# **ORIGINAL CONTRIBUTIONS**





# The Preoperative Dietary Inflammatory Index Predicts Changes in Cardiometabolic Risk Factors After 12 Months of Roux-en-Y Gastric Bypass

Sônia L Pinto <sup>1,2</sup> · Leidjaira L Juvanhol<sup>2</sup> · Alessandra da Silva<sup>2</sup> · Nitin Shivappa<sup>3,4,5</sup> · James R Hébert<sup>3,4,5</sup> · Josefina Bressan<sup>2</sup>

© Springer Science+Business Media, LLC, part of Springer Nature 2020

# Abstract

**Background** The objective of this study was to evaluate Dietary Inflammatory Index (DII®) in the preoperative period as well as 3 and 12 months post-surgery and its association with cardiometabolic risk factors after RYGB.

**Materials and Methods** This is a prospective cohort study of 50 patients (both sexes) who underwent RYGB. All data were collected in 3 phases: before surgery, 3, and 12 months post-surgery. To calculate DII scores, we utilized mean nutrients from three 24-h recalls at each time point.

**Results** The patients had median age of  $39.1 \pm 7.9$  years (70% women). Mean preoperative DII® score of  $0.39 \pm 1.49$  was slightly pro-inflammatory. Mean DII score reduced to  $-1.52 \pm 1.27$  after 3 months post-surgery and was classified anti-inflammatory. This value rebounded to  $-0.88 \pm 1.49$  at 12 months but was still anti-inflammatory. From the adjusted linear regression analysis, we observed that preoperative DII score was statistically associated with variations in neck circumference ( $\beta = -0.50$ ; p = 0.03), waist-hip ratio ( $\beta = 0.01$ ; p = 0.02), total cholesterol ( $\beta = 6.47$ ; p = 0.002), and LDL cholesterol ( $\beta = 6.42$ ; p = 0.001) after 12 months post-surgery. Changes in DII® at 3 and 12 months were not associated with changes in cardiometabolic risk factors.

**Conclusion** We observe significant changes in the inflammation potential of diet after 3 and 12 months of RYGB. Patients with higher preoperative E-DII scores have a greater metabolic improvement after 12 months of surgery.

Keywords Gastric bypass · Inflammation · Dietary inflammatory index · Cardiometabolic risk factors

Sônia L Pinto sonialopes@uft.edu.br

> Leidjaira L Juvanhol leidjaira@ufv.br

Alessandra da Silva alessan.drasg94@gmail.com

Nitin Shivappa shivappa@email.sc.edu

James R Hébert jhebert@mailbox.sc.edu

Josefina Bressan jbrm@ufv.br

- <sup>1</sup> Nutrition Course of the Federal University of Tocantins/TO, Federal University of Viçosa, Quadra 109 North, NS-15, ALCNO-14, Plano Diretor Norte, Palmas, Tocantins 77001-090, Brazil
- <sup>2</sup> Department of Nutrition and Health, Federal University of Viçosa/ MG, University Campus, Viçosa, Minas Gerais 36570-900, Brazil
- <sup>3</sup> Cancer Prevention and Control Program, University of South Carolina, Columbia, SC 29208, USA
- <sup>4</sup> Department of Epidemiology and Biostatistics, Arnold School of Public Health, University of South Carolina, Columbia, SC 29208, USA
- <sup>5</sup> Connecting Health Innovations LLC, Columbia, SC 29201, USA

# Introduction

Obesity is characterized by adipose tissue expansion, lowgrade chronic inflammation [1], and the development of cardiometabolic risk factors, such as dyslipidemia and insulin resistance. Adipose tissue expansion culminating in tissue hypoxia is one of the causes of inflammation. In this context, the secretion of pro-inflammatory adipokine decreases the production of anti-inflammatory cytokines [2]. Bariatric surgery is the most effective method for the treatment of severe obesity in terms of weight loss and long-term maintenance [3]. In addition, the surgery improves cardiometabolic profile linked to reduced inflammation from weight loss. Among bariatric patients, studies have reported a decrease and increase in pro-inflammatory cytokines and anti-inflammatory adipokines, respectively, at 3 [4, 5] and 12 [6] months postsurgery.

