Effects of long-term consumption and single meals of chickpeas on plasma glucose, insulin, and triacylglycerol concentrations1–3

Paul Nestel, Marja Cehun, and Andriana Chronopoulos

ABSTRACT
Background: Legumes are recommended for better glucose control in persons with diabetes. Whether subjects with normal insulin sensitivity would also benefit from legume consumption is not clear.

Objective: Our goal was to compare the effects on insulin sensitivity of chickpea-based and wheat-based foods when eaten as single meals or over 6 wk.

Design: Acute and long-term studies were conducted in healthy middle-aged men and women. In the acute study (n = 19), plasma glucose, insulin, and calculated homeostasis model assessment (HOMA; an index of insulin sensitivity) were measured on 3 separate days over 3 h after the subjects consumed 50-g available carbohydrate loads from either chickpeas, wheat-based foods, or white bread. The long-term comparison (n = 20) was a randomized, crossover study in which chickpea-based and wheat-based foods were eaten for 6 wk each. Plasma glucose, insulin, and HOMA were measured in the fasting state and 2 h after a 75-g glucose load.

Results: After single meals, plasma glucose was substantially lower 30 and 60 min after the chickpea meal than after the other 2 meals (P < 0.05), and plasma insulin and HOMA were lower at 120 min (P < 0.05 for both). Despite this, the long-term study failed to show significant differences in plasma glucose, insulin, or HOMA either in the fasting state or after a glucose load.

Conclusion: Compared with a wheat-based meal, a single chickpea-based meal led to a lesser response in plasma glucose and insulin concentrations, but this was not translated into long-term improvement in insulin sensitivity over 6 wk, at least in healthy subjects.


KEY WORDS Chickpeas, wheat-based foods, insulin sensitivity, healthy subjects, HOMA, homeostasis model assessment

INTRODUCTION
The consumption of carbohydrates that result in low postprandial glucose and insulin concentrations, known as low–glycemic index foods, has been associated with reduced rates of coronary artery disease (1) and type 2 diabetes (2). The main legumes eaten by humans have in common starches that are slowly digested. In chickpeas, about one-third of the starch is amylose (3), which, even after gelatinization, is resistant to rapid and total hydrolysis in the small intestine (4). The botanical structure of legumes may also be a contributing factor to their rate of digestion (5, 6).

The health benefits of eating legumes reportedly include improved glucose disposal through greater insulin sensitivity. Jenkins et al (7) reported in 1980 that equal portions of carbohydrate derived from various legumes including chickpeas led to 45% lower peak glucose concentrations than did carbohydrate derived from grains and cereals. Wolever et al (8) emphasized the importance of including legumes in the diets of patients with type 2 diabetes to achieve the desired overall glycemic index.

It is possible, however, that the demonstration of reduced glucose and insulin responses to single meals may not translate into long-term improved insulin sensitivity. Kiens and Richter (9) showed that diurnal values for plasma glucose and insulin were higher with high–glycemic index than with low–glycemic index foods initially, but that the difference was no longer significant after 1 mo. This may explain the inconsistent improvement in diabetic control with low–glycemic index foods, although reviews show that a benefit accrues in a majority (10). Juntunen and colleagues observed reduced postprandial insulin responses to whole-kernel rye bread (5) but also observed that 8 wk of consumption of high-fiber rye bread did not improve insulin sensitivity (11). Jenkins et al (12) failed to find a benefit on carbohydrate metabolism in the intermediate term in healthy subjects consuming low–glycemic index foods despite a significantly lower 12-h blood glucose profile.

The focus of the present study was a comparison of the short-term and long-term effects of chickpeas on plasma glucose and insulin responses. The legume is a major staple in large parts of the world, yet less is known about its potential health benefits than about other commonly eaten legumes such as soybeans. We report comparisons between chickpea- and wheat-based foods on the acute responses to single meals and after 6-wk interventions with each category of these starch- and fiber-rich foods.

