

Evaluation of DNA damage through cytogenetic approach in smokers and vapers with and without nicotine compared with control group

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Available from. <http://dx.doi.org/10.21931/BJ/2024.01.01.75>

ABSTRACT

The use of tobacco and electronic cigarettes is harmful to health and can cause genetic damage, increasing the risk of cancer and other serious diseases. Although e-cigarettes contain fewer toxic chemicals than conventional tobacco, recent studies have shown that the vapor caused by burning produced by e-cigarettes can also be toxic and carcinogenic. Various studies have found that those exposed to tobacco and vaping have significantly higher levels of damage to their DNA in different types of cells and tissues. Evidence has accumulated that e-cigarette vaping can alter cellular functions and DNA itself, increasing the risk of cancer and aging. The present work evaluates the cytogenetic damage in individuals exposed to conventional cigarette vapors by burning with nicotine and vapors without nicotine, compared with a non-smoking population. The study included participants with an average age of 30 years (+/-10), with a majority of men representing 70% of the sample. The Chi-square test found no significant statistical differences between the men and women exposed ($p < 0.05$). The results of chromosomal fragility found in the four groups studied (control group, conventional smokers, vapers with nicotine and vapers without nicotine) showed breaks and gaps in one or both of the chromatids in all exposed individuals, with highly significant statistical differences ($p < 0.001$) compared to the unexposed control group. No statistically significant differences were found between the group of conventional smokers and the vapers with and without nicotine, nor between the two types of vapers ($p > 0.05$). In conclusion, cytogenetic evidence of DNA damage produced by vaping is shown in the same proportions as a normal cigarette. This will significantly impact public health, which must be considered in preventive actions.

Keywords: cigarettes, vaping, chromosome breakage, DNA damage

INTRODUCTION

Tobacco use is one of the leading causes of death worldwide. It is estimated that more than eight million people die each year due to tobacco-related diseases. Tobacco contains chemicals that are harmful to health, such as tar, carbon monoxide, and nicotine. These substances can cause genetic damage and increase the risk of cancer and other serious diseases¹⁻³.

In recent years, electronic cigarettes have emerged as an alternative to conventional tobacco. Although initially believed to be a safer option than tobacco¹⁻⁵, many studies have shown otherwise¹⁻⁵. The vapor produced by e-cigarettes contains toxic and carcinogenic chemicals, albeit at lower levels than tobacco smoke. Additionally, e-cigarette devices may also contain additional ingredients that are toxic and inhaled directly into the lungs¹⁻⁵.

Tobacco and e-cigarettes are two of the most common forms of nicotine consumption today. However, there is significant controversy about which is more harmful to health. Recently, the DNA of the epithelial cells of the oral cavity of 72 tobacco users, vapers and non-consumers was analyzed, finding that those exposed to smoking and vaping had significantly higher levels ($p < 0.005$) of DNA damage between vapers and smokers⁶. It has been known for many years that the consumption of conventional tobacco and electronic cigarettes, depending on their exposure time and composition, causes various cellular, cytogenetic and DNA damage¹⁻¹¹. In a previous *in silico* study¹ we identified 50 compounds in e-cigarettes with possible carcinogenic effects and genes reported to be deregulated in the oral epithelium of e-cigarette users. The most important health risks include respiratory tract irritation, eyes and skin, with a 50% incidence and 10% cytotoxicity. The types of cancer with the highest risk identified are ovary, uterus, bladder, lung, esophagus and stomach. Additionally, in a previous study that we carried out in smokers¹², we found an increase in chromosomal damage and telomeric associations (TA), a sign of predisposition of cells to genetic instability reflected in chromosomal damage and DNA breaks. We found that the group exposed to tobacco showed a higher percentage of TA than the control group ($p < 0.001$).

Currently, evidence has accumulated that the vapors of electronic cigarettes alter cell functions and DNA itself. According to the WHO classification, ENDS (Electronic Nicotine Delivery Systems) and conventional cigarettes share some 27 chemicals that have been proven to be harmful to DNA. In our previous study, we found 234 chemical compounds: 150 for conventional cigarettes and 84 for e-cigarettes; we found 34 compounds in common with e-cigarettes and 50 present exclusively in e-cigarettes. Additionally, in 11% of the compounds, their effect on health issues has not been established yet, although half of these have been tested in animals as damaging to DNA¹.

