic groups.

We excluded studies in which the outcome was not explicitly stated as mortality (e.g., combined outcomes of morbidity and mortality), studies of infant mortality, single-case designs, and reports with exclusively aggregated data (e.g., census-level statistics). We included all other types of quantitative research designs that were longitudinal and yielded a statistical estimate of the risk of mortality among Hispanic populations compared with that of other racial/ethnic groups. There were no age limitations other than those related to studies of infant mortality. However, the published literature on mortality was largely skewed toward older ages, as reflected here.

Data Abstraction

Articles were independently coded by 2 teams with 2 members each. A third independent member then compared the 2 ratings, resolving discrepancies through joint review with the teams. Coders extracted several objectively verifiable characteristics of the studies: (1) the number of participants and their composition by age, ethnicity, gender, and preexisting health conditions (if any), as well as the cause of mortality; (2) length of follow-up; and (3) research design. Given the substantial heterogeneity among Hispanic peoples exemplified by differences in culture, traditions, and importantly, health outcomes, we further sought to code by country of origin or nativity when such data were available.

Data within studies were often reported in terms of odds ratios (ORs), the likelihood of mortality contrasted by ethnic group. Because OR values cannot be meaningfully aggregated, all effect sizes reported within studies were transformed to the natural log ORs for analyses and then transformed back to ORs for interpretation. When effect size data were reported in any metric other than ORs or the natural log ORs, we transformed those values using statistical software programs and macros (*Comprehensive Meta-Analysis*[14]). In many cases, we calculated effect sizes from frequency data in matrixes of mortality status by ethnicity. In cases when frequency data were not reported, we recovered the cell probabilities from the reported risk ratio and marginal probabilities. Across studies, we assigned OR values less than 1.00 to data indicative of decreased mortality among Hispanics and OR values greater than 1.00 to data indicative of increased mortality among Hispanics relative to the comparison group(s).

When multiple effect sizes were reported within a study at the same time, we averaged the values (weighted by SE) to avoid violating the assumption of independent samples. When a study contained multiple effect sizes across time, we extracted the data from the longest follow-up period. If a study used statistical controls in calculating an effect size, we extracted the data from the model utilizing the fewest statistical controls. We coded the research design used rather than the estimate risk of individual study bias. The coding protocol is available from the authors.

Information obtained from the studies was extracted directly from the reports. As a result, the interrater agreement was high for categorical variables (mean Cohen's $\kappa = 0.97$; SD = 0.02) and for continuous variables (mean intraclass correlation = 0.93; SD = 0.14). Discrepancies across coders were resolved through further scrutiny of the article until consensus was obtained.

Aggregate effect sizes were calculated using random effects models following confirmation of heterogeneity. A random effects approach yields results that generalize beyond the sample of studies actually reviewed.[15] We assumed that the results would differ as a function of participant characteristics (i.e., age, gender) and study design (i.e., length of follow-up). Random effects models take this between-studies variation into account, whereas fixed effects models do not.[16]

