

Environmental health effects of exposures originating from the workplace

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I will focus on diseases occurring from workplace exposures which also cause environmental health risks to residents nearby with three examples

- asbestos
- arsenic
- dioxin

Asbestos was the first established workplace exposure leading to environmental health risks from the workplace, in particular malignant mesothelioma

- Mesotheliomas occur in persons living near work sites including near asbestos mines, asbestos factories, and shipyards, due to drifting of asbestos dust.
- Mesotheliomas occur in spouses and children due to asbestos dust coming home on workers clothes.

Asbestos use is not declining

The asbestos disease epidemic: here today, here tomorrow.

Cullinan P, Pearce N. Thorax. 2012 Feb;67(2):98-9.

“Global asbestos production and use had not declined; rather, the problem was simply being moved from Western countries to emergent economies. Unhappily, the situation has not improved in the intervening 17 years. In India, for example, the use of asbestos has doubled in the last decade to about an estimated 300 000 tonnes a year by an industry that now employs an estimated 100,000 people”.

The often repeated claim has been that the chrysotile form of asbestos is relatively harmless

Smith AH and Wright CC.

Chrysotile asbestos is the **main** cause of pleural mesothelioma.

Am J Industr Med, 30:252-266, 1996.

- We did not say it was the most potent cause
- We concluded that crocidolite might be 2-4 times more potent than chrysotile, but chrysotile was much more widely used.
- There have been many snide remarks about this paper but only one substantive criticism in the literature, and that is that in our analysis we assumed that chrysotile and crocidolite were about equally potent in causing lung cancer.

Hodgson JT and Darnton A
The Quantitative Risks of Mesothelioma and Lung
Cancer in Relation to Asbestos Exposure

Ann. occup. Hyg., Vol. 44, No. 8, pp. 565–601, 2000

**At the other extreme, it
has been argued (Smith and Wright, 1996), that there
is virtually no difference between the risks presented
by the different fibre types.**

Hodgson JT and Darnton A
The Quantitative Risks of Mesothelioma and Lung
Cancer in Relation to Asbestos Exposure

Ann. occup. Hyg., Vol. 44, No. 8, pp. 565–601, 2000

**However this argument is based on the
assumption that all fibre types are equally potent for
lung cancer. If this review is correct in suggesting
that this is not the case, these arguments are not valid.**

Hodgson JT and Darnton A
The Quantitative Risks of Mesothelioma and Lung
Cancer in Relation to Asbestos Exposure

Ann. occup. Hyg., Vol. 44, No. 8, pp. 565–601, 2000

**At exposure levels seen in occupational cohorts it is concluded
that the exposure specific risk of mesothelioma to the principal
commercial asbestos types is broadly in the ratio**

1:100:500

for chrysotile, amosite and crocidolite respectively.

**D Mirabelli, R Calisti, F Barone-Adesi, E Fornero, F Merletti
and C Magnani**

**Excess of mesotheliomas after exposure to chrysotile in
Balangero, Italy**

Occupational and Environmental Medicine 2009

Mesothelioma deaths among workers at the Balangero Chrysotile mine.

- 631 the number of workers alive in 1987
- 9 number of deaths in employees from mesothelioma among employees

**If amosite were 100 times more potent than chrysotile,
then if it had been an amosite mine, there should have
been 900 deaths**

Mesothelioma deaths among workers at the Balangero Chrysotile mine.

- 631 the number of mine workers alive in 1987
- 9 the number of deaths from mesothelioma among employees

If crocidolite were 500 times more potent than chrysotile, then if it had been an crocidolite mine, there should have been 4500 deaths

- These are rough and ready back of the envelope calculations,
- but you get the idea?

Mesothelioma deaths among workers at the Balangero Chrysotile mine.

- 631 the number of mine workers alive in 1987
- 9 the number of deaths from mesothelioma among employees

in addition, there were another 5 mesothelioma deaths among contractors who worked at the mine,

Mesothelioma deaths among workers at the Balangero Chrysotile mine and those with non- occupational exposure

- 631 the number of mine workers alive in 1987
- 9 the number of deaths from mesothelioma among employees
- 5 the number of deaths in contractors

**in addition, there were another 5 mesothelioma deaths
due to household or residential exposure originating
from the mine,**

Environmental exposure cases

1. No definite/likely occupational exposure. Husband asbestos packer at the mining site, work clothes cleaned and washed at home (1948–1973). Lived close to the mining area (1926–1981).
2. No definite/likely occupational exposure. Lived close to the mining area (1925–1926 and 1983–2003).
3. No definite/likely occupational exposure. Lived close to the mining area (1935–2003).
4. No definite/likely occupational exposure. Lived close to the mining area (1943–1980).
5. No definite/likely occupational exposure. Lived close to the mining area (1943–1980).

Do you believe this????

Main messages insert for this paper:

Potency for mesothelioma induction was estimated to be two to three orders of magnitude lower for chrysotile than for amphiboles, based on findings from Quebec miners and millers and because of the absence or very small number of cases in other cohorts, including Balangero miners and millers.

This study identified 14 cases of malignant mesothelioma in workers active in the mine and 13 in other persons exposed to Balangero chrysotile, a situation less reassuring and more complex than previously reported.

The message should have been: this study, and others, demonstrate that, contrary to some claims made, chrysotile asbestos is a highly potent cause of mesothelioma.

Conclusions concerning asbestos

- Workplace risks of disease are extremely high
- The risks go beyond the workplace into peoples homes
- Any further use of asbestos requires asbestos mines, asbestos factories and asbestos use of end-products
- If this is allowed to continue workers will continue to die from mesotheliomas and other diseases
- **An even greater tragedy is that family members of workers may die.**

I will focus on diseases occurring from workplace exposures which also cause environmental health risks to residents nearby with three examples

- asbestos
- **arsenic**
- dioxin

Urinary arsenic levels in timber treatment operators.

Gollop BR, Glass WI.

N Z Med J. 89:10-1, 1979.

An investigation was carried out into arsenic levels in urine of timber treatment operators at six treatment plants in the Waikato-Rotorua area. The mean arsenic level for treatment operators was 222 micrograms/l compared with the normal range of 5-40 micrograms/l. In order to reduce the present significant exposure to treatment chemicals such as arsenic and chromium, it is recommended that the wood preservation industry take engineering measures to reduce the present air emissions and adopt strict work practices in hygiene and protective clothing in similar manner to those handling mercury and lead.

