Occupational asthma due to chrome and nickel electroplating

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Abstract

Background – Exposure to chromium during electroplating is a recognised though poorly characterised cause of occupational asthma. The first series of such patients referred to a specialist occupational lung disease clinic is reported.

Methods – The diagnosis of occupational asthma was made from a history of asthma with rest day improvement and confirmed by specific bronchial provocation testing with potassium dichromate and nickel chloride.

Results – Seven workers had been exposed to chrome and nickel fumes from electroplating for eight months to six years before asthma developed. One subject, although exposed for 11 years without symptoms, developed asthma after a single severe exposure during a ventilation failure. This was the only subject who had never smoked. The diagnosis was confirmed by specific bronchial challenges. Two workers had isolated immediate reactions, one a late asthmatic reaction, and four a dual response following exposure to nebulised potassium dichromate at 1–10 mg/ml. Two of the four subjects were also challenged with nebulised nickel chloride at 0.1–10 mg/ml. Two showed isolated late asthmatic reactions, in one at 0.1 mg/ml, where nickel was probably the primary sensitising agent. Four workers carried out two hourly measurements of peak expiratory flow over days at and away from work. All were scored as having occupational asthma using OASYS-2. Breathing zone air monitoring was carried out in 60 workers from four decorative and two hard chrome plating shops from workers with similar jobs to those sensitised. No measurement exceeded the current occupational exposure standard for chromate or nickel, the mean levels of chromate exposure for jobs similar to those of the affected workers were 9–15 µg/m³.

Conclusion – Chrome used in electroplating is a potential cause of occupational asthma. Sensitivity to chrome in electroplaters may occur in situations where exposure levels are likely to be within the current exposure standards. There may be cross reactivity with nickel. Inhalation challenge with nebulised potassium dichromate solution is helpful in making the specific diagnosis where doubt exists.

Keywords: occupational asthma, electroplating, chrome, nickel, bronchial provocation testing.

Chromium is the major industrial contact allergen that causes dermatitis1 and is widely used in electroplating, dyes, leather tanning, and cement works. Single cases of asthma caused by chrome have been described since the nineteenth century and in the 1930s both Smith2 and Joules3 reported chromium induced asthma due to exposure at work. Since then there have been very few reports of respiratory symptoms in workers associated with electroplating.

Electroplating is the application of a metallic coating to articles using inorganic salts of metals such as chromium or nickel. In the electroplating process the gases released at the electrodes rise to the surface of the bath and generate a fine aerosol. Nickel electroplating is 95% efficient and gas generation is minimal whereas chromium electroplating is inefficient and 80–90% of the total energy used may be directed to the generation of potentially respirable aerosols of chromic acid. Epidemiological studies have suggested increased respiratory morbidity in electroplaters compared with galvanisers that is related to exposure to chromium.4 We report here our experience with chromium induced asthma in workers referred to a specialist occupational lung disease clinic over 10 years.

Methods

Subjects were identified at a specialist occupational lung disease clinic. The current report includes all those investigated by occupational type bronchial provocation testing over the last 10 years. The clinic has an additional six workers from the same industry who have not had challenge testing, but whose diagnosis is also likely to be occupational asthma from plating shop fumes. None were identified as part of epidemiological surveys.

Skin testing

Skin prick tests were performed using a range of common environmental solutions (Bencard, UK). Atopy was defined as a weal of 2 mm or
more than that caused by a diluent control
solution. Skin prick tests were also performed
with solutions of nickel chloride and potassium
dichromate at concentrations of 1 and 10 mg/
ml diluted in isotonic saline.

