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Review Article

Natural Herbs for Insomnia: A Promising Approach in Obesity Management

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Abstract

Sleep, a fundamental regulator of neuroendocrine function and glucose metabolism, has been associated with metabolic and endocrine alterations. Sleep deprivation leads to decreased glucose tolerance, insulin sensitivity, and elevated cortisol levels in the evenings, increased ghrelin, decreased leptin, and heightened hunger and appetite. Recent epidemiological and laboratory evidence supports previous findings, suggesting a connection between sleep loss and an increased risk of obesity. Obesity, resulting from a calorie imbalance, significantly impacts quality of life and elevates the risk of various diseases. Insufficient sleep and circadian misalignment contribute to obesity by disrupting appetite hormones and energy expenditure. Millions worldwide face challenges in obtaining sufficient sleep due to modern lifestyles and technological advancements, affecting metabolic functions and body weight regulation. Sleep is essential in regulating physiological functions and influencing behavior, development, and overall health. Sleep deprivation and disorders are linked to obesity and may hinder weight loss efforts. Studies demonstrate that sleep restriction disrupts energy balance. Hence, use of natural remedies for treating insomnia might be an effective therapy in controlling insomnia associated overweight and obesity.

Keywords: *Orexin System; Insomnia; Obesity; Ghrelin; Leptin; Natural Herbs*

Introduction

Obesity, a chronic illness, arises from an imbalance between the body's energy expenditure and calorie intake. According to the World Health Organization (WHO), a person's body mass index (BMI) of 30 or higher indicates obesity. Between 1990 and 2022, the global prevalence of obesity is doubled, with a significant increase in children and adolescents aged 5 to 19 from 7% to 16% and adults aged 18 and over from 2% to 8% [1]. Several factors contribute to the development of obesity, including lifestyle choices such as food preferences, nutritional status, and sedentary behaviors. These choices are influenced by a complex interplay of social, psychological, behavioral, economic, and environmental factors. Additionally, family genetics and epigenetic modifications, as well as interactions between the gastrointestinal (GI) tract and central nervous system (CNS), play crucial roles. Furthermore, alterations in the gut microbiome, which regulates appetite, contribute to obesity. Beyond its negative impact on quality of life, obesity significantly increases the risk of cardiovascular disease and type 2 diabetes mellitus, which can shorten life expectancy [2].

Sleep is a means of recovery which is crucial to maintain the equilibrium of one's physical, mental, and emotional well-being [3]. It's an important modulator of neuroendocrine function and glucose metabolism [4]. Lack of sleep or insufficient sleep is termed as insomnia; it is the most prevalent sleep disorder and the second most prevalent neuropsychiatric disorder [5]. Symptoms include difficulty falling asleep, waking up throughout the night or sooner than desired, and difficulty falling back asleep [6]. The metabolic and endocrine changes

brought on by insomnia include impaired insulin sensitivity, lower glucose tolerance, elevated evening cortisol concentrations, elevated ghrelin, decreased leptin, and increased appetite and hunger according to epidemiological and laboratory investigations [7]. Short sleep duration or poor sleep quality i.e., Insomnia is emerging as a new risk factor for obesity [8].

Natural herbs have been used for centuries in traditional medicine to treat insomnia [9]. These herbs work by influencing the nervous system, promoting relaxation, and improving sleep quality. These are generally safe, effective and cheap when compared to modern allopathy medicines with minimal side effects [10].

The current review gives an insight about how obesity is associated with insomnia, which includes the prevalence, risk factors, molecular mechanism of obesity and insomnia and finally utilization of natural herbs to alleviate insomnia as a part of obesity management.

Epidemiology

Current world statistical studies revealed that there are over 1 billion obese people worldwide, including around 880 million adults and 159 million children and adolescents between the ages of 5 and 19 [11]. According to the World Obesity Federation's study statistics; around 3 billion individuals suffer from being overweight or obese. Between 1975 and 2022, adult obesity rates almost tripled in women (6.6% to 18.5%) and quadrupled in men (3% to 14.0%) [12].

