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# **HEALTH HAZARD EVALUATION REPORT**

**HETA 88-0140-2517  
BOISE CASCADE  
UNITED PAPERWORKERS  
INTERNATIONAL UNION  
RUMFORD, MAINE**

## **PREFACE**

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer and authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to federal, State, and local agencies; labor; industry; and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

**HETA 88-0140-2517  
AUGUST 1995  
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UNITED PAPERWORKERS  
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**NIOSH INVESTIGATORS:  
Robert Mouradian, Ph.D.  
Susan Burt, M.Sc., M.S.N.  
Allison Tepper, Ph.D.  
Kevin Hanley, M.S.P.H., C.I.H.**

## **SUMMARY**

In May 1988, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation (HHE) from the United Paperworkers International Union (UPIU). The request asked NIOSH to assess employee exposures to polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and chlorinated volatile organic compounds (CVOCs) formed as by-products during wood pulp bleaching at pulp and paper mills. In response to this request, NIOSH investigators conducted walk-through surveys at a number of pulp and paper mills in order to select a facility for a more in-depth site survey. The Boise Cascade Paper Company in Rumford, Maine, was selected for further study since it had high PCDD/PCDF waste-water effluent levels reported by the Environmental Protection Agency (EPA), an older stable work population with many years of potential exposure, complete employment records, and the willingness of workers to participate. The survey at the Boise Cascade plant consisted of environmental monitoring and analysis of serum samples from workers and community residents to assess exposures to PCDDs, PCDFs, and CVOCs.

In August 1989, five area air samples were collected and analyzed for the 2,3,7,8-tetra PCDD/PCDF isomers and total tetra PCDD/PCDF isomers. In August 1991, five surface wipe samples were collected and analyzed for the 2,3,7,8-tetra PCDD/PCDF isomers as well as specific and total tetra-chlorinated through octa-chlorinated PCDD and PCDF isomers. The PCDD/PCDF concentrations are reported as 2,3,7,8-TCDD toxicity equivalents (I-TEQ) using the 1989 International Toxicity Equivalency Factors (I-TEFs). Airborne I-TEQ concentrations ranged from .01 to .06 picograms per cubic meter (pg/m<sup>3</sup>). (These results however, may be a slight underestimate of the true I-TEQ due to analytic limitations.) Of the five air sample locations, the highest I-TEQ concentrations were detected in the hardwood bleach plant and at the hardwood rewind station. The wipe sample results indicated greater amounts of PCDDs/PCDFs near the dry end of a paper machine and on the side of a hardwood chlorine bleaching rinse tank. The TEQ levels on the surface wipe samples ranged from 13 to 651 picograms per square meter (pg/m<sup>2</sup>) in the bleach plant and from 86 to 1049 pg/m<sup>2</sup> in the paper mill. All I-TEQ concentrations were well below the National Research Council (NRC) guideline of 10.0 pg/m<sup>3</sup> for airborne PCDDs/PCDFs and 25,000 pg/m<sup>2</sup> for PCDD and PCDF surface contamination. Neither the Occupational Safety and Health Administration (OSHA) nor the American Conference of Governmental Industrial Hygienists (ACGIH) have evaluation criteria for PCDDs/PCDFs. NIOSH considers 2,3,7,8-TCDD to be a potential human carcinogen and recommends that occupational exposure be controlled to the lowest feasible level.

Area and personal breathing zone (PBZ) air samples were also collected and analyzed for CVOCs during the August 1991 survey. Mass spectroscopy analysis qualitatively identified many compounds. Chloroform, 1,1,1-trichloroethane, carbon tetrachloride, dibromochloromethane, and bromodichloromethane were the major compounds detected and these were quantitatively analyzed with gas chromatography (GC). Area concentrations of chloroform obtained near ports of pulp bleaching rinse tanks ranged from 1.8 milligrams per cubic meter ( $\text{mg}/\text{m}^3$ ) to  $116 \text{ mg}/\text{m}^3$  and were highest at the hypochlorite stage. During the initial sampling survey in August 1991, some PBZ exposures of bleach plant operators exceeded the NIOSH Recommended Exposure Limit (REL) for chloroform of  $10 \text{ mg}/\text{m}^3$ . A follow-up visit was conducted in March 1992 to assess the impact of process changes on airborne chloroform concentrations. This survey found chloroform levels to be substantially lower, indicating that the process changes resulting in reduced usage of hypochlorite in the bleaching process were successful in lowering workers' exposures below the NIOSH REL.

Serum samples were collected from two groups of Boise Cascade workers and community residents and analyzed for PCDDs and PCDFs. Overall, there were no appreciable differences among the exposure groups (community resident, low exposure potential worker, high exposure potential worker) for specific PCDDs or PCDFs. The highest values, however, occurred consistently in seven workers, all but one of whom had worked for some time in the paper mill. Neither exposure group nor duration in high exposure potential jobs was related to PCDD and PCDF serum levels. Serum levels of PCDDs and PCDFs in both workers and community residents generally were within the range previously reported for persons with no known occupational exposure.

Industrial hygiene data collected at the Rumford mill indicate the potential for workers to be exposed to low levels of PCDDs and PCDFs. Current body burdens of PCDDs and PCDFs were within the background ranges previously reported for persons with no known occupational exposure. Differences between mill workers and community residents in serum concentrations of PCDDs and PCDFs were small. Although the highest levels for specific PCDDs and PCDFs occurred in workers, these results were not clearly related to occupational exposure. No relationship was seen between PCDD or PCDF levels in serum and work in jobs thought to have a potential for high exposure. Air sampling results indicate that process modifications have substantially reduced the potential for overexposure to chloroform. The prohibition of smoking and eating in exposure areas, use of personal protective equipment to reduce dermal contact, and periodic exposure monitoring are recommended.

**KEYWORDS:** SIC 2621 (Pulp and Paper Mills), wood pulping, bleaching, paper machines, chlorine, hypochlorite, polychlorinated dibenzo-*p*-dioxins, 2,3,7,8 tetrachlorinated dibenzo-*p*-dioxins, TCDD, polychlorinated dibenzofurans, toxicity equivalents, I-TEQ, chloroform, serum.

## **INTRODUCTION**

In May 1988, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation (HHE) from the United Paperworkers International Union (UPIU). The UPIU asked NIOSH to evaluate workplace exposures to polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and chlorinated volatile organic compounds (CVOCs) that are formed as by-products when pulp is bleached with chlorine-based chemicals. Concern over potential worker exposures to PCDDs and PCDFs arose when a study conducted by the Environmental Protection Agency (EPA) and the American Paper Institute (API) identified these compounds in sludge, pulp, and effluent from bleached pulp mills.<sup>1,2</sup> These low-volatility polychlorinated compounds have also been identified in finished paper products. Concern with potential exposures to other chlorinated compounds, including potential carcinogens such as chloroform, is based on studies that have detected these compounds as by-products of the bleaching process.<sup>3</sup>

In response to the UPIU request, NIOSH investigators made a series of walk-through evaluations at pulp and paper mills in the Northwest, Northeast, and Southeast areas of the U.S. to become familiar with paper making processes, identify jobs with potential exposure to PCDDs/PCDFs and CVOCs, and select appropriate sites for further study. Potential study sites were initially selected on the basis of PCDD and PCDF levels reported in the EPA/API survey of contaminant levels in pulp, effluent, and sludge in 104 mills.<sup>2</sup> Only mills that fell in the top 15 in terms of reported PCDD/PCDF levels in effluent, sludge, or pulp were considered for exposure assessments. Sites were evaluated for further study on the basis of several factors, including stability of the work force, quality of employment records, current and past bleaching technologies, product line, and willingness of the workers to participate.

Following the initial walk-through inspections, a more complete environmental evaluation was conducted at the Boise Cascade pulp and paper mill in Rumford, Maine. The Rumford mill was selected because of relatively high levels of PCDD/PCDF reported by the EPA, because it is a large and relatively old facility with a stable employee population, and because NIOSH investigators were already conducting another HHE at that site.

In August 1989, air sampling was conducted for tetrachlorinated PCDDs and PCDFs. In August 1991, surface wipe samples were collected for PCDDs and PCDFs. In addition, air sampling was conducted for CVOCs and for paper dust, the latter as an indicator of potential respiratory exposure to PCDDs and PCDFs. In March 1992, a follow-up air sampling survey was conducted to repeat the monitoring for chloroform and CVOCs. Letters discussing the chloroform and CVOCs results were sent to company and local union representatives in January 1991 and January 1992.

A medical evaluation was conducted in May 1992. This evaluation consisted of a questionnaire and biological monitoring to determine serum concentrations of PCDDs and PCDFs in selected workers. In June 1993, letters were sent to each study participant providing individual test

results. An interim letter describing the preliminary findings from the serum analyses was also sent to the local union representative, Boise Cascade management in Rumford, and all study participants.

## **BACKGROUND**

The Boise Cascade Rumford facility is a fully integrated Kraft pulp and paper bleach mill capable of producing 1400 tons per day (tpd) of finished paper products. Approximately 1600 workers were employed at the time of the NIOSH surveys. The mill has both a hardwood and softwood line and also processes approximately 140 tpd of groundwood (thermomechanical process) products. The mill generates its own power (power boilers) and steam and has a primary water treatment system (clarifiers) for effluent. The Kraft pulping process is a chemical treatment method that uses a solution of sodium hydroxide and sodium sulfide (white liquor) to dissolve the lignin in wood and release the cellulose fibers. Approximately 80% of pulp mills in the United States use the Kraft process. A principal feature of the Kraft process is efficiency, as most of the chemicals can be recovered, regenerated, and recycled back into the process.

Some products (coated paper, magazine paper, copy paper, etc.) call for the use of bleached pulp. The bleaching process chemically changes the color of the pulp from brown to white and also removes impurities. Although the processes may vary somewhat, most bleaching operations entail treatment, in stages, with chlorine, sodium hypochlorite, sodium hydroxide (extraction), and chlorine dioxide. The pulp is usually washed after each stage and undergoes a final rinsing prior to concentration (dewatering) of the final stock, addition of various additives, and delivery to the paper machines. Because of the concern with PCDD and PCDF formation, many bleaching operations have reduced the use of chlorine, and substituted, for example, chlorine dioxide.

At the Boise Cascade plant the bleaching sequence during the time period the samples were collected was chlorine gas, sodium hydroxide extraction, oxygen, peroxide, sodium hypochlorite, and chlorine dioxide soaking. Chlorine dioxide was generated on-site. Liquid chlorine was delivered from 90-ton rail car containers and processed through a vaporization system to produce chlorine gas. During the last follow-up site visit, the hypochlorite stage was eliminated in the softwood bleaching process and was substantially reduced in the hardwood process.

There were numerous paper machines present at the Boise Cascade plant, each generally dedicated for a class of products using similar pulp (or paper) feed stock. After additives are blended, the pulp stock is pumped to the wet end of the paper machine and a thin layer of the pulp is applied to a carrier screen. The screen is conveyed continuously through multiple series of rollers within the paper machine, which applies heat to dry the pulp into a sheet of paper. Coatings, if applicable, are applied onto the paper surface, and the finished paper is wound into very large rolls at the dry end of the machine. At the calenders, paper is passed between rollers

to improve the finish and polish the paper surface. The rolls are then cut into smaller sizes by paper slitters at the rewinders.

## **EVALUATION CRITERIA**

As a guide for the assessment of hazards posed by workplace exposures, evaluation criteria have been developed for a number of chemical and physical agents. The primary sources of evaluation criteria for the workplace environment are the following: **1)** NIOSH Criteria Documents and Recommended Exposure Limits (RELs), **2)** the U.S. Department of Labor, Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs), and **3)** the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLVs).<sup>4,5,6</sup> These criteria establish levels of inhalation exposure to which it is believed most workers may be exposed without experiencing adverse health effects.

Full-shift and shorter duration inhalation criteria are available for over 700 chemical substances. Full-shift limits are based on the time-weighted average (TWA) airborne concentration of a substance to which most workers may be repeatedly exposed during a normal eight- or ten-hour day, up to 40 hours per week for a working lifetime, without adverse effect. Some substances have short-term exposure limits (STELs) or ceiling limits which are intended to supplement the full-shift criteria when there are recognized irritative or toxic effects from brief exposures to high airborne concentrations. STELs are usually based on average concentrations over 15 minute time periods, whereas ceiling limits are concentrations which should not be exceeded even momentarily.

Occupational exposure limits are generally based on information from industrial experience, animal or human experimentation, and epidemiological studies. When comparing exposure criteria, it should be noted that employers are legally required to meet those levels (and any conditions) specified by an OSHA PEL.

