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Amounts of Hepatic Glucose and Lipids Induced by Honey Feeding in Wistar Rats

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ABSTRACT

Recent findings indicated that a high fructose diet induces dyslipidaemia and accumulation of lipid in the liver. Honey contains high amount of fructose and it's uses is currently being promoted in diverse ways. Whether honey consumption elicits similar dyslipidaemic effects is clearly documented. Therefore, the aim of this study is to determine liver glucose and lipids induced by honey in rats. A total of 49 Wistar Albino rats, of both gender weighing between 60 – 110g were divided into 7 groups (n=7 rats/group). The control rats (group A) were fed with 100% grower s' mash. The experimental groups (Group B-G) were respectively given feed containing 20%, 30%, 40% honey and fructose quantities equivalent to amounts in 20%, 30% and 40% honey, for four weeks. Results showed that there were increases (p<0.05) in hepatic glucose, triacylglycerol and total cholesterol concentrations in rats fed with honey or fructose compared with the control group. Fructose at higher amounts significantly (p<0.05) reduced hepatic HDL-cholesterol but increased (p<0.05) LDL-cholestgerol. Honey increased (p<0.05) hepatic HDL-cholesterol at 40%, but LDL-cholesterol was minimally elevated. Honey and fructose feeding alike increase the amounts of glucose, triacylglycerol and LDL-cholesterol, but lower HDL-cholesterol level in the liver. The results present a measure of fatty liver and associated fibrotic risk in both groups of experimental animals. The increased hepatic glucose level suggests a degree of oxidative stress. Further studies involving the metabolic activities of the liver during honey feeding are desirable in order to fully document the observed risks.

Key words: Honey, Lipids, Glucose, Liver, Fructose.

INTRODUCTION

Honey is a popular viscous sweetener and a common household product used throughout the World. Popularity comes not only in being a natural sweetener but also because of its several proven or unproven benefits (Bansal, *et al*., 2005). Honey is a natural substance produced by honey bees, *Apis mellifera*, from the nectar of blossom or from exuduates of trees and plants.

The composition of honey is rather variable and primarily depends on the floral source. Honey contains at least 181 substances (Chow, 2002). It is a super saturated solution of sugars, mainly composed of fructose (38%) and glucose (31%). It also contains minerals, proteins, free amino acids, enzymes and vitamins (Perez, 2002).

The physiological and health effects of honey have been related to its antibacterial activity (Bogdanor, 1997), antioxidant capacity (Gheldof, *et al*., 2003), antimutagenic, antitumour and anti-inflammatory activity (Molan, 2001), gastroenterology and cardiovascular effects. Yaghobi, *et al*. (2008), reported that honey ameliorates cardiovascular risk factors in healthy individuals and in patients with elevated risk factors. Yoghoobi, *et al*. (2008) and Al-Waili (2004) have reported the effects of honey on total cholesterol, low density cholesterol (LDL-C), high density cholesterol (HDL-C), triacylglycerol (TAG), fasting blood glucose and C-reactive protein (CRP). Busserolles, *et al*. (2002), reported that substitution of honey for refined carbohydrates protects rats from hypertriglyceridemic and prooxidative effects of fructose. Munstedt, et al. (2009) also observed similar effect of honey on serum cholesterol and lipid values. However, this study compares the amounts of glucose and lipids in the livers of rats fed with honey and fructose.

MATERIAL AND METHODS

Experimental animals

Both male and female Wistar rats weighing between 60g-110g and obtained from the Animals House, Faculty of Basic Medical Sciences, Delta State University, Abraka, Nigeria, were used for the experiment. They were fed on growers' mash purchased from Top Feeds, Sapele, Delta State, and given water *ad libitum*. The animals were housed in metal cages under controlled condition of 12 hours light/12 hours dark cycle.

Experiment design

A total of 49 rats were used for the experiment. The rats were fed growers' mash that was either sugar free or contained 20%, 30%, 40% honey or mixed sugar (fructose and glucose) equivalent to that in 20%, 30% and 40% honey for four weeks. The rats were divided into 7 groups of 7 rats per groups.

Preparation of liver tissue homogenate

At the completion of the 4 – week feeding period, the rats were fasted overnight, sacrificed under anaesthesia (chloroform) and their livers were excised and collected for biochemical analysis. One gramme (1g) of wet liver tissue was homogenized in 9.0ml of normal saline. The supernatant obtained after centrifuging was kept frozen until required for assay which was performed within 72 h.

Biochemical Assays

The amount of hepatic triacyglycerol was determined by the Trinder reaction (McGowan, *et al*., 1983). Liver total cholesterol and HDL-cholesterol levels were estimated colorimetrically by the methods of Beaumont *et al*. (1972) and Castelli *et al*. (1977), respectively. Hepatic LDL-cholesterol content was calculated by the Friedewald formula (Wang, *et al*., 1996). The concentration of liver glucose was assessed by the glucose oxidase method (Trinder, 1969).

All the reagent test kits used for these assays were purchased from TECO Diagnostics, Anheim CA, USA.

Statistics

All data were analyzed using ANOVA and group means were compared by Duncan's multiple range. Value of $p<0.05$ were considered significant.

RESULTS

The biochemical parameters measured which include liver glucose, liver triacylglycerol, total cholesterol, high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) are shown in Tables 1 and 2 respectively.

The results in Table 1, indicate that there were significant decreases ($p<0.05$) in the hepatic glucose, triacylglycerol and total cholesterol concentrations in rats fed with 100% feed (control) compared with the other six experimental groups.

Fructose at higher amounts significantly ($p<0.05$) reduced hepatic HDL-cholesterol but increased ($p<0.05$) LDL-cholesterol. Honey increased ($p<0.05$) hepatic HDL-cholesterol at 40%, but LDL-cholesterol levels were minimally elevated (Table 2).

DISCUSSION

Honey is a biological product with very complex chemical composition (Busserolles, *et al*., 2002). It is a popular viscous sweetener and a common household product used through out the world. From this research, high amounts of honey and fructose feeding caused significant increase in hepatic glucose level when compared with the control value. The present experiment confirms the hypertriglyceridaemic reaction induced by dietary fructose (Table 1). It is noteworthy that substituting honey for refined carbohydrates lowers the triacylglycerol levels. Honey reduced total cholesterol and increased high density lipoprotein cholesterol (HDL-C) in the liver tissue. This experiment is in agreement with previous studies (Yoghoobi, *et al*., 2008; Al-Waili, 2004). Low density lipoprotein cholesterol levels were increased in the liver of experimental rats feed honey ($p>0.05$) and fructose ($p<0.05$) compared with control (Table 2).

Honey and fructose feeding alike increase the amounts of glucose, triacylglycerol and LDL-cholesterol but lower HDL-cholestgerol levels in the liver. The results present a measure of fatty liver

Table 1: Liver levels of glucose, triacylglycerol and total cholesterol in control and experimental rats

Groups	Glucose (mmol/L)	TAG (mmol/L)	Total
A	0.84±0.16	0.62±0.04	5.73±0.56
B	1.23±0.28	0.68±0.03	5.90±0.24
C	1.97±0.44	0.73±0.02	6.71±0.54
D	1.54±0.09	0.78±0.05	6.71±0.37
E	2.18±0.31	0.74±0.05	7.05±0.46
F	1.86±0.12	0.78±0.06	7.08±0.64
G	1.85±0.28	0.83±0.07	7.09±0.37

Values are expressed as mean ± SD for n=7 rats/groups
Group 1: Treated as control (100% feed)
Group 2: Treated with 20% honey, 80% feed
Group 3: Treated with 30% honey, 70% feed
Group 4: Treated 40% honey, 60 feed
Group 5: Treated with fructose/glucose equivalent to amounts in 20% honey
Group 6; Treated with fructose/glucose equivalent to amounts in 30% honey.
Group 7: Treated with fructose/glucose equivalent to amounts in 40% honey.

Table 2: Changes in hepatic HDL and LDL-cholesterol levels in control and experimental rats

Groups	HDL Cholesterol (mmol/L)	LDL-Cholesterol (mmol/L)
A (100% Feed)	4.82±0.35	0.63±0.04
B (20% Honey)	4.93±0.24	0.66±0.05
C (30% Honey)	5.45±0.43	0.09±0.08
D (40%) Honey)	5.48±0.28	0.95±0.07
E(Fructose/Glucose 20%)	4.82±0.46	1.89±0.12
F(Fructose/Glucose 30%)	4.63±0.31	2.10±0.16
G(Fructose/Glucose 40%)	4.47±0.32	2.24±0.12

Values are expressed as mean ± SD for n=7 rats/groups
Group 1: Treated as control (100% feed)
Group 2: Treated with 20% honey, 80% feed
Group 3: Treated with 30% honey, 70% feed
Group 4: Treated 40% honey, 60 feed
Group 5: Treated with fructose/glucose equivalent to amounts in 20% honey
Group 6; Treated with fructose/glucose equivalent to amounts in 30% honey.
Group 7: Treated with fructose/glucose equivalent to amounts in 40% honey.

and associated fibrotic risk in both groups of experimental animals. The increased hepatic glucose level suggests a degree of (oxidative) stress. Further studies involving the metabolic activities of the liver during honey feeding are desirable in order to fully document the observed risks.

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