

## EDITORIAL

# Colophony hypersensitivity revisited

Colophony (or rosin as it is sometimes called) is a common cause of occupational sensitization. In the UK it ranks in the top five causes of occupational asthma [1] and the top 10 causes of contact dermatitis. It is also a cause of rhinitis and eye irritation, and perhaps of headache and allergic alveolitis [2–4]. It is an ‘old fashioned’ small molecular weight chemical used in the early days of the plastics industry as a basis for semisynthetic resins. It is derived from the resin of pine trees, either by direct tapping of the sap, by extraction from dead stumps (wood rosin), or from tall oil left over after paper manufacture (tall oil rosin); the latter two products tending to have more oxidized resin acids than native colophony. Colophony consists of a variable mixture of resin acids, some of which are relatively unstable, and a neutral fraction. Occupational asthma usually results from inhalation of the fume from heated colophony in soft soldering fluxes [5], or occasionally in hot melt glues [6] or depilatory mixtures [7]. However, cases have occurred in a chemical worker with colophony heated to 90°C [8] and from unheated colophony dust [9]. Colophony or colophony derivatives have caused occupational asthma when added as a deodorant to coolant oils [10] and floor cleaning products [11].

The occupational asthma resulting from colophony exposure usually has the features of a sensitizing reaction, with a latent interval from first exposure to first symptom (averaging 6.5 years in one heavily exposed group [12]), only a proportion of similarly exposed workers are affected, and asthma is induced in affected workers with exposures which have no effect in asymptomatic exposed workers [13]. There is only a weak relationship between non-specific reactivity and the concentration of colophony required to elicit an asthmatic reaction [13], again pointing to some mechanism requiring specificity. Colophony fumes at levels found in some soldering operations can sometimes produce reactions which are probably irritant in some individuals who have no interval between first exposure and first symptom. Colophony has induced asthma in previously unexposed hyperresponsive asthmatics. Colophony and plicatic acid have both damaged pulmonary epithelial cells following direct application [14].

Some small molecular weight chemicals elicit specific immunoglobulin (Ig) E to hapten–protein conjugates, which can be infrequent (about 20% with isocyanates [15]), moderately prevalent (about 40% with plicatic acid, the principal resin acid from Western Red Cedar [16]), or usual (such as with acid anhydrides [17,18]). In the first two situations workers with and without specific IgE are

clinically indistinguishable and appear to have the same prognosis. So far there are no reports where specific IgE to colophony conjugates have been described.

In this issue Elms and colleagues speculated that the lack of colophony IgE antibodies was due to inappropriate conjugates. They investigated the *in vitro* potential of colophony to interact with immune cells and the cells response to colophony [19]. Colophony at 5–50 µg/mL was incubated with various cell lines. Cells with neutrophil or monocyte differentiation showed increased intracellular peroxide after a 1-min colophony incubation, suggesting that reactive oxygen species were being induced which could oxidize colophony *in vivo* to resin acid epoxides and hydroperoxides. These epoxides are thought to be implicated in colophony contact dermatitis [20], and might be a hapten for IgE antibodies.

The control of a hazard causing occupational asthma requires the causative agent to be identified and a method devised for its measurement. Until recently the control standard for colophony fume was based on its aldehyde content which correlated poorly with fume concentration [5] and had many sources of bias (e.g. from off-gassing of particle board). A method has been developed for estimation of the resin acid content of soldering fume [21] and an exposure standard based on this assay introduced [22]. The ingredients are now in place for the control of one of the commoner causes of occupational sensitization.

## References

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