



## Original Research

## Comorbidity as a predictor of racial and ethnic disparities in cancer in the United States population

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## ABSTRACT

**Aims:** This study aims to examine the racial and ethnic disparity in cancer prevalence and determine if comorbidities can explain this disparity.

**Study design:** This was a cross-sectional study.

**Methods:** The study examined cancer prevalence among adults who self-identified as White, Black, and Other races in the US population according to data from the 2017 National Health Interview Survey.

**Results:** Cancer was 58.5% [OR = 0.415; 95% CI: 0.346–0.498] and 57.5% [OR = 0.425; 95%CI: 0.346–0.522] more likely to be found in the White compared to the Black adults and White compared to Other race adults, respectively. After adjusting for the comorbidities, the odds of cancer in White adults increased marginally compared to Black adults [OR = 0.407; 95%CI: 0.338–0.490] and decreased marginally compared to Other race adults [OR = 0.462; 95%CI: 0.374–0.569] even though the odds remained significant. Ever smoking, age of 50 years or more, Former and current alcohol consumption, overweight and obesity, being female and physical inactivity were found to be significantly associated with higher odds of cancer.

**Conclusions:** This study did identify a racial and ethnic disparity in cancer prevalence between White and Black adults and White and Other adult races. However, this racial and ethnic disparity could not be explained by comorbidities.

## 1. Introduction

Cancer is a leading cause of mortality and morbidity globally with an estimated 1,806,590 new cases and 606,520 deaths occurring in 2020 in the United States (US) [1]. This translates into about 4950 cases diagnosed each day. While the etiology of most cancers is not well understood, multifactorial causes including lifestyle, infections and socioeconomic factors, are said to play a role [2,3]. Hence, cancer prevalence and mortality are affected by these multifactorial causes as well. There is a racial inequality in the prevalence and mortality of cancer cases [4,5]. Although in the last 20 years, there is a declining trend in cancer incidence and mortality, and the incidence in most types of cancers in women are lower in Black women compared to White woman, cancer mortality remains higher in Black compared to White populations [1,6,7]. Because of lower death rates in White populations, the prevalence of cancer appears higher in this race compared to Black populations. While evidence exists on this health disparity, there is insufficient data to adequately explain this variability by race and

ethnicity. Race or ethnicity alone cannot sufficiently explain this disparity either. Race surrogated cancer pathway factors such as socioeconomic factors, access to healthcare, cigarette smoking, alcohol consumption and genetic factors were found to be some explanatory factors for cancer disparities [8–10].

The racial or ethnic disparity in cancer prevalence may also be due to the racial or ethnic distribution of comorbidities. Reducing racial and ethnic disparity in cancer prevalence and mortality entails reducing the race surrogate pathway factors. This involves reducing risk factors including comorbidities. To the best of our knowledge, there is little data from population-based studies examining the role of comorbidities on the racial/ethnic disparity in cancer prevalence in the US population. Therefore, this study was designed to evaluate the relationship between cancer and race and ethnicity using the National Health Interview Survey (NHIS) adult sample data, and to examine if the racial and ethnic disparity in cancer prevalence could be explained or predicted by comorbidities. Establishing this, will help in the realignment of policies on cancer interventions and the appropriate allocation of essential

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resources for cancer programs. We hypothesized that the presence of comorbidities may be a predictor of the racial and ethnic disparity in cancer prevalence, hence controlling for known comorbidities would eliminate this disparity.

