Dietary Intake Association with IFG and Responses of a Lifestyle Changing Protocol in a Community-Based Adult Cohort

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Abstract

Objective: Investigate the association of diet on Impaired Fasting Glucose (IFG) and response of a lifestyle changing protocol (LISC) on a community sample of adults.

Methods: A cross-sectional study of LISC was conducted with 1004 subjects. From those, 264 adults individuals participated in a 20-week intervention based on physical exercises and dietary counseling and were divided in three groups, normoglycemic, IFG, and T2DM. Evaluations were done at baseline (M0) and after a 20-week intervention (M1). The analyses were performed by using SAS, version 9.2., and results were discussed based on the level of significance of p<0.05.

Results: At baseline, the three groups differed for plasma triglycerides, and number of altered metabolic syndrome (MetS) components. T2DM differed from normoglycemic by presenting higher intake of meat, lower of sugar, and less dietary variety, along with higher plasma levels of uric acid. After 20-week intervention, normoglycemics, IFG and T2DM responded similarly to LISC. Both genders increased body fatness. Men increased fasting plasma insulin, saturated fatty acid intake, along with a decrease of vegetable oil intake while women showed a significant increase in HEI and dietary fiber intake and a trend to higher sugar and protein intake and lower vegetable oil intake. Overall T2DM decreased 68% from M0 (9.5%) to M1 (6.4%) of LISC.

Conclusion: Our data showed a significant difference in food composition on altered plasma glucose, and its further normalization with lifestyle intervention was independent of significant body weight and body fat changes.

Keywords: Type 2 diabetes mellitus; Diet; Lifestyle intervention; Obesity; Behavioral modification

Introduction

Type 2 diabetes mellitus (T2DM) prevalence is increasing worldwide and is an emerging problem in public health [1]. It is a complex genetic disorder influenced by interactions between susceptible loci and environmental perturbation [2]. Environmental contributions to the development of T2DM potentially include exposures such as suboptimal in utero environment, low birth weight, obesity physical inactivity and advancing age [3]. Such situations might affect the expression of key genes regulating insulin resistance [4].

Many studies have shown the protective effects from a balanced healthy diet on preventing chronic metabolic diseases. Diets rich in whole grain cereal, legumes, fruits, vegetables, fish, nuts, and with low-fat dairy products are related to disease preventive risk factors, such as hypertension, dyslipidemia, and obesity [5]. Diet is considered one of the main modifiable lifestyle risk factors related to the prevention of T2DM. Studies have shown a positive association of insulin resistance (HOMA-IR) with dietary fat and low dietary fiber intake [6,7]. On the other hand, HOMA-IR correlated inversely with consumption of fruits and dietary fiber, and also with VO2max.

This study aims to investigate further those associations of the role of diet on impaired fasting glucose and response of a lifestyle changing protocol (supervised physical exercises along with dietary counseling) on a community sample of adults.

Methods

Individuals and study design

The studied subjects were part of a subgroup (convenience sample) of a cross-sectional study conducted with 1004 participants screened for the intervention program of lifestyle changing “Mexa-se Pró-Saúde [Move for Health]”, from the years of 2004 to 2011. Of those 264 individuals, (53 men and 211 women, mean age of 53.3 ± 10.4 years old) had complete information of dietary intake and were selected for the longitudinal quasi-experimental study, with evaluations at baseline (M0) and after a 20-week (M1) of lifestyle changing intervention based on weekly dietary counseling and supervised physical activity sessions three times a week. The intervention program of lifestyle changing...
Laboratory analyses

Blood samples were collected by vacuum venous puncture, after a 10 to 12 hour fasting period, and centrifuged to obtain serum and plasma samples which were stored at -80°C until the end of the study. The individuals were previously advised not to perform vigorous physical exercises 24-hours and/or consume alcohol 72-hours prior to blood collection. Serum glucose was assayed by dry-chemistry (Vitros System 5600®, Ortho Clinical Diagnostics, Johnson & Johnson Company, Raritan, NJ, USA). Laboratory analysis of lipids parameters (total cholesterol, high density lipoprotein cholesterol and triglycerides), urea, creatinine, uric acid, and gamma-glutamyltransferase (Gamma GT) were performed within 4 hours after blood collection by dry chemistry method (Vitros® 5600, Ortho Clinical Diagnostics, Johnson & Johnson Company, Raritan, NJ, USA). The plasma low density lipoprotein cholesterol (LDL-c) concentrations were estimated using the formula proposed by Friedewald [10]. Serum concentrations of insulin were quantified by a chemiluminescent method (Immulite 2000®, Siemens Healthcare Diagnostics, Marburg, Germany).