SUBJECTS AND METHODS
The experimental design comprised 3 separate studies in which chickpea- and wheat-based foods were compared at approximately similar macronutrient contents. The studies were approved by the Alfred Hospital’s Human Ethics Committee.

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Acute study

Nineteen middle-aged men and women were recruited for the acute study; the subjects’ characteristics are shown in Table 1. The subjects were aged < 70 y and had an average body mass index indicating mild overweight; none had a waist circumference exceeding the Adult Treatment Panel III criteria for the metabolic syndrome in men or women (13). In all subjects, fasting plasma glucose concentrations were < 6 mmol/L and plasma insulin concentrations were within the range for healthy subjects. Only LDL cholesterol was on average moderately above the normal value of 3.6 mmol/L. None of the subjects had any other metabolic disorders, cardiovascular disease, or renal or hepatic dysfunction. Vegetarians and others who had habitually eaten substantial amounts of legumes were excluded. Daily alcohol consumption exceeding 4 standard drinks in men or 2 in women was also an exclusion criterion (although none of the subjects drank that much). Other exclusion criteria included smoking, taking supplements or therapy for lowering lipids or blood pressure, and unusual physical activity.

Subjects were asked to avoid eating legumes during the study period, to not drink alcohol for 24 h before each test day, and to abstain from food other than water for 12 h before each study day. Studies began at 0800. The 3 test meals, which were based on white bread (standard), chickpeas, or wheat, were separated by at least 1 wk and no more than 2 wk.

Each meal provided 50 g available carbohydrate containing ≈42 g starch plus 10 g sugars, 12 g protein, and < 2 g fat and 10 g dietary fiber. Chickpeas (200 g) had been cooked and drained, gently mashed, and prepared with 100 mL low-fat milk. Wheat-based foods, mainly shredded whole-grain, low-sugar wheat cereal with a minor addition of wheat bran served in 100 mL low-fat milk, also contained 10 g dietary fiber. The standard meal based on white bread and jam to equal the carbohydrate and glucose contents of the other meals contained only 3 g fiber and was supplemented with similar milk. The meals were eaten within 10 min. Chickpeas, including canned chickpeas, have a reported glycemic index value between 47 and 59 (against white bread) (14); shredded wheat and wheat bran have reported glycemic index values of 99 and 60, respectively (14).

Blood was collected from an indwelling small catheter in an antecubital vein at times 0, 30, 60, 120, and 180 min for measurement of plasma glucose and insulin concentrations. Homeostasis model assessment (HOMA) was calculated with the following equation (15):

\[ \text{HOMA} = \frac{\text{plasma insulin (mU/L)}}{\text{plasma glucose (mmol/L)/22.5}} \]

Long-term study

The design was a randomized, crossover, open study with chickpea- or wheat-based foods eaten for 6 wk each. Of the 21 subjects recruited, 20 subjects completed the study, one having dropped out because of abdominal discomfort when eating chickpeas; however, a second subject was found to have been noncompliant, leaving 19 subjects for analysis. The characteristics of the subjects as shown in Table 1 were not significantly different from those of the participants in the acute study, although only a few subjects volunteered for both studies. Inclusion and exclusion criteria were similar to those required for the acute study. A 2-wk familiarization period preceded the randomization, during which time the subjects were instructed about dietary compliance, avoidance of legumes, and completing food-frequency questionnaires.

Chickpea foods were derived from cooked, drained canned chickpeas (140 g) and bread and biscuits baked with 30% chickpea flour. Total chickpea protein was 38 g daily. Analysis of the flour showed that it contained 41% starch (30% amylose), 22% protein, 10% fiber and lignins, 5% soluble sugars, 3% fructooligosaccharide, 2% fat, and > 10% moisture. Wheat-based foods included whole-grain shredded wheat cereal and bread and biscuits made from whole-grain flour. The macronutrient contents of the 3 diets (baseline, chickpea, and wheat; Table 2) were calculated with a program designed by the Anti-Cancer Council of Victoria, Australia, based on Australian food tables. The subjects’ baseline diet, as assessed with a 3-d food-frequency questionnaire, contained an average fat intake of 32% of energy, energy intake consistent with average normal body weight, and a relatively high intake of fiber. Similar questionnaires were completed at the end of the 2 intervention periods.

