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Research Article

Cancer Epidemiology, Biomarkers & Prevention

Baby Boomers and Birth Certificates: Early-Life Socioeconomic Status and Cancer Risk in Adulthood

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Abstract

Background: Early-life socioeconomic status (SES) may play a role in cancer risk in adulthood. However, measuring SES retrospectively presents challenges. Parental occupation on the birth certificate is a novel method of ascertaining early-life SES that has not been applied in cancer epidemiology.

Methods: For a Baby-Boom cohort born from 1945–1959 in two Utah counties, individual-level Nam-Powers SES (Np-SES) was derived from parental industry/occupation reported on birth certificates. Neighborhood SES was estimated from average household income of census tract at birth. Cancer incidence was determined by linkage to Utah Cancer Registry records through the Utah Population Database. Hazard ratios (HR) for cancer risk by SES quartile were estimated using Cox proportional hazards regression.

Results: Females with low Np-SES at birth had lower risk of breast cancer compared with those in the highest Np-SES group [$HR_{Q1/Q4}$ = 0.83; 95% confidence interval (CI), 0.72–0.97; $HR_{Q2/Q4}$ = 0.81; 95% CI, 0.69–0.96]. Np-SES was inversely associated with melanoma ($HR_{Q1/Q4}$ = 0.81; 95% CI, 0.67–0.98) and prostate cancer ($HR_{Q1/Q4}$ = 0.70; 95% CI, 0.56–0.88). Women born into lower SES neighborhoods had significantly increased risk for invasive cervical cancer ($HR_{Q1/Q4}$ = 1.44; 95% CI, 1.12–1.85; $HR_{Q2/Q4}$ = 1.33; 95% CI, 1.04–1.72). Neighborhood SES had similar effects for melanoma and prostate cancers, but was not associated with female breast cancer. We found no association with SES for pancreas, lung, and colon and rectal cancers.

Conclusions: Individual SES derived from parental occupation at birth was associated with altered risk for several cancer sites.

Impact: This novel methodology can contribute to improved understanding of the role of early-life SES on cancer risk. *Cancer Epidemiol Biomarkers Prev; 26(1); 1–10. ©2016 AACR.*

Introduction

Cancer disparities and the role of socioeconomic status (SES) is a major focus of the National Cancer Institute's (NCI) strategic plan (1). Studies have observed that SES as defined by income or education is associated with cancer incidence, late-stage diagnosis, and survival (2–15). However, the majority of population-based studies are based on area-level measures (16–20) because measures of individual SES are not generally available in medical records or through cancer registries (18, 21). This approach may also misclassify SES for some individuals and bias results (22).

Early-life conditions are increasingly recognized as playing an important role in the etiology of chronic diseases (23), including coronary heart disease (24–26) and cardiac dysfunction (27), type II diabetes (28–30), chronic obstructive pulmonary disease (31–33), asthma (34), osteoporosis (35–37), and hypertension (38, 39). In addition, social and material disadvantages experienced in early-life have been associated with poor health behaviors in adulthood, such as smoking and excessive alcohol consumption (40–42), obesity (42–44), and poor overall mental and physical health (45). The role of early-life SES and cancer incidence, however, is surprisingly underdeveloped. Vohra and colleagues (46) conducted a rapid review of the literature from 1996 to 2012 and found only three of 22 studies evaluated childhood socioeconomic position in relation to cancer incidence (47–49). Although there is a growing interest to explore the association between early-life SES and cancer risk in adulthood (23, 46, 48, 50–55), there remains a need for a new approach to collecting high-quality, early-life, individual-level SES data that may be applied across states and nations to advance our understanding of the etiology of the disease.

To address this need, we utilized birth certificates for a cohort of Utah Baby Boomers born from 1945 to 1959 to obtain early-life, individual-level SES based on parental occupation and neighborhood SES based on the birth certificate census tract (CT). Studying

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the health outcomes of the Baby-Boom cohort is important particularly because of their large contribution toward the profile of an aging nation (56) and the demands they will place on the U.S. health care system (57). We hypothesized that early-life SES, based on parental occupation at birth, will be associated with cancer risk in adulthood.

Materials and Methods

Sample

The cohort was drawn from the Utah Population Database (UPDB), a longitudinal, population-based resource that comprehensively links cancer incidence data from the Utah Cancer Registry (UCR) and birth certificate data from the Utah Department of Health's Office of Vital Statistics (58). An individual identified within the UPDB was selected if he or she appeared as a child on a birth certificate, born between 1945 and 1959 in Salt Lake and Weber Counties, survived and lived in Utah to at least age 18, and linked to a UPDB record other than their parents' record or another UCR record ($N = 125{,}335$). The cohort was restricted to Salt Lake and Weber Counties because they were the only two counties with CTs in the 1960 census. The cohort was limited to children who lived in Utah to age 18 because of our focus on cancer incidence in adulthood. The requirement that birth records link to other non-UCR records in the UPDB was imposed to reduce the likelihood of ascertainment bias.

Data linkage

Birth certificate records (1945–1959) were linked to cancer records (1996–2009) by UPDB staff using Quality Stage, a part of IBM's Websphere Information Integration Solution(™) family of tools and applications. Every potential match is assigned a numeric weight based on probabilistic record linkage. Extensive details on the linkage algorithms have been described previously (59–61). Approximately 7% ($n = 8{,}989$) of birth certificates from our cohort linked to a UCR record.

