Cell. Mol. Biol. (ISSN: 1165-158X)

Journal Homepage: www.cellmolbiol.org

Cellular and Molecular Biology

Original Article

Congruency of exosome-mediated blood–brain barrier dysfunction by activation of Na/K-ATPase in obstructive sleep apnea and Alzheimer's disease

Abdelnaby Khalyfa[1*], Jose M. Marin[2], David Sanz-Rubio[2], Sneha S. Pillai[3], Xiaoman Zhang[4], David Gozal[5], Komal Sodhi[3]

[1]Department of Biomedical Sciences, Joan C. Edwards School of Medicine, Marshall University, Huntington, West Virginia, USA
[2]Translational Research Unit, Hospital Universitario Miguel Servet & IISAragon, CIBERES, 50009 Zaragoza, Spain
[3]Department of Surgery, Internal Medicine and Biomedical Sciences, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV 25701, USA
[4]Department of Otolaryngology Head and Neck Surgery & Shanghai Key Laboratory of Sleep Disordered Breathing & Otolaryngology Institute of Shanghai Jiao Tong University, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai 200233, China
[5]Department of Pediatrics and Office of the Dean, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV 25755, USA

Article Info

Article history:

Received: March 03, 2025
Accepted: May 12, 2025
Published: May 31, 2026

Use your device to scan and read the article online

Abstract

Obstructive sleep apnea (OSA) and Alzheimer's disease (AD) are highly prevalent chronic disorders that share several pathological features, including oxidative stress, inflammation, vascular dysfunction, and cognitive impairment. Although epidemiological studies have demonstrated a strong association between OSA and an increased risk of AD, the mechanisms linking these disorders remain poorly understood. We hypothesized that circulating plasma exosomes from patients with OSA or AD disrupt blood–brain barrier (BBB) integrity through activation of Na^+/K^+-ATPase signaling. Plasma exosomes were isolated from adult male patients with OSA or AD (n = 10/group) and applied to naïve human brain microvascular endothelial cells (hCMEC/D3). BBB integrity was continuously monitored using electric cell-substrate impedance sensing (ECIS) in the presence or absence of the Na^+/K^+-ATPase signaling inhibitor pNaKtide (0–2.0 μM). Exosomes derived from both OSA and AD patients significantly impaired endothelial barrier function, indicating that circulating extracellular vesicles carry bioactive cargo capable of disrupting BBB integrity. Pharmacological inhibition of Na^+/K^+-ATPase signaling with pNaKtide significantly restored barrier function at concentrations of 1.0 and 2.0 μM, although the degree of protection differed between the OSA- and AD-derived exosomes, suggesting disease-specific mechanisms of endothelial injury. These findings identify Na^+/K^+-ATPase signaling as a key mediator of exosome-induced BBB dysfunction and provide mechanistic evidence linking circulating extracellular vesicles to neurovascular injury in both OSA and AD. Collectively, our results suggest that plasma-derived exosomes contribute to BBB disruption and highlight Na^+/K^+-ATPase signaling as a promising therapeutic target for preventing or attenuating neurovascular dysfunction associated with OSA and AD.

Keywords: Obstructive sleep apnea; Alzheimer's disease; Plasma exosomes; Blood–brain barrier; Na^+/K^+-ATPase signaling; pNaKtide; Endothelial dysfunction

1. Introduction

The presence of obstructive sleep apnea (OSA) is associated with cognitive decline and dementia in older adults [1, 2]. Alzheimer's disease (AD) is a progressive neurodegenerative disorder that impairs cognitive function and places a substantial socioeconomic burden on society. OSA is also recognized as a significant risk factor that may contribute to both the development and progression of AD [3]. Evidence from animal, epidemiological and human AD studies suggests a bidirectional relationship, with OSA potentially worsening existing AD. These deficits are likely mediated, at least in part, by alterations in blood–brain barrier (BBB) integrity [4, 5]. The BBB is a selective barrier in brain capillaries that regulates the exchange of molecules, ions, and cells, and alterations in these cellular components have been implicated in several neurodegenerative disorders including AD [6-8]. BBB dysfunction can impair brain function, and intermittent hypoxia, such as that characteristically occurs in OSA, exacerbates this process by promoting endothelial dysfunction, oxidative stress, and neuroinflammation, ultimately increasing BBB permeability and contributing to cognitive decline [5].