中国古典文学名著全集

Atomic Properties of the Elements

NIST

11

The Berkeley Arsenic Health Effects Research Group (ASRG)

Arsenic Research Group

Not

Allan Smith's Research Group

Associate Director: Craig Steinmaus









CHILE

Region II



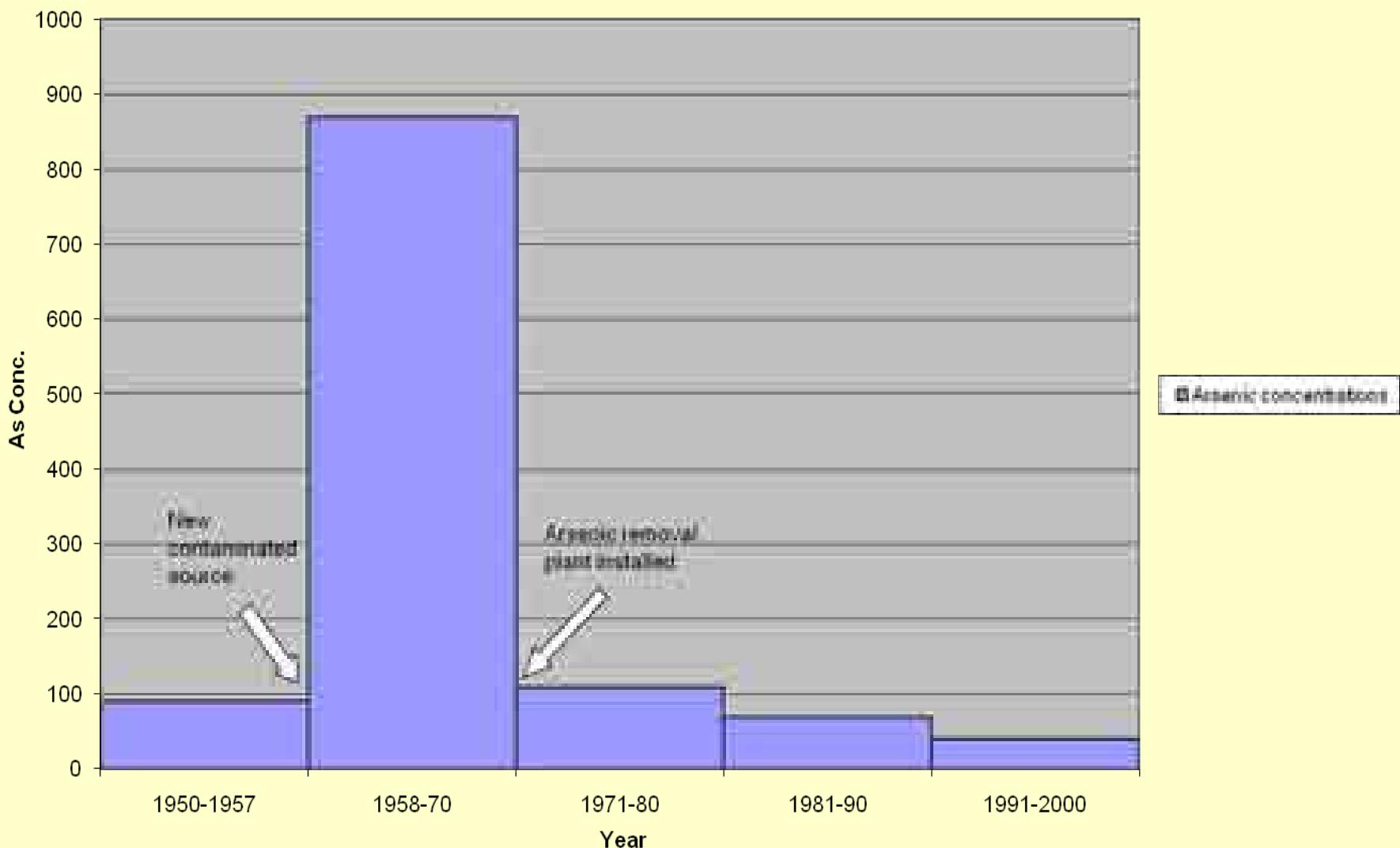
Lung Cancer Mortality Region II Chile, 1989-1993

Age Group	30-39	40-49	50-59	60-69	70-79	SMR	p value
<hr/>							
Women							
Observed	5	23	21	41	47		
Expected	1.2	3.0	8.0	16.0	13.3		
O/E	4.2	7.7	2.6	2.6	3.5	3.1	p<0.001
Men							
Observed	14	48	142	177	129		
Expected	1.2	8.1	28.5	61.8	32.1		
O/E	11.7	5.9	4.9	2.9	4.0	3.8	p<0.001





Arsenic concentrations in drinking water in the city of Antofagasta (popn 200,000) in Chile



Marshall G, Ferreccio C, et al.

