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# An outbreak of occupational asthma due to chromium and cobalt

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| **Background** | Five metal turners employed by an aerospace manufacturer presented to the Birmingham Chest Clinic occupational lung disease unit. Four cases of occupational asthma (OA) due to chromium salt (3) and cobalt (1) were diagnosed by serial peak-expiratory flow measurements and specific inhalation challenge testing. |
| **Aims** | To measure the extent of the outbreak and to provide epidemiological data to ascertain the aetiology. |
| **Methods** | Participants answered a detailed, self-administered questionnaire, designed to detect occupational lung disease. Urine chromium and cobalt excretion, spirometry and exhaled nitric oxide measurements were taken. Those with possible, probable or definite non-OA or OA, after questionnaire, were invited to undertake two-hourly peak flow measurements and received specialist follow-up. |
| **Results** | A total of 62 workers (95% of workforce) participated. Sixty-one per cent of employees were working in higher metalworking fluid (MWF) exposure areas. Ninety per cent of workers had urinary chromium excretion indicating occupational exposure. Sixty-six per cent of workers reported active respiratory symptoms, although there were no significant differences between exposure groups. Two further workers with probable OA were identified and had significantly higher urinary chromium and cobalt concentration than asymptomatic controls. Eighteen cases of occupational rhinitis (OR) were identified, with significantly raised urinary chromium concentration compared with asymptomatic controls. |
| **Conclusions** | Chromium salt and cobalt can be responsible for OA and OR in workers exposed to MWF aerosols. Onset of symptoms in those with positive specific challenges followed change in MWF brand. Workers with OA had increased urinary concentrations of chromium and cobalt, and those with OR had increased urinary concentrations of chromium. |
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| **Key words** | Chromium; cobalt; metal working fluids; occupational asthma; occupational rhinitis. |



## Introduction

Occupational asthma (OA) from sensitization due to inhalation of chromium salt has been reported in electroplaters, stainless steel welders and construction workers [1–4] but never before in metalworkers exposed to metalworking fluid (MWF). Chromium-related OA occurs in situations where exposure levels are likely to be within the current exposure standards [1]. Users of hard metal-tipped tools may also develop cobalt OA from dry grinding [5,6] and exposure to MWF.

Five employees of a medium-sized manufacturer of precision engineering parts who were toolmakers or grinders by trade presented to the Birmingham Chest Clinic Occupational Lung Disease Unit in 2009 with work-related respiratory symptoms and were investigated. The aim of the subsequent clinical investigation was to measure the extent of the outbreak of occupational respiratory disease and to provide epidemiological data to ascertain the aetiology.

## Methods

The five workers were investigated using (i) methacholine reactivity (using the Yan method) [7], (ii) two-hourly peak flow measurements (analysed using Occupational Asthma SYStem (OASYS)) [8], (iii) spirometry, (iv) fractional exhaled nitric oxide (FeNO), (v) skin prick tests

to chromium and cobalt salts and (vi) after a subsequent workplace visit specific inhalation challenge (SIC) tests. These were performed via a nebuliser over three exposures totalling 32–35 min for potassium dichromate (2 mg/ml) and cobalt chloride (1 mg/ml and 10 mg/ml), and 50–70 min for used and unused MWF.

The index workers were employed by a medium-sized manufacturer of precision engineering parts for aerospace applications, using pre-forged alloys. Several metals were used including Nimonic alloys containing high percentages of nickel and chromium, light steel and titanium. Most turning was done with tungsten carbide tipped tools, which also raised the possibility of cobalt exposure. The machine shop was split into two areas: (i) old machine shop, where slow speed cutting of metal occurred using carbide tipped tools, and (ii) main machine shop, where computer numerical controlled (CNC) machining was used. In the old machine shop, manual turning machines were open; in the main machine shop, CNC machines were either fully enclosed or open at the top. Each CNC machine had its own sump and the MWF was tested regularly for pH, bacteria and cobalt levels. The machines did not have local exhaust ventilation; large roof extraction units were installed in the main machine shop in 2007, re-circulating filtered air. The MWF was changed to Castrol Hysol G in 2004.

