CRITIQUE OF DIOXIN FACTORIES

prepared for:

The Vinyl Institute
Wayne Plaza II
155 Route 46 West
Wayne, New Jersey 07470

prepared by:

ChemRisk®
A Division of McLaren/Hart Environmental Engineering
1685 Congress Street
Portland, Maine 04102
(207) 774-0012
(207) 774-8263 (FAX)

September 1, 1993

ChemRisk®
A Division of McLaren/Hart
Environmental Engineering

CTL014677

CRITIQUE OF DIOXIN FACTORIES

TABLE OF CONTENTS

APPENDIX
ARTICLES CITED IN *DIOXIN FACTORIES* AND OBTAINED BY CHEMRISK

VOLUME I - A THROUGH F

- Adelt. 1990. Letter to the Regierungsprasident, Dusseldorf. "Untersuchung des Abwassers auf Dioxine und Furane, Deutsche Solvay Werke Rheinberg." 23.08.1990. From Dr. Adelt, Staatliches amt fur Wasser - und Abfallwirtschaft (STAWA), Dusseldorf.

- Behrens, C. 1990. VCM-fabrikk, Rafnes - Dioksiner. Docnr 90C.BA6. Norsk Hydro Research Centre, Forskningssenter Porsgrunn. Project nr. R70035. July 18.

- Cato, I. 1992. *Sedimentological Investigations of the Bohus Coast 1990 - The Coastal Water Monitoring Program of the Goteberg and Bohus County*. Geological Survey of Sweden, Sveriges Geologiska Undersokning. Rapporter och meddelanden nr 74. 97 pp. Uppsala, Sweden.

- Claus, F., H. Friege, and D. Gremler. 1990. "Es Geht auch ohne PVC: Einsatz-Entsorgung-Ersatz ein Ratgeber,"Hamburg: Rasch und Rohring Verlag. (In German).

TABLE OF CONTENTS (CONT'D)

- Erickson, M.D., J.S. Stanley, J.K. Turman, and J.E. Going. 1988. Determination of byproduct polychlorobiphenyls in commercial products and wastes by high-resolution gas chromatography / electron impact mass spectrometry. *Environ. Sci. Technol.* 22:71-76.

- Evers, E.H.G., H. Govers, K. Olie, J.W. van Berghem, and V. Bakker. 1989. *De Vorming van PCDFs, PCDDs en Gerelateerde Verbingdingen Bij de Oxychlorering van Etheen.* MTC-publikatie nr. MTC89EE, University of Amsterdam. December.

- Evers, E.H.G., K.C.M. Ree, and K. Olie. 1988. Spatial variations and correlations in the distribution of PCDDs, PCDFs, and related compounds in sediments from the River Rhine - Western Europe. *Chemosphere.* 17(12):2271-2288.

- Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland, and A.J. Suruda. 1991a. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *New England J. Med.* 324(4):212-218.

- Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland, and A.J. Suruda. 1991b. Mortality among U.S. workers employed in the production of chemicals contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Industry Studies Branch, Cincinnati, OH. January. (Submitted to New England Journal of Medicine.)

VOLUME II - G THROUGH M

- Greenpeace International. 1992. *PVC: Toxic Waste in Disguise.* Greenpeace International, Amsterdam, The Netherlands.

- Hagen, S. 1993. Svensk undersokelse av sedimenter pa vestkysten. Letter of February 24, 1993, from the Norwegian EPA to Ministry of Environment. Statens forurensningstilsyn (SFT), Oslo, Norway.

- Hagen, S. 1993. Kartlegging av dannelse av dioksin ve produksjon av VCM. Letter of January 19, to Greenpeace from the Norwegian EPA. Statens forurensningstilsyn (SFT), Oslo, Norway.

- Heindl, A. and O. Hutzinger. 1987. Search for industrial sources of PCDD/PCDF III. Short-chain chlorinated hydrocarbons. *Chemosphere.* 16(8-9):1949-1957.

- Hydro Plast. 1992. Erinringar med anledning av Nya yttranden 1992-09-23 och 1992-10-02 fran Greenpeace i arende. M92/610-8

TABLE OF CONTENTS (CONT'D)

- Jenkins, C. 1991. Affidavit of Cate Jenkins, Ph.D., Exhibit H, Recent scientific evidence developed after 1984 supporting a causal relationship between dioxin and human health effects. In the United States District Court for the Eastern District of New York. Shirley Ivy, Individually and as Representative of the Estate of Donald Ivy, et al., Plaintiffs v. Diamond Shamrock Chemicals Company et al., Defendants. CV-89-03361 (E.D.N.Y.) (J.B.W.), [B-89-00559-CA (E.D.TEX)].

- Kallqvist, T. 1991. *Kjemisk/Biologisk Karakterisering av avlopsvann fra Norsk Hydro A.S. Rafnes*. Projektnr: 0-90223. Norsk Institutt for Vannforskning (NIVA), Senter for Industriforskning, Oslo, Norway.

- Knutzen, J. and M. Oehme. 1989. Polychlorinated dibenzofuran (PCDF) and dibenzo-p-dioxin (PCDD) levels in organisms and sediments from the Frierfjord, Southern Norway. *Chemosphere*. 19(12):1897-1909.

- Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46:279-303.

- Kollman, H., R. Heckler, W. Huber, S. Husung, K.-D. Sturm, and F. Wendland. 1990. Stoffstrome und Emissionen durch Produktion, Verwendung und Entsorgung von PVC. Angewandte Systemanalyse nr. 59. Forshungszentrum Julich GmbH, Programmgruppe Systemforschung und Technologische Entwicklung, Julich. July.

- Lykke, E. 1993. Hydro's expansion at Rafnes - Huge mistaken investment. First chapter in new book on environmental performance. (In Press).

- Manz, A., J. Berger, J.H. Dwyer, D. Flesch-Janys, S. Nagel, and H. Waltsgott. 1991. Cancer mortality among workers in chemical plant contaminated with dioxin. *The Lancet*. 338:959-964.

VOLUME III - N THROUGH Z

- NILU. 1990. *Sporstoffundersokelse pa Norsk Hydros VCM-fabrikk, Rafnes*. Rapportnr. OR 64/90. Norsk Institutt for Luftforskning (NILU), Lillestrom, Norway. NILU Prosjekt Nr. 0-90057. September.
(Cited as: Vurdering av diffuse utslipp fra VCM fabrikken pa Hydro Rafnes.)

- Norsk Hydro. 1993. *Egenrapportering av Utslippstall fra Bedrifter med Utslippstillatelse. Rapporteringsskjema: Rapportering for 1992*. Statens forurensningstilsyn (SFT), Oslo, Norway. 11 pp. February 1.

- Norsk Hydro. 1992. *PVC and The Environment*. Norsk Hydro a.s., Petrochemical Division, Oslo, Norway. September.

TABLE OF CONTENTS (CONT'D)

- Oehme, M., S. Mano, and B. Bjerke. 1989. Formation of polychlorinated dibenzofurans and dibenzo-p-dioxins by production processes for magnesium and refined nickel. *Chemosphere.* 18(7-8):1379-1389.
- Oehme, M., S. Mano, E.M. Brevik, and J. Knutzen. 1989. Determination of polychlorinated dibenzofuran (PCDF) and dibenzo-p-dioxin (PCDD) levels and isomer patterns in fish, crustacea, mussel and sediment samples from a fjord region polluted by Mg-production. *Fresenius Z. Anal. Chem.* 335:987-997.
- Peterson, R.E., T.A. Mably, R.W. Moore, and R.W. Goy. 1992. In-utero and lactational exposure of male rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin: Effects on sexual behavior and the regulation of luteinizing hormone secretion in adulthood. *Chemosphere.* 25(1-2):157-160.
- Wang-Anderson, G. 1993. En vurdering av undersokelsen: Sedimentundersokningar langs Bohuskysten 1990 - Goteborg och Bohus Lans kustvattenkontroll. (Sedimentological investigations of the Bohus Coast 1990 - The Coastal Water Monitoring Program of the Goteberg and Bohus County.) Ingemar Cato. Sveriges Geologiska Undersokning. Rapporter och meddelanden nr 74, 1992. Mot kjent kunnskap om dannelse av miljoskadelige komponenter under produksjon av PVC til Hydro Plast i Stenungsund. Greenpeace Report.
- WHO. 1991. *Summary Report: Consultation on Tolerable Daily Intake from Food of PCDDs and PCDFs*. World Health Organization, Regional Office for Europe. EUR/ICP/PCS 030 (S). EUR/HFA Target 19; 036n. Bilthoven, Netherlands, December 4-7, 1990.

The following citations in the report were not obtained by ChemRisk®:

- Greenpeace. 1992 Norsk Hydro, PVC och Dioxiner. Fact Sheet.
- Nationen. 1993. Newspaper article. March 4.
- Niklasson, R. 1992. Samanstallning av utslapp til luft og vatn under 1990 fra Hydro Plast AB.
- Norwegian Parliament. 1993. Question and Answer given to the Minister of Environment. March 11.
- Verhoog, W., J.W. Duffer, Stichting Reinwater. 1990. De rijn - Dioxinen en andere Microverontreiningnen in industriel Afvalwaterfozingen.
- Wahlqyist, L. 1993. Dioxinmafningar VCM-fabriken. Letter describing the test program. February 25.

