Sleep quality and duration and cardiovascular diseases: is there an association?

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Abstract

The article reviews the relationship between the quality and duration of sleep and cardiovascular diseases (CVD). Chronic sleep deprivation and sleep disorders are common in modern society, however, many people underestimate their effects on health. Experimental data, population-based epidemiologic and interventional studies have shown that both short and long sleep duration are associated with CVD and their major risk factors. Thus, sleep duration could be considered an additional modified risk factor. Social factors, irregular sleep-wake cycle, some somatic and mental diseases, and sleep disorders themselves may decrease quality and duration of sleep. Normal sleep is a complex and dynamic process that significantly affects homeostasis of the cardiovascular system. The main pathophysiological mechanisms involved in CVD development and progression in subjects with the insufficient sleep considered to be the following: increased activity of the sympathetic nervous system, blunted circadian rhythm of blood pressure, impaired lipid metabolism, glucose intolerance and changes in the secretion of hormones that affect appetite. Thus, the novel goals of the cardiovascular prevention should include timely diagnosis and treatment of sleep disorders and sufficient sleep quality and duration.

Key words: cardiovascular diseases, hypertension, coronary heart disease, diabetes mellitus, obesity, sleep duration, sleep disorders, circadian rhythms, melatonin.

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Продолжительность и качество сна — есть ли связь с сердечно-сосудистыми заболеваниями?

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Резюме

В статье обсуждается взаимосвязь между качеством и продолжительностью сна и сердечно-сосудистыми заболеваниями (ССЗ). В современном обществе хроническая депривация сна и нарушения сна широко распространены, но многие недооценивают их эффекты на здоровье. Данные экспериментальных, популяционных эпидемиологических и интервенционных исследований показали, что продолжительность сна (как короткая, так и длинная) ассоциирована с ССЗ и их основными факторами риска и может являться дополнительным модифицируемым фактором риска. Социальные факторы, нерегулярный режим сна-бодрствования, некоторые соматические и психические заболевания и сами нарушения сна могут приводить к изменению продолжительности сна и снижению его качества. Учитывая, что нормальный сон является сложным и динамичным процессом, значительно влияющим на гомеостаз сердечно-сосудистой системы, в качестве основных патофизиологических механизмов, участвующих в становлении и прогрессировании ССЗ у лиц с нарушениями сна, рассматриваются следующие изменения: повышение активности симпатической нервной системы, нарушение суточного ритма артериального давления, нарушение липидного обмена, нарушение толерантности к глюкозе и изменения секреции гормонов, влияющих на аппетит. Для профилактики сердечно-сосудистых заболеваний в современном обществе представляется очень важным соблюдение режима с достаточной продолжительностью сна, а также выявление и лечение нарушений сна.

Ключевые слова: сердечно-сосудистые заболевания, артериальная гипертензия, ишемическая болезнь сердца, сахарный диабет, ожирение, продолжительность сна, нарушения сна, циркадианные ритмы, мелатонин.

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Cardiovascular diseases (CVD) remain to be a major cause of mortality in Russian Federation, constituting 38,8 % and 36,8 % of all-cause mortality structure in male and female population, respectively [1]. The search fo new CVD risk factors is ongoing. Their correction will contribute individual risk reduction by better control of overweight and obesity, arterial hypertension (HTN), lipid and carbohydrate metabolism etc. The growing data evidence that sufficient sleep duration is important for normal mental and physical functioning. Normalization of sleep quality and its adequate duration are crucial for primary and secondary CVD prevention [2].

A need for sleep decreases with aging, accounting for more than 13 hours for newborn, 9–10 hours for adolescents (14–18 years old) [3]. However, normal sleep duration for adults is not yet established, the optimal duration is considered as equal to 7 hours in the absence of daytime sleepiness [4]. The association between risk of cardiovascular events and sleep duration has the U-shape curve, which implies different personal mechanisms influencing the prognosis in long or short sleepers [4–6]. At the same time, the number of people with insufficient sleep duration (due to the shifting of sleep onset to later hours and early awakenings because of the work schedule [7, 8], psychosocial stress and sleep disturbances [2]) is increasing. About a quarter of the working population do shift-work. After night shifts duration of daytime sleep (that is compensatory in order to restore sleep deficiency) is 1–4 hours less than usual night sleep. Sleep disorders are known to appear in three-quarter of shift-workers [9, 10]. Shift-work is associated with increased cardiovascular morbidity (including coronary heart disease, CHD; myocardial infarction, MI; and atherosclerosis) and mortality.

