Association of β-cell function with periodontal pocketing and alveolar bone loss

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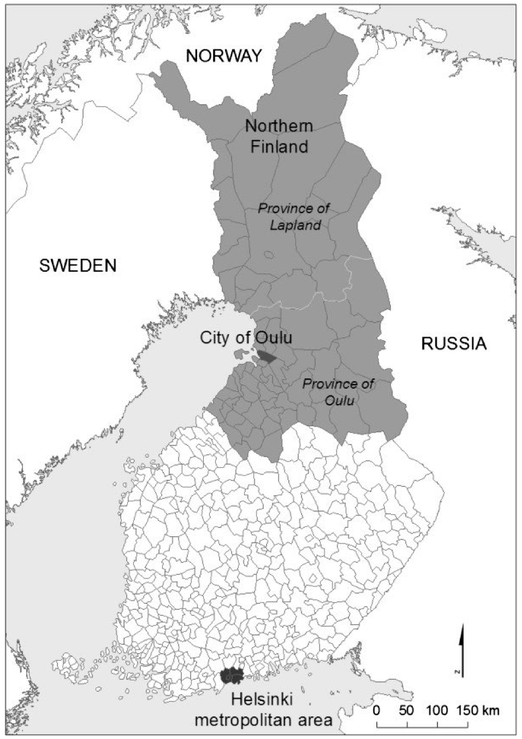

Figure 1. Surveyed areas of Northern Finland Birth Cohort 1966 study as shaded. (Näyhä et al. 2013, BMC Public Health; 8:13:938).

Background and aim

Insulin resistance occurs when insulin action in the liver, adipose tissue, and skeletal muscle is insufficient. It induces increased gluconeogenesis in the liver, decreased glucose disposal in the muscle, and increased release of free fatty acids from the adipose tissue.

Insufficient insulin action leads to an increased compensatory insulin production. Hyperglycaemia exists when compensatory mechanisms of hyperinsulinemia fail.

Diabetic subjects with poor metabolic control, i.e., with high blood glucose, are at higher risk for periodontitis than those with good metabolic control. The prevailing hypothesis emphasizes the role of a hyper-inflammatory state as the main cause for periodontal tissue destruction in diabetes patients. Currently, the role of insulin resistance and associated disturbed beta cell function in this process is unclear.

The aim of this study was to investigate whether beta cell function (HOMA-2B) is associated with periodontal tissue destruction, measured as periodontal pocketing and alveolar bone level.

Materials and methods

The study is based on the follow-up study of The Northern Finland Birth Cohort 1966. In 2012–2013, when the subjects were 46–47 years old, an oral health examination, including periodontal examination, was conducted for the first time for 1,964 persons living in the city of Oulu or within a 100 km radius from Oulu.

Subjects with diabetes mellitus (DM 1 and 2), rheumatic and inflammatory intestinal and lung diseases were excluded from the study. Different sub-populations were formed according to the smoking status and body mass index (Table 1). The study population was categorized based on HOMA-2B levels at ages 31 and 46 (under the median = high level; over the median = low level).

Periodontal pocketing was measured as the number of sites with ≥ 4 mm of periodontal pocket depth (PD), and alveolar bone loss as the number of sites with bone level ≥ 5 mm (BL). Relative risks (RRs) and 95% confidence intervals (CI) were estimated using Poisson regression models.

Results

Low beta cell levels between ages 31 (HOMA-2B: 0–96.30) and 46 (HOMA-2B:0–76.50) were associated with PD ≥ 4 mm among never smokers. In those never smokers whose BMI was under 30, the association was stronger (RR 1.6). The association with BL ≥ 5 mm was found only among never smokers with BMI < 30 (Table 1).

Table 1. Association HOMA-2B with the numbers of sites with ≥ 4 mm deep periodontal pockets (PD) and ≥ 5 mm alveolar bone level (BL) in never-smokers and never-smokers with BMI < 30 using Poisson regression models [relative risk (RR) with 95 % confidence intervals (95 % CI)]

	Never-smokers		*Never-smokers BMI < 30*
	Unadjusted RR (95% CI) *n* = 647	Adjusted RR[1] (95% CI) *n* = 647	Adjusted RR[2] (95% CI) *n* = 532
HOMA-2B between ages 31 and 46 years			
Number of sites with PD ≥ 4 mm			
high and high (reference)	1	1	1
high and low	0.6 (0.5–0.8)	0.7 (0.5–0.8)	0.7 (0.6–0.9)
low and high	1.1 (0.9–1.3)	1.1 (0.9–1.3)	1.1 (0.9–1.4)
low and low	1.4 (1.2–1.6)	1.4 (1.2–1.7)	1.6 (1.4–2.0)
Number of sites with BL ≥ 5 mm			
high and high (reference)	1	1	1
high and low	1.0 (0.7–1.2)	0.9 (0.7–1.2)	1.0 (0.8–1.4)
low and high	1.3 (1.0–1.7)	1.2 (1.0–1.6)	1.6 (1.2–2.1)
low and low	1.2 (1.0–1.5)	1.2 (0.9–1.5)	1.4 (1.0–1.8)

HOMA-2B, BMI, body mass index
[1] Adjusted for gender, BMI, education, number of teeth with plaque and number of tooth sites (offset)
[2] Adjusted for gender, education, number of teeth with plaque and number of tooth sites (offset).

Conclusions

Recently reported studies using these data showed that long-term prediabetes, long-term obesity, and weight gain are associated with periodontal pocketing and alveolar bone loss (Tegelberg et al. 2021, 2023). Results of the present study suggest that the altered beta cell function is associated with the process leading to periodontal destruction, periodontal pocketing and alveolar bone loss.

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