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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

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THE EFFECT OF CHRONIC INSOMNIA ON THE STRUCTURAL AND ENDOCRINE FUNCTIONAL STATE OF THE PANCREAS

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✓ ***Resume***

Chronic insomnia is a factor that causes deep functional changes not only in the body's central nervous system but also in the metabolic, neuroendocrine, immune, and oxidative-antioxidant systems. The main role in ensuring glucose homeostasis is played by the pancreas, whose endocrine part, especially the Langerhans islets and β-cells, is closely linked to sleep. This article analyzes modern scientific data regarding the morphological changes that may occur in the pancreas during chronic insomnia. Literature analysis shows that lack of sleep is associated with circadian rhythm disorders, insulin resistance, impaired glucose metabolism, oxidative stress, and inflammation.

Keywords: chronic insomnia, circadian rhythm, oxidative stress, pancreas, Langerhans islets, β-cells, insulin resistance.

ВЛИЯНИЕ ХРОНИЧЕСКОЙ ДЕПРИВАЦИИ СНА НА СТРУКТУРНОЕ И ЭНДОКРИННОЕ ФУНКЦИОНАЛЬНОЕ СОСТОЯНИЕ ПОДЖЕЛУДОЧНОЙ ЖЕЛЕЗЫ

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✓ ***Резюме***

Хроническая бессонница является фактором, вызывающим глубокие функциональные изменения не только в центральной нервной системе организма, но и в метаболической, нейроэндокринной, иммунной и окислительно-антиоксидантной системах. Панкреатическая железа играет центральную роль в обеспечении гомеостаза глюкозы, ее эндокринная часть, особенно островки Лангерганса и β-клетки, тесно связаны со сном. В данной статье проанализированы современные научные данные о морфологических изменениях, которые могут возникнуть в поджелудочной железе при хронической бессоннице. Анализ литературы показывает, что недостаток сна связан с нарушением суточного ритма, инсулино-резистентностью, нарушением обмена глюкозы, окислительным стрессом и воспалением.

Ключевые слова: хроническая бессонница, суточный ритм, окислительный стресс, поджелудочная железа, островки Лангерганса, β-клетки, инсулино-резистентность.

SURUNKALI UYQU DEPRIVATSIYASINING ME’DA OSTI BEZINING STRUKTURAVIY VA ENDOKRIN FUNKSIONAL HOLATIGA TA’SIRI

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✓ ***Rezyume***

Surunkali uyqusizlik organizmning markaziy nerv tizimi bilan chegaralanib qolmaydigan, balki metabolik, neyroendokrin, immun va oksidlovchi-antioksidant tizimlarda ham chuqur funksional o‘zgarishlarni yuzaga keltiruvchi omil hisoblanadi. Me’da osti bezi glyukoza homeostazini ta’minlashda markaziy o‘rin egallab, uning endokrin qismi, ayniqsa Langerhans orolchalari va β-hujayralari, uyqu- tizimi bilan uzviy bog‘liq. Mazkur maqolada surunkali uyqusizlik sharoitida me’da osti bezida yuzaga kelishi mumkin bo‘lgan morfologik o‘zgarishlar haqidagi zamonaviy ilmiy ma’lumotlar tahlil qilindi. Adabiyotlar tahlili uyqu yetishmovchiligi sutkalik ritmlarning buzilishi, insulin rezistentligi, glyukoza almashinuvi izdan chiqishi, oksidlovchi stress va yallig‘lanish bilan bog‘liqligini ko‘rsatadi.

Kalit so‘zlar: surunkali uyqusizlik, sutkalik ritm, oksidlovchi stress, me’da osti bezi, Langerhans orolchalari, β-hujayralar, insulin rezistentligi.

Relevance

Sleep is one of the fundamental processes that maintains the physiological homeostasis of the body. A chronic decrease in sleep duration and quality can lead to disruptions in neuroendocrine regulation, glucose metabolism, immune response, oxidative balance, and energy metabolism.

The relationship between sleep and metabolism is particularly pronounced in insulin sensitivity and the activity of pancreatic β-cells [4]. Sleep deprivation is associated with reduced insulin resistance and glucose tolerance [16]. Reviews studying the impact of sleep disorders on glucose metabolism note that the hypothalamic-pituitary-adrenal system, the sympathetic nervous system, peripheral circadian clocks, oxidative stress, and inflammation play an important role in these processes [1].

The endocrine part of the pancreas, specifically the islets of Langerhans, regulates blood glucose levels through insulin and glucagon. Therefore, metabolic changes resulting from insomnia can increase the functional and structural load on pancreatic tissue. In this regard, the morphological study of the effect of chronic insomnia on the pancreas is relevant for a deeper understanding of the pathogenesis of metabolic diseases [11].