In India, incidence of obesity raised up from 1.2% in 1990 to 9.8% in 2022 for women and 0.5% to 5.4% in 2022 for men [13]. According to a study undertaken by the NCD Risk Factor Collaboration with World Health Organization, the prevalence of obesity in India is 182nd place for women and the 180th place for men in 2022 in the world. Obesity incidence rates were higher among people aged 45 - 54 than aged 18-24 [14].

Insomnia is commonly a chronic disorder, with a 40% persistence rate over a 5-year period. Approximately 10% of the adult population has an insomnia condition, while another 20% experiences occasional insomnia symptoms [15]. Insomnia affects more women, older persons, and people with socioeconomic issues as mentioned in figure 1 [16]. Growing data from laboratory and epidemiological investigations suggests that short sleep duration and poor sleep quality are one of the reasons for obesity development. A meta-analysis of 18 research studies revealed that individuals who slept fewer than 5 hours each night had a 1.55-fold increased likelihood of obesity [17].

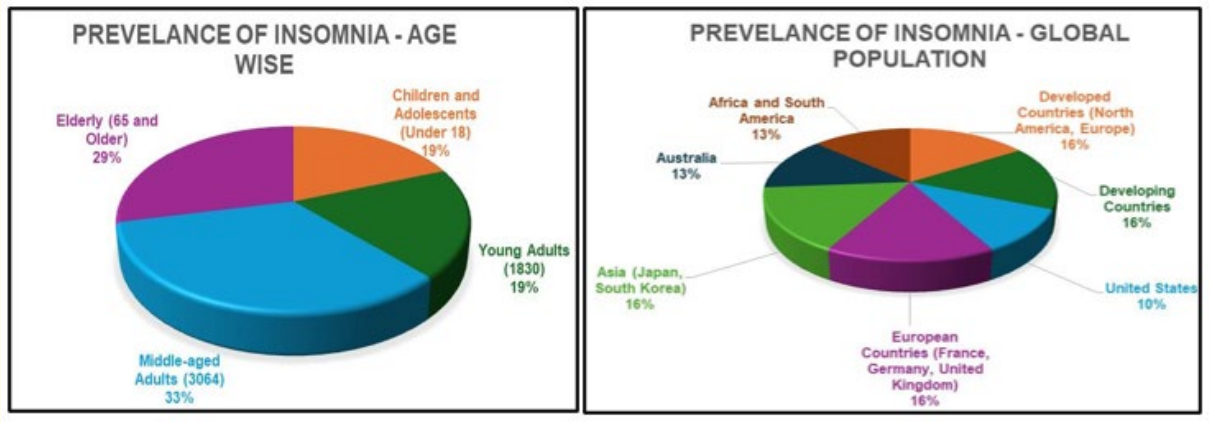

Figure 1: *Epidemiology of insomnia.*

Risk factors for insomnia

Age

Insomnia can occur at any age, although your chances of developing rise with age [18].

Genetic profile

Genes may increase the risk of insomnia, as insomnia can be inherited [19].

Profession and surroundings

Profession and surroundings can disrupt the sleep-wake cycle such as late-night job, exposing to screen for long period, extremely high or low temperatures, traveling frequently to different time zones [20].

Gender and stress

Frequent alteration in the daily routine, including change in the bed time, sleep disruptions due to infant care, taking extended naps throughout the day, getting too little exercise throughout the day, abusing alcohol, nicotine, or caffeine. Additionally, using electronics or watching TV right before bed has an increased risk of developing insomnia. Lately, stress or concern about your job, relationships, finances, or the passing of a loved one can also increase the chances of getting insomnia [21]. Women are more likely prone to insomnia than males due to alteration in hormones that occur during pregnancy and menopause [22].

Risk factors for obesity

Lack of physical activity, coupled with excessive screen time, is associated with a high body mass index (BMI). Excessive added sugar intake, insufficient sleep (Poor sleep quality), high saturated fat intake and unhealthy eating habits increase the risk of being overweight and obese. Obesity leads to occurrence of cardiovascular disorders, such as heart disease and stroke, are the major causes of death globally. Being overweight can also cause diabetes and its complications, like blindness, limb amputations, and the need for dialysis [23].

