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Health Care Reform

Black/White Racial Disparities in Health: A Cross-Country Comparison of Canada and the United States

Research on health disparities in the United States has consistently reported poorer health outcomes among racial/ethnic minorities relative to whites, particularly among African Americans.1,2 In Canada, there are limited studies on racial/ethnic groups, presumably because of concerns about small samples, confidentiality, and an emphasis on socioeconomic inequalities.3 The body of literature regarding black Canadians, which compose 2.5% of the nation, is beginning to emerge.

The existing literature indicates that the burden of disease may be greater for black Canadians compared with their white counterparts, and that black Canadians face a number of barriers to achieving good health, including poverty, difficulty accessing health care, discrimination, and poor health behaviors.4,6 We obtained nationally representative estimates of health indicators among native-born black Canadians, and compared these estimates with those of native-born white Canadians. We replicated the analyses using a US sample of African Americans and white Americans to compare racial disparities in health in Canada vs the United States.

Methods. Individual-level data came from the Canadian Community Health Survey (CCHS) and the National Health Interview Survey (NHIS). For both data sets, we pooled data from 4 survey cycles (2003-2008) into a single sample to increase sample size. Analyses in both countries were limited to native-born adults to isolate the effect of race from that of nativity. Final sample sizes were 729 blacks and 280 672 whites (CCHS) and 14 211 blacks and 64 625 whites (NHIS).

Outcomes included smoking status, body mass index (BMI), general health status, and various chronic conditions (ie, asthma, hypertension, diabetes, heart disease, cancer). Self-reported race was categorized as black vs white (respondents reporting multiple races were excluded). Sociodemographic characteristics were considered as covariates, including age, sex, marital status, education, annual household income, employment status, and health insurance coverage status (in the United States).

We compared health outcomes across black and white respondents in each country. Logistic regressions assessed associations between race and health outcomes. Estimates were adjusted for various sociodemographic factors. Sampling weights were incorporated to account for complex sampling. Adjustments were made for multiple comparisons, with P < .01 considered statistically significant.

Results. In Canada, native-born whites had higher rates of current (25%) or former smoking (45%), while native-born blacks had higher rates of smoking abstinence (52%; P < .001). Whites had higher rates of hypertension (21% vs 9%), dia-
betes (6% vs 2%), heart disease (5% vs 0.6%), and cancer (6% vs 1%) compared with blacks (P < .001 for all).

In the United States, blacks had higher rates of never smoking than whites (63% vs 53%; P < .001) but also higher rates of obesity (34% vs 24%; P < .001) and lower rates of excellent/very good health status (52% vs 64%; P < .001). Blacks had higher rates of asthma (9% vs 8%), hypertension (35% vs 28%), and diabetes (11% vs 8%) but lower rates of heart disease (6% vs 9%) and cancer (4% vs 9%) compared with whites (P < .001 for all).

The Table provides results of logistic regression models. In adjusted analyses for Canada, blacks had lower odds of current or former smoking, heart disease, or cancer compared with whites. In the United States, blacks had higher adjusted odds of obesity, hypertension, diabetes, and fair/poor health relative to whites. Blacks had better outcomes than whites for current smoking, former smoking, heart disease, and cancer.

Comment. Native-born black Canadians generally reported comparable or better health outcomes than their white counterparts in contrast to the findings in the United States, where African Americans fared worse than white Americans on many health indicators.

The study had several limitations. Multiple cycles of the CCHS were pooled to produce larger samples of native-born blacks, yet despite these efforts the sample remained small, raising questions concerning generalizability. This study also examined self-reported responses rather than medical records. Recall bias is possible if blacks and whites remembered or perceived their health differently.

Another consideration is the diverging country histories: a much larger proportion of the black population in Canada comes from recent immigration flows, compared with the black population in the United States, which predominantly consists of multiple generations dating back to slavery. The United States has a long history of slavery, which was much more limited in Canada. Thus, African Americans faced a dramatically more disadvantaged social and economic trajectory compared with Canadian counterparts. These differences might contribute to our findings. Finally, there were important sociodemographic differences in the black populations across Canada and the US black Canadians were younger, had higher education and income, and were more frequently employed compared with African Americans (results not shown). And of course, Canada provides universal health coverage while the US health care system is fragmented and employment based, leaving many individuals uninsured. Our adjusted analyses may not have adequately accounted for these differences, which might explain some of the disparate health findings across countries.

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Previous Presentations: Preliminary findings from this study were previously presented at the American Public Health Association annual meeting, November 8, 2010, Denver Colorado; Academy for Health Equity conference, August 18, 2010, Littleton, Colorado; and the Canadian Public Health Association conference, June 13, 2010, Toronto, Ontario, Canada.

Additional Contributions: This analysis is based on Statistics Canada’s Canadian Community Health Survey Microdata File, which contains “anonymized” data collected in the 2003, 2005, 2007, and 2008 CCHS, as well as the Centers for Disease Control and Prevention, National Center for Health Statistics’ NHS datasets from the same years.


