CLINICAL RESEARCH

Salt and the glycaemic response

ANNE W THORBURN, JENNIE C BRAND, A STEWART TRUSWELL

Abstract

The possibility that salt increases plasma glucose and insulin responses to starchy foods was investigated. Six healthy adults took four morning test meals randomly: 50 g carbohydrate as cooked lentils or white bread, with or without 4·25 g of added salt (an amount within the range of salt found in a meal). When salt was added to the lentils the incremental area under the three hour plasma glucose curve was significantly greater than that for lentils alone (43·2 mmol.min/l *v* 11·1 mmol.min/l (778 mg.min/100 ml *v* 200 mg.min/100 ml)). When salt was added to bread the peak glucose concentration was significantly higher than that for unsalted bread (6·96 mmol/l *v* 6·35 mmol/l (125 mg/100 ml *v* 114 mg/100 ml)), and this was followed by relative hypoglycaemia. Plasma insulin concentrations at 45 minutes were higher after a meal of salted lentils and salted bread than after the unsalted foods ($p<0·05$). The high insulin concentration after salted bread was sustained for one hour after the meal, thus the mean area under the three hour curve was 39% greater than that for unsalted bread ($p< 0·05$).

Salt may increase the postprandial plasma glucose and insulin responses to lentils and bread by accelerating the digestion of starch by stimulating amylase activity or accelerating small intestinal absorption of the liberated glucose, or both. The findings of this preliminary study, if confirmed by others, would support the recommendation that diabetics, as well as the general population, should reduce their intake of salt.

Introduction

The size and duration of the increase in plasma glucose concentration after the consumption of standardised meals of various foods rich in carbohydrates vary considerably in both normal adults[1] and diabetics.[2,3] These differences may result from effects on the rate of gastric emptying, digestion, and absorption and on the chemical nature of the constituent monosaccharides. Viscous dietary fibre,[4] phytic acid,[5] protein in the food,[6] the type of starch,[7] and the physical form of the food (raw or cooked, whole or ground)[8] may all affect postprandial plasma glucose concentrations, and we have found that highly processed forms of staple starchy foods give high glycaemic responses.[9]

In an analysis of published reports and our own studies on carbohydrate digestion we found a positive correlation between the sodium content of food and the rate of starch digestion or glycaemic response.

In this study we tested the effect of the moderate addition of salt on the plasma glucose and insulin responses to two foods containing 50 g carbohydrate. The foods studied were boiled lentils, a slowly absorbed carbohydrate food,[10] and white bread, a rapidly absorbed staple food.[1]

Methods

Six healthy subjects (three men and three women) took part in the experiments with salted and unsalted lentils and six healthy subjects (four men, two women) took part in the experiments with salted and unsalted bread. Five subjects participated in all four experiments. All subjects were non-smokers aged 21-32 with normal body mass index. The study was approved by the medical ethics review committee of the University of Sydney.

Brown lentils, cooked by boiling in excess unsalted water for 30 minutes, and commercial white sliced wheat bread ("Tip Top" 1·3% weight/weight sodium chloride) were studied. The lentils were freshly cooked and the bread freshly baked on the morning of the experiment; the lentils were eaten whole and the bread as slices. The starch and sugar contents of the bread and cooked lentils were measured by isolating sugars by methanolic extraction, hydrolysing the starch with acid,[11] and quantifying the hexoses with anthrone reagent.[12] The tests were performed during the morning after the subjects had fasted for 12 hours. The following four test meals were consumed randomly: 50 g carbohydrate as cooked lentils (200 g) or bread (98 g) with or without 4·25 g of added sodium chloride (AR grade). Two cups of white tea (400 ml) containing 80 ml of milk (0·06 g intrinsic sodium) were consumed at the same time. No butter was taken with the bread. Zero time was taken as the time that eating started, and the meal was consumed within 15 minutes.

