Source and Amount of Carbohydrate Affect Postprandial Glucose and Insulin in Normal Subjects

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ABSTRACT To determine if source and amount of carbohydrate affected postprandial glucose and insulin responses, seven nondiabetic subjects consumed 0, 25, 50, 75 or 100 g carbohydrate (total carbohydrate minus total dietary fiber) portions of barley, spaghetti, bread or potato. By ANOVA, both source and amount of carbohydrate had significant effects on incremental response areas for capillary glucose (P = 0.001), plasma glucose (P = 0.01) and plasma insulin (P = 0.03), but there was no source x amount interaction. By regression analysis, source of carbohydrate explained a similar amount of the variability of glucose and insulin responses, 46–64%, as the amount of carbohydrate, 47–57%. Together, carbohydrate source and amount accounted for 85–94% of the variability of mean glucose and insulin responses. We conclude that, for individual foods with different glycemic indices, both source and amount of carbohydrate influence the postprandial glucose and insulin responses of nondiabetic subjects. J. Nutr. 126: 2798–2806, 1996.

INDEXING KEY WORDS:
• humans • glucose • insulin • glycemic index • carbohydrate

The current position of the American Diabetes Association (1994) regarding dietary carbohydrate is that first priority should be given to the amount of carbohydrate consumed rather than its source. Nevertheless, numerous studies have shown that postprandial plasma glucose and insulin responses are influenced by both the amount of carbohydrate consumed and its source (Brand et al. 1985, Crapo et al. 1977, Gannon et al. 1989, Jenkins et al. 1981, Kreuzowski et al. 1987, Rasmussen 1993a and 1993b, Wolever et al. 1994). The effect of source of carbohydrate on blood glucose responses is indicated by the glycemic index (GI) which is defined as the glycemic response of a 50-g carbohydrate portion of a food expressed as a percentage of that to an equal carbohydrate portion of a standard, usually white bread or glucose (Wolever et al. 1991). The effects on plasma glucose and insulin of varying the amount of a single carbohydrate source have been examined (Gannon et al. 1989, Jenkins et al. 1981, Rasmussen 1993a and 1993b, Wolever et al. 1994) as have the effects of varying the source of carbohydrate at a single dose level (Brand et al. 1985, Crapo et al. 1977, Jenkins et al. 1981, Kreuzowski et al. 1987, Wolever et al. 1994). However, few, if any, studies have determined the effects of various amounts of different foods on postprandial glucose and insulin responses. Thus, the main purpose of this study was to determine whether the source and amount of carbohydrate from single foods affected postprandial glucose and insulin responses in nondiabetic subjects. The secondary purposes were to determine 1) how much of the variability of glucose and insulin responses could be explained by amount and source of carbohydrate and 2) if the relative glycemic effects of foods were the same when measured in capillary blood or venous plasma.

SUBJECTS AND METHODS

Subjects and protocol. Seven normal university students (3 female, 4 male; age 25.1 ± 0.8 y; body mass

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4 Abbreviations used: GI, glycemic index; GR, glucose relative response; IDDM, insulin-dependent diabetes mellitus; II, insulin-nemic index; IR, insulin relative response; NIDDM, noninsulin-dependent diabetes mellitus.
TABLE 1
Composition of test foods