Promulgation of OSHA PELs has historically been a lengthy legal process and must consider the technical and economical feasibility of achieving the proposed limit. NIOSH RELs, however, are primarily based upon the prevention of occupational disease. The ACGIH is an organization of industrial hygienists and other professionals in related disciplines employed in the public or academic sector. TLVs are developed by consensus agreement of the ACGIH TLV committee and are published annually. The documentation supporting the TLVs (and proposed changes) is periodically reviewed and updated if believed necessary by the committee.<sup>7</sup>

Not all workers may be protected from adverse health effects if their exposures are maintained below these occupational health exposure criteria. A small percentage of workers may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, previous exposures, and/or a hypersensitivity (allergy). In addition, some hazardous substances may act in combination with other workplace exposures, or with medications or

personal habits of the worker (such as smoking, etc.) to produce health effects even if the occupational exposures are controlled at or below the recommended level. These combined effects are often not considered by chemical-specific evaluation criteria. Furthermore, many substances are appreciably absorbed by direct contact with the skin and thus potentially increase the overall exposure and biologic response beyond that expected from inhalation alone. Finally, evaluation criteria may change over time as new information on the toxic effects of an agent become available.

The pertinent evaluation criteria and toxicological background information for the chemical substances evaluated during this HHE are presented below.

### **Polychlorinated Dibenzo-*p*-Dioxins and Furans (PCDDs/PCDFs)**

PCDDs and PCDFs are two types of chlorine-substituted tricyclic aromatic compounds. The number of chlorine atom substitutions can vary between one and eight (mono- through octa-chloro homologs), resulting in 75 PCDDs and 135 PCDF positional congeners. The toxicity of these compounds varies with the number and specific placement of the chlorine atoms in the molecule. The tetra-, penta- and hexachlorinated isomer groups exhibit greater toxicity than other chlorinated forms.<sup>8,9,10</sup> PCDDs and PCDFs with chlorine at positions 2,3,7, and 8 are particularly toxic.<sup>11,12,13</sup>

PCDDs and PCDFs are highly toxic in experimental animals when administered acutely, subchronically, or chronically.<sup>14,15,16,17,18,19,20,21</sup> Adverse health effects in experimental animals include severe weight loss, liver necrosis and hypertrophy, skin lesions, immunosuppression, reproductive toxicity, teratogenesis, and death. Of the 75 PCDD and 135 PCDF isomers, only 2,3,7,8-TCDD and a mixture of hexachlorinated dibenzo-*p*-dioxins with four of the six chlorines in positions 2,3,7, and 8 have been tested for carcinogenicity. Two independent studies of 2,3,7,8-TCDD showed significant increases in the incidence of liver and/or lung tumors in exposed rodents.<sup>21,22</sup> A mixture of two 2,3,7,8-substituted hexachlorinated dibenzodioxins was found to produce an increased incidence of liver tumors or neoplastic nodules in exposed rats and mice.<sup>23</sup>

Numerous studies have been conducted to examine the possible relationship between exposure to PCDDs, primarily 2,3,7,8-TCDD, and cancer in humans. Groups that have been studied include chemical manufacturing workers,<sup>24,25,26</sup> pulp and paper mill workers,<sup>27,28,29,30,31</sup> the general population in several countries,<sup>32,33,34,35,36,37,38</sup> and the population of Seveso, Italy, where a chemical explosion exposed a community to very high levels of 2,3,7,8-TCDD.<sup>39</sup> The results of these studies provide consistent information about risks for some types of cancer, but offer mixed results for most cancers. The epidemiological evidence is strongest for an increase in risk for soft tissue sarcoma (although there is debate about the magnitude of the risk) and for an increase in risk for all cancers as a group. There also appears to be an increase in risk for lung cancer, but this is probably limited to people with very high levels of exposure.



Epidemiologic studies also have examined the possible relationship between TCDD exposure and other health effects in humans. Although exposure can cause chloracne,<sup>40</sup> these studies have not found an association between exposure and long-term effects on the gastrointestinal system (including the liver),<sup>41</sup> the immune system,<sup>42</sup> and the lung.<sup>43</sup> Additionally, there is no evidence that exposure is related to peripheral neuropathy or mood disorders.<sup>42,44,45</sup> There is some indication, however, of alterations in glucose metabolism and in male reproductive hormones among individuals with very high levels of 2,3,7,8-TCDD in serum.<sup>46,47</sup>

Occupational exposure criteria for PCDDs and PCDFs are not currently available from NIOSH, OSHA, or ACGIH. However, NIOSH recommends that 2,3,7,8-TCDD be regarded as a potential occupational carcinogen and that occupational exposure to 2,3,7,8-TCDD be controlled to the lowest feasible level.<sup>19</sup> This recommendation is based on studies demonstrating carcinogenicity in rats and mice.

Exposure to PCDDs/PCDFs are generally expressed in terms of 2,3,7,8-TCDD toxicity equivalents. Because of the many different isomers of chlorinated dioxin and "dioxin-like" compounds, each with varying levels of toxicity, the Toxicity Equivalent (I-TEQ) system was developed.<sup>48</sup> This weighting method reduces the amount of data researchers need to consider when estimating the toxicity of a complex mixture of dioxin-like compounds. The system is also valuable because it allows comparison of mixtures that have differing compositions. With the I-TEQ system, each isomer is assigned an equivalency factor which reflects its toxicity relative to 2,3,7,8 TCDD. 2,3,7,8-TCDD equivalents are defined as the concentration of 2,3,7,8-TCDD which, by itself, would exhibit the same toxicological potency as the mixture of structurally-related PCDD and PCDF compounds that are actually present in a sample. The structurally-related PCDDs and PCDFs that are considered in the calculation of 2,3,7,8-TCDD equivalents include the tetra- through octa-chloro homologs and 2,3,7,8-substituted isomers.

The weighting factors, referred to as toxicity equivalency factors (TEFs), were proposed in 1987 by the EPA.<sup>49</sup> In 1989, the EPA adopted the International TEFs (Table 1).<sup>50</sup> The International (I-89) TEFs were used in this report. The concentrations of the PCDD and PCDF compounds are converted to TCDD equivalents by multiplying measured values of each isomer by the appropriate TEF. The TCDD equivalents for each measured isomer are then summed to calculate the total I-TEQ, which is compared to the guideline value. Hence, the I-TEQ provides an estimate of the 2,3,7,8-TCDD concentration that would be as toxic as the mixture of isomers that was actually measured.

The Dioxin Subcommittee of the National Research Council (NRC) released a report on acceptable levels of dioxin contamination in office buildings following transformer fires.<sup>51</sup> Exposure guidelines adopted by the subcommittee were 10 pg/m<sup>3</sup> for air and 25 ng/m<sup>2</sup> for surfaces of 2,3,7,8-TCDD equivalents calculated using the 1987 EPA TEFs. Reported lifetime cancer risk estimates were 9 x 10<sup>-8</sup> to 2 x 10<sup>-4</sup> at the recommended guidelines. Risks correspond to a single source contamination (either air or surface). Risks for simultaneous exposures are considered to be additive. For example, risks apply for exposure to 10 pg/m<sup>3</sup> of air only,

25 ng/m<sup>2</sup> of surface only, or 5 pg/m<sup>3</sup> of air plus 12.5 ng/m<sup>2</sup> of surface. Simultaneous exposure at 10 pg/m<sup>3</sup> of air and 25 ng/m<sup>2</sup> of surface implies twice the risk.

### **Chloroform**

Chloroform (trichloromethane) is a colorless, non-flammable, volatile organic solvent with a sweet characteristic odor similar to ether; it is a liquid at standard temperature and pressure.<sup>52</sup> The chemical industry uses chloroform as an intermediate in the manufacture of fluorocarbon refrigerants, resins, and plastics; it is also used as an extractant and industrial solvent in the pharmaceutical industry.<sup>7,53</sup> Prior to World War II, chloroform was frequently used as an anesthetic but this use was discontinued due to the risk of cardiac arrest and delayed hepatic injury.<sup>53</sup>

Inhalation exposure to chloroform can cause hepatotoxicity and central nervous system (CNS) depression.<sup>53</sup> Symptoms of the latter include vertigo, dizziness, drowsiness, incoordination, headache, and, if exposures are sufficient, unconsciousness.<sup>54,55</sup> Direct contact with the liquid or high concentrations of vapor may cause irritation of the mucous membranes, eyes, and skin.

Animal studies have suggested that chloroform is carcinogenic.<sup>53</sup> The National Toxicology Program, an interagency research program, has found evidence supporting an association between chloroform exposure and the development of cancer in experimental animals and has classified chloroform as a compound that "can be reasonably anticipated to be a human carcinogen."<sup>56</sup> The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence to establish chloroform as a potential human carcinogen (Group 2B).<sup>57</sup> ACGIH also lists chloroform as a suspect human carcinogen (class A2); based on the available evidence, ACGIH considers chloroform an animal carcinogen at dose levels and routes of exposure relevant to occupational exposure, but the epidemiological evidence is insufficient to confirm an increased cancer risk to exposed humans.<sup>7</sup>

The OSHA PEL for chloroform is a ceiling limit of 50 parts per million (ppm) (240 milligrams per cubic meter ( mg/m<sup>3</sup>)).<sup>5</sup> The ACGIH TLV for chloroform is 10 ppm (49 mg/m<sup>3</sup>), as an 8-occupational carcinogen and recommends that exposure be reduced to the lowest feasible level. NIOSH has established a 2 ppm (10 mg/m<sup>3</sup>) 60-minute short-term REL (the lowest level that can be reliably measured).<sup>58</sup>

### **Paper Dust**

Air sampling for paper dust was conducted to compare dust exposures between job categories and plant locations. This does not provide a direct measure of PCDD and PCDF exposure but allows a comparison of the relative potential for exposure to PCDDs and PCDFs that may be adhered to suspended paper particles.

The NIOSH REL for cellulose (paper dust) is 10 mg/m<sup>3</sup> (total dust) and 5 mg/m<sup>3</sup> (respirable dust) as a full-shift time-weighted average.<sup>58</sup> The REL is based on eye and skin irritation. Regulatory standards also exist for a more general category termed "particulate not otherwise classified" (PNOC). Dusts considered to be physical irritants for which no substance-specific toxicological data are available are generally placed in this category by the Occupational Safety and Health Administration (OSHA) for enforcement purposes.<sup>5</sup> It is important to note that the NIOSH REL and OSHA PNOC criteria are not intended to address the issue of contaminants, such as PCDDs and PCDFs, that may be present in the dust.

The respirable fraction is considered to be that portion of inhaled dust which penetrates to the non-ciliated portions of the lung. In general, particles greater than 7-10 micrometers in diameter (µm) are removed in the nasal passages and have little probability of penetrating to the lung. Particles smaller than this can reach the air-exchange regions (alveoli, respiratory bronchioles) of the lung, and are considered more hazardous.<sup>59</sup>

## **STUDY DESIGN AND METHODS**

To address the UPIU HHE request it was necessary to determine current worker exposure levels to all compounds of interest by environmental monitoring and assess cumulative exposure to PCDDs and PCDFs by biological monitoring. Because PCDDs and PCDFs have a long biological half-life (approximately 7 years) and some health effects may have long latency periods, it is important that past exposures be evaluated as well as current work conditions. Because most pulp and paper manufacturers modified their bleaching process over the last few years, there was concern that retrospective assessments based on current environmental measurements might underestimate past exposures. Direct measurement of blood levels was determined to be the most appropriate approach to evaluating exposures.

### **Industrial Hygiene**

Data collected by the EPA and the paper industry have demonstrated that most, if not all, of the PCDDs/PCDFs found in mill effluent and pulp are formed during the bleaching process.<sup>2</sup> Occupational exposures to PCDDs and PCDFs are most likely to occur in locations where there is contact with bleaching effluent, or bleached pulp and paper. Potential exposure routes include dermal absorption following skin contact with pulp or paper and respiratory exposure resulting from inhalation of vapor or particulate.<sup>60,61</sup> Dermal absorption maybe a significant intake route.<sup>60</sup>

Because dioxins and furans have very low vapor pressures, most respiratory exposures would probably occur as a result of inhalation of contaminated paper dust. Work areas with the highest concentrations of airborne paper dust (and to a lesser extent, bleached pulp mist or dust) would

likely have the greatest potential for inhalation exposure to PCDDs and PCDFs. Unless otherwise noted, all air sampling was conducted for the duration of the work-shift or activity of interest.

