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Title: Tobacco smoking alters the relationship between airway inflammation and airway hyperresponsiveness in asthma

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Body: Background: Smoking in asthma patients constitutes a major health problem, due to impaired steroid responsiveness, poor symptom control and an accelerated loss of lung function. This may relate to alterations in the airway inflammation. The aim of the present study was to evaluate the effect of smoking on the relationships between airway inflammation and airway hyperresponsiveness in asthma patients not on steroid treatment. Material and methods: A group of smoking asthma patients (n=27) was compared to a group of non smoking asthma patients (n=34) with induced sputum, exhaled nitrogenous oxide (eNO) and airway hyperresponsiveness with a bronchial challenge with mannitol. Airway eosinophilia was defined as a sputum % eosinophils > 2 %. Results: A significantly higher proportion of patients with non-eosinophilic asthma was observed in the smoking group than in the non-smoking group (44% and 18%, p=0.023). The mean eNO was lower in the smoker group compared to the non-smoker group (11.7 ppb vs 38.2 ppb (p=0.001)). The proportion of subjects with a positive mannitol test was comparable among smokers (68%), and non-smokers (50%) (p=0.19). Smokers with a positive mannitol test (n=15) had a lower mean eNO (15.4 ppb) than the non-smokers (mean eNO=46.3 ppb, n=15), p=0.006. Conclusion: Our data showed that despite of a lower proportion of eosinophilic phenotype amongst the smoking asthma patients than amongst the non-smokers, no reduced degree of airway hyperresponsiveness to mannitol in the smoker group was observed, indicating that pathogenetic mechanisms other than eosinophilic airway inflammation are responsible for the tendency to airway narrowing in smoking asthma patients.