

THE EFFECT OF ONE ANASTOMOSIS GASTRIC BYPASS ON SERUM

FATTY ACID PROFILE IN PATIENTS WITH MORBID OBESITY

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BACKGROUND Bariatric surgery can result in an improvement of patient's metabolic status e.g. remission of type 2 diabetes (T2M), dyslipidaemia or hypertension. Recently, we showed that one anastomosis gastric bypass (OAGB) resulted in improvement of serum amino acid profiles in patients with morbid obesity [1]. **Here we aim to establish the OAGB's effect on serum fatty acids (FAs).**

RESULTS ♦ Patients with morbid obesity after the OAGB had significant decrease in BMI (Table 1).

♦ There was an improvement in the serum levels of triglycerides, total, LDL-cholesterol and glucose (Table 1).

♦ PCA plot based on the whole FA profile revealed grouping of pre- and post-OAGB patients and separation of the control group (Figure 1).

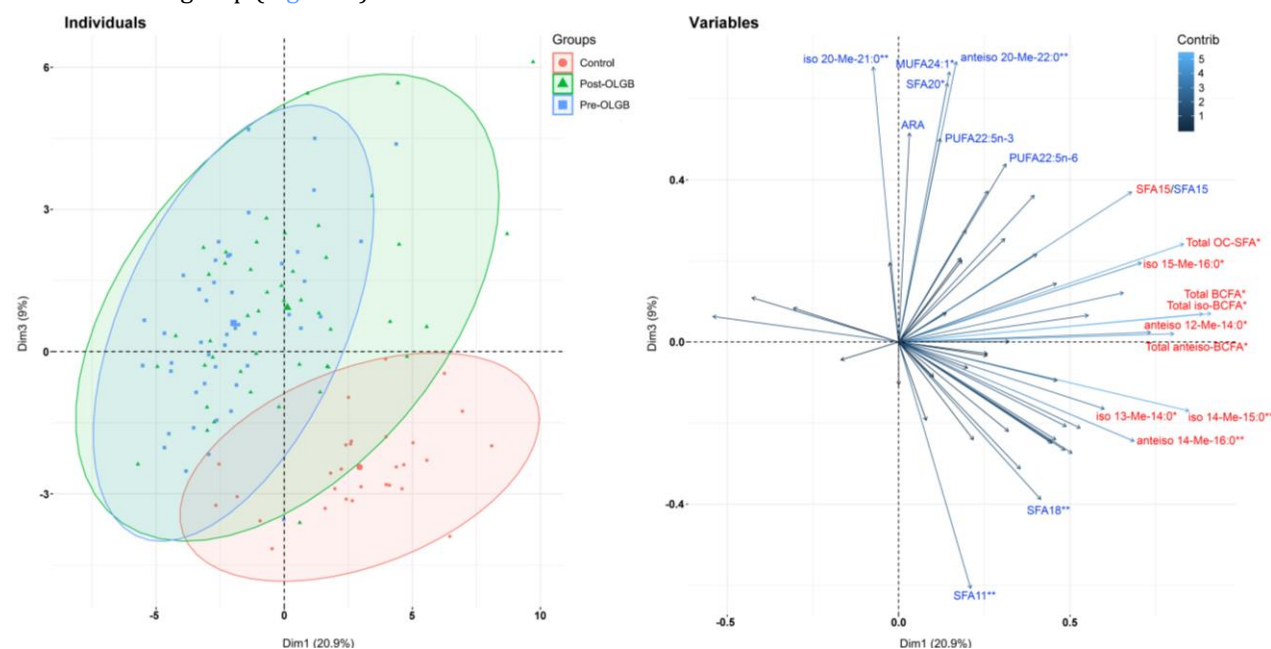


Figure 1. PCA results of the FA profiles in study subjects. Score plot (left) and variables plot (right) for the first and third PCs. * - variables that differ significantly between one control-OAGB pair (control and pre-OAGB or control and post-OAGB), ** - variables that differ between control and both pre- and post-OAGB group, p-value was set as significant at p < 0.01. Red - FAs with the most significant contribution into PC1, blue - FAs with the most significant contribution into PC3.

MATERIALS & METHODS

Patients: 46 patients, 23 with and 23 without T2M. Fasting blood serum was collected before surgery, and 6-9 months post-surgery.