RESULTS

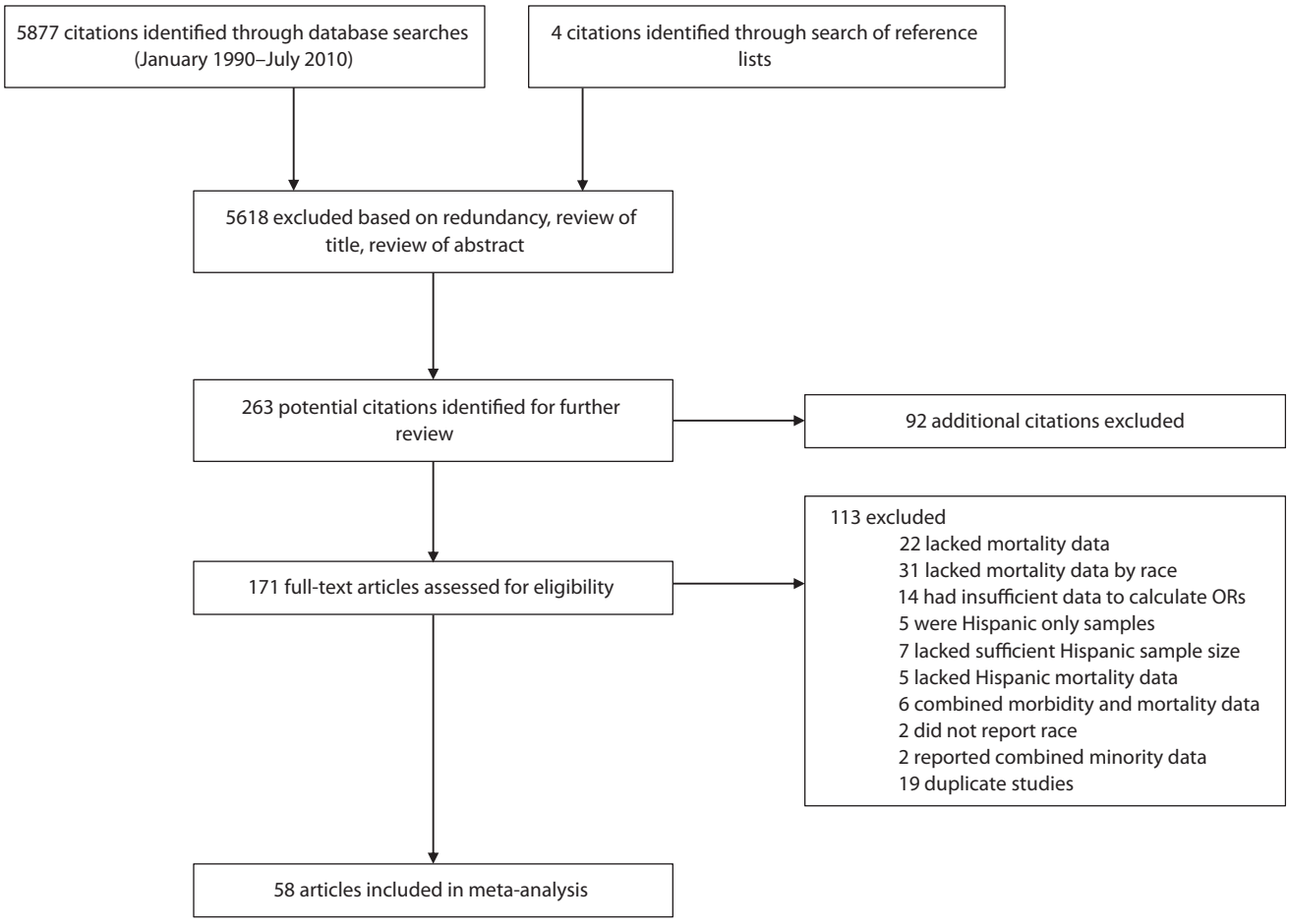
Figure 1 shows the study selection process. Statistically nonredundant effect sizes were extracted from 58 studies (Table 1).[17–74] Data were reported from 4 615 747 total participants, with an average composition of 26% Hispanic participants within studies. The mean ages of participants at initial evaluation were 54.6 years (SD = 11.6) for Hispanics and 56.1 years (SD = 11.7) for comparison groups. Hispanic participants consisted of 44% women, and comparison groups included 45% women.

Research reports typically failed to describe the specific ethnic heritage of the Hispanic participants (80% omitted this information), but 8 studies (15%) were specific to Mexican Americans,[20,29,33,42,47,52,67,72] 1 study was specific to Puerto Rican Americans,[48] and 5 studies (9%) involved participants from a variety of ethnic backgrounds.[24,25,31,36,51] Several studies (22%) involved initially healthy participants, but 24% of studies involved patients with cardiovascular disease (CVD), 12% with cancer, 10% with HIV infection, 7% with diabetes, 5% with renal disease, and the remaining 20% with a variety of conditions, including liver disease and dementia. Research reports most often (91%) considered all-cause mortality, but some restricted evaluations to mortality associated with CVD (5%) or other specific causes (4%). Only 8 studies (14%) involved a medical intervention[21,24,25,33,35,45,62,73]; most merely tracked participants' mortality over time. Participants were followed for an average of 6.9 years (SD = 5.9; range = 1 month to 33 years). Preferred Reporting Items for Systematic Reviews and Meta-Analyses and Meta-analysis of Observational Studies in Epidemiology guidelines were adhered to in the design and reporting of this study.[75,76]

Omnibus Analysis

Across the 58 studies, the random effects weighted average effect size was OR = 0.825 ($P < .001$; 95% confidence interval [CI] = 0.75, 0.91). Consistent with the hypothesis, Hispanic ethnicity was associated with a 17.5% mortality advantage.