Exosomes are small extracellular vesicles (EVs) ranging from 30 to 150 nm in size that are detected and secre-

* Corresponding author.
E-mail address: khalyfa@marshall.edu (A. Khalyfa).
Doi: http://dx.doi.org/10.14715/cmb/2026.72.5.4

ted by numerous cell types in a variety of tissues including blood and other body fluids [9]. These exosomes have been identified as pivotal mediators of intercellular communication with crucial roles in physiological and pathological conditions, as well as possible diagnostic markers for disease [10]. Furthermore, exosomes are involved in a wide range of physiological processes such as immune responses, tissue repair, stem cell maintenance and pathological processes involving cardiovascular diseases and neurodegeneration [11].

Repetitive cycles of intermittent hypoxia followed by reoxygenation in OSA promote reactive oxygen species (ROS) production and the resulting oxidative stress and chronic inflammatory state are the main contributors to several comorbidities, including neurodegenerative diseases like AD [12, 13]. Sodium/potassium ATPase (NKA), a member of the P-type ATPase family, has a signaling function that augments oxidative stress in a manner distinct from its ion-pumping function [14].

We hypothesized that circulating plasma-derived exosomes from patients with Alzheimer's disease (AD) and obstructive sleep apnea (OSA) activate Na^+/K^+-ATPase signaling in brain microvascular endothelial cells, leading to oxidative stress, inflammatory signaling, and disruption of blood–brain barrier (BBB) integrity, as illustrated in Figure 1. Beyond its role as an ion transporter, Na^+/K^+-ATPase functions as a signaling receptor that amplifies reactive oxygen species (ROS) production, promoting endothelial dysfunction and neurovascular injury. We further hypothesized that inhibition of this pathway with pNaKtide would interrupt this oxidative signaling cascade and preserve BBB function. Because OSA is associated with an increased risk of cognitive decline and AD, targeting Na^+/K^+-ATPase signaling may represent a novel therapeutic strategy to reduce neurovascular dysfunction. To test this hypothesis, we examined the effects of plasma-derived exosomes from OSA and AD patients on BBB integrity using an established *in vitro* human brain microvascular endothelial cell (hCMEC/D3) model in the presence or absence of pNaKtide.

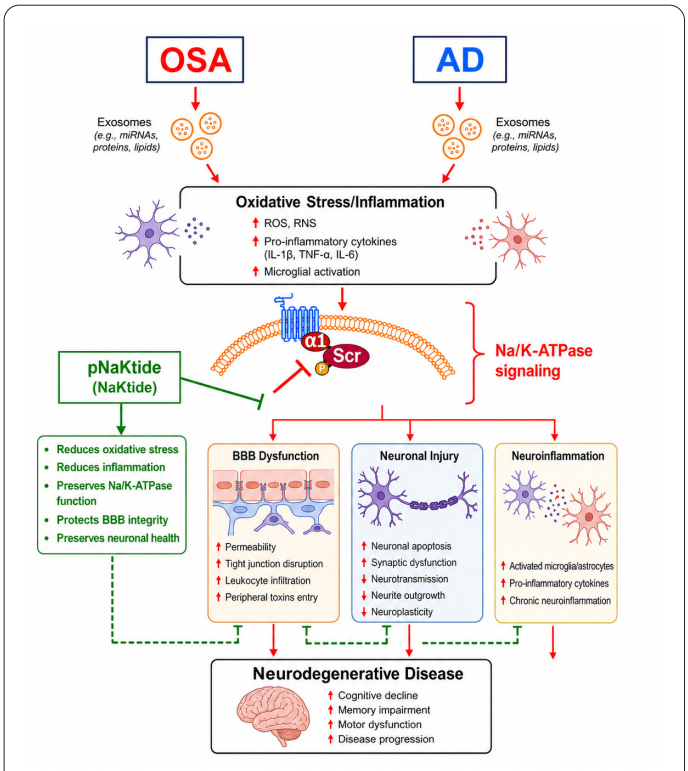


Fig. 1. Mechanistic link between OSA, AD, and neurodegeneration. OSA and AD release exosomes that drive oxidative stress and inflammation, marked by increased reactive oxygen and nitrogen species (ROS/RNS), pro-inflammatory cytokines, and microglial activation. This activates Na^+/K^+-ATPase–dependent signaling through the α1 subunit and Src kinase signaling, amplifying redox imbalance. Downstream effects include BBB dysfunction, neuronal injury, and chronic neuroinflammation, leading to neurodegenerative disease. pNaKtide inhibits this pathway, reducing oxidative stress and inflammation while preserving BBB integrity and neuronal health.

