lic beverage the previous day. The coefficient of agreement (Kappa) was 73% [14].

Blood lead analysis

Venous blood samples were taken with a vaccutainer® free of heavy metals, containing sodium heparin. The samples were stored at a maximum of 4° Celsius until they were mailed to an analytical laboratory. Mailing was without cooling (average mailing time 1–2 days), but never over a weekend. Samples from the same day and/or the same medical practice were distributed to different analytical batches. The laboratory and the analytical method were identical to those used in the 1984 Western Switzerland blood lead study (and its repetition in 1991). This allowed a comparison of the two data sets. Methods, and quality control data showing the data's precision and accuracy, have been described previously [4,5,15].

Place of residence

Each subject's data was matched with the hourly car frequency on the street of his/her residence. This information was provided by the noise emission registry of Basle [16].

Statistical analysis

The data were analysed with the SPSS-X software package. Blood lead concentrations were distributed log-normally, and the analyses were therefore carried out on logarithms of blood lead concentrations. The results of statistical analysis are reported as geometrical means ($\bar{x}_g$). We used multiple linear regression analysis to control for important confounders. Discrete independent variables were coded as dummy variables. For model building the forced entry method was selected: gender and age were introduced first, followed by other variables according to their predictive value in univariate regression analysis. No interaction terms were introduced into the model, because analysis of the data did not reveal any major interactions for the determinants of interest. Results are reported as cumulated R^2 and as partial regression coefficients with associated t-statistics.

Results

The results of the crude analysis are presented in table 1. The average geometrical mean blood lead level in the sample was 0.33 ± 0.19 µmol/l whole blood, men having an age-adjusted average blood level about 30% above women (men 0.37 µmol/l; women 0.29 µmol/l). Within each age/sex category participants had an arithmetical average blood lead level 30% lower than subjects in a study of blood lead in Western Switzerland examined approximately 5 years earlier (figure 1) [4,5].

(µmol/l) by different predictor variables [1].

	Male			Female		
	N	$\bar{x}_g$	SD_g	N	$\bar{x}_g$	SD_g
Occupation [2]						
≥ 50% in painting-, printing-, construction-, metal-processing industry	33	0.51	0.21	–	–	–
else	153	0.36	0.16	–	–	–
	$t = 4.4$; $p \leq 0.0001$					
alcohol intake						
never	17	0.29	0.16	28	0.27	0.19
seldom	121	0.34	0.17	161	0.28	0.16
regularly (nearly every day or more)	93	0.44	0.20	50	0.35	0.17
	$F = 12.8$; $p \leq 0.0001$			$F = 6.5$; $p = 0.0017$		
smoking						
never	64	0.31	0.14	106	0.28	0.17
former	74	0.38	0.21	52	0.28	0.18
casual	13	0.41	0.23	8	0.26	0.17
regular	79	0.41	0.18	70	0.34	0.16
	$F = 4.9$; $p = 0.0025$			$F = 4.08$; $p = 0.0076$		
place of residence						
Basle	199	0.38	0.18	208	0.30	0.17
Riehen	33	0.32	0.20	31	0.25	0.13
	$t = 2.1$; $p = 0.02$			$t = 1.44$; $p = 0.08$		

[1] Age-adjustment did not influence comparisons and geometric means.
[2] comparison only among males, since no females in exposed groups.

Multiple linear regression analysis

Gender, age, occupation, alcohol consumption and smoking were identified as having an independent predictive value in multiple linear regression analysis (table 2). In the crude analysis participants living in the area of Basle had an average blood lead level 20% higher than those living in Riehen, a suburban community without much traffic (table 1). Place of living and hourly car frequency were, however, not significant independent predictors when they were introduced last into the multiple linear regression model.

Introducing occupation and alcohol consumption into the model considerably reduced the estimated regression coefficient for gender. Since gender differences in smoking prevalence were less prominent, and owing to its correlation with alcohol intake, entering smoking into the model did not further decrease the regression coefficient for gender. Part

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Alcohol consumption and other lifestyle factors: Avoidable sources of excess lead exposure

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Blood lead studies in different countries have shown a downward trend in the population's lead burden parallelling the reduction of lead in petrol. Between 1976 and 1980 the National Health and Nutrition Survey found an average blood lead reduction of 37% in the U.S. In the same time the total amount of lead used in petrol production decreased from approximately 53000 to 24000 tons per quarter[1]. A correlation between lead content of all petrol sold in Massachusetts between April 1979 and April 1981 and the concentration of lead in umbilical cord blood from births at a Boston hospital could be demonstrated[2]. In Sweden a follow-up study found an average decrease in blood lead of 34% between 1980 and 1984 for residents in the inner city area of Stockholm[3].

