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## **Oral Inflammatory Load and Salivary Flow Rate in Morbidly Obese Patients in Saudi Arabia -2023**

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### **Abstract**

Obesity and diabetes promote periodontal disease (PD). The bariatric care protocol, which is a very-low-calorie diet (VLCD) followed by bariatric surgery (BSx), is an effective treatment for Obesity and diabetes but little is known about its effect on oral health. Study objectives are: 1) to Assess PD prevalence in obese subjects using oral inflammatory load (OIL); 2) to determine the Difference in OIL and salivary flow rate (SFR) between obese patients with and without diabetes; and 3) to assess the effects of VLCD, BSx and bariatric care protocol on OIL and SFR. Findings suggest that, based on OIL, PD prevalence is similar to that reported in the literature. Both



VLCD and BSx improve glucose metabolism and weight, but have no impact on oral parameters. However, patients with low SFR at baseline are more prone to increased OIL post-surgery, suggesting a higher risk of developing PD.

## **1. Introduction:**

### **Definition**

Obesity is defined as the presence of excess fat and is measured by calculating body mass index (BMI), which is an individual's body weight divided by the square of their height. A BMI score of 30–39.9 kg/m<sup>2</sup> is considered obese and morbid obesity is represented by a score of 40 kg/m<sup>2</sup> or more. Multiple etiological factors predispose individuals to gain weight. Behavioral causes include overnutrition, unhealthy food choices, sedentary lifestyle, and insufficient sleep [2]. Others include the gut microbiome [3], genetic factors, medications, or diseases including hypothyroidism and polycystic ovarian syndrome[2].

### **1.1.Obesity:**

#### **a) introduction leading to obesity**

The amount of daily energy requirement differs among individuals based on their basal metabolic rate and activity level, the energy their bodies use to maintain essential bodily functions, and the thermic effect of food. When dietary intake is in excess of the dietary requirement, the result is a positive energy balance. The body must then store the extra energy, causing weight gain. Ref?

#### **1- Overnutrition**

Overnutrition is defined as the overconsumption of food, especially food high in refined sugar, carbohydrate, and saturated fat. This increases levels of both glucose [6] and free fatty acids



(FFA)[2]. Digested food is carried to target cells, and any extra is stored in adipose tissue. Adipocytes undergo hypertrophy and hyperplasia in order to accommodate a large amount of stored energy. Once adipocytes reach maximum volume and cannot expand further, the number of adipocytes increases [7].

## **2- Diet quality**

The quality of diet is important to supplement body with healthy type of nutrients which depends on personal choices and availabilities. Since the concept of food shifted from nourishing body to source of energy rich and poor nutritive diet. It is important to address the effect of frequent consumption of this diet. It negatively impacts weight especially with no proper physical activity. In the response to the importance influence of food choices on weight management, supporting healthier diet with active lifestyle will help to reduce obesity rate.

Frequent consumption of unhealthy, energy dense diet greatly influence weight gain especially with no focus on diet quality which is important to supplement body with healthy type of nutrients. This depends on personal choices and availabilities.

## **3- Sedative life style**

Lack of physical activity greatly contribute to an increase in body weight and fat mass. Many studies have shown that inactivity is linked to increased rate of obesity. Regular moderate-intensity exercise with calorie restriction are effective in weight management. However, it might be conformed with other factors such as genes that might contribute to obesity.

Sedative lifestyle might not be the primary cause to obesity. It is influenced by other factors such as genes. Risk of obesity increase. Inactivity and unhealthy diet increase the risk of weight gain. The risk increase with different genes.

## **4- Genetics**

Genetics factors influence the susceptibility of weight gain in certain conditions? Environment

## **5- Intestinal microbiome**



## 2 Objectives and Hypotheses

### 3.1 Objectives:

In a cross-sectional study:

1) To determine the differences between obese with and without type 2 diabetes in oral measurements (oral inflammatory load (OIL) and stimulated salivary flow rate (SFR))

In a prospective cohort study:

2) To determine the effect of very low calorie diet (VLDC; Optifast) on oral measurements (OIL, SFR) in obese patients.

3) To determine the effect of bariatric surgery (BSx) on OIL and SFR in obese patients. Separately, from Optifast in order to assess if the effects from the 2 interventions are different.

4) To determine if the changes in oral parameters (OIL, SFR) during the bariatric care protocol correlate with changes in BMI as well as metabolic changes such as insulin resistance, HbA1c or improvement in T2D.

