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## Low-Energy Dense Potato- and Bean-Based Diets Reduce Body Weight and Insulin Resistance: A Randomized, Feeding, Equivalence Trial

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**ABSTRACT** We evaluated the effect of diets low in energy density (1 kcal/g) and high in either potatoes (Potato) or pulses (Bean) on blood glucose control in participants with insulin resistance. We hypothesized that the Potato and Bean diets would have equivalent effects. This was an 8-week randomized, parallel design, controlled feeding study comparing Potato and Bean diets (50–55% carbohydrate, 30–35% fat, 15–20% protein). Equivalence was prespecified as the mean change in the blood glucose concentration for Potato that was within  $\pm 20\%$  of the Bean diet. Thirty-six participants (age: 18–60 years, body mass index: 25–40 kg/m<sup>2</sup>) with insulin resistance (homeostatic model assessment of insulin resistance [HOMA-IR] >2) were enrolled. Body weight was measured, and subjects underwent a mixed meal tolerance test at baseline and after 8 weeks. Intent-to-treat (ITT) and completer analyses were conducted. Equivalence between the two diets in the area under the curve for serum glucose was attained within  $\pm 10\%$ , but the reduction from baseline was not statistically significant. For the Bean diet, insulin (area under the response curve:  $-2136.3 \pm 955.5$  mg/[dL·min],  $P = .03$ ) and HOMA-IR ( $-1.4 \pm 0.6$ ,  $P = .02$ ) were lower compared with baseline. ITT and completer analyses were similar, except that HOMA-IR was also reduced by the Potato diet ( $-1.3 \pm 0.6$ ,  $P < .05$ ). Compliance with the diets was 87–88%, and body weight was reduced in both diets (Potato:  $-5.6\% \pm 0.6\%$ ; Bean:  $-4.1\% \pm 0.6\%$ ,  $P < .001$ ) with no significant difference between the two diets. Potato and Bean diets low in energy density were equally effective in reducing insulin resistance and promoting weight loss in individuals with impaired blood glucose control.

**Clinical Trial:** The trial was registered with ClinicalTrials.gov Identifier: NCT04203238.

**KEYWORDS:** • Beans • energy density • glucose • insulin • lipids • Potato

### INTRODUCTION

ENERGY DENSITY REFERS to the amount of energy in a particular weight of food or the kcal/g of food. The role of energy density in regulating food intake was recognized and tested in a number of studies conducted in the 1990s.<sup>1</sup> In most of these studies, dietary advice was offered, acute effects were evaluated, fat mimetics were used, or participants were provided with a selection of foods, which they had to incorporate into their meal plans.<sup>2,3</sup> The positive

outcomes notwithstanding, the true effect of modulating the energy density of the diet warranted investigation in a more controlled fashion where foods customarily consumed are provided to participants to meet their energy needs. Furthermore, the effect of reducing the energy density for individuals with insulin resistance, which frequently accompanies obesity has never been investigated.

Potatoes have negligible fat and a low energy density.<sup>4</sup> Although potatoes are generally considered a high glycemic index food,<sup>5</sup> cooling of gelatinized potatoes generates appreciable levels of slowly digested starch (resistant starch type 3) and substantially lowers the blood glucose response that potatoes elicit.<sup>6–9</sup> Nevertheless, most but not all epidemiological studies have grouped potatoes in the “less healthy plant food” category and associated its intake with weight gain and increased risk for type 2 diabetes.<sup>10–16</sup>

Manuscript received 5 December 2021. Revision accepted 15 September 2022.

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Like potatoes, pulses (dry beans and peas) contain resistant starch, which improves insulin sensitivity and glucose tolerance.<sup>17</sup> The United States dietary guidelines and the evidence in the scientific literature unequivocally support increased intake of pulses.<sup>18,19</sup> A systematic review and meta-analysis of 41 randomized controlled clinical trials provided evidence that pulses as part of a low glycemic index diet improve blood glucose control.<sup>20</sup>

Research suggests that people tend to eat a constant weight of food.<sup>21,22</sup> Foods vary in many ways and environmental influences confound our ability to judge how much to eat. The weight of food is a cue that often guides consumption.<sup>21</sup> We investigated the effect of a diet low in energy density (0.6–1.5 kcal/g)<sup>23</sup> on blood glucose control in individuals with insulin resistance. We hypothesized that potatoes can be used to enhance the health benefits of the diet and will be equivalent to pulses in controlling blood glucose and lipids. We followed the Extension of the 2010 CONSORT statement for reporting of equivalence in randomized trials with the Potato diet as the group under evaluation and the Bean diet as the reference group.<sup>24</sup>

## MATERIALS AND METHODS

### Study design

An 8-week randomized parallel trial in free-living participants was conducted between September 2019 and May 2021 at the Pennington Biomedical Research Center (PBRC) in Baton Rouge, Louisiana. The participants were randomized to consume a diet high in white potatoes (Potato) or pulses (Bean) for 8 weeks. The randomization was conducted by the study biostatistician using a uniform random-number generator. The diet assignment was kept in a sealed envelope and assigned to the participant by the study dietitian. Participants were blinded but they were likely able to identify the diet assignment because of the nature of the diet. The investigators, study staff, and sponsors were masked to the study treatment assignments. Participants met with the study dietitian weekly to receive study foods and to be weighed. Nutrition counseling was provided weekly to facilitate adherence to the intervention.

### Study population

Participants included men and women 18–60 years of age who were overweight or had obesity (body mass index [BMI]  $\geq 25$  and  $\leq 40$  kg/m<sup>2</sup>) and waist circumference  $> 88$  cm (if female) and 102 cm (if male). Previously, consumption of pulses has been shown to improve glycemia compared with control in participants with newly diagnosed type 2 diabetes.<sup>25</sup> In our study, we included participants with insulin resistance (homeostatic model assessment of insulin resistance [HOMA-IR]  $> 2.0$ ). This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by PBRC Institutional Review Board. All partic-

TABLE 1. PARTICIPANT CHARACTERISTICS AT SCREENING

	Potato	Bean
Age <sup>a</sup>	44.6 (2.6)	45.4 (2.6)
Gender		
Females	14	14
Males	4	4
Race		
White	11	12
Non-White	7	6

<sup>a</sup>Values are mean (SE).  
SE, standard error.

ipants provided written informed consent. Participant characteristics at screening are presented in Table 1.