The first general population survey on blood lead in Switzerland, conducted in 1984, revealed average blood concentrations comparable to those of other European and North American populations at that time[4,5]. Since then, lead emission has decreased drastically in Switzerland. Emission from traffic, accounting for 65 to 75% of total lead output (680 tons in 1984), decreased by about 40%[6]. However, lead in airborne particulate matter and in soil still exceeds the legal limits in Basle, an urban industrialised agglomeration in northwestern Switzerland (the Swiss legal ambient air quality standard for lead is 1 $\mu g/m^3$ (annual mean) in airborne particulate matter and 100 $\mu g/(m^2 \cdot day)$ (annual mean) in dust precipitation). It was therefore of interest to determine the population's mean lead exposure and the risk factors associated with lead absorption.

A population's basal lead level – as a result of lead in atmosphere, food and beverages – can be lowered by *collective* preventive actions. The first aim of this study was to determine the effects of the achieved reduction in petrol-related lead emissions on the population's exposure, and to compare the data with previous national and international data. The second aim was to identify environmental and lifestyle factors which result at the *individual* level in blood lead concentrations exceeding the basal level[7–9].

Materials and methods

Population

Blood samples were available from participants in a study on cardiovascular risk factors and nutritional behavior[10].

In August 1989 a random sample of residents of Basle aged 20 to 74 years was selected from the register of inhabitants. Turkish-speaking persons were excluded. A letter with an invitation to participate was followed by telephone contact. Only 3% of all subjects could not be contacted. 471 persons participated (59%); there was no difference between the sexes. Participation was lower for persons aged 20 to 24 years (45%) and for foreign residents (52%). The sample's sex and nationality distribution was representative of Basle's population aged 20 to 74, Turkish residents excluded. The age distribution for males, however, was not representative of Basle's male population, with a higher proportion of older subjects in the sample.

Data collection

Between October 1989 and March 1990 participants were examined in different medical practices. Examination included: blood pressure, height and weight, and venous blood sample for determination of cholesterol levels and lead. A questionnaire derived from previous Swiss studies[11–13] requesting information on demographic factors and nutrition, as well as smoking and drinking habits, was filled in by the participants. The participants were subdivided into two different categories on the basis of their job-descriptions: those working less than half-time, or working in occupations where lead exposure is unlikely, and those working at least half-time in occupations where it is possible or known from occupational medicine that lead exposure may occur.

Questions on alcohol consumption were validated by comparing the general statement with the twenty-four hour recall. 124 out of 143 participants (87%) who had reported daily alcohol consumption on the food frequency questionnaire also reported having consumed an alcoholic beverage the day before the examination. 44 of 45 self-reported

Tab. 4. Mean lead content (µg/l) by colour of wine on Basle market.

Colour	N	mean	SD	range
red wine	81	53	16	21–102
rosé	5	44	12	18–54
white wine	13	31	10	23–53
Total	99	50	17	18–102

red versus white wine: $t = 6{,}86$; $p < 0.001$.

lead in wine on sale in Basle. Wine was sampled from five appropriate supermarkets in Basle. The samples represented mainly the lowest price segment, which includes two thirds of the total amount of wine sold in Basle. We determined the lead concentration of 99 wines of different origin and colour, and with different types of bottle cap. The wines were mainly from Switzerland, Italy, France, Spain, and Algeria. The analytical method and quality control data have been described[17]. The average lead concentration was 50 ± 17 µg/l. Red wine contained significantly more lead than white wine (table 4). The 31 Italian red wines contained significantly more lead (62 ± 15 µg/l) than the 50 red wines from other countries. When wine in bottles with different types of closure was compared, essentially no differences in the lead content were found if the sample analysed was taken directly from the bottles. However, high lead concentrations of up to more than 300 µg/l were found for wine bottles with tin-lead closures if the wine was poured into a glass before being analysed. Lead corrosion products on the edge of the bottle, coming from the closure, were obviously dissolved in the wine.

Discussion

Average blood lead levels in Basle ($\tilde{x}_g = 0.33 \pm 0.19$ µmol/l) were low compared to those found in Western European and North American cities some 5 to 10 years ago (0.5–1.0 µmol/l)[18]. The same low level was, however, already found in inner city residents of Stockholm in 1980. Daily nutritional lead intake in Switzerland and Sweden is estimated to be 25–30 µg[3,19], whereas in Great Britain – with an average blood lead level twice today's Basle level – it is 75 µg[20,21]. In addition, lead in drinking water is neglectably low in Basle (well below 25 µg/l), and lead in can solder is legally limited (< 10% lead)[19,22–24].

