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

Objectives: The current objective was to compare Hispanic mortality rates to those of other racial/ethnic groups in order to investigate the possibility of a Hispanic mortality advantage.

Methods: We conducted a systematic review and meta-analysis of the published longitudinal literature reporting Hispanic individuals’ mortality of any cause compared with that of any other racial/ethnic group.

Results: Across 58 studies (4,615,747 participants), the random effects weighted average effect size was $OR = 0.825$ ($P < .001$, 95% CI = 0.75 to 0.91), corresponding to a 17.5% lower risk of mortality among Hispanic populations compared to other racial groups. The difference in mortality risk tended to be greater among older populations and varied as a function of pre-existing health condition, with effects apparent for initially healthy samples and for those with cardiovascular diseases. The results also differed by racial group comparison: Hispanics had lower overall risk for mortality than non-Hispanic Whites and non-Hispanic Blacks, but overall higher risk for mortality than Asian Americans.

Conclusions: These findings provide evidence of a small Hispanic mortality advantage, with implications for conceptualizing and addressing racial/ethnic health disparities.
INTRODUCTION

Despite a significantly worse risk factor profile, Hispanics in the U.S. often experience similar or better health outcomes across a range of health and disease contexts compared to non-Hispanic Whites (NHW), an epidemiological phenomenon commonly referred to as the “Hispanic Paradox”. Amongst the most salient features of this advantage is evidence that Hispanics appear to live longer than NHW.\textsuperscript{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 due to Hispanics returning to their countries of origin prior to death (i.e., the “Salmon bias hypotheses”) or that the numerator is not representative due to the healthiest Hispanics migrating to the U.S. (i.e., the “Healthy migrant hypothesis”) have been largely refuted.\textsuperscript{4} The contemporary overarching concern is that the statistical estimation approach remains flawed due to underreporting of ethnicity on death certificates. Despite recent data suggesting that the associated error is negligible,\textsuperscript{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 five narrative literature reviews of the associated data\textsuperscript{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.

The current objective was to systematically review the longitudinal literature comparing Hispanic mortality rates to those of other racial/ethnic groups and to conduct 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 will facilitate future research efforts to identify contributing resilience factors which may 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 improve on prior reviews by using meta-analytic procedures that take into account the differences in available studies regarding sample size, participant characteristics, selection criteria, and methodology.

**METHODS**

**Ethics Statement**

Following 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.

**Identification of Studies**

Studies were identified through two techniques. First, we conducted extensive electronic database searches for the time frame of January 1990 to July 2010, using Medline, PubMed, EMBASE, HealthSTAR, and PsycINFO. January 1990 was used as the beginning search date due to 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 Hispanic Paradox
articles, three search term categories were used: (1) Hispanic (*Hispanic, Latino, Mexican, Puerto Rican, Cuban*); (2) Mortality (*mortality, death, longevity, survival, lifespan*); (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**

In the meta-analysis we included only published studies meeting the following criteria: (1) written in English or Spanish, (2) used a longitudinal design; and (3) provided quantitative data regarding Hispanic individuals’ mortality 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/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 to 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 is largely skewed towards higher ages as reflected here.

**Data Abstraction**

Articles were independently coded by two teams with two members each. A third independent member then compared the two 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 pre-existing health conditions (if any), as well as the cause of mortality; (2) length of follow up; and (3) research design. Given the substantial heterogeneity amongst Hispanic peoples exemplified

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by differences in culture, traditions, and importantly, health outcomes, we further sought to code by country of origin/nativity when such data were available. Although we intended to include data regarding participant acculturation/enculturation, no studies reported information regarding participants’ immigration status or years living in the U.S., two studies reported information regarding percentage of foreign-born participants, and only ten studies reported participant average level of education, so our analyses could not include those variables.

Data within studies were often reported in terms of odds ratios (OR), 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 OR (lnOR) for analyses and then transformed back to OR for interpretation. When effect size data were reported in any metric other than OR or lnOR, we transformed those values using statistical software programs and macros (e.g., Comprehensive Meta-Analysis\textsuperscript{14}). In many cases we calculated effect sizes from frequency data in matrices 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 standard error) 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 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 inter-rater 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 manuscript 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. 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.

RESULTS

Literature Search and Study Characteristics

Figure 1 shows the study selection process. Statistically non-redundant effect sizes were extracted from 58 studies (see Table 1). Data were reported from 4,615,747 total participants, with an average composition of 26% Hispanic participants within studies. The average 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% omitting this information), but eight studies (15%) were specific to Mexican Americans, one study was specific to Puerto Rican Hispanic Paradox.
Americans\textsuperscript{48}, and five studies (9\%) involved participants from a variety of ethnic backgrounds.\textsuperscript{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 eight studies (14\%) involved a medical intervention;\textsuperscript{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). Note that PRISMA and MOOSE guidelines were adhered to in the design and reporting of this study.\textsuperscript{75-76}

**Omnibus Analysis**

Across the 58 studies, the random effects weighted average effect size was $OR = 0.825$ ($P< .0001$, 95\% Confidence interval = 0.75 to 0.91). As shown in Figure 2, odds ratios ranged from 0.39 to 2.75, with an extremely large degree of heterogeneity across studies ($I^2 = 96\%$; $Q_{(57)} = 1564, p< .001$; $\tau^2 = .12$), 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) may 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.