In April 2010 an epidemiological investigation of the workforce was carried out in order to identify any further workers with respiratory disease. This was done with the cooperation of company management and the payroll department in order to identify all current employees. All workers were invited to answer a detailed, self-administered questionnaire [9] designed to detect OA, rhinitis, alveolitis and humidifier fever (available online at: www.occupationalasthma.com/resources/outbreak_respiratory_survey.doc). Questions included demographic information (gender, ethnicity and age), smoking status, job history and clinical information. Workers were asked whether symptoms were 'better', the 'same' or 'worse' on 'days away from work' and 'holiday'. Symptoms were classified as work related if they improved on days off or on holiday. A provisional clinical diagnosis was made following a 10-min interview with a specialist occupational respiratory physician including a review of questionnaire responses. Asthma, OA, rhinitis, occupational rhinitis (OR), humidifier fever, chronic bronchitis and allergic alveolitis were assessed as definite, probable, possible or absent. Those with possible, probable or definite asthma or OA were invited to undertake two-hourly peak flows and received a follow-up appointment at the Birmingham Chest Clinic occupational lung disease clinic. Chromium and cobalt concentrations (μg/l) and excretion (normal chromium and cobalt excretion less than 1 μg/g creatinine) were measured on random urine samples taken at the time of the questionnaire. Spirometry was measured on a Viasys Microlab portable spirometer (Micromedical Ltd, Rochester, Kent, UK) according to European Respiratory Society/American Thoracic Society (ERS/ATS) standards using European Community for Coal and Steel predicted values [10]. FeNO was measured on a Niox Mino handheld machine (Aerocrine AB, Solna, Sweden) at 50 ml/s compliant with ERS/ATS recommendations for measurement of FeNO [11]. Workers undertook spirometry and FeNO measurements in an office adjacent to the main machine shop, during a week day shift.

Data regarding demographic information, job history and clinical symptoms were tabulated and analysed by comparison between workers with medium or higher exposure and those with low or no exposure to MWFs. Comparisons of workers with probable or definite asthma, OA or OR, were made with asymptomatic workers. Categorical data were analysed using chi-squared tests with Yates' correction, non-parametric data analysed using Mann–Whitney *U*-test, and parametric data analysed using two-tailed Student's *t*-test.

Ethics approval for the cross-sectional study was not sought, as this report is the result of respiratory surveillance at the company. The investigation was approved by both management and employee representatives and was voluntary.

## Results

Four out of the five index presentations to the specialist clinic were diagnosed with OA in or after 2009, and the fifth (Case 5) was diagnosed with asthma probably unrelated to work, with an OASYS plot not suggestive of OA (Table 1). Latency of symptoms to presentation was 6–30 months, in all workers. Workers' age range was 35–56 years; three had never smoked and two were ex-smokers. On skin prick testing, two had 1–2 mm wheals to potassium dichromate and three had 3 mm wheals to cobalt chloride. Two workers, both never smokers, had a raised FeNO level (greater than 22.1 ppb) [12]. Three had normal spirometry at diagnosis and three had normal methacholine challenges after SIC. The OASYS score for the four workers with OA ranged from 3.25 to 4.00 (see Table 1 and Figure 1) and was 1.15 in the non-OA case. In the OA group there was one dual asthmatic reaction after SIC to potassium dichromate, two early asthmatic reactions to potassium dichromate (one of those with an additional dual reaction to used MWF) and one late asthmatic reaction to cobalt chloride.