The most plausible reason for Basle's low blood lead level, though, seems to be the fact that the study was conducted at least five years later than comparable investigations, namely after a period of drastically reduced airborne lead emission in Switzerland. A comparison of the Basle results with data from the 1984 Western Switzerland blood lead study supports this hypothesis[4,5] (figure 1). In terms of environmental and demographic determinants, comparability of the populations in Basle and Western Switzerland is a valid assumption. It is true that a higher percentage of participants in the Basle study did not consume alcohol, and this may have accounted in part for the lower exposure. However, a comparison of the Basle results with only the urban subjects of the study in Western Switzerland would have resulted in an even greater difference since the latter included a rural population with lower exposure. The comparability assumption is further validated by the repetition of the survey in Western Switzerland in 1988/89, which resulted in blood lead concentrations comparable to the levels found in our study[25]. Furthermore, in 1984 the Swiss results were not low in an international comparison. In view of all these facts, the reduction in lead emissions since 1984 is the most plausible reason for today's low lead levels in Switzerland.

The association between lead and *gender, age, occupation, smoking, and alcohol consumption* confirmed the results of earlier studies[4,5,26–30]. *Men's* higher blood lead levels may partly be due to their higher hemoglobin concentration. The *age* dependency – though small – correlated with comparable data and is probably the result of accumulation and the long half-life of lead in bone.

Sources of lead exposure in *construction, painting, printing, and metal-processing* are lead in dust and soil, old paint or lead containing alloys[26,31]. Since lead type is no longer used in the printing industry, printers' high blood lead levels might be a sign of high exposure in the past.

The finding that alcohol consumption and *smoking* – despite their correlation – were both predictors of blood lead supports the results of previous studies that identified them as independent risk factors[4,5,18,20,29,32,33]. Lead persistence in soil as a result of the widespread use of lead arsenate as an insecticide in tobacco fields in the past might still give rise to lead dust contamination of tobacco plants. But the average lead content of a cigarette is estimated to be 1–2 µg, which is too small to account for the total difference observed between the blood lead levels of smokers and non-smokers[33,34]. The smokers' higher hand-to-mouth-activity, leading to a higher lead dust absorption is therefore another possible source of exposure. Occupationally exposed smokers have been shown to have the highest blood lead levels if they smoke at their working place[9,21].

Regular *alcohol* consumption (at least one glass of wine a day) has been found in other studies to increase the basal lead level by 30–50%[29,35]. Grandjean and co-workers concluded on the basis of their multivariate model that a daily intake of 1.35 cl of pure alcohol (one glass of wine) increases the blood lead level by 0.025 to 0.050 µmol/l whole

Tab. 2. Multiple linear regression model on log(Pb-B) as dependent variable:
$\log(\text{Pb-B}) = b_0 + b_1\,(\text{gender}) + b_2\,(\text{age}) + b_3\,(\text{occupation}) + b_4\,(\text{alcohol intake}) + b_5\,(\text{tobacco consumption}) + e$

N = 471	b	SEb	t/p	R^2 cumulation	F_{change}/p
sex 0 = female; 1 = male	0.056	0.016	3.4/0.0007	7.0	36.5/< 0.0001
age (years)	0.002	0.001	3.9/0.0001	9.8	15.7/0.0001
occupational exposure 0 = no; 1 = yes	0.139	0.031	4.5/< 0.0001	14.5	26.6/< 0.0001
alcohol intake					
seldom	0.034	0.026	1.3/0.19	19.6	19.0/< 0.0001
regularly (reference: never)	0.114	0.028	4.1/0.0001		
tobacco consumption					
1–20 cigarettes/day	0.048	0.019	2.6/0.009	23.0	11.2/0.0009
> 20 cigarettes/day	0.095	0.028	3.4/0.0009		
various tobacco products (reference: never)	0.136	0.041	3.3/0.0009		

of the difference in blood lead levels between men and women was therefore due to the fact that men were occupationally more often exposed and drank more alcohol than women. But even if four confounders were taken into account men still had an average blood lead level 6% higher than that of women.

Age was a robust, but very weak predictor. Logarithmic transformation of the variable did not substantially improve its predictive value.

The high blood lead level of workers in painting or printing, construction or metal-processing industries was also partially explained by their high alcohol and tobacco consumption. But an unconfounded average blood lead level 14% above the level of all other participants was estimated to be associated with occupational exposure.