With the chickpea diet, dietary fiber was significantly higher than during the wheat-based period or at baseline. When compared with baseline nutrient consumption, fatty acid and cholesterol intakes during the chickpea period were significantly lower, suggesting a shift in food choices due to the apparently satiating effects of the chickpeas. However, only fiber intake differed between the 2 test diets.

The foods were eaten across 3 meals; for example, bread made with chickpea flour was eaten at breakfast, canned chickpeas and biscuits made from the flour were eaten at lunch, and canned chickpeas plus bread were eaten in the evening. Similarly, wheat-based cereals were eaten in the morning and the bread at the other 2 meals. Compliance was judged from the consistency of the food-frequency questionnaires across the 3 periods and the negligible returns of uneaten foods.

Blood samples were collected as above twice, at baseline after randomization and on ≈2 consecutive days at the end of the dietary period. On the final day of each dietary period, a 75-g glucose load was administered and blood samples were collected over 120 min through an indwelling small catheter in a forearm vein. Plasma glucose and insulin concentrations were assayed in each sample, the former by a standard automated method and the latter by radioimmunoassay.
Acute lipemia study

Ten subjects also participated in this phase of the trial, which was designed to test whether the addition of chickpeas to a standard fat meal would reduce the postprandial lipemia. The characteristics of the group resembled those in the acute study group from which the subjects were recruited. The core component of the test meal was 56 g fat derived from cream, cheese, butter, and whole milk. This was eaten together with 65 g rice cereal plus 2 slices of bread made from chickpea flour, or 3) shredded wheat cereal plus 2 slices of whole-grain bread. Apart from starch, the meals contained 11-14 g protein, 2-7 g additional fat, and 12-14 g fiber and were isoen-ergetic. The subjects fasted overnight and ate the meals at 1–2 weekly intervals. Blood samples were drawn through a small indwelling catheter after 1, 2, 3, 5, and 7 h. Plasma triacylglycerol concentrations were measured by a standard automated method.

RESULTS

Acute study

The baseline values for plasma glucose, insulin, and calculated HOMA did not show significant within-subject differences on the 3 test days; nevertheless, subsequent analyses took baseline values into account. The effects of the 3 treatments are shown in Table 3. The initial analysis tested the effects of time, treatment, and time x treatment for plasma glucose, insulin, and HOMA. Glucose was measured at 30, 60, 120, and 180 min, but insulin and therefore HOMA were measured at only 120 min. Because the time x treatment effect on glucose was significant, the results were further analyzed. Plasma glucose concentrations after the chickpea treatment differed significantly from those after the wheat and standard treatments at 30 and 60 min. Plasma insulin and HOMA were significantly lower at 120 min after the chickpea treatment than after the other 2 treatments. In several subjects, plasma glucose was also measured at 4 h, and glucose concentrations had not risen further (data not shown).

Long-term study

Mean body mass did not change significantly during the study, being 71.8, 71.8, and 72.6 kg at baseline, after the wheat-based diet, and after the chickpea-based diet, respectively. Two variables of glucose disposal and insulin sensitivity were measured. First, the fasting values (means of 2 measurements) for plasma glucose, insulin, and HOMA were not significantly different between the 2 treatments (Table 4). Second, the 2-h post-glucose-load values for plasma glucose, insulin, and HOMA also did not differ significantly between treatments (Table 4). Thus, there was no evidence for improved insulin sensitivity with long-term chickpea treatment. Note that only 2 subjects had HOMA

