

RURAL, SUBURBAN AND URBAN DIFFERENCES IN THE SELF-DIAGNOSIS OF CORONARY HEART DISEASE IN THE UNITED STATES

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Summary. This study explores rural, suburban and urban differences in coronary heart disease (CHD) using the 2005 Behavior Risk Factor Surveillance Survey conducted in the United States. Although areal context is not often considered in morbidity studies, this study evaluates the importance of place of residence given that areas offer differential access to health infrastructures and different contextual factors that could affect health. Also examined is the role of geographic heterogeneity on the recent racial divergence in CHD in the United States. Results indicate that area of residence is associated with CHD diagnosis, net of health and demographic variables. The area-stratified analysis documents that rural residents are most impacted by exercise and smoking, while being male or above age 50 are most detrimental for suburban residents. In addition, the racial divergence in CHD is driven by differences in rural locales. These findings indicate a disparate impact of geography on CHD and highlight the need for health research to take into account areal context.

Introduction

Life expectancy estimates indicate that the mortality gap between Whites and Blacks in the United States has narrowed consistently over the past century (National Center for Health Statistics, 2006). More specifically, in 1900, the difference in life expectancy between Whites and Blacks was 14.6 years. By 2002, however, that difference fell to 5.4 years (National Center for Health Statistics, 2006). This decrease is primarily the result of a massive decline in communicable diseases and advances in medical technology (Manton & Singer, 2002). These factors also aided in the shrinking of various racial disparities in cause-specific mortality. For example, Blacks and Whites now have similar probabilities of dying from certain diseases (e.g. liver and colon cancer), outcomes that reflect a 50-year trend in the convergence of these racial differences (Mensah *et al.*, 2005; National Center for Health Statistics, 2006).

Although the racial disparity in life expectancy and some cause-specific mortalities has narrowed, mortality due to coronary heart disease (CHD) has recently experienced a racial divergence (Ford & Giles, 2003). Although CHD is now the leading cause of death among Americans (Lethbridge-Çejku & Vickerie, 2005), Blacks are less likely to be diagnosed with CHD than Whites (Davidson *et al.*, 2000; Knox *et al.*, 2002). In addition, those Blacks diagnosed with CHD tend to die sooner than Whites with the same disease (Mensah *et al.*, 2005). Blacks, however, are more likely to be diagnosed with hypertension, a precursor to CHD (Liu *et al.*, 1996). This trend indicates that the racial disparity in CHD is not necessarily due to diagnosis, but instead could be attributable to various other factors (Keil *et al.*, 1977; James *et al.*, 1984; Dressler, 1990, 1991; Dressler, 1993; Jonas *et al.*, 1997; Dressler *et al.*, 1998; Dyer *et al.*, 1999; Jonas & Lando, 2000; Levenstein *et al.*, 2001; Swapan *et al.*, 2002; Schum *et al.*, 2003; Stamler *et al.*, 2003).

Medical research examining racial disparities in health typically focuses on lifestyle differences. The risk factors that tend to be more pronounced among Blacks include: high levels of sedentary behaviour, low vegetable or fruit intake, a higher likelihood of smoking, high blood pressure and diabetes (Dressler *et al.*, 2003).

However, prior national and international research has clearly documented the importance of geographical context on disease prevalence and health outcomes. For instance, Yankauer's (1950) pioneering work demonstrated how residential segregation among African Americans during the 1940s was linked to high infant mortality rates (IMR). Polednak's (1996) more recent research provided additional insights into the effect of segregation on health as he established a direct relationship between segregation and IMR in 92 Metropolitan Statistical Area (MSAs). Internationally, Howe's (1964, 1986) examination of disease mortality in the United Kingdom illustrated that place of residence had a remarkable impact on cause-specific mortality. In particular, Howe asserted that places have both unique biological mechanisms that operate to concentrate certain pathologies (e.g. the number of mosquitoes that carry malaria) and socio-cultural resources (e.g. laws regarding public sanitation and pollution sites). As such, places are not only inhabited by ill persons, but also the conditions within the place that can create illness within a population.

