

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

Roberto Carlos Burini<sup>1</sup>, Gabriel Augusto Torezan<sup>1,2</sup>, Lance A Sloan<sup>3</sup>, José Eduardo Corrente<sup>4</sup> and Katia Cristina Portero McLellan<sup>1,3\*</sup>

<sup>1</sup>Center for Exercise Metabolism and Nutrition (CeMENutri), Department of Public Health, School of Medicine, Sao Paulo State University (UNESP), Botucatu/SP, Brazil

<sup>2</sup>Post Graduate Student of Pathology, School of Medicine, Sao Paulo State University (UNESP), Botucatu/SP, Brazil

<sup>3</sup>Texas Institute for Kidney and Endocrine Disorders, Lufkin/TX, USA

<sup>4</sup>Department of Bioestatistic, Bioscience Institute, School of Medicine, Sao Paulo State University (UNESP), Botucatu/SP, Brazil

\*Corresponding author: Katia Cristina Portero McLellan, Texas Institute for Kidney and Endocrine Disorders, Lufkin/TX, USA, Tel: 14-3880-1344; E-mail: [kmclellan@fmb.unesp.br](mailto:kmclellan@fmb.unesp.br)

Received date: Jan 17, 2014; Accepted date: Mar 24, 2014; Published date: Mar 26, 2014

Copyright: © 2014 Burini RC, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### 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  $VO_2$ max.

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 “Mexe-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  $53.3 \pm 10.4$  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

(LISC) has been described elsewhere [8]. This study was conducted in accordance with the Declaration of Helsinki (1964). Subjects gave their written consent to participate in the study, which was approved by the Medical Ethics Committee of Sao Paulo State University – Medical School, Botucatu/SP.

Participants (n=264) were divided into three groups, normoglycemic (n=192), IFG (n=47), and T2DM (n=25). They were considered normoglycemic when fasting plasma glucose was  $\leq 5.5$  mmols/L ( $\leq 100$  mg/dL) and hyperglycemic when plasma glucose was  $\geq 5.5$  mmols/L ( $\geq 100$  mg/dL). T2DM was defined by plasma glucose  $>7.0$  mmols/L ( $\geq 126$  mg/dL). All participants were assessed at baseline (M0) and at the end of the study (M1) for biochemical, anthropometric, and dietary intake data. Participants were not taking medication to control blood glucose (insulin or oral antidiabetic therapy).

Dietary counseling was delivered by a Brazilian certified dietitian that met weekly with all participants to discuss the dietary intervention program. The dietary intervention discussed diet quality and dietary recommendation for nutrients such as: protein (10-35% of total calories); carbohydrates (45-65% of total calories); total fat (20-35% of total calories); saturated fat ( $<10\%$  of total calories), cholesterol ( $<300$  mg/day); sodium ( $<2,400$  mg/day) and dietary fiber ( $>25$  g/day for women and  $>30$  g/day for men). Individuals were also informed about ingredients substitution in meals to increase fiber intake through adding fruits and vegetables to the recipe and different cooking methods. In addition, participants were encouraged to increase the daily intake of fruits and vegetables, whole grain cereals, legumes, low-fat dairy products, and lean meat, fish, or poultry as recommended in the Brazilian food guide [9].

### 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  $-80^{\circ}\text{C}$  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<sup>®</sup>, 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<sup>®</sup> 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<sup>®</sup>, 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<sup>®</sup>, 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  $\text{MM (kg)}/\text{height}^2$ .

### 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 dietitian, 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 non-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  $>$  normoglycemic) and thus differing them by the number of altered MetS components. T2DM group was discriminated from normoglycemic 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 (normoglycemic, IFG, and T2DM) responded similarly to LISC (Table 2).

