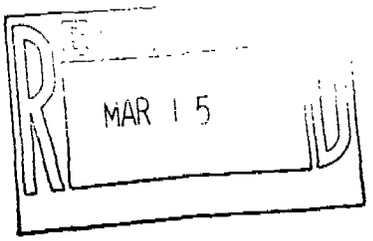
CHEMICAL MANUFACTURERS ASSOCIATION



March 13, 1989

To: VDC Panel

Re: OSHA "Z-Table" Rule for VDC

For your information, attached is a review of the final Z-Table regulation providing exposure limits for VDC for possible legal issues, as prepared by Mr. Bruce Dickson.

Sincerely yours,

Robert R Romano

Robert R. Romano, Ph.D.
Associate Director, Special
Programs Division &
Manager, Vinylidene Chloride Program

LAW OFFICES OF
PAUL, HASTINGS, JANOFSKY & WALKER
A PARTNERSHIP INCLUDING PROFESSIONAL CORPORATIONS
TWELFTH FLOOR
1050 CONNECTICUT AVENUE, N. W.
WASHINGTON, D. C. 20036
TELEPHONE (202) 223-9000
TWX 710-822-9062
TELECOPIER (202) 452-8149

COUNSEL
LEE G. PAUL
ROBERT P. HASTINGS
LEONARD S. JANOFSKY
CHARLES M. WALKER

LOS ANGELES OFFICE
555 SOUTH FLOWER STREET
LOS ANGELES, CALIFORNIA 90071
TELEPHONE (213) 683-6000

ORANGE COUNTY OFFICE
695 TOWN CENTER DRIVE
COSTA MESA, CALIFORNIA 92626
TELEPHONE (714) 641-1100

WEST LOS ANGELES OFFICE
1299 OCEAN AVENUE
SANTA MONICA, CALIFORNIA 90401
TELEPHONE (213) 451-1200

ATLANTA OFFICE
GEORGIA-PACIFIC CENTER
133 PEACHTREE STREET, N. E.
ATLANTA, GEORGIA 30303
TELEPHONE (404) 586-9900

CONNECTICUT OFFICE
ONE CANTERBURY GREEN
STAMFORD, CONNECTICUT 06901
TELEPHONE (203) 357-0100

NEW YORK OFFICE
9 WEST 57TH STREET
NEW YORK, NEW YORK 10019
TELEPHONE (212) 832-6100

TOKYO OFFICE
TORANOMON OHTORI BUILDING
4-3, TORANOMON 1-CHOME
MINATO-KU, TOKYO 105
TELEPHONE (03) 507-0730

March 7, 1989

WRITER'S DIRECT DIAL NUMBER

OUR FILE NO:

MEMORANDUM TO CMA VINYLIDENE CHLORIDE PROGRAM PANEL

Re: OSHA "Z-Table" Rule for VDC

The Panel has asked that we review the final Z-Table regulation providing exposure limits for vinylidene chloride for possible legal issues. We have reviewed it and discussed it with Dr. Romano. Specifically, we have looked into the question of whether the Panel has any reasonable objection to the final rule on the basis of an argument that the proposal gave insufficient notice that OSHA was considering a reduced exposure limit. Our conclusion is that the final rule probably was not sufficiently different from the proposed rule to warrant challenge on a theory of insufficient notice under the Administrative Procedure Act. This memorandum summarizes the law with respect to that issue and reaches the conclusion that litigation would probably not be fruitful.

We have not addressed the question of whether OSHA's conclusions about the adequacy of the proposed 5 ppm TWA or the need to adopt a 1 ppm TWA are supported by the record. OSHA's rationale for adopting the final standard -- that studies show damage at 25 ppm and that VDC is a potential carcinogen -- does not seem to be so flawed as to justify an argument that the standard is not supported by the record.

A. Proposed Rule

In its proposed "Z-Table" rule, the Occupational Safety and Health Administration ("OSHA") or ("the Agency") suggested setting permissible exposure limits ("PELs") for

vinylidene chloride ("VDC") at an 8-hour time weighted average ("TWA") of 5 parts per million ("ppm") and a 15-minute short-term exposure limit ("STEL") of 20 ppm. 53 Fed. Reg. 21106 (June 7, 1988). The Agency stated that it was concluding preliminarily that these limits would protect workers from the "risk of liver and kidney damage and carcinogenity potentially associated with exposure to VDC at the levels permitted by the absence of any OSHA limit." Id. The Agency noted, however, that this standard might be an interim limit and that OSHA would promulgate a new limit "if it determined that such a new limit would substantially reduce significant risk." Id.

