Red meat, dairy, and insulin sensitivity: a randomized crossover intervention study¹–³

Kirsty M Turner, Jennifer B Keogh, and Peter M Clifton

ABSTRACT
Background: Epidemiologic studies have linked high consumption of red and processed meat with risk of developing type 2 diabetes, whereas high dairy consumption has been associated with decreased risk, but interventions have been limited.

Objective: We compared the effects on insulin sensitivity of consuming a diet high in lean red meat with minimal dairy, a diet high in dairy primarily low fat (from milk, yogurt, or custard) with no red meat, and a control diet that contained neither red meat nor dairy.

Design: A randomized crossover study was undertaken with 47 overweight and obese men and women divided into 2 groups as follows: those with normal glucose tolerance and those with impaired fasting glucose or impaired glucose tolerance. Participants followed the 3 weight-stable dietary interventions for 4 wk with glucose, insulin, and C-peptide measured by using oral-glucose-tolerance tests at the end of each diet.

Results: Fasting insulin was significantly higher after the dairy diet than after the red meat diet (P < 0.01) with no change in fasting glucose resulting in a decrease in insulin sensitivity after the high-dairy diet (P < 0.05) as assessed by homeostasis model assessment of insulin resistance (HOMA-IR). A significant interaction between diet and sex was observed such that, in women alone, HOMA-IR was significantly lower after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with red meat and sex was observed such that, in women alone, HOMA-IR was significantly lower after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01). Insulin sensitivity calculated by using the Matsuda method was 14.7% lower in women after the red meat diet compared with dairy diet (1.33 ± 0.8 compared with 1.71 ± 0.8, respectively; P < 0.01).

Conclusion: In contrast to some epidemiologic findings, these results suggest that high consumption of dairy reduces insulin sensitivity compared with a diet high in lean red meat in overweight and obese subjects, some of whom had glucose intolerance. This trial was registered at the Australian New Zealand Clinical Trials Registry as ACTRN12613000441718. Am J Clin Nutr doi: 10.3945/ajcn.114.104976.

Keywords: dairy, dietary proteins, insulin sensitivity, red meat, type 2 diabetes

INTRODUCTION
Insulin resistance increases the likelihood of impaired glucose tolerance (IGT)⁴ and development of type 2 diabetes (T2D) (1). Improved diet quality, energy restriction, and weight loss and increased physical activity lowers risk of T2D, but the role of specific dietary components is still debated (2).

Red meat is a good source of protein as well as vitamins and minerals (3); however, high consumption of red meat has been linked to risk of developing T2D. A meta-analysis of 12 cohort studies showed a 20% increase in risk of diabetes per 120-g/d increase in red meat intake and, for processed red meat, a 57% increase in risk per 50-g/d increase (4). There have been only a few intervention studies that assessed meat intake and insulin sensitivity without a weight-loss component, and these studies showed mixed results. A crossover study in healthy young women compared an 8-wk diet high in oily fish to one high in red meat. Fasting glucose concentrations decreased after both diets, and the effect of diet was NS. Fasting insulin concentrations increased after the red meat diet and decreased significantly after the oily fish diet with an almost 20% difference between the 2 diets, which resulted in increased insulin sensitivity after the oily fish diet (5). Fasting glucose concentrations did not change after a comparison of lean lamb and chicken in a crossover intervention of two 5-wk diets, but insulin was not measured (6).

Dairy foods such as milk, yogurt, and cheese, which are sources of high-quality protein, calcium, and other vitamins and minerals (7), have been associated with protection from developing T2D. A recent meta-analysis examined 14 cohorts and showed a significant inverse linear association between consumption of total dairy products, low-fat dairy, cheese, and yogurt and risk of T2D (8). A United Kingdom study showed 24% lower risk with 4.5 (125-g) servings of low-fat fermented dairy (primarily yogurt but including low-fat cheese), whereas total dairy, high-fat dairy, milk, cheese, and high-fat fermented dairy showed no association (9). In contrast, high-fat dairy had the strongest inverse association with T2D in a recent Swedish study, with no association shown for low-fat dairy intake, which indicated a possible protective effect of dairy fat (10). The most-recent meta-analysis (11) showed no effect of total or low fat dairy, and

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⁴Abbreviations used: BCAA, branched-chain amino acid; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral-glucose-tolerance test; T2D, type 2 diabetes.
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only yogurt was protective. There was no suggestion of harm from dairy despite its high saturated fat content.

