Bull. Environm. Contam. Toxicol. 26, 131-136 (1981)

Human Exposure to Environmental Trichloroethylene and Tetrachloroethylene: Preliminary Data on Population Groups of Milan, Italy

Giuliano Ziglio

Institute of Hygiene, University of Milan Medical School, Via F. Sforza, 35, I-20122 Milano, Italy

Trichloroethylene (TRI) and tetrachloroethylene (PER), as well as chloroform, trichloroethane, methylchloroform and carbon tetrachloride, were first found in a well of the Milan water system in May of 1975. By August 30, 1979, 83 out of 540 wells were closed because the total concentration of various chlorinated compounds exceeded 250 ug/L, i.e., the work-limit presently set for excluding a single well (GIOVANARDI 1979).

Unofficial estimates set the annual use of TRI and PER in the Milan area at 1500-2000 tons. It is thought that approximately 25% of this amount is discharged into the ground or the subsoil, while the rest is dispersed into the atmosphere.

The Hygiene Institute of the University of Milan Medical School has begun to study the exposure to TRI and PER of an initial group of 24 persons residing in Milan. These two contaminants are present in the tap water consumed in the most highly polluted area of the city at concentrations as high as 150-200 ug/L (GIOVANARDI 1979), and are carcinogenic in animals (NCI 1976; NIOSH/NCI 1978). To measure the actual exposure to TRI/PER, we used the plasmatic level of trichloroacetic acid (TCA), a metabolite common to both molecules.

Numerous findings from occupational exposure (MULLER et al. 1974; FERNANDEZ et al. 1977; MONSTER et al. 1979a,b) consistently show that plasmatic TCA correlates with a cumulative, progressive exposure rather than with a single recent dose, and it is therefore traceable in the biological monitoring of chronic exposures. Tests performed on volunteers expo-

0007-4861/81/0026-0131 $01.20

sed to TRI and PER vapours show that 50-70% of the absorbed TRI is eliminated as TCA and trichloroethanol (TCE), while only about 2% of PER is metabolized and eliminated as TCA. Finally, it should be emphasized that the carcinogenic action of TRI/PER is due to the formation of a primary intermediate oxirane during the process of biotransformation (HENSCHLER 1977; BARTSCH et al. 1979).

MATERIALS AND METHODS

Plasmatic TCA was measured by gas chromatography using a Ni^{63} electron capture detector. Chloroform formed by the decarboxylation of TCA at 90°C was measured. The procedure can measure up to 2 ug of TCA/L and has a coefficient of variation of 3.8% (based on 10 tests: ZIGLIO 1979).

Atmospheric concentrations of TRI/PER were measured by absorption on granular activated carbon followed by extraction with CS_2 and gas chromatographic analysis (NIOSH 1975).

RESULTS

In 24 subjects, the majority of whom were from our Institute, the plasmatic levels of TCA were measured in the following periods: June-July 1978 (S 78), November-December 1978 (W 78), and June-July 1979 (S 79). On the basis of their personal habits and the type of water they drank, the subjects were divided into two groups: Exposed (those who drank water with TRI/PER $\geq$ 50 ug/L), and Non-Exposed (those who drank water with TRI/PER $\leq$ 1 ug/L or who drank other liquids). Results of the tests are given in Table 1.

For the period June-July 1978, the concentrations of TCA are shown to be higher, on the average, in exposed subjects than in Non-Exposed. Repeated tests performed on two subjects during the summer 1978 period did not show any appreciable variations.

In the same subjects, regardless of the type of exposure, the plasmatic levels in winter (W 78) were higher than those in summer (S 78 and S 79). There was no significant difference between the two summer

Table 1. TCA plasmatic levels in subjects with different exposures

Subjects	Exposure	TCA, ug/L		
		S 78	W 78	S 79
1	E	25	80	38
2	E	28	--	35
3	NE	10	38	14
4	E	19	--	22
5	E	41	--	45
6	E	15	--	15
7	E	52	--	66
8	NE	28	--	15
9	NE	12	64	30
10	NE	18	--	31
11	E	21	61	29
12	E	70	--	29
13	NE	11	47	14
14	NE	8	22	6
15	NE	7	39	13
16	NE	26	--	--
17	NE	21	--	15
18	NE	16	--	--
19	NE	15	--	9
20	NE	10	--	--
21	E	42	--	--
22	NE	--	25	--
23	NE	9	--	--
24	E	--	159	--

S 78 = Summer 1978; W 78 = Winter 1978; E 79 = Summer 1979
E = Exposed subject; NE = Non-Exposed subject
COMPARISONS: TCA E vs NE: $p<0.01$; TCA W 78 vs S 78: $p<0.01$
TCA W 78 vs S 79: $p<0.01$; TCA S 78 vs S 79: $p>0.05$

levels (Table 1).