OSHA indicated that the proposed rule adopted the same standard as the one advanced by the American Conference of Governmental Industrial Hygienists -- Threshold Limit Values ("ACGIH-TLV"). The proposal also noted that the National Institute on Occupational Health and Safety ("NIOSH") proposed that "employee exposure to VDC be reduced to the lowest feasible level," and stated that NIOSH considered VDC to be a carcinogen. Although there does not appear to be a specific statement in the proposal defining what NIOSH meant by "the lowest feasible level," the final rule indicates that this NIOSH recommended exposure limit ("REL") was 1 ppm -- the same level as the one finally adopted by OSHA. 54 Fed. Reg. 2567 (January 19, 1989).

The proposed OSHA rule also outlined various studies and the conclusions drawn therefrom on the toxicity and carcinogenicity of VDC.

B. Final Rule

After reviewing the studies cited and several comments (most notably, those submitted by CMA and the Worker's Institute for Safety and Health ("WISH")), the Agency concluded that "these studies clearly demonstrate that VDC can cause adverse liver and kidney damage at airborne concentrations as low as 25 to 50 ppm and suggest that VDC is a potential occupational carcinogen." Id. Thus, on January 19, 1989, OSHA adopted a final rule setting the allowable limit for exposure to 1 ppm as an 8 hour TWA.

C. Notice of Rulemaking

The Administrative Procedure Act, 5 U.S.C. §§ 551 et seq., requires agencies to give public notice of a proposed rule and to give interested parties the "opportunity to participate in rule making through submission of written data, views, or arguments, with or without opportunity for oral presentation." Id. at § 553(b). This provision does not require a proposal to specify every aspect of the rule which may ultimately be adopted in final form. It does, nonetheless, require a sufficient description of the subjects and issues involved so as to allow interested parties a meaningful opportunity to comment. Trans-Pacific Conference of Japan/Korea v. Federal Maritime Comm'n, 650 F.2d 1235 (D.C. Cir. 1980), cert. denied, 101 S. Ct. 2315 (1980). If notice of the proposed rule is too broad or the final agency rule deviates too sharply from the proposal, the affected parties will be deemed to have been deprived of notice and of an opportunity to comment. Small Refiner Lead Phase-down Task Force v. EPA, 705 F.2d 506, 547 (D.C. Cir. 1983); Chocolate Manufacturers Ass'n of the U.S. v. Block, 755 F.2d 1098 (4th Cir. 1985.

This does not mean, however, that when an agency adopts a final rule which contains substantial changes from the proposed rule the new rule is automatically invalid and the agency needs to open a new comment period. International Harvester Co. v. Ruckleshaus, 478 F.2d 615, 632 n.51 (D.C. Cir. 1973). Nevertheless, to be valid, the change must represent a "logical outgrowth" of the prior notice and comments. South Terminal, 504 F.2d at 659.

In determining whether a final regulation is a "logical outgrowth" of a proposed rule, the court must "proceed to compare carefully the specific language of the proposal with that of the final rule in light of the evidence adduced at the hearings." United Steelworkers v. Marshall, 647 F.2d 1180, 1221 (D.C. Cir. 1980). This standard essentially means that "given a new opportunity to comment, commenters would not have their first occasion to offer new and different criticisms which the agency might find convincing." BASF Wyandote Corp. v. Costle, 598 F.2d

637, 642 (1st Cir. 1979), cert. denied, 444 U.S. 1096, 100 S. Ct. 1063 (1980).

D. Case Law

In the following cases, the court found that adequate notice had been given:

1. American Iron and Steel Institute v. EPA, 568 F.2d 284 (3rd Cir. 1977).

EPA proposed regulations establishing maximum permissible levels of pollutants which could be discharged by certain manufacturing operations within the iron and steel industry. The proposed regulations surveyed the available pollution control techniques -- the best practable control technology currently available ("BPCTCA") -- that could be used in meeting these limitations. The court held that interested parties were sufficiently apprised that there was an issue as to whether recycling of partially clarified effluent was the BPCTCA and, therefore, that the Agency might adopt more stringent treatment technologies than those embodied in the proposed rule. Thus, the final rule was a "logical outgrowth" of the proposal.

