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Early Diabetic Nephropathy - Saying no to NO?

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Diabetic nephropathy is the leading cause of end stage renal failure in the Western world; however advancements in therapies preventing the pathogenesis of this disease process have been limited. The nitric oxide (NO) system has been attributed to cause glomerular hyperfiltration in the early stages of diabetic nephropathy, a state that has been implicated in the pathogenesis and progression of the disease. The literature regarding the NO system has been contradictory and studies have reported evidence proposing alternative factors. This includes aspects related to superoxide, insulin and tubuloglomerular feedback that must all be acknowledged before considering the notion of targeting this system. The idea of targeting the NO system and preventing progression of diabetic nephropathy is an enticing concept, however, practically it may present with complications to the systemic vasculature and 'saying no to NO' may be a concept too complex to be feasible. Nevertheless, recent findings involving C-peptide and Peroxisome proliferator-activated receptor alpha (PPARα) agonists have provided insight into novel approaches that alter components of the NO system and may exert beneficial effects on diabetic nephropathy. This review aims to explore the physiology of the NO system and address whether targeting this system is a viable option in the future of treating diabetic nephropathy.

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