#### *PCDD and PCDF - Air Samples*

Air samples for PCDD/PCDF compounds were collected using methods developed by the New York State Health Department.<sup>62</sup> The air sampling device for PCDD/PCDF compounds consists of two stages. The first stage was a 47 millimeter (mm) glass microfiber filter (EM 2000) with a 0.3 micrometer ( $\mu\text{m}$ ) pore size for collecting particulate. The second stage was used to collect vapors and contains a glass cartridge with eight grams of activated 30/70 mesh silica gel adsorbent. The silica gel cartridges were spiked with 2,3,7,8-TCDD and 2,3,7,8-TCDF with radioactive carbon ( $^{13}\text{C}$ ) or chlorine ( $^{37}\text{Cl}$ ) markers, both for quantification and to account for any retention losses during sampling. The glass cartridge containing the spiked silica gel adsorbent was sealed in a rugged Teflon® housing with fluorelastomer Viton® "O" rings. During the monitoring, the sampler was placed in a vertical position and attached via Tygon® tubing to a calibrated rotary vane vacuum pump at a flow rate of 20 liters per minute (Lpm). Flow rates were regulated and adjusted using precision control valves and rotometers. After collection the samples were shipped to a contract laboratory for analysis.

Sample analysis was performed by a contract laboratory using procedures similar to that described by EPA procedure 8290, and included the use of high resolution gas chromatography/high resolution mass spectrometry (HRGC/HRMS) techniques.<sup>63,64</sup> The samples were desorbed with toluene for 16 hours using a Soxhlet apparatus to extract the PCDD and PCDF. The samples were then purified by passing the extract through various columns containing silica gel, acid alumina, carbon, and neutral alumina using various mixtures of hexane, methylene chloride, and benzene to elute the analytes.

The purified samples were analyzed by HRGC/HRMS equipped with a DB-5 chromatographic column. For those samples (all except for the field blank) which had detectable amounts of 2,3,7,8-TCDF present, the analysis was repeated using a OV-225 GC Column, a confirming column which adequately resolves 2,3,7,8-TCDF from the other TCDF isomers. Selected  $^{13}\text{C}$  or  $^{37}\text{Cl}$  labeled PCDD and PCDF isomers were included as internal standards and recovery (surrogate) standards.

Because of resource limitations, only "screening" analyses were conducted to measure total tetra-chlorinated dibenzofurans and dibenzodioxins, and specific PCDD and PCDF isomers containing chlorine substitution in the 2, 3, 7, and 8 positions.

#### *PCDD and PCDF - Surface Wipe Samples*

During the August 1991 site visit, five surface wipe samples were collected in work areas with potential for frequent exposure to bleached pulp. Sampling sites selected for wipe sampling were

laboratory benches in the hardwood and softwood bleach plants, a section of wall below a hardwood (chlorine) bleaching rinse tank sampling port, a wall beside the wet end of paper machine #7, and the back of an electrical control box at the dry end of paper machine #7. These areas were selected because there appeared to be frequent contact with bleached pulp or paper dust and some potential for skin contact.

Wipe samples were collected with pre-extracted, hexane-soaked gauze according to the methods used in other NIOSH HHEs.<sup>65</sup> For each sample, a one-square meter surface area was wiped once vertically and once horizontally. After collection, the samples were stored in dark glass containers to protect the samples from ultraviolet degradation and shipped to the laboratory for analysis. The samples were analyzed by a contract laboratory using standard EPA methodology, which included Soxhlet extraction, sample clean-up, GC-separation, and peak identification with HRMS64. Improvements in the analytical method allowed for the quantification of more specific isomers, including total tetra-, penta-, hexa-, hepta-, and octachlorinated dibenzofurans and dibenzodioxins, and specific PCDD and PCDF isomers containing chlorine at the 2, 3, 7, and 8 positions. It should be noted that wipe sampling results provide only a qualitative indicator of exposure potential. Wipe sampling can be used to identify areas with contamination, but will not provide a direct measure of exposure or absorption. Exposure to PCDDs/PCDFs could occur through direct skin contact with bleached pulp and paper, thus contamination of surfaces is only one possible route of dermal exposure.

#### *CVOCs*

Air sampling for chlorinated volatile organic compounds (CVOCs) was conducted in August 1991 and March 1992 at the Boise Cascade mill. Air samples were collected in the hardwood and softwood bleach plant and at the wet end of five paper machines. Both personal breathing zone (PBZ) and area air samples were collected using calibrated sampling pumps. The sampling was conducted according to NIOSH method #1003, using activated charcoal as the collection media and analysis by GC equipped with a flame ionization detector (FID).<sup>66</sup> A limited number of duplicate samples were also collected by the same sampling method and analyzed qualitatively using Gas Chromatography-Mass Spectrometry (GC-MS) to confirm the identity of the halogenated volatile organic compounds.

#### *Paper Dust*

Air sampling for paper dust was conducted at the dry end of each paper machine and at each of the rewind and paper cutting operations. The samples were collected on PVC filters according to NIOSH method #0500.<sup>67</sup> Both personal and area samples were collected using battery-powered personal sampling pumps and total dust was determined gravimetrically. Initially, a number of samples were selected for microscopic analysis to determine the sample proportion composed of cellulose fibers and total organic dust. Due to analytical difficulties, however, no meaningful information was obtained from the microscopic analyses.

## **Medical**

### *Study Participants*

The objective of the medical component of the HHE was to determine serum concentrations of PCDDs and PCDFs in selected mill employees and compare them to concentrations in community residents who never worked in the mill. Long-term workers in jobs that were most likely to result in exposure to PCDDs and PCDFs were identified. High-exposure-potential jobs and areas were selected on the basis of data collected in the EPA/API study, knowledge of processes and jobs gained during site visits to several mills, and from environmental monitoring data. Input from the UPIU and Boise Cascade was utilized to confirm job categories and exposure classification. Similarly, long term workers who worked in jobs that were less likely to result in exposure to PCDDs and PCDFs were identified.

Two important considerations used for exposure classification of mill workers were job location and duration of employment in the job category of interest. Workers with the longest duration of employment in jobs involving contact with bleached pulp or paper and process water/effluent were expected to most likely have serum PCDDs/PCDFs levels above background. The high-exposure-potential group included workers with at least 10 years duration of employment in selected job categories. The low-exposure-potential group included workers with at least 10 years duration of employment in selected jobs that did not involve regular contact with bleached pulp/paper or effluent and less than one year in a high-exposure-potential area. Jobs classified as having high exposure potential involved frequent skin contact with bleached pulp, paper, or effluent, and those entailing potential inhalation of bleached paper dust or mists formed from process water and/or effluent. Mill locations considered to involve contact with bleached pulp or paper were: (a) the bleach plant, (b) the dry end of the paper machines, (c) rewind areas, (d) finishing areas, and (e) the effluent treatment plant. The potential for exposure was expected to be significantly less in the following work areas: (a) the groundwood mill/long log area, (b) the woodyard, and (c) the Kraft mill (those jobs that are strictly within the Kraft mill, and not in the bleach plant).

A community referent group of Rumford residents who had never worked at a pulp and paper mill was included as a comparison group. Because Rumford is a relatively isolated community located on a river with two nearby paper mills which are potential sources of PCDD/PCDF contamination, background levels in this community could be slightly elevated relative to the levels measured in other communities. For this reason, data from people who do not work at the mill was considered necessary to differentiate the relative contributions of occupational and non-occupational exposures.

Each worker who agreed to participate was asked to identify an individual close in age who lived in the community, but never worked in the mill. The workers and identified individuals were sent letters explaining the study and asking for their participation. Follow-up phone calls were made to discuss the study and, if the individual agreed to participate, schedule times for

interviews and collection of blood. The interviews and blood collection took place at the community hospital in Rumford.

### *Questionnaire*

Each study participant was interviewed about personal characteristics related to serum concentrations of PCDDs and PCDFs, such as age, cigarette smoking, height, and weight. (The latter two values were used to calculate body mass index.) In addition, participants were asked about exposures other than from work at the mill that may affect serum concentrations of PCDDs and PCDFs. Questions were asked about past work in jobs with potential PCDD exposure (including waste incineration, reclamation or hazardous waste work, work with transformers or capacitors, and herbicide manufacturing), military experience in Vietnam, consumption of fish caught in local rivers, use of herbicides at home, and use of pentachlorophenol to treat wood.

### *Serum Collection and Analyses*

Study participants each provided 250 milliliters of blood. Fasting was not required because of concern about the safety of workers who had to work before or after the blood draw. One previous study found no statistically significant differences between fasting and non-fasting serum levels of PCDDs and PCDFs.<sup>68</sup> To account for the possible effect of using non-fasting levels, however, the blood draw was scheduled so that workers and community residents were both tested throughout the day, and all results were adjusted for lipid content of the blood.<sup>69</sup> Serum was analyzed for PCDDs and PCDFs using HRGC/HRMS.<sup>70,71</sup> Each analytical run consisted of a method blank, three unknown samples, and a quality-control pool sample.

### *Statistical Analysis*

When analyzing biological monitoring data, some compounds are often undetected in many people. When this happens, it is assumed that the concentration is below the limit of detection (LOD) of the analytic methods. For analyses of these data, we calculated an imputed value when a compound was not detected.<sup>72</sup>

When the compound was not detected in more than 50% of the samples, the imputed value was one-half the minimum detectable concentration for the compound in the sample. This method was used for the following compounds:

tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD)  
pentachlorodibenzo-*p*-dioxin (1,2,3,7,8-PeCDD)  
hexachlorodibenzo-*p*-dioxin (1,2,3,4,7,8-HxCDD; 1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDD)  
heptachlorodibenzo-*p*-dioxin (1,2,3,4,6,7,9-HpCDD)  
octachlorodibenzo-*p*-dioxin (OCDD)  
pentachlorodibenzofuran (2,3,4,7,8-PeCDF)  
hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF; 1,2,3,6,7,8-HxCDF; 2,3,4,6,7,8-HxCDF)

heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF)

When the compound was detected in at least 50% of the samples, the imputed value is equal to the minimum detectable concentration for the compound divided by the square root of two. When the imputed value was less than the median value for each exposure group it was retained; if it was greater than the median, it was considered to be too imprecise and the median value is used. This method was used for the following compounds:

tetrachlorodibenzofuran (2,3,7,8-TCDF)  
pentachlorodibenzofuran (1,2,3,7,8-PeCDF)  
hexachlorodibenzofuran (1,2,3,7,8,9-HxCDF)  
heptachlorodibenzofuran (1,2,3,4,7,8,9-HpCDF)

To ensure that the imputation method did not artifactually impact on our findings, statistical analyses were repeated by replacing nondetectable values with zero. The conclusions were unchanged.

Differences between individual congeners in workers and community residents were examined. Because PCDD and PCDF levels were log-normally distributed, the median levels in the two groups are reported. The Wilcoxon rank-sum test was used to test the statistical significance of group differences.

In addition to conducting a statistical analysis for each congener individually, we also calculated the total I-TEQ. We looked at total I-TEQ and the portion of the I-TEQ contributed by PCDDs and PCDFs. Because the I-TEQ was log-normally distributed, the natural logarithm (ln) of the I-TEQ was used in the analysis. Multiple linear regression models were used to examine the relationship between the ln(I-TEQ) and exposure while controlling for the effects of potential confounders. Exposure was assessed as a categorical variable (high exposure potential, low exposure potential, community resident) and as a continuous variable (time spent in high exposure potential areas). Based on results from published studies and examination of the bivariate relationships in this study, we assessed the effects of age, body mass index, cigarette smoking status (current, former, never), and consumption of locally caught fish (dichotomized as ever yes or no). Main effects and all two-way interactions were considered. Terms remained in the model only if the level of statistical significance was  $< .10$ .



## **RESULTS**

### **Industrial Hygiene**

#### *PCDD/PCDF Air Sampling (August 1989)*

Although PCDDs and PCDFs had not previously been measured in air, there was concern that the respiratory route of exposure might be significant for workers who are exposed to bleached paper dust or other potentially contaminated particulate.<sup>73</sup> Low but detectable air levels of PCDD and PCDF were found during the initial survey conducted at this facility in 1989.<sup>74</sup> These results are shown in Table 2. 2,3,7,8-TCDD was not detected in any of the samples. Other tetrachlorinated dioxins were detected near the rewind operation of paper machine #7, but not in the other sampling locations. 2,3,7,8-TCDF and other tetra-chlorinated furans were found in all samples. Due to the low PCDD and PCDF air sample results from this survey and the expensive analytical method, only surface contamination samples were collected during the follow-up survey in August 1991.

Using the I-TEQ weighting technique, the mixture of isomers detected in the rewind area of paper machine #7 is considered equivalent to a concentration of 0.06 picograms of 2,3,7,8-TCDD per cubic meter of air ( $\text{pg}/\text{m}^3$ ). The highest I-TEQ concentration was detected in the hardwood bleach plant. As previously noted, NIOSH has not established an REL for PCDDs/PCDFs. However, the concentrations detected were well below the  $10 \text{ pg}/\text{m}^3$  exposure limits recommended by the National Research Council. (The analytical method available for PCDDs/PCDFs at the time these air samples were collected did not measure all congeners that contribute to the I-TEQ. Hence, the air sampling results reported may have slightly underestimated the actual I-TEQ.)