For the workforce investigation 62 out of a total of 65 workers at the company completed the questionnaire and undertook physiological measurements. There were 54 males (87% of workforce); mean age was 39.5 (standard deviation (SD) = 12.5). There were 38 employees working in areas of medium or higher exposure to MWF (manual and CNC milling or turning, grinding),

**Table 1.** Demographic and sensitization characteristics of index workers

| | Age/ Gender | Smoking | Symptom latency to presentation (months) | $FEV_1$ at diagnosis (% of predicted) | $FE_{NO}$ level at diagnosis (parts per billion) | SPT wheal size (mm) to chromium/cobalt/ histamine control | Methacholine $PD_{20}$ (μg) after SIC | OASYS score | Potassium dichromate 2 mg/ml SIC (% fall in $FEV_1$) | | Cobalt chloride 10 mg/ml SIC (% fall in $FEV_1$) [or 1 mg/ml] | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | Early | Late | Early | Late |
| Case 1 | 56/Male | Ex-smoker (18 pyh) | 24 | 70 | 17 | 0/3/7 | 1909 | 3.67 | −23.8 | −25.8 | −2.5 | −12.7 |
| Case 2 | 48/Male | Never | 24 | 93 | 64 | 2/3/9 | >3000 | 3.87 | −17.8 | −7.0 | −5.5 | −4.6 |
| Case 3 | 35/Male | Never | 6 | 115 | 20 | 1/3/8 | >4800 | 4.00 | −7.0 | −1.9 | −4.9 | −11.2 |
| Case 4 | 39/Male | Never | 6 | 119 | 33 | Flare/0/7 | 1200 | 3.25 | −26.0 | −19.7 | −0.3 | 1.1 |
| Case 5 | 47/Male | Ex-smoker (9 pyh) | 30 | 105 | 17 | 0/0/7 | >4800 | 1.15 | n/a | n/a | n/a | n/a |

$FEV_1$, forced expiratory volume in one second; $FE_{NO}$, fractional exhaled nitric oxide; $PD_{20}$, dose of methacholine by the Yan method producing a 20% fall in $FEV_1$ (normal $PD_{20}$ > 2000 μg methacholine); PYH, smoking pack year history; SPT, skin prick test (chromium = 1 or 2 mg/ml potassium dichromate; cobalt = cobalt chloride 10 mg/ml).

compared with 24 in areas of low or no exposure to MWF (de-burring, inspection, stores, packing or offices). Ninety per cent (43/48) of workers who submitted urine samples for analysis had urinary chromium excretion greater than normal (more than 1 μg/g creatinine) [13]. Sixty-six per cent (41/62) of workers reported active respiratory symptoms (see Table 3). Spirometry, FeNO and urinary chromium and cobalt measurements, or reporting of respiratory, eye and nasal symptoms, did not differ significantly between groups, although all were generally more prevalent in the higher MWF exposure group. Dry or sore throat was significantly more common in the higher exposure group. Six workers with probable/definite OA (two new workers, in addition to four index cases previously described) and 12 workers with probable/definite non-OA were identified. There were 18 with probable/definite OR but no workers with a history suggestive of humidifier fever or alveolitis. Table 4 shows the comparisons between probable/definite OA, non-OA or OR with asymptomatic controls. There were significantly higher urinary chromium and cobalt concentrations in the probable/definite OA group over the asymptomatic controls. There were significantly higher urinary chromium concentrations in the group with probable/definite OR.

Twenty-three workers (those with possible/probable or definite OA based on questionnaire, and who reported a work effect on their asthma symptoms) were asked to provide two-hourly peak flow measurements from wake to sleep, for 4 weeks. Thirteen workers who completed the questionnaire subsequently returned electronic peak-expiratory flow measurements; none of these revealed definite OA, and no further investigations were completed. All four index cases of OA received follow-up at the occupational lung disease clinic and their health and employment outcomes are shown in Table 5. Patients with a diagnosis of OR received a letter from the Birmingham Chest Clinic advising them of the diagnosis and the likelihood of onset of OA with continued exposure to a sensitizing agent [14].