	Normoglycemic (n=660)	IFG (n=243)	T2DM (n=101)
Age (years)	52.1 ± 9.9 <sup>a</sup>	54.8 ± 9.4 <sup>b</sup>	56.3 ± 1 <sup>b</sup>
BMI	28.5 ± 5.2 <sup>a</sup>	30.1 ± 5.0 <sup>b</sup>	30.7 ± 5.3 <sup>b</sup>
WC (cm)	92.8 ± 13.5 <sup>a</sup>	98.4 ± 12.4 <sup>b</sup>	101.4 ± 12.4 <sup>b</sup>
BF (%)	31.8 ± 8.6 <sup>a</sup>	32.6 ± 7.6 <sup>a</sup>	33.7 ± 8.8 <sup>a</sup>
MMI (kg/m <sup>2</sup> )	10.0 ± 5.8 <sup>a</sup>	13.2 ± 5.5 <sup>b</sup>	12.8 ± 5.8 <sup>b</sup>
Uric Acid (mg/dL)	4.65 ± 1.48 <sup>a</sup>	4.8 ± 1.7 <sup>a</sup>	5.12 ± 1.84 <sup>b</sup>
HDL-c (mg/dL)	51.8 ± 15.1 <sup>a</sup>	53.3 ± 16.1 <sup>a</sup>	49.6 ± 17.7 <sup>a</sup>
Glucose (mg/dL)	88.3 ± 7.6 <sup>a</sup>	108.5 ± 7.2 <sup>b</sup>	177.2 ± 50.1 <sup>c</sup>
Triglycerides (mg/dL)	148.2 ± 104.7 <sup>a</sup>	178.4 ± 99.6 <sup>b</sup>	207.8 ± 129.0 <sup>c</sup>
Gamma GT (U/L)	36.0 ± 29.1 <sup>a</sup>	43.2 ± 29.8 <sup>b</sup>	53.3 ± 37.1 <sup>b</sup>
SBP (mmHg)	125.8 ± 16.2 <sup>a</sup>	132.1 ± 18.8 <sup>b</sup>	134.2 ± 18.1 <sup>b</sup>
DBP (mmHg)	80.3 ± 10.5 <sup>a</sup>	82.7 ± 11.5 <sup>b</sup>	84.7 ± 12.4 <sup>b</sup>
MetS components	1.3 ± 1.1 <sup>a</sup>	2.9 ± 1.1 <sup>b</sup>	4.0 ± 1.0 <sup>c</sup>
Meat (servings)	1.8 ± 1.6 <sup>a</sup>	2.1 ± 1.4 <sup>ab</sup>	2.3 ± 1.8 <sup>b</sup>
Sugar (servings)	1.6 ± 1.9 <sup>a</sup>	1.5 ± 1.8 <sup>ab</sup>	0.9 ± 1.3 <sup>b</sup>
Vegetable oil (servings)	2.6 ± 6.4 <sup>a</sup>	2.4 ± 1.8 <sup>a</sup>	2.2 ± 2.3 <sup>a</sup>
Variety (items)	13.1 ± 4.2 <sup>a</sup>	12.9 ± 4.4 <sup>ab</sup>	11.8 ± 3.7 <sup>b</sup>
Fiber (g)	13.5 ± 7.5 <sup>a</sup>	13.8 ± 7.1 <sup>a</sup>	14.2 ± 8.0 <sup>a</sup>
HEI (points)	79.3 ± 15.1 <sup>a</sup>	77.2 ± 16.8 <sup>a</sup>	82.1 ± 13.0 <sup>a</sup>

**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

	Normoglycemic (n=192)		IFG (n=47)		T2DM (n=25)	
	Baseline	After 20 weeks	Baseline	After 20 weeks	Baseline	After 20 weeks
Fasting glucose (mg/dl)	87.27 (6.75)Aa	90.60 (17.66)Aa	107.28 (7.10)Ba	101.50 (16.35)Ba	182.54 (51.49)Ca	159.29 (53.04)Cb
Fasting Insulin (mg/dl)	9.27 (6.52)Aa	11.17 (10.15)Aa	8.66 (5.65)Aa	10.24 (6.79)Aa	16.68 (10.05)Aa	12.34 (2.51)Aa
BMI (kg/m <sup>2</sup> )	30.81 (6.48)Aa	30.18 (5.62)Aa	33.48 (6.88)Aa	32.28 (6.24)Aa	32.31 (5.80)Aa	31.81 (6.28)Aa
WC (cm)	97.31 (15.16)Aa	94.46 (12.86)Aa	103.79 (14.24)Aa	102.45 (14.76)Aa	101.05 (13.54)Aa	99.23 (15.73)Aa
BF (%)	36.29 (8.87)Aa	35.71 (8.65)Aa	37.43 (8.70)Aa	34.57 (8.60)Aa	40.76 (8.02)Aa	40.27 (8.50)Aa
MMI (kg/m <sup>2</sup> )	8.33 (1.52)Aa	8.27 (1.53)Aa	9.26 (2.02)Aa	9.23 (2.10)Aa	7.90 (1.09)Aa	7.99 (1.07) Aa
Protein (%)	17.35 (5.25)Aa	17.80 (5.50)Aa	17.88 (4.57)Aa	18.16 (4.17)Aa	18.14 (4.71)Aa	21.73 (6.16)Aa
CHO (%)	53.16 (10.36)Aa	53.62 (8.64)Aa	52.98 (7.59)Aa	54.29 (8.79)Aa	52.76 (8.61)Aa	52.84 (6.88)Aa
Total Fat (%)	30.19 (10.82)Aa	28.36 (7.14)Aa	29.13 (6.40)Aa	27.61 (8.21)Aa	29.09 (8.26)Aa	25.42 (4.20)Aa

