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Sleep-related erectile dysfunction

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ABSTRACT

Introduction: Sleep is crucial for preserving general health, and the emergence of numerous chronic illnesses is associated with sleep deprivation. Recent studies suggest that short sleep duration may also have negative effects on the reproductive system. Sleep disturbances are increasingly associated with erectile dysfunction through their impact on hormonal balance and physiological regulation.

Aim of the study: This article summarizes the interplay between physiological processes such as sleep and erection, based on an analysis of information regarding the functioning of the autonomic nervous system, hormones, neurotransmitters, and pro-inflammatory cytokines, as well as the NOS-NO-cGMP signaling pathway and REM-related erections.

Material and Methods: A literature review was conducted using PubMed and Google Scholar to identify studies on the physiology of erections and the impact of sleep disorders on the erectile mechanism. Peer-reviewed clinical trials, systematic reviews, and other studies were analyzed to provide a comprehensive overview of the current state of knowledge.

Conclusion: Erectile dysfunction is becoming increasingly common, affecting not only older men but also younger men as well. One important contributing factor is poor sleep, which can lead to serious hormonal and neurological disturbances. This disrupts both the molecular mechanism and the functioning of the central nervous system in relation to the development of an erection.

Keywords: erectile dysfunction, penile erection, short sleep duration, sleep disorder, mechanism, regulation

1. INTRODUCTION

Male erection is a natural physiological phenomenon essential for reproduction [1]. The mechanism underlying it is complex and requires the interaction of multiple systems, including biochemical (NOS-NO-cGMP signaling pathway), cellular (smooth muscle relaxation mechanisms), neural (autonomic and somatic pathways), and hormonal (testosterone) systems [2]. The central nervous system can induce erections without peripheral stimuli, e.g., in nocturnal erections. This is a natural

reaction that indicates homeostasis, which is maintained, among other things, by proper sleep hygiene [3].

Nowadays, due to a variety of factors—such as longer working hours, shift work, urbanization, exposure to artificial cold light, participation in nighttime activities, as well as stress, mood disorders (depression), and chronic illnesses—the quality of sleep has declined, and the average duration of sleep has shortened [4],[5].

Sleep plays a key role in regulating hormones that are essential for male reproductive health. Adequate sleep is necessary for the optimal functioning of the hypothalamic-pituitary-gonadal (HPG) axis, which regulates hormone production and influences various aspects of male reproductive health. Disruptions in sleep quality and rhythm can lead to significant hormonal imbalances, which may adversely affect fertility [6].

2. AIM OF THE STUDY

This article summarizes the interplay between physiological processes such as sleep and erection, based on an analysis of information regarding the functioning of the autonomic nervous system (ANS), hormones, neurotransmitters, and pro-inflammatory cytokines, as well as the NOS-NO-cGMP signaling pathway and REM-related erections.

3. MATERIALS AND METHODS

This review was conducted using scientific literature available in the PubMed and Google Scholar database. Publications related to erectile dysfunction, physiological erection and sleeping duration were analyzed. Search terms included:

“physiological process of erection" | "erectile dysfunction" | "the role of sleeping" | "stages of sleeping" | "sleep-related erecrion" | "sleep disorders and chronic diseases" | “molecular mechanisms of penile erection”

Peer-reviewed clinical trials, systematic reviews, and other studies were analyzed to provide a comprehensive overview of the current state of knowledge. A total of 45 publications were selected and used to prepare this review.

4. STATE OF KNOWLEDGE

4.1 Autonomic Nervous System

The sympathetic pathway begins in the twelfth thoracic, travels to the second lumbar spinal segment, and then travels to the sympathetic chain ganglia via the white rami. After reaching the inferior mesenteric and superior hypogastric plexuses via the lumbar splanchnic nerves, some fibers go to the pelvic plexus via the hypogastric nerves. The sympathetic fibers in humans typically originate from the T10 to T12 segments, while the sacral and caudal ganglia contain the chain ganglia cells that project to the penis. Neurons in the columns of interneurons of the second, third, and fourth segments of the sacral spinal cord form the parasympathetic pathway. Sympathetic nerves from the superior hypogastric plexus join the preganglionic fibers, passing through the pelvic nerves to the pelvic plexus. The penis is innervated by branches of the pelvic plexus known as the cavernous nerves. While flaccidity results from stimulation of the sympathetic trunk or the hypogastric nerve, erection is induced by activation of the pelvic plexus and the cavernous nerves. This clearly suggests that flaccidity is caused by the thoracolumbar sympathetic pathway, and erection by a sacral parasympathetic impulse [7] .