### 3.2 Hypotheses:

- 1) Differences will be observed between diabetic and non-diabetic obese subjects in oral measures.
- 2) Oral measurements will be improved with the VLCD.
- 3) Oral measurements will change similarly when the effect of bariatric surgery is compared to the effect of the VLCD.
- 4) Oral measurements changes will correlate with changes in BMI and metabolic changes such as HbA1c, insulin resistance, and improvement in T2D through the bariatric care protocol.

## Methods and Materials



### 3.3 Study Design and Protocol

This study is a prospective cohort study looking at the changes of oral health prior and post-bariatric surgery. Clinical data and oral samples collection from each participant corresponded with the standard bariatric clinical visits (Pre-Optifast<sup>®</sup>, post-Optifast<sup>®</sup>/surgery day, and 1-month post-surgery). This study is a part of a large project that looks at intestinal microbiota and other oral measurements in the same patient population with different outcomes measures featured in future studies.

### 3.4 Data collection

#### 3.4.1 Oral inflammatory load

This test is a simple non-invasive method developed by Dr. Glogauer team to detect periodontal disease, which has been used in different patient populations, including pregnant women, and patients with restricted mobility [88, 89]. This analysis identifies the level of polymorphonuclear cells (neutrophils), major inflammatory response cells, present in an oral rinse giving the oral inflammatory load (OIL). Neutrophils counts correlate with the severity of periodontal disease [90]. Moreover, neutrophil count has been shown to decrease after periodontal treatment, root scaling, and planing was administered in patients with chronic periodontitis [9]. This analysis is suitable for detecting moderate-to-severe PD in an oral rinse, with a cut-off neutrophil count of  $>3 \times 10^6$  provided a sensitivity of 0.83 and a specificity of 0.79 with a 0.94 positive predictive value and 0.55 negative predictive value [9]. The study used this rinse as an alternative of dental examination for PD with morbidly obese patients because of its feasibility in a non-dental clinic [88, 89]. In addition, obesity-related restrictions may support the practicality of oral rinse as a diagnostic tool for PD since visiting dental clinics is difficult for morbidly obese individuals due to the absence of larger chairs or facilities that accommodate them comfortably [9].



The collection protocol and analysis were as follows. Subjects were instructed to rinse their mouths with 10 ml of tap water for 5 seconds and wait a minimum of 2 minutes. Then, they were given 10 ml saline to rinse their mouths for 30 seconds and release into a 50-ml falcon tube. The samples were kept at 4 °C degrees and transported to the lab for analysis within 24 hours of collection [91]. Sample analysis was performed with some modifications, following a recent study from Glogauer's lab [90]. The rinse was prepared by adding 50 ul of 37% formaldehyde to 500 ul of oral rinse. The sample was centrifuged for 5 minutes at 4 °C at 13.0 x 1000 rpm (Hettich Rotina 35R, Rare Scientific, Edmonton, Canada) after incubation for 15 min on ice or 4 °C. The supernatant was removed and 100ul of 1xPBS added to each tube. To visually count neutrophils, samples were stained with 1 ul of acridine orange, which allows the stain to identify neutrophils from other cells. They were incubated in a dark place with room temperature for 15 minutes. Then, neutrophils were measured using the hemocytometer method. The number of neutrophils was counted from the middle square of the hemocytometer slide. Finally, the result was multiplied by 20,000 to determine the number of PMNs in the 10-ml mouth rinse.

### 3.4.2 Salivary flow rate

Stimulated saliva was collected in a well-ventilated room with the patient comfortably seated. The patient was asked to chew on a piece of Parafilm (1.40 g). Saliva produced during the first 30 seconds was discarded. The participant continued chewing and spat into a pre-weighed 50 ml Falcon tube every 30 seconds until a volume of 5 ml was reached. The time was recorded after the first 30 seconds until the end of saliva collection. The 50-ml Falcon tube was weighed after collection to calculate stimulated salivary flow rate, which is the volume of saliva collected over the collection time (ml/min) [9]. A result of  $\geq 1$  ml/min was considered a normal salivary flow rate. Below this number, patients were diagnosed with hyposalivation [22].



### 3.5 Blood Profile

12-hours fasting blood tests: insulin, glucose, HbA1C and C-peptide protein were measured in all visits with the standard protocol in Toronto Western Hospital. Furthermore, homeostasis model assessment estimated insulin resistance (HOMA-IR) was calculated using this formula fasting glucose (mmol/L)  $\times$  fasting insulin (mU/L)/22.5.