As shown in Figure 2, ORs ranged from 0.39 to 2.75, with a very large degree of heterogeneity across studies ($I^2 = 96\%$; $Q_{(57)} = 1564$; $P < .001$; $\tau^2 = 0.12$), suggesting that systematic effect size variability was unaccounted for. Thus, it was likely that factors associated with the studies themselves (e.g., publication status), participant characteristics (e.g., age, health status), and, or the research design (e.g., length of follow-up) might have moderated the overall results. We therefore conducted additional analyses to determine the extent to which the variability in the effect sizes was moderated by these variables.



Note. OR = odds ratio.

FIGURE 1—Selection of articles for meta-analysis: 1990 to 2010.

Evaluation for Publication Bias

To assess the possibility of publication bias,[77] we conducted 4 analyses. First, we calculated Orwin's fail-safe *N*,[78] the theoretical number of unpublished studies with effect sizes averaging zero (no effect) that would need to be located to reduce the overall magnitude of the results obtained to a trivial estimate of $1.0 > OR > 0.95$. Based on this calculation, at least 367 additional studies averaging OR = 1.0 would need to be found to render the results of the present meta-analysis as negligible. Second, we utilized both Egger's regression test[79] and the alternative to that test recommended by Peters et al.,[80] which is better suited to data in OR format. The results of these 2 analyses failed to reach statistical significance ($P > .05$). Third, we generated a "funnel plot"[81] of the studies' log ORs by the SEs. The data obtained from this meta-analysis were not symmetrically distributed around the grand mean; there appeared to be multiple studies "missing" from the bottom left corner of the distribution. However, these studies were in the opposite corner from what would have been expected. Typically, "missing" studies were in the region of nonsignificance if publication bias was present. In this case, the data underrepresented studies with relatively fewer participants that demonstrated lower mortality rates among Hispanics. Finally, we employed the "trim and fill" methodology described by Duval and Tweedie.[82,83] This analysis indicated that when 14 estimated "missing" studies were included in the analysis, the overall effect size was calculated to be OR = 0.70 (95% CI = 0.64, 0.77), indicating that Hispanic participants were 30% less likely to die than were comparison group members over the same time period.

Based on these 4 analyses, we concluded that the data did not reflect publication bias per se, but that they might represent a conservative estimate of risk for mortality among Hispanic populations.

Moderation by Participant and Study Characteristics

To investigate whether the lower risk of mortality among Hispanic populations varied as a function of participant characteristics within studies, we conducted analyses involving participants' age, gender, and preexisting diagnoses. We also investigated