Molecular mechanisms in obesity

Central and peripheral mechanism plays an important role in appetite regulation. It is very important to gain a deeper understanding of the mechanisms underlying the development of obesity i.e., hunger, satiation, satiety, homeostatic and non-homeostatic food intake which fuel the eating and fasting mechanism [24]. The amount of a meal and the time it is finished are determined by satiation, linked with sensations like fullness or nausea. Satiety starts once satiation is reached and hunger is suppressed throughout this time. As satiety fades, feeling of hunger progresses, which increases desire to have food [25].

Food intake may be homeostatic (synchronizes food consumption and energy expenditure to control energy balance and motivation to eat increases when energy stores depleted) or non-homeostatic (eating too much or too little in comparison to what the body needs also called as hedonic eating) [26].

Central mechanism in appetite regulation

At the central level, the brain stem and hypothalamus are key components of appetite regulation process. Signals are sent to brain stem via vagus nerve in response to peripheral stimulation of mechano and chemoreceptors via afferent conduction. They are further sent to the amygdala, stria terminalis, and hypothalamic regions (dorsomedial, paraventricular, and arcuate nuclei) [27]. Two distinct types of neurons, Neuropeptide Y (NPY) and agouti-related peptide (AgRP) work against one another inside the arcuate nucleus, which have an orexigenic (or appetite-stimulating) impact. Other neurons such as, Proopiomelanocortin (POMC) and cocaine-and amphetamine-regulated transcript (CART) peptide suppresses appetite (anorexigenic action) [28].

GABA blocks the action of POMC/CART, while β-endorphin blocks the action of AgRP/NPY. Moreover, each type of hypothalamic signals are initiated at paraventricular nucleus level by interacting with melanocortin 3 and 4 receptors (MC3R and MC4R) and alterations in leptin levels. Furthermore, it leads to the production of anorexigenic hormones such as corticotropin-releasing hormone (CRH), thyrotropin-releasing hormone (TRH), oxytocin, and brain-derived neurotrophic factor (BDNF) [29].

The hormones produced in the stomach, such as ghrelin (hormone that stimulates hunger by AgRP/NPY neuron activation). Ghrelin's messenger ribonucleic acid is primarily found in stomach tissue and works with the hypothalamus to maintain energy homeostasis [30]. A sequence of post-transcriptional enzymes activates ghrelin and converts it from preproghrelin to proghrelin. There are two types of ghrelin in the bloodstream, acylated ghrelin and non-acylated ghrelin. The non-acylated form of ghrelin is much more prevalent than acetylated ghrelin. Ghrelin's main receptor is G-protein coupled growth hormone secretagogue receptor type 1a (GHS-R1a). GHS-R1a is seen everywhere in the body, even in hypothalamus and helps in integration and maintenance of energy homeostasis. Additionally, it also maintains some anorexigenic enter hormones like peptide YY (PYY), cholecystokinin (CCK), or glucagon-like peptide 1 (GLP-1), as they have an impact on multiple brain regions in addition to their peripheral effects [31].

Leptin mostly present in white adipose tissues and produced by the obese gene on chromosome 7x, release of leptin into bloodstream commands the brain to maintain homeostasis. The major receptor for leptin is LepR, which has several subtypes that functions at the level of the arcuate nucleus in hypothalamus, and activates neurons that express POMC/CART by preventing AgRP/NPY from forming. It also suppresses hunger and increases energy expenditure by activating the BDNF cascade and inhibiting the lateral hypothalamus and mesolimbic dopaminergic neurons [32]. Like leptin, insulin also reduces appetite, which is linked to an increase in POMC in the arcuate nucleus and a decrease in NPY secretion. When free fatty acids are abundant, intermediate metabolites of intracellular fatty acid metabolism, such as long-chain fatty acyl-CoA (LCFA-CoA) molecules, serve as a signal of satiety. In hypothalamus, elevated LCFA-CoA has anorexigenic effects, causing weight loss and NPY gene expression suppression.