If smoking status (see table 1) was entered into the model, the regularly smoking subjects had an unconfounded average blood lead level 8% above the level of those who never smoke. Smokers were further subdivided into persons smoking 1–20 cigarettes/day, > 20 cigarettes/day, and several tobacco products or something other than cigarettes (table 2). The partial regression coefficients were significant for all three subgroups of tobacco consumption, indicating a dose-effect relationship. Five men who smoked pipes had a high blood lead concentration ($\bar{x}_g = 0.69$ µmol/l, 95% CI = 0.381–1.240).

Subjects who drank alcohol regularly had blood lead concentrations on average 12% higher than those of abstinent subjects after controlling for confounding.

Data on the average alcohol intake was further stratified by the reported consumption of alcoholic beverages the day before the examination (table 3). Regularly-drinking participants who had consumed only wine the previous day had a higher blood lead level than persons who had taken only beer, although the difference was not significant (probably owing to the small number of participants in both subgroups). The alcoholic beverage consumed the previous day by regularly-drinking subjects in most cases represents the preferred alcoholic beverage, therefore giving an idea of the effect depending on the kind of alcohol.

Tab. 3. Sex specific geometrical mean blood lead concentrations (µmol/l) by average alcohol intake and by previous day's alcohol consumption [1].

alcohol intake	Male			Female		
	N	$\bar{x}_g$	SD_g	N	$\bar{x}_g$	SD_g
"never" and "nothing yesterday"	17	0.29	0.16	27	0.27	0.19
"regularly" and "something yesterday":						
– beer only yesterday	18	0.35	0.21	6	0.30	0.08
– wine only yesterday	30	0.48	0.20	26	0.37	0.17
– various alcoholic beverages yesterday	32	0.50	0.18	8	0.39	0.15
	F = 8.0; p < 0.0001			F = 3.4; p = 0.02		
Scheffe multiple comparison p < 0.05	"wine only" vs. "never"; "different" vs. "never"; and vs. "beer only"			not significant		

[1] 4 participants with regular alcohol intake had consumed cider or liqueurs the previous day; this limited number does not allow any inference about the effect of these beverages.

Lead in wine

The observed association between alcohol consumption and blood lead levels led to the analysis of

Despite today's low lead levels there are several reasons to strive for the lowest possible lead exposure in the general population. The lead levels observed today still exceed the lead content found in human bones from pre-industrial cultures. Lead serves no function in the human organism. Adverse health effects have been identified at blood lead concentrations observed in the general population; for these effects no threshold has been found as yet. Little is known about interactions with other environmental pollutants[42,43]. The successful reduction of blood lead levels in the general population over the last decade is an excellent and motivating example for the introduction of measures for environmental protection, like the introduction of unleaded petrol. The results of this study further highlight the importance of the prevention of alcohol abuse and smoking. Pregnant women are an important target group. Future research is required to investigate whether alcohol, smoking, and lead have synergistic effects on the fetus.

Summary

Lead concentration in whole blood of a representative sample of 471 subjects aged 20 to 74 years and living in Basle was determined in 1989/90 by flame atomic absorption spectrophotometry. The participants in the cross-sectional study filled in a questionnaire on demographic factors, nutrition and drinking habits. The age-adjusted geometrical mean blood lead level was 0.38 ± 0.19 µmol/litre of whole blood for males, and 0.29 ± 0.17 µmol/litre for females. The average lead burden was about 30% below the mean concentration found in a comparable population in Western Switzerland five years earlier. Lead emissions in Switzerland decreased by about 40% from 1984 to 1990 through restriction of lead in petrol. This offers the most plausible explanation for the low blood lead levels found in Basle. Sex, age, occupation (employment in painting or printing, construction or the metal processing industry), smoking and alcohol intake (especially wine consumption) were identified as independent blood lead predictors in a multiple linear regression analysis. Participants who consumed alcohol daily had blood lead concentrations on average 12% higher than those of abstinent subjects. Regular smokers had an unconfounded average blood lead level 8% above that of people who never smoked. Place of residence and hourly frequency of cars in that area were not identified as independent predictors in the model. The analysis of 99 different wines on the market in Basle showed an average lead concentration of 50 ± 17 µg/litre wine. Compared to an estimated 25 µg daily nutritional intake of lead in Switzerland the value appeared to be high. The hypothesis is presented that there is a causal relationship between lead in wine and lead in blood.

