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REVIEW ARTICLE

Mitochondrial Stress and Axonal Degeneration in Ramsay Hunt Syndrome: A Bioenergetic Perspective

Malaz KamalEl-din Mirgani Ali[1*], Muhammad Uzair[2]

[1] Sudan International University, Khartoum, Sudan

[2] Liaquat University of Medical and Health Sciences; Jamshoro, Pakistan

***Corresponding Author:** Malaz KamalEl-din Mirgani Ali
Sudan International University, Khartoum, Sudan.
Email: malaz.kamal999@gmail.com

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Abstract

Ramsay Hunt Syndrome was caused by the reactivation of the varicella-zoster virus in the geniculate ganglion. It is a debilitating neurological disorder characterized by facial paralysis, otalgia, and vesicular eruptions. Despite antiviral and corticosteroid therapies, recovery remains suboptimal for many patients. Recent evidence suggests that mitochondrial dysfunction plays a central role in the progression of neural damage in Ramsay Hunt Syndrome. Mitochondria as power house of the cells are critical in maintaining axonal health, energy homeostasis, and redox balance. In Ramsay Hunt Syndrome, viral reactivation initiates an inflammatory cascade that disrupts mitochondrial dynamics, induces oxidative stress, and impairs ATP synthesis. These bioenergetic deficits compromise axonal transport and integrity, leading to degeneration. This review explores the intersection of mitochondrial stress and axonal degeneration in Ramsay Hunt Syndrome, with a focus on the bioenergetic consequences of varicella-zoster virus induced neuroinflammation. By elucidating these mechanisms, we aim to provide a comprehensive understanding of Ramsay Hunt Syndrome pathophysiology and lay the groundwork for novel bioenergetic-based interventions to mitigate nerve injury and promote recovery.

1. Introduction

Ramsay Hunt Syndrome is a neurological disorder caused by the reactivation of the varicella-zoster virus in the geniculate ganglion of the facial nerve (Goswami and Gaurkar, 2023). It is characterized by a constellation of symptoms including unilateral facial paralysis, otalgia, vesicular rash in the ear canal or auricle, and occasionally sensorineural hearing loss and vertigo. While the hallmark clinical features and virologic underpinnings of Ramsay Hunt Syndrome have been extensively described, the precise cellular mechanisms contributing to axonal degeneration and sustained neurological deficits remain poorly understood. Recent advances in neurobiology and mitochondrial physiology suggest that mitochondrial stress may serve as a critical nexus in the pathogenesis of Ramsay Hunt Syndrome, linking viral reactivation, neuroinflammation, and axonal degeneration through bioenergetic dysregulation.

Mitochondria as the powerhouses of the cell are integral to neuronal function due to their central roles in ATP production, calcium homeostasis, redox regulation, and apoptotic signaling (Feofilaktova et al., 2025). Neurons, particularly long axons of cranial nerves, are metabolically demanding and highly dependent on intact mitochondrial dynamics and function (Yang et al., 2023). Any compromise in mitochondrial integrity due to oxidative stress, altered mitochondrial dynamics (fission/fusion), impaired mitophagy, or disrupted bioenergetic signaling which can lead to profound axonal dysfunction (W. Chen et al., 2023). In the context of Ramsay Hunt Syndrome, the reactivated varicella-zoster virus triggers a localized inflammatory cascade, recruiting immune cells and inducing oxidative stress, which can impair mitochondrial function in affected neurons (Gershon et al., 2015). This may be especially detrimental in the facial and vestibulocochlear nerves, which exhibit high energy requirements and limited regenerative capacity.