Blood samples were drawn with an indwelling antecubital venous cannula for the measurement of glucose and insulin concentrations in the fasting state and 15, 30, 45, 60, 90, 120, 150, and 180 minutes after eating. Blood samples were collected into tubes containing edetic acid, and the plasma was stored at −80°C. Plasma glucose concentrations were measured by the glucose hexokinase method using the glucose "rapid" centrifichem system (Roche Diagnostica, Basle, Switzerland) and were analysed single blind. Plasma insulin concentrations were measured using a double antibody radioimmunoassay (Bio-RIA, Montreal, Canada).

Results are given as mean (SEM). Incremental areas under the three hour plasma glucose and plasma insulin curves above fasting values were

Human Nutrition Unit, University of Sydney, New South Wales 2006, Australia

ANNE W THORBURN, BSC, research scholar
JENNIE C BRAND, PHD, lecturer in human nutrition
A STEWART TRUSWELL, MD, FRCP, Boden professor of human nutrition

Correspondence to: Dr Brand.

calculated. Peak plasma glucose and insulin concentrations were compared using the same fasting baseline for salted and unsalted meals. Two tailed Student's *t* test for paired data was used for statistical comparisons between salted and unsalted meals, assuming glucose and insulin responses are normally distributed.

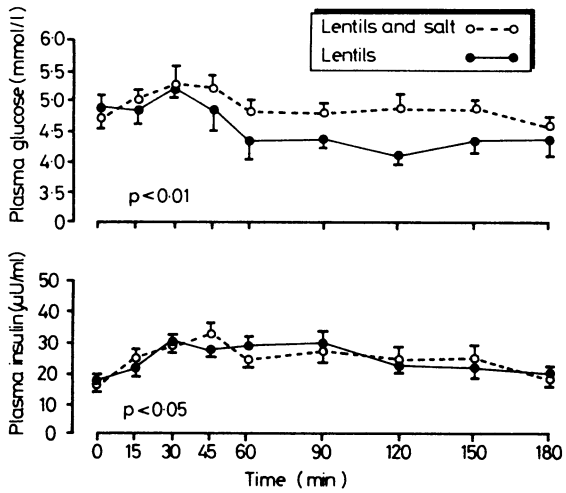

FIG 1—Mean (SEM) plasma glucose and insulin concentrations in six healthy subjects after eating 50 g carbohydrate as lentils with or without 4·25 g added salt.

Conversion: SI to traditional units—Glucose: 1 mmol/l≈18 mg/100 ml.

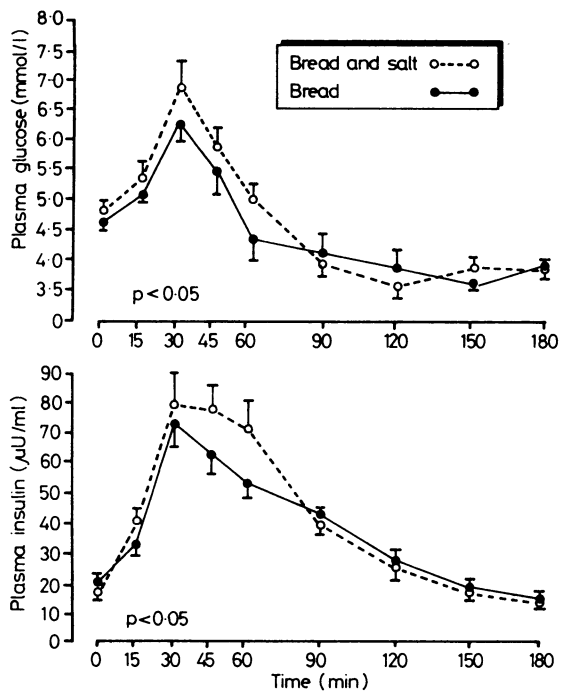

FIG 2—Mean (SEM) plasma glucose and insulin concentrations in six healthy subjects after eating 50 g carbohydrate as bread with or without 4·25 g added salt.