2. South Terminal Corp. v. EPA, 504 F.2d 646 (1st Cir. 1974).

In order to reduce carbon monoxide emissions in the Boston area, EPA proposed (1) a ban on street parking in downtown; (2) prohibiting travel on certain routes one day a week through a $5 sticker system; and (3) limiting the supply of gasoline. The Agency also warned the public that other alternatives being considered, including a plan for fewer available parking spaces downtown. After extensive public comment, the sticker and gas supply options were dropped. The final rule adopted a plan which had not been previously recommended and that required a freeze on present and a review of future parking spaces. The court upheld the final rule stating that "[a]lthough the changes were substantial, they were in character with the original scheme and were additionally foreshadowed in proposals and comments advanced during the rulemaking. Parties had been

warned that strategies might be modified in light of their suggestions." Id. at 658.

3. Small Refiner Lead-Phase Down Task Force v. EPA, 705 F.2d 506 (D.C. Cir. 1983).

EPA proposed setting lead content limits for small refineries ("SRs") at 2.5 grams per leaded gallon ("gplg") which was more lenient than the level proposed for large refineries ("LRs"). The final rule set a much stricter level of 1.1 gplg, which was based on past production levels, for both SRs and LRs. This final rule reflected an attempt to close certain loopholes created by the disparate treatment of SRs and LRs. The notice has raised this concern but had not specifically listed which loopholes EPA might attempt to close. Nevertheless, the court found that SRs were on adequate notice of the past production requirements because they were generally aware that EPA was seeking to seal unidentified loopholes that might affect them and they were in fact aware that the LRs had proposed considering past production requirements. Thus, the final rule flowed logically from the earlier proposal. In addition, the court noted that the SRs' attorney had attended the public hearings and was closely monitoring the docket and that the SRs had responded to other information to which they objected.

In the following cases the Court found that there had not been adequate notice:

1. AFL-CIO v. Donovan, 757 F.2d 330 (D.C. Cir.; 1985).

Prior to the final regulation amending the Service Contract Act, the definition under that Act of "in the United States" covered situations where any part of the contract was performed in the U.S. The two proposals amending the Act highlighted the sections to be amended. Neither notice indicated that the definition of "in the United States" was to be changed to cover only those actions actually performed in the U.S. The court held that because the change appeared only in the final rule itself without indication of the pending change, the modification was not a "logical outgrowth" of the proposed rule.

2. Chocolate Manufacturers Ass'n ("CMA") v. Block, 755 F.2d 1098 (4th Cir. 1985).

Where the Food and Nutrition Services of the USDA proposed a rule that would limit the sugar content allowed in supplemental foods -- without indicating that chocolate flavored milk would be affected by the final regulation -- the court held that the agency had not given CMA adequate notice. The court noted that although the final rule was an outgrowth of the proposed regulation, it wasn't a logical one, as chocolate milk had always been a permissible supplemental food in the program and there had never been any hint that it would be removed therefrom. Thus, CMA could not have anticipated such a provision in the final rule and therefore had not been adequately alerted of the need for comment.

Conclusion

Insufficient notice does not appear to be a valid basis for objecting to OSHA's final rule on VDC exposure limits. In its proposal, OSHA implied that several alternatives were being considered, including NIOSH's REL. CMA knew that OSHA and NIOSH had jointly recommended a 1 ppm VDC exposure level in 1978 and that NIOSH proposed that VDC be designated an occupational carcinogen. In its comments, CMA responded directly to that allegation and attempted to establish the NIOSH's proposal was unsupported by scientific evidence. CMA also recommended that ACGIH-TLVs be adopted (5/20 ppm) rather than NIOSH RELs (1 ppm).

Because NIOSH's low TLV recommendation was mentioned in the proposed rule, even if in passing, and CMA referred to it in its comments, the proposed rule would probably be held to give adequate notice, and the final rule would be held to be a "logical outgrowth" of the proposed standard. In addition, if a comment period were to be re-opened, CMA would not be getting its first opportunity to offer new criticisms or suggestions that the agency might find convincing. CMA has already advocated that VDC is not a likely carcinogen and that ACGIH-TLVs be used as the basis for updating the Z-Table limits. OSHA addressed these claims but concluded that a lower exposure limit was warranted.

Further, OSHA specifically stated in the proposal that it might promulgate a new limit in the final rule if doing so would substantially reduce a significant risk. This statement, coupled with OSHA's direct evaluation of CMA's comment and other studies in the final rule and its notice in the proposal that it was considering the 1 ppm limit, makes it difficult to argue that the final rule deviated too sharply from the proposed one, thereby depriving CMA of notice.

