

Butter flavoring in microwave popcorn, thought safe for food industry workers, is respiratory hazard

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The ingredient 2,3-pentanedione (PD), used to impart the flavor and aroma of butter in microwave popcorn, is a respiratory hazard that can also alter gene expression in the brain of rats. Manufacturers started using PD when another butter flavoring, diacetyl, was found to cause bronchiolitis obliterans, a life-threatening and nonreversible lung disease in workers who inhaled the substance. New research on PD with implications for "popcorn workers' lung" is published in *The American Journal of Pathology* and indicates that acute PD exposure has respiratory toxicity which is comparable to diacetyl in laboratory animals.

"Our study demonstrates that PD, like diacetyl, damages airway epithelium in laboratory studies. This finding is important because the damage is believed to be the underlying cause of bronchiolitis obliterans," says lead investigator Ann F. Hubbs, DVM, PhD, DACVP, Health Effects Laboratory Division of the National Institute for [Occupational Safety and Health](#), [Centers for Disease Control and Prevention](#), Morgantown, WV. "Our study also supports established recommendations that flavorings should be substituted only when there is evidence that the substitute is less toxic than the agent it replaces."

The study included groups of rats exposed for six hours to different concentrations of PD, a comparable concentration of diacetyl, or filtered air. Since the investigators observed signs of delayed toxicity, they

exposed additional rats to PD, and further microscopically examined the brains, lungs, and nasal tissues from these rats 0-2 hours, 12-14 hours, and 18-20 hours after exposure. investigators then evaluated changes in [gene expression](#) in discrete [brain regions](#).

The investigators found respiratory epithelial injury in the upper nose, comparable to that caused by diacetyl that progressed through 12 to 14 hours post-exposure. They also found that PD exposure caused necrosis and apoptosis in the olfactory neuroepithelium and activation of caspase 3, a protein that plays a role in cell death, in axons of olfactory nerve bundles. Signs consistent with neurotoxicity included increased expression of the inflammatory mediators, interleukin-6 and nitric oxide synthase-2, as well as decreased expression of vascular endothelial growth factor A in the olfactory bulb, striatum, hippocampus, and cerebellum.

"Our study is a reminder that a chemical with a long history of being eaten without any evidence of toxicity can still be an agent with respiratory toxicity when appropriate studies are conducted," says Dr. Hubbs. "It suggests several intriguing potential mechanisms for the toxicity of inhaled volatile α -diketones, reveals mRNA changes in the brain, documents olfactory neurotoxicity, and clearly demonstrates that the remarkable airway [toxicity](#) of diacetyl is shared with its close structural relative, PD."

More information: "Respiratory and Olfactory Cytotoxicity of Inhaled 2,3-Pentanedione in Sprague-Dawley Rats," by A.F. Hubbs, A.M. Cumpston, W.T. Goldsmith, et al.. It appears in *The American Journal of Pathology*, Volume 181, Issue 3 (September 2012) dx.doi.org/10.1016/j.ajpath.2012.05.021

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