

# THE VARIABILITY OF SLEEP HABITS AND DISORDERS IN ELDERLY POPULATION

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**Abstract.** The sleep habits have a very broad spectrum and varies through history with culture and age. Of course, there are a lot of myths about sleep some true but most of them false. Perhaps that the most common myth is that elderly need less sleep than adults. Once we age, we did not require less sleep, but the quality and architecture of sleep changes. The main changes in elderly are: decreasing the amplitude (phase advanced/delayed, internal desynchronization, increasing in variability); cellular/molecular changes (expression of the clock genes, neurobiochemical changes in the SCN and decreased ability of the SCN to drive peripheral oscillators); decreasing the light input (age related losses in photoreception like cataracts, decreased exposure or decreased response). From neurobiological point of view, The orexin levels increases in elderly, and higher value could be found in elderly with insomnia. This is important because the orexin level is correlated with Alzheimer’s disease markers and cognitive decline. Moreover, the growth hormone secretion decreases with age and this may have a serious impact on the person health. The presence of insomnia has a serious consequence on the elderly: increased risk for psychiatric disorders and the risk for cardio-metabolic diseases; it decreased quality of life; may produce cognitive; it increased pain and healthcare utilization and; it induces absenteeism and poor occupational; may increase the risk of falls and hip fractures; may increase the risk of motor vehicle crashes and workplaces accidents and overall increases mortality. In elderly, the presence of sleep disorders may have serious consequences, so they should address the specialist as soon as they notice sleep problems.

**Key words:** sleep disorders, insomnia, cardiovascular death, cognitive decline, elderly

**Rezumat.** Somnul și obiceiurile de a dormi ocupă un spectru foarte larg atât în cadrul istoriei, cât și în aceeași perioadă de timp variind de la cultură la cultură. Sunt foarte multe mituri legate de somn, dar multe sunt false. Probabil cel mai folosit este acela că vârstnici au nevoie de mai puțin somn. O dată cu înaintarea în vârstă se schimbă doar arhitectura somnului, calitatea lui și nivelul anumitor hormoni. Principalele modificări la vârstnici sunt: scăderea amplitudinii (și modificări de fază avansată/întârziată, desincronizare internă, creșterea variabilității); modificări celulare/moleculare (expresia genelor ceasului intern, modificări neurobiochimice în SCN și scăderea capacității SCN de a se sincroniza cu oscilatorii periferici); scăderea aportului de lumină (pierderi legate de vârstă în foto-recepție, cum ar fi cataracta, scăderea expunerii sau scăderea răspunsului). Din punct de vedere neurobiologic, nivelul de orexină crește la vârstnici, iar la vârstnicii cu insomnie valorile sunt cu mult mai mari. Acest lucru este important deoarece nivelul de orexină este corelat direct proporțional cu markerii bolii Alzheimer și cu declinul cognitiv. În plus, secreția hormonului de creștere scade odată cu vârsta și acest lucru poate avea un impact grav asupra sănătății persoanei. Prezența insomniei are consecințe grave asupra vârstnicilor: crește riscul de tulburări psihice și boli cardio-metabolice; scade calitatea vieții; poate produce deficite cognitive; crește sensibilitatea la durerea și utilizarea serviciilor medicale; induce absenteeism și prezenteism; crește riscul de căderi și fracturi de șold; poate crește riscul de accidente auto și de accidente la locul de muncă și în general, crește mortalitatea. La vârstnici, prezența tulburărilor de somn poate avea consecințe grave, așa că ar trebui să se adreseze specialistului imediat ce observă probleme de somn.

**Cuvinte cheie:** tulburări de somn, insomnie, mortalitate cardiovasculară, deficit cognitiv, vârstnici

## INTRODUCTION

The sleep has been fascinating humans from the beginning of humanity to nowadays. Throughout history, people have attempted to understand it from different perspectives, starting with esoteric terms all the way through to psychiatric ones. Given the high variety of

angles applied to the research of sleep, it becomes quite clear that one size does not fit all. The present paper aims to summaries some of these angles in the context of geriatric care. It thus consists of three sections: one is to offer a historical overview of this phenomenon, followed by an attempt to classification and finishing

with a section zooming into the phenomenon of insomnia.