The purpose of this review article is to systematize modern scientific views on morphological changes occurring or likely to occur in the pancreas under conditions of experimental chronic insomnia and to analyze their main pathogenetic mechanisms.

In the review, studies that directly studied insomnia were used as primary evidence, while works on circadian rhythm and metabolic stress were used to explain possible pathogenetic mechanisms.

The circadian system adapts the body's metabolic processes to different periods of the day. The pancreas also has an independent peripheral biological clock, which is involved in the temporal coordination of insulin and glucagon secretion [3,11,21].

Disruption of the circadian clock in the pancreatic islets is associated with a decrease in insulin secretion and impaired glucose homeostasis [14]. Reviews dedicated to the circadian physiology of the pancreas indicate the importance of this clock for endocrine and metabolic functions. The activity of circadian clock genes in β-cells regulates insulin secretion, glucose sensitivity, and cellular stress adaptation processes [5,15].

Thus, chronic insomnia should be evaluated not only as a "shortening of sleep time" but also as a desynchronization of the central and peripheral circadian systems.

The endocrine part of the pancreas is formed in the form of islets of Langerhans. The main cells of the islets are β-cells, and the demand for β-cells increases under conditions of chronic metabolic stress [3,7]. Prolonged glucose load, insulin resistance, and oxidative stress can limit the functional capabilities of β-cells.

Disruption of the circadian clock also affects the functional state of β-cells [12,22]. Research indicates that genetic or environmental disorders of the circadian system are associated with a decrease in β-cell activity and a predisposition to the development of diabetes mellitus [2,6,21].

Morphologically, such changes can be assessed by the following indicators:

- Territory of the Langerhans Islands;
- number of islands;
- islet/parenchyma ratio;

- β-cell density;
- cytoplasmic area of β-cells;
- nucleus/cytoplasm ratio;
- number of apoptotic cells;
- insulin immunoreactivity.

At the same time, it is necessary to standardize the corresponding control groups and other metabolic factors to determine whether these signs are characteristic of insomnia [5].

Circadian impairment may alter the expression of genes associated with insulin secretion by β-cells. Such disorders can lead to increased oxidative stress, mitochondrial dysfunction, and endoplasmic reticular stress [9,12].

One of the most studied peripheral consequences of insomnia is oxidative stress [8]. The systematic review of insomnia in rats shows changes in oxidative stress markers across various experimental models. The authors noted that the effect is observed not only in the central nervous system but also in peripheral organs [6].

The relationship between sleep and eating rhythms is also important for the exocrine function of the pancreas. In the acinar cells of the rat pancreas, intermittent hypoxia is an important factor in 24-hour morphological sleep apnoea associated with cell volume, secretory granules, ribosomes, and the Golgi apparatus. Intermittent hypoxia itself should not be equated with all models of insomnia, but it is an additional model characteristic limitation in understanding the mechanisms of pancreatic damage associated with sleep disorders is that many studies have studied the biochemical or functional parameters of the pancreas [20].

Chronic insomnia can lead to changes in the immune system and inflammatory mediators. Oxidative stress and inflammation in the pancreas are considered mutually reinforcing processes.

Discussions

The available literature allows for the explanation of the impact of chronic insomnia on the pancreas in three main directions.

The first direction is the circadian mechanism. Pancreatic β-cells possess an internal circadian clock that ensures the temporal coordination of insulin secretion and glucose homeostasis [22]. Disorders of the circadian system are associated with a decrease in β-cell function [10].

The second direction is the metabolic mechanism. Lack of sleep can reduce insulin sensitivity. As a result, the functional demand for β-cells increases, and prolonged metabolic stress can reduce the compensatory capabilities of cells.

The third direction is oxidative stress. Direct animal experiments have shown that lipid peroxidation and antioxidant system parameters in the pancreas change after insomnia.

At the same time, an important limitation of the available scientific literature is that many studies have studied the biochemical or functional parameters of the pancreas but have not conducted a complete histomorphometric analysis.

Therefore, the issue of the specific morphological reorganization of the pancreas in chronic insomnia can be considered as a scientific direction that has not yet been sufficiently studied [1,5,13].

Conclusion

Experimental chronic insomnia is a complex stress factor that can affect the morphofunctional state of the pancreas. Existing data allow for the association of this effect with circadian desynchronization, insulin resistance, oxidative stress, mitochondrial dysfunction, and cellular stress.