### 3.6 Anthropometric measures

The following anthropometric measurements were measured: weight, height and waist circumference. Weight and Height were measured using a hospital beam scale with a stadiometer. Waist circumference was measured at the umbilicus by using a flexible tape measure at the end of normal exhale. Body mass index (BMI) was calculated by dividing body weight by height squared.

### 3.7 Clinical Data and Questionnaires

Clinical data were collected directly from participants or their charts which included demographic information, alcohol consumption, the method of birth and breastfeeding as an infant. Supplements and medications list were also recorded. Questionnaires were administered to collect additional information that might influence health. Patients completed an environmental survey, which included country of origin, pets and pro/prebiotic/antibiotic use. was administered to assess current oral health and hygiene practices inclusive of the brushing times, flossing, the number of lost teeth and presence of oral issues.

### 3.8 Statistical Analysis:

Descriptive statistics were calculated for all measurements using means, medians and standard deviations or percentages as appropriate. All statistical analyses were conducted in IBM SPSS version 23. (Will be written after writing preliminary results)



Descriptive statistics were calculated for all measurements using means, medians and standard deviations or percentages as appropriate. A non-parametric test, Spearman's rank correlation, was used to assess the relationship between variables.

### 3.8.1 Sample Size Calculation

The sample size for the main variable oral inflammatory load was calculated based on Bender et al.'s study, which has a similar design [91]. SD of  $1.8 \times 10^6$  and effect size (ES) of  $1 \times 10^6$  were used. A minimum sample size of 29 subjects was required to detect statistical significance at a  $\alpha = 0.05$  significance level, using a two-sided test ( $H_0: \rho = 0$  versus  $H_1: \rho \neq 0$ ) with 80% power ( $1 - \beta = 0.80$ ).

## 4 Results:

### 4.1 Cross-sectional study:

#### 4.1.1 Baseline Characteristics

A total of 46 obese patients were eligible and completed baseline measurements with a mean age of  $47.3 \pm 10$  (SD) and BMI of  $47.8 \pm 7.3$ . Based on Diabetes Canada guidelines, 12 patients had type 2 diabetes. Environmental questionnaires were completed by 44 subjects. The patient population was mostly Caucasian (84%) and predominantly born in Canada (79.1%). Sample demographics, anthropometrics and diagnoses information are presented in table 1 below.

**Table 1:** Sample Demographics, anthropometric and diagnoses data

Variable	n	%
Age group: Young adults (18-35 years)	4	8.7
Middle-aged adults (35-55 years)	32	69.6
Older adults (> 55 years)	10	21.7
Sex: Female	40	87
Male	6	13



Born in Canada	34	79.1
Caucasian	37	84
Insulin resistance	31	83.8
Diabetes	12	26.1
Diabetes medications	9	20
High Oral Inflammatory Load	7	15.9
Low Salivary Flow Rate	23	52.3
Xerostomia	11	28.9

Biomedical and oral health data are shown in table #. The mean OIL of the 30-second oral rinse was  $7.79 \pm 8.70 \times 10^5$  (SD) neutrophils/10 ml. Seven patients had a high oral inflammatory load at the baseline. As for saliva rate, the mean was  $1.2 \pm .9$  ml/min and 23 patients had a salivary flow below 1 ml/min. A dental questionnaire was completed by 44 subjects. Only 11.3% of patients reported being previously diagnosed with periodontal disease in which they were treated at least two years ago. Xerostomia prevalence in this population was 22%. Approximately 50% of the participants had visited dentists in the past 6 months.

**Table 2:** Sample biomedical and oral health data

Variable	n	Mean $\pm$ SD
Weight (kg)	46	131.6 $\pm$ 24.3
BMI (kg/m <sup>2</sup> )	46	47.8 $\pm$ 7.3
Waist Circumference (cm)	33	133.8 $\pm$ 15.2
Glucose (mmol/L)	46	6.2 $\pm$ 2.9
Insulin (pmol/L)	37	135.5 $\pm$ 52.5
HOMA-IR	37	6.2 $\pm$ 4.5
HbA1c (%)	43	5.8 $\pm$ 0.8
C-peptide (pmol/L)	38	1262 $\pm$ 492
OIL (neutrophils count/10 ml)	44	$7.79 \pm 8.70 \times 10^5$
SFR (ml/min)	44	1.2 $\pm$ .9

**Sub-analyses in obese patients with and without high OIL and low SFR:**

Exploratory sub-analyses were conducted in obese patients with and without high OIL and low SFR. The first analysis of patients who had normal and high OIL is presented in table #. The highest percentage of patients with high OIL were over 55 years old (57.1%), followed by middle-aged adults (42.9%) with none in the young adult group. The difference in age groups was near significant with a p value of .053. No significant differences were found between the groups other than the significant difference in OIL. About 43% of subjects with high OIL were diabetic compared to 24.4% in the obese group. A low percentage of patients with high OIL had a low saliva rate. Similar percentages of patients in both groups brushed their teeth twice a day and had visited the dentist in the past 6 months. Xerostomia prevalence was lower in the high OIL group compared to obese patients with normal OIL.