In recent years, studies have shown that habitual eating is related to inflammation [7], providing further evidence that diet influences inflammatory response [8]. The inflammatory potential of diet is measured by the Dietary Inflammatory Index (DII®), which quantifies the inflammatory response to an ingested food or nutrient [9]. It has been shown that individuals on a high DII diet have significantly higher C-reactive protein levels than those on a low DII diet, suggesting that DII can determine the association between dietary inflammatory potential and non-communicable diseases [9, 10]. The literature reports association of DII with cardiovascular disease [8], osteoporosis [10], insulin resistance, hepatic steatosis [11], higher BMI, [12] and mortality [13].

Despite evidence linking many obesity-related conditions with the DII, changes in the inflammatory potential of diet among patients with severe obesity after bariatric surgery is unknown. To our knowledge, no study has evaluated the impact of Roux-en-Y gastric bypass (RYGB) surgery on DII, as well as the influence of DII on changes in cardiometabolic risk factors after the procedure. Therefore, this study aimed to evaluate DII® in the preoperative period as well as 3 and 12 months post-surgery and its association with cardiometabolic risk factors after Roux-en-Y gastric bypass (RYGB).

# Methods

# **Study Population**

This is a prospective cohort study of 50 patients who underwent RYGB bariatric surgery and received 12 months of post-surgery care. This study was approved by the Research Ethics Committee of Federal University of Viçosa (UFV), Brazil. Informed consent was obtained from all individual participants included in the study. All information was collected in 3 stages: before surgery and at 3 and 12 months after RYGB. There were no losses from the follow-up, that is, all patients attended all stages of data collection in the study.

## **Food Consumption**

We used the mean of three 24-h recalls (24HR) at each phase of the research (preoperative period, 3, and 12 months after surgery). At each phase of the research, three 24HR were applied with a minimum interval of 3 days between each recall. The 24HR was administered by a trained nutritionist and the patients reported all foods and beverages consumed the previous day. The 24HR data were entered digitally, and variables such as energy, fiber, macro, and micronutrients intake were evaluated using the Brasil Nutri® software based on the Brazilian nutritional composition tables constructed for the Family Budget Survey (POF, acronym in Portuguese) of the Brazilian Institute of Geography and Statistics (IBGE, acronym in Portuguese) [14].

The inflammatory potential of the diet was evaluated using the DII®, an index developed by Shivappa et al., to quantify the effect of diet on inflammation [9]. DII® scores were calculated using an algorithm based on 1943 articles that presented findings on associations between 45 dietary parameters and six inflammatory biomarkers (IL-1b, IL-4, IL-6, IL-10, tumor necrosis factor- $\alpha$ , and protein C-reactive). Food and nutritional intake derived from the 24HR were adjusted for total energy and, subsequently, standardized with a representative global dietary database. Standard energy-adjusted dietary intake (i.e., per 1000 kcal) was multiplied by the literature-derived inflammatory score for each DII component. Individual scores were defined to determine the overall energy-adjusted DII (E-DII) score for each individual, with positive values representing a more pro-inflammatory diet and negative values representing a more anti-inflammatory diet.

To construct the E-DII, we considered the following food consumption variables obtained from the average of three 24HRs at each phase of the study: carbohydrate, protein, lipid, fiber, calcium, magnesium, manganese, phosphorus, iron, sodium, potassium, copper, zinc, thiamine, riboflavin, pyridoxine, vitamin C, cholesterol, saturated, monounsaturated and polyunsaturated fats, linoleic and linolenic acids, trans fat, vitamin D, vitamin E, vitamin B12, selenium, folate, retinol, and niacin.

### **Cardiometabolic Risk Factors**

To assess cardiometabolic risk, the following variables were considered: triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-c) and high-density lipoprotein (HDL-c), glucose, insulin, and gamma-glutamyl transferase (GGT) besides the following indexes: homeostasis model assessment-insulin resistance (HOMA-IR), triglyceride/glucose (TyG), and fatty liver index (FLI).

Serum glucose, TG, TC, HDL-c, and LDL-c were determined by enzymatic colorimetric test. For the determination of serum insulin concentration, electrochemiluminescence immunoassay was used. All the analyses were performed in an outsourced laboratory.

Insulin resistance was determined by HOMA-IR and TyG. HOMA-IR index was calculated from the formula: fasting insulin ( $\mu$ U/L) × fasting glycemia (nmol/L)/22.5 [15]. TyG index was calculated based on the TG and fasting glucose values by the equation (ln (fasting TG (mg/dl) × fasting glycemia (mg/dL))/2 [16].