Individual-level SES: industry/occupation and Nam-Powers SES

Individual-level SES measures were coded from the text on birth certificates about parents' industry and occupation (I/O). Parental occupation is the most common measure of childhood SES in cancer studies (46). Records were manually reviewed, and all abbreviations, erroneous characters, and misspellings were removed. Records were run through the Standardized Occupation & Industry Coding (SOIC) software distributed by the CDC (62). Because SOIC software does not perform well when company names and other ambiguous information are included in the I/O text, I/O text strings that did not code were manually reviewed by an expert coder and corrected. This increased the percent of I/O coding from 81.7% to 98.3%.

The I/O codes were used as inputs to construct Nam-Powers socioeconomic scores (Np-SES; ref. 63). Np-SES scores classify occupations into an interval scale according to their respective median education and income levels from 0 (low) to 100 (high) and have been used in several large studies of mortality (58, 64, 65). We classified Np-SES at birth using the father's I/O code or mother's I/O code if father's occupation was unknown or no father was listed on the birth certificate. If an I/O was not codable for either parent, the Np-SES for that record was classified as unknown. Birth certificates with parental I/O of "student" or "homemaker" were also classified as unknown as these activities do not have Np-SES values (63). Quartile ranks of Np-SES (Q1–Q4, unknown) were used in analyses.

Neighborhood-level SES at Birth: 1960 CT household income

Neighborhood SES, or birth certificate census tract SES (BiCT-SES), was defined as the average household income at the CT level, and based on the birthmother's residence at the time of the child's birth. The 1960 CT boundary geographic information system files were obtained from the National Historical Geographic Information System (https://www.nhgis.org/). The residential street address on the birth certificate were geocoded and spatially joined based on geocoded location to one of 119 CTs for Salt Lake and Weber Counties. These accounted for 50% of all Utah births. A comparison of birth characteristics in Salt Lake and Weber counties to the rest of Utah indicated they were similarly distributed by birth year, gender, race/ethnicity, cancer occurrence, vital status, and SES characteristics. The final sample include geocodes for 85% ($n = 107{,}153$) of the births.

The 1960 census did not include average household income for CTs. Instead, family income was reported in one of 13 income categories ranging from less than $1,000 to $25,000 annually. We calculated "average household income" by multiplying the number of households within each income range by the midpoint of each income range of that group and dividing the total by the number of households in the CT. Quartile ranks of BiCT-SES (Q1–Q4, unknown) were used in analyses.

Follow-up

UPDB linkages to other datasets including Utah death certificates, driver licenses, and hospital discharge records were used to ascertain each individual's last date of documented residence in Utah. Cohort members were followed for cancer incidence beginning either from age 18 or from 1966 for those who turned 18 before 1966 when the UCR began cancer surveillance. The end of follow-up was December 31, 2009, or date of diagnosis of first invasive primary cancer, death, or end of Utah residence. 2009 was the last year in which cancer incidence and follow-up information were available at the time this study was conducted. Month and year of diagnosis were assigned on the basis of UCR data. Analysis focused on cancer sites ranking among the highest, including sites for which incidence was strongly influenced by widely disseminated cancer screening during the study period (female breast, cervical, colorectal, and prostate), and sites for which no widely applicable screening programs were available and therefore participation in screening would not influence incidence (melanoma, lung and bronchus, and pancreatic cancers).

Statistical analysis

We conducted descriptive analysis tabulating demographic, birth, and SES characteristics by presence or absence of a cancer diagnosis during follow-up. We generated cumulative cancer incidence curves based on the cumulative hazard function by cancer site, sex, and SES. The time scale was age in months starting from age 18 ($t_{=0}$). Log-rank tests were conducted for all seven cancers and for both SES measures to assess survival differences across the strata with significance set at $P < 0.05$.

We used Cox proportional hazards regression models to estimate the relative hazards and 95% confidence intervals for the diagnosis of cancer by Np-SES and BiCT-SES quartile rank for each cancer site. All covariates met the assumption of proportional

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Table 1. Births 1945–1959 in two Utah counties (Salt Lake and Weber): demographics and subsequent cancer diagnosis