Cholesterol (mg)	186.08 (130.46)Aa	157.97 (106.24)Aa	245.62 (211.81)Aa	196.84 (111.00)Aa	171.24 (67.82)Aa	158.27 (68.02)Aa
Fiber (g)	15.74 (8.48)Aa	20.84 (13.64)Aa	14.00 (8.65)Aa	18.87 (17.43)Aa	14.23 (10.37)Aa	21.11 (8.52)Aa
Meat (serving)	1.44 (1.12)Aa	1.35 (1.02)Aa	1.70 (1.35)Aa	1.43 (0.89)Aa	1.53 (0.87)Aa	1.56 (1.09)Aa
Sugar (serving)	1.37 (1.70)Aa	0.96 (1.16)Aa	1.16 (1.69)Aa	1.29 (1.62)Aa	1.03 (1.69)Aa	0.60 (1.34)Aa
Vegetable oil (serving)	2.14 (2.35)Aa	1.86 (1.38)Aa	2.17 (2.39)Aa	1.87 (1.06)Aa	1.90 (1.51)Aa	1.83 (1.14)Aa
Variety (items)	13.16 (4.26)Aa	13.91 (4.47)Aa	12.69 (4.27)Aa	13.64 (4.61)Aa	11.58 (4.04)Aa	14.21 (4.00)Aa
HEI (points)	76.99 (14.17)Aa	81.45 (12.57)Aa	72.35 (14.87)Aa	77.58 (19.85)Aa	80.35 (12.19)Aa	84.62 (9.97)Aa

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

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

Capital letters compare groups and small letters compare moments (baseline and After 20 weeks). Different letters p<0.05

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).

	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/m <sup>2</sup> )	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/m <sup>2</sup> )	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 (itens)	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)

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

\* p<0.05

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 normoglycemic and 75.0% with T2DM and IFG had abnormal WC.

After the intervention, the prevalence of normal WC increased by 2.2% among normoglycemics subjects and 6.2% among T2DM subjects, but IFG subjects had an increased in the prevalence of altered abdominal WC (6.2%) (Table 4).

With LISC T2DM decreased from 9.5% (M0) to 6.4% (M1). A calculated reduction of 68% (Table 5). The better responses of T2DM to LISC were from either obeses or eutrophics but with normalized WC.

	Baseline			After 20 weeks			p- value*
	Normo-glycemic (n=192)	IFG (n=47)	T2DM (n=25)	Normo-glycemic (n=192)	IFG (n=47)	T2DM (n=25)	
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/m <sup>2</sup> )							
≤ 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 4:** Percentage of Healthy Eating Index, waist circumference and body mass index classifications of groups at baseline and after the intervention

IFG=Impaired fasting glucose; T2DM=Type 2 Diabetes Mellitus; HEI=Healthy Eating Index; BMI=Body Mass index; WC=Waist Circumference

\* trend chi-square test

	Baseline	After 20 weeks		
		Normoglycemic	IFG	T2DM
Normoglycemic	192 (72.7)	166 (62.9*; 86.4)	21 (7.9*; 10.9)	5 (1.9*; 2.6)
IFG	47 (17.8)	25 (9.6*; 53.2)	17 (6.5*; 36.2)	5 (1.9*; 10.6)
T2DM	25 (9.5)	2 (0.6*; 8.0)	6 (2.3*; 24.0)	17 (6.4*; 68.0)
Total	264 (100.0)	193 (73.1)	44 (16.7)	27 (10.2)

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

IFG=Impaired fasting glucose; T2DM=Type 2 Diabetes Mellitus

\*Percentage based on total population (n=264)