Non-alcoholic fatty liver disease (NAFLD) was defined by FLI, calculated by the algorithm of Bedogni et al. [17]. The calculation considers TG values in milligram per deciliter, GGT in unit per liter, body mass index (BMI) in kilogram per square meter, and waist circumference (WC) in centimeter.

#### Anthropometry and Associated Measurements

Anthropometric data such as body weight, height, waist, hip, and neck circumferences were collected. Weight and height were measured according to the technique recommended by Jellife [18]. From these measurements, BMI was calculated by dividing weight (kg) by height (m) squared.

All circumferences were measured with a flexible and inelastic anthropometric tape. Waist and hip circumferences were measured according to Callaway et al. [19] and neck circumference (NC), according to Ben-Noun et al. [20]. Waist-hip ratio (WHR) was calculated by dividing waist circumference (cm) by hip circumference [21].

After the surgery, weight loss was evaluated by absolute measurement in kilograms. Additionally, percentage excess weight loss (% EWL) was quantified and calculated considering an ideal BMI of 24.9 kg/m<sup>2</sup>. From this, excess weight (EW) in kilogram was calculated: pre-weight (kg) – ideal weight (kg). The percentage EWL was calculated considering the following: absolute weight loss (kg)/EW × 100.

Body composition was assessed by tetrapolar electrical bioimpedance (BIA) using the *Biodynamics*® 310 instrument, as instructed by the manufacturer's manual. Body fat (BF) was expressed as a percentage.

#### **Statistical Analysis**

All data were analyzed using the Stata® software version 13.0 and SPSS® version 20.0. Numerical variables were first tested for normality by the Shapiro–Wilk test and expressed as mean  $\pm$  standard deviation.

Repeated measures ANOVA test, followed by the Bonferroni post hoc test, was used to assess differences in mean DII and cardiometabolic risk factors during the phases of the study: preoperative, 3, and 12 months of RYBG. To evaluate changes in cardiometabolic risk and DII variables throughout the post-surgery period, delta ( $\Delta$ ) was calculated, being baseline—value at 3 or 12 months. For the linear regression analysis, each cardiometabolic risk variable was considered dependent ( $\Delta$ 12 months) and E-DII score as independent (preoperative,  $\Delta$ 3 months, and  $\Delta$ 12 months). The regression models of the preoperative DII were adjusted by the preoperative cardiometabolic risk factor values. The regression models of the DII for  $\Delta$ 3 months and  $\Delta$ 12 months were simultaneously adjusted by the preoperative cardiometabolic risk factor values and the preoperative DII values. For the variables blood glucose, TyG, and HOMA-IR, the adjustment was performed by the use of hypoglycemic medication. For all analyses, a significance level of 5% was adopted.

# Results

Fifty RYGB patients with a mean age of  $39.2 \pm 7.9$  years participated in this study, of whom 70% were female. After 12 months of surgery, percentage EWL was 83.7%, corresponding to an average weight loss of 38.5 kg. All cardiometabolic risk factors significantly decreased after 3 and 12 months of surgery, except for TC, LDL-c, glucose, and GGT, which significantly reduced from baseline to 3 months, and HDL-c, which decreased at 3 months but increased after 1 year of surgery (Table 1).

In contrast, compared with the average preoperative E-DII score  $(0.39 \pm 1.49)$ , a pro-inflammatory diet, the average E-DII values at 3 and 12 months of surgery were significantly lower:  $-1.52 \pm 1.27$  and  $-0.88 \pm 1.49$ , respectively. Despite the significant increase in E-DII scores observed at 12 months compared with 3 months, both values are characteristic of an anti-inflammatory diet (Fig. 1).

When assessing the possible associations between E-DII (preoperative,  $\Delta 3$  months, and  $\Delta 12$  months) and cardiometabolic risk factors ( $\Delta 12$  months), we observed a negative association between preoperative DII and  $\Delta NC$  ( $\beta$ , -0.50; p = 0.030), indicating that higher E-DII values before surgery are associated with smaller changes in NC after 12 months. On the other hand, we observed a positive association of preoperative E-DII with  $\Delta$  WHR ( $\beta$ , 0.01; p = 0.018),  $\Delta TC$  ( $\beta$ , 6.47; p = 0.002), and  $\Delta LDL$ -c ( $\beta$ , 6.42; p = 0.001). Thus, higher E-DII values before surgery were associated with greater changes in these markers after 12 months (Fig. 2). Such associations were independent of the cardiometabolic variable in the preoperative period. Changes in DII at 3 and 12 months were not associated with  $\Delta 12$  months of cardiometabolic risk variables (Table 2).