The neurotropism of varicella-zoster virus further complicates the pathology. After primary infection, varicella-zoster virus remains latent in sensory ganglia, including the geniculate ganglion (Shapiro, 2009). Upon reactivation the virus replicates and induces inflammation in sensory and motor neurons. The geniculate ganglion, which houses both motor and sensory fibers of the facial nerve, becomes a focal point of viral replication and immune-mediated damage (Wang et al., 2025). Inflammation within the confined bony facial canal can cause ischemia and compression of neural structures, further compounding mitochondrial dysfunction and axonal injury. This convergence of viral, inflammatory, and metabolic insults suggests a crucial role for mitochondria as both targets and amplifiers of neuronal damage in Ramsay Hunt Syndrome. In addition to direct viral cytopathy and bystander immune damage, emerging evidence indicates that mitochondrial reactive oxygen species (ROS) play a significant role in neural degeneration. Mitochondria are major sources of ROS under stress conditions, and excessive ROS generation can lead to oxidative damage of lipids, proteins, and mitochondrial DNA (mtDNA), triggering a vicious cycle of mitochondrial injury and bioenergetic collapse (Bhat et al., 2015). In neurons, such mitochondrial distress can impair axonal transport, synaptic function, and ultimately result in Wallerian degeneration (Conforti et al., 2014). It is a process marked by distal axon breakdown following injury. Ramsay Hunt Syndrome involves facial nerve palsy and potentially auditory and vestibular dysfunction, the role of mitochondrial-induced axonal degeneration needs close investigation. Recent insights into mitochondrial quality control pathways such as mitophagy (selective degradation of damaged mitochondria), biogenesis, and fusion/fission dynamics further support the hypothesis that mitochondrial failure contributes to the poor regenerative outcome seen in Ramsay Hunt Syndrome patients. Studies have shown that impaired mitophagy leads to the accumulation of dysfunctional mitochondria, which exacerbates neuroinflammation and axonal loss (Wu et al., 2021). Furthermore, mitochondrial fission protein DRP1 and fusion proteins such as MFN1/2 and OPA1, which regulate mitochondrial morphology and distribution, are often dysregulated in neurodegenerative diseases and viral neuropathies (Kathiresan et al., 2025). It is plausible that similar disruptions occur in Ramsay Hunt Syndrome, especially in neurons under viral attack and metabolic stress.

Beyond the facial nerve, the involvement of cranial nerves VIII and IX in some Ramsay Hunt Syndrome cases points to a broader spectrum of neural injury potentially mediated by common mitochondrial mechanisms (Ananthapadmanabhan et al., 2022). Sensorineural hearing loss and vertigo, seen in up to 30–50% of patients, implicate mitochondrial compromise in cochlear and vestibular neurons. Studies on other auditory neuropathies and vestibular pathologies reveal that mitochondrial dysfunction plays a central role in hair cell loss, spiral ganglion neuron degeneration, and impaired synaptic transmission (Feng et al., 2020). This review aims to provide a comprehensive analysis of the role of mitochondrial stress in axonal degeneration observed in Ramsay Hunt Syndrome.

2. Mitochondrial Dysfunction in varicella-zoster virus Reactivation

Varicella-zoster virus reactivation is a key pathological event in Ramsay Hunt Syndrome, initiating a cascade of neural damage centered around the geniculate ganglion and facial nerve. While viral replication and immune-mediated cytotoxicity are primary contributors, recent studies suggest that mitochondrial dysfunction plays a crucial intermediary role (Qu et al., 2019). The varicella-zoster virus infected neuron experiences both direct viral stress and a pro-inflammatory microenvironment, conditions that disrupt mitochondrial integrity. This dysfunction manifests as impaired oxidative phosphorylation (OXPHOS), leading to ATP depletion and bioenergetic crisis in the axon. Compromised ATP production affects axonal transport, neurotransmission, and the maintenance of ion gradients functions which are essential for neuronal survival (Chamberlain and Sheng, 2019). Additionally, varicella-zoster virus infection can modulate mitochondrial dynamics, promoting excessive fission via Drp1 activation and inhibiting fusion proteins like Mfn1/2 (Duan et al., 2025), thus favouring mitochondrial fragmentation and dysfunction. Viral proteins may also localize to mitochondria, further impeding their function and triggering the release of pro-apoptotic factors. Moreover, mitochondrial DNA (mtDNA) damage caused by ROS can impair transcription of essential respiratory enzymes, amplifying the dysfunction (Liu and Chen, 2017). The loss of mitochondrial membrane potential also compromises mitophagy, preventing the clearance of damaged organelles. These combined events set the stage for axonal degeneration and prolonged neural deficits. Understanding mitochondrial responses to varicella-zoster virus reactivation is thus essential in identifying therapeutic targets that preserve bioenergetic health and prevent irreversible nerve injury in Ramsay Hunt Syndrome (Table 1).

