

Editorial

Occupational rhinitis and asthma due to metal salts

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In the current issue of *Allergy*, Cristaudo and coworkers publish interesting prevalence data on sensitization to platinum salts and respiratory symptoms in workers of a secondary industry (1). Metals are common causes of allergic sensitization and symptoms in the workplace, as reviewed (2). Among metals, several 'first long period of transitional elements', including nickel, chromium, cobalt, vanadium and zinc, have been incriminated. Second and third long periods of transitional elements include those metals that are the focus of the work by Cristaudo and colleagues: platinum, iridium, rhodium, and palladium. This article is relevant and worthwhile for various reasons. First, the authors compare sensitization to various metal salts of the second and third long periods of transitional elements to which the workers were exposed. Platinum salts were described as a cause of allergic sensitization and occupational asthma in the late 19th and early 20th centuries, as reviewed (3). Contrary to most other low-molecular-weight agents, an IgE-dependent mechanism has clearly been elucidated (4–6). Extensive epidemiologic studies in a refinery by Merget and coworkers have shown that platinum salts are highly allergenic (7). Cristaudo and coworkers confirm this by showing that platinum salts are a frequent cause of sensitization (14% incidence in their study). These authors further demonstrate that, among platinum salts that also include potassium and sodium hexachloroplatinate s, hexachloroplatinic acid is clearly the most allergenic. Indeed, all of the 22 sensitized subjects reacted to this salt. However, eight of these 22 subjects (36%) reacted to all platinum salts. Other second- and third-group elements (iridium, rhodium, palladium) were found to be very uncommonly allergenic: only two subjects reacted to both iridium and rhodium and one to iridium only, while all of the latter subjects also reacted to all platinum salts. Linnet and Hughes have also shown that tetraammine platinum dichloride in which the halide is present as an ion and not as a ligand coordinated to platinum, is not allergenic (8). The explanation for these enhanced allergenic properties of hexachloroplatinic acid

when compared with other platinum salts and other metal salts of the second- and third-group elements is hypothetical, although most likely related to the molecular structure of the agent (9, 10). It is also interesting that only two subjects reacted to patch tests, whereas all but one showed reactions to prick testing, further confirming that the reaction is IgE-mediated.

Secondly, this valuable contribution is a cross-sectional study carried out in a secondary industry that employs a relatively small group of workers. The incidence of sensitization is clearly as elevated as in the primary industry. Prevalences of sensitization and work-related symptoms are often higher in the secondary industry because preventive means and medical surveillance do not operate as efficiently in these workplaces as in large-scale production plants.

Thirdly, this study suggests, as other studies have clearly found, that cigarette smoking is a risk factor (11, 12). Most other causes of occupational asthma have not been associated with cigarette smoking. This further emphasizes the need to encourage workers in such workplaces to quit smoking by promoting smoking cessation programmes.

Finally, the study by Cristaudo and coworkers demonstrates that skin-prick testing is a highly valuable surveillance tool in the case of workers exposed to platinum salts. It is also a useful indicator of the validity of surveillance programmes in which workers are offered jobs with no exposure to platinum salts. Merget and coworkers have indeed shown that sensitization to platinum disappears when workers are no longer exposed (13).

Of the 22 subjects with skin reactivity to platinum salts, 18 had symptoms of rhinitis and 10 reported asthma, although not necessarily work-related. The prevalences of probable occupational rhinitis and asthma were 12 and 6.6%, respectively. These figures are comparable with prevalence figures generally reported in workers exposed to low-molecular-weight agents, i.e. approximately 5–10% for occupational asthma. In most prevalence studies, work-related rhinitis is more common than

occupational asthma (14). In the current study, bronchial responsiveness was not assessed; this would have been a further means, now more often used in epidemiological surveys, to confirm the asthmatic status. Using criteria of immunological reactivity along with bronchial hyperresponsiveness and symptoms would have indicated a stronger likelihood of occupational asthma (15).

Interventions are clearly warranted to reduce the burden of sensitization, rhinitis and asthma caused at the workplace. Reducing exposure levels should be a priority, as it has proved consistently effective. Newman-Taylor and coworkers have shown that intensity exposure

and HLA phenotype (HLA-DR6 increases the risk whereas HLA-DR3 is associated with reduced risk) interact in the case of sensitization to platinum (16). Moreover, early identification of workers who acquire sensitization and transfer to areas with reduced exposure may result in reversal of sensitization to platinum (13). Pre- and regular post-employment assessments of sensitization, bronchial responsiveness and symptomatic status should be carried out in these workers so as to provide incidence figures, more rarely evaluated than prevalence figures in the case of occupational asthma (17), in order to guide efficient interventions.

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