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Sleep duration and incidence of type 2 diabetes: the Multiethnic Cohort

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ABSTRACT

Objectives: As an emerging risk factor for the rising incidence of type 2 diabetes, we examined sleep duration in relation to type 2 diabetes and several biomarkers.
Design: Prospective cohort recruited 1993-1996.
Setting: The Multiethnic Cohort in Hawaii and California.
Participants: A cohort of 151,691 White, African American, Japanese American, Native Hawaiian, and Latino participants; 9695 cohort members had biomarker measurements.
Measurements: Sleep duration was self-reported at cohort entry. Diabetes status was obtained from 3 questionnaires and confirmed by 3 administrative data sources. Biomarkers were measured by standard assays 9.6 ± 2.1 years after cohort entry. We estimated diabetes risk as a time-varying outcome using Cox regression adjusted for body mass index assessed at 3 time points and other known confounders and computed adjusted means of biomarkers by sleep hours.
Results: During 7.9 ± 3.5 years of follow-up, 8487 new diabetes cases were diagnosed. Long sleep duration (≥9 hours), as compared with 7-8 hours, was significantly associated with higher incidence (hazard ratio, 1.12; 95% confidence interval 1.04-1.21), but the 4% elevated incidence for short sleep duration (≤6 hours) did not reach significance (95% confidence interval 0.99-1.09). After stratification, the associations appeared stronger in Japanese American than other ethnic groups and in participants without comorbidity. Hours of sleep were positively associated with C-reactive protein and triglycerides and inversely related to high-density lipoprotein cholesterol and adiponectin but not with leptin levels and homeostatic model assessment of insulin resistance.
Conclusion: In this multiethnic population, the 12% higher diabetes risk for long sleep hours may be mediated through inflammation, a poor lipid profile, and lower adiponectin levels.

Introduction

The rising health burden due to type 2 diabetes around the world has stimulated a search for new etiologic factors.[1] Besides excess body weight, physical activity, and dietary composition, the role of the pineal hormone melatonin in transmitting circadian timing information to the pancreatic islets has emerged because it affects the blood glucose–regulating function of the islet cells and inhibits insulin secretion during the night.[2] In the last 50 years, the average self-reported sleep duration in the United States has decreased by 1.5-2 hours in parallel with an increasing prevalence of obesity and type 2 diabetes.[3] Strong epidemiologic evidence supports an association of sleep duration with diabetes development. In a pooled study[4] and in a later meta-analysis,[5] low (≤6) and high (≥8) hours of sleep predicted a higher risk in a U-shaped pattern with the lowest risk at 7-8 hours. The respective risk estimates per 1-hour shorter or longer sleep were 1.09 (95% confidence interval [CI], 1.04-1.15) and 1.14 (95% CI, 1.03-1.26). In the Multiethnic Cohort (MEC), short and long sleep duration was associated with 15%-25% higher all-cause and cardiovascular mortality.[6] In women only, diabetes-specific mortality was also elevated with short and long sleep duration. Despite this extensive evidence on the role of sleep in diabetes etiology,[7] a number

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of questions remain open. First, less is known about this relation in non-White ethnic groups, although ethnic differences are likely given the high prevalence of obesity in some ethnic groups and stronger associations of obesity with type 2 diabetes incidence in Asians and Native Hawaiians than in Whites.[8–10] Second, confounding by obesity status is likely, and detailed information on body mass index (BMI) is needed. Third, biological mechanisms for a potential association are not well understood. C-reactive protein (CRP), high-density lipoprotein cholesterol (HDL-C), triglycerides, leptin, and adiponectin are some of the possible mediators for sleep duration on diabetes risk.[11,12] The current analysis determined the association of self-reported sleep duration assessed at cohort entry with diabetes incidence in 5 ethnic groups using self-reported diagnoses confirmed by administrative data and controlling for repeated BMI measures. In addition, possible biologic mechanisms for an association were examined using biomarkers of inflammation, adiposity, and metabolism among a subset of cohort members.

Participants and methods

Study population

The MEC was established in 1993-1996 to study the association of lifestyle and genetics with cancer and other chronic diseases among primarily Whites, Native Hawaiians, and Japanese Americans in Hawaii and African Americans and Latinos in California.[13] The study has been approved by the Institutional Review Boards of the University of Hawaii and the University of Southern California. A total of 215,831 participants aged 45-75 years at recruitment entered the cohort with a self-reported ethnic distribution of 26% Japanese American, 23% White, 22% Latino, 16% African American, 7% Native Hawaiian, and 6% Other. Depending on sex and ethnicity, the response rate varied between 19% and 51%. The MEC includes all education levels, although cohort members were somewhat better educated than the general population.[13]