Studies conducted exposing human and animal cells to vapors from conventional and e-cigarettes show an increase of more than three times in cell damage: decreased cell division and DNA degradation, increased apoptosis, shortening of telomeres, and genotoxicity. All of this is associated with a higher risk of cancer and aging⁶⁻²¹.

As far as we have been able to review, there is no study of DNA damage reflected in chromosomal breaks in individuals exposed to vaping with nicotine or Electronic Nicotine Delivery Systems (ENDS) and Electronic Non-Nicotine Delivery Systems (ENNDS). The present work evaluates the cytogenetic damage, according to the consumption of conventional cigarettes, vapers with nicotine and vapers without nicotine, compared with a non-smoking population.

MATERIALS AND METHODS

The experimental and quantitative study was conducted with 120 individuals divided into four groups aged 25 to 40. Each group underwent a survey and had clinical records taken. The study complies with all Ecuadorian regulations' legal and ethical aspects, and the informed consent was thoroughly discussed with the study participants.

The first group exposed to cigarettes included 30 smokers (>5 packs a year). The second group exposed to vaping by burning with nicotine (ENDS) included 30 individuals who consumed 3 to 6 milligrams of nicotine, with at least one year of exposure and no more than four years. The third group of non-nicotine-burning vapers (ENNDS) included 30 individuals with at least one year of exposure and no more than four years. The fourth group was the Control Group (CG), consisting of 30 healthy individuals of similar ages to those exposed to

vaping and tobacco and who were not in contact with vaping or tobacco or other known genotoxic potentials. Additionally, in the analysis of results, it will be considered whether or not there is variation depending on the sex of the individuals, men or women.

Peripheral blood T lymphocyte cultures were implemented using the standard technique: 5 milliliters of RPMI-1640 culture medium, supplemented with fetal bovine serum (10%), penicillin (10,000 IU/ml), streptomycin (10 mg/ml), L-glutamine (200 mM), HEPES buffer (1 mM) and phytohemagglutinin (1 mg/ml). Cultures were maintained for 48 h at 37°C. Harvest was carried out using standard techniques. Slides were air-dried and stained with 5% Giemsa. One hundred metaphases per individual were analyzed at 1000X magnification with an optical photomicroscope, recording chromosome fragility: breaks and gaps in one or both chromatids and radial figures. The chi-square test was used for statistical analysis with $p < 0.05$. This methodology has been validated in several studies^{12, 22, 23}.

RESULTS

The average age of the participants was 30 years (+/-10). Although the study did not include the classification of women and men, on average, men represented 70% of the sample. Despite this differentiation, we did not find significant statistical differences between the groups of men and women exposed to these toxins ($p < 0.05$). Table 1 shows the results of chromosomal fragility found in the four groups studied: CG, conventional smokers, vapers with nicotine (ENDS) and vapers without nicotine (ENNDS). The cytogenetic study showed breaks and gaps in one or both of the chromatids and occasional radial figures (<1%) (Figure 1), without finding other alterations such as dicentric chromosomes, rings or fragments. All exposed individuals presented highly significant statistical differences ($p < 0.001$) when compared to the unexposed control group (Table 2). When we compared the group of conventional smokers with vapers with nicotine (ENDS) and without nicotine (ENNDS), as well as when comparing the damage between the two types of vapers, we found no statistically significant differences between them ($p > 0.05$). All exposed groups present significant statistical differences compared to the unexposed 30 healthy individuals control group ($p < 0,001$).

Subsection

	Number of individuals (70% men)	% of chromosome damage: breaks, gaps and figures
Normal Controls	30	2
Smokers	30	16
Vapers without nicotine (ENNDS)	30	11
Vapers with nicotine (ENDS)	30	14

Table 1. Cytogenetic damage produced by conventional tobacco and vaping by burning electronic cigarettes, compared with the control group

Samples	CN/CN	CN/Smokers	CN/Vapers without nicotine	CN/Vapers with nicotine	Smokers/Vapers without nicotine	Smokers/Vapers with nicotine	vapers without nicotine/vapers with nicotine
p	>0,05	<0,001	<0,01	<0,001	>0,05	>0,05	>0,05

Table 2. Comparative statistical analysis was done using a Chi-square test and p-value between the exposed groups versus the control normal group (CN).

