Why We Must Put The Precautionary Principle to Work at Work

Occupational Disease in the US Semiconductor Industry as case-in-point

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Workplace standards for carcinogens and reproductive toxins are much weaker than environmental standards.

Compare these “strong” OSHA standards to environmental standards for the same carcinogens:

Toxic Agent	**Best OCC STDD** 8 hr. TWA	**Best Env. STDD** NSRL or MCL	**Env. STDD** converted to 8 hr. TWA in Air	**Yield** in improved worker protection
Benzene	1 ppm	7 ug/day	1 ppb	1,000:1
TCE	25 ppm	80 ug/day	7 ppb	3,571:1
Perchlorethylene	25 ppm	14 ug/day	.3 ppb	8,333:1
Dichloromethane	25 ppm	0.005 mg/L	1 ppb	25,000:1

These disparities are akin to speed ‘limits’ of 25,000 km/hour in residential areas when we know that 25 km/hr limits are required to prevent death and disability.

Clean room workers have no respiratory protection from noxious vapors and fumes

For decades semiconductor fabrication and electronics assembly has used carcinogens and developmental toxics to produce integrated circuits. Ethylene glycol ethers, TCE, methylene chloride, xylene, n-methyl pyrrolidone, perchloroethylene, arsenic, cadmium, chromium, nickel, and epoxy resins have all been mainstays of “high tech” manufacturing.

The 1986 Microelectronics Industry monograph further alerted the scientific and medical community to this cancer potential and to the toxic components of photoresist mixtures.[2]

Early warnings about reproductive effects from ethylene glycol ethers noted adverse health effects in test animals exposed at levels below the PELs then in effect.[3]

Table 1. Profile of carcinogens in the electronics industry circa 1986,
(from Garabrandt el al "Carcinogens in the electronics industry" ibid.)

Material	IARC Survey Evaluation of Carcinogenic Risk to Humans*	Target Organ or Malignancy, and Strength of Human Evidence which Links Organ to Chemical †
Solvents		
Benzene	1	Leukemia[a]
Carbon tetrachloride	2B	Liver[c]
Chloroform	2B	Unclear[e]
Dichloromethane (methylene chloride)	3	Unclear[e]
1,4-Dioxane	2B	Unclear[e]
Tetrachlorethylene (perchloroethylene)	3	Pharynx[d], esophagus[d], colon[d], liver[d], pancreas[d], lung[d], skin[d], lymphoma[d], leukemia[d]
Tetrachlorethylene	3	Pharynx[d], esophagus[d], colon[d], liver[d], pancreas[d], lymphoma[d]
Metals and Their Compounds		
Arsenic and certain arsenic compounds	1	Lung[a], skin[a], angiosarcoma of liver[b], lymphatic and hematopoetic symptoms[c]
Beryllium and Beryllium compounds	2A	Lung[b]
Cadmium and cadmium compounds	2B	Lung[b], prostate[b], kidney[c], pharynx[c], colon[c], rectum[c]
Chromium and certain chromium compounds	1	Lung[b], nose and nasal sinuses[c], gastrointestinal tract[c]
Nickel and certain nickel compounds	2A	Nose and nasal sinuses[b], larynx[b], lung[b]
Other Materials		
Asbestos	1	Lung[a], pleural mesothelioma[a], pleural mesothelioma[a], peritoneal mesothelioma[a], larynx[b], gastrointestinal tract[b]
P-Dichlorobenzene	3	Leukemia[c]
Epichlorohydrin	2B	Lung[c]
Formaldehyde (gas)	2B	Gastrointestinal tract[d], skin[d], prostate[d], kidney[d], brain[d], Hodgkin's disease[d]

† Strength of human evidence which links organ to chemical (*Adapted from* Merletti, F, Heseltine, E, Saracci, L, et al.: Target organs for carcinogenicity of chemicals and industrial exposures in humans: A review of results in the IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Cancer Res, 44: 2244-2250, 1984.)

a. Target organ-chemical association on which IARC evaluation of sufficient evidence of carcinogenicity rests
b. Target organ-chemical association plausible or possible
c. Target organ-chemical suggested to exist
d. Target organ-chemical association hinted but not substantiated
e. Data inadequate to specify a target organ-chemical association

Efforts to control exposures and protect workers from chronic disease

• 1978 Santa Clara Center for Occupational Safety and Health (SCCOSH) petitioned California OSHA to ban TCE because of its carcinogenicity. Though not banned, California workplace PEL for TCE was lowered from 100 to 25 ppm, a reduction that prompted an industry phase-out of TCE.