Additionally, nutrients may indirectly control vagus nerve function by affecting the production of gut peptides and neurotransmitters from enteroendocrine cells [33]. For example, glucose may stimulate serotonin release, increase receptor exposure and initiate satiety signalling. Endogenous mediators such as cytokines like interleukin-6 (IL-6) or tumor necrosis factor-α (TNF-α) may have an impact on the suppression of food intake [34]. Some of the conditions that are caused by obesity include:

- **Dysregulation of hormones:**
  - Obesity makes the brain become less sensitive to leptin, which raises hunger and lowers feelings of fullness [35].
  - Obesity makes pancreatic cells become less sensitive to insulin, which raises blood sugar and elevates fat accumulation [36].
- **Inflammation and its mediators:**
  - Chronic low-grade inflammation in adipose tissue and other organs is linked to obesity. Resistance to insulin and other metabolic disorders can worsen through this inflammation, which can also interfere with metabolic processes [37].
  - Adipose tissue in obesity releases inflammatory cytokines such as TNF-α and IL-6, which cause insulin resistance and other metabolic diseases [38].
- **Dysregulation of neuroendocrine system:**
  - Obesity affects the hypothalamus in brain which controls hunger and energy expenditure. This may result in consuming more food while utilizing less energy [39].

- Dopamine and serotonin influence appetite and reward-seeking behavior. Overeating and weight gain may be caused by these neurotransmitters' dysregulation [40].
- **Gut microbes:** Alteration in the gut microbiota's constitution can impact inflammation and energy metabolism, and result to obesity [41].

Reward system dysregulation

The desire for pleasure promotes unhealthy consumption of food. Long-term exposure to these stimuli ultimately leads to down-regulation, as well as a drop in reward system sensitivity. Thus, to have the same rewarding impact, fat people need to eat more of a particular item, which feeds the cycle of energy overload [42].

Another factor contributing to obesity is having cravings for food types, especially those high in fat, sugar, and salt, is another factor that contributes to weight gain. Insulin and leptin resistance, which are common in obese people, also contribute to reward system dysregulation. Likewise, the relationship between the reward system and the parts of the brain involved in vision and decision-making makes it harder for fat people to regulate their impulses. Because of this impairment, they are unable to resist eating unhealthy foods regardless of whether they are aware of its negative effects [43].

