INTRODUCTION

Habits have been defined as repetitive action of certain conditional phenomenon controlled by neuromuscular impulses. Certain habits are likely to bring about pathological changes in tissues, if continued for a long time. Amongst our people, the habit of smoking and tobacco chewing appears to be wide spread and is cutting through all cross-sections of the society. On a world scale, tobacco consumption is increasing by 2.1% per annum. In the developed countries, like U.S.A., European continent, it is decreasing by 0.2% per annum, while in the developing countries like India, tobacco consumption is increasing by about 3.4% per annum, and mostly the youth, especially during adolescent period, are getting hooked to smoking and chewing under peer influence. About 50% of the total tobacco use in India is in smokeless form, i.e. zarda, gutkha, khaini, pan-masala, etc. Over the last decade, with commercial availability of qualitatively consistent tobacco, arecanut, lime, catechu etc. preparations in attractive polythene pouches and their heavy promotion through mass media and sporting events, have led to increase in use of these smokeless tobacco preparations among the youth, which foster the nicotine addiction and dependence more stronger than cigarette smoking.

A correlation has been experimentally established by various research workers, both in India and abroad, between smoking, smokeless tobacco to periodontal disease, premalignant lesions as well as oral cancer by studies on laboratory animals and human beings supported by epidemiological data.
This effect of tobacco smoking and chewing on the periodontium has been the subject of many studies since a few decades. The clinical appreciation of differences in the periodontal health status and the tissue response to treatment in smokers, chewers and non-smokers has stimulated extensive research activity. Nevertheless, descriptive cross-sectional studies of Western countries indicate that smokers and chewers experience more periodontal breakdown and depression of host defence posture\textsuperscript{18,22,60,80}.

A lot of actions and mechanisms are implicated to be the cause for increased incidence of intra-oral neoplastic disease among tobacco habiters. These include increased irritation, various products of tobacco combustion and tobacco-specific nitrosamines acting as vaso-constrictors of peripheral blood vessels of oral mucous membrane resulting in restricted circulation to these tissues\textsuperscript{1,8,9}. Interference of function of polymorphonuclear leukocytes from oral cavity\textsuperscript{13,88}, interference of natural killer cell (NK cell) function\textsuperscript{19}, and generalized immunologic impairment\textsuperscript{29} are also suggested. Interpretation of changes in immune function associated with tobacco usage, however, must be constrained by limitations of the current knowledge concerning the immune system and carcinogenesis\textsuperscript{2}.

Although the extent to which the changes in immune system may affect the host, or the tissues to mount and maintain an effective defensive reaction is still unclear, a number of studies do suggest impairment of an efficient response in users of smokeless tobacco and smokers\textsuperscript{8,107}. A number of mechanisms act to reduce the defense, but their relative contributions remain to be elucidated.
Bennet and Reade measured the salivary immunoglobulin A (S-IgA) levels in normal subjects, tobacco smokers and patients with minor aphthous ulcerations arriving at the conclusion that there is definite decrease of S-IgA in chronic tobacco smokers. They hypothesized the significance of decrease in S-IgA concentrations in chronic tobacco smokers in the light of this, which is being due to the immunosuppressive effect of combustion products of tobacco and the possibility of intra-oral neoplastic disease being increased in tobacco smokers by this effect.

The concentration of secretory immunoglobulin A is the highest of all immunoglobulins in unstimulated whole saliva. To quote Sir Macfarlane Barnett "S-IgA is antiseptic coat over mucous surfaces of oral cavity and is very important for structural integrity and barrier function of oral mucous membrane."

There is limited literature available on cigarette smoking and immunological parameters from oral cavity, including the study of S-IgA levels by Bennet and Reade (1982). A large section of the Indian youth is hooked to the use of smokeless tobacco with its commercial availability since a decade or so. It will be relevant in the Indian context to find out scientifically whether Indian smokeless tobacco has similar effect on S-IgA levels or not and, if so, whether they are comparable to cigarette smoking.

Reports indicate a gain/reversal in immunological impairment and Remission/Disappearance of premalignant lesions on cessation of habitual tobacco usage either by smoking or by smokeless tobacco. Hence, this study was planned to determine periodontal health status of Indian tobacco habiters including prevalence of various grades of recession at tobacco placement site in
chewers and to assess the salivary immunoglobulin-A in unstimulated whole saliva of cigarette smokers and users of smokeless tobacco in form of commercially available preprepared tobacco pouches as an indicator of local immunologic reactivity in mouth. Secondarily, in addition to determining immunotoxicity, local immune recovery and change in severity/disappearance of leukoplakia on cessation of smoking and tobacco chewing was studied in pre-selected highly motivated subjects with adjunctive use of tobacco deterrent tablets. Therefore, the present study has been planned to evaluate and to elucidate the immunological status of urban young smokers and chewers with following objectives:

(1) To determine periodontal health status of tobacco habiters.

(2) To determine the incidence and prevalence of various grades of recession at placement site of tobacco in chewers.

(3) To determine immunotoxicity related to quantitative salivary immunoglobulin-A (S-IgA) level in tobacco habiters, i.e. smokers and chewers with secondary aim to determine local immune recovery (S-IgA) in pre-selected tobacco habiters on tobacco cessation.

(4) To correlate the local immune recovery (S-IgA) on tobacco cessation to change in severity/disappearance of leukoplakia in tobacco habiters.