
Vozoris’ recently reported that menthol cigarette use was associated with increased stroke risk compared with nonmenthol cigarette use among US smokers (odds ratio, 2.25; 95% CI, 1.33-3.78). These results, however, were not consistent across all demographic groups. For example, no increased risk was observed among African American smokers, a group with a high menthol smoking prevalence. I recently reported that menthol cigarette use was associated with lower lung cancer mortality among US smokers, although I found no difference in mortality risk for other causes. This study reexamines stroke risk among US menthol smokers using national health survey data and mortality follow-up.

Methods | I used data from the 1999 through 2010 National Health and Nutrition Examination Survey (NHANES), a nationally representative health survey of the US civilian noninstitutionalized population conducted by the National Center for Health Statistics. It includes a health interview as well as a physical examination and collection of biospecimens. Approximately 10,000 individuals participate in the NHANES every 2 years. I analyzed data for NHANES current smokers 20 years and older using logistic regression analysis to examine the association between having been diagnosed with a stroke by a health professional and menthol smoking. I included pack years of smoking, sex, age, race and ethnicity (using the NHANES race and ethnicity categories), educational attainment, ratio of family income to poverty threshold, use of other tobacco products, and body mass index as control variables in the analysis. Of the 7055 NHANES participants who reported that they were current smokers, 5745 had information for all regression variables, including menthol cigarette use (600 smokers did not have family income information and 294 were missing menthol information). In total, 1765 smokers were identified as menthol smokers and 3980 as nonmenthol smokers. Of these smokers, 195 reported having had a stroke. Stroke prevalence was 3.4% (95% CI, 2.9%-4.0%) among nonmenthol smokers and 3.3% (95% CI, 2.6%-4.3%) among menthol smokers. Stroke prevalence was similar among all NHANES smokers at 3.5% (95% CI, 3.0%-4.1%) for nonmenthol smokers and 3.8% (95% CI, 3.0%-4.7%) for menthol smokers. I conducted the analyses using the appropriate NHANES sample weights, taking into account the NHANES complex sample design.

I also analyzed stroke mortality risk using 1987 National Health Interview Survey (NHIS) Cancer Control Supplement data linked for mortality follow-up. The NHIS is an annual questionnaire-based health survey of the US civilian noninstitutionalized population. Approximately 22,000 NHIS participants 18 years and older completed the Cancer Control Supplement in 1987. These individuals were observed for mortality through the end of 2006 by linking their records to the National Death Index, which is maintained by the National Center for Health Statistics and contains death certificate information for all US decedents since 1979. I conducted a survival analysis of current smokers at baseline by menthol cigarette use, using a Cox proportional hazards regression model. I controlled for sex, age, race and ethnicity, educational attainment, family income, and use of other tobacco products. Of the 4832 smokers who had information for all variables, 1356 were identified as menthol smokers and 3476 as nonmenthol smokers. Among these individuals, 1221 deaths were ascertained through linkage with the National Death Index, of which 56 were caused by stroke (International Classification of Diseases, Tenth Revision, codes 160-169).

All NHANES and NHIS data used in this analysis have been released for public use by the National Center for Health Statistics, so institutional review board approval was not needed or obtained.

Results | The Table presents adjusted odds ratios for stroke for NHANES menthol smokers compared with nonmenthol smokers. No difference in risk was observed among smokers overall or among male or female smokers. Odds of stroke were lower for African American menthol smokers, but it is possible that this estimate was affected by a limited sample size and/or residual confounding. I also did not observe a difference in stroke mortality risk for menthol cigarette use among NHIS smokers (hazard ratio, 0.67; 95% CI, 0.34-1.33).

Discussion | I found no evidence in national health survey data of a higher stroke risk for US menthol smokers compared with nonmenthol smokers. I examined an expanded set of NHANES data compared with that used by Vozoris’...
(1999-2010 vs 2001-2008 participants) and controlled for a similar set of covariates. Results were consistent by sex, generally stable across race and ethnicity groups, and robust to alternative specifications for variables such as smoking exposure and income. Using NHIS data, I also found no evidence of higher stroke mortality associated with menthol cigarette use among smokers.