Epidemiological studies on sleep duration

Surveys of the US population have shown that duration of sleep in adults is gradually reducing [11]: in 2002 it constituted 6 hours and 54 minutes at weekdays and 7 hours and 30 minutes — at weekends [12], and in 2013 it was 6 hours 31 minutes and 7 hours 13 minutes, respectively. Also, the duration of sleep varies in different countries, e. g. accounting in Germany 7 hours and 1 minute on weekdays and 8 hours

Table 1

AN ASSOCIATION BETWEEN SLEEP DURATION AND CARDIOVASCULAR DISEASE AND MORTALITY BASED ON THE RESULTS OF THE MAIN COHORT STUDIES

Estimated end point	Name of study, sleep evaluation method	Groups	Age of participants	Study period (years)	Sleep duration (hours)	Results RR (95 % CI), adjusted for the key risk factors
1	2	3	4	5	6	7
HTN development	NHANES, 2006 [19], Canvass	5 806 males 3 983 females	32–59	8–10	≤ 5	RR 1.60 (1.19–2.14)
					< 6	RR 1.05 (0.83–1.31)
			60–86	8–10	≤ 5	RR 0.85 (0.50–1.45)
					< 6	RR 0.86 (0.56–1.33)
Change in BMI	NHANES, 2005 [30], Canvass	8 073	25–74	8–10	2–4	2.51 (0.83–7.53)
					5	1.07 (0.58–1.97)
					6	1.24 (0.84–1.82)
HTN development	Nurses' Health Study, 2013 [49], Canvass	82 969	30–55	14	≤ 5	RR 1,01 (0.91–1.12)
Weight gain	Nurses' Health Study, 2006 [24], Canvass	68 183	45–65	4		7–8-hours sleep duration is associated with lower BMI; U-phenomenon
HTN development	Whitehall II Study , 2014 [21] Canvass	3 691	35–55	–	≤ 5	RR 1.31 (0.65–1.42)
					< 6	RR 1.42 (0.94–2.16)
Development of obesity (BMI ≥ 30 kg/m2)	Whitehall II Study, 2008 [31], Canvass	4 378	35–55	16	≤ 5h	OP 1.05 (0.6–1.82)

1	2	3	4	5	6	7
All-cause mortality	Finnish Twin Cohort, 2007 [17], Canvass	9 529 males 10 265 females	35–55	22	< 7	OP 1.27 (1.16–1.39)
					> 8	OP 1.27 (1.17–1.38)
All-cause mortality	JACC Study 2004 [18], Canvass	41 489 males 57 145 females	40–79	9,9	< 6 > 9	7-hour sleep duration is associated with lower mortality; U-phenomenon
Development of CHD and CVD	The MORGEN Study, 2011 [23], Canvass	20 432	20–65	12	< 6	CVD: RR 1.15 (1.00–1.32); CHD: RR 1.23 (1.04–1.45)
					Low sleep quality	CVD: RR 1.04 (0.87–1.26); CHD: RR 1.19 (0.95–1.5)
Change in BMI	The Penn State Cohort, 2014 [32], polysomnography	815	48,9 ±13,4	7,5	< 5	RR 1.08 (95% CI 0.48–2.41); p > 0.05
Mortality	The Penn State Cohort, 2010 [25], polysomnography	741 males	50,2 (14,5)	13,9	< 5	RR 1.34 (0.79–2.28) In combination with insomnia RR 4 (1.14–13.99)
		1000 females	47,4 (12,6)	10,3		RR 1.41 (0.47–4.19) In combination with insomnia RR 0.36 (0.03–4.33)
HTN development	The SWAN (Study of Women's Health Across the Nation), 2014 [26], polysomnograpy	355	42–52	7	< 6	RR 2.26 (1.19–5.86)
HTN development	The CARDIA Sleep Study, 2009 [27], actigrapy	578	33–45	6	< 6	RR 1.30 (0.96–1.75)

