



GroEL Reactive T Cells In Atherosclerosis And Chronic Periodontitis

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Introduction

The relationship between chronic periodontitis and atherosclerosis is now well established. Chronic infections together with systemic inflammation are both accepted as risk factors for the development of atherosclerosis (Muhlestein, 2000).

Biological mechanisms that may explain the link between chronic periodontitis and atherosclerosis have been proposed (reviewed in Seymour et al. 2007). The finding of T cells cross-reacting with both oral bacteria and heat shock protein (HSP) 60 positive endothelial cells has supported the concept of cross-reactivity or molecular mimicry as one of the hypotheses explaining this association (Ford et al 2005).

Objective:

The objective of the present study was to determine the profile of cytokine gene expression of *P. gingivalis* GroEL reactive T cells in the peripheral blood of patients with atherosclerosis (A) and those with chronic periodontitis (P) in order to gain some insight into the functional activity of these cells in atherosclerosis and chronic periodontitis.

Materials and Methods

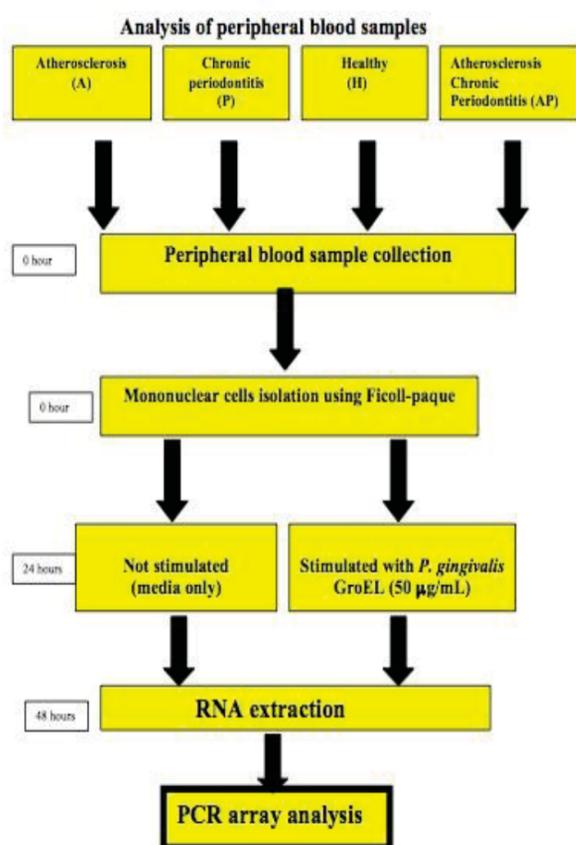


Figure 1: Outline of the study

Results

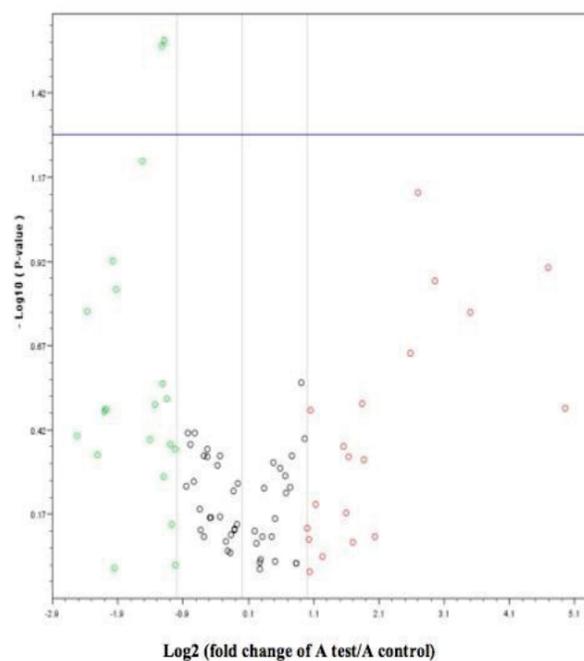


Figure 2: Volcano plot of differential expression of cytokine genes in the atherosclerosis group (A) following stimulation with *P. gingivalis* GroEL