<table>
<thead>
<tr>
<th>Food</th>
<th>Weight</th>
<th>Energy</th>
<th>Fat</th>
<th>Protein</th>
<th>Carbohydrate¹</th>
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<tr>
<td>Barley</td>
<td>30.0</td>
<td>485</td>
<td>0.1</td>
<td>2.4</td>
<td>25.0</td>
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<tr>
<td></td>
<td>60.0</td>
<td>975</td>
<td>0.2</td>
<td>4.8</td>
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</tr>
<tr>
<td></td>
<td>120.0</td>
<td>1950</td>
<td>0.3</td>
<td>9.8</td>
<td>100.0</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>33.8</td>
<td>505</td>
<td>0.6</td>
<td>4.0</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td>67.5</td>
<td>1015</td>
<td>1.2</td>
<td>8.1</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td>135.0</td>
<td>2030</td>
<td>2.4</td>
<td>16.2</td>
<td>100.0</td>
</tr>
<tr>
<td>Bread</td>
<td>50.0</td>
<td>505</td>
<td>0.2</td>
<td>3.9</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td>100.0</td>
<td>1015</td>
<td>0.3</td>
<td>7.9</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td>150.0</td>
<td>1520</td>
<td>0.5</td>
<td>11.8</td>
<td>75.0</td>
</tr>
<tr>
<td></td>
<td>200.0</td>
<td>2025</td>
<td>0.7</td>
<td>15.8</td>
<td>100.0</td>
</tr>
<tr>
<td>Potato</td>
<td>30.5</td>
<td>495</td>
<td>0.2</td>
<td>1.8</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td>60.9</td>
<td>990</td>
<td>0.4</td>
<td>3.5</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td>121.8</td>
<td>1980</td>
<td>0.9</td>
<td>7.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

¹ Moisture, fat, protein and ash were measured using standard methods (AOAC 1980). Total carbohydrate was calculated by difference. Total dietary fiber was measured by the AOAC method (Prosky et al. 1984). Carbohydrate was taken to be total carbohydrate minus total dietary fiber.

index 23.8 ± 1.0 kg/m²) were studied on 14 separate occasions after 12-h overnight fasts. On each occasion, an indwelling catheter for taking blood samples was placed into a forearm vein and kept open by flushing with 2–3 mL of normal saline after each blood sample. The saline was cleared before each blood sample by withdrawing and discarding 1 mL. After venous and finger-prick capillary blood samples were obtained in the fasting state, subjects were asked to consume a test meal within 15 min and remain seated for the next 2 h. Additional simultaneous venous and finger-prick capillary blood samples were taken 15, 30, 45, 60, 90 and 120 min after subjects started to eat the test meal. Each subject took all 14 different test meals at approximately weekly intervals. On the first occasion, the subjects chose to drink either 250 mL water or either coffee or tea with 30 mL 2% milk and aspartame sweetener as desired. Four subjects chose coffee with milk and sweetener, one chose coffee alone and two chose water; the drink chosen by each subject was the same for each subsequent test. Subjects tested three different doses [25, 50 and 100 g carbohydrate (total carbohydrate minus total dietary fiber) portions] of pearled barley, white spaghetti and instant mashed potato and four different doses of white bread [25, 50, 75 and 100 g carbohydrate portions, Table 1]. In addition, on one occasion, nothing but the standard drink was consumed. The tests were grouped into five blocks consisting of the drink only (1 test), barley (3 tests), spaghetti (3 tests), bread (4 tests) and potato (3 tests). The order of the blocks was randomized and the order of the tests within each block alternated between ascending or descending order of carbohydrate dose. The protocol was approved by the Human Subjects Review Committee of the University of Toronto, and all subjects gave informed consent.

Capillary blood samples were taken into fluro-oxalate tubes and frozen at −20°C prior to glucose analysis within 24 h. Venous blood was taken into fluro-oxalate tubes [3-mL grey top Vacutainer, Beckton-Dickinson, Rutherford, NJ] and placed on ice prior to centrifugation to remove the plasma. Plasma aliquots were frozen at −20°C prior to glucose and insulin analysis. Capillary blood glucose and plasma glucose were measured using an automatic analyzer (2300 Stat Glucose Analyzer, Yellow Springs Instruments, Yellow Springs, OH). Insulin was measured at the Banting and Best Diabetes Core Laboratory, University of Toronto, by RIA [Pharmacia Insulin RIA, Dorval, Quebec].

