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Depletion of glutathione S-transferase P1 induces apoptosis in human lung fibroblasts.

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Glutathione S-transferase P1 (GSTP1) is one of the xenobiotic-metabolizing and antioxidant enzymes, identified in the peripheral lungs. Recently, the authors reported the association between GSTP1 gene polymorphism and susceptibility to chronic obstructive pulmonary disease (COPD), and protective effect of GSTP1 against cigarette smoke in human lung fibroblasts in vitro. In this study, the authors investigated that depletion of GSTP1 by itself could induce cell death, including apoptosis, in human lung fibroblast-derived HFL-1 cells. The level of apoptosis and necrosis was increased significantly with GSTP1 antisense vector transfection. It was also observed that the transfection efficiency and the expression level of the vector were weaker in the transfectant of the antisense vector than in those of the sense and control vectors, which is also thought to indicate that inhibition of GSTP1 expression by the antisense vector alone affects cellular viability. However, there was no difference among these transfectants neither on glutathione (GSH) level nor on c-Jun NH2-terminal kinase (JNK) activation. Therefore, the authors report here that underexpression of GSTP1 appeared to induce apoptosis on lung fibroblasts, which suggests that GSTP1 may have protective effects against apoptosis in the airway cells, though the mechanism of this apoptotic pathway is still to be elucidated.

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