INDUSTRIAL TOXINS AND THE COMMUNITY

Arsenic Contamination Near a Copper Smelter

Samuel Milham, Jr., MD

The first, and most convincing, evidence that environmental exposures could cause chronic diseases and cancers came from the occupational setting. The larger and more recent question is whether community exposures to the various man-made pollutants in air causes disease. The National Research Council (1) and other groups have addressed this problem in general. In this paper, I would like to share the experiences I have had in the past five years studying the community around a large copper smelter in Tacoma, Washington.

Since 1889, the American Smelting and Refining Company has operated a smelter in Ruston, Washington, an administrative enclave north of, and contiguous with, the city of Tacoma. The smelter is located on a peninsula which extends into Puget Sound (Figure 1). Although built originally as a lead smelter, since 1913 the facility has been a copper smelter specializing in processing copper ores high in arsenic; and it is a major producer of arsenic trioxide (AS_2O_3).

There are homes within 50 yards of the smelter stack, and there is a public elementary school (Ruston School) within 300 yards of the smelter complex. Current estimates place smelter arsenic emissions at about 1,000 pounds per day.

Two facts have emerged in the past five years, which place a special burden on those responsible for community health in Ruston:

1. Ruston residents have increased exposure to arsenic trioxide.
2. Inhaled arsenic trioxide is a respiratory carcinogen.

Children living near the smelter have been shown to have increased levels of arsenic in hair and urine (2). Urinary arsenic levels decrease with distance of residence from the smelter. Evidence that exposure to arsenic may be by inhalation comes from three different pieces of information:

1. Community air contains elevated levels of arsenic, especially when the smelter is operating.
2. Urinary arsenic levels were reduced in the community during a period of smelter inactivity related to a strike (Table 1).
3. Urine arsenic levels in children varied synchronously in samples obtained once a week over a five-week period (2).

The evidence that inhaled arsenic trioxide is a carcinogen is summarized in a NIOSH criteria document (3). Workers at copper smelters, including the Tacoma smelter, have been shown to have increased mortality from lung cancer (4,5). Workers at pesticide formulating plants with arsenic trioxide exposure have also been shown to have a similar lung cancer incidence increase (6,7).

Studies done at the Tacoma smelter have shown that smelter air arsenic levels are positively correlated with urinary arsenic levels in workers (8), and that measures of arsenic exposure in workers (urinary arsenic) are directly related to lung cancer incidence.

Given the above relationships, it is natural to consider whether there might be a lung cancer risk in the community around the smelter due to inhalation of arsenic. An assessment of lung cancer rates by census tract in Tacoma was unrevealing. Excess lung cancer rates in areas around the smelter disappeared when smelter workers were removed from the counts.

Initial urinary arsenic sempling in the community began in June, 1972. Over the past five years, numerous samplings have been done in Ruston, adjacent Tacoma, and Fern Hill Elementary School, a school about 12 miles from the smelter used as a control. Table 2 shows sampling results from the study period. For comparison, normal urinary arsenic levels are 0.014 ppm (14 micrograms/liter). No obvious trends are visible in the data. For comparable groups, arsenic levels are about the same now as they were when sampling began. The smelter has undertaken extensive emission control measures over this time period, which resulted in a halving of arsenic emissions over the study period. No similar trend is discernible in the urinary arsenic sampling.

Table 3 shows daily urinary arsenic sampling for nine consecutive days for a group of children collected in family groups. It is my feeling that long-term sampling in the same individual is more revealing of community pollution patterns than 1-day sampling of large numbers of children. Over the sampling period, marked day-to-day variation in urinary arsenic level is seen in the same individual. The highest levels of urinary arsenic seen in the study are well into the range of urinary arsenic values seen in smelter workers.

Urinary arsenic levels on a given day will be affected by many variables: smelter emission rates, wind direction and velocity, place and motion in the community, respiratory rate, and ingestion of arsenic. Early in the sampling it became evident that seafood ingestion could cause increase in urinary arsenic. To make our results comparable with others, we also found it necessary to resort to urine specific gravity adjustments. Since published normal or control values for arsenic are based on data nearly 30 years old (9), I think it is important that more recent normal level information be developed.

Studies of morbidity in Ruston students in terms of growth (10), school attendance, hearing, and hematologic values (11) were unrevealing.

To make rational judgment about whether the current ambient air levels of arsenic in the community are hazardous in terms of causing lung cancer, more information is needed. To obtain this information, two mortality studies are currently in progress. With the cooperation of smelter officials, we have obtained urinary arsenic data for workers going back as far as the 1940's. This exposure information will be correlated with a concurrent mortality study in smelter workers to examine the relationship between arsenic exposure and mortality from respiratory cancer. We hope this information will allow us to examine the "threshold" question: Is there a level of ambient air arsenic below which no lung cancer is seen? The Occupational Safety and Health Administration has proposed an 8-hour workroom ambient air standard for arsenic of 4 micrograms per cubic meter of air. This level is, on occasion, exceeded in the community air a mile from the smelter for three or more consecutive 8-hour periods (9). The study should also provide a rational basis for setting the workplace arsenic standard.