On the basis of the above discussion, it would be difficult to argue that the VDC Panel did not have sufficient notice of OSHA's consideration given to reducing the exposure levels for VDC to a level below the proposal. Consequently, it is unlikely that CMA could argue that there was insufficient notice of the proposed change in exposure limits to VDC.

R. Bruce Dickson
Leslie A. Gonzalez

RBD/mbl

tungsten (1977h, as cited in ACGIH 1986/Ex. 1–3, p. 614), NIOSH states that information on the effects of exposure to soluble tungsten compounds in the working population is not available. The ACGIH (1986/Ex. 1–3, p. 614) recommends a lower TLV for the soluble, as compared to the insoluble, compounds of tungsten because of the former's greater systemic toxicity. No comments other than those of NIOSH (Ex. 8–47) were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA of 1 mg/m³ and a STEL of 3 mg/m³ for tungsten and its soluble compounds, measured as tungsten. The Agency concludes that these limits will protect workers against the significant risks of systemic toxicity, anorexia, colic, incoordination, trembling, and dyspnea, all of which constitute material health impairments that are associated with exposure to these compounds at levels above the new PELs.

VINYLIDENE CHLORIDE (1,1-DICHLOROETHYLENE)

CAS: 75–35–4; Chemical Formula: $CH_2 = CCl_2$ H.S. No. 1428

Previously, OSHA's Z tables did not include a limit for vinylidene chloride (VDC). The ACGIH has established 5 ppm as an 8-hour TWA and 20 ppm as a 15-minute STEL. NIOSH and OSHA, in 1978, jointly recommended that employee exposure to VDC be reduced to the lowest feasible level on the basis of VDC's carcinogenicity (NIOSH/OSHA 1978/Ex. 1–1119). OSHA proposed a PEL of 5 ppm (8-hour TWA) and a STEL of 20 ppm. However, in response to record comments, the final rule promulgates a 1-ppm limit as an 8-hour TWA. Vinylidene chloride is a colorless liquid that polymerizes readily.

The acute oral LD_{50} for male rats is 2500 mg/kg (Jenkins, Trabulus, and Murphy 1972/Ex. 1–960). The LC_{50} for rats exposed to a single four-hour exposure of VDC vapor was reported as 6350 ppm in one study (Siegel, Jones, Coon, and Lyon 1971/Ex. 1–371) and 32,000 ppm in an earlier study (Carpenter, Smyth, and Pozzani 1949/Ex. 1–722). Liquid VDC causes transient irritation to the eyes of rats but has little effect on exposed skin if the VDC is allowed to evaporate (Torkelson and Rowe 1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628).

Prendergast and co-workers (1967/Ex. 1–926) exposed rats, rabbits, guinea pigs, and monkeys eight hours/day, five days/week for six weeks to 395 mg/m³ (100 ppm); these authors saw no visible signs of toxicity while the exposure was in process, but rabbits and monkeys lost weight. These same species were exposed continuously to VDC concentrations of 5, 15, 25, or 47 ppm for 90 days; only the animals exposed to 5 ppm showed no increases in mortality (Prendergast, Jones, Jenkins, and Siegel 1967/Ex. 1–926).

Nasal irritation, liver cell degeneration, and retarded weight gain were reported in rats following 20 six-hour exposures to 500 ppm VDC (Gage 1970/Ex. 1–318); at 200 ppm, only nasal irritation occurred. Studies by Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) in which rats, rabbits, guinea pigs, and dogs were exposed to 25, 50, or 100 ppm VDC for eight hours per day, five days per week for six months revealed injury of the kidneys and liver in all animals at all levels of exposure. Maltoni (1977/Ex. 1–985) and Maltoni, Cotti, Morisi, and Chieco (1977/Ex. 1–1090) conducted an evaluation of VCD's carcinogenicity in which mice, rats and hamsters were exposed to levels from 10 to 150 ppm for four hours per day, five days per week for 52 weeks, with results reported through week 98 of the study. In those mice exposed to 25 ppm VCD, 21 percent of the males and 1.5 percent of the females developed kidney adenocarcinomas; these tumors were not seen in rats exposed to amounts of VDC up to 150 ppm. Exposures of 100 or 150 ppm in rats did produce a significant increase in mammary adenocarcinomas, and this response was dose-related (Maltoni 1977/Ex. 1–985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1–1090). Overt toxicity and mortality occurred early in the studies after four-hour exposures at levels of 50 ppm in mice and 200 ppm in rats; hamsters exposed to 20 ppm VDC showed no increase in tumor incidence (Maltoni 1977/Ex. 1–985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1–1090).

