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TITLE: ULTRAFINE CARBON BLACK PARTICLES INHIBIT HUMAN LUNG FIBROBLAST-MEDIATED COLLAGEN GEL CONTRACTION

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ABSTRACT: Both acute and chronic exposure to particulates have been associated with increased mortality and morbidity from a number of causes, including chronic obstructive pulmonary disease and other chronic lung diseases. The current study evaluated the hypothesis that ultrafine carbon particles, a component of ambient particulates, could affect tissue repair. To assess this, the three-dimensional collagen gel contraction model was used. Ultrafine carbon black particles, but not fine carbon black, inhibited fibroblast-mediated collagen gel contraction. Although previous research has indicated that inflammatory effects of ultrafine carbon black particles are mediated by oxidant mechanisms, the current study suggests that ultrafine carbon black's inhibition of fibroblast gel contraction is mediated by the binding of both fibronectin and transforming growth factor (TGF)-beta to the ultrafine particles. Binding of TGF-beta was associated with a reduction in nuclear localization of Smads, indicative of inhibition of TGF-beta signal transduction. There was also a decrease in fibronectin mRNA, consistent with a decrease in TGF-beta-mediated response. Taken together, these results demonstrate the ability of ultrafine particles to contribute to altered tissue repair and extend the known mechanisms by which these biologically active particles exert their effects.