ChemRisk® - A Division of McLaren/Hart
Vinyl Institute
September 1, 1993

EXECUTIVE SUMMARY

A critical analysis is presented of the Greenpeace report entitled *Dioxin Factories: A Study of the Creation and Discharge of Dioxins and Other Organochlorines from the Production of PVC*, a publication which targets the polyvinyl chloride (PVC) manufacturing industry as a major source of environmental levels of organochlorine compounds. The Greenpeace assessment contains numerous factual errors and unsupported allegations concerning the following key subject areas:

- EDC/VCM/PVC Production Processes
- Dioxins and Organochlorines as Byproducts of EDC/VCM/PVC Production
- Sources of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans (PCDD/PCDFs) to the Environment
- Toxicity Issues
- Human Exposure Issues

In Greenpeace's cursory consideration of the EDC/VCM/PVC production processes, they concluded that the industry has become "the sink for surplus chlorine," while the widespread uses and benefits of PVC are wholly overlooked. Greenpeace's assumption that production processes at all EDC/VCM/PVC facilities are comparable is the basis for their generalization from a single facility to all facilities worldwide, without regard for differences in regulations, facility designs, processes, or operating practices.

Greenpeace also presents numerous unfounded conclusions pertaining to discharge rates of dioxins and other organochlorines, each of which is critically evaluated and determined to be without scientific basis. Additionally, in Greenpeace's effort to lay blame on the PVC industry, other sources of PCDD/PCDF to the environment are completely overlooked. The inherent bias of the report is also evident through several misleading statements and misinterpretations of the cited literature. Five examples of clear misinterpretations of the literature are presented.

Furthermore, the scientific basis for Greenpeace's assumptions regarding the toxicity of dioxin and risks associated with potential exposures to dioxin is extremely limited and tenuous. The sources

referenced by Greenpeace as confirming dioxin's carcinogenicity in humans do not support Greenpeace's conclusions. Similarly, Greenpeace's assertions that dibenzofurans affect the growth and mental development of children exposed *in utero* and that dioxin causes noncancer effects other than chloracne in humans were also examined and found to be equivocal. Other erroneous conclusions refuted in this analysis include: results of an incorrect interpretation of dose response, misinterpretation of carcinogenic versus noncarcinogenic effects, and the inappropriate estimation of the dioxin dose received by the general population.

In summary, many of Greenpeace's conclusions pertaining to EDC/VCM/PVC production, dioxins and organochlorines as byproducts of EDC/VCM/PVC production, source contribution, toxicity, and exposure are unsupported by the scientific literature. Interestingly, many of the sources referenced by Greenpeace do not support their assertions. An analysis is presented which examines the scientific basis for such conclusions and offers scientific interpretations of the available data and literature.

1.0 INTRODUCTION

ChemRisk®, a Division of McLaren/Hart Environmental Engineering was retained by the Vinyl Institute to critically evaluate the Greenpeace report entitled *Dioxin Factories: A study of the creation and discharge of dioxins and other organochlorines from the production of PVC*. The nineteen-page Greenpeace publication targets the polyvinyl chloride (PVC) manufacturing industry as a major source of environmental levels of organochlorine compounds including dioxins and furans. In its report, Greenpeace raised a number of claims concerning the PVC production industry, based on the assumption that the measured dioxin emissions from one facility were characteristic of industry-wide dioxin emissions on a global scale. Greenpeace also assessed the industry's contribution to environmental contamination (such as river sediments), toxicity of chemical byproducts of PVC production, and human exposures to byproducts, such as dioxins.

Overall, the Greenpeace assessment of dioxin and organochlorines in PVC production contains factual errors and unsupported conclusions. A critical flaw in the Greenpeace report is that it discusses toxicity and distributions of dioxins and furans in certain media but does not identify differences among the congeners or isomers of concern. For example, Greenpeace cited Evers et al. (1989) in its allegation that $CuCl_2$, a catalyst used in the oxychlorination process, contained dioxins at the ng/kg (ppt) level. However, Greenpeace failed to mention that Evers et al. (1989) reported that $CuCl_2$ contained only octa- and heptachlorinated dioxins and furans, two of the least toxic congener groups (Heindl and Hutzinger, 1986). Other examples of factual errors and inaccurate reporting of study results are discussed in this report.

In this report, the results of ChemRisk's critical evaluation and review of Greenpeace's allegations and claims are presented. Comments were organized into the following categories which encompass the majority of Greenpeace's allegations:

- PVC Production Process Issue
- Dioxins and Organochlorines as Byproducts of PVC Production
- Source Contribution of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans (PCDD/PCDFs)
- Toxicological Issues
- Human Exposure Issues

Within each category, selected specific or general allegations made by Greenpeace in *Dioxin Factories* are presented in italics, and are followed by ChemRisk's comments. Direct citations from *Dioxin Factories* are presented in quotation marks and are also italicized, followed by ChemRisk's comments. Our comments are provided in Sections 2.0 through 6.0 of this report, while sections 7.0 and 8.0 present our conclusions and references, respectively.

2.0 PVC PRODUCTION PROCESS

In general, Greenpeace has overlooked the widespread uses and benefits of PVC, which is heavily used in electronics, appliances, consumer goods, packaging, and medical products. The construction and automotive industries also rely on the versatility of PVC. Being exceptionally versatile and durable, PVC is the only plastic that can be modified to suit a variety of applications.

PVC is the second largest volume plastic produced in the world. In 1992, the U.S. alone produced over 4.5 million tonnes (Vinyl Institute, 1993) and worldwide consumption is estimated at 18 million tonnes annually (Norsk Hydro, 1992). Because the production of PVC is based on natural resources, such as sodium chloride, it is an economical plastic to produce. Furthermore, it is one of the most efficient construction materials available when analyzed on an energy-equivalent basis (Cowfer and Migistro, 1985).

The manufacture of PVC is essentially a closed production process (Vinyl Institute, 1993). Because of this closed system, production efficiencies are maximized, while environmental emissions and potential worker exposures are minimized. As individual plants improve environmental controls, solid waste generation and air and water emissions will continue to decrease.

Greenpeace : PVC industry has become "the sink for surplus chlorine"

According to the Greenpeace report, the PVC industry has become "the sink for surplus chlorine". Greenpeace neither quantified "surplus chlorine" nor provided a reference for the accompanying discussion that led to this conclusion. In fact, the entire chemical process industry consumes chlorine in order to create beneficial materials such as organic and inorganic chlorine compounds, refrigerants, pulp and paper, and fabrics (Windholtz, 1983; Parmeggiani, 1983; Charles River Associates Inc., 1993). These materials, in turn, are used as the starting materials for products that are used worldwide on a daily basis.

Greenpeace: There is a level of comparability among all PVC facility emissions

Throughout the Greenpeace report, site-specific data provided by Norsk Hydro (Norsk Hydro, 1992) were heavily relied upon and considered representative of ethylene dichloride and vinyl chloride monomer (EDC/VCM) and PVC facilities worldwide. However, Norsk Hydro explicitly stated that their data should not be assumed to be valid for all EDC/VCM/PVC production plants because regulatory and technology standards worldwide vary widely (Norsk Hydro, 1992). It should also be noted that the production of EDC/VCM uses different processes than PVC production and the terms should not be interchanged.

For example, in the U.S., all air and water emissions resulting from the PVC production process are regulated by the USEPA (Vinyl Institute, 1993). Additionally, all U.S. manufacturers of PVC and/or VCM must report their compliance with USEPA and State environmental agency standards. Prior to issuing permits in the United States, numerous site-specific factors are considered by the environmental agencies. The USEPA conducts assessments of risk and technology in order to determine the effects of different concentrations of pollutants and the appropriate technology to be applied to an effluent stream. Also taken into account is the site-specific technology being used and whether the Best Available Control Technology (BACT) has been adopted by the facility. Thus, the USEPA takes these factors into consideration in issuing a permit and allowable emission rates may vary among facilities.

Regulatory requirements also influence the types and efficiencies of air pollution control devices installed at EDC/VCM production facilities. Differences in facility designs, processes, and operating practices also contribute to the difficulty of extrapolating from one facility to another. Despite these factors, Greenpeace assumed that dioxin emissions data, which were specific to a Norsk Hydro EDC/VCM plant in Norway, were representative of levels found at other EDC/VCM facilities. Because data do not exist to support this claim, such an extrapolation is inappropriate.

Because this fundamental assumption has not been validated, the resulting conclusions made by Greenpeace, based on this assumption are unfounded. Greenpeace contended that chlorinated byproducts, formed during EDC/VCM production, are divided into heavy and light ends, or high molecular weight and low molecular weight compounds, respectively, which are created in

approximately the same amounts. Greenpeace supported this by citing the percentage amounts of heavy and light ends resulting from a single German PVC production process. However, the actual production of PVC from EDC/VCM does not generate heavy and light ends. These residuals are generated in the production of EDC/VCM only. It is impossible to ascertain whether Greenpeace was aware of this distinction between EDC/VCM and PVC production because a description of the German process was not provided. Details, such as production rate, starting materials, reactor type, and reaction conditions are necessary to identify potential byproducts and, subsequently, to determine how the byproduct components will behave under separation. Because the amount and proportions of resultant byproduct components depend upon individual facility operating conditions, it appears that Greenpeace did not account for process variabilities when stating that heavy and light ends are produced in almost equal proportions.