Test meals. Bread was baked in the diet kitchen in loaves containing 250 g carbohydrate. For each loaf, 334 g all-purpose flour [Maple Leaf Mills, Toronto, Ontario], 7 g sucrose, 6 g yeast, 4 g salt and 250 mL water were placed into an automatic bread maker [model SD-BT2P, Matsushita Electric of Canada, Mississauga, Ontario] which mixed, kneaded, and baked the bread over a 4-h period. Loaves were cooled at room temperature for 1 h, weighed, cut into portions containing 50 g carbohydrate [discarding crust ends], packed into plastic bags and frozen. Prior to consumption, bread was warmed in a microwave oven. Barley [McNair Products, Montreal, Quebec], spaghetti [Primo Foods, Woodbridge, Ontario] and instant mashed potato [Oetker, Mississauga, Ontario] were cooked on the morning of the test according to package directions. Barley was boiled for 20 min in 3 volumes of salted water [all water was absorbed]. Spaghetti was boiled for 20 min in an excess of salted water and drained. Instant potatoes were mixed with boiling water in the ratio of 5:1 water/potato by weight.

Data analysis. Results are expressed as means ± SEM. Incremental areas under the glucose and insulin curves, ignoring area beneath the fasting level, were calculated geometrically [Wolaver et al. 1991]. Glucose responses in capillary blood and venous plasma were compared by paired t test and linear regression analysis. To determine the effect of amount and source of carbohydrate, glucose and insulin responses to the 25, 50 and 100 g carbohydrate doses, which were common to all four foods, were assessed by three-way ANOVA for the effects of source, amount, source × amount interaction, and subject.

To determine how much of the variance of mean glucose and insulin responses could be explained by amount and source of carbohydrate, regression analysis was performed first with linear models and then with curvilinear models to establish the best fit class of model. The dependent variables (y-axis) were the mean glucose and insulin response areas for the 14 different test meals. The independent variables (x-axis) were the amount of carbohydrate (total carbohydrate minus dietary fiber) in grams and the source of carbohydrate,
expressed as the glycemic index (GI; the 0-g carbohydrate test was assigned a GI of 0). For source of carbohydrate, a linear model approached the maximum explained variance. However, for amount of carbohydrate, an exponential association model was considerably better. For each model, the correlation coefficient, \( r \), was determined, and \( r^2 \) gave the proportion of the variance of the dependent variable (glucose or insulin response) which was explained by the independent variable (grams of carbohydrate or GI).

The GI is defined as the incremental area under the glucose response curve for a 50-g carbohydrate portion of a food expressed as a percentage of that after 50 g carbohydrate from white bread is taken by the same subject. To obtain more reliable values, we recommend that the mean of at least three white bread tests in each subject be used for calculating the GI [Wolever et al. 1991]. To achieve this in this study, subjects took four different doses of white bread, and curves were fitted by nonlinear regression for each subject's blood glucose, plasma glucose and plasma insulin responses to the 0, 25, 50, 75 and 100 g carbohydrate portions of white bread. Each subject's mean glucose and insulin response to 50 g carbohydrate from bread was calculated from these curves. The capillary blood glucose, plasma glucose and plasma insulin responses for each of the 14 different test meals for each subject were expressed as a percentage of the respective mean responses to 50 g carbohydrate from white bread (calculated from the fitted curves). The resulting values are termed "relative responses." For each food, a curve was fitted to the mean relative responses for the 0, 25, 50 and 100 g doses, and the GI was taken to be the value predicted by the curve for 50 g carbohydrate.

To determine how much of the variance of glucose and insulin responses was explained by both source and amount of carbohydrate, a necessary assumption was that the relative differences between foods are the same at any dose of carbohydrate. To test this assumption, the areas under the capillary glucose response curves for the 25-g carbohydrate dose of the foods for each subject were expressed as a percentage of that subject's mean response to 25 g carbohydrate from white bread calculated from that subject's regression of glucose response on bread carbohydrate dose. The same was done for the 50- and 100-g carbohydrate doses of foods and bread. The resulting values were assessed by repeated measures ANOVA examining for the main effects of amount (25, 50 or 100 g) and source of carbohydrate.