## Discussion

We have described an outbreak of OA and OR in a factory machining Nimonic alloys with high nickel and chromium content. Three workers had positive SIC tests with chromium and one a probable reaction to cobalt, derived from aerosols of MWF. This is the first description of chromium asthma from MWF contamination, which started after a change in MWF. The subsequent survey identified 18 workers (27% of the workforce) with OR, which often precedes OA [14]. There were significantly higher urinary chromium and cobalt concentrations in workers with OA and significantly higher urinary chromium concentrations in workers with OR compared with asymptomatic workers.

All index cases showed asthmatic reactions to SIC with potassium dichromate (2 mg/ml) or cobalt chloride (1 mg/ml), and SIC testing is generally regarded as the gold standard for diagnosis of OA. Although the reaction for Case 3 to cobalt chloride did not reach a 15% fall, there were six statistically significant points below the three other exposure days where no response was seen [15]. Workers with OA had features of sensitization with latent intervals of 6–24 months following a change in MWF, had positive challenges to non-irritant concentrations of chrome or cobalt salts and significantly higher urinary chromium and cobalt concentrations than asymptomatic controls. The mean chromium excretion of the workforce was 2.2 μg/g creatinine; a level indicating occupational exposure (more than 1 μg/g creatinine). The mean levels in the low/no