**Fifty-year study of lung and bladder
cancer mortality in Chile related to
arsenic in drinking water.**

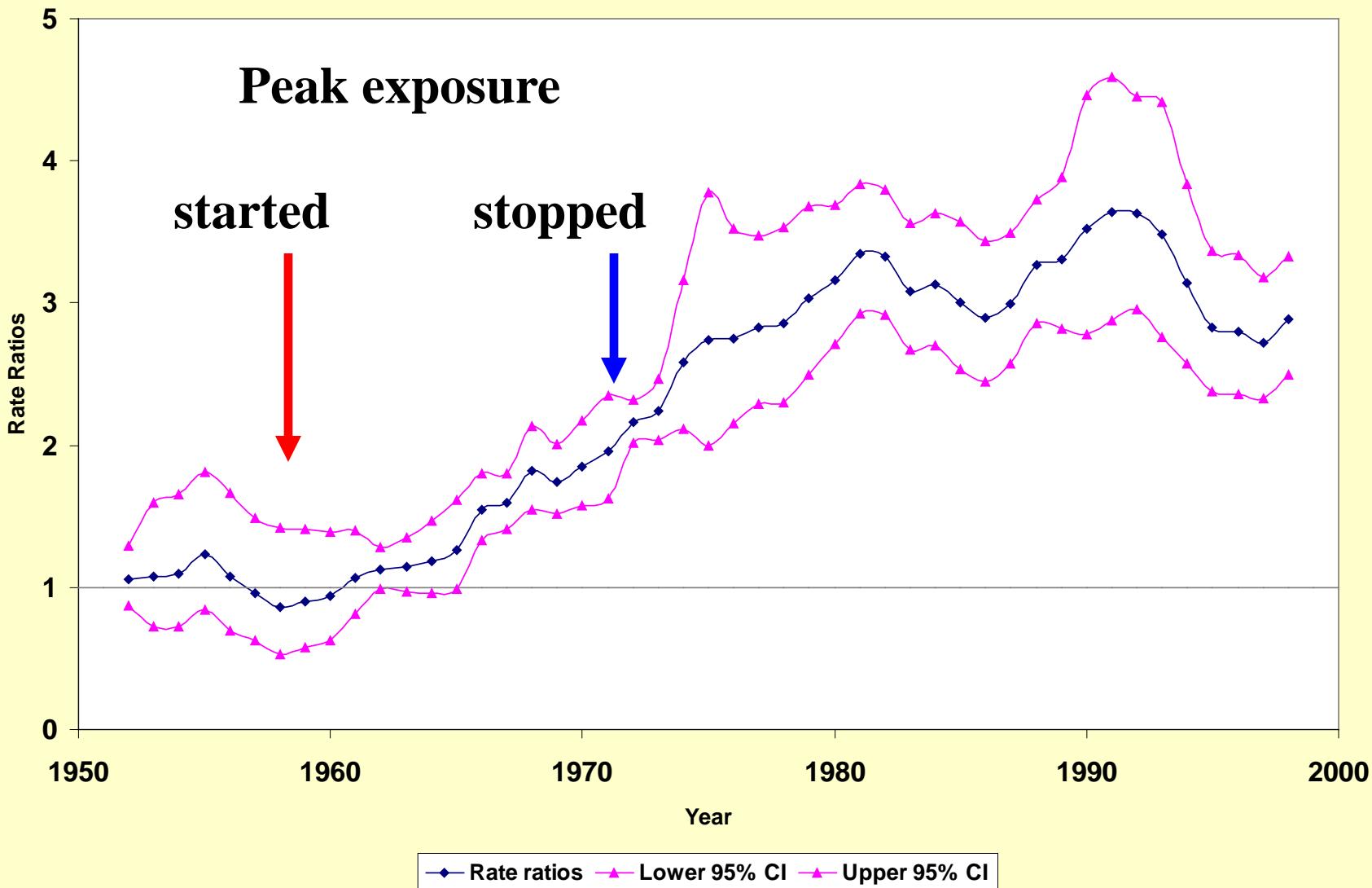


J Natl Cancer Inst 99:920-928, 2007

Mortality data were already available computerized for 1971-2000.

For the years 1950-1971, 200,000 death certificates were digitally photographed and coded for this study.

Mortality from lung cancer among men, Region II Chile Marshall et al, J. Natl Cancer Inst, 2007



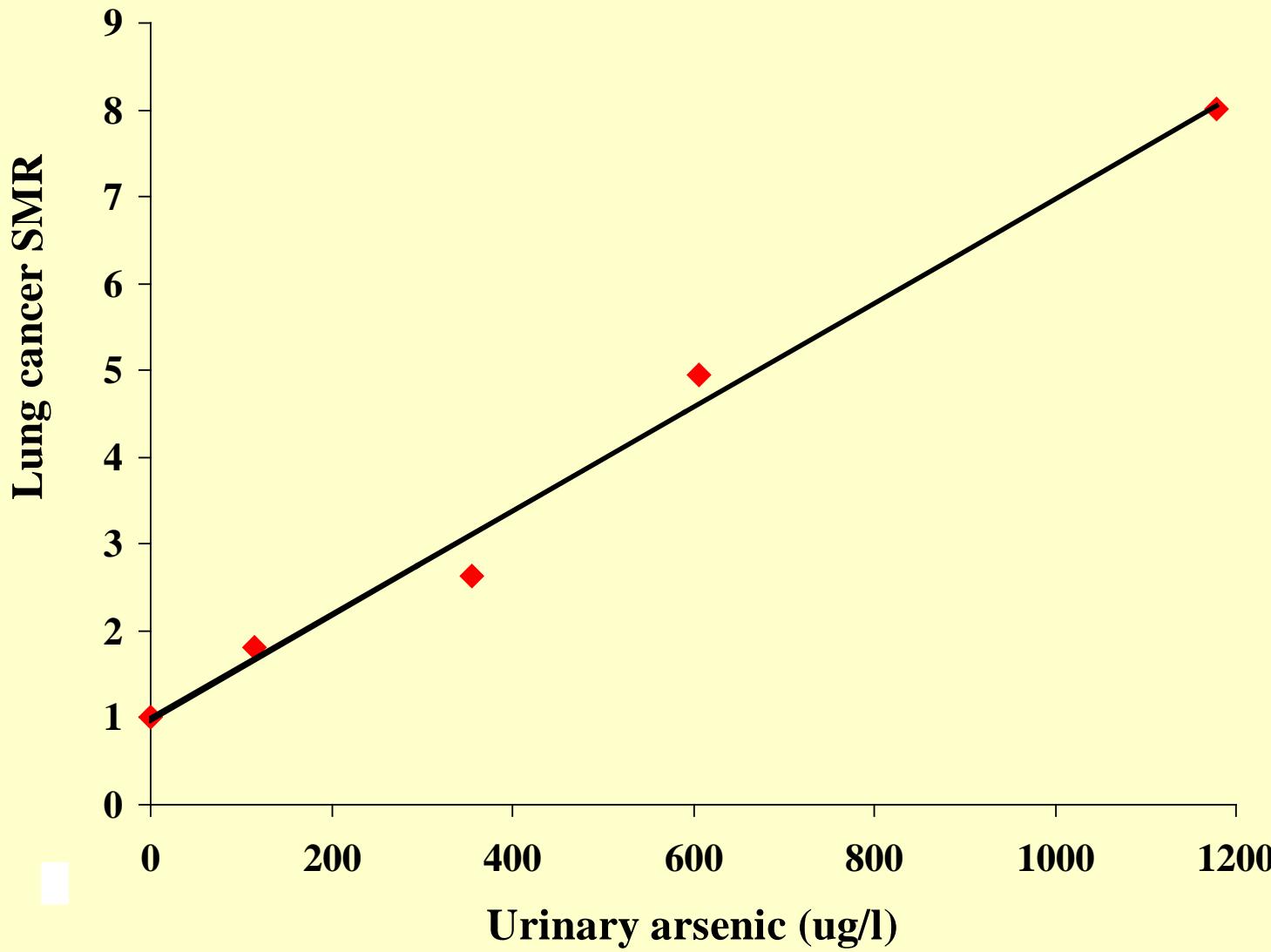
**It is surprising that arsenic in
drinking water would have major
effects in the lungs**

And people preferred to believe it
was the bad mining company that was
the cause of their high cancer rates

Known causes of lung cancer involve inhalation

- smoking
- passive smoking
- asbestos
- radon
- silica
- chromium
- diesel exhaust
- coke oven PAHs
- bischlormethyl ether
- nickel
- arsenic

Lung Cancer and Inhalation of Arsenic



Enterline PE et al, Am J Epidemiol 1987;125:929-38

Lung cancer among women residing close to an arsenic emitting copper smelter

**Frost F, Harter L, Milham S, Royce R. Smith AH, Hartley
J, Enterline P. Archives Environ Hlth 42:148-152, 1987**

Selection of cases

All lung cancer deaths among female
residents of Tacoma or Rushton
1935-69, identified from State death
certificates

Selection of controls

Individual matching

The next death certificate for a woman who died within 5 years of the case, had the same year of death (moving numerically forward or backwards from the case)