Characteristics	Total *N* (%)	Cancer[a] *N* (%)	No cancer[a] *N* (%)	χ^2 *P*
Total births	126,335 (100.0)	8,989 (7.1)	117,346 (92.9)	—
Gender				
Male	65,838 (52.1)	3,786 (42.1)	62,052 (52.9)	<0.001
Female	60,497 (47.9)	5,203 (57.9)	55,294 (47.1)	
Year of birth				
1945–1949	31,744 (25.1)	3,294 (36.6)	28,450 (24.2)	<0.001
1950–1954	42,851 (33.9)	3,086 (34.3)	39,765 (33.9)	
1955–1959	51,740 (41.0)	2,609 (29.0)	49,131 (41.9)	
Birth certificate race				
White	124,564 (98.6)	8,880 (98.8)	115,684 (98.6)	<0.001[b]
Black	472 (0.4)	29 (0.3)	443 (0.4)	
AIAN	<5 (–)	<5 (–)	<5 (–)	
API	600 (0.5)	38 (0.4)	562 (0.5)	
Other	637 (0.5)	40 (0.4)	597 (0.5)	
Unknown	58 (<0.0)	<5 (–)	56 (<0.0)	
Birth certificate Hispanic origin				
Known Hispanic	402 (0.3)	23 (0.3)	379 (0.3)	<0.001
Not specified	125,933 (99.7)	8,966 (99.7)	116,967 (99.7)	
Birth order				
1	31,803 (25.2)	2,376 (26.4)	29,427 (25.1)	<0.001
2	31,895 (25.2)	2,262 (25.2)	29,633 (25.3)	
3+	61,317 (48.5)	4,269 (47.5)	57,048 (48.6)	
Unknown	1,320 (1.0)	82 (0.9)	1,238 (1.1)	
Sibling in cohort				
No	31,175 (24.7)	2,140 (23.8)	29,035 (24.7)	<0.001
Yes	95,160 (75.3)	6,849 (76.2)	88,311 (75.3)	
Mother married at birth				
Yes	124,903 (98.9)	8,903 (99.0)	116,000 (98.9)	<0.001
No	428 (0.3)	36 (0.4)	392 (0.3)	
Unknown	1,004 (0.8)	50 (0.6)	954 (0.8)	
Multiple birth				
No	122,299 (96.8)	8,832 (98.3)	113,467 (96.7)	<0.001
Yes	2,258 (1.8)	148 (1.6)	2,110 (1.8)	
Unknown	1,778 (1.4)	9 (0.1)	1,769 (1.5)	
Birth weight				
Low (<2,500 g)	6,170 (4.8)	402 (4.5)	5,768 (4.9)	<0.001
Normal (2,500–4,200 g)	96,742 (76.6)	6,484 (72.1)	90,258 (76.9)	
Above normal (>4,200 g)	3,261 (2.6)	185 (5.7)	3,076 (2.6)	
Unknown	20,162 (16.0)	1,918 (21.3)	18,244 (15.6)	
1960 Census tract income (BiCT-SES)				
Total coded	107,153 (84.8)	7,627 (84.8)	99,526 (84.8)	<0.001
Q1 (lowest)	32,031 (25.4)	2,396 (26.7)	29,635 (25.3)	
Q2	29,225 (23.1)	2,108 (23.5)	27,117 (23.1)	
Q3	29,597 (23.4)	2,037 (22.7)	27,560 (23.5)	
Q4 (highest)	16,300 (12.9)	1,086 (12.1)	15,214 (13.0)	
Unknown	19,182 (15.2)	1,362 (15.2)	17,820 (15.2)	
Nam-Powers score (Np-SES)[c]				
Total coded	118,158 (93.5)	8,418 (93.6)	109,740 (93.5)	<0.001
Q1 (lowest)	32,690 (25.9)	2,334 (26.0)	30,356 (25.9)	
Q2	23,492 (18.6)	1,696 (18.9)	21,796 (18.6)	
Q3	32,230 (25.5)	2,363 (26.3)	29,867 (25.5)	
Q4 (highest)	29,746 (23.5)	2,025 (22.5)	27,721 (23.6)	
Unknown	8,177 (6.5)	571 (6.4)	7,606 (6.5)	

[a]Cancer incidence through December 31, 2009.
[b]Fisher exact due to 0 cell value; cells less than 5 masked to safeguard confidentiality.
[c]Father's Nam-Powers score at birth; if unknown, then Mother's Nam-Powers score; otherwise, unknown.

hazards. High SES (Q4) was the referent group. All models controlled for birth weight (continuous pounds), year of birth (continuous 1945–1959), presence of a sibling in the cohort, and gender (male/female) where appropriate. We controlled for whether the child had a sibling born in the same birth cohort to control for additional circumstances that may impact early-life socioeconomic conditions, which may subsequently impact cancer risk later in life. The very low proportion of nonwhites made adjusting for race impracticable; therefore, analyses were restricted to whites. Hispanic ethnicity was not reported on birth certificates from this era. Deaths or loss to follow-up were right censored. Test for trends by SES quartile were evaluated at the $P < 0.05$ level. We conducted shared frailty (random effect) models to account for the nonindependence of observations within CT and sibship