The results of this analysis and the method by which nonlinear regression analysis was used to determine regression equations describing the relative glucose and insulin responses as a function of carbohydrate amount and GI are described in the Results section. Finally, the regression equations developed were used to calculate predicted values for the glucose and insulin responses. The correlation between these predictions and the observed mean glucose and insulin responses indicated the extent to which both source and amount of carbohydrate determined postprandial glucose and insulin responses.

When ANOVA was performed, individual means were compared using the Newman-Kuels method to adjust for multiple comparisons (Snedecor and Cochran 1980). Regression analysis and curve fitting were done using GraphPad Prism (GraphPad Software, San Diego, CA). Differences were considered significant if \( P < 0.05 \).

### RESULTS

The test meals were well received but, due to a large volume, some subjects took longer than 15 min to consume the following test meals (times of subjects taking > 15 min in parentheses) 50 g carbohydrate barley (23 and 35 min); 100 g carbohydrate barley (23, 26, 35 and 40 min); 100 g carbohydrate spaghetti (25 and 30 min); 100 g carbohydrate potato (29, 30, 35 and 35 min).

The mean glucose and insulin concentrations after the 0, 25, 50 and 100 g doses of carbohydrate are shown in Figure 1. Capillary glucose concentrations were lower than those for plasma glucose. However, the overall mean capillary glucose response area, 132 ± 19 [mmol·min]/L, was significantly greater than the mean plasma glucose response area, 121 ± 16 [mmol·min]/L \( (P = 0.042) \). There was a significant relationship between the mean capillary and plasma glucose response areas \( (y\text{-intercept forced through }0, r = 0.975) \), and the slope, 0.90 ± 0.025, was significantly different from 1 \( (P = 0.002; \text{Fig. 2}) \). By contrast, the mean relative glucose response measured in capillary blood, 92.0 ± 13.1%, was not different from that measured in plasma glucose, 93.8 ± 14.0%, and the slope of the regression line \( (forced through the origin, r = 0.975) \), 0.94 ± 0.027, was not significantly different from 1 \( (\text{Fig. 2}) \).

Both amount and source of carbohydrate had significant effects on the areas under the curve for capillary and plasma glucose and plasma insulin \( (\text{Table 2}) \). As the amount of carbohydrate increased from 25 to 100 g, the mean capillary glucose response for all four foods increased from 88 ± 11 to 187 ± 16 [mmol·min]/L \( (P = 0.001) \), the plasma glucose response increased from 86 ± 11 to 158 ± 18 [mmol·min]/L \( (P = 0.012) \) and the plasma insulin response increased from 6.2 ± 1.4 to 21.3 ± 4.3 [mmol·min]/L \( (P = 0.024) \). The overall mean glucose and insulin responses for the 25, 50 and 100 g carbohydrate doses were highest for potato and lowest for barley, the differences being 192 ± 17 vs. 86 ± 10 [mmol·min]/L \( (P = 0.001) \) for capillary glucose, 169 ± 16 vs. 73 ± 10 [mmol·min]/L \( (P = 0.006) \) for plasma glucose and 20.6 ± 4.2 vs. 5.2 ± 8.9 [mmol·min]/L for plasma insulin \( (P = 0.032) \). There was no food × dose interaction for the capillary glucose, plasma glucose or plasma insulin response areas \( (\text{Table 2}) \).
The relationships between amount of carbohydrate and glucose and insulin responses were not linear. The model which appeared to fit the data best was an exponential association of the form \( y = A(1 - e^{-Bx}) + C \) where \( y \) is the response, \( x \) is the amount of carbohydrate, and \( A, B \) and \( C \) are constants. The correlations between amount of carbohydrate and glucose and insulin responses were statistically significant \( (P = 0.007 \text{ to } P = 0.003) \), but, because the different foods produced different responses for any given amount of carbohydrate, the data were widely scattered about the regression lines. Amount of carbohydrate accounted for only 57% of the variance of mean capillary glucose responses, 50% of the variance of mean plasma glucose responses and 47% of the variance of mean plasma insulin responses [Fig. 3].