SERIAL PEAK FLOW MEASUREMENTS
For workers still exposed at the time of referral
serial readings of peak expiratory flow (PEF)
were recorded two hourly from waking until
sleeping using a mini-Wright peak flow meter
on days at and away from work. Workers were
instructed to record the best of three readings
reproducible within 20 l/min, and the records
were subsequently analysed using the OASYS-
2 analysis program.5

AIRWAY CHALLENGES
Airway challenges to potassium dichromate sol-
lutions (0.1 mg/ml) were performed on an
inpatient basis using a Wright nebuliser driven
with air at 8 l/min by a compressor unit. On
the day of challenge inhaled and oral broncho-
dilators were withheld but any inhaled cortico-
steroids were continued. After a stable baseline
period subjects inhaled the test solution during
tidal breathing initially for one minute. Forced
expiratory volume in one second (FEV1) was
measured using a dry bellows spirometer
(Vitalograph, Buckingham, UK) at 5 and 10
minutes thereafter. If the FEV1 fell by less than
10% the subject inhaled further two minute
doses up to a total of five minutes. Spirometric
tests were subsequently repeated every five
minutes for the first 30 minutes, every 10 min-
utes for the next 30 minutes, at 90 minutes, and
then at hourly intervals throughout the day.
If no early or late response was seen the
challenge was repeated on the following days
with solutions at concentrations of 1 mg/ml and
then 10 mg/ml. Nebulised saline was used as a
negative control and nickel chloride solution
as a second challenge in some cases. Subjects
were not aware of the challenge solution. In
order to control for both the acidity and the
tonicity of the solutions, a further control ex-
posure to potassium chloride solution at pH 3.8
was made in one subject.

AIR SAMPLING
As part of an epidemiological survey of elec-
troplaters in the West Midlands UK an ex-
tensive air monitoring programme was
undertaken in 60 workers from the larger elec-
troplating shops. All workers present on the
days of sampling (always Thursdays) took part
(86.5% of the total workforce). Two hour per-
sonal samples breathing zone air were collected
on a 25 mm glass fibre membrane with a pore
size of 0.8 μm mounted on a closed face seven
hole cassette with airflow of 2 l/min. Nickel
and chrome levels were measured by atomic
absorption spectroscopy using the Health and
Safety Executive method.6 The results for the
occupational categories corresponding to the
workers studied are reported.

Results
Details of the seven workers studied are shown
in table 1. All came from factories where both
chrome and nickel (or zinc in one case) were
used for plating or passivating. Six came from
factories where decorative plating was taking
place, and one (subject 4) from a factory hard
plating large objects. Three workers were em-
ployed as platers, working directly with the
plating baths, the other four were only in-
cidentally exposed within the same general en-
vironment.

In the first subject asthma started after an
unusual acute exposure when the extraction
fans on both chrome and nickel plating baths
failed, filling the factory with a visible cloud of
fume; he had been asymptomatic in this job
for the previous 11 years. In the others there
was a latent interval between first exposure
and the onset of symptoms ranging from eight
months to six years, with no unusual exposures
before symptoms developed. Of these, all but
two were current smokers and one out of six
was atopic. Two of the seven subjects had a
2 mm weal following prick testing with potas-
sium dichromate (10 mg/ml) and two had pos-
tive reactions to nickel chloride (10 mg/ml).

Five workers had significant (>20% fall from
baseline) immediate asthmatic reactions fol-
lowing exposure to potassium dichromate at
concentrations between 1 and 10 mg/ml. In
addition, four developed significant late asth-
matic reactions. One worker had a borderline
early reaction (subject 7) and one (subject 1)
a borderline late asthmatic reaction. Chal-
lenges to nickel chloride in concentrations of
0.1–10 mg/ml were performed in five workers
resulting in one borderline immediate reaction
and two late asthmatic reactions. In the first
subject the reaction was at 0.1 mg/ml for nickel
and 10 mg/ml for chrome, suggesting that the
principal sensitisation was to nickel; the others
had greater reactions to chrome. The pH of
the challenge solutions was measured for con-
centrations from 0.1 to 10 mg/ml. Nickel chlor-
ide had a pH ranging from 5.25 to 5.27,
whereas the pH of potassium dichromate de-
creased from 4.21 to 3.83 with increasing con-
centration. The last subject was challenged
with potassium chloride (10 mg/ml), with pH
adjusted to 3.83, with negative results (fig 1).

Serial PEF records during exposure were
available in four workers as indicated in table
2. All the PEF records had evidence of a work
related effect as determined by the OASYS-2
analysis. An OASYS-2 score of more than 2.51
indicated the presence of a significant work
related effect in the serial PEF record. The
symptoms in subject 1 were so severe on re-
turning to the work place that only a short
period of exposure (two days) could be tol-
erated. The PEF record from subject 1 and
part of the record from subject 4 are shown in
fig 2.