TABLE 1—Characteristics of Included Studies: 1990–2010

Source	Total, No.	Hispanic, No. (%)	Female, %	Mean Age, Years	Follow-Up, Years	Health Status at Study Entry	Analysis Category
Alexander et al.[17]	90 316	9835 (11)	55	69	1	CVD	CVD
Assassi et al.[18]	250	71 (28)	87	47	6	Scleroderma	Other
Brogan et al.[19]	1027	31 (3)	35	35	< 1	Respiratory failure	Other
Brown et al.[20]	327	125 (38)	38	37	5	HIV/AIDS	HIV/AIDS
Bush et al.[21]	2486	92 (4)	40	65	5	CVD	CVD
Chen et al.[22]	281	100 (36)	19	59	3	Cancer	Cancer
Cohen et al.[23]	15 610	2600 (17)	17	36	3	HIV/AIDS	HIV/AIDS
Cohen et al.[24]	27 788	734 (3)	26	59	< 1	CVD	CVD
Cooper-Dehoff et al.[25]	22 576	8045 (36)	61	66	3	CVD	CVD
Cromwell et al.[26]	692 574	9868 (1)	NA	> 65	1	CVD	CVD
Cunningham et al.[27]	200	36 (18)	5	38	6	HIV/AIDS	HIV/AIDS
Echols et al.[28]	7007	344 (5)	38	63	1	CVD	CVD
Eden et al.[29]	107	64 (60)	73	62	7	Stroke	Other
Feinglass et al.[30]	25 568	3628 (14)	44	72	5	Extremity bypass	Other
Fernandez et al.[31]	396	220 (56)	86	35	10	Autoimmune	Other
Frankenfield et al.[32]	7723	994 (13)	46	59	1	Kidney disease	Other
Freedman et al.[33]	15 329	970 (6)	55	44	12	Cancer	Cancer
Gomez et al.[34]	41 901	2061 (5)	50	> 65	7	Cancer	Cancer
Gortmaker et al.[35]	1028	358 (35)	50	7	4	HIV/AIDS	HIV/AIDS
Hartmann et al.[36]	980	483 (41)	50	66	5	Stroke	Other
Harzke et al.[37]	1 238 317	311 082 (25)	0	28	5	None apparent	None/community
Havranek et al.[38]	7495	1789 (24)	49	56	< 1	CVD	CVD
Helzner et al.[39]	323	179 (55)	70	87	4	Dementia	Other
Henderson et al.[40]	71 798	41 665 (58)	52	63	6	None apparent	None/community
Jokela et al.[41]	8544	1736 (20)	50	20	25	None apparent	None/community
Lee et al.[42]	446	312 (70)	61	> 60	8	None apparent	None/community
Liao et al.[43]	696 697	52 725 (8)	53	38	9	None apparent	None/community
Lin et al.[44]	553 307	33 954 (6)	54	> 25	11	None apparent	None/community
Mak et al.[45]	15 376	1613 (10)	34	64	3	CVD	CVD
Manoharan et al.[46]	400	67 (17)	33	67	14	Cancer	Cancer
Medina et al.[47]	584	236 (40)	60	62	4	Diabetes	Other
Mendenhall et al.[48]	428	63 (15)	0	49	5	Liver Disease	Other
Murthy et al.[49]	100 618	10 393 (10)	47	59	2	Kidney disease	Other
Ostir et al.[50]	506	153 (30)	51	81	5	None apparent	None/community
Palmas et al.[51]	1178	451 (38)	55	72	7	Diabetes	Other
Patel et al.[52]	66 397	1114 (2)	56	73	8	None apparent	None/community
Peralta et al.[53]	39 550	12 076 (31)	59	62	4	Kidney disease	Other
Perez E et al.[54]	312	91 (29)	46	58	20	Cancer	Cancer
Perez M et al.[55]	44 171	2625 (6)	9	54	8	CVD	CVD
Plurad et al.[56]	3998	2495 (62)	18	33	7	Sepsis	Other
Robinson et al.[57]	6677	673 (10)	45	57	5	Kidney disease	Other
Sacco et al.[58]	394	82 (21)	51	63	1	Stroke	Other
Sacco et al.[59]	2670	1443 (54)	63	66	9	None apparent	None/community
Schupf et al.[60]	2247	876 (39)	66	76	3	None apparent	None/community
Segev et al.[61]	79 034	9846 (12)	59	39	6	None apparent	None/community
Serna et al.[62]	5122	413 (8)	41	NA	5	Cancer	Cancer

Continued

any differences across studies that may be due to length of follow-up, type of research design, and cause of mortality.

