Periodontal Disease: The Influence of Metabolic Syndrome

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ABSTRACT

Nearly 50% of people over the age of 29 in the U.S. develop periodontitis, commonly known as gum disease. This percentage increases by 20% for those over 65. Periodontitis causes more than just dental damage; *Porphyromonas gingivalis* (Pg), a periodontopathic bacteria produced at the onset of infection undergoes an inflammatory pathway. Moreover, local inflammation caused by Pg results in systemic inflammation and impaired immune response, suggesting that periodontitis plays a role in the development of metabolic syndrome. Systemic inflammation and fat metabolism are directly related in effect with this immune response, however our understanding of the relationship between periodontitis and the development of metabolic syndrome is unclear. My aim was to investigate the hypothesis that Pg has a direct effect by triggering insulin resistance and decreasing glucose uptake alongside altering healthy gut microbiota which causes an increase in obesity, a risk factor for metabolic syndrome. To do this, I reviewed several scientific journal articles that conducted cohort research analysis on mice infected with Pg and human participants with preexisting periodontitis. Methods used involved measurement of inflammatory cytokine levels, adipose tissue production, and insulin and glucose levels. Major results supported that gut bacteria alteration elicited by Pg causes gut inflammation via the release of pro-inflammatory cytokines. The mice showed increased insulin resistance, which resulted in fat storage in white and brown adipose tissues, suggesting that Pg causes obesity, a significant precursor for metabolic syndrome. Moreover, my review of these articles emphasizes the importance of oral hygiene to avoid downstream health problems.

METHODS

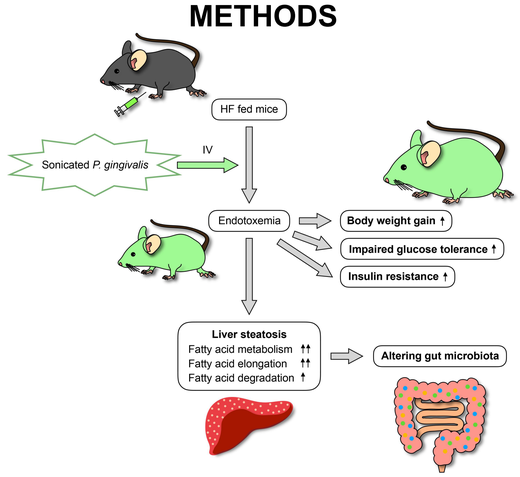
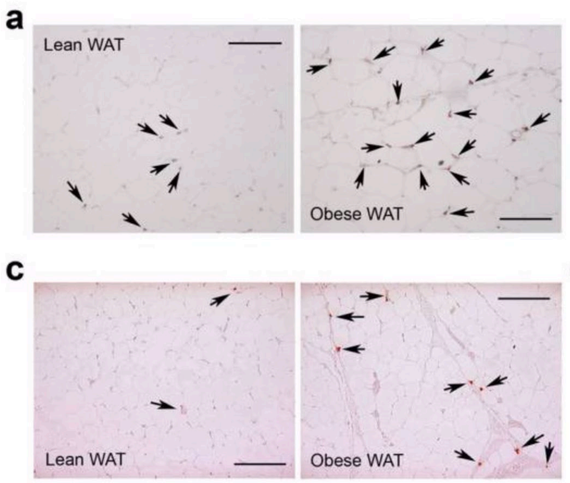


Figure 1. Mechanism of *P. Gingivalis* infection in mice induces pro-inflammatory cytokines causing glucose and lipid metabolism impairment and altered gut microbiota. Blaco-Baque et al. (2017) showed mass macrophage and Th1 cell production in HF fed mice. Winer et al. (2011) found increased Th1 cell count in obese mice causing the release of macrophages into phenotypic M1. Lumeng et al. (2007) observed more M1 markers in obese mice promoting fat storage and adipocyte apoptosis. Upregulation of tumor necrosis factor (TNF) in adipose tissue by macrophage and Th1 cells was found by Thastasa et al. (2021) to alter triglyceride turnover causing liver steatosis. Together these studies showed impairment of metabolic function in adipose tissue by Pg.