Additionally, the possible associations of the E-DII (preoperative,  $\Delta 3$  months, and  $\Delta 12$  months) and changes in cardiometabolic risk variables at 3 months in relation to the preoperative period were tested; however, no statistically significant differences were observed (data not shown).

**Table 1** Characteristics of studyparticipants before and after 3 and12 months of RYGB (n = 50)

Variables	Preoperative $n = 50$	3 months $n = 50$	12 months $n = 50$
Weight (kg)	$115.5 \pm 16.7^{a}$	$94.2 \pm 13.7^{b}$	$77.0 \pm 11.7^{c}$
BMI (kg/m <sup>2</sup> )	$42.2\pm4.9^a$	$34.5 \pm 4.1^{b}$	$28.2\pm3.8^{\rm c}$
BF (%)	$42.4\pm4.5^a$	$37.7\pm5.4^{b}$	$29.4\pm7.4^{\rm c}$
WC (cm)	$122.4\pm11.7^a$	$106.4 \pm 10.9^{b}$	$92.5 \pm 11.3^{\circ}$
HC (cm)	$128.3\pm9.4^{\rm a}$	$115.2 \pm 9.2^{b}$	$103.5 \pm 7.9^{\circ}$
NC (cm)	$42.1 \pm 4.3^{a}$	$38.0\pm3.5^b$	$35.2 \pm 3.5^{\circ}$
WHR	$0.9\pm0.1^{\rm a}$	$0.9\pm0.1^{\mathrm{a}}$	$0.8\pm0.1^{\rm b}$
%TWL	-	$18.5 \pm 2.6^{a}$	$33.2\pm6.3^{b}$
% EWL	-	$46.8\pm9.8^a$	$83.7\pm19.2^{b}$
TG (mg/dl)	$140.2 \pm 67.7^{\rm a}$	$92.6 \pm 31.5^{b}$	$75.5\pm28.4^{\rm c}$
TC (mg/dl)	$181.7\pm30.8^a$	$151.5 \pm 30.4^{b}$	$154.7 \pm 26.7^{b}$
LDL-c (mg/dl)	$104.8\pm28.3^a$	$91.3\pm27.3^{b}$	$85.8\pm24.8^{b}$
HDL-c (mg/dl)	$48.8\pm11.1^{\rm a}$	$41.7 \pm 10.4^{\text{b}}$	$53.8 \pm 12.0^{\circ}$
Glucose (mg/dl)	$103.8\pm38.2^a$	$82.9\pm8.7^{\rm b}$	$83.5\pm6.7^{b}$
GGT (U/L)	$37.5\pm60.2^{\rm a}$	$22.8\pm20.0^{b}$	$20.1\pm21.9^{b}$
Insulin (µUI/ml)	$16.4\pm10.6^{\rm a}$	$6.7 \pm 3.5^{\mathrm{b}}$	$3.9\pm2.1^{\circ}$
HOMA-IR	$4.5\pm6.0^{\rm a}$	$1.4\pm0.7^{ m b}$	$0.8\pm0.5^{ m c}$
TyG index	$8.7\pm0.5^{\rm a}$	$8.2\pm0.3^{\mathrm{b}}$	$7.9\pm0.4^{\rm c}$
FLI	$94.4\pm7.1^a$	$69.1 \pm 21.7^{b}$	$34.2 \pm 21.7^{c}$

Variables expressed in mean and standard deviation. \*Different letters indicate a statistically significant difference (p < 0.05) between the groups. ANOVA of repeated measures test was used for variables with normality with Bonferroni post hoc and the Friedman and Wilcoxon test for the others

*RYGB* Roux-en-Y gastric bypass, *BMI* body mass index, *BF* body fat, *WC* waist circumference, *HC* hip circumference, *NC* neck circumference, *WHR* waist-hip ratio, *EWL* excess weight of loss, *TWL* total weight loss, *TG* triglycerides, *TC* total cholesterol, *LDL-c* low-density lipoprotein, *HDL-c* high-density lipoprotein, *GGT* gama glutamyl transferase, *HOMA-IR* homeostasis model assessment for insulin resistance, *TyG* triglyceride-glucose index, *FLI* fatty liver index

# Discussion

In this study, we observed changes in the inflammatory potential of the diet after 3 and 12 months of surgery, changing the pro-inflammatory profile in the preoperative to anti-