## 2. Methods

### 2.1. Data

This study utilized cross-sectional data of the 2017 National Health Interview Survey (NHIS) to examine the relationship between cancer and race and ethnicity in the US population and how the racial and ethnic cancer disparity is influenced by the distribution of comorbidities among different races. The NHIS is household survey which aims to monitor and update the health of the US population and has been conducted since 1957. The 2017 NHIS included 32,617 households with 66.5% household response rate. A face-to-face interview was conducted from one sample adult from each participating family, and 26,617 of the 33,143 eligible adults were interviewed representing 80.7% response rate. Details of the survey instrument, sampling methodology, and study protocols are published elsewhere [11]. In summary, the 2017 NHIS household survey was designed to consist of a sample of 319 primary sampling units (PSUs) drawn from about 1700 geographically defined PSUs in each of the 50 states and the District of Columbia. A PSU consisted of a county, small group of contiguous counties, or a metropolitan statistical area. The PSUs are stratified by state and subdivided into four separate panels such that each panel is representative of the US civilian noninstitutionalized population. This design is advantageous as it allows flexibility. Each household selected is mailed a letter prior to the visit of a trained interviewer who conducts face-to-face interview. The study utilized a complete case analysis protocol and a weighted sample size of 22,842 was included in the final analysis.

The 2017 NHIS was approved by the Research Ethics Review Board of the National Center for Health Statistics (NCHS) and the US Office of Management and Budget. Additionally, each participant provided an informed consent.

### 2.2. Study variables

The dependent variable was cancer and was measured as self-reported and dichotomized as “yes” or “no”. Participants in the NHIS were asked if they were ever told by a doctor, they had cancer. The independent variables included the following race and ethnicity, comorbidities, and other covariates:

### 2.3. Independent variables

**Race and Ethnicity** – Race and ethnicity was the primary predictor variable for this study. Race and ethnicity were self-reported and categorized into (a) White only, (b) Black/African American only, (c) American Indian and Alaskan Native (AIAN) only, (d) Asian only and (e) multiple race. This variable was recoded and categorized into White, Black and Others.

**Comorbidities** – Comorbidities were the secondary predictor variables which were measured by the presence of other health or disease conditions. The health or disease conditions examined in this study included hypertension, heart attack, stroke, diabetes, weak or failing kidneys, liver conditions, hepatitis, asthma, ulcer, Chronic Obstructive Pulmonary Disease (COPD), sinusitis, angina, other heart conditions, bronchitis, coronary heart disease and hypercholesterolemia. These were self-reported and dichotomized as “yes” or “no”. They were treated individually and entered into the model after examining their individual association with the dependent variable.

### 2.4. Covariates

Sociodemographic variables examined included age, sex, marital status, and employment. Age was measured as a continuous variable but dichotomized as “< 50 years” or “≥50 years” given the risk of cancer in older age [12,13]. Sex was reported as either “Male” or “Female” and both were included in the study. Marital status which was initially reported as “Married (spouse in household)”, “married (spouse not in the household)”, “married (spouse in household unknown)”, “widowed”, “divorced”, “separated”, “Never married” or “Living with partner” was dichotomized as “Married” and “Not married”. The employment status was also recoded to examine the impact of employment and unemployment in different races on cancer.

The Body Mass Index (BMI) was determined from the weight and height information that were collected as continuous variables. This was recoded into four categories as Underweight (<18.5kg/m<sup>2</sup>), Normal weight (18.5kg/m<sup>2</sup> – 24.9kg/m<sup>2</sup>), Overweight (25kg/m<sup>2</sup> -29.9kg/m<sup>2</sup>) and Obese (≥30kg/m<sup>2</sup>) using the Centers for Disease Control and Prevention cut-off points for BMI [14].

Alcohol consumption was measured by the number of drinks within a defined time period and was recoded as “Current drinker”, “Former drinker” or “Never drinker”. The smoking status was elicited with the question “Ever smoked 100 cigarettes” with “Yes” or “No” response included in the analysis. The responses “refused”, “not ascertained” and “do not know” were excluded. Physical activity which was categorized into ten groups was recoded into “Ever exercised” and “Never exercised”.

### 2.5. Statistical analysis

Weighted analysis was performed using SAS version 9.4. Frequencies and percentages were used to describe categorical variables. Chi-square statistic was used to compare the racial differences in the distribution of sociodemographic characteristics such as sex, age, BMI, marital status, physical exercise, smoking status, alcohol drinking status, and employment status. The prevalence of cancer and other comorbidities were also examined across the different races and chi-square comparative analyses were conducted to find out any differences among the racial groups.

