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Functional capacity is a better predictor of coronary heart disease than depression or abnormal sleep duration in Black and White Americans

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Abstract

Objective—To assess whether functional capacity is a better predictor of coronary heart disease (CHD) than depression or abnormal sleep duration.

Methods—Adult civilians in the USA ($n=29,818$, mean age 48 ± 18 years, range 18–85 years) were recruited by a cross-sectional household interview survey using multistage area probability sampling. Data on chronic conditions, estimated habitual sleep duration, functional capacity, depressed moods and sociodemographic characteristics were obtained.

Results—Thirty-five percent of participants reported reduced functional capacity. The CHD rates among White and Black Americans were 5.2% and 4%, respectively. Individuals with CHD were more likely to report extreme sleep durations [short sleep (< 5 h) or long sleep (> 9 h); odds ratio (OR) 1.65, 95% confidence interval (CI) 1.38–1.97; $P<0.0001$], less likely to be functionally active [anchored by the ability to walk one-quarter of a mile without assistance (OR 6.27, 95% CI 5.64–6.98; $P<0.0001$)] and more likely to be depressed (OR 1.78, 95% CI 1.60–1.99; $P<0.0001$) than their counterparts. On multivariate regression analysis adjusting for sociodemographic factors and health characteristics, only functional capacity remained an independent predictor of CHD (OR 1.81, 95% CI 1.42–2.31; $P<0.0001$).

Conclusion—Functional capacity was an independent predictor of CHD in the study population, whereas depression and sleep duration were not independent predictors.

Keywords

Functional capacity; Depression; Sleep duration; Coronary heart diseases; Physical activity; Mood

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1. Introduction

Aberrant sleep duration, depression and impaired functional capacity are associated with coronary heart disease (CHD). There is a strong and consistent association between both short and long sleep durations and CHD [1–4]. Sleep disturbance constitutes a form of stress [5], which is directly related to the degree of objective sleep disturbance [6]. This has been shown to predict coronary events, and is linked to increased myocardial infarction (MI) [7].

An increasing body of evidence also suggests that psychological illness, such as depression, may have a negative impact on cardiovascular health [8–12]. Depression is associated with increased risk of adverse outcome and worse prognosis in individuals with congestive heart failure [13,14] and acute MI [15–20], and in patients who have undergone coronary artery bypass surgery [11,21,22].

Converging data indicate that depression, sleep disturbance and poor functional capacity often co-exist [23,24]. As these factors cluster together in a variable fashion, it is difficult to discern the potential mechanism by which they exert their pathophysiological effects. Both physical activity and sleep disturbance constitute diagnostic indices for major depression [25]. Individuals experiencing depression or life stress are likely to be functionally limited [23,24], which is an independent risk factor for CHD [26,27]. Pathophysiological mechanisms that are potentially important in the development of CHD have been linked to physical inactivity and/or poor functional capacity [28]. Despite the growing evidence linking CHD to aberrant sleep duration, depression and poor functional capacity, independent associations between these factors and CHD have not been established. Hence, this cross-sectional study tested the hypothesis that functional capacity is a better predictor of CHD than depression or sleep duration.

2. Methods

2.1. Participants

This study was approved by the Institutional Review Board of State University of New York Downstate Medical Center. Data were obtained from 29,818 Americans (age range 18–85 years) participating in the 2005 National Health Interview Survey (NHIS). The present analysis included data from White (85%) and Black (15%) Americans of both genders (males 44%, females 56%).

2.2. Procedures

The NHIS is a cross-sectional household interview survey conducted annually by the National Center for Health Statistics of the Centers for Disease Control and Prevention. The NHIS uses a multistage area probability design that permits representative sampling of US households. Probability samples of the civilian adult population of all 50 states and the District of Columbia were obtained. It is estimated that the final sample was characterized by a response rate of 69%. No significant differences were found in the demographic characteristics between responders and non-responders. Details on sample design can be found in Design and Estimation for the National Health Interview Survey, 1995–2005 [29].

Participants provided sociodemographic data and information about physician-diagnosed chronic conditions during face-to-face interviews conducted by trained interviewers from the US Census Bureau. The chronic conditions included hypertension, heart disease, cancer, diabetes and arthritis. Participants also estimated habitual sleep duration (using full hour units, i.e. 5 h, 6 h, 7 h, etc.); no information on specific sleep disorders was elicited during the interview. Participants also reported depressed moods (i.e. feeling of sadness, hopelessness, worthlessness and poor effort) experienced in the past 30 days. The depression

severity score used in this analysis represents a composite score, summing answers from the above-mentioned four items; scores ranged from 0 to 20 with low scores indicating greater depression. Functional capacity was measured by asking respondents whether they were able to walk one-quarter of a mile without assistance; respondents were classified as 'limited' if they answered that it would be very difficult or they would be unable to perform this activity, or 'not limited' if they were able to perform this activity.