Greenpeace: Heavy and light ends are waste streams

Greenpeace referred to heavy and light ends as two types of waste streams. This is misleading because byproducts are often used as marketable and recyclable commodities. For example, heavy ends such as 1,1,2-trichloroethane and 1,1,2,2-tetrachloroethane are useful as feedstock for thermal chlorination processes, or to produce high-quality muriatic acid (Norsk Hydro, 1992; McNaughton, 1983). Light ends, including ethyl chloride and *cis*-dichloroethylene, are also recoverable and sold as raw materials (Norsk Hydro, 1992; McNaughton, 1983). It is incorrect and misleading, therefore, to classify these materials as wastes.

3.0 DIOXIN AND ORGANOCHLORINES AS BY-PRODUCTS OF PRODUCTION

Greenpeace: *"There is also reason to believe that other organochlorines are discharged in at least the same percentage as for dioxin type compounds."*

No supporting documentation was provided to substantiate this claim. It is unclear whether Greenpeace is asserting that each organochlorine is discharged in an equal and fixed proportion relative to dioxin or that total organochlorine discharges represent a proportion of the waste stream equal to that of dioxin. In any case, given the variety of facility designs, feedstocks, operating procedures, and physicochemical behaviors of organochlorines, it is highly unlikely that discharges would be as uniform as Greenpeace states. In fact, Table 1 of the Greenpeace report, which lists the proportions of several chlorinated hydrocarbons produced during VCM manufacture, rebuts this claim. In this table it is shown that other organochlorines discharged range from <0.0001% to 0.8%, while the discharge of dioxin-type compounds from this facility was not reported.

Greenpeace: *"...if dioxins follow the EDC streams, and the percentage levels of dioxin to EDC in the gas streams is the same as those found in the discharge to water, the level of dioxin discharged to air would be: 74.52 grams TCDD-equivalents/year for this factory."*

The incorrect assumption inherent in this calculation is that dioxin partitions equally between gas and liquid phases. The physical properties of dioxin have been described by various investigators and are readily available in the literature (Bopp et al., 1991; Norwood et al., 1989; Schroy et al., 1985). The use of these data would allow a better and more accurate calculation to be provided.

Greenpeace assumed that laboratory experimentation replicates industrial manufacture of EDC/VCM

In Case Study B, Greenpeace cited a laboratory analysis of the oxychlorination of ethylene and concluded, from a comparison of the PCDD and PCDF congeners from the experiment, that the oxychlorination process is responsible for the PCDD and PCDF present in sediment at the investigated site. This allegation is based on the assumption that the laboratory was able to replicate actual conditions. To directly extrapolate from laboratory data to the actual byproducts created during manufacturing is an oversimplification which does not take into consideration the differences in impurity production due to varying reactor volumes.

Greenpeace: [production results in] "...5 - 10 grams of dioxin TCDD equivalents per 100,000 tons.."

Many of the studies cited by Greenpeace do not support the contention that EDC/VCM manufacturing produces TCDD equivalents at the rate of 5 to 10 grams per 100,000 tonnes. In fact, Norsk Hydro (1992) data indicate that emissions of TCDD equivalents is lower by two orders of magnitude.

Greenpeace: Components can be quantified by the AOX method

Greenpeace is incorrect in asserting that the molecular weight of constituents can be determined by an AOX analysis. The compounds present cannot be identified by this methodology and most AOX analyses do not adequately separate inorganic from organic fractions. Greenpeace has attempted to use total AOX results to quantify the components. This is conjecture, not science, and the ratio of inorganic constituents to organic constituents cannot be determined with the results presented. Furthermore, in contrast to Greenpeace's statements that Norsk Hydro "tried to discredit the use of AOX as a measurement of the discharge, claiming that inorganic chlorine was interfering in the process" and that "the Center for Industrial Research stated that the AOX measurement is adequate to give an idea of the size of unidentified organochlorines in the

wastewater", the Center for Industrial Research (Källquist, 1991) concluded that the analysis of organic material in the outlet water was uncertain, due to interference from inorganic salts.

Greenpeace: Bench-scale prototypes are capable of replicating VCM production

Research by Evers (1989) is an attempt to use a bench-scale prototype to emulate manufacturing facilities. Although this research is valuable from the perspective of optimizing conditions for VCM production, it is always questionable as to how the trace impurities production will compare when the process is scaled up for manufacturing. For example: Is the ratio of dioxin to EDC constant for increasing production of EDC? No graphs are offered in the report that would support this contention. In fact, the majority of impurities may be produced at the start-up of a process; as the process continues, the ratio of impurities to product will likely continually decline. These types of confounding factors are not addressed nor are they accounted for by the simple ratio that Greenpeace applied to arrive at mass of TCDD equivalents for 100,000 tons of EDC.

Greenpeace: "...discharges to the environment were less than 0.3 g TCDD equivalents."

The use of uncited material to support this claim, as in Case Study C does not allow the data to be reviewed or its subsequent interpretation possible. It should be noted, however, that the production of a compound in one of a series of processes does not imply that there will be a release to the environment. Rather, there is potential for chemical alteration or destruction during subsequent reaction steps. Without the report, this subject cannot be adequately addressed.

4.0 POTENTIAL SOURCES OF PCDD/PCDF IN THE ENVIRONMENT

Greenpeace : Greenpeace disregarded multiple sources of PCDD/PCDF in the environment

The Greenpeace report has assumed that dioxins present in sediments of Sweden and the Netherlands were entirely attributable to EDC/VCM production. Other known or potential sources of contamination were not considered in spite of the fact that the areas of most concern to Greenpeace, i.e. along the Rhine River in Germany and the Netherlands, and the Bohus Coast in Norway and Sweden are heavily industrialized. The southeastern coast of Norway, in the vicinity of Norsk Hydro's chlorine/EDC/VCM facility in Rafnes, houses several metals processing and pulp and paper manufacturing facilities (P. Balerin, personal communication, 1993). The western coast of Sweden, in the vicinity of another Norsk Hydro EDC/VCM/PVC facility in Stenungsund, is well known for its automobile production facilities, petrochemical and pulp and paper plants, shipyards, and smelters which process specialty steels (G. Couey, personal communication, 1993). In Germany and the Netherlands, the proximity of the river Rhine encouraged many chemical companies to establish operations in the region. These companies were attracted to the Rhine's water supply and transport facilities. Production facilities along the Rhine include manufacturers of organic and inorganic industrial chemicals, organic intermediates, fertilizers, pigments, plastics, and synthetic rubbers (Chemische Industrie, 1991).

Several studies indicate that PCDDs and PCDFs are widely distributed in industrialized and heavily populated environments (Hutzinger and Blumich, 1985; Sheffield, 1985; Czuczwa and Hites, 1986; Rappe et al. 1987a; 1987b; Rappe and Kjeller, 1987; 1988; Southerland et al., 1987; Tiernan et al., 1989; Smith et al., 1990). A growing number of processes which can potentially lead to TCDD and PCDF emissions to the environment have been identified. These include large and small combustion engines, chemical manufacturing, production of pulp and paper, the chlorination of sewage, smelting, incineration, heating systems and forest fires. Although the variety of sources of PCDD and PCDF in the environment have been well established in the scientific literature (Langhorst and Shadoff, 1980; Czuczwa et al., 1985; Sheffield, 1985; Ballschmiter et al., 1986; Hagenmaier et al., 1986; Jones and Konheim, 1986; Konheim 1986; Patterson et al., 1986; Stanley et al., 1986; Clement et al., 1985, 1987; Marklund et al., 1987; Rappe et al., 1987c,

1989; Thoma, 1988; Fries and Paustenbach, 1990), Greenpeace failed to acknowledge multiple contributors.

Research by Ballschmiter et al. (1986) demonstrated a striking similarity in the distribution patterns of PCDD and PCDF congeners in used automobile oil with environmental patterns found in urban air particulates, human tissues, and municipal solid waste (MSW) incinerator emissions. These authors concluded that environmental patterns of PCDDs could be correlated equally with motor vehicle emissions and MSW incinerator emissions. Ballschmiter et al. (1986) further concluded that the non-point source character of motor vehicles was a major source of PCDD and PCDF in the environment. Jones and Konheim (1986) and Konheim (1986) calculated that automobiles were the primary source of chlorinated PCDDs and PCDFs prior to 1975. Furthermore, in 1987, Marklund et al. showed that total emissions from Swedish automobiles, even if well maintained, may contain as much PCDD and PCDF as 2 to 20 municipal waste incinerators.