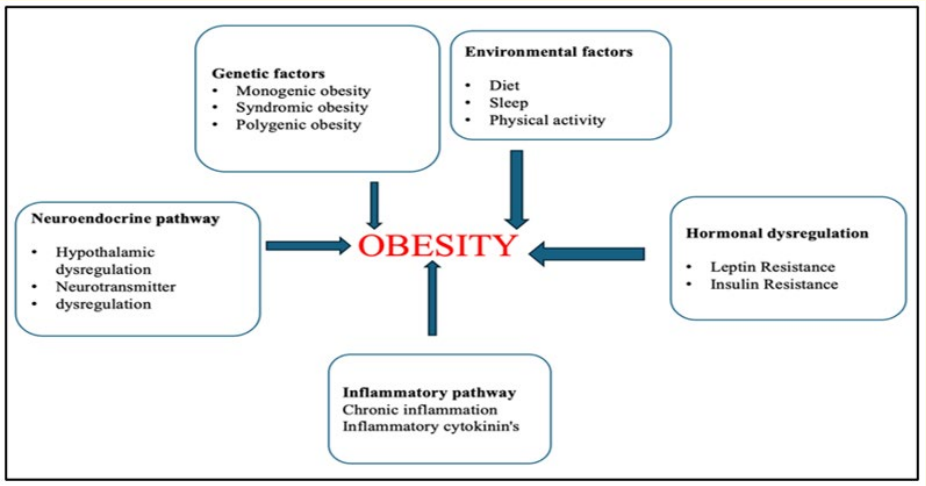

Figure 2: *Mechanistical insights of obesity.*

Molecular mechanism in insomnia

Sleep is regulated by two internal mechanisms that operate largely independently which include the sleep homeostatic mechanism and the circadian mechanism. The sleep homeostatic drive is primarily regulated by the adenosine and melatonin, which accumulate throughout the waking day. The 24-hour rhythms of the sleep/wake cycle are synchronized to the external environment, primarily influenced by light. The suprachiasmatic nucleus (SCN) serves as the primary circadian pacemaker of the body, controlling the circadian oscillators through neural and endocrine mechanisms [44]. A decrease in SCN neuron firing rate due to reduced light exposure leads to an increase in sympathetic action potential, more norepinephrine (NE) release on the pineal gland, and an increase in melatonin production. Melatonin, in turn, decreases the activity of SCN neurons, thereby lowering the circadian drive for arousal in a positive

feedback loop. Lesions on the hypothalamus, specifically affecting and restricting the SCN, can disrupt the sleep-wake cycle. However, the synchronization of the circadian clock is not a strictly hierarchical SCN-driven system. Peripheral clocks may be regulated independently of the SCN, influenced by light and other external factors [45].

Insomnia's etiology encompasses a complex interplay of genetic, environmental, physiological, and behavioral factors [46]. Recent studies have stated that insomnia is not solely caused by sleep deprivation; instead, they propose that insomnia is a state of 24-hour hyper-arousal. This hyper arousal manifests in various physiological changes, including alterations in EEG recordings, increased sympathetic activity, higher ATP utilization in gray matter, and heightened activation of the HPA axis. Conversely, there's reduced prefrontal cortex metabolism during wakefulness. These changes align with an alternating daytime cytokine secretion pattern, with hyper-secretion observed in primary insomnia patients [47]. Furthermore, alterations in the endocrine and immune systems support this hypothesis. Long-term sympathetic hyperactivity can lead to elevated plasma insulin, decreased high-density lipoproteins, increased triglycerides, total cholesterol, plasma angiotensin, hematocrit, and an increased risk of cardiac arrhythmias and hypertension. Chronic HPA axis activation can also cause depression, anxiety, hypertension, visceral obesity, and other pathologies [48].

Neurohormonal system

Orexin-A and Orexin-B, hypothalamic neuropeptides, play a crucial role in regulating wakefulness. Whereas, orexinergic neurons, which innervate brain nuclei, stabilize wakefulness. The dorso-medial nucleus of the hypothalamus provides essential circadian rhythm information to influence orexin neurons. During wakefulness, these neurons are active and they cease during sleep [49]. Inappropriately activated orexin neurons at night can lead to insomnia, characterized by signs of hyperarousal. Conversely, orexin deficiency causes narcolepsy. During wakefulness, the lateral hypothalamus (LH) is highly active. Other neuronal populations including noradrenergic, histaminergic, serotonergic, cholinergic, and tegmental nuclei, also contribute to enhancing wakefulness [50].

Orexin-A and Orexin-B suppress REM sleep through protein-coupled receptors (OX1R and OX2R), extending waking stretch. Orexin-A exhibits a similar affinity to OX1R and OX2R, while Orexin-B has a tenfold preference for OX2R [51]. During NREM and REM sleep, orexin neurons are inhibited by GABAergic neurons that are acting on GABA-A and GABA-B receptors. GABA-A receptor antagonism increases orexin neuron activity during NREM sleep. Orexin antagonists block OX2R or both Orexin-1 and Orexin-2 receptors, promoting sleep. Notably, orexin receptor antagonists enhance sleep onset and maintenance without significant tolerability issues or withdrawal effects in patients with chronic insomnia [52].

Melatonin

Melatonin regulates various physiological processes, including immunity, hormone secretion, reproductive rhythms, and sleep. Melatonin synthesis gradually declines with aging [53]. Melatonin enhances sleep quality by reducing sleep onset latency, fragmentation, and increasing efficiency. It may also extend total sleep duration. Melatonin secretion peaks during sleep onset but decreases with age, leading to sleep decline. Exogenous melatonin enhances sleep duration, efficiency, and quality in patients with primary insomnia and also increases endogenous melatonin levels during the evening and night, enhancing daytime function and alertness. Melatonin interacts with MT1 and MT2 receptors, which regulate circadian oscillations. These receptors are expressed by the suprachiasmatic nucleus (SCN) and bind to melatonin, suppressing neuronal firing and promoting sleep.

Melatonin synthesis ceases after eight hours of physiological desensitization. Ramelteon, a synthetic melatonin analogue, is FDA-approved for insomnia characterized by sleep onset difficulty. It exhibits a stronger affinity for MT1 receptors than melatonin and is highly selective for these receptors. Ramelteon is a chrono-biotic and hypnotic agent that promotes sleep initiation and maintenance. Agomelatine, a acetamide naphthalene analogue of melatonin, acts as a potent agonist for MT1 and MT2 receptors, regulating circadian rhythms. It also binds to the 5-HT2C receptor [54].

Molecular mechanism involved in insomnia induced obesity

The association between food and sleep can be regulated by several pathways. Food intake may be impacted by changes in hormones that regulate hunger and an increase in orexin neuron activity [55]. Several studies have found that sleep deprivation causes a rise in the hunger-promoting hormone ghrelin and a fall in the satiety-promoting hormone leptin. The important mechanisms are discussed below.

Orexin system

Two orphan G-protein-coupled receptors in the lateral hypothalamus zone are identified as endogenous ligands for orexins, also referred to as hypocretins. Even though they are significant modulators of the sleep/wakefulness cycle, they were first identified as regulators of feeding behavior [56]. To maintain a prolonged, consistent awake period, orexins stimulate orexin neurons situated in the hypothalamus and brainstem regions. Orexin neurons play a vital role in arousal, reward systems, energy balance, and emotion coordination. Ghrelin, leptin, and glucose levels are all mediated by orexin neurons, implies a connection between arousal states and energy homeostasis [57]. Orexins are also involved in reward systems [58].

In addition to controlling the hypothalamic arcuate nucleus homeostatic feeding center, orexigenic neurons also influence hedonic feeding, which is mediated via reward centers (nucleus accumbens and ventro-tegmental region) [59]. Peripheral hormones like leptin and ghrelin interact directly with the arcuate nucleus via central nervous system modifying the function of the orexin system to reduce and enhance food intake, respectively. The orexin system is inhibited by sleep-promoting neurons in the ventrolateral preoptic area (VLPO) containing gamma-aminobutyric acid (GABA) [60].

The orexigenic neurons, located in the lateral hypothalamic area (LHA) and posterior hypothalamus (PH), play a major role in the maintenance of arousal. Orexin activity affects homeostatic feeding via raising the activity of neuropeptide Y (NPY) neurons in the hypothalamus arcuate nucleus, which in turn affects food intake. Further by stimulating the dopaminergic ventrotegmental area (VTA) and nucleus accumbent (NA, the reward centers regulates non homeostatic food intake. Increasing sympathetic activity in turn inhibits leptin release and stimulates ghrelin release. Lower leptin and higher ghrelin levels will act simultaneously to further activate orexin neurons resulting in an increased drive for both homeostatic and non-homeostatic food intake [61].

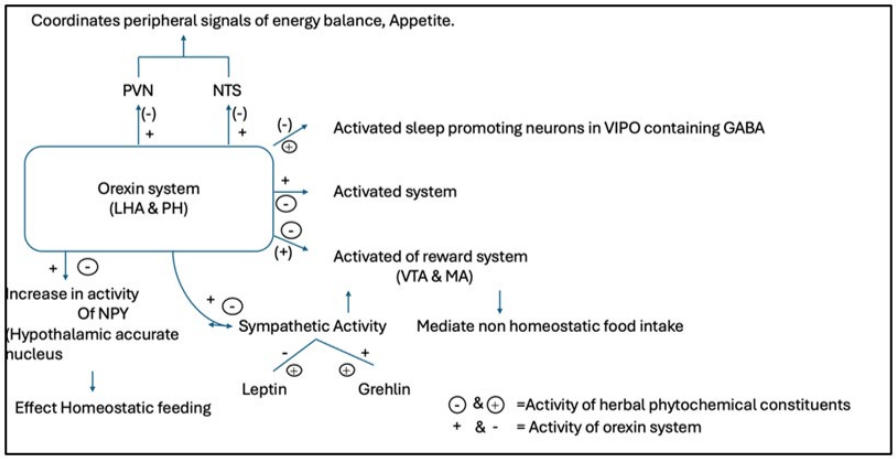

Figure 3: *Mechanistical relationship of orexin system with eating and sleeping.*