Exposure

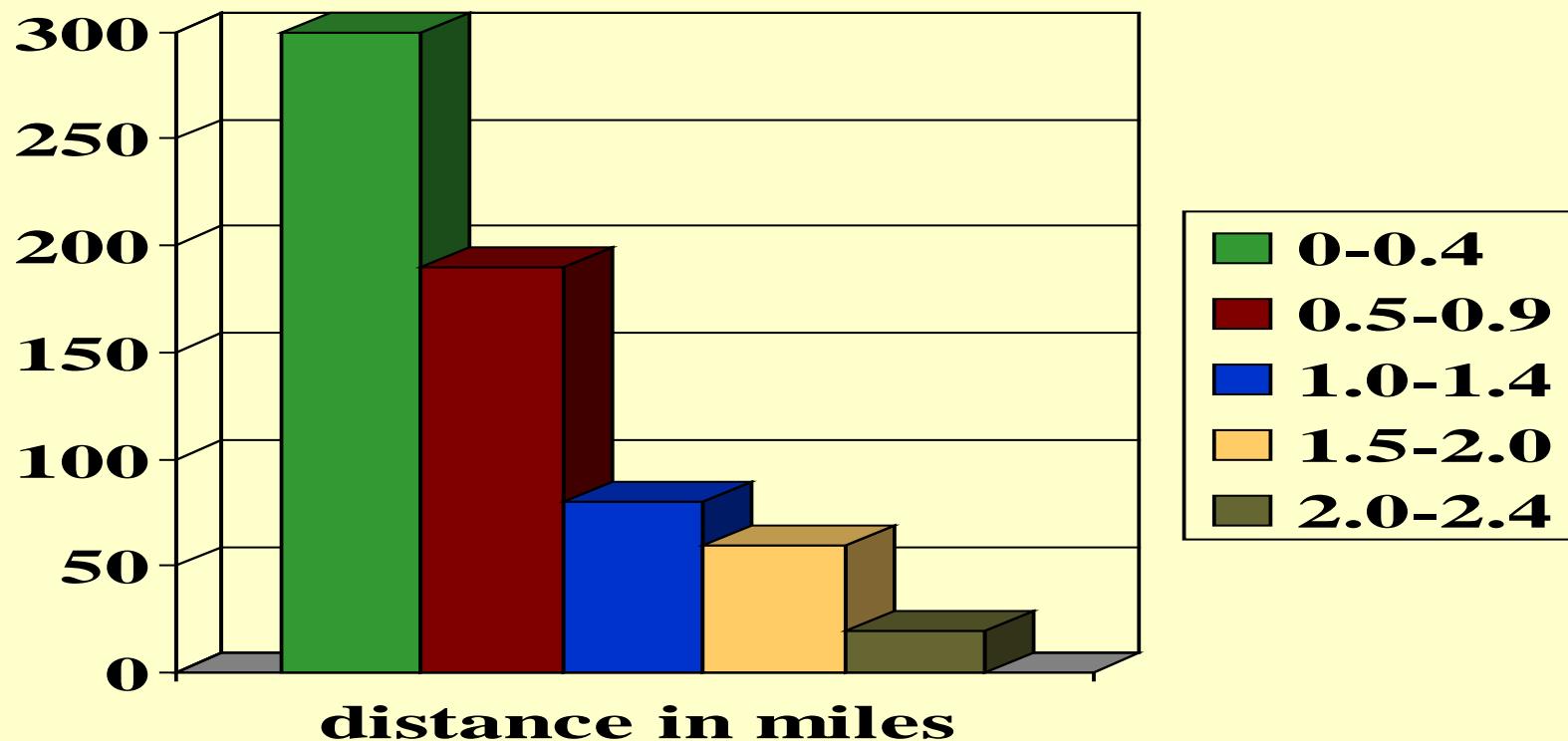
- Address abstracted from death certificate
- distance from smelter identified from a geocoding system
- duration of residence obtained from the death certificate
- cumulative exposure index calculated:

exposure = (years * weighting factor) / (distance)

Urine arsenic concentrations (ug/L) in relation to residential distance from the Tacoma smelter

adapted from Milham S and Strong T. Environmental Research

6:176-182, 1974



Lung cancer odds ratios by exposure index derived from calendar year and distance of residence from the smelter

Index	0	1	2	3	4
Quintile midpoint	2	6	10	16	211
Case	29	29	30	32	36
Control	33	34	32	31	26
Odds ratios	1	1.0	1.1	1.2	1.6

