

14th World Congress on
Endocrinology & Diabetes

November 21-22, 2018 | Paris, France

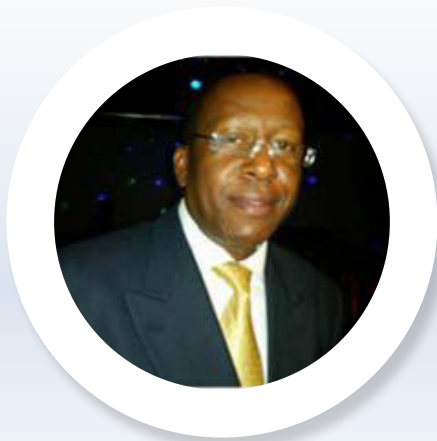
Heme oxygenase is a molecular switch that ameliorates cardio-renal complications in diabetes

Impaired insulin signaling and deregulated glucose metabolism are associated with cardiac and renal dysfunction. Our recent studies indicate that upregulating heme-oxygenase (HO) potentiates insulin signaling and improve glucose metabolism in different animal models of type-1 and type-2 diabetes This was accompanied by: (i) the attenuation of pro-inflammatory cytokines (TNF- α , IL-6, IL-1 β) and chemokines (MCP-1 and MIP-1 α); (ii) the suppression of transcription factors/mediators of oxidative stress (NF- κ B, activating-protein (AP)-1, AP-2, and c- Jun-N-terminal-kinase and 8-isoprostane); (iii) the potentiation of fundamental proteins implicated in the insulin signal transduction pathway like IRS-1, PI3K and PKB; (iv) the reduction of insulin/glucose intolerance (IPITT); (v) the enhancement of insulin sensitivity, and (vi) the inability of insulin to enhance GLUT4 was overturned. Correspondingly, renal histological lesions such as glomerulosclerosis, tubular necrosis, tubular vacuolization, interstitial macrophage-M1 infiltration were attenuated. Similarly, pro-fibrotic/extracellular-matrix proteins like collagen and fibronectin that deplete nephrin, an important transmembrane protein which forms the scaffolding of the podocyte slit-diaphragm allowing ions to filter but not proteins were reduced, while proteinuria/albuminuria decreased and creatinine clearance increased suggesting improved renal function. Similarly, HO reduced cardiac hypertrophy, abated collagen deposition in cardiomyocytes and suppressed of left ventricular longitudinal muscle fiber thickness. These data suggest that HO may be considered an important switch that can be potentiated to rescue kidney damage in diabetes.

Biography

Joseph Fomusi Ndisang is an Associate Professor in the University of Saskatchewan College of Medicine, Department of Physiology. He received postdoctoral training in Physiology at the University of Saskatchewan College of Medicine from 2000-2005. He obtained a PhD in Pharmacology & Toxicology from the University of Florence, Italy, 2000. He obtained a Doctor of Pharmacy degree from University of Florence, Italy in 1995. He has received several distinguished awards and distinctions including: (i) Fellow of the Canadian Cardiovascular Society (FCCS) in 2016; (ii) Fellow of the American Heart Association (FAHA) in 2011; (iii) Fellow of the International College of Angiology (FICA) in 2007; (iv) Young Investigator Award by International College of Angiology (2007); (v) Young Investigator Award by the American Society of Pharmacology & Experimental Therapeutics-Division for Drug Discovery, Development & Regulatory Affairs (2005); (vi) Young Investigator Award by the Society of Experimental Biology and Medicine (2005); (vii) Caroline tum Suden/Frances A Hellebrandt Professional Opportunity Award for Meritorious Research by the American Physiological Society (2005); and (viii) Recognition Award for Meritorious Research by a Young Investigator by the American Physiological Society (2004). Top 5% of cited authors in journals of Biology and Biochemistry in 2011, by Thomson-Reuters. Currently, Dr. Ndisang is an Editor for Frontiers in Bioscience (impact factor 3.8) and Executive Guest Editor for Current Medicinal Chemistry (impact factor 3.7) He has published more than 65 -full length manuscripts in peer-reviewed journals and more than 80 abstracts.

joseph.ndisang@usask.ca



Joseph F Ndisang

University of Saskatchewan College of Medicine, Canada

Notes: