COFFEE ATTENUATES INSULIN RESISTANCE IN RATS FED ON HIGH-SUCROSE DIET



MORAKINYO AO¹, ADEKUNBI DA², ADEGOKE OA¹

¹Department of Physiology, College of Medicine, University of Lagos, Nigeria.

²Department of Physiology, Benjamin S. Carson (Snr) School of Medicine, Babcock University, Ilishan Remo, Nigeria

SUMMARY

Several epidemiological evidence indicate that consumption of coffee is associated with a lower risk of type 2 diabetes mellitus; however, there is dearth of experimental data to support these observations. Given that associations do not necessarily infer causality; the present study investigate the impact of coffee (300 mg/kg BW) by oral gavage on normal and high sucrose-diet (HSD) fed (30 % w/v) rats. HSD rats were insulin resistant and had significantly elevated levels of insulin, MDA, triglyceride, LDL while HDL level, SOD and GSH activities were significantly reduced. In contrast, coffee consumption significantly up-regulate glucose tolerance, insulin sensitivity, HDL level, SOD and GSH activities while MDA, triglyceride, LDL levels were reduced. These findings suggest that coffee attenuates glucose intolerance, insulin insensitivity, lipid disorders, peroxidation and oxidative stress in sucrose-induced insulin resistant rat.



INTRODUCTION

Coffee is probably the most frequently ingested beverage worldwide.

The numerous beneficial health effects of coffee consumption have received considerable scientific attention (Ranheim and Halvorsen, 2005).

Epidemiological evidence indicates that consumption of coffee is consistently associated with a lower risk of T2DM (Bidel et al 2006).

Since associations do not necessarily infer causality; experimental study using a representative diabetic animal model is importantly meaningful.

A small number of these studies have been performed (Yamacuchi et al, 2010; Matsuda et al, 2011).

No report of its effects on diet-induced diabetic model and diabeticinduced oxidative stress.

Figure 2: Glucose response curves during an ITT in control and experimental rats after 12 weeks of treatment.

Table 1: Insulin level and Area under Curve (AUC) for OGTT and ITT after 12 weeks of treatment.

	ND	ND+COF	HSD	HSD+COF
INSULIN	7.69±0.22	8.63±0.53	12.25±0.51*	9.21±0.66 [#]
AUC _{OGTT}	1017±59.9	906±48.4	2016±119.2*	1132±64.5 [#]
AUCITT	414±29.1	424±30.2	536±36.4*	389±26.3 [#]

Table 2: Cholesterol, Triglyceride, HDL and LDL levels after 12 weeks of treatment. All units in nmol/L.

	ND	ND+COF	HSD	HSD+COF
тс	1.80±0.10	1.71±0.11	2.20±0.14	2.14±0.11
TG	0.35±0.03	0.32±0.06	1.43±0.32*	0.44±0.06 [#]
HDL	0.89±0.06	1.19±0.08	069±0.02*	1.10±0.06 [#]

In the present study, we employed sucrose-induced diabetic rats to investigate the effects of coffee on glucose regulation, T2DM, and its antioxidant potential under diabetic condition.

METHODS

Male Sprague-Dawley rats (120-150g) were used.

- Group 1: normal diet (ND).
- Group 2: ND supplemented with coffee, 300mg/kg BW (ND+COF).
- Group 3: high sucrose-diet (HSD).
- Group 4: HSD supplemented with coffee, 300mg/kg BW (HSD+COF).

Free access to HSD (30 % w/v) as drinking water; coffee solution administered by oral gavage (Ribeiro et al, 2005).

OGTT and ITT (Sanjay et al, 2005); Serum insulin (Latha & Pari 2004); TC, TG, HDL and LDL (Padee et al, 2010); MDA, GSH and SOD (Morakinyo et al 2011) were determined.

Data shown as mean±SEM, compared by ANOVA followed by SNK's multiple comparison test. *P < 0.05 vs. ND; #P < 0.05 vs. HSD.



Table 3: MDA, SOD and GSH after 12 weeks of treatment. MDA (nmol/ml), SOD (mmol/ml) and GSH (umol/ml)

	ND	ND+COF	HSD	HSD+COF
MDA	17.3±1.41	18.6±1.74	37.4±3.59*	21.4±2.36 [#]
SOD	5.45±0.38	5.91±0.72	1.63±0.01*	4.38±0.23 [#]
GSH	0.62±0.04	0.66±0.04	0.24±0.01*	0.53±0.03 [#]

CONCLUSION

Coffee consumption attenuates the severity of glucose intolerance, insulin insensitivity, lipid abnormalities and oxidative stress induced by HSD.

The anti-diabetic effect of coffee solution is possibly related to its strong anti-oxidant potential. The need to identify the active component of coffee and the exact mechanism of action also merits further investigation.







Figure 1: Glucose response curves during OGTT after 12 weeks of treatment. Each point represents mean \pm SEM, n=6.

Bidel S et al (2006) Diabetologia 49: 2618-2626. Ranheim T and Holversen B (2005) Mol Nuti Food Res, 49: 272-294. Matsuda Y et al (2011) Bio Sci Biotechnol Biochem, 75: 2309-15. Ribeiro RT et al (2005) Diabetologia 48: 976-983. Padde P et al (2010) J Ethnopharmacol, 132:491-496.

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