Environmental Health Perspectives
Vol. 17, pp. 107–115, 1976

PVC: Health Implications and Production Trends

by Myra Karstadt*

Poly(vinyl chloride) (PVC) is a complex plastic system. Individual components of the PVC system, including residual vinyl chloride monomer (RVCM) and certain additives, may pose risks of harm to human health. There have been significant reductions in the RVCM content of PVC resin since 1974, reducing the cancer risk of workers in PVC fabrication plants and consumers of PVC products. A "no-effect" level for vinyl chloride monomer (VCM)-induced carcinogenesis has not been found to date; therefore, the significance of human exposure to low levels of RVCM remains to be determined. Exposure to PVC dust may cause pulmonary dysfunctions. Pulmonary and other possible health effects of PVC dust require further study. The PVC plastics system should be characterized as to interactions among its various components and as to interactions of the components and the PVC system as a whole with biological systems.

In January 1974, B.F. Goodrich Chemical Company announced that three reactor workers in a poly(vinyl chloride) (PVC) polymerization plant had developed a rare cancer, angiosarcoma of the liver. Exposure to vinyl chloride was linked to development of angiosarcoma of the liver by subsequent epidemiological studies. Animal studies, several initiated before the B.F. Goodrich announcement, also link exposure to vinyl chloride (VCM) with development of cancer.

Regulations promulgated in the United States of America since early 1874 have resulted in the lowering of VCM concentrations in the atmosphere of PVC manufacturing plants, and decreased concentrations of VCM in PVC resin. This has reduced the cancer risk of PVC production workers, workers in PVC fabrication plants and the ultimate consumers of fabricated PVC products.

We are now at a crucial stage as regards studies of the effects of plastics and plastics components on human health. Human exposure to massive doses of VCM, whether due to uncontrolled venting of PVC polymerization vessels or the voluntary venting in a home bathroom of an aerosolized VCM hair spray, is essentially over. The time has come for study of possible health hazards posed by the complex plastic system called PVC.

* 3003 Van Ness, Washington, D.C. 20008.

Why be concerned about PVC? A publication (*1*) states that: "Total world employment in the VCM and PVC producing industries is likely to be well over 70,000 workers. Those employed in industries which use PVC as a basic element are likely to total in the millions. Those who come in contact with PVC every day in some form or other probably make up about at least one-third of the human race."

What are the human health effects of PVC? There are several components of PVC which, on their own, could affect human health adversely: VCM, PVC dust considered as a dust, additives of various sorts. ("Additives" for our purposes includes stabilizers, colorants, flame retardants, plasticizers, fillers, etc.)

But one cannot consider the components of PVC solely as independently acting potentially toxic agents. PVC is a complex chemical system with little-understood interactions, both physical and chemical, among its components.

Leaving aside the multicomponents interaction problem for the time being, consider the individual components of PVC which may pose risk of harm to humans.

VCM is already identified as a cancer-causing agent. Even given the reductions of VCM in PVC resin, which have been achieved by changes in both production and compounding procedures, some RVCM (residual vinyl chloride monomer) is still present in PVC.

Table 1. April 1975 status of suspension resin stripping.[a]

Company	Number of suspension resin grades	Suspension resin production, %	RVCM, ppm
A[a]	—	60	2000–4000
		40	4000–6000
B[b]	—	100	2000–5000
C	5	100	<400
D	—	80	400
		20	500–700
E	13	86.8	0–50[c]
		5.4	100
		3.8	500
		3.2	1000
		.8	4000
F	15	100	<400
G	4 (Homopolymer)	6	1500
		14	2500
		80	100
	4 (Copolymer)	16	300
		84	400
H	4	100	2000–10,000
I	—	38	400
		9	600
		25	800
		5	1800
		15	3600
		8	4000

[a] Based on data from responses to 3/31/75 section 114 request (*4*).

[b] All suspension resins to be reduced to <400 ppm RVCM by 7/75.

[c] Some values are speculative until improved stripping is installed.