The second mortality study in progress is an attempt to see whether exposure to community air in the past caused any unusual mortality patterns. Children who attend Ruston School and Fern Hill School in the early 1900's will be identified and, hopefully, followed to determine whether their mortality patterns differ from one another, or from expectation based on U.S. mortality rates. If childhood exposure to inhaled arsenic causes lung cancer in adult life, this study should be able to document it.

In the next year, I hope to develop more current normal or background arsenic information for urinary arsenic levels by sampling unexposed populations and populations whose direct exposures to arsenic ceased over 30 years ago.

In summary, studies of communities around well characterized sources of pollution offer the best chance for understanding health-pollution relationships. Our studies, of course, would be impossible without the cooperation of the Tacoma smelter, which provides historical arsenic dose information as well as mortality information for its workers. The situation in large urban areas, in contrast, is so complex as to defy understanding.

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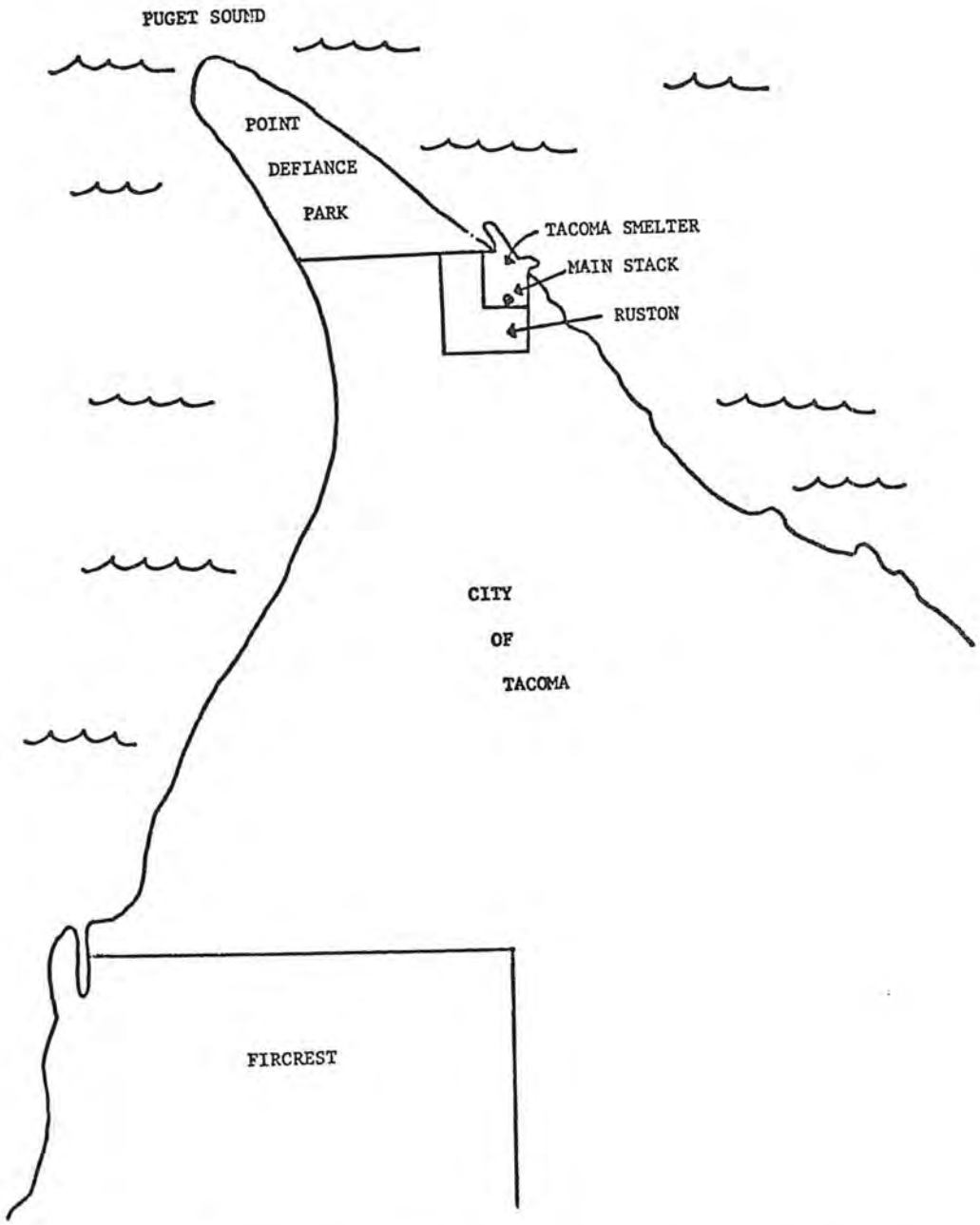

Figure 1. Location of Tacoma Smelter and Ruston In Relation to the City of Tacoma

