題名:Porphyromonas gingivalis fimbriae-dependent interleukin-6 autocrine regulation by increase of Gp130 in endothelial cells

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摘要:BACKGROUND AND OBJECTIVE: Local persistent infection by Porphyromonas gingivalis leads to inflammatory systemic diseases, such as atherosclerosis. We have reported previously that avirulent P. gingivalis fimbriae-dependent invasion into endothelial cells might be involved in progression of atherosclerosis. Although interleukin-6 (IL-6) regulates progression of atherosclerosis, little is known about the relationship of P. gingivalis fimbriae-dependent invasion to IL-6 regulation in endothelial cells. MATERIAL AND METHODS: We examined the secretion of IL-6 and the expression of the IL-6 signal transducer gp130 in human umbilical vein endothelial cells (HUVEC) infected with the wild-type FDC381 strain of P. gingivalisand a fimbriae-deficient mutant (fimA) by enzyme-linked immunosorbent assay, quantitative reverse transcriptase-polymerase chain reaction (RT-PCR) and flow cytometry (fluorescence-activated cell sorting, FACS) analysis. RESULTS: Coculture of HUVEC with P. gingivalis resulted in increase of IL-6 secretion at 24 h postinfection. Interestingly, the increase was inhibited significantly in HUVEC infected with the P. gingivalis fimA mutant. In addition, the increase of IL-6 secretion induced by P. gingivalis infection was significantly impaired by the meiosis specific kinase 1 inhibitor, PD98059, or the nuclear factor kappaB inhibitor, Bay11-7082. Furthermore, we demonstrated that gp130 expression increased with P. gingivalis infection. Importantly, gp130 expression was significantly impaired by P

gingivalis fimA mutant infection compared with wild-type P. gingivalis infection, as assessed by both quantitative RT-PCR and FACS analysis. CONCLUSION: Our findings indicate that P. gingivalis fimbriae are important factors in the autocrine regulation of IL-6, by increasing gp130 in endothelial cells.