In 1974 Rowe (*2*) estimated the average monomer concentration of PVC resin to be approximately 1000 ppm. As shown in Table 1, RVCM concentrations of 100 ppm or less for suspension resins before compounding were not unusual by April 1975. An advertisement (*3*) run in July 1975 by a major manufacturer of PVC stated that general-purpose PVC homopolymers and blending resins were being produced with a maximum of 10 ppm, and usually less than 5 ppm, RVCM.*

Tables 1–3, taken from a U.S. Environmental Protection Agency (EPA) publication (*4*) give some idea of the present RVCM in several PVC resins. In particular, note company E in Table 1. 86.8% of suspension resin production is said to be at 0–50 ppm RVCM (with a footnoted qualification that "some values are speculative until improved stripping is installed".) Note also that for company A, 60% of whose resin had a RVCM concentration of 2000–4000 ppm in April 1975, with the remaining 40% at 4000-6000 ppm, "all suspension resins (were) to be reduced to less than 400 ppm RVCM by 7/75."

Health problems which may be associated with RVCM in finished PVC resin are of importance to workers and consumers. The residual VCM in the old high-residue PVC evidently posed problems for workers in fabrication plants. Data collected by the United States National Institute for Occupational Safety and Health (NIOSH) indicate that several angiosarcoma cases were identified in fabrication plant workers in this country and abroad (Table 4) (*5*).

* The advertisement states (in part): "At Tenneco, we've developed a unique new production technology that allows us to produce our general purpose PVC homopolymer, blending and dispersion resins with no more than 10 parts per million of VCM(vinyl chloride monomer). In fact, the typical VCM content of these homopolymer and blending resins is less than 5 parts per million and under 3 parts for our dispersion resins. And, we are producing our flexible compounds at nondetectable(less than 1 ppm) levels.

"This is a 98% to 99% reduction from what was once the typical practice of the industry for residual VCM."

Table 2. Suspension resins.

ASTM cell class[a]	Prod, %	VCM into stripper, ppm	ppm (avg.) VCM out of stripper (dry basis),	VCM out of dryer, ppm (avg.)	Emission factor, kg VCM/ 100 kg PVC
GP1–26250	5	(40–60) × 10^3	3,050	308	0.27
GP2–16340	5	(40–60) × 10^3	2,050	162	0.18
GP3–15340	13	(50–70) × 10^3	1,200	104	0.09
GP4–15340	35	(60–90) × 10^3	1,050	95	0.09
GP6–15343	24	(90–120) × 10^3	950	82	0.08
GP1–14443	0	(80–100) × 10^3	600[b]	25	0.06
GP2–14443	1	(80–100) × 10^3	550	16	0.06
GP3–15433	1	(80–100) × 10^3	500	21	0.05
GP5–15433	5	(90–110) × 10^3	450	15	0.05
GP6–15433	9	(100–140) × 10^3	450	18	0.05
Copolymers					
C11–8500	1	(40–60) × 10^3	2,100	280	0.18
C11–3500	1	(90–110) × 10^3	1,000	95	0.09

[a] ASTM cell class is standard definition of resin (ASTM–D–1755).

[b] Based on limited data (less than three samples) (*4*).

NIOSH data (Table 5) presented in late 1974 (*6a*) indicated that breathing zone samples in fabrication plants turned up relatively low concentrations of VCM–even at a time when RVCM concentrations were high compared to today's values. Even at that time, as Wagoner put it: "60% of the samples were less than 1 ppm" (*6b*). This level (1 ppm) is the maximum exposure level (8-hr time-weighted average) currently permitted by the United States Occupational Safety and Health Administration (OSHA) standard (*7*). Again quoting Wagoner (*6b*): "I think it [the occurrence of angiosarcoma of the liver in fabrication plant workers] points to the possibility of low level exposure being obviously very hazardous."

A recent contract study (*8*) prepared for NIOSH reported on fabricating plant VCM concentrations in 1975. The VCM concentrations are, in a number of cases, very low (less than 0.01 ppm, the limit of detection). However, certain work stations had values which

Table 3. April 1975 status of dispersion resin stripping with protection of future capabilities.[a]

Plant	Current RVCM, after stripper	ppm in product	Projected RVCM after stripper, ppm	Time to implement controls needed to meet projected levels, months
A	30,000	1–3	20,000	6
B	50,000	5	10,000	6
C	20,000	5–15	4000–6000	48
D	1500–4000	—	—	—
E	15,000	1–10	6000	12–18
F	13,000–18,000	10	4500 500–1000	30 42
G	2000–10,000	25	2000 400	30 48
H	14,000–18,000	5–30	1200	30
I	2000	<1	—	—

[a] From responses to March 31, 1975, section 114 request (*4*).

Table 4. Reported cases of angiosarcoma of the liver among nonpolymerization workers exposed to vinyl chloride.