A study by Murray, Nitschke, Rampy, and Schwetz (1979/Ex. 1–920) investigated the embryotoxic, fetotoxic, and teratogenic effects of inhaled and ingested VDC (in rats) and inhaled VDC (in rabbits). In the inhalation studies, rats were exposed to 20, 80, or 160 ppm VDC for seven hours per day. VDC was toxic to both the adults and their embryos at levels of 80 and 160 ppm among the rats, and at 160 ppm in rabbits. At exposure levels of 20 ppm in rats and 80 ppm in rabbits, neither maternal toxicity nor effects on embryonic or fetal development were noted. In the ingestion study with rats, drinking water containing 200 ppm VDC caused no toxic effects in either the rats or their offspring.

Two strains of rats exposed to 75 or 100 ppm VDC for five days/week, six hours/day for 12 months did not show a significant increase in tumors (Viola and Caputo 1977/Ex. 1–937). Other investigators exposed rats to 25 or 75 ppm by inhalation for six hours/day, five days/week for 18 months, or to 60, 100, or 200 ppm VDC in their drinking water for two years, and found no increase in tumor incidence in these animals (Rampy, Quast, Humiston et al. 1977, as cited in ACGIH 1986/Ex. 1–3, p. 628). In mice, VDC was not active either as a whole mouse skin carcinogen or by subcutaneous injection.

In other studies, VDC proved mutagenic in both *E. coli* and *S. typhimurium* strains (Greim, Bonse, Radwan et al. 1975/Ex. 1–904; Bartsch, Malaveille, Montesano, and Tomatis 1975/Ex. 1–869). VDC has been implicated as a tumor initiator in a carcinogenesis bioassay by Van Duuren, Goldschmidt, Loewengart et al. (1979/Ex. 1–936). Studies by Reitz, Watanabe, McKenna et al. (1980/Ex. 1–927) suggest that VCD's tumorigenicity is a result of its ability to initiate cell injury, rather than of its ability to alter the genetic material of an injured cell. However, VDC has been shown to alkylate DNA *in situ* and increase the rate of DNA repair to a small extent in mice (Norris and Reitz 1984/Ex. 134B). The actual cell injury is caused by VDC metabolites, which are highly reactive and cytotoxic (Maltoni 1977/Ex. 1–985; Hathway 1977/Ex. 1–906; Henschler and Bonse 1977/Ex. 1–908).

A cohort study of 138 VCD-exposed workers did not identify any VCD-related health effects in these workers (Ott, Fishbeck, Townsend, and Schneider 1976/Ex. 1–924). The cohort was too small to provide any evidence that VDC is not likely to be carcinogenic.

The Chemical Manufacturers Association submitted the results of an NTP gavage study of VDC in mice and rats (NTP 1982/Ex. 134B). The only observed significant increase in tumor incidence occurred in low-dose female mice; this increase was not considered to be related to VDC administration because similar effects were not observed in high-dose female mice, male mice, or rats. The NTP (1982/Ex. 134B) concluded that VDC was not carcinogenic in mice or rats exposed by gavage, but cautioned that a maximum tolerated dose had not been demonstrated and that previously reported studies had shown that carcinogenicity is associated with VDC inhalation by animals.

Based on the carcinogenicity evidence described above, NIOSH (Ex. 8–47, Table N6B) indicated that VDC is a suitable candidate for an individual 6(b)

final rule

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rulemaking. However, the CMA (Ex. 165) was of the opposite opinion, stating that the demonstrated lack of tumor response in most studies, coupled with evidence that VDC metabolism is species-specific, "demonstrates that VDC is unlikely to pose an oncogenic risk to humans" (Ex. 165, p. 42). CMA also objected to the statement by NIOSH and OSHA in the joint *Current Intelligence Bulletin on VDC* (NIOSH/OSHA 1978/Ex. 1–1119) that VDC be considered a potential carcinogen because of its structural similarity to vinyl chloride; the CMA considered this statement inappropriate, given the toxicity data available.