2. Materilas and Methods

2.1. Study population

For AD patients, the Ethics Committee of Marshall University Institutional Review Board approved the study (Protocol #: 1757741 & 2183533) and the study was conducted at the Cabell Huntington Hospital, Huntington, West Virginia, USA. The study was performed according to the guidelines and regulations specified in the Declaration of Helsinki for the use and recruitment of human research subjects. All patients were informed about the use of their blood samples and provided written informed consent.

For OSA patients, human studies were conducted at the Sleep Clinic of the Hospital Universitario Miguel Servet, a large teaching hospital in Zaragoza, Spain, as part of the EPIOSA study (NCT02131610) as previously described [15]. Eligible participants were 18 to 60 years old with polysomnographically confirmed OSA. All subjects completed overnight sleep studies conducted in accordance with American Academy of Sleep Medicine guidelines by trained personnel who were blinded to the study aims. Blood samples were collected and plasma was separated by centrifugation, aliquoted and stored at -80°C.

2.2. Plasma and EVs isolation

Blood samples were collected in Vacutainer tubes and centrifuged at 2000 × g for 20 min at 4 °C. Plasma was collected and stored at −80°C. Exosomes were isolated from plasma using the Total EVs Isolation Kit (Life Technologies) following the manufacturer's protocol. Briefly, plasma was centrifuged at 2000 × g for 20 min to remove debris, and the supernatant was mixed with 0.2 volumes of isolation reagent and incubated at 4 °C for 30 min. Samples were then filtered (0.22 µm), centrifuged at 10,000 ×g for 10 min, and the resulting pellet was resuspended in 1× PBS. Exosomes were isolated and characterized from plasma using a previously established enrichment protocol [16, 17].

2.3. Cell cultures

Human brain microvascular endothelial cells (hCMEC/D3; Sigma-Aldrich) were maintained according to the manufacturer's instructions. hCMEC/D3 Cells were cultured in EndoGRO™-MV Complete Media Kit (Cat. No. SCME004) supplemented with 1 ng/mL FGF-2 (Cat. No. GF003) and incubated at 37°C with 5% CO_2 and saturated humidity for 2-3 days.

2.4. Blood-brain barrier integrity

2.4.1. Electric cell–substrate impedance sensing (ECIS)

Endothelial barrier resistance was measured by ECIS, which continuously monitors impedance across cell monolayers. hCMEC/D3 cells were cultured in low-FBS

media for 24 h, then seeded (50,000 cells/well) on collagen II–coated 8W10E arrays and grown to confluence. Impedance was recorded across multiple frequencies until a stable barrier formed. Exosomes were then added ± pNaKtide and monitored for up to 24 h. Varying doses of pNaKtide were added (0.5, 1.0 and 2 μM respectively) to the wells. Of note, pNaKtide is a peptide derived from α1 Na/K-ATPase that disrupts the NKAα1–Src interaction, blocking Src activation and downstream ROS production [18]. Changes in impedance reflect alterations in endothelial junction integrity.

2.5. Statistical analysis

All statistical analyses were conducted using GraphPad Prism, version 11.0. Results are presented as mean ± standard deviation (SD). The effects of exosome and cell treatments on the BBB monolayer were analyzed using two-way repeated-measures ANOVA, followed by Tukey's *post hoc* test for multiple comparisons. Statistical significance was set at $p \leq 0.05$.