Table 1: Mitochondrial Dysfunctions in Ramsay Hunt Syndrome and Corresponding Therapeutic Strategies

Mitochondrial Dysfunction	Pathological Effect in Ramsay Hunt Syndrome	Targeted Therapeutic Strategy
Impaired Oxidative Phosphorylation	ATP depletion; axonal transport and synaptic failure	Bioenergetic support
Excess ROS	Oxidative damage to lipids, proteins, mtDNA	Mitochondria-targeted antioxidants
Mitochondrial Fragmentation (↑ Fission / ↓ Fusion)	Loss of structural integrity; energy imbalance	Modulators of mitochondrial dynamics
Mitophagy Deficiency	Accumulation of dysfunctional mitochondria; chronic inflammation	Mitophagy enhancement
Loss of Mitochondrial Membrane Potential (ΔΨm)	Blocked mitophagy; apoptosis initiation	Membrane stabilizers; PINK1-Parkin pathway support
mtDNA Damage	Reduced respiratory chain enzyme expression	Antioxidants; mitochondrial biogenesis stimulators
Viral Protein Interference	Direct inhibition of mitochondrial function	Early antiviral therapy

3. Oxidative Stress and Energy Failure in Facial Nerve Degeneration

Oxidative stress and impaired energy metabolism are intimately linked and represent major contributors to facial nerve degeneration in Ramsay Hunt Syndrome. Following varicella-zoster virus reactivation, immune cells such as macrophages and T cells infiltrate the affected ganglion and nerves, releasing pro-inflammatory cytokines (e.g., TNF-α, IL-1β) and generating high levels of ROS (Hakami et al., 2024). Mitochondria, as both sources and targets of ROS, become compromised under these conditions. Excess ROS causes lipid peroxidation, protein oxidation, and mtDNA damage culminating in mitochondrial dysfunction and energy failure. In neurons, ATP is critical for axonal transport, synaptic transmission, and calcium buffering. When mitochondrial ATP synthesis is impaired, axons lose the capacity to maintain ion gradients and cytoskeletal dynamics, leading to degeneration (Cheng et al., 2022). Additionally, oxidative stress activates stress-responsive kinases such as JNK and p38 MAPK, which can further exacerbate mitochondrial injury and initiate apoptotic pathways (Sinha et al., 2013). The facial nerve, confined within the bony facial canal, is particularly vulnerable to ischemia and energy deficits due to limited capacity for metabolic compensation. Furthermore, impaired mitochondrial calcium handling contributes to excitotoxicity and axoplasmic stasis. Together, these factors trigger Wallerian degeneration, characterized by fragmentation of the axon distal to the injury site. Therapeutic strategies that target oxidative stress such as mitochondrial-targeted antioxidants may protect facial nerve integrity by preserving redox balance and sustaining ATP production. Addressing this bioenergetic crisis is vital for improving recovery outcomes and minimizing long-term facial paralysis in Ramsay Hunt Syndrome.