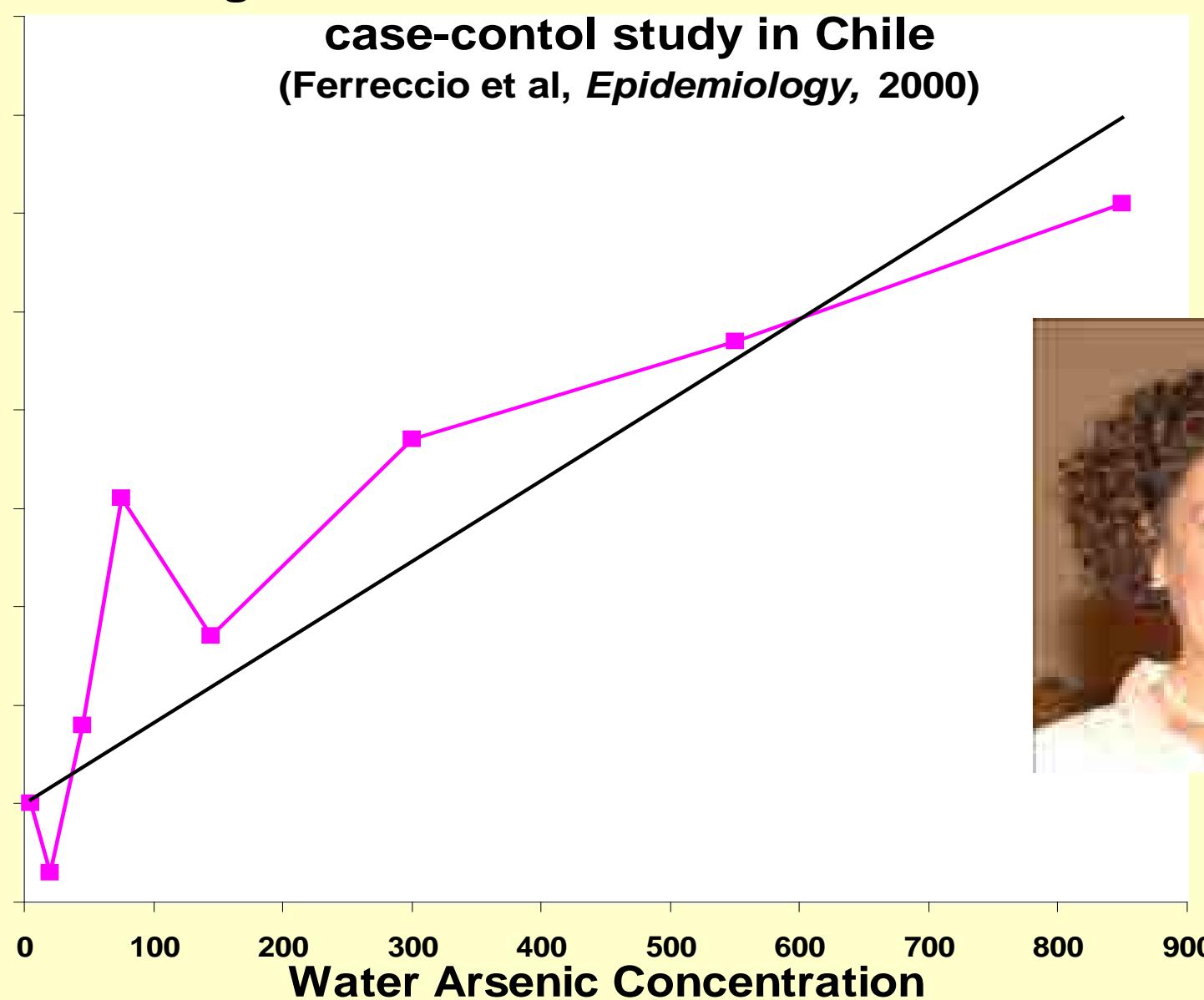
Test for trend, 1-tailed, p = 0.07

**Woman being
congratulated
for participating
in the study of
women living
near
the Tacoma
smelter**

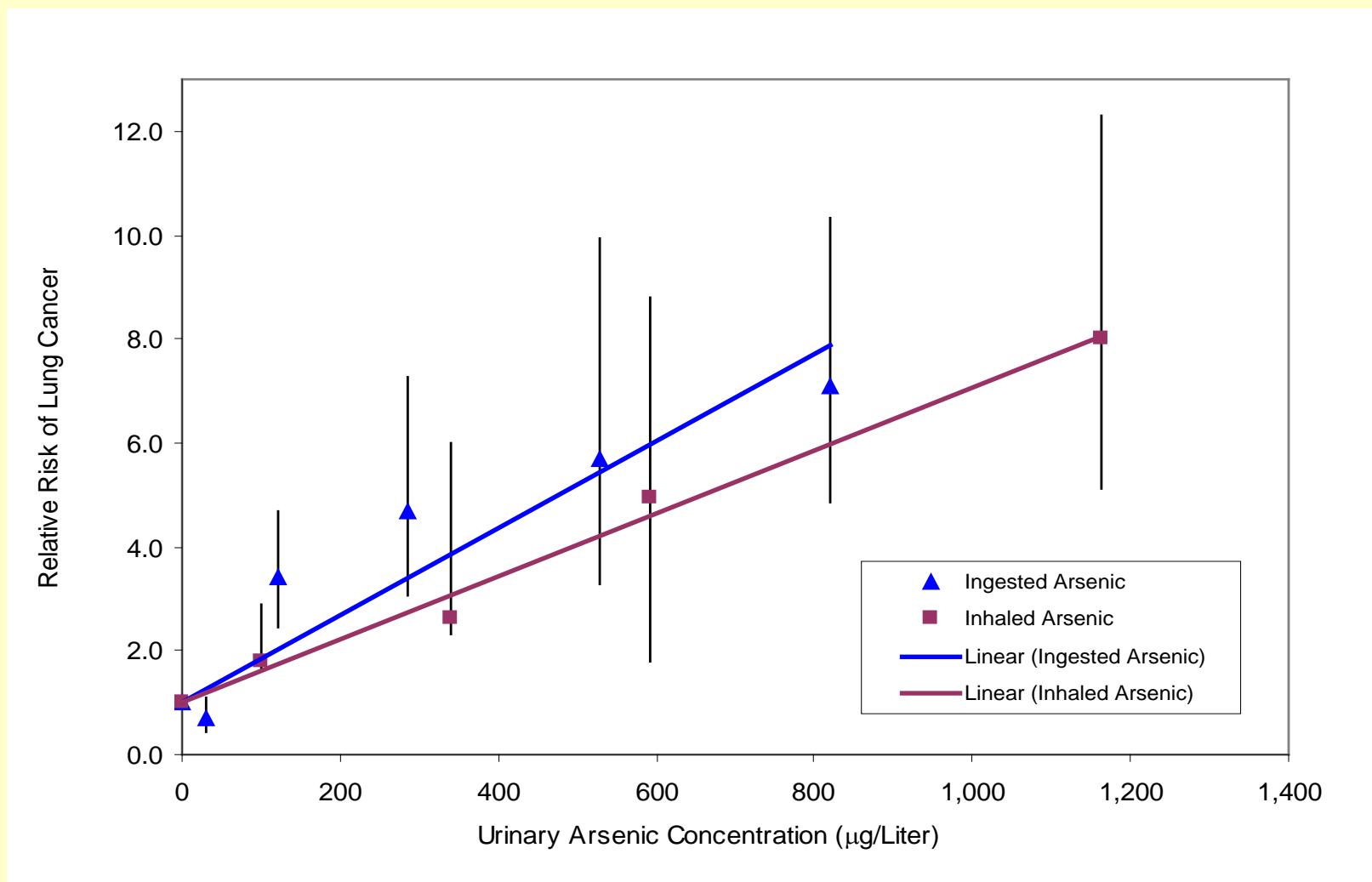


Lung cancer relative risk estimates from a case-control study in Chile (Ferreccio et al, *Epidemiology*, 2000)

Relative Risk



Increased lung cancer risks are similar whether arsenic is ingested or inhaled.



Arsenic is unique

The risks from environmental exposure in drinking water are commensurate with very high exposure workplace risks

And there are marked increased risks of adult disease among those exposed in early life