## 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 normoglycemics 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 and oxidative stress [32], which may explain the hyperglycemia and higher plasma levels of Gamma GT among the T2DM subjects studied. Gamma GT is a nonspecific marker of oxidative stress [33], and it is possible that increased oxidative stress plays an important role in the association of red meat intake and hyperglycemia. Red meat is a major food source of protein and fat, and its consumption is associated with cardiovascular disease and diabetes [34,35]. The high consumption of red meat also increases the intake of SFA, nitrates and nitrite, glycation end products [36,37]. An increase of plasma insulin and SFA intake was seen in men after the intervention, which may be related to the high consumption of red meat. A decrease of soybean oil intake was also seen among men, that could be a result of a decreased intake of deep fried foods. One of the topics discussed in the dietary intervention program was related to decreasing intake of soybean oil by avoiding deep fried foods. Men had a better adherence to this recommendation than women. On the other hand, women increased dietary fiber consumption and healthy eating index after the intervention compared to men, even though all participants were encouraged to increase daily intake of fruits, vegetables, legumes, and whole-grain cereals.

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.

## References

1. Shaw JE, Sicree RA, Zimmet PZ (2010) Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 87: 4-14.
2. Pinney SE, Simmons RA (2010) Epigenetic mechanisms in the development of type 2 diabetes. *Trends Endocrinol Metab* 21: 223-229.
3. Jin W, Patti ME (2009) Genetic determinants and molecular pathways in the pathogenesis of Type 2 diabetes. *Clin Sci (Lond)* 116: 99-111.
4. Wells JC (2011) The thrifty phenotype: An adaptation in growth or metabolism? *Am J Hum Biol* 23: 65-75.
5. Hu FB, Willett WC (2002) Optimal diets for prevention of coronary heart disease. *JAMA* 288: 2569-2578.
6. McLellan KCP, Lerário AC, Burini RC (2011) Prevention of Diabetes type 2: Effects of a Lifestyle Intervention. In: Zimring MB, Topics in the prevention, treatment and complications of Type 2 Diabetes (1st Edn), Intech Open Access Publisher.
7. Mota JF, Moreto F, Burini FHP, Medina WL, Rimm EB, et al. (2011) Effect of physical conditioning with lifestyle intervention on a