4. Mitophagy and Mitochondrial Quality Control in Ramsay Hunt Syndrome

Mitochondrial quality control mechanisms, particularly mitophagy know for the selective autophagic removal of damaged mitochondria play a fundamental role in neuronal homeostasis. In Ramsay Hunt Syndrome, the reactivation of varicella-zoster virus and subsequent neuroinflammation impair these protective pathways, contributing to axonal degeneration. Under physiological conditions, mitophagy is tightly regulated by proteins such as PINK1 and Parkin, which tag depolarized mitochondria for degradation via the autophagosome-lysosome system (Springer and Kahle, 2011). In neurons affected by Ramsay Hunt Syndrome, however, excessive ROS and mitochondrial membrane depolarization can inhibit PINK1 stabilization on the outer mitochondrial membrane, preventing Parkin recruitment and mitophagy initiation (Xiao et al., 2017) (Figure 1). Furthermore, viral infection can downregulate autophagic gene expression or alter lysosomal function, leading to the accumulation of dysfunctional mitochondria (T. Chen et al., 2023). This promotes further oxidative damage, loss of ATP, and release of pro-apoptotic factors such as cytochrome c. Inefficient mitophagy also contributes to inflammasome activation, sustaining a chronic inflammatory state that perpetuates tissue injury. In axons, where mitochondrial turnover is slower and dependent on retrograde transport, impaired quality control leads to localized energy deficits and structural collapse. Interventions aimed at enhancing mitophagy, such as the use of pharmacologic agents like urolithin A or NAD+ boosters, have shown promise in preclinical neurodegeneration models and could be translated into Ramsay Hunt Syndrome therapy. Evaluating mitophagy status via biomarkers such as LC3-II, p62, and mitochondrial mass may also provide insights into disease progression. Restoration of mitochondrial quality control mechanisms is essential for preserving neuronal function and promoting recovery in Ramsay Hunt Syndrome.

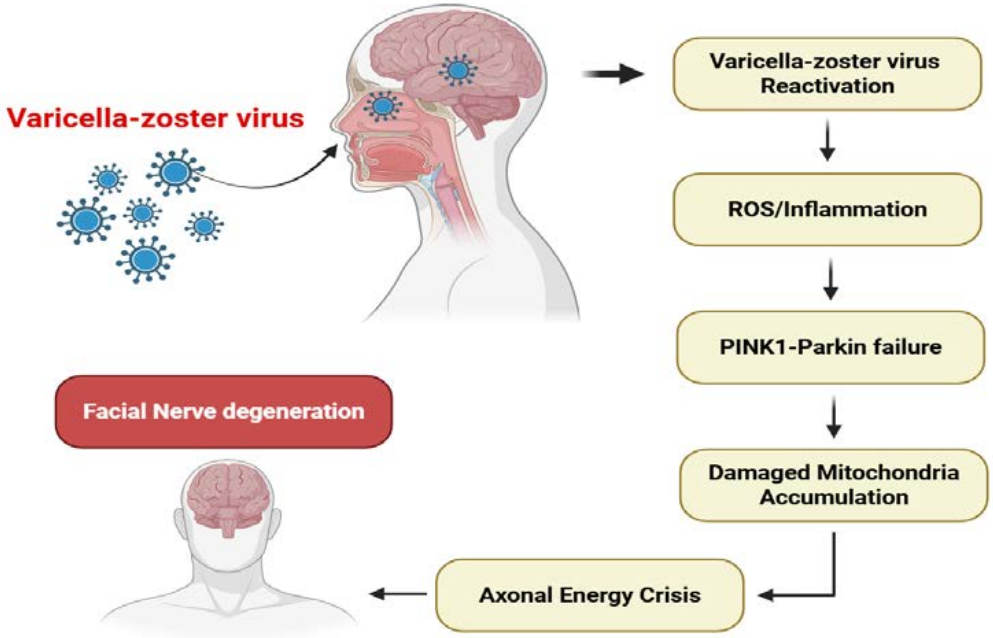

Figure 1: Varicella-zoster virus (VZV) reactivation to axonal degeneration in Ramsay Hunt Syndrome. Viral reactivation triggers reactive oxygen species (ROS) overproduction and neuroinflammation, leading to PINK1-Parkin mitophagy pathway failure. This results in accumulation of damaged mitochondria, bioenergetic crisis (ATP depletion), and subsequent facial nerve degeneration.