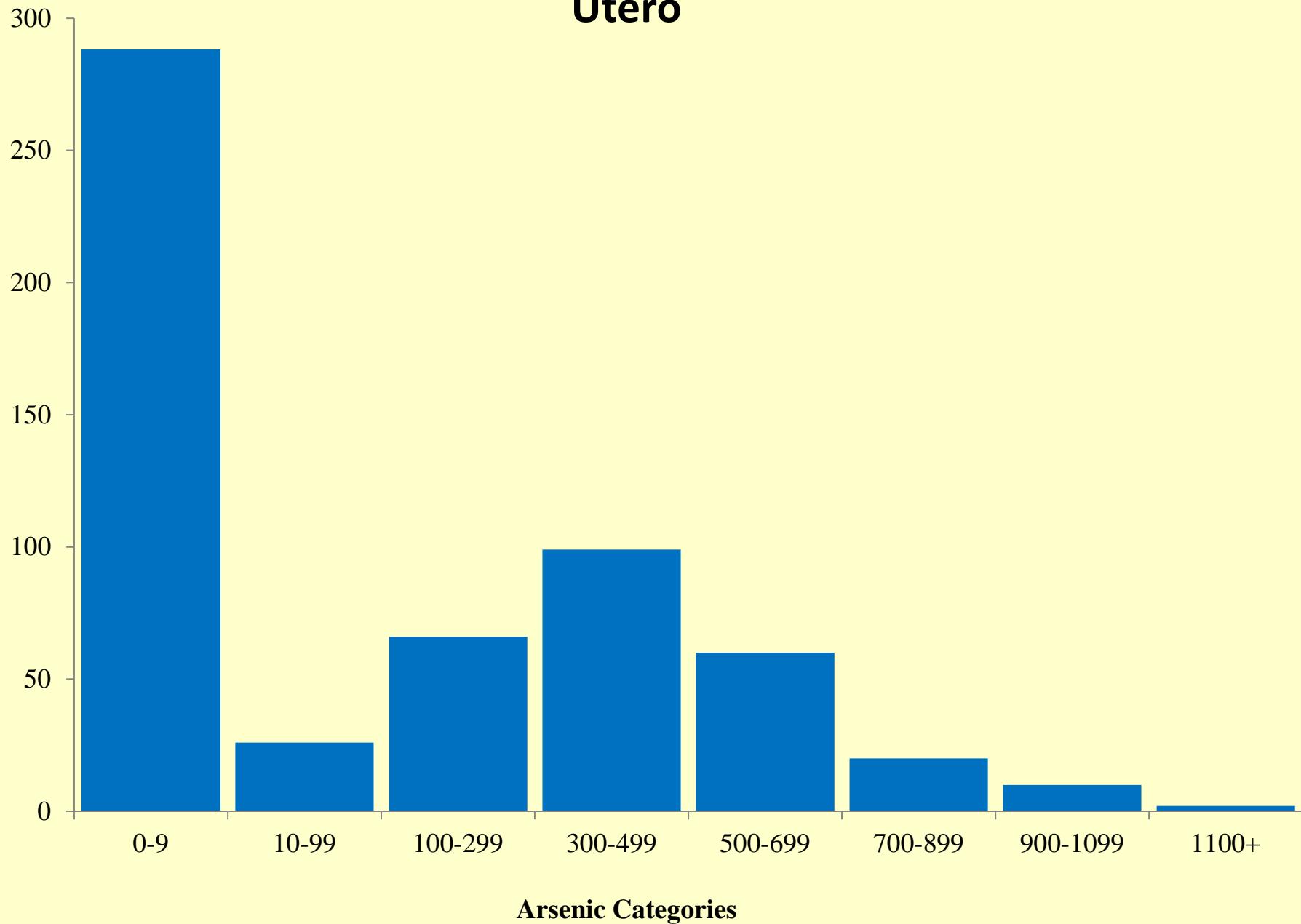




Source: Project Well, West
Bengal, India, 2003

Distribution of Children's Arsenic Exposure (ug/L) In Utero

Number of Subjects (total=571)

