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Association of sleep duration with kidney function and albuminuria: NHANES 2009-2012☆

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ABSTRACT

Objective: The purpose of this study was to examine the association between self-reported sleep duration and markers of kidney function.
Design: A cross-sectional survey from the 2009-2012 National Health and Nutrition Examination Survey.
Setting and Participants: The participants were 8690 adults (≥20 years) without a previous sleep disorder diagnosis, end-stage kidney failure, or other kidney or liver problems. Subsamples with pre-diabetes and pre-hypertension were examined.
Measurements: Participants reported habitual sleep duration, coded as ≤5, 6, 7, 8, and ≥9 hours per night. Biomarkers of kidney function were determined, including glomerular filtration rate (eGFR) estimated from the Chronic Kidney Disease Epidemiology Collaboration equation, urine albumin-to-creatinine ratio (ACR), microalbuminuria status, and glomerular hyperfiltration status. Weighted and adjusted general linear models assessed associations between sleep duration with eGFR and ACR. Logistic regression analyses evaluated the associations of microalbuminuria and glomerular hyperfiltration status with sleep duration.
Results: Greater eGFR was related to short sleep duration in the total sample and among participants with pre-diabetes. Greater ACR was associated with short and long sleep duration. Short sleep duration (≤5 hours) was associated with an increased odds for glomerular hyperfiltration (OR, 1.41; 95% CI, 0.97-2.06) and microalbuminuria (OR, 1.31; 95% CI, 0.96-1.79).
Conclusions: In a US representative sample of adults, self-reported short and long sleep duration were related to higher ACR. Short sleep duration was associated with higher eGFR and microalbuminuria. Research is needed to understand whether these associations indicate increased risk for kidney damage and cardiovascular risk.

Introduction

Short and long reported sleep durations are consistently associated with mortality,[1,2] and numerous chronic medical conditions, including cardiovascular disease (CVD), [3] stroke,[4] hypertension,[5,6] and type 2 diabetes.[7] Extremes in sleep duration have also been associated with risk factors for chronic illnesses, including high cholesterol,[8,9] high blood pressure,[10] and greater circulation of inflammatory markers[11]; however, there are limited data describing the association between sleep duration and indicators of current kidney function. Kidney function is intimately tied to the vascular system, and poor kidney function is an important, independent risk factor for CVD.[12,13] If short and long sleep durations are associated with indices of cardiovascular risk, then it is possible they are also associated with harmful alterations in kidney function that may lead to clinical conditions, including chronic kidney disease (CKD). Extremes

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in sleep duration may contribute to the development of CKD directly through detrimental changes in the sympathetic nervous and renin-angiotension-aldosterone systems, and/or indirectly through known risk factors for CKD such as obesity, hypertension, and diabetes.[14]

Few studies have investigated the relationship between sleep duration and kidney function among community-based adults. Short and long sleep durations were found to be associated with prevalent CKD.[15,16] An analysis of estimated glomerular filtration rate (eGFR) in the 2005-2008 National Health and Nutrition Examination Survey (NHANES) found that self-reported short sleep (≤6 hours) was associated with greater odds for the prevalence of CKD stages 1 and 2 than moderate sleep duration.[17] Two longitudinal studies found similar results. One study found self-reported short sleep (<5 hours) was related to a 28% increase in the incidence of proteinuria over a median of 2.5 years among young to middle-aged Japanese adults with no indicators of kidney dysfunction or injury at baseline.[18] Notably, the other longitudinal study assessed sleep using wrist actigraphy and measured kidney function continuously with eGFR from serum creatinine. Among young to middle-aged US adults without a history of CVD, CKD, proteinuria, or hypertension, each 1-hour decrease in sleep duration was associated with a 1.5 mL/min per 1.73 m^2 increase in eGFR over 10 years.[19]

Petrov et al suggested that in healthy younger individuals, short sleep duration may initially lead to glomerular hyperfiltration followed by increased risk for renal injury, intraglomerular hypertension, and accelerated development of CKD. Such a pathophysiological progression occurs in the context of Type I diabetes mellitus and hypertension, as well as increasing stages of pre-diabetes and pre-hypertension.[20–22] By the time a person has progressed to diagnosed diabetes and/or hypertension, the association between reported sleep duration and eGFR reverses such that short sleep duration is then associated with reduced eGFR,[23] although the association between extremes in sleep duration and elevated levels of protein and albumin excretion (i.e., microalbuminuria) may persist throughout this process.[24,25] Hence, extremes in sleep duration may be a risk factor for early progression toward CKD through glomerular hyperfiltration. This hypothesis is difficult to test because there is no standardized, clinical cut-off for glomerular hyperfiltration due to strong associations between eGFR with age and sex.[26]