PCDDs and PCDFs have also been detected in chimney soot and bottom ash from oil, wood, and coal burning furnaces at levels that are almost as high as levels reported in MSW incineration emissions (Thoma, 1988). PCDDs and PCDFs have been found in chimney soot from oil-fired central heating systems and coal ovens, and in the bottom ash of wood burning stoves from private homes in West Germany (Thoma, 1988). These data indicate that at least three types of commonly used heating systems contribute to the presence of PCDDs and PCDFs in ambient air and, consequently, to their deposition in the environment.

The generation of PCDDs and PCDFs by municipal solid waste (MSW) incinerators has been well established in the scientific literature (USEPA, 1984; Hutzinger et al., 1985a; Rappe et al., 1987a; Rappe and Kjeller, 1987b; Southerland et al., 1987; Buser et al., 1978). Dioxins and furans have been detected as contaminants of the fly ash from municipal waste and smaller hospital waste incinerators (Dickson et al., 1989; Hutzinger et al., 1985a; Tong et al., 1989). These incinerators generally burn household wastes as well as some industrial refuse, but no chemical waste as a rule (Buser et al., 1978). The median rate of production and the composition of emissions from 24 MSW facilities located worldwide has been estimated by Jones et al. (1987). This study, and numerous similar studies, indicate that MSW facilities can generate part per billion levels of

PCDDs and PCDFs in the flue gas and part per million levels in the fly ash, depending on the technology employed at a given facility (Tong et al., 1989)

Greenpeace: *The Greenpeace report contained misleading statements and misinterpretations of the literature*

In their report, Greenpeace made misleading inferences and misinterpreted the scientific literature. A clear example is Case Study A in which Greenpeace cited a study conducted by Evers et al. (1988), who determined PCDF isomer distributions from sediments in the Rhine at river km 660. Although Greenpeace contributed the presence of PCDF to VCM production, the VCM facility to which Evers et al. referred was nine river kilometers downstream of river km 660. However, it is generally expected (except in the case of tidal mixing) that the source of chemical contamination occurs upstream from a "hot spot." In addition, Greenpeace alleged that the location of Akzo's Rotterdam VCM plant is at river km 669 and that this plant contributed to PCDF levels in sediment. This contradicts Evers et al. (1988), who stated that Rotterdam is at river km 1000, over 330 river kilometers downstream from river km 669, which further disputes the possibility that the Rotterdam VCM facility is responsible for those levels. Finally, although a figure provided by Evers et al. (1988) displays the heavily industrialized area of the river Rhine from the 159 river km mark to the 1000 km river mark, Greenpeace does not acknowledge the density of the industrialization.

In addition, Greenpeace incorrectly reported the scientific literature in the following statement:

> "The researchers (Evers et al., 1988) looked at the available scientific evidence, and found that the compounds found in the sediments were also produced during VCM production (Greenpeace reference 17 (Erickson et al., 1988)) and during synthesis of short chain hydrocarbons (Greenpeace reference 18 (Heindl and Hutzinger, 1987))."

The "compounds" related to VCM production wastes reported by Erickson et al. (1988) were not PCDDs or PCDFs, as implied by Greenpeace. The conclusions of Heindl and Hutzinger (1987) were also taken out of context. These authors determined that PCDDs and PCDFs were formed

from chlorinated aliphatics only under severe alkaline conditions, as opposed to conditions conducive to VCM production. The above statement is further misleading because since Evers et al. (1988) was published, new information has been made available on the sources of PCDDs and PCDFs, as discussed earlier in this section. In addition, methods for fingerprinting PCDD and PCDF isomers have been refined to more accurately identify contributors (Wenning et al., 1992). Greenpeace failed to address these recent studies.

The translation of several referenced sources into English revealed several cases in which Greenpeace took the conclusions of researchers out of context or otherwise misinterpreted their work. Copies of original and translated documents are provided in Appendix A. In the Executive summary, Greenpeace presented estimates of risks associated with dioxin emissions and then concluded that, "revelations of these findings in Norway and Sweden have forced both Environmental Protection Agencies (EPA) and Norsk Hydro to conduct further studies," citing Hagen (1993). While Hagen (1993) did specify that Greenpeace's request for further study had been granted, he explicitly stated that this did not mean that EPA doubted the results of the previous study, but rather, wished to confirm previous findings. In his letter, Hagen (1993) indicated that EPA considered the previous study program satisfactory. Perhaps more misleading is that Greenpeace implied that further studies on potential health risks were to be undertaken, when in fact, the requested studies related to the chemical characterization of media. In the presentation of Case Study I, Greenpeace again cited Hagen (1993) in the assertion that EDC/VCM production gives an isomer pattern dominated by the hepta-, and octachlorodibenzo-furans. In studying Hagen's (1993) letter to Greenpeace, no such statement could be found. In fact, the letter includes no discussion whatsoever of isomer patterns.

A final example of Greenpeace's misrepresentation of referenced sources was provided by their statement that Norsk Hydro admits that dioxins are found in the VCM used for PVC production. In fact, the referenced source (Behrens, 1990) stated that the likelihood of producing dioxin while burning VCM is small and that the formation of dioxins through this route is considered unimportant in the overall mass balance. Behrens, 1990, also provided supporting data: groundwater samples uniformly indicated that dioxin levels in the vicinity of the plant were below background.

5.0 TOXICOLOGICAL ISSUES

Greenpeace: TCDD is a Human Carcinogen

Greenpeace cited Manz et al. (1991), Fingerhut et al. (1991a,b), and Jenkins (1991) as the sources of their allegations that "recent research confirms carcinogenicity in humans." A review of these studies indicates that they do not support this conclusion. The Jenkins (1991) report is simply a compilation of scientific data and anecdotal information, some of which is dioxin-related and some that is not. A second evaluation of causation is absent from the Jenkins affidavit. Manz et al. (1991) and Fingerhut et al. (1991a,b) report only simple associations between exposure (defined as length of employment) and increased mortality. Manz et al. (1991) measured adipose TCDD levels in a small group of workers for the purpose of confirming exposure categories. However, those individuals sampled were not members of the study cohort and adipose TCDD levels were not directly used to evaluate mortality. Fingerhut et al. (1991a,b) correlated serum TCDD levels with length of employment, and separately evaluated the relationship between length of employment and increased mortality. Although Fingerhut et al. (1991a,b) found a correlation between length of employment and serum TCDD levels in chemical workers, this correlation is a simple association and does not suggest a causal association. Because these chemical workers were exposed to many different persistent chemicals, it is likely that such a simple correlation would exist for any number of chemicals. Neither group of researchers evaluated causation. Moreover, neither group directly correlated 'dioxin exposure' with health effects.

Although there is sufficient evidence to suggest that 2,3,7,8-TCDD, and possibly other dioxin and furan isomers, are carcinogenic in laboratory animals (Kociba et al., 1978; NTP, 1982; USEPA, 1985), there is no conclusive evidence that TCDD is a human carcinogen (Zober et al., 1990; Fingerhut et al., 1991a,b; Kimbrough, 1991; Manz et al., 1991; Saracci et al., 1991; Tollefson et al., 1991). In fact, the evidence obtained from dozens of epidemiologic studies of herbicide sprayers, chemical workers, American servicemen exposed to Agent Orange in Vietnam, and the residents of Seveso, Italy (who received the highest doses of any population studied), indicates that TCDD is unlikely to be carcinogenic in humans at the very low doses currently associated with environmental exposures.

A scientifically based answer to the question, "Is TCDD a human carcinogen?", can only be answered by examination of the evidence of causation. When a statistically significant relationship is found between a chemical exposure and an adverse health effect, a separate analysis must be conducted to determine whether the observed association is indeed a causal association or just a simple association (Susser, 1986). The evaluation of causation requires the consideration of at least nine criteria (Hill, 1965; Susser, 1973, 1976; Ibrahim, 1985; Rothman, 1986). These criteria, often referred to as the Hill Criteria, are: (1) strength , (2) consistency of the association, (3) specificity of the association, (4) temporal correctness of the association, (5) biologic gradient, (6) plausibility, (7) coherence, (8) experimental evidence, and (9) analogy. A causal association can only be inferred by an evaluation of each criteria and a final determination should be based on the total weight of evidence (Hill, 1965; Rothman, 1986). With respect to dioxin and human cancer, such an evaluation of causation has not been conducted.

Although not all of the most recent epidemiologic studies were available at the time of the reviews of Kimbrough (1991) and Tollefson (1991), they concluded that the evidence for a causal association of TCDD with human cancer is equivocal. The majority of the available epidemiologic studies on the association of cancer with TCDD exposure provide little evidence that TCDD is a potent carcinogen in humans (Tollefson, 1991). While the converse (that TCDD is not a human carcinogen) cannot be ruled out on the basis of currently available data (Tollefson, 1991), it is unlikely that TCDD is a human carcinogen at low doses (Bond et al., 1989). The results of more recent studies (Zober et al., 1990; Fingerhut et al., 1991a,b; Manz et al., 1991; Saracci et al., 1991) are not inconsistent with this conclusion.