#### *PCDD/PCDF Wipe Sampling (August 1991)*

The results of the wipe samples collected August 9-11, 1991, are shown in Tables 3 through 6. As previously noted, analytical developments allowed for the identification and quantification of more specific PCDD/PCDF isomers than in the air samples. Tables 3 and 5 show the specific concentration in units of picogram per square meter ( $\text{pg}/\text{m}^2$ ) for each isomer detected, and the adjusted I-TEQ concentration after I-TEF weighting of the specific isomers. Tables 4 and 6 show the relative contribution (percent of total I-TEQ) of each specific isomer detected, and the relative rank of each isomer.

As shown in Tables 3 and 5, a wide variety of PCDDs/PCDFs were detected in the samples. All wipe sample results, however, were below the NAS guideline of  $25 \text{ ng}/\text{m}^2$  for total I-TEQ. The highest I-TEQ concentration ( $1049 \text{ pg}/\text{m}^2$ ) was detected on the sample collected at the back of the electrical control panel near the dry end of paper machine # 7. The lowest I-TEQ concentration ( $13 \text{ pg}/\text{m}^2$ ) was found on the sample collected from the lab bench in the softwood

bleach plant. 2,3,7,8-TCDD was found on three of the five wipe samples. No 2,3,7,8-TCDD was found on samples collected from lab benches in the hardwood or softwood bleach plants. . The compounds that contributed the most to the I-TEQ in the bleach plant were PCDFs, while in the paper mill it was the PCDDs that contributed the most to the I-TEQ. Furthermore, in all three of the bleach plant wipe samples, the % of I-TEQ relative rank for 2,3,4,7,8-PeCDF and 2,3,7,8-TCDF was 1 and 2, respectively. In the paper mill, hepta and octa PCDDs were well represented in the wipe sampling results. (Although the actual concentration of 2,3,7,8-TCDD was much less, it also contributed substantially to the I-TEQ since its I-TEF factor is one.)

*CVOCs (August 1991)*

Qualitative analysis of air samples collected in August 1991 at various locations in the bleach plant indicated the presence of chloroform, carbon tetrachloride, dichloro-bromo-methane, and chloro-dibromo-methane. PBZ sampling to quantitate exposure to these compounds is shown in Table 7. As noted in the table, both the hardwood and softwood bleach plant operators were exposed to chloroform concentrations exceeding the NIOSH REL of 10 mg/m<sup>3</sup> as a 60-minute STEL. PBZ chloroform concentrations in the bleach plant ranged from 1.3 mg/m<sup>3</sup> to 15.2 mg/m<sup>3</sup> with an average value of 6.2 mg/m<sup>3</sup>. No other halogenated organic compounds were detected on the PBZ samples.

Area monitoring at the wet end of several paper machines (Table 8) also detected chloroform at concentrations up to 1.1 mg/m<sup>3</sup>, indicating the potential for some low level exposure in those areas. To obtain information about the source of chlorinated organics, monitoring was conducted at the sampling ports on each of the bleach lines. The data collected from these "source" samplers, shown in Tables 9 (hardwood bleach plant) and 10 (softwood bleach plant), showed chloroform concentrations ranging from 1.8 to 116 mg/m<sup>3</sup>. Although many of the "source" samplers detected chloroform levels above the NIOSH REL, these data do not represent worker PBZ exposures since the workers are present at these locations infrequently. The source sampling data indicates chloroform and other chlorinated organics are released at each of the rinse stages, but that the hypochlorite stage is the strongest source. No significant difference was detected between the hardwood and softwood bleach lines.

*Follow-up Chloroform Monitoring (March 1992)*

After the August 1991 site visit, Boise Cascade management implemented process changes to substantially reduce the amount of hypochlorite used in the Hardwood Bleach Plant, and eliminate the use of hypochlorite in the softwood bleach plant. The use of hypochlorite in the bleaching stage is the largest source of chloroform emissions. On March 25-26, 1992, NIOSH investigators conducted a follow-up site visit to conduct personal and area monitoring for chloroform and other halogenated organics. The purpose of the follow-up visit was to assess the effectiveness of the process changes on chloroform generation.

PBZ samples were collected from workers in the same jobs that were monitored during the August 9-11, 1991, survey. The results of this monitoring, shown in Table 11, show that worker exposure to chloroform was significantly reduced when compared with the results of the previous monitoring. For all samples, the results were either less than the analytical limit of detection (LOD) or between the LOD and the limit of quantification (LOQ). All were well below the NIOSH REL for chloroform. Three PBZ samples (Chemical Unloader, Hardwood Operator, Head Bleacher) had detectable concentrations of 1,1,1-trichloroethane. The highest concentration of 1,1,1-trichloroethane detected ( $4.8 \text{ mg/m}^3$ ) was well below the NIOSH REL of  $1910 \text{ mg/m}^3$  as a 15-minute limit.

Area air samples were collected at both the hardwood and softwood bleach plant, and at several paper machines. The results of this monitoring are shown in Tables 12 (hardwood bleach plant), 13 (softwood bleach plant), and 14 (paper machines). As shown in Table 12, chloroform was detected at two hypochlorite rinse tanks in the hardwood bleach plant, although the concentrations detected were much lower than those found prior to the process changes. No chloroform was detected (concentrations were below the analytical limit of detection) in the softwood bleach plant or at the paper machine. During the March 1992 follow-up survey some process interruption occurred. However, lower chloroform results were found during periods when the pulp and paper plants were operating normally.

#### *Paper Dust (August 1991)*

The results of the PBZ monitoring for total dust are presented in Table 15. As shown in this table, only low levels of dust were detected on the samples. All results were well below the NIOSH REL for cellulose. The NIOSH REL, however, would not be applicable if PCDDs, PCDFs, or other contaminants were present in the dust. The area sampling results, shown in Table 16, also indicate dust levels were low in most areas. The highest concentrations were detected at the dry ends of older paper machines (#7 and #4), where slitters are used to cut the initial large roll into a series of smaller rolls.

#### *Workplace Observations*

Dermal contact to wet bleached pulp can occur while collecting process control samples, correcting process disruptions, cleaning up spills and process leaks, as well as during maintenance activities. Dermal contact to bleached pulp that is contaminated with PCDDs/PCDFs increases the potential to absorb these compounds directly through the skin or by ingestion. During the survey, it was also observed that food consumption, as well as cigarette smoking was occurring in work areas. This practice is a potential exposure pathway when conducted in areas where toxic material is present.

## **Medical**

Based on seniority lists in selected areas of the mill, 76 workers were identified as potential study subjects. We were successful in contacting 68 of these workers by phone; 10 refused to participate in the study. Forty-six of the 58 workers with the highest seniority were selected for the study. Thirty-two workers worked for 10 or more years in high-exposure-potential areas. Seven of these no longer worked in these areas at the time of the study. Fourteen workers worked 10 or more years in low-exposure-potential areas. Seven of these workers also had been in high-exposure-potential areas for one to eight years. (These seven workers were considered to have mixed exposure potential.) Twenty-three community workers identified by participating worker were contacted. Twelve agreed and seven refused to participate, and four were excluded because of prior work at a mill. Four replacements were identified by workers or the union; all participated.

Table 17 compares mill workers and community residents with respect to the relevant covariates. All study participants were white males. As a group, community residents were somewhat younger and leaner than mill workers. Fewer community residents currently or ever smoked cigarettes. The proportion of persons who drank more than one alcoholic beverage per week at the time of the study was similar in the two groups. None of the community residents and only two mill workers were Vietnam veterans. Neither Vietnam veteran reported assignment to the Air Force Ranch Hand unit or the Army Chemical Corps, units that potentially handled dioxin-containing compounds. Community residents were less likely than mill workers to have ever eaten fish caught in local rivers, to have ever applied weed or brush killer outside work, or to have lived in a house heated by wood or coal, though the differences were small.

Eight PCDDs and 10 PCDFs were found in the sera of study participants (Table 18). The percentage of samples with values below the limit of detection ranged from 0 to 26 for the PCDDs and from 2 to 98 for the PCDFs. For four of the 10 PCDFs, more than 50% of the persons tested had levels below the limit of detection. Because there were analytical problems for 95% of the samples of OCDF analyses, data for this congener are not included in subsequent findings.

Overall, there were no appreciable differences among the three exposure groups in the median values for specific PCDDs or PCDFs (Tables 19 and 20). The median value in high exposure potential workers was greater than in community residents for four of the eight PCDDs --2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,7,8,9-HxCDD, and 1,2,3,4,6,7,9-HpCDD-- and four of the nine PCDFs --1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 2,3,4,6,7,8-HxCDF, and 1,2,3,4,6,7,8-HpCDF. The relative differences for 2,3,7,8-TCDD (6%) and 1,2,3,7,8-PeCDD (2%) were very small and probably not meaningful ( $p=0.65$  and  $0.44$ , respectively). There was a 20% difference for 1,2,3,7,8,9-HxCDD ( $p=0.36$ ) and a 35% difference for 1,2,3,4,6,7,8-HpCDD ( $p=.14$ ), but workers with low exposure potential had higher medians than workers with high exposure potential. The relative differences for 1,2,3,4,7,8-HxCDF (6%) and 1,2,3,6,7,8-HxCDF (2%) were also very small and probably not meaningful ( $p=0.55$  and  $0.50$ , respectively). There was an

18% difference for 2,3,4,6,7,8-HxCDF ( $p=0.77$ ) and a 12% difference for 1,2,3,4,6,7,8-HpCDF ( $p=0.88$ ), but workers with low exposure potential had a similar or higher median than workers with high exposure potential.

For all PCDDs and PCDFs except 2,3,7,8-TCDD and 1,2,3,7,8,9-HxCDD, high exposure potential workers no longer in these jobs had slightly higher levels than those whose presumed exposure was current. PCDD and PCDF levels in workers with mixed exposure did not consistently follow a pattern suggestive of occupational exposure (i.e., intermediate between those with high and those with low exposure potential).

The highest values for specific PCDDs and PCDFs consistently occurred in seven workers. These individuals ranged in age from 37 to 64 and had worked in the mill for 10 to over 40 years in a variety of jobs. One had a job involving exposure to bleached paper dust in the finishing department for four years and to effluent for five years. Five had jobs involving exposure to bleached paper dust at the paper machines for six to 44 years. One never worked in an area with high exposure potential. Six of the seven had potential exposure outside work that could have contributed to these findings. Six ate locally caught fish, four had lived in a home heated with wood, two had applied weed killer at home, and one had used pentachlorophenol to treat wood.

Both the median and maximum values of the total I-TEQ were higher in workers in the low-exposure-potential group than in workers in the high-potential-exposure group or community residents (Table 21). In all instances, the lowest value for the median I-TEQ occurred in workers in the high-exposure-potential group. The relative differences between the highest and the other median values, however, were small (11% to 26%,  $0.32 \leq p \leq 0.96$ .) Two workers had considerably higher values than other workers or community residents (Figure 1). Both worked in the wood yard for more than 15 years. When these workers are excluded from the I-TEQ comparisons, the median and maximum values are decreased slightly for low exposure potential workers, but the pattern of the results remain unchanged.

None of the exposure variables (worker with low exposure potential, worker with high exposure potential, years in the mill, years in high exposure potential areas) remained in the final multivariate regression model for  $\ln(\text{I-TEQ})$  (Table 22).  $\ln(\text{I-TEQ})$  was positively related to age ( $p=0.001$ ), body mass index ( $p=0.01$ ), and the consumption of locally caught fish ( $p=0.02$ ), and was negatively related to current cigarette smoking ( $p=0.05$ ). Age, total duration in the mill, and duration in high exposure potential areas, however, were highly correlated. To clarify whether age or duration was actually more important, we examined the correlation between  $\ln(\text{I-TEQ})$  and duration in high exposure potential areas among workers stratified by age (30-39, 40-49, 50-59 years) in workers. For each age group, the correlation was small in magnitude and negative in direction ( $r=-.07$ ,  $-.21$  and  $-.37$ , respectively).

## **DISCUSSION AND CONCLUSIONS**

Industrial hygiene data collected at the Rumford mill indicates the potential for worker exposure to PCDDs, PCDFs, chloroform, and possibly other chlorinated organics. Although only limited environmental monitoring data were collected, the sampling results suggest that airborne concentrations of PCDDs and PCDFs are below the National Research Council guidelines. However, the air sampling data can not be used as an indicator of potential dermal exposure or to evaluate the significance of past exposures. Initial air sampling data indicated that workers were potentially exposed to chloroform levels exceeding NIOSH exposure criteria. However, process modifications to eliminate hypochlorite in the softwood bleach plant and to reduce hypochlorite in the hardwood bleach line resulted in a reduction in chloroform concentrations to well below the NIOSH REL. Exposure to paper dust was found to be well below NIOSH exposure criteria for cellulose. As previously noted, however, the NIOSH REL is not intended to address the issue of contaminants, such as PCDD and PCDF, that may be present in the dust.