Atmospheric concentrations of TRI and PER were found to be higher in the winter than in the summer. This is particularly true for samples taken during the daytime (Table 2). No substantial differences were observed in the concentrations of TRI and PER in the first set of measurements taken in the downtown area, in the residential suburbs and in the industrial outskirts of Milan (Table 3). However, concentrations of PER in the immediate vicinities of dry-cleaning devices were constantly shown to be

twice those measured in urban areas further away from these sources (20 vs 10, 17 vs 8, 16 vs 5, 26 vs 12 ug/m^3 respectively).

Table 2. TRI and PER ambient air concentrations in Milan (ug/m^3)

PERIOD	TRI		PER	
	day	night	day	night
NOV 78-JAN 79	76	10	71	16
	50	43	15	38
	74	17	60	20
	10	35	86	31
	60		61	
FEB 79-MAR 79	6	34	6	12
	5	40	10	21
	25	14	12	13
	8	9	14	11
		7		7
JUNE 79	15	5	23	7
	8	7	8	9
	18	15	22	13
AUGUST 79	1	2	3	3
	4	2	1	1

day: 09.00 am to 07.00 pm night: 07.00 pm to 09.00 am

DISCUSSION

These initial findings show the validity of plasmatic TCA as a tracer for monitoring chronic exposure to TRI and PER. In fact, at comparable exposures (S 78 and S 79), no significant statistical variations in TCA levels are noticed; when there is an increase in exposure (W 78), the level of TCA increses. In addition, the plasmatic level of the non-exposed subjects show that water is not the only source of TRI/PER. These solvents can be taken in through food (McCONNELL et al. 1975), through the respiratory system (atmospheric contamination) and by occasional domestic exposure (home cleaning with solvents and use of other products containing TRI and PER).

Table 3. TRI and PER ambient air concentration (ug/m^3) by different areas (duration of sampling: 4-5 daytime hours)

	downtown Milan	residential suburbs	industrial outskirts
TRI	6	-	7
	34	-	15
	11	14	10
	15	12	11
	8	5	9
	11	20	8
	9	11	7
	25	10	14
	7	4	4
PER	6	-	14
	13	-	16
	12	19	15
	16	13	17
	10	5	16
	11	21	9
	9	11	11
	25	10	16
	7	4	15

With regard to the differences between exposed and non- exposed subjects, it should be pointed out that the sample was too small to take into consideration the age, sex, body build, or personal habits (smoking and alcohol consumption) of the subjects tested, nor the separate levels of TRI and PER in the water consumed. For the most part, the levels of TCA were indicative of exposure to TRI, since only a small percentage of PER is biotransformed, as opposed to 50-70% of TRI. Plasmatic level of TCE, a metabolite only of TRI, can be used to distinguish between exposure to TRI and exposure to PER.

The importance of TRI/PER intake through the respiratory system is documented by the large increase from summer to winter consistently observed in the plasmatic TCA of the same subjects. While intake by drinking water affects only a part of the population,

exposure to contaminated air represents an equal, if not greater, risk for all residents.

Research is now being carried out in two different areas of Milan. Therefore, additional data on TCA plasmatic levels and environmental concentrations of TRI and PER will make it possible to confirm and better understand the role of various sources of TRI and PER.

Acknowledgments. I thank G. M. Fara for revising the manuscript and B. Elbaum for her skillful translation.

REFERENCES

BARTSCH, H., C. MALAVILLE, A. BARBIN and G. PLANCHE: Arch. Toxicol. 41, 249 (1979).

FERNANDEZ, J. G., B. E. HUMBERT, P. O. DROZ and J. R. CAPEROS: Br. J. Ind. Med. 34, 43 (1977).

GIOVANARDI, A.: Riv. Ital. Ig. 39, 323 (1979).

HENSCHLER, D.: J. Environ. Path. Toxicol. 1, 125 (1977).

McCONNELL, G., D. M. FERGUSON and C. R. PEARSON: Endeavour 34, 13 (1975).

MONSTER, A. C., G. BOERSMA and W. C. DUBA: Int. Arch. Occup. Environ. Health 42, 283 (1979a).

MONSTER A. C., G. BOERSMA and G. H. STEENWAG: Int. Arch. Occup. Environ. Health 42, 303 (1979b).

MULLER, G., M. SPASSOWSKI and D. HENSCHLER: Arch. Toxicol. 32, 283 (1974).

NATIONAL CANCER INSTITUTE (NCI): Carcinogenesis Technical Report Series. No. 2: 'Carcinogenesis Bioassay of Trichloroethylene'. Bethesda, Md 20014, June 14 (1976).

NATIONAL INSTITUTE OCCUPATIONAL SAFETY HEALTH (NIOSH): Manual of Analytical Methods. NIOSH New Publication No. 75-121 (1975).

NATIONAL INSTITUTE OCCUPATIONAL SAFETY HEALTH / NATIONAL CANCER INSTITUTE (NIOSH / NCI): Current Intelligence Bulletin no. 21. Tetrachloroethylene. DHEW (NIOSH) Publication no. 78-112, January 20 (1978).

ZIGLIO, G.: Ig. Mod. 72, 876 (1979).