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Polymorphisms of Surfactant Protein Gene *A*, *B*, *D*, and of *SP-B*-linked Microsatellite Markers in COPD of a Mexican Population *

Xiaoxuan Guo, Hung-Mo Lin, Zhenwu Lin, Martha Montaño, Raul Sansores, Guirong Wang, Susan DiAngelo, Annie Pardo, Moises Selman and Joanna Floros

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traction (60.8 ± 2.5%; $p < 0.05$) but had little effect on A549 contraction (63.6 ± 1.3%; $p > 0.05$). As cigarette smoke is an oxidant stress, we next evaluated if A549 was resistant to the effect of cigarette smoke due to glutathione content. Consistent with this, A549 cells contained significantly more glutathione (3.23 vs 0.88 nmol/10^6 cells; $p < 0.05$) than did HFL-1 cells. A549 cells also secreted glutathione into the culture medium. Both A549 culture medium and exogenous glutathione were capable of protecting HFL-1 cells from cigarette smoke inhibition of fibroblast gel contraction. Taken together, these studies suggest that alveolar epithelial cells may be able to participate in repair responses following injury including tissue contraction. Such capabilities may be important in limiting alveolar enlargement characteristic of emphysema. Intracellular antioxidant mechanisms may be important in protecting against adverse effects of cigarette smoke, and epithelial cells antioxidants may protect mesenchymal cells.

Cognitive Improvement Following Rehabilitation in Patients With COPD*

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Abbreviations: BDI = Beck depression inventory

Thirty patients with COPD who completed 3 weeks of rehabilitation were evaluated before and after treatment on measures of cognition, depression, pulmonary function, and exercise capacity. These patients were compared to 29 untreated COPD patients and 21 healthy control subjects tested over a similar interval on these measures (including a 2-h neuropsychological assessment). Using analysis of covariance, significant group differences were found on the digit vigilance test (sustained visual attention, $p = 0.006$), visual retention (visual memory, $p = 0.03$) and semantic fluency (ability to generate words to a category, $p = 0.04$) scores. *Post hoc* pairwise comparisons found a greater improvement in the treated vs untreated COPD groups on digit vigilance and semantic fluency ($p < 0.05$). Additional results indicate that the treated COPD group significantly improved on the 6-min walk (mean, 265.6 feet) compared to the untreated COPD group (mean, 43.2 feet), and that this improvement was significantly correlated with improved digit vigilance scores ($p = 0.03$). Although the treated COPD group did not have a significant decline in depressive symptoms compared to the other groups on the Beck Depression Inventory (BDI), the change scores suggested a "clinical decline" in symptoms from the mildly distressed (mean BDI, 13.6) to the normal range (mean BDI, 8.3). Overall, these results indicate that changes in select cognitive functions and exercise capacity occur in COPD patients in rehabilitation compared to untreated COPD patients and healthy control subjects.

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COPD is a major medical problem and leads to a significant morbidity and mortality among the adult population. Several factors have been suggested as risk factors for COPD, including environmental and genetic, supporting the notion that the etiology of COPD is multifactorial and/or multigenic. One of the important risk factors is cigarette smoking, where a significant number of smokers develop COPD.[1–3] Although inherited deficiency of α_1-antitrypsin is one of the documented risk factors, this accounts for < 1% of the COPD cases.[1,4] Other genetic polymorphisms have been associated with increased susceptibility to developing COPD.[5,6] In addition, the expression of the disease itself is complex, *ie*, some COPD patients may develop predominately airway disease and others parenchymal disease.

Pulmonary surfactant, a lipoprotein complex, is essential for normal lung function.[7] In addition, pulmonary surfactant or its components are shown to play important roles in the innate host defense of the lung and the regulation of inflammatory processes.[8] Because there is a chronic inflammation in COPD, and because deranged surfactant composition has the potential to contribute to both surfactant dysfunction and to altered defenses and altered regulation of inflammatory processes in the lung, we reasoned that the surfactant system is a good candidate

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