

EFFECTS OF THE INTERACTION OF TOBACCO SMOKE AND ALCOHOL CONSUMPTION ON BUCCAL MICRONUCLEUS IN WORKERS EXPOSED OCCUPATIONALLY TO FORMALDEHYDE



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INTRODUCTION

Occupational exposure to FA has been shown to induce nasopharyngeal cancer and has been classified as carcinogenic to humans (group 1) on the basis of sufficient evidence in humans. Tobacco smoke has been associated to a higher risk of development of cancer, especially in the oral cavity, larynx and lungs, as these are places of direct contact with many carcinogenic tobacco's compounds. Alcohol is a recognized agent that influence cells in a genotoxic form, been cited as a strong agent with potential in the development of carcinogenic lesions. Epidemiological evidence points to a strong synergistic effect between cigarette smoking and alcohol consumption in the induction of cancers in the oral cavity. Approximately 90% of human cancers originate from epithelial cells. Therefore, it could be argued that oral epithelial cells represent a preferred target site for early genotoxic events induced by carcinogenic agents entering the body via inhalation and ingestion. The MN assay in buccal cells was also used to study cancerous and precancerous lesions and to monitor the effects of a number of chemopreventive agents.

AIM OF THE STUDY

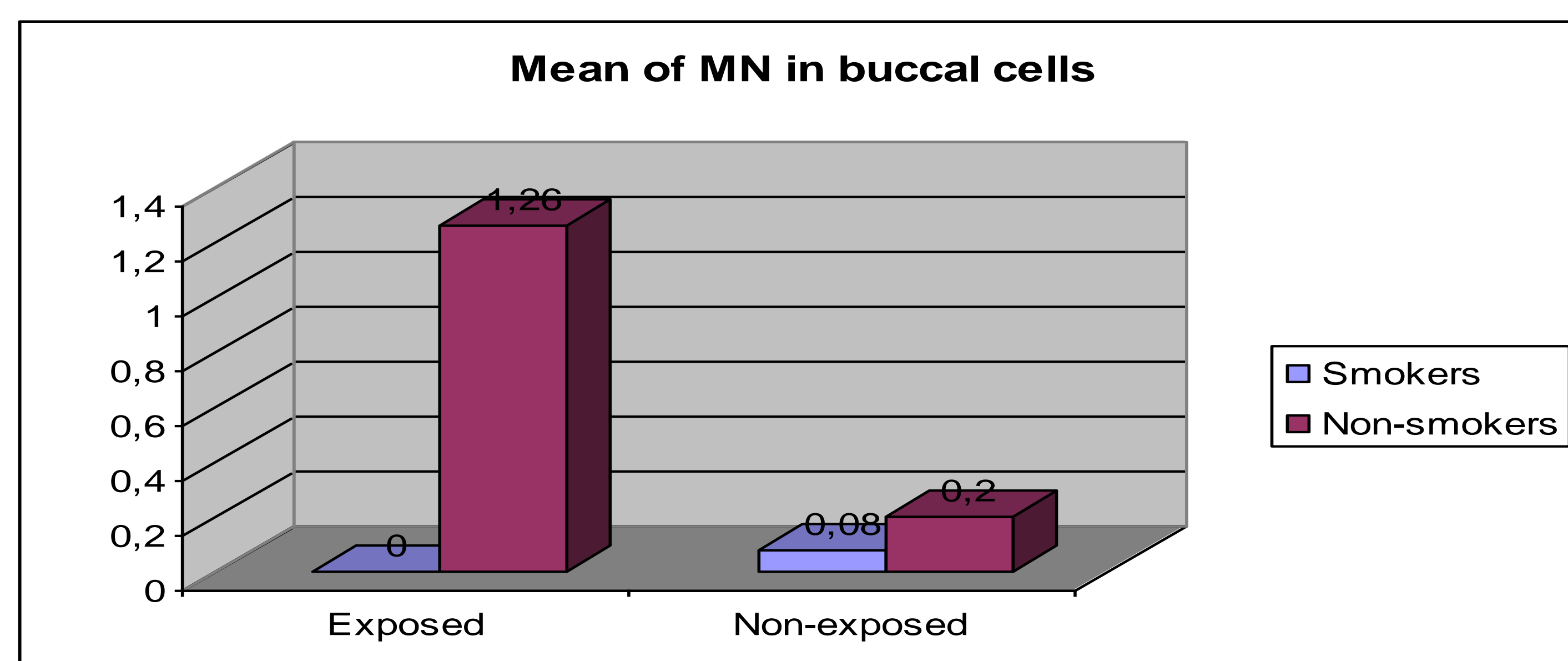
Determine the influence of tobacco smoke and alcohol consumption in workers exposed occupationally to formaldehyde by measuring the frequency of micronucleus in buccal mucosa cells