The results for the personal samples breath-
ing zone air for nickel and chromate are shown
in table 3. The results came from four plants
where decorative plating took place, and two
where hard plating occurred. Subject 4 worked
at one of the hard plating shops where air
Table 1  Demographic data and results of skin and specific bronchial challenge tests on seven workers with asthma due to chrome

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Occupation</th>
<th>Latent interval</th>
<th>Smoking history</th>
<th>FEV₁ (% predicted)</th>
<th>Atopy</th>
<th>Prick tests</th>
<th>FEV₁ (% fall from baseline)</th>
<th>FEV₁ (% fall from baseline)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A</td>
<td>34</td>
<td>M</td>
<td>Plater</td>
<td>Acute</td>
<td>None</td>
<td>96</td>
<td>Yes</td>
<td>Negative</td>
<td>4 mm</td>
<td>22 (10)</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>F</td>
<td>Assembler</td>
<td>Current</td>
<td>67</td>
<td>No</td>
<td>Negative</td>
<td>ND</td>
<td>23 (1)</td>
<td>0</td>
</tr>
<tr>
<td>3A</td>
<td>20</td>
<td>F</td>
<td>Jigger</td>
<td>Current</td>
<td>98</td>
<td>No</td>
<td>Negative</td>
<td>Negative</td>
<td>53 (10)</td>
<td>0</td>
</tr>
<tr>
<td>4A</td>
<td>58</td>
<td>M</td>
<td>Manager</td>
<td>Current</td>
<td>71</td>
<td>Yes</td>
<td>Negative</td>
<td>Negative</td>
<td>44 (1)</td>
<td>28</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>M</td>
<td>Plater</td>
<td>Ex-smoker</td>
<td>61</td>
<td>No</td>
<td>4 mm</td>
<td>Negative</td>
<td>5 (1)</td>
<td>43</td>
</tr>
<tr>
<td>6</td>
<td>48</td>
<td>M</td>
<td>Plater</td>
<td>Current</td>
<td>54</td>
<td>No</td>
<td>4 mm</td>
<td>27 (10)</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>7A</td>
<td>55</td>
<td>F</td>
<td>Packer</td>
<td>&lt;1</td>
<td>91</td>
<td>No</td>
<td>4 mm</td>
<td>17 (1)</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0 ND</td>
<td></td>
</tr>
</tbody>
</table>

ND = not done; A = peak flow record undertaken while still exposed.

Figures in parentheses for challenge tests indicate the concentration of solution used in mg/ml.

monitoring took place and the other workers all came from other plating shops not studied in the epidemiological study. The results show that the plating bath operators are not necessarily the most heavily exposed, and that exposure levels are similar in those with incidental exposure. The heat from plating baths and the local exhaust extraction often removes the fume from the plating operators, resulting in higher than expected exposures away from the plating baths. No measurement of chromate or nickel exceeded the occupational exposure standard. Apart from subject 1 who was exposed to subjectively high levels of fume after a breakdown of the extraction system, the work

![Figure 1](image1.png)

**Figure 1** Bronchial provocation testing in subject 7 showing responses to nebulised acetic potassium chloride (control), potassium dichromate 1 mg/ml, and nickel chloride 10 mg/ml.

![Figure 2](image2.png)

**Figure 2** Plots of serial PEF records for subjects 1 and 4. Shaded areas indicate days at work, unshaded areas days away from work. Diurnal variation (% predicted) is illustrated at the top of each figure. The three lines in each figure represent, from top to bottom, the daily maximum, mean and minimum.
Occupational asthma due to chrome and nickel electroplating