The GI values of the foods were linearly related to the glucose and insulin responses and the correlation coefficients were statistically significant \( (P = 0.007 \text{ to } P < 0.001) \). However, the data points were widely scattered about the regression lines, because for any GI, the glucose and insulin responses depended upon the amount of carbohydrate fed. GI accounted for only 60% of the variance of the mean capillary glucose responses, 64% of the variance of plasma glucose responses and 46% of the variance of plasma insulin responses [Fig. 3].

The mean capillary glucose responses of the four foods, expressed as a percentage of that for bread at the same level of carbohydrate, did not differ significantly at different levels of carbohydrate, but the variability of the values decreased as the amount of carbohydrate increased: 25 g carbohydrate, 99 ± 13%, 50 g carbohydrate, 86 ± 10% or 100 g carbohydrate, 83 ± 6% \( (F_{2,62} = 0.47) \). Thus, for any specific amount of carbohydrate,
the mean glucose response of different foods taken by the same individuals and expressed as a percentage of food GI should result in the same value.

From the above three analyses, it appeared that the postprandial glucose and insulin response areas could be described by a family of curves, one for each food. For each food, the glucose and insulin response areas would increase nonlinearly with amount of carbohydrate to reach a maximum. The relative distance between the curves for the different foods would be the same at any level of carbohydrate and would be proportional to the GI values of the food. Below is a detailed explanation of how an expression was derived to describe the blood glucose response data in this way.

Because the relative responses for capillary and plasma glucose were not different (Fig. 2), an average was taken for each test. The glucose relative responses (GR) were normalized by expressing them as a percentage of food GI. A curve was fitted to all 98 individual points (7 subjects, 14 test meals) by nonlinear regression analysis using the model $y = A[1 - e^{-Bx}] + C$. The values obtained for the constants A, B and C, respectively, were 150, 0.018 and 13 ($r = 0.68, P < 0.001$). Here, $x$ is the amount of carbohydrate and $y$ is the glucose relative response (GR). The constant B describes the rate at which the curve reaches its maximum value; at a value of $x = 0.69/B$, $y$ is half of its maximum value. Because it was shown above that the relative glycemic effects of foods is independent of amount of carbohydrate, the value of $B$ is the same for all foods. $C$ is the response at $x = 0$ (i.e., the $y$-intercept). The maximum glucose response, which occurs at $x = \infty$, equals $A + C$. Because the maximum response for different foods is determined by the food GI, and the constant $A$ was determined from the normalized GR values (100·GR/GI), the term $GI\cdot A/100$ was substituted for $A$. Thus, the equation describing the relative glucose responses (GR) as a function of amount of carbohydrate (D) and glycemic index (GI), was as follows:

$$GR = 1.5 \cdot GI \cdot (1 - e^{-0.018D}) + 13.$$ 

The family of curves generated for the four foods tested (GI = 48, 71, 100, 129) are shown along with mean relative glucose responses (GR) in Figure 4. The equation was used to calculate GR for all 14 test meals: e.g., for 25 g carbohydrate from spaghetti (GI = 71), $GR = 1.5 \cdot 71 \cdot (1 - e^{-0.018 \cdot 25}) + 13 = 35$. The GR predicted from the equation were highly correlated with the observed mean capillary and plasma glucose response areas ($r = 0.97$ and $r = 0.96, P < 0.001$), accounting for 94% and 92% of the variance of the mean capillary and plasma glucose response areas, respectively (Fig. 3). When the amount of protein in the test meals was added to the model, using stepwise multiple linear regression, the unexplained variance of mean capillary and plasma glucose response areas was reduced nonsignificantly by 0.4% ($P = 0.34$) and 0.3% ($P = 0.52$), respectively. Fat had a similar, nonsignificant effect.