Coronary heart disease continues to be unequally spread across the United States and to affect subpopulations in different ways (Barnett & Halverson, 2000, 2001). For instance, in a community-based sample, Cort & Stewart-Fahs (2001) found that rural Black women had significantly lower mortality rates from CHD than did the general population of US women. In contrast, Barnett & Halverson (2000) discovered that over time, Black women have seen a major decrease in CHD-related deaths, particularly in the urban South. Other scholars, however, have found that urban areas are plagued by external factors that are directly associated with negative health outcomes, such as environmental dumping (Anderton *et al.*, 1994; Davidson & Anderton, 2000) and poor air quality (Pastor Jr *et al.*, 2004). Thus, there is some disagreement at present regarding the extent to which areal context plays a role in CHD.

The purpose of this research is to examine the role of geographic heterogeneity in an attempt to account for the recent racial divergence in CHD. Specifically, this

research draws from the health behaviour model, which posits that racial differences in CHD could be due to the discrete behaviours voluntarily adopted by individuals (Dressler *et al.*, 2003). The use of this model will enable us to determine if there are any geographic differences in the likelihood of having CHD when controlling for health behaviours. By modelling the differential effects of health behaviours based on area of residence, it becomes possible to compare the relative effects of certain health behaviours and demographic characteristics on having CHD *within* and *across* geographic locales.

Race, socioeconomic status and health

Socioeconomic status (SES) is sometimes used to explain how racial inequality in CHD persists. However, there are two reasons why SES should be used with caution in race-based health research. First, there is an 'inextricability of race and socioeconomic status' (Balibar & Wallerstein, 1992/96) such that the mechanisms that produce the class-based differences in morbidity and health are really race-based differences. It has been argued in early literature that controlling for SES either causes racial disparities to disappear or reveals the 'true' effect of race (Kaufman *et al.*, 1997; Dressler *et al.*, 2003). However, very few studies have found this to be the case (Keil *et al.*, 1977). The SES indicators typically considered are some combination of education, income or occupation (status or prestige). These three measures are each related in different ways to health and disease. For example, income reflects spending power and medical care options; occupation measures monitor work exposure and physical activity; while education affects knowledge about diseases. By controlling for these synergistic measures, one may not be uncovering the true effect of SES. Indeed, review articles by Williams (1990) and Williams & Collins (1995) suggest that controlling for SES may exacerbate race-based differences in morbidity and disease, rather than explain it.

Second, individual SES may be of lesser importance than areal SES. For example, there is a correlation between individual income and neighbourhood levels of poverty (LeClere *et al.*, 1997). Likewise, access to quality health care for preventative or chronic care may be more dependent on neighbourhood characteristics than those of the individuals who make up a given neighbourhood (Geronimus *et al.*, 2001). As such, using individual SES as a proxy for areal SES is conceptually problematic.

Because of the aforementioned substantive concerns, SES will not be considered in the analyses presented below. In addition, the inclusion of the SES measures in the dataset examined (to be discussed in additional detail below) would result in a substantial reduction in sample size due to missing values for the income and education measures. Although imputation procedures such as logistic iterative regression can be utilized, the authors believe it is important to maintain the integrity of the population dataset by including all individuals regardless of SES reporting.

Data and Methods

This research utilizes the 2005 Behavioral Risk Factor Surveillance Survey (BRFSS) data to address whether distinct areas of residence have different impacts on acquiring

CHD (Centers for Disease Control and Prevention, 2006a). The BRFSS is a collaborative project of the Centers for Disease Control (CDC) and US states and territories. The BRFSS, administered and supported by CDC's Behavioral Surveillance Branch, is an ongoing data collection programme designed to measure cross-sectional behavioural risk factors in the adult population (i.e. 18 years of age or older) living in households. The BRFSS includes all 50 US states, the District of Columbia, Puerto Rico, Guam and the Virgin Islands. This study's sample is limited to the 50 states and the District of Columbia.

The objective of the BRFSS is to collect uniform, state-specific data on preventive health practices and risk behaviours that are linked to chronic diseases, injuries and preventable infectious diseases in the adult population. Factors assessed by the BRFSS include tobacco use, health care coverage, HIV/AIDS knowledge and prevention, physical activity, and fruit and vegetable consumption. Data are collected from a random sample of adults (one per household) through a telephone survey. Respondents are identified through telephone-based methods. Although approximately 95% of all US households have telephones, coverage ranges from 87 to 98% across states and varies for subgroups (Mokdad *et al.*, 2003). In particular, people living in the South, minorities and those in lower socioeconomic groups typically have lower telephone coverage. There is no direct method of compensating for non-telephone coverage in the BRFSS.