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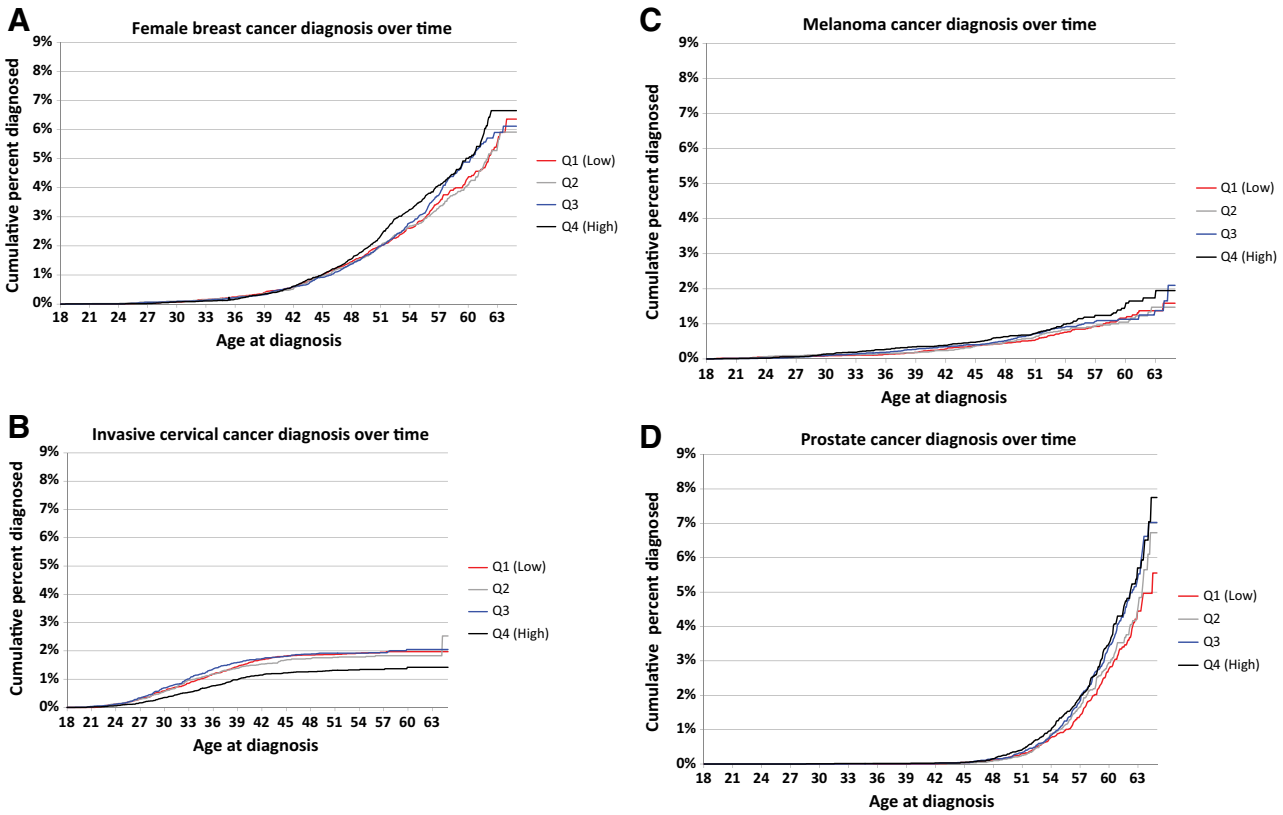

Figure 1.

Cumulative incidence in Baby-Boom Cohort 1940–1959 by cancer site and Np-SES: female breast cancer (log rank $P < 0.05$; **A**), Invasive cervical cancer (log rank $P < 0.001$; **B**), melanoma (log rank $P < 0.05$; **C**), and prostate cancer (log rank $P < 0.01$; **D**).

during *post hoc* analyses and found frailty did not change the conclusions. Therefore, we present results from the standard Cox models. All analyses were conducted using SAS v9.2.

Results

There were 60,497 (47.9%) female births and 65,838 (52.1%) male births included in the study cohort (Table 1). More than 98% were white, 48.5% were birth order 3 or higher, <2% were part of multiple births, and 98.9% of mothers were married. Seventy-five percent had siblings in the cohort, representing 65,418 unique families. Seventy-six percent were normal birth weight (2,500–4,200 g), but a significantly larger percent of Baby Boomers who later developed cancer were above the normal birth weight range (>4200 g, 5.7% vs. 2.6%). Average age of mothers was 26 years at the time of birth and mean gestational age was 39 weeks. Np-SES scores were assigned to 118,158 births (93.5%) as not all I/O codes had corresponding Np-SES scores (i.e., students, home makers). The mean and median population in each CT was 1,235 and 1,106, respectively (range 153–3,113). Although there were at least 10 cases per CT (mean = 88, median = 78, max = 223), the range of cases per CT varied by cancer site (breast 0–39, pancreas 0–6, melanoma 0–23, cervical 0–25, prostate 0–19, colorectal 0–23, lung 0–9). The correlation between Np-SES and BiCT-SES quartiles were low but significant ($P < 0.0001$) for both the continuous ($r = 0.24$) and quartile measures ($\rho = 0.20$).

SES and cancer incidence

Mean follow-up time was about 34 years from age 18 and 91% of cohort members were alive at last follow-up. A total of 46,311 individuals were alive but censored before the end of follow-up (December 31, 2009). Most were censored in 2008 because this was the latest year in which the UPDB obtained follow-up data. More females were diagnosed with invasive cancer in adulthood than males (8.6% vs. 5.8%). Just over a third (33.9%) of all female cancers were breast, followed by invasive cervical cancer (18.4%), melanoma (9.4%), colorectal (4.6%), lung (2.6%), and pancreas (0.8%). Among males, the most frequent cancer diagnosis in adulthood was prostate (25.2%), followed by melanoma of the skin (16.1%), colorectal (8.0%), lung (4.5%), and pancreas (2.3%).

