



# Can Renalase Enzyme Control the Fate of Parkinson's and Schizophrenia?

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## Editorial

Dopamine is a catecholamine that is involved in the etiopathology of neurodegenerative diseases like Parkinson's and schizophrenia [1,2]. Parkinson's disease develops as a result of dopamine deficiency in the glial beds of the brain [1]. Schizophrenia, on the other hand, is characterized by excessive dopamine [2]. The limited number of drugs used in Parkinson's disease either makes up the dopamine deficiency (L-Dopa), or exercise dopamine-like effects, or prevent the breakdown of dopamine in the brain [1]. Neuroleptics used in the treatment of schizophrenia, on the other hand, act by decreasing the dopamine quantity [3]. In some cases, these neuroleptics reduce the dopamine amounts below the normal physiological level and cause Parkinson's-like symptoms. Thus, dopamine is a catecholamine which is like a double-edged sword.

This editorial hypothesizes that Parkinson's and schizophrenia may be associated with the synthesis and release of the renalase enzyme synthesized in many tissues, including the kidneys and the brain in particular [4]. Renalase is an enzyme that catabolizes catecholamines, including dopamine, epinephrine and norepinephrine [4, 5]. Theoretically, excessive renalase synthesis would lead to more catecholamine destruction, resulting in reduced dopamine and Parkinson's disease. In the reversed situation, when renalase is synthesized below the physiological dose, catecholamines, including dopamine, would be destroyed to a lesser extent, and diseases such as high blood pressure and schizophrenia will develop. Since the formation and destruction of catecholamines depend of the renalase enzyme, designing the drugs used in the treatment of diseases like Parkinson's and schizophrenia in consideration of this situation can produce more effective results. Molecular weight of renalase is 38 kDa [5]. A molecule as large as this cannot pass through the blood-brain barrier. Therefore, renalase blockers administered through the peripheral route (in Parkinson's) cannot be effective in schizophrenia. When the renalase-secreting part of the brain is operated on in Parkinson's disease, renalase synthesis, and therefore renalase-dependent dopamine destruction will be reduced; thus, this can be a new therapeutic alternative in Parkinson's. In the case of schizophrenia, it may be that renalase-synthesizing renalase cells in the brain which atrophy over time cannot produce renalase and the resulting failure of dopamine destruction leads to schizophrenia. A

general evaluation of the data suggests that renalase is involved in the mechanism underlying Parkinson's and schizophrenia, and therefore, its involvement can be considered in the treatment of these diseases.

## References

1. Connolly BS, Lang AE (2014) Pharmacological treatment of parkinson disease: a review. JAMA 311: 1670-1683.
2. Archer T, Ricci S, Garcia D, Ricciardi MR (2014) Neurodegenerative aspects in vulnerability to schizophrenia spectrum disorders. Neurotox Res 26: 400-413.
3. Matute MS, Matute R, Merino P (2016) Design and synthesis of dopaminergic agonists. Curr Med Chem 23: 2790-2825.
4. Hennebray SC, Eikelis N, Socratous F, Desir G, Lambert G, et al. (2010) Renalase, a novel soluble FAD-dependent protein, is synthesized in the brain and peripheral nerves. Mol Psychiatry 15: 234-236.
5. Xu J, Li G, Wang P, Velazquez H, Yao X et al. (2005) Renalase is a novel, soluble monoamine oxidase that regulates cardiac function and blood pressure. J Clin Invest 115: 1275-1280.

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