STATISTICAL ANALYSIS PLAN

Glycemic Effects of a Mediterranean-Style Dietary Pattern Containing Potatoes in Adults with Prediabetes

Penn State University IRB: STUDY00027785

ClinicalTrials.gov Identifier: NCT07229781

Version: July 13, 2026

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1. Administrative information

Clinicaltrials.gov Identifier: NCT07229781 (Registered November 17, 2025)

Key Personnel

Principal Investigator: Dr. Kristina Petersen PhD, APD, FAHA is an Associate Professor in the Department of Nutritional Sciences at Penn State University. Dr. Petersen is the Principal Investigator of the clinical trial. Dr. Petersen will be responsible for general study oversight and administration, protocol development and implementation, institutional review board (IRB) submission, data analysis and management, and training study personnel required for protocol execution.

Statistician: Dr. Prabhani Kuruppumullage Don PhD is an Associate Research Professor in the Department of Statistics at Penn State University. Dr. Kuruppumullage Don is a Co-investigator and will oversee all aspects of statistical design and analysis for the study. Specifically, she assisted with the power calculations and the development of the statistical analysis plan. She will also provide consultation on execution of the statistical analysis plan and critically review all of the manuscripts for statistical considerations.

Clinical Research Center: The Clinical Research Center (CRC) at Penn State University is staffed with experienced clinical research employees consisting of physicians, a nurse practitioner, and registered nurses who will work closely with the PI and study personnel throughout the clinical trial to facilitate the research protocol.

Metabolic Kitchen Manager: The Metabolic Kitchen manager will be responsible for overseeing food preparation, procurement, and provision to study participants and will conduct adherence monitoring during the controlled-feeding study.

Study Coordinators: Study personnel involving the research laboratory coordinator and research staff will be responsible for recruitment activities, data collection, and study procedures and will facilitate clinical trial operations.

2. Introduction

Background and Rationale

Low-quality evidence suggests potato intake is associated with a higher risk of type 2 diabetes (T2D), with a stronger relationship observed for fried potatoes compared to non-fried potatoes. A 2017 meta-analysis of 7 prospective cohort studies showed potato intake was positively associated with the risk of T2D, such that per 150 g/day increase in potato intake, the relative risk of T2D was increased by 18%.[1] However, the relative risk of T2D was increased by 66% per 150 g/day increase in fries compared with 9% for boiled/ baked/mashed potatoes. In this meta-analysis, no association was found between potato intake and coronary heart disease risk. Observational evidence is limited by confounding from diet and lifestyle factors that co-occur with potato intake. Specifically, foods commonly eaten with potatoes (e.g., burgers) and the dietary patterns (i.e., poor-quality diets, e.g., western-style diets) in which potatoes are commonly consumed may confound

the relationship between potato intake and T2D risk, and therefore, causation cannot be established from observational studies. Furthermore, potatoes can be consumed in an infinite number of ways and dietary patterns,[2] which makes it difficult to disentangle the independent effects of potato intake.

In contrast to observational findings, short-term (<8 weeks) clinical trials show that higher intake of potatoes does not worsen markers of glycemic control or risk factors for T2D compared to diets lower in potatoes and higher in refined grains, beans, or nuts.[3–6] In a randomized crossover trial conducted by the PI, intake of one non-fried potato per day by generally healthy adults did not affect markers of glycemic control or cardiovascular disease (CVD) risk factors after 4 weeks compared to an isocaloric amount of refined grains.[3] Similarly, in another 4-week trial, no differences in body composition or markers of glycemic control were observed with fried potato intake (300 kcal/day) vs. almond intake in generally healthy adults.[4] In an 8-week randomized controlled feeding study including adults with overweight or obesity, enlarged waist circumference, and insulin resistance, intake of a low glycemic index (GI) diet with non-fried potatoes reduced insulin resistance to a similar extent as a low GI diet with beans.[5] Findings from these short-term trials suggest potato intake has similar effects on glycemic control and other CVD risk factors compared to refined grains, beans, or nuts. However, longer-term (>8 weeks) trials examining the glycemic effects of habitual intake of potatoes as part of healthy dietary patterns are needed to inform recommendations for potato intake and guidance for healthy dietary patterns, particularly for individuals at high risk for T2D.