To establish whether the average age of the sample accounted for significant between-studies variance, the effect sizes from the 53 studies that reported participants' average age at intake were correlated with the corresponding effect size for that study. The resulting random effects weighted correlation was –0.28 ($P = .03$), indicating that studies with older populations tended to demonstrate lower risk of mortality among Hispanic participants relative to comparison groups. As a first step to verify that this association was specific to chronological age, we investigated the possible confounding association with trends over time. However, when we correlated the effect sizes with the year of initial data collection and with a variable created by subtracting the average age of participants at the start of the study from the year of initial data collection (an estimate of the average year of participant birth), the resulting values of $r = –0.08$ and $r = 0.22$ did not approach statistical significance ($P > .1$). Thus, the findings within studies did not consistently change over time. Because older populations were more likely to receive treatment than were younger populations, we conducted a second analysis to verify the association observed with participant age by simultaneously regressing participant age and the type of research study (intervention vs observation) on study effect size. In this model, the average age of participants remained statistically significant ($b = –0.28$, $P = .04$), but the type of research study (intervention vs observation) did not. The differences observed in risk for

TABLE 1—*Continued*

Shaw et al.[63]	346 075	7823 (2)	47	61	< 1	CVD	CVD
Silverberg et al.[64]	4787	661 (14)	10	37	9	HIV/AIDS	HIV/AIDS
Smyth et al.[65]	581	323 (56)	0	25	33	Heroin addiction	Other
Stefanidis et al.[66]	408	296 (73)	44	54	16	Cancer	Cancer
Steffen-Batey et al.[67]	406	196 (48)	41	59	7	CVD	CVD
Sudano et al.[68]	8400	723 (9)	52	56	6	None apparent	None/community
Swenson et al.[69]	1862	921 (49)	57	52	11	Diabetes	Other
Tedaldi et al.[70]	1301	225 (17)	20	38	5	HIV/AIDS	HIV/AIDS
Waring et al.[71]	956	37 (4)	73	72	13	Dementia	Other
Wei et al.[72]	3735	2630 (70)	59	43	8	None apparent	None/community
Wolf et al.[73]	9303	979 (11)	44	61	1	Kidney disease	Other
Young et al.[74]	337 870	26 544 (8)	1	64	2	Diabetes	Other

Note. CVD = cardiovascular disease; NA = not available.

mortality appeared to be moderated by participant age.

Similar random effects weighted correlations with the gender composition of each sample (using percentage who were female; $r = -0.23$) and the length of time participants were followed ($r = 0.07$) did not reach statistical significance ($P > .05$). Furthermore, no differences in the average effect sizes were found between studies using prospective versus retrospective designs ($Q_{1,57} = 0.1$; $P > .05$). Studies evaluating all-cause mortality had effect sizes of equivalent magnitude to those from the studies in which a specific cause of death was evaluated (i.e., cancer; $Q = 0.3$; $P > .05$). Thus, the omnibus results presented earlier were not moderated by these variables.

As shown in Table 2, statistically significant differences were found across participants' type of health condition at the point of initial evaluation ($Q = 11.5$; $P = .02$). Community samples of Hispanics with no identified health impairment had the greatest mortality advantage (estimated 30%) relative to non-Hispanics. Hispanic ethnicity was also associated with a 25% reduced mortality advantage among individuals with CVD and an estimated 16% advantage among persons with a variety of other preexisting health conditions. However, Hispanics diagnosed with HIV/AIDS or cancer had a risk of mortality that did not significantly differ from non-Hispanics.

Because studies compared Hispanic participants with different ethnic groups, we conducted a random effects weighted analysis of variance across the several comparisons conducted within studies (such that each study contributed as many effect sizes as it had unique comparisons with different ethnic groups[84]). As shown in Table 3, there was a significant difference across ethnicity ($Q = 6.5$; $P < .05$). Hispanic participants were less likely to die over time compared with both NHWs and non-Hispanic Blacks (NHBs), but they were more likely to die than were Asian Americans during the same follow-up period.