The data for plasma insulin (IR, insulin relative response; II insulenic index) were handled in the same way as for glucose to derive the following equation: $IR = 2.9 \cdot GI \cdot (1 - e^{-0.0078D}) + 5$ where IR is the plasma insulin response area expressed as a percentage of that to 50 g carbohydrate from white bread, II is the food insulenic index and $D$ is the amount of carbohydrate in grams. The relationship between the mean II and GI for the four foods was determined by multiple linear
regression to be about II = GI⋅0.6 + GI²⋅0.003. Thus, substituting for II, the following equation describes the relative insulin responses as a function of carbohydrate amount and food glycemic index: IR = 2.9⋅[GI⋅0.6 + GI²⋅0.003][1 − e^{−0.0078P}] + 5 [Fig. 4]. The insulin responses predicted from this equation were highly correlated with the observed mean insulin responses (r = 0.92; P < 0.001) explaining 85% of their variability [Fig. 3]. When protein was added to the model, the unexplained variance of mean insulin responses was nonsignificantly reduced by 2.2% (P = 0.20). Fat had an even smaller effect, 0.3%.

### DISCUSSION

The results showed that both dose of carbohydrate consumed and food GI significantly influence blood glucose and insulin responses in nondiabetic subjects. Consequently, when both source and amount of carbohydrate in test meals were varied, amount of carbohydrate alone was a poor predictor of postprandial glucose and insulin responses, explaining only 47–57% of their variability [Fig. 3, top]. Similarly, source of carbohydrate alone was a poor predictor of postprandial glucose and insulin responses, explaining only 46–64% of their variability [Fig. 3, middle]. However, by taking both source and amount of carbohydrate into account, we were able to derive an expression which explained 85–94% of the variability of postprandial glucose and insulin responses [Fig. 3, bottom].

This is at variance with current recommendations (American Diabetes Association 1994) on dietary carbohydrate which state: "... from a clinical perspective first priority should be given to the total amount of carbohydrate consumed rather than the source of the carbohydrate." One reason for the discrepancy may be that, for this study, we selected carbohydrate sources with a wide range of GI which we expected to produce a wide range of glucose and insulin responses. However, in practice, common foods from different sources may not have large differences in GI and, thus, would not be expected to affect glucose or insulin responses. For example, we have shown that the GI value for 14 different bread products did not differ significantly (Wolever et al. 1994). Food sources as different as melba toast, bagels, angel food cake, gram crackers, whole wheat crackers, couscous, corn chips, oatmeal muffins, french fries, mashed potatoes, canned green pea soup, and the breakfast cereals Cream of Wheat™, Cheerios™ and Golden Grahams™ have almost identical GI values (range 94–106) [Wolever et al. 1994]. Thus, source of carbohydrate will affect postprandial glucose responses only if the GI values of the different foods are different. The chance of detecting a difference in an experimental situation depends upon the within-subject variability of blood glucose responses in the

### TABLE 2

<table>
<thead>
<tr>
<th>Amount of carbohydrate</th>
<th>ANOVA²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Source</td>
</tr>
<tr>
<td>25 g</td>
<td>50 g</td>
</tr>
<tr>
<td>Means³</td>
<td></td>
</tr>
<tr>
<td>Capillary glucose, (mmol⋅min/L)</td>
<td></td>
</tr>
<tr>
<td>Barley</td>
<td>51 ± 9</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>97 ± 21</td>
</tr>
<tr>
<td>Bread</td>
<td>—</td>
</tr>
<tr>
<td>Potato</td>
<td>109 ± 16</td>
</tr>
<tr>
<td>Means⁴</td>
<td>86 ± 10a</td>
</tr>
<tr>
<td>Plasma glucose, (mmol⋅min/L)</td>
<td></td>
</tr>
<tr>
<td>Barley</td>
<td>48 ± 13</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>93 ± 20</td>
</tr>
<tr>
<td>Bread</td>
<td>—</td>
</tr>
<tr>
<td>Potato</td>
<td>78 ± 13</td>
</tr>
<tr>
<td>Means⁴</td>
<td>73 ± 10a</td>
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<tr>
<td>Plasma insulin, (mmol⋅min/L)</td>
<td></td>
</tr>
<tr>
<td>Barley</td>
<td>2.8 ± 1.1</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>6.0 ± 1.6</td>
</tr>
<tr>
<td>Bread</td>
<td>—</td>
</tr>
<tr>
<td>Potato</td>
<td>6.8 ± 1.7</td>
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<tr>
<td>Means⁴</td>
<td>5.2 ± 0.9a</td>
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</tbody>
</table>