The objective of this study was to investigate the cross-sectional association between self-reported sleep duration and markers of kidney function including eGFR and urinary albumin to creatinine ratio (ACR) in the NHANES 2009-2012 data collection cycles. We hypothesized that shorter sleep duration would be associated with greater eGFR and ACR. This study expands upon the previous literature in that it : 1) analyzed both short and long sleep durations; 2) assessed the moderating roles of age, sex, race/ethnicity, and body mass index (BMI) in the association between sleep duration and ACR, as well as the moderating role of BMI on the association between sleep duration and eGFR (age, sex, and race/ethnicity are already accounted for in the calculation of eGFR); 3) examined the relationships between sleep duration and markers of kidney function among subsamples of individuals with pre-diabetes and pre-hypertension; and 4) evaluated the associations of sleep duration with glomerular hyperfiltration status and microalbuminuria status.

Participants and methods

Sample and study design

Participants in this study were from the NHANES 2009-2010 and 2011-2012 surveys conducted by the US Centers for Disease Control and Prevention. Both surveys used a stratified, multistage probability design to recruit a representative sample of the non-institutionalized U.S. civilian population. Details on study design and sampling can be found elsewhere.[27] The ethics review board of the Centers for Disease Control and Prevention reviewed and approved the protocol and all participants gave written, informed consent. Participants underwent a standardized in-home interview as well as a physical examination at a mobile examination center during which blood and urine were collected. This analysis was limited to participants ≥20 years old with complete sleep duration data, and complete or in part complete serum creatinine, and urine albumin and creatinine assessments (n = 11,752). Participants were further excluded from the analyses if they were pregnant, had a previous sleep disorder diagnosis, end-stage kidney failure (eGFR <15 mL/min per 1.73 m^2), were currently taking insulin, reported urinating five or more times per night, or self-reported that a doctor had told them they had kidney or liver problems. The total, eligible analysis sample was 8690 participants.

Measurements

Sleep duration

Habitual nightly sleep duration was reported by the participants at the in-home visit by trained interviewers using the Computer-Assisted Personal Interviewing system. The question stated "How much sleep do you usually get at night on weekdays or workdays (hours)?" Sleep duration was categorized as ≤5, 6, 7, 8, and ≥9 hours per night in whole integers.

Markers of kidney function

Serum creatinine was measured using the Jaffe rate method and calibrated with a standardized isotope dilution mass spectrometry reference method. Glomerular filtration rate was estimated from serum creatinine using the Chronic Kidney Disease Epidemiology Collaboration equation.[28] The eGFR was operationalized both continuously and categorically. The eGFR was categorized by CKD stages as follows: no CKD; Stages 1 and 2 (≥60 mL/min per 1.73 m^2 + ACR ≥30 mg/mmol); and Stages 3 and 4 (15 – 59 mL/min/1.73 m^2). In addition, eGFR was further subdivided into participants with and without glomerular hyperfiltration. Glomerular hyperfiltration was defined as exceeding age and sex specific 95th percentile for eGFR levels as determined by a healthy subset of the eligible study sample. This healthy subset of participants did not have pre-diabetes, diabetes, pre-hypertension, hypertension, microalbuminuria (ACR ≥30 mg/mmol), or cancer. This subset was divided into 10-year age categories by sex. The number of participants ≥70 years of age were too few to provide hyperfiltration reference levels. Therefore, these participants were categorized with the 60 - 69 year age group. ACR was determined with a single, spot urine sample. Urinary albumin and creatinine were measured with a solid-phase fluorescent immunoassay and modified Jaffe kinetic method. ACR was analyzed both continuously and categorically. Microalbuminuria status was categorized as ACR ≥30 mg/mmol.

Covariates

Participants self-reported demographics (ie, age, sex, race/ethnicity), socioeconomic status (ie, family income to poverty guidelines ratio; range, 0-5), medical conditions (ie, hypertension, CVD, stroke, diabetes), moderate physical activity (ie, moderate-intensity activity for at least 10 minutes continuously), and self-rated general health (5-point scale from excellent to poor). Ethnicity/race was categorized as Mexican-American, Other Hispanic, non-Hispanic White, non-Hispanic Black, and Other Race including Multiracial. At the physical examination, height and weight were measured to calculate BMI, and resting blood pressure was taken. BMI was categorized as underweight to normal weight (BMI <25), overweight (25 < BMI < 29.99), and obese (BMI ≥30). Along with blood and urine samples to