Résumé

Consommation d'alcool et autres facteurs liés au style de vie: Sources évitables d'exposition au plomb

La plombémie d'un échantillon représentatif de la population (n = 471) âgée de 20 à 74 ans et vivant à Bâle a été déterminée par spectrophotométrie à flamme en 1989–1990. Les participants à cette étude transversale ont rempli un questionnaire relevant des données démographiques, les habitudes en matière de nutrition, de consommation alcoolique et tabagique. La moyenne géométrique de la plombémie, ajustée pour l'âge, était de $0,38 \pm 0,19$ µmol/l pour les hommes et de $0,29 \pm 0,17$ µmol/l pour les femmes. Ces valeurs moyennes étaient d'environ 30% inférieures à celles mesurées dans une population comparable de Suisse orientale cinq ans auparavant. Les émissions de plomb en Suisse ont diminué d'environ 40% entre 1984 et 1990, suite à la diffusion de plus en plus large des carburants sans plomb, ce qui représente l'explication la plus plausible aux valeurs de plombémie basses mesurées à Bâle. Le sexe, l'âge, la profession (peintres en bâtiment, imprimeurs, employés de la construction ou de la métallurgie), les habitudes tabagiques et la consommation d'alcool (de vin en particulier) ont été identifiés comme des prédicteurs indépendants de la plombémie dans une analyse de régression linéaire multiple. Les consommateurs quotidiens d'alcool avaient en moyenne une plombémie de 12% supérieure aux abstinents et les fumeurs habituels, une valeur de 8% plus élevée que les sujets n'ayant jamais fumé. Le lieu de résidence et la densité du trafic automobile à proximité du domicile n'ont pas été identifiés comme des prédicteurs indépendants. L'analyse de 99 vins disponibles sur le marché bâlois a révélé une concentration de plomb moyenne de 50 ± 17 µmol/l de vin. Comparée à un apport par les aliments estimé à 25 µg/j en Suisse, cette valeur est élevée. L'hypothèse d'une relation causale entre plombémie et consommation de vin est émise.

Zusammenfassung

Vermeidbare Quellen der Bleizufuhr: Alkoholkonsum und individuelle Einflussfaktoren

Im Rahmen einer Querschnittstudie wurde 1989/90 die Bleibelastung der 20–74jährigen Bevölkerung von Basel-Stadt bestimmt. Für die Gehaltsanalyse mittels Atomabsorptions-Spektrophotometrie wurde einer repräsentativen Stichprobe (n = 471) venöses Blut entnommen. Mittels Fragebogen wurden soziodemographischer Hintergrund, Ernährungsgewohnheiten, Rauchen und Alkoholkonsum erfasst. Die alterskorrigierte mittlere (geometrische) Bleibelastung des Kollektivs betrug 0.38 ± 0.19 µg/l Vollblut für Männer und 0.29 ± 0.17 µmol/l für Frauen. Die mittlere Bleibe-