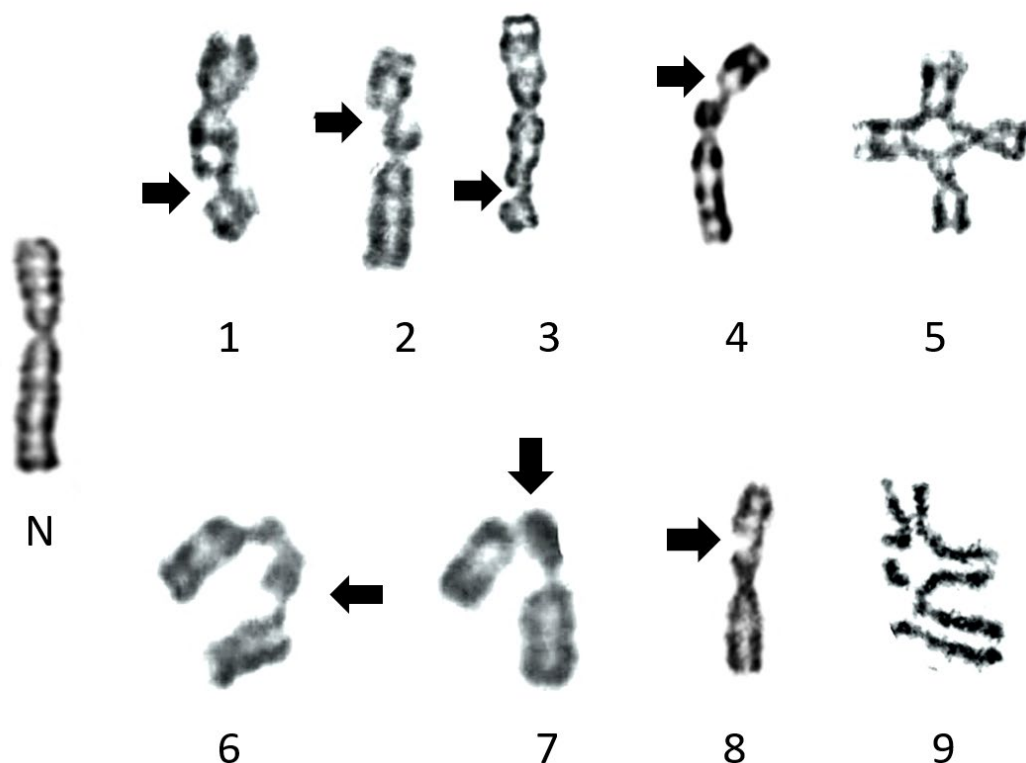


Figure 1. Variety of breaks in one and two chromatids on different chromosomes (1 to 4 and 6 to 8). Tetradial figure (5). Triradial figure (9). Normal Chromosome (N).

DISCUSSION

Our data show the chromosomal damage produced by traditional tobacco, vaping with nicotine and without nicotine, when compared with the control group, in the same way as conventional cigarettes when compared with electronic ones, do not show statistical differences; their behavior in the percentage of chromosome fragility is the same. Chromosomal damage in one or two chromatids defines vape compounds as genotoxic agents, as evidenced in several articles^{1-14, 16-21, 24}.

The significance of genotoxicity is widely highlighted. Conventional tobacco contains more than 70 chemicals known to be carcinogenic. The tar in tobacco smoke contains substances capable of causing mutations in cells¹⁻¹⁴. A study conducted by the National Cancer Institute found that smokers are up to 15 times more likely

to develop lung cancer than non-smokers¹⁴. Other studies have also shown that tobacco can increase the risk of cancer of the bladder, pancreas, mouth, pharynx, esophagus, larynx, kidney, liver, stomach, and cervix.^{3, 24} E-cigarettes are consumed as a safer alternative to conventional tobacco. However, studies show that nicotine-burning e-cigarettes can also cause genetic damage, using liquids containing nicotine, flavorings, and others such as formaldehyde and acetaldehyde, known to cause DNA damage and increase the risk of cancer^{1, 9-14, 16-21}.