### TABLE 2

<table>
<thead>
<tr>
<th>Energy (kJ/d)</th>
<th>Baseline</th>
<th>Wheat-based diet</th>
<th>Chickpea-based diet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7989 ± 3717</td>
<td>7524 ± 3947</td>
<td>7424 ± 2938</td>
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<table>
<thead>
<tr>
<th>Protein (g/d)</th>
<th>Baseline</th>
<th>Wheat-based diet</th>
<th>Chickpea-based diet</th>
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<tbody>
<tr>
<td></td>
<td>92 ± 45</td>
<td>89 ± 49</td>
<td>83 ± 32</td>
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<table>
<thead>
<tr>
<th>Carbohydrates (g/d)</th>
<th>Baseline</th>
<th>Wheat-based diet</th>
<th>Chickpea-based diet</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>221 ± 92</td>
<td>211 ± 100</td>
<td>222 ± 81</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Fiber (g/d)</th>
<th>Baseline</th>
<th>Wheat-based diet</th>
<th>Chickpea-based diet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>45 ± 6</td>
<td>44 ± 14</td>
<td>47 ± 11</td>
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</table>

<table>
<thead>
<tr>
<th>Cholesterol (mg/d)</th>
<th>Baseline</th>
<th>Wheat-based diet</th>
<th>Chickpea-based diet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>240 ± 168</td>
<td>235 ± 167^a</td>
<td>200 ± 119^b</td>
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</table>

^a ± SD; n = 19. Values were derived from 3-d food-frequency questionnaires completed at baseline after a 2-wk familiarization period and during the final weeks of each intervention. Means in a row with different superscript letters are significantly different, P < 0.05 (two-factor repeated-measures ANOVA with Bonferroni post hoc adjustment).

Statistical analysis

Statistical analysis was performed by using SAS version 8.0 (SAS Institute Inc, Cary, NC). Outcome variables were all found to be well approximated by log-normal distribution and were thus log-transformed before analysis. The values presented in the tables that provide statistical assessment are therefore given as geometric means. Statistically significant differences between treatments (chickpea, wheat, and standard) were determined by using a repeated-measures analysis of covariance, with adjustment for baseline concentrations. Interactions between treatments and time were calculated when appropriate. Post hoc comparisons between treatments were made by using Bonferroni adjustment. A two-sided P value of 0.05 was considered to be statistically significant.

Acute lipemia study

Ten subjects also participated in this phase of the trial, which was designed to test whether the addition of chickpeas to a standard fat meal would reduce the postprandial lipemia. The characteristics of the group resembled those in the acute study group from which the subjects were recruited. The core component of the test meal was 56 g fat derived from cream, cheese, butter, and whole milk. This was eaten together with 65 g rice cereal plus 2 slices of bread made from chickpea flour, or 3) shredded wheat cereal plus 2 slices of whole-grain bread. Apart from starch, the meals contained 11-14 g protein, 2-7 g additional fat, and 12-14 g fiber and were isoen-ergetic. The subjects fasted overnight and ate the meals at 1–2 weekly intervals. Blood samples were drawn through a small indwelling catheter after 1, 2, 3, 5, and 7 h. Plasma triacylglycerol concentrations were measured by a standard automated method.
values that were indicative of diminished insulin sensitivity (4.0 and 5.7 after wheat-based diets).

**Acute lipemia study**

In the 10 subjects in whom a standard fat meal was combined with 1 of 3 supplements (chickpea, wheat, or standard), baseline and corresponding postprandial triacylglycerol concentrations did not differ significantly over 7 h (Table 5). These results show that the addition of chickpeas to a meal does not modify postprandial lipemia and in the context of the effects on acute glycemnic response appear to exclude a significant effect on gastric emptying. Changes in LDL cholesterol and HDL cholesterol values that were indicative of diminished insulin sensitivity (4.0 and 5.7 after wheat-based diets).

**DISCUSSION**

The results of the present study confirm that a low-glycemic index food such as chickpeas results in lower plasma glucose, insulin, and HOMA responses after a single meal than after meals of similar available carbohydrate content derived from wheat-based or higher glycemic index foods. However, our results also suggest that this benefit need not translate into longer-term improved insulin sensitivity or glucose disposal. Such comparisons have been infrequently carried out. Note also that whereas the 2 populations in our acute and long-term studies were not dissimilar in characteristics relevant to this comparison, they consisted of mostly different persons.