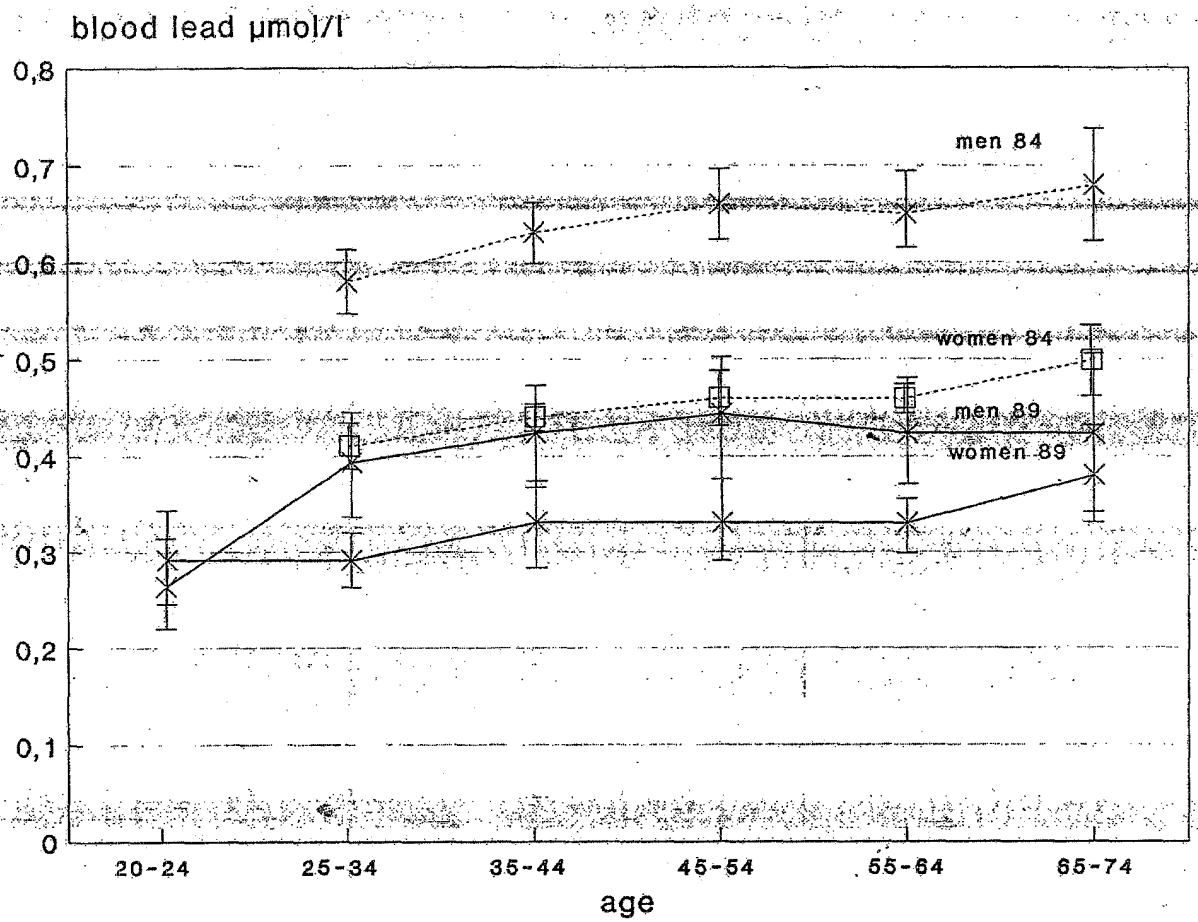

Fig. 1. Arithmetical means for blood lead levels (± one standard error) by sex and age decade for Basel, sample (1989–90) compared to values for Western Switzerland collected in 1984.

blood[36]. With an average blood lead level of 0.36 µmol/l in Basle, and assuming that regularly-drinking subjects consume a daily average of 1–2 glasses of wine, the 12% increase in average lead level as compared to abstinent persons is in good agreement with the above-mentioned results.

Previous studies have also found a stronger effect of wine than of beer[37,38]. Wine is, furthermore, the most frequently consumed alcoholic beverage in Switzerland[39]. The analysis of 99 different wines on Basle's wine market showed an average lead concentration of 50 ± 17 µg/l, which appeared to be high compared to an estimated 25 µg daily nutritional lead intake in Switzerland[19]. Assuming that lead from alcoholic beverages is absorbed to a higher degree (20%) than lead from food (10%)[40] and that about 125 000 Swiss consume at least 1 litre of wine per day, lead in wine is likely to be a direct cause of high lead levels in regularly-drinking subjects.

The *place of residence* had no independent predictive value in this urban population. A difference between rural and urban subjects' blood lead levels was shown in earlier studies[4,5,20,28,32], but no difference existed between urban subjects living on highways or inside streets[3]. To identify the effect of traffic density on blood lead, information about the indoor-/outdoor ratio for lead and about the time spent at home is needed.

Conclusions

The low basal blood lead level found in this study population is expected to further decrease with systematic lead reduction in petrol. Because there are lead reservoirs in the skeleton, and owing to the long half-life this decrease might pause at some intermediate blood lead level. An individual can reduce his/her blood lead level primarily by reducing alcohol and tobacco consumption. The neck of a wine bottle should be cleaned before pouring out wine. In Switzerland a provisional legal limit of 0.3 mg/kg wine has been set based on the described results, and caps containing lead alloys must be replaced by 1994[41]. The issue of lead in wine has been carefully investigated by the U.S. FDA. Major efforts to reduce lead in wine and to inform the public about safety aspects are currently being undertaken.

33 *Brockhaus A, Freier I, Ewers U, Baginski B, Kraemer U, Dolgner R.* Epidemiologische Untersuchungen zur ökologischen Bleibelastung von männlichen Grossstadtbewohnern in Nordwestdeutschland. Int Arch Occup Environ Health 1980; *46*:59–70.
34 *Watanabe T, Kasahara M, Nakatsuka H, Ikeda M.* Cadmium and lead contents of cigarettes produced in various areas of the world. Sci Total Environ 1987; *66*:29–37.
35 *Bortoli A, Fazzin G, Marchiori M, Trabuio G, Zotti S.* High levels of Pb-B and Cd-B induced by alcohol and smoke. In: Lekkas TD. Heavy metals in the environment: International conference, Athens, September 1985. Edinburgh, CEP Consultants Ltd., 1985; 439–441.
36 *Grandjean P, Olsen NB, Hollnagel H.* Influence of smoking and alcohol consumption on blood lead levels. Int Arch Occup Environ Health 1981; *48*:391–397.
37 *Elinder CG, Lind B, Nilsson B, Oskarsson A.* Wine – an important source of lead exposure. Food Add Contam 1988; *5*:641–644.
38 *Sherlock JC, Pickford J, White GF.* Lead in alcoholic beverages. Food Add Contam 1986; *3*:347–354.
39 *Fahrenkrug H, Mueller R.* Gesünder ins Jahr 2000? Eine Untersuchung der Schweizerischen Fachstelle für Alkoholprobleme (SFA) zu Gesundheit und Alkoholkonsum der 15–74jährigen Schweizer Bevölkerung. ISPA-Press, 1990.
40 *Smart GA, Pickford J, Sherlock JC.* Lead in alcoholic beverages: a second survey. Food Add Contam 1990; *7*: 93–99.
41 Bundesamt für Gesundheitswesen: Bulletin, Bern 1992; 10.
42 *Levin R.* Reducing lead in drinking water: a benefit analysis. Office of Policy, Planning and Evaluation. Washington, U.S. Environmental Protection Agency, 1986.
43 *Royce SE, Needleman HL.* Case studies in environmental medicine. Lead toxicity. Atlanta, U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1990.

