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From discovering "calcium paradox" to Ca2+/cAMP interaction: Impact in depression

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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 Ca2+ 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 Ca2+/cAMP interaction (Bergantin et al., Cell Calcium, 2013; ScienceDirect TOP 25 Hottest Articles - Cell Calcium - TOP 1 July to September 2013/ TOP 5 October to December 2013/ TOP 1 January to December 2013 full year/TOP 6 January to March 2014). 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 (Caricati-Neto et al. 2015, Pharmacol Res Perspectives; Bergantin and Caricati-Neto 2016, Eur J Pharmacol).

Biography

Bergantin received his academic education at UNIFESP-EPM (Brazil) and UAM (Spain): degree in biomedicine (2008), MSc (2010) and PhD (2014). His research involves cell signaling mediated by Ca2+ 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 Ca2+ channel blockers (ScienceDirect TOP 25 Hottest Articles, including TOP 1 positions: 2013 and 2014, Cell Calcium) .

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