### Dietary intervention

The diets were developed and prepared in our metabolic kitchen and were based on a 5-day cycle menu at six levels of energy intake (1500, 1800, 2100, 2400, 2700, and 3000 kcal). The main entrées (lunch and dinner) in the Potato diet contained 3 ounces of cooked meat or fish and 2 ounces of potatoes. Each meal was served with a side of potatoes. To enhance resistance to starch and dietary fiber components, the potatoes were boiled with skins, refrigerated for 12 to 24 h before the whole potato was incorporated into the meals. Similarly, in the Bean diet, each meal contained 3 ounces of cooked meat or fish, 2 ounces of cooked pulses, and a side of bread, rice, or pasta. Participants received three main entrées with fish each week. The current study was a controlled feeding study where participants' energy needs were met with a diet that included 4 ounces/day of the reference food (pulses). In previous studies, only the pulses servings were provided and participants were instructed to incorporate it into their usual diet, or they were advised to increase pulses intake.<sup>20,26</sup>

At each calorie level, the macronutrient composition, dietary fiber, and energy densities of the two meal plans were closely matched (Table 2). The macronutrient composition of the diets was designed to meet the Acceptable Macronutrient Distribution Range (Carbohydrates: 50–55%;

TABLE 2. MACRONUTRIENT, DIETARY FIBER, AND ENERGY DENSITY OF THE DIET

Nutrient	Potato	Bean
Total fat, %	30.7	31.5
Carbohydrate, %	54.0	53.9
Protein, %	15.3	14.6
Fiber, g	35.8	37.4
Energy density, kcal/g <sup>a</sup>	1.0	1.0
Energy density, kcal/g <sup>b</sup>	1.1	1.2

Values are shown for 5-day, 2100-kcal menu cycle and are based on nutritional analysis using MENU 6.0 (Pennington Biomedical Research Center).

<sup>a</sup>Energy density with the beverages provided in the meal plan.

<sup>b</sup>Without beverages.

Protein: 15–20%; and Fat: 30–35% of energy). The nutrient compositions were analyzed using Menu 6.0 (PBRC Nutrition Analysis software).

Participants' basal metabolic rate was calculated using the Mifflin St-Jeor equation.<sup>27</sup> Energy requirements were determined as the product of basal metabolic rate and an activity factor, which ranged from 1.2 to 1.5 depending on each participant's activity level. The energy needs were not adjusted for any changes in body weight. Participants were advised to maintain their usual physical activity levels during the study. Participants returned their food containers and checked off their daily consumption from the meal plan for each day, which was reviewed by the study dietitian at the weekly visits as a measure of compliance with the intervention.

### Experimental procedures

A mixed meal tolerance test was performed after an 8-h overnight fast before starting the intervention and after 8 weeks. Participants were given a 500-kcal standardized meal containing either potatoes or pulses matched for the macronutrient composition and dietary fiber content, depending upon whether they were assigned to the Potato or Bean diet, respectively. Blood samples were obtained before the meal for analysis of biochemical measures. Participants were presented with their meal and given 10 min to eat it. Test meals were supervised to ensure that the entire meal was eaten. Before serving the mixed meal, hunger, fullness, desire to eat, and prospective intake were assessed using visual analog scales (VAS).<sup>28</sup> Blood samples were obtained at 30, 60, 90, 120, and 180 min after meal ingestion to measure glucose and insulin, and the VAS was also administered at these time points.

Adverse events were recorded throughout the study. Plasma lipoprotein particle number and size were assessed by a proton magnetic resonance spectroscopy assay (NMR LabCorp, Morrisville, NC, USA), which measures the particle concentrations of lipoprotein subclasses and average particle size of lipoproteins as previously described.<sup>29</sup> All other biochemical measures were analyzed by PBRC clinical chemistry core.

### Statistical analysis

The *a priori* primary goal was to conduct an equivalence study comparing the area under the curve (AUC) for blood glucose in the Potato and Bean groups. In a previous study, a single serving of pulses was shown to reduce the AUC for blood glucose.<sup>25</sup> In the absence of data on the insulin AUC, we assumed that the difference in glucose AUC between the two groups would be no larger than 720 mg/(dL·min) which approximates to margins of  $\pm 20\%$ .<sup>30</sup> Secondary outcomes included changes in insulin, HOMA-IR, and lipid and lipoprotein profiles. At 80% power, a sample of at least 15 subjects per group was expected to yield equivalence in glucose AUC between the 2 groups. Two one-sided tests were used to determine equivalence between the two diets. Baseline differences were assessed with two-sided *t*-tests or chi-squared tests, as appropriate.

An intent-to-treat (ITT) analysis was conducted with a linear mixed effects model to test for differences between groups over time. Estimates were based on the least square mean derived from the mixed effects model and tested with a two-sided *t*-test. We also conducted a similar analysis of participants who completed the study (completers) since the trial was interrupted by the COVID-19 pandemic and some participants had to withdraw or be withdrawn from the study (Fig. 1). Alpha of 0.05 was considered statistically significant.