Surveys were conducted using computer-assisted personal interviewing, which uses a computer program for data collection that guides the interviewer through the questionnaire. The interviewer enters survey responses directly into the computer. The program uses a computer algorithm to determine whether data entered by the user match against all possible responses to specific questions; the program also checks for consistency against other data collected during the interview, and saves the responses into a survey data file [30].

2.3. Statistical analysis

As the NHIS dataset amalgamates data from different samples using a multistage area probability sampling design, all analyses performed in this study used weighted statistics based on the final weights provided with the NHIS dataset. These weights represent a product of weights for corresponding units computed in each of the sampling stages. Frequency and measures of central tendency were used to describe the sample. In preliminary analyses, Pearson and Spearman correlations were used to explore relationships between variables of interest. Fisher's exact test was used to assess differences in categorical variables, and analysis of variance was used for parametric variables.

Univariate logistic regression analyses were performed to assess the relationships between the three main factors: sleep duration, depression and functional capacity. Before constructing the final model assessing the best predictor of CHD, binary relationships were assessed between hypothesized predictors and the dependent variable; only predictors showing a P -value <0.05 were entered into the final model [31]. Covariates entered into the model were: sex, age, race/ethnicity, income, body mass index (BMI), and a history of hypertension, diabetes, arthritis or heart disease. All analyses were performed using Statistical Package for the Social Sciences Version 17.0 (IBM Corporation, NY, USA).

3. Results

The health characteristics of the individuals participating in the NHIS are provided in Table 1. The sample included White (85%) and Black (15%) Americans of both genders (males 44%, females 56%). Thirty-five percent of participants reported functional limitation due to chronic conditions: hypertension (28%), heart disease (8%), diabetes (9%) and arthritis (23%). The CHD rates among White and Black Americans were 5.2% and 4%, respectively.

Results of univariate regression analysis showed that individuals with CHD were more likely to report short (< 5 h) or long (> 9 h) sleep durations [odds ratio (OR) 1.65, 95% confidence interval (CI) 1.38–1.97; $P<0.0001$] than their counterparts. Results also showed that individuals with CHD were less likely to be functionally active [anchored by the ability to walk one-quarter of a mile (OR 6.27, 95% CI 5.64–6.98; $P<0.0001$)], and were more likely to be depressed [OR 1.78, 95% CI 1.60–1.99; $P<0.0001$] than their counterparts. Functional capacity was directly correlated with both short and long sleep durations [$r=0.23$ ($P<0.0001$) and $r=0.25$ ($P<0.0001$), respectively], and inversely correlated with depression [$r=-0.27$ ($P<0.0001$)].

Results of multivariate logistic regression analysis indicated that functional capacity remained a significant independent predictor of CHD (OR 1.81, 95% CI 1.42–2.31;

$P < 0.0001$). As shown in Table 2, adjusting for the effects of age, gender, race/ethnicity, income, BMI, and a history of hypertension, diabetes and arthritis demonstrated that extreme sleep duration and depressed mood were not significant independent predictors of CHD. However, in subgroup analysis, depression was an independent predictor of CHD in White Americans but not in Black Americans (Table 3).

4. Discussion

This study found that depression, sleep duration and poor functional capacity are associated with CHD. Interestingly, functional capacity remained an independent predictor of CHD after adjusting for sociodemographic factors and health characteristics, whereas depression and sleep duration did not remain independent predictors. This finding is consistent with data suggesting that physical activity predicts the likelihood of cardiovascular disease beyond that explained by commonly measured cardiometabolic risk factors [32]. Although the mechanism of this association is not entirely clear, research has shown that exercise improves coronary artery flow reserves in patients with CHD [33]. Among patients with congestive heart failure, research indicates that exercise increases the anaerobic threshold [34], and improves ventilator response and heart rate variability [35]. This evidence provides justification for the integration of regular exercise into the management of CHD and psychosocial disorders.

The present finding of a positive association between depression and CHD is consistent with prior reports [8–12]. In the subgroup analysis, depression was found to be an independent predictor of CHD in White Americans but not in Black Americans. This may be related to differences in population size and the limited power of subgroup analysis. In a meta-analysis involving healthy volunteers, the presence of major depression was associated with increased risk of adverse coronary events [12]. In patients who had undergone coronary artery bypass surgery, the occurrence of depressive symptoms was associated with a greater risk of graft disease progression [11]. Although the mechanism is unclear, poor psychosocial functioning may impact on cardiovascular health via biological mechanisms such as cortisol hypersecretion, disturbed hypothalamic–pituitary–adrenal axis function, elaboration of pro-inflammatory cytokines, endothelial dysfunction, atherogenic lipid profile and development of metabolic syndrome [8,10]. Unhealthy lifestyle changes (smoking, unhealthy diet, sedentary lifestyle, non-compliance with medication) have also been suggested as a possible explanation for the association between psychosocial factors and CHD [8,36–39]. Consistent with the present finding, a recent study involving 49,321 young Swedish men who were followed for 37 years showed that early-onset depression is not an independent predictor of CHD after adjusting for well-established vascular risk factors [40]. These observations raise concerns about the true association between depression and CHD.