1	2	3	4	5	6	7
Dyslipidemia	The CARDIA Sleep Study, 2013 [28], actigraphy	503	32–51	10	Every 1 hour to increase sleep duration	TC raise to 5.2 (1.7–8.6) mg/dl. p = 0.003 HDL raise to 3.4 (0.2–6.6) mg/dl; p = 0.039 Risk of dyslipidemia assessed by the ratio TC/HDL ≥ 5.0: RR 1.23 (0.99–1.63). p = 0.012
Change in BMI	The CARDIA Sleep Study, 2009 [34], actigraphy	612	33–45	5	< 6	RR –0.02 (–0.30–0.25); p = 0.86

Note: HTN — hypertension; BMI — body mass index; CHD — coronary heart disease; CVD — cardiovascular diseases; RR — relative risk; CI — confidence interval; TC — total cholesterol; LDL — low density lipoproteins; HDL — high density lipoproteins.

00 minutes on weekends, and in Japan — 6 hours 22 minutes and 7 hours 12 minutes, respectively [13]. None epidemiological study of sleep duration and quality was carried out in Russia. In a survey of 1,552 residents of St. Petersburg [mean age 50 (21–68) years; 56.2% female] participated in the Epidemiological study of cardiovascular morbidity in different regions of Russia (ESSE-RF), the average sleep duration was 7.3 ± 1.2 hours. At the same time 22.5% participants reported 6-hour or shorter sleep, and 12.4% respondents slept more than 9 hours per day [14]. Moreover, among St Petersburg residents every fourth adult respondent complained of sleep disturbances (such as difficulties in falling asleep, frequent awakenings) occurring more often in women and elderly people [15].

Both short and long sleep is associated with unfavorable health effects (Table 1) leading to the increased overall mortality [4–6, 16–18], CVD incidence [19–21], diabetes mellitus (DM) [22], dyslipidemia, and obesity in adults, as well as in children [23]. However, the underlying mechanisms of these relationships still remain poorly understood and require further careful investigation [24–31].

A meta-analysis of large epidemiological studies (Cancer Prevention Study I and II, National Health and Nutrition Examination Survey, Framingham Study, Japan Collaborative Cohort Study), which includes data of 474,684 subjects observed for 6.9–25 years demonstrated [16] an association between short sleep duration and a higher risk of coronary heart disease (CHD), heart failure (HF), including fatal cases [relative risk (RR) 1.48, 95% confidence interval (CI) (1.22–1.80), p < 0.0001], and stroke [RR 1.15, 95% CI (1.00–1.31), p = 0.047]. At the same time a short nap was not associated with the increase in total cardiovascular risk [RR 1.03; 95% CI (0.93–1.15), p = 0.52]. Long sleep duration (more than 9 hours per day) was also associated with an increased risk of death due to HF [RR 1.38, 95% CI (1.15–1.66), p = 0.0005], with a higher risk of stroke [RR 1.65, 95% CI (1.45–1.87), p < 0.0001] and the overall CVD risk [RR 1.41, 95% CI (1.19–1.68) p < 0.0001].

Sleep duration, metabolic syndrome and obesity

Both metabolic syndrome and obesity are well-known CVD risk factors [29]. The impact of sleep duration on obesity was analyzed in a meta-analysis (2008) [23]. The data from 634,511 respondents from 30 studies were included (of those 12 studies were conducted among children and 18 were conducted among adults). Risk of obesity in children was 1.89 (95% CI 1.46–2.43; p < 0.0001) and 1.55 (95% CI 1.43–1.68; p < 0.0001) in adults with short sleep duration. The influence of sleep duration on weight was similar almost in all age groups. Regression analysis confirmed an association between 1-hour shortening of adult

sleep and 0.35 kg/m2 increase in body mass index (BMI). Cohort Nurses' Health Study [24] and the Rotterdam study [32] showed an U-shaped association between sleep duration and obesity. However, other studies, e. g. Whitehall II Study [23] (subjective assessment of sleep duration by questionnaires), The Penn State Cohort (sleep assessment by polysomnography [33]) and the Coronary Artery Risk Development in Young Adults Study (CARDIA) (sleep assessment by actigraphy [34]) did not show any effect of short sleep duration on weight gain.