## RESULTS

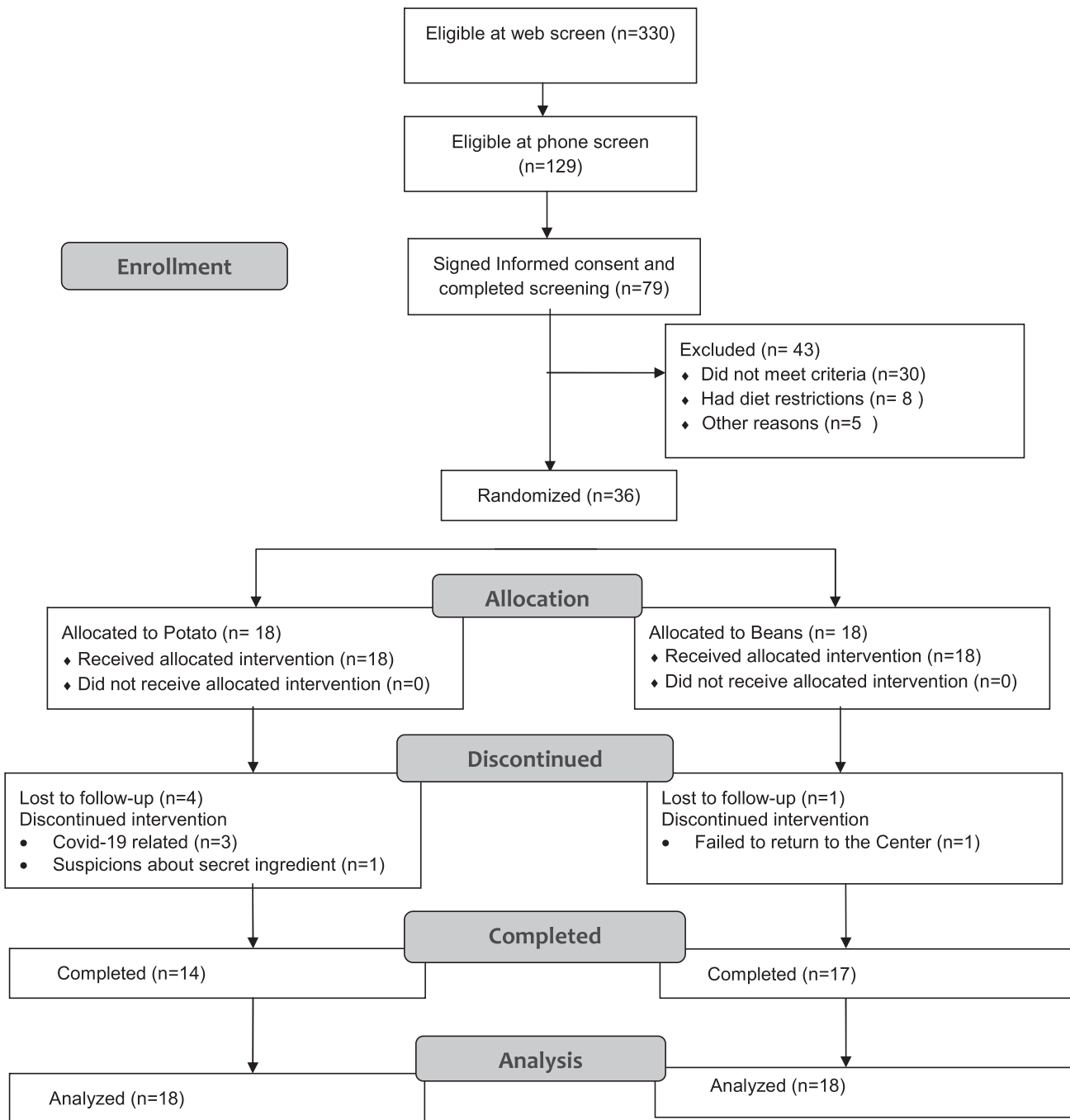
### Study participants

The study was conducted between September 2019 and May 2021. Figure 1 shows the participant flow. After clinical screening, 36 participants were enrolled, and 86% (31 participants) completed all visits. The Center closed during the pandemic in March 2020 and the study was stopped until June 2020. When the study resumed, we restarted participants (who were in progress at the time that the study stopped) from the baseline visit. One participant could not restart as her life circumstances had changed. Subsequently, two participants had to be dropped from the study due to COVID-19. Two participants withdrew from the study for personal reasons.

The distribution of age, gender, and race was not significantly different between the groups (Table 1). Two participants in the Bean diet group reported mild-to-moderate gastrointestinal effects (flatulence, cramps, and diarrhea). These effects resolved and participants completed the study. The results of the ITT and completer analyses were similar, except for HOMA-IR. We present herein the ITT analysis, except for HOMA-IR, which includes the ITT and completer analyses. Compliance with the diet was 87% in the Potato diet and 88% in the Bean diet.

### Effect of the diets on serum glucose and insulin, body weight, and BMI

The statistical evaluation of the treatment on measurements relating to glycemia, body weight, and blood pressure are presented in Table 3. The change in the AUC for serum glucose concentrations in the Potato diet was within equivalence margins of  $\pm 10\%$  of the Bean diet (90% confidence interval:  $-6.02$  to  $4.18$ , Fig. 2), although the Bean diet did have a significantly higher glucose AUC at baseline compared with the Potato diet (difference at baseline:  $2045.4 \pm 618.6$ ,  $P = .003$ ). In both diets, fasting glucose concentrations were in the normal range at baseline ( $< 100$  mg/dL),<sup>31</sup> and there was no significant reduction from baseline in the AUC for serum glucose concentrations. In both diets, baseline fasting insulin concentration was  $> 10.6$   $\mu$ U/mL, which is considered insulin resistance.<sup>32</sup> Compared with baseline, the insulin response (AUC:  $-2136.3 \pm 955.5$  mg/[dL·min],  $P = .034$ ) was reduced for the Bean diet and equivalence was attained within  $\pm 21\%$  (90% confidence interval:  $-20.80$  to  $15.44$ , Fig. 2).



**FIG. 1.** Consort diagram of enrollment and follow-up.

The HOMA-IR was reduced in response to the treatment in both diets and statistical significance was attained with the Bean diet in both the ITT and completer analyses. The HOMA-IR attained significance in response to the Potato diet only in the completer analysis (Fig. 3A–D).

Significant treatment effects were observed for body weight such that Potato and Bean diets produced reductions in body weight (Potato:  $-5.82 \pm 0.70$  kg; Bean:  $-4.0 \pm 0.63$  kg,  $P < .001$ ) and BMI (Potato:  $-2.04 \pm 0.22$  kg/m<sup>2</sup>; Bean:  $-1.35 \pm 0.20$  kg/m<sup>2</sup>,  $P < .001$ ). Although there were no baseline differences in body weight between the two diets, BMI at

baseline was higher and the reduction in response to the treatment was significantly greater in the Potato diet compared with the Bean diet (difference at baseline:  $2.93 \pm 1.19$  kg/m<sup>2</sup>,  $P = .02$ ; difference in postintervention change:  $-0.70 \pm 0.29$  kg/m<sup>2</sup>,  $P = .03$ ). The percent change in body weight is presented in Figure 4.