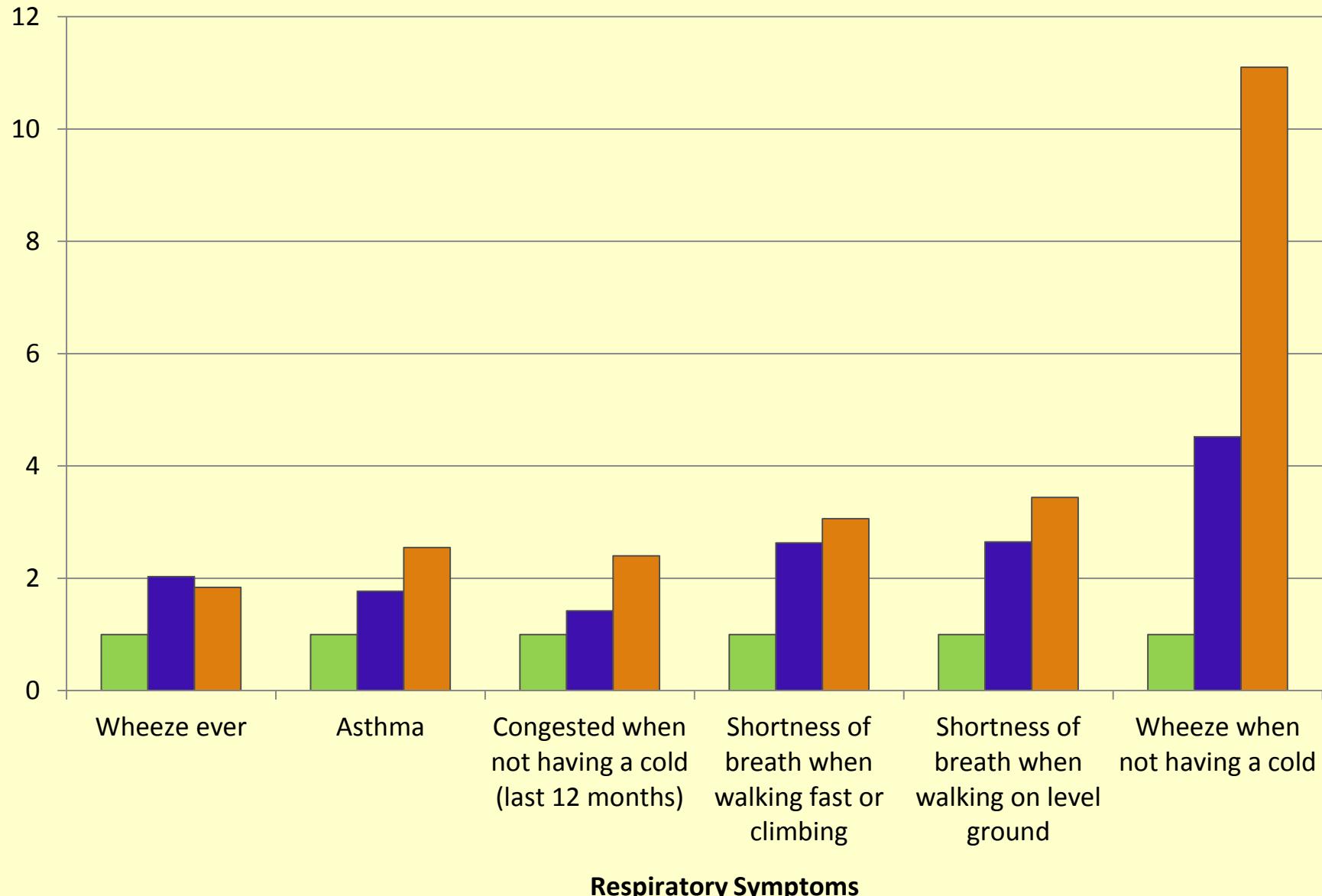






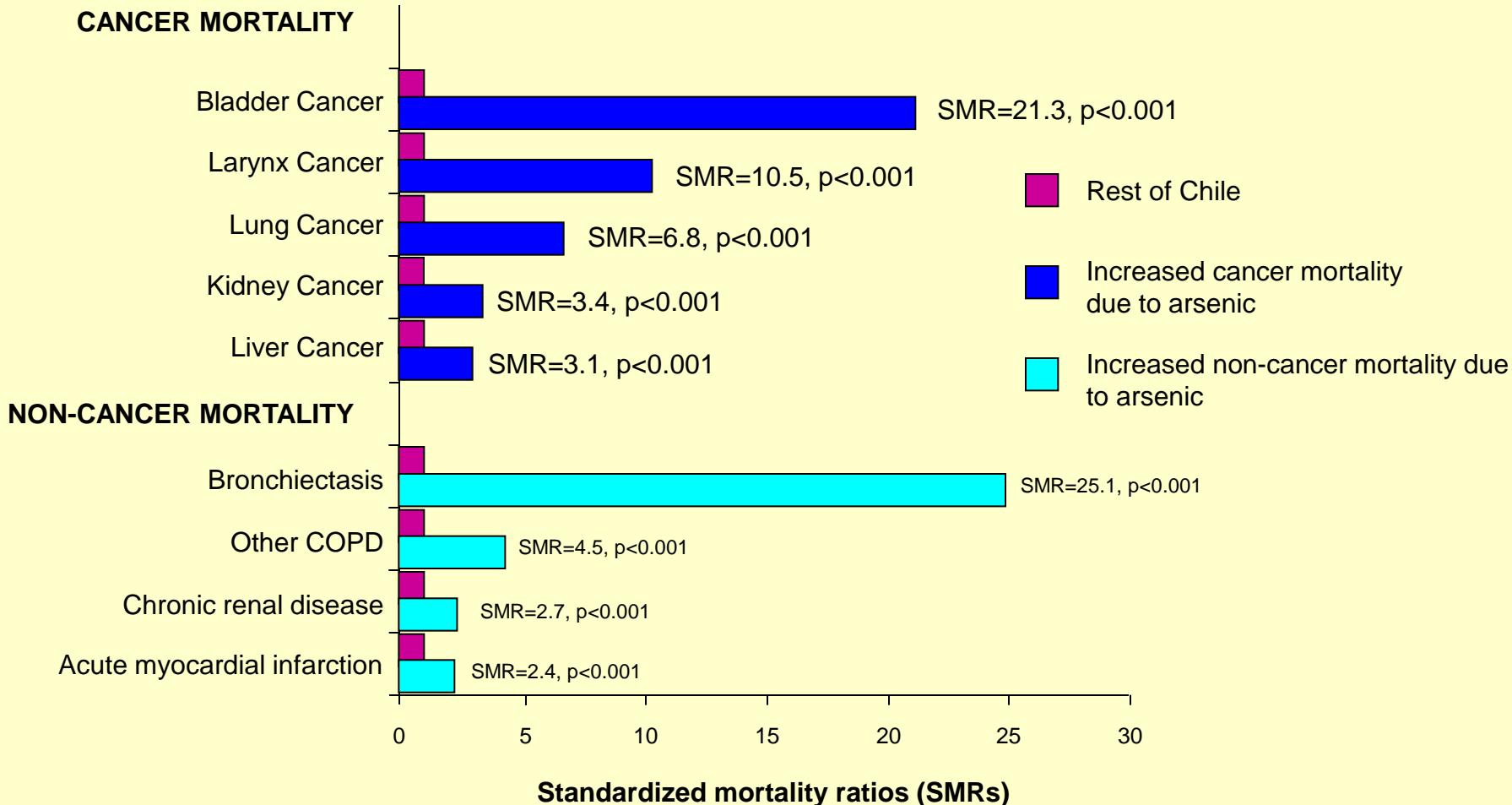
Respiratory Symptoms for Which Adjusted* Odds Ratios for Highly Exposed Compared with Never Exposed In Utero are Greater Than 2

Never Exposed 10-499 ug/L 500+ ug/L



* Adjusted for age, gender, mother's education, father's education, father's smoking status and rooms in the house

Ecologic study of mortality of young adults aged 30-49 following exposure to high concentrations of arsenic in drinking water in early life (not yet published)



Conclusions concerning arsenic

- Workplace risks of disease can be very high
- The risks can go beyond the workplace into surrounding residents, but proving it is hard.
- It happens there is an environmental exposure to arsenic independent of workplace sources which is associated with very high disease risks.

I will focus on diseases occurring from workplace exposures which also cause environmental health risks to residents nearby with three examples

- asbestos
- arsenic
- **dioxin**

BASIS FOR IARC WORKING GROUP EVALUATION

- Human evidence: There is *limited evidence* in humans for the carcinogenicity of 2,3,7,8-TCDD
- Animal evidence: There is *sufficient evidence* in experimental animals for the carcinogenicity of 2,3,7,8-TCDD
- Mechanistic evidence: There is *strong evidence* in exposed humans that 2,3,7,8-TCDD acts through a relevant mechanisms

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC)

Volume 69
Polychlorinated Dibenzo-*para*-Dioxins and
Polychlorinated Dibenzofurans
1997