Biochemical and physiological pathways

Lack of sleep can affect the physiological pathways by diminished levels of physical activity and hormones linked to energy expenditure, which minimize energy expenditure. In addition, lack of sleep and excess alcohol consumption could also encourage people to choose foods that are high in calories lacking in nutrients [62].

Lack of sleep can affect the biochemical or metabolic pathways by altering hormone balances that controls metabolism and appetite. Ghrelin may rise while leptin may fall which causes the orexin system to activate and in turn leads to stimulation of NYP neurons at arcuate nucleus of hypothalamus responsible for food intake. Additionally, lack of sleep can also make difficult for the body to respond to insulin, which raises the blood glucose level and causes insulin resistance. Weight gain and raise chances of type 2 diabetes [63]. Furthermore, the cycle of sleep and wakefulness has a significant impact on glucose control. The whole-brain metabolism decreases during sleep compared to being awake, which leads to a decreased utilization of glucose. Since insulin does not mediate brain glucose, a decrease in brain glucose utilization leads to diminished glucose efficiency, causing impaired glucose tolerance and promoting insulin resistance [64].

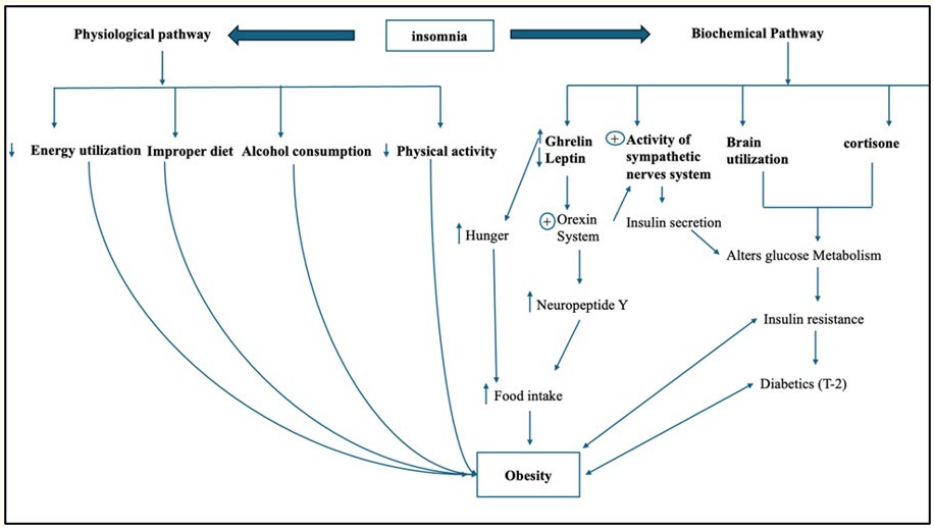

Figure 4: *Overview of biochemical and physiological pathways relating insomnia and obesity.*

Natural herbs in alleviating insomnia

Table 1 lists the medicinal plants along with information on them based on the most recent research on medicinal plants and how they can be used to cure insomnia in both preclinical and clinical studies. Hence, it might be suggested that consumption of these medicinal herbs can ameliorate obesity associated with insomnia.