#### *Effect of the diets on blood lipids and lipoproteins*

The Bean diet produced a significant reduction in total cholesterol compared with baseline ( $-11.79 \pm 3.25$  mg/dL,

TABLE 3. INTENT-TO-TREAT ANALYSES (N=36) OF THE EFFECT OF DIET ON BODY WEIGHT, BODY MASS INDEX, GLUCOSE, INSULIN, HIGH-SENSITIVITY C-REACTIVE PROTEIN, AND BLOOD PRESSURE

Outcome	Baseline potato		Baseline bean		Baseline P-value, Potato versus Bean		Postintervention Potato		Postintervention Bean		P-value, Potato versus baseline		P-value, Bean versus baseline		Postintervention change P-value, Potato versus Bean	
	SE	Mean	SE	Mean	P-value	SE	SE	Mean	SE	Mean	SE	P-value	SE	P-value	SE	P-value
Body weight, kg	100.6	3.63	95.21	3.63	0.3	3.65	94.78	3.65	<0.001 <sup>a</sup>	91.21	3.65	<0.001 <sup>a</sup>	3.65	<0.001 <sup>a</sup>	3.65	0.061
BMI, kg/m <sup>2</sup>	35.64	0.84	32.72	0.84	0.02 <sup>a</sup>	0.85	33.6	0.85	<0.001 <sup>a</sup>	31.37	0.84	<0.001 <sup>a</sup>	0.84	<0.001 <sup>a</sup>	0.84	0.026 <sup>a</sup>
Glucose AUC, mg/(dL·min)	17679	437.42	19725	437.42	0.003 <sup>a</sup>	475.26	17576	475.26	0.812	19223	454.36	0.228	454.36	0.228	454.36	0.506
Insulin AUC, $\mu$ U/(mL·min)	7708.9	1152.8	10942	1152.8	0.057	1236.2	6742.3	1236.2	0.348	8805.51	1190	0.034 <sup>a</sup>	1190	0.034 <sup>a</sup>	1190	0.408
HOMA-IR	3.86	0.6	4.7	0.6	0.334	0.66	2.75	0.66	0.075	3.28	0.61	0.017 <sup>a</sup>	0.61	0.017 <sup>a</sup>	0.61	0.715
hsCRP, mg/L	6.55	1.52	5.97	1.52	0.79	1.64	7.06	1.64	0.725	6.21	1.55	0.858	1.55	0.858	1.55	0.891
SBP, mmHg	117.07	2.66	113.18	2.66	0.312	2.91	114.13	2.91	0.4	118.33	2.74	0.124	2.74	0.124	2.74	0.098
DBP, mmHg	72.07	1.99	69.28	1.8	0.762	2.02	65.64	2.02	0.089	69.85	1.85	0.706	1.85	0.706	1.85	0.137

Values are mean and SE.

<sup>a</sup>Significantly different values ( $P < .05$ ).

AUC, area under the curve; BMI, body mass index; DBP, diastolic blood pressure; HOMA-IR, homeostatic model assessment of insulin resistance; hsCRP, high-sensitivity C-reactive protein; SBP, systolic blood pressure.

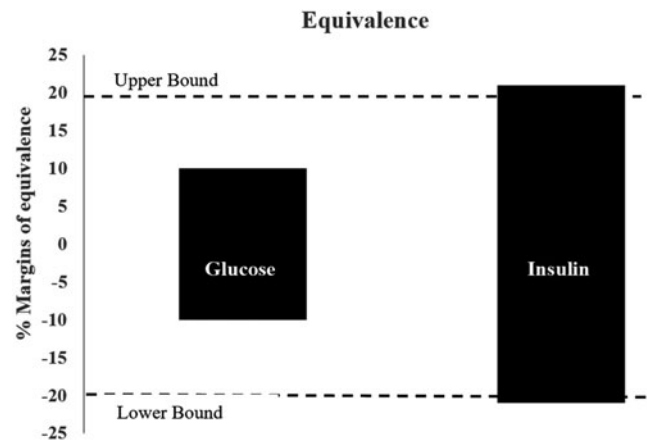


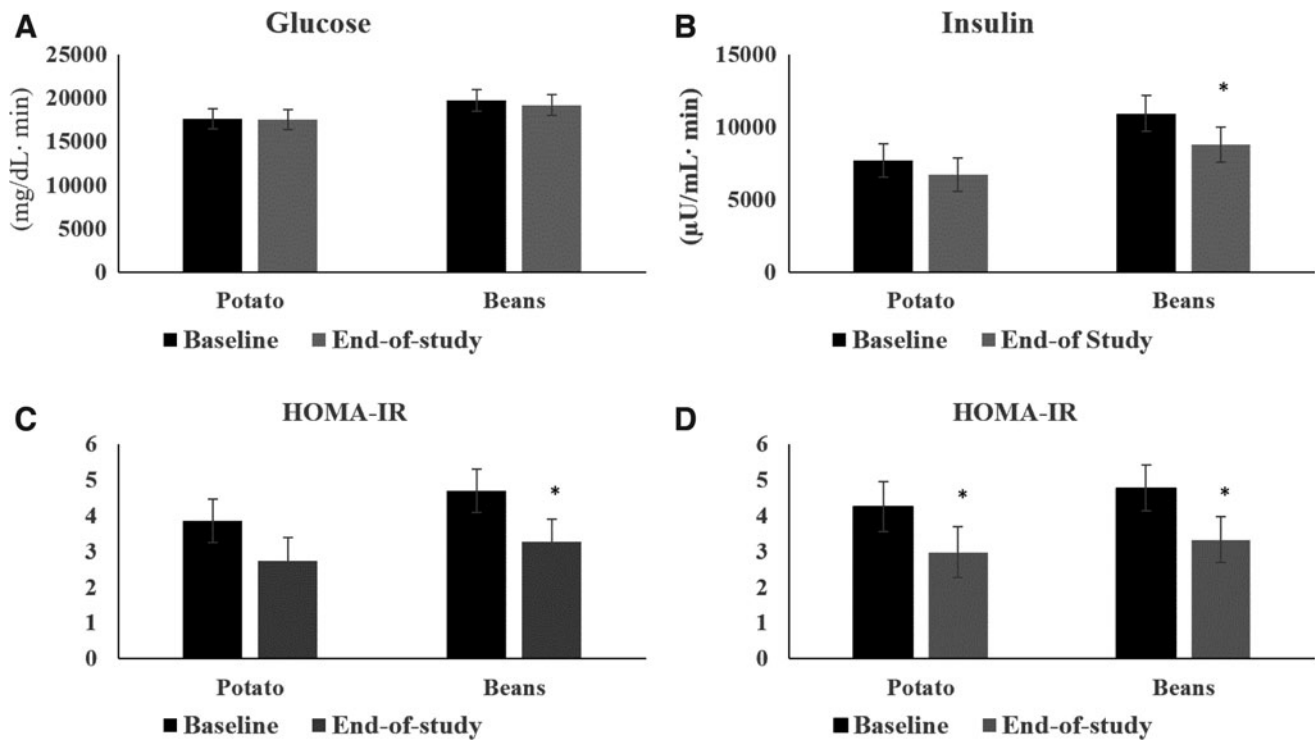
FIG. 2. Equivalence in change in the area under the curve for glucose and insulin between the Potato and Bean diets.

