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Major Professor: James Ntambi

Degree Objective: Ph.D Endocrinology and Reproductive Physiology

Background:

2012	MSc in Clinical Chemistry, Rochester Institute of Technology
2008	Teaching Assistant, Department of Basic Medical Sciences, College of medicine King Saud bin Abdul-Aziz University for Health Sciences, Riyadh, Saudi Arabia
2008	Completed Internship (Clinical Labs Rotation/ 1 Year), King Khalid University Hospital
2007	Bachelor in Clinical Laboratory Sciences, King Saud University Riyadh, Saudi Arabia

Current Research Project:

Obesity is a major health problem around the world. According to a recent report, more than one third of adults are overweight in the United States. One of the major factors that contribute to increased body weight is the genetic control of lipogenesis. Stearoyl CoA Desaturase (SCD1) is a key player in lipogenesis; it catalyzes the rate limiting step in the production of monounsaturated fatty acids (MUFA's). We have previously shown that mice with liver specific SCD-1 (LKO) deficiency showed normal lipogenesis when fed with high fat diet (HFD), however they fail to induce de-novo lipogenesis when fed with high carbohydrate diet (HCD). mTOR mediated activation of hepatic de-novo lipogenesis showed significant upregulation of SCD-1, suggesting significant relationship between mTOR and SCD1. We designed an experiment to study the impact of SCD1 deficiency on mTOR signaling. Interestingly, SCD1 deficient mice fed HCD showed significant induction in the phosphorylation of both Akt and mTOR compared with LOX mice. Akt and mTOR activation was further confirmed by determining the phosphorylation status of the downstream targets such as GSK3, Rp6, and 4EBP1, which showed higher phosphorylation levels. Induction of de novo lipogenesis despite activated Akt and mTOR was not evident in HCD fed LKO mice. In contrast LKO mice fed HFD showed almost similar induction of Akt, mTOR and de novo lipogenesis when compared to LOX mice. The failure of activated AKT and mTOR to induce lipogenesis upon HCD could be attributed to induced mRNA expression and plasma levels of Fibroblast growth factor

21(FGF21) in LKO mice compared to LOX mice. In vivo deoxy-glucose experiment showed that LKO mice have higher glucose uptake in brown adipose tissue, white adipose tissue and liver suggesting that increased liver FGF21 secretion might increase glucose uptake in these tissues. Furthermore, feeding LKO mice with triolein supplemented HCD reduced FGF21 expression and plasma levels. These results suggest that a decrease in MUFAs/or an increase in SFAs, in response to SCD1 deficiency, activates mTORC1 and subsequently increases FGF21 expression. Increased FGF21 expression is expected to decrease hepatic lipogenesis, increase glucose uptake and increase energy expenditure in BAT that could explain decreased adiposity observed in SCD1 deficient mice.

Publication:

Mohammad Imran Khan, **Ahmed Al Johani**, Abid Hamid, Bushra Ateeq, Vaqar Mustafa Adhami, Rahul K. Lall, Suvasmita Rath, Mario Sechi, Imtiaz Ahmad Siddiqui, Bilal Bin Hafeez, Ajit Kumar Verma, Thomas C. Havighurst, Wei Huang, James M. Ntambi, and Hasan Mukhtar. Pro-Proliferative Role of Adaptor Protein GRB10 in Prostate Carcinoma.JBC (**under review**).

Presentations:

Poster Presentation: **Aljohani AM**, Khan IM, Mukhtar H, Ntambi JM. (2014) Hepatic Stearoyl-CoA desaturase-1 deficiency activates mTOR. 11th Annual Molecular Pharmacology Research Training Symposium. Madison, WI

Selected for oral presentation: **Aljohani AM**, Khan IM, Mukhtar H, Ntambi JM. Hepatic stearoyl-CoA desaturase deficiency suppresses Akt-mTORC1 mediated de novo lipogeneisis. Endocrinology-Reproductive Physiology Training Program 2014 Annual Scientific Symposium.

Oral presentation: **Aljohani AM**, Khan IM, Mukhtar H, Ntambi JM. Hepatic stearoyl CoA desaturase-1 deficiency induces FGF21-dependent glucose uptake in adipose tissue on high carbohydrate diet. Endocrinology-Reproductive Physiology Seminar December 11, 2011

Oral presentation: **Aljohani AM**, Khan IM, Mukhtar H, Ntambi JM. Hepatic stearoyl CoA desaturase-1 deficiency induces FGF21 expression.

Endocrinology-Reproductive Physiology Seminar January 21, 2016

Oral presentation: **Aljohani AM**, Khan IM, Mukhtar H, Ntambi JM. Hepatic stearoyl CoA desaturase-1 deficiency promotes FGF21 expression.

Endocrinology-Reproductive Physiology Seminar September 29, 2016