An erection may be triggered by a nerve impulse traveling through a neuron in the somatic nervous system, but the autonomic nervous system (ANS) is responsible for the mechanical process of achieving an erection [8]. An erection is a hemodynamic process that primarily involves the dilation of arteries and the restriction of venous outflow, through the ANS’s control of the smooth muscles of blood vessels and also of corpus cavernosum [9], [10]. One or more venules emerge from the lacunar area and combine to form a subtunical venule, which drains as the emissary vein through the tunica albuginea. The intralacunar smooth muscle is constricted when the penis is flaccid. The emissary veins are physically constricted, the lacunar space and helicine arteries dilate, and the subtunical venules are physically and potentially neurologically compressed. As a result, the lacunar gap turns into a sizable vascular "sink" where blood quickly flows and is trapped, raising the pressure inside the lacunar space to mean arterial pressure [11].

4.2 Hormones, Neurotransmitters, and Pro-inflammatory cytokines

The body goes through significant hormonal changes when you sleep. The natural circadian cycle of testosterone levels causes them to peak in the early morning and then progressively decrease during the day [12]. Research from the National Health and Nutrition Examination Survey, for instance, revealed a drop in testosterone levels of roughly 5.9 ng/dL for each hour of sleep lost [13]. This suggests that inadequate sleep can result in decreased testosterone production. Moreover, a 10–15% decrease in testosterone levels in just one week is linked to sleep deprivation, indicating that long-term sleep loss may result in serious hormonal imbalances [14]. Testosterone plays a direct role in penile erection by being a component of the NOS-NO-cGMP signaling pathway. Testosterone has been shown to have both central and peripheral effects on erectile function, particularly in the preservation of libido. A hundred Testosterone-therapy restores libido and sexual activity in hypogonadal males, but decreased testosterone levels in males invariably result in a fall in sexual interest and erectile performance [45]. Additionally, a fascinating new study discovered a negative relationship between testosterone levels and the degree of penile deformity in males with Peyronie's disease, suggesting that testosterone may have a role in avoiding fibrosis. This would suggest that both primary and secondary erectile dysfunction are influenced by testosterone levels [15].

One of the hormones whose concentration was a result of a response to poor-quality sleep was TSH. [16] Human and rat CC (corpus cavernosum), endothelium, and SMC (smooth muscle cells) have been shown to have thyroid hormone receptors [17]. Hyperthyroidism decreased the neurogenic and endothelium-dependent relaxation of SMC in the CC in rabbits, suggesting a compromised NO-dependent relaxation of the CC [18]. Rats with hypothyroidism brought on by propyl thiouracil (PTU) had limited erectile response and a marked decrease in T and thyroid hormones. Treatment with either levothyroxine or T could partially restore erectile responses, but combined hormonal treatment was able to fully restore erectile responses [19]. One study looked at the connection between erectile dysfunction, low libido, and thyroid dysfunction. The study discovered that erectile dysfunction and low libido are frequently linked to both hypothyroidism and hyperthyroidism in men [20].