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lastung betrug rund 30% weniger als 1984 in einem vergleichbaren Westschweizer Kollektiv. Seit 1984 gingen die Bleiemissionen durch den Verkehr in der Schweiz um 40% zurück. Somit ist der erfolgreich gesenkte Bleiausstoss die wahrscheinlichste Ursache für die heute vergleichsweise niedrige Belastung in Basel. In einer multiplen linearen Regressionsanalyse wurden Geschlecht, Alter, Beruf (Tätigkeit in Metallverarbeitung, Maler-, Druckerei- und Baugewerbe), Rauchen und Alkoholkonsum (insbesondere Wein) als unabhängige Determinanten identifiziert, die beim Einzelnen zu einer Blutbleibelastung über das durch die Umwelt gegebene Grundmass hinaus führen können. Teilnehmer, die täglich Alkohol konsumierten, hatten durchschnittlich 12% höhere Blutbleiwerte als abstinente Personen. Regelmäßige Raucher hatten 8% höhere mittlere Blutbleiwerte als Personen, die nie geraucht hatten. Das Verkehrsaufkommen an der Wohnadresse konnte nicht mit dem Blutbleigehalt assoziiert werden. Die Analyse von 99 Weinen des Basler Marktes zeigte in der Tat, dass der mittlere Bleigehalt mit 50 ± 17 µg/l hoch ist, verglichen mit der für die Schweiz geschätzten täglichen Nahrungs-Bleizufuhr von 25 µg. Dies deutet auf einen Kausalzusammenhang zwischen Blei im Wein und – bei regelmässigem Weinkonsum – im Blut hin. Ein individueller Ansatzpunkt für die Prävention einer unnötig hohen Bleibelastung scheint damit identifiziert.

References

1 *Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG.* Chronological trend in blood lead levels between 1976 and 1980. N Engl J Med 1983; *308*:1373–1377.

2 *Rabinowitz MB, Needleman HL.* Petrol sales and umbilical cord blood lead levels in Boston, Massachusetts. Lancet 1983; *1*:63.

3 *Elinder CG, Friberg L, Lind B, Nilsson B, Svartengren M, Oevermark I.* Decreased blood lead levels in residents of Stockholm for the period 1980–1984. Scand J Work Environ Health 1986; *12*:114–120.

4 *Rickenbach M, Wietlisbach V, Berode M, Guillemin M.* La plombémie en Suisse en 1985: résultats de l'enquête MONICA. Soz Präventivmed 1987; *32*:87–90.

5 *Berode M, Wietlisbach V, Rickenbach M, Guillemin M.* Lifestyle and environmental factors as determinants of blood lead levels in a Swiss population. Environ Res 1991; *55*:1–17.

6 BUWAL Bundesamt für Umwelt, Wald und Landschaft. Vom Menschen verursachte Schadstoff-Emissionen in der Schweiz 1950–2010. Schriftenreihe Umweltschutz 1987; *76*.

7 *Roberts J, Mahaffey KR, Annest JL.* Blood lead levels in general populations. In: Mahaffey KR. Dietary and environmental lead: human health effects. Amsterdam, Elsevier Science Publishers, 1985; *355*:72.

8 *Hunter J.* The distribution of lead. In: Mahaffey KR. Dietary and environmental lead: human health effects. Amsterdam, Elsevier Science Publishers, 1985; 96–126.

9 *Lehnert G, Szadkowski D.* Die Bleibelastung des Menschen. Weinheim, Verlag Chemie 1983; 20–48.

