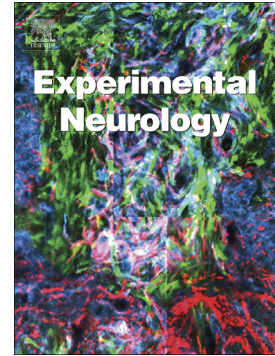


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## Calcium, mitochondrial dysfunction and slowing the progression of Parkinson's disease

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### Abstract

Parkinson's disease is characterized by progressively distributed Lewy pathology and neurodegeneration. The motor symptoms of cPD are unequivocally linked to the degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNc). Several features of these neurons appear to make them selectively vulnerable to factors thought to cause cPD, like aging, genetic mutations and environmental toxins. Among these features, Ca<sup>2+</sup> entry through Cav1 channels is particularly amenable to pharmacotherapy in early stage cPD patients. This review outlines the linkage between these channels, mitochondrial oxidant stress and cPD pathogenesis. It also summarizes considerations that went into the design and execution of the ongoing Phase 3 clinical trial with an inhibitor of these channels – isradipine.

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