Analysis of DNA damage by e-cigarettes exposure in a youth population

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The global consumption of electronic cigarettes has significantly increased, particularly among the youth population, while the genetic damage associated with their use remains poorly understood. Consequently, this study aimed to evaluate the genotoxic effects and susceptibility related to the consumption of both electronic and conventional cigarettes.

A total of 156 university students were categorized into control, vaper, and smoker groups. Serum cotinine levels were measured as a biomarker for nicotine exposure. DNA damage was assessed using the comet assay (measuring the percentage of tail DNA and tail length) and a competitive ELISA for detecting 8-hydroxy-2'-deoxyguanosine (8-OHdG). The variant rs16969968 (G>A) of the *CHRNA5* gene was identified, which is associated with increased susceptibility to nicotine consumption frequency. Null genotypes of *GSTM1* and *GSTT1*, which are linked to reduced xenobiotic detoxification, were also evaluated.

Cotinine levels confirmed the classification of groups. The percentage of tail DNA and tail length were significantly higher in vapers (7.33 ± 2.01 and 23.09 ± 7.98) and smokers (7.65 ± 2.67 and 24.59 ± 11.03) compared to controls (4.64 ± 1.79 and 14.00 ± 5.88). The 8-OHdG levels were elevated in vapers (124.66 ± 53.86) and smokers (114.05 ± 40.86) compared to controls (96.51 ± 42.14). The *CHRNA5* polymorphism showed a tendency toward higher cigarette/puff consumption. Additionally, the null genotypes of *GSTM1* and *GSTT1* were associated with increased tail length and 8-OHdG levels, respectively.

These findings suggest that electronic cigarettes induce clastogenic damage to DNA and that certain genetic variants may increase individual susceptibility. Further research is needed to understand their impact better and inform policies countering the perception of e-cigarettes as harmless.

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