10 *Bodenmann A, Ackermann-Liebrich U, Keller U.* Fleischkonsum und Serum-Cholesterinkonzentration. Dtsch Med Wschr 1991; *116*:1089–1094.

11 *Zemp E.* Frauen und Gesundheit. Ergebnisse der Gesundheitsbefragung SOMIPOPS. Dissertation Medizinische Fakultät der Universität Basel 1988.

12 *Muster E.* Zahlen und Fakten zu Alkohol- und Drogenproblemen 1988. Lausanne, Schweizerische Fachstelle für Alkoholprobleme 1988.

13 MONICA-Suisse. Première enquête de population dans les cantons de Vaud, Fribourg et Tessin 1984–1986. Soz Präventivmed supp 1987; *32*:52–86.

14 *Cohen J.* A coefficient of agreement for nominal scales. Educational and Psychological Measurement 1960; *20*:37–46.

15 *Mitchell DG, Ryan FJ, Aldous KM.* The precise determination of lead in whole blood by solvent extraction-atomic absorption spectrometry. Atomic Absorption Newsletter 1972; *11*:120–121.

16 Grüner und Partner AG. Verkehrslärm. Lärmbelastungsplan des Kantons Basel-Stadt. Basel 1987.

17 *Probst-Hensch N, Wagmann M, Herrmann A.* Monitoring-Programm Schwermetalle in Lebensmitteln. IV. Blei in Weinen auf dem Schweizer Markt. Teil A: Analytische Qualitätskontrolle. Mitt Gebiete Lebensm Hyg 1991; *82*:159–173.

18 *Grandjean P.* Blood lead concentrations reconsidered. Nature 1981; *291*:188.

19 *Knutti R, Zimmerli B.* Untersuchung von Tagesrationen aus schweizerischen Verpflegungsbetrieben. Mitt Gebiete Lebensm Hyg 1985; *76*:206–232.

20 *Quinn M.* Factors affecting blood lead concentrations in the UK: results of the EEC Blood Lead Surveys, 1979–1981. Int J Epidemiol 1985; *14*:420–431.

21 *Moore MR.* Sources of lead exposure. In: Lansdown R, Yule W. The lead debate: the environment, toxicology and child health. London, Sydney, Croom Helm Ltd. 1986; 96–126.

22 Kantonales Laboratorium Basel-Stadt. Schwermetallgehalte im Trinkwasser. In: Die Durchführung der Lebensmittelkontrolle in der Schweiz im Jahre 1984. Mitt Gebiete Lebensm Hyg 1984; *75*:312–314.

23 *Elwood PC, Phillips KM, Lowe N, Phillips JK, Toothill C.* Hardness of domestic water and blood lead levels. Human Toxicol 1983; *2*:645–648.

24 *Mahaffey KR.* Factors modifying susceptibility to lead toxicity. In: Mahaffey KR. Dietary and environmental lead: human health effects. Amsterdam, Elsevier Science Publishers, 1985; 373–419.

25 *Rickenbach M, Wietlisbach V, Berode M, Guillemin M, Probst-Hensch N.* Moins de plomb dans l'essence, moins de plomb dans le sang. Submitted for publication to Bundesamt für Gesundheitswesen: Bulletin.

26 *Olsen NB.* Indicators of lead exposure in an adult Danish suburban population. Dan Med Bull 1981; *28*:168–176.

27 *Huel G, Boudène C, Jouan M, Lazar P.* Assessment of exposure to lead of the general population in the French community through biological monitoring. Int Arch Occup Environ Health 1986; *58*:131–139.

28 *Mahaffey KR, Annest JL, Roberts J, Murphy RS.* National estimates of blood lead levels: United States, 1976–1980. N Engl J Med 1982; *307*:573–579.

29 *Pocock SJ, Shaper AG, Walker M, et al.* Effects of tap water lead, water hardness, alcohol, and cigarettes on blood lead concentrations. J Epidemiol Commun Hlth 1983; *37*:1–7.

30 *Grandjean P, Hollnagel H, Hedegaard E, Christensen TM, Larsen S.* Blood lead-blood pressure relations: alcohol intake and hemoglobin as confounders. Am J Epidemiol 1989; *129*:732–739.

31 *Grandjean P.* Aspekte der Arbeitshygiene im Baugewerbe. In: WHO Regionalbüro für Europa. Kopenhagen, EURO-Berichte und Studien 1986.

32 *Ewers U, Brockhaus A, Dölgner R, et al.* Blutblei- und Blutcadmiumkonzentrationen bei 55–66jährigen Frauen aus verschiedenen Gebieten Nordrhein-Westfalens. Entwicklungstrends 1982–1988. Zbl Hyg 1990; *189*:405–418.