$P = .001$ ). There were no treatment effects for serum concentrations of low-density lipoprotein (LDL) cholesterol, but LDL particle size was reduced significantly in both diets with no difference between diets (Potato:  $-0.27 \pm 0.13$  nm,  $P = .04$ ; Bean:  $-0.38 \pm 0.12$  nm,  $P = .003$ ). Particle concentrations of large LDL decreased in both diets (Potato:  $-104.45 \pm 28.87$  nmol/L,  $P = .001$ ; Bean:  $-72.40 \pm 26.40$  nmol/L,  $P = .010$ ). High-density lipoprotein (HDL) cholesterol (Potato:  $-5.17 \pm 1.56$  mg/dL,  $P = .002$ ; Bean:  $-6.04 \pm 1.42$  mg/dL,  $P < .001$ ) and total particle concentrations were reduced in both diets with no difference between diets. Medium very low-density lipoprotein (VLDL) particle concentration increased significantly in the Potato diet with no difference between the diets (Table 4). There were no differences in the satiety measures.

## DISCUSSION

The Potato and Bean diets were equivalent in their effect on the blood glucose response, although the reductions from baseline were not statistically significant. However, both diets improved measures of insulin resistance. Both diets resulted in significant reductions in body weight and BMI without affecting appetite. Although serum total cholesterol concentrations were reduced in the Bean diet, the two diets did not affect serum LDL cholesterol concentrations, but reduced LDL particle size. There were no significant treatment differences between the diets in body weight, other serum measures, or satiety.

To our knowledge, this is the first randomized trial investigating the effect of potatoes as part of a daily meal pattern for 8 weeks on metabolic outcomes in a controlled feeding setting. We demonstrated that contrary to observations from epidemiological studies, potatoes do not adversely affect the glycemic response. Indeed, consistent with studies showing that consumption of potatoes with meat or fat reduces the glycemic response,<sup>7,8,30</sup> the Potato diet reduced HOMA-IR in the analysis of participants who completed the study. Compared with baseline, the Bean diet



**FIG. 3.** Observed least squares mean and SE in the area under the curve for (A) serum glucose (B) serum insulin,  $*P = .034$ ; (C) intent-to-treat analysis of HOMA-IR at baseline and after 8 weeks,  $*P = .017$ . Potato versus Beans,  $P = .715$ . (D) Completers analysis of HOMA-IR at baseline and after 8 weeks,  $*P < .05$ . Potato versus Beans,  $P = .839$ . HOMA-IR, homeostatic model assessment of insulin resistance; SE, standard error.

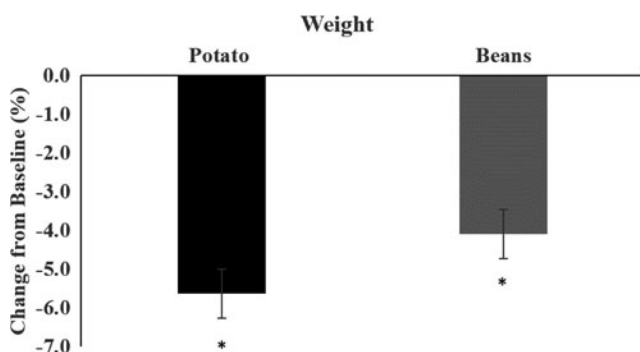
reduced the insulinemic response and HOMA-IR, which supports the results of randomized controlled trials of the metabolic effects of beans.<sup>20,26</sup> The serum glucose concentrations were in the normal range at baseline in both diets, but insulin concentrations were in the insulin resistance range.<sup>31,32</sup> These baseline concentrations may explain why there was no change in the AUC for glucose, but the AUC for insulin and HOMA-IR were reduced.

We demonstrated a reduction in body weight in individuals with insulin resistance by reducing the energy density of the diet with potatoes or beans. The results are consistent with previous studies on energy density ranging from 3 days to 3 weeks demonstrating reductions in energy in-

take, as well as longer term studies demonstrating weight loss in generally healthy individuals.<sup>2,3</sup> Metabolizable energy content of mixed diets have been shown to decrease by 7.2 kcal/g of total dietary fiber intake.<sup>33</sup> Thus, the high dietary fiber content of the diet coupled with reduced intake (87–88% completion of meals) may have contributed to a reduction in total daily energy intake and resulted in weight loss. Although both diets led to a reduction in body weight, the Potato diet reduced BMI compared with the Bean diet. By contributing to reducing the energy density of the diet, potatoes and beans promoted weight loss without affecting appetite and without the need for calorie restriction. This weight loss if sustained over time could have a substantial impact on body weight.<sup>34</sup>

Triglyceride (TG) concentrations were well within the normal range in participants at baseline and did not change in response to the two diets. The lipoprotein pathway arising from low TGs leads to the assembly and secretion of either large or medium VLDL cholesterol that result in large and medium LDL cholesterol, respectively.<sup>35</sup> This pathway is juxtaposed to the atherogenic pathway, where high TGs drive secretion of large TG-enriched VLDL particles that stimulate the exchange of cholesteryl esters from HDL and LDL for VLDL-TG. The TG-enriched LDL then undergo lipolysis and become small and dense.<sup>36</sup>

We observed an increase in medium VLDL particle concentration reminiscent of the nonatherogenic pathway in both diets and statistical significance was attained in the Potato diet. Unfortunately, the methodology used in this



**FIG. 4.** Observed least squares mean and SE percentage change from baseline in body weight at 8 weeks in the Potato and Bean diet,  $*P < .001$ . Potato versus Beans,  $P = .684$ .