Moreover, it is documented that vapers have higher levels of toxic chemicals in their urine than non-smokers and conventional cigarette smokers²⁵ or have more significant amounts of DNA damage in mouth cells compared to non-smokers¹⁸. There is evidence that e-cigarettes can damage lung function and cause inflammation in lung tissues and liver damage^{26, 27}. Our data reinforce the criteria for genetic damage from exposure to both types of cigarettes, conventional and e-cigarettes.

It has been shown that the damage of e-cigarette vapor depends on the power of the vaping system. Powers from 18 to 30 Watts produce an increase in DNA damage evaluated by the comet assay, the micronucleus test and the *Pig-a* gene mutation assay, depending on whether the exposure is subacute (4 days), subchronic (3 months) and chronic. (6 months)²⁶. The high-power e-cigarette and a conventional cigarette induced oxidative damage to DNA in the lungs and liver of exposed mice²⁶. Our data agrees with these chronic exposure results since the individuals analyzed in this study were exposed between 6 months and 4 years.

Cytogenetic damage from conventional and e-cigarettes depends on the amount and type of chemicals present in the smoke and vapor¹⁻²¹. Vapes do not contain tar, carbon monoxide, or other harmful chemicals found in conventional tobacco smoke. However, the e-liquids used in e-cigarettes contain other chemicals that can harm health^{1, 19, 26, 27}.

The genotoxic action is demonstrated in studies of DNA alterations. The results of Tommasi et al. (2023), detected significantly higher levels of DNA damage in both vapers and smokers ($p < 0.005$), compared to non-users ($p = 0.020$)⁶, data very similar to our study, which shows highly significant differences between smokers and vapers, compared to the control group.

In the study by Tommasi et al., vapers who used sweet, mint/menthol, and fruit-flavored e-liquids showed higher levels of DNA damage than the control group⁶. Although our study did not analyze these vaping characteristics, it is legitimate to think that chromosomal damage has the same characteristic if we compare controls versus users, which has been widely documented in other studies^{1-14, 16-27}.

In our study, as found in other publications^{6, 26, 27}, the nicotine content of vaping liquids did not predict DNA damage. We evaluated e-cigarettes by burning with at least 3 mg or more of nicotine per load, evidencing cytogenetic damage.

Additional studies have shown, including with adolescents and at the prenatal level^{25, 28}, that while nicotine levels in e-cigarettes can vary, just because the vapors do not contain the same chemicals found in conventional cigarettes does not mean they are safe.

Moreover, in a previous study¹, in which we reviewed the exclusive chemical compounds of e-cigarettes through in silico selection and which included articles that reported chemical composition and risks associated with health, we detected that, of the chemical compounds classified according to the code According to the "Chemical Abstracts Service"²⁹ for chemical substances, at least 50 dangerous compounds are present in e-cigarettes.

In addition, the potential carcinogenic effects of vaping are related to the deregulation of epithelial genes in 33 tumors; at least 878 genes published and analyzed in silico are associated with this deregulation in e-cigarette users³⁰.

According to our data from the present study and previous reports^{1, 11}, we conclude that there is actual damage to chromosomes associated with consuming conventional tobacco, ENDS and ENNDS. According to the last findings¹, it could additionally be associated with 11% of new compounds found in e-cigarettes, apart from the 123 compounds typical of conventional cigarettes, 50 typical of e-cigarettes and 34 common to both. This translates into an increased carcinogenic risk that would affect individual health, evidenced in chromosome breaks, since they produce irritation of the respiratory tract, eyes and skin with a 50% higher incidence in consumers, as well as a 10% evidenced by cytotoxic effects, and more significant risks of ovarian, uterine, bladder, lung, esophagus and stomach^{1, 11}.

Although various studies suggest that vaping may be less harmful than conventional cigarettes, there has not been enough long-term research to assess the health risks associated with vaping entirely. Furthermore, some studies suggest that the use of e-cigarettes may be a step towards conventional tobacco consumption and not an effective alternative¹³.

Regarding the impact on public health, some studies^{5, 7, 8, 14, 15, 20} analyze the relationship between the use of e-cigarettes and the reduction of tobacco consumption. The results showed that although some people may use e-cigarettes as a tool to give up tobacco smoking, it is also common for people who have never smoked to start using them, which can tempt new smokers with their respective genotoxic and carcinogenic risks.