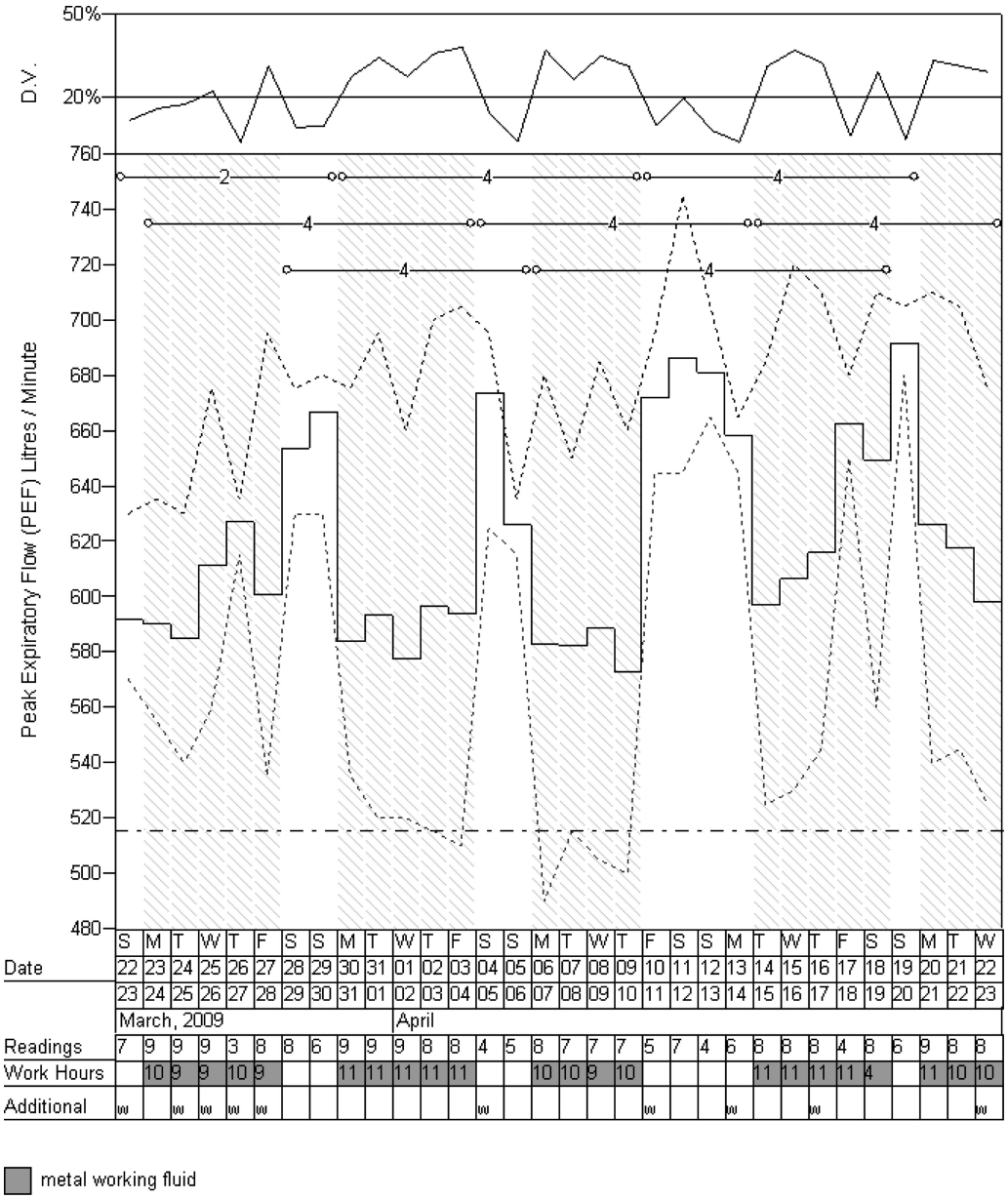


**Figure 1.** Serial peak flow measurements from Case 2 using a Mini-Wright digital meter were analysed using OASYS. The plot showed OA and OASYS score was 3.87. The top part of the chart shows the diurnal variation for each day. The middle of the chart shows the maximum-, mean- and minimum peak flow for each day. Work days are shaded (diagonal back slash bars are morning shifts) and the rest days are blank. The horizontal lines containing numbers in this part of the chart are scores for the work-rest-work and rest-work-rest complexes (eight complexes in total in this record). The bottom of the record shows the days, dates and number of readings per day for the record.

exposure group are probably an overestimate, since only one worker in that group described no exposure at all, that is, never entering the machine area. Interpretation of results would have been improved by the availability of inhalable exposure data and further investigation of workers with possible OA identified through the survey.

MWFs are mixtures of oils, emulsifiers, anti-weld agents, corrosion inhibitors, buffers and other additive agents. Once used, they become contaminated with other manufacturing by-products, including metal particles, and they also grow microbes requiring addition of biocide. Thus there is an array of potential sensitizers many of which can cause OA, making it difficult to determine a precise cause [9,16]. An emerging cause of OA, MWFs are cited as the offending agent in 11% of reported cases of OA in the West Midlands [17] (the second leading cause behind isocyanates) and were implicated in a large outbreak of 74 cases of OA at a manufacturing plant [9]. From our series, negative challenges to unused MWF and a positive test to used MWF in one of the chromium

**Table 2.** Demographic and physiological characteristics of workers by level of exposure to MWF

| | All workers ($n$ = 62) | (I) Workers with higher/medium MWF exposure ($n$ = 38) | (II) Workers with low/no MWF exposure ($n$ = 24)[a] |
|---|---|---|---|
| Mean age, years (SD) | 39.5 (12.5) | 37 (11.8) | 43 (12.9) |
| Sex (number of males) (%) | 54 (87) | 38 (100) | 16 (67)* |
| Mean hours worked per week (SD) | 45.1 (7.3) | 45.9 (6.0) | 43.8 (9.0) |
| Mean work loss due to chest in preceding 12 months (in days) (SD) | 0.48 (1.68) | 0.6 (1.9) | 0.26 (1.1) |
| Number of active smokers (%) | 17 (27) | 11 (29) | 6 (25) |
| Number of ex-smokers (%) | 13 (21) | 9 (24) | 4 (17) |
| Number of non-smokers (%) | 32 (52) | 23 (61) | 9 (38) |
| Mean FEV (% predicted) (SD) | 100.4 (11.5) | 102.1 (10.4) | 97.6 (12.7) |
| Mean forced vital capacity (% predicted) (SD) | 108.6 (10.7) | 109.6 (10.8) | 107.0 (10.7) |
| Mean FeNO (ppb) (SD) | 18.5 (15.7) | 18.9 (17.8) | 17.7 (11.9) |
| Mean urine chromium (µg/L)[b] (SD) | 1.56 (0.8) | 1.65 (0.9) | 1.4 (0.7) |
| Mean urine chromium excretion (µg/g creatinine)[b] (SD) | 2.2 (4) | 2.3 (5) | 2.0 (0.9) |
| Mean urine cobalt (µg/l)[b] (SD) | 0.6 (1.1) | 0.8 (1.3) | 0.3 (0.3) |
| Mean urine cobalt excretion (µg/g creatinine)[b] (SD) | 0.6 (1) | 0.7 (1.2) | 0.3 (0.3) |