### **Short overview of the mythology and history of sleep**

The ancients were attracted by sleep and dreams, so the Greeks attributed a god to this body function: Hypnos (with the Roman equivalent, Somnus). Hypnos was the son of Nyx (i.e., Night) and Erebus (i.e., Darkness) and he had a brother, Thanatos (i.e., Death). This association took place because in the ancient Greek culture the sleep usually took place in the night and in darkness, while people also noticed the resemblance between a deep sleep and death [1]. Morpheus is one of the sons of Somnus and sent human shapes (Greek morphai) of all kinds to the dreamer, while his brothers Phobetor (or Icelus) and Phantasus sent the forms of animals and inanimate things, respectively. Some similarities can be drawn to the Norse mythology, where the goddess Nótt is the night personified.

Sometime later, during the Middle Ages many superstitions and beliefs revolved around sleep disorders and nightmares explained that these were provoked by all kinds of gods and creatures like: Lamiae and Empusae in the Roman Empire, Lilith/Lilim in the Jewish culture, Old Hag in Anglo-Saxon culture, Bakhtak in Iran and Persia, Noctnisa in Eastern Europe, incubus or succubus in the old Europe and The Flyer in Romania and Bulgaria. Interestingly, the old Teutonic word mar (meaning devil) could be at the base of the German word nachtmar or the English word nightmare, because in that Christian medieval Europe the superstition was that the parasomnias were caused by the evil night visitors. Also, in French, the word for nightmare is cauchemar (caucher – to press + mar-devil), that shows its medieval superstition origin. Interestingly, during the Middle Ages, early scientists started developing the neuropsychiatric approach to sleep disorders. In his book “De Miraculis Occultis Naturae” (1564), the Dutch physician Lemnius Levinus (1505-

1568) linked the psychosomatic disease to somnambulism and his theory was supported by other scientists in their books of the epoch [1].

### **Attempt at a classification**

The ways in which people around the world sleep are as heterogeneous as the cultures they come from and it can be surprising how much can be learned from the sleep habits of the cultures around the world.

### ***The ‘where’ of sleep***

In Japan for example, people prefer sleeping on tatami mats. A tatami mat is commonly made of rice straw, wood chip boards or polystyrene foam. It is rectangular in shape, its width is always half the size of its length, and it comes in various sizes. These gentle-yet-firm mats were traditionally used as special seating for nobles. In order to sleep on one, a person usually places a thin mattress on top of it, which is called a futon.

In south India, mothers often put their sleeping babies in indoor hammocks made of the light, breathable fabric Sari and mount them so they hang from the ceiling in a bedroom. Also, in areas where malaria is a threat (such as the sub-Saharan African countries, like Ethiopia, Mali, Rwanda, Senegal, Tanzania, and Uganda) beds are often surrounded by nets. Malaria is a serious and sometimes life-threatening disease that is usually transmitted through the bite of an infected mosquito. Because it is a lot harder to fight off mosquitoes during sleep, bed nets, which are often made of strong multi-filament polyester fibers as an attempt to reduce the transmission of the disease by keeping mosquitoes at bay [2].

In South and Central America, for their “siesta” (or nap), people prefer a hammock, which is typically made of cloth, twine, or rope and dips with flexibility as one’s body presses into it. There is a practical reason for this choice: since hammocks swing above the ground, they can protect sleepers from ground-based ants, snakes and other critters that are common in tropical

countries. Navy ships also popularized the hammock, because when a sailor sways back and forth in one, he or she is less likely to be tossed to the ground by the lurching boat.

One interesting napping novelty can be observed in Scandinavia: dozens of strollers parked outside. Scandinavian parents often leave strollers with snoozing newborns outside while they finish up their errands inside. This can be, for many peoples from US, a nightmare waiting to happen. For Nordics, it is such a popular belief, that many daycares and preschools in these countries hold nap time outside to expose infants to fresh air more [2].

### ***The 'how' of sleep***

In some parts of Asia, getting a few minutes of nap during your lunch break is not only normal, but expected. During lunchtime employees will retire to a dedicated "nap room" to get a few minutes of relaxation. Others will simply lean back in their chair and nod off. Depending on the company and the culture, naps could last anywhere from a few minutes to a full hour. This curious napping habit stems from the fact that many locals living in these countries start their days much earlier than their Western counterparts, with some locals rising as early as 4 am [3].