**Fig. 1** Dietary inflammatory index obtained before (preoperative) and after (3 and 12 months) Roux en-Y-gastric bypass surgery (n = 50). Data expressed as mean  $\pm$  standard deviation. E-DII dietary inflammatory index. Different letters indicate the presence of a statistically significant difference in the ANOVA test of repeated and post hoc measurements of Bonferroni

inflammatory. Besides, we investigated whether preoperative E-DII scores and changes in E-DII scores after 3 and 12 months of RYGB were associated with changes in cardiometabolic risk markers. To our knowledge, this is the first study to assess the possible impact of the DII (or E-DII) or its variation on the cardiometabolic risk of patients undergoing RYGB bariatric surgery. We observed that the E-DII before surgery was negatively associated with  $\Delta$ NC and positively associated with  $\Delta$ WHR,  $\Delta$ TC, and  $\Delta$ LDL-c. These results indicate that the inflammatory potential of diet in the preoperative period, assessed by the E-DII, can predict changes in NC, WHR, TC, and LDL-c after 12 months of RYGB.

These results are contrary to the hypothesis that a diet with a more pro-inflammatory profile in the preoperative period would be associated with smaller changes in cardiometabolic risk markers. In this sense, only the marker NC showed expected behavior, indicating that although the patients had lower NC due to surgery, such changes were negatively associated with preoperative E-DII scores. Regarding WHR, TC, and LDL, we observed that the higher the E-DII before surgery, the greater the changes after 12 months. These results suggest that bariatric surgery can have a greater impact on individuals



Fig. 2 Correlation between the DII in the preoperative and the change in the cardiometabolic risk variables after RYGB. RYGB Roux-en-Y gastric bypass, DII dietary inflammatory index, NC neck circumference, TC total cholesterol, WHR waist-hip ratio, LDL-c low-density lipoprotein cholesterol

who eat poor quality diets; thus, they could significantly benefit from the procedure.

To date, only one study evaluated the relationship between the DII and bariatric surgery, where a more inflammatory diet profile before surgery was inversely associated with changes in weight and BMI after 6 months of RYGB. However, the data were not adjusted for preoperative BMI. In the cited study, the average DII before surgery was 0.35, a value close to that observed in our study (0.39) and considered to be proinflammatory [22].

We also observed a significant change in E-DII scores after 3 and 12 months of RYGB compared with the preoperative period. The reduction in the E-DII after a short period of surgery, 3 months, is expected since during this period, the patient usually eats a diet rich in fresh foods, vitamins, minerals, antioxidants, which tend to be strongly anti-inflammatory. According to previously published results, at 3 months of surgery, we observed an increase in the consumption of fresh or minimally processed foods, such as fruits, vegetables, milk, and meat, and a reduction in ultra-processed foods, such as sweets, sugary drinks, and snacks, which promote inflammation [23]. At 1-year post-surgery, patients are adapted to their new gastrointestinal condition, presenting less food intolerance and greater acceptance of foods rich in sugar and fat. Thus, there is a tendency to lapse into old eating habits. This trend was observed in our study, since at 12 months, the E-DII score was higher than at 3 months, although an anti-inflammatory profile was maintained.

Several studies have been conducted around the world aimed at understanding the association of a more proinflammatory diet with the prevalence or incidence of risk factors associated with chronic non-communicable diseases [24–26] such as neoplasms [27, 28], high blood pressure [29], cardiovascular disease [30–32], and psychiatric disorders [33, 34]. However, the findings are inconsistent [35, 36].

In general, some studies show a positive association between a pro-inflammatory diet and the risk of developing metabolic syndrome and its isolated components, such as high blood pressure, TG [37], BMI, WC, and WHR [12]. In addition, a study found that in individuals with obesity, a more pro-inflammatory diet was associated with a higher degree of liver damage (fatty liver index—FLI > 60) compared with overweight individuals [11]. In the current study, the FLI

**Table 2** Association of E-DII at preoperative,  $\Delta 3$  months, and  $\Delta 12$  months (independent variables) with  $\Delta 12$  months of cardiometabolic risk factors (dependent variable) of patients submitted to RYGB (n = 50)