Bivariate logistic regression was performed to examine the associations between cancer and the main predictor variable as well as other known risk factors such as smoking status, alcohol status, BMI, physical exercise, age, sex and marital status as crude odds ratios (COR). Similar analyses were performed between cancer and each of the comorbidities. Any covariate yielding a *p*-value <0.05 in the crude analyses was used in the multivariable analyses. This strategy was followed for each interaction term; however no significant association was identified. All analyses were two-sided, with a *p*-value <0.05 or included confidence intervals not containing the null value of 1 with respect to odd ratios considered statistically significant.

## 3. Results

The characteristics of the participants are presented in Table 1. A weighted total of 22,842 participants was included with 78.40% being White, 12.29% Black and 9.31% Other races. There were more females (55.01%) than males (47.99%) with most of the participants being <50 years of age (55.07%). Overall, 34.55% of the participants were obese and 32.91% were overweight. There was a higher proportion of Black adults who were obese (44.40%) compared to the Other race adults. Only 33.35% of White adults participated in some form of physical activity. The prevalence of ever smoking and current alcohol drinking was 36.14% and 67.31% respectively. 70.06% of White adults were current drinkers compared to 56.79% of Black adults and 58.01% of Other race adults. More White adults (38.83%) were ever smokers compared to Black adults (26.76%) and Other race adults (25.88%). Unemployment was 5.04% among all participants with White adults (4.38%) having the

**Table 1**  
Characteristics of the participants stratified by the race or ethnicity.

Variable	Total	White 17908 (78.40)	Black 2808 (12.29)	Other 2126 (9.31)	X [2] (df)	p-value
<b>Sex</b>						
Female	11,879 (52.01)	9191 (51.32)	1563 (55.66)	1125 (52.93)	19.0807 (2)	<0.0001
Male	10,963 (47.99)	8717 (48.68)	1245 (44.34)	1001 (47.07)		
<b>Age</b>						
<50years	12,578 (55.07)	9494 (53.02)	1695 (60.36)	1389 (65.34)	152.9387 (2)	<0.0001
≥50years	10,264 (44.93)	8414 (46.98)	1113 (39.64)	737 (34.66)		
<b>BMI</b>						
<18.5	295 (1.29)	220 (1.23)	38 (1.36)	37 (1.73)	266.7882 (6)	<0.0001
18.5–24.9	7136 (31.24)	5569 (31.10)	681 (24.26)	886 (41.70)		
25–29.9	7518 (32.91)	601 (33.57)	842 (29.97)	666 (31.32)		
≥30	7892 (34.55)	6108 (34.11)	1247 (44.40)	547 (25.24)		
<b>Marital Status</b>						
Married	12,069 (52.84)	9986 (55.76)	918 (32.70)	1166 (54.82)	521.9691 (2)	<0.0001
Not Married	10,772 (47.16)	7922 (44.24)	1890 (67.30)	960 (45.18)		
<b>Physical Activity</b>						
Ever	7556 (33.08)	5973 (33.35)	864 (30.77)	719 (33.81)	7.8522 (2)	0.0197
Never	15,286 (66.92)	11,935 (66.65)	1944 (69.23)	1407 (66.19)		
<b>Smoke</b>						
Yes	8256 (36.14)	6954 (38.83)	751 (26.76)	550 (25.88)	260.3486 (2)	<0.0001
No	14,586 (63.86)	10,954 (61.17)	2057 (73.24)	1576 (74.12)		
<b>Alcohol</b>						
Never	4481 (19.62)	2987 (16.68)	832 (29.63)	663 (31.16)	469.3592 (4)	<0.0001
Current	15,374 (67.31)	12,546 (70.06)	1594 (56.79)	1233 (58.01)		
Former	2986 (13.07)	2375 (13.26)	382 (13.59)	230 (10.82)		
<b>Job</b>						
Ever	21,693 (94.97)	17,124 (95.62)	2614 (93.08)	1955 (91.96)	77.3410 (2)	<0.0001
Never	1149 (5.04)	784 (4.38)	194 (6.92)	171 (8.04)		

BMI=Body Mass Index; X [2] – Chi square value; df = degree of freedom.

lowest unemployment value compared to Black adults (6.92%) and Other race adults (6.92%). Irrespective of race, participants who never did any physical activity, never smoked, ever employed and females were statistically higher (as presented in Table 1).