The differences in serum levels of PCDDs and PCDFs between mill workers and community residents were small and probably not meaningful. These findings are consistent with those of Rosenberg et al.,<sup>75</sup> who did not find any statistically significant differences in PCDD or PCDF concentrations in blood plasma between unexposed workers and potentially exposed workers in the bleach plant or the paper mill of a Finnish pulp and paper mill.

The individuals who consistently had the highest serum levels of PCDDs and PCDFs were mill workers, suggesting a possible effect of occupational exposure. Five of these seven workers had a job at some time in the past at the paper machines. All but one of the seven workers, however, also had nonoccupational exposure to materials potentially contaminated with PCDDs or PCDFs. The finding that the two highest values for I-TEQ occurred in workers with jobs in the wood yard was surprising, as this was initially considered to be a low exposure potential area. Recent information obtained from Boise Cascade suggests that there was some potential for occasional exposure to bleach plant effluent used in a log flume to aid in moving logs from the wood pile to a saw, and then to a debarker. Additionally, the emission stack from the refuse burner, another potential source of PCDD/PCDF exposure, was lower in the past than now and the predominant wind direction is over the wood yard. It is not possible to determine, however, how much these potential exposures may have contributed to serum PCDD/PCDF levels in wood yard workers.

Long-term Boise Cascade workers in this study currently do not have high body burdens of PCDDs or PCDFs. Current levels, however, may not accurately describe maximum exposure levels in the past, particularly if exposure has declined over time. The possibility of previously higher exposure is suggested by the finding that workers whose exposure was not current had slightly higher levels of PCDDs and PCDFs than workers with current exposure. PCDDs and PCDFs in blood of exposed and comparison populations have been measured in studies of other occupational groups,<sup>76,77,78,79,80</sup> veterans exposed to Agent Orange<sup>81,82,83</sup> and in the general population.<sup>84,85</sup> Although there are some difficulties in comparing results among studies, the

findings from this study indicate that both workers and community residents had serum PCDD and PCDF levels within the ranges reported for unexposed groups in most other studies.

### **RECOMMENDATIONS**

1. All workers who may be dermally exposed to materials contaminated with significant levels of PCDDs/PCDFs should be provided with appropriate chemical protective clothing and equipment to minimize skin contact.
2. Food and beverage consumption and cigarette smoking should be restricted to designated areas isolated from potential exposure areas. Workers should wash their hands before handling food, food or beverage containers, cigarettes, or eating utensils.
3. Periodic industrial hygiene assessments should be conducted to evaluate worker exposures and assess the impact of process modifications on contaminant generation.

**REFERENCES**

1. U.S. Environmental Protection Agency [1986]. National Dioxin Study: Draft Report to Congress.
2. U.S. Environmental Protection Agency [1988]. U.S. EPA - Paper Industry Cooperative Dioxin Screening Study. EPA-440/1-88-025.
3. Suntio LR, Shiu WY, Mackay D [1988]. A review of the nature and properties of chemicals present in pulp mill effluents. *Chemosphere* 17(17):1249-1290 .
4. CDC [1994]. Pocket guide to chemical hazards. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication No. 94-116.
5. Code of Federal Regulations [1989]. 29 CFR 1910.1000. Washington, DC: U.S. Government Printing Office, Federal Register.
6. ACGIH [1994]. Threshold limit values and biological exposure indices for 1994-1995. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
7. ACGIH [1992]. Documentation for threshold limit values and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
8. Goldstein JA [1979]. The structure-activity relationships of halogenated biphenyls as enzyme inducers. *Ann NY Acad Sci* 320:164-78.
9. Youshimura H, Youshihara S, Ozawa N, Miki M [1979]. Possible correlation between induction modes of hepatic enzymes by PCBs and their toxicity in rats. *Ann NY Acad Sci* 320:179-92.
10. Poland A, Greenlee W, Kende AS [1979]. Studies on the mechanism of action of the chlorinated dibenzo-*p*-dioxins and related compounds. *Ann NY Acad Sci* 320:214-30.
11. McConnell EE, Moore JA, Haseman JK, Harris MW [1978]. The comparative toxicity of chlorinated dibenzo-*p*-dioxins in mice and guinea pigs. *Toxicol Appl Pharmacol* 44:335-56.
12. Nagayama J, Kuroki H, Masuda Y, Kuratsune M [1983]. A comparative study of polychlorinated dibenzofurans, polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on aryl hydrocarbon hydroxylase inducing potency in rats. *Arch Toxicol* 53:177-84.



13. Yoshihara S, Nagata K, Youshimura H, et al. [1981]. Inductive effect on hepatic enzymes and acute toxicity of individual polychlorinated dibenzofuran congeners in rats. *Toxicol Appl Pharmacol* 59:580-8.
14. Moore JA, McConnell EE, Dalgard DW, Harris MW [1979]. Comparative toxicity of three halogenated dibenzofurans in guinea pigs, mice, and rhesus monkeys. *Ann NY Acad Sci* 320:151-63.
15. Schwetz BA, Norris JM, Sparschu GL, et al [1973]. Toxicology of chlorinated dibenzo-*p*-dioxins. *Environ Health Perspect* 5:87-99.
16. McConnell EE, Moore JA, Dalgard DW [1978]. Toxicity of 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin in rhesus monkeys (*Macaca mulatta*) following a single oral dose. *Toxicol Appl Pharmacol* 43:175-87.
17. Garthoff LH, Cerra FE, Marks EM [1981]. Blood chemistry alterations in rats after single multiple gavage administration of polychlorinated biphenyl. *Toxicol Appl Pharmacol* 60:33-44.
18. Moore JA, Gupta BN, Vos JG [1976]. Toxicity of 2,3,7,8-tetrachlorodibenzo-furan - preliminary results. In: *Proceedings of the National Conference on Polychlorinated Biphenyls*. Chicago, IL: Environmental Protection Agency, Office of Toxic Substances, EPA-560/6-75-004, pp. 77-80.
19. NIOSH [1984]. Current intelligence bulletin 40 - 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Cincinnati, OH: National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 84-104.
20. Hassoun E, d'Argy R, Deneker L [1984]. Teratogenicity of 2,3,7,8-tetrachloro-dibenzofuran in the mouse. *J Toxicol Environ Health* 14:337-51.
21. Kociba RJ, Keyes DG, Beyer JE, et al [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.
22. NTP [1982]. Bioassay of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin for possible carcinogenicity (gavage study). Bethesda, MD and Research Triangle Park, NC: National Toxicology Program, DHHS (NIH) Publication No. 82-1765.
23. NTP [1980]. Bioassay of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin for possible carcinogenicity (gavage study). Bethesda, MD and Research Triangle Park, NC: National Toxicology Program, DHHS (NIH) Publication No. 80-1758.
24. Fingerhut MA, Halperin WE, Marlow DA, et al. [1991]. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *New Engl J Med* 324:212-218.

25. Manz A, Berger J, Dwyer JH, et al. [1991]. Cancer mortality among workers in chemical plant contaminated with dioxin. *Lancet* 338:959-964.
26. Zober A, Messerer P, Huber P [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* 62:138-157.
27. Wingren G, Persson B, Thoren K, Axelson O [1991]. Mortality pattern among pulp and paper mill workers in Sweden: a case-referent study. *Am J Industr Med* 20:769-774.
28. Henneberger PK, Ferris BG, Monson RR [1989]. Mortality among pulp and paper workers in Berlin, New Hampshire. *Brit J Industr Med* 46:658-664.
29. Milham S, Demers RY [1984]. Mortality among pulp and paper workers. *J Occ Med* 26:844-846.
30. Schwartz E [1988]. A proportionate mortality ratio analysis of pulp and paper mill workers in New Hampshire. *Brit J Industr Med* 45: 234-238.
31. Robinson CF, Waxweiler RJ, Fowler DP [1986]. Mortality among production workers in pulp and paper mills. *Scand J Work Environ Health* 12:552-560.
32. Hardell L, Sandstrom A [1979]. Case-control study: soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. *Br J Cancer* 39: 711-717.
33. Hardell L, Eriksson M [1988]. The association between soft tissue sarcomas and exposure to phenoxyacetic acids: a new case-referent study. *Cancer* 62:652-656.
34. Hardell L, Eriksson M, Lenner P, et al. [1981]. Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case-control study. *Br J Cancer* 43:169-176.
35. Eriksson M, Hardell L, Adami HO [1990]. Exposure to dioxins as a risk factor for soft tissue sarcoma: a population-based case-control study. *J Natl Cancer Inst* 82:486-490.
36. Smith AH, Pearce NE, Fisher DO, et al. [1984]. Soft tissue sarcoma and exposure to phenoxyherbicides and chlorophenols in New Zealand. *J Natl Cancer Inst* 73:1111-1117.
37. Smith AH, Pearce NE [1986]. Update on soft tissue sarcoma and phenoxyherbicides in New Zealand. *Chemosphere* 15:1795-1798.
38. Pearce NE, Smith AH, Howard JK, et al. [1986]. Non-Hodgkin's lymphoma and exposure to phenoxyherbicides, chlorophenol, fencing work, and meat works employment: a case-control study. *Br J Ind Med* 43:75-83.

39. Bertazzi PA, Zocchetti C, Pesatori AC, et al. [1989]. Ten-year mortality study of the population involved in the Seveso incident in 1976. *Am J Epidemiol* 129:1187-1200.
40. Ideo G, Bellati G, Bellobuono A, Bissanti L [1985]. Urinary D-glucaric acid excretion in the Seveso area, polluted by tetrachlorodibenzo-*p*-dioxin (TCDD): five years of experience. *Environ Health Perspect* 60:151-157.
41. Calvert GM, Hornung RW, Sweeney MH, et al. [1992]. Hepatic and gastrointestinal effects in an occupational cohort exposed to 2,3,7,8-tetrachlorodibenzo-*para*-dioxin. *JAMA* 267:2209-2214.
42. Roegner RH, Grubbs WD, Lustik MB, et al. [1991]. Air Force Health Study: an epidemiologic investigation of health effects in Air Force personnel following exposure to herbicides. Serum dioxin analysis of 1987 examination results. NTIS# AD A-237-516 through AD A-237-524.
43. Calvert GM, Sweeney MH, Morris JA, et al. [1991]. Evaluation of chronic bronchitis, chronic obstructive pulmonary disease (COPD) and ventilatory function among workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). *Am Rev Respir Dis* 144:1302-1306.
44. Sweeney MH, Fingerhut MA, Arezzo JC, et al. [1993]. Peripheral neuropathy after occupational exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). *Am J Ind Med* 23:845-858.
45. Alderfer R, Sweeney M, Fingerhut M, et al. [1992]. Measures of depressed mood in workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Chemosphere* 25:247-250.
46. Sweeney MH, Hornung RW, Wall DK, et al. [1992]. Prevalence of diabetes and increased fasting serum glucose in workers with long-term exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Presented at: 12th International Symposium on Dioxins and Related Compounds; August 24-28, Tempere, Finland.
47. Egeland GM, Sweeney MH, Fingerhut MA, et al. [1994]. Total serum testosterone and gonadotropins in workers exposed to dioxin. *Am J Epidemiol* 139:272-281.
48. Eadon G, Aldouos K, Frenkel G, et al. [1982]. Comparisons of chemical and biological data on soot samples from the Binghamton State Office building. Albany, NY: Center for Laboratories and Research, New York State Department of Health.
49. Environmental Protection Agency [1987]. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-*p*-dioxins and dibenzofurans (CDDs and CDFs). United States Environmental Protection Agency, EPA 625/3-87/012.

50. Environmental Protection Agency [1989]. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs) - 1989 update. United States Environmental Protection Agency, EPA 625/3-89/016.
51. NRC [1988]. Acceptable levels of dioxin contamination in an office building following a transformer fire. Washington, DC: National Academy Press..
52. NIOSH [1976]. Criteria for a recommended standard: occupational exposure to chloroform. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 75-114.
53. Hathaway GJ, Proctor NH, Hughes JP, Fischman MF [1991]. Chemical hazards of the workplace, 3rd ed. New York: Van Nostrand Reinhold Company.
54. NIOSH [1977]. Occupational diseases: a guide to their recognition. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 77-181.
55. NIOSH [1978]. Occupational safety and health guidelines for chemical hazards. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 81-123.
56. NTP [1994]. Seventh annual report on carcinogens: 1994 summary. Research Triangle Park, NC: U.S. Department of Health and Human Services, National Toxicology Program.
57. IARC [1982]. IARC monographs on the evaluation of the carcinogenic risks of chemicals to humans, supplement 4. Lyon, France: International Agency for Research on Cancer.
58. NIOSH [1992]. Recommendations for occupational safety and health: compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92-100.
59. Glenn RE, Craft BF [1986]. Air sampling for particulates. In: Occupational respiratory diseases. Merchant JE, ed. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 86-102.