Few intervention studies have evaluated red meat or dairy for their effect on insulin sensitivity in the absence of weight loss; therefore, this study was designed to maintain weight stability to isolate the effect that lean red meat and dairy have on insulin sensitivity. The primary hypothesis was that a red meat diet would produce greater insulin resistance than would a high-dairy diet with secondary hypotheses that the high-dairy diet would produce greater insulin sensitivity than would the diet without dairy and that these changes would be greater in individuals with impaired fasting glucose (IFG) or IGT than in those with normal glucose tolerance (NGT).

METHODS

Participants

Participants were recruited by public advertisement and screened for eligibility. Inclusion criteria included overweight and obese men and women >20 y old. Exclusion criteria included diagnosed diabetes, medication or supplements that would influence glucose metabolism, pregnancy or breastfeeding, recent weight gain or weight loss, or a history of metabolic illness such as kidney or liver disease. Participants were excluded if they had a known allergy or intolerance to dairy or lactose or were considered unlikely to comply with the study protocol. Participants were separated into 2 groups as follows: those with NGT and those with IGT or IFG as established by a 75-g oral-glucose-tolerance test (OGTT) performed at the baseline visit or from a previous medical diagnosis of IGT. The University of South Australia Human Research Ethics committee approved the study, and all participants provided written informed consent before participating. The trial was registered with the Australian New Zealand Clinical Trials Registry as ACTRN12613000441718. AUD $150 was offered to participants at completion of the study.

Dietary intervention

During the high–red meat diet, participants were instructed to consume ≥200 g red meat/d for 6 d/wk and consume minimal (<1 serving) dairy per day. During the high-dairy diet, participants were instructed to consume 4–6 servings of primarily low-fat dairy (from milk, yogurt, or custard) and cheese per day with chicken and fish as additional sources of protein but no red meat. Serving sizes were defined by using the guidelines of the Australian National Health and Medical Research Council (7) (e.g., 250 g milk, 200 g yogurt, 40 g hard cheese, or 120 g ricotta cheese). The low-dairy, no red meat control diet contained ≥200 g fish or chicken/d with <1 serving of dairy/d. Usual food items were replaced with red meat or dairy for weight to remain stable. Participants attended the clinic on 3 occasions during each diet to monitor weight and ensure dietary compliance. Participants were asked not to consume processed meat for the duration of the study. The diet order was randomized with all participants completing each 4-wk diet with a 2-wk washout period in between. Verbal and written instructions, including explanations of serving sizes, were provided for each diet along with digital kitchen scales (Homemaker Slimline Electronic Scale; KMart Australia).

<table>
<thead>
<tr>
<th>TABLE 1 Baseline characteristics of the participants¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>NGT</td>
</tr>
<tr>
<td>Sex (M/F), n</td>
</tr>
<tr>
<td>Age, y</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
</tr>
<tr>
<td>Baseline SBP, mm Hg</td>
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<tr>
<td>Baseline DBP, mm Hg</td>
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<tr>
<td>Total fat mass, %</td>
</tr>
<tr>
<td>Total lean mass, %</td>
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<tr>
<td>Total fat mass, kg</td>
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<td>Total lean mass, kg</td>
</tr>
</tbody>
</table>

¹Means that do not share a common superscript letter were significantly different at P < 0.05. DBP, diastolic blood pressure; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; SBP, systolic blood pressure.

²Mean ± SD (all such values).