When considering sleep duration in weekdays and weekends separately, each hour difference in sleep duration in working and non-working days (so called «social jet lag») led to the increase in risk of overweight and obesity by 33 % [7]. In recent years, a «social jet lag» is considered to be an important indicator of diurnal biological rhythms stability. The larger the difference in sleep duration in weekdays and weekends, the more profound is «social jet lag», and the higher the rate of smoking, drinking and coffee consumption [35].

Changes in sleep and wake cycle are known to affect basal metabolism, feeding behavior and physical activity [36]. Imbalance between feed and energy consumption may lead to metabolic disorders, including obesity and DM. The main component of homeostasis is the ability to coordinate the daily pattern of activity and food intake, energy consumption and conservation during the day. It is supported by the synchronization of the endocrine system functioning with daylight [37]. Even one night of sleep deprivation leads to an increase in food intake and reduced energy consumption regardless of serum glucose levels in healthy humans [38, 39]. Moreover, people tend to eat more food high in carbohydrate and fat after partial sleep deprivation [40]. In a three-week interventional study the increase in sleep duration from usual 6.5 hours to 7.1 per day in overweight adults ($p < 0.001$) reduced the drowsiness and appetite by 14 % ($p = 0.034$), as well as the desire for sweet and salty food by 62 % ($p = 0.017$) [41].

Different experimental and epidemiological studies showed that the prevalence of impaired glucose tolerance (IGT) and DM increases among individuals with changed sleep quality and duration [42]. One of the first epidemiological studies was the Sleep Heart Health Study that demonstrated an association between short sleep and the risk of DM and IGT [43]. In a prospective study CARDIA, only sleep quality was associated with serum glucose and insulin levels but not sleep duration assessed by actigraphy [44].

One of the explanations for the relationship between sleep duration and appetite might be circadian rhythm of appetite-regulating hormones coupled with the «sleep-wake» cycle. Disruption in neuropeptides ghrelin and leptin interaction affects the energy balance and leads to increased appetite and uncontrolled food intake in short and fragmented sleep [45]. The duration of sleep less than 5 hours per day is associated with decreased ghrelin levels [46].

Thus, some epidemiological studies have established an association between short sleep duration and increased incidence of DM and obesity. Experimental studies have shown a decreased insulin sensitivity and glucose tolerance in subjects with low sleep quality and short sleep duration. Experimental sleep deprivation also leads to the behavioral and physiological changes resulting in a positive energy balance by increasing food intake and weight gain.

Sleep duration, blood pressure and risk of hypertension

Recent experimental and epidemiological studies show that short sleep increases blood pressure (BP) and may lead to hypertension [21, 26, 27]. Normal sleep is a complex and dynamic process, significantly affecting the cardiovascular homeostasis. Some changes are associated with diurnal (circadian) regulatory pathway activity rhythms during sleep. Other changes correspond to certain sleep stages. Changes in the autonomic nervous system (ANS) affect BP dynamics and heart rate (HR) while sleeping. Microneurography studies demonstrated that there is a continuous decrease of sympathetic drive to the peripheral vascular bed with NREM-sleep onset and deepening and its increase in REM-sleep. This phenomenon is reflected in HR and BP changes in different sleep phases [47].