Some aboriginal communities from Australia, follow age-old customs when it comes to sleep. Rather than sleeping in separate rooms or areas, people sleep in large groups that are designed to keep members of the community safe. Beds are arranged in long rows, with the strongest members of the community sleeping on the perimeter while the younger individuals and the elderly sleep toward the center of the group. This stems from a cultural belief that protection and togetherness is an important ingredient for a restful sleep. This custom can also be seen in some Arabic countries. Many Afghan families will fold up their mattresses and blankets after a night's sleep so that the room can play host to other activities, like meals and entertaining guests. It is also common for

entire families to snooze in one room together, rather than retiring to separate bedrooms to sleep.

In Botswana, Zaire and some other African countries, certain traditional hunter-gatherer groups do not follow a set sleep schedule. Instead, they sleep when they feel they need it. Some sleep experts suggest that sleeping in this way could fend off "sleep anxiety", and could even translate to better, more restful sleep [3].

According to a study from the National Sleep Foundation, the UK has the highest percentage of self-proclaimed nocturnal nudists than any other country surveyed. Almost a third of the UK population expresses a preference towards sleeping without any clothes at all. In Spain, children's sleep schedules are the same as the ones of their parents, staying awake until well past 10 pm. This is likely because of two cultural elements: siestas (long midday meals with naps), and much later dinners. Families in Spain often eat dinner as late as 9 pm, so heading off to bed an hour or two following a meal is not out of the ordinary [3].

### ***The biology of sleep***

- NREM and REM

Sleep is ubiquitous among mammals and may also be found among lower life forms. As discussed above, the accommodations made to allow and support sleep are different from one environment to another and differ from culture to culture. Sleep is not just the absence of wakefulness but it is rather a complex and multi-dimensional state. Sleep influences all major systems and this relationship is bidirectional, with many of the body's systems also influencing a person's sleep. Sleep problems, including sleep deprivation can have a great deal of influence on one's health, wellness, and longevity [4].

The need for sleep varies between individuals and across the life span. Normally, adults sleep about 7 to 9 hours per night although this can vary considerably; people spend one-quarter to one-third of their lives asleep [3]. The

classical sleep patterns consist of 4 to 5 cycles of paradoxical sleep (rapid eye movement sleep- REM) that are alternating with some deep sleep periods (non-REM sleep) that is further divided into four stages. The sleep-wake cycle is controlled by two separated but interacting processes: the homeostatic (or the recovery) process and the circadian process [5].

It is known that NREM sleep is characterized by some distinctive patterns on the electroencephalography (EEG): “sleep spindles” (12 to 14 Hz in humans) high amplitude and/or slow "delta" waves (circa 0.5 to 1.0 Hz in humans). The NREM sleep constitutes the major portion

of the sleep period (about 75 to 80 percent in humans), since the sleep process usually starts with NREM sleep (the main features of the NREM sleep from the literature [5] are presented in Tab. I) which is then interrupted periodically by episodes of REM sleep (at about 90 min. intervals in humans). The REM sleep is characterized by the occurrence of rapid eye movements, a low voltage-mixed frequency EEG pattern and muscle atonic state. Reports of dreaming can be elicited on awakenings from all stages but are the most vivid and most frequent on awakenings from REM sleep [5].

Tab. I The main cerebral, systemic and endocrine features of NREM sleep

Type of features		
Cerebral	Systemic	Endocrine
Cerebral temperature decreases	Reductions in motor activity, postural tonus, behavioral responsiveness, metabolic rate, heart rate, respiration rate, ventilatory response to CO <sub>2</sub> , vasomotor tone, arterial blood pressure, brain, and body temperatures, thermoregulatory setpoint, renal function, decreased intestinal motility.	Reductions in release of cortisol and thyrotropin
Decreased neuronal firing in some areas; increases in others		
Burst pause firing pattern of neurons in several major brain areas		
Drifting, unfocused thought; occasional dreams; occasional reports of no mental activity	General parasympathetic dominance	Increased secretion of growth hormone, aldosterone, testosterone, prolactin, insulin
Decreased activation of forebrain by reticular system		
Hyperpolarization of thalamocortical neurons		
"Sleep-active" neurons in anterior hypothalamus, basal forebrain, amygdala and nucleus of the solitary tract		Increased glucose levels
Reduced cerebral metabolism during slow wave sleep		
Cerebral blood flow varies regionally		