Botanical Name	Common Name	Family	Active Constituents	Neurochemical Pathways	Reference
Achillea millefolium	Yarrow	Asteraceae	Flavonoids, sesquiterpene lactones, Di caffeoylquinic acids	Unknown	[65]
Aloysia polystachya	Aloysia	Rubiaceae	Thujone, carvone	GABA	[66]
Albies pindrow	Western Himalayan fir	Pinaceae	Terpenoids, flavonoids, glycosides	Unknown	[67]

Albizia julibrissin	Mimosa	Fabaceae	Flavonoids, triterpenoid saponins	Serotonin 5-HT1A	[68]
Annona spp.	Annona	Annonaceae	Palmitone, annomontine, β-sitosterol	GABA,5-HT, DA	[69]
Apocynum venetum	Luobuma	Apocynaceae	Flavonoids	GABA, 5-HT	[70]
Casimiroa edulis	White sapote	Rutaceae	Histaminergic compounds	Unknown	[71]
Coriandrum sativum	Coriander	Apiaceae	Volatile oils, flavonoids	Unknown	[72]
Crocus sativus	Saffron	Iridaceae	Safranal, crocins, picrocrocin	5-HT, NE, DA, GLU, GABA	[73]
Equisetum arvense	Horsetail	Equisetaceae	Silica, flavonoids	Unknown	[74]
Erythrina spp.	Velutina, mulungu	Fabaceae	Erythravine alkaloids	GABA	[75]
Eschscholzia californica	Californian poppy	Papaveraceae	Benzophenanthridine alkaloids	GABA	[76]
Eurycoma longifolia	Tongkat ali	Simaroubaceae	Alkaloids	Unknown	[77]
Euphorbia hirta	Asthma weed	Euphorbiaceae	Alkaloids, phenolics	GABA	[78]
Gastrodia elata	Gastrodia	Orchidaceae	Phenolics (4-hydroxybenzaldehyde)	GABA, 5-HT	[79]
Juncus effusus	Soft rush	Juncaceae	Polyphenols, phenanthrenes, dehydroeffusol	GABA	[80]
Leea indica	Bandicoot berry	Leeaceae	Triterpenoid glycosides, hydrocarbons, ursolic acid	Unknown	[81]
Magnolia spp.	Magnolia (Saiboku-to, Hange-ko-boku)	Magnoliaceae	Honokiol, magnolol, obovatol	GABA	[82]
Nauclea latifolia	African peach	Rubiaceae	Isoquinoline alkaloids	Unknown	[83]
Panax ginseng	Korean ginseng	Araliaceae	Triterpenoid saponins (ginsenosides)	Monoamines, HPA-axis, BDNF	[84]
Rubus brasiliensis	Brazilian raspberry	Rosaceae	Tannins, flavonoids	GABA	[85]
Stachys lavandulifolia	Wood betony	Lamiaceae	Flavonoids, terpenoids, essential oils	Unknown	[86]
Tilia spp.	Lime blossom	Malvaceae	Tiliroside, quercetin, kaempferol glycosides	Unknown	[87]
Uncaria rhynchophylla	Chinese cat's claw	Rubiaceae	Rhynchophylline alkaloid	5-HT	[88]
Zizyphus jujuba	Sour date	Rhamnaceae	Jujubosides, spinosin	GABA, DA, 5-HT, GLU	[89]

Table 1: *List of natural herbs in alleviating insomnia.*

Summary and Conclusion

Obesity and central obesity are linked to both short and extended sleep durations, as well as symptoms of insomnia. Today, many young people seeking insomnia treatment are overweight or obese. However, because their insomnia is less severe, young people who are overweight or obese may exhibit more symptoms of insomnia and sleep issues, but they also react similarly to behavioral insomnia treatment. Future research should examine the impact of family, cultural, and societal determinants on weight and sleep health, long-term outcomes related to insomnia remission or symptom return, and changes in both weight status and insomnia during treatment. People suffering from insomnia may benefit from herbal medicines, although there is currently insufficient evidence to support that claim. This field needs more comprehensive, superior random clinical studies. The long-term safety and effectiveness of herbal insomnia therapies, as well as the effects of various administration, extraction, and preparation parameters on patient outcomes, must all be thoroughly investigated. Furthermore, because there is a link between insomnia and obesity, these herbal medicines can be utilized to treat obese persons who are suffering from insomnia.

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Conflicts of Interest

The authors declare no conflict of interest regarding this article.

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Volume 13 Issue 3 March 2025