METHODOLOGY

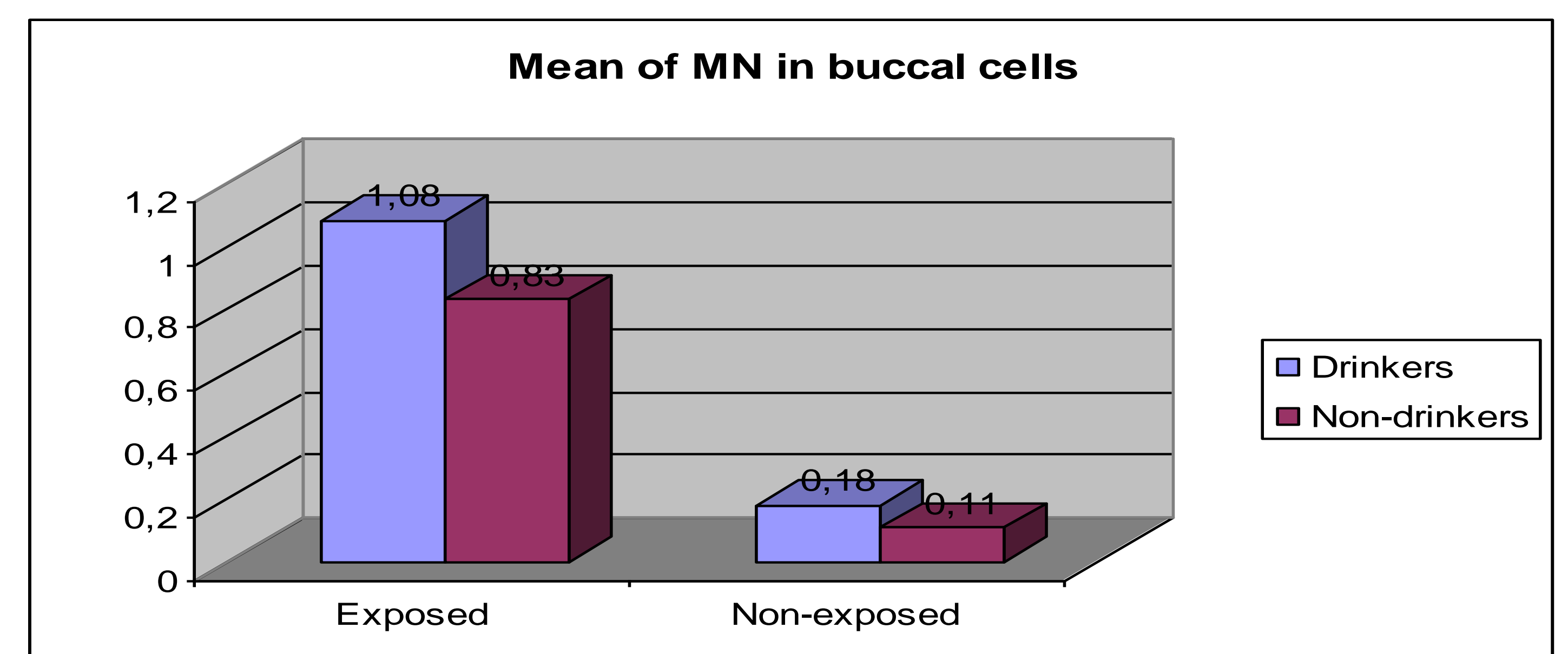
The study was carried out in Portugal in a sample of 56 workers occupationally exposed to FA in pathology anatomy laboratories and in 85 non-exposed subjects. Both groups were asked about their smoking and drinking habits. The evaluation of genotoxic effects was conducted by applying MN test in exfoliated cells from buccal mucosa. Buccal cells were removed by endobrush and stained with Feulgen technique without counterstain.

RESULTS

TOBACCO SMOKE



ALCOHOL CONSUMPTION



Subjects in study presented higher means of MN in buccal cells were found in drinkers exposed to FA (1.08 ± 0.399) in comparison with non-drinkers exposed to FA (0.83 ± 0.326). About tobacco smoke, non-smokers presented higher means of MN in comparison with smokers. The analysis of the interaction between the alcohol consumption and smoking habits demonstrate an interaction between subjects that did not drink and did not smoke and subjects who drink and smoke but that interaction did not reach statistical significance ($p=0.054$).

CONCLUSIONS

Epidemiological evidence points to a strong effect between cigarette smoking and alcohol consumption in the induction of cancers in the oral cavity. In several studies of lifestyle factors, results are controversial in what concern to tobacco and alcohol. In this study the results about tobacco were unexpected and this can be explain at part by the distribution not normal of the subjects in all classes.

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