• 1980 Federal OSHA conceded the so-called "clean" semiconductor industry is actually chemically-intensive .

> *"The electronics industry is misleading. People think of it as wires, soldering and transistors. But when you get to the semiconductor industry, you're really talking about chemical reactions. It's a chemical industry".* – Hamilton Fairburn, assistant regional administrator, US Occupational Safety and Health Admin. "The Chemical Handlers", San Jose Mercury News April 6-8, 1980.

• 1980 NIOSH Health Hazard Evaluation found "a significant occupationally-related health problem" existed at Signetics in Sunnyvale, Calif. The investigators found that narcotic and irritant symptoms arose in a mixed solvent environment where the individual solvents were all maintained well within their OSHA exposure limits.[4]

- 1982 California OSHA's study of semiconductor industry fails to investigate reproductive hazards, cancer or other and chronic disease.

- 1985 Silicon Valley chip makers are charged in a KRON-TV expose with keeping two sets of records for on-the-job exposures to toxics and systematically underreporting the number of workers affected by poison gases and liquids.[5]

- 1986 Health impact of work in chemically-intensive clean rooms documented at Digital Equipment Corporation, with reports of elevated rates of miscarriage and overall malaise. Newspapers editorialize on "Malaise in the Chip Rooms" as SCCOSH and Silicon Valley Toxics Coalition call for replacement of ethylene glycol ethers.

Table 6. Summary Table for Symptoms* Significantly Associated with Exposure, by Sex Group†

Exposure Group	Reported Symptom	Reporting Symptoms				Prevalence Ratio	95% CIE
		Exposed		Non-exposed			
		N	%	N	%		
Women							
Photolithographic	Arthritis	5	7.9	7	2.1	3.81	1.30, 11.06
Photolithographic	Nausea	7	10.6	12	3.6	2.94	1.22, 6.97
Photolithographic	Rash	9	13.4	19	5.7	2.38	1.13, 4.89
Photolithographic	Sore Throat	6	9.1	11	3.3	2.76	1.08, 6.94
Diffusion	Headache	31	47.0	73	21.7	2.16	1.54, 2.92
Men							
Diffusion	Nausea	7	7.9	1	0.9	8.73	1.44, 54.24

* A symptom with a prevalence ration whose confidence interval estimate does not include 1.0.

† Reports of symptoms whose first "onset" preceded employment in the facility were excluded from this analysis.

Pastides, H., E. J. Calabrese. et al. (1988). "Spontaneous Abortion and General Illness Symptoms Among Semiconductor Manufacturers." J Occup Med 30 (7): 543-51

• 1991 Johns Hopkins Univ. and UC Davis report significant elevations in miscarriage rates in clean room workers where exposures to EGEEA in photoresist were markedly below OSHA PELs. Cancer not studied.

> *"Miscarriages and Chip-Making Chemicals Linked" by John Markoff The New York Times, Oct. 12, 1992, p. A1. "The new concerns about worker health and safety may prove a potential black eye for a high-technology industry that has long sought to portray itself as clean and with little impact on the environment. "*
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> *"Danger of Miscarriage Found in Chip Workers" by John Markoff The New York Times, Dec. 4, 1992. Women exposed to certain chemicals while working in the nation's semiconductor factories face a significantly higher risk of miscarriage, a broad industry-financed study has found.* ***The study is the third in four years to find that a class of chemicals called glycol ethers****—widely used in the process of etching microelectronic circuits on semiconductors—****have toxic effects****."*

- 1991-2003 US semiconductor industry continues to resist doing occupational cancer studies, stating there is no evidence of a problem.

- Sorahan et al (1985, 1992 and 2004) report elevated SMRs for stomach, colon and pancreatic cancer and elevated SIRs for melanoma*, rectal* and pancreatic cancer in study of 1800 UK semiconductor workers; 17% of cohort dead by 2002.

- McElvenny et al (2003) report elevated SMRs for melanoma, lung and brain cancer, elevated SIRs for stomach*, lung,* and breast cancer* in study of 4388 Nat. Semi workers in UK. (only 2% of cohort dead).

• 2003 Dr. Richard Clapp PhD does PMR analysis of IBM's Corporate Mortality File, containing cause of death information and work history data on over 30,000 IBM employees in the US. Clapp finds cohort has significantly elevated cancer PMRS for lymphohematopoetic cancer, brain cancer, kidney cancer, breast cancer and all cancers. These same significantly elevated PCMRs also found for specific IBM manufacturing sites and for chemical-handling jobs.