The total sample for the 2005 BRFSS includes 356,112 persons. In the present study, those records with missing data were not analysed. Likewise, because of small cell sizes, persons who were not identified as Black or White were excluded. As such, the sample analysed in this study includes 270,635 persons.

The BRFSS is a rich, national-level dataset that can adequately test rural, suburban and urban differences in CHD (Centers for Disease Control and Prevention, 2006b). This study's main dependent variable is self-reported CHD diagnosis, which is measured by respondents' reports of whether or not they have ever been diagnosed with CHD. The focal independent variable is area type. Area type is measured by a variable that indicates if the respondent lives in a MSA, in a suburban area or outside of an MSA. If the respondent lives in an MSA, the respondent is coded as urban. Likewise, if the respondent lives in a suburban area, or outside of a city centre, they are coded as suburban. Lastly, those living outside of an MSA are coded as rural. This method of areal coding is consistent with other research using the BRFSS (Rohrer *et al.*, 2005).

Several independent variables monitor key aspects of health and health behaviour. Body mass index (BMI) is a continuous measure of body fat based on height and weight that applies to both adult men and women. Body mass index was first divided into four categories, indicating underweight, normal weight, overweight and obese. These divisions correspond to the CDC guidelines for defining these categories (NHLBI Obesity Education Initiative Expert Panel, 1998). However, building on more recent research, the obese category was further sub-divided into the groupings moderately obese, severely obese and extremely obese (see Goldberg *et al.*, 2006). Thus, BMI was coded as six different categories. Exercise rigour indicates the number of days per week respondents undertake at least 10 minutes of physical activity excluding work-related endeavours. Exercise rigour is further separated into tertiles of

low (0–2 days), *medium* (3–5 days) and *high* (6–7 days). Smoking status indicates whether respondents are current smokers, former smokers or have never smoked.

Three demographic measures are used in these analyses. Race is captured by a binary variable indicating whether the respondent is Black or White. Because the sample is restricted to Blacks and Whites, only one race variable is created. Gender is coded as a dummy variable with females as the contrast category. Age is partitioned into six groups as follows: less than 30, 30–39, 40–49, 50–59, 60–69 and greater than 70.

Logistic regression models are utilized to determine the effects of area on CHD. Results are reported as odds ratios, which are exponentiated from the standardized logistic regression coefficient estimates. All data manipulation and analyses were conducted using Stata 9.2.

Results

Table 1 presents the unweighted descriptive statistics of the health and demographic variables controlling for area type. Most respondents resided in suburban and urban areas (39.3% and 37.4%, respectively) while rural residents constitute the least-represented area (23.4%). Although rural residents have the smallest sample presence, they have the highest percentage of CHD diagnoses, as over 6% of the rural subsample indicated they were diagnosed with CHD. Suburban and urban residents reported slightly lower percentages of CHD diagnoses, at 5.4% and 5.2% respectively. The distribution of BMI is similar for all areas. That is, about 1.5% are underweight, 35% are normal weight, 39% are overweight, 15.6% are moderately obese, 5.3% are severely obese and around 3% are extremely obese. In addition, exercise rigour and smoking status are similarly distributed among rural, suburban and urban locales.

Two of the three demographic variables indicate some variation by area type. Gender was the measure that did not, however, as similar proportions of men and women were interviewed in all three areas. However, Blacks comprise a higher proportion of the urban sample (16.3%) than they do for the suburban (7.2%) or rural ones (6.1%). Furthermore, although the age distribution is approximately normal for all area types, urban areas have the highest proportion of the young, while rural areas have the highest proportion of the elderly. Meanwhile, suburban respondents were more likely to be between the ages of 40 and 59. This age–region phenomenon is pertinent to our research question as it suggests that the geographic variation in CHD could be a product of the age-specific residential patterns that exist within American society. The multivariate analyses presented will explore this age–region phenomenon in additional detail.

