The elements in reactive thought resulting from tobacco, betel, and alcohol are carcinogenic and can be responsible for the induction of chromatin/chromosomal aberrations resulting in production of micronuclei. The genotoxic and carcinogenic chemicals released from betel nut and tobacco and also the calcium hydroxide content of lime present in the betel quid are thought to be responsible for promotion of reactive oxygen species from areca nut extracts. These reactive oxygen species can in turn cause damage to the DNA.

FORMATION OF MICRONUCLEI: Micronuclei are extra nuclear cytoplasmic bodies. They are induced in cells by numerous genotoxic agents that damage the chromosomes. The damaged chromosomes, in the form of acentric chromatids or chromosome fragments, lag behind in anaphase when centric elements move towards the spindle poles. After telophase, the undamaged chromosomes, as well as the centric fragments, give rise to regular daughter nuclei.
The lagging elements are included in the daughter cells, too, but a considerable proportion is transformed into one or several secondary nuclei, which are, as a rule, much smaller than the principal nucleus and are therefore called micronuclei.3

**DISCUSSION:** Squamous cell carcinoma of the oral mucosa accounts for 90% to 95% of all oral malignancies.3 Oral exfoliative cytology has been used extensively for screening cellular alteration in oral squamous cell carcinoma cases. An accuracy of 95% and a reliability of more than 96% in detection of squamous cell carcinoma in mass screening have been reported in the literature.4 Oral exfoliative cytology can reveal various cellular alterations in squamous cell carcinoma. It includes karyorrhexis, karyolysis, micronucleus formation, pyknosis, binucleation, broken-egg nucleus, anucleation, etc.5,6

Micronuclei in oral exfoliated cells is a marker of chromosomal damage caused by genotoxic agents from tobacco and tobacco-related substances, alcohol, etc.7,13 The micronucleus assay has been used to assess the genotoxic damage in oral squamous cell carcinoma and oral premalignancies.8,9 It has been established that genomic damage is produced by genotoxic substances, medical procedures (radiation, chemicals), micronutrient deficiency (folic acid), lifestyle factors (alcohol, smoking, stress, drugs), and genetic factors such as defects in metabolism and/or in repair of DNA.10,11

The Micronuclei assay has been reported to correlate well with the histological grading of oral squamous cell carcinoma and leukoplakia. Incidence of micronuclei has been analyzed by various studies in normal patients, oral premalignancies, and oral squamous cell carcinoma.12 Micronuclei has been used as a biomarker for assessment of DNA damage.14 When compared with other body sites, mouth provides with a distinct opportunity for defining biomarkers because the mouth permits noninvasive, repetitive examinations in longitudinal studies of tobacco-associated acute and chronic diseases.15

There are many studies (more than 200) in which epithelial cells from other site such as nasal mucosa, cervix, bladder, esophagus and bronchi had been used for micronuclei assay.16 Biomarkers help to detect high-risk patients. They are divided into three groups: First to define the exposure to carcinogenic agents, the second to show biological effects on the target tissue and the third to give information about the individual susceptibility.17 The micronucleus assay in exfoliated buccal cells is a useful and minimally invasive method for monitoring genetic damage in humans.18

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