³n = 45.
## Table 2

Effect of diet on glucose, insulin, and insulin-sensitivity indexes

<table>
<thead>
<tr>
<th>Diet</th>
<th>Men (n = 29)</th>
<th>Women (n = 18)</th>
<th>All (n = 47)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Red meat</td>
<td>Dairy</td>
<td>Control</td>
</tr>
<tr>
<td></td>
<td>Fasting glucose, mmol/L</td>
<td>5.24 ± 0.6</td>
<td>5.23 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>Fasting insulin, mU/L</td>
<td>5.47 ± 2.4</td>
<td>5.31 ± 2.6</td>
</tr>
<tr>
<td></td>
<td>HOMA-IR</td>
<td>1.30 ± 0.14</td>
<td>2.78 ± 0.39</td>
</tr>
<tr>
<td></td>
<td>Matsuda index</td>
<td>0.084 ± 0.027</td>
<td>0.082 ± 0.029</td>
</tr>
</tbody>
</table>

### Dietary measurements

A food-frequency questionnaire was completed at the baseline visit to assess usual dietary intake over the previous 12 mo. The food-frequency questionnaire is a validated tool for measuring dietary intake in the Australian population (12) and provides information regarding food choice, portion size, and frequency of consumption. A daily checklist was completed during each dietary period to obtain the amounts of red meat, dairy, or alternate protein sources consumed each day. A 3-d weighed food record was also completed within each 2-wk period. All food and beverages consumed over these 3-d weighed periods were recorded and entered into FoodWorks Professional Edition 7.0 software (Xyris) for dietary analysis.

### Clinical measurements

Height was measured on a wall-mounted stadiometer (Seca) at the baseline visit. Body weight was measured at each visit by using electronic digital scales (Tanita Corp.) in light clothing and without shoes. BMI (in kg/m²) was calculated as weight divided by height squared. Body composition was assessed at baseline by using whole-body dual-energy X-ray absorptiometry (Lunar Prodigy; Lunar Radiation Corp.). After an overnight fast, participants came to the Sansom Institute for Health Research Clinical Trial facility at the University of South Australia for OGTTs. These tests were performed at the end of each diet with blood samples taken every 30 min for a total of 5 time points. Blood for serum was collected in tubes containing sodium fluoride and stored on ice until processed. Blood samples were separated by a centrifuge at 1780 g at 4°C for 10 min (Universal 32R; Hettich Zentrifugen). Plasma glucose was measured by using an automated spectrophotometric analyzer (Konelab 20XTi; Thermo Electron), and serum insulin and C-peptide were measured by using commercial ELISA kits (kit 0030N for insulin, kit 0040 for C-peptide; Alpha Diagnostic).

### Analysis

Insulin sensitivity was assessed from the OGTT by using the methods of Stumvoll et al. (13), calculated as

\[
0.226 - \left( 0.0032 \times \text{BMI} \right) - \left( 0.0000645 \times \text{Ins}_{120} \right) (\text{pmol/L})
- \left( 0.0037 \times G_{90} \right) (\text{mmol/L})
\]

whereby \( \text{Ins}_{120} \) denotes insulin at 120 min and \( G_{90} \) denotes glucose at 90 min, and Matsuda and DeFronzo (14), calculated as

\[
10,000 \div \sqrt{ \left( \left[ G_{\text{fasting}} \right] \times \text{Ins}_{\text{fasting}} \right) \times \left[ G_{\text{meanOGTT}} \times \text{Ins}_{\text{meanOGTT}} \right]}
\]

whereby \( \text{Ins} \) denotes insulin, and \( G \) denotes glucose. HOMA-IR was also calculated from fasting glucose and insulin as

\[
\text{HOMA-IR} = \left[ FPI (\text{mU/L}) \times \text{FPG} (\text{mmol/L}) \right] \div 22.5
\]

whereby \( FPI \) denotes fasting plasma insulin, and \( \text{FPG} \) denotes fasting plasma glucose. Each of these methods previously showed strong correlations with the euglycemic hyperinsulinemic clamp method, which is considered the reference standard for assessing
insulin sensitivity (15). A sample size of 68 was calculated initially from the literature, and thus, the aim was to recruit 80 participants, allowing for withdrawals. This calculation was revised after the first 5 volunteers completed the study, and we showed the SD of insulin and HOMA-IR was lower than expected, and a sample size of 45 would have provided 90% power to see a 20% change in insulin sensitivity as assessed by using the Matsuda index.