Kiens and Richter (9) came to a conclusion similar to ours in their study of healthy subjects and attributed the long-term ab-

**TABLE 3**

<table>
<thead>
<tr>
<th>Time</th>
<th>Chickpea</th>
<th>Standard</th>
<th>Wheat</th>
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</thead>
<tbody>
<tr>
<td>Glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min</td>
<td>6.01 (5.50, 6.55)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.63 (6.99, 8.33)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.74 (7.09, 8.45)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>60 min</td>
<td>5.99 (4.66, 5.56)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.98 (6.39, 7.61)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.86 (6.28, 7.48)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>120 min</td>
<td>4.80 (4.39, 5.23)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.89 (4.48, 5.33)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.90 (4.49, 5.35)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>180 min</td>
<td>4.38 (4.01, 4.78)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.98 (3.64, 4.34)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.89 (3.56, 4.24)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Insulin, 120 min (mU/L)</td>
<td>9.47 (3.73, 13.33)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>20.91 (14.82, 29.50)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>20.97 (14.90, 29.53)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>HOMA index, 120 min</td>
<td>2.14 (1.42, 3.27)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.74 (3.11, 7.21)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.86 (3.21, 7.37)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>1</sup> Geometric i; 95% CI in parentheses. n = 19. Means within a row with different superscript letters are significantly different, P < 0.05. For glucose values, analysis was by two-factor repeated-measures ANOVA with post hoc Bonferroni adjustment; the treatment x time interaction was significant, P < 0.05. For insulin and HOMA index values, analysis was by one-factor repeated-measures ANOVA with post hoc Bonferroni adjustment.

**TABLE 4**

<table>
<thead>
<tr>
<th>Diet</th>
<th>Glucose</th>
<th>Insulin</th>
<th>HOMA index&lt;sup&gt;2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fasting</td>
<td>Post glucose load</td>
<td>Fasting</td>
</tr>
<tr>
<td>mmol/L</td>
<td>mU/L</td>
<td>mmol/L</td>
<td>mU/L</td>
</tr>
<tr>
<td>Baseline</td>
<td>5.2 ± 0.4</td>
<td>5.0 ± 2.0</td>
<td>6.6 ± 3.6</td>
</tr>
<tr>
<td>Wheat</td>
<td>5.1 ± 0.5</td>
<td>4.4 ± 1.5</td>
<td>8.2 ± 4.7</td>
</tr>
<tr>
<td>Chickpea</td>
<td>4.9 ± 0.4</td>
<td>4.4 ± 1.7</td>
<td>7.9 ± 4.5</td>
</tr>
</tbody>
</table>

<sup>1</sup> ± SD; n = 19. Baseline values represent concentrations after a 2-wk familiarization period. Post-glucose-load values were measured 2 h after the 75-g glucose load. None of the differences between the wheat-based and the chickpea-based periods were significant by two-factor repeated-measures ANOVA with interaction.

<sup>2</sup> Fasting HOMA = (fasting insulin (mU/L) x fasting glucose (mmol/L))/22.5. Post-glucose-load HOMA = (post-glucose-load insulin (mU/L) x post-glucose-load glucose (mmol/L))/22.5.
leguminous foods in overweight, insulin-resistant subjects (19), and the inclusion of legumes in the diets of persons with diabetes has resulted in better glucose disposal (8). As shown by Jarvi et al (20), improved glycemic control in patients with type 2 diabetes can be achieved by low–glycemic index foods independently of the fiber content of the diet, and little benefit has been reported of dietary fiber on glucose disposal in healthy subjects (21). The absence in the present study of significant differences in the glycemic and insulimemic responses after the meals containing wheat-based foods and the standard refined-carbohydrate foods despite a large difference in dietary fiber confirms that the glycemic index of foods is likely to be more relevant than the fiber content. A similar conclusion was reached by Jenkins et al (22) in a study of dietary wheat fiber in patients with type 2 diabetes.