Matthew Gillen and Scott Schneider of the Workers Institute for Safety and Health (WISH) commented that the proposed 5-ppm PEL and 20-ppm STEL for VDC would not provide sufficient protection from systemic effects (Ex. 116). They pointed out that the study by Prendergast et al. (1967/Ex. 1–926) found 15 ppm to be the lowest effect level for increased mortality in animals, and that the Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) study found liver and kidney injury in animals. These commenters stated that the ACGIH TLV cannot be considered to provide adequate protection for this substance. Given this fact, OSHA should consider the NIOSH REL of 1 ppm as an interim value until further risk assessment studies can be carried out" (Ex. 116).

OSHA has re-examined the health evidence in light of the comment by WISH, and has determined that the proposed 5-ppm TWA PEL for VDC does not afford workers sufficient protection from systemic effects. Although it is questionable, in the Prendergast et al. (1967/Ex. 1–926) study, that the observed deaths at lower exposure levels were compound-related, histopathologic examination of animals exposed to 47 ppm showed treatment-related liver and kidney damage. Using an exposure regimen similar to occupational exposure (i.e., eight hours/day, five days/week), Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) demonstrated kidney and liver toxicity in four species of animals after exposure to VDC levels as low as 25 ppm were administered for only six months.

OSHA believes that these studies clearly demonstrate that VDC can cause adverse liver and kidney damage at airborne concentrations as low as 25 to 50 ppm and suggest that VDC is a potential occupational carcinogen. Liver and kidney damage and cancer clearly constitute material health impairments within the meaning of the Act. Therefore, OSHA concludes that the proposed limits of 5 ppm as an 8-hour TWA and 20 ppm as a STEL will not sufficiently protect workers from the significant risk of organ damage, and that a further reduction in the PEL is warranted. Accordingly, OSHA is establishing a 1-ppm 8-hour TWA limit for vinylidene chloride in the final rule.

WELDING FUMES

CAS: None; Chemical Formula: Not available
H.S. No. 1430

OSHA formerly had no limit for exposure to welding fumes, which are defined as fumes that are generated by the manual metal arc or oxy-acetylene welding of iron, mild steel, or aluminum. The ACGIH has set an 8-hour TWA of 5 mg/m³ for these welding fumes, measured as total particulate in the welder's breathing zone. OSHA proposed an 8-hour TWA of 5 mg/m³ for these fumes; this limit is established in the final rule. This limit applies to the total fume concentration generated during the welding of iron, mild steel, or aluminum; the fumes generated by the welding of stainless steel, cadmium, or lead-coated steel, or other metals such as copper, nickel, or chrome are considerably more toxic and should be kept at or below the levels required by their respective PELs. Welding fumes consist of metallic oxides generated by the heating of metal being welded, the welding rod, or its coatings.

Although these types of welding generally produce fumes consisting of aluminum, iron, or zinc oxides, other toxic gases may also be produced in large amounts (Ferry and Ginther 1952/Ex. 1–900; Ferry 1954/Ex. 1–782; Silverman 1956/Ex. 1–1169; Homer and Mohr 1957/Ex. 1–787). The welding of iron metals may give off fumes of manganese, silicate, and various organic binders. Aluminum welding may generate fumes consisting of fluorine, arsenic, copper, silicon, and beryllium (NIOSH 1975b and American Welding Society 1974, both as cited in ACGIH 1986/Ex. 1–3, p. 634). Eighteen different substances, including fluoride, manganese, silicon, titanium, and sodium and potassium silicates, have been measured in the fumes resulting from the welding of mild steel (ACGIH 1986/Ex. 1–3, p. 634).

Excessive exposure to welding fume can cause a variety of disorders, most notably metal fume fever. It has been estimated that 30 to 40 percent of all welders have experienced metal fume fever at some time (Abraham 1983, in *Environmental and Occupational Medicine*, W.N. Rom, ed., p. 146). This disorder, which results from exposure to freshly formed metal fume, results in the appearance of delayed, flu-like symptoms, including dyspnea, coughing, pains in muscles and joints, fever, and chills. Recovery usually requires one or two days of time away from work. In addition to fume fever, exposure to welding fume may damage the small airways, causing interstitial pneumonia (Abraham 1983).

Several commenters, the American Iron and Steel Institute (Exs. 129, 188), the Abbott Laboratories (Tr. 9–155 to 9–156), and the American Welding Society (Ex. 3–860), were of the opinion that OSHA's discussion of welding fumes in the NPRM was not clear with regard to whether the limit applied to exposure samples taken inside or outside of the welding helmet. OSHA wishes to clarify that welding fume is to be measured in the breathing zone of the welder; the specific details of the appropriate positioning of the sampler should be determined on the basis of guidance in the *Field Operations Manual* (OSHA 1984). This is consistent with a past OSH Review Commission decision (8 OSHRC 1049).