[a]Analysis I versus II by chi-squared test with Yates' correction, for categorical data; analysis by unpaired two-tailed Student's $t$-test for parametric data.
[b]48 out of 62 workers provided urine samples for analysis of chromium and cobalt concentration and excretion.
*$P < 0.001$.

**Table 3.** Presence of symptoms by exposure group, based on questionnaire

| | All workers ($n$ = 62) $n$ (%) | (I) Higher or medium MWF exposure ($n$ = 38) $n$ (%) | (II) Low or no MWF exposure ($n$ = 24)[a] $n$ (%) |
|---|---|---|---|
| Are you disabled from walking by breathlessness? | 11 (18) | 9 (23) | 2 (8) |
| Work effect[b] | 5 (8) | 5 (13) | 0 (0) |
| Do you cough? | 27 (44) | 19 (50) | 8 (33) |
| Work effect | 10 (16) | 9 (24) | 1 (4) |
| Do you cough up phlegm from your chest? | 22 (36) | 16 (42) | 6 (25) |
| Work effect | 6 (10) | 6 (16) | 0 (0) |
| Have you been wheezy in the last 12 months? | 19 (3) | 14 (37) | 5 (21) |
| Work effect | 8 (13) | 7 (18) | 1 (4) |
| In the last 12 months has your chest ever felt tight? | 22 (36) | 15 (40) | 7 (29) |
| Work effect | 10 (16) | 7 (18) | 3 (13) |
| In the past 12 months have you suffered recurrent flu like symptoms? | 16 (26) | 12 (32) | 4 (17) |
| Work effect | 3 (5) | 3 (8) | 0 (0) |
| In the past 12 months have you had more than two episodes of irritation or watering of the eyes? | 23 (37) | 17 (45) | 6 (25) |
| Work effect | 10 (16) | 8 (21) | 2 (8) |
| In the past 12 months have you had more than two episodes of blocked or stuffy nose? | 35 (57) | 25 (66) | 10 (42) |
| Work effect | 15 (24) | 12 (32) | 3 (13) |
| In the past 12 months have you had more than two episodes of a dry or sore throat? | 30 (49) | 23 (61)* | 7 (29) |
| Work effect | 12 (19) | 10 (16) | 2 (8) |

[a]Analysis of I versus II by chi-squared test with Yates' correction, for categorical data.
[b]Possible work effect is suggested by answer 'better' to either or both of 'Is your symptom better (a) on days away from work or (b) on holiday?'.
*$P < 0.05$.

**Table 4.** Characteristics and comparisons of workers (completing questionnaires) with respiratory symptoms, and an asymptomatic control group