The significantly and substantially lower plasma glucose and insulin concentrations after the single chickpea meals may reflect the higher amylose content and the botanical structure of chickpeas. It is likely that the starch was digested and absorbed more slowly in the small intestine from chickpeas than from wheat. Botham et al (4) reported that 15% of starch in chickpeas escaped hydrolysis in the small intestine of ileostomy patients. We do not believe that this alone explains the substantially lower glycemia and insulinemia after single meals of chickpeas than after wheat-based meals, however. The total loss of chickpeas is lessened through fermentation in the large intestine and is consistent with the absence of weight loss in the long-term study. When other foods with less readily available starch as a result of the physical characteristics of the plant are eaten, glycemia may be attenuated minimally, whereas the insulimemic response is significantly reduced (5). It is therefore likely that several physicochemical properties of chickpeas contributed to the reduced glycemia and insulinemia in the first hour after the chickpea-based meal.

The form in which the chickpeas were eaten during the long-term study and the acute single-meal study was not identical. Although both studies included cooked chickpeas (140 g daily in the long-term study and 200 g in the acute study), additional chickpea flour was consumed as bread and biscuits in the long-term study. The processing of the flour and its products may have attenuated the low–glycemic index status of the original chickpeas. Whereas we cannot exclude this completely, the delivery of test starches in bread, biscuits, cereals, and muffins is not unusual. In a study by Behall and Howe (23), low- and high-glycemic index starches were compared during long-term consumption and after single meals as in the present study. The starches tested were presented in the same foods during the acute studies and during the long term (bread, cookies, cereal, and muffins). Behall and Howe’s findings resembled ours, with no significant changes in fasting plasma glucose and insulin concentrations after several weeks, yet significantly lower plasma glucose and insulin concentrations with the low–glycemic index starch over the 3 h of the acute study. Their study also showed that the processing of the starch need not negate its glycemic status.

The results of the postprandial lipemia study make it unlikely that delayed gastric emptying was responsible for the diminished glycemia after the chickpea meal. Although we did not find an effect on the degree of postprandial lipemia over 7 h after the different meals, Liljeberg and Bjorck (24) reported a reduced rise in plasma triacylglycerol after eating fat at lunchtime if the breakfast contained low–glycemic index starch.

We conclude that, at least in apparently insulin-sensitive subjects, the daily consumption of relatively large amounts of chickpeas and chickpea flour does not improve insulin sensitivity over a 6-wk period more than does the consumption of wheat-based foods. These negative findings occurred despite substantial and significantly lesser glycemic and insulimemic responses after single meals of chickpeas than after single meals of wheat-based foods.

We thank Michael Bailey of Monash University, Melbourne, for assistance with statistical analysis.

All 3 authors contributed to the design, execution, and analysis of the studies and none had a conflict of interest.

REFERENCES


### TABLE 5

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Standard</th>
<th>Wheat</th>
<th>Chickpea</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.2 ± 0.5</td>
<td>1.2 ± 0.5</td>
<td>1.2 ± 0.4</td>
</tr>
<tr>
<td>60</td>
<td>1.4 ± 0.6</td>
<td>1.5 ± 0.6</td>
<td>1.6 ± 0.6</td>
</tr>
<tr>
<td>120</td>
<td>2.1 ± 1.1</td>
<td>2.0 ± 0.7</td>
<td>2.0 ± 0.7</td>
</tr>
<tr>
<td>180</td>
<td>2.3 ± 1.2</td>
<td>2.3 ± 1.1</td>
<td>2.4 ± 1.0</td>
</tr>
<tr>
<td>300</td>
<td>2.3 ± 1.7</td>
<td>2.2 ± 1.4</td>
<td>2.5 ± 1.4</td>
</tr>
<tr>
<td>420</td>
<td>2.0 ± 1.5</td>
<td>2.0 ± 1.1</td>
<td>2.2 ± 1.3</td>
</tr>
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</table>

\[\text{mmol/L} \times \text{SD; } n = 10\]There was a significant treatment 
\(P < 0.05\) (two-factor repeated-measures ANOVA with post hoc Bonferroni adjustment), although differences between treatments were not significant \((P > 0.05)\).