A second analysis compared obese patients who had a normal and low SFR, presented in table #. Age groups and gender did not differ between groups. The only significant difference between the groups, beside the SFR, was glucose. It was significantly higher in patients with a low SFR versus patients with a normal SFR. About 39% of patients with a low SFR were diabetic compared to 14.3% in the obese group. Regarding oral hygiene practices, about half of the patients had acceptable hygiene habits in which they had visited the dentist within the past 6 months and brushed two times a day. Most of the subjects in both groups reported flossing. Bad breath and loss of one or more teeth were higher in patients with a low SFR.

**Table 3:** Sub-analysis in obese patients with and without high oral inflammatory load

Variable	n	Obese patients with high oral inflammatory load n=7	n	Obese patients n=37	P value
Young adults	7	0% (0)	37	10.8% (4)	.053
Middle aged adults	7	42.9% (3)	37	73% (27)	.053
Older adults	7	57.1% (4)	37	16.2% (6)	.053

Gender % female	7	71.9% (5)	37	89.4% (33)	.238
Weight (kg)	7	137.5 ± 21.4	37	130 ± 25.2	.297
BMI (kg/m <sup>2</sup> )	7	46.9 ± 6.7	37	48 ± 7.6	.683
Waist circumference (cm)	6	135.9 ± 7.6	25	133.9 ± 16.9	.510
Glucose	7	6 ± 1	37	6.3 ± 3.2	.312
Insulin	5	156.6 ± 49	30	133.8 ± 53.5	.237
HOMA-IR	5	7.2 ± 1.9	30	6.2 ± 4.9	.116
Insulin resistance (based on HOMA-IR)	5	100% (5)	30	83.3%(25)	.439
HbA1c	5	6.2 ± .76	36	5.8 ± .8	.217
C-peptide	6	1469 ± 293	30	1242 ± 522	.103
T2DM	7	42.9% (3)	37	24.4% (9)	.369
Oral inflammatory load	7	2.33 ± 1.31 x10 <sup>6</sup>	37	4.8 ± 2.36 x10 <sup>5</sup>	< .001***
Saliva flow rate	7	.94 ± 42	35	1.2 ± 1	.766
Low Salivary flow rate	7	18.2% (4)	35	81.8% (18)	1.00
Xerostomia	6	16.7% (1)	31	29 % (9)	1.00
Dental visit in the past 6 months	7	57.1% (4)	34	52.9% (18)	1.00
Brushing at least 2x/day	7	57.1 % (4)	34	50 % (17)	.878
No floss	7	0% (0)	34	29.4% (10)	.164
Bleeding when brushing	7	28.6 % (2)	35	22.9 % (8)	1.00
Inflamed gum	6	33.3 % (2)	35	8.6 % (3)	.148
bad breath	7	14.3% (1)	35	37.1 % (13)	.239
Lost at least 1 tooth	7	57.1 % (4)	35	48.6 % (17)	1.00

Values are expressed as mean ± standard deviation or percentages (count)

