Guest Editorial

Cytogenetic Biomonitoring of Oral Mucosa Cells for Protecting Individuals against Potential Harm: A Multidisciplinary Approach

Cytogenetic biomonitoring have been employed in human health during several decades for diagnosing, staging disease, as well as to measure the risk assessment. Such information gives statements concerning the level of risk and further evidence on the status of susceptibility. To date, a variety of assays have been proposed as potential tools for evaluating cytogenetic damage, including those that assess metaphase chromosomal aberrations, sister chromatid exchanges, DNA damage, DNA repair. The findings are used to define the exposure to carcinogenic agents, to show biological effects on the target tissue, and/or to give information about the individual susceptibility. In the last few decades, a great enthusiasm has raised by the application of cytogenetic studies in oral mucosa cells.

Considering the strong evidence for a relationship between genetic damage, and carcinogenesis,[1] as far as > 90% of all human cancers are of epithelial origin,[2] the approach is relevant to measure the side health effects induced by exposure to harmful agents. Particularly, our research group has employed with success cytogenetic biomonitoring studies for evaluating cytogenetic damage in oral mucosa cells by means of multidisciplinary approach including, dentistry, toxicology, oncology, physical education and mutagenesis.

For example, we designed a cytogenetic study making use of 39 healthy adults who had been submitted to panoramic dental radiography. Exfoliated oral mucosa cells were collected immediately before the X-ray exposure and after 10 days. Our results indicated no statistically significant differences ($p > 0.05$) in micronucleated oral mucosa cells before and after dental X-ray exposure as a result of DNA damage.[3] On the other hand, X-ray exposure did increase ($p<0.05$) other nuclear alterations closely related to cellular death (cytotoxicity), such as karyorrhexis, pyknosis, and karyolysis.[3] Moreover, we designed a cytogenetic study consisting of 15 adult males who practise weight lifting and are anabolic steroid users or 15 adult males who practise weight lifting, but are non-anabolic steroid users. No significant statistical differences ($p > 0.05$) were noticed in individuals who practise physical activity only. On the other hand, an increase of micronucleated cells in anabolic steroid (decadurabulin and Winstrol) users was observed.[4] A number of experimental studies, as well as epidemiological evidence, indicate that gasoline and diesel engine exhausts are mutagenic and carcinogenic to laboratory animals and possibly to humans.[5] A previous study conducted by our research group have demonstrated that the micronucleus frequencies in oral mucosa cells were significantly different between control and exposed subjects.[6]

In summary, this is an area that warrants investigation, since the estimation of risk from using such cytogenetic methods, will be added to those already established for regulatory purposes as a way to improve health status and prevention of oral carcinogenesis.

References

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