Cell Compositions in White Adipose Tissue

Mast Cell Immunostaining



Macrophages & Mast Cells

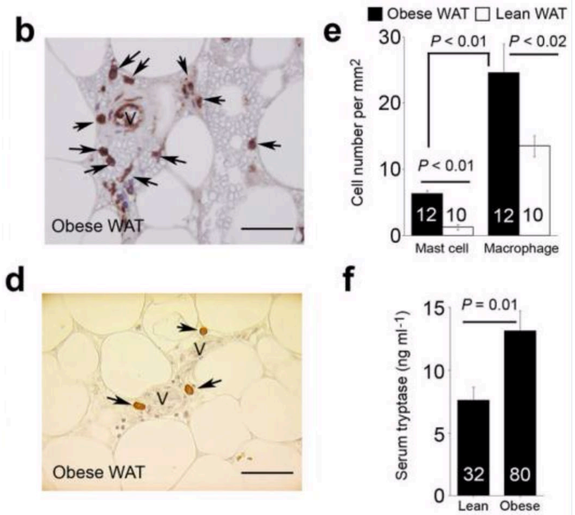
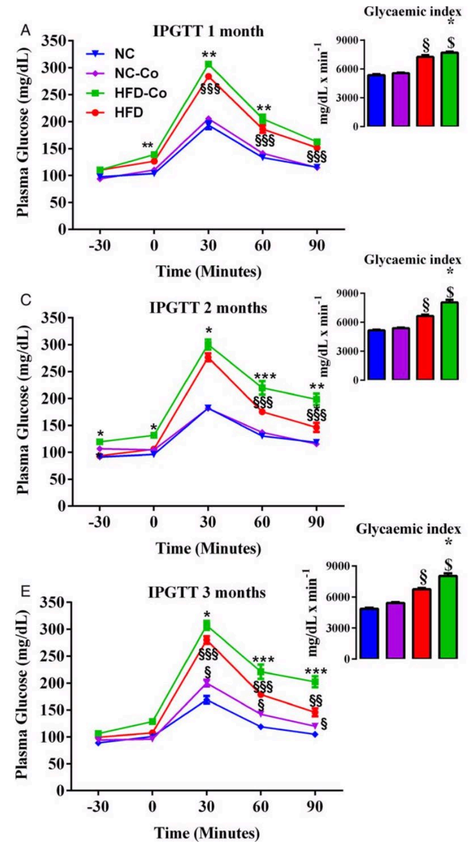
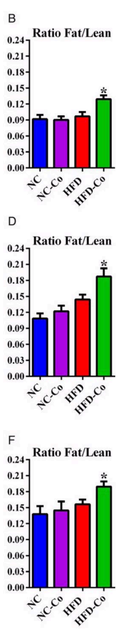


Figure 2. Lui et al. (2009) examined macrophage and mast cell composition in White Adipose Tissue (WAT). **a.** HAM56 Macrophage and **c.** Tryptase mast cell immunostaining in lean and obese WAT. **b.** HAM56 Macrophages and **d.** Tryptase mast cells in obese WAT. **e.** increased macrophage and mast cell concentration in obese mice and **f.** higher serum tryptase.

High-Fat Diet Enhanced by Periodontitis





Normal chow | Normal chow colonized | HF diet | HF diet colonized

Figure 3. Monitored glucose tolerance (mg/dL) in mice **A,B.** displays glycemic index and ratio fat/lean for each group of mice at 1 month and **C,D.** at 2 months and **E,F.** at 3 months. Blaco-Baque et al. (2017) concluded that on the onset of periodontitis infection, HF fed mice became significantly glucose intolerant.

Gene Expression in Brown Adipose Tissue

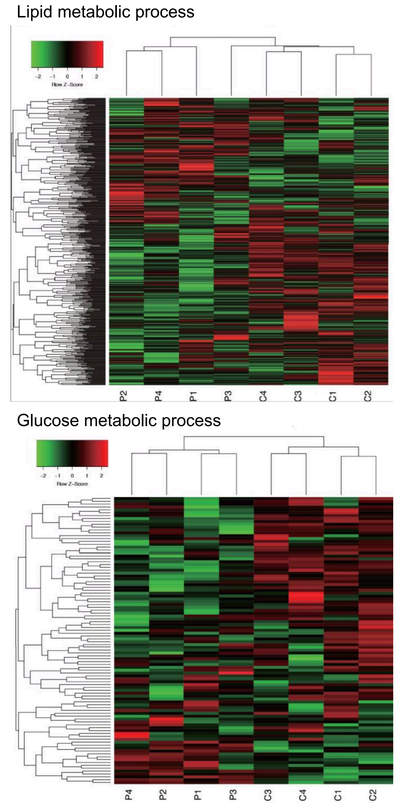


Figure 4. Nakajima et al. (2016) examined the downregulation of genes responsible for insulin sensitivity using a heatmap.

CONCLUSIONS

- A pattern of the upregulation of pro-inflammatory cytokines impaired insulin signaling pathways.
- Pg resulted in the upregulation of genes responsible for lipid synthesis.
- Pg caused fatty liver disease and obesity, significant precursors of metabolic syndrome.
- Practicing good oral hygiene is key to avoiding serious health implications.
- With today's medical technology, anti-inflammatory agents could decrease insulin resistance.

REFERENCES