The study has some limitations, such as the variety of vaping products, the concentrations of vaping liquids, and the quality of products used for vaping. This is a current public health problem with so many brands and types of vapes available, which determines that there must be controls and standardization of vaping products. Given our data, the genotoxic risk should be evaluated more broadly in vaper populations. More excellent controls on marketing and consumption will undoubtedly be proposed, as well as education campaigns about the dangers of exposure to vaping products.

CONCLUSIONS

According to our data, electronic cigarettes, like traditional tobacco, are shown to be genotoxic, evaluated by chromosomal studies for fragility. Although electronic cigarettes without nicotine produce less chromosome damage, when compared to electronic cigarettes that do contain nicotine, the statistical differences show that they are equally genotoxic ($p < 0.01$). Electronic cigarettes for vaping should be considered genotoxic whether they contain nicotine or not; the results, when compared with the control population, demonstrate this ($p < 0.001$). A more significant number of studies and analyzed individuals are needed to evaluate the real genotoxicity of electronic cigarettes and to carry out educational and public health campaigns on the risks of their consumption.

Author Contributions:

All authors contributed equally to the research, both in the laboratory analysis and in the construction of this article.

Funding: This research received no external funding

Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Institutional Ethics Committee of Universidad UTE y Ministerio de Salud.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: This section details where data supporting reported results can be found, including links to publicly archived datasets analyzed or generated during the study. Please refer to the suggested Data Availability Statements in the "Bionatura Research Data Policies" section at <https://www.revistabionatura.com/policies.html>. You might exclude this statement if the study did not report any data.

Acknowledgments: Acknowledgments to the Committee on Human Research (CISH) No. 2018-226E and Ministry of Public Health MSP-DIS-2019-0283-O for approval of this study.

Conflicts of Interest: The authors declare no conflict of interest.

REFERENCES

1. Armendáriz-Castillo, I.; Guerrero, S.; Vera-Guapi, A.; Cevallos-Vilatuña, T.; García-Cárdenas, J.M.; Guevara-Ramírez, P.; López-Cortés, A.; Pérez-Villa, A.; Yumiceba, V.; Zambrano, A.K.; Leone, P.E.; Paz-y-Miño, C. Genotoxic and carcinogenic potential of compounds associated with electronic cigarettes: A systematic review. *BioMed Res Int.* 2019; 1386710. DOI: 10.1155/2019/1386710.
2. National Academies of Sciences, Engineering, and Medicine. *Public Health Consequences of E-Cigarettes*. Washington, DC: The National Academies Press. 2018. [Internet]. [Consulted Jul 2023]. Available in: <https://www.nap.edu/catalog/24952/public-health-consequences-of-e-cigarettes>
3. Centers for Disease Control and Prevention (CDC). *Health Effects of Cigarette Smoking*. 2021. [Internet]. [Consulted Jul 2023]. Available in: https://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/index.htm
4. US Department of Health and Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. 2014. [Internet]. [Consulted Jul 2023]. Available in: <https://www.ncbi.nlm.nih.gov/books/NBK179276/>
5. World Health Organization (WHO). *Tobacco*. 2021. [Internet]. [Consulted Jul 2023]. Available in: <https://www.who.int/news-room/fact-sheets/detail/tobacco>
6. Tommasi, S.; Blumenfeld, H.; Besaratinia, A. Vaping Dose, Device Type, and E-Liquid Flavor are Determinants of DNA Damage in Electronic Cigarette Users. *Nicotine Tob Res*, 2023. [Internet]. [Consulted Jul 2023]. Available in: <https://doi.org/10.1093/ntr/ntad003>.
7. Environmental Health Perspectives. *Metals in emissions of electronic cigarettes: results from a risk assessment*. 2018. [Internet]. [Consulted Jul 2023]. Available in: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5936451/>
8. Levy, D.T.; Yuan, Z.; Li, Y.; Prevalence and Characteristics of E-Cigarette Users in the United States. *Int J Environ Res Public Health*, 2017, 14(10):1200. DOI: 10.3390/ijerph14101200
9. Farsalinos, K.E.; Poulas, K.; Voudris, V.; Le Houezec, J. Electronic cigarette use in the European Union: analysis of a representative sample of 27 460 Europeans from 28 countries. *Addiction*, 2016, 111:2032–40. <https://doi.org/10.1111/add.13506>.
10. Lee, H.W.; Park, S.H.; Weng, M.W.; Wang, H.T.; Huang, W.C.; Lopor, H.; Tang, M.S. E-cigarette smoke damages DNA and reduces repair activity in mouse lung, heart, and bladder as well as in human lung and bladder cells. *Proc Nat Acad Sci*, 2018, 115(7), E1560-E1569. <https://doi.org/10.1073/pnas.171818511>
11. Tsai, J.C.; Saad, O.A.; Magesh, S.; Xu, J.; Lee, A.C.; Li, W.T.; Chakladar, J.; Fuster, M.M.; Chang, E.Y.; Wang-Rodriguez, J.; Ongkeko, W.M. Tobacco Smoke and Electronic Cigarette Vapor Alter Enhancer RNA Expression That Can Regulate the Pathogenesis of Lung Squamous Cell Carcinoma. *Cancer (Basel)*. 2021, 13(16): 4225. DOI: 10.3390/cancers13164225
12. Paz-y-Miño, C.; Pérez, J.C.; Dávalos, V.; Sánchez, M.E.; Leone, P.E. Telomeric associations in cigarette smokers exposed to low levels of X-rays. *Mutat Res.* 2001, 490(1), 77-8016. [https://doi.org/10.1016/S1383-5718\(00\)00156-X](https://doi.org/10.1016/S1383-5718(00)00156-X)
13. Barrington-Trimis, J.L.; Samet, J.M.; McConnell, R. Flavorings in Electronic Cigarettes An Unrecognized Respiratory Health Hazard? *JAMA*, 2014, 312(23), 2493-2494. DOI: 10.1001/jama.2014.14830
14. National Cancer Institute (NCI). *Smoking and Cancer*. 2021. Recuperado de <https://www.cancer.gov/about-cancer/causes-prevention/risk/tobacco/smoking-fact-sheet>