Body composition

Weight, height and waist circumference (WC) were measured with standardized protocols. Body Mass Index (BMI) and waist circumference were evaluated according to the World Health Organization [11]. Body fat percentage (BF%) was assessed by a bioelectrical impedance device (Biodynamics®, model 450, USA).

The percentage of muscle mass (%MM) was obtained using the Janssen et al. [12] equation, and the muscle mass index (MMI) was calculated as MM (kg)/height².

Dietary intake

Dietary intake data was assessed using a single 24-hour dietary recall at baseline and after the intervention. Dietary intake was documented by a certified dietician, and to obtain precise information, the subjects were asked if that was a typical day of intake from them, how often they usually ate during the day, what variety of food was consumed, how the food was prepared, what the serving size was, and what the brand of the food/meal was. Total caloric intake was computed using the Brazilian food tables [13,14]. The Healthy Eating Index (HEI) modified for the Brazilian population was used to assess the quality of the participants diet [15]. Eight food groups and 12 components to measure the variety and quality of food intake were evaluated.

Physical activity

All participants were submitted to supervised exercise of 80 minutes, including warm up (20 min) walking (40 min) and stretching (20 min), 3x/wk complemented with 60 min (2x/wk) of strength (40 min) and stretching (20 min) at a gym [16]. Only participants with frequency of 3x/wk were included in the study.

Statistical Analysis

The analyses were performed by using SAS, version 9.2. The data were described as mean ± SD. Sample normality was tested by means of the Shapiro-Wilk test. For comparison of groups the ANOVA with repeated measure mode was used for normal distribution. For not-normal distribution the same model was fitted with a gamma distribution. For comparison of each category of the following variables HEI, BMI, and WC for baseline and after 20 weeks of intervention it was used trend chi-square test. The results were discussed based on the level of significance of p<0.05.

Results

Characteristics of the cross sectional study with 1004 individuals are shown in Table 1. At baseline the hyperglycemic individuals (IFG and T2DM) were older and heavier (body weight and BMI), with higher WC and MMI (Table 1). Additionally, they presented with higher blood pressure (SBP and DBP) and Gamma GT than normoglycemic. Triglycerides were the only metabolic syndrome (MetS) component that discriminated the 3 groups (T2DM>IFG> normoglicemic) and thus differing them by the number of altered MetS components. T2DM group was discriminated from normoglicemic group only by higher plasma uric acid and by consuming more meat, lower vegetable oil, lower sugar, and having lower dietary variety (Table 1).

When evaluating the subjects selected for the longitudinal study it was noticed that all three groups (normoglicemic, IFG, and T2DM) responded similarly to LISC (Table 2).
Table 1: Characteristics of individuals at baseline (n=1004)

IFG: Impaired fasting glucose; T2DM: Type 2 Diabetes Mellitus; BMI: Body Mass Index; WC: Waist Circumference; BF: Body Fat; MMI: Muscle Mass Index; HDL-c: High Density Lipoprotein cholesterol; Gamma GT: gamma-glutamyltransferase; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MetS components: Metabolic Syndrome Components; HEI: Healthy Eating Index