Interestingly the brain does not shut down during sleep. According to one comprehensive study of human sleep, cerebral metabolism is substantially reduced from waking levels only during the high-voltage, slow-wave portion of NREM sleep, which normally constitutes only about 20 percent of total sleep [4]. Therefore, the reductions in energy during sleep are relatively modest. The metabolic savings of sleep over quiet wakefulness

have been estimated at around 10 to 15%. Of course, the reductions of activity might be functionally targeted to some specific organ systems. However, the specific organs that require sleep to rest have not been identified.

Moreover, there are complex changes in neuronal firing which vary with the state and with the brain site instead of a massive decrease in neuronal firing. Neurons in thalamic and cortical areas tend to show

modest decreases in rate during NREM sleep followed by increases to the waking level or above during REM sleep. For many of these neurons, the most striking change is the development of a burst-pause firing pattern, which has been interpreted by some as functionally important. The hypothalamic and limbic neurons may show increases or decreases in firing rates in NREM or REM sleep, depending on the specific nuclei recorded by the studies. Brain stem neurons generally show decreased firing rates during NREM sleep, but in REM sleep they may show their highest firing rates in some areas, such as the mesencephalic or pontine reticular formation, or their lowest firing rates in other areas, such as the dorsal raphe or locus coeruleus nuclei [4, 6].

- The sleep/ wake cycle and the importance of their relationship

In the passage from waking to NREM sleep, there is increased firing in neurons of the anterior hypothalamus, nucleus of the solitary tract, and amygdala, which are believed to be involved in sleep generation or behavioral inhibition. Also, some complex changes in patterns of neural activity can occur without changes in average firing rates: for example, in the visual cortex, association cortex, and brain stem of the cat, neurons that fire rapidly during NREM sleep tend to fire even more rapidly during waking, while neurons which fire more slowly during NREM sleep tend to fire even more slowly during waking. Regarding these changes, there is no simple descriptive characteristic of brain activity that points to the sleep functions [4].

From the neurobiological perspective, both sleep and the arousal spectrum are important to be evaluated together because they tend to be a marker for mental health. The arousal spectrum is believed to be linked to 5 major neurotransmitters that form some circuits, in the ascending reticular activating system: histamine, serotonin, dopamine, norepinephrine, and acetylcholine [7]. There is another set of

circuits in the hypothalamus, called the sleep/wake switch. The wake center is in the tuberomammillary nucleus (TMN) and the sleep center is in the ventrolateral preoptic nucleus (VLPO) of the hypothalamus. Two sets of neurons are involved: orexin neurons of the lateral hypothalamus that promotes and stabilizes wakefulness (via two peptide neurotransmitters: orexin and hypocretin) and the melatonin sensitive neurons of the suprachiasmatic nucleus (SCN). The SCN is the body internal clock that regulates the circadian activity [7]. Melatonin is secreted by consecutive acetylation and methylation of serotonin in the pineal gland during the night. Its production is turned on by the noradrenergic (NA) neurons of the SNS in the upper spinal cord that passes into the gland through the superior sympathetic ganglions. The NA action on a beta-adrenoreceptor turn on the gene of the enzyme that it requires to produce melatonin. During daytime, melatonin production is suppressed by a SCN output. Also, some beta-blockers prevent melatonin production [8]. This is why for example, agomelatine (and MT1 and MT2<sub>2</sub> agonist and 5HT2C antagonist), may improve REM sleep behavior disorder symptoms [9].

