

Anti-adipogenic Effects of a Diet High in Resistant Starch

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INTRODUCTION

The long term effectiveness of low-fat diets on obesity is debatable [1] and alternative approaches are being considered, including the intake of low glycaemic index (GI) foods. GI is a measure of the postprandial increase in blood glucose and lipid levels following food consumption. The beneficial effects of a low GI diet is unclear, although one thought is these diets tend to contain high amounts of resistant starch which is not digested in the small intestine and so, does not give rise to high postprandial glucose levels. Induction of high blood glucose levels as following intake of foods of high GI status, leads to hyperinsulinaemia as the body secretes insulin to restore normal glucose levels. However, hyperinsulinaemia is linked to the 'metabolic syndrome' - insulin resistance, hyperlipidemia, hypertension and visceral obesity. In this study, the effect of diets containing different amounts of resistant starch was studied on adiposity and intrahepatocellular lipid (IHCL) levels by ¹H MRS in a murine model.

METHODS

Animals and Treatment: Forty mice (C57BL/6, 3 weeks old, Harlan UK) were obtained and whole body ¹H MRS performed following a 16h fast. Based on adiposity measurements by MRS, the mice were allocated into groups of five with similar adiposities for housing in individual cages. Mice were then maintained for 8 weeks on either a low resistant starch diet (LRS, 6 cages) or a high resistant starch diet (HRS, 4 cages). The LRS and HRS diets were similar except the former contained AMIOCA, and the latter, Hi-Maize starch: Hi-Maize is a slow glucose releasing starch. ¹H MRS was also performed at weeks 4 and 8 post-dietary intervention and *in vivo* localised ¹H MRS of the liver performed only at the latter time-point for a number of the animals (HRS, 2 cages and LRS, 4 cages). Weekly bodyweights and food intakes were also recorded.

¹H MRS: Anaesthesia was induced and maintained by inhalation of 1-2% isoflurane/oxygen mix. Following a 16h fast, whole body ¹H MRS spectra, to assess adiposity, were obtained on a Varian Inova 4.7T system (Palo Alto, USA): TR 20s, 45° pulse and 4 averages. The total percentage adiposity was calculated by multiplying the lipid peak by 0.38 to correct for the ratio of total body water compared to total fat free mass [3]. For localised ¹H MRS of the liver, data was collected from a 3x3x3mm voxel using PRESS: TR 10s, TE=9ms and 64 averages. Values are quoted as mean ± SEM.

RESULTS AND DISCUSSION

Both groups of mice maintained for 8 weeks on the LRS (high-GI) and HRS (low-GI) diets had similar bodyweights throughout the time-course (Fig. 1). However, assessment of total percentage adiposity by whole body ¹H MRS, indicated that despite similar weight gains between the two groups, the weight gained by the LRS group arises from deposition of adipose fat rather than lean tissue mass (Fig. 2). Furthermore, the similarities in bodyweights between the two groups occurred despite the significantly lower intake of food by the mice maintained on the LRS diet (Fig. 3). It is apparent that the degree of tissue deposition between the LRS and HRS groups is similar but in the latter, adipose tissue deposition is increased at the expense of lean tissue deposition. Further, IHCL levels are also elevated in the mice maintained on the LRS diet. The LRS diet leads to hyperinsulinaemia, promoting carbohydrate oxidation at the expense of fatty acid oxidation, thereby encouraging fat storage in adipose tissue and hepatic triglyceride synthesis [4], and explains the increased adiposity and higher IHCL levels in the mice maintained on the LRS diet.

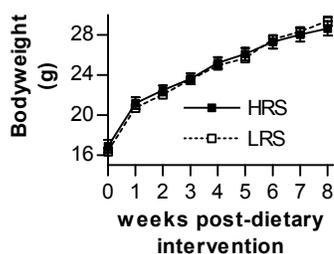


Fig. 1 – Effect of LRS and HRS diets on weekly bodyweights.

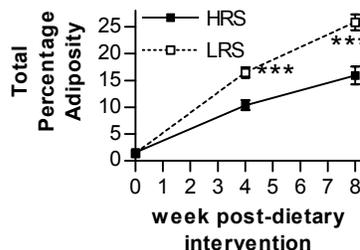


Fig. 2 - Effects of LRS and HRS diets on total percentage adiposity.

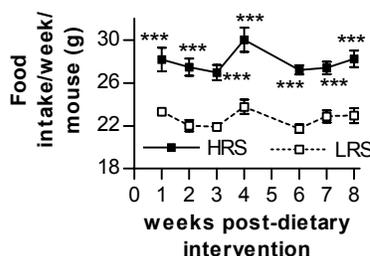


Fig. 3 – Influence of LRS and HRS diets on food intake.

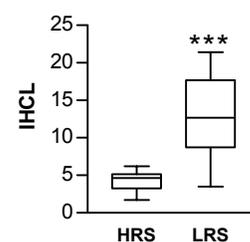


Fig. 4 – Effect of LRS and HRS diets on IHCL.

CONCLUSION

Maintenance for 8 weeks on either the LRS (high-GI) or HRS (low-GI) diets lead to similar weight gain but differences in whole body adiposity. Coupled with the elevation in IHCL levels, it is apparent that LRS diets leads to perturbation of fatty acid metabolism. The results of this study may help to explain some of the beneficial effects of low GI reported in humans.

REFERENCES

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