Figure 1 describes the cumulative hazard functions for cancers that were significantly associated with Np-SES (log-rank $P < 0.05$): female breast (Fig. 1A), invasive cervical (Fig. 1B), prostate (Fig. 1C), and melanoma (Fig. 1D). For female breast cancer, we see an increase in incidence around age 40. By age 50, we begin to see a separation among women in the highest SES group. SES differences in the incidence of invasive cervical cancer appear much earlier in time. Women from the lowest Np-SES groups were being diagnosed with invasive cervical cancer at a higher rate than the highest SES group beginning around age 26. In addition, the differences between low and high SES groups continues to expand until around age 40, at

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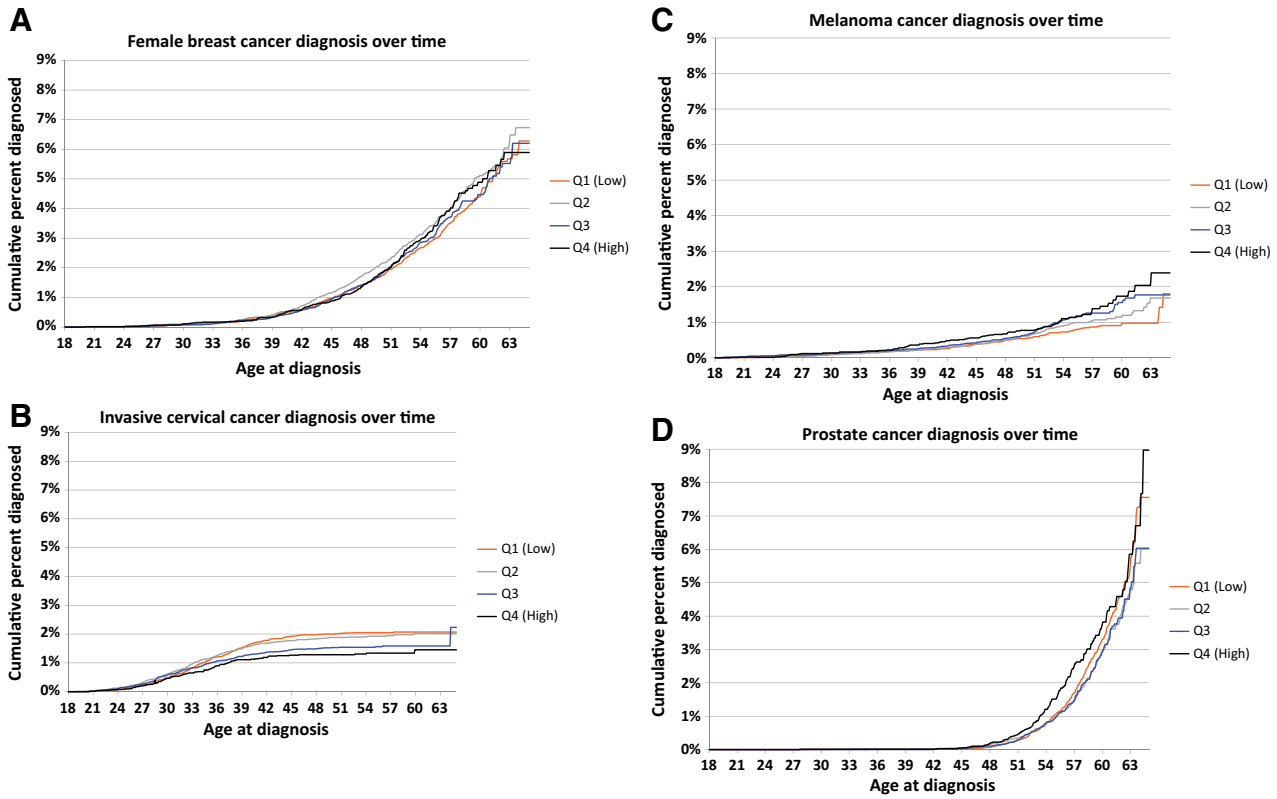

Figure 2.

Cumulative incidence in Baby-Boom Cohort 1940–1959 by cancer site and 1960 CT SES (BiCT-SES): female breast cancer (log rank $P < 0.01$; **A**), invasive cervical cancer (log rank $P < 0.01$; **B**), melanoma (log rank $P < 0.01$; **C**), and prostate cancer (log rank $P < 0.05$; **D**).

which time the SES differences of invasive cervical cancer stabilizes through age 60. Melanoma incidence follows expected patterns with the highest SES group experiencing the highest rates of melanoma. Prostate cancer incidence follows the natural history of the disease with substantial increases in incidence beginning around age 55–60, but we also see differences between the high Np-SES groups and low Np-SES groups. There were no significant associations between Np-SES and colorectal ($P = 0.12$), lung ($P = 0.06$), or pancreatic ($P = 0.33$) cancers.

Cumulative hazard functions based on neighborhood BiCT-SES for the same four cancer types show no differences in female breast cancer incidence, and slightly different patterns for the other sites when compared with individual Np-SES (Fig. 2). For cervical cancer, women in Q3 of neighborhood BiCT-SES look more similar to women in Q4, and there is no clear separation between high (Q3–Q4) versus low (Q1–Q2) neighborhood BiCT-SES groups until about age 39. For melanoma, the differences in incidence by neighborhood BiCT-SES are more pronounced between all four groups and the separation between the lowest BiCT-SES and highest BiCT-SES group are seen at an earlier age compared with individual Np-SES. Finally, for prostate cancer, neighborhood BiCT-SES differences are seen in earlier ages compared with individual Np-SES, and men in the lowest neighborhood BiCT-SES converge with men in the highest neighborhood BiCT-SES group by age 63. There were no significant associations between BiCT-SES and lung ($P = 0.23$) and pancreatic ($P = 0.11$) cancer risk in adulthood, but the log rank for colorectal cancer was significant ($P = 0.02$) with individuals at the highest BiCT-SES experiencing lower rates starting around age 60 when compared with individuals in the lower BiCT-SES groups (results not shown).