Table 2 presents the analysis on the prevalence of cancer and the comorbidities in the participants. Overall, the prevalence of cancer was 9.24%. Cancer was more prevalent among White adults (10.49%) compared to the Black adults (4.64%) and Other race adults (4.74%). The prevalence of hypertension was 27.95%, with the highest prevalence observed in Black adults (34.97%). Hypercholesterolemia (27.95%) was the most prevalent comorbidity after hypertension (29.63%) in the participants followed by asthma (13.26%) and sinusitis (12.36%). The two least reported comorbidities in the participants were angina (1.64%) and liver conditions (1.74%). Heart attack, angina, coronary heart disease (CHD), other heart conditions, COPD, ulcer and sinusitis were more prevalent in the White adults (3.01%, 1.72%, 4.38%, 8.21%, 3.44%, 6.31% and 13.14% respectively) compared to the Black adults (2.75%, 1.41%, 3.56%, 5.76%, 2.53%, 5.28% and 10.89% respectively) and Other race adults (1.98% and 1.29%, 2.75%, 4.10%, 2.02%, 4.73% and 7.75% respectively). On the other hand, stroke,

**Table 2**  
Prevalence of cancer and other comorbidities in the population stratified by race or ethnicity.

Condition	White	Black	Other	X [2] (df = 2)	P-value	
<b>Cancer</b>						
Yes	2110 (9.24)	1879 (10.49)	130 (4.64)	101 (4.74)	155.6766	<0.0001
No	20,732 (90.76)	16,029 (89.51)	2678 (95.36)	2025 (95.26)		
<b>Hypercholesterolemia</b>						
Yes	6385 (27.95)	5188 (28.97)	650 (23.13)	547 (25.74)	46.7493	<0.0001
No	16,457 (72.05)	12720 (71.03)	2158 (76.87)	1579 (74.26)		
<b>Hypertension</b>						
Yes	6767 (29.63)	5255 (29.35)	982 (34.97)	530 (24.92)	61.7434	<0.0001
No	16075 (70.37)	12653 (70.65)	1826 (65.03)	1596 (75.08)		
<b>Heart Attack</b>						
Yes	658 (2.88)	539 (3.01)	77 (2.75)	42 (1.98)	7.4353	0.0243
No	22184 (97.12)	17369 (96.99)	2731 (97.25)	2084 (98.02)		
<b>Stroke</b>						
Yes	696 (3.05)	548 (3.06)	107 (3.81)	42 (1.96)	14.1186	0.0009
No	22146 (96.95)	17360 (96.94)	2701 (96.19)	2084 (98.04)		
<b>Diabetes</b>						
Yes	2184 (9.56)	1663 (9.29)	312 (11.12)	209 (9.8)	9.6507	0.0080
No	20658 (90.44)	16245 (90.71)	2496 (88.88)	1917 (90.20)		
<b>Renal condition</b>						
Yes	459 (2.01)	369 (2.06)	65 (2.30)	25 (1.18)	8.8413	0.0120
No	22383 (97.99)	17539 (97.94)	2743 (97.70)	2101 (98.82)		
<b>Liver condition</b>						
Yes	397 (1.74)	314 (1.75)	36 (1.28)	48 (2.23)	6.4276	0.0402
No	22445 (98.26)	17594 (98.25)	2772 (98.72)	2078 (97.77)		
<b>Hepatitis</b>						
Yes	613 (2.68)	482 (2.69)	64 (2.29)	68 (3.18)	3.6719	0.1595
No	22229 (97.32)	17426 (97.31)	2744 (97.71)	2058 (96.82)		
<b>Asthma</b>						
Yes	3030 (13.26)	2407 (13.44)	400 (14.23)	223 (10.48)	17.1375	0.0002
No	19812 (86.74)	15501 (86.56)	2408 (85.77)	1903 (89.52)		
<b>Ulcer</b>						
Yes	1379 (6.04)	11300 (6.31)	149 (5.28)	101 (4.73)	11.5951	0.0030
No	21463 (93.96)	16778 (93.69)	2659 (94.72)	2025 (95.27)		
<b>COPD</b>						
Yes	729 (3.19)	615 (3.44)	71 (2.53)	43 (2.02)	16.91183	0.0002
No	22113 (96.81)	17293 (96.56)	2737 (97.47)	2083 (97.98)		
<b>Sinusitis</b>						
Yes	2824 (12.36)	2353 (13.14)	306 (10.89)	165 (7.75)	57.3913	<0.0001
No	20018 (87.64)	15555 (86.86)	2502 (89.11)	1961 (92.25)		
<b>Angina</b>						
Yes	375 (1.64)	308 (1.72)	40 (1.41)	28 (1.29)	3.2442	0.1975
No	22467 (98.36)	17600 (98.28)	2768 (98.59)	2098 (98.71)		
<b>Other Heart Conditions</b>						
Yes					60.2900	<0.0001