Individuals with prediabetes are at high risk for T2D,[7] and approximately 38% of US adults have prediabetes.[8] Prediabetes is defined by the American Diabetes Association as a condition that precedes T2D and is diagnosed by elevated hemoglobin A1C (HbA1c; 5.7-6.4%), impaired fasting glucose (100-125 mg/dL), or impaired glucose tolerance assessed with a 75 g oral glucose tolerance test.[7] For individuals with prediabetes at age 45 years, the lifetime risk of T2D is 74%; the risk is higher for those with concomitant obesity.[9] In addition, prediabetes is associated with a heightened risk of CVD.[7] Therefore, the goal of prediabetes management is to reduce the risk for progression to T2D and the associated complications, including CVD risk.[7]

The American Diabetes Association recommends that individuals with prediabetes be prescribed an eating pattern known to be effective in preventing T2D, such as a Mediterranean-style diet.[7] This is based on evidence showing that Mediterranean-style diets reduce the risk of T2D.[10] Notably, in the PREDIMED randomized controlled trial, a Mediterranean diet enriched with extra virgin olive oil reduced the risk of T2D by 40% after approximately 4 years compared to the control group.[11] The Mediterranean diet is characterized by high intake of fruits, vegetables, legumes, fish, whole grains, nuts, and olive oil; moderate intake of dairy products and wine; and low intake of red and processed meats and foods that contain high amounts of added sugar.[10] While the Mediterranean diet emphasizes vegetables, potatoes are not considered a defining feature, and intake of potatoes is generally low in Mediterranean countries.[12] Research investigating the glycemic and cardiovascular effects of potato consumption as part of a Mediterranean-style diet will further understanding of the cardiometabolic effects of potatoes and inform

recommendations for potato intake within healthy dietary patterns, particularly for people at risk for T2D.

This is of clinical relevance because of longstanding concerns about the potential for adverse effects of starchy vegetables on glucose metabolism, particularly in individuals with impaired glucose metabolism or T2D. Currently, the American Diabetes Association recommends that people with diabetes follow a healthy dietary pattern that emphasizes non-starchy vegetables only.[13,14] Further research directly examining the glycemic and CVD effects of a healthy dietary pattern higher in potatoes, a starchy vegetable, compared to an already recommended healthy dietary pattern for people with prediabetes and T2D is needed to empirically investigate the concerns about the effect of potatoes on glucose metabolism. Specifically, a trial designed using the equivalence framework will have the greatest clinical impact because it has the potential to show that a healthy dietary pattern higher in potatoes has the same effect as a healthy dietary pattern that is already recommended for people with impaired glucose metabolism. This conclusion cannot be made from a trial designed using the superiority framework.

Specific Aims

The goal of this 12-week randomized, parallel arm, controlled-feeding trial is to determine if a Mediterranean-style dietary pattern that includes various forms of potatoes (MED-P) has an equivalent effect or non-different effect on HbA1c compared to a traditional-style Mediterranean dietary pattern (MED). Additionally, the effect of the MED-P vs. the MED on other markers of glycemic control and risk factors for CVD will be assessed.
The study will address the following objectives:

1. Determine the effect of the MED-P vs. the MED on markers of glycemic control, including HbA1c, fasting glucose, fasting insulin, and insulin resistance assessed by the homeostatic model of insulin resistance (HOMA-IR), and continuous glucose monitor assessed mean glucose, time in range and glycemic variability.
2. Examine the effect of the MED-P vs. the MED on risk factors for CVD including lipids/lipoproteins, apolipoprotein B (apoB), C-reactive protein (CRP), blood pressure, and vascular health.
3. Assess the acceptability and satisfaction of both diets.