In addition to understanding the nature of the observed associations, it is critical that the pertinent epidemiologic studies be interpreted within the constraints of the study design, particularly with respect to exposure definition. In every so-called "dioxin" epidemiologic investigation published in the scientific literature, exposure has been characterized not as dioxin exposure, but rather by some surrogate of chemical exposure, most often the length of employment. In each of these studies, the exposed populations have experienced multiple chemical exposures. While some of these chemicals had been contaminated with TCDD (i.e., 2,4,5-T, trichlorophenol (TCP)), most were probably not. Moreover, for those chemicals that were contaminated with TCDD, levels were only about one part per million (ppm) (USEPA, 1980). Thus, workers handling 2,4,5-T and

TCP were exposed to the raw chemicals at levels approximately one million fold greater than they were exposed to TCDD. Interestingly, in the largest study of chemical workers and herbicide sprayers ever conducted, Saracci et al. (1991) found the highest mortality rates associated with soft tissue sarcoma occurred in those who had been exposed to the types of phenoxy herbicides not contaminated with TCDD.

In summary, the "dioxin" epidemiologic studies conducted to date have not evaluated causation. The poor characterization of "dioxin" exposure in the exposed subjects precludes any definitive conclusions regarding the simple associations reported by Manz et al. (1991) and Fingerhut et al. (1991a,b). Finally, because these studies have not evaluated other chemical exposures, the "dioxin" effects cannot be distinguished from the potential effects associated with other chemicals.

Greenpeace: Furans Affect The Growth and Mental Development of Children Exposed In Utero

According to Greenpeace, "The furans affect the growth and mental development of children born to exposed women." This allegation is not referenced, but it is likely that Greenpeace refers to the Yusho and Yu-Cheng incidents which occurred in Japan in 1968 and in Taiwan in 1979, respectively (Kuratsune, 1989). Both poisoning incidents occurred from the ingestion of PCB-contaminated rice oil. Because the PCB-ladened rice had been heated, other compounds including polychlorinated dibenzofurans (PCDFs) and polychlorinated quarter phenyls (PCQPs) were also present. It has been suggested that the clinical effects observed in the Yusho and Yu-Cheng victims were due to the PCDFs, not the PCBs (Kimbrough, 1987; Kuratsune, 1989). The primary basis for this hypothesis was the decline in PCB tissue levels over time, with little reduction in PCDF levels over time, and the lack of a concurrent change in the observed clinical manifestations (Kuratsune, 1989). More recently, Chen et al. (1992) evaluated cognitive development in Taiwanese children who were exposed *in utero* to these compounds as a result of the Yu-Cheng incident. Cheng et al. (1992) found only a mild deficit in cognitive development and noted that "...the PCBs or PCDFs responsible for the physical findings may not be those responsible for the developmental delay." With this limited information potentially relating PCDFs to physical effects, no definitive conclusions can be drawn regarding the chemical-specific causes of the Yusho and Yu-Cheng health effects. Finally, no evidence has been provided to suggest that the very high

exposures incurred by Yusho and Yu-Cheng subjects are at all relevant to the levels typical of environmental exposures.

Greenpeace: *TCDD Causes Noncancer Effects In Humans Other Than Chloracne*

Greenpeace stated that, "...noncancer toxicity [of TCDD] in humans includes effects on the central nervous system, disruption of metabolism and suppression of immune system". No reference to the scientific literature is given for this allegation. Numerous epidemiologic studies have evaluated noncancer effects in human populations exposed to dioxin-contaminated chemicals (Suskind and Hertzberg, 1984; Moses et al., 1984; Lathrop et al., 1984, 1987; Sweeney et al., 1990; Wolfe et al., 1991). The populations studied by these researchers are generally the same populations that have been evaluated for cancer mortality. Reported noncancer effects include chloracne, porphyria, hepatomegaly, changes in liver enzyme levels and lipid metabolism, diabetes, and cardiovascular disease. However, there has not been one condition or series of long-term health effects that has been consistently demonstrated among every exposed population (USEPA, 1992). Furthermore, these studies all suffer from the same shortcomings as the cancer epidemiologic studies; that is, it is not possible to separate out the effects of TCDD from the effects caused by the other chemical exposures.

Greenpeace: *The Toxicity of Chemicals Used In Production Are Relevant to Environmental Exposures*

Greenpeace contends that 29 different chemicals are created by Norsk Hydro's Stenungsund facility (Table 1 of the Greenpeace report). It should be pointed out that the simple production of intermediate compounds does not imply their release to the environment from a closed production system. If there is no release, then there is no exposure.

6.0 EXPOSURE ISSUES

Greenpeace: *"The World Health Organization has established a tolerable daily intake (TDI) for 2,3,7,8-TCDD of 10 pg/kg body weight/day, which has subsequently been adopted by many countries. However, it is fundamentally flawed in that it is based on the assumptions that dioxins do not cause cancer."*

This statement is incorrect. The World Health Organization (WHO) Tolerable Daily Intake (TDI) value of 10 pg/kg-day is based on general toxicological effects, including carcinogenicity, reproductive effects, and immunotoxicity in various laboratory animals (WHO, 1990). WHO analyzed tissue levels and health effects data from animal studies and, from these data, identified a No-Observed-Adverse-Effect-Level (NOAEL) of 1000 pg/kg-day for 2,3,7,8-TCDD. Based on this NOAEL, WHO calculated a TDI by applying a safety factor of 10 to extrapolate from animal to humans and an uncertainty factor of 10 to account for uncertainty in reproductive effects (WHO, 1990). Based on its analysis, WHO (1990) concluded that TCDD is carcinogenic in animals, but the evidence regarding the carcinogenicity of TCDD in humans is inconclusive. Similarly, although the WHO considered reproductive and immunotoxic effects in developing the TDI, the only noncarcinogenic effect which has been causally associated with TCDD is chloracne.

The TDI of 10 pg/kg-day is consistent with the allowable daily intakes (ADIs) developed by Canada, the Netherlands, West Germany, and the United Kingdom (Ontario, 1985; van der Heijden et al., 1982; NCASI, 1987; U.K., 1989; Tollefson, 1991). These countries have historically used a safety factor approach to estimate ADIs for TCDD based on the NOAEL of 1,000 pg/kg-day, reported in the Kociba et al. (1978) cancer bioassay, and a safety factor of 100. The Kociba et al. (1978) study has been used by nearly all regulatory agencies for setting standards for TCDD.

Based on the evidence that TCDD is nongenotoxic and that TCDD mediated carcinogenesis is thought to proceed through a receptor-mediated event, it is appropriate to suggest that there is an exposure level at which no carcinogenic response will occur. Therefore, use of a threshold model like that used by WHO to develop a dose for TCDD that is protective of general toxicological health

effects of TCDD, including cancer, is consistent with the scientific evidence regarding the carcinogenic potential of this compound.

Greenpeace: A dose of 0.006 pg/kg-day 2,3,7,8-TCDD is sufficient to cause 1 excess cancer per million population in the U.S. (Introduction)

This statement is grossly misleading and requires further qualification. Greenpeace has interpreted estimated hypothetical risks to represent actual risks of death caused by cancer due to 2,3,7,8-TCDD exposure. Risk estimates generated from exposure and dose-response models are not intended by regulatory agencies to represent actual risk probabilities for individuals in the population. Cancer risk estimates calculated using USEPA methodology are considered upper-bound estimates and are associated with a high degree of conservatism. As USEPA states, actual risks associated with cancer risk estimates may be as low as zero (USEPA, 1986).

The assumptions underlying the U.S. cancer risk estimates are associated with significant uncertainty. These include the use of a nonthreshold, linearized multistage dose-response model, the 95 percent upper bound slope value, and extrapolation of high-dose animal bioassay data to low doses in humans. Because conclusive evidence regarding the carcinogenicity of TCDD in humans is lacking, estimates of the carcinogenic potential must be calculated from extrapolation of data from animal bioassays. In addition to the physiological differences among species, there may be differences in sensitivity and mechanism of toxic action for chemical agents. Doses administered to experimental animals are relatively large in comparison with environmental levels to which humans may be exposed. Because of the high degree of uncertainty associated with animal to human dose response modeling, U.S. regulatory agencies have built a high degree of conservatism into their dose-response methodology.

There is general consensus within the scientific community that the linearized multistage model does not accurately describe the mechanism by which TCDD causes toxicity (USEPA, 1992). Consequently, because of this shortcoming and new epidemiologic and mechanistic evidence, the USEPA is currently reevaluating the health assessment of TCDD toxicity (USEPA, 1991). The anticipated outcomes of the USEPA reevaluation include the development of a biologically based

dose-response model for TCDD and development of a toxicity value that is more reflective of the potential for TCDD to induce a carcinogenic response in humans (USEPA, 1992).

Collectively, use of these conservative assumptions in estimating risks is likely to overestimate actual risks perhaps by orders of magnitude. As such, Greenpeace's interpretation of risk estimates is flawed and the allegation is unjustifiable.

Greenpeace: The WHO TDI of 10 pg/kg-day and the risk-specific dose of 0.006 pg/kg-day (which is based on 10-6 risk level and the EPA cancer slope factor of 156,000 mg/kg-day^{-1} for 2,3,7,8-TCDD) do not address the "...most obvious noncancer effects that are thought to occur at levels that are 1/100th of those at which cancer occurs; neither addresses the impacts to the unborn, through exposure via transplacental transfer, or the newborn, through exposure via breast milk..."

