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Renal dopamine receptors, oxidative stress, and hypertension

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Dopamine, synthesized in the kidney, independent of renal nerves, is important role in the regulation of fluid and electrolyte balance and blood pressure. Physiological concentrations of dopamine counteract oxidative stress but high concentrations (≥10 μM) can lead to oxidative stress due to auto/enzymatic oxidation. Lack of any of the five dopamine receptor subtypes (D1R, D2R, D3R, D4R, and D5R) results in hypertension. D1R, D2R, and D5R are important in the maintenance of a normal redox balance. In the kidney, the antioxidant effects of these receptors are caused by direct inhibition of pro-oxidant enzymes, specifically, NADPH oxidase, and stimulation of anti-oxidant enzymes (e.g., paraoxonase 2 [PON2], heme oxygenase [HO]) which can also inhibit NADPH oxidase activity. D2R increases the expression of endogenous anti-oxidants, such as Parkinson protein 7 (PARK7 or DJ-1), PON2, and HO-2. DJ-1 exerts its antioxidant effect, in part, via Nrf2 and PON2 exerts its antioxidant effect, in part, via sestrin2. The D5R decreases NADPH oxidase activity, via the inhibition of PLD2, and increases the expression of HO-1, another antioxidant. D1R inhibits NADPH oxidase activity via PKA and PKC cross-talk. The role of D3R and D4R in the regulation of oxidative stress is not clear. The hypertension and salt sensitivity in D3R-/- or D4R-/- mice are not associated with increased renal oxidative stress. There is, however, oxidative stress in resistance vessels of D4R-/- mice. Therefore, the protective role of dopamine on oxidative stress depends on not only its concentration but also to the specific dopamine receptor subtype and its organ expression.

Biography

Pedro A Jose received his MD degree, magna cum laude, from the University of Santo Tomas, Philippines and received his PhD degree in Physiology from Georgetown University. He has published more than 330 articles; 4 as covers of scientific journals and 5 as subject of editorial commentaries. His research has been recognized (2007 Ernest H. Starling Lecture, 2003 Lewis K. Dahl Memorial Lecture, and NIH MERIT Award). Deciphering the role of variations of the GRK4 in the causation of human essential hypertension was cited by the Director of the NHLBI for its FY 2004 Budget Justification to the US Congress.

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