There was an U-shaped relationship between sleep duration and hypertension prevalence in the

Sleep Heart Health Study (SHHS), and 7–8 hours was considered an optimal sleep duration [48]. In a prospective National Health and Nutrition Examination Survey (NHANES) there was a 1.6-fold increase in the risk of hypertension development in people aged 32–59 years old with sleep duration less than 5 hours. However, it remained unchanged in the elderly group [19]. Wherein, sleep duration equal to 6 hours or less was not associated with an increased risk of hypertension in the elderly. At the same time, the 14-year follow-up did not show an increase in hypertension risk in nurses with short sleep duration [49]. A prospective Whitehall II study showed that risk of hypertension increased only in females with short sleep [21]. The larger percentage of slow wave sleep in young people and females may serve a possible explanation. CARDIA study showed an association between short sleep and hypertension development even in young healthy individuals when sleep duration was objectively assessed by actigraphy [27]. The seven-year prospective study of women (The Study of Women's Health Across the Nation) demonstrated a 2.26-fold risk of hypertension when the «gold standard» for sleep evaluation (polysomnography) was used [26]. Russian researchers (Martynov AI et al, 2002) found a 5-fold risk of nocturnal hypertension in older hypertensive patients with self-reported impaired sleep quality [50].

The main mechanisms for BP elevation in sleep disorders include the impairment of circadian BP profile and HR. Short-sleepers are characterized by increased daytime activity. It leads to a long-term sympathetic activation, resulting in increased catecholamine secretion and, as a consequence, in HR and BP elevation [51]. Kato M. et al (2000) in an experiment work showed an increase of systolic blood pressure (SBP) by 6 mmHg and of diastolic blood pressure (DBP) 3 mmHg for after one night of partial sleep deprivation (up to 3.6 hours on average) [52]. They discussed increased norepinephrine excretion and sympathetic activity as the possible mechanisms to cause such an effect. Ekstedt et al. (2004) found an increase of BP, cholesterol and cortisol in a group of people with frequent nocturnal awakenings, and normal sleep duration. They suggested that both sleep duration and quality are important [53]. Haack M. et al (2013) hypothesized that an increased sleep duration might contribute to BP reduction. An experimental study was conducted in order to prove the hypothesis that the increase in sleep duration in people with habitual sleep duration of less than 7 hours can lead to BP reduction. It demonstrated that sleep elongation for 35 ± 9 minutes on average for 6 weeks resulted in reduction of SBP for 14 ± 3 mmHg and of DBP for 8 ± 3 mmHg ($p < 0.05$) in participants with high normal BP or HTN of 1st grade [54].

The synchronization between circadian (daily) BP rhythm and sleep-wake cycle is regulated by the suprachiasmatic nucleus (SCN), located in the hypothalamus, and its impact on ANS and hormone levels. Light transmits the information through the retina and retinohypothalamic tract to the SCN and pineal gland, leading to a change in melatonin secretion [55]. Mismatch of lighting with sleep-wake rhythm may lead to an impaired perception of circadian rhythms by SCN, disrupting circadian BP rhythm in susceptible individuals [56]. In experimental studies single dose of short-acting exogenous melatonin caused neither BP decrease nor sleep quality enhancement in hypertensive subjects. Opposite results were found in long-term use of melatonin. SBP and DBP decreased by 6 and 4 mmHg, respectively, during sleep three weeks after melatonin use. Furthermore, melatonin contributed to sleep onset latency reduction from 33 to 22 minutes ($p = 0.036$). At the same time sleep efficiency increased from 80 to 85% ($p = 0.017$), and sleep duration — from 5.6 to 6.1 hours ($p = 0.013$) [57]. Herewith, the hypotensive effect of short-acting melatonin was not confirmed by meta-analysis of randomized controlled trials. SBP and DBP reduced by –0.3 mmHg [95% CI (–5.9; –5.30); $p = 0.92$], and –0.2 mmHg [95% CI (–3.8; –3.3; $p = 0.89$] when short-acting melatonin was used. At the same time slow-release form of melatonin led to SBP falling by –6.1 mmHg [95% CI (–10.7; 1.5), $p=0.009$] and DBP — by –3.5 mmHg [95% CI (–6.1, –0.9), $p = 0.009$] [58].