	CMF				SJ					ROC
			US	US		PMR		CA	CTY	
	US	US	PMR	PCMR	PMR	SJ	PCMR	PMR	PMR	
	PMR	PCMR	Mfg.	Mfg.	SJ	Mfg.	SJ	SJ	SJ	ROC
Males										
KI	146††	136††	179††	162††	217††	146	167†	217††	209††	268††
CNS	192††	162††	190††	165††	341†	324†	247††	314††	296††	407††
NHL	150††	135††	142††	126	147	51	111	139	127	237††
MM	139††	133††	116	107	164	---	132	160	145	149
All	107††		110††		127††	114				142††
ME					198			171	180	
Females										
BR	137††	115††	109	97	209††	243††	144††	202††	193††	148
KI	135	120	231††	212††	128	---	92	135	138	343
CNS	132††	109	136	119	71	---	48	69	69	644††
NHL	140††	122	159	144	248	122	177	246	232	161
MM	108	99	189	178	---	---	---	---	---	1989††
All	115††		110††		141††	146††				191††

† = significant at 95%
†† = significant at 99%
BR = Breast
CNS = Central Nervous System
KI = Kidney
NHL = Non-Hodgkins lymphoma
MM = Multiple Myeloma

CMF = Corporate Mortality File
SJ = San Jose, CA IBM plant
PCMR = Proportionate Cancer Mortality Ratio
ROC = Rochester, MN IBM plant

Clean room work and birth defects

Typically, major malformations occur in 1% of US live births; malformations involving the central nervous system account for 5% of all major malformations. Thus, in 1000 live births, only .5 CNS malformations would be expected

Fewer than 1000 children were born between 1980 and 1989 to clean room workers at the IBM facilities where Johns Hopkins reported elevated miscarriage rates. There were 3 cases of hydrocephaly in the cohort.

If the 3 cases of hydrocephaly were the only CNS malformations in the cohort, they caused more than a 6-fold excess in CNS malformations over expected.

But they were not the only CNS malformations; cases of microcephaly and spina bifida were also found [6]

Zachary was born to a young IBM couple exposed to ethylene glycol ethers in the “clean room” at conception and through gestation. Zach is blind, requires a tracheal tube to breathe, and has had multiple surgeries for micrognathia.

Candace was born to a young IBM employee exposed to lead, chromium and organic solvents. She has microcephaly, numerous longbone anomalies, and requires a tracheal tube to breathe. Industrial hygienist Dr. Mark Nicas estimated her mother’s 8-hr TWA exposure to lead at 0.0267 mg/m3 (OSHA PEL = 0.05 mg/m3). He estimates her mother’s 8-hr TWA exposure to chromium VI at 0.023 mg/m3 (OSHA PEL = 0.05 mg/m3). [7]

Ashley's father was working with ethylene glycol ethers and other solvents when she was conceived and during her gestation. She was born with agenesis of the corpus collosum and hydrocephaly. Dr. Nicas estimates her father's 8 hr TWA exposure to ethylene glycol ethers at 5.1 ppm (OSHA PEL 5 ppm); xylene at 0.48 ppm (PEL 100 ppm, n-butyl acetate .44 ppm (PEL 150 ppm). [8]

Treatment costs for children with birth defects are staggering. The Cost of Birth Defects [9] calculates $8 billion as the cost of lifetime care for the US children born with major malformations in a single year.

Table 1-2. The cost of birth defects in the United States, by cost category, discounted at 5% ($1,000s, 1992)

Condition	Direct Costs			Indirect Costs (c)	Total Costs
	Medical (a)	Special Education (b)	Developmental Services (b)		
Spina bifida	204,512	41,672	1,781	241,324	489,289
Truncus arteriosus	107,578	602	--	101,496	209,676
Transposition/ DORV	166,334	4,402	--	343,794	514,529
Tetralogy of Fallot	185,122	3,974	--	171,390	360,486
Single ventricle	61,659	871	--	110,101	172,631
Cleft lip or palate	97,126	17,551	2,937	578,888	696,501
TE fistula	61,558	--	--	103,444	165,002
Atresia, small intestine	63,156	--	--	46,905	110,061
Colorectal atresia	57,213	--	--	162,049	219,262
Renal agenesis	24,713	--	--	399,466	424,159
Urinary obstruction	46,294	--	--	296,929	343,223
Upper-limb reduction	11,138	24,280	--	134,619	170,036
Lower-limb reduction	16,560	11,851	--	138,656	167,067
Diaphragmatic hernia	62,772	--	--	301,576	364,348
Gastroschisis	54,520	--	--	54,243	108,763
Omphalocele	27,871	--	--	104,133	132,004
Down syndrome	278,696	293,960	95,029	1,180,068	1,847,752
Cerebral palsy	851,809	226,718	217,899	1,129,355	2,425,781
Total Cost	**2,104,419**	**591,842**	**295,557**	**5,038,854**	**8,030,672**

Waitzman et al, The Cost of Birth Defects: Estimates of the Value of Prevention, University Press of America, Inc. 1996.