Prior to presenting the analyses stratified by area of residence, first the multivariate results computed for the entire sample are discussed. The zero-order model (not shown) indicates significant differences in the propensity with which rural, suburban and urban residents acquire CHD. The key finding is that area of residence is a strong predictor of CHD diagnosis, as suburban and urban residents had significantly lower odds of being diagnosed than did rural residents. This finding is inconsistent with some prior research which found persons in urban areas associated with neighbourhood disadvantage are more likely to have CHD than residents of

Table 1. Unweighted descriptive statistics of health and demographic variables (as percentages) by area type

	Rural	Suburban	Urban
CHD diagnosed			
Yes	6.13	5.39	5.18
No	93.87	94.61	94.82
Body mass index			
Underweight	1.52	1.46	1.73
Normal weight	34.33	35.08	36.28
Overweight	39.09	39.50	38.31
Moderately obese	16.18	15.64	15.02
Severely obese	5.58	5.32	5.38
Extremely obese	3.29	2.99	3.28
Exercise rigour			
High	40.21	38.64	38.13
Medium	38.28	40.42	40.55
Low	21.51	20.93	21.32
Smoking status			
Never smoked	50.56	52.09	52.38
Former smoker	29.03	28.98	28.31
Current smoker	20.41	18.93	19.31
Race			
White	93.86	92.77	83.72
Black	6.14	7.23	16.28
Gender			
Female	61.81	61.53	62.43
Male	38.19	38.47	37.57
Age group (years)			
Less than 30	9.94	9.64	12.03
30–39	13.79	16.28	16.17
40–49	18.97	21.22	19.26
50–59	21.21	20.77	20.46
60–69	16.90	15.59	17.49
70+	19.19	16.51	17.49
<i>N</i> (%)	62,873 (23.35)	107,671 (39.28)	100,091 (37.38)

Source: 2005 Behavior Risk Factor Surveillance Survey.

Note: Totals may not add to 100% due to rounding.

other areas (Winkleby *et al.*, 2007). However, prior research also clearly documents that Blacks predominately reside in US urban areas (Bajari & Kahn, 2005), a result also found here. Thus, the authors believe that the observed lower effect of urban

residence is essentially the aggregate effect of a concentrated Black population, one already associated with lower odds of acquiring CHD. When considering the demographic makeup of urban areas, the lower odds of CHD among the urban population become more understandable.

Table 2 presents the odds ratios for the complete sample on the likelihood of developing coronary heart disease. Moving beyond area type, witness that individuals with above-normal BMI are associated with higher odds of having CHD. Exercise levels also clearly affect CHD diagnosis, as low exercise levels are associated with significantly higher odds of CHD than the highest exercise group. There is, however, no significant difference between the high and medium exercise levels. Smoking, not surprisingly, exacerbates the likelihood of having CHD. In particular, current and former smokers are associated with higher odds of being diagnosed with CHD than those who never smoked. However, former smokers have lower odds relative to current smokers, indicating that there is some protective benefit to quitting smoking on CHD diagnosis, relative to continuously smoking.

Table 2 also reveals that the Black coefficient is lower than that of Whites, indicating that Blacks are less likely ($p < 0.03$) to have been diagnosed with CHD. This finding is consistent with previous research (e.g. Ford & Giles, 2003).

The variable gender indicates that males experience significantly higher odds of CHD diagnosis than females. The final demographic measure, age, is associated with a monotonic increase in the likelihood of having CHD. Specifically, the older people are, the greater their odds of being diagnosed with this illness.

Because Table 2 results for area type were significant, this suggested that it might be appropriate to stratify the complete sample into rural, suburban and urban sub-samples. A Chow test (DeMaris, 2004) was used to evaluate whether the health and demographic variables had different effects on predicting CHD when controlling for area type. This test was highly significant ($p < 0.001$), indicating that it is statistically appropriate to stratify the full sample by area type. Three separate multivariate logistic models were then run to ascertain the relative impact of the demographic and health behaviour covariates on the likelihood of being diagnosed with CHD. Table 3 presents the odds ratios for these three multivariate models.

Most of the results in the stratified models (see Table 3) are consistent with the Table 2 findings, indicating similar effects within the distinct areal types. For example, both Tables 2 and 3 reveal a highly significant, positive effect of BMI on CHD within rural, suburban and urban locales. Likewise, low exercise is associated with higher odds of CHD within each area. Former or current smoking is also significant and positively associated with CHD in all areal types. Finally, in all three environments males were more likely to have CHD than females.