Hypothesis

It is hypothesized that after 12 weeks, the MED-P and MED will have equivalent effects on HbA1c, fasting glucose, mean glucose, time in range, glycemic variability, HOMA-IR, lipid/lipoprotein concentrations, lipoprotein particle size and concentrations, apoB, CRP, blood pressure and vascular health.

3. Study Methods

Trial design

A 12-week, 2-arm parallel, randomized controlled feeding trial will be conducted. Participants will be randomized to either a Mediterranean-style diet that includes 1 medium potato/day/2000 kcal replacing some grains (MED-P), or a traditional-style Mediterranean dietary pattern (MED). The MED-P will represent a traditional-style Mediterranean diet and

include 1 medium potato (~150 g) per day/2000 kcal that will displace 1 serving of refined grains and 1 serving of whole grains (1 oz-eq of each). Potatoes will be prepared in a variety of fried and non-fried recipes. The MED will not contain potatoes and will contain approximately 1 additional serving per day of both refined grains and whole grains (1 oz-eq of each).

Randomization method, allocation concealment, blinding

Participants will be randomized at the individual level. The randomization sequence will have block sizes of 2, 4, and 8 and be computer-generated by a person not involved in recruitment or data collection, and be uploaded to REDCap. REDCap will be used to ensure allocation concealment. Prior to baseline testing, the metabolic kitchen manager will use the randomization module in REDCap to reveal the participant's randomization. The person generating the randomization sequence and the metabolic kitchen manager will be the only study team members with user access to the randomization module in REDCap. The nurses drawing blood and laboratory staff conducting the assays will be blinded to the randomization schedule. The randomization code will be unsealed only once all participants have completed the study and the dataset has been reviewed and cleaned. Due to the nature of the study interventions, participants will not be blinded.

Sample size estimate

A sample size of 64 subjects (32/group) will enable equivalency to be demonstrated at a between-diet difference in the 12-week change in HbA1c (primary outcome) of 0 % [SD 0.16][15] with an equivalency margin ±0.12% at 80% power, $p<0.05$. 74 subjects will be recruited to account for a ~15% attrition rate.

Hypothesis testing framework

Primary Endpoint (12-week change from baseline):

- **HbA1c:** HbA1c will be assessed on one day at baseline and endpoint to calculate the change (endpoint minus baseline). The equivalency hypothesis will be tested using an independent samples *t*-test with a two one-sided test (TOST) equivalence analysis with equivalence bounds set at −0.12, 0.12 percentage point. Equivalence will be demonstrated if the 90% CI of the between-group mean difference is entirely within the equivalence bounds (i.e., −0.12, 0.12). Generally, a 0.3 to 0.5 percentage point change in HbA1c is considered clinically relevant.[16,17] Thus, trial is powered to establish equivalency based on an equivalence margin that is not considered clinically meaningful (±0.12 percentage points).

Secondary Endpoints (12-week change from baseline):

- **Fasting plasma glucose**: Glucose will be assessed on two consecutive days at baseline and endpoint. The change in glucose will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at −4.2, 4.2 mg/dL. The minimally important difference for fasting glucose is 9 mg/dL.[18] With 64 participants we have ~80% power ($p<0.05$; SD 5.6 based on a previous trial from our lab[15]) to detect between-diet equivalency with an equivalency margin of ± 4.2 mg/dL.

