

## CASE REPORT

# Superficial copper staining of the teeth in a brass foundry worker

A. M. Donoghue\* and M. M. Ferguson†

\*Occupational Safety and Health Service, Department of Labour,  
Dunedin, New Zealand and †School of Dentistry, University of Otago,  
Dunedin, New Zealand.

A 21 year-old man developed green surface staining of the cervical margins of his teeth 10 months after starting work in a brass foundry. During this time he was exposed intermittently to brass fumes which contained approximately 75% copper and 2-5% lead. The staining of his teeth was attributed to the absence of respiratory protection in the knock-out process and was accompanied by a rising blood lead concentration. Staining of the teeth by copper was described early this century but seems to have been neglected in the recent literature and texts on occupational medicine. We suggest that in brass foundry workers it is a warning of failure to control fume or dust exposure with the attendant risk of lead toxicity.

Occup. Med. Vol. 46, 233-234, 1996

Received 19 July 1995; accepted in final form 1 November 1995.

## HISTORY OF COMPLAINT

A 21 year-old man began work in August 1993 at a brass foundry. Initially he worked in several different areas within the foundry, specifically: the casting, knock-out, fettling, core and mould making areas. The cast metal was brass and contained approximately 75% copper and 2-5% lead. He worked 50-60 hours per week. Disposable dust/fume half-masks were worn in the casting, fettling and mould making areas. Respiratory protection was not however worn in the knock-out room or in the core making area, the latter being relatively isolated from brass dust and fume.

In May 1994 he started a 9-week period working exclusively in the knock-out room. The casts in the knock-out room were knocked out of the moulds while they were still hot and visible fumes were released into the breathing zone. No respiratory protection was worn in the knock-out area at that time. Six weeks after starting this job he noticed green surface staining of his teeth. For a week or so he was able to remove the staining by vigorous application of his tooth brush, but after 2 further weeks, during which he was unable to do this, he decided to seek dental treatment. At this time (mid July) he left the knock-out room and began working exclusively in the mould making area where

disposable dust/fume half-masks were routinely worn because of proximity to the casting area. Previous employment had not involved exposure to metal dust or fume.

The medical history was unremarkable, he was taking no medication and had no significant family history. His recreational activities included shooting but he did not undertake the reloading of ammunition or any other activities exposing him to metal dust or fume.

## CLINICAL EXAMINATION

On the 4th of August 1994 examination of his teeth revealed a superficial green staining of the cervical margins. Clinical examination on the 22nd of September 1994 did not reveal signs of copper or lead toxicity. In particular: the hair and skin was blonde with no green discolouration, the nasal septum was normal with no signs of ulceration or perforation, Kayser-Fleischer rings were not seen, the neurological system was normal and the gums did not display blue/grey lines.

## INVESTIGATIONS

The whole blood lead concentration results from biological monitoring are given in Table 1.

Correspondence and reprint requests to: Dr A Michael Donoghue, Departmental Medical Practitioner, Occupational Safety and Health, Department of Labour, P.O. Box 537, Dunedin, New Zealand.

**Table 1.** Whole blood lead concentrations ( $\mu\text{mol/l}$ )

Date	13.12.93	22.04.94	20.06.94	18.07.94	02.08.94
Blood lead	1.37	1.75	2.15	2.18	1.51

On the 22nd of September 1994 serum ceruloplasmin was 0.23 g/l (normal range 0.17–0.56 g/l) and the full blood screen and liver function tests were normal.

## DISCUSSION

This man developed green surface staining of the cervical margins of his teeth 10 months after starting work in a brass foundry. During this time he was exposed intermittently to brass fume in the knock-out process without respiratory protection. Exposure to brass dust and fume elsewhere in the foundry was minimized by the use of disposable dust/fume half-masks. In the 6 weeks immediately preceding the appearance of the staining he worked exclusively in the knock-out area. The brass was known to contain about 75% copper. We concluded that the surface staining of his teeth was most likely due to the adherence of copper from the brass fume present in his breathing zone during knock-out and its subsequent conversion to a copper salt, probably copper carbonate. A normal serum ceruloplasmin and an absence of the relevant symptoms and signs reasonably excluded Wilson's Disease.<sup>1</sup>

The green staining was removed from his teeth and workers in the knock-out area of the foundry are now required to wear disposable dust/fume half-masks

thereby minimizing the risk of new cases developing.

We were surprised during our investigation of this case to find no mention of this condition in the recent medical and dental literature. However Sir Thomas Legge in his book *Industrial Maladies* (1934) commented that 'A faint green to a dark olive-green deposit of basic carbonate of copper is often seen on the teeth of brass polishers, and is a characteristic sign of a brass- or bronze-worker.'<sup>2</sup> In 1902 he had investigated brass-workers in Birmingham and found the condition in 65% of brass polishers and 16% of brass casters.<sup>2</sup> The condition appears to have been neglected in the recent literature and texts on occupational medicine, perhaps because of a declining prevalence due to generally improved industrial hygiene. Nevertheless its occurrence in today's setting may be of use, as in this case, as a crude marker for isolated failure of brass fume or dust exposure control measures. It is important to recognize such failures because of the associated exposure to lead, a constituent of brass, and the potential for lead toxicity. This is highlighted in our case by the rise in whole blood lead concentration during the period of uncontrolled exposure in the knock-out room and its fall following departure from this area.

## REFERENCES

1. Scheinberg H. Wilson's Disease. In: Wilson JD *et al.* (eds). *Harrison's Principles of Internal Medicine*. 12th Ed. New York: McGraw-Hill, 1991: 1843–1845.
2. Legge TM, Henry SA (eds). *Industrial Maladies*. London: Oxford University Press, 1934: 92–95.