However, just because the same variables are significant for all areal types does not mean there are no differences in effects across areas. For example, although the coefficients for males are all highly significant ($p < 0.001$), suburban men are much more likely to be diagnosed with CHD than rural or urban men. These and other such differences *across* geographic locales are noted in Table 3 by the superscript letters (i.e. a, b or c) following the individual levels of significance. Note that statistically significant effects were found across areas for the measures exercise, smoking status, gender and age. These findings are elaborated below.

Table 2. Multivariate odds ratio estimates for coronary heart disease, combined sample

	Odds ratio	95% CI
Area type		
Rural (Ref.)		
Suburban	0.95*	(0.91, 0.99)
Urban	0.92**	(0.88, 0.96)
Body mass index		
Normal weight (Ref.)		
Underweight	0.99	(0.85, 1.16)
Overweight	1.25**	(1.20, 1.30)
Moderately obese	1.67**	(1.59, 1.76)
Severely obese	2.24**	(2.09, 2.41)
Extremely obese	2.68**	(2.45, 2.94)
Exercise rigour		
High (Ref.)		
Medium	0.98	(0.94, 1.02)
Low	1.30**	(1.25, 1.36)
Smoking status		
Never smoked (Ref.)		
Former smoker	1.61**	(1.55, 1.68)
Current smoker	1.74**	(1.65, 1.83)
Race		
White (Ref.)		
Black	0.93*	(0.87, 0.99)
Gender		
Female (Ref.)		
Male	1.66**	(1.60, 1.72)
Age group (years)		
Less than 30 (Ref.)		
30–39	1.47**	(1.18, 1.82)
40–49	4.01**	(3.31, 4.86)
50–59	10.70**	(8.89, 12.87)
60–69	21.36**	(17.76, 25.69)
70+	38.19**	(31.78, 45.90)
<i>N</i>		270,635

Source: 2005 Behavior Risk Factor Surveillance Survey.

** $p < 0.001$; * $p < 0.05$.

Because Table 3 controls for area of residence, it is possible to distinguish areal effects not present in Table 2. For instance, low levels of exercise were significant predictors of CHD diagnosis in Table 2 and for all three areal types presented in

Table 3. Multivariate odds ratio estimates for coronary heart disease, stratified sample

	Rural		Suburban		Urban	
	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI
Body mass index						
Normal weight (Ref.)						
Underweight	0.96	(0.72, 1.30)	0.94	(0.72, 1.23)	1.07	(0.84, 1.36)
Overweight	1.24***	(1.13, 1.35)	1.21***	(1.13, 1.30)	1.30***	(1.21, 1.40)
Moderately obese	1.72***	(1.56, 1.91)	1.67***	(1.54, 1.81)	1.64***	(1.50, 1.80)
Severely obese	2.39***	(2.08, 2.75)	2.17***	(1.93, 2.44)	2.21***	(1.96, 2.50)
Extremely obese	2.63***	(2.20, 3.14)	2.62***	(2.26, 3.03)	2.80***	(2.41, 3.24)
Exercise rigour						
High (Ref.)						
Medium	1.04 ^a	(0.96, 1.13)	0.99 ^a	(0.92, 1.05)	0.92 ^{*b}	(0.86, 0.99)
Low	1.31*** ^a	(1.20, 1.42)	1.39*** ^a	(1.29, 1.49)	1.20*** ^b	(1.12, 1.29)
Smoking status						
Never smoked (Ref.)						
Former smoker	1.61***	(1.49, 1.74)	1.61***	(1.52, 1.72)	1.62***	(1.52, 1.73)
Current smoker	1.96*** ^a	(1.77, 2.16)	1.68*** ^b	(1.54, 1.82)	1.65*** ^b	(1.52, 1.80)
Race						
White (Ref.)						
Black	0.84 [*]	(0.72, 0.99)	0.96	(0.85, 1.09)	0.94	(0.87, 1.03)
Gender						
Female (Ref.)						
Male	1.47*** ^a	(1.37, 1.57)	1.81*** ^b	(1.71, 1.91)	1.66*** ^c	(1.56, 1.76)
Age group (years)						
Less than 30 (Ref.)						
30–39	1.48	(0.95, 2.30)	1.83**	(1.22, 2.73)	1.30	(0.94, 1.78)
40–49	4.06***	(2.76, 5.98)	5.05***	(3.50, 7.30)	3.51***	(2.66, 4.64)
50–59	10.49*** ^{a,b}	(7.22, 15.24)	14.52*** ^a	(10.14, 20.78)	8.82*** ^b	(6.76, 11.52)
60–69	20.83*** ^{a,b}	(14.36, 30.21)	30.57*** ^a	(21.39, 43.70)	16.62*** ^b	(12.74, 21.67)
70+	38.20*** ^{a,b}	(26.37, 55.33)	53.20*** ^a	(37.25, 75.99)	30.24*** ^b	(23.24, 39.36)
<i>N</i>	62,873		107,671		100,091	