Conversion: SI to traditional units—Glucose: 1 mmol/l≈18 mg/100 ml.

Results

All subjects had normal fasting plasma glucose (4·78 (0·11) mmol/l (86 (2) mg/100 ml)) and insulin (17·1 (1·2) μU/ml) concentrations. After consumption of the unsalted boiled lentils the plasma glucose concentration gave a small peak that gradually declined to below the fasting concentration (fig 1). When salt was added to the lentils mean plasma glucose concentrations were higher throughout compared with those for unsalted lentils, and the differences were significant at 120 minutes ($p<0·01$). The area under the plasma glucose curve after the consumption of salted lentils was four times greater (43·2 mmol.min/l *v* 11·1 mmol.min/l (778 mg.min/100 ml *v* 200 mg.min/100 ml); $p<0·05$) than after unsalted lentils (table I). Plasma insulin concentrations rose only slightly after the consumption of lentils and were similar with or without salt, but the mean concentration was 22% higher ($p<0·05$) 45 minutes after salted lentils (fig 1).

The peak plasma glucose concentration after consumption of the bread was higher than after lentils (fig 2). When salt was added to bread the peak plasma glucose concentration at 30 minutes was significantly greater (9%) than that for unsalted bread ($p<0·05$). By 120 minutes the plasma glucose curve with added salt showed relative hypoglycaemia. Plasma insulin concentrations rose higher after bread than lentils and were raised 15, 30, 45, and 60 minutes after the consumption of salted bread (fig 2). The insulin concentration at 45 minutes was 29% higher ($p<0·05$) after salted bread than after unsalted bread. The mean (SEM) area under the three hour plasma insulin curve after salted bread was 39% greater ($p<0·05$) than after unsalted bread (table I).

Mean (SEM) area under three hour plasma glucose and plasma insulin curves in six subjects after eating 50 g carbohydrate as lentils or bread with or without 4·25 g added salt

	Area under plasma glucose curve (mmol.min/l)	p value	Area under plasma insulin curve (μU.min/ml)	p value
Lentils	11·1 (6·5)	<0·05	1695 (322)	NS
Lentils plus salt	43·2 (8·5)		1883 (422)	
Bread	59·0 (14·9)	NS	3395 (352)	<0·05
Bread plus salt	66·7 (11·7)		4716 (644)	

Conversion: SI to traditional units—Glucose: 1 mmol/l≈18 mg/100 ml.

Discussion

We have shown that moderate addition of salt increases the postprandial plasma glucose and insulin responses to two common foods. The amount of salt used in this study, 4·25 g (73 mmol sodium), represents about one third of the daily sodium intake of many people in Western countries.[13]

The two most likely ways that salt increases the postprandial plasma glucose and insulin responses are by accelerating the digestion of starch or accelerating the absorption of glucose, or both. Fifty years ago Clifford[14] showed that sodium chloride accelerated in vitro hydrolysis of pure raw starch by salivary and pancreatic amylases; maximum activation occurred between 3 mmol/l and 300 mmol/l sodium chloride (17·5 mg/100 ml and 1755 mg/100 ml). Chloride rather than sodium may have been the responsible factor as chloride ions are potent activators of amylase.[14] In vivo salivary amylase digests about half the dietary starch[15] and its concentration can be increased by salt stimuli.[16] Salt may therefore have an effect on starch digestion by increasing both the amount of salivary amylase and its activity as well as the activity of pancreatic amylase.

Sodium facilitates the absorption of glucose in the small intestine.[17] The added salt to carbohydrate ratio in our in vivo study (1:12) was similar to the salt to glucose ratio found in oral rehydration fluid (1:8).[18] This fluid replaces the loss of electrolytes and water that occurs during diarrhoea by using sodium to help the transport of glucose by the sodium potassium dependent adenosine triphosphatase in the small intestine. This mechanism may also facilitate the transport of glucose, which results from starch digestion, across the epithelium; rice powder may be used in oral rehydration mixtures in place of glucose. We therefore suggest that sodium may increase the glycaemic response to starches as well as to sugars.