Different Letters ANOVA p ≤ 0.05

|                      | Normoglicemic (n=660) | IFG (n=243) | T2DM (n=101) |
|----------------------|-----------------------|-------------|--------------|
| Age (years)          | 52.1 ± 9.9a           | 54.8 ± 9.4b | 56.3 ± 1b    |
| BMI                  | 28.5 ± 5.2a           | 30.1 ± 5.0b | 30.7 ± 5.3b  |
| WC (cm)              | 92.8 ± 13.5a          | 98.4 ± 12.4b| 101.4 ± 12.4b|
| BF (%)               | 31.8 ± 8.6a           | 32.6 ± 7.6a | 33.7 ± 8.8a  |
| MMI (kg/m²)          | 10.0 ± 5.8a           | 13.2 ± 5.5b | 12.8 ± 5.8b  |
| Uric Acid (mg/dL)    | 4.65 ± 1.48a          | 4.8 ± 1.7ª  | 5.12 ± 1.84b |
| HDL-c (mg/dL)        | 51.8 ± 15.1a          | 53.3 ± 16.1ª| 49.6 ± 17.7a |
| Glucose (mg/dL)      | 88.3 ± 7.6a           | 108.5 ± 7.2b| 177.2 ± 50.1c|
| Triglycerides (mg/dL)| 148.2 ± 104.7a        | 178.4 ± 99.6b| 207.8 ± 129.0c|
| Gamma GT (J/L)       | 36.0 ± 29.1a          | 43.2 ± 29.8b| 53.3 ± 37.1b |
| SBP (mmHg)           | 125.8 ± 16.2a         | 132.1 ± 18.8b| 134.2 ± 18.1b|
| DBP (mmHg)           | 80.3 ± 15.1a          | 82.7 ± 11.5b | 84.7 ± 12.4b |
| MetS components      | 1.3 ± 1.1a            | 2.9 ± 1.1b  | 4.0 ± 1.0c   |
| Meat (servings)      | 1.8 ± 1.6a            | 2.1 ± 1.4ab | 2.3 ± 1.8b   |
| Sugar (servings)     | 1.6 ± 1.9a            | 1.5 ± 1.8ab | 0.9 ± 1.3b   |
| Vegetable oil (servings) | 2.6 ± 6.4a        | 2.4 ± 1.8ª  | 2.2 ± 2.3a   |
| Variety (items)      | 13.1 ± 4.2a           | 12.9 ± 4.4ab| 11.8 ± 3.7b  |
| Fiber (g)            | 13.5 ± 7.5a           | 13.8 ± 7.1ª | 14.2 ± 8.0a  |
| HEI (points)         | 79.3 ± 15.1a          | 77.2 ± 16.8ª| 82.1 ± 13.0a |

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Table 2: Characteristics of individuals (n=264) according to plasma glucose levels at baseline and after the intervention (20-week)

|                      | Women (n=211) | Men (n=53) |
|----------------------|--------------|------------|
|                      | baseline     | After 20 weeks | baseline     | After 20 weeks |
| Fasting glucose (mg/dl) | 99.99 (34.99) | 99.20 (32.53) | 97.87 (15.15) | 97.36 (17.26) |
| Fasting Insulin (mg/dl) | 9.59 (6.76)   | 10.67 (9.54)  | 10.05 (7.33)  | 12.36 (7.84)* |
| BMI (kg/m²)           | 30.65 (5.99)  | 30.83 (5.77)  | 30.61 (7.24)  | 32.06 (8.20)  |
| Body Fat (%)          | 40.05 (94.98) | 41.29 (3.60)* | 29.49 (6.11)  | 31.80 (4.98)* |
| WC (cm)               | 94.98 (14.02) | 95.03 (13.50) | 98.25 (8.95)  | 104.18 (16.22) |
| BF (%)                | 32.88 (3.70)  | 32.59 (4.33)  | 32.77 (5.30)  | 32.76 (6.43)  |
| MMI (kg/m²)           | 7.99 (1.22)   | 7.91 (1.17)   | 9.46 (1.38)   | 9.51 (1.29)   |
| Protein (%)           | 17.45 (5.62)  | 18.62 (5.50)  | 18.93 (4.83)  | 19.06 (5.46)  |
| CHO (%)               | 52.95 (10.18) | 56.35 (9.20)  | 51.46 (8.16)  | 50.43 (10.07) |
| Total Fat (%)         | 29.41 (8.83)  | 27.75 (6.56)  | 29.60 (7.58)  | 30.59 (7.76)  |
| SFA (%)               | 9.01 (3.96)   | 8.68 (4.21)   | 7.75 (2.37)   | 9.51 (3.87)*  |
| Fiber (g)             | 14.65 (8.22)  | 17.32 (9.52)* | 18.53 (10.80) | 23.30 (16.26) |
| Meat (serving)        | 1.38 (0.91)   | 1.37 (0.95)   | 2.25 (1.53)   | 2.46 (1.92)   |
| Sugar (serving)       | 1.58 (1.77)   | 1.22 (1.68)   | 1.73 (1.92)   | 1.48 (2.52)   |
| Vegetable oil (serving) | 1.84 (2.00) | 1.58 (1.55) | 3.29 (2.67) | 2.25 (1.81)* |
| Variety (items)       | 13.22 (4.22)  | 13.52 (4.34)  | 12.54 (4.65)  | 12.72 (4.61)  |
| HEI                   | 77.46 (14.23) | 80.81 (14.03)* | 78.65 (13.24) | 79.98 (15.98) |