The statistical analysis was performed with SPSS V22 software (IBM). The Kolomogorov-Smirnov test, Q-Q plots, and histograms were used to test for the normality of distribution. Variables that were not normally distributed were log transformed. Differences between groups were tested by using a repeated-measures ANOVA and paired samples t tests. A mixed-model analysis was used to examine the influence of weight changes during each dietary period. Analyses were conducted with and without outliers 2 SDs from the mean to assess any effect on outcomes. Outliers were included in the final analysis. The incremental AUC was calculated by using the trapezoidal equation. Data are expressed as means ± SDs, and significance was set at P < 0.05.

RESULTS

Of 304 people who initially responded to advertising, 176 people were screened, and 86 people satisfied the inclusion criteria. Figure 1 outlines the recruitment and withdrawal of participants. Forty-seven people (age: 47.8 ± 13.0 y; BMI: 31.1 ± 5.1) completed the study. Twenty-seven subjects had NGT, 6 subjects had IFG, and 14 subjects had IGT. Baseline characteristics of each group are shown in Table 1.

Sensitivity indexes were not normally distributed, and thus, data were log transformed before analysis. A repeated-measures analysis of variance showed a significant difference between diets for fasting insulin concentrations (Table 2). Fasting insulin was significantly higher in the dairy diet compared with the red meat diet (6.6 ± 4.1 compared with 5.5 ± 2.4 mU/L, respectively; P < 0.01). Because there was no difference in fasting glucose concentrations between diets, this resulted in a 16% decrease in insulin sensitivity after the high-dairy diet as assessed by using HOMA-IR (P < 0.05). There was no effect of age, BMI, percentage of fat mass, percentage of lean mass, or glucose-tolerance group when added as covariates or factors; however, a post hoc analysis revealed a significant interaction between diet and sex (P < 0.05), with insulin and HOMA-IR significant for women between red meat and dairy diets (P < 0.01). In women alone, the glucose-tolerance group or percentage of fat mass was NS.

Similarly, when red meat and dairy were compared, the Matsuda index showed a 14.7% reduced sensitivity after the dairy diet in women (P = 0.01) with no difference between diets in men (P-diet by sex interaction < 0.05). The Stumvoll sensitivity index showed a significant effect for women between red meat and dairy diets (P < 0.05); however, the removal of an outlier 2 SDs from the mean attenuated the significance. No interaction between group (NGT or IFG and IGT), BMI, age, or diet order was seen when added as covariates or factors. Fasting insulin concentrations after the dairy diet were also higher than after the control diet (P < 0.05), but HOMA-IR and the Matsuda index were only significantly different between the 2 diets in women (P-both comparisons < 0.05). The Stumvoll index was not significantly different between dairy and control diets.

The glucose incremental AUC was significantly different between glucose-tolerance subgroups (P < 0.01) (Figure 2), but there were no differences between diets overall and no effect when age, BMI, sex, or diet order was used as a covariate or factor. Insulin and C-peptide incremental AUCs were not significantly different between diets or groups (Table 3).

Energy intake was higher with the dairy diet (Table 4) than with both red meat and control diets (P-both comparisons < 0.001), and total and saturated fat intakes were also higher during the dairy diet than during either the red meat or control diet (P-both comparisons < 0.01). Carbohydrate intake was

FIGURE 2 Postprandial glucose, insulin, and C-peptide concentrations in response to a 75-g glucose OGTT after three 4-wk diets. NGT; n = 27, IFG/IGT; n = 20. Between glucose-tolerance group difference, P < 0.01 for glucose; NS for insulin or C-peptide (repeated-measures ANOVA by glucose-tolerance group). IFG, impaired fasting glucose; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral-glucose-tolerance test.
DISCUSSION