Results from multivariable proportional hazards analyses are described in Figs. 3 and 4. Women in low Np-SES groups were less likely to be diagnosed with breast cancer than women in the highest Np-SES group ($HR_{Q1/Q4} = 0.83$ 95% CI, 0.72–0.97; $HR_{Q2/Q4} = 0.81$; 95% CI, 0.69–0.96; $P_{trend} = 0.030$; Fig. 3). Similarly, men in the lower Np-SES groups had lower incidence of prostate cancer than men in the highest individual Np-SES group ($HR_{Q1/Q4} = 0.70$; 95% CI, 0.56–0.88; $HR_{Q2/Q4} = 0.76$; 95% CI, 0.60–0.96; $P_{trend} = 0.009$). For melanoma, the disparity was only evident for those born in the lowest SES group as they were significantly less likely to be diagnosed with melanoma in adulthood ($HR_{Q1/Q4} = 0.81$; 95% CI, 0.67–0.98; $P_{trend} = 0.008$). Although there was not a consistent gradient across SES quartiles, there was some indication that women born into low SES families had an increased risk for cervical cancer than women born into the highest SES group ($HR_{Q1/Q4} = 1.35$; 95% CI, 0.63–2.89; $HR_{Q2/Q4} = 1.11$; 95% CI, 0.63–1.96; $HR_{Q3/Q4} = 1.66$; 95% CI, 1.03–2.70; $P_{trend} = 0.044$).

Associations between melanoma and neighborhood BiCT-SES was similar to Np-SES ($P_{trend} = 0.001$), but were not significant for female breast cancer ($P_{trend} = 0.237$; Fig. 4). Women born into lower BiCT-SES neighborhoods had a

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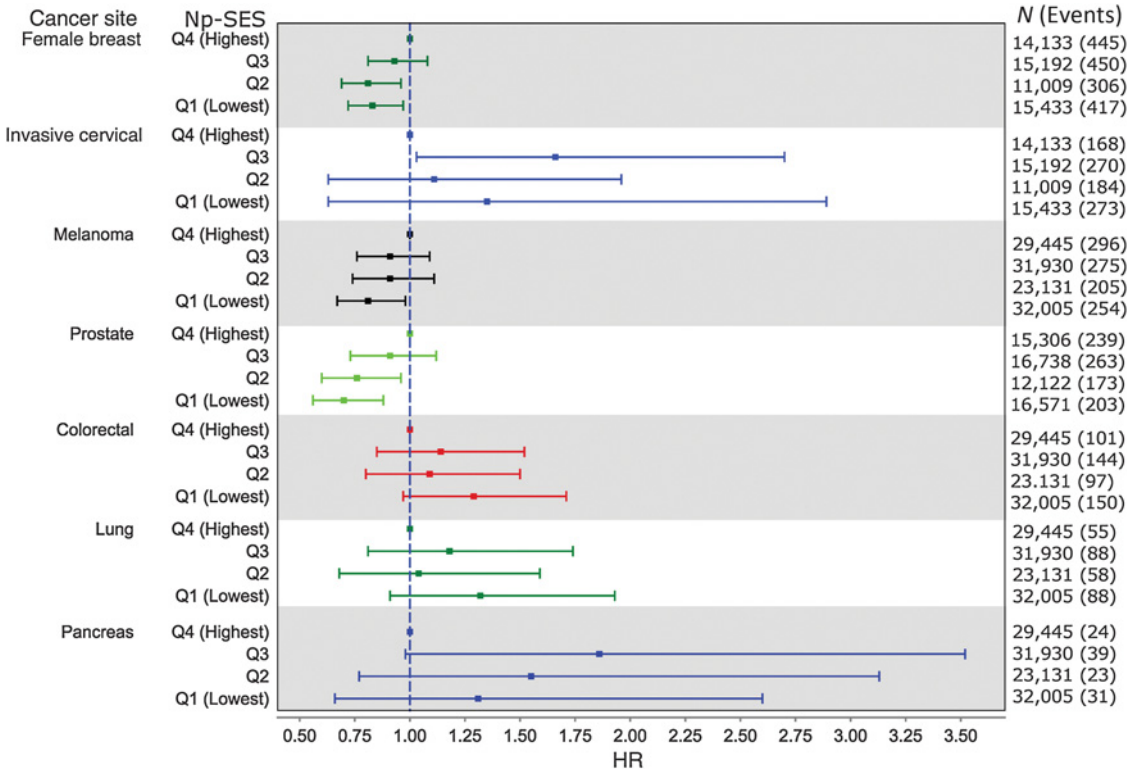

Figure 3.

HRs and 95% confidential Intervals for cancer in adulthood by cancer site and quartile of individual-level SES at birth (Np-SES), controlling for birth weight (pounds), year of birth, sibling in cohort, and gender (if applicable).

significantly increased risk for invasive cervical cancer ($HR_{Q1/Q4}$ = 1.44; 95% CI, 1.12–1.85; $HR_{Q2/Q4}$ = 1.33; 95% CI, 1.04–1.72; P_{trend} = 0.012). Prostate cancer risk was significantly lower among men born in lower SES neighborhoods across all SES quartiles ($HR_{Q1/Q4}$ = 0.76; 95% CI, 0.59–0.97; $HR_{Q2/Q4}$ = 0.65; 95% CI, 0.50–0.85; $HR_{Q3/Q4}$ = 0.68; 95% CI, 0.52–0.89; P_{trend} = 0.010). We found no significant association with either SES variable for pancreas, lung, and colon and rectal cancers; and the two SES variables performed similarly in predicting cancer risk (−2 Log L within ≤10 points).