5. Bioenergetic Crosstalk Between Cranial Nerves VII and VIII

Ramsay Hunt Syndrome often presents with involvement of both the facial (cranial nerve VII) and vestibulocochlear (cranial nerve VIII) nerves, manifesting as facial paralysis, hearing loss, and vertigo (Perszke and Egierska, 2022). This dual nerve involvement suggests shared pathogenic mechanisms particularly at the level of mitochondrial function and bioenergetic regulation. Both nerves contain long, myelinated axons with high metabolic demands, rendering them susceptible to energy deficits. varicella-zoster virus induced inflammation and mitochondrial stress within the geniculate ganglion can propagate to adjacent neural structures, especially as the facial and vestibulocochlear nerves traverse the internal auditory canal in close anatomical proximity. Mitochondrial dysfunction in this confined space leads to accumulation of ROS, reduced ATP availability, and impaired calcium handling (Pivovarova and Andrews, 2010), affecting signal conduction and synaptic transmission. Auditory neurons and cochlear hair cells are particularly sensitive to oxidative damage, and their mitochondrial reserves are often insufficient to withstand prolonged insult. Additionally, energy failure in vestibular neurons disrupts spatial orientation and balance. Crosstalk between cranial nerves may also occur via shared glial support cells and overlapping vascular territories, both of which are affected by mitochondrial stress. Inflammatory cytokines and mitochondrial damage-associated molecular patterns (mtDAMPs) released from one nerve can trigger bystander injury in neighboring structures (Ye et al., 2023). Understanding this bioenergetic crosstalk may aid in explaining the multisystem presentation of Ramsay Hunt Syndrome and guide targeted therapies to protect both facial and auditory function. Mitochondrial support therapies, anti-inflammatory agents, and antioxidants could be beneficial when applied early in the disease course to preserve the function of both cranial nerves.

6. Future Perspectives

The growing recognition of mitochondrial stress as a central mechanism in Ramsay Hunt Syndrome provides exciting opportunities for both mechanistic research and therapeutic innovation. Future investigations should focus on characterizing mitochondrial alterations in neuronal tissues using high-resolution imaging, transcriptomics, and proteomics. Such studies could uncover specific molecular signatures of mitochondrial dysfunction, including altered dynamics, disrupted mitophagy, and impaired bioenergetic pathways.

Animal models of varicella-zoster virus reactivation and facial nerve inflammation will be essential for evaluating mitochondrial-targeted therapies in vivo. The use of advanced tools like mitochondrial-specific biosensors and optogenetic control of mitochondrial function may provide real-time insights into axonal energy failure and recovery processes. Further, longitudinal studies in patients with Ramsay Hunt Syndrome tracking mitochondrial biomarkers in blood, saliva, or cerebrospinal fluid may help correlate bioenergetic disruption with clinical severity and outcomes. Therapeutically, combining antiviral agents with mitochondrial-protective drugs may offer synergistic benefits. Additionally, understanding mitochondrial crosstalk between cranial nerves may improve management of vestibulocochlear symptoms in

Ramsay Hunt Syndrome. Personalized medicine approaches, guided by mitochondrial genomic analysis, could further refine treatment strategies. Ultimately, integrating mitochondrial health as a diagnostic and therapeutic focus has the potential to significantly improve long-term outcomes in patients affected by Ramsay Hunt Syndrome.

7. Conclusion

Mitochondrial dysfunction plays a pivotal role in axonal degeneration and symptom progression in Ramsay Hunt Syndrome. Viral reactivation, oxidative stress, impaired mitophagy, and energy failure contribute to the vulnerability of facial and auditory nerves. This bioenergetic perspective highlights mitochondria as both mediators and therapeutic targets of nerve injury. By addressing mitochondrial stress early and precisely, clinicians may mitigate long-term neurological deficits. Advancing our understanding of mitochondrial biology in Ramsay Hunt Syndrome can inform the development of targeted interventions, potentially transforming patient outcomes and shifting the paradigm in managing viral neuropathies.