Table 3: Characteristics of individuals (n=264) according to gender at baseline and after the intervention (20-week)

|                      | Women (n=211) | Men (n=53) |
|----------------------|--------------|------------|
|                      | baseline     | After 20 weeks | baseline     | After 20 weeks |
| Fasting glucose (mg/dl) | 99.99 (34.99) | 99.20 (32.53) | 97.87 (15.15) | 97.36 (17.26) |
| Fasting Insulin (mg/dl) | 9.59 (6.76)   | 10.67 (9.54)  | 10.05 (7.33)  | 12.36 (7.84)* |
| BMI (kg/m²)           | 30.65 (5.99)  | 30.83 (5.77)  | 30.61 (7.24)  | 32.06 (8.20)  |
| Body Fat (%)          | 40.05 (94.98) | 41.29 (3.60)* | 29.49 (6.11)  | 31.80 (4.98)* |
| WC (cm)               | 94.98 (14.02) | 95.03 (13.50) | 98.25 (8.95)  | 104.18 (16.22) |
| BF (%)                | 32.88 (3.70)  | 32.59 (4.33)  | 32.77 (5.30)  | 32.76 (6.43)  |
| MMI (kg/m²)           | 7.99 (1.22)   | 7.91 (1.17)   | 9.46 (1.38)   | 9.51 (1.29)   |
| Protein (%)           | 17.45 (5.62)  | 18.62 (5.50)  | 18.93 (4.83)  | 19.06 (5.46)  |
| CHO (%)               | 52.95 (10.18) | 56.35 (9.20)  | 51.46 (8.16)  | 50.43 (10.07) |
| Total Fat (%)         | 29.41 (8.83)  | 27.75 (6.56)  | 29.60 (7.58)  | 30.59 (7.76)  |
| SFA (%)               | 9.01 (3.96)   | 8.68 (4.21)   | 7.75 (2.37)   | 9.51 (3.87)*  |
| Fiber (g)             | 14.65 (8.22)  | 17.32 (9.52)* | 18.53 (10.80) | 23.30 (16.26) |
| Meat (serving)        | 1.38 (0.91)   | 1.37 (0.95)   | 2.25 (1.53)   | 2.46 (1.92)   |
| Sugar (serving)       | 1.58 (1.77)   | 1.22 (1.68)   | 1.73 (1.92)   | 1.48 (2.52)   |
| Vegetable oil (serving) | 1.84 (2.00) | 1.58 (1.55) | 3.29 (2.67) | 2.25 (1.81)* |
| Variety (items)       | 13.22 (4.22)  | 13.52 (4.34)  | 12.54 (4.65)  | 12.72 (4.61)  |
| HEI                   | 77.46 (14.23) | 80.81 (14.03)* | 78.65 (13.24) | 79.98 (15.98) |

BMI: Body Mass Index; WC: Waist Circumference; BF: Body Fat; MM: Muscle Mass; MMI: Muscle Mass Index; CHO: Carbohydrates; SFA: Saturated Fatty Acids; HEI: Healthy Eating Index

Both genders responded similarly to LISC by increasing body fatness. Men increased fasting plasma insulin, saturated fatty acid intake, along with a decrease of vegetable oil (soybean oil) intake. Women showed a significant increase in HEI and dietary fiber intake and a trend to higher sugar and protein intake and lower vegetable oil intake (Table 3).

Only 5.4% of normoglycemics had a recommended HEI score (higher than 100 points) at baseline and even less were seen in IFG (0%) and T2DM (0.8%). After the intervention, there was no improvement in the dietary quality among groups noted. At baseline, IFG was present in obese (15.4%) and overweight (14.6%), and absent...
in normal weight individuals; however, T2DM was similarly frequent in obese (16.9%) and normal weight individuals (13.3%), and less frequent in overweight (4.9%).