15. 15. US Food and Drug Administration (FDA). Vaporizers, E-Cigarettes, and other Electronic Nicotine Delivery Systems (ENDS). 2021. [Internet]. [Consulted Jul 2023]. Available in: <https://www.fda.gov/tobacco-products/products-ingredients-components/vaporizers-e-cigarettes-and-other-electronic-nicotine-delivery-systems-ends>
16. 16. Alexandrof, L.B.; Ju, Y.K.; Haase, K.; Van Loo, P.; Martincorena, I.; Nik-Zainal, S.; Totoki, Y.; Fujimoto, A.; Nakagawa, H.; Shibata, T.; Campbell, P.J.; Vineis, P.; Phillips, D.H.; Stratton, M.R. Mutational signatures associated with tobacco smoking in human cancer. *Science*, 2016, 354(6312), 618-622. DOI: 10.1126/science.aag0299
17. 17. Kaur, G.; Pinkston, R.; Mclemore, B.; Dorsey, W.C.; Batra, S.K.; Singh, A.P. Immunological and toxicological risk assessment of e-cigarettes. *Eur Respir Rev*, 2018, 27(147), 170119. DOI: 10.1183/16000617.0119-2017
18. 18. Lee, H.W.; Park, S.H.; Weng, M.W.; Wang, H.T.; Huang, W.C.; Lepor, H.; Wu, X.R.; Chen, L.C.; Tang, M.S. E-cigarette smoke damages DNA and reduces repair activity in mouse lung, heart, and bladder as well as in human lung and bladder cells. *Proc Natl Acad Sci*, 2018, 115(7), E1560-E1569. DOI: 10.1073/pnas.1718185115
19. 19. Park, J.Y.; Kim, H.J.; Kim, H.J. The effects of cigarette smoke on immune responses and the immune system in respiratory tissues. *Clin Exp Otorhinolaryngol*, 2018, 11(2), 69-74. DOI: 10.21053/ceo.2017.00833
20. 20. Zhang, Y.; Sumner, W.; Chen, D.R. In vitro particle size distributions in electronic and conventional cigarette aerosols suggest comparable deposition patterns. *Nicotine Tob Res*, 2013, 15(2), 501-508. DOI: 10.1093/ntr/nts165
21. 21. Zhang, Y.; Sumner, W.; Chen, D.R. In vitro exposure systems and dosimetry assessment tools for inhaled tobacco products: Workshop proceedings, conclusions and paths forward for in vitro model use. *Altern Lab Anim*, 2017, 45(4), 195-213. DOI: 10.1177/026119291704500305
22. 22. Galloway, S.M.; Aardma, M.J.; Ishidate, M.; Ivett, J.L.; Kirkland, D.J.; Morita, T.; Mosesso, P.; Sofuni, T. Report from working group on in vitro test for chromosomal aberrations. *Mutat Res*, 1994, 312(1994) 241–261. DOI: 10.1016/0165-1161(94)00012-3
23. 23. Carrano, A.V.; Natarajan, A.T. Considerations for population monitoring using cytogenetic techniques, in: *Proceedings of the International Commission for Protection Against Environmental Mutagens and Carcinogens*, Publication no.14, *Mutat Res*, 1998, 204, 379–406. DOI: 10.1016/0165-1218(88)90036-5
