

Modelling Whole Smoke exposure: developing a superior tool for Smoke research.

### **Oliver Reed**

Epidemiological studies and research have linked smoking to a number of different diseases including lung cancer and chronic obstructive pulmonary disease (COPD), however the molecular mechanisms and pathways underpinning the smoking disease processes are poorly understood.

In vitro model systems, in which human lung cells are exposed to appropriate doses of cigarette smoke, may provide useful tools to interpret the processes.

Two models are currently used to study the effect of cigarette smoke on Human Bronchial Epithelial cells: Cigarette Smoke Extract (CSE) and the Whole Smoke model (**Fig. 1**).

In the CSE model, cells are directly submerged in medium containing dissolved CSE. With regards to Human Bronchial epithelial cells, CSE impairs development of cilia and epithelial damage repair mechanisms. In the Whole smoke model, cells are placed in an air/liquid interface (ALI). The ALI method results in culture medium on the basolateral side and air/smoke dilution on the apical side. The Whole smoke model allows for the induction of cilia formation and exposure to both soluble and insoluble particles (in CSE model, only soluble particles are retained by the liquid). Thus the Whole smoke model allows for a more accurate model.

Undifferentiated Epithelial cells (A549 cells) were used to identify an adequate exposure model of cigarette smoke. The release of lactate dehydrogenase (LDH), interleukin (IL)-6 and IL-8 were measured as markers of cell death and inflammation respectively.