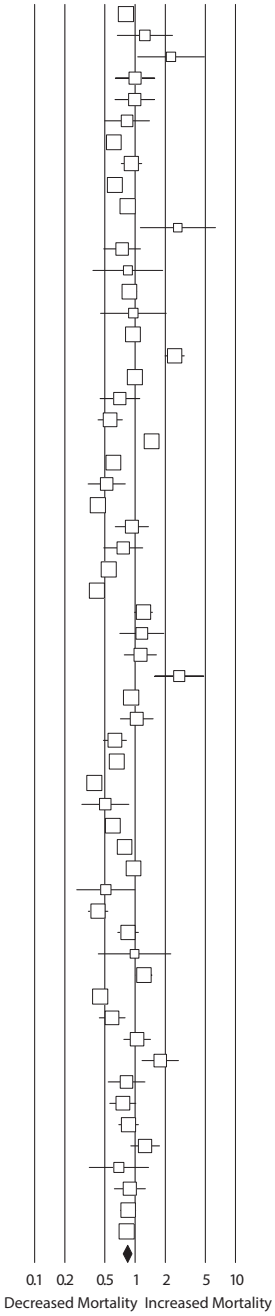
DISCUSSION

Results of this meta-analysis showed that Hispanic ethnicity was associated with a 17.5% lower mortality rate relative to non-Hispanics, a rate that was highly comparable to the 20% advantage reported by Arias et al.[5] using the alternative death statistic estimation strategy. The omnibus finding in the present study was moderated by age, such that the effect became stronger among older participants, a finding similar to that which was recently reported using the estimation approach.[85] However, the date of data collection did not moderate the effect, suggesting that the trajectory of this mortality effect did not change (i.e., weaken) over time. The Hispanic mortality advantage varied as a function of preexisting health status at study entry. Specifically, Hispanics displayed a significant mortality advantage among studies of initially healthy samples and in the context of CVD and other health conditions, such as renal disease. With respect to studies of persons with cancer and HIV/AIDS, Hispanics and non-Hispanics experienced equivalent mortality risk. Findings also indicated that although Hispanics had a significant overall mortality advantage relative to NHWs and NHBs, they were marginally disadvantaged relative to Asian Americans.

When considered along with the consistent state and national vital statistics evidence, including the recent Centers for Disease Control and Prevention report clearly stating a Hispanic ethnicity mortality advantage,[3] it might be time to move beyond the question of the existence of the Hispanic mortality paradox and onto investigations into the causes of such resilience. An important conceptual consideration was that the observed mortality advantage, as well as the broader health outcome advantages evident in the Hispanic paradox, may reflect resilience at several points in the course of disease. Hispanics might be less susceptible than some other races to illness in general or to specific conditions with high mortality rates, such as CVD. It was also possible that the rate of disease progression might be slower among Hispanics, resulting in lower morbidity and greater longevity. Finally, the mortality advantage might reflect an advantage in survival and recovery from acute clinical events (e.g., myocardial infarction, stroke). Hence, further research is needed to ascertain whether the observed Hispanic mortality advantage reflects advantages at specific points in the disease course and whether such time-point differences vary by disease context.

Several risk and resilience factors might contribute to these effects, including potential biological (e.g., genetics, immune functioning), behavioral (e.g., diet, smoking), psychological (e.g., stress, personality), and social (e.g., acculturation, social cohesion) differences.[86] Although not assessed in the present study, lower socioeconomic status (SES) is a robust predictor of worse health outcomes.[87] However, the present findings challenged the