(continued on next page)

Table 2 (continued)

Condition	White	Black	Other	X [2] (df = 2)	P-value
	1719 (7.52)	1470 (8.21)	162 (5.76)	87 (4.10)	
No	21123 (92.48)	16438 (91.79)	2646 (94.24)	2039 (95.90)	
<b>Bronchitis</b>					
Yes	759 (3.32)	615 (3.44)	101 (3.58)	43 (2.01)	12.6346 0.0018
No	22083 (96.68)	17293 (96.56)	2707 (96.42)	2083 (97.99)	
<b>CHD</b>					
Yes	944 (4.13)	785 (4.38)	100 (3.56)	59 (2.75)	15.4237 0.0004
No	21898 (95.87)	17123 (95.62)	2708 (96.44)	2067 (97.25)	

COPD – Chronic Obstructive Pulmonary Disease; CHD – Coronary Heart Disease; X [2] = Chi square value; df = degree of freedom.

diabetes, renal failure, asthma and Bronchitis were more prevalent among the Black adults (3.81%, 11.12%, 2.30%, 14.23% and 3.58%) compared to the White adults (3.06%, 9.29%, 2.06%, 13.44% and 3.32%) and Other race adults (1.96%, 9.80%, 1.18%, 10.48% and 2.01%). Hepatitis and liver conditions were the only two comorbidities more prevalent among Other race adults (hepatitis and liver conditions: 3.18% and 2.23%, respectively) compared to the Black adults (hepatitis and liver conditions: 2.29% and 1.28%, respectively) and White adults (hepatitis and liver conditions: 2.69% and 1.75%, respectively). Apart from angina (p-value = 0.1975) and hepatitis (p-value = 0.1595), significant differences in prevalence were observed across racial and ethnic groups.

From Table 3, hypertension was the most prevalent comorbidity among the cancer subjects with 52.11% of all cancer subjects reported having hypertension. This was followed by hypercholesterolemia with 48.68% of cancer subjects reporting to have high cholesterol. 3.62% and 4.58% of the subjects with cancer compared to only 1.55% and 1.75% in the subjects with no cancer reported some liver and renal conditions, respectively. Diabetes was 15.74% prevalent in the cancer subjects compared to 8.93% in those without cancer whilst CHD was 11.43% among the cancer compared to 3.39% among those without cancer. All comorbidities were significantly higher among those with cancer compared to those without cancer.

Participants who were overweight and obese were 17.8% [OR = 1.178; 95% CI: 1.052–1.319] and 30.0% [OR = 1.30; 95%CI: 1.010–1.265] more likely to have cancer compared to participants with normal weight. Also, participants who smoked and drank alcohol were 79.9% [OR = 1.799; 95% CI: 1.644–1.969] and 23.6% [OR = 1.236; 95% CI: 1.091–1.401] more likely to have cancer compared to participant who did not smoke and drink. Being ≥50 years (p-value <0.0001), a female (p-value = 0.0058) and married (p-value <0.0001) were significantly associated with cancer prevalence as presented in Table 4.