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001

**Table 4:** Sub-analysis in obese patients with and without low salivary flow rate

Variable	n	Patients with low salivary rate n=23	n	Obese patients n=21	P value
Young adults	23	8.7% (2)	21	9.5% (2)	.439
Middle aged adults	23	60.9% (14)	21	76.2% (16)	.439
Older adults	23	30.4% (7)	21	14.3% (3)	.439
Gender % female	23	82.6% (19)	21	90.5% (19)	.666
Weight (kg)	23	133 ± 29	21	129.3 ± 18.6	.760
BMI (kg/m <sup>2</sup> )	23	47.5 ± 8.4	21	47.9 ± 6.2	.474
Waist circumference (cm)	19	136.4 ± 17	13	130.3 ± 12.3	.388
Glucose	23	7 ± 3.9	21	5.4 ± 1.2	.021*
Insulin	18	143 ± 50	17	124 ± 51	.181
Homa-IR	18	7.1 ± 5.5	17	5.1 ± 3.2	.113
Insulin resistance (based on HOMA-IR)	18	88.9% (16)	17	76.4% (13)	.496
HbA1c	21	6.1 ± 1	20	5.6 ± .6	1.00
C-peptide	18	1312.6 ± 533	18	1192 ± 470	.496
T2DM	23	39.1% (9)	21	14.3% (3)	.094
Oral inflammatory load	22	8.90 ± 11.4 x10 <sup>5</sup>	20	6.95 ± 4.72 x10 <sup>5</sup>	.678
High oral inflammatory load	22	18.2% (4)	20	15% (3)	1.00
Saliva flow rate	23	.59 ± .2	23	1.8 ± 1	< .001***
Xerostomia	20	20% (4)	17	41.2 % (7)	.279
Dental visit in the past 6 months	22	54.5% (12)	20	60% (12)	.764
Brushing at least 2x/day	22	45.5 % (10)	20	55 % (11)	.611
No floss	22	31.8% (7)	20	15% (3)	.284
Bleeding when brushing	22	22.7 % (5)	21	28.6 % (6)	.736
Inflamed gum	21	9.5% (2)	21	19 % (4)	.663

bad breath	22	40.9% (9)	21	33.3 % (7)	.755
Lost at least 1 tooth	22	59.1% (13)	21	38.1% (8)	.227

Values are expressed as mean  $\pm$  standard deviation or percentages (count)

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001

#### 4.1.2 Differences between obese subjects with and without diabetes

Biomedical, clinical and oral health measures in obese patients with and without diabetes are presented in table #. Glucose, HOMA-IR and HbA1c were significantly higher in patients with diabetes versus patients without. Sex also significantly differed between the groups in which all male subjects were diabetic. Other biomedical and clinical measures were not statistically different except for xerostomia. All reported cases were only in non-diabetic obese patients (p value=.016). Although OIL and SFR did not show significance, patients with diabetes tended to have a lower mean of saliva rates and a higher OIL mean. A correlation of oral hygiene practices was tested between the groups. Patients with diabetes showed a higher percentage of bad breath, no flossing and not brushing every day compared to non-diabetic obese with no statistically significant difference. Both groups had similar percentages of bleeding gums and losing one or more teeth.

**Table 5: Biomedical, clinical and oral health measure in obese with and without diabetes**

Variable	n	Diabetic patients	n	Non-diabetic patients	P value
Age (years)	12	49.5 $\pm$ 9.3	34	46.5 $\pm$ 10.2	.278
Age group: Young adults	12	0% (0)	34	11.8% (4)	.097
Middle aged adults	12	58.7% (7)	34	73.5% (25)	.097
Older adults	12	41.7% (5)	34	14.7 (5)	.097
Sex (% female)	12	50% (6)	34	100% (34)	< .001 ***
Weight (kg)	12	136 $\pm$ 29	34	130 $\pm$ 23	.612
BMI (kg/m <sup>2</sup> )	12	45.8 $\pm$ 3.3	34	48.8 $\pm$ 8.4	.558
Waist circumference (cm)	8	139.6 $\pm$ 19	25	132.5 $\pm$ 14	.237
Glucose (mmol/L)	12	9 $\pm$ 4.8	34	5.3 $\pm$ 0.7	< .001 ***
Insulin (pmol/L)	9	165 $\pm$ 59.5	28	127 $\pm$ 48	.073

HOMA-IR	9	10.2 ± 7.2	28	5 ± 2.2	.008 **
HbA1c (%)	11	7 ± 0.8	32	5.5 ± 0.4	< .001 ***
C-peptide (pmol/L)	9	1473± 716	29	1215 ± 394	.368
OIL (neutrophils count/10 ml)	12	9.58 ± 11.8 x10 <sup>5</sup>	32	7.12 ± 7.33 x10 <sup>5</sup>	.668
High OIL	12	25% (3)	32	12.5% (4)	.369
SFR (ml/min)	12	1 ± 0.8	32	1.3 ± 1	.170
Low SFR	12	75% (9)	32	43.8% (14)	.094
Xerostomia	11	0% (0)	27	40.7%	.016*
Not brushing everyday	12	8.3 % (1)	31	6.5 % (2)	.124
Not flossing	12	33.3 % (4)	31	19.4 % (6)	.427
Bleeding gum	12	25 % (3)	32	25 % (8)	1.00
Bad breath	12	50 % (6)	32	31.3 % (10)	.303
Lost ≥ 1 teeth	12	50 % (6)	32	50 % (16)	1.00

Values are expressed as: mean ± standard deviation or percentages (count)

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001

#### 4.1.3 Relationships between oral measurements and HbA1c, insulin resistance and BMI

Analyses were conducted to assess possible relationships between oral measurements (OIL, SFR), HbA1c and BMI. Also, the potential influence of diet on oral health were investigated. As shown in table #, OIL did not correlate with HbA1c, BMI or insulin resistance at the baseline. The SFR showed a weak negative correlation only with HbA1c ( $r = -0.343$ ,  $p$  value = .032) (figure 1). This significance was not found when the correlation was done pairwise. Dietary carbohydrate, fat, omega-6, omega-3, fiber, vitamin C and D did not correlate with OIL and the SFR.