Source: 2005 Behavior Risk Factor Surveillance Survey.

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$.

Note: Odds ratios with no superscript letters indicate that the global test for interactions was insignificant at 0.05 alpha level. Odds ratios with different letters within the same row indicate significant differences between areal types.

Table 3. However, Table 3's area-stratified results reveal that low exercise levels most often lead to CHD diagnosis among suburban dwellers, while such diagnoses occur significantly less among sedentary urban residents.

While Table 2 documents a clear racial difference in CHD, in the area-stratified sample, race is an insignificant predictor of CHD among suburban and urban residents. In fact, the racial effect observed in Table 2 is driven by racial differences in CHD in *rural* locales. An argument could be made that because there are accessible health infrastructures within suburban and urban areas, the likelihood of being diagnosed with CHD should be similar among Blacks and Whites. These health facilities are accessible because of proximity and because there are similar levels of socioeconomic advantage within these areas. However, in rural areas, socioeconomic differences may be exacerbated by race, as only wealthier, white individuals have the means to travel to hospitals or health care facilities where they are diagnosed with CHD.

Lastly, while Table 2 indicates the monotonic effect of age group on CHD, Table 3 indicates that in rural and urban locales, the category 30–39 years is *not* associated with increased risk of CHD relative to those less than 30. However, in suburban areas, those between the ages of 30 and 39 are about 1.8 times more likely to have CHD. Suburban residents in this age group might have higher levels of CHD diagnosis because their neighbourhoods, physicians and hospitals are relatively affluent, perhaps enabling them to receive various types of early detection examinations (such as the exercise treadmill test or electron-beam computerized tomography) that might not be as readily available to rural or urban residents.

Among current smokers, there was a significant difference between rural residents and those from both suburban and urban areas, although there was no difference between suburban and urban residents. While currently smoking increases the odds of CHD in all three areal types, rural residents who were current smokers were the group most likely to be diagnosed with CHD. Smoking in rural areas has been shown to be associated with a lack of access to social networks, particularly among adolescents and young adults (Substance Abuse and Mental Health Services Administration, 2003; Doescher *et al.*, 2006). If this were the case, smoking could be seen as a pastime that begins early in life to compensate for the lack of social network activities. Eventually, this type of compensational smoking could become a lifetime habit and lead to the observed detrimental effects on one's health.

Males have a definite disadvantage within all three geographic types but they are more likely to be diagnosed with CHD in suburban locales and least likely in rural areas. Perhaps this trend occurs because of stressors associated with occupational choice (such as middle-management or professional jobs with a high amount of mental work) and socioeconomic standing (e.g. owning a home, school choices for children, and an income salary lower than needed to maintain a middle-class lifestyle). Both stressors tend to be more pronounced for men, given the nature and persistence of work–family gender roles (Saegert, 1980; Marsh, 1988).

Age is the final covariate with effects that vary by geographic location. Increasing age is generally associated with higher CHD propensity. However, there was a significant difference between suburban and urban residents among those older than age 50. Although both suburban and urban residents over 50 are associated with a high risk of CHD, for suburbanites this risk is higher. While it may seem counterintuitive that a geographic locale associated with a relatively lower risk of CHD and resources to facilitate healthy behaviours would yield a higher risk of CHD for those over 50, this result is consistent with the literature. Specifically, studies using

data from 1983–1987 found that persons aged 45–64 in non-metropolitan areas had the highest prevalence of CHD deaths (Gillum, 1990). Thus, this aspect of the present study reinforces and builds on past research by finding that areal differences in CHD prevalence are consistent with areal differences in CHD deaths.

