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Thirdhand smoke causes DNA damage in human cells.

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Abstract

Exposure to thirdhand smoke (THS) is a newly described health risk. Evidence supports its widespread presence in indoor environments. However, its genotoxic potential, a critical aspect in risk assessment, is virtually untested. An important characteristic of THS is its ability to undergo chemical transformations during aging periods, as demonstrated in a recent study showing that sorbed nicotine reacts with the indoor pollutant nitrous acid (HONO) to form tobacco-specific nitrosamines (TSNAs) such as 4-(methylnitrosamino)-4-(3-pyridyl)butanal (NNA) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). The goal of this study was to assess the genotoxicity of THS in human cell lines using two in vitro assays. THS was generated in laboratory systems that simulated short (acute)- and long (chronic)-term exposures. Analysis by liquid chromatography-tandem mass spectrometry quantified TSNAs and common tobacco alkaloids in extracts of THS that had sorbed onto cellulose substrates. Exposure of human HepG2 cells to either acute or chronic THS for 24h resulted in significant increases in DNA strand breaks in the alkaline Comet assay. Cell cultures exposed to NNA alone showed significantly higher levels of DNA damage in the same assay. NNA is absent in freshly emitted secondhand smoke, but it is the main TSNA formed in THS when nicotine reacts with HONO long after smoking takes place. The long amplicon-quantitative PCR assay quantified significantly higher levels of oxidative DNA damage in hypoxanthine phosphoribosyltransferase 1 (HPRT) and polymerase β (POLB) genes of cultured human cells exposed to chronic THS for 24h compared with untreated cells, suggesting that THS exposure is related to increased oxidative stress and could be an important contributing factor in THS-mediated toxicity. The findings of this study demonstrate for the first time that exposure to THS is genotoxic in human cell lines.

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