**Table 6 :** Correlation between OIL, SFR and BMI and HbA1c

Oral health measurements	BMI	HbA1c
Oral inflammatory load	$r_s = -.222$ P= .174	$r_s = -.017$ P= .916
Salivary flow rate	$r_s = -.205$ P= .211	$r_s = -.343$ P= .032*

Listwise N = 39

Values with superscript are significantly different.

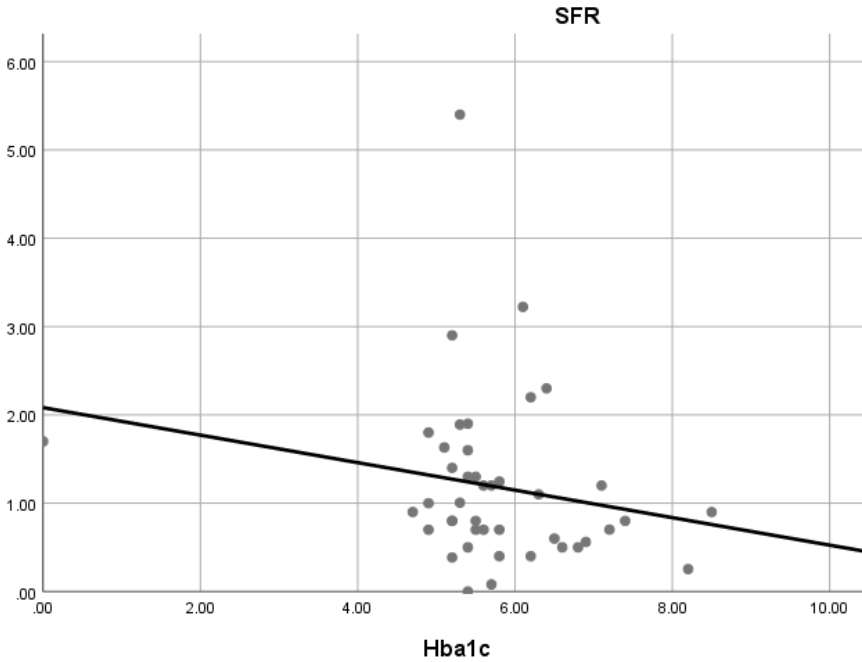
Analyses were conducted to assess relationships between Body Mass Index (BMI) and the (Age in Years), the potential relationships is negative relation equal -0.429 but its significant relation at 0.01 level .the negative sign in relation means that when age is great then BMI is Skinny , And when the age decreases, the weight increases were investigated. As shown in table 7,

**Table7:** Correlation between BMI, Age in year

Correlations			
		BMI	Age in Years
BMI	Pearson Correlation	1	-.429**
	Sig. (2-tailed)		.003
	N	46	46
Age in Years	Pearson Correlation	-.429**	1
	Sig. (2-tailed)	.003	
	N	46	46

\*\* . Correlation is significant at the 0.01 level (2-tailed).

**Figure1:** HbA1c and Saliva flow rate correlation



**Table8:** Correlation between dietary intake OIL and SFR at baseline.

Dietary components	Oral inflammatory load (n=38)	Salivary flow rate (n=40)
Carbohydrates	$r_s = .201$ $P = .233$	$r_s = .013$ $P = .936$
Sugar	$r_s = -.073$ $P = .668$	$r_s = .028$ $P = .865$
Sucrose	$r_s = -.045$ $P = .792$	$r_s = -.054$ $P = .740$
Fat	$r_s = -.004$ $P = .983$	$r_s = -.124$ $P = .446$
Omega-6	$r_s = .328$ $P = .071$	$r_s = -.101$ $P = .577$
Omega-3	$r_s = .218$ $P = .239$	$r_s = -.276$ $P = .119$
Fiber	$r_s = -.270$ $P = .106$	$r_s = .029$ $P = .860$



Vitamin C	$r_s=.147$ P= .385	$r_s= .065$ P= .688
Vitamin D	$r_s= .314$ P= .066	$r_s=-.264$ P= .110