60. Kelada FS [1990]. Occupational intake by dermal exposure to polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans in the pulp mill industry. *Am Industr Hyg Assoc J* 51:519-521.
61. Sullivan MJ, Fisher RP, Gillespie WJ [1989]. Risks associated with potential dioxin exposure through inhalation of paper dust. *Chemosphere* 19:869-872.
62. Smith RM, O'Keefe PW, Hilker DR, Aldous KM [1986]. *Analytical Chemistry*, 58(12): 2414-2420.
63. U.S. Environmental Protection Agency [1984]. Compendium of methods for the determination of toxic organic chemicals in ambient air (method T09). U.S. EPA #600/4-84-027.
64. Hudson J L, Moray DA [1989]. Evaluation of method performance for measuring 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in ambient air. *Chemosphere* 18(1-6):141-148.
65. NIOSH [1986]. Health hazard evaluation report: Staniford Street Office Building, Boston Massachusetts. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 86-092-1870.
66. NIOSH [1984]. NIOSH manual of analytical methods, third edition, method 1003. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health.
67. National Institute for Occupational Safety and Health [1984]. NIOSH manual of analytical methods, third edition, method 0500. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health.
68. Hansson M, Rappe C, Gochfeld M, et al. [1989]. Effects of fasting on blood levels of 2,3,7,8-TCDD and related compounds. *Chemosphere* 18:525-530.
69. Akins JR, Waldrep K, Bernert JT [1989]. The estimation of total serum lipids by a completely enzymatic summation method. *Clin Chem Acta* 184:219-226.
70. Patterson DG Jr, Hampton I, Lapeza CR Jr, et al. [1987]. High resolution gas chromatographic high resolution mass spectrometric analysis of human serum on a whole weight and lipid bases for 2,3,7,8,-tetrachlorodibenzo-*p*-dioxin. *Anal Chem* 59:2000-2005.

71. Patterson DG Jr, Holler JS, Lapeza DR Jr, et al. [1986]. High-resolution gas chromatography/high-resolution mass spectrometric analysis of human adipose tissue for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Anal Chem* 58:705-713.
72. Hornung RW, Reed LD [1990]. Estimation of average concentration in the presence of nondetectable values. *Appl Occ Environ Hyg* 5:46-51.
73. NCASI [1988]. Risks associated with dioxin exposure through inhalation of paper dust in the workplace. NCASI Technical Bulletin No. 537.
74. NIOSH [1993]. Health hazard evaluation, Boise Cascade, Rumford, ME. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, NIOSH HHE 89-326.
75. Rosenberg C, Kintsas H, Tornaues J, et al. [1994]. PCDD/PCDF levels in the blood of workers in a pulp and paper mill. *Organohalogen Compounds* 21:101-104, Kyoto University, Kyoto, 606-01, Japan.
76. Hesso A, Maneila M, Tornaues J, et al. [1992]. Polychlorinated dioxins, furans and non-ortho polychlorinated biphenyls in blood of exposed laboratory personnel. *Chemosphere* 25:1053-1059.
77. Papke O, Ball M, Lis A [1992]. Various PCDD/PCDF patterns in human blood resulting from different occupational exposures. *Chemosphere* 25:1101-1108.
78. Patterson DG Jr, Fingerhut MA, Roberts DW, et al. [1989]: Levels of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Am J Industr Med* 16:135-146.
79. Piacitelli LA, Sweeney MH, Fingerhut MA, et al. [1992]. Serum levels of PCDDs and PCDFs among workers exposed to 2,3,7,8-TCDD contaminated chemicals. *Chemosphere* 25:251-254.
80. Smith AH, Patterson DG, Warner ML, et al. [1992]. Serum 2,3,7,8-tetrachlorodibenzo-*p*-dioxin levels of New Zealand pesticide applicators and their implication for cancer hypotheses. *J Natl Cancer Inst* 84:104-108.
81. Kahn PC, Gochfeld M, Nygren M, et al. [1988]. Dioxins and dibenzofurans in blood and adipose tissue of Agent Orange-exposed Vietnam veterans and matched controls. *J Amer Med Assoc* 259:1661-1667.

82. Schechter A, Constable J, Bangert JV, et al. [1989]. Isomer specific measurement of polychlorinated dibenzodioxin and dibenzofuran isomers in human blood from American Vietnam veterans two decades after exposure to Agent Orange. *Chemosphere* 18:531-538.
83. Schechter A, McGee H, Stanley J, Boggess K [1992]. Dioxin, dibenzofuran, and PCD, including coplanar PCD levels in the blood of Vietnam veterans in the Michigan Agent Orange study. *Chemosphere* 25:205-208.
84. Papke O, Ball M, Lis A [1994]. PCDD/PCDF in humans, a 1993-update of background data. *Chemosphere* 29:2355-2360.
85. Patterson DG Jr, Todd GD, Turner WE, et al. [1994]. Levels of non-*ortho*-substituted (coplanar), mono- and di-*ortho*-substituted polychlorinated biphenyls, dibenzo-*p*-dioxins, and dibenzofurans in human serum and adipose tissue. *Environmental Health Perspectives* 102 (Suppl 1):195-204.

**AUTHORSHIP AND ACKNOWLEDGMENTS**

Evaluation Conducted and  
Report Prepared By:

Robert Mouradian, PhD, Industrial Hygienist  
Kevin Hanley, MSPH, CIH, Industrial Hygienist  
Industrial Hygiene Section  
Hazard Evaluations and Technical  
Assistance Branch (HETAB)  
Division of Surveillance, Hazard Evaluations, and  
Technical Assistance (DSHEFS)  
National Institute for Occupational Safety and  
Health (NIOSH)

Allison Tepper, PhD, Supervisory Epidemiologist  
Susan Burt, MSc, MSN, Senior Research Officer  
Medical Section, HETAB, DSHEFS, NIOSH

Max Kiefer, MS, CIH  
Industrial Hygienist, Atlanta Regional Office,  
HETAB, NIOSH

Field Assistance Provided by:

Michael Barsan  
Rick Rinehart, MS  
Industrial Hygiene Section, HETAB, DSHEFS,  
NIOSH

Patricia McGraw  
Robert Schutte  
Medical Section, HETAB, DSHEFS, NIOSH

BJ Haussler  
Support Services Branch, DSHEFS, NIOSH

Charles Dodson  
National Center for Environmental Health

Report Formatted by:

Tamme Ottlinger, Office Automation Assistant  
Medical Section, HETAB, DSHEFS, NIOSH



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Laboratory Support:

Staff  
Measurements Research Support Branch  
Division of Physical Sciences and Engineering  
NIOSH

Wayman Turner  
Louis Alexander  
National Center for Environmental Health

Originating Office:

HETAB, DSHEFS, NIOSH

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1. United Paperworkers International Union
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3. Department of Labor/OSHA Region I

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

**Table 1**  
**2,3,7,8-TCDD Toxicity Equivalency Factors**  
**Boise Cascade: Rumford, Maine**  
**HETA 88-0140-2517**

Compound	Toxic Equivalency Factors (TEFs)/89 <sup>50</sup>
Mono-,Di-, and TriCDDs	0
2,3,7,8-TCDD	1
Other TCDDs	0
2,3,7,8-PeCDD	0.5
Other PeCDDs	0
2,3,7,8-HxCDDs	0.1
Other HxCDDs	0
2,3,7,8-HpCDDs	0.01
Other HpCDDs	0
OCDD	0.001
Mono-,Di-, and TriCDFs	0
2,3,7,8-TCDF	0.1
Other TCDFs	0
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
Other PeCDFs	0
2,3,7,8-HxCDFs	0.1
Other HxCDFs	0
2,3,7,8-HpCDFs	0.01
Other HpCDFs	0
OCDF	0.001

TCDD-tetra chlorinated dibenzo-*p*-dioxin

CDD-chlorinated dibenzo-*p*-dioxin

CDF-chlorinated dibenzofurans

Pe-penta

Hx-hexa

Hp-hepta

O-octa

**Table 2  
Airborne Dioxin and Furan Sampling Results  
Boise Cascade: Rumford, Maine  
August 9-11, 1989  
HETA 88-0140-2517**

<b>Sample Location</b>	<b>2378 TCDD</b>	<b>Other TCDDs</b>	<b>2378 TCDF</b>	<b>Other TCDFs</b>	<b>Total I-TEQ</b>
#7 Rewind (Hardwood)	<0.16	0.55	0.49	0.36	0.05
Hardwood Bleachery	<0.06	<0.06	0.63	2.75	0.06
Softwood Bleachery	<0.11	<0.11	0.27	0.21	0.03
#44 Rewind (Coated)	<0.10	<0.10	0.10	0.10	0.01
Effluent Plant	<0.06	<0.06	0.08	0.08	0.01

Values marked as "<" were below the method's limit of detection, and were assumed to be zero when calculating I-TEQs (toxic equivalents).  
All values are reported in picograms of contaminant per cubic meter of air.  
I-TEQs were calculated using the International-89 toxicity equivalent factors.

**Table 3**  
**Wipe Sample Concentrations For Chlorinated Dioxins and Furans**  
**Bleach Plant, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Compound	Hardwood Cl, Rinse		Hardwood Lab Bench		Softwood Lab Bench	
	<i>Conc</i>	<i>I-TEQ Conc</i>	<i>Conc</i>	<i>I-TEQ Conc</i>	<i>Conc</i>	<i>I-TEQ Conc</i>
2,3,7,8-TCDD	21.1	21.1	<7.2	0	<5.6	0
Other TCDDs	152	0	7.1	0	<4.8	0
1,2,3,7,8-PeCDD	<42	0	<11	0	<2.7	0
Other PeCDDs	<42	0	25.6	0	<9.3	0
1,2,3,4,7,8-HxCDD	<77	0	<14	0	<3.4	0
1,2,3,6,7,8-HxCDD	<45	0	<11	0	6.5	.65
1,2,3,7,8,9-HxCDD	<64	0	<13	0	<3.2	0
Other HxCDDs	147	0	54	0	141	0
1,2,3,4,6,7,8-HpCDD	219	2.19	125	1.25	80.0	.8
Other HpCDDs	429	0	231	0	153	0
OCDD	1660	1.66	912	.91	417	.42
2,3,7,8-TCDF	1340	134	60.5	6.05	30.8	3.08
Other TCDFs	6640	0	272	0	104	0
1,2,3,7,8-PeCDF	505	25.2	17.1	.86	7.1	.36
2,3,4,7,8-PeCDF	464	232	20.0	10.0	9.5	4.75
Other PeCDFs	5190	0	169	0	53.5	0
1,2,3,4,7,8-HxCDFs	1260	126	18.5	1.85	<10	0
1,2,3,6,7,8-HxCDFs	413	41.3	<8.0	0	6.8	.68
2,3,4,6,7,8-HxCDFs	186	18.6	<9.3	0	15.6	1.56
1,2,3,7,8,9-HxCDFs	177	17.7	<11	0	<2.6	0
Other HxCDFs	3320	0	74.3	0	69.5	0
1,2,3,4,6,7,8-HpCDFs	1310	13.1	48.9	.49	40.8	.41
1,2,3,4,7,8,9-HpCDFs	1260	12.6	<26	0	<11	0
Other HpCDFs	3840	0	116	0	69.5	0
OCDF	5290	5.29	109	.11	55.4	.055
Total I-TEQ (I-89)		651		22		13

All values are reported in picograms of contaminant per square meter.