Variables ( $\Delta 12$ months)	DII		
	Preoperative*	$\Delta 3 \text{ months}^{**} \beta (p \text{ value})$	$\Delta 12 \text{ months}^{**}$
$\Delta$ Weight (kg)	-0.14 (0.84)	- 1.17 (0.17)	0.61 (0.42)
$\Delta \text{ BMI (kg/m^2)}$	0.01 (0.96)	0.37 (0.22)	-0.32 (0.22)
$\Delta \operatorname{BF}(\%)$	-0.01 (0.99)	0.86 (0.14)	-0.33 (0.50)
$\Delta$ WC (cm)	0.79 (0.25)	0.37 (0.65)	-0.34 (0.62)
$\Delta$ HC (cm)	-0.31 (0.57)	0.62 (0.38)	-0.53 (0.36)
$\Delta$ NC (cm)	-0.50 (0.03)	0.18 (0.50)	-0.25 (0.29)
$\Delta$ WHR	0.01 (0.02)	-0.00 (0.34)	0.00 (0.89)
% EWL	-	0.87 (0.36)	-0.28 (0.88)
$\Delta$ TG (mg/dl)	2.69 (0.19)	2.59 (0.30)	- 1.12 (0.60)
$\Delta$ TC (mg/dl)	6.47 (0.002)	0.19 (0.94)	- 1.44 (0.49)
$\Delta$ LDL-c (mg/dl)	6.42 (0.001)	-0.86 (0.71)	0.15 (0.94)
$\Delta$ HDL-c (mg/dl)	-0.52 (0.62)	1.08 (0.38)	- 1.37 (0.20)
$\Delta$ glucose (mg/dl)	0.04 (0.70)	0.05 (0.65)	0.05 (0.69)
$\Delta$ GGT (U/L)	-0.64 (0.57)	-0.56 (0.68)	-0.78 (0.50)
$\Delta$ insulin (µUI/ml)	0.07 (0.64)	0.05 (0.79)	0.08 (0.64)
$\Delta$ HOMA-IR	-0.00 (0.94)	-0.00 (0.97)	-0.01 (0.88)
$\Delta$ TyG index	0.13 (0.29)	0.12 (0.33)	0.12 (0.32)
Δ FLI	1.10 (0.53)	1.28 (0.54)	-0.71 (0.69)

\*Adjusted for the preoperative value of the cardiometabolic risk factor analyzed. \*\*Simultaneously adjusted by the preoperative values of the analyzed cardiometabolic risk factor and the DII.  $\beta$  and p values according to linear regression

*RYGB* Roux-en-Y gastric bypass,  $\Delta$  delta, *DII* E-dietary inflammatory index, *BMI* body mass index, *WC* waist circumference, *HC* hip circumference, *NC* neck circumference, *WHR* waist-hip ratio, % *EWL* excess weight loss, *TG* triglycerides, *TC* total cholesterol, *LDL-c* low-density lipoprotein cholesterol, *HDL-c* high-density lipoprotein cholesterol, *GGT* gama glutamyl transferase, *HOMA-IR* homeostasis model assessment for insulin resistance, *TyG* triglyceride-glucose index, *FLI* fatty liver index

index, which evaluates liver fat, was not associated with DII but showed a significant reduction after bariatric surgery.

Among the limitations of this study, we highlight the instrument used to collect the food consumption data. Although considered the "gold standard" of dietary assessment, the 24HR is not error-free, including the possibility of underestimation or omission of information on actual food consumption. However, this bias, which is typical of traditional food consumption assessment instruments, is smaller in the 24HR than in structured assessments [38, 39]. Furthermore, to minimize bias, the data were collected by only one researcher, appropriately trained and qualified. In addition, data from three 24HR on non-consecutive days were used, which allowed an adequate characterization of the food intake of the patients, strengthening our results. We recognize, however, that modern instruments not based on self-report can provide a more accurate assessment of food consumption, as smartphone-based food recognition applications [40] and laboratory-based food intake measurements [41], which have already been introduced in studies on bariatric surgery.

# Conclusion

We observed significant reductions in dietary inflammation, as measured by the E-DII, after 3, and 12 months of RYGB. Also, we observed by linear regression analysis that patients with higher preoperative E-DII scores have a greater metabolic improvement in parameters such as WHR, CT, and LDL after 12 months of surgery. However, other studies should be carried out, with a control group, to assess whether these changes are associated exclusively with surgery.

Acknowledgments To the Coordination for the Improvement of Personnel at the - CAPES for the financing of the project

**Authors' Contributions** Sônia L Pinto contributed in the design of the study, data collection, analysis and interpretation, manuscript writing, and final version approval. Sônia L Pinto, Leidjaira L Juvanhol, Alessandra da Silva, and Josefina Bressan contributed in the design of the study, analysis and interpretation of the data, critical revision of the manuscript, and approval of the final version. Nitin Shivappa and James R Hébert contributed in analysis and interpretation of the final version.