Country	Case no.	Birth date	1st VC or PVC exposure[a]	Diagnosis of angiosarcoma[a]	Age at diagnosis	Time from 1st exposure to diagnosis, yr	Tot exposure, yr	Date of death[a]
Fed. Rep. Germany	03	07-16-30	09-09-52[b]	03-00-73[c]	43	21	14	10-10-73
Fed. Rep. Germany	07	05-09-36	00-00-60[d]	00-00-74[c]	38	14	03	12-16-74
Great Britain	02	09-08-14	00-00-46[e]	02-00-70[c]	55	24	11	12-00-70
Italy	01	06-15-34	00-00-65[f]	04-19-71[g]	36	6	3	04-16-71
Sweden	02	11-27-11	00-00-45[h]	05-15-72[c]	61	27	23	08-16-72
United States	14	00-00-13	01-18-38[i]	06-00-73[j]	60	36	00	07-03-73
United States	15	00-00-25	00-00-00[k]	07-00-72[c]	47	00	00	02-15-73

[a] "00" indicates unknown data.

[b] Loading pesticide cans with VC propellant.

[c] Microscopically confirmed angiosarcoma of the liver.

[d] Assistant factory chemist.

[e] Pouring PVC oil mixture onto fabric bases.

[f] Production of PVC sacks.

[g] Angiosarcoma involving liver, lung, and pericardium. Although difficult to determine, primary site seems to be pericardium.

[h] Production of vinyl chloride.

[i] Machine operator covering electrical wire with PVC plastic insulation.

[j] Diagnosis: sarcoma (possibly "angiosarcoma"), liver. Possibility of generalized neoplasm of the reticuloendothelial cell system cannot be ruled out.

[k] Accountant at plant making PVC fabric.

exceeded the 1 ppm 8-hr time-weighted average currently permitted by OSHA.

How low must you go before RVCM ceases to represent a hazard? A "no-effect" level for VCM carcinogenesis has not been found to date; cancer has been found in all groups of animals tested with nonzero concentrations of inhaled VCM. Just how low are the VCM concentrations in fabrication plants now? Publication of monitoring data would be useful for analysis of possible risks of harm to health.

A fabricated PVC product category with wide consumer exposure is PVC automobile upholstery, rooftops, and interior fittings. The

Table 5. Summary of VCM concentrations in PVC fabrication facilities.

Type of plant	Maximum	Minimum	Mean	Fraction >1 ppm
Firm and mold (7 plants)				
NIOSH data				
Breathing zone samples	12	<0.3	3	2/5
Area samples	8	<0.3	1.2	1/11
Source samples	340	<0.3	18	10/21
Pipe and mold (2 plants)				
Industry data				
Breathing zone samples	<10	<0.1	<1	4/21
Area samples	35	<0.1	6	16/20
Source samples	540	<0.1	142	10/17

EPA has a contract study, entitled, "Sampling of Automobile Interiors for Vinyl Chloride Monomer" (*9*) in progress. According to Hedley, who is working on the EPA contract study (personal communication, February 23, 1976), of 7 cars in which 30–60 min grab samples were taken in 1975 under varying heat and sunlight conditions, one reading was 1.2 ppm VCM, one 0.4 ppm and the remaining five less than 0.05 ppm (the detection limit). The car with the highest reading was sampled one month after assembly under the following temperature conditions: ambient 36°C, interior air of car 60°C, seat surface 66°C. No attempt was made to follow any decrease in off-gassing of RVCM over time.

Given the number of individuals exposed to "new car atmospheres" and cars or other motor vehicles generally, some further data on the RVCM content of PVC auto fittings might be useful.

Even if risk to consumers from RVCM in PVC seat covers is low, what is the risk of harm to persons working in automobile factories?

A recent study by Infante et al (*10*) is especially interesting. The data collected by Infante have been interpreted to suggest that VCM may exert a mutagenic effect on sperm cells of PVC production workers, leading to an increase in fetal deaths among pregnancies of workers' wives. Infante's findings are not surprising in light of the demonstration by Ducatman et al. (*11*) of chromosome aberrations in PVC production workers and the finding by Ames (*12*) that VCM is mutagenic in a *Salmonella* test system.

The sewing of vinyl automobile upholstery and other vinyl fabric automobile fittings is, evidently, in large part woman's work. Even given the multicomponent nature of the PVC in auto fittings, it might be interesting to determine the incidence of cancer, stillbirths, and birth defects in offspring among women vinyl auto upholstery workers.