3. Results

3.1. Demographic characteristics

The study included 10 patients with Alzheimer's disease (AD) and 10 patients with obstructive sleep apnea (OSA). Participants in the AD group had a mean age of 80.5 ± 7.05 years, whereas those in the OSA group were substantially younger, with a mean age of 38.81 ± 10.48 years. Body mass index (BMI) was comparable between groups, averaging 25.38 ± 3.34 kg/m² in the AD group and 28.82 ± 3.21 kg/m² in the OSA group. Similarly, systolic blood pressure (SBP) was 127.4 ± 21.18 mmHg in the AD group and 125.11 ± 11.38 mmHg in the OSA group, while diastolic blood pressure (DBP) averaged 73.5 ± 13.31 mmHg and 75.61 ± 11.01 mmHg, respectively. These findings indicate that both groups had generally comparable blood pressure profiles despite their marked difference in age.

Among participants with OSA, the mean apnea–hypopnea index (AHI) was 41.6 ± 15.52 events/hour, consistent with severe OSA. Although the AD and OSA cohorts differed considerably in age, BMI and blood pressure were relatively similar, minimizing the likelihood that these cardiovascular variables were major contributors to the observed differences in exosome-induced endothelial responses. The severe AHI observed in the OSA cohort confirms a substantial burden of intermittent hypoxia and sleep disruption, supporting the biological relevance of investigating circulating plasma exosomes as mediators of blood–brain barrier dysfunction in this population.

3.2. Barrier integrity

To determine whether circulating plasma-derived exosomes impair blood–brain barrier (BBB) function, confluent human brain microvascular endothelial cells (hCMEC/D3) were exposed to equal numbers of exosomes isolated from patients with Alzheimer's disease (AD) or obstructive sleep apnea (OSA). BBB integrity was continuously monitored by electric cell-substrate impedance sensing (ECIS), which measures transendothelial electrical resistance as an indicator of endothelial barrier function. Exosomes were applied to duplicate wells in the presence or absence of the Na^+/K^+-ATPase signaling inhibitor pNaKtide (0.5–2.0 μM), and impedance was recorded for 24 hours.

Exposure to plasma-derived exosomes from either AD or OSA patients significantly reduced endothelial resistance compared with untreated control cells, indicating progressive disruption of BBB integrity (Figure 2A–D). In both groups, barrier dysfunction developed in a time-dependent manner, with the greatest decline observed in the absence of pNaKtide.

For AD-derived exosomes, normalized resistance progressively declined throughout the 24-hour recording period (n = 10/experimental condition), reaching approximately 63% of baseline in untreated cells. Co-treatment with pNaKtide significantly attenuated this reduction in a concentration-dependent manner. The lowest concentration (0.5 μM) partially preserved barrier function, maintaining resistance at 71% of baseline ($p = 0.08$), whereas 1.0 μM significantly improved resistance to 75% ($p = 0.0003$). The highest concentration (2.0 μM) produced the greatest protective effect, preserving approximately 91% of baseline resistance ($p = 0.0001$) (Figure 2A, B). Differences between treated and untreated cells became evident after approximately 8–12 hours and increased throughout the remainder of the experiment.

A similar response was observed following exposure to OSA-derived exosomes. In the absence of pNaKtide, endothelial resistance declined rapidly within the first 4–8 hours, reaching approximately 63% of baseline by 24 hours (n = 10/experimental condition). pNaKtide significantly attenuated this loss of barrier function in a dose-dependent manner, preserving resistance at 73% (0.5 μM, $p = 0.0001$), 76% (1.0 μM, $p = 0.0001$), and 87% (2.0 μM, $p = 0.0001$) (Figure 2C, D). Unlike the AD group, the protective effects of pNaKtide against OSA-derived exosomes