Cancer was 58.5% [OR = 0.415; 95% CI: 0.346–0.498] and 57.5% [OR = 0.425; 95% CI: 0.346–0.522] more likely to be prevalent in the White adults compared to Black and Other race adults, respectively. After adjusting for all comorbidities the odds of cancer in White adults increased marginally compared to Black adults [OR = 0.407; 95% CI: 0.338–0.490] and decreased marginally compared to Other races [OR = 0.462; 95% CI: 0.374–0.569] even though the odds remained significant as seen in Table 5.

4. Discussion

Cancer remains a significant cause of disability and death globally. Multiple risk factors have been found to be associated with cancer. Race and ethnicity have long been established as a risk factor. However, race and ethnicity have not sufficiently explained the racial disparity in the

Table 3

Prevalence of the comorbidities in the participants stratified by the cancer status.

Comorbidity	Total	Cancer status		X [2](df = 2)	p-value
		Yes	No		
<b>Hypercholesterolemia</b>					
Yes	6385 (27.95)	1027 (48.68)	5358 (25.84)	496.1679	<0.0001
No	16457 (72.05)	1083 (51.32)	15384 (74.16)		
<b>Hypertension</b>					
Yes	6767 (29.63)	1100 (52.11)	5668 (27.34)	563.8648	<0.0001
No	16075 (70.37)	1011 (47.89)	15064 (72.66)		
<b>Heart Attack</b>					
Yes	658(2.88)	143(6.74)	516(2.49)	123.7962	<0.0001
No	22184 (97.12)	1968 (93.26)	20216 (97.51)		
<b>Stroke</b>					
Yes	696(3.05)	161(7.62)	536(2.58)	164.2301	<0.0001
No	22146 (96.95)	1950 (92.38)	20196 (97.42)		
<b>Diabetes</b>					
Yes	2184 (9.56)	333 (15.74)	1852 (8.93)	102.5969	<0.0001
No	20658 (90.44)	1778 (84.26)	18880 (91.07)		
<b>Renal condition</b>					
Yes	459(2.01)	97(4.58)	362(1.75)	78.3828	<0.0001
No	22383 (97.99)	2014 (95.42)	20369 (98.25)		
<b>Liver condition</b>					
Yes	398(1.74)	77(3.62)	321(1.55)	47.9204	<0.0001
No	22444 (98.26)	2034 (96.38)	20410 (98.45)		
<b>Hepatitis</b>					
Yes	614(2.68)	83(3.92)	531(2.56)	13.5436	0.0002
No	22228 (97.32)	2028 (98.08)	20201 (97.44)		
<b>Asthma</b>					
Yes	3030 (13.26)	321 (15.22)	2708 (13.06)	7.7581	0.0053
No	19812 (86.74)	1789 (84.78)	18023 (86.94)		
<b>Ulcer</b>					
Yes	1379 (6.04)	247 (11.69)	1132 (5.46)	131.1620	<0.0001
No	21463 (93.96)	1863 (88.31)	19599 (94.54)		
<b>COPD</b>					
Yes	729(3.19)	166(7.88)	563(2.71)	165.5998	<0.0001
No	22113 (96.81)	1944 (92.12)	20169 (97.29)		
<b>Sinusitis</b>					
Yes	2824 (12.36)	384 (18.21)	2439 (11.76)	73.4081	<0.0001
No	20018 (87.64)	1726 (81.79)	18292 (88.24)		
<b>Angina</b>					
Yes	375(1.64)	90(4.25)	285(1.38)	97.7889	<0.0001
No	22467 (98.36)	2021 (95.75)	20446 (98.62)		
<b>Heart Conditions</b>					
Yes	1719 (7.52)	348 (16.46)	1371 (6.61)	266.9406	<0.0001
No	21123 (92.48)	1763 (83.54)	19360 (93.39)		
<b>Bronchitis</b>					
Yes	759(3.32)	121(5.71)	638(3.08)	41.4870	<0.0001
No	22083 (96.68)	1990 (94.29)	20093 (96.92)		
<b>CHD</b>					
Yes	944(4.13)	241 (11.43)	702(3.39)	312.7193	<0.0001
No	21898 (95.87)	1869 (88.57)	20029 (96.61)		



COPD – Chronic Obstructive Pulmonary Disease; CHD – Coronary Heart Disease; X<sup>2</sup> = Chi square value; df = degree of freedom.

**Table 4**  
Risk factors for cancer in the participants.

Covariate	Odd ratio	95% CI	p-value
<b>Smoke</b>			
No	1		
Yes	1.799	1.644–1.969	<0.0001
<b>Alcohol</b>			
Never	1		
Current	1.236	1.091–1.401	0.0009
Former	2.023	1.736–2.358	<0.0001
<b>BMI</b>			
Normal	1		
Underweight	0.951	0.620–1.457	0.8166
Overweight	1.178	1.052–1.319	0.0045
Obese	1.130	1.010–1.265	0.0327
<b>Physical Activity</b>			
Never	1		
Ever	1.335	1.208–1.475	<0.0001
<b>Age</b>			
<50years	1		
≥50years	8.420	7.440–9.528	<0.0001
<b>Sex</b>			
Male	1		
Female	1.135	1.037–1.242	0.0058
<b>Marital Status</b>			
Nonmarried	1		
Married	1.564	1.426–1.715	<0.0001

CI = Confidence Interval.

**Table 5**  
Racial or ethnic disparity in cancer prevalence adjusting for comorbidities.

Race	Unadjusted		Adjusted	
	OR	95%CI	OR	95% CI
White	1	Referent	1	Referent
Black	0.415	0.346–0.498	0.407	0.338–0.490
Other	0.425	0.346–0.522	0.462	0.374–0.569

OR = Odd ratio; CI = Confidence Interval.

prevalence, incidence, and mortality of cancer. This study reevaluated some traditional cancer risk factors such as cigarette smoking, age, physical activity, alcohol consumption and body mass index and examined comorbidity as an explanatory variable for the racial and ethnic disparity in cancer using prevalence odds ratios. Ever smoking, age of 50 years or more, former, and current alcohol consumption, overweight and obesity, being female and physical inactivity were found to be significantly associated with higher odds of cancer. There was not enough evidence indicating that comorbidities were a significant predictor of the racial/ethnic disparity in cancer prevalence.

Physical inactivity resulted in 33.5% more cancer cases compared to people who ever exercised. Similar positive associations between the risk of some cancers and physical activity have been reported [15–17]. There are several known mechanisms that establish the impact of physical activity on cancer risk. While some of these mechanisms may be specific to some particular cancers, one unifying mechanism suggest that physical inactivity reduces the sensitivity to insulin thereby leading to a growth promotional environment and hence facilitating neoplasia [17]. Again, physical activity may improve the non-specific immune system which can offer protection against uncontrolled cellular growth. While being underweight was observed to be protective against cancer compared to normal weight, overweight and obese were associated with increased cancer risk. Wang et al., observed 0.65% of cancer cases in the Chinese population was attributable to overweight and obesity combined [18]. Physical activity levels are associated with overweight and obesity outcomes. Physical inactivity leads to gain in weight and

subsequently overweight and/or obesity [19].

People who were 50 years or older were 7.42 times more likely to have cancer compared to those younger than 50 years. Aging comes with a lot of biological and physiological changes including a decline in organ function. Mechanisms involved include telomere attrition, loss of proteostasis, altered metabolism, stem cell function and cellular senescence [20]. Cancer is therefore, considered an aging disease even though the mechanisms remain unclear. Other traditional factors such as ever smoking, alcohol status and sex remained significantly associated with cancer prevalence as observed in previous studies [21–24]. People who ever smoked were 80% more likely to have or have had cancer compared to those who have never. Smoking affects nearly every organ and system of the body thereby reducing the overall health of the person. The carcinogens in the cigarette cause a wearing of the tissues lining the lungs where the cells eventually are forced to act abnormally after a series of repairs. Smoking also leads to inflammation and a reduction in the immune function. Alcohol also causes damages to the body. It can be converted to acetaldehyde, a chemical substance which has the potential to damage the human DNA which can lead to cancer. Thus, this study found that adults who currently drank alcohol were 23.6% more likely to have or have had cancer compared to those who have never consumed any alcohol. Further, former alcohol consumers were 2.023 times as likely to be a cancer case compared to adults who never consumed alcohol.