Coulston *et al* showed that there is considerable disagreement about the plasma glucose response to specific foods.[19] For example, Jenkins *et al* found a plasma glucose response to carrots almost equivalent to glucose,[1] whereas Ionescu-Tirgoviste *et al* found a much lower response.[2] Jenkins *et al*, unlike other workers, added 2 g salt into the cooking water of the dry grains, legumes, and vegetables that were studied. Such variations in salt content may account for some of the discrepancies.

Recently, the importance of measuring plasma insulin concentra-

tions after the consumption of different carbohydrate foods has been emphasised as the insulin responses can vary considerably even when glucose responses are similar and might be of more clinical significance in the non-diabetic population.[19] In our study the sustained high insulin concentration after salted bread was probably responsible for the steep drop in plasma glucose concentration after 30 minutes (fig 2). Our findings of the small rise in plasma glucose concentration after the consumption of lentils and the fall below fasting concentrations (fig 1) have been seen after the consumption of other test meals of lentils and soya beans.[20]

The findings of this preliminary study, if confirmed by others, including those of diabetics, would support the recommendation that diabetics,[21-23] as well as the general population,[24-26] should reduce their intake of salt. The differences seen after the consumption of unsalted and salted foods justify a controlled trial of the use of salt restriction in a group of stable diabetic patients as an additional way to lower postprandial plasma glucose concentrations.

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Assessment of oral candidiasis in patients with respiratory disease and efficacy of a new nystatin formulation

P J THOMPSON, H J WINGFIELD, R F COSGROVE, B O HUGHES, M E TURNER-WARWICK

Abstract

Fifty consecutive patients with respiratory diseases who developed oropharyngeal candidiasis were assessed clinically and microbiologically before and after seven days' treatment with nystatin suspension or pastilles (a new formulation). In 45 patients in whom microbiology yielded positive results there was frequent associated use of oral corticosteroids, antibiotics, sedatives, and inhaled corticosteroid, while in a few patients atropine analogues may have predisposed to infection. Dentures were worn by 32 of the infected patients. Concomitant treatment of dentures in chronically infected patients appeared to improve the therapeutic response. Pastilles and suspension were equally efficacious both clinically and microbiologically.

The potential for enhanced drug delivery to the oropharynx suggests that nystatin pastilles may be useful in patients in whom poor compliance seems likely.

Professorial Unit, Department of Medicine, Cardiothoracic Institute, London
P J THOMPSON, FRACP, lecturer
M E TURNER-WARWICK, PHD, FRCP, professor

Brompton Hospital, London
H J WINGFIELD, chief medical laboratory scientific officer, microbiology department
B O HUGHES, principal pharmacist, pharmacy department

The Squibb Institute for Medical Research, Moreton, Wirral, Merseyside
R F Cosgrove

Correspondence to: Dr P J Thompson, University Department of Medicine, Queen Elizabeth II Medical Centre, Nedlands 6009, Western Australia.

Introduction

Oral candidiasis is common in patients with respiratory disease and predisposing states have been described.[1] Despite repeated treatment chronic infection occurs, and nystatin pessaries have therefore been prescribed to achieve greater contact time between drug and oropharynx.[2,3] Pessaries have an unpleasant taste, however, so that compliance with treatment may be poor. To overcome these problems a pastille of nystatin has been formulated. We have assessed its clinical efficacy and carried out a clinical audit of 50 patients with respiratory diseases who developed oral candidiasis.

Patients and methods

After removing antifungal agents from the wards we investigated 50 consecutive patients who had been prescribed this treatment. All were questioned about symptoms, primary chest disease, and current drug treatment. Other predisposing factors such as diabetes, malignancy, recent infection, and dentures were recorded.[1] Chronic infection was defined as four or more infections in the past 12 months. Six clinical signs of oral