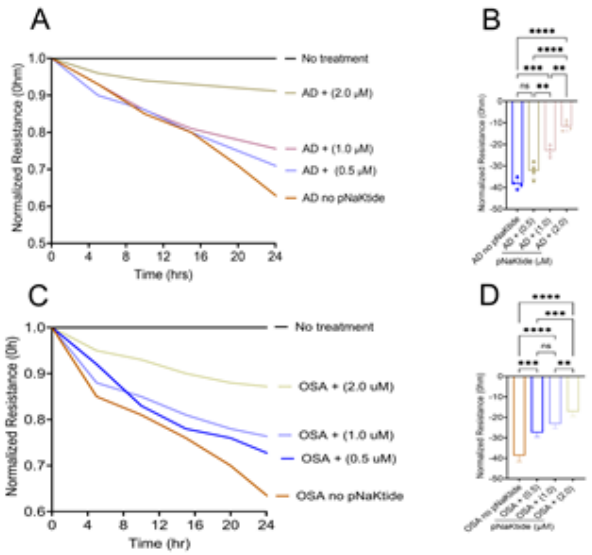


Fig. 2. Effects of the Na^+/K^+-ATPase inhibitor pNaKtide on circulating exosomes from AD or OSA patients and the impact on BBB endothelial monolayer barrier integrity. Panels (A) and (B) show representative time-course impedance recordings following the addition of exosomes from aged male AD and from OSA patients, respectively, to endothelial monolayers with or without pNaKtide. Panels (C) and (D) show the quantified average disruption of endothelial monolayer resistance for AD and OSA patients, respectively, with or without pNaKtide. Data were recorded using an ECIS Zθ instrument equipped with ECIS software. Data are presented as mean ± S.D.; n = 10.

became apparent during the early phase of the experiment, suggesting a more rapid onset of exosome-induced endothelial injury.

Collectively, these findings demonstrate that plasma-derived exosomes from both AD and OSA patients induce progressive BBB dysfunction in human brain endothelial cells. Pharmacological inhibition of Na^+/K^+-ATPase signaling with pNaKtide consistently and dose-dependently preserved endothelial barrier integrity, with the highest concentration providing the greatest protection in both disease groups. These results support a central role for Na^+/K^+-ATPase-mediated signaling in exosome-induced BBB dysfunction and identify this pathway as a potential therapeutic target for neurovascular injury associated with both OSA and AD.

4. Discussion

Here's a pretty concerning finding from this study. It turns out that exosomes circulating in the blood plasma of patients with sleep apnea and Alzheimer's can actually damage the blood–brain barrier (BBB). What's interesting is that this doesn't happen randomly—at least part of the damage is driven by the activation of a specific signaling pathway linked to the sodium-potassium pump.

To make sure we were on the right track, we blocked that pathway pharmacologically using a compound called pNaKtide. And the result? The endothelial barrier held up remarkably well. So there's really no doubt left, this Na^+/K^+-ATPase signaling is a major player in the exosome-induced breakdown of the BBB.

Now, where does this chain of events lead? These exosomes carry all sorts of cargo, proteins, lipids, and nucleic acids, and they aren't just passive passengers. By gradually damaging endothelial cells and weakening that protective shield around the brain, they pave the way for more serious injuries to the central nervous system.

And here's the kicker: the more fragile that barrier becomes, the easier it is for inflammatory mediators and immune cells to sneak into brain tissue. This breach seems to happen much earlier than we might expect. In fact, it could very well be the critical early link that connects peripheral diseases to neuroinflammation, and eventually, to neuronal dysfunction and cognitive decline.

We all know that a leaky blood–brain barrier is a major red flag in neurodegenerative diseases. But here's the thing: we still don't have the full picture. The exact molecular steps that tear that barrier down, and whether that breakdown actually kicks off the disease or just shows up later, are largely still a mystery.

Our data, however, point to a pretty compelling piece of the puzzle. Those circulating extracellular vesicles aren't just cellular debris floating around; they're active messengers. They seem to be picking up pathological signals from the body and carrying them straight to the brain's smallest blood vessels. And given what we already know about the strong link between sleep apnea and Alzheimer's risk, this exosome-driven activation of the sodium-potassium pump starts to look like a common thread, a shared mechanism that could be fueling neurovascular damage in both conditions.

Now, let's be upfront: this is still early-stage work, and we're drawing these conclusions from an in vitro model of the BBB, not from live human brains. So we're not jumping to clinical conclusions just yet. But what this does give us is a solid launchpad. It lays out a clear roadmap for where to go next—first, to pinpoint exactly which specific molecules inside those exosomes are doing the damage, and second, to see if we can actually hit the brakes on this whole process. If we can block those vesicles or shut down that signaling pathway, could we actually preserve the barrier and slow down neurodegeneration? That's the big question we're itching to answer next.

OSA and AD are common conditions with substantial adverse impact on health systems. OSA is linked to cognitive impairment, particularly in attention, memory and executive functions. AD is the leading cause of dementia, with vascular and other neurodegenerative forms making up most remaining cases. OSA has also been associated with cognitive decline and dementia in the elderly. Its underlying features—disrupted sleep, intermittent hypoxia, oxidative stress, intrathoracic pressure changes, and cardiovascular comorbidities—may accelerate the onset and enhance the severity of AD. The current findings corroborate previous in vivo animal studies showing the unique vulnerability of the BBB in conditions such as OSA or AD [4, 8].