- **Fasting insulin**: Insulin will be assessed on two consecutive days at baseline and endpoint. The change in insulin will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -0.83, 0.83 µIU/mL. The minimally important difference for fasting insulin is 0.83 µIU/mL.[18]
- **Homeostatic model of insulin resistance (HOMA-IR):** Fasting glucose and insulin will be assessed on two consecutive days at baseline and endpoint; the mean of the two measurements taken at each timepoint will be used to calculate HOMA-IR. HOMA-IR will be calculated as (insulin µIU/mL × glucose mg/dL) / 405. The change in HOMA-IR will be calculated as the endpoint value minus the baseline value. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -0.5, 0.5. Limited data exist to define the minimally important difference; this margin is estimated to reflect a 25% change. Baseline HOMA-IR is expected to be ~2.0.
- **Mean glucose assessed by continuous glucose monitor (CGM):** Participants will wear a FreeStyle Libre Pro (Abbott) CGM in blinded mode to measure interstitial glucose concentrations every 15 minutes over a 7-day period at baseline and endpoint. All data collected during the CGM wear time will be included in the analyses.[19] Mean glucose (mg/dL) will be calculated across the wear time at each timepoint. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -5, 5 mg/dL. There is no consensus on a clinically important difference in mean glucose.[19] Observational evidence suggests the difference in mean glucose between individuals with prediabetes and those with normal glycemia is approximately 8 mg/dL.[20,21]
- **Time in range assessed by CGM:** Participants will wear a FreeStyle Libre Pro (Abbott) CGM in blinded mode to measure interstitial glucose concentrations every 15 minutes over a 7-day period at baseline and endpoint. All data collected during the CGM wear time will be included in the analyses.[19] Mean time in range will be calculated as the percentage of time spent in the time in tight range (70-140 mg/dL), which is appropriate for prediabetes,[21] across the wear time at each timepoint. The between-group difference in time in tight range will also be expressed as minutes. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -3, 3 percentage points. A change in time in range of 5 percentage points is considered clinically meaningful, and it is recommended to consider a 3% difference in the change as meaningful when examining intervention responses.[19]
- **Glycemic variability assessed by CGM:** Participants will wear a FreeStyle Libre Pro (Abbott) CGM in blinded mode to measure interstitial glucose concentrations every 15 minutes over a 7-day period at baseline and endpoint. All data collected during the CGM wear time will be included in the analyses.[19] Glycemic variability will be assessed by percent coefficient of variability across the wear time at each timepoint. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set -1.3, 1.3 percentage points. There is no consensus on a clinically important difference in glycemic variability.[19] Observational evidence suggests that the difference in

the coefficient of variability between individuals with prediabetes and those with normal glycemia is 1.54 percentage points.[20]

- **Triglycerides:** Triglycerides will be assessed on two consecutive days at baseline and endpoint. The change in triglycerides will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -8.9, 8.9. The minimally important difference for triglycerides is 8.9 mg/dL.[18]
- **Total cholesterol:** Total cholesterol will be assessed on two consecutive days at baseline and endpoint. The change in total cholesterol will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -10, 10 mg/dL. This is expected to represent an approximate 5% difference.
- **LDL-cholesterol:** LDL-cholesterol will be assessed on two consecutive days at baseline and endpoint using the Martin-Hopkins equation. The change in LDL-cholesterol will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -4, 4. The minimally important difference for LDL-cholesterol is 4 mg/dL.[18]
- **HDL-cholesterol:** HDL-cholesterol will be assessed on two consecutive days at baseline and endpoint. The change in HDL-cholesterol will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -4.6, 4.6 mg/dL. A 4.6 mg/dL increase in HDL-cholesterol is associated with a 10% reduction in risk for coronary heart disease.[22]
- **Non-HDL cholesterol:** non-HDL-cholesterol will be assessed on two consecutive days at baseline and endpoint by subtracting HDL-cholesterol from total cholesterol. The change in non-HDL cholesterol will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -4, 4. The minimally important difference for non-HDL-cholesterol is 4 mg/dL.[18]
- **ApoB:** ApoB will be assessed on one day at baseline and endpoint. The change will be calculated as the endpoint value minus the baseline value. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -2.6, 2.6 mg/dL. A 4 mg/dL difference in LDL-C equates to a 2.6 mg/dL difference in apoB in untreated populations.[23]
- **Lipoprotein particle size and concentration (LDL, HDL, triglyceride-rich lipoproteins):** Given the number of lipoprotein outcomes assessed and the lack of defined minimally important

differences for these outcomes, mean differences and 90% CIs will be reported for all of these outcomes and equivalence hypotheses will not be formally assessed.

