

# “Popcorn worker’s lung” in Britain in a man making potato crisp flavouring

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## ABSTRACT

The case history is presented of a patient who developed “popcorn worker’s lung” following exposure to 2,3-butanedione (diacetyl). Other similar cases are reviewed, and it is concluded that “food flavourer’s lung” may be a more suitable diagnostic label.

Evidence that bronchiolitis obliterans could result from inhaled popcorn flavouring ingredients was first published in 2002.<sup>1,2</sup> Its recognition in eight former workers from a single American factory packaging popcorn for microwave use led to the appellation “popcorn worker’s lung”. It was sufficiently severe in four cases that lung transplantation was considered. Investigations by the National Institute for Occupational Safety and Health showed that the prevalence of fixed airway obstruction among continuing employees was 3.3-fold that expected; that bronchiolitis obliterans could be detected in workers from four of five subsequently studied microwave popcorn plants; and that a prominent ingredient providing a butter taste (the oily organic chemical 2,3-butanedione (diacetyl)) could cause airway necrosis when inhaled by laboratory animals.

Diacetyl has also been used in a factory in Britain as a flavouring ingredient for potato crisps, and a member of the workforce developed, subacutely, fixed airway obstruction consistent with bronchiolitis obliterans. His case illustrates the likelihood that popcorn worker’s lung is occurring in other countries than the USA and in other settings than the production of microwave popcorn. It illustrates too that not all cases of fixed airway obstruction are the result of emphysema and smoking.

## CASE REPORT

The patient was a 36-year-old man who had never smoked and had no relevant family history. He kept dogs and a cat and for 4 years had spent about 30 min daily in pig husbandry. From 2004 he worked in a factory producing food flavourings, principally for potato crisps/chips, having worked previously as a baker/food processor, warehouse packer/dispatcher, shop stock controller, driver and pest controller. A routine health check in May 2004 was unremarkable, and spirometry in November 2004 was normal. In mid to late summer 2005 he developed undue exertional breathlessness which was moderately disabling when first reported in August 2005. It showed no obvious variability, and so appeared unrelated to his animal contacts. He was not aware of wheeze or cough, but had chest tightness. He noted no benefit from the use of

asthma medications, including oral steroid, and a serum  $\alpha_1$ -antitrypsin measurement was normal.

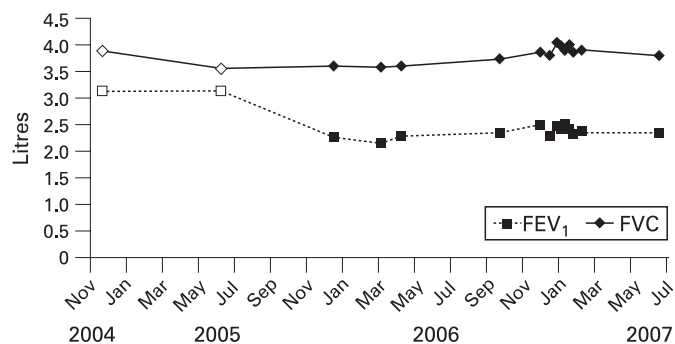
He worked irregularly with diacetyl, which was used at various concentrations in a number of flavouring products. During a particular work shift in April 2005 he used diacetyl at the highest concentration in a product that was needed urgently. Because of this, the heated diacetyl-containing mixture was managed unusually in unsealed containers and, because he substituted for an absent workmate, he worked with diacetyl (and recognised its odour) for an unusually long period—several hours compared with the usual 30 min. During the last few hours of this shift his eyes became sore and reddened, and a sticky conjunctival secretion developed soon after. He was treated topically with steroid and antihistamine, but resolution required several days. He had no respiratory symptoms, and spirometry remained normal when remeasured in June 2005.

By the time of his evaluation by a respiratory physician in December 2005 his breathlessness was stable, and there was no abnormality on physical examination. Spirometry gave 2.25/3.60 (respectively 64% and 87% predicted), and both the carbon monoxide transfer factor and transfer coefficient exceeded predicted values. A flow-volume study was suggestive of small airway disease, with flow at 75%, 50% and 25% of total lung capacity being reduced disproportionately to the reduction in forced expiratory volume in 1 s (to 41%, 33% and 27% of predicted, respectively, vs 64%). Flow between 75% and 25% of total lung capacity was 30% of predicted. Plain chest radiographs and CT scans (inspiratory and expiratory) were unremarkable, and skin prick tests with common allergens were negative. Figure 1 shows the serial spirometric measurements. From December 2005 to July 2007 there was no change, nor any change in exercise capacity.

Ongoing exposure to diacetyl was restricted from December 2005 and he was placed on permanent “sick leave” in December 2006 before ceasing employment in March 2007. None of these changes was associated with any change in disability or spirometry.

## DISCUSSION

The development, over a few months, of fixed airway obstruction of moderate severity is most readily attributed to bronchiolitis obliterans, and exposure to diacetyl provides the most likely explanation. Although no other examples had been published from outside the USA by the time this case came to light, a paper has very recently described four Dutch cases from a chemical plant



**Figure 1** Spirometric measurements of patient between November 2004 and July 2007. FEV<sub>1</sub>, forced expiratory volume in 1 s; FVC, forced vital capacity. Bronchodilator inhalation produced no more than a minor and inconsistent improvement. NB. The initial measurements of November 2004 and June 2005 were not obtained from an accredited lung function laboratory and may be underestimated.

making diacetyl.<sup>3</sup> “Popcorn worker’s lung” is consequently more widespread than is generally supposed, and is occurring in settings unrelated to popcorn. “Food flavourer’s lung” might be a more appropriate diagnostic label.

It is tempting with the present case to speculate whether the unusual work shift of April 2005, with high diacetyl exposure, played a critical role. Diacetyl is known to pose a risk of ocular toxicity, and American experience has shown that bronchiolitis obliterans may progress for some months after exposure ceases. However, spirometry in June 2005 remained normal, and symptoms were not recognised for a further 1–2 months.

No evidence at present suggests that diacetyl is hazardous when ingested, but the possibility is not lightly dismissed. A number of orally administered drugs are known to cause inflammatory/fibrotic reactions at the bronchiolar level, similar to paraquat and a notorious batch of adulterated cooking oil.<sup>4</sup> More relevant is the ingestion, as an appetite suppressant, of leaf extracts from the Asian shrub *Sauropus androgynus* which also induces life-threatening bronchiolitis obliterans.<sup>5</sup> The inhalation of acramin (a garment dye) and fragmented synthetic polymer fibres completes a recently observed causal spectrum for the development of subacute bronchiolar disease.<sup>6,7</sup> With causes of such a disparate nature—both inhaled and ingested—it may be prudent to look out for others.

**Competing interests:** None.

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