The BBB regulates substance exchange vital for brain metabolism and neuronal function, making its integrity essential for maintaining brain homeostasis. Endothelial cells of the BBB form tightly sealed junctions that generate high trans-endothelial electrical resistance that limits both paracellular and transcellular permeability. Preserving BBB integrity is therefore essential for maintaining the chemical balance of the brain interstitial fluid [19] and for regulating communication within the central nervous system (CNS). Consequently, BBB dysfunction is implicated in the development of many neurological diseases [20].

In summary, in this pilot study, we show for the first time that plasma exosomes from patients with either OSA or AD disrupt barrier integrity in naïve hCMEC/D3 cells. In the absence of pNaKtide, these circulating exosomes impair BBB function, as reflected by reduced trans-endothelial impedance. This effect is attenuated by pNaKtide in a concentration-dependent manner, further suggesting a major contributory role played by activation of Na^+/K^+-ATPase, and therefore pointing to a therapeutic strategy opportunity in future studies.

Data availability

Data supporting the results of this study can be obtained from the corresponding author upon reasonable request.

Funding

This study was supported by unrestricted startup funds to A.K from the Joan C. Edwards School of Medicine at Marshall University (Marshall University Research Corporation, MURC, and by a grant from the National Institutes of Health (NIH) grant R01 HL164460 to K.S. DG is supported in part by NIH grants HL166617 and HL169266. This research was also funded in part by grants from the Spanish Fondo de Investigación Sanitaria (Health Research Fund) and European Regional Development Funds PI18/01524 and PI21/01954 to J.M.M.

Declaration of Competing Interests

We declare that the authors have no known conflict of interest or personal relationship that could have a detrimental

effect on the work described in this paper, as well as any financial interests or personal relationships.

Use of Artificial Intelligence Tools

Some sentences in this manuscript were revised with the assistance of artificial intelligence, using language models to improve clarity and readability. All intellectual content, interpretations, and conclusions are solely the responsibility of the authors.