| | (I) Probable/ definite OA, ($n$ = 5)[a] | (II) Probable/ definite non-OA ($n$ = 12) | (III) Probable/ definite OR ($n$ = 18)[b] | (IV) Controls (normals) ($n$ = 37) | IV versus I[c] | IV versus II[c] | IV versus III[c] |
|---|---|---|---|---|---|---|---|
| Mean age years (SD) | 34.4 (4.6) | 38.8 (12.8) | 36.6 (10) | 40.2 (13.5) | $t$ = 0.08 | $t$ = 0.75 | $t$ = 0.33 |
| Number of active smokers | 1 (20) | 2 (17) | 4 (22) | 12 (32) | NS | NS | NS |
| Number of ex-smokers | 0 (0) | 6 (50) | 4 (22) | 4 (11) | NS | $P$ = 0.01 | NS |
| Number of non-smokers | 4 (80) | 4 (33) | 10 (56) | 21 (57) | NS | NS | NS |
| $FEV_1$ (%-predicted) (SD) | 100 (13) | 90 (13) | 100 (11) | 103 (10) | $t$ = 0.49 | $t$ < 0.001 | $t$ = 0.32 |
| FVC (%-predicted) (SD) | 113 (17) | 108 (7) | 110 (11) | 108 (11) | $t$ = 0.39 | $t$ = 0.95 | $t$ = 0.54 |
| Ever worked with MWF (%) | 5 (100) | 9 (75) | 18 (100) | 27 (73) | NS | NS | $P$ < 0.05 |
| Currently working with MWF (%) | 5 (100) | 5 (42) | 3 (17) | 18 (49) | NS | NS | $P$ < 0.05 |
| Mean FeNO (ppb) (SD) | 17.1 (9.5) | 23.6 (13.3) | 21 (11.8) | 16.6 (17.3) | $t$ = 0.92 | $t$ = 0.17 | $t$ = 0.45 |
| Mean urine chromium (µg/l) (SD) | 2.2 (1) | 1.3 (0.6) | 1.9 (1) | 1.4 (0.7) | $t$ = 0.05 | $t$ = 0.66 | $t$ < 0.05 |
| Mean urine cobalt (µg/l) (SD) | 1.6 (1.5) | 0.4 (0. 5) | 1.2 (1.8) | 0.4 (0.3) | $t$ < 0.001[d] | $t$ = 0.92 | $t$ = 0.07 |
| Working in medium/higher exposure area (%) | 5 (100) | 6 (50) | 15 (83) | 20 (54) | NS | NS | $P$ < 0.05 |
| Duration of employment at company (months), Median (interquartile range) | 187 (157–243) | 93.5 (41–124.3) | 106 (61.3–156) | 110 42–245) | NS | NS | NS |

NS, not significant.
[a]$n$ = 5 for the analysis, because one worker with OA (an index case) did not complete the questionnaire; all 5 also had probable/definite OR.
[b]Of these, 5 also had probable/definite OA; 4 also had non-OA.
[c]Analysis by chi-squared test with Yates' correction, for categorical data; analysis by unpaired two-tailed Student's $t$-test, for parametric data (age, $FEV_1$, FVC, urine cobalt, urine chromium, FeNO); analysis by Mann–Whitney $U$-test for non-parametric data (duration of employment).
[d]Done assuming equal variance.

**Table 5.** Health and employment outcomes of Index cases ≥2 years after investigation

| | Cause of OA | Ongoing asthma symptoms? | Asthma Treatment | $FEV_1$ at 2 years after diagnosis (% of predicted) | $FE_{NO}$ level at 2 years after diagnosis (parts per billion) | Methacholine $PD_{20}$ (µg) | Employment | Tasks undertaken |
|---|---|---|---|---|---|---|---|---|
| Case 1 | Chromium | Ongoing mild symptoms, but no work-related symptoms | ICS/LABA and SABA | 67 | 26 | Not available | New employer | Machining low-carbon steels and bronze with no chrome content |
| Case 2 | Chromium | Asthma with exercise and infection | ICS/LABA and SABA | 96 | 41 | >4800 | With same company | Relocated to quality control; urine chrome now unmeasurably low. FeNO 24 ppb |
| Case 3 | Cobalt | Symptom free | SABA only | 115 | Not available | >4800 | With same company | Relocated to gear cutting without hard metal-tipped tools (urine cobalt now normal |
| Case 4 | Chromium | Ongoing mild symptoms, with episodic deterioration at work | ICS and SABA | 100 | Not available | 1603 | With same company | Management role, with occasional machining with MWF |

ICS, inhaled corticosteroids; SABA, short-acting beta-agonists; ICS/LABA, ICS and LABA combination inhaler; LABA, long-acting beta-agonists.

sensitized cases, suggest that metal constituents in the used oil were likely responsible.

