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Implant Surface Characteristics Affect Macrophage Polarization, Modulating Osteoblast Mineralization

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Objectives Successful osseointegration of dental implants relies on an intimate crosstalk between immune cells and osteoprogenitors. We hypothesized that the characteristics of the implant surface might be important to modulate the polarization of macrophages, thus contributing to the creation of a micro-environment favorable to osseointegration. The aim of this study was to investigate how various implant surfaces modulate the activation of human macrophages, and how the released cytokines affect osteoblast differentiation.
Methods Human monocytes (THP1) were seeded on four different dental implant surfaces: hydrophobic or hydrophilic sandblasted acid-etched titanium (SLA and SLA+), and hydrophobic or hydrophilic sandblasted acid-etched mixed titanium(85%)/zirconia(15%) (R and R+) surfaces. The release of pro-inflammatory interleukins (IL) 6 and 8, as well as of the anti-inflammatory IL4 and IL10 was studied by qRT-PCR and cytokine arrays. Subsequently, MG63 osteoblast-like cells were culture under osteogenic condition with THP1 conditioned medium. MG63 mineralization was assessed after 21 days by Alizarin Red staining.
Results R-like surfaces showed to promote the release of pro-inflammatory cytokines compared to the SLA counterparts. *Vice versa*, the SLA-like surfaces supported the release of the anti-inflammatory cytokines. The increased hydrophilicity for both SLA and R surfaces reduced the release of pro-inflammatory cytokines compared to the hydrophobic surfaces, while it did not show any effect with regard of the anti-inflammatory cues. Overall, the SLA+ surface markedly reduced the release of IL6 compared to SLA, R and R+ surfaces. IL6 is a potent pro-inflammatory cytokine, which is known to inhibit osteogenesis. Accordingly, mineralization of MG63 was stronger when THP1 cells were cultured on the SLA+.
Conclusions Taken together, these data prove that the type of implant surface affects the activation of immune cells, which consequently regulate the behavior of osteoprogenitors.