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Making the Case for Selective Use of Statins in the Primary Prevention Setting

Members of the Archives of Internal Medicine editorial board recently wrote, 1(p19)

...opioid medications for persons with chronic nonmalignant pain, and statin medications for persons without coronary artery disease are... examples of the widespread use of medications with known adverse effects despite the absence of data for patient benefit for these indications.

We disagree that “less is more” when it comes to statins for primary prevention of cardiovascular disease (CVD). In accord with current National Cholesterol Education Program Adult Treatment Panel III (ATP III) guidelines, we believe there is compelling evidence to support the use of statins for primary prevention in patients at high risk (Framingham risk score, >10%) for developing coronary heart disease (CHD) over the next 10 years. 2

The argument made by the Archives Redberg et al 3 is based on the assertion that statin therapy does not decrease near-term (<5-year) all-cause mortality. 4 Before disregarding the benefits of statins, we must place this assertion in context—can we expect any drug or lifestyle intervention to reliably produce a survival benefit in asymptomatic individuals within a few years?

An effective primary prevention intervention must either prolong survival or reduce patient suffering. A fundamental goal of primary prevention must also include reduction of morbidity—particularly myocardial infarction, stroke, ischemia-related hospitalizations, and invasive revascularization procedures resulting in over $475 billion in direct and indirect costs in 2009. 4 While not always fatal, major vascular events often result in severe pain and lifelong disability.

All-Cause Mortality Data for Statin Therapy. Three meta-analyses of the large statin primary prevention randomized controlled trials (RCTs) have been published since 2009. 3,5,6 Among these publications is a 2010 study by Ray et al, 3 which included 11 RCTs involving 65,229 participants without known CHD. Using previously unpublished data of strictly primary prevention patients, the authors found that statins resulted in a nonsignificant 9% risk reduction in all-cause mortality (relative risk [RR], 0.91; 95% confidence interval [CI], 0.83-1.01) over an average treatment duration of 3.7 years. 3 Unfortunately, an analysis of cardiovascular morbidity was not undertaken.

A 2009 meta-analysis by Brugts et al 5 examined 10 RCTs, 9 of them the same as those analyzed by Ray et al 3 but also including the primary prevention subgroup from the Heart Protection Study. A total of 70,388 participants were included, 6% of whom had known CHD but could not be eliminated owing to data constraints. Treatment with statins significantly reduced the risk of all-cause mortality by 12% (odds ratio [OR] 0.88; 95% CI, 0.81-0.96) over a mean follow-up of 4.1 years. 5 A significant decrease in all-cause mortality remained after excluding the 3 trials that included participants with known CHD (OR, 0.87; 95% CI, 0.78-0.97) or after excluding JUPITER (Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin), the largest and most controversial of the primary prevention trials (OR, 0.89; 95% CI, 0.81-0.97).

A 2011 Cochrane meta-analysis of statins in primary prevention analyzed 14 RCTs (including 16 trials arms) dating from 1994 to 2006, representing a total of 34,272 patients. For inclusion, the authors required enrollment of 10% of participants or less with known CVD. The individual trials had observation periods ranging from 1 to 5.3 years. Treatment with a statin resulted in a 17% reduction in all-cause mortality (RR, 0.83; 95% CI, 0.73-0.95). 6 The authors cautioned that any conclusions about mortality benefit with statin therapy are limited owing to inconsistencies in individual RCT data quality, study design, and emphasis on combined end points as primary outcomes.

Overall, 2 of the 3 statin primary prevention meta-analyses indicate a modest but statistically significant 12% to 17% RR reduction in all-cause mortality at 5 years of follow-up or less. If the only goal of statin therapy is to prolong near-term (=5-year) survival, then the evidentiary support would have to be considered less than robust.

Cardiovascular Morbidity Data for Statins—The Message Is Clear. The meta-analysis by Brugts et al 5 also analyzed the effect of statins on cardiovascular morbidity. These findings suggest a significant decrease in major coronary events (OR, 0.70; 95% CI, 0.61-0.81), major cerebrovascular events (OR, 0.81; 95% CI, 0.71-0.93), nonfatal myocardial infarction 0.56 (OR, 0.56; 95% CI, 0.41-0.76) and revascularizations (OR, 0.67; 95% CI, 0.59-0.76). 5 These results corroborate those of the Cochrane meta-analysis demonstrating a 34% reduction in revascularizations (RR, 0.66; 95% CI, 0.53-0.83) and a 30% reduction in combined fatal and nonfatal CVD end points (RR, 0.70; 95% CI, 0.61-0.79). 5

Despite these impressive treatment effects on CVD morbidity and modest effects on mortality, the authors of the Cochrane review emphasize that “caution should be taken in prescribing statins for primary prevention among people at low cardiovascular risk.” 6 We agree that statins are not likely to benefit patients with low coronary risk (10-year Framingham risk score, <10%) but emphasize that it is paramount to make the distinction between low-risk and high-risk primary prevention cohorts. Patients without known CHD but with diabetes,