**Table 4**  
**Percent of I-TEQ and Relative Rank of Specific Chlorinated Dioxins**  
**Furans Measured in Wipe Samples**  
**Bleach Plant, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Compound	Hardwood Cl <sub>2</sub> Rinse		Hardwood Lab Bench		Softwood Lab Bench	
	% of I-TEQ	Relative Rank	% of I-TEQ	Relative Rank	% of I-TEQ	Relative Rank
2,3,7,8-TCDD	3.2	6	0	-	0	-
Other TCDDs	0	-	0	-	0	-
1,2,3,7,8-PeCDD	0	-	0	-	0	-
Other PeCDDs	0	-	0	-	0	-
1,2,3,4,7,8-HxCDDs	0	-	0	-	0	-
1,2,3,6,7,8-HxCDDs	0	-	0	-	5.1	6
1,2,3,7,8,9-HxCDDs	0	-	0	-	0	-
Other HxCDDs	0	-	0	-	0	-
1,2,3,4,6,7,8-HpCDDs	0.33	12	5.8	4	6.3	4
Other HpCDDs	0	-	0	-	0	-
OCDD	0.26	13	4.2	5	3.3	7
2,3,7,8-TCDF	20.6	2	28.1	2	24.1	2
Other TCDFs	0	-	0	-	0	-
1,2,3,7,8-PeCDF	3.9	5	4.0	6	2.8	9
2,3,4,7,8-PeCDF	35.6	1	46.5	1	37.2	1
Other PeCDFs	0	-	0	-	0	-
1,2,3,4,7,8-HxCDFs	19.4	3	8.6	3	0	-
1,2,3,6,7,8-HxCDFs	6.3	4	0	-	5.3	5
2,3,4,6,7,8-HxCDFs	2.9	7	0	-	12.2	3
1,2,3,7,8,9-HxCDFs	2.7	8	0	-	0	-
Other HxCDFs	0	-	0	-	0	-
1,2,3,4,6,7,8-HpCDFs	2.0	9	2.3	7	3.2	8
1,2,3,4,7,8,9-HpCDFs	1.9	10	0	-	0	-
Other HpCDFs	0	-	0	-	0	-
OCDF	0.8	11	0.5	8	0.4	10

**Table 5**  
**Analysis Of Wipe Samples For Chlorinated Dioxins and Furans**  
**Paper Mill, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Compound	Wall Near Wet End		Control Panel Near Dry End	
	Conc.	I-TEQ Conc.	Conc.	I-TEQ Conc.
2,3,7,8-TCDD	31.1	31.1	32.8	32.8
Other TCDDs	75.2	0	73.1	0
1,2,3,7,8-PeCDD	<20	0	<19	0
Other PeCDDs	42.6	0	115	0
1,2,3,4,7,8-HxCDDs	<31	0	59.7	5.97
1,2,3,6,7,8-HxCDDs	55.5	5.55	1050	105
1,2,3,7,8,9-HxCDDs	19.5	1.95	<219	0
Other HxCDDs	301	0	11700	0
1,2,3,4,6,7,8-HpCDDs	496	4.96	72400	724
Other HpCDDs	1010	0	116000	0
OCDD	5190	5.19	139000	139
2,3,7,8-TCDF	195	19.5	188	18.8
Other TCDFs	615	0	500	0
1,2,3,7,8-PeCDF	31.2	1.56	<10	0
2,3,4,7,8-PeCDF	19.9	9.95	11.2	5.6
Other PeCDFs	202	0	138	0
1,2,3,4,7,8-HxCDFs	26.7	2.67	29.6	2.96
1,2,3,6,7,8-HxCDFs	10.2	1.02	<13	0
2,3,4,6,7,8-HxCDFs	12.6	1.26	24.2	2.42
1,2,3,7,8,9-HxCDFs	<19	0	<19	0
Other-HxCDFs	321	0	893	0
1,2,3,4,6,7,8-HpCDFs	111	1.11	854	8.54
1,2,3,4,7,8,9-HpCDFs	<30	0	56.3	.56
Total-HpCDFs	278	0	4070	0
OCDFs	136	.14	3650	3.65
Total I-TEQ (I-89)		86		1049

All values are reported in picograms of contaminant per square meter.

**Table 6**  
**Percent of I-TEQ and Relative Rank Of Specific Chlorinated Dioxins and Furans**  
**Measured In Wipe Samples**  
**Paper Mill, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Compound	Wall Near Wet End		Control Panel Near Dry End	
	<i>% of I-TEQ</i>	<i>Relative Rank</i>	<i>% of I-TEQ</i>	<i>Relative Rank</i>
2,3,7,8-TCDD	36.2	1	3.1	4
Other TCDDs	0	-	0	-
1,2,3,7,8-PeCDD	0	-	0	-
Other PeCDDs	0	-	0	-
1,2,3,4,7,8-HxCDDs	0	-	0.6	7
1,2,3,6,7,8-HxCDDs	6.4	4	10.0	3
1,2,3,7,8,9-HxCDDs	2.3	8	0	-
Other HxCDDs	0	-	0	-
1,2,3,4,6,7,8-HpCDDs	5.8	6	69.0	1
Other HpCDDs	0	-	0	-
OCDD	6.0	5	19.2	2
2,3,7,8-TCDF	22.7	2	1.8	5
Other TCDFs	0	-	0	-
1,2,3,7,8-PeCDF	1.8	9	0	-
2,3,4,7,8-PeCDF	11.6	3	0.5	8
Other PeCDFs	0	-	0	-
1,2,3,4,7,8-HxCDFs	3.1	7	0.3	10
1,2,3,6,7,8-HxCDFs	1.2	12	0	-
2,3,4,6,7,8-HxCDFs	1.5	10	0.2	11
1,2,3,7,8,9-HxCDFs	0	-	0	-
Other-HxCDFs	0	-	0	-
1,2,3,4,6,7,8,-HpCDFs	1.3	11	0.8	6
1,2,3,4,7,8,9-HpCDFs	0	-	.05	12
Other-HpCDFs	0	-	0	-
OCDFs	0.2	13	0.3	9



**Table 7**  
**Analysis Of Personal Breathing Zone Samples For Halogenated Organics**  
**Bleach Plant, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Job Title	Concentration (mg/m <sup>3</sup> )				
	CHCL <sub>3</sub>	CCL <sub>3</sub> CH <sub>3</sub>	CCL <sub>4</sub>	BRCL <sub>2</sub> CH	BR <sub>2</sub> CLCH
Head Bleacher	1.1	ND	ND	ND	ND
Hardwood Operator	4.3	ND	ND	ND	ND
Softwood Operator	7.5	ND	ND	ND	ND
Chemical Unloader	1.3	ND	ND	ND	ND
Head Bleacher	6.0	ND	ND	ND	ND
Hardwood Operator	11.2	ND	ND	ND	ND
Softwood Operator	15.2	ND	ND	ND	ND
Chemical Unloader	2.9	ND	ND	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air  
 ND = None Detected (results were below the analytical limit of detection)  
 CHCL<sub>3</sub> = chloroform  
 CCL<sub>3</sub>CH<sub>3</sub> = 1,1,1-trichloroethane  
 CCL<sub>4</sub> = carbon tetrachloride  
 BRCL<sub>2</sub>CH = dichlorobromomethane  
 BR<sub>2</sub>CLCH = chlorodibromomethane

**Table 8**  
**Analysis Of Area Samples For Halogenated Organics**  
**Paper Mill, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Location	Concentration (mg/m <sup>3</sup> )				
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>	CCL <sub>4</sub>	BRCL <sub>2</sub> CH	BR <sub>2</sub> CLCH
Machine #4, Wet End, Booth	1.1	0.1	ND	ND	ND
Machine #4, Wet End, Back Side	1.0	ND	ND	ND	ND
Machine #7, Wet End, Booth	0.5	ND	ND	ND	ND
Machine #7, Wet End, Back Side	0.8	ND	ND	ND	ND
Machine #8, Wet End, Press El	ND	ND	ND	ND	ND
Machine #9, Wet End, Front Side	ND	ND	ND	ND	ND
Machine #9, Wet End, Back Side	ND	ND	ND	ND	ND
Machine #15, Wet End, Front Side	ND	ND	ND	ND	ND
Machine #15, Wet End, Back Side	ND	ND	ND	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air sampled  
 ND = None Detected (results were below the analytical limit of detection)  
 CHCL<sub>3</sub> = chloroform  
 CCL<sub>3</sub>CH<sub>3</sub> = 1,1,1-trichloroethane  
 CCL<sub>4</sub> = carbon tetrachloride  
 BRCL<sub>2</sub>CH = dichlorobromomethane  
 BR<sub>2</sub>CLCH = chlorodibromomethane

**Table 9**  
**Analysis Of Area-Source Samples For Halogenated Organics**  
**Hardwood Bleach Plant, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Sampling Location	Concentration (mg/m <sup>3</sup> )				
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>	CCL <sub>4</sub>	BRCL <sub>2</sub> CH	BR <sub>2</sub> CLCH
Hardwood, Chlorine, Rinse Tank	9.0	ND	ND	ND	ND
Hardwood, Chlorine, Rinse Tank	15.2	ND	ND	ND	ND
Hardwood, Chlorine dioxide, Rinse Tank	3.6	ND	ND	ND	ND
Hardwood, Chlorine dioxide, Rinse Tank	3.6	ND	ND	ND	ND
Hardwood, Hypochlorite, Rinse Tank	54.7	ND	ND	1.1	ND
Hardwood, Hypochlorite, Rinse Tank	116.6	ND	ND	1.3	ND
Hardwood, Hypochlorite, Rinse Tank	59.1	ND	ND	0.7	ND
Hardwood, Hypochlorite, Rinse Tank	9.8	ND	ND	ND	ND
Hardwood, Hypochlorite, Rinse Tank	83.2	ND	ND	2.4	ND
Hardwood, Hypochlorite, Rinse Tank	4.4	ND	ND	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air  
 ND = none detected (results were below the analytical limit of detection)  
 CHCL<sub>3</sub> = chloroform  
 CCL<sub>2</sub>CH<sub>3</sub> = 1,1,1-trichloroethane  
 CCL<sub>4</sub> = carbon tetrachloride  
 BRCL<sub>2</sub>CH = dichlorobromomethane  
 BR<sub>2</sub>CLCH = chlorodibromomethane

**Table 10**  
**Analysis Of Area-Source Samples For Halogenated Organics**  
**Softwood Bleach Plant, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Sampling Location	Concentration (mg/m <sup>3</sup> )				
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>	CCL <sub>4</sub>	BRCL <sub>2</sub> CH	BR <sub>2</sub> CLCH
Softwood, Chlorine, Rinse Tank	2.8	ND	ND	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	1.8	ND	ND	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	2.8	ND	ND	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	9.9	ND	ND	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	12.0	ND	ND	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	2.9	ND	ND	ND	ND
Softwood, Hypochlorite, Rinse Tank	23.9	ND	ND	ND	ND
Softwood, Hypochlorite, Rinse Tank	5.5	ND	ND	ND	ND
Softwood, Hypochlorite, Rinse Tank	2.6	ND	ND	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air

ND = none detected (results were below the analytical limit of detection)

CHCL<sub>3</sub> = chloroform

CCL<sub>2</sub>CH<sub>3</sub> = 1,1,1-trichloroethane

CCL<sub>4</sub> = carbon tetrachloride

BRCL<sub>2</sub>CH = dichlorobromomethane

BR<sub>2</sub>CLCH = chlorodibromomethane

**Table 11**  
**Analysis Of Personal Breathing Zone Samples**  
**For Halogenated Organics**  
**Bleach Plant, Boise Cascade: Rumford, Maine**  
**March 25-26, 1992**  
**HETA 88-0140-2517**

Job Title	Concentration (mg/m <sup>3</sup> )	
	CHCL <sub>3</sub>	CCL <sub>3</sub> CH <sub>3</sub>
Head Bleacher	(0.01)	ND
Hardwood Operator	(1.18)	ND
Softwood Operator	ND	ND
Chemical Unloader	ND	ND
Head Bleacher	(1.79)	3
Hardwood Operator	(0.01)	3.1
Softwood Operator	ND	ND
Chemical Unloader	(0.01)	4.8

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air

ND = none detected (results were below the analytical limit of detection [LOD])

CHCL<sub>3</sub> = chloroform

CCL<sub>3</sub>CH<sub>3</sub> = 1,1,1-trichloroethane

() = values in parentheses indicate the concentration detected was between the LOD and the limit of quantification (LOQ)

**Table 12**  
**Analysis Of Area-Source Samples For Halogenated Organics**  
**Hardwood Bleach Plant, Boise Cascade: Rumford, Maine**  
**March 25-26, 1992**  
**HETA 88-0140-2517**

Sampling Location	Concentration (mg/m <sup>3</sup> )	
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>
Hardwood, Chlorine, Rinse Tank	ND	ND
Hardwood, Chlorine, Rinse Tank	ND	ND
Hardwood, Chlorine dioxide, Rinse Tank	ND	ND
Hardwood, Chlorine dioxide, Rinse Tank	ND	ND
Hardwood, Hypochlorite, Rinse Tank	8.7*	ND
Hardwood, Hypochlorite, Rinse Tank	44.1*	ND
Hardwood, Quality Control Lab	ND	ND

\*Data only reported for duration of industrial production observed during the monitoring.