The two main neurotransmitters that regulate sleep, from the hypothalamus are GABA (gamma-aminobutyric acid) from the Ventrolateral Preoptic Nucleus (VLPO) and histamine from the tuberomammillary nucleus (TMN). When the wake promoter from the TMN center becomes active and releases histamine to facilitate arousal and in the VLPO to inhibit the sleep promoter. After that, during the day, the homeostatic sleep drive increases and the circadian wake drive diminishes to an eventual tipping point where the sleep promoter from the VLPO is triggered, and GABA is released in the TMN to inhibit wakefulness [7]. So, for example, in treating a disorder with excessive daytime sleepiness, products like modafinil during the day to balance the circadian cycle back to

wakefulness can be recommended. Notably, the exact way that modafinil works is still unclear; some theories hypothesize that by blocking the dopamine transporter (DAT) from the dopamine neurons it increases the dopamine level that promotes the release of histamine from the TMN neurons. For disorders characterized by insomnia, which tend to be more frequent in the geriatric presentation, products that block histamine or that enhance GABA actions can be used. For the homeostatic sleep drive, it is presumed that the sleep is due to fatigue and linked with a neurotransmitter called adenosine which is accumulating during the day and diminishing during the night [7]. Caffeine is the most used antagonist of

adenosine, but there are also some endogenous antagonists.

### **SLEEPING DISORDERS**

Examining the sleep patterns of a patient is a basic part of the psychiatric evaluation. The sleep disorders are generally classified in ICD-10 (codes F 51.0, G 47, R and Z) and DSM IV/5, but more fully described in the ICSD (International Classification of the Sleeping Disorders) [10]. In terms of symptomatology, the sleeping disorders can be classified by insomnia (not enough sleep or impossibility to sleep), hypersomnia (excessive daytime sleepiness or drowsiness) and parasomnias (unusual events during sleep). A more detailed classification of sleep disorders can be found in Tab. II.

Tab. II Classification of sleep disorders with their corresponding ICD-10 codes

Superordinary type	Disorder name	Code
General sleep disorders	Insomnia (NOS)	G47.00
	Adjustment Insomnia	F51.02
	Other insomnia not due to a substance or known physiological condition	F51.09
	Primary insomnia	F51.01
	Paradoxical insomnia	F51.03
	Sleep Deprivation	Z72.820
	Insomnia Due to Medical Condition	G47.01
	Hypersomnia (NOS)	G47.10
	Hypersomnia Due to Medical Condition	G47.14
	Narcolepsy with Cataplexy	G47.411
	Narcolepsy Without Cataplexy	G47.419
	Recurrent Hypersomnia	G47.13
	Idiopathic Hypersomnia with Long Sleep Time	G47.11
	Idiopathic Hypersomnia Without Long Sleep Time	G47.12
	Primary Hypersomnia	F51.11
	Insufficient sleep syndrome	F51.12
Other hypersomnia not due to a substance or known physiological condition	F51.19	
Sleep related breathing disorders	Obstructive Sleep Apnea	G47.33
	Sleep Related Nonobstructive Alveolar Hypoventilation	G47.34
	Obesity Hypoventilation Syndrome	E66.2
	Sleep Related Hypoventilation/Hypoxemia	G47.36
	Primary Central Sleep Apnea	G47.31
	Cheyne Stokes Breathing Pattern	R06.3
	Central Sleep Apnea/Complex Sleep Apnea	G47.37
	Other Sleep Apnea	G47.39
	Dyspnea, unspecified	R06.00
	Other forms of dyspnea	R06.09
	Periodic breathing	R06.3
	Snoring	R06.83
	Other abnormalities of breathing	R06.89
	Apnea, not elsewhere specified	R06.81
Unspecified Sleep Apnea	G47.30	