- **C-reactive protein (CRP):** high sensitivity CRP will be assessed on two consecutive days at baseline and endpoint. The change in CRP will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). Values above 10 mg/L will be removed from analyses because this reflects acute inflammation. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -0.5, 0.5 mg/L. The minimally important difference for CRP is 0.5 mg/L.[18]
- **Central blood pressure:** Systolic and diastolic blood pressure will be measured on two consecutive days at baseline and endpoint. The change in systolic and diastolic will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis. The equivalence margins will be set at the same bounds are for peripheral blood pressure because evidence suggests similar risk prediction for central and peripheral blood pressure.[24]
- **Peripheral blood pressure:** Systolic and diastolic blood pressure will be measured on two consecutive days at baseline and endpoint. The change in systolic and diastolic will be calculated as the mean of the endpoint measures (i.e., mean of day 1 and day 2 values) minus the mean of the baseline measures (i.e., mean of day 1 and day 2 values). The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis. For systolic blood pressure the equivalence bounds will be set at -4, 4 mmHg. This is based on the FDA requirement for approval of new antihypertensive medications.[25] For diastolic blood pressure, the equivalence bounds will be set at -2, 2 mmHg based on the minimally important difference of 2 mmHg. [18]
- **Pulse wave velocity (PWV):** carotid-femoral PWV will be measured on one day at each timepoint and the change calculated as the endpoint value minus the baseline value. The equivalency hypothesis will be tested using an independent samples *t*-test with a TOST equivalence analysis with equivalence bounds set at -0.7, 0.7 m/s. A 0.7 m/s higher PWV is associated with a 10% increase in cardiovascular events.[26]

Additional Secondary Endpoints

For all other secondary endpoints, the *superiority framework* will be used for hypothesis testing:

- Diet satisfaction at 12 weeks
- Diet acceptability at 12 weeks

Interim analyses

No interim analyses will be performed.

Timing of outcome assessment
Participants will wear a CGM for 7 days prior to baseline and during week 11 of the study. Participants will also attend visits on two consecutive days at the beginning and at the end of the 12 weeks study for outcome assessment.

4. Trial Population

Recruitment
Participants will be recruited from University Park and State College, PA and surrounding areas using public advertisements and recruitment flyers posted on campus and in the local community (State College/University Park area).

Screening and eligibility criteria
Individuals responding to advertising will be emailed information about the study and complete a pre-screening survey via REDCap. Potentially eligible individuals will be telephone screened. Based on the answers to the questions, participants will be deemed eligible or ineligible by the staff member assessing eligibility in consultation with the PI. Eligible individuals will be scheduled for a clinic screening visit. At the clinic screening appointment, anthropometrics and blood pressure will be measured. Fasting blood samples will be assessed for HbA1c, glucose, a complete blood count, including liver and kidney function, and a blood lipid panel. Inclusion/exclusion criteria will be assessed.

At the screening visit, participants must meet all the following inclusion criteria and none of the exclusion criteria to participate in this study.

Inclusion criteria

- Age 25-65 years
- Prediabetes assessed by an HbA1c of 5.7-6.4% or fasting glucose 100-125 mg/dL
- BMI 25-40 kg/m^2

Exclusion criteria

- HbA1c ≥6.5% (diagnostic of type 2 diabetes)
- LDL-C (calculated with the Martin-Hopkins equation) ≥190 mg/dL
- Hemoglobin <13.2 g/dL for men or <11.7 g/dL for women
- Fasting triglycerides >350 mg/dL
- ≥10% change in body weight within the 6 months prior to enrollment
- Blood pressure >140/90 mmHg
- Reported diagnosis of type 1 or type 2 diabetes
- Prescription of anti-hypertensive, lipid-lowering, or glucose-lowering drugs
- Intake of supplements that affect the outcomes of interest (i.e., lipid, blood pressure, or glucose lowering; vitamin C or multi-vitamins containing vitamin C) and are unwilling to cease during the study period
- History of liver, kidney, or autoimmune disease
- Prior cardiovascular event (e.g., stroke, heart attack)
- Current pregnancy or intention of pregnancy within the next 6 months
- Lactation within the prior 6 months