Overall Evaluation:
2,3,7,8-TCDD is carcinogenic to humans
Group 1

Point source exposures

2,4,5-T manufacture, New Plymouth

Timber treatment with PCP

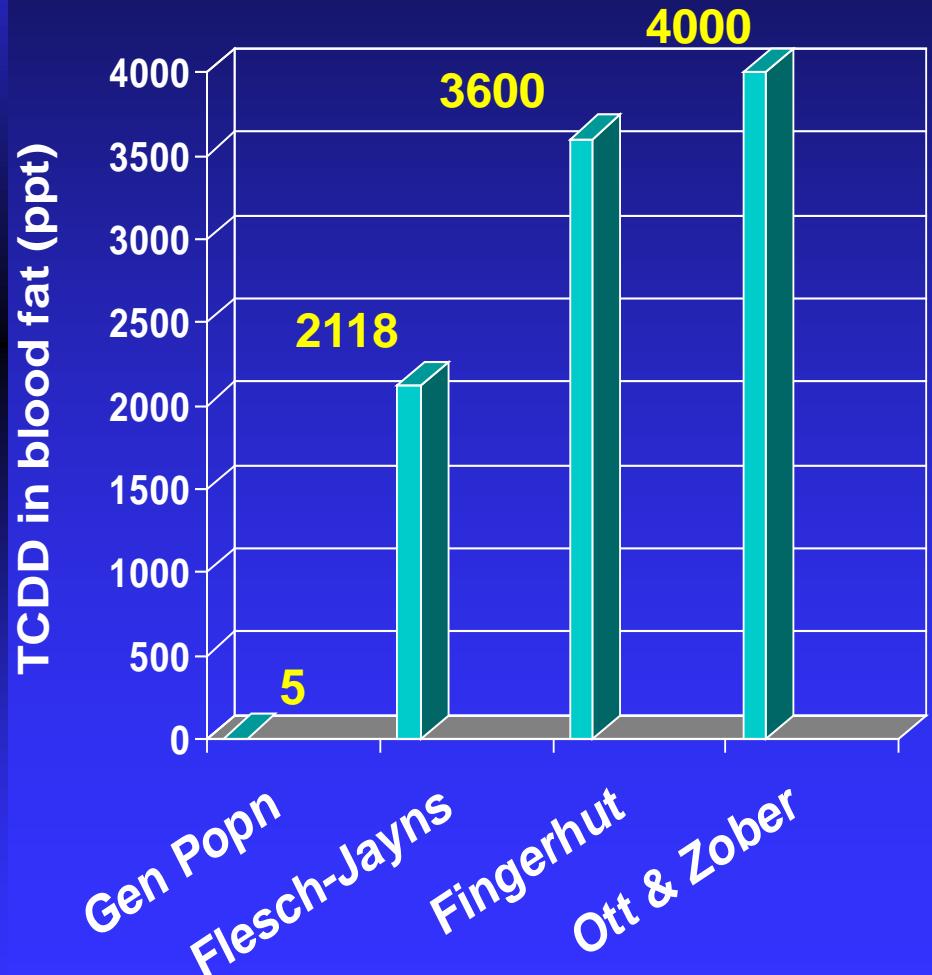
Comparison of dioxin concentrations

Combined U.S. cohorts	3600
BASF cohort Germany	1000-2400
Chlorophenol plant Germany	345-3890
Chlorophenol plants, Netherlands	1842
Seveso, Zones A and B	136
Paritutu, New Plymouth	6.5
General population	1

Comparison of approximate population numbers

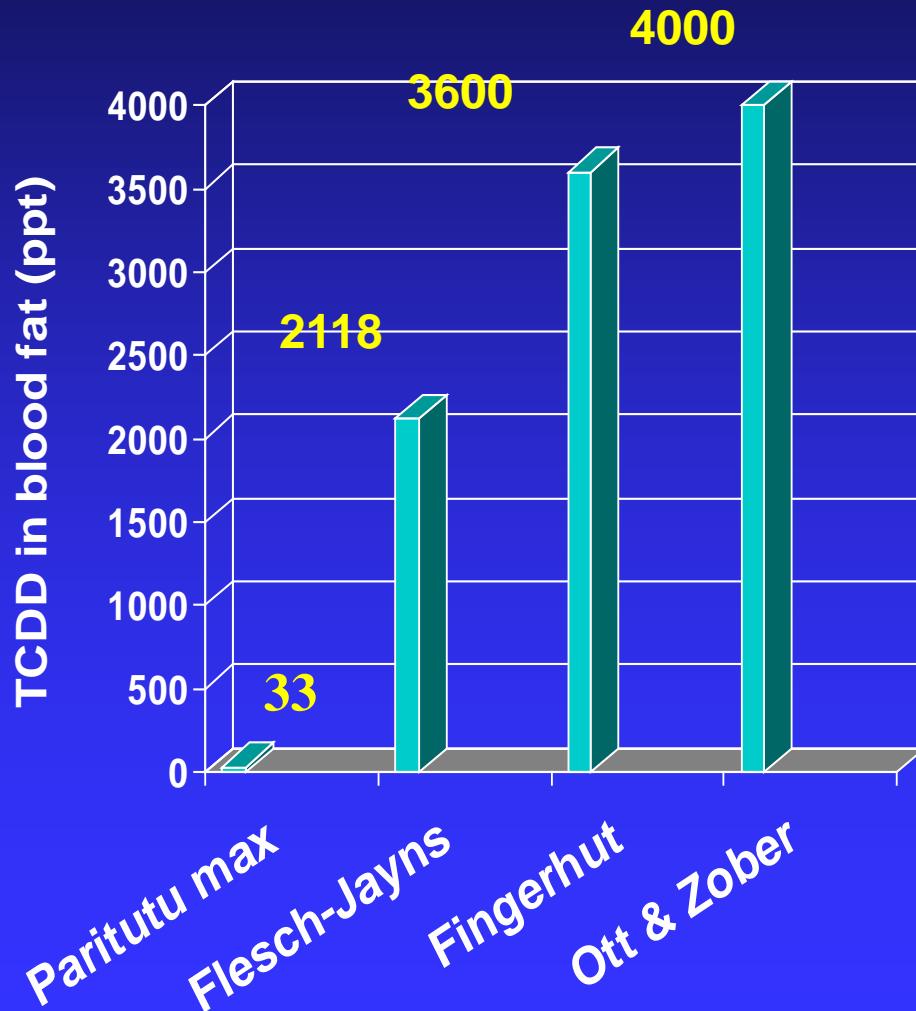
Combined U.S. cohorts	5000
BASF cohort Germany	243
Chlorophenol plant Germany	
Chlorophenol plants, Netherlands	
Seveso, Zones A and B	6800
Paritutu, New Plymouth	50
General population	-

Serum TCDD Levels for the General Population and Three Occupational Cohorts Back-extrapolated to the End of their Exposure



- TCDD concentration for general population is ~5 ppt
- Midpoint of highest exposure group from Flesch-Jayns *et al.*
- Mean for group with >/ 20 years latency and >/ 1 yr exposure from Fingerhut *et al.*,

Serum TCDD Levels for the General Population and Three Occupational Cohorts Back-extrapolated to the End of their Exposure and Paritutu max current



- TCDD concentration for general population is ~5 ppt
- Midpoint of highest exposure group from Flesch-Jayns *et al.*
- Mean for group with >/ 20 years latency and >/ 1 yr exposure from Fingerhut *et al.*,

Serum TCDD Levels for the General Population and Three Occupational Cohorts Back-extrapolated to the End of their Exposure and Paritutu max back calculated



- TCDD concentration for general population is ~5 ppt
- Midpoint of highest exposure group from Flesch-Janys *et al.*
- Mean for group with >/ 20 years latency and >/ 1 yr exposure from Fingerhut *et al.*,
- Highest exposure group from Ott and Zober, 1996

A Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) Exposures in Paritutu, New Zealand

- People with these levels of exposure should be reassured that although their dioxin concentrations are above average, they are way below levels which have been shown to cause health effects.
- There is no good basis for doing epidemiological studies of health effects, although there is a good basis for monitoring exposure.

Need to further study the cohort of workers

- in contrast to those living nearby, there are good reasons to study the workers in the plant who would have experienced much higher exposure to dioxin

Conclusions concerning dioxin

- Workplace risks of disease can be moderately increased.
- Exposure can go beyond the workplace into surrounding residents, but proving any health effects is not possible.
- Once a community becomes concerned about low exposure without rapid assessment and reassurance, then it may become necessary to do health effect studies even knowing that any health effects attributed to the exposure would not be valid.
- And beware of multiple comparisons.

Lessons to be learned from these three examples

- Health effects from exposure to chemical substances are usually detected by workplace studies
- However we need to be alert to potential health effects in surrounding populations, and conduct studies if appropriate
- As soon as concerns are raised we should investigate exposure levels and if high, conduct health effect studies
- If exposures are low then we must immediately provide reassurance with clearly explained data
- If we delay, the community may respond with anger when we tell them their fears are not warranted

• **THE END**