What happens to PVC on pyrolysis? There seems to be some disagreement as to whether VCM is liberated (*13*) or whether the principal health threat from burning PVC is caused by evolution of HCl (*14, 15*). Has there been a definitive demonstration that VCM is not evolved during pyrolysis of PVC? What about depolymerization on aging? in sunlight? in heat?

PVC exposure has been linked both to dermatitis and Raynaud's syndrome or occupational acroosteolysis (*16*). Dermatitis could be attributable to several of the additives in PVC, or possibly to VCM itself. A catalog of resin components which have caused skin sensitization reactions would be useful.

One of the important consumer uses of PVC is in food packaging. Regulations recently proposed by the United States Food and Drug Administration (FDA) (*17*) deal with PVC food packaging. Although industry data (*18*) have been presented to FDA which indicate migration of VCM from PVC as below 50 ppb (the "lowest detectable" level established by the FDA proposal), challenges (*19*) have been raised to permitting use of PVC food wrap if there is any RVCM capable of migrating.

The debate about PVC food wrap points up what may be the salient characteristic of PVC considered as a source of residual VCM: RVCM contained in PVC has now joined the ranks of other carcinogenic substances whose presence in trace amounts—the amounts limited only by the means of detection—in the US food supply is forbidden by the Delaney Clause, an amendment to the United States Food, Drug and Cosmetic Act. That clause reads in pertinent part:

". . . no additive shall be deemed to be safe if it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal . . ." (*20*).

FDA's attempt to set a nonzero tolerance for VCM in food wrap has been challenged as illegal due to conflict with the Delaney Clause (*19*). It may be that final promulgation of a proposal setting a nonzero tolerance for RVCM in food wrap will be challenged in the courts.

Efforts to reduce VCM residual in PVC have come a long way in just over two years. The industry has demonstrated its ability to reduce VCM levels in PVC production plants from hundreds or thousands of ppm to a level close to or at the OSHA 8-hr TWA of 1 ppm.

Since chemical reactions rarely go to completion and are usually reversible, will it ever be possible to produce a PVC resin completely free of residual VCM and incapable of breaking down to release VCM? The stumbling block for the continued health and growth of the PVC industry may well prove to be not the threat of massive doses of VCM and high potential risk

of cancer for the few, but the threat of low doses of VCM and a low but nonzero risk of cancer for the many.

The presence of RVCM in PVC represents a potential health hazard for the large number of humans exposed to resin or, more commonly, finished plastic goods. The dusty nature of PVC resin may represent a health hazard for persons with an occupational exposure to PVC.

Where could PVC dust represent a significant hazard? In a polymerization plant, work stations which could be dusty include drying and bagging areas, storage rooms or silos where PVC resin is held for shipment, mixing areas in compounding and/or fabrication facilities, and baghouses or cyclones in production or fabrication facilities. If the dusty operations are not completely isolated from the remainder of the production or fabrication facility, dust hazards could be present throughout the plant.

How dusty do PVC plants get? Two photographs (Figs. 1 and 2) taken in 1975 in a large PVC polymerization facility in the U.S. (*21*) may be of interest. Figure 1 gives a general idea of the powdery coating on the equipment in the bagging area; Figure 2 is a picture of the floor of the plant: note the depth of the dust, as indicated by the footprints.

PVC dust concentration data are not readily available; at least, the author had difficulty obtaining such data from labor, government, academic, and industry sources. It would be very helpful to those interested in problems of dust-caused disease for industry, labor or government to take measurements of PVC dust in the workplace and make them public.

Particle sizes within the respirable range have been noted (W. J. Nicholson, Mt. Sinai School of Medicine, personal communication, February 18, 1976) in a U.S. PVC polymerization plant, not the same plant as the one in which the photographs displayed as Figures 1 and 2 were taken.

