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From discovering “calcium paradox” to Ca^{2+} /cAMP interaction: Impact in human health and disease

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The hypothesis of the so-called “calcium paradox” phenomenon in the sympathetic neurotransmission has its origin in experiments done in models of neurotransmission since 1970's. Historically, “calcium paradox” originated several clinical studies reporting that acute and chronic administration of L-type Ca^{2+} Channel Blockers (CCBs), drugs largely used for antihypertensive therapy such as verapamil and nifedipine, produces reduction in peripheral vascular resistance and arterial pressure, associated with a paradoxical sympathetic hyperactivity. Despite this sympathetic hyperactivity has been initially attributed to adjust reflex of arterial pressure, the cellular and molecular mechanisms involved in this paradoxical effect of the L-type CCBs remained unclear for four decades. Also, experimental studies using isolated tissues richly innervated by sympathetic nerves showed that neurogenic responses were completely inhibited by L-type CCBs in high concentrations, but paradoxically potentiated in low concentrations, characterized as a “calcium paradox” phenomenon. We discovered in 2013 that this paradoxical increase in sympathetic activity produced by L-type CCBs is due to Ca^{2+} /cAMP interaction. Then, the pharmacological manipulation of this interaction could represent a potential cardiovascular risk for hypertensive patients due to increase of sympathetic hyperactivity. In contrast, this pharmacological manipulation could be a new therapeutic strategy for increasing neurotransmission in psychiatric disorders such as depression, and producing neuroprotection in the neurodegenerative diseases such as Alzheimer's and Parkinson's diseases.

Biography

Leandro Bueno Bergantin received his academic education at UNIFESP-EPM (Brazil) and UAM (Spain) i.e., degree in Biomedicine in 2008, MSc in 2010 and PhD in 2014. His research involves cell signaling mediated by Ca^{2+} and cAMP, skeletal and smooth muscles, peripheral and central nervous systems. His research work solved the enigma of the paradoxical effects produced by L-type Ca^{2+} channel blockers. He is currently pursuing Post-doctoral fellowship (FAPESP) at UNIFESP-EPM.

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