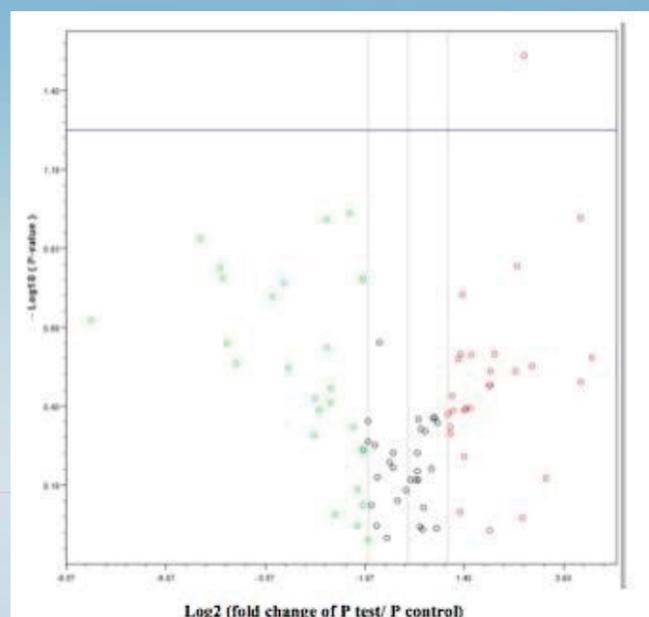


Figure 3: Volcano plot of differential expression of cytokine genes in the chronic periodontitis (P) group following stimulation with *P. gingivalis* GroEL

Gene Symbol	Atherosclerosis (Fold Regulation)	Chronic periodontitis (Fold regulation)
BMP1	2.007	2.091
BMP5	5.986	2.678
CSF2	3.245	24.672
IFNA4	25.963	8.743
IFNA5	2.938	6.504
IL17C	2.359	4.174
IL1A	2.191	2.666
IL1B	4.098	20.086
TGFB2	7.754	2.972

Table 1: Up-regulated genes in atherosclerosis (A) and chronic periodontitis (P) groups

Gene Symbol	Atherosclerosis (Fold Regulation)	Chronic periodontitis (Fold regulation)
BMP6	-4.331	-2.166
BMP8B	-3.814	-2.191
IFNA1	-5.175	-3.510
IFNG	-5.755	-10.496
IFNK	-4.252	-23.347
IL13	-4.621	-25.785
IL3	-2.310	-2.191
IL5	-2.145	-7.936
IL18	-2.034	-2.376

Table 2: Down-regulated genes in atherosclerosis (A) and chronic periodontitis (P) groups

- 39 genes were differentially expressed in the atherosclerosis group, of which 19 were up regulated and 20 down regulated (Figure 2)
- 55 genes were differentially expressed in the periodontitis group, of which 28 were up regulated and 27 down regulated (Figure 3)
- 9 genes were up regulated and a further 9 genes were down regulated in both groups (Tables 1 & 2)

The present study has shown the presence of *P. gingivalis* GroEL specific T cells in the peripheral blood of patients with atherosclerosis but without chronic periodontitis and in the peripheral blood of patients with chronic periodontitis but without atherosclerosis. Further, despite some degree of differential cytokine gene expression, there was a degree of similarity in the gene expression profiles of these groups. This degree of similarity lends support to the concept of molecular mimicry contributing to the mechanism underpinning the association between these two chronic diseases. At the same time, the degree of differential gene expression raises the possibility of epitopic variation with peripheral blood T cells from each group recognising a range of GroEL epitopes.

References

- Ford, P., Gemmell, E., Walker, P., West, M., Cullinan, M. & Seymour, G. (2005) Characterization of heat shock protein-specific T cells in atherosclerosis. *Clinical and Diagnostic Laboratory Immunology* 12, 259-267
- Muhlestein, J. B. (2000) Chronic infection and coronary artery disease. *Medical Clinics of North America* 84, 123-148.
- SEYMOUR GJ, FORD PJ, CULLINAN MP, LEISHMAN S, YAMAZAKI K. Relationship between periodontal infections and systemic disease. *Clinical Microbiology and Infection* 2007; 13: 3-10.