Research Article

Chronic Administration of Oil Palm (Elaeis guineensis) Leaves Extract Attenuates Hyperglycaemic-Induced Oxidative Stress and Improves Renal Histopathology and Function in Experimental Diabetes

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Oil palm (Elaeis guineensis) leaves extract (OPLE) has antioxidant properties and because oxidative stress contributes to the pathogenesis of diabetic nephropathy (DN), we tested the hypothesis that OPLE prevents diabetes renal oxidative stress, attenuating injury. Sprague-Dawley rats received OPLE (200 and 500 mg kg⁻¹) for 4 and 12 weeks after diabetes induction (streptozotocin 60 mg kg⁻¹). Blood glucose level, body and kidney weights, urine flow rate (UFR), glomerular filtration rate (GFR), and proteinuria were assessed. Oxidative stress variables such as 8-hydroxy-2'-deoxyguanosine (8-OHdG), glutathione (GSH), and lipid peroxides (LPO) were quantified. Renal morphology was analysed, and plasma transforming growth factor-beta1 (TGF-β1) was measured. Diabetic rats demonstrated increase in blood glucose and decreased body and increased kidney weights. Renal dysfunction (proteinuria, elevations in UFR and GFR) was observed in association with increases in LPO, 8-OHdG, and TGF-β1 and a decrease in GSH. Histological evaluation of diabetic kidney demonstrated glomerulosclerosis and tubulointerstitial fibrosis. OPLE attenuated renal dysfunction, improved oxidative stress markers, and reduced renal pathology in diabetic animals. These results suggest OPLE improves renal dysfunction and pathology in diabetes by reducing oxidative stress; furthermore, the protective effect of OPLE against renal damage in diabetes depends on the dose of OPLE as well as progression of DN.

1. Introduction

Diabetic nephropathy (DN) is a major microvascular complication of diabetes and is the leading cause of chronic renal failure and end-stage renal disease (ESRD) worldwide. Traditionally, DN has been described as a glomerular disease with the following stages: glomerular hyperfiltration, incipient nephropathy, microalbuminuria, overt proteinuria, and ESRD [1]. The morphological changes that occur in DN include glomerular hypertrophy, thickening of glomerular basement membrane, and mesangial expansion. These morphological changes probably give rise to proteinuria, renal dysfunction, and eventually to development of glomerulosclerosis and tubulointerstitial fibrosis [2]. At present no adequate treatment is available for diabetic renal injury [3–7] and thus other agents that can affect the molecular mechanisms which contribute to the pathogenesis of DN are essential.

Studies in both humans and animal models clearly implicate the contribution of oxidative stress to the pathogenesis of DN [8–11]. Chronic hyperglycaemia is probably the most important factor in the generation of oxidative stress. Oxidative stress has an important role in the pathogenesis