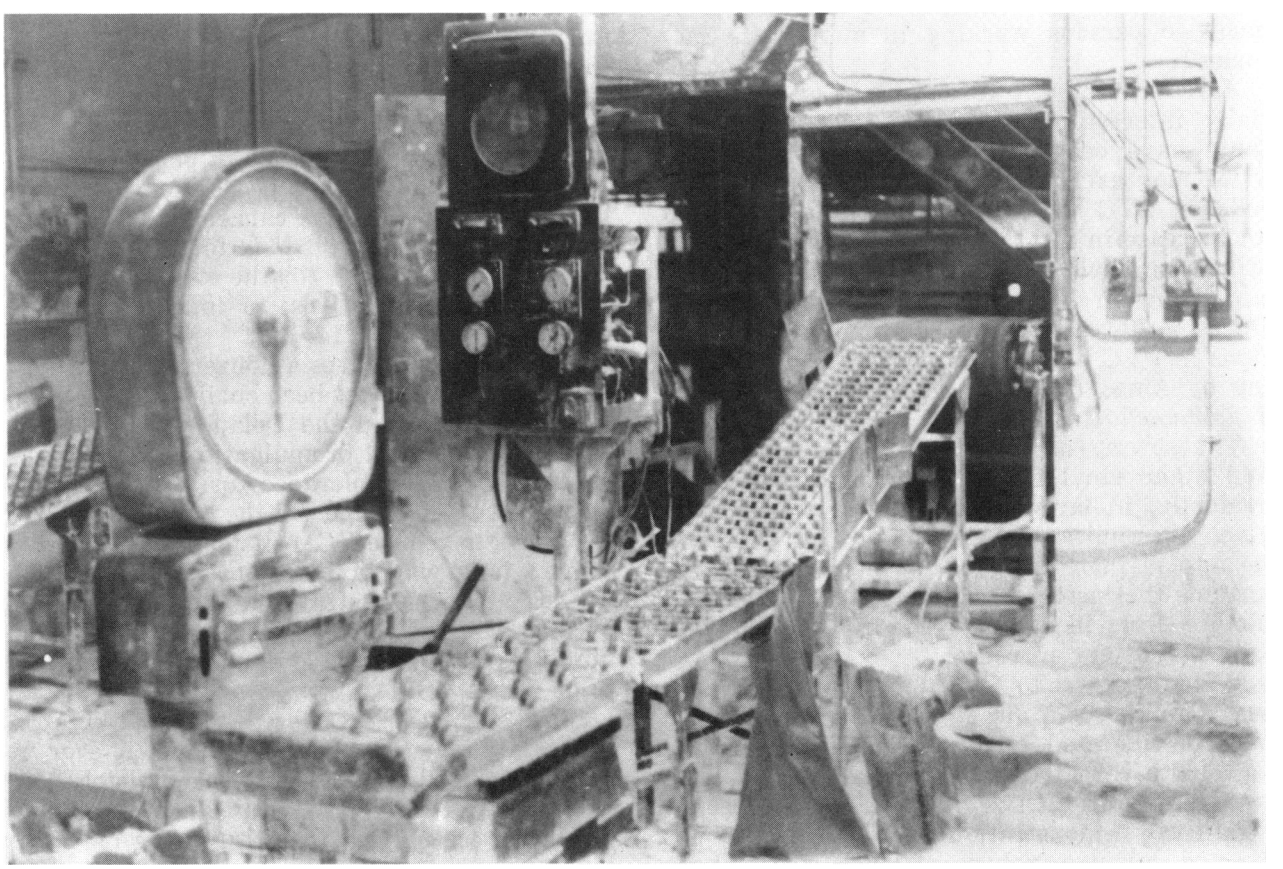

FIGURE 1. Bagging area in PVC manufacturing facility.

