SUMMARY
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Cigarette smoking is a reprehensible habit that has spread all over the world and is also associated with development of various pulmonary cardiac, vascular and ocular diseases causing mortality and morbidity. Cigarette consumption has risen over the past two decades in India and most other countries. Its stimulatory, euphoric, reinforcing properties and effects such as mood control, weight control and improving the performance and health of people to cope with daily stress make people not to quit the habit. Cigarette smoke contains > 4,000 known constituents and > 1,00,000 unknown constituents in its gas and tar phases among which nicotine, tobacco specific nitrosamines, certain aldehydes, peroxides are some. Inhaled smoke passes through parts of respiratory as well as digestive tract to enter circulation and thereby affecting blood and tissues causing oxidative damage leading to atherosclerosis and various diseases. Actual mechanisms underlying the physiological and biochemical events associated with cigarette smoking are not clear. Now the role of nitric oxide has been recognized in various physiological processes and surprisingly nitric oxide is a component of cigarette smoke also. However its precise role in various biochemical events associated with smoking is yet to be understood. Hence the present study is aimed at understanding the biochemical changes associated with cigarette smoke induced impairment, and, the status of atherogenic factors as well as the role of nitric oxide and associated molecular interactions in plasma, red cell and red cell membrane in chronic cigarette smoking human volunteers. Cigarette smoking has been associated with oxidative stress and impaired endothelium dependent vasodilation, primarily causing atherogenic damage. Observed increase in plasma cholesterol points to a
derangement/impairment in cholesterol metabolism and homeostasis and this finding corroborates the reports of Criqui et al., Craig et al and Latha et al. Increased LDL and fall in HDL with unaltered triglycerides and VLDL plus hike in plasma and membrane lipid peroxidation observed in the present study have confirmed the increased oxidant stress and endogenous generation of free radicals resulting in tissue damage in cigarette smokers which has been implicated in the pathogenesis of atherogenesis and various smoking associated cardiovascular, pulmonary, ocular and cerebral diseases. This is evident from the observed changes in above parameters in this study.

Further, results of this study suggested that cigarette smoking induced increase in hemolysis – when red cells were exposed to different concentrations of NaCl (osmotic fragility), and changes in membrane cholesterol and phospholipid moieties with no significant change in the consequent C/P ratio plus changes in plasma and red cell enzymes strongly suggest the operation of certain protective mechanisms amidst oxidant stress of smoking. An important finding of the present study is an increase in the concentration of nitrites and nitrates of plasma indicating increased production of nitric oxide. Though various endogenous, environmental and dietary and other factors may contribute for the observed hike in plasma NO2 and NO3 in the present study, available literature strongly suggests the relationship between nitric oxide and cigarette smoking addiction. An up regulation of endothelial nitric oxide synthase protein and NO generation by increased eNOS activity in presence of increased eNOS protein expression in cigarette smokers cannot be ruled out in the present study. Besides the study also suggests the role of NO in addition to hormonal and other factors in the causation of observed findings such as undisturbed glucose homeostasis, increased