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**Title:** LSC 2013 abstract - Cr(VI)stimulated fibroblasts and epithelial mesenchymal transition induction in epithelial cells

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**Body:** EMT permits epithelial cells (EC) to reprogram and acquire mesenchymal motile phenotype; happens during embryonic development, in physiological response to injury and in cancer progression metastasis formation. Normal human bronchial fibroblasts were first rendered senescent following exposure to Cr(VI). A normal bronchial fibroblasts culture (E2A, established from normal redundant tissue) was exposed to either 0.25/0.5  $\mu$ M Cr(VI) for 4 weeks. To evaluate how normal and senescent fibroblasts and EC co-evolved together, normal/senescent bronchial fibroblasts were then co-cultured with epithelial BEAS-2B cells in transwells in the absence/presence of 0.25/0.5  $\mu$ M Cr(VI). BEAS-2B cells exposed to 0.5  $\mu$ M Cr(VI) co-cultured with non-senescent fibroblasts showed cuboidal regular morphology and expressed vimentin/MNF-116/CK7. Non-treated epithelial cells co-cultured with senescent fibroblasts exposed to 0.25  $\mu$ M Cr(VI), revealed mesenchymal-like phenotype positivity for LP34/vimentin and weak expression of CK7. The fibroblasts, in both conditions, showed a crisscross organization and stellate morphology predominated. Fusiform vimentin-positive and  $\alpha$ -SMA-positive cells were detected as myofibroblasts. Simultaneous expression of vimentin/MNF-116/CK7 and cuboidal morphology shown by epithelial cells exposed to Cr(VI) is consistent with acquisition of pleomorphic-like carcinoma phenotype. On the other, the positivity for LP34/vimentin, revealed by non-treated BEAS-2B cells in the presence of senescent fibroblasts is characteristic of basal cell epidermoid carcinoma phenotype. These results suggest that the exposure to Cr(VI) and the presence of senescent fibroblasts induce malignant features in epithelial cells.