Discussion

Population-based investigations of the relationship between SES and cancer incidence have traditionally used area-based measures from residential locations at the time of diagnosis (2–12, 16–20, 66). Two novel approaches were taken in this study by capturing SES at the individual and neighborhood levels at birth. This study also provides important information about a large and aging segment of the U.S. population (Baby Boomers) who have reached the age in which the risk of cancer increases (56, 57).

To our knowledge, only one U.S. study used population-based cancer surveillance data in combination with individual-level SES (3). Clegg and colleagues (3) linked data from the NCI's SEER Program to the National Longitudinal Mortality Study (SEER-NLMS) to assess the availability and utility of self-reported adult SES in a population-based study of cancer. The results from our study are consistent with Clegg and colleagues, in that individual-level SES disparities were detected for breast, prostate, and melanoma cancers, but unlike Clegg and colleagues (3), we were not able to detect a significant individual-level SES effect on cancers of the colon and rectum and lung. The low prevalence of smoking in Utah (67) and the relatively young age of the cohort could make finding a significant SES association for these cancers difficult to detect. The Baby-Boom cohort in this study had a maximum age at follow-up of 64 and, therefore, had not reached the age when lung, colorectal, and pancreatic cancers are commonly diagnosed (age 65–75; refs. 68–70). The differences between our study and Clegg and colleagues (3) could be attributed to operationalization of individual-level SES. Clegg and colleagues (3) defined individual-level SES as self-reported educational attainment, family income, and employment status at the time of survey completion when respondents were ages 25 years and older. We defined individual-level SES using occupational/industry data obtained at the time of birth.

For cancers with widespread screening programs implemented during the decades studied—cervical, breast, and prostate—patterns in relation to SES in the Utah Baby-Boom cohort were consistent with the literature (19, 71–79). The increased risk of prostate cancer among men born into high SES families is likely associated with greater participation in PSA screening (80). We also found that women born into high SES families were at increased risk for breast cancer, which is likely due to greater use of mammography screening (72, 73, 75, 78, 79) and higher prevalence of known risk factors associated with high SES such

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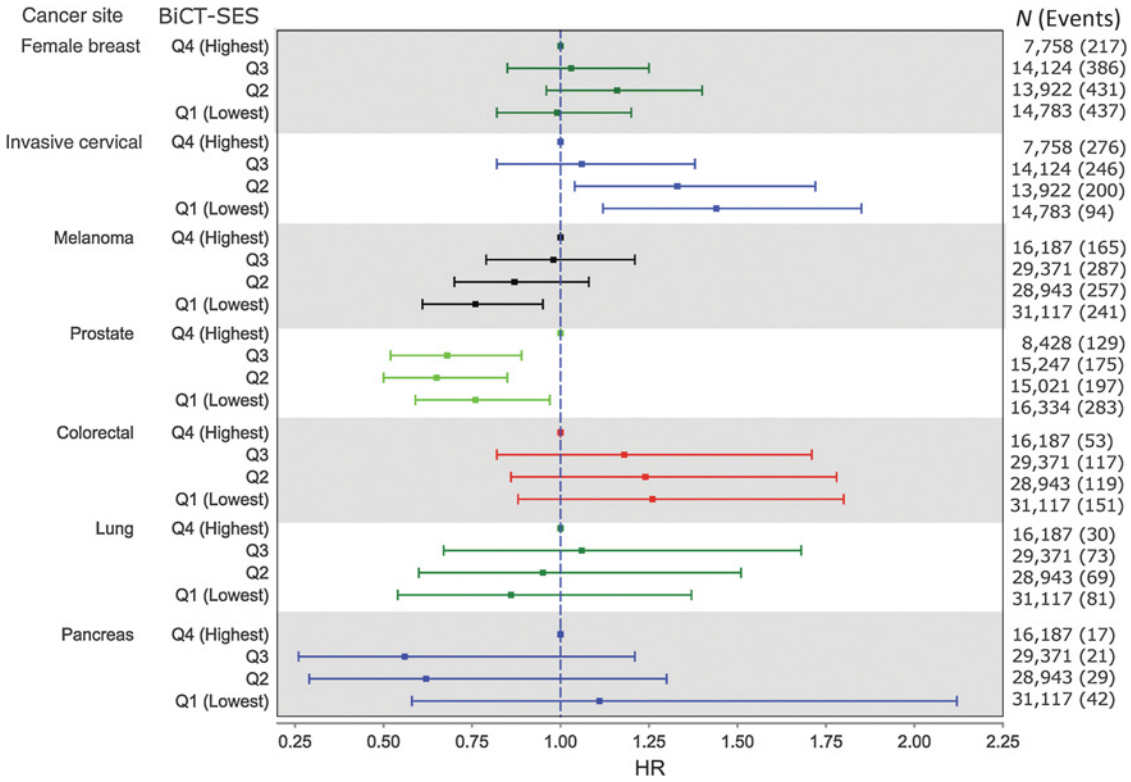

Figure 4.