The intervention resulted in a greater decrease of T2DM among normal weight individuals (-6.6%) than obese (-1.5%). The promotion of normoglycemia after the intervention was poorly seen in obese (-3.1%) and overweight (0%). At baseline, 60.9% of normoglicemic and 75.0% with T2DM and IFG had abnormal WC.

### Table 4: Percentage of Healthy Eating Index, waist circumference and body mass index classifications of groups at baseline and after the intervention

|                  | Baseline | After 20 weeks |
|------------------|----------|----------------|
|                  | Normo-glicemic (n=192) | IFG (n=47) | T2DM (n=25) | Normo-glicemic (n=192) | IFG (n=47) | T2DM (n=25) | p-value* |
| HEI (points)     |          |                |            |                |            |            |          |
| <100             | 60.5     | 18.6           | 14.7       | 62.0           | 18.6       | 15.5       | 0.2706   |
| >= 100           | 5.4      | -              | 0.8        | 3.9            | -          | -          | 0.4106   |
| BMI (kg/m²)      |          |                |            |                |            |            |          |
| <= 25            | 86.7     | -              | 13.3       | 73.3           | 20.0       | 6.7        | 0.1025   |
| >25 - 30         | 80.5     | 14.6           | 4.9        | 80.5           | 12.2       | 7.3        | 0.6122   |
| > 30             | 67.7     | 15.4           | 16.9       | 64.6           | 20.0       | 15.4       | 0.6555   |
| WC (cm)          |          |                |            |                |            |            |          |
| Normal           | 39.1     | 25.0           | 25.0       | 41.3           | 18.8       | 31.2       | 0.6244   |
| Abnormal         | 60.9     | 75.0           | 75.0       | 58.7           | 81.2       | 68.8       | 0.7700   |

### Table 5: Changes in plasma glucose levels of groups after the intervention

|                  | Baseline | After 20 weeks |
|------------------|----------|----------------|
|                  | Normoglicemic | IFG | T2DM |
| Normoglicemic    | 192 (72.7) | 166 | 21 | 5 (1.9); 2.6 |
| IFG              | 47 (17.8)  | 25 | 17 | 5 (1.9); 10.6 |
| T2DM             | 25 (9.5)   | 2  | 6 | 17 (6.4); 68.0 |
| Total            | 264 (100.0)| 193 | 44 | 27 (10.2) |

### Discussion

In the present study, we found that the intervention protocol for lifestyle changing benefited T2DM subjects with abnormal waist circumference who are either obese or at normal body weight, but not overweight. The protocol, however, failed for normoglycemic subjects with abnormal waist circumference who were obese and normal weight, but not overweight. Confusingly, there was an increase of T2DM and IGT after the intervention, with a reciprocal decreased in normoglycemics, nevertheless, our study contained no control group to determine if our intervention decreased the number of patients with impaired glucose metabolism compared to control. This was associated with weight gain and increase in abnormal waist circumference in normal weight individuals, independently of the dietary quality. On the other hand, a decrease in T2DM prevalence was seen among obese with abnormal waist circumference after the intervention. This may mean that our intervention had a much greater impact on individuals with higher abdominal fat than those with less abdominal fat at baseline.

The prevalence of T2DM has been increasing worldwide, and it is now recognized that developing countries are facing the greatest burden of this disease [1]. The prevalence of T2DM in our study was 9.5%, which is compatible with those obtained in other national surveys [17-19]. In our study, hyperglycemic subjects were older and heavier than normoglycemics which is consistent with other studies showing that aging and obesity are well known risk factors for diabetes [20].

Waist circumference is an indirect measurement of visceral fat, and has been shown to be a significant independent risk factor for T2DM [21]. Visceral fat is a precursor to the increased lipolysis and fatty free acids seen in MetS subjects [22]. It also modulates insulin action and is
more closely associated with insulin resistance than overall obesity or subcutaneous fat is [22,23]. Our study showed that increased WC was associated with hyperglycemia, altered blood pressure, plasma insulin, and Gamma GT. Gamma GT was significantly higher for IFG and T2DM which is consistent with increasing fatty liver from normoglicemics to IFG to T2DM. Obesity is the most common risk factor associated with non-alcoholic fatty liver disease (NAFLD) and NAFLD incidence is increasing globally, because of the epidemic of obesity and T2DM [24]. The high prevalence of NAFLD and insulin resistance among obese individuals may be attributed to the increased caloric intake (particularly high fat and sugar and low fiber) and decreased caloric expenditure (lack of exercise) [25]. The results of our study are consistent to the literature, as we showed an association of visceral obesity with increased plasma levels of Gamma GT and insulin; however, the interpretation of these results should be taken with caution, since information on alcohol consumption was not collected.