### 3.1.1 Anthropometric measurements and Glucose metabolism

Changes in anthropometric and biomedical measurements were assessed after a VLCD, 1 month after surgery and after the whole bariatric care protocol (VLCD and BSx). Changes after a VLCD, prior to surgery, are presented in table #. The mean VLCD duration was  $16 \pm 3$  days. A VLCD significantly reduced weight and BMI. It also significantly improved glucose metabolism including glucose, insulin, HOMA-IR and c-peptide. Changes of these measurements at 1 month after surgery are presented in table 9. Similarly, bariatric surgery significantly reduced weight and BMI. However, it did not show a significant effect on glucose, insulin, HOMA-IR and c-peptide expect HbA1c which had a statistically significant difference (p value= .013). As for the whole protocol, shown in table 9, all measurements were statistically significant.

**Table 9:** Anthropometrics and clinical data post-VLCD

Value	n	Baseline	Post-VLCD / BSx	P value
Weight (kg)	38	$126.6 \pm 17.4$	$121.5 \pm 16.3$	< .001***
BMI (kg/m <sup>2</sup> ) n= 36	36	$46.6 \pm 5.8$	$44.6 \pm 5.6$	< .001 ***
Glucose (mmol/L) n=34	34	$6.3 \pm 3.2$	$5.3 \pm 1.5$	.001**
Insulin (pmol/L) n=33	33	$127 \pm 37$	$64.5 \pm 44$	< .001***
HOMA-IR n=33	33	$5.7 \pm 3$	$2.7 \pm 3$	< .001***
HbA1c (%) n=34	34	$5.8 \pm 0.7$	$5.6 \pm 0.8$	< .001***
C-peptide (pmol/L) n=32	32	$1246 \pm 416$	$1163 \pm 1569$	< .001***

Values are expressed as: mean  $\pm$  standard deviation

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001

**Table 10:** Anthropometrics and clinical data 1 month post-surgery

Value	n	Post-VLCD / BSx	1-month post-BSx	P value
Weight (kg)	34	121.5 ± 16.2	112.9 ± 41	< .001***
BMI (kg/m <sup>2</sup> )	31	44.6 ± 5.6	40.9 ± 5	< .001***
Glucose (mmol/L)	30	5.3 ± 1.5	4.9 ± 0.7	.533
Insulin (pmol/L)	28	64.5 ± 44	84.9 ± 61	.631
HOMA-IR	28	2.7 ± 3	3.1 ± 2.3	.614
HbA1C (%)	30	5.6 ± 0.8	5.4 ± 0.5	.013
C-peptide (pmol/L)	28	1163 ± 1569	883 ± 458	.929

Values are expressed as: mean ± standard deviation

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001

**Table 11:** Anthropometrics and clinical data post bariatric care protocol

Variable	n	Baseline	Post-VLCD	Post-surgery	P value
Weight (kg)	31	127.7 ± 17.7	122.3 ± 16.5	112.3 ± 14.5	< .001***
BMI (kg/m <sup>2</sup> )	30	46.6 ± 5.8	44.6 ± 5.6	40.9 ± 5	< .001***
Waist circumference (cm)	9	134 ± 15	-	119.6 ± 15.7	.008**
Glucose (mmol/L)	29	6.3 ± 3.2	5.3 ± 1.5	4.9 ± 0.7	< .001***
Insulin (pmol/L)	22	127 ± 37	64.5 ± 44	84.9 ± 61	< .001***
HOMA-IR	22	5.7 ± 3	2.7 ± 3	3.1 ± 2.3	< .001***
HbA1C (%)	25	5.8 ± 0.7	5.6 ± 0.8	5.4 ± 0.5	< .001***
C-peptide (pmol/L)	21	1246 ± 416	1163 ± 1569	883 ± 458	< .001***

Values are expressed as: mean ± standard deviation

\*= P<0.05 \*\*= P<0.01 \*\*\*= P<0.001



## Conclusion

This project provides a new perspective on the care of bariatric patients that has not been well assessed. Our study is the first to evaluate the effects of VLCD with Optifast, BSx and the overall bariatric care protocol on oral health in obese patients. We did not detect a significant effect regarding the two oral health parameters measured, OIL and SFR. However, this study contributed further to the body of the literature by using OIL as a less invasive method of screening for PD in obese patient population, as well as by using SFR to identify those with low SFR who may be at risk of developing PD post-BSx. A longer follow-up period may be required to detect the effects on these conditions. We plan to re-assess these oral parameters at 6 months post-surgery to determine this. A multidisciplinary team may find it worthwhile to incorporate oral health assessments when evaluating patients to minimize the risk of PD afterwards.