HRs and 95% confidential intervals for cancer in adulthood by cancer site and quartile of neighborhood SES at birth (BiCT-SES), controlling for birth weight (pounds), year of birth, sibling in cohort, and gender (if applicable).

as older age at first child birth and nulliparity (81). For cervical cancer, we found that women from low SES groups were significantly more likely to be diagnosed with the disease, which again is consistent with the literature (19, 71, 72, 76, 78, 79). The more immediate SES disparities observed among young women diagnosed with invasive cervical cancer is likely due to disparities in access and utilization of routine PAP screening and higher prevalence of human papillomavirus (HPV) infections (82). The age-delay in SES disparities for female breast cancer was consistent with known genetic risks linked to early onset breast cancer (83).

This is one of the first studies to report a trend of increasing melanoma incidence by early-life SES. Possible mechanisms may include differences in behavioral risk or preventive factors associated with SES, and/or early-life exposures that have biologic consequences decades later (84). Given the known influence of exposure to UVA/UVB on melanoma risk, the results suggest that Utah residents with high SES had higher levels of sun exposure than low-SES residents. Examples of SES-related exposure patterns might include intermittent intense sun exposure while skiing or while traveling to sunnier climates during winter (85, 86), residence at higher elevation (87), and employment in outdoor jobs (i.e., ski resorts; ref. 88). Future investigations should address these risk factors in conjunction with SES, the role of latent, high-intensity direct exposures, and cumulative exposures that begin early in the life course; and, an examination of the geographic location of cases to assess the likelihood of clustering around high altitude areas with potentially high sun exposures.

We were also one of the first to assess both neighborhood SES at birth as well as individual-level SES at birth in relation to cancer incidence in adulthood. We found that neighborhood SES and individual-level SES performed similarly when estimating the risk of incident cancers, with one exception. For female breast cancer, individual-level SES at birth was significantly associated with increased risk, whereas neighborhood SES at birth was not. This is in direct contrast to the known positive association between breast cancer and neighborhood SES in adulthood (6, 15, 20, 78). However, when considered in the context of life course epidemiology, our findings provide evidence to support the critical period model wherein exposures to certain physical and social conductions in early life will impact cancer risk in adulthood through direct and indirect pathways (48). Our individual SES measure was derived from father's occupation at birth, which is associated with reproductive factors (46, 89) that in turn increase breast cancer risk. Because we were unable to obtain individual-level SES in adulthood and at cancer diagnosis, we were unable to examine the role of adult SES as a mediator of risk.

One of the strengths of this study is that we geocoded residential locations at birth. Unfortunately, Utah counties with the exception of Salt Lake and Weber were not assigned CTs until the 1960 Decennial Census, which may or may not be representative of the conditions in the earlier years of the cohort. The measurement error could result in information bias (22) and underestimate the true effect of SES on cancer incidence. Although our study population was limited geographically, Salt Lake

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and Weber Counties were representative of the larger Utah population.

Potential surveillance and selection bias were also concerns in this study. We addressed this by using multiple sources of residential and follow-up data from the UPDB to establish residence at age 18 with follow-up through the end of 2009. As a result, a majority of alive censored observations were not censored until 2008, thereby contributing all but 1 year of follow-up time to the analysis. Residential mobility at the time of birth might also be a concern. If outmigration was more common among those with lower SES, then we should see a larger effect because the low SES individuals who do not move would be worse off. If outmigration was more common among those with higher SES, then the high SES groups that did not move may be more like the lower SES and effect sizes would be weaker. For either high or low SES, staying may represent more intense exposures in which case it may be difficult to predict the effects on the risk estimates.

Our analysis of cancer risk was limited to the first primary invasive cancer diagnosed from 1966 to 2009. Because a few members of the cohort would have turned 18 at least 3 years before cancer surveillance began (1963), the number of cancer cases could be underestimated, but this effect should be small as incidence of cancer at ages 18 to 21 is extremely low. Restricting analyses to first primary cancers would also underestimate the true cancer risk in the cohort as individuals remain at risk for subsequent cancers even after their first diagnosis. These underestimations of cancer incidence are not expected to be related to SES, and, therefore, any bias derived from these issues is likely to be nondifferential.

Assessing the simultaneous or cumulative contributions of individual-level SES and neighborhood SES was beyond the scope of this study. Nor were we able to fully adjust for adult SES at cancer diagnosis and over the life course. Future research is needed to disentangle their independent and joint contributions and their role in the causal pathway toward cancer risk in order to identify potential interventions.

Conclusion

This study applied a method for determining early-life SES that is novel in its application to cancer epidemiology. I/O coding to determine early-life SES for epidemiologic studies should increase as populations with electronic birth certificates reach middle-aged with higher cancer risk. Through this novel approach, we observed SES associations with breast, cervical, and prostate cancer that are likely indicative of the SES differences in participation in cancer screening; and, report evidence that supports life course determinants of breast cancer incidence. The SES gradient in melanoma that we observed is intriguing and calls for future research into potentially deleterious sun/UV exposures in early childhood and cumulative exposures over the life course.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Authors' Contributions

Conception and design: A.M. Stroup, K.A. Herget, H.A. Hanson, K.A. Henry, C.J. Harrell, K.R. Smith

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Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): A.M. Stroup, K.A. Herget, H.A. Hanson, C.J. Harrell, K.R. Smith

Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): A.M. Stroup, K.A. Herget, H.A. Hanson, J.T. Butler, K.A. Henry, C.J. Harrell, C. Sweeney, K.R. Smith

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Baby Boomers and Birth Certificates: Early-Life Socioeconomic Status and Cancer Risk in Adulthood

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