The hormone whose levels significantly increased as a result of sleep deprivation was cortisol [16]. The pituitary and adrenal glands are involved in the stimulating and inhibitory loops that make up the HPA (Hypothalamic–Pituitary–Adrenal) axis. These loops control the production of glucocorticoids, including cortisol from the adrenal cortex [21]. It is noted that cortisol-secreting patterns during the

day are linked to short sleep duration, recurrent sleep disturbance, and symptoms of chronic insomnia [22]. In summary, brief sleepers' altered cortisol secretion may be a sign of HPA axis malfunction and negatively impact erectile performance. In fact, cortisol increases vascular responses to vasoconstrictors, which helps to maintain the tone of vascular smooth muscles (VSMCs) [16]. Serum cortisol levels have been found to be adversely connected with various domains of the International Index of Erectile Function-5 (IIEF-5) score in relation to sexual function, and males who have high serum cortisol levels may be more susceptible to ED [23]. The dysfunctions between the HPA axis and cortisol might be an important cog between sleep disorders and ED. Meanwhile, short sleep, stress and disordered HPA axis may coexist inevitably and have the complex interplay in sleep-related conditions [16].

Poor-quality sleep or sleep disorders lead to a decrease in dopamine levels. Research conducted over the years has demonstrated the important biological role of dopamine in regulating mood, the sleep-wake cycle, and circadian rhythms. [24]. This relationship creates a vicious cycle, so to speak, because a lack of restful sleep lowers dopamine levels, which in turn regulates sleep and the circadian rhythm. Innovative studies with fMRI (functional magnetic resonance imaging) have also demonstrated its particular role in the context of male sexual performance. Dopamine is specifically engaged in the consumatory phase of sexual activity in the dorsal caudate-putamen and the appetitive/anticipatory phase in the nucleus accumbens. This is in line with dopamine's broader roles in emotion and cognition in limbic regions and motor regulation in the striatum, respectively. Dopamine facilitates the manifestation of copulatory behavior in the medical pre-optic region. In the end, dopamine most likely plays a role in the paraventricular nucleus and possibly the lumbosacral spinal cord in initiating the erectile response [25].

Penile erection, energy metabolism, sleep, and brain activity are all connected by the neurotransmitter adenosine [26]. Numerous sleep variables, as well as the adenosine A1 receptor and adenosine A2, may have a similar route with adenosine signalling. According to reports, a receptor is involved in controlling sleep homeostasis by stimulating sleep-active neurones [27]. Furthermore, in vivo evidence has demonstrated the up-regulation of adenosine A1 receptors in cortical and subcortical regions following sleep deprivation, indicating that brief sleep may alter gene transcription linked to homeostatic sleep regulation and even other cellular functions. Like NO (nitric oxide II), it has been shown that adenosine, which has a short half-life and strong vasodilation, relaxes the cavernosal smooth muscles, which in turn promotes penile erection [28]. Adenosine receptors have been shown to perform a variety of biological functions. Among these, adenosine

receptor A1 is abundant on neurones and its downstream signalling seems to lower the release of NE and promote penile erection, whereas adenosine receptor A2B is necessary for adenosine signalling to induce penile erection [29].

Inflammation is thought to be a key link between sleep and the emergence of a number of illnesses [30]. In fact, there is ample evidence that elevated inflammatory markers are a result of inadequate sleep. Among them, greater CRP levels were apparently linked to shorter sleep durations [31]. The nuclear factor kappa B (NF-κB) inflammatory signalling and hormone response pathways may be responsible for the markedly elevated monocyte production of IL-6 and TNF-α in the morning following a night of sleep deprivation [32]. Furthermore, after a night of sleep deprivation, real-life models showed elevated levels of interferon γ (IFN-γ) with unaltered levels of TNF-α, interleukin-2 (IL-2), and interleukin-10 (IL-10) [33]. Meanwhile, it appears that certain cytokines mediate vasoconstriction by inhibiting the endothelial-dependent NO-cGMP pathway in animal models, thereby influencing cardiovascular risk [34]. Increased levels of inflammatory cytokines in the blood are linked to the occurrence and severity of ED, which is consistent with the mechanism underlying the development of cardiovascular diseases [35]. Studies on the molecular processes behind ED and chronic pelvic pain syndrome (CPPS) have shown similar results. Rat models of CPPS through experimental autoimmune prostatitis appear to have decreased eNOS expression in the corpus cavernosum, decreased erectile function, and increased levels of serum inflammatory chemicals, such as IL-6, CRP, and TNF-α [36]. Additionally, clinical data shows elevated IL-6 levels 24 hours following prostatectomy, and in animal models of cavernous nerve dissection, IL-6 suppression may reduce ED. Therefore, the association between ED and short sleep may be explained by the systemic inflammatory state [37].