1 Values are means ± SEM. abc Means with different letter superscripts are significantly different (P < 0.05); NS = not significant.

2 Right three columns show F values from ANOVA and their significance for the main effects of source and amount of carbohydrate and source × amount interaction (S × A).

3 Means of values for the 25, 50 and 100 g doses of carbohydrate for each food.

4 Means of values for the four foods.
subjects being studied, the difference in GI, and the number of subjects being studied, or the number of times the tests are done [Wolever et al. 1989].

The American Diabetes Association has minimized the importance of source of carbohydrate because of the belief that differences in source of carbohydrate make no difference to glycemic responses in the context of mixed meals. However, we have pointed out that such an interpretation is erroneous because faulty methods were used in a number of the studies cited to support this position [Wolever and Jenkins 1986, Wolever et al. 1991]. In addition, there are a number of studies, which were ignored, showing that the GI retains its predictive ability in the mixed meal setting [Chew et al. 1988, Collier et al. 1986, Indar-Brown et al. 1992]. Furthermore, there is a large amount of evidence that therapeutic maneuvers which reduce postprandial glucose responses, either by altering diet GI [Brand et al. 1991, Collier et al. 1988, Fontvieille et al. 1988, Wolever et al. 1992a] or through pharmacologic inhibition of digestive enzymes [Chiaison et al. 1994] improve overall blood glucose control in diabetes. Thus, we believe it is warranted and important to characterize and understand the factors which influence postprandial glucose and insulin responses.

The glycemic indices for spaghetti and instant potato estimated here, 71 and 129, respectively, are similar to previously published averages for these foods, 67 and 120 (Wolever 1990). The value for barley, 48, is somewhat higher than the average of 36 from two other studies [Wolever 1990], but the difference of 12 is probably within experimental error, given that the mean SD of GI values for the same foods tested in different studies is 12 [Wolever et al. 1991].

We measured glucose in simultaneously obtained capillary blood and plasma glucose because these methods are used by different groups to determine glycemic responses. It is well known that plasma glucose concentrations are greater than those of capillary whole blood because the concentration of glucose in red cells is lower than that in plasma. On the other hand, after a meal, the glucose concentration in arterial plasma is higher than in venous plasma because of glucose utilization by peripheral tissues [Coppack et al. 1990]. Because capillary blood is arterialized, our finding that postprandial increments of glucose were greater in capillary glucose response at 50 g carbohydrate from white bread. Values are means ± SEM. Lines represent the following equations: GR = 1.5 × GI × [1 − e^{−0.016D}] + 13 and IR = 2.9 × GI × D + 0.003[1 − e^{−0.0078D}] + 5 where GR = relative glucose response, IR = relative insulin response, GI = glycemic index (the 4 lines represent GI values of 129, 100, 71, and 48) and D = amount of carbohydrate consumed. A line was drawn for each of the four foods tested, potato, bread, spaghetti and barley, using the GI values determined in this study. See text for method of deriving the equations.
illary blood than in venous plasma was not unexpected. In this study, we compared the relative glycemic effects of different foods determined from glucose measurements in capillary blood and venous plasma. Previously, it has been assumed that measuring glucose responses in capillary blood or venous plasma did not affect the glycemic index values of foods. This study provides evidence that this assumption is valid. Nevertheless, the current data also suggest that the use of capillary blood glucose is a more precise way of assessing the glycemic responses of foods because there were greater absolute differences between foods and greater heterogeneity between means (Table 2), and, thus, more experimental power was obtained.