NIOSH (Ex. 8–47) stated at the hearing that welding fumes should be designated as a carcinogen. This view was also endorsed by Dr. James Melium, of the New York State Department of Health (Tr. p. 11–104). In response to these commenters, OSHA notes that there are few data sufficient to establish a dose-response for the fumes. Accordingly, OSHA believes it would be premature to identify these fumes as potential occupational carcinogens.

OSHA concludes that a PEL for welding fumes is needed to protect workers involved in the welding of aluminum, iron, or mild steel from the significant risk of metal fume fever and respiratory irritation associated with the generation of welding fumes. In the final rule, OSHA is establishing a TWA of 5 mg/m³ for these particular types of welding fumes, measured as total particulate inside the welder's breathing zone. The Agency finds that this limit will substantially reduce the significant risk of material health impairment to which manual metal arc or oxy-acetylene welders of iron, mild steel, or aluminum were previously exposed in the absence of any OSHA limit.

ZINC OXIDE (FUME)

CAS: 1314–13–2; Chemical Formula: ZnO
H.S. No. 1437

OSHA's former exposure limit for zinc oxide fume was 5 mg/m³ as an 8-hour TWA. The ACGIH recommends a 5-mg/m³ TWA and also has a STEL of 10 mg/m³. NIOSH recommends a 5-mg/m³ 10-

basis for proposing a new limit for tungsten and compounds (soluble). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

VINYLIDENE CHLORIDE (1,1-DICHLOROETHYLENE)

CAS: 75-35-4; Chemical Formula: $CH_2=CCl_2$
H.S. No. 1428

Currently, OSHA's Z tables do not have a limit for vinylidene chloride (VCD). The ACGIH has established 5 ppm as an 8-hour TWA and 20 ppm as a 15-minute STEL. NIOSH recommends that employee exposure to VCD be reduced to the lowest feasible level, and considers VCD a carcinogen. Vinylidene chloride is a colorless liquid that polymerizes readily.

The acute oral LD_{50} for male rats is 2500 mg/kg (Jenkins, Trabulus, and Murphy 1972). The LC_{50} for rats exposed to a single 4-hour exposure of VCD vapor was reported as 6350 ppm in one study (Siegel, Jones, Coon, and Lyon 1971) and 32,000 ppm in an earlier study (Carpenter, Smyth, and Pozzani 1949). Liquid VCD causes transient irritation to the eyes of rats but has little effect on exposed skin if the VCD is allowed to evaporate (Torkelson and Rowe 1981).

Prendergast and co-workers exposed rats, rabbits, guinea pigs, and monkeys 8 hours/day, 5 days/week for 6 weeks to 395 mg/m³ (100 ppm); these authors saw no visible signs of toxicity while the exposure was in process, but rabbits and monkeys lost weight. These same species were exposed continuously to VCD concentrations of 5, 15, 25, or 47 ppm for 90 days; only the animals exposed to 5 ppm showed no increases in mortality (Prendergast, Jones, Jenkins, and Siegel 1967).

Nasal irritation, liver cell degeneration, and retarded weight gain were reported in rats following twenty 6-hour exposures to 500 ppm VCD (Gage 1970); at 200 ppm, only nasal irritation occurred. Studies by Torkelson and Rowe (1981) in which rats, rabbits, guinea pigs, and dogs were exposed to 25, 50, or 100 ppm VCD for 8 hours per day, 5 days per week for 6 months revealed injury of the kidneys and liver in all animals at all levels of exposure. Maltoni (1977) and Maltoni, Cotti, Morisi, and Chieco (1977) conducted an evaluation of VCD's carcinogenicity in which mice, rats and hamsters were exposed to levels from 10 ppm to 150 ppm for 4 hours per day, 5 days per week for 52 weeks; with results reported through week 98 of the study. In those mice exposed to 25 ppm VCD, 21 percent of the males and 1.5 percent of the females developed kidney adenocarcinomas; these tumors were not seen in rats exposed to amounts of VCD up to 150 ppm. Exposures of 100 or 150 ppm in rats did produce a significant increase in mammary adenocarcinomas, and this response was dose-related (Maltoni 1977; Maltoni, Cotti, Morisi, and Chieco 1977). Overt toxicity and mortality occurred early in the studies after 4-hour exposures at levels of 50 ppm in mice and 200 ppm in rats; hamsters exposed to 20 ppm VCD showed no increase in tumor incidence (Maltoni 1977; Maltoni, Cotti, Morisi, and Chieco 1977).