TABLE 4. INTENT-TO-TREAT ANALYSES (N=36) OF THE EFFECT OF DIET ON LIPIDS AND LIPOPROTEINS

Lipid or lipoprotein	Baseline Potato		Baseline Bean		Baseline P-value, Potato versus Bean		Postintervention Potato		Postintervention Bean		P-value, Potato versus baseline		P-value, Bean versus baseline		Postintervention change P-value, Potato versus Bean		
	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	
Triglycerides, mg/dL	104.94	15.85	100.56	15.9	0.846	100.28	17.7	172.77	17.7	0.81	97.68	16.24	0.874	0.946	175.6	7.21	0.001 <sup>a</sup>
Total cholesterol, mg/dL	177.78	7.17	187.39	7.17	0.351	172.77	7.35	172.77	7.35	0.171	175.6	7.21	0.001 <sup>a</sup>	0.171	175.6	7.21	0.001 <sup>a</sup>
LDL cholesterol, mg/dL	111.38	6.24	119.86	6.21	0.344	109.22	6.35	109.22	6.35	0.472	114.76	6.24	0.06	0.461	114.76	6.24	0.06
HDL cholesterol, mg/dL	48.58	2.33	47.42	2.33	0.729	43.4	2.44	43.4	2.44	0.002 <sup>a</sup>	41.38	2.36	<0.001 <sup>a</sup>	0.684	41.38	2.36	<0.001 <sup>a</sup>
Non-HDL cholesterol, mg/dL	131.72	6.58	141.11	6.58	0.322	132.12	6.83	132.12	6.83	0.922	136.42	6.63	0.208	0.354	136.42	6.63	0.208
apoB, mg/dL	94.39	3.8	101.94	3.8	0.532	94.28	4.02	94.28	4.02	0.969	99.36	3.85	0.335	0.353	99.36	3.85	0.335
VLDL size, nm	50.56	1.9	49.77	1.85	0.769	50	2.01	50	2.01	0.733	50	1.91	0.881	0.725	50	1.91	0.881
LDL size, nm	20.56	0.14	20.77	0.14	0.305	20.29	0.15	20.29	0.15	0.038 <sup>a</sup>	20.39	0.14	0.003 <sup>a</sup>	0.554	20.39	0.14	0.003 <sup>a</sup>
HDL size, nm	9.51	0.09	9.42	0.09	0.538	9.44	0.1	9.44	0.1	0.415	9.32	0.1	0.154	0.707	9.32	0.1	0.154
Lipoprotein particle concentration																	
VLDL, nmol/L	43.3	5.04	45.73	5.04	0.74	49.03	5.39	49.03	5.39	0.2	49.82	5.12	0.313	0.78	49.82	5.12	0.313
Total VLDL and chylomicron																	
Large	4.53	0.92	4.65	0.92	0.926	4.95	0.99	4.95	0.99	0.618	4.51	0.93	0.856	0.624	4.51	0.93	0.856
Medium	10.49	2.39	12.68	2.39	0.521	16.05	2.57	16.05	2.57	0.016 <sup>a</sup>	15.69	2.43	0.143	0.395	15.69	2.43	0.143
Small	28.28	3.36	28.39	3.36	0.982	28.36	3.67	28.36	3.67	0.982	29.6	3.43	0.707	0.811	29.6	3.43	0.707
IDL, nmol/L	126.89	24.53	161.83	24.5	0.322	237.9	27.5	237.9	27.5	0.002 <sup>a</sup>	202.03	25.18	0.186	0.114	202.03	25.18	0.186
LDL, nmol/L																	
Total	1008.17	61.64	1118.17	61.6	0.217	1050.03	66.6	1050.03	66.6	0.477	1144.88	62.7	0.622	0.849	1144.88	62.7	0.622
Large	253.78	42.53	313.89	42.5	0.326	149.33	44.5	149.33	44.5	0.001 <sup>a</sup>	241.49	42.94	0.010 <sup>a</sup>	0.419	241.49	42.94	0.010 <sup>a</sup>
Small	627.33	51.04	642.28	51	0.837	661.78	55	661.78	55	0.469	699.48	51.89	0.196	0.724	699.48	51.89	0.196
HDL, $\mu$ mol/L																	
Total	32.82	1.13	31.19	1.13	0.319	28.19	1.25	28.19	1.25	<0.001 <sup>a</sup>	26.94	1.16	<0.001 <sup>a</sup>	0.822	26.94	1.16	<0.001 <sup>a</sup>
Large	7.32	0.58	6.64	0.58	0.416	6.55	0.6	6.55	0.6	0.067	5.63	0.58	0.01 <sup>a</sup>	0.651	5.63	0.58	0.01 <sup>a</sup>
Medium	12.26	1.35	11.19	1.35	0.58	9.51	1.5	9.51	1.5	0.094	9	1.38	0.147	0.8	9	1.38	0.147
Small	13.26	1.32	13.37	1.32	0.953	12.1	1.45	12.1	1.45	0.435	12.31	1.35	0.443	0.96	12.31	1.35	0.443
Lipoprotein ratios																	
Total cholesterol:HDL	3.83	0.2	4.02	0.2	0.512	4.1	0.21	4.1	0.21	0.035 <sup>a</sup>	4.34	0.2	0.009 <sup>a</sup>	0.791	4.34	0.2	0.009 <sup>a</sup>
Triglycerides:HDL	2.59	0.51	2.29	0.51	0.682	2.46	0.57	2.46	0.57	0.839	2.49	0.52	0.743	0.71	2.49	0.52	0.743

Values are mean and SE.

<sup>a</sup>Significantly different values ( $P < .05$ ).