Declarations

Ethics approval statement

Not applicable

Consent to participate

Not applicable

Consent to publish

Not applicable

Data Availability Statement

The data are available from the corresponding author upon reasonable request

Competing Interests

The authors declare that they have no conflict of interest

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During the preparation of this work the authors used AI-assisted technology QuillBot, in order to check the grammar and spell in some sentences

Reference

1. Ananthapadmanabhan, S., Soodin, D., Sritharan, N., Sivapathasingam, V., 2022. Ramsay Hunt syndrome with multiple cranial neuropathy: a literature review. Eur. Arch. Oto-Rhino-Laryngology 279, 2239–2244. https://doi.org/10.1007/s00405-021-07136-2
2. Bhat, A.H., Dar, K.B., Anees, S., Zargar, M.A., Masood, A., Sofi, M.A., Ganie, S.A., 2015. Oxidative stress, mitochondrial dysfunction and neurodegenerative diseases; a mechanistic insight. Biomed. Pharmacother. 74, 101–110. https://doi.org/10.1016/j.biopha.2015.07.025
3. Chamberlain, K.A., Sheng, Z., 2019. Mechanisms for the maintenance and regulation of axonal energy supply. J. Neurosci. Res. 97, 897–913. https://doi.org/10.1002/jnr.24411
4. Chen, T., Tu, S., Ding, L., Jin, M., Chen, H., Zhou, H., 2023. The role of autophagy in viral infections. J. Biomed. Sci. 30, 5. https://doi.org/10.1186/s12929-023-00899-2
5. Chen, W., Zhao, H., Li, Y., 2023. Mitochondrial dynamics in health and disease: mechanisms and potential targets. Signal Transduct. Target. Ther. 8, 333. https://doi.org/10.1038/s41392-023-01547-9
6. Cheng, X.-T., Huang, N., Sheng, Z.-H., 2022. Programming axonal mitochondrial maintenance and bioenergetics in neurodegeneration and regeneration. Neuron 110, 1899–1923. https://doi.org/10.1016/j.neuron.2022.03.015
7. Conforti, L., Gilley, J., Coleman, M.P., 2014. Wallerian degeneration: an emerging axon death pathway linking injury and disease. Nat. Rev. Neurosci. 15, 394–409. https://doi.org/10.1038/nrn3680
8. Duan, X., Liu, R., Lan, W., Liu, S., 2025. The Essential Role of Mitochondrial Dynamics in Viral Infections. Int. J. Mol. Sci. 26, 1955. https://doi.org/10.3390/ijms26051955
9. Feng, B., Jin, C., Cheng, Z., Zhao, X., Sun, Z., Zheng, X., Li, X., Dong, T., Tao, Y., Wu, H., 2020. Mitochondrial Dysfunction and Therapeutic Targets in Auditory Neuropathy. Neural Plast. 2020, 1–10. https://doi.org/10.1155/2020/8843485
10. Feofilaktova, T., Kushnireva, L., Segal, M., Korkotian, E., 2025. Calcium signaling in postsynaptic mitochondria: mechanisms, dynamics, and role in ATP production. Front. Mol. Neurosci. 18. https://doi.org/10.3389/fnmol.2025.1621070
11. Gershon, A.A., Breuer, J., Cohen, J.I., Cohrs, R.J., Gershon, M.D., Gilden, D., Grose, C., Hambleton, S., Kennedy, P.G.E., Oxman, M.N., Seward, J.F., Yamanishi, K., 2015. Varicella zoster virus infection. Nat. Rev. Dis. Prim. 1, 15016. https://doi.org/10.1038/nrdp.2015.16
12. Goswami, Y., Gaurkar, S.S., 2023. Ramsay Hunt Syndrome: An Introduction, Signs and Symptoms, and Treatment. Cureus. https://doi.org/10.7759/cureus.33688
13. Hakami, M.A., Khan, F.R., Abdulaziz, O., Alshaghdali, K., Hazazi, A., Aleissi, A.F., Abalkhail, A., Alotaibi, B.S., Alhazmi, A.Y.M., Kukreti, N., Binshaya, A.S., 2024. Varicella-zoster virus-related neurological complications: From infection to immunomodulatory therapies. Rev. Med. Virol. 34. https://doi.org/10.1002/rmv.2554