Study Name	OR	Lower limit	Upper limit	z-Value	P-Value
Alexander et al.[17]	0.811	0.774	0.850	-8.708	<.001
Assassi et al.[18]	1.252	0.652	2.405	0.676	.499
Brogan et al.[19]	2.282	1.040	5.008	2.057	.04
Brown et al.[20]	1.000	0.622	1.607	0.000	>.999
Bush et al.[21]	0.992	0.617	1.594	-0.033	.974
Chen et al.[22]	0.834	0.489	1.421	-0.669	.503
Cohen et al.[23]	0.615	0.524	0.722	-5.927	<.001
Cohen et al.[24]	0.920	0.713	1.187	-0.638	.523
Cooper-Dehoff et al.[25]	0.629	0.564	0.702	-8.268	<.001
Cromwell et al.[26]	0.845	0.807	0.883	-7.348	<.001
Cunningham et al.[27]	2.670	1.103	6.462	2.177	.029
Echols et al.[28]	0.743	0.478	1.155	-1.320	.187
Eden et al.[29]	0.849	0.374	1.926	-0.392	.695
Feinglass et al.[30]	0.880	0.730	1.060	-1.347	.178
Fernandez et al.[31]	0.961	0.443	2.084	-0.101	.919
Frankenfield et al.[32]	0.951	0.804	1.126	-0.581	.561
Freedman et al.[33]	2.477	1.950	3.146	7.434	<.001
Gomez et al.[34]	0.997	0.902	1.102	-0.059	.953
Gortmaker et al.[35]	0.706	0.439	1.137	-1.432	.152
Hartmann et al.[36]	0.564	0.418	0.762	-3.739	<.001
Harzke et al.[37]	1.462	1.255	1.704	4.872	<.001
Havranek et al.[38]	0.609	0.526	0.705	-6.613	<.001
Helzner et al.[39]	0.521	0.333	0.815	-2.860	.004
Henderson et al.[40]	0.426	0.400	0.454	-26.656	<.001
Jokela et al.[41]	0.930	0.622	1.389	-0.356	.722
Lee et al.[42]	0.763	0.478	1.219	-1.130	.259
Liao et al.[43]	0.548	0.518	0.579	-21.500	<.001
Lin et al.[44]	0.418	0.391	0.446	-25.676	<.001
Mak et al.[45]	1.214	0.969	1.521	1.687	.092
Manoharan et al.[46]	1.169	0.691	1.976	0.582	.561
Medina et al.[47]	1.131	0.766	1.670	0.618	.537
Mendenhall et al.[48]	2.751	1.534	4.934	3.396	.001
Murthy et al.[49]	0.915	0.869	0.963	-3.423	.001
Ostir et al.[50]	1.039	0.699	1.543	0.188	.851
Palmas et al.[51]	0.630	0.473	0.839	-3.164	.002
Patel et al.[52]	0.658	0.584	0.742	-6.852	<.001
Peralta et al.[53]	0.393	0.364	0.423	-24.605	<.001
Perez E et al.[54]	0.506	0.289	0.886	-2.385	.017
Perez M et al.[55]	0.602	0.512	0.707	-6.195	<.001
Plurad et al.[56]	0.786	0.655	0.943	-2.591	.010
Robinson et al.[57]	0.965	0.810	1.148	-0.404	.686
Sacco et al.[58]	0.511	0.257	1.017	1.912	.056
Sacco et al.[59]	0.428	0.334	0.548	-6.738	<.001
Schupf et al.[60]	0.852	0.659	1.102	-1.221	.222
Segev et al.[61]	0.989	0.422	2.320	-0.025	.98
Serna et al.[62]	1.225	1.001	1.499	1.971	.049
Shaw et al.[63]	0.450	0.413	0.490	-18.410	<.001
Silverberg et al.[64]	0.590	0.430	0.811	-3.253	.001
Smyth et al.[65]	1.048	0.756	1.454	0.281	.778
Stefanidis et al.[66]	1.779	1.147	2.759	2.571	.01
Steffen-Batey et al.[67]	0.822	0.528	1.280	-0.867	.386
Sudano et al.[68]	0.756	0.549	1.040	-1.718	.086
Swenson et al.[69]	0.862	0.675	1.102	-1.184	.236
Tedaldi et al.[70]	1.259	0.886	1.788	1.285	.199
Waring et al.[71]	0.691	0.342	1.393	-1.034	.301
Wei et al.[72]	0.887	0.609	1.292	-0.625	.532
Wolf et al.[73]	0.854	0.703	1.037	-1.596	.11
Young et al.[74]	0.824	0.780	0.871	-6.893	<.001
Combined	0.825	0.746	0.912	-3.767	<.001



Note. CI = confidence interval; OR = odds ratio.

FIGURE 2—Meta-analysis of Hispanic ethnicity and all-cause mortality: 1990–2010.

generalizability of this relationship given the typically lower SES of Hispanics relative to NHWs. It is possible that SES either does not contribute to risk among Hispanics or confers risk only as moderated by some third variable. For example, emerging data suggested that acculturation moderates the relationship between SES and disease risk among Hispanics, such that there is a buffering effect of SES associated with low levels of acculturation and a more traditional SES gradient effect at higher acculturation levels.[88] Acculturation might be a proxy for social behaviors and cultural values that buffer against the stress of economic and environmental disadvantages. It was also possible that the relative impact of traditional risk factors, such as diabetes and lipids, differ by ethnicity and contribute to the observed paradox. More research is needed to identify risk and resilience mechanisms as well as to understand potentially complex interaction patterns that may explain the observed effects.

