Lead Risks Overlooked in Sandblasters?

Steenland, *et al*, reported that sandblasters had the highest odds ratio (3.83) for end stage renal disease of any occupational group in Michigan.[1] Sandblasters were categorized as silica exposed. Lead exposed workers were considered as a separate category and were also found to be at an elevated risk for renal disease in reference to the area populations, although their risk was found to be lower than sandblasters (odds ratio = 1.73).

Lead may be a more important etiologic agent than silica in association with the observed excess of kidney disease in sandblasters. Sandblasting surfaces painted with lead can cause environmental contamination and lead poisoning in sandblasters.[2] Because of this risk, Maryland has banned open abrasive sanding (sandblasting) as a method of lead abatement.[3] According to US census data, 20–30 percent of Michigan housing was built before 1940 and is thus likely to contain high concentrations of lead paint.[4] In the Detroit area where 244/325 cases lived, the majority of residential property was built before 1970, and thus well before lead content of new residential paint was reduced to 600 ppm by the Consumer Product Safety Commission. Sandblasting is also used to clean nonresidential property such as bridges, for which there is no current prohibition against the use of leaded paint.

I would be interested to know if the occupational histories obtained for the sandblasters in the Michigan study contain any indications of lead exposure, such as blasting bridges or older buildings. Also, it should be possible to determine if the sandblasters in the Michigan study were construction workers or manufacturing/general industry workers. If the "silica exposed" sandblaster cases with renal disease worked in construction rather than manufacturing, their disease could be related to the weaker occupational health protection standards available for construction compared to general industry workers. The US Department of Labor OSHA permissible exposure level (PEL) for lead is 200 micrograms per cubic meter of air for construction workers, compared to 50 $\mu g/m^3$ for manufacturing workers.[5]

By comparison, hand sanding for 5 to 22 minutes on lead painted surfaces has generated concentrations of 510 to 550 μg lead/m^3 air.[6] Sandblasting could generate even higher concentrations. Sandblasters' risk of high dose lead exposure is increased further because typical respiratory protection used by sandblasters had been reported to be inadequate, especially in regards to respirable particles.[7]

In order to evaluate the relative contributions of silica and lead to sandblasters' risks of end stage renal disease, it would be helpful to locate silica-exposed sandblasters with less risk of lead exposure.

REFERENCES

1. Steenland K, Thun MJ, Ferguson CW, Port FK: Occupational and other exposures associated with male end stage renal disease: A case/control study. Am J Public Health 1990: 80:153–157.
2. Landrigan PJ, Baker EL, Himmelstein JS, *et al:* Exposure to lead from the Mystic river bridge: The dilemma of deleading. N Engl J Med 1982; 306:673–676.
3. Maryland Title 26, Department of the Environment, 1988: 26.02.07 Procedures for Abating Lead Containing Substances from Buildings.
4. Agency for Toxic Substances and Disease Registry: The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. Atlanta, GA: US Department of Health and Human Services, July 1988.
5. 29 CFR 1910.1025 (US Dept. of Labor General Industry lead standard) and 29 CFR 1926.55 (US Dept of Labor Construction Industry lead standard).
6. Feldman RG: Urban lead mining: lead intoxication among deleaders. N Engl J Med 1978; 298:1143–1145.
7. Glindmeyer HW, Hammad YY: Contributing factors to sandblasters' silicosis: inadequate respiratory protection equipment and standards. JOM 1988; 30:917–921.

Joseph Schirmer, CIH, MS
Public Health Educator-Epidemiologist, Environmental and Chronic Disease Epidemiology Section, Bureau of Community Health and Prevention, Wisconsin Department of Health and Social Services 1 West Wilson St., PO Box 309, Madison, WI 53701–0309

Drs. Steenland and Thun Respond

We appreciate the suggestion from Mr. Schirmer that lead exposure may have contributed to the increased risk of end-stage renal disease observed in sandblasters in our study of end stage renal disease. Sandblasting may indeed involve exposure from leaded paint. There are several factors, however, which strengthen the case for silica as an independent risk factor.

1. In our study, sandblasting had an odds ratio of 3.83, compared to 1.73 for lead workers. The higher risk in sandblasters, only some of whom are exposed to lead, suggests that an exposure other than lead is responsible.
2. Our review of the work history of 8 renal cases and 3 controls in our study who were exposed to sandblasting (Table 1) suggests that only two (cases 2 and 4) worked in jobs with probable exposure to leaded paint.

TABLE 1—Cases and Controls with Exposure to Sandblasting

Case 1,	toolmaker, 35 yrs, 5 hrs/wk sandblasting tools
Case 2,	construction worker, 1 yr, 4 hrs/wk sandblasting
Case 3,	toolmaker, 5 yrs, 4hrs/wk sandblasting tools
Case 4,	brewery worker, 14 yrs, 5 hrs/wk sandblasting trucks before painting
Case 5,	meatworker, 2 yrs, 40 hrs/wk, sandblasting meat container
Case 6,	motor repairman, 1 yr, 10 hrs/wk, sandblasting motors
Case 7,	sandblaster, 3 yrs, 40 hrs/wk, sandblasting caskets
Case 8,	sandblaster, 10 yrs, 40 hrs/wk in sandblasting company
Control 1,	miller, 9 yrs, 1 hr/wk, sandblast parts
Control 2,	auto repair, 12 yrs, 1 hr/wk sandblast parts
Control 3,	setup man (tools), 5 yrs, 40 hrs/wk, sandblast parts

3. Rats and rabbits which have been implanted or injected with silica gel develop interstitial nephritis and glomerular lesions.[1]
4. Sandblasters with silicosis have an unusually high prevalence of antinuclear antibody (ANA) positivity without other stigmata of lupus erythematosis,[2] suggesting that silica-induced immunologic abnormalities may provide a mechanism for renal injury.
5. Cases of silica-associated nephropathy, reported in the literature, have all shown evidence of glomerulopathy as well as interstitial disease.[3] Similar data have been reported from an autopsy series of silicotics.[4] Glomerular involvement is not a common feature of lead-induced kidney disease.

In summary, the literature on silica and glomerulonephritis suggests that the occurrence of renal disease in silica-exposed workers, particularly in those with silicosis, should be examined further.

REFERENCES

1. Policard A, Collet A: Experimental study of renal lesions caused by the elimination of silica. J d'Urol Med Chir 1954; 60:164–171.