In agreement with previous studies, the present study found a strong association between abnormal sleep duration and CHD [1–4]. Although the mechanism underlying the observed association is not well understood, it may relate to adverse physiological changes relating to CHD risk that are associated with sleep disturbance [41,42]. Both long and short sleep durations are associated with elevated markers of inflammation, abnormal lipid profile, insulin resistance, increased BMI, diabetes mellitus and hypertension [43–46], all of which are independent predictors of CHD [47]. Sleep disturbance in itself can constitute a form of stress [5], which is directly related to the degree of objective sleep disturbance [6]. This has been shown to predict coronary events, possibly mediated through increased sympathetic nervous system activity [7,8]. Investigators have observed that sympathetic arousal and early-morning insomnia were associated with cardiac disease in a sample of 4041 patients [48]. Among older adults, the severity of insomnia was directly related to use of cardiovascular drugs, general fatigue, exhaustion, angina pectoris, cardiac insufficiency,

worsened objective and subjective health, presence of negative T waves on electrocardiogram, and lower survival rates [49]. However, in the present study, abnormal sleep duration was not an independent predictor of CHD after controlling for other sociodemographic factors and health characteristics.

This study has some limitations. First, the findings may not be generalized to Black and White individuals across the lifespan, as the majority of the study population were White Americans aged <65 years. Also, research has shown clear evidence of race/ethnicity-based neurobiological differences in sleep regulation, depression and CHD risk [50]. Second, this analysis was based on subjective reports of depression, functional capacity and sleep duration. Future studies should examine associations between these factors using objective data.

In conclusion, this study found that functional capacity was an independent predictor of CHD after adjusting for sociodemographic factors and health characteristics, whereas depression and sleep duration were not independent predictors.

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Table 1

Comparison of sociodemographic and health data of Black and White Americans participating in the 2005 National Health Interview Survey.

Variable	Black Americans (15%)	White Americans (85%)	F/χ^2	P-value
Age (mean $\pm$ SD)	45.56 $\pm$ 16.91	48.02 $\pm$ 18.02	72	0.0001
Female sex (%)	61	56	52	0.0001
Income >\$35,000 (%)	16	24	140	0.0001
Body mass index (% obese)	52	38	193	0.0001
Hypertension (%)	36	27	133	0.0001
Diabetes (%)	12	8	62	0.0001
Heart disease (%)	6	8	29	0.0001
Arthritis (%)	22	24	11	0.0001

SD, standard deviation.

Table 2

Regression coefficients for coronary heart disease with sleep, depression, medical and sociodemographic factors, and functional capacity of the participants in the 2005 National Health Interview Survey.

Variables	Odds ratio	95% Confidence interval		P-value
		Lower	Upper	
Age	1.058	1.049	1.067	0.000
Gender	0.486	0.392	0.603	0.000
Race/ethnicity	0.674	0.489	0.930	0.016
Income	0.701	0.497	0.988	0.043
Hypertension	2.564	2.005	3.279	0.000
Diabetes	1.795	1.390	2.319	0.000
Arthritis	1.246	0.987	1.574	0.064
Body mass index	1.275	1.006	1.616	0.045
Sleep	1.083	0.830	1.412	0.557
Depression	1.236	0.988	1.546	0.064
Functional capacity	1.811	1.417	2.314	0.000

Table 3

Regression coefficients for coronary heart disease with sleep, depression, medical and sociodemographic factors, and functional capacity of Black and White Americans in the 2005 National Health Interview Survey.

Race	Variable	Odds ratio	95% Confidence interval		P-value
			Lower	Upper	
White	Age	1.055	1.048	1.062	0.000
	Gender	0.405	0.449	0.483	0.000
	Income	0.665	0.509	0.870	0.000
	Hypertension	2.644	2.179	3.209	0.000
	Diabetes	1.504	1.205	1.877	0.000
	Arthritis	1.360	1.128	1.641	0.001
	Body mass index	1.112	0.990	1.249	0.074
	Sleep	1.160	0.930	1.447	0.187
	Depression	1.289	1.070	1.552	0.008
	Functional capacity	1.731	1.418	2.112	0.000
Black	Age	1.034	1.013	1.056	0.001
	Gender	0.719	0.418	1.237	0.234
	Income	0.239	0.056	1.025	0.054
	Hypertension	7.330	2.955	18.181	0.000
	Diabetes	4.010	2.341	6.867	0.000
	Arthritis	1.074	0.593	1.943	0.814
	Body mass index	0.926	0.641	1.336	0.681
	Sleep	0.676	0.359	1.273	0.225
	Depression	1.555	0.915	2.641	0.103
	Functional capacity	2.407	1.305	4.439	0.005