ABSTRACT

The rising worldwide ingestion of fructose enriched diets has coincided with the global epidemics of diabetes mellitus, obesity and ectopic lipid deposition in other organs such as the liver and pancreas. The fructose diet induced lipid deposition in the liver is termed non-alcoholic fatty liver disease (NAFLD) and often coexists with the lipid deposition in the pancreas which is known as non-alcoholic fatty pancreas disease (NAFPD). The prevalence of NAFLD and NAFPD is estimated at 25% and 16% in the general population respectively, and is rising steadily. Posing huge health and economic challenge, and yet no standard approved treatment guidelines. The few, synthetic drugs being used to treat these diseases are associated with significant adverse side effects like constipation, hepatitis, and pancreatitis. Hence, the current study explored the potential of the phytochemical quercetin in preventing the occurrence of NAFLD and NAFPD, as well as the metabolic dysfunction associated with a high fructose feeding in growing female Sprague Dawley rats. Thirty seven, 21 day old, female Sprague Dawley rats were randomised into one of four experimental groups and fed for with their respective diets and treatments. Control group (C) received SRC (Standard rat chow), tap water, and plain gelatine cubes daily; quercetin group (Q) received SRC, tap water, and quercetin cubes (100 mg/kg body weight) daily; fructose group (F) received SRC, and 20% fructose solution (FS) to drink *ad libitum,* and plain gelatine cubes daily; fructose plus quercetin group (F + Q) received SRC, 20% FS *ad libitum* and quercetin cubes (100 mg/kg body weight) daily. At the end of 12 weeks feeding and treatments, the animals were terminated; blood and tissues samples were collected.

The growth performance was unaffected ($P > 0.05$), however, fructose consumption significantly ($P < 0.05$) reduced food consumption and increased overall total caloric intake, visceral fat mass, liver mass, hepatosomatic index (HSI), hepatic lipid yield, and induced NAFLD. However, the fructose feeding did not induce NAFPD or insulin resistance and except for the reduced high density lipoprotein cholesterol (HDL-C) level, all other circulating metabolic substrates were unaffected ($P > 0.05$). Quercetin did not improve the visceral fat mass, liver mass and HSI, but improved the HDL-C level, hepatic steatosis, inflammation and fibrosis significantly ($P < 0.05$). There was a trend towards the reduction of hepatic lipid yield below the 10% mean value cut off mark applicable for the diagnosis of NAFLD. Red blood cell indices, markers of liver,

and renal functions were unaffected by either the fructose solution or quercetin treatment, and no adverse effects were observed. Therefore, the use of quercetin as a preventative intervention against high fructose induced NAFLD should be further examined as a natural alternative in curtailing the rising prevalence of dietary fructose induced NAFLD globally.