Comorbidities have long been acknowledged as risk for cancer occurrence and mortality. All comorbidities included in this study were significantly more prevalent in the cancer subjects compared to the no cancer subjects. Hypertension was the most common comorbidity in both groups, followed by hypercholesterolemia. Some drugs used in the treatment of cancer, especially angiogenesis inhibitors, have been found to significantly increase the risk of hypertension[25–27]. Even though the mechanism is not well understood, the drugs used in cancer treatment have anti-vascular endothelial growth factor (VEGF) antibody and certain tyrosine kinase inhibitors associated with endothelial dysfunction due to reduced nitric oxide bioavailability. This can lead to increased vascular and renal endothelin production. Endothelin causes increased vascular tone; decreased density of micro-vessels; and renal thrombotic microangiopathy with secondary glomerular structural and functional changes that lead to proteinuria and hypertension [26].

Cancer was 58.5% and 57.5% more likely in White adults compared to the Black adults and Other race adults, respectively. Cancer prevalence is highest in the White adults because of higher cancer survival rates. Cancer survival in Black adults are lower and mostly attributed to socioeconomic factors including access to healthcare [28,29]. After adjusting for comorbidities, the prevalence odds did not change much and remained significantly different across the different racial and ethnic groups with cancer being 59.3% and 53.8% more likely in White adults compared to Black adults and Other race adults, respectively. This is likely due to the variation in the distribution of different comorbid conditions. For instance, while hypercholesterolemia, heart attack, liver conditions, COPD, hepatitis, ulcer and CHD were significantly higher in White adults compared to the Black adults, hypertension, stroke, diabetes, asthma and renal conditions were significantly higher in the Black adults compared to the White adults.

This study examined the possibility of comorbidity being a predictor of the racial and ethnic disparity in cancer prevalence in the United States. The findings indicate that comorbidities could not explain or predict the reasons for the higher cancer prevalence in the White adults compared to the Black and Other race adults. Again, this study identified traditional cancer risk factors, such as smoking, alcohol consumption, physical inactivity, and age, were significantly associated with cancer prevalence.

While this study has several strengths including the large sample size, the randomization in the data collection and the generalizability of the findings to US populations, there are some limitations. First, the data from the cross-sectional study design limits the ability to know when the

development of cancer disparities occurred in the population. Furthermore, the lack of a temporal relationship complicates the interpretation of whether comorbidities such as hypertension, hypercholesterolemia and diabetes occurred before the cancer or vice versa. Again, the race and ethnicity data collected could not be separated into specific races such as African Americans, Caucasians, Hispanics and American Indian and Alaskan Native. Thus, the analyses were limited to the use of Black, White and Other races. As mentioned previously, the cancer and comorbidity data were based on self-reporting and hence could have suffered some misclassification reporting and recall biases especially for the comorbidities. These biases might be non-differential misclassification and therefore are unlikely to influence this study's outcome substantially. Finally, even though the analyses adjusted for all comorbidities that were reported, the list of possible comorbid conditions is inexhaustible and hence residual confounding remained. However, it is unlikely that the study findings are driven solely by residual confounding.

In summary, this study found a racial disparity in cancer prevalence; however, this disparity could not be explained by the presence of comorbidities. Therefore, there is a need to examine other risk factors along with comorbidities to better understand the racial disparity in cancer prevalence in the US.

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## Ethical approval

The study did not require any ethical approval since the data was taken from a secondary source which was coded anonymously and not linked to individuals that could be traced.

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## Declaration of competing interest

None declared.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.puhip.2021.100175>.

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