The present study is a reminder to physicians and researchers about the heterogeneity in racial/ethnic minority health. Despite similar risk factor profiles, Hispanics had significantly lower all-cause mortality relative to NHBs. Such findings support a need for Hispanic-specific comparative research to determine where such differences occur in specific disease courses and outcomes and to investigate potential racial and ethnic differences in the relative weight or influence of identified risk factors for disease. Given evidence of Hispanic heterogeneity in health outcomes, subgroup comparative research is also warranted.

Limitations

We could not entirely rule out the possibility of selection bias as an alternative explanation for the findings. Although we made significant efforts to identify all relevant published studies, and data checks indicated no significant violations of publication distribution, our results might yet reflect

TABLE 2—Analyses of Weighted Average Effect Sizes Across Type of Preexisting Health Condition: 1990–2010

Type of Health Condition[a]	Studies, No.	OR (95% CI)
None apparent (community samples)	13	0.70 (0.58, 0.85)
Cardiovascular disease	11	0.75 (0.61, 0.91)
Cancer	7	1.21 (0.92, 1.59)
HIV/AIDS	6	0.86 (0.64, 1.17)
Other conditions	21	0.84 (0.72, 0.99)

Note. CI = confidence interval; OR = odds ratio, transformed from random effects weighted natural log OR; Q_b = Q-value for variance between groups.
[a]$Q_b = 115$; $P = .02$.

some degree of bias. For example, limiting inclusion to only those studies in English or Spanish might have resulted in a language bias. The number of available studies also limited our ability to examine mortality in specific contexts, including diabetes, autoimmune conditions, injury, neurologic disorders, and others, as well as test effects of acculturation or generational status. We were also unable to address questions regarding whether the observed effect was constant or decreased over time. Study availability might also have limited our ability to detect subtle effects, as in the context of cancer and HIV, where observed effects might have been significant with a larger number of studies. Lack of reporting also limited our ability to examine several key moderators, including SES and health behaviors, which were shown to influence outcomes.[89] To these points, we would note that we did not examine unpublished manuscripts that could also affect findings. Finally, the analyzed sample was predominantly Mexican American, which likely limited generalizability across Hispanic subgroups, particularly given evidence of significant heterogeneity in Hispanic subgroup mortality outcomes.[90,91]

Conclusions

These findings should serve as a cornerstone to document a comparative Hispanic mortality advantage in the context of a disadvantaged risk factor profile and to demonstrate important heterogeneity in racial/ethnic minority health. Furthermore, these findings highlighted the need for specific comparative studies involving Hispanics as opposed to generalizing findings of Black-White differences. A next challenge is to identify factors that promote resilience across the life span, and in turn, have the potential for informing interventions for all. ■

TABLE 3—Odds of Survival by Race Compared With Hispanics: 1990–2010

Race[a]	Studies, No.	OR (95% CI)
Non-Hispanic Black	40	0.87 (0.76, 0.99)
Asian	9	1.19 (0.90, 1.56)
Non-Hispanic White	53	0.81 (0.73, 0.91)

Note. CI = confidence interval; OR = odds ratio, transformed from random effects weighted natural log OR; Q_b = Q-value for variance between groups.
[a]$Q_b = 6.5$; $P = .04$.

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Contributors

J. M. Ruiz had the idea for the project. J. M. Ruiz, P. Steffen, and T. B. Smith conceptualized the study design. J. M. Ruiz and P. Steffen oversaw the literature review and article procurement. P. Steffen and T. B. Smith oversaw data extraction. T. B. Smith conducted the statistical analyses. J. M. Ruiz, P. Steffen, and T. B. Smith drafted the article. J. M. Ruiz, P. Steffen, and T. B. Smith had full access to all the data in the meta-analysis and take responsibility for the integrity of the data and the accuracy of the data analysis.

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Human Participant Protection

No protocol approval was necessary because data were obtained from secondary sources. After consultation with our respective institutional review boards, approval was not sought given the nature of the study and its use of published, de-identified data.

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