The clinical features of chrome induced asthma in seven subjects exposed in metal electroplating works are described. All had positive airway challenges to chrome solution and in two there was also a significant reaction to nickel, which was probably the primary sensitizer in the worker with the high exposure following the breakdown of an extractor fan. There was no association between atopy and skin test response to metal salts and the airways response to inhaled potassium dichromate solution. It is interesting that all those sensitised to usual electroplating shop exposures were current or ex-smokers. Cigarette smoking may enhance the absorption of chrome, perhaps due to contamination of the cigarettes or to bronchial inflammation induced by the cigarette smoke. Smokers are at increased risk of developing specific IgE to some occupational allergens such as platinum salts. The first report of chromium induced asthma was in 1886. Since then, there have been several reports demonstrating that bronchoconstriction can be experimentally induced either by subcutaneous injection or by aerosol inhalation. There have only been isolated case reports of occupational asthma due to chrome exposure in electroplaters. This is the first report of a series of such cases and the first to include air measurements from the workplace. Occupational asthma due to chromium exposure is not unique to workers in the electroplating industry. Previous reports have also demonstrated occupational asthma or bronchitis in workers welding stainless steel, in concrete or construction workers, or chromite ore miners. There have also been some reports of occupational asthma in nickel platers. Specific bronchial provocation testing is generally regarded as the gold standard by which to identify the specific cause of occupational asthma. All the reported workers reacted to potassium dichromate at concentrations from 1 to 10 mg/ml. Reactions can, however, be induced by irritant mechanisms if the exposures are too high. Potassium dichromate solution is acidic and it is at least possible that this contributes to the reactions seen. Previous studies of the effects of non-isotonic aerosols on airways calibre in mild asthmatics using ultrasonic nebulisers have demonstrated that the degree of hyperosmolarity, the pH, and the type of ion itself are all important factors in determining the airways response. It is unlikely, however, that the airway responses to challenges in this study were due simply to an irritant effect as challenge with hypertonc acid aerosol in subject 7 did not change the airway calibre. Non-specific bronchial responsiveness was not measured routinely at the time of specific challenge, but in the only worker measured it was within the normal range. The airway challenges demonstrate the wide variety of responses to inhaled chromium salts with both single early and dual responses being seen. The role of skin tests and RAST measurements in the diagnosis of metal induced asthma is not clear. The size of the skin reactions obtained in subjects who were clearly responsive to chrome solutions, it seems, that the skin responsiveness is not a good predictor of airways response. This may suggest that metal solutions need to be presented with other proteins before becoming antigenic. Skin prick tests with potassium dichromate (10 mg/ml) relate to exposure but not to asthmatic symptoms. Positive reactions (>2 mm weal) were seen in 10% of exposed workers with symptoms suggestive of occupational asthma, 15% of exposed workers with rhinitis, 11% of asymptomatic exposed workers, and 2% of unexposed controls in a cross sectional epidemiological study of plating workers. For workers with exposure to nickel, positive reactions to a solution of 10 mg/ml were seen in 2% of workers with asthma-like symptoms, no workers with rhinitis had positive skin tests, and 9% of asymptomatic exposed workers had positive reactions; only 1% of unexposed controls had positive skin reactions to nickel. The results with serum IgE antibodies to metal salts are equally inconsistent. Novey et al demonstrated specific IgE antibodies to chromium and nickel conjugates in an electroplater who developed occupational asthma. Inhalation challenges were positive but skin tests to chromic acid were negative. Malo et al described a single electroplater with a positive nickel inhalation challenge in whom both skin tests and RAST were negative. Dolovich suggested, in a case of nickel induced asthma, that the antigenic determinant was dependent on the combination of Ni²⁺ with human serum albumin at the primary copper binding site. Similar results were reported to nickel in workers with hard metal asthma in whom cobalt is the principal allergen. The differences may be due in part to the ability of the metal salt to form complex with human serum albumin.

These results suggest that airways sensitivity to chrome and nickel exists and is a significant cause of occupational asthma in electroplating workers. It is likely that, in most workers, exposure to chrome was maintained at levels below the occupational exposure limit, suggesting that still lower levels will need to be achieved to prevent sensitisation. It is important, however, to note that one worker developed his first symptoms after a single large exposure. Cross reaction with nickel may exist. Where there is doubt about the diagnosis, specific bronchial provocation testing with potassium dichromate solution may be useful.

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2 Smith AR. Chrome poisoning with manifestations of sensitisation. JAMA 1931;97:95–8.


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