Hypertension Research (2022) 45:1213–1216
https://doi.org/10.1038/s41440-022-00942-z

COMMENT

Effectiveness of regular physical activity in preventing the progression of arterial hypertension: improved cardiovascular autonomic control during sleep

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Received: 10 April 2022 / Accepted: 16 April 2022 / Published online: 17 May 2022

Regular physical activity is widely recognized as an effective nonpharmacological intervention to treat systemic arterial hypertension. Indeed, physical activity is a part of comprehensive lifestyle interventions aimed at managing arterial pressure, as acknowledged by the recommendations of several professional organizations [1, 2]. For example, a joint report from the American College of Cardiology and the American Heart Association recommends a non-pharmacological lifestyle modification treatment for patients with elevated blood pressure, initial (stage I) hypertension, and an atherosclerotic cardiovascular disease risk score <10% [2]. The cardiovascular benefits of an active lifestyle include a blood-pressure-lowering effect, which is observed after different types of regular physical activity, such as aerobic, dynamic resistance, or combined exercise [3]. These benefits are mediated mainly by reduced peripheral resistance resulting from neurohumoral and structural adaptations [4].

Aside from regular physical activity, adequate sleep is an essential component of cardiovascular health. Notably, sleep deprivation is a common issue in modern societies [5], and its prevalence has been increasing due to constant exposure to artificial light and interactive activities, which combine with social and economic pressures to shorten the time spent asleep [6]. From an acute perspective (i.e., one night without sleeping), sleep deprivation results in arterial pressure elevation and the activation of mechanisms to counteract this hypertensive response, such as increased natriuresis and parasympathetic activity [7]. In contrast, from a chronic perspective (i.e., several nights without sleeping), sleep deprivation is associated with adverse consequences, including daytime sleepiness, decreased cognitive performance, increased sympathetic tone, inflammation, and impaired glucose tolerance [8]. These impairments are observed, for example, in shift workers who have inadequate sleep due to circadian misalignment and irregular shifts. Habitual decreased sleep duration and the subsequent physiological changes contribute to the increased risk of hypertension, cardiovascular disease, obesity, and diabetes in this population [8].

The study by Chen et al. [9] investigated associations between the elements mentioned in the previous paragraphs by examining the roles of autonomic function and sleep in mediating the antihypertensive effects of physical activity. The authors also explored the chronological sequence of changes in arterial pressure, cardiovascular autonomic regulation, and sleep patterns while the spontaneously hypertensive rats (SHRs) had access to a running wheel.

The effects of regular physical activity were assessed in male SHRs (from 10 to 20 weeks of age), an experimental model with similar features to those observed in humans with essential hypertension [10]. By their 16th week of life, SHRs have sustained elevated arterial pressure [11, 12], sympathetic hyperactivity, increased peripheral resistance [13], and endothelial dysfunction [12]. These hypertensive rats also have a limited ability to regulate their body temperature, at least in part due to central dysfunctions, including altered neuronal activation in the paraventricular hypothalamic nucleus [14], an autonomic premotor nucleus. Finally, SHRs have impaired endurance on treadmill tests [15], and as reported earlier by the same research group, these rats have less sleep time, fewer quiet and paradoxical sleep episodes, and a greater tendency to wake up from quiet sleep than normotensive rats [16].

A wireless sensor was mounted on the rats' heads, and this equipment acquired electroencephalographic and