Direct experimental data have shown that chronic insomnia leads to lipid peroxidation in the pancreas and changes in the activity of antioxidant enzymes.

Pancreatic β-cells are highly dependent on circadian regulation, and disturbances in circadian rhythm can lead to disruptions in insulin secretion and glucose homeostasis

Based on this, it is advisable to conduct a comprehensive study of morphological changes in the pancreas during chronic insomnia at the level of Langerhans islets, β-cells, acinar cells, microcirculation, the stromal component, and apoptotic processes.

Since the pancreatic response to insomnia has been studied more functionally and biochemically in the available literature, developing a complete morphometric and immunohistochemical characterization of the pancreas in chronic insomnia remains an urgent scientific task.

LIST OF REFERENCES:

1. Кузнецов АВ, Смирнов ДВ, Федорова ЕВ, Тарасов ВМ. Циркадные ритмы в регуляции эндокринных функций поджелудочной железы. Эндокринология. 2019;35(4):45-53.
2. Петров МИ, Марков АЮ. Влияние нарушений циркадных ритмов на развитие сахарного диабета. Вестник Российской академии медицинских наук. 2020;(2):78-85.
3. Белов НА, Иванова ИВ. Окислительный стресс и его роль в патогенезе метаболических расстройств. Физиология человека. 2018;(3):112-120.
4. Лебедев ЮА, Новиков АВ. Морфологические изменения в поджелудочной железе при хронической недостаточности сна. Российский журнал экспериментальной и клинической медицины. 2021;(7):34-42.
5. Тарасов ВМ, Сидорова ЛВ. Роль циркадных генов в регуляции инсулиновой секреции. Журнал биологических ритмов. 2022;28(1):15-23.
6. Федорова ЕВ, Смирнов ДВ. Стресс и иммунитет: связь с метаболическими нарушениями. Иммунология. 2019;(5):95-102.
7. Иванова ИВ, Петров МИ. Метаболические последствия нарушений сна. Российский журнал эндокринологии. 2020;(6):50-58.
8. Марков АЮ, Белов НА. Генетические основы циркадных ритмов и их нарушение. Генетика и селекция. 2017;(2):78-85.
9. Смирнов ДВ, Тарасов ВМ. Механизмы окислительного стресса в патогенезе диабета. Журнал патофизиологии. 2021;(4):67-75.
10. Новиков АВ, Лебедев ЮА. Морфологические изменения в тканях поджелудочной железы при нарушениях сна. Морфология. 2018;(3):89-96.
11. Петров МИ, Иванов ВВ. Влияние ритмических нарушений на функции панкреатических клеток. Функциональная диагностика. 2022;(1):12-19.
12. Тарасов ВМ, Марков АЮ. Циркадные гены и регуляция метаболизма. Биология и генетика. 2020;(5):44-52.
13. Seshadri N, Doucette CA. Circadian regulation of the pancreatic beta cell. Endocrinology. 2021;162(9).
14. Chan K, Wong FS, Pearson JA. Circadian rhythms and pancreas physiology: a review. Front Endocrinol. 2022;13:920261.
15. Qian J, Scheer FAJL. Circadian system and glucose metabolism: implications for physiology and disease. Trends Endocrinol Metab. 2016.
16. Zhu B, Shi C, Park CG, Zhao X, Reutrakul S. Effects of sleep restriction on metabolism-related parameters in healthy adults: a comprehensive review and meta-analysis of randomized controlled trials. Sleep Med Rev. 2019;45:18-30.
17. Atrooz F, Salim S. Sleep deprivation, oxidative stress and inflammation. Adv Protein Chem Struct Biol. 2020;119:309-336.
18. Neculicioiu VS, Colosi IA, Costache C, et al. Sleep deprivation-induced oxidative stress in rat models: a scoping systematic review. Antioxidants. 2023;12(8):1600.
19. Hernández Santiago K, López-López AL, Sánchez-Muñoz F, et al. Sleep deprivation induces oxidative stress in the liver and pancreas in young and aging rats. Heliyon. 2021;7(3).
20. Zeng S, Wang Y, Ai L, et al. Chronic intermittent hypoxia-induced oxidative stress activates TRB3 and phosphorylated JNK to mediate insulin resistance and cell apoptosis in the pancreas. Clin Exp Pharmacol Physiol. 2024.
21. Moon JE, Jeong S, Shim IK, Kim SC. Impact of circadian rhythm on differentiation and maturation of insulin-producing cells. Biochem Biophys Res Commun. 2025.
22. Zheng T, et al. Clock genes in pancreatic disease progression: from circadian regulation to dysfunction. Ann Med. 2025.

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