The Greenpeace allegation is misleading and unsubstantiated. Two lines of evidence contradict the Greenpeace assertions. First, the only noncancer effect that has been causally associated with TCDD exposure in humans is the skin disease or condition known as chloracne (Kimbrough, 1990; Tollefson, 1991). No other causal association between TCDD exposure and noncancer effects in humans has been demonstrated. This is even true of TCDD exposure to the human fetus and newborn. Although there is evidence suggesting that TCDD can cross the placental barrier into the human fetus (Schecter et al., 1990), there is no evidence that historical exposures to TCDD caused these adverse health effects.

The second line of evidence comes from the evaluation of TCDD's noncancer effects in various animal species. These animal studies typically use large doses of TCDD in an attempt to produce adverse effects in the reproductive system, immune system, or developmental effects in fetal or newborn animals (USEPA, 1985). The lowest dose which produces a noncancerous effect is referred to as a LOAEL (lowest observable effect level). The next lower dose, which has no adverse effect, is termed a NOAEL (no adverse effect level). A survey of the literature reporting TCDD animal studies provided the lowest NOAELs for reproductive and immune system effects. A NOAEL for reproductive effects in monkeys is 127 pg/kg-day (Bowman et al., 1989). A NOAEL for immune system effects in the guinea pig is 5700 pg/kg-day (Vos et al., 1973).

Applying a conservative safety factor of 100, which includes a factor of 10 for between-species differences and a factor of 10 to protect sensitive human individuals, results in an estimated safe dose for reproductive and immune system effects in humans of 1.3 to 57 pg TCDD/kg-day, respectively. Even using this conservative 100-fold safety factor, these values are 1,000 to 10,000-fold greater than the USEPA's risk specific dose of 0.006 pg/kg-day. Thus, the statement by Greenpeace that noncarcinogenic effects occur at levels 100 times lower than cancerous effects is not justified. Furthermore, the indication by Greenpeace that the WHO TDI and U.S. risk-specific doses are not adequately protective of human health is scientifically unjustified.

Greenpeace: *Assuming an annual global production of VCM of 18 million tons, the dioxin level created would give 8.1 billion people the annual maximum dose (based on the WHO TDI of 10 pg/kg-day and an average body weight of 60 kg). Assuming the WHO TDI of 10 pg/kg-day and an average body weight of 60 kg, one gram of TCDD would provide the annual dose for 4,500,000 people.*

These statements are grossly misleading and the underlying assumptions in their assertions are flawed. The numerous assumptions on which this allegation is based are unrealistic and unsupported. First, Greenpeace assumed that 100% of the TCDD, expressed in Toxic Equivalency (TEQ) units, that were discharged had contaminated the entire annual global food supply. Thus, it was assumed that the entire estimated mass of TCDD TEQ discharged from VCM production facilities partitions to an undefined global food supply, and none partitions to air, surface water, sediment, or soil. Greenpeace's allegation was also based on the assumption that TCDD does not degrade in the environment. This assumption is highly misleading; indeed, even the most conservative environmental fate and transport models would invalidate this argument. Greenpeace also assumed that the entire global population will be exposed daily to dioxin in food. Inherent in this assumption is that uniform concentrations of TCDD are present in all food types consumed worldwide and that ingested TCDD is 100% bioavailable. In summary, Greenpeace's extrapolations are based on numerous assumptions, each of which are scientifically unsupportable and beyond reason in the context of meaningful human health risk assessment.

Greenpeace: No levels of exposure to dioxin are acceptable

The concept of acceptable risk has been a central issue for regulatory agencies and organizations worldwide concerned with the quality of the environment and the regulation of chemical agents. Due to technological and economic constraints, attaining zero environmental levels of TCDD (i.e., zero risk) is not feasible. In response to such issues, agencies charged with protecting public health and the environment have set guidelines for *de minimis* incremental risk levels which trigger regulatory action. *De minimis* risks are calculated from theoretical models utilizing conservative and protective assumptions regarding toxicity and exposure potential. Such risk values represent upper-bound estimates rather than actual risk probabilities.

Clearly, attainable environmental and health-protective standards or guidelines should be reevaluated when new scientific data become available. The USEPA is currently engaged in an extensive and exhaustive reevaluation of the toxicity of TCDD and related compounds. The goal of this reevaluation is to insure that regulatory standards for dioxin are based on the best and most current scientific understanding of the potential for adverse health effects from TCDD exposure.

Greenpeace's position that no exposure to dioxin is acceptable fails to acknowledge technological feasibility and the existence of natural sources of dioxins and furans in the environment (i.e. forest fires) (Hutzinger and Fiedler, 1989). Due to dioxin's ubiquitous distribution and the fact that it is a product of a number of common processes i.e., automobiles, combustion, etc. it would be impossible to eliminate every last molecule in the environment even though laboratory instruments may become sophisticated enough to measure chemical concentrations at extremely low levels. In addition, Greenpeace fails to recognize the importance of historical and ongoing advances made by the regulated industrial community to reduce emissions.

7.0 CONCLUSIONS

Of all the issues raised in this critique, ChemRisk is most concerned with Greenpeace's misinterpretation of the contents of cited sources, unsupported calculations and statements of risks to human health, and the assumption that byproduct and emissions data are comparable from one production facility to another. Although Greenpeace raised important and interesting questions, a responsible, thoughtful, and scientific approach should be taken by the reader when evaluating the Greenpeace report.

8.0 REFERENCES

Asplund, G., A. Grimvall. 1991. Organohalogens in nature: More widespread than previously assumed. *Environ. Sci. Technol.* 25(8):1346-1350

Asplund, G., A. Grimvall, and C. Pettersson. 1989. Naturally produced adsorbable organic halogens (AOX) in humic substances from soil and water. *Sci. Total Environ.* 81/82:239-248.

Ballschmiter, K., H. Buchert, R. Niemczyk, A. Munder, and M. Swerev. 1986. Automobile exhausts versus municipal-waste incineration as sources of the polychloro-dibenzodioxins (PCDD) and -furans (PCDF) found in the environment. *Chemosphere* 15(7):901-915.

Bopp, R.F., M.L. Gross, H. Tong, M.J. Simpson, S.J. Monson, B.L. Deck, and F.C. Moser. 1991. A major incident of dioxin contamination: sediments of New Jersey esturaries. *Environ. Sci. Technol.* 25: 951-956.

Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross, and D.A. Barsotti. 1989. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 or 25 parts per trillion in the monkey: TCDD kinetics and dose-effect estimate of reproductive toxicity. *Chemosphere* 18(1-6):243-252.

Buser, H.R., H.P. Bosshardt, and C. Rappe. 1978. Identification of polychlorinated dibenzo-p-dioxin isomers found in fly ash. *Chemosphere* 2:165-172.

Charles River Associates. 1993. Assessment of the Economic Benefits of Chlor-Alkali Chemicals to the United States and Canadian Economics. Prepared for Chlorine Institute, Washington, D.C.

Chemsche Industrie. 1993. V. Partner. Chemische Industrie 0 November pS3-5. (Cited in Chemical Search). June 27.

Chen, Y.C.J., Y.L. Guo, C.C. Hsu, and W.J. Rogan. 1992. Cognitive development of Yu-Cheng ('oil disease') children prenatally exposed to heat-degraded PCBs. *JAMA* 268(22):3213-3218.

Clement, R.E., H.M. Tosine, and B. Ali. 1985. Levels of polychlorinated dibenzo-p-dioxins and dibenzofurans in wood burning stoves and fireplaces. *Chemosphere* 14(6/7):815-819.

Clement, R.E., H. M. Tosine, V. Taguchi, C.J. Musial, and J.F. Uthe. 1987. Investigation of American lobster, Homarus Americanus, for the presence of chlorinated dibenzo-p-dioxins and dibenzofurans. *Bull. Environ. Contam. Toxicol.* 39:1069-1075.

Czuczwa, J.M. and R.A. Hites. 1986. Sources and fate of PCDD and PCDF. *Chemosphere* 15(9-12):1417-1420.

Czuczwa, J.M., F. Niessen, and R.A. Hites. 1985. Historical record of polychlorinated dibenzo-p-dioxins and dibenzofurans in Swiss lake sediments. *Chemosphere* 14(9):1175-1179.

Deutsch, D. and T. Goldfarb. 1988. PCDD/PCDF Contamination Following a Plastics Fire in a University Lecture Hall Building. *Chemosphere* 17(12):2423-2431.

Dickson, L.C., D. Lenoir, and O. Hutzinger. 1989. Surface-catalyzed formation of chlorinated dibenzxodioxins and dibenzofurans during incineration. *Chemosphere* 19(1/6):277-282.

Erickson, M.D., J.S. Stanley, J.K. Turman, and J.E. Going. 1988. Determination of byproduct polychlorobiphenyls in commercial products and wastes by high-resolution gas chromatography/electron impact mass spectrometry. *Environ. Sci. Technol.* 22:71-76.

Evers, E.H.G., K.C.M. Ree, and K. Olie. 1988. Spatial variations and correlations in the distribution of PCDDs, PCDFs and related compounds in sediments from the River Rhine - Western Europe. *Chemosphere* 17(12):2271-2288.