Fasting insulin increased after the dairy diet with no change in fasting glucose, which resulted in a higher HOMA-IR index. Calculated insulin sensitivity from the OGTT showed reduced sensitivity after the dairy diet in women. The red meat diet had a similar insulin and glucose response to that of the control diet that contained white meat. In contrast to some epidemiologic findings, these results suggest that, in overweight and obese individuals, high consumption of dairy may reduce insulin sensitivity compared with a diet high in lean red meat. The low carbohydrate amount in our diets may have influenced the results. Hoppe et al. (16) showed similar results when groups of healthy 8- to 11-yr-old boys consumed either 1.5 L skim milk each day for 1 wk or 250 g lean red meat, although weight gain in the dairy group may have played a role in the decreased insulin sensitivity seen.

In contrast to the current study, a decrease in fasting insulin was observed when an adequate dairy diet was compared with a low dairy diet in overweight and obese subjects (17) over 12 wk, but there was a 1.7-kg fall in fat mass in the adequate dairy group. Fasting insulin concentrations were also significantly different between 2 groups of overweight and obese subjects (n = 121) assigned to either increase dairy to 3–5 servings/d or to continue their habitual intake of <2 servings/d over a 6-mo period (18). However, this change was due solely to increased insulin concentrations in the control group in Norway in the absence of weight change. In studies with normal-weight volunteers, high dairy consumption appeared to have no effect (19, 20), whereas for overweight and obese subjects, some studies showed an improvement in homeostasis model assessment with higher dairy intake (17, 21), and other studies reported no difference (22, 23). Our increase in dairy consumption was similar to increases in these studies. Although the amounts consumed in these studies were higher than Australian National Health and Medical Research Council dietary guidelines, which recommend 2.5 servings of dairy each day (24), typical intake in Australia is <2 servings/d (25). Our data were partly consistent with that of Chiu et al. (26) who showed, in a large study, that increasing predominantly dairy fat and dairy protein did not improve insulin sensitivity. Although no impairment of insulin sensitivity was shown, dairy branched-chain amino acids (BCAAs) were related to fasting insulin concentrations and insulin clearance.

Whey proteins in dairy foods were shown to increase serum insulin concentrations more than casein or other animal and plant proteins (27, 28). This finding may have been due to the activation of the incretin hormones glucagon-like peptide-1 and glucose-dependent insulinotropic polypeptide by BCAAs, known to enhance insulin secretion (29). Whey protein consumed before or with a carbohydrate meal was shown to substantially increase the AUC for insulin, glucagon-like peptide-1, and glucose-dependent insulinotropic polypeptide (P < 0.05 in people with diet-controlled T2D (30). In contrast with our study, after a chronic 12-wk parallel intervention study, Pal et al. (21) noted a decrease in fasting plasma insulin concentrations after whey supplementation compared with a glucose control. However, within the Framingham cohort, higher fasting concentrations of BCAAs along with tyrosine and phenylalanine were higher in subjects who later developed diabetes than in their matched controls (31). Other metabolomic studies also confirmed a correlation between

<table>
<thead>
<tr>
<th>TABLE 3</th>
<th>Incremental AUC for glucose, insulin, and C-peptide by glucose-tolerance group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Red meat</td>
</tr>
<tr>
<td>Glucose</td>
<td>6.6 ± 4.5</td>
</tr>
<tr>
<td>Insulin</td>
<td>118.2 ± 76.9</td>
</tr>
<tr>
<td>C-peptide</td>
<td>19.03 ± 6.08</td>
</tr>
</tbody>
</table>

\(^1\)All values are means ± SDs. No effect of diet or diet by group was shown for the incremental AUC (3-diet repeated measures ANOVA by glucose-tolerance group). Between-group difference: glucose, P < 0.01. NS for insulin or C-peptide. IFG, impaired fasting glucose; IGT, impaired glucose tolerance; NGT, normal glucose tolerance.