Discussion

The present study investigated the mortality divergence in coronary heart disease among Blacks and Whites in the United States over the past half-century. The potential for a racial mortality bias was noted since Whites are more likely to report having CHD but Blacks are more likely to die from it. It is suggested that this divergence could be better understood by controlling for the geographic context where people reside since racial groups are not uniformly distributed across the US. Furthermore, distinct areal types offer differential access to health infrastructures and different contextual factors that could affect health such as availability of fitness centres, healthy food selections and the presence or absence of stressful conditions such as crime. It is further postulated that because areas have distinct mechanisms and institutions for facilitating healthy behaviour, that controlling for health behaviours would also explain part of the observed racial gap in CHD.

Health behaviours were found to be associated with CHD in the expected directions. Specifically, positive health behaviours such as maintaining a healthy weight and frequent rigorous exercise were protective against CHD, while negative health behaviours such as smoking were detrimental and enhanced one's odds of developing the disease. In addition, certain demographic factors were found to be associated with CHD. For instance, males were at higher odds of having CHD. Likewise, as age increased, so did the odds of CHD diagnosis.

Once the sample was stratified by place of residence, the relative effects of the examined variables within each type of geographic local were further clarified. For instance, current smokers in rural areas were significantly more likely than current smokers in other areas to be diagnosed with CHD, a finding consistent with Hartley's (2004) research. Meanwhile, in suburban areas males were significantly most likely to report CHD. In sum, this research found that there are health advantages and disadvantages to be found all of the environments examined.

This study highlights the importance of geographic stratification in investigating health disparities. It also illustrates that the binary rural–urban distinction does not fully capture the complexities of how place of residence is associated with health outcomes. Furthermore, like others (Probst *et al.*, 2004; National Center for Health Statistics, 2006), this study has documented the importance of considering suburban areas as a distinct geographic unit of analysis.

There are several limitations and potential biases that may influence these analyses. The first is sample representation. The BRFSS data were collected via telephone interviews, which indicates some class bias regarding sample participants. While there were no sample selection effects with regard to income (supplemental analyses not shown), it is important to note that by virtue of this sampling method, severely impoverished persons may not be adequately represented. The inclusion of more people in the omitted categories could introduce distinct results.

Second, if the data were collected longitudinally it would be possible to link death outcomes with these processes. Thus, the second limitation is that information regarding deaths of respondents is not available. In addition, because the data are cross-sectional, it is difficult to understand the total effect of place, as place is measured at the time of data collection and not throughout the life course. As such, the degree to which current geographic area of residence affects CHD is unclear. It is also uncertain whether movement from one type of area to another impacts or is predicated by CHD. Similarly, it was not possible to address how length of exposure to distinct areal types impacts health and survival.

The final limitation is that health status is self-reported and not based on medical examinations or records. The reporting of health depends on whether patients choose to consult their general practitioner and their own self-assessment at the time the data were collected. This is problematic for two reasons. First, there could be various types of bias embedded in the study. If this research assumes that people accurately report their morbidities based on a doctor's evaluation, then it fails to acknowledge those people who are unable to seek medical advice. Second, self-reported health status may be self-diagnosed, which could lead to errors in misdiagnosis. This could result in significant variability in self-reported health, which problematizes the validity of the measurements.

Despite the aforementioned limitations, this research is meritorious. The use of recent, nationally representative data to document an important historical trend with measures of health, health behaviours and demographic variables is the most appealing aspect of this research. Additionally, separating analyses by geographic type allowed for the determination of different patterns and effects for rural, suburban and urban residents, something neglected by prior studies of CHD. Finally, using recently collected data to analyse these effects enables us to document a contemporaneous effect of health behaviours on CHD.

Because the racial disparity regarding CHD has yet to be accounted for, future research should attempt to parse out why a racial effect persists in both the diagnosis of, and deaths due to, CHD. In addition, future research should address whether the race-specific mortality patterns are mediated by health and demographic information, including access to health care, socioeconomic status and socio-ecologic stress that could emerge from living in certain area types, such as densely populated urban areas.

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