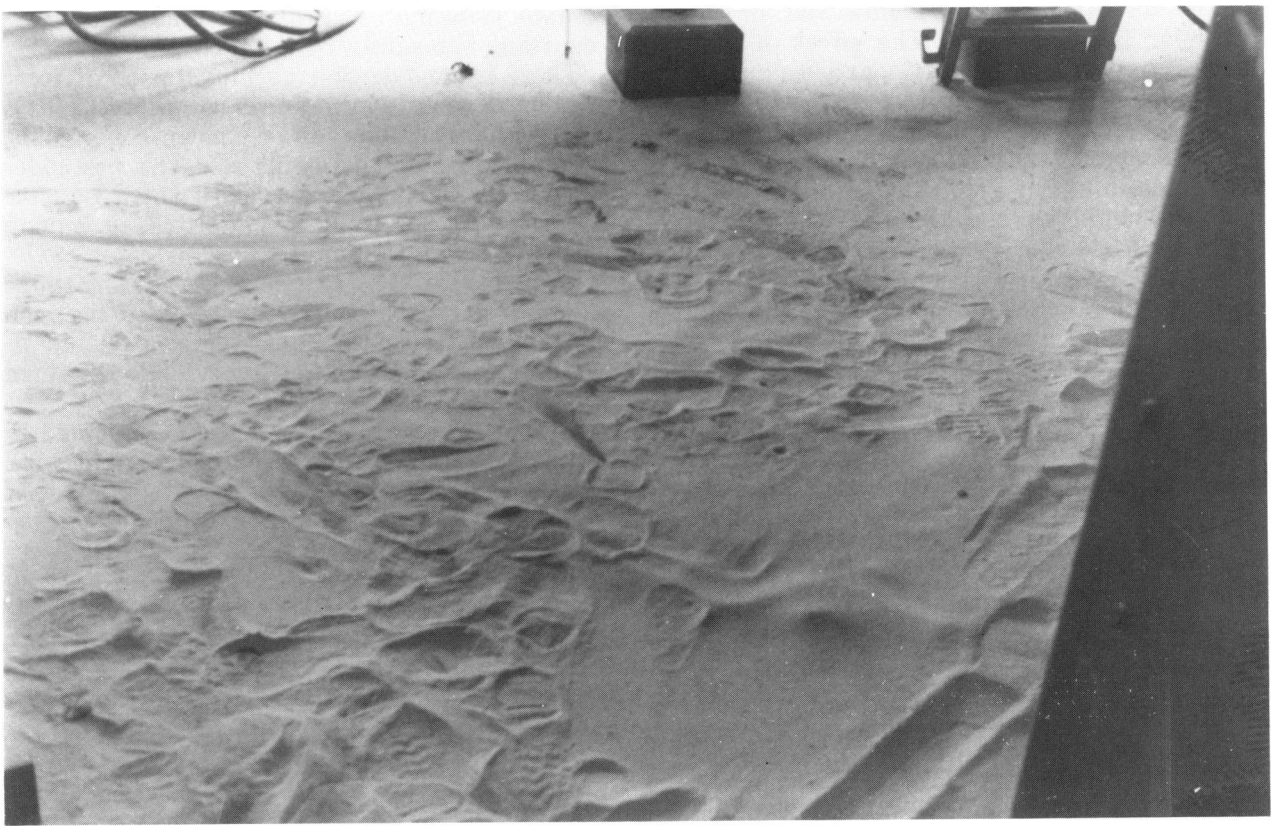

FIGURE 2. Floor of PVC manufacturing plant.

A number of clinical studies (*22–24*) have demonstrated pulmonary abnormalities in PVC production and fabrication workers. Abnormalities have ranged from decreased pulmonary function as measured by FEV to pulmonary fibrosis. Given the data currently available, work is needed to separate the effects on the lung and respiratory system of VCM from those of PVC dust.

Experimental studies associate pulmonary fibrosis with exposure of animals to PVC dust. In a recent paper from Italy, Frongia et al. (*25*) performed a series of studies with a simple basic plan-place guinea pigs or rats in cages in the bagging area of a PVC polymerization plant and observe the effects on the animals' lungs. These animal experiments were inspired by a clinical report of PVC-induced pneumoconiosis (*26*). The Frongia group, after microscopic examination of tissues of the animals under study, concluded that they had demonstrated a pneumoconiotic reaction brought on by PVC dust. Experiments reported as "in progress" as of late 1974 were designed to determine whether the pulmonary fibrosis was reversible on cessation of exposure to PVC. Data on reversibility of pulmonary and respiratory effects of PVC exposure should also be obtained for human populations.

Work is needed to determine whether VCM exposure heightens pulmonary response to PVC dust or whether, conversely, PVC dust may convey low levels of VCM or additives to the lungs.* Possible enhancement of dust effects by other chemicals in the work atmosphere and/or the additives in the PVC resin should also be investigated.

PVC dust could also be swallowed. Volkheimer (*27*) has found that PVC particles can

* According to Miller et al. (*23*) "... If exposure to vinyl chloride and/or PVC is causally related to air flow impairment, we do not know which substance is more pathogenic. The fine dust of the polymer might be more likely to concentrate in and damage the small airways. This dust, and other small respirable particles, perhaps from tobacco smoke, may also serve as carriers for molecules of a monomer gas to settle in these airways."

be "persorbed" from the intestine into the lymph or blood systems. The effect of locally high concentrations of PVC particles carrying minute amounts of potentially leachable RVCM or additives merits investigation, especially as concerns contact with lung or liver tissue.