HDL, high-density lipoprotein; LDL, low-density lipoprotein; IDL, intermediate density lipoprotein; VLDL, very low-density lipoprotein.

study provides an average of the medium and small particles (small LDL) and although it changed in the right direction, the analysis did not specifically provide medium LDL concentrations.<sup>29</sup> However, like published observations for the dietary approaches to stop hypertension (DASH) diet, large LDL particle concentration decreased, and the overall effect was a reduction in LDL particle size.<sup>37</sup> Similarly, the Mediterranean diet also reduces LDL particle size.<sup>38</sup> Importantly, the high-fat DASH diet, which is composed of 14% and 47% of the energy content from saturated fat and carbohydrate, respectively, increases LDL particle size compared with the DASH diet (9% and 55% of energy from saturated fat and carbohydrate, respectively).<sup>37</sup> Dietary carbohydrates have been shown to drive the secretion of large TG-enriched particles that undergo intravascular lipolysis and remodeling to generate small atherogenic LDL.<sup>35</sup>

We hypothesize that reducing the carbohydrate content of the DASH diet as well as the low energy–dense diet of the present study (14% and 55% of energy from saturated fat and carbohydrate, respectively) will likely increase LDL particle size and warrants investigation. HDL concentrations reduced in both groups expectedly with weight loss, which has been observed in several studies.<sup>39–41</sup>

Despite the demonstration of beneficial metabolic outcomes, the relatively short time frame of 8 weeks is a limitation of the study. An equivalence study between a food associated with adverse outcomes compared with one that has health benefits is a plausible comparison, but the addition of a typical Western diet would have enhanced our understanding of the effect of low energy–dense diets on metabolic outcomes. The study was a randomized trial, but there were differences in BMI and fasting insulin between the Potato and Bean groups at baseline. Although our primary outcome was the change from baseline in each diet, baseline values appear to affect the outcome.

In conclusion, data from this study indicate that reducing the energy density of the diet by incorporating potatoes or pulses improves the insulinemic response and promotes weight loss in individuals with insulin resistance. The reductions in LDL particle size and serum HDL concentrations are consistent with other healthy eating patterns such as the DASH diet. Whether the carbohydrate contents of these healthy eating patterns adversely affect the lipoprotein profile warrants evaluation in future studies.

#### AUTHORS' CONTRIBUTIONS

C.J.R., J.P.K., and F.L.G. designed research (project conception, development of overall research plan, and study oversight); C.J.R., K.C.A., and K.K.H. conducted research; and R.A.B. performed the statistical analysis. K.K.H. works now for Novo Nordisk, Inc. with a business address 800 Scudders Mill Road, Plainsboro NJ 08536.

#### AUTHOR DISCLOSURE STATEMENT

No competing financial interests exist.

#### FUNDING INFORMATION

This work was supported in part by an investigator-initiated grant from the Alliance for Potato Research and Education and in part by a grant from the National Institute on Aging (Candida J. Rebello, 5K99AG065419-02) and from the National Institute of General Medical Sciences of the National Institutes of Health, which funds the Louisiana Clinical and Translational Science Center (John P. Kirwan, U54 GM104940). The funders (Alliance for Potato Research and Education and the National Institutes of Health) had no role in the design, analysis, or writing of this article. The content is solely the responsibility of the authors and does not necessarily represent the official views of the sponsors or the National Institutes of Health.

#### REFERENCES

1. Rolls BJ. Dietary energy density: Applying behavioural science to weight management. *Nutr Bull* 2017;42(3):246–253; doi: 10.1111/mbu.12280
2. Karl JP, Roberts SB. Energy density, energy intake, and body weight regulation in adults. *Adv Nutr* 2014;5(6):835–850; doi: 10.3945/an.114.007112
3. Poulsen SK, Due A, Jordy AB, et al. Health effect of the New Nordic Diet in adults with increased waist circumference: A 6-mo randomized controlled trial. *Am J Clin Nutr* 2014;99(1):35–45; doi: 10.3945/ajcn.113.069393
4. King JC, Slavin JL. White potatoes, human health, and dietary guidance. *Adv Nutr* 2013;4(3):393S–401S; doi: 10.3945/an.112.003525
5. McGill CR, Kurilich AC, Davignon J. The role of potatoes and potato components in cardiometabolic health: A review. *Ann Med* 2013;45(7):467–473; doi: 10.3109/07853890.2013.813633
6. Fernandes G, Velangi A, Wolever TM. Glycemic index of potatoes commonly consumed in North America. *J Am Diet Assoc* 2005;105(4):557–562; doi: 10.1016/j.jada.2005.01.003
7. Collier G, O'Dea K. The effect of co-ingestion of fat on the glucose, insulin, and gastric inhibitory polypeptide responses to carbohydrate and protein. *Am J Clin Nutr* 1983;37:941–944; doi: 10.1093/ajcn/37.6.941
8. Collier G, McLean A, O'Dea K. Effect of co-ingestion of fat on the metabolic responses to slowly and rapidly absorbed carbohydrates. *Diabetologia* 1984;26(1):50–54; doi: 10.1007/BF00252263
9. Leeman M, Ostman E, Bjorck I. Vinegar dressing and cold storage of potatoes lowers postprandial glycaemic and insulinemic responses in healthy subjects. *Eur J Clin Nutr* 2005;59(11):1266–1271; doi: 10.1038/sj.ejcn.1602238
10. Satija A, Bhupathiraju SN, Spiegelman D, et al. Healthful and unhealthful plant-based diets and the risk of coronary heart disease in U.S. adults. *J Am Coll Cardiol* 2017;70(4):411–422; doi: 10.1016/j.jacc.2017.05.047
11. Satija A, Bhupathiraju SN, Rimm EB, et al. Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: Results from three prospective cohort studies. *PLoS Med* 2016;13(6):e1002039; doi: 10.1371/journal.pmed.1002039
12. Schulze MB, Fung TT, Manson JE, et al. Dietary patterns and changes in body weight in women. *Obesity (Silver Spring)* 2006;14(8):1444–1453; doi: 10.1038/oby.2006.164