Closing the gap between environmental and workplace PELS

There are 68 chemicals known to the State of California to cause cancer or reproductive harm that are either totally unregulated by Cal-OSHA or regulated only for non-cancer effects such as irritation. California Assembly Bill 815 would close the gap between workplace and environmental "PELs" by directing Cal-OSHA to make use of the state health department's risk assessments for chemicals known to cause cancer and birth defects in setting workplace standards for these chemicals.

The same huge disparity between workplace and environmental protections against known carcinogens and developmental toxicants exists everywhere. The same rationales for closing the gap apply everywhere as well:

- *MSDSs and other hazard communications wrongly imply that compliance with PELs and TLVs protects against cancer and reproductive harm*

- *Chemical manufacturers invest heavily in challenging sound science, and seek to obscure the body of evidence that chemically-exposed workers suffer disproportionately from cancer and chronic disease*

- *Chemical manufacturers are well aware not only of the irrelevance of PELS when workers are exposed to mixtures but also of the vast disparity between occupational and environmental PELS*

- *Environmental containment is harder and more costly when workplace emissions are out-of-control*

- *Requiring real controls on workplace toxics creates incentives for industrial chemical substitution that do not exist with the current weak PELs;*

- *the employer who does not have to invest in engineering controls to comply with a chemical's PEL lacks a clear economic incentive to stop using the chemical*

- *Legal remedies for industrial disease too often provide too little too late. Legal systems impose only modest fines and penalties for the kinds of employer negligence and failures to warn that lead to serious worker illness and death*.

- *"No fault" workers compensation systems provide no real incentives to prompt employers to improve working conditions*.

Conclusion:

Unless and until non-protective workplace PELS are brought into parity with health-protective environmental standards, the best-intentioned efforts to bolster enforcement of existing standards, improve hazard communication, combat industry disinformation, develop incentives to use safe alternatives, and hold wrongdoers accountable will not protect workers and their offspring from toxic harm.

References:

[1] Toxics attorney in San Jose, CA, founder of SCCOSH and Cal-COSH. Ahawes@alexanderlaw.com.

[2] State of the Art Reviews, Occupational Medicine "The Microelectronics industry" 1986. See chapters by Daniel Teitelbaum "Photoactive Chemicals Used in Photoresist Systems" and by David Garabrant and Robert Olin, "Carcinogens and Cancer Risks in the Microelectronics Industry"

[3] See e.g. Nagano, K., E. Nakayama, et al. (1981). "Embryotoxic effects of ethylene glycol monomethyl ether in mice." Toxicology 20(4): 335-43.; Nagano, K., E. Nakayama, et al. (1984). "Experimental studies on toxicity of ethylene glycol alkyl ethers in Japan." Environ Health Perspect 57: 75-84; Hardin, B.D., G. P. Bond, et al. (1981). "Testing of selected workplace chemicals for teratogenic potential." Scand J Work Environ Health 7 (Suppl 4).: 66-75.; Hardin, B.D., P. T. Goad, et al. (1984). "Developmental toxicity of four glycol ethers applied cutaneously to rats." Environ Health Perspect 57: 69-74.; Hardin, B. D. and J. P. Lyon (1984). "Summary and Overview: NIOSH Symposium on Toxic Effects of Glycol Ethers." Environmental Health Perspectives 57: 273-275

[4] NIOSH 1980 US Dept Health Education and Welfare, CDC, NIOSH Interim Rept #2, Health Hazard Evaluation proj. No. HHH 79-66; Signetics Corporation, Sunnyvale Ca.

[5] Hard Copy. "Chip Makers: Doctoring the Books?" San Jose Metro June 6-12 1985.

[6] Expert opinion of Dr. Cynthia Bearer MD, PhD in Kardas el al v IBM et al

[7] Expert opinion of Dr. Mark Nicas in Ruffing et al v IBM et al

[8] Expert opinion of Dr. Mark Nicas in Ruffing et al v IBM et al

[9] Waitzman et al, The Cost of Birth Defects: Estimates of the Value of Prevention, University Press of America, Inc. 1996.