- community-based hyperglycemic-overweight adults. *J US-China Med Sciences* 8: 581-587.
8. Portero-McLellan KC, Pimentel GD, Corrente JE, Burini RCC (2010) Association of fat intake and socioeconomic status on anthropometric measurements of adults. *Rev Saúde Coletiva* 18: 266-274.
  9. Brasil. Ministério da Saúde, Secretaria de Atenção à Saúde (2008) Guia alimentar para a população brasileira: promovendo a alimentação saudável/Ministério da Saúde, Secretaria de Atenção à Saúde, Brasília: Ministério da Saúde.
  10. Friedewald WT, Levy RI, Fredrickson DS (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 18: 499-502.
  11. (2000) Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 894: i-xii, 1-253.
  12. Janssen I, Heymsfield SB, Baumgartner RN, Ross R (2000) Estimation of skeletal muscle mass by bioelectrical impedance analysis. *J Appl Physiol* (1985) 89: 465-471.
  13. NEPA/UNICAMP. Tabela brasileira de composição de alimentos. TACO. 1 ed. Campinas/SP, 2004.
  14. Philippi ST (2002) Tabela de Composição de Alimentos: Suporte para decisão nutricional. (2nd Edn), São Paulo.
  15. Mota JF, Rinaldi AEM, Pereira AF, Maestá N, Scarpin MM, et al. (2008) Adaptação do Índice de Alimentação Saudável ao Guia Alimentar da População Brasileira. *Rev Nutr* 21: 545-552.
  16. American College of Sports Medicine (2009) American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. *Med Sci Sports Exerc* 41: 687-708.
  17. Sartorelli DS, Franco LJ (2003) [Trends in diabetes mellitus in Brazil: the role of the nutritional transition]. *Cad Saude Publica* 19 Suppl 1: S29-36.
  18. Torquato MT, Montenegro Júnior RM, Viana LA, de Souza RA, Lanna CM, et al. (2003) Prevalence of diabetes mellitus and impaired glucose tolerance in the urban population aged 30-69 years in Ribeirão Preto (São Paulo), Brazil. *Sao Paulo Med J* 121: 224-230.
  19. da Costa JS, Olinto MT, Assunção MC, Gigante DP, Macedo S, et al. (2006) [Prevalence of Diabetes Mellitus in Southern Brazil: a population-based study]. *Rev Saude Publica* 40: 542-545.
  20. Finucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, et al. (2011) National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet* 377: 557-567.
  21. de Vegt F, Dekker JM, Jager A, Hienkens E, Kostense PJ, et al. (2001) Relation of impaired fasting and postload glucose with incident type 2 diabetes in a Dutch population: The Hoorn Study. *JAMA* 285: 2109-2113.
  22. Bosello O, Zamboni M (2000) Visceral obesity and metabolic syndrome. *Obes Rev* 1: 47-56.
  23. Lefebvre AM, Laville M, Vega N, Riou JP, van Gaal L, et al. (1998) Depot-specific differences in adipose tissue gene expression in lean and obese subjects. *Diabetes* 47: 98-103.
  24. Farrell GC (2003) Non-alcoholic steatohepatitis: what is it, and why is it important in the Asia-Pacific region? *J Gastroenterol Hepatol* 18: 124-138.
  25. Fabbrini E, Sullivan S, Klein S (2010) Obesity and nonalcoholic fatty liver disease: biochemical, metabolic, and clinical implications. *Hepatology* 51: 679-689.
  26. Bays HE, Chapman RH, Grandy S; SHIELD Investigators' Group (2007) The relationship of body mass index to diabetes mellitus, hypertension and dyslipidaemia: comparison of data from two national surveys. *Int J Clin Pract* 61: 737-747.
  27. Hjellvik V, Sakshaug S, Strøm H (2012) Body mass index, triglycerides, glucose, and blood pressure as predictors of type 2 diabetes in a middle-aged Norwegian cohort of men and women. *Clin Epidemiol* 4: 213-224.
  28. Salas-Salvadó J, Martínez-González MÁ, Bulló M, Ros E (2011) The role of diet in the prevention of type 2 diabetes. *Nutr Metab Cardiovasc Dis* 21 Suppl 2: B32-48.
  29. McNaughton SA, Dunstan DW, Ball K, Shaw J, Crawford D (2009) Dietary quality is associated with diabetes and cardio-metabolic risk factors. *J Nutr* 139: 734-742.
  30. Gopinath B, Rochtchina E, Flood VM, Mitchell P (2013) Diet quality is prospectively associated with incident impaired fasting glucose in older adults. *Diabet Med* 30: 557-562.
  31. Mizoue T, Yamaji T, Tabata S, Yamaguchi K, Ogawa S, et al. (2006) Dietary patterns and glucose tolerance abnormalities in Japanese men. *J Nutr* 136: 1352-1358.
  32. Montonen J, Boeing H, Fritsche A, Schleicher E, Joost HG, et al. (2013) Consumption of red meat and whole-grain bread in relation to biomarkers of obesity, inflammation, glucose metabolism and oxidative stress. *Eur J Nutr* 52: 337-345.
  33. Lee DH, Blomhoff R, Jacobs DR Jr (2004) Is serum gamma glutamyltransferase a marker of oxidative stress? *Free Radic Res* 38: 535-539.
  34. Micha R, Wallace SK, Mozaffarian D (2010) Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. *Circulation* 121: 2271-2283.
  35. Pan A, Sun Q, Bernstein AM, Schulze MB, Manson JE, et al. (2011) Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. *Am J Clin Nutr* 94: 1088-1096.
  36. Peppas M, Goldberg T, Cai W, Rayfield E, Vlassara H (2002) Glycotoxins: a missing link in the "relationship of dietary fat and meat intake in relation to risk of type 2 diabetes in men". *Diabetes Care* 25: 1898-1899.
  37. Song Y, Manson JE, Buring JE, Liu S (2004) A prospective study of red meat consumption and type 2 diabetes in middle-aged and elderly women: the women's health study. *Diabetes Care* 27: 2108-2115.
  38. Slavin J (2003) Why whole grains are protective: biological mechanisms. *Proc Nutr Soc* 62: 129-134.
  39. Sorriquer F, Rojo-Martínez G, Valdés S, Tapia MJ, Botas P, et al. (2013) Factors determining weight gain in adults and relation with glucose tolerance. *Clin Endocrinol (Oxf)* 78: 858-864.