14. Kathiresan, D.S., Balasubramani, R., Marudhachalam, K., Jaiswal, P., Ramesh, N., Sureshbabu, S.G., Puthamohan, V.M., Vijayan, M., 2025. Role of Mitochondrial Dysfunctions in Neurodegenerative Disorders: Advances in Mitochondrial Biology. Mol. Neurobiol. 62, 6827–6855. https://doi.org/10.1007/s12035-024-04469-x
15. Liu, X., Chen, Z., 2017. The pathophysiological role of mitochondrial oxidative stress in lung diseases. J. Transl. Med. 15, 207. https://doi.org/10.1186/s12967-017-1306-5
16. Perszke, M., Egierska, D., 2022. Ramsay Hunt syndrome. J. Educ. Heal. Sport 12, 876–882. https://doi.org/10.12775/JEHS.2022.12.09.101
17. Pivovarova, N.B., Andrews, S.B., 2010. Calcium-dependent mitochondrial function and dysfunction in neurons. FEBS J. 277, 3622–3636. https://doi.org/10.1111/j.1742-4658.2010.07754.x
18. Qu, C., Zhang, S., Li, Y., Wang, Y., Peppelenbosch, M.P., Pan, Q., 2019. Mitochondria in the biology, pathogenesis, and treatment of hepatitis virus infections. Rev. Med. Virol. 29. https://doi.org/10.1002/rmv.2075
19. Shapiro, J.S., 2009. Does varicella-zoster virus infection of the peripheral ganglia cause Chronic Fatigue Syndrome? Med. Hypotheses 73, 728–734. https://doi.org/10.1016/j.mehy.2009.04.043
20. Sinha, K., Das, J., Pal, P.B., Sil, P.C., 2013. Oxidative stress: the mitochondria-dependent and mitochondria-independent pathways of apoptosis. Arch. Toxicol. 87, 1157–1180. https://doi.org/10.1007/s00204-013-1034-4
21. Springer, W., Kahle, P.J., 2011. Regulation of PINK1-Parkin-mediated mitophagy. Autophagy 7, 266–278. https://doi.org/10.4161/auto.7.3.14348
22. Wang, A., Xie, W., Zhang, J., 2025. The synergistic role of viral infection and immune response in the pathogenesis of facial palsy. J. Neurovirol. https://doi.org/10.1007/s13365-025-01258-7
23. Wu, X., Gong, L., Xie, L., Gu, W., Wang, X., Liu, Z., Li, S., 2021. NLRP3 Deficiency Protects Against Intermittent Hypoxia-Induced Neuroinflammation and Mitochondrial ROS by Promoting the PINK1-Parkin Pathway of Mitophagy in a Murine Model of Sleep Apnea. Front. Immunol. 12. https://doi.org/10.3389/fimmu.2021.628168
24. Xiao, B., Goh, J.-Y., Xiao, L., Xian, H., Lim, K.-L., Liou, Y.-C., 2017. Reactive oxygen species trigger Parkin/PINK1 pathway–dependent mitophagy by inducing mitochondrial recruitment of Parkin. J. Biol. Chem. 292, 16697–16708. https://doi.org/10.1074/jbc.M117.787739
25. Yang, S., Park, J.H., Lu, H.-C., 2023. Axonal energy metabolism, and the effects in aging and neurodegenerative diseases. Mol. Neurodegener. 18, 49. https://doi.org/10.1186/s13024-023-00634-3
26. Ye, J., Hu, X., Wang, Z., Li, R., Gan, L., Zhang, M., Wang, T., 2023. The role of mtDAMPs in the trauma-induced systemic inflammatory response syndrome. Front. Immunol. 14. https://doi.org/10.3389/fimmu.2023.1164187