Cigarette smoking is one of the major risk factors for the development of lung cancer [1]. However, little is known about nicotine's effects on human bronchial epithelial cells. This study aimed to investigate the effects of nicotine on immortalized BEAS-2B and BZR cells, and whether these effects contribute to tumorigenesis.

**Results**

- **Effect of Chronic Nicotine Treatment on Cell Proliferation**
  - BEAS-2B and BZR cells were grown in collagen I-coated 6-well plates or cell culture flasks (VWR, Dietikon, Switzerland) at 37°C in a humidified 5% CO2 atmosphere. Cell counting was performed weekly to assess nicotine's impact on cell proliferation.
  - Nicotine did not increase the proliferation of immortalized BEAS-2B or BZR cells as determined by cell counting.

- **Effect of Chronic Nicotine Treatment on Staurosporin-Induced Apoptosis**
  - Staurosporine is a potent P38 inhibitor that induces apoptosis. The EC50 values of staurosporine-induced caspase 3/7-mediated apoptosis were calculated by fitting the points to a straight line.
  - Control and nicotine-treated cells were stained for caspase 3/7 activity and imaged using the Cellomics® ArrayScan™ VTI High Content Screening Reader (Thermo Fisher Scientific Inc.).

- **Effect of Chronic Nicotine Treatment on MMP levels**
  - MMP-1 and MMP-9 levels were measured in cell culture supernatants using the Luminex® Performance Human MMP magnetic bead panel according to the manufacturer's instructions (Merck Millipore, Darmstadt, Germany).

**Conclusions**

- Chronic nicotine treatment in immortalized non-tumorigenic BEAS-2B and tumorigenic BZR cells did not promote cell proliferation or survival, did not suppress apoptosis, and did not initiate any mechanisms that favor tumorigenesis.

**References**