The equations developed here for predicting relative glucose and insulin responses would be useful if they could be applied to subjects regardless of their glucose tolerance status. There is some evidence that the model for glucose responses may be useful in a wide variety of situations. The GI of foods is not affected by age (Wolever et al. 1988), and is similar in subjects with normal glucose tolerance, noninsulin-dependent diabetes (NIDDM), and insulin-dependent (IDDM) diabetes (Wolever et al. 1987 and 1990). The shape of the dose-response curve for incremental glucose areas may also be similar in normal and diabetic subjects. Gannon et al. (1989) tested 0–50 g glucose in subjects with NIDDM, and found a virtually linear increase in glucose responses. Rasmussen (1993a and 1993b) tested 20, 40 and 60 g carbohydrate from rice and found a linear response in NIDDM, but, in IDDM, there tended to be a flattening of the curve. We tested 0, 25, 50, 75 and 100 g carbohydrate from bread in diabetic subjects and found a marked flattening of the curve above 50 g carbohydrate (Wolever et al. 1994). For insulin responses, the model may not be valid for all subjects, particularly in NIDDM in which insulin secretion is reduced. For example, Gannon et al. (1989) found that subjects with NIDDM had the same insulin response after 50 g oral glucose as after 35 g. However, this may reflect the poor insulin-stimulating effect of glucose; Rasmussen (1993a) found a nearly linear insulin dose-response curve in NIDDM subjects given 20, 40 and 60 g carbohydrate from rice.

The other major factor which may influence the utility of the equations developed here is the potential effects of fat and protein in mixed meals on glucose and insulin responses. Fat delays gastric emptying and tends to flatten postprandial plasma glucose and insulin responses (Collier et al. 1984, Welch et al. 1987), whereas protein stimulates insulin and reduces glucose responses (Nuttall et al. 1984). In addition, different proteins have different effects (Gannon et al. 1988). In the present study, the small variation in protein and fat content of the test meals did not account for a detectable amount of variation in the observed glucose and insulin responses. The amount of fat required to have an impact on the incremental area under the glucose curve is rather large; 50 g fat markedly reduces the glucose and insulin response to a 50-g carbohydrate load. However, the difference in fat between most meals would probably be much smaller than this. We estimated that the composition of most meals would range from about 16 to 55% energy from fat, representing, for 2090 kJ meals, 9–31 g, or a range of 22 g. Equivalor substitution of fat for carbohydrate across this range was shown to have no effect on the glycemic response of spaghetti relative to bread in subjects with NIDDM (Wolever et al. 1992b). We found that adding 22 g fat to cornmeal delayed the rise of glucose somewhat, but had no effect on the overall glycemic response area (Wolever et al. 1994). Similar arguments apply for protein, at least in subjects with NIDDM, in whom 30 g protein stimulated insulin, but for whom 50 g was required to have a significant effect on glucose (Nuttall et al. 1984). In normal subjects, however, 16 g protein influenced both glucose and insulin responses (Spiller et al. 1987). There is some evidence that large amounts of protein and fat may affect the relative difference in glucose and insulin responses of foods (Gulliford et al. 1989), but other studies suggest that fat does not have this effect (Collier et al. 1984, Wolever et al. 1992b). Thus, the models developed here have to be tested in the context of mixed meals, and may apply less well for insulin than for glucose.

It is concluded that, for individual foods with different GI, both source and amount of carbohydrate influence the postprandial glucose and insulin responses of non diabetic subjects. Relative glucose responses are similar if glucose is measured in capillary blood or venous plasma. However, the use of capillary blood to measure glucose may provide more experimental power to detect differences among foods. Using equations developed by nonlinear regression analysis, source and amount of carbohydrate explained 85–94% of the variability of observed glucose and insulin responses. Further studies are required to determine if these conclusions are valid for mixed meals and for subjects with diabetes.

LITERATURE CITED