It is not clear to me how Vozoris' obtained his findings, given that I cannot replicate his general results for stroke using the NHANES data and analyses that he specified. Moreover, the absence of observed differences in stroke prevalence among NHANES menthol smokers would suggest that methodological or analytical issues may have affected his results.

These findings are consistent with my research that found lower lung cancer mortality for US menthol smokers compared with nonmenthol smokers but no difference in mortality risk for other causes. Lower lung cancer risk for menthol smokers has been observed in previous studies6,7 and among certain groups in meta-analysis.8

### Conflicts of Interest Disclosures: None reported.

### Corresponding Author: Brian Rostron, PhD, 8000 Wildwood Dr, Takoma Park, MD 20912 (brianr@demog.berkeley.edu).

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### End-Stage Renal Disease From Human Immunodeficiency Virus–Associated Nephropathy in the United States, 2001 Through 2010

Before the advent of effective antiviral therapy, AIDS was associated with extremely high mortality.1 Human immunodeficiency virus (HIV)-associated nephropathy (HIVAN) emerged as a common cause of progressive kidney disease in inadequately treated patients predominantly of African descent, characterized by an increasingly evident genetic predisposition.2,3

With access to optimal care, the outlook for HIV-infected individuals has changed substantially in the past 2 decades.4 However, temporal trends in end-stage renal disease (ESRD) from HIVAN in the United States have not been defined systematically.

### Methods | The main objectives of this study were to enumerate trends in incidence ratios, standardized to 2001-2002, of ESRD from HIVAN treated with renal replacement therapy (RRT) in the United States for 2001 through 2010. In this retrospective study, we used United States Renal Data System standard analysis files to study US patients who initiated maintenance RRT between 2001 and 2010 (N = 1 048 867). Cases of ESRD from HIVAN were those with the primary cause listed as “AIDS nephropathy” on the ESRD Medical Evidence Report.

US census data were used to determine population denominators for each year examined, with race or ethnicity classified as non-Hispanic white, non-Hispanic black, Hispanic, and other.5 The Poisson distribution was applied to calculate incidence rates of RRT-requiring ESRD due to HIVAN. For standardized incidence ratios, expected incidence rates were calculated by applying incidence rates in 2000 for each of the 168 possible combinations of age (21 subgroups), sex (2 subgroups), and race or ethnicity (4 subgroups) to the corresponding subgroup of the US population for each year. Binary logistic regression was used for adjusted between-era comparisons of patients at initiation of RRT. SAS version 9.1.3 (SAS Institute) was used for data analysis. Specific institutional review board approval was not sought for this retrospective registry-based study.

### Results | Table 1 shows characteristics at the time of RRT initiation in patients with HIVAN in 2 eras, 2001-2005 and 2006-2010. Age 45 to 64 years and 65 years or older, white race, diabetes mellitus, drug abuse, glomeruliferous filtration rate of more than 15 mL/min/1.73 m², and body mass index of 25 or higher (calculated as weight in kilograms divided by height in meters squared) were more prominent from 2006 through 2010. In 2001-2002, the rate of RRT-requiring ESRD due to HIVAN was 2.9 cases per million per year (Table 2). Standardized incidence ratios declined for the overall population between the 2001-2002 and 2009-2010 biennia, with a stepwise decline from 2005 through 2006. However, standardized incidence ratios increased for patients 65 years and older and those of non-Hispanic white race or ethnicity.

### Discussion | Our study of ESRD from HIVAN suggests both meaningful progress and challenges for the future, with declining overall incidence contrasted by an emergence in older and non-Hispanic white populations.

This retrospective and registry-based study lacks desirable data elements that a prospective design could provide. While a true tissue diagnosis in all patients would be desirable, this aspiration is likely utopian.6 Questions about HIV positivity and AIDS as a comorbid illness at dialysis initiation were removed from the 2005 Medical Evidence Report. Thus, it is not possible to refute with certainty the hypothesis that the apparently salutary trends for HIVAN reflect changing fashions in labeling the cause of renal disease in patients living with HIV and not an alteration in the incidence of HIVAN.