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Influence of salt on glycaemic response to carbohydrate loading

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Abstract

The effect of dietary salt on glycaemic responses to different test meals was investigated. Eight healthy male volunteers ate four test meals on consecutive mornings and in random order; the meals were 50 g carbohydrate taken as a 20% glucose solution or as boiled macaroni with and without supplementation with 6 g salt. In contrast with other reports, no significant differences in peak plasma glucose concentrations or areas under the plasma glucose curves could be established.

These findings do not support a beneficial effect of salt restriction on glycaemic control in diabetes.

Introduction

The glycaemic response to standard test meals of various foods varies considerably in both normal volunteers and diabetics.^{1,2} Thorburn *et al* recently reported that adding salt to two common starchy foods resulted in an increase of the postprandial plasma glucose and insulin responses.³ A possible effect of dietary salt on the digestion of starch or absorption of glucose was postulated. This observation was even more interesting in the light of the observed

association between plasma insulin concentrations and blood pressure. Both Berglund *et al* and Lucas *et al* found higher insulin concentrations in obese hypertensive patients than in normotensive subjects with the same body mass index.^{4,5} Also in non-diabetic normotensive subjects significant associations of systolic and diastolic blood pressure with insulin concentrations have been observed even after allowing for adiposity.⁶ The possible influence of salt on glucose and insulin responses might therefore be implicated in the effect of dietary salt restriction on blood pressure in essential hypertension.

We have studied the plasma glucose response to a moderate amount of salt added to two foods containing 50 g carbohydrate—namely, a readily absorbed glucose solution and boiled macaroni.

Subjects and methods

Eight healthy normotensive volunteers with a normal body mass index took a test meal on four consecutive mornings after an overnight fast. Meals were allocated at random according to a Latin square model and comprised 50 g carbohydrate as either 71 g macaroni boiled for 10 minutes (intrinsic salt content 3.6 mg) or 250 ml 20% glucose with and without the addition of 6 g sodium chloride. The salt supplement was the only additive allowed. Zero time was taken as the time that eating started, and the meal had to be finished in 10 minutes.

Blood samples were drawn from an indwelling antecubital venous cannula for measurement of glucose concentration in the fasting state and every 15 minutes thereafter until 180 minutes after eating was begun. Plasma glucose concentrations were measured by a glucose oxidase method (Yellow Springs glucose analyser, Ohio, USA).

Results are expressed as medians and ranges. Incremental areas under the three hour glucose curves above fasting values were calculated. The Wilcoxon test for paired observations was used for assessing differences between salted and unsalted meals. The 95% confidence intervals for the

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eight subjects were calculated as the mean of differences $\pm 2.36 \times$ standard error.

Results

All subjects had normal fasting plasma glucose concentrations (table I). Peak plasma glucose concentrations after the glucose solutions whether supplemented or not with salt were higher than those after the macaroni meals ($p < 0.05$ in both cases). No significant effect on plasma glucose responses was observed after adding salt to the two test meals (table I). Similarly, no differences in the areas under the plasma glucose curves were discernible (table II).

Discussion

In contrast with Thorburn *et al.*,³ we found no significant difference in glycaemic responses to test meals with and without added salt. The median peak plasma glucose values were almost identical for each test meal with and without the addition of salt. Though there was a trend towards an increase in the glucose absorption rate after the glucose solution with added salt, this did not reach significance owing to the considerable variability in glucose responses. On the other hand, the opposite trend was seen after the macaroni meal, suggesting that adding salt to a carbohydrate meal is unlikely to have a substantial influence on the glycaemic response. As the rate of ingestion of the carbohydrate load was standardised and no significant differences were found in the glycaemic responses to salted and unsalted test meals at the various

TABLE I—Fasting and peak plasma glucose concentrations after test meals in the eight subjects

	Median (range) fasting plasma glucose (mmol/l)	Median (range) peak plasma glucose (mmol/l)	p Value; 95% confidence interval
Glucose solution	4.6 (4.1-4.9)	7.8 (5.7-8.5)	0.401 (NS); -0.53 to 1.03
Glucose solution with salt	4.6 (4.1-4.9)	7.6 (6.3-9.0)	
Macaroni	4.6 (4.2-5.0)	6.1 (5.6-7.0)	0.726 (NS); -0.84 to 0.69
Macaroni with salt	4.6 (4.5-4.9)	6.5 (4.6-7.5)	

TABLE II—Areas under plasma glucose curves before and after adding salt to the two test meals

	Median (range) area under plasma glucose curve (mmol min/l)	p Value; 95% confidence interval
Glucose solution	57.8 (-63.00 to 220.50)	0.484 (NS); -49.05 to 101.71
Glucose solution with salt	103.5 (-163.00 to 223.50)	
Macaroni	93.4 (-12.00 to 151.50)	0.263 (NS); -69.46 to 25.46
Macaroni with salt	60.5 (-88.50 to 154.50)	

time intervals we did not think it necessary to assess plasma insulin concentrations.^{7,8} In these healthy volunteers no differences were expected, as neither insulin sensitivity nor β cell function was influenced during these acute carbohydrate loading experiments. As expected, we confirmed the lower peak plasma glucose concentrations after the macaroni meal as compared with glucose.

This short term study does not support a beneficial effect of salt restriction on glycaemic control in diabetes. Nevertheless, the reported relation between insulin concentrations and blood pressure warrants further study.

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Delayed cerebellar ataxia: a new complication of falciparum malaria?

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Abstract

Twelve cases of an unusual phenomenon of ataxia were investigated in otherwise well, conscious patients recovering from a febrile attack of presumed falciparum malaria. The ataxia occurred as the fever was subsiding, usually after an afebrile period of two to four days. The delay between onset of fever and the ataxia was three to four weeks. Peripheral blood of all the patients contained gametocytes of *Plasmodium falciparum*, and

in some cases ring stages. The ataxia was most noticeable in the legs and the clinical picture suggested selective impairment of the cerebellar system. Signs of improvement appeared in a few weeks but complete recovery took one to four months.

The most likely pathogenic mechanism of the ataxia in these cases was an immune reaction triggered by the malaria parasite and affecting the cerebellum or its connections, or both.

Introduction

Neurological manifestations of falciparum malaria range from coma¹ to muscular paralysis.² This paper draws attention to a unique ataxic syndrome seen recently in Sri Lanka, which may prove to be a new complication of the disease. The outstanding feature was

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