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**Treatments for diabetes mellitus type II: new perspectives regarding the possible role of calcium and cAMP interaction**

Diego Soares Carvalho<sup>1,2,1</sup>, Alexandre Aparecido de Almeida<sup>1,3</sup>, Aurélio Ferreira Borges<sup>2</sup>, Diego Vannucci Campos<sup>1</sup>

<sup>1</sup>Departamento de Fisiologia, Universidade Federal de São Paulo (UNIFESP), Brazil.

<sup>2</sup> Instituto Federal de Educação, Ciência e Tecnologia de Rondônia (IFRO), Campus Colorado do Oeste - RO, Brazil.

<sup>3</sup> Instituto Federal Goiano, Campus Ceres, Ceres - GO, Brazil.

professordiegovcampos@gmail.com

**ABSTRACT**

Diabetes mellitus (DM) is among the top ten causes of death worldwide. It is considered to be one of the major global epidemics of the 21<sup>st</sup> century, with a significant impact on public health budgets. DM is a metabolic disorder with multiple etiologies. Its pathophysiology is marked by dysfunction of pancreatic  $\beta$ -cells which compromises the synthesis and secretion of insulin along with resistance to insulin action in peripheral tissues (muscle and adipose). Subjects presenting insulin resistance in DM type 2 often also exhibit increased insulin secretion and hyperinsulinemia. Insulin secretion is controlled by several factors such as nutrients, hormones, and neural factors. Exocytosis of insulin granules has, as its main stimulus, increased intracellular calcium ( $[Ca^{+2}]_i$ ) and it is further amplified by cyclic AMP (cAMP). In the event of this hyperfunction, it is very common for  $\beta$ -cells to go into exhaustion leading to failure or death. Several animal studies have demonstrated pleiotropic effects of L-type  $Ca^{2+}$  channel blockers (CCBs). In animal models of

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<sup>1</sup> **Mailing Address:** Tel:55115576-4848. - Brazil.

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