- Allergy/intolerance/sensitivity/dislike of any foods in the study menus
- Antibiotic use within the prior 1 month
- Oral steroid use within the prior 1 month
- Use of tobacco or nicotine-containing products within the past 6 months
- History of cancer at any site within the past 10 years (eligible if ≥10 years without recurrence) or non-melanoma skin cancer within the past 5 years (eligible if ≥5 years without recurrence)
- Participation in another clinical trial within 60 days of baseline
- Currently following a restricted or weight-loss diet
- Prior bariatric surgery
- Intake of >14 alcoholic drinks/week and/or not willing to avoid alcohol consumption for 48 hours prior to test visits
- Does not speak and/or understand English
- Unwilling to refrain from donating blood during the study
- Weight <110 lb
- Principal Investigator discretion related to the potential participant's ability to adhere to the study requirements, including being able to come to attend visits

Early withdrawal of participants

Participants will be withdrawn from the study for the following reasons:

- Risks to the other participants/research team members, disruptive behavior during the study visit or food pick-ups
- Diagnosis of a disease listed as an exclusion criterion or a serious medical condition requiring active intervention (assess by review of medical history form).
- Not meeting the inclusion criteria assessed at the clinical screening visit
- Prescription of anti-hypertensive, lipid-lowering or glucose-lowering drugs
- Prescription of steroids for longer than 1 week
- Pregnancy
- Lack of adherence defined as intake of <90% of provided food for more than 5 consecutive days (not including the 5 days participants may request to be off the diet).

Presentation of baseline characteristics

Baseline demographic and clinical characteristics will be reported for the total analysis population and by randomization according to CONSORT guidelines.

5. Analysis Population

Analyses will be conducted consistent with intent-to-treat principles. All available data from randomly assigned participants will be included in data analyses.

6. Hypothesis Testing

Primary outcome:
The primary outcome is 12-week change in HbA1c. Change in HbA1c will be calculated as the end of diet measure minus the baseline measure.

Secondary outcomes:
The secondary outcome variables will be 12-week change (end of diet period minus baseline) in:

- Glucose
- Insulin
- HOMA-IR (Homeostatic Model Assessment for Insulin Resistance)
- Continuous glucose monitor (CGM) assessed mean glucose, time in range, and glycemic variability
- Lipid/lipoprotein concentrations (triglycerides, total cholesterol, LDL-C, HDL-C, non-HDL-C)
- Apolipoprotein B
- Lipoprotein particle size and concentration (LDL, HDL, triglyceride-rich lipoproteins)
- C-reactive protein (CRP)
- Central and peripheral blood pressure
- Pulse wave velocity (PWV)

Secondary outcome variables assessed a 12-weeks will be:

- Diet satisfaction
- Diet acceptability

7. Statistical Analyses

Statistical model assumptions will be evaluated and confirmed prior to analyses for hypothesis testing being conducted, and necessary transformations will be considered if deviations from normality or homoscedasticity assumptions are observed. All primary analyses will follow *intent-to-treat* principles. A sensitivity per-protocol analysis will be conducted that only includes those who consumed ≥90% of the energy provided on ≥90% of the study days. For the CGM-assessed outcomes, a sensitivity analysis will be conducted whereby only data from timepoints where ≥70 % of readings were obtained during the CGM wear time.

For endpoints with an equivalency hypothesis, independent samples *t*-tests with a TOST equivalence analysis, with equivalence bounds set, as described in section 3, will be used.

For the diet satisfaction and acceptability outcomes, hypothesis testing will follow the superiority framework with use of independent samples t-tests to assess the between-group differences for each outcome. Statistical significance $p<0.05$.

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