The possibility of PVC dust serving as a skin irritant should be explored. PVC dust could serve as an efficient means for bringing a sensitizing compound into contact with the skin.

The suggestive nature of the clinical and experimental data available at this time supports reduction of PVC dust levels in polymerization, compounding and fabrication facilities. The OSHA standard (*28*) does not include PVC dust protection or reduction regulations, although such regulations were recommended by NIOSH.†

Major categories of PVC additives are colorants, fibrous reinforcements (e.g., asbestos), flame retardants, plasticizers (e.g., phthalic acid esters) and stabilizers (e.g., organotin compounds) (*28*). Based on experimental and/or clinical data, questions are in order as to the safety of representatives of each of these chemical classes.

For instance, Ames (*29*) has recently reported that a certain flame retardant compound used in children's sleepwear is highly mutagenic in a *Salmonella* test system. Compounds exhibiting mutagenic activity in this bacterial test system are likely to be carcinogens as well. What about the toxicity of flame retardants used in PVC plastics?

In September 1972, NIEHS held a conference on phthalic acid esters (*30*). What is the current thinking of regulators and researchers regarding the safety of these chemicals?

There is considerable documentation of toxic effects of certain organotin compounds (*31*). The biocidal activity of organotin compounds, several of which are being used as pesticides at present, should cause concern about possible effects of organotin plastics additives on human health.

The possibility of complex interactions among PVC plastics components and between the components and living systems should be emphasized. Most probably, very little is known about these interactions.

NIEHS has in progress projects designed to identify chemicals which should be studied for possible adverse health effects for humans. Because of its wide population exposure due to enormous production volume and broad spectrum of uses and health problems already associated with VCM and PVC dust and certain additives, PVC seems a likely candidate for further investigation.

Given the current state of scientific knowledge—at least as regards data available for public scrutiny—producers and fabricators of PVC cannot assure the public that PVC plastics are safe for human use.

Therefore, interested scientists the world over, whether working for industry, government, or labor unions, or engaged in research at universities, should make a concerted effort to collect and analyze such data as are available at present on possible risks of harm to human health attributable to PVC. Scientists should identify research which should be carried out to determine whether or not the family of PVC plastics is safe for general use, and seek the necessary funding to carry out whatever studies are needed.

† To date, neither the US OSHA nor the US EPA has taken any specific steps to reduce PVC dust considered as dust. Dust reduction and protection was recommended to the OSHA by NIOSH; EPA in its standard support document (*4*), states:

"With regard to the potential problem of polyvinyl chloride particulate as a possible cause of pneuumoconiosis, NIOSH is currently involved in experimental studies on the effects of the particulate in animals. The extent of public exposure (as opposed to occupational exposure) to ambient concentrations of the particulate is unknown at this time. Ambient measurements of polyvinyl chloride particulate have not been made by EPA in the vicinity of industrial sources because no technology is currently available for separating polyvinyl chloride particulate from total suspended particulate. As data become available from NIOSH and other sources on the health effects of polyvinyl chloride particulate, EPA may find that it is necessary to re-evaluate the need to propose standards for polyvinyl chloride particulate."

REFERENCES

1. Levinson, C. Vinyl Chloride: A Case Study of the New Occupational Health Hazard. International Chemical Federation, Geneva, Switzerland, 1974, p. 15.
2. Rowe, V. K. discussion to Ann. N.Y. Acad. Sci. 246: 317 (1975).
3. Anonymous. Tenneco PVC resins have VCM down for a 1 count . . . down to 10 parts per million (advertisement). Wall Street Journal, July 9, 1975, p. 4.
4. Environmental Protection Agency. Standard Support and Environmental Impact Statement: Emission Standard for Vinyl Chloride. EPA-450/2-75-009, U.S. Environmental Protection Agency, Office of Air and Waste Management, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, October 1975.

