Leandro Bueno Bergantin et al., J Neurol Neurophysiol 2016, 7:4 (Suppl)
http://dx.doi.org/10.4172/2155-9562.C1.034



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

**Leandro Bueno Bergantin** and **Afonso Caricati-Neto**
UNIFESP-Escola Paulista de Medicina (EPM), Brazil

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 has received his academic education from UNIFESP-EPM (Brazil) and did his MSc (2010) and PhD (2014) degrees in Biomedicine. 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 Ca2+ channel blockers. He is currently a Post-doctoral fellow (FAPESP) at UNIFESP-EPM.

leanbio39@yahoo.com.br

**Notes:**