TABLE 1

AVERAGE URINARY ARSENIC LEVELS (PPM)

During and After Strike at the
Tacoma Smelter 1974

	DURING			AFTER		
	7-19-74	7-25-74	Average	8-20-74	8-22-74	Average
Child 1	.17	.34	.25	.68	.63	.655
Child 2	.27	.07	.17	.16	.41	.285
Child 3	.01	.08	.045	.11	.06	.085
Child 4	x	.14	.14	.10	.13	.115
Child 5	.01	.04	.025	.03	.05	.04
Child 6	.01	.04	.025	.04	.08	.06
Child 7	.04	.08	.06	.09	.10	.095
Child 8	.03	.08	.055	.06	.05	.055
Child 9	x	.06	.06	x	.22	.22
Child 10	.02	.01	.015	.02	.03	.025
ALL			.08			.16

Normal urinary arsenic = .015-.020 ppm.

TABLE 2
URINARY ARSENIC (micrograms per liter)

Sampling date	Number of individuals sampled	Group Studied	Minimum	Maximum	Average
6-6-72	19	Ruston School	10	150	81.8
6-7-72	16	Fern Hill School	10	50	20.0
6-7-72	9	Ruston Preschool Children	40	620	270.0
9-12-72		Traverse Study of Ruston and Tacoma within:			
	7	.5 mi. of stack	50	620	300.0
	8	.5 - 1.0 mi. of stack	50	420	190.0
	6	1.0 - 1.5 mi. of stack	40	140	80.0
	5	1.5 - 2.0 mi. of stack	40	100	60.0
	6	2.0 - 2.5 mi. of stack	N.D	50	20.0
	5	2.5 - 3.0 mi. of stack	10	100	46.0
	5	3.0 - 3.5 mi. of stack	10	50	34.0
	10	3.5 - 4.0 mi. of stack	10	110	48.0
10-11-72 10-23-72 10-30-72 11-6-72 11-13-72	14	Ruston Children Average of 5 weekly samples	20	470	99.0
9-18-72	107	Ruston School Children	N.D.	430	81.0
10-25-73	106	Ruston School Children	10	470	55.0
7-19-74	8	Ruston Children (Smelter	10	270	70.0
7-25-74	10	on strike)	10	340	94.0
8-20-74	9	Ruston Children (after	20	680	143.0
8-22-74	10	Smelter strike)	30	630	176.0
6-3-75		Ruston School Children			
	5	Seafood ingestion	30	190	102.0
	36	No seafood ingestion	20	660	87.0
6-3-75		Fern Hill School Children			
	13	Seafood ingestion	10	270	62.0
	48	No seafood ingestion	10	230	25.0
11-17-75	102	Ruston School Children	10	200	40.0
	17	Seafood ingestion	10	150	68.0
	85	No seafood ingestion	10	200	35 0
7-23-thru 8-1-76	10	Ruston School Children for 10 days each	20	890	114.0
8-26 thru 9-1-76	6	Ruston Preschool Children for 7 days each	10	400	122.0
11-30 thru 12-3-76	7 (21 samples)	Ruston School children	60	170	101.9

Normal urinary arsenic = .015-.020 ppm or 15-20 micrograms per liter.
N.D. = not detectable

TABLE 3
URINARY ARSENIC (PARTS PER MILLION) FOR RUSTON SCHOOL CHILDREN

		Date/Day										
	Age	7/23 Fri.	7/24 Sat.	7/25 Sun.	7/26 Mon.	7/27 Tues.	7/28 Wed.	7/29 Thur.	7/30 Fri.	7/31 Sat.	8/1 Sun.	Average
Family No. 1												
Child 1	10	.03	.03	.02	.05	.02	.08	.08	.03	X	X	.043
Child 2	9	.03	.08	.04	.06	.07	.04	.04	X	X	X	.051
Child 3	12	.03	.04	.04	.04	.03	.03	.03	.01	X	X	.031
Family No. 2												
Child 1	8	.16	.11	.05	.08	.13	.26	.11	.08	.10	.07	.115
Child 2	10	.04	.08	.04	.07	.07	.06	.09	.04	.05	.11	.065
Child 3	6	.14	.22	.10	.17	.12	.22	.07	.06	.19	.07	.136
Family No. 3												
Child 1	6	.05	.11	.03 SF	.59	.13	.04	.18	.15	.20	.80	.228
Child 2	9	.09	.10	.09	.06	.07	.18	.17	.06	.17	.19	.118
Child 3	10	.04	.10	.03	.05	.10	.08	.06	.07	.04	.46	.103
Child 4	7	.10	.09	.06 SF	.20	.06	.11	.14	.17	.20	.89	.202
Average		.071	.096	.050	.135	.080	.112	.097	.074	.136	.370	.114

SF = ate salmon
X = no specimen

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NIOSH Project Officer: Loren L. Hatch, DO, PhD
Principal Investigators: Theodore C. Doege, M.D.
Robert H. Wheater, M.S.

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