	Circadian Rhythm Sleep Disorders (NOS)	G47.20
	Delayed Sleep Phase Type	G47.21
	Advanced Sleep Phase Type	G47.22
	Irregular Sleep-Wake Type	G47.23
	Free-Running Type	G47.24
	Shift Work Type (Shift Work Disorder)	G47.26
Parasomnias	Confusion Arousals	G47.51
	Sleepwalking	F51.3
	Night Terrors	F51.4
	REM Sleep Behavior Disorder	G47.52
	Recurrent Isolated Sleep Paralysis	G47.53
	Nightmare Disorder	F51.5
	Other sleep disorders not due to a substance or known physiological condition	F51.8
	Others parasomnia	G47.59
Sleep Related Movement Disorders	Restless Legs Syndrome RLS	G25.81
	Periodic Limb Movement Disorder	G47.61
Other	Opioid abuse with opioid-induced sleep disorder	F11.182
	Opioid dependence with opioid-induced sleep disorder	F11.282
	Opioid use, unspecified with opioid-induced sleep disorder	F11.982
	Sedative, hypnotic, or anxiolytic abuse with sedative, hypnotic or anxiolytic-induced sleep disorder	F13.182
	Sedative, hypnotic, or anxiolytic dependence with sedative, hypnotic or anxiolytic-induced sleep disorder	F13.282
	Sedative, hypnotic, or anxiolytic use, unspecified with sedative, hypnotic or anxiolytic-induced sleep disorder	F13.982
	Cocaine abuse with cocaine-induced sleep disorder	F14.182
	Cocaine dependence with cocaine-induced sleep disorder	F14.282
	Cocaine use, unspecified with cocaine-induced sleep disorder	F14.982
	Other stimulant abuse with stimulant-induced sleep disorder	F15.182
	Other stimulant dependence with stimulant-induced sleep disorder	F15.282
	Other stimulant use, unspecified with stimulant-induced sleep disorder	F15.982
	Other psychoactive substance abuse with psychoactive substance-induced sleep disorder	F19.182
	Other psychoactive substance dependence, in remission	F19.21
	Other psychoactive substance dependence with psychoactive substance-induced sleep disorder	F19.282
	Other psychoactive substance use, unspecified with psychoactive substance-induced sleep disorder	F19.982
	Opioid abuse with opioid-induced sleep disorder	F11.182
	Opioid dependence with opioid-induced sleep disorder	F11.282
	Opioid use, unspecified with opioid-induced sleep disorder	F11.982

When evaluating sleep problems, the whole 24 circadian rhythms (sleep journal), the presence of affective disorders and other psychiatric comorbidities (dementia, RLS, PMLS) or somatic comorbidities (asthma, obstructive sleep apnea syndrome, thyroidal afflictions, cardiac problems, etc.) or substance use and habits must be assessed. Additionally, some patient rated questionnaires like the Bristol Sleep Profile, or more research-oriented scales like the Pittsburg Sleep Quality Index, the

St. Mary's Sleep Questionnaire, the Leeds Sleep Evaluations Questionnaire or other similar scales can be administered. The most frequent medical investigations required in the cases of sleep problems are: TSH, glycemic value, vitamin D<sub>3</sub> and B<sub>12</sub> and the ions values, actigraphy (movement assessment), overnight video recording, multiple sleep latency test (helpful for diagnosing narcolepsy or excessive daytime sleepiness) and polysomnography.

Another important aspect of the disorders is characterized by circadian rhythm perturbation that can be perceived as phase delay of phase advance. The disorders with phase delay have the characteristic that the sleep/wake cycle is turned on too late in the 24 hours sequence. In contrast, the phase advance disorder is characterized by the fact that the sleep/wake cycle and the sleep promoter is turned on too early. A clear example is the case of adolescents and the case of the depressed patients who have a phase delay; therefore, their sleep/wake cycle turns on in the mornings when it is time to get out of bed. To reset their cycle, exposure to light in the morning and melatonin intake at night can be recommended. Contrary to this example, the elderly patient is usually phase advanced, therefore recommending them light in the evening and melatonin in the morning can reset their SCN and the sleep/wake cycle should stay off a little bit longer, retuning the patient to a normal rhythm [7].

The authors' perspective is that the psychiatric interview should ask questions about the onset of sleep, as well as about the sleep disturbances after sleep initiation, about early morning wakening, sleep quality, the sensation in the mornings, the frequency of the sleep disorder, about the impact of sleep on daytime activities and about the patient's quality of life. Nap-taking during the day and feeling sleepy may indicate either poor sleeping habits or hypersomnia. The sleep disorders in elderly are usually only the tip of the iceberg; further investigations are needed to seek for the presence of any affective

disorders or other comorbidities, including the presence of the periodic leg movement during sleep or the restless leg syndrome. The screening for narcolepsy, obstructive sleep apnea, Sunday night insomnia (i.e., anxiety related to work), Monday morning blues (i.e., tendency towards excessive alcohol consumption during the weekend), the use of caffeine, nicotine and any other drugs, as well as the working shifts and daily schedule should also be considered and assessed [7].