The growing evidence suggests that sleep duration impairment might lead to BP increase and alter its daily profile. According to the interventional studies the increase in sleep duration in short-sleepers and exogenous melatonin (as a medicine) use can contribute to the BP lowering.

Sleep duration and coronary heart disease

Effect of sleep duration on the development of CHD may be mediated through intermediate risk factors such as an increase of BMI, BP, lipid and carbohydrate metabolism. The MORGEN (Monitoring Project on Risk Factors and Chronic Diseases in the Netherlands), a Dutch cohort study, showed an increase of CHD risk by 23% in short-sleepers. The relative CHD-associated mortality risk was 1.57 and 1.79 (sleep duration less than 5 and more than 9 hours, respectively) according to the prospective observation of the Singapore population [59]. According to the survey of Russian population within WHO project "MONICA — psychosocial», at 8-year follow-up risk of MI was 9.25 times higher in men aged 25–44 years who reported their sleep quality as "bad" compared to age-matched men who assessed their sleep as «good» [60]. Unfortunately, sleep duration was not evaluated in this study. The five-year prospective study CARDIA showed an increased risk of coronary artery calcification with 1-hour shortening of sleep. By the way, one extra hour of sleep had a beneficial effect on the risk of coronary artery calcification, comparable with the effect of SBP lowering by 16.5 mmHg [61]. An increased risk of hyperlipidemia with a shorter sleep was also shown in the mentioned study: the rate of elevated total cholesterol and low density lipoprotein (LDL) ≥ 5,0 mmol/l was 1.23 times higher (95% CI 0.99–1.63; p = 0.012) [28].

A J-shaped association between intima-media thickness (IMT) and sleep duration was established in epidemiological Study of Health in Pomerania (varying from 0.81 ± 0.21 mm or less in subjects sleeping 5 hours per day to 0.89 ± 0.29 mm in those sleeping 11–12 hours per day). The tiniest IMT 0,76 ± 0,15 mm was in people with the subjective sleep duration of 7 hours per day (p <0.001). Moreover, the last ones were characterized by the lowest SBP levels and the lowest glycated hemoglobin HbA1c, as well as the highest level of physical activity and lower weight [62].

Activation of pro-inflammatory markers may be another mechanism mediating the effect of impaired sleep quality and duration on the CHD development. It is widely known that C-reactive protein (CRP) is one of the predictors of stroke and MI risk. Partial sleep deprivation was shown to be associated with CRP elevation [63]. However, no association was found between CRP and subjective and objective sleep duration in a large-scale epidemiological study Wisconsin Sleep Cohort Study [64]. A number of studies named Withal demonstrated an inverse relationship between the sleep quality and CRP levels in certain subgroups of subjects reflecting a higher pro-inflammatory activity in patients with low sleep quality (based on the questionnaires) [65].

Sleep fragmentation and cardiovascular diseases

The quality of sleep can be affected by various diseases and sleep disorders themself that are the most common psychopathological conditions. The most frequent sleep disorders are insomnia (its prevalence is 30% in the general population and increases to 50% in elderly) [66], and obstructive sleep apnea (OSA) or paroxysmal nocturnal dyspnea (PND) (diagnosed in 10% women and 30% men [67]). A meta-analysis of epidemiological cohort followed up for 3–20 years showed up to 45% increase of CVD-related mortality risk associated with insomnia (RR 1.45; 95% CI 1.29–1.62; p<0.00001). Sympathetic nervous system activation, inflammatory cytokine and cortisol levels, circadian BP profile alteration and impaired glucose tolerance are considered as the main mechanisms underlying insomnia impact on cardiovascular system [68].

OSA is characterized by occurrence of repetitive occlusions in upper respiratory tract, leading to the episodes of apnea and hypopnea (lasting for 10 seconds or more) with continuing efforts of the respiratory muscles and movements of the chest and abdominal wall. To date, accumulated evidence indicates the relationship between sleep breathing disorders and CVD, e. g. BP elevation, the risk of adverse cardiovascular events (myocardial infarction, acute cerebrovascular events, cardiac arrhythmias) [67, 69].