The conclusions of the study can be summarized as follows:

- Based on OIL, the prevalence of PD in our patient population is similar to that reported in the literature, 15.2% versus 17.65%.
- This study did not find significant differences in OIL or SFR between diabetic and nondiabetic obese subjects but the findings suggest an inverse relationship between SFR and HbA1c.
- Our study suggests that VLCD with Optifast<sup>®</sup> improves metabolic parameters and weight, but there was no significant impact on oral measurements.
- Bariatric surgery is effective in improving glycemic control and inducing weight loss, with no significant impact on oral measurements.
- Other factors may have influenced oral measurements, such as diet pattern or other surgical side effects, namely potential increases in reflux and/or vomiting.
- Patients with hyposalivation may be at risk of developing PD after bariatric surgery.



## References

1. WHO / *Obesity and overweight*, in WHO. 2017, World Health Organization.
2. Ryan, M.E. and V. Raja, *Diet, Obesity, Diabetes, and Periodontitis: a Syndemic Approach to Management*. Current Oral Health Reports, 2016. **3**(1): p. 14-27.
3. Nieuwdorp, M., et al., *Role of the microbiome in energy regulation and metabolism*. Gastroenterology, 2014. **146**(6): p. 1525-33.
4. Sjostrom, L., et al., *Association of bariatric surgery with long-term remission of type 2 diabetes and with microvascular and macrovascular complications*. JAMA, 2014. **311**(22): p. 2297-304.
5. Chaffee, B.W. and S.J. Weston, *Association between chronic periodontal disease and obesity: a systematic review and meta-analysis*. Journal of periodontology, 2010. **81**(12): p. 1708-1724.
6. Ye, J., *Mechanisms of insulin resistance in obesity*. Front Med, 2013. **7**(1): p. 14-24.
7. Atif B. Awad, P.G.B., *Adipose Tissue and Inflammation*. 2009: CRC Press.
8. Van der Velden, U., D. Kuzmanova, and I.L. Chapple, *Micronutritional approaches to periodontal therapy*. J Clin Periodontol, 2011. **38 Suppl 11**: p. 142-58.
9. Weisberg, S.P., et al., *Obesity is associated with macrophage accumulation in adipose tissue*. J Clin Invest, 2003. **112**(12): p. 1796-808.
10. Canadian Diabetes Association Clinical Practice Guidelines Expert, C., R. Goldenberg, and Z. Punthakee, *Definition, classification and diagnosis of diabetes, prediabetes and metabolic syndrome*. Can J Diabetes, 2013. **37 Suppl 1**: p. S8-11.
11. *Diabetes in Canada*. 2016.
12. *Diabetes in Ontario*. 2016, Canadian Diabetes Association.
13. Kumanyika, S., et al., *Obesity prevention: the case for action*. Int J Obes Relat Metab Disord, 2002. **26**(3): p. 425-36.
14. Bank, J., *Diabetes in Canada: Facts and figures from a public health perspective*.
15. Hotamisligil, G.S., et al., *Increased adipose tissue expression of tumor necrosis factor- $\alpha$  in human obesity and insulin resistance*. J Clin Invest, 1995. **95**(5): p. 2409-15.
16. P., D., et al., *Tumor Necrosis Factor- $\alpha$  in Sera of Obese Patients- Fall with Weight Loss*. Journal of Clinical Endocrinology and Metabolism, 1998. **83**.
17. Tsai, C., C. Hayes, and G.W. Taylor, *Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population*. Community Dent Oral Epidemiol, 2002. **30**(3): p. 182-92.
18. Pihlstrom, B.L., B.S. Michalowicz, and N.W. Johnson, *Periodontal diseases*. The Lancet, 2005. **366**(9499): p. 1809-1820.
19. Nibali, L., et al., *Tooth loss in aggressive periodontitis: a systematic review*. J Dent Res, 2013. **92**(10): p. 868-75.



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20. Armitage, G.C., *Periodontal diagnoses and classification of periodontal diseases*. Periodontology 2000, 2004. **34**(1): p. 9-21.
21. Eke, P.I., et al., *Prevalence of periodontitis in adults in the United States: 2009 and 2010*. J Dent Res, 2012. **91**(10): p. 914-20.
22. Genco, R.J. and W.S. Borgnakke, *Risk factors for periodontal disease*. Periodontology 2000, 2013. **62**(1): p. 59-94.