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Calcium Signaling in Neuronal and Cardiac Function

Petrica Anaïs*

Department of Clinical Pharmacy, College of Pharmacy, Jouf University, Sakaka 72388, Saudi Arabia

***Corresponding author**: Petrica Anaïs, Department of Clinical Pharmacy, College of Pharmacy, Jouf University, Sakaka 72388, Saudi Arabia, E-mail: Anais.petri@ju.edu.sa

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Introduction

Mitochondria are traditionally regarded as the powerhouses of the cell, generating Adenosine TriphosPhate (ATP) through oxidative phosphorylation to sustain vital biological processes. However, this narrow perspective has expanded dramatically in recent years, revealing mitochondria as highly dynamic and multifunctional organelles that play pivotal roles in metabolism, calcium signaling, apoptosis, innate immunity, and redox balance. Central to their function is the concept of mitochondrial dynamics, which encompasses the continuous processes of fission, fusion, biogenesis, and mitophagy. These mechanisms enable mitochondria to adapt their morphology, distribution, and quality in response to cellular demands and environmental stresses. Mitochondrial dynamics are therefore not mere structural phenomena but are deeply intertwined with cellular homeostasis. Dysregulation of these processes has been linked to a broad spectrum of diseases, including neurodegenerative disorders, metabolic syndromes, cardiovascular conditions, and cancer. Understanding the molecular regulators of mitochondrial dynamics thus provides key insights into both normal physiology and pathological states [1].

Description

Calcium signaling is highly dynamic, relying on finely tuned mechanisms that regulate intracellular Ca^{2+} concentrations. In resting conditions, cytosolic Ca^{2+} is maintained at nanomolar levels, while extracellular and organelle concentrations, particularly in the Endoplasmic or Sarcoplasmic Reticulum (ER/SR), are much higher. This steep gradient enables rapid calcium fluxes upon activation of specific channels [2]. In neurons, calcium influx is central to communication and plasticity. Action potentials depolarize the presynaptic membrane, opening Voltage-Gated Calcium Channels (VGCCs) such as N-type and P/Q-type channels. The resulting Ca^{2+} entry triggers the fusion of synaptic vesicles with the presynaptic membrane, releasing neurotransmitters into the synaptic cleft. This process, known as excitation–secretion coupling, is both highly localized and temporally precise, relying on calcium microdomains near the channels. Postsynaptically, NMDA receptors, a subtype of ionotropic glutamate receptors, allow Ca^{2+} influx during synaptic activity, contributing to Long-Term Potentiation (LTP) and Long-Term Depression (LTD), key mechanisms of synaptic plasticity underlying learning and memory [2].

Calcium signaling in neurons extends beyond synaptic transmission. Activation of metabotropic receptors can stimulate inositol trisphosphate (IP_3) production, leading to calcium release from the ER through IP_3 receptors. Ryanodine receptors (RyRs) also mediate Calcium-Induced Calcium Release (CICR), amplifying signals. These intracellular calcium elevations regulate gene transcription via calcium-dependent transcription factors such as CREB, influencing neuronal growth, differentiation, and survival. Moreover, calcium interacts with calmodulin and downstream kinases, including CaMKII, which is critical for synaptic strengthening and memory consolidation. Dysregulation of neuronal calcium signaling has been implicated in epilepsy, neurodegeneration, and psychiatric disorders. For instance, excessive Ca^{2+} entry through NMDA receptors contributes to excitotoxicity in stroke and Alzheimer's disease, while impaired calcium buffering is associated with Parkinson's disease.

In the heart, calcium signaling governs the rhythmic contraction of cardiomyocytes through excitation contraction coupling. An action potential propagating along the sarcolemma and into the transverse tubules activates L-type calcium channels, permitting a small influx of Ca^{2+}. This triggers a much larger release of calcium from the sarcoplasmic reticulum via ryanodine receptors in a process known as CICR. The resulting transient rise in cytosolic Ca^{2+} binds to troponin C on the actin filaments, enabling cross-bridge cycling between actin and myosin and thus muscle contraction. Relaxation occurs when Ca^{2+} is removed from the cytosol by reuptake into the SR via SERCA pumps, extrusion via the sodium–calcium exchanger (NCX), and buffering by mitochondria. This cycle, repeated with each heartbeat, allows the heart to contract and relax in a coordinated manner [1].

The regulation of cardiac calcium dynamics is essential for maintaining rhythm and force of contraction. Modulation by sympathetic stimulation, through β-adrenergic signaling, enhances L-type calcium channel activity and SERCA function, thereby increasing contractility and heart rate. Conversely, parasympathetic signals dampen calcium influx to reduce cardiac output. Any disruption in this balance can have severe consequences. Abnormal calcium handling underlies arrhythmias, as inappropriate Ca^{2+} release can trigger delayed afterdepolarizations. Impaired SERCA activity contributes to diastolic dysfunction in heart failure, while mutations in RyRs or calcium-handling proteins cause inherited arrhythmia syndromes such as Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) [2].

Conclusion

Calcium signaling stands at the crossroads of neuronal and cardiac physiology, orchestrating processes as diverse as neurotransmission, synaptic plasticity, and heartbeat. Its versatility lies in the ability to generate localized, transient signals as well as global, sustained responses, all governed by a finely tuned network of channels, exchangers, pumps, and sensors. In neurons, calcium dictates communication and adaptation, serving as a molecular substrate for memory and cognition, while in the heart it ensures the precise coupling of electrical activity to contraction.

Dysregulation of calcium signaling carries profound consequences, from neurodegeneration and epilepsy to arrhythmias and heart failure, emphasizing its centrality to health and disease. Advances in molecular biology, imaging, and therapeutics continue to unravel the complexity of calcium dynamics, offering new avenues for intervention. Ultimately, calcium signaling exemplifies how a simple ion can regulate the most intricate functions of life, maintaining balance at the cellular and organismal levels.

Acknowledgement

None.

Conflict of Interest

None.

Reference

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