OSA is known to be an independent risk factor for hypertension [70], leading to resistant hypertension in some cases [71]. Russian prospective studies with 5–7-year follow-up demonstrated a threefold increase of DM risk in patients with OSA, regardless to other risk factors (RR 3.13; 95% CI 1.22–8.07; p = 0.019) [72]. The main mechanisms of OSA impact

on the cardiovascular system include impaired sleep quality and continuity (fragmentation), hypoxia and hypercapnia, leading to sympathetic activation, BP elevation, metabolic disorders, endothelial dysfunction and hypercoagulation. Negative intrathoracic pressure and its nocturnal oscillations additionally increase left ventricular overloading [69].

Recent data evidence that periodic leg movements (PLM) are the risk factor for CVD development. PLM are often diagnosed only based on polysomnography data and are not accompanied by subjective complaints [73, 74]. Furthermore, children with PLM index ≥ 5 per hour of sleep had significantly higher risk of hypertension (both systolic and diastolic BP elevation): RR 6.25 (95% CI 1.87–20.88) and RR 4.83 (95% CI 1.66–14.07), respectively, $p < 0.05$, adjusted for key risk factors) [75]. The mechanisms underlying PLM effects on the cardiovascular system include sleep fragmentation due to repeated arousals, thereby increasing sympathetic activity. However, they require further studying.

The main sleep disorders — insomnia and OSA — contribute substantively to the development of sleep structure disruption. They are also associated with the development of CVD and risk factors manifestation. Considering this, timely diagnosis and correction of sleep disorders are extremely important.

General limitations of research

There are similar limitations in lots of investigations studying the effects of sleep duration and quality on CVD development. Epidemiological studies often consider only self-reported sleep duration. The peculiarities of questionnaire wording may also cause differences in survey results. The standard question is "How long is your night sleep?" It does not include daytime naps (which have particular importance in older persons, in patients with hypersomnia, shift-workers and others). Night sleep quality usually is not regarded in these questionnaires. Moreover, sleep duration can be shorter due to the longer time of falling asleep and frequent awakenings. These lead to sleep fragmentation. Older people may overestimate or underestimate their sleep duration because of cognitive decline [76]. Also, patients with sleep disorders (e. g., OSA, PLM etc.) and severely disrupted sleep structure often do not report changes in sleep duration. CV risk in these patients may increase due to sleep disturbances per se rather than a change in its duration or quality (e. g., the risk of hypertension in patients with OSA is mediated by different mechanisms, while night sleep duration may be normal and the total (diurnal) sleep time exceeds 10–11 hours due to severe daytime sleepiness). These factors are not always taken into account in surveys.

On the other hand, small sample size with the low statistical power is a common limitation in studies assessing objective sleep measures. Also, these studies do not include repeated measurements of sleep duration, thus impeding the validity assessment of a single measurement of sleep duration for the follow-up. This is especially important in long-term prospective studies.

When assessing CVD risk in sleep disorders, the possible deleterious effects of common concomitant somatic diseases regarding sleep quality should be taken into account, in particular, gastro-esophageal reflux disease [77] and irritable bowel syndrome [78], cancer [79], musculoskeletal disorders often associated with severe pain [80], acromegaly [81], mental illness [82], Parkinson's disease [83], asthma [84], chronic kidney disease [85].

Conclusion

Current data show the importance of adequate sleep duration in CVD prevention. It is necessary to identify the causes of sleep duration disorders in time and to conduct activities to improve sleep quality. Sleep apnea and insomnia are main sleep disorders, resulting in poor sleep quality and short duration.

Interventional studies have shown that an improvement of the insufficient duration and poor quality of sleep lead to a lowering in appetite and BP, which may be beneficial regarding the target-organ damage and the CVD risk reduction. Though, this statement requires confirmation in further prospective studies.

When planning the research on sleep duration and its association with the somatic diseases, the objective methods for sleep duration assessment should be included. The following indicators should be also evaluated: sleep time, duration, and frequency (number) of nighttime awakenings and time of a day in which the subject was sleeping.

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