Questionnaire and follow-up data

At cohort entry in 1993-1996 (QX1), detailed exposure information was collected by means of a 26-page, self-administered questionnaire (Spanish version for Latinos in California in addition to English)[13] that included an extensive validated food frequency questionnaire specifically developed to include foods commonly consumed by the 5 ethnic groups,[14] height, body weight, physical activity, smoking, medications, medical conditions, family history, reproductive history, and demographics. Based on a large recipe database,[15] intakes of foods, nutrients, and total energy were computed. Physical activity was assessed by items asking about hours of sedentary, moderate, and vigorous activities; this information was used to compute daily metabolic equivalents of tasks (METs). The question “On the average, during the last year, how many hours in a day did you sleep including naps?” offered 6 categories (≤5, 6, 7, 8, 9, and ≥10 hours). As partial validation, the association of habitual sleep duration with objective measures of energy balance was demonstrated in a small subset of White and African American cohort members.[16] Updated information on BMI and diabetes status was available from QX2 (1999-2002) returned by 84% of the cohort and QX3 (2003-2009) completed by approximately 50% of cohort members. Each of the questionnaires included the question “Has your doctor ever told you that you had diabetes?” At cohort entry, 25,858 of 215,831 (12%) participants self-reported diabetes; all of these were excluded as prevalent cases. The percentage increased in QX2 and QX3 to 14% and 18%. Given the age structure of the MEC, all cases were considered type 2 diabetes. Self-reports were consistent across subsequent questionnaires for 37,728 (93%) individuals. Regular linkages with death record files in Hawaii and California were performed and provided information on all deaths until 2009 when the last QX3s were returned.

For this analysis, only participants with self-reported diabetes confirmed by administrative data were considered incident cases. Three sources of administrative data were available: Medicare claims,[17] a linkage with health insurance plans in Hawaii,[8] and hospital discharge diagnosis data in California.[18] For 1999-2012, a Medicare linkage provided information on 114,309 cohort members who were fee-for-service beneficiaries.[17] The information for 69,061 (32% of cohort) beneficiaries enrolled in managed care plans does not offer sufficient detail to establish a diagnosis. For fee-for-service plans, the Chronic Condition Warehouse supplies information on diabetes using at least 1 inpatient, skilled nursing facility, or home health claim or 2 hospital outpatient or carrier claims during a 2-year period.[19] In Hawaii only, records for MEC members alive in 2007 were linked to the diabetes care registries of the 2 major insurers in Hawaii covering at least 90% of the population.[8] The State of California provides diagnosis data for each patient who is treated as an inpatient in a licensed general acute care hospital in California[20] using the same algorithm as Medicare. Of 114,309 Medicare beneficiaries with fee-for-service plans, 44,718 (39%) were classified as cases. The percentages were lower in the California hospital discharge data (24%) and the Hawaii health plan linkage, which classified 15,110 of 88,004 (17%) participants as cases. When all 3 data sources were considered, 83% of self-reports were confirmed by at least 1 administrative data source. The major reasons for lack of confirmation were not being part of Medicare due to young age or managed care plans, not being a member of a linked health plan in Hawaii, and death.

Biomarker assays

In 2001-2006, a MEC biospecimen subcohort of 68,740 cohort members (49.7% of eligible) with morning blood samples from predominantly fasting individuals was established. Part of this cohort ($n = 12,578$) was characterized for a number of biomarkers by selecting controls from genetic case-control studies (breast, prostate, colorectal, and lung cancer). Only individuals who had fasted ≥8 hours and had not been diagnosed with diabetes before the blood draw were included in the analysis ($N = 9700$). Adiponectin and leptin from plasma were measured by enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, Inc, Minneapolis, MN; cat. nos. DRP300 and DLP00). Insulin was assessed in serum using an ELISA kit (EMD Millipore, Billerica, MA; cat. no. EZHI-14K). All ELISA protocols were followed in accordance with the manufacturer's instructions. A Cobas Mira Plus chemistry autoanalyzer (Roche Diagnostics, Indianapolis, IN) was used to measure serum glucose (kit from Randox, Kearneysville, WV), CRP, HDL-C, and triglycerides (Pointe Scientific, Inc, Canton, MI) per manufacturer's instructions. Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated as (fasted insulin [mU/L] × fasted glucose [mg/dL])/405.

Statistical analysis

Of the total cohort, the following exclusions were made (some overlap), resulting in 151,691 observations: 13,994 other ethnicity, 28,153 prevalent diabetes at cohort entry, 9152 invalid diet, 7662 missing information on sleep variables, 3132 missing BMI, 22,045 missing physical activity, and 4 no follow-up time. Participants with missing smoking status were coded accordingly and included as a separate category. After applying a diabetes definition requiring a positive self-report and a claims diagnosis to maximize specificity, 8487 incident diabetes cases were identified.

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