Nicotine induces overexpression of low affinity p75 NGF receptor in monocytes

Mario Rende, Alessandra Pistilli, Anna Maria Stabile, Maria Grazia Rambotti, Antonio Spreca and Claudia Montagnoli
School of Medicine, sect. of Human Anatomy, University of Perugia, Perugia, Italy

In inflammatory pulmonary diseases, cigarette smoking is a major risk factor influencing the phenotype of immune cells and their functions (Arnson et al., 2010). Nicotine is a relevant constituent of cigarette smoking and, on monocytes, it binds nicotinic acetylcholine receptors inducing a quantitative increase in the levels of CD14 and a decrease in TNF-α. Altogether, these data supports an anti-inflammatory effect of nicotine on these cells. Our previous studies have shown that NGF and its receptors TrKA and p75 are involved in inflammatory pulmonary diseases. In particular, we have shown that smokers present an increase in p75 expression on monocytes, but the cause that triggers this increase is actually unknown. In addition, cigarette smoking seems not to vary the TrKA expression on these cells.

The aim of our study was to investigate in vitro the biological effects of nicotine on the percentage of TrKA- and p75-positive monocytes from human healthy non-smoker donors. Cytofluorimetric investigation confirms that in monocytes nicotine treatment does not influence the percentage of TrKA, but induces a relevant dose-dependent increase in the percentage of p75 that, in turn, could be an element in the general anti-inflammatory effects of nicotine. Consequently, in order to investigate the involvement of p75 in inflammatory/non-inflammatory mechanisms, we added in vitro the inflammatory stimulus MCP-1 on monocytes. Cytofluorimetric investigation have shown that, in this latest inflammatory condition, the percentage of p75-positive monocytes was greatly reduced.

In conclusion, our data support the hypothesis that nicotine-induced p75 overexpression could be involved in its anti-inflammatory effect.

References

Keywords: NGF, TrKA, p75, nicotine.