# **Compliance with Ethical Standards**

**Conflict of Interest** The authors declare that they have no conflicts of interest.

**Ethical Approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

# References

- Cox AR, Chernis N, Masschelin PM, et al. Immune cells gate white adipose tissue expansion. Endocrinology. 2019;160:1645–58.
- Ouchi N, Parker JL, Lugus JJ, et al. Adipokines in inflammation and metabolic disease. Nat Rev Immunol Nature Publishing Group. 2011;11:85–97.
- Mechanick JI, Kushner RF, Sugerman HJ, et al. Guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. Endocr Pract. 2008;14:1–83.
- Illán-Gómez F, Gonzálvez-Ortega M, Orea-Soler I, et al. Obesity and inflammation: change in adiponectin, C-reactive protein, tumour necrosis factor-alpha and interleukin-6 after bariatric surgery. Obes Surg. 2012;22:950–5.
- Viana EC, Araujo-Dasilio KL, Miguel GPS, et al. Gastric bypass and sleeve gastrectomy: the same impact on IL-6 and TNF-α. prospective clinical trial. Obes Surg. 2013;23:1252–61.
- Muller MK, Gero D, Reitnauer D, et al. Response to letter to the editor: the impact of Roux-en-Y gastric bypass on bone remodeling expressed by the P1NP/βCTX ratio: a single-center prospective cohort study. Obes Surg. 2019;29:3708–9.
- Namazi N, Larijani B, Azadbakht L. Dietary inflammatory index and its association with the risk of cardiovascular diseases, metabolic syndrome, and mortality: a systematic review and meta-analysis. Horm Metab Res. 2018;50:345–58.
- Shivappa N, Hebert JR, Marcos A, et al. Association between dietary inflammatory index and inflammatory markers in the HELENA study. Mol Nutr Food Res. 2017;61:1–23.
- 9. Shivappa N, Steck SE, Hurley TG, et al. Dietary inflammatory index. Public Health Nutr. 2014;17:1689–96.
- Kim HS, Sohn C, Kwon M, et al. Positive association between dietary inflammatory index and the risk of osteoporosis: results from the KoGES\_Health Examinee (HEXA) cohort study. Nutrients. 2018;10:1999.
- Cantero I, Abete I, Babio N, et al. Dietary inflammatory index and liver status in subjects with different adiposity levels within the PREDIMED trial. Clin Nutr. 2018;37:1736–43.
- Ruiz-Canela M, Zazpe I, Shivappa N, et al. Dietary inflammatory index and anthropometric measures of obesity in a population sample at high cardiovascular risk from the PREDIMED (PREvención con Dleta MEDiterránea) trial. Br J Nutr. 2015;113:984–95.
- Garcia-Arellano A, Martínez-González MA, Ramallal R, et al. Dietary inflammatory index and all-cause mortality in large cohorts: the SUN and PREDIMED studies. Clin Nutr. 2018;38:1–11.
- IBGE. Instituto Brasileiro de Geografia e Estatística Pesquisa de Orçamentos Familiares: 2008–2009. Análise do Consumo Alimentar Pessoal no Brasil. Bibl. do Minist. do Planejamento, Orçamento e Gestão. 2011.
- Matthews DR, Hosker JP, Rudenski AS, et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28:412–9.

- Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord. 2008;6:299–304.
- Bedogni G, Bellentani S, Miglioli L, et al. The fatty liver index: a simple and accurate predictor of hepatic steatosis in the general population. BMC Gastroenterol. 2006;6:33.
- Jellife D. Evoluación del estado nutricion dela comunidad. Ginebra: Organización Mundial de la Salud. Série Monogr. 1968;53.
- Callaway CW. et al. Circumferences. Apud Lohman, TG; Roche, AF; Martorell, R Anthr Standarization Ref Manual Hum Kinet Books Illinois. 1988.
- Ben-Noun L, Laor A. Relationship of neck circumference to cardiovascular risk factors. Obes Res. 2003;11:226–31.
- Salud. OM de la. El estado físico: uso e interpretación de la antropometría: informe de un Comitê de Expertos de la OMS. Ginebra Oragización Mund la Salud. 1995.
- 22. Andrade PA, Hermsdorff HHM, Leite JIA, et al. Baseline proinflammatory diet is inversely associated with change in weight and body fat 6 months following-up to bariatric surgery. Obes Surg. 2019;29:457–63.
- Pinto SL, da Silva DCG, Bressan J. Absolute and relative changes in ultra-processed food consumption and dietary antioxidants in severely obese adults 3 months after Roux-en-Y gastric bypass. Obes Surg. 2019;29:1810–5.
- 24. Phillips C, Shivappa N, Hébert J, et al. Dietary inflammatory index and biomarkers of lipoprotein metabolism, Inflammation and Glucose Homeostasis in Adults. Nutrients. 2018;10:1033.
- Mazidi M, Shivappa N, Wirth MD, et al. Dietary inflammatory index and cardiometabolic risk in US adults. Atherosclerosis. 2018;276:23–7.
- Kim H-Y, Lee J, Kim J. Association between dietary inflammatory index and metabolic syndrome in the general Korean population. Nutrients. 2018;10:648.
- Shivappa N, Godos J, Hébert JR, Wirth MD, Piuri G, Speciani AF, et al. Dietary inflammatory index and colorectal cancer risk—a meta-analysis. Nutrients. 2017;9:1043.
- Shivappa N, Hébert JR, Polesel J, et al. Inflammatory potential of diet and risk for hepatocellular cancer in a case-control study from Italy. Br J Nutr. 2016;115:324–31.
- Vissers LET, Waller MA, van der Schouw YT, et al. The relationship between the dietary inflammatory index and risk of total cardiovascular disease, ischemic heart disease and cerebrovascular disease: findings from an Australian population-based prospective cohort study of women. Atherosclerosis Elsevier Ltd. 2016;253: 164–70.
- Ramallal R, Toledo E, Martínez-González MA, et al. Dietary inflammatory index and incidence of cardiovascular disease in the SUN cohort. Eckel J, editor. PLoS One. 2015;10:e0135221.
- Garcia-Arellano A, Ramallal R, Ruiz-Canela M, et al. Dietary inflammatory index and incidence of cardiovascular disease in the PREDIMED study. Nutrients. 2015;7:4124–38.
- 32. Shivappa N, Godos J, Hébert JR, et al. Dietary inflammatory index and cardiovascular risk and mortality—a meta-analysis. Nutrients. 2018;10:1–15.
- Phillips CM, Shivappa N, Hébert JR, et al. Dietary inflammatory index and mental health: a cross-sectional analysis of the relationship with depressive symptoms, anxiety and well-being in adults. Clin Nutr. 2018;37:1485–91.
- Wirth MD, Shivappa N, Burch JB, et al. The dietary inflammatory index, shift work, and depression: results from NHANES. Health Psychol. 2017;36:760–9.
- Ren Z, Zhao A, Wang Y, et al. Association between dietary inflammatory index, C-Reactive Protein and Metabolic Syndrome: A Cross-Sectional Study. Nutrients. 2018;10:831.

- Sokol A, Wirth MD, Manczuk M, et al. Association between the dietary inflammatory index, waist-to-hip ratio and metabolic syndrome. Nutr Res. 2016;36:1298–303.
- Neufcourt L, Assmann KE, Fezeu LK, et al. Prospective association between the dietary inflammatory index and cardiovascular diseases in the SUpplémentation en VItamines et Minéraux AntioXydants (SU.VI.MAX) cohort. J Am Heart Assoc. 2016;5: e002735.
- Shook RP, Hand GA, O'Connor DP, et al. Energy intake derived from an energy balance equation, validated activity monitors, and dual x-ray absorptiometry can provide acceptable caloric intake data among young adults. J Nutr. 2018;148:490–6.
- Hebert JR, Ebbeling CB, Matthews CE, et al. Systematic errors in middle-aged women's estimates of energy intake: comparing three

self-report measures to total energy expenditure from doubly labeled water. Ann Epidemiol. 2002;12:577–86.

- O'Loughlin G, Cullen SJ, McGoldrick A, et al. Using a wearable camera to increase the accuracy of dietary analysis. Am J Prev Med. Elsevier Inc. 2013;44:297–301.
- Nielsen MS, Christensen BJ, Ritz C, et al. Roux-en-Y gastric bypass and sleeve gastrectomy does not affect food preferences when assessed by an ad libitum buffet meal. Obes Surg. 2017;27:2599– 605.

**Publisher's Note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.