References

1. Yaffe K, Laffan AM, Harrison SL, Redline S, Spira AP, Ensrud KE, Ancoli-Israel S, Stone KL (2011) Sleep-disordered breathing, hypoxia, and risk of mild cognitive impairment and dementia in older women. JAMA 306 (6): 613-619. doi: 10.1001/jama.2011.1115
2. Yang Y, Li M, Leng B, Yao R, Xue S, Tan M, Sun H, Zhang J (2023) Alzheimer's Disease Biomarkers and Complement Proteins Mediate the Impact of Sleep Fragmentation on Cognitive Impairment in Obstructive Sleep Apnea Patients Without Dementia. J Alzheimers Dis 95 (4): 1685-1696. doi: 10.3233/JAD-221288
3. Bubu OM, Andrade AG, Umasabor-Bubu OQ, Hogan MM, Turner AD, de Leon MJ, Ogedegbe G, Ayappa I, Jean-Louis GG, Jackson ML, Varga AW, Osorio RS (2020) Obstructive sleep apnea, cognition and Alzheimer's disease: A systematic review integrating three decades of multidisciplinary research. Sleep Med Rev 50: 101250. doi: 10.1016/j.smrv.2019.101250
4. Puech C, Badran M, Barrow MB, Gozal D (2025) Cognitive Function, Sleep, and Neuroinflammatory Markers in Mice Exposed to Very Long-Term Intermittent Hypoxia. Int J Mol Sci 26 (5). doi: 10.3390/ijms26051815
5. Puech C, Badran M, Runion AR, Barrow MB, Cataldo K, Gozal D (2023) Cognitive Impairments, Neuroinflammation and Blood-Brain Barrier Permeability in Mice Exposed to Chronic Sleep Fragmentation during the Daylight Period. Int J Mol Sci 24 (12). doi: 10.3390/ijms24129880
6. Wu D, Chen Q, Chen X, Han F, Chen Z, Wang Y (2023) The blood-brain barrier: structure, regulation, and drug delivery. Signal Transduct Target Ther 8 (1): 217. doi: 10.1038/s41392-023-01481-w
7. Profaci CP, Munji RN, Pulido RS, Daneman R (2020) The blood-brain barrier in health and disease: Important unanswered questions. J Exp Med 217 (4). doi: 10.1084/jem.20190062
8. Puech C, Sadanand A, Coleman N, Ikhane M, Badran M, Wang R, Gozal D, Parent AT (2026) APP-mediated intracellular signaling rescues sleep impairment and blood-brain barrier leakage in Alzheimer's disease mouse model. Alzheimers Dement 22 (2): e71134. doi: 10.1002/alz.71134
9. Zhou X, Huang J, Zhang D, Qian Z, Zuo X, Sun Y (2025) Small extracellular vesicles: the origins, current status, future prospects, and applications. Stem Cell Res Ther 16 (1): 184. doi: 10.1186/s13287-025-04330-5
10. Vucemilovic A (2024) Exosomes: intriguing mediators of intercellular communication in the organism's response to noxious agents. Arh Hig Rada Toksikol 75 (4): 228-239. doi: 10.2478/aiht-2024-75-3923
11. Singh S, Paul D, Nath V, A R (2024) Exosomes: current knowledge and future perspectives. Tissue Barriers 12 (2): 2232248. doi: 10.1080/21688370.2023.2232248
12. Lavie L (2003) Obstructive sleep apnoea syndrome--an oxidative stress disorder. Sleep Med Rev 7 (1): 35-51. doi: 10.1053/smrv.2002.0261
13. Andrade AG, Bubu OM, Varga AW, Osorio RS (2018) The Relationship between Obstructive Sleep Apnea and Alzheimer's Disease. J Alzheimers Dis 64 (s1): S255-S270. doi: 10.3233/JAD-179936
14. Liang M, Tian J, Liu L, Pierre S, Liu J, Shapiro J, Xie ZJ (2007) Identification of a pool of non-pumping Na/K-ATPase. J Biol Chem 282 (14): 10585-10593. doi: 10.1074/jbc.M609181200
15. Marin JM, Artal J, Martin T, Carrizo SJ, Andres M, Martin-Burriel I, Bolea R, Sanz A, Varona L, Godino J, Gallego B, Garcia-Erce JA, Villar I, Gil V, Forner M, Cubero JP, Ros L (2014) Epigenetics modifications and Subclinical Atherosclerosis in Obstructive Sleep Apnea: The EPIOSA study. BMC Pulm Med 14: 114. doi: 10.1186/1471-2466-14-114
16. Khalyfa A, Marin JM, Sanz-Rubio D, Lyu Z, Joshi T, Gozal D (2023) Multi-Omics Analysis of Circulating Exosomes in Adherent Long-Term Treated OSA Patients. Int J Mol Sci 24 (22). doi: 10.3390/ijms242216074
17. Khalyfa A, Verma M, Alexander MM, Qiao Z, Rood T, Kapoor R, Joshi T, Gozal D, Francisco BD (2025) Childhood Asthma Biomarkers Derived from Plasma and Saliva Exosomal miRNAs. Int J Mol Sci 26 (15). doi: 10.3390/ijms26157043
18. Liu J, Chaudhry M, Bai F, Chuang J, Chaudhry H, Al-Astal AY, Nie Y, Sollars V, Sodhi K, Seligman P, Shapiro JI (2022) Blockage of the Na-K-ATPase signaling-mediated oxidant amplification loop elongates red blood cell half-life and ameliorates uremic anemia induced by 5/6th PNx in C57BL/6 mice. Am J Physiol Renal Physiol 322 (6): F655-F666. doi: 10.1152/ajprenal.00189.2021
19. Bosma EK, van Noorden CJF, Schlingemann RO, Klaassen I (2018) The role of plasmalemma vesicle-associated protein in pathological breakdown of blood-brain and blood-retinal barriers: potential novel therapeutic target for cerebral edema and diabetic macular edema. Fluids Barriers CNS 15 (1): 24. doi: 10.1186/s12987-018-0109-2
20. Sonar SA, Lal G (2018) Blood-brain barrier and its function during inflammation and autoimmunity. J Leukoc Biol 103 (5): 839-853. doi: 10.1002/JLB.1RU1117-428R