Adsorption of Lipoproteins onto Mineral Dust Surfaces: A Possible Factor in the Pathogenesis of Particle-induced Pulmonary Fibrosis?

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We compare the adsorption behavior of high density lipoproteins (HDL) and low density lipoproteins (LDL) on "fibrogenic" and "nonfibrogenic" mineral dusts. The adsorption tests with bovine lipoprotein concentrate and human serum produced the following results: 1) All seven examined fibrogenic dusts (SiO_2 DQ12, SiO_2 F600, silica, graphite, TiC, kaolin, talc) adsorbed significantly more high density lipoproteins (HDL), than the five examined nonfibrogenic (inert) dusts (TiO_2, SnO_2, Al_2O_3, Fe_2O_3, Fe_3O_4). This different behavior was particularly conspicuous in the presence of competing adsorbates (serum proteins). 2) In contrast, the adsorption of LDL did not correlate with the fibrogenicity of the mineral dusts. 3) The known silicosis-protective substance polyvinylpyridine-*N*-oxide inhibits the HDL adsorption of α-quartz. These results indicate that the adsorption of HDL could have a causal relationship with the triggering of a fibrotic reaction. The adsorption on the surface of fibrogenic dust particles provides an exceptional opportunity for the intake of HDL by macrophages. During the phagocytosis of the inhaled dust particles, the HDL adsorbed on the surface of the particles could be taken up by macrophages regardless of the receptor. There the HDL particles and/or compounds associated with them, such as lecithin-cholesterol-acyltransferase, could stimulate the macrophages to release fibrogenic mediators by some yet unknown mechanism.

Key words: Fibrosis, Mineral Dusts, High Density Lipoproteins