#### **ZOOMING IN ON SLEEP DISORDERS: INSOMNIA AND ITS IMPACT ON THE GERIATRIC POPULATION**

Insomnia is a common sleep disorder representing the patient's troubles in falling asleep, staying asleep, or both. As a result, one may get too little and/or poor-quality sleep. An overview of insomnia is presented in Tab. III [10]. As one's sleep changes with age, the quality of sleep is the aspect that suffers the most [11-14]. The main changes of the circadian rhythms with age are: decreases in amplitude (phase advanced/ delayed, internal desynchronization, increasing in variability) [11, 12]; cellular/molecular changes (expression of the clock genes, neurobiochemical changes in the SCN and decreased ability of the SCN to drive peripheral oscillators) [13]; decreases in the light input (age related losses in photoreception like cataracts, decreased exposure or decreased response) [14]. All these changes may disrupt the tonic orexin secretion [15]. This means that the sleep and wakefulness phases are out of synchronicity with the environment [16].

Tab. III Overview of insomnia

Insomnia descriptors	Description of characteristics
General characteristics	lying awake for a long time before you fall asleep
	sleeping for only short periods
	being awake for much of the night
	feeling as if you have not slept at all
	waking up too early
Precipitating factors	psychiatric (e.g., anxiety, depression)
	sleep-wake rhythm (e.g., work shifts, jet lag, irregular routine)
	pharmacological (e.g., stimulants, caffeine, alcohol, antidepressants, beta-blockers, etc.)



	physical (e.g., pain, cardiac/urinary/respiratory illnesses, pregnancy)
	other (e.g., stress/noise/bereavement/worry/arousal/children)

Importantly, insomnia can have serious impact on the patients' health. Such examples are: increased risk for psychiatric disorders; increased risk for cardio-metabolic diseases; decreased quality of life; cognitive decline; increased pain sensitivity; increased healthcare utilization and costs; absenteeism and poor occupational performances; falls and hip fractures; motor vehicle crashes and workplace accidents; increased mortality [17-20]. A possible explanation for this could be that those with primary chronic insomnia showed significantly higher midnight salivary cortisol concentrations (1.46 vs. 0.76 nmol/l,  $p=0.02$ ), indicating dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis [21]. Also some other studies showed that people with insomnia have a higher peripheric resistance to insulin, but it seems to work both ways, because a recent Japanese study confirmed that diabetes and anxiety are independently and significantly associated with insomnia [22].

Another neurobiological aspect that can explain the consequences of untreated insomnia could be that the orexin levels are elevated with age and much higher in people with insomnia [23]. This aspect is very important because orexin levels are correlated with Alzheimer disease biomarkers. Another study showed a direct correlation between the presence of the chronic insomnia and the cognitive decline [24].

Interestingly, the amount of slow wave sleep decreases with age and along with this phenomenon lower levels of growth hormone secretion are also registered [25]. The consequences of growth hormone (GH) secretion declining with the ageing process, may result in GH deficiency. For the older

adults, this could mean a decrease in lean body mass, an increase in body fat, especially in the visceral/abdominal compartment, adverse changes in lipoproteins, and a reduction in aerobic capacity. The increase in central obesity can further inhibit GH secretion.

A controversial and important aspect is linked with the common treatment of insomnia that uses benzodiazepines and the possible risk of fall within the geriatric population as a possible common side effect of hypnotic use. A study that included 34.163 older adults with insomnia (age above 65) that were followed up for 210 days answered this issue. The study concluded that insomnia alone, but not hypnotic use, is associated with a higher risk of fall [26]. So it is better for the patient to receive treatment, even with hypnotics, with the possible risk of sides' effects, rather than to let the insomnia untreated.

Moreover, a recent prospective study of 10.330 adult participants revealed that people with excessive daytime sleepiness had 2.85-times greater risk (95% CI, 1.33–6.09) of cardiovascular death than those without daytime sleepiness in multivariable analysis corrected for sociodemographic factors, comorbidities and cardiovascular risk factors including depression [27].

In conclusion, the sleep has a broad spectrum and it varies with culture and age. With age, people do not require less sleep, since it is the quality of sleep that changes. The presence of sleep disorders may have serious consequences especially for the geriatric population, therefore they ought to address a specialist as soon as they notice any sleep irregularities.

### ***Conflicts of interest***

The authors declare no conflicts of interest.

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