Evers, E.H.G., J.W. van Berghem, and K. Olie. 1989. Exploratory data analysis of PCDD and PCDF measurements in sediments from industrialized areas. *Chemosphere* 19(1-6):459-466.

Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland, and A.J. Suruda. 1991a. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *N. Eng. J. Med.* 324(4):212-218.

Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland, and A.J. Suruda. 1991b. Mortality among U.S. workers employed in the production of chemicals contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health. January.

Fries, G.F. and D.J. Paustenbach. 1990. Evaluation of potential transmission of 2,3,7,8-tetrachlorodibenzo-p-dioxin contaminated incinerator emissions to humans via foods. *J. Toxicol. Environ. Health* 29:1-43.

Grimvall, A., S. Jonsson, S. Karlsson, R. Savenhed, and H. Boren. 1991. Organic halogens in unpolluted waters and large bodies of water receiving bleach plant effluents. *TAPPI* 74(5):197-203.

Hagenmaier, H., H. Brunner, R. Haag, and A. Berchtold. 1986. PCDDs and PCDFs in sewage sludge, river and lake sediments from South West Germany. *Chemosphere* 15(9/12):1421-1428.

Heindl, A. and O. Hutzinger. 1986. Search for industrial sources of PCDD/PCDF II. Metal Chlorides. *Chemosphere* 16(8/9):1949-1957.

Heindl, A. and O. Hutzinger. 1987. Search for industrial sources of PCDD/PCDF III. Short-chain chlorinated hydrocarbons. *Chemosphere* 16(8/9):1949-1957.

Hill, A.B. 1965. The environment and disease: Association or causation? *Proc. Roy. Soc. Med.* pp. 7-12.

Hutzinger, O. and M.J. Blumich. 1985. Sources and fate of PCDDs and PCDFs: An overview. *Chemosphere* 14(6/7):581-600.

Hutzinger, O. and H. Fiedler. 1989. Sources and emissions of PCDD/PCDF. *Chemosphere* 18(1-6):23-32.

Hutzinger, O., G.G. Choudhry, B.G. Chittim, and L.E. Johnston. 1985. Formation of polychlorinated dibenzofurans and dioxins during combustion, electrical equipment fires and PCB incineration. *Environ. Health Perspect.* 60:3-9.

Ibrahim, M.A. 1985. Time Trends, Ecologic Correlation, and Modeling. In: *Epidemiology and Health Policy.* An ASPEN Publication. pp. 53-61.

International Labour Oganisation (ILO). 1983. *Encyclopaedia of Occupational Health and Safety*. L. Parmeggiani (ed.) Third edition. Geneva, Switzerland.

Jeckel, M.R., and P.V. Roberts. 1980. Total organic halogen as a parameter for the characterization of reclaimed waters: Measurement, occurrence, formation and removal. *Environ. Sci. Technol.* 14:970.

Jenkins, C. 1991. *Recent Scientific Evidence Developed after 1984 Supporting a Causal Relationship Between Dioxin and Human Health Effects.* Affidavit of Cate Jenkins, Exhibit H, in the United State district court for the Eastern District of New York, Shirley Ivy et al. vs. Diamond Shamrock Cehmicals Company et al. CV989-03361 (E.D.N.Y.XJBW).

Jones, K.H. and C.S. Konheim. 1986. A comparative analysis of leaded gasoline auto exhaust versus MSW incinerators as sources of U.S. population dioxin/furan exposure. Paper presented at annual meeting, Government Refuse Collection and Sisposal Association, Reno, NV. August 11-14.

Jones, K.H., J. Walsh, and D. Alton. 1987. The statistical properties of available worldwide MSW combustion dioxin/furans emissions as they apply to the conduct of risk assessment. *Chemosphere* 16(8/9):2183-2186.

Kimbrough, R.D. and V.N. Houk. 1987. Effects of Chlorinated Dibenzodioxins. In: *Solving Hazardous Waste Problems.* American Chemical Society (eds.), Washington, D.C. pp. 68-79.

Kimbrough, R.D. 1990. How toxic is 2,3,7,8-tetrachlorodibenzodioxin to humans? *J. Toxicol. Environ. Health* 30:261-271.

Kimbrough, R.D. 1991. *Examining and communicating risk from dioxin exposure.* Proceedings from the Rifkin Conference, "Dioxin: National Conference on Establishing Multi-Media Controls", May 9-10, 1991, Washington, D.C. March 1.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46:279-303.

Konheim, C.S. 1986. Dioxin exposure and motor vehicle emissions. *Waste Age* pp. 69-76.

Kuratsune, M. 1989. Chapter 13: Yusho, with reference to Yu-Cheng. In: *Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products.* Kimbrough, R.D. and A.A. Jensen. (eds.). New York, NY: Elsevier Publishing Co.

Langhorst, M.L. and L.A. Shadoff. 1980. Determination of parts-per-trillion concentrations of tetra-, hexa-, hepta-, and octachlorodibenzo-p-dioxins in human milk. *Anal. Chem.* 52:2037-2044.

Lathrop, G.D., W.D. Wolfe, R.A. Albanese, and P.M. Moynahan. 1984. An epidemiologic investigation of health effects in Air Force personnel following exposure to herbicides. In: *Banbury Report 18. Biological Mechanisms of Dioxin Action.* A. Poland and R.D. Kimbrough (eds.). New York, NY: Cold Spring Harbor Laboratory. pp. 471-474.

Lathrop, G.D., W.H. Wolfe, S.G. Machado, J.E. Michalek, T.G. Karrison, J.C. Miner, W.D. Grubbs, M.R. Peterson, and W.F. Thomas. 1987. Air Force Health Study: An Epidemiologic Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicides. Science Applications International Corporation, VA and Epidemiology Division, United States Air Force. October.

Lucier, G.W., A. Tritscher, T. Goldsworthy, J. Foley, G. Clark, J. Goldstein, and R. Maronpot. 1991. Ovarian hormones enhance 2,3,7,8-tetrachlorodibenzo-p-dioxin-mediated increases in cell proliferation and preneoplastic foci in a two-stage model for rat hepatocarcinogenesis. *Can. Res.* 51:1391-1397.

Manz, A., J. Berger, J.H. Dwyer, D.F. Janys, S. Nagel, and H. Waltsgott. 1991. Cancer mortality among workers in chemical plant contaminated with dioxin. *Lancet* 338:959-964.

Marklund, S., C. Rappe, M. Tysklind, and K.E. Egeback. 1987. Identification of polychlorinated dibenzofurans and dioxins in exhausts from cars run on leaded gasoline. *Chemosphere* 16(1):29-36.

McNaughton, K.J. 1983. Ethylene dichloride process. *Chemical Engineeing* December 12, pp. 54-58.

Moses, M., R. Lilis, K.D. Crow, J. Thornton, A. Fischbein, H.A. Anderson, and I.J. Selikoff. 1984. Health status of workers with past exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in the manufacture of 2,4,5-tetra-chlorophenoxy-acetic acid: Comparison of findings with and without chloracne. *Am. J. Ind. Med.* 5:161-182.

National Academy Science (NAS). 1987. *Pharmacokinetics in Risk Assessment.* Vol. 8. National Academy of Science. Washington, D.C.: National Academy Press.

NCASI. 1987. *Dioxin: A Critical Review of its Distribution, Mechanism of Action, Impacts on Human Health, and the Setting of Acceptable Exposure Limits.* National Council of the Paper Industry for Air and Stream Improvements, Inc. NCASI Special Report No. 87-07. May.

Norsk Hydro. 1992. *PVC and The Environment.* Norsk Hydro a.s., Petrochemical Division, Oslo, Norway. September.

Norwood, C.B., M. Hackett, R.J. Powell, B.C. Butterworth, K.J. Williamson, and S.M. Naumann. 1989. Polychlorinated dibenzo-p-dioxins and dibenzofurans in selected estuarine sediments. *Chemosphere* 18: 553-560.

NTP. 1982. *Carcinogenesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Osborne-Mendel Rats and B6C3F1 Mice.* U.S. National Toxicology Program, Research Triangle Park, NC. PB82-163445. February.

Ontario. 1985. *Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs).* Intergovernmental Relations and Hazardous Contaminants Coordination Branch. Scientific Criteria Document for Standard Development No. 4-84. September.

Parmeggiani, L. 1983. Acetylaminofluorene. In: *Encyclopaedia of Occupational Health and Safety.* Third Edition, Vol. 1. Geneva, Switzerland: International Labour Organisation. pp. 39-40.

Patterson, D., R. Hoffman, L. Needham, D. Roberts, J. Bagby, J. Pirkle, H. Falk, E. Sampson, and V. Houk. 1986. 2,3,7,8-Tetrachlorodibenzo-p-dioxin levels in adipose tissue of exposed and control persons in Missouri. *JAMA* 256(19):2683-2686.

Rappe, C. 1987a. Global Distribution of Polychlorinated Dioxins and Dibenzofurans. Dioxin 88, Umea, Sweden.

