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Letter to the Editor

Only Objectively Measured Sleep Duration and the Inclusion of the Entire Spectrum of Cardiovascular Disease can Establish a Reliable Link between the Two

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Letter to the Editor

We were interested to read the article by Xu *et al*. on a retrospective study on the association between sleep duration and cardiovascular disease (CVD) to investigate possible causality and provide a potential approach for early intervention in CVD [1]. Data were obtained from genome-wide association studies (GWAS) on sleep duration and 12 CVDs [1]. A negative association was found between sleeping too long (>8 hours) and valvular heart disease, myocardial infarction and heart failure [1]. A positive association was found between too little sleep (<7h) and non-ischemic cardiomyopathy, cardiac arrhythmias, valvular heart disease and atrial fibrillation [1]. The study is remarkable, but some points require further discussion.

The first point is that sleep duration does not depend on a single disease, but is multifactorial. Attributing sleep duration to a single disease is a simplification of the phenomenon of sleep. As far as we know, sleep is a complex phenomenon and is determined by numerous influencing factors that should not be neglected when conducting sleep studies. In general, sleep determinants can be endogenous or exogenous. Endogenous determinants include personality type, acute and chronic stress levels, sympathetic tone, a subject's ability to cope with exogenous or endogenous stressors, genetic background, comorbidities, comedications, and the ability to follow one's own rules and adhere to a structured lifestyle (e.g. always going to bed at the same time, the willingness to turn off lights, TV, headphones, radio, cell phone, i-Pad and lights in the bedroom and to remove all devices that generate electrosmog). Exogenous determinants include noise, light vibrations, draughts, insects, pets, children, partners, the time of the last meal or fluid intake, the intake of alcohol, adrenergic stimulants or pathogenic drugs.

The second problem is that the relationship between sleep and CVD is also highly dependent on the degree of CVD. Sleep cannot be disturbed by first-degree aortic valve stenosis, but it can be disturbed by third-degree aortic valve stenosis. Similarly, a fractional shortening of 35% may allow a subject to sleep well, whereas a fractional shortening of 15% may be associated with dyspnea at rest or on exertion and thus may severely disturb sleep.

The third point is that CVD includes not only arterial hypertension, coronary artery disease, pulmonary heart disease and arrhythmias, as mentioned in the introduction, but also left ventricular systolic or diastolic dysfunction, endocarditis, myocarditis, pericarditis, pericardial effusion, primary or secondary malignancy, benign neoplasm, valvular stenosis or insufficiency, non-compaction, Takotsubo cardiomyopathy, right-to-left interventricular or interatrial shunts or congenital heart disease. Therefore, including only a limited number of CVDs may introduce bias and over- or underestimate the impact of CVDs on sleep duration.

The fourth point relates to the way in which sleep duration was determined in the index study [1]. Self-assessment of sleep duration is inadequate because an individual is usually not able to accurately determine the time of falling asleep and waking up. The subjective assessment of poor sleep can be very different if the person is monitored in a sleep laboratory at the same time. Therefore, accurate determination of sleep duration can only be made by recording the electroencephalogram, oxygen saturation, muscle activity, respiratory rate, heart function and limb movements during sleep.

The fifth point is that valvular heart disease was negatively correlated with oversleeping and positively correlated with undersleeping [1]. This result is contradictory and suggests that valvular heart disease may not be a relevant factor for sleep duration. The results might be different if the correlation between sleep duration and specific valvular heart diseases such as aortic stenosis or insufficiency, pulmonary stenosis or insufficiency, or tricuspid stenosis or insufficiency is calculated.

In summary, it can be said that the index study has limitations that put the results into perspective. Addressing these limitations could strengthen the findings of the study. Before a reliable correlation between sleep duration and CVDS can be established,

sleep duration must be objectively measured and a specific CVD selected for analysis. Different types of CVD can affect sleep duration in different ways.
Addressing these limitations could strengthen the study's message. Before reliably correlating sleep duration with CVD, sleep duration needs to be objectively measured and specific CVD chosen for the analysis. The different types of CVD may influence sleep duration differentially.

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