Control group: 29 age-matched lean individuals without metabolic disorders.

FAs were analysed with GC-MS [2].

Data analysis was performed using SigmaPlot 11 (Systat Software, Inc, 2008), computing environment R (R Core Team, 2019).

Table 1. Metabolic characteristics of study subjects.

	Control	Pre-OAGB	Post-OAGB
BMI (kg/m²)	24.9 ± 2.57	38.5 ± 4.31 ^a	29.6 ± 3.85 ^{b,c}
TG (mg/dL)	109 ± 47.7	113 ± 37.3	87.8 ± 26.7 ^b
HDL (mg/dL)	55.3 ± 13.2	50.1 ± 9.33	50.9 ± 11.7
LDL (mg/dL)	128 ± 41.7	114 ± 33.8	88.3 ± 25.3 ^{b,c}
TC (mg/dL)	208 ± 44.5	201 ± 40.9	180 ± 49.9 ^{b,c}
CRP (mg/L)	1.57 ± 1.22	1.65 ± 0.53	1.02 ± 0.55 ^b
Glucose (mg/dL)	93.1 ± 9.36	111 ± 32.1 ^a	91.5 ± 11.2 ^b
Insulin (μU/mL)	9.11 ± 3.97	14.9 ± 7.85 ^a	7.72 ± 6.49 ^b
HOMA-IR	2.13 ± 1.02	4.37 ± 3.04 ^a	2.04 ± 1.96 ^b

p < 0.05: a - control vs pre-OAGB, b - pre- vs post-OAGB, c - control vs post-OAGB

Table 2. FA main groups composition [%] in serum.

	Control	Pre-OAGB	Post-OAGB
Even-chain FAs (ECFA)	32.0 ± 0.350	32.5 ± 0.261	32.6 ± 0.292
Odd-chain FAs (OCFA)	0.644 ± 0.020	0.577 ± 0.013 ^a	0.606 ± 0.019
Saturated FAs (SFA)	33.1 ± 0.343	33.4 ± 0.264	33.6 ± 0.302
Branched-chain FAs (BCFA)	0.422 ± 0.017	0.295 ± 0.011 ^a	0.396 ± 0.017 ^b
Monounsaturated FAs (MUFA)	29.9 ± 0.647	33.4 ± 0.392 ^a	34.3 ± 0.462 ^c
Polyunsaturated FAs n-3 (PUFA n-3)	2.97 ± 0.238	2.49 ± 0.102	2.30 ± 0.079 ^{b,c}
Polyunsaturated FAs n-6 (PUFA n-6)	33.9 ± 0.686	30.7 ± 0.539 ^a	29.8 ± 0.612 ^{b,c}

p < 0.05: a - control vs pre-OAGB, b - pre- vs post-OAGB, c - control vs post-OAGB

♦ Patients with obesity had lowered total OCFA, BCFA, both iso- and anteiso- and PUFA n-6 content.

♦ Post-OAGB we observed improvement of OCFA and BCFA content.

CONCLUSIONS

1. Following OAGB there was an improvement of glucose homeostatis and lipoprotein profile in serum of treated patients.
2. Despite the differences in FA content post-OAGB (Table 2), the bariatric treatment did not result in the complete normalization of FA profiles in serum of treated patients (Figure 1).
3. Among beneficial changes observed post-OAGB is a slight elevation of OCFA and BCFA levels, which exhibit antitumor effects and inverse correlations with inflammation, dyslipidaemia, and insulin resistance [3].
4. In the follow up MUFAs were elevated and PUFAs were decreased. These FAs are consumed with diet, respectively mainly plant oils and seafood.
5. **Considering the beneficial effects of PUFAs, OCFAs and BCFAs (cardioprotective, antitumour, anti-inflammatory [4]) the use of new dietary guidelines to supplement these FAs may be recommended.**

[1] Halinski, L. P. et al. *J. Clin. Med.* **9**, 100 (2019)

[2] Pakiet, A. et al. *Nutr. Metab.* **16**, 81 (2019)

[3] Mika, A. et al. *Obes. Rev.* **18**, 247-272 (2017)

[4] Mika, A. et al. *Nutrients* **12**, 187 (2020)



Grant number:
NCN 2016/21/D/NZ5/00219.