Obesity is associated with hypertriglyceridemia and hypertension [26]. In our study population, plasma triglycerides and blood pressure increased with increasing plasma glucose levels. A similar outcome was found in a middle-aged Norwegian cohort of men and women [27]. This study showed that levels of triglycerides and blood pressure were independent risk factors for T2DM [27]. Therefore, our study is consistent with other studies [25] showing that obesity is associated with hypertriglyceridemia, high blood pressure, and hyperglycemia which may lead to T2DM.

It is well established that a healthy lifestyle, including adequate diet and exercises, is associated with the prevention of T2DM [28]. Although, there is still a lack of evidences about the optimal eating pattern for the prevention of T2DM [29]. The dietary assessment in our study considered not only the nutrients, but the quality of the diet, which may represent an approach to assessing the role of diet on epidemiological studies.

In our study, T2DM subjects differed from normoglycemics by consuming more meat, less sugar, and having less dietary variety. The inverse association between dietary quality and risk of incident IFG was shown by Gopinath et al. [30] and by previous cross-sectional population-based studies [29,31]. High consumption of red meat, probably from its high fat content, is associated with increase insulin resistance among obese individuals may be attributed to the increased caloric intake (particularly high fat and sugar and low fiber) and decreased caloric expenditure (lack of exercise) [25]. The results of our study are consistent to the literature, as we showed an association of visceral obesity with increased plasma levels of Gamma GT and insulin; however, the interpretation of these results should be taken with caution, since information on alcohol consumption was not collected.

Obesity is associated with hypertriglyceridemia and hypertension [26]. In our study population, plasma triglycerides and blood pressure increased with increasing plasma glucose levels. A similar outcome was found in a middle-aged Norwegian cohort of men and women [27]. This study showed that levels of triglycerides and blood pressure were independent risk factors for T2DM [27]. Therefore, our study is consistent with other studies [25] showing that obesity is associated with hypertriglyceridemia, high blood pressure, and hyperglycemia which may lead to T2DM.

After the intervention, plasma insulin levels remained the same among women, while it was increased among men. High intake of dietary fiber and whole grains are associated with higher insulin sensitivity, lowering plasma insulin, glucose, and decreased inflammation and oxidative stress [32,38]. Despite the intensive dietary counseling in our study, subjects did not show much difference in their quality of diet, as they presented with a poor quality of diet (HEI less than 100 points) at the start of the study and maintained the poor quality of diet at the end of the study.

The association between obesity and the risk for T2DM is well known. In our study, hyperglycemic individuals were heavier than normoglycemics, and both men and women increased body fat percentage after the intervention. A study conducted with two population-based cohorts from the north and south of Spain showed an association between a greater weight gain, and the onset of T2DM in both those who were previously obese and those who were not obese, suggesting that the effect of weight gain is independent of the weight itself for increasing the risk of becoming diabetic [39].

This study has some limitations: such as, the sample size, it is a small subset of a larger study divided in three groups with uneven numbers of cohort, which may not have been large enough for estimating the prevalence of IFG and T2DM. Furthermore, our study contained no control group to determine if our intervention decreased the number of patients with impaired glucose metabolism. A dietary intake assessment using only a single 24-hour dietary recall has limitations, since one 24-hour recall does not detect dietary intake variations.

In summary, the results of this study highlight the hypothesis that dietary intake may influence altered plasma glucose, and its further normalization with lifestyle intervention was independent of significant body weight and body fat changes. Moreover, the intervention had a much greater impact on individuals with abnormal waist circumference than those with normal waist circumference.

Authors' Contributions

RCB designed the study. RCB and KM wrote the manuscript. LAS contributed to and revised the draft manuscript prepared by RCB and KM. GT and JEC was responsible for the acquisition and analysis of data. All authors had substantial contributions to the manuscript, read, and agreed to the final version.

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