24. 24. Natarajan, A.T. Origin and significance of chromosomal alterations, in: G. Obe (Ed.), *Mutation in Man*, Springer, Berlin, Germany, 1988, pp. 156–176
25. 25. Rubinstein, M.L.; Delucchi, K.; Benowitz, N.L.; Ramo, D.E. Adolescent exposure to toxic volatile organic chemicals from e-cigarettes. *Pediatrics*, 2018, 141(4), e20173557. DOI: 10.1542/peds.2017-3557
26. 26. Platel, A.; Dusautoir, R.; Gwenola, R.; Kervoaze, G.; Dourdin, G.; Gateau, E.; Talahari, S.; Huot, L. Simar, S. Ollivier, A. Laine, W. Kluza, J. Gosset, P. Garçon, G. Anthérieu, S.; Guidice, J-M.; Nesslany, F. Comparison of the in vivo genotoxicity of electronic and conventional cigarettes aerosols after subacute, subchronic and chronic exposures. *J. Hazard Mater*, 2022, 5, 423. DOI: 10.1016/j.jhazmat.2021.127246
27. 27. Moses, E.; Wang, T.; Corbett, S.; Jackson, G.R.; Drizik, E.; Perdomo, C.; Perdomo, C.; Kleerup, E.; Brooks, D.; O'Connor, G.; Dubinett, S.; Hayden, P.; Lenburg, M.E.; Spira, A. Molecular Impact of Electronic Cigarette Aerosol Exposure in Human Bronchial Epithelium. *Toxicol Sci*, 2017, 155(1), 248–257. DOI: 10.1093/toxsci/kfw198
28. 28. Römer, P.; Gómez Putzer, A.; Kemmerich, R.; Mathes, B. Effects of Prenatal Electronic Cigarette Exposure On Foetal Development: a Review of the Literature. *GebFra Science, Review/Übersicht*, 2021, 81(11),1224-1237. DOI: 10.1055/a-1524-5155
29. 29. CAS, "Chemical Abstracts Service". American Chemical Society, 2023, <https://www.cas.org/>.
30. Hoadley, K.A.; Yau, C.; Hinoue, T.; Wolf, D.M.; Lazar, A.J.; Drill, E.; Shen, R.; Taylor, A.M.; Cherniack, A.D.; Thorsson, V.; Akbani, R.; Bowlby, R.; Wong, C.K.; Wiznerowicz, M.; Sanchez-Vega, F.; Robertson, A.G.; Schneider, B.G.; Lawrence, M.S.; Noushmehr H.; Malta, T.M. Cancer Genome Atlas Network. Stuart, J. M. Benz, C. C. Laird P, W. Cell-of-Origin Patterns Dominate the Molecular Classification of 10,000 Tumors from 33 Types of Cancer. *Cell*, 2018, 173(2) 291–304,e6, <https://doi.org/10.1016/j.cell.2018.03.022>

Received: December 14, 2023/ **Accepted:** February 25, 2024 / **Published:** March 15, 2024

Citation: César Paz-y-Miño, Ligia Ocampo, María Eugenia Sánchez, Paola E. Leone. Evaluation of DNA damage through cytogenetic approach in smokers and vapers with and without nicotine compared with control group. Bionatura journal 2024; 1 (1) 75. <http://dx.doi.org/10.21931/BJ/2024.01.01.75>

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