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air

ND = none detected (results were below the analytical limit of detection)

CHCL<sub>3</sub> = chloroform

CCL<sub>2</sub>CH<sub>3</sub> = 1,1,1-trichloroethane

**Table 13**  
**Analysis Of Area-Source Samples For Halogenated Organics**  
**Softwood Bleach Plant, Boise Cascade: Rumford, Maine**  
**March 25-26, 1992**  
**HETA 88-0140-2517**

Sampling Location	Concentration (mg/m <sup>3</sup> )	
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>
Softwood, Chlorine, Rinse Tank	ND	ND
Softwood, Chlorine, Rinse Tank	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	ND	ND
Softwood, Chlorine dioxide, Rinse Tank	ND	ND
Softwood, Hypochlorite, Rinse Tank	ND	ND
Softwood, Hypochlorite, Rinse Tank	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air

ND = none detected (results were below the analytical limit of detection)

CHCL<sub>3</sub> = chloroform

CCL<sub>2</sub>CH<sub>3</sub> = 1,1,1-trichloroethane

**Table 14**  
**Analysis Of Area Samples For Halogenated Organics**  
**Paper Mill, Boise Cascade: Rumford, Maine**  
**March 25-26, 1992**  
**HETA 88-0140-2517**

Location	Concentration (mg/m <sup>3</sup> )	
	CHCL <sub>3</sub>	CCL <sub>2</sub> CH <sub>3</sub>
Machine #4, Wet End, Start of Drying Rolls	ND	ND
Machine #4, Adjacent to Drying Rollers	ND	ND
Machine #7, Wet End, Begining of Drying Rolls	ND	ND
Machine #7, Adjacent to Drying Roller	ND	ND
Beater Room, Near #7 Sample Port	ND	ND
Machine #4, Wet End, Guard Rail	ND	ND
Machine #4, Adjacent to Drying Rollers	ND	ND
Machine #11, Wet End, Front Side	ND	ND
Machine #11, Inside Drying Roller Enclosure	ND	ND
Machine #11, Dry End, Control Bench	ND	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air  
 ND = none detected (results were below the analytical limit of detection)  
 CHCL<sub>3</sub> = chloroform  
 CCL<sub>3</sub>CH<sub>3</sub> = 1,1,1-trichloroethane



**Table 15  
Paper Dust Personal Samples  
Paper Mill, Boise Cascade: Rumford, Maine  
August 9-11, 1991  
HETA 88-0140-2517**

<b>Paper Machine - Job Title</b>	<b>Time (min.)</b>	<b>Dust Concentration (mg/m<sup>3</sup>)</b>
#4 - Fourth Hand	277	0.13
#4 - Third Hand	269	0.02
#7 - Third Hand	337	0.28
#8 - Back Tender	338	ND
#8 - Third Hand	324	0.14
#9 - Operator B	235	0.10
#10 - Fourth Hand	292	0.08
#10 - Utility	339	0.16
#11 - Third Hand	333	0.05
#12 - Fifth Hand	275	0.02
#15 - Back Tender	368	0.02
#15 Finishing - Operator B	357	ND
#15 Finishing - Winder Operator	321	0.04
#38 Rewinder - C Box Operator	283	ND

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air

ND = none detected (results were below the analytical limit of detection)

**Table 16**  
**Paper Dust Area Samples**  
**Paper Mill, Boise Cascade: Rumford, Maine**  
**August 9-11, 1991**  
**HETA 88-0140-2517**

Sample Location	Time (min.)	Dust Concentration (mg/m <sup>3</sup> )
# 4 Back side of reel, machine	445	ND
# 4 Paper machine, back side of reel	446	0.20
# 5 Front side winder controls, machine	433	ND
# 5 Paper machine, front side of winder	432	0.03
# 7 Back side tension drive, machine	459	ND
# 7 Paper machine winder	473	0.21
# 7 Paper machine, back side tension drive	462	0.24
# 7 Paper machine, back side winder	475	0.41
# 8 and # 9 Paper machines, between winders	473	0.07
# 9 Paper machine back side of winder	398	0.18
# 9 Paper machine front side of winder	449	0.09
#10 Back side of winder, machine	415	ND
#10 Paper machine back side of winder	412	0.03
#10 Paper machine front side of reel	448	0.16
#11 Paper machine, back side of reel	445	0.22
#12 Back side, roll detector, machine	399	0.01
#12 Back side, roll detector, machine	424	ND
#15 Paper machine front side, calender	429	0.05
#15 Paper machine front side, reel	430	0.05
#15 Paper machine, back side of rewinder	441	0.05
#15 Paper machine, front side of winder	409	0.03
#35 Winder drive	382	0.06
#35 Winder drive	386	ND
#38 Upper deck, winder	376	ND
#38 Upper deck, winder	375	ND
#39 Winder operator booth, front side	451	0.05
#40 winder top of stairs, tender side	455	0.05
#40 winder, top of stairs, drive side	452	0.03

mg/m<sup>3</sup> = milligrams of contaminant per cubic meter of air  
 ND= None detected

**Table 17**  
**Demographic and Non-occupational Exposure Characteristics**  
**of Mill Workers and Community Residents**  
**Boise Cascade: Rumford, Maine**  
**HETA 88-0140-2517**

	Community Resident		Mill Worker	
	No. of Persons	%	No. of Persons	%
Age (years)				
<20	3	19	0	0
30-39	3	19	11	24
40-49	4	25	8	17
50-59	4	25	17	37
60+	2	13	10	22
Body mass index				
1st quartile (Lowest)	5	31	11	24
2nd quartile	4	25	11	24
3rd quartile	3	19	13	28
4th quartile (Highest)	4	25	11	24
Cigarette smoking status				
Never	8	50	9	20
Former	5	31	26	57
Current	3	19	11	24
Current alcohol consumption				
More than 1 drink/week	8	53	25	54
One or less drinks/week	7	47	21	46
Vietnam veteran	0	0	2	4
Eats locally caught fish	12	75	37	80
Ever applied pentachlorophenol	0	0	1	2
Ever applied weed or brush killer	0	0	8	17
Current house heated by wood or coal	4	25	16	35

**Table 18  
PCDDs and PCDFs in Serum  
Boise Cascade: Rumford, Maine  
HETA 88-0140-2517**

Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)	Tetrachlorodibenzofuran (2,3,7,8-TCDF)
Pentachlorodibenzo-p-dioxin (1,2,3,7,8-PeCDD)	Pentachlorodibenzofuran (1,2,3,7,8-PeCDF) (2,3,4,7,8-PeCDF)
Hexachlorodibenzo-p-dioxin (1,2,3,4,7,8-HxCDD) (1,2,3,6,7,8-HxCDD) (1,2,3,7,8,9-HxCDD)	Hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF) (1,2,3,6,7,8-HxCDF) (1,2,3,7,8,9-HxCDF) (2,3,4,6,7,8-HxCDF)
Heptachlorodibenzo-p-dioxin (1,2,3,4,6,7,8-HpCDD) (1,2,3,4,6,7,9-HpCDD)	Heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF) (1,2,3,4,7,8,9-HpCDF)
Octachlorodibenzo-p-dioxin (OCDD)	Octachlorodibenzofuran (OCDF)

**Table 19**  
**Lipid-adjusted Serum Concentrations of PCDDs**  
**in Mill Workers and Community Residents<sup>1</sup>**  
**Boise Cascade**  
**Rumford, Maine**  
**HETA 88-0140-2517**

Congener	Exposure Group	N <sup>2</sup>	Concentration (ppt) <sup>1</sup>		
			Median	Low	High
2,3,7,8-TCDD	Community Resident	16	1.8	1.5	3.5
	Worker - Low <sup>3</sup>	14	1.9	0.9	5.2
	Worker - High <sup>4</sup>	32	1.9	0.7	4.9
1,2,3,7,8-PeCDD	Community Resident	15	5.6	2.0	7.8
	Worker - Low	14	5.3	3.7	12.3
	Worker - High	32	5.7	2.6	11.3
1,2,3,4,7,8-HxCDD	Community Resident	16	6.2	1.8	11.3
	Worker - Low	12	7.4	1.8	19.1
	Worker - High	31	5.6	2.5	14.7
1,2,3,6,7,8-HxCDD	Community Resident	16	67.0	48.3	101
	Worker - Low	14	79.7	33.1	145
	Worker - High	25	65.9	29.4	117
1,2,3,7,8,9-HxCDD	Community Resident	16	6.9	3.3	12.8
	Worker - Low	14	9.4	1.9	19.7
	Worker - High	29	8.3	2.9	21.4
1,2,3,4,6,7,8-HpCDD	Community Resident	15	95.2	64.1	115
	Worker - Low	13	91.2	47.5	230
	Worker - High	31	73.9	34.0	161
1,2,3,4,6,7,9-HpCDD	Community Resident	15	5.2	3.3	14.7
	Worker - Low	9	7.7	3.5	26.8
	Worker - High	19	7.0	3.3	36.5
OCDD	Community Resident	11	547	230	1042
	Worker - Low	10	673	288	1600
	Worker - High	23	541	285	1489

<sup>1</sup>ppt = parts per trillion. Imputed values were calculated for nondetectable results (see text)

<sup>2</sup>The number of samples may be fewer than 16 for community residents, 32 for high exposure workers, and 14 for low exposure workers because quality control criteria were not met in some samples.

<sup>3</sup>Low = low exposure potential (groundwood mill/long log area, wood yard, Kraft mill)

<sup>4</sup>High = high exposure potential (bleach plant, dry end of the paper machines, rewind areas, finishing areas, effluent treatment plant)

**Table 20**  
**Lipid-adjusted Serum Concentrations of PCDFs**  
**in Mill Workers and Community Residents**  
**Boise Cascade**  
**Rumford, Maine**  
**HETA 88-0140-2517**

Congener	Exposure Group	N <sup>2</sup>	Median	Concentration (ppt) <sup>1</sup>	
				Low	High
2,3,7,8-TCDF <sup>3</sup>	Community Resident	14	1.3	0.8	4.9
	Worker - Low <sup>4</sup>	14	1.4	0.7	6.7
	Worker - High <sup>5</sup>	32	1.3	0.4	11.1
1,2,3,7,8-PeCDF <sup>3</sup>	Community Resident	15	1.3	0.8	2.8
	Worker - Low	14	1.3	0.4	2.1
	Worker - High	30	1.2	0.5	2.4
2,3,4,7,8-PeCDF	Community Resident	16	6.4	3.5	13.0
	Worker - Low	14	7.8	3.9	14.1
	Worker - High	31	5.9	2.1	11.3
1,2,3,4,7,8-HxCDF	Community Resident	16	6.4	2.4	9.6
	Worker - Low	14	6.9	2.2	16.2
	Worker - High	32	6.8	2.9	13.2
1,2,3,6,7,8-HxCDF	Community Resident	16	4.8	2.5	7.7
	Worker - Low	14	5.3	2.2	11.7
	Worker - High	31	4.9	2.5	9.4
1,2,3,7,8,9-HxCDF <sup>3</sup>	Community Resident	16	1.6	0.8	2.3
	Worker - Low	14	1.4	0.4	3.4
	Worker - High	31	1.3	0.5	3.3

cont'd

<sup>1</sup>ppt = parts per trillion. Imputed values were calculated for nondetectable results (see text)

<sup>2</sup>The number of samples may be fewer than 16 for community residents, 32 for high exposure workers, and 14 for low exposure workers because quality control criteria were not met in some samples.

<sup>3</sup>The proportion of detectables for this congener was less than 50% of all samples.

<sup>4</sup>Low = low exposure potential (groundwood mill/long log area, wood yard, Kraft mill)

<sup>5</sup>High = high exposure potential (bleach plant, dry end of the paper machines, rewind areas, finishing areas, effluent treatment plant)

**Table 20**  
**Lipid-adjusted Serum Concentrations of PCDFs**  
**in Mill Workers and Community Residents**  
**Boise Cascade**  
**Rumford, Maine**  
**HETA 88-0140-2517**

Congener	Exposure Group	N <sup>2</sup>	Median	Concentration (ppt) <sup>1</sup>	
				Low	High
2,3,4,6,7,8-HxCDF <sup>3</sup>	Community Resident	15	3.9	2.5	14.6
	Worker - Low	7	4.7	2.3	7.9
	Worker - High	15	4.6	1.9	7.8
1,2,3,4,6,7,8-HpCDF	Community Resident	13	15.5	9.5	31.3
	Worker - Low	9	19.5	13.3	28.1
	Worker - High	22	17.3	7.5	33.7
1,2,3,4,7,8,9-HpCDF <sup>3</sup>	Community Resident	12	2.7	0.8	11.0
	Worker - Low	8	3.4	1.6	4.7
	Worker - High	20	2.6	0.6	7.0

<sup>1</sup>ppt = parts per trillion. Imputed values were calculated for nondetectable results (see text)

<sup>2</sup>The number of samples may be fewer than 16 for community residents, 32 for high exposure workers, and 14 for low exposure workers because quality control criteria were not met in some samples.

<sup>3</sup>The proportion of detectables for this congener was less than 50% of all samