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Does socioeconomic status account for racial and ethnic disparities in childhood cancer survival?

Rebecca D Kehm, PhD[1], **Logan G Spector, PhD**[2], **Jenny N Poynter, PhD**[2], **David M Vock, PhD**[3], **Sean F Altekruse, PhD**[4], and **Theresa L Osypuk, SD**[1]

[1]University of Minnesota School of Public Health, Division of Epidemiology & Community Health, West Bank Office Building, 13000 S. Second Street, Minneapolis, MN 55455

[2]University of Minnesota, Division of Epidemiology & Clinical Research, Department of Pediatrics

[3]University of Minnesota School of Public Health, Division of Biostatistics

[4]National Cancer Institute, Epidemiology Branch, Prevention and Population Sciences Program, Division of Cardiovascular Sciences, National Heart Lung and Blood Institute

Abstract

Background—For many childhood cancers, survival is lower in non-Hispanic blacks and Hispanics compared to non-Hispanic whites, which may be attributed to underlying socioeconomic factors. However, prior childhood cancer survival studies have not formally tested for mediation by socioeconomic status (SES). We applied mediation methods to quantify the role of SES in racial/ethnic differences in childhood cancer survival.

Methods—We used population-based cancer survival data from the Surveillance, Epidemiology, and End Results 18 database for black, white, and Hispanic children, ages 0-19 years, diagnosed 2000-2011 (N=31,866). We estimated black-white and Hispanic-white mortality hazard ratios (HR) and 95% confidence intervals (CI), adjusted for age, sex, and stage at diagnosis. We used the inverse odds weighting (IOW) method to test for mediation by SES, measured with a validated census tract composite index.

Results—Whites had a significant survival advantage over blacks and Hispanics for several childhood cancers. SES significantly mediated the race/ethnicity-survival association for acute lymphoblastic leukemia, acute myeloid leukemia, neuroblastoma, and non-Hodgkin lymphoma; SES reduced the original association between race/ethnicity and survival by 44% ((log hazard ratio total effect – log hazard ratio direct effect)/log hazard ratio total effect), 28%, 49%, and 34% respectively for blacks vs. whites, and by 31%, 73%, 48%, and 28% respectively for Hispanics vs. whites.

Conclusions—SES significantly mediates racial/ethnic childhood cancer survival disparities for several cancers. However, the proportion of the total race/ethnicity-survival association explained

[corresponding author] kehmx003@umn.edu, (p) 612-626-6426.

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by SES varied between black-white and Hispanic-white comparisons for some cancers, suggesting that mediation by other factors differs across groups.

Keywords

socioeconomic status; childhood cancer; cancer survival; racial and ethnic disparities; mediation

Introduction

Despite improvements over the last four decades in cancer survival among the US pediatric population, marked racial and ethnic disparities persist.[1] Compared to non-Hispanic white (white) children, non-Hispanic black (black) and Hispanic children experience lower survival from many cancers including leukemias,[2, 3] lymphomas,[4, 5] central nervous system (CNS) tumors,[6] and extracranial solid tumors.[7–9] The underlying causes of racial/ethnic survival differences are not well understood, and may vary by cancer type. As outlined in Figure 1, both biological and socioeconomic pathways have been proposed in the literature.[10, 11] Underlying genetic variations associated with ancestry may lead to differences in tumor biology and pharmacogenetics for some childhood cancers.[10] However, race/ethnicity is a socially constructed taxonomy that is not synonymous with ancestry.[12] Race/ethnicity is highly correlated with socioeconomic status (SES), especially in the United States where embedded institutionalized racism continues to place racial and ethnic minorities at high risk of low SES.[13] Given emerging evidence of a positive association between SES and survival from some childhood cancers,[11] racial/ethnic survival disparities may also be explained by socioeconomic differences.

Quantifying the relative role of SES in explaining racial/ethnic survival disparities will help inform practice and intervention efforts. If SES accounts for racial/ethnic survival differences, then interventions addressing social and economic barriers to treatment and care are warranted. However, if SES does not fully account for survival differences by race/ethnicity, then other social factors (e.g. immigration), and biological mechanisms (e.g. tumor biology), must be considered. To date, formal mediation methods have not been employed to disentangle race/ethnic disparities in childhood cancer survival. Therefore, we conducted a mediation analysis using population-based data, representative of the US pediatric cancer population, to measure the role of SES in racial and ethnic childhood cancer survival disparities. We assessed survival from several childhood cancers to determine if mediation by SES differs across cancer type.

Methods

Study Population

We obtained population-based cancer registry data from the Surveillance, Epidemiology, and End Results (SEER) 18 database, excluding the Alaska Native Tumor Registry. We restricted to black, Hispanic, and white cases, ages 0 to 19 years, with microscopically confirmed first primary malignancies. Race was assigned in SEER through medical record abstraction. Hispanic ethnicity was assigned in SEER based on self/guardian-report of Spanish origin in the medical record, or by a computer algorithm that searches surnames and maiden names to

determine Spanish origin. Individuals of Spanish origin were categorized as Hispanic regardless of racial background.[14] SES data were available in SEER for diagnostic years 2000 to 2012. Therefore, we restricted our sample to cases diagnosed 2000-2011, followed through December 31, 2012, to allow for at least one year of follow-up. We excluded 45 cases with *in situ* tumors, 707 cases with missing/zero months of follow-up, and 725 cases missing SES data. We assessed cancers with ≥200 cases for each racial/ethnic group, classified using the International Classification of Childhood Cancer, third edition.[15] Our final analytic sample consisted of 31,866 cases.

Measures

Overall Survival—Overall Survival was calculated in SEER as months from date of cancer diagnosis to date of death from any cause, or censored at date of last contact.

Socioeconomic Status—Socioeconomic Status was measured at the neighborhood level, based on residential address at date of cancer diagnosis, using a validated census tract composite index.[16] As described in prior literature,[17] the index was constructed through factor analysis of nationwide 2000 decennial census data and 2005-2009 American Community Survey (ACS) data.[16] Seven indicators of neighborhood SES, previously specified by Yost et al. (2001), were included in the index: proportion employed in working-class occupations, proportion aged 16+ unemployed, education index,[18] median household income, proportion below 200% poverty level, median rent, and median house value.[19] Addresses were geocoded to census tracts, 2000 geographic boundaries. 2000 census values were assigned to cases diagnosed 2000-2003; 2005-2009 ACS values were assigned to cases diagnosed 2004-2011.[16] The index is available in SEER as a five-level variable categorized into quintiles (Q1=lowest SES; Q5=highest SES).

Covariates—We controlled for diagnostic age group (<1, 1-4, 5-9, 10-14, 15-19 years), sex, and stage at diagnosis (SEER Summary Stage 2000 (1998+): localized, regional, distal, unknown/unstaged).[20]

Statistical Analysis

For each cancer type, we estimated black-white and Hispanic-white mortality hazard ratios (total effects) from multivariable Cox proportional hazards regression models. No substantial violations of the proportional hazards assumption were identified. For cancers with a statistically significant total effect, we used the inverse odds weighting (IOW) method to test for mediation by SES.[21, 22] IOW analyses were conducted separately for black-white and Hispanic-white comparisons to account for the possibility that SES may mediate differently by race/ethnicity, although sensitivity analyses using multinomial models of all three racial/ethnic groups documented comparable results. IOW is a semiparametric weight-based approach that overcomes many limitations of traditional parametric mediation methods.[23] For example, IOW is appropriate for any functional form (rather than just linear models), can test multiple mediators simultaneously (as opposed to testing them one by one), and is valid even in the presence of exposure-mediator interactions.[24]

Applying the IOW method, we estimated the (natural)[25] direct effect of race/ethnicity on survival by fitting a weighted multivariable Cox proportional hazards model. Weighting by the inverse odds of exposure creates a pseudo-population in which the exposure and mediator are independent, thus estimating the race/ethnicity-survival association (direct effect) that remains after accounting for the pathway through SES.[25] To obtain the IOW weights, we first estimated the odds of exposure (i.e. race/ethnicity) for each subject from a multivariable logistic regression model specifying SES and covariates. We then took the inverse of the predicted odds to create the IOW weight for whites; non-whites were assigned a weight of one. The non-white racial/ethnic group was selected as the reference to minimize extreme weighting values. Next, we estimated the (natural)[25] indirect effect of race on survival operating through SES by subtracting the direct (log hazard ratio (β)) from the total effect, and bootstrapping to obtain standard errors (500 replications). A significant indirect effect provides statistical evidence of mediation. To quantify the magnitude of mediation by SES, we calculated the percent reduction from the total to the direct effect $((\beta_{total} - \beta_{direct})/\beta_{total})$. Statistical significance was determined as $p<0.05$ for a 2-sided hypothesis test. Analyses were performed using Stata 14.2 (Stata Corporation, College Station, Texas).[26]

Secondary Analyses—The tract-level SES index likely captures an array of socioeconomic factors contributing to survival. One such factor may be health insurance status. To empirically test this, we compared indirect effects of mediation by tract SES index and also by individual-level health insurance status (private versus otherwise), testing each of these mediators separately and simultaneously. This analysis was confined to cancers with a significant tract indirect SES effect in the primary analysis and to cases diagnosed 2007-2011, when health insurance data were available in SEER. We also explored whether we inadvertently overly adjusted IOW models by including stage at diagnosis as a covariate. Stage at diagnosis could theoretically operate as a downstream mediator of the SES-survival association if, for example, SES influences diagnostic timing.[10] We tested logistic models of SES predicting tumor stage (local versus otherwise; distal versus otherwise), and we compared SES indirect effect estimates from IOW models unadjusted and adjusted for stage at diagnosis.

Results

Descriptive characteristics by cancer type are provided in Table 1. All-cause mortality from 2000 to 2012 varied across cancers, ranging from 5.2% among Hodgkin lymphoma (HL) cases to 33.8% among acute myeloid leukemia (AML) cases. Mean age at diagnosis varied across cancers, ranging from 2.5 years (SD=3.3) among neuroblastoma cases to 14.9 years (SD=3.8) among HL cases. There was a higher proportion of males compared to females for all cancers except Wilms tumor (53.4% female). The distribution of tumor stage varied across cancers (stage does not apply to leukemias). For example, only 1.9% of astrocytoma cases were classified as distal stage at diagnosis compared to 48.9% of neuroblastoma cases. The distribution of cases across SES categories was consistent across cancers. Sample characteristics by race/ethnicity are available in the supplemental materials (Table S2).

We compare all-cause mortality between black versus white and Hispanic versus white cases (total effects) in Table 2. Compared to whites, black cases had a statistically significant

higher hazard of death for all cancers except Wilms tumor, osteosarcoma, and germ cell tumors (GCT). Across the 9 cancers with significant racial disparities in mortality, black compared to white children exhibited from 38% (neuroblastoma) to 95% (astrocytoma) higher risk of mortality ($p<0.05$). Compared to whites, Hispanic cases had a statistically significant, or marginally significant (AML and non-rhabdomyosarcoma soft tissue sarcomas (NRSTS)), higher hazard of death for all cancers except HL, non-astrocytoma CNS tumors, rhabdomyosarcoma, and osteosarcoma. Among the 6 cancers exhibiting significant ethnic disparities in mortality, Hispanic children, compared to their white counterparts, exhibited from 31% (neuroblastoma) to 65% (non-Hodgkin lymphoma (NHL)) higher risk of mortality ($p<0.05$).

In Table 3, we present IOW results for mediation by SES of the racial (black-white) disparity in all-cause mortality among childhood cancer cases. SES was determined to be a significant mediator of the race-survival association if the indirect effect of race on survival operating through SES was statistically significant. SES significantly mediated the black-white survival disparity for ALL (indirect effect HR (iHR)=1.17; CI=1.07, 1.28; $p<0.01$; 44% reduction from the total to the direct effect of the racial disparity in mortality), AML (iHR=1.15; CI=1.03, 1.29; p=0.01; 28% reduction), and neuroblastoma (iHR=1.17; CI=1.03, 1.33; p=0.02; 49% reduction). SES was a marginally significant mediator of the black-white survival disparity for NHL (iHR=1.16; CI=0.97, 1.37; p=0.10; 34% reduction). First-leg mediation results are available in the supplemental materials (Table S1).

In Table 4, we present IOW results testing mediation by SES of the ethnic (Hispanic-white) disparity in all-cause mortality. SES significantly mediated the ethnic mortality disparity for ALL (iHR=1.16; CI=1.08,1.26; $p<0.001$; 31% reduction from the total to the direct effect of the ethnic disparity in mortality), AML (iHR=1.13; CI=1.03, 1.25; p=0.01; 73% reduction), neuroblastoma (iHR=1.14; CI=1.03, 1.26; p=0.01; 48% reduction), and NHL (iHR=1.15; CI=1.01, 1.31; p=0.04; 28% reduction). Notably, SES significantly mediated both the racial and ethnic disparity in survival for the same four cancers.

Secondary Analyses

Except for NHL, the mediating effect of tract-level SES was greater than the mediating effect of health insurance status among black-white and Hispanic-white comparisons (supplemental materials, Table S3). For example, the indirect effect of tract SES on the black-white mortality disparity for ALL was 1.22 (CI=1.01, 1.48; p=0.04; 44% reduction), while the indirect effect of health insurance was 1.09 (CI=0.94, 1.27; p=0.24; 19% reduction). Among cancers with significant SES indirect effects, SES was not associated with stage at diagnosis (supplemental materials, Table S4). Exclusion of stage at diagnosis from IOW models did not lead to notably stronger indirect SES effects (supplemental materials, Tables S5–S6).

Discussion

This is the first study to use formal mediation methods to unpack childhood cancer survival disparities by race/ethnicity, which generated several findings. First, we replicated results from prior studies that whites have a significant survival advantage over blacks and

Hispanics for several childhood cancers including leukemias,[2, 3] lymphomas,[4, 5] CNS tumors,[6] neuroblastoma,[7] and NRSTS.[9] In no instance was survival among whites significantly worse than that of either black or Hispanic children. Racial and ethnic survival differences were not uniform across cancers, and some variability between black-white and Hispanic-white comparisons was observed. Second, we demonstrated that SES contributes to racial/ethnic survival disparities (e,g. ALL, AML, neuroblastoma, and NHL), and mediation conclusions were consistent between the black-white and Hispanic-white mediation models for these four cancers. However, SES did not fully account for racial/ethnic survival disparities (indicated by some direct effects that remained significant after testing the SES pathway); this finding aligns with prior research suggesting that race captures more than just SES, including racism and differential treatment, which we did not have the data to test in this study.[27] Finally, secondary findings suggest that the association between tract SES and childhood cancer survival is not explained by differential access to health insurance, since both SES measures contributed independently to mediate the disparity; nor is it explained by tumor stage at diagnosis.

Among childhood cancers with significant mediation by SES (ALL, AML, neuroblastoma, and NHL), indirect HRs fell within a narrow range (1.13 to 1.17) for both black-white and Hispanic-white comparisons. This suggests that the association between SES and survival is not modified by, and may be shared across, race/ethnicity. Conversely, the proportion of the overall survival disparity explained by SES (i.e. % reduction) did vary by race/ethnicity for some cancers. For example, among AML cases, SES explained only 28% of the black-white survival disparity compared to 73% of the Hispanic-white disparity. This may indicate that mediation by other factors, not captured by the SES index, differs across racial/ethnic groups for some cancers. Such factors may include differences in tumor biology, pharmacogenomics, or other social factors, such as health care quality. For example, prior evidence suggests that, among AML cases, a significantly lower proportion of black children have matched family donors available compared to white and Hispanic children.[28]

The downstream mechanisms through which SES influences childhood cancer survival are not fully understood. Prior literature suggests that the strong association between SES and ALL survival may be explained by differences in treatment adherence.[10] Adherence to the prolonged maintenance phase required for treatment of ALL may be difficult for low SES families due to social and economic constraints.[10] This is supported by prior evidence of lower treatment adherence among children with ALL living in a single-mother household compared to a two-parent household.[29] Research on SES and survival is less developed for other types of childhood cancer. Secondary findings from this study suggest that factors beyond health insurance status and stage at diagnosis contribute to the SES-survival association, at least for some childhood cancers. Thus, additional studies are needed to further unpack the association between SES and childhood cancer survival.

Limitations

We relied on an area-based variable as our primary measure of SES given the lack of individual-level SES measures in SEER data; moreover we selected an SES index in order to operationalize the SES construct over a meaningful period of time. Though this improves

upon many prior population-based cancer studies that lacked any measures of SES or relied on county-level measures, tract-level SES is still a proxy for individual-level SES in this study because we could not comprehensively control for SES at the individual level.[30, 31] Because the tract-level SES index was only available in SEER for years 2000-2012, sample size and follow-up time were limited. This prevented us from testing more homogenized cancer and racial/ethnic subgroups or stratifying by age. Additional research is thus needed for other smaller populations of racial and ethnic groups not considered in this analysis due to the rarity of childhood cancer that limited power. We also lacked geographic variables to explore potential spatial variations in survival. Further, the lack of clinical data in SEER limited our ability to account for diagnostic, therapeutic, and biological factors, such as cytogenetic or molecular features. Finally, there is the potential for differential loss to follow-up by race and SES.

Conclusion

Through the application of formal mediation methods, we demonstrated that SES significantly contributes to racial and ethnic survival disparities for several childhood cancers including ALL, AML, neuroblastoma, and NHL. Thus, for these cancers in particular, racial/ethnic survival disparities could theoretically be addressed through initiatives that reduce social and economic barriers to effective care. Such efforts may include expanded health insurance coverage, improved patient care coordination, increased health literacy, and supplementation of transportation and childcare costs during treatment. However, because SES did not fully account for survival disparities, we cannot rule out the potential role of other mediating pathways, such as tumor biology, pharmacogenomics, health care quality, or other social factors. A multipronged intervention approach that both addresses socioeconomic barriers to care and invests in personalized treatment regimens may ultimately be needed to fully eliminate childhood cancer survival disparities.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Significant Conclusions

Socioeconomic status mediates the association between race/ethnicity and childhood cancer survival, though to varying degrees across cancers. The proportion of the total effect accounted for by socioeconomic status varied by race/ethnicity for some cancers.

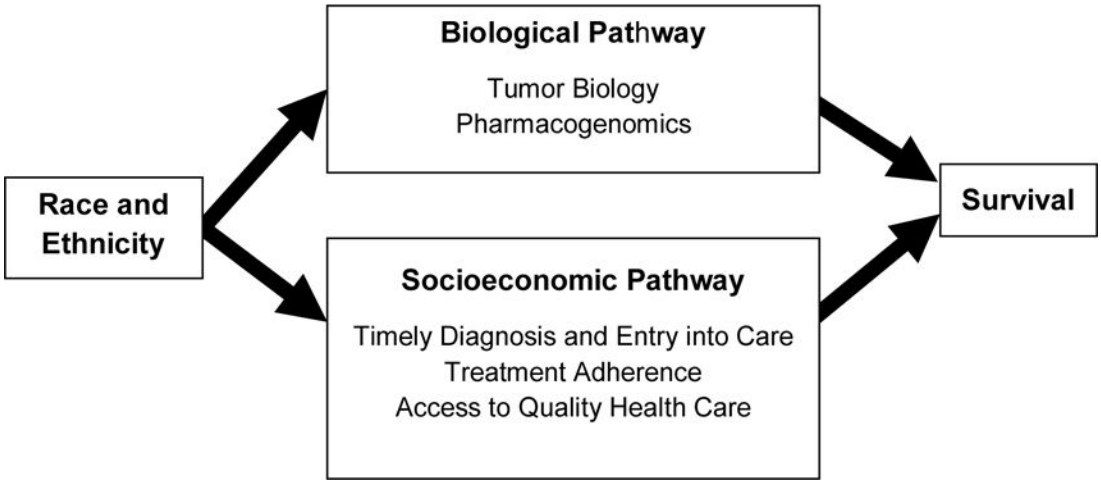

Figure 1.
Proposed mediating pathways between race/ethnicity and childhood cancer survival

Table 1

Characteristics of childhood cancer cases, ages 0-19 years, diagnosed 2000-2011, SEER 18 registries

		Race/Ethnicity N						Stage at Diagnosis %				Tract-Level SES Index[a] %				
Cancer Type	**N**	**W**	**B**	**H**	**Survival Months Mean (SD)**	**All-Cause Mortality %**	**Age at Diagnosis Mean (SD)**	**Female %**	**L**	**R**	**D**	**U**	**Q1**	**Q2**	**Q3**	**Q4**	**Q5**
Acute Lymphoblastic Leukemia	8,492	4,357	634	3,501	70.2 (42.6)	12.9	6.9 (5.3)	43.1	n/a	n/a	n/a	n/a	23.2	21.4	19.3	17.9	18.3
Acute Myeloid Leukemia	1,832	965	253	614	54.6 (43.8)	33.8	8.9 (6.7)	48.0	n/a	n/a	n/a	n/a	25.4	20.5	18.6	18.7	16.8
Neuroblastoma	1,901	1,214	264	423	63.5 (43.0)	21.6	2.5 (3.3)	47.6	20.8	25.3	48.9	5.0	20.5	19.7	21.0	18.6	20.3
Non-Hodgkin Lymphoma	2,065	1,169	343	553	68.1 (44.2)	15.5	12.6 (5.0)	37.1	30.9	19.6	44.4	5.2	22.4	19.1	19.3	19.4	19.9
Hodgkin Lymphoma	3,078	1,947	384	747	76.8 (41.5)	5.2	14.9 (3.8)	46.6	14.5	48.3	34.3	2.9	20.0	20.2	18.2	19.7	21.9
Astrocytoma	3,195	2,080	360	755	69.2 (45.4)	17.2	9.1 (5.6)	47.9	82.7	11.5	1.9	3.8	19.2	19.2	19.5	20.3	21.9
Non-Astrocytoma CNS Tumors	2,827	1,718	326	783	60.7 (45.1)	31.2	7.5 (5.8)	42.1	69.7	13.3	12.8	4.2	21.1	19.1	19.6	19.7	20.4
Non-Rhabdomyosarcoma Soft Tissue Sarcomas	1,784	974	296	514	65.3 (44.9)	22.9	11.9 (5.9)	46.6	56.6	22.8	15.3	5.4	20.0	21.5	19.9	19.0	19.7
Rhabdomyosarcoma	1,202	656	208	338	59.0 (43.1)	32.6	7.9 (5.7)	42.9	31.9	34.1	29.7	4.3	20.7	22.0	18.1	19.1	20.1
Wilms Tumor	1,430	789	254	387	71.0 (43.2)	8.3	3.4 (3.0)	53.4	42.4	29.7	24.8	3.2	22.5	21.1	19.7	18.1	18.6
Osteosarcoma	1,247	619	217	411	60.8 (42.1)	32.6	13.2 (3.7)	45.1	32.3	44.0	20.6	3.1	21.3	22.5	20.2	18.3	17.6
Germ Cell Tumors	2,813	1,549	231	1,033	72.7 (43.5)	7.5	13.8 (6.0)	35.4	55.5	22.7	18.2	3.6	20.9	20.2	19.8	19.1	19.9

Abbreviations: B, non-Hispanic black; D, distal; H, Hispanic; L, localized; N, sample size; Q, quintile; R, regional; SD, standard deviation; SES, socioeconomic status; U, unknown/unstaged; W, non-Hispanic white

[a]Higher quintiles represent higher SES (i.e. Q1=lowest SES quintile; Q5=highest SES quintile)

Table 2

Comparison of all-cause mortality by race/ethnicity among childhood cancer cases, ages 0 to 19 years, diagnosed 2000-2011, SEER 18 registries

	Black versus White			Hispanic versus White		
Cancer Type	**Mortality HR[a]**	**95% CI**	**P**	**Mortality HR[a]**	**95% CI**	**P**
Acute Lymphoblastic Leukemia	1.43	1.15, 1.77	<0.01	1.63	1.44, 1.85	<0.001
Acute Myeloid Leukemia	1.68	1.35, 2.08	<0.001	1.19	0.99, 1.42	0.06
Neuroblastoma	1.38	1.07, 1.78	0.02	1.31	1.03, 1.66	0.03
Non-Hodgkin Lymphoma	1.53	1.15, 2.05	<0.01	1.65	1.28, 2.13	<0.001
Hodgkin Lymphoma	1.66	1.09, 2.53	0.02	1.11	0.76, 1.64	0.59
Astrocytoma	1.95	1.55, 2.46	<0.001	1.34	1.10, 1.64	<0.01
Non-Astrocytoma CNS Tumors	1.53	1.26, 1.86	<0.001	1.07	0.92, 1.25	0.36
Non-Rhabdomyosarcoma Soft Tissue Sarcomas	1.40	1.08, 1.82	0.01	1.22	0.98, 1.53	0.08
Rhabdomyosarcoma	1.44	1.10, 1.88	0.01	1.11	0.88, 1.41	0.37
Wilms Tumor	0.96	0.57, 1.62	0.88	1.60	1.06, 2.39	0.02
Osteosarcoma	0.88	0.67, 1.16	0.37	0.99	0.79, 1.23	0.91
Germ Cell Tumors	0.98	0.57, 1.69	0.94	1.63	1.23, 2.18	<0.01

Abbreviations: CI, confidence interval; HR, hazard ratio

[a]Adjusted for age at diagnosis, sex, and stage at diagnosis (stage not applicable for leukemias)

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Table 3

Mediation by SES of racial (black versus white) survival disparities among childhood cancer cases, ages 0 to 19 years, SEER 18 registries, 2000-2011 diagnoses

	Total Effect of race on survival through all mediating pathways			Direct Effect of race on survival after blocking the SES pathway			Indirect Effect of race on survival operating through the SES pathway			Percent Reduction from total to direct effect
Cancer Type	**Mortality HR[a]**	**95% CI**	**P**	**Mortality HR[a]**	**95% CI**	**P**	**Mortality HR[a]**	**95% CI**	**P**	**%[b]**
Acute Lymphoblastic Leukemia	1.43	1.15, 1.77	<0.01	1.22	0.96, 1.54	0.10	1.17	1.07, 1.28	<0.01	44
Acute Myeloid Leukemia	1.68	1.36, 2.07	<0.001	1.45	1.15, 1.84	<0.01	1.15	1.03, 1.29	0.01	28
Neuroblastoma	1.38	1.08, 1.75	0.01	1.18	0.91, 1.52	0.22	1.17	1.03, 1.33	0.02	49
Non-Hodgkin Lymphoma	1.53	1.14,2.07	0.01	1.33	0.94, 1.88	0.11	1.16	0.97, 1.37	0.10	34
Hodgkin Lymphoma	1.66	1.06, 2.60	0.03	1.50	0.87, 2.58	0.15	1.11	0.83, 1.48	0.50	20
Astrocytoma	1.95	1.57, 2.43	<0.001	1.80	1.42, 2.30	<0.001	1.08	0.98, 1.20	0.12	12
Non-Astrocytoma CNS Tumors	1.53	1.25, 1.88	<0.001	1.41	1.11, 1.78	<0.01	1.09	0.97, 1.22	0.14	20
Non-Rhabdomyosarcoma Soft Tissue Sarcomas	1.40	1.06, 1.84	0.02	1.34	0.96, 1.87	0.08	1.04	0.87, 1.26	0.65	13
Rhabdomyosarcoma	1.44	1.10, 1.88	0.01	1.33	0.98, 1.81	0.07	1.08	0.93, 1.25	0.31	21

Abbreviations: CI, confidence interval; HR, hazard ratio; SES, socioeconomic status

[a] Adjusted for age at diagnosis, sex, and stage at diagnosis (stage not applicable for leukemias)

[b] Percent reduction from the total to the direct effect ((βtotal − βdirect)/βtotal)

Table 4

Mediation by SES of ethnic (Hispanic versus white) survival disparities among childhood cancer cases, ages 0 to 19 years, SEER 18 registries, 2000-2011 diagnoses

	Total Effect of ethnicity on survival through all mediating pathways			Direct Effect of ethnicity on survival after blocking the SES pathway			Indirect Effect of ethnicity on survival operating through the SES pathway			Percent Reduction from total to direct effect
Cancer Type	**Mortality HR[a]**	**95% CI**	**P**	**Mortality HR[a]**	**95% CI**	**P**	**Mortality HR[a]**	**95% CI**	**P**	**%[b]**
Acute Lymphoblastic Leukemia	1.63	1.43, 1.86	<0.001	1.40	1.21, 1.63	<0.001	1.16	1.08, 1.26	<0.001	31
Acute Myeloid Leukemia	1.19	0.99, 1.43	0.071	1.05	0.85, 1.29	0.66	1.13	1.03, 1.25	0.011	73
Neuroblastoma	1.31	1.04, 1.65	0.021	1.15	0.89, 1.49	0.27	1.14	1.03, 1.26	0.011	48
Non-Hodgkin Lymphoma	1.65	1.29, 2.12	<0.001	1.44	1.08, 1.92	0.013	1.15	1.01, 1.31	0.04	28
Astrocytoma	1.34	1.10, 1.64	<0.01	1.26	1.01, 1.56	0.041	1.07	0.98, 1.16	0.12	23
Non-Rhabdomyosarcoma Soft Tissue Sarcomas	1.22	0.96, 1.55	0.10	1.13	0.87, 1.46	0.38	1.08	0.95, 1.24	0.25	41
Wilms Tumor	1.60	1.04, 2.45	0.032	1.57	0.95, 2.52	0.061	1.02	0.83, 1.24	0.883	3
Germ Cell Tumor	1.63	1.19, 2.24	<0.01	1.70	1.19, 2.42	<0.01	0.96	0.81, 1.15	0.685	-8

Abbreviations: CI, confidence interval; HR, hazard ratio; SES, socioeconomic status

[a] Adjusted for age at diagnosis, sex, and stage at diagnosis (stage not applicable for leukemias)

[b] Percent reduction from the total to the direct effect ((βtotal − βdirect)/βtotal)