Rappe, C., R. Andersson, P.A. Bergqvist, C. Brohede, M. Hansson, L.O. Kjeller, G. Lindstrom, S. Marklund, M. Nygren, S.E. Swanson, M. Tysklind, and K. Wiberg. 1987b. Overview on environmental fate of chlorinated dioxins and dibenzofurans: sources, levels and isomeric pattern in various matrices. *Chemosphere* 16(8/9):1603-1618.

Rappe, C., R. Andersson, P.A. Bergqvist, C. Brohede, M. Hansson, L.O. Kjeller, G. Lindstrom, S. Marklund, M. Nygren, S.E. Swanson, M. Tysklind and K. Wiberg. 1987c. Sources and relative importance of PCDD and PCDF emissions. *Waste Manage. Res.* 5:225-237.

Rappe, C. and L. Kjeller. 1987. PCDDs and PCDFs in environmental samples air, particulates, sediments and soil. *Chemosphere* 16(8/9):1775-1780.

Rappe, C. and L. Kjeller. 1988. Identification and concentration of PCDDs and PCDFs in urban air. *Chemosphere* 17(1):3-20.

Rappe, C., P.A. Bergqvist, and L.O. Kjeller. 1989. Levels, trends and patterns of PCDDs and PCDFs in Scandinavian environmental samples. *Chemosphere* 18(1-6):651-658.

Rappe, C., I. Fangmark, S. Marklund, and M. Tysklind. 1990. *Formation of dioxins and dibenzofurans during incineration and pyrolysis of PVC A Controversial Issue.* Proceedings from the Dioxin '90 Conference, Tenth International Conference on Organohalogen Compounds, Beyreuth, F.R.G.

Reckhow, D.A., P.C. Singer, and R.L. Malcolm. 1990. Chlorination of humic materials: Byproduct formation and chemical interpretations. *Environ. Sci. Technol.* 24(11):1655-1664.

Rothman, K.J. 1986. Chapter 2: Causal Inference in Epidemiology. In: *Modern Epidemiology.* Boston, MA: Litte, Brown and Company. pp. 15-21.

Saracci, R., M. Kogevinas, P.A. Bertazzi, B.H. Bueno De Mesquita, D. Coggon, L.M. Green, T. Kauppinen, K.A. L'Abbe, M. Littorin, E. Lynge, J.D. Mathews, M. Neuberger, J. Osman, N. Pearce, and R. Winkelmann. 1991. Cancer mortality in workers exposed to chlorophenoxy herbicides and chlorophenols. *Lancet* 338(8774):1027-1032.

Schecter, A., O. Papke, and M. Ball. 1990. Evidence for transplacental transfer of dioxins from mother to fetus: chlorinated dioxin and dibenzofuran levels in the livers of stillborn infants. *Chemosphere* 21(8):1017-1022.

Schroy, J.M., F.D. Hilleman, and S.C. Cheng. 1985. Physical/chemical properties of 2,3,7,8-TCDD. *Chemosphere* 14(6/7): 877.

Sheffield, A. 1985. Sources and releases of PCDDs and PCDFs to the Canadian environment. *Chemosphere* 14(6/7):811-814.

Southerland, J.H., W.B. Kuykendal, W.H. Lamason, A. Miles, and D.A. Oberacker. 1987. Assessment of Combustion Sources as Emitters of Chlorinated Dioxin Compounds: A Report on the Results of Tier 4 of the National Dioxin Study. *Chemosphere* 16(8/9):2161-2168.

Smith, R.M., P.W. O'Keefe, K.M. Aldous, H. Valente, S.P. Connor, and R.J. Donnelly. 1990. Chlorinated dibenzofurans and dioxins in atmospheric samples from cities in New York. *Environ. Sci. Technol.* 24(10):1502-1506.

Stanley, J.S., K.E. Boggess, J. Onstot, T.M. Sack, J.C. Remmers, J. Breen, F.W. Kutz, J. Carra, P. Robinson, and G.A. Black. 1986. PCDDs and PCDFs in human adipose tissue from the EPA Fy82 NHATS repository. *Chemosphere* 15(9-12):1605-1612.

Suskind, R.R. and V.S. Hertzberg. 1984. Human health effects of 2,4,5-T and its toxic contaminants. *JAMA* 251(18):2372-2380.

Susser, M. 1973. Criteria of Judgement. In: *Causal Thinking in the Helath Sciences Concepts and Strategies of Epidemiology.* New York, N.Y.: Oxford university Press. pp. 140-162.

Susser, M. 1986. Reviews and Commentary: The logic of Sir Karl Popper and the practice of epidemiology. *Am. J. Epidemiol.* 124(5):711-718.

Sweeney, M.H., M.A. Fingerhut, D.G. Patterson, L.B. Connally, L.A. Piacitelli, J.A. Morris, A.L. Greife, R.W. Hornung, D.A. Marlow, J.E. Dugle, W.E. Halperin, and L.L. Needham. 1990. Comparison of serum levels of 2,3,7,8-TCDD in TCP production workers and in an unexposed comparison group. *Chemosphere* 20(7-9):993-1000.

Thoma, H. 1988. PCDD/F-concentrations in chimney soot from house heating systems. *Chemosphere* 17(7):1369-1379.

Tiernan, T.O., D.J. Wagel, G.F. VanNess, J.H. Garrett, J.G. Solch, and L.A. Harden. 1989. PCDD/PCDF in the ambient air of a metropolitan area in the U.S. *Chemosphere* 19(1-6):541-546.

Tollefson, L. 1991. Use of epidemiology data to assess the cancer risk of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Regul. Toxicol. Pharmacol.* 13(2):150-169.

Tong, H.Y., S. Arghestani, M.L. Gross, and F.W. Karasek. 1989. Polychlorodiben-zodioxins and Polychlorodibenzofurans in the Dust of the Ambient Atmosphere in a Municipal Waste Incinerator. *Chemosphere* 18(1/6):577-584.

USEPA. 1980. *Dioxins.* U.S. Environmental Protection Agency, Industrial Environmental Research Lab, Cincinnati, OH. EPA-600/2-80-197. November.

USEPA. 1984a. *Ambient Water Quality Criteria for 2,3,7,8-Tetrachloro-Dibenzo-p-Dioxin.* U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-84-007.

USEPA. 1985. *Health Assessment Document for Polychlorinated Dibenzo-p-dioxins.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-84/014F.

USEPA. 1986. *Guidelines for Carcinogen Risk Assessment.* U.S. Environmental Protection Agency, 15 FR 185:33992-34003.

USEPA. 1987. *Hazardous Waste Treatment, Storage, and Disposal Facilities (TSDF) - Air Emission Models*. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA/450/3-87-026.

USEPA. 1991. Memo from W.K. Reilly Re: Dioxin Reassessment and Research. U.S. Environmental Protection Agency, Washington, D.C.

USEPA. 1992. *Review Draft: Chapter 8. Dose-Response Relationships.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/AP-92/001h. August.

United Kingdom (UK). 1989. Dioxins in the Environment. Report of the Interdepartmental Working Group on Polychlorinated Dibenzo-para-Dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs). Department of the Environment, United Kingdom.

vanderHeijden, C., A. Knaup, P. Kramers, and M. Van Logten. 1982. *Evaluation of the Carcinogenicity and Mutagenicity of 2,3,7,8-Tetrachlorodibenzo-1,4-Dioxin (TCDD): Classification and No-effect Level.* State Institute of National Health, Bilthoven, The Netherlands. Report DOC/LCM 300/292 (cited in Shu et al., 1987).

vanderHeijen, C.A. 1990. Statement on the outcome of the Banbury Conference: Biological Basis for Risk Assessment of Dioxins and Related Compounds.

Vinyl Institute. 1993. *Important Information about PVC, its Users, and The Vinyl Industry.* The Vinyl Institute A Division of the Society of The Plastics Industry, Inc. Wayne, NJ.

Vos, J.G., J.A. Moore, and J.G. Zinki. 1973. Effect of 2,3,7,8-tetrachlorodibenzo-p-dioxin on the immune system of laboratory animals. *Environ. Health Perspect.* September:149-162.

Wenning, R.J., M.A. Harris, D.J. Paustenbach, and H. Bedbury. 1992. Potential sources of 1,2,8,9-tetrachlorodibenzo-p-dioxin in the aquatic environment. *Ecotoxicol. Environ.Saf.* 23:133-146.

WHO. 1990. Press release: Consultation on tolerable daily intake from food of PCDDs and PCDFs. World Health Organization, Bilthoven, Netherlands.

Windholtz, M., S. Budavari, R.F. Blumetti, and E.S. Otterbein. 1983. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals.* Tenth Edition. Rahway, NJ: Merck & Co., Inc.

Wolfe, W.H., J.E. Michalek, and J.C. Miner. 1991. *Air Force Health Study: An Epidemiological Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicides: Mortality Update - 1991.* Armstrong Laboratory, Epidemiological Research Division, Human Systems Division (AFSC), Brooks Air Force Base, TX. AD-A241 874. October 15.

Zober, A., P. Messerer, and P. Huber. 1990. Thirty-four-year mortality follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident. *Int. Arch. Occup. Environ. Health* 62:139-157.