<table>
<thead>
<tr>
<th>TABLE 4</th>
<th>Dietary intake from weighed food records</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Red meat</td>
</tr>
</tbody>
</table>

\(^1\)All values are means ± SDs. Means that do not share a common superscript letter were significantly different at P < 0.05 (2-diet repeated-measures ANOVA).
raised BCAA and insulin resistance (32, 33). The leucine content is comparable between dairy and red meat (34), and in the current study, protein intake did not differ between dairy and red meat diets, and thus, leucine was unlikely to be an explanation for the differences.

Dairy fat contains \( \sim 70\% \) saturated fat (35), and in healthy subjects, insulin sensitivity was significantly impaired with an SFA diet compared with a diet containing MUFAs (36); therefore, it is surprising that dairy is not associated with more T2D rather than less. Higher concentrations of dairy fat were inversely associated with fasting plasma glucose in an observational study, and an OGTT showed higher systemic and hepatic insulin sensitivity in high-dairy consumers (37). A trial in overweight individuals showed no effect when saturated fats were replaced with either MUFAs or carbohydrates (38), and a review in this area (39) similarly did not find an association between fat quality and insulin sensitivity. Only 3 of 12 interventions evaluated showed a negative effect of saturated fat; and thus, it is not clear that dairy saturated fat would be adverse. Food diaries indicated 4% significantly higher saturated fat intake during the dairy diet, but an analysis of serum lipids or fatty acids would be unlikely to detect this difference. Carbohydrate intake was similarly higher in the dairy diet than in either the red meat or control diets. Adjustment for carbohydrate differences in this study did not abolish the effect.

A recent meta-analysis showed that, although higher intake of yogurt was associated with reduced risk of T2D, other dairy products showed no association (11). Participants in this study could choose from a range of dairy products including yogurt and high-fat products, and the latter may have had adverse effects. Participants were instructed to consume lean red meat and avoid processed meat, which may be why the red meat diet resulted in an insulin response that was not different from that of the control diet that contained white meat.

Glucose concentrations did not change between diets, which was consistent with the effect of increased insulin balancing the effect of increased insulin resistance. The IFG and IGT group was not more sensitive to dairy than the NGT group, which suggested that dietary recommendations should cover all individuals.

The sex difference was not expected, and the reasons were unclear but not related to glucose-tolerance group or the percentage of fat mass. Bedard et al. (40) showed that only men benefited from a Mediterranean diet, and women had an increase in insulin AUC with an OGTT. Similarly, Sumner et al. (41) showed that, relative to their fat-free mass, African American women were more insulin resistant than were men. A 14% reduction in insulin sensitivity was seen in women in this study with dairy. If maintained, this reduction would increase risk of T2D by a similar amount in the population genetically at risk, which may be one-third of the whole population, leading to perhaps a 5% increase in T2D incidence.

One of the strengths of this study was the crossover design with each participant serving as their own control. This was a free-living study and, as far as is possible to determine from the checklists and diaries provided, adherence to the protocol was good. Although the checklists indicated that compliance to the protocol was met, food diaries indicated that overall energy intake was significantly higher in the dairy diet than both red meat and control diets; however, the weight gain was minimal and unrelated to insulin sensitivity. Self-reporting of food intake is a limitation, as underreporting is a common issue across all methods of dietary accounting (42), and it was possible that participants underreported dietary intake in red meat and control diets or, perhaps, may have overestimated dairy consumption to meet the expectations of the protocol. An analysis of biomarkers of dairy consumption may also be useful to assess compliance; however, this analysis was not performed in this study.

In conclusion, in contrast to some epidemiologic findings, these results suggest that high consumption of mixed dairy reduces insulin sensitivity compared with that of a diet high in lean red meat in this population of overweight and obese individuals. Interventions with yogurt only are required.

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The authors’ responsibilities were as follows—PMC: designed the research; KMT: conducted the research and had primary responsibility for the final content of the manuscript; KMT and PMC: analyzed data; and all authors: wrote the manuscript and read and approved the final manuscript. None of the authors reported a conflict of interest related to the study.

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