Smoking Intensity and its Relationship with Lung Function and Antioxidants in Healthy Subjects

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Sir,

The gas and tar phases of tobacco are rich in reactive oxygen and nitrogen species and disrupt the respiratory and pulmonary function leading to development of COPD, cancer etc.¹ Antioxidants form the first line of defense against oxidative insult. Studies on lung function and oxidant/antioxidant imbalance relating to the intensity of smoking in healthy subjects was sparse. Hence, prospective cross-sectional study was undertaken to test the lung function, oxidant and antioxidant imbalance, in healthy smokers focusing on the effect of duration and quantity of smoking. Data was collected from a random sample of 150 smokers (males) who were >40 years of age with a smoking history >10 pack years from rural villages in Chittoor District, Andhra Pradesh. Sample size was established assuming a 95% confidence interval with 3% sampling error. Informed consent was obtained from all the subjects. Spirometry was carried out on individuals with no respiratory problems strictly with an FEV1/forced expiratory volume in 6 s ratio >70%. Information on demographics, smoking status and anthropometry were collected. Pack years were calculated as per the standard procedure. Erythrocyte superoxide dismutase, catalase, glutathione S-transferase, glutathione peroxidase and reduced glutathione and plasma malondialdehyde was measured as per the standard procedures.

Study participants had a mean age of 60 years, average number of cigarette/bidi per day 26, average duration of smoking 42 years, average pack years was 46 respectively. FEV1% and FEV1/FVC% were negatively correlated with age and duration of smoking status. Systolic blood pressure exhibited a linear relationship with age and duration of smoking. Pack years was positively correlated with malondialdehyde. Glutathione peroxidase activity negatively correlated with duration of smoking, while reduced glutathione was negatively correlated with number per day and pack years. Pack years had exerted about 23% of the variance in the malondialdehyde level and 10% of the variance in reduced glutathione level. An elevation of one pack year increases 0.6 nmol/ml of malondialdehyde and decreases 0.3 nmol/ml of reduced glutathione level.

The present investigation confirms the role of oxidant and antioxidant imbalance in smokers. Intensity of smoking status elevated the plasma malondialdehyde level and depletion of antioxidant levels. Further, both FEV1% and FEV1/FVC% decreased significantly upon exposure to smoking, indicating the restrictive pattern of disease as against obstructive or mixed as noticed in several studies. These observations are in line with our previous work showing declined pulmonary function test parameters in smokers with COPD.² Even though respiratory function ratio failed to show association either with oxidative stress and antioxidants, exposure to smoking has significantly elevated oxidative stress and decreased antioxidant capacity. Increased malondialdehyde levels and decreased antioxidant levels were observed upon exposure to smoking. Interestingly lung function was positively correlated with malondialdehyde in the study sample. On the other hand malondialdehyde was negatively correlated with reduced glutathione. The correlations observed in the study could be speculated that antioxidant supplementation may ameliorate the deterioration of lung damage. Thus, stoppage of smoking, before any clinically evident respiratory impairment takes place, bears potential scope for preventive programs.

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References

