Title *

The role of IL-1 in silica nanoparticle-induced pro-inflammatory responses in human lung epithelial cells

Abstract *

Non-crystalline silica particles of submicro- and nanosize have a wide range of applications for consumers, and also for potential medical products. A major challenge in nanoparticle (NP) research is to elucidate critical initial targets that mediate NP signalling events leading to cytotoxicity and inflammation. We have previously reported that silica nanoparticles (SiNPs) of nominal size 50 nm induce the pro-inflammatory cytokines CXCL8 and IL-6 in human lung epithelial cells (BEAS-2B), via mechanisms involving MAPK p38, TACE-mediated TGFα release and the NFκB-pathway. In the present study, we examined the role of interleukin-1 (α and β) in induction of cytokine responses by silica nanoparticle of nominal size 10 nm (Si10) in BEAS-2B cells. The cells were exposed to Si10 and the time-dependent gene expression and cytokine release were assessed by qPCR and ELISA, respectively. The cells were pretreated with the IL-1 receptor antagonist (Anakinra) and examined for inhibition of IL-6, CXCL8, TNFα, IL-1 (α and β) and COX-2. The results showed a time-dependent increase of the cytokines, preceded by an up-regulation of the respective genes. The IL-1 receptor antagonist reduced the Si10-induced gene expression of all the cytokines, but much less in the initial phase (1.5-3 h) than upon prolonged exposure (4.5-12 h). A similar pattern was observed for the cytokine releases. In conclusion, the initial Si10-induced gene expression and cytokine release seem to be mediated via an IL-1 receptor-independent mechanism. However, the prolonged gene up-regulation and release of all the cytokines seem to be mediated via a mechanism involving the IL-1 receptor.

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