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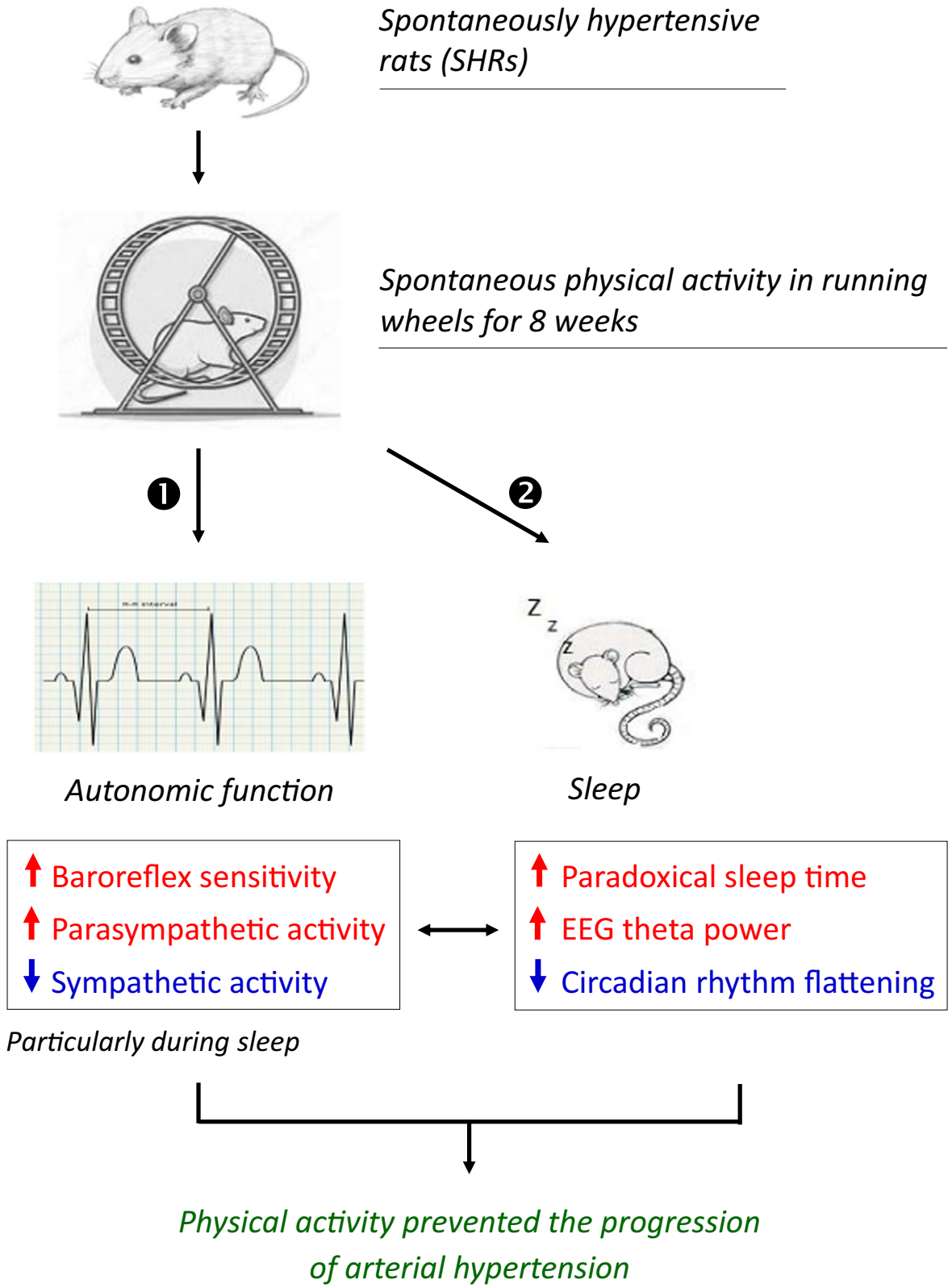

Fig. 1 The physiological mechanisms underlying the effects of regular physical activity in preventing the progression of arterial hypertension. The numbers 1 and 2 indicate that adaptations related to autonomic control occurred earlier than those related to sleep. The upward arrows indicate "increase/improvement", whereas the downward arrows indicate "reduction/attenuation". EEG electroencephalogram

electrocardiographic (ECG) signals. These signals were recorded for 24 h once a week over 11 weeks [9]. The R–R interval (RR) was determined continuously from the digitized ECG signals and then used to quantify the heart rate variability, a measure of autonomic activity. Another telemetry transmitter was implanted in the abdomen to collect pulsatile arterial pressure signals via the tip of an arterial catheter inserted into the abdominal aorta. The equipment used, which allowed the maintenance of signals over a long period in rats exposed to wheel running, is a critical methodological advance of the present study [9].

In the current study, because wheel running serves as an experimental model of spontaneous physical activity, the rats also had access to a running wheel for 8 weeks [9]. Interestingly, the research group previously reported the suppression of age-related increases in blood pressure and vascular sympathetic activity during sleep in SHRs chronically exercised on a treadmill [17]. Notably, as evidenced by Li et al. [18], wheel running causes lower stress levels than treadmill running, which is known as a forced physical exercise paradigm.

The findings of the current study, summarized in Fig. 1, revealed that wheel running not only prevented the progression of hypertension but also upregulated parasympathetic activity and baroreflex sensitivity and downregulated sympathetic activity, especially during sleep [9]. These adaptations may account for the effective improvement in hypertension management in physically active SHRs. Furthermore, wheel running reduced wakefulness and augmented paradoxical sleep during the early-light period, with the opposite findings during the dark period (i.e., wheel running attenuated the circadian rhythm flattening). Remarkably, the autonomic changes occurred earlier than the sleep pattern changes induced by regular physical activity, even though these adaptations were correlated [9].

In summary, the present findings are exciting and open the door to new lines of investigation to understand the mechanisms underlying the effects of regular physical activity in preventing the progression of hypertension, which may ultimately reduce the incidence of cardiovascular diseases. One important question that still needs to be addressed is whether regular physical activity will also induce favorable adaptations in SHRs subjected to pharmacological treatment, which would be an experimental model even more similar to human hypertension. It may also be helpful to understand the central adaptations (i.e., brain areas and neurotransmitters) mediating the improved cardiovascular autonomic control and baroreflex sensitivity, particularly during sleep. In this sense, the readership of Hypertension Research looks forward to the novel contributions of Dr. Cheryl Yang's research group on this topic.

Compliance with ethical standards

Conflict of interest The authors declare no competing interests.

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