4.3 NOS-NO-cGMP signaling pathway

The NOS-NO-cGMP signaling pathway is a key mechanism involved in the physiological process of penile erection. This pathway begins with NO synthesis, mediated by neuronal nitric oxide synthase (nNOS) and endothelial nitric oxide synthase (eNOS) [38]. Both synthases are activated, for example, by sexual stimuli, which are components of neurons and endothelial cells in the corpus cavernosum of the penis. Subsequently, NO activates guanylate cyclase (GC) in smooth muscle cells, which catalyzes the conversion of guanosine triphosphate (GTP) to cGMP. The generated cGMP acts primarily by activating cGMP-dependent protein kinases (PKG) and other effector proteins.

Phosphorylation of target proteins by PKG produces a number of downstream effects, including lowering intracellular calcium concentrations by inhibiting calcium influx through L-type calcium channels and promoting calcium sequestration. The decrease in calcium levels is necessary for the relaxation of smooth muscles, which facilitates increased blood flow to the tissues of the penis, contributing to an erection [39].

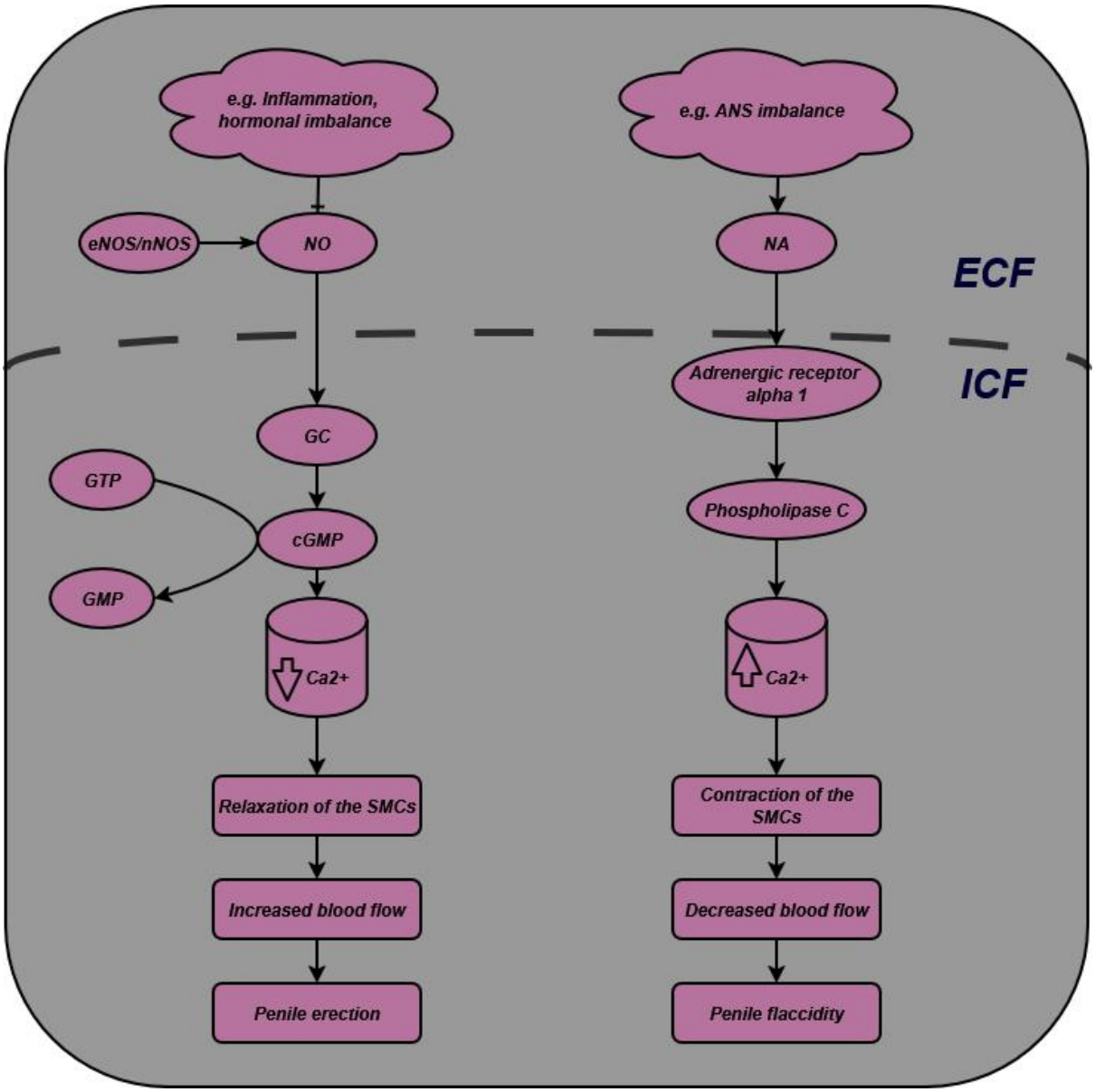
The signaling pathway described above can stall at any stage under unfavorable conditions. NO synthesis can be disrupted by inflammation, which increases the levels of proinflammatory cytokines (e.g., IL-6, IFN-γ, TNF-α), or by hormonal imbalances. In the mechanism of male erection, one of the hormones that significantly influences NO production by regulating eNOS is testosterone. Therefore, low testosterone levels result in reduced nitric oxide synthesis. Another mechanism causing erectile dysfunction involves the so-called stress axis. Cortisol, whose levels increase during sleep disorders, may be a trigger for such stress. This hormone stimulates the sympathetic nervous system and inhibits the parasympathetic nervous system. The ANS produces norepinephrine, which affects alpha adrenergic receptors, which increase the concentration of the enzyme phospholipase C. This enzyme, by releasing inositol triphosphate (IP3) molecules, in turn leads to the release of Ca2+ through calcium channels. Calcium causes smooth muscle contraction, impeding increased blood flow to the penis and maintaining its flaccid state [40] (Figure 1).

