Biological response of tissues with macrophagic activity to titanium dioxide.

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The titanium dioxide layer is composed mainly of anatase and rutile. This layer is prone to break, releasing particles to the milieu. Therefore, corrosion may cause implant failure and body contamination. We have previously shown that commercial anatase-titanium dioxide (TiO(2)-anatase) is deposited in organs with macrophagic activity, transported in the blood by phagocytic-mononuclear cells, and induces an increase in the production of reactive oxygen species (ROS). In this study, we evaluated the effects of rutile-titanium dioxide (TiO(2)-rutile). Male Wistar rats were injected i.p. with a suspension of TiO(2)-rutile powder at a dose of 1.60 g/100 g b.w. Six months postinjection, the presence of Ti was assessed in serum, blood cells, liver, spleen, and lung. Titanium was found in phagocytic mononuclear cells, serum, and in the parenchyma of all the organs tested. TiO(2)-rutile generated a rise in the percentage of reactive cells, which was smaller than that observed when TiO(2)-anatase was employed in a previous study. Although TiO(2)-rutile provoked an augmentation of ROS, it failed to induce damage to membrane lipids, possibly due to an adaptive response. The present study reveals that TiO(2)-rutile is less bioreactive than TiO(2)-anatase. (c) 2007 Wiley Periodicals, Inc.

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Ultrafine titanium dioxide particles in the absence of photoactivation can induce oxidative damage to human bronchial epithelial cells.

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Ultrafine titanium dioxide (TiO(2)) particles have been shown to exhibit strong cytotoxicity when exposed to UVA radiation, but are regarded as a biocompatible material in the absence of photoactivation. In contrast to this concept, the present results indicate that anatase-sized (10 and 20 nm) TiO(2) particles in the absence of photoactivation induced oxidative DNA damage, lipid peroxidation, and micronuclei formation, and increased hydrogen peroxide and nitric oxide production in BEAS-2B cells, a human bronchial epithelial cell line. However, the treatment with anatase-sized (200 and >200 nm) particles did not induce oxidative stress in the absence of light irradiation; it seems that the smaller the particle, the easier it is for the particle to induce oxidative damage. The photocatalytic activity of the anatase form of TiO(2) was reported to be higher than that of the rutile form. In contrast to this notion, the present results indicate that rutile-sized 200 nm particles induced hydrogen peroxide and oxidative DNA damage in the absence of light but the anatase-sized 200nm particles did not. In total darkness, a slightly higher level of oxidative DNA damage was also detected with treatment using an anatase-rutile mixture than with treatment using either the anatase or rutile forms alone. These results suggest that intratracheal instillation of ultrafine TiO(2) particles may cause an inflammatory response.

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