A study by Murray, Nitschke, Rampy, and Schwetz (1979) investigated the embryotoxic, fetotoxic, and teratogenic effects of inhaled and ingested VCD (in rats) and inhaled VCD (in rabbits). In the inhalation studies, rats were exposed to 20, 80, or 160 ppm VCD for 7 hours per day. VCD was toxic to both the adults and their embryos at levels of 80 and 160 ppm among the rats, and 160 ppm in rabbits. At exposure levels of 20 ppm in rats and 80 ppm in rabbits, neither maternal toxicity nor effects on embryonic or fetal development were noted. In the ingestion study with rats, drinking water containing 200 ppm VCD caused no toxic effects in either the rats or their offspring.

Two strains of rats exposed to 75 or 100 ppm VCD for 5 days/week, 6 hours/day for 12 months did not show a significant increase in tumors (Viola and Caputo 1977). Other investigators exposed rats to 25 or 75 ppm by inhalation for 6 hours/day, 5 days/week for 18 months or to 60, 100, or 200 ppm VCD in their drinking water for 2 years and found no increase in tumor incidence in these animals (Rampy, Quast, Humiston et al. 1977). In mice, VCD was not active either as a whole mouse skin carcinogen or by subcutaneous injection.

In other studies, VCD proved mutagenic in both E. coli and S. typhimurium strains (Greim, Bonse, Radwan et al. 1975; Bartsch, Malaveille, Montesano, and Tomatis 1975). VCD has been implicated as a tumor initiator in a carcinogenesis bioassay by Van Duuren (1979). Studies by Reitz, Watanabe, McKenna et al. (1980) suggest that VCD's tumorigenicity is a result of its ability to initiate cell injury and not of its ability to alter the genetic material of an injured cell. The actual cell injury is caused by VCD metabolites which are highly reactive and cytotoxic (Maltoni 1977; Hathaway 1977; Henschler and Bonse 1977).

A cohort study of 138 VCD-exposed workers did not identify any VDC-related health effects in these workers (Ott, Fishbeck, Townsend, and Schneider 1976).

OSHA is proposing an 8-hour TWA of 5 ppm and a 15-minute STEL of 20 ppm for vinylidene chloride. The Agency preliminarily concludes that these limits will protect workers from the risk of kidney and liver damage and carcinogenicity potentially associated with exposure to VCD at the levels permitted by the absence of any OSHA limit. This limit may be an interim limit; as future priorities permit, the Agency may perform a quantitative risk assessment for VCD and consider further rulemaking. This health evidence forms a reasonable basis for proposing a new limit for vinylidene chloride (1,1-dichloroethylene). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

WELDING FUMES

CAS: None; Chemical Formula: Not available
H.S. No. 1430

OSHA currently has no limits for exposure to welding fumes, which it defines as fumes that are generated by the manual metal arc or oxy-acetylene welding of iron, mild steel, or aluminum. The ACGIH has set an 8-hour TWA of 5 mg/m³ for welding fumes, measured as total particulate inside the welding helmet.

Although welding of these types generally produces fumes made up of aluminum, iron, or zinc oxides, other toxic gases may be produced in large amounts (Ferry and Ginther 1952; Ferry 1954; Silverman 1956; Homer et al. 1957). Iron metals may give off fumes of manganese, silicate, and various organic binders. Aluminum welding may result in fumes consisting of fluorine, arsenic, copper, silicon, and beryllium (NIOSH n.d.; American Welding Society 1973). Eighteen different substances, including fluoride, manganese, silicon, titanium, and sodium and potassium silicates, have been measured in the fumes resulting from the welding of mild steel (ACGIH 1986, p. 634). The process of shielded arc welding is known to produce ozone, and when carbon dioxide is used as a shield gas, carbon monoxide is given off (NIOSH n.d.; American Welding Society 1973).

The adverse health effects associated with over exposure to welding fumes are those of metal fume fever—chills and fever, profuse sweating, and weakness—and respiratory irritation.

OSHA preliminarily concludes that a PEL for welding fumes is needed to protect workers involved in the welding of aluminum, iron, or mild steel from the

proposed rule