Cobalt causes OA in hard metal workers and those using hard metal-tipped tools. This can arise through dry grinding or with MWF use and is known to cause sensitization at low exposure levels [6]. In our series, one of the workers had a positive SIC test to cobalt chloride (1 mg/ml) but not used or unused MWF. His symptoms were worse when CNC milling; changing the metal being turned had no effect, but avoiding the use of tungsten-tipped tools abolished work-related symptoms.

A small number of cases of OA following chromium inhalation have been reported—in electroplaters, stainless steel welders and construction workers [1–4]. This, however, is the first report of an outbreak of OA due to chromium salts in MWF. All workers were sensitized in or after 2004, when the MWF brand and composition were changed, which would be compatible with leaching of the metals into this particular oil. SIC tests revealed early and dual asthmatic responses to inhaled chromium salts, which reproduced the variety of responses that have been seen previously [1,3]. However, the mechanism of sensitization remains unclear. Some authors have proposed an IgE-mediated immunological mechanism where the metal salts act as haptens [3,4,18]. Cruz [3] reviewed all cases of chromium OA to date and found that 58% cases (7/12) had positive skin prick tests, and 3/3 had high serum IgE levels. Furthermore, Sastre *et al.* [18] had reported an increase in sputum eosinophilia in nickel and chromium induced asthma. In our series skin prick responsiveness was variable with two cases having 1–2 mm wheals to potassium chromate and three out of four cases having 3 mm wheals to cobalt chloride.

This paper provides strong evidence for OA due to chromium sensitization, and describes the first series in metal turners. Sensitization occurred in a small number of individuals within a larger number of exposed workers. Additionally, 27% of the workforce had symptoms of OR.

## Key points

- We have described an outbreak of occupational asthma in metal turners, due to chromium salts in metalworking fluid, which was accompanied by an additional case of cobalt occupational asthma.
- Positive specific inhalation challenge tests to potassium dichromate and cobalt chloride provided evidence for sensitization to metal salts.
- A large minority of the workforce and those exposed to metalworking fluids had features of occupational rhinitis, with significantly higher urinary chromium concentration than asymptomatic workers.

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## Conflicts of interest

None declared.

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# Risk assessments: good and bad

Many years ago, I had the responsibility once a month of doing a tuberculosis clinic in deepest West Virginia. The small town that I drove to was the site of the State penitentiary—it was said the citizens had been polled on whether they wished to have this or the State university and had opted for the gaol in the belief it would provide more jobs. On one occasion, a small and inoffensive-looking patient was led in, handcuffed on both wrists to burly warders. I ensured that he was taking his drugs and making satisfactory progress and arranged to see him again the next month.

After he left, curious to know why the security had been so apparently excessive, I asked the nurse, a local woman, what he was in for. She told me that he had been done for armed robbery. The story was that he had lived in the town all his life and was known to most in the community. One day he had walked in to his local bank, pulled out a pistol and demanded of the cashier that he hand over all the money. The cashier, recognizing that death from acute lead poisoning was an occupational hazard of bank employees, wisely complied but took the sensible step of calling the police when the robber left, giving them his name and address which, as he was also a customer, was known to the bank. The police went round to his house where he was found counting the money on his kitchen table. He received a severe sentence, an occupational hazard of robbers.

Hazards, foreseeable adverse consequences of an activity, are quantified as risks in our Control of Substances Hazardous to Health (COSHH) assessments. The risk of being a victim of an armed robbery is low for any individual, even in Appalachia, but the outcome may be fatal and is always traumatic. The cashier clearly made a snap-risk assessment and decided on a wise course of action. The risk of being apprehended after performing an armed robbery on one's own bank in one's own community must be high, even in Appalachia. My poor patient must have been unfamiliar with the practical importance of risk assessment and learnt that failure to make one could lead to serious consequences. A lesson to us all.

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