4.4 REM-related erections

In healthy men of all ages, from young children to the elderly, sleep-related erections (SREs) happen cyclically during the REM period [41]. Erectile dysfunction has long been assessed and categorised using polysomnography in sleep medicine [42]. The precise mechanism underpinning the genesis of SRE is still unknown, despite the well-studied mechanisms of REM sleep. However, erectile dysfunction brought on by sleep may be a precursor to several illnesses [43]. In this context, people with diabetes, renal illness, hypertension, hypogonadism, and certain sleep disorders have been found to have defective SREs. In the meantime, those with serious depressive illnesses, which are marked by changes in REM sleep, are said to have lower SREs [44]. Significantly, sleep fragmentation or sleep deprivation—an uncommon and poorly understood condition—may result from an aberrant extension of SREs, known as sleep-related painful erection (SRPE), which is characterised by penile pain during REM sleep. SREs and REM sleep may occur less frequently as a result of the brief sleep period. Reduced SREs may also result in insufficient arterial inflow and

metabolite buildup in erectile tissue, which causes the cavernosum to become hypoxic and acidotic, impairing erectile function [40].

Figure 1.



Molecular mechanisms of penile erection in corpus cavernosum smooth muscle cell

Source: authors' diagram

5. CONCLUSION

Erectile dysfunction is one of the most common sexual dysfunctions. Nowadays, more and more men—and younger men—are affected by this problem. The causes of this condition are varied. Studies show that one of the factors may be poor sleep, which is associated with a number of adverse consequences. One of these is hormonal imbalance. Low testosterone levels disrupt both the molecular mechanism underlying erection and affect the central nervous system, negatively impacting sexual responses. The functioning of the autonomic nervous system and excessive stimulation of the sympathetic nervous system, in turn, result from significantly elevated cortisol levels, indicating that the body is in a state of stress. Stress facilitates the development of inflammatory reactions and also increases susceptibility to chronic diseases. All of this, through a vicious cycle and mutual dependence, exacerbates sleep disorders and, consequently, erectile dysfunction.

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