5. NIOSH Register of VCM-related anglosarcoma cases; this is one of the continuing series of tabulations published by the agency.
6. Wagoner, J. K. Vinyl chloride. Hearing before the Subcommittee on Environment of the Committee on Commerce, United States Senate, August 21, 1974, Serial No. 93–110, U.S. Government Printing Office, Washington, D.C. 1974, (a) p. 51; (b) p. 50.
7. Standard for Exposure to Vinyl Chloride, Occupational Safety and Health Administration, Department of Labor, 39 Fed. Reg. 35890 (October 4, 1974).
8. Barnhart, W. L., Toney, C. R., and Devlin, J. B. Environmental/industrial hygiene surveys of vinyl chloride monomer manufacturing operations and operations where polyvinyl chloride and copolymers of polyvinyl chloride are processed. Contract No. CDC–99–74–50, U.S. Department of Health, education and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health (August 1975).
9. Feairheller, W.R., Cheng, J. T., and McCormick, R. J. Sampling of automobile interiors for vinyl chloride monomer. EPA Contract No. 68–02–1404, Task 1, Cost and Performance Report, 1 June 1975 to 30 June 1975, Environmental Protection Agency, Research Triangle Park, North Carolina.
10. Infante, P. F., et al. Genetic risks of vinyl chloride. Lancet (i) 1976: 743.
11. Ducatman, A., Hirshchhorn, K., and I. J. Selikoff. Vinyl chloride exposure and human chromosome aberrations, Mutation Res. 31: 163 (1975).
12. McCann, J., et al. Detection of carcinogens as mutagens in the *Salmonella*/microsome test: assay of 300 chemicals. Proc. Nat. Acad. Sci. U.S. 72: 5135 (1975).
13. Boettner, E. A., Ball, G. L., and B. Weiss. Combustion products from the incineration of plastics. EPA Grant No. EP–00386, Program Element No. 1D2063, EPA–670/2–73–049, 25, 26 (July 1973).
14. Dyer, R. F., and Esch, V. H. Polyvinyl chloride toxicity in fires. J. Amer. Med. Assoc. 235: 393 (1976).
15. Hornblower, M. Plastic fumes insidious. Washington Post, December 11, 1975, p. A8.
16. Proceedings of International Workshop on Vinyl Chloride, May 1974. Ann. N.Y. Acad. Sci. 246, 1975.
17. Department of Health, Education and Welfare, Food and Drug Administration: Vinyl Chloride polymers in contact with food, Notice of Proposed Rulemaking, Fed. Register 40: 40529 (September 3, 1975).
18. Heckman, J. Society of the Plastics Industry, Inc.: Submission for the Record in FDA Docket No. 75–N–0190; Vinyl Chloride Polymers in Contact with Food; Notice of Proposed Rulemaking, 40 Fed. Reg. 40529, September 3, 1975 (December 19, 1975).
19. Johnson, A., and Wolfe, S. Submission for the record in proposed FDA regulation Vinyl Chloride Polymers in Contact with Food (December 19, 1975).
20. 21 USC ¶ 348(c) (3) (a).
21. Wodka, S. Oil, Chemical and Atomic Workers International Union, Citizens-Legislative Department, Washington, D.C. Photographs taken in unidentified PVC polymerization plant.
22. Lilis, R., et al. Prevalence of disease among vinyl chloride and polyvinyl chloride workers. Ann. N.Y. Acad. Sci. 246: 22 (1975).
23. Miller, A., et al. Changes in pulmonary function in workers exposed to vinyl chloride and polyvinyl chloride. Ann. N.Y. Acad. Sci. 246: 42 (1975).
24. Wegman, D. comments in discussion to paper of Lange, C. E., et al. Further results in polyvinyl chloride workers. Ann. N.Y. Acad. Sci. 246:20 (1975).
25. Frongia, N., Spinazzola, A., and A. Bucarelli. Lesioni pulmonari sperimentali da inalazione prolungata di polveri di PVC in ambiente di lavoro (Experimental lung damage from prolonged inhalation of airborne PVC dust). Med. Lavor 5: 321 (1974).
26. Szende, B., et al. Pneumoconiosis caused by the inhalation of PVC dust, Med. Lavor 61: 433 (1970).
27. Volkheimer, G. Hematogenous dissemination of ingested polyvinyl chloride particles, Ann. N.Y. Acad. Sci. 246: 164 (1975).
28. U.S. Department of Labor, Occupational Safety and Health Administration, Standard for Exposure to Vinyl Chloride. Fed. Register 29: 35890 (October 4, 1974).
29. Blum, A., and Ames, B. Flame retardant additives as possible cancer hazards: chemicals added to children's pajamas may be the wrong solution to the problem of fires. Science, in press.
30. Perspective on PAEs. Environ. Health Perspect. Exp. No. 3, 1973.
31. Piver, W. T. Organotin compounds: Industrial applications and biological investigation. Environ. Health Perspect. 4: 61 (1973).