13. Mozaffarian D, Hao T, Rimm EB, et al. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med* 2011;364(25):2392–2404; doi: 10.1056/NEJMoa1014296
14. Muraki I, Rimm EB, Willett WC, et al. Potato consumption and risk of type 2 diabetes: Results from three prospective cohort studies. *Diabetes Care* 2016;39(3):376–384; doi: 10.2337/dc15-0547
15. Darooghegi Mofrad M, Milajerdi A, Sheikhi A, et al. Potato consumption and risk of all cause, cancer and cardiovascular mortality: A systematic review and dose-response meta-analysis of prospective cohort studies. *Crit Rev Food Sci Nutr* 2020;60(7):1063–1076; doi: 10.1080/10408398.2018.1557102
16. Borch D, Juul-Hindsøgaard N, Veller M, et al. Potatoes and risk of obesity, type 2 diabetes, and cardiovascular disease in apparently healthy adults: A systematic review of clinical intervention and observational studies. *Am J Clin Nutr* 2016;104(2):489–498; doi: 10.3945/ajcn.116.132332
17. Jenkins DJ, Kendall CW, Augustin LS, et al. High-complex carbohydrate or lente carbohydrate foods? *Am J Med* 2002; 113(Suppl 9B):30S–37S; doi: 10.1016/s0002-9343(01)00989-5
18. Havemeier S, Erickson J, Slavin J. Dietary guidance for pulses: The challenge and opportunity to be part of both the vegetable and protein food groups. *Ann N Y Acad Sci* 2017;1392(1):58–66; doi: 10.1111/nyas.13308
19. USDA. Dietary Guidelines for Americans 2020–2025. 2020. Available from: [https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary\\_Guidelines\\_for\\_Americans-2020-2025.pdf](https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf) [Last Accessed: August 13, 2021].
20. Sievenpiper JL, Kendall CW, Esfahani A, et al. Effect of non-oil-seed pulses on glycaemic control: A systematic review and meta-analysis of randomised controlled experimental trials in people with and without diabetes. *Diabetologia* 2009;52(8):1479–1495; doi: 10.1007/s00125-009-1395-7
21. Stubbs RJ, Whybrow S. Energy density, diet composition and palatability: Influences on overall food energy intake in humans. *Physiol Behav* 2004;81(5):755–764; doi: 10.1016/j.physbeh.2004.04.027 S0031938404001751
22. Rebello CJ, Johnson WD, Martin CK, et al. Instant oatmeal increases satiety and reduces energy intake compared to a ready-to-eat oat based breakfast cereal: A randomized crossover trial. *J Am Coll Nutr* 2016;35(1):41–49; doi: 10.1080/07315724.2015.1032442
23. Smethers AD, Rolls BJ. Dietary management of obesity: Cornerstones of healthy eating patterns. *Med Clin North Am* 2018; 102(1):107–124; doi: 10.1016/j.mcna.2017.08.009
24. Piaggio G, Elbourne DR, Pocock SJ, et al. Reporting of non-inferiority and equivalence randomized trials: Extension of the CONSORT 2010 statement. *JAMA* 2012;308(24):2594–2604; doi: 10.1001/jama.2012.87802
25. Jang Y, Lee JH, Kim OY, et al. Consumption of whole grain and legume powder reduces insulin demand, lipid peroxidation, and plasma homocysteine concentrations in patients with coronary artery disease: Randomized controlled clinical trial. *Arterioscler Thromb Vasc Biol* 2001;21(12):2065–2071; doi: 10.1161/hq1201.100258
26. Jenkins DJ, Kendall CW, Augustin LS, et al. Effect of legumes as part of a low glycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus: A randomized controlled trial. *Arch Intern Med* 2012;172(21):1653–1660; doi: 10.1001/2013.jamainternmed.70
27. Mifflin MD, St Jeor ST, Hill LA, et al. A new predictive equation for resting energy expenditure in healthy individuals. *Am J Clin Nutr* 1990;51(2):241–247; doi: 10.1093/ajcn/51.2.241
28. Flint A, Raben A, Blundell JE, et al. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord* 2000;24(1):38–48; doi: 10.1038/sj.ijo.0801083
29. Jeyarajah EJ, Cromwell WC, Otvos JD. Lipoprotein particle analysis by nuclear magnetic resonance spectroscopy. *Clin Lab Med* 2006;26(4):847–870; doi: 10.1016/j.cll.2006.07.006
30. Hatonen KA, Virtamo J, Eriksson JG, et al. Protein and fat modify the glycaemic and insulinaemic responses to a mashed potato-based meal. *Br J Nutr* 2011;106(2):248–253; doi: 10.1017/S0007114511000080
31. American Diabetes A. Understanding A1C. 2022. Available from: <https://www.diabetes.org/diabetes/a1c/diagnosis> (accessed May 1, 2022).
32. Tam CS, Xie W, Johnson WD, et al. Defining insulin resistance from hyperinsulinemic-euglycemic clamps. *Diabetes Care* 2012; 35(7):1605–1610; doi: 10.2337/dc11-2339
33. Baer DJ, Rumpler WV, Miles CW, et al. Dietary fiber decreases the metabolizable energy content and nutrient digestibility of mixed diets fed to humans. *J Nutr* 1997;127(4):579–586; doi.org/10.1093/jn/127.4.579
34. Hall KD, Sacks G, Chandramohan D, et al. Quantification of the effect of energy imbalance on bodyweight. *Lancet* 2011; 378(9793):826–837; doi: 10.1016/S0140-6736(11)60812-X
35. Siri-Tarino PW, Chiu S, Bergeron N, et al. Saturated fats versus polyunsaturated fats versus carbohydrates for cardiovascular disease prevention and treatment. *Annu Rev Nutr* 2015;35:517–543; doi: 10.1146/annurev-nutr-071714-034449
36. Dobiasova M, Urbanova Z, Samanek M. Relations between particle size of HDL and LDL lipoproteins and cholesterol esterification rate. *Physiol Res* 2005;54(2):159–165.
37. Chiu S, Bergeron N, Williams PT, et al. Comparison of the DASH (dietary approaches to stop hypertension) diet and a higher-fat DASH diet on blood pressure and lipids and lipoproteins: A randomized controlled trial. *Am J Clin Nutr* 2016;103(2):341–347; doi: 10.3945/ajcn.115.123281
38. Fleming JA, Kris-Etherton PM, Petersen KS, et al. Effect of varying quantities of lean beef as part of a Mediterranean-style dietary pattern on lipids and lipoproteins: A randomized crossover controlled feeding trial. *Am J Clin Nutr* 2021;113(5):1126–1136; doi: 10.1093/ajcn/nqaa375
39. Hall KD, Ayuketah A, Brychta R, et al. Ultra-processed diets cause excess calorie intake and weight gain: An inpatient randomized controlled trial of ad libitum food intake. *Cell Metab* 2020;32(4):690; doi: 10.1016/j.cmet.2020.08.014
40. Follick MJ, Abrams DB, Smith TW, et al. Contrasting short- and long-term effects of weight loss on lipoprotein levels. *Arch Intern Med* 1984;144(8):1571–1574; doi: 10.3945/ajcn.115.123281
41. Hallak MH, Nomani MZ. Body weight loss and changes in blood lipid levels in normal men on hypocaloric diets during Ramadan fasting. *Am J Clin Nutr* 1988;48(5):1197–1210; doi: 10.1093/ajcn/48.5.1197