**Evaluation for Publication Bias**

To assess the possibility of publication bias\textsuperscript{77}, we conducted four analyses. First, we calculated Orwin’s fail-safe N,\textsuperscript{78} the theoretical number of unpublished studies with effect sizes
averaging zero (no effect) that would need to be located in order to reduce the overall magnitude of the results obtained to a trivial estimate of $1.0 > \text{OR} > 0.95$. Based on this calculation, at least 367 additional studies averaging $\text{OR} = 1.0$ would need to be found to render negligible the results of the present meta-analysis. Second, we utilized both Egger’s regression test\textsuperscript{79} and the alternative to that test recommended by Peters and colleagues\textsuperscript{80} that is better suited to data in OR format. The results of these two analyses failed to reach statistical significance ($P > .05$). Third, we generated a “funnel plot”\textsuperscript{81} of the studies’ log odds ratios by the standard errors. 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 are in the region of non-significance if publication bias is 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.\textsuperscript{82-83} This analysis indicated that when 14 estimated “missing” studies were included in the analysis, the overall effect size was calculated to be $\text{OR} = 0.70$ (95% CI = 0.64 to 0.77), indicating that Hispanic participants were 30% less likely to die than comparison group members over the same period of time. Based on these four analyses, we concluded that the data do not reflect publication bias per se, but that they may 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

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participants’ age, gender, and pre-existing diagnoses. We also investigated any differences across studies 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 \(-.28\) (\(P = .03\)), 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 (i.e., age cohort).

However, when we correlated the effect sizes 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 value of \(r = .22\) did not reach statistical significance (\(P = .10\)). Because older populations are more likely to receive treatment than 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 (\(b = -.28, P = .04\)), but the type of research study (intervention vs. observation) did not. The differences observed in risk for mortality appear to be moderated by participant age.

Similar random effects weighted correlations with the gender composition of each sample (using percent female; \(r = -.23\)) and the length of time participants were followed (\(r = .07\)) did not reach statistical significance (\(P > .05\)). Furthermore, no differences in the average effect sizes were found between studies using prospective vs. retrospective designs (\(Q_{1.57} = 0.1, P > .05\)).

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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 can be seen 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 pre-existing 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). 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 when compared with both NHWs and non-Hispanic Blacks (NHB), but they were more likely to die than Asian Americans during the same follow-up period.

**DISCUSSION**

Results of this meta-analysis show that Hispanic ethnicity is associated with a 17.5% lower likelihood of mortality relative to non-Hispanics, which figure is comparable to the 20% advantage reported by Arias and colleagues using the alternative death statistic estimation strategy. The omnibus finding in the current study is moderated by age such that the effect
becomes stronger among older participants, a finding similar to that which was recently reported using the estimation approach.\(^8\) In addition, the Hispanic mortality advantage varied as a function of pre-existing health status at study entry. Specifically, Hispanics displayed a significant mortality advantage among studies of initially healthy samples as well as in the context of cardiovascular disease 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 CDC report clearly stating a Hispanic ethnicity mortality advantage,\(^3\) it may 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 is that the observed mortality advantage, as well as the broader health outcome advantages evident in the Hispanic Paradox, may be due to resilience at several points in the course of disease. Hispanics may be less susceptible than some other races to illness in general or to specific conditions with high mortality rates, such as cardiovascular disease. It is also possible that the rate of disease progression may be slower among Hispanics, resulting in lower morbidity and greater longevity. Finally, the mortality advantage may reflect an advantage in survival and recovery from acute clinical events (e.g., MI, 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.

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Several risk and resilience factors may 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. Although not assessed in the current study, lower SES is a robust predictor of worse health outcomes. However, the current findings challenge the generalizability of this relationship given the typically lower SES of Hispanics relative to NHWs. It’s 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 suggest 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. Acculturation may be a proxy for social behaviors and cultural values which buffer against the stress of economic and environmental disadvantages. It is 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 understand potentially complex interaction patterns which may explain the observed effects.

The current 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 as well as 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

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We cannot 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 may yet reflect some degree of bias. For example, limiting inclusion to only those studies in English or Spanish may result in a language bias. The number of available studies also limited our ability to examine mortality in specific contexts including diabetes, autoimmune conditions, injury, neurological 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 has been constant or is decreasing over time. Study availability may also have limited our ability to detect subtle effects, as in the context of cancer and HIV where observed effects may 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 are shown to influence outcomes.\textsuperscript{89} To these points we would note that we did not examine unpublished manuscripts which could also affect findings. Finally, the analyzed sample was predominantly Mexican American, which likely limits generalizability across Hispanic subgroups, particularly given evidence of significant heterogeneity in Hispanic subgroup mortality outcomes.\textsuperscript{90-91}

Conclusions

These findings should serve as a cornerstone to documenting a comparative Hispanic mortality advantage in the context of a disadvantaged risk factor profile and demonstrate important heterogeneity in racial/ethnic minority health. Further, these findings highlight 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 lifespan, and in turn, have the potential for informing interventions for all. REFERENCES

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34. Gomez SL, O'Malley CD, Stroup A, Shema SJ, Satariano WA. Longitudinal, population-based study of racial/ethnic differences in colorectal cancer survival: impact of

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Table 1. Characteristics of Included Studies

<table>
<thead>
<tr>
<th>Source</th>
<th>Total N</th>
<th>Hispanic N</th>
<th>% Hispanic</th>
<th>% Women</th>
<th>Mean Age Sample</th>
<th>Follow-Up (Years)</th>
<th>Health Status at Study Entry</th>
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Hispanic Paradox
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Abbreviations: CVD, Cardiovascular disease; NA, not available.
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Note. $k =$ number of studies. $OR =$ odds ratio, transformed from random effects weighted lnOR. $Q_b =$ Q-value for variance between groups.
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Note. k = number of studies. OR = odds ratio, transformed from random effects weighted lnOR. Qb = Q-value for variance between groups.
Figure Legend

**Figure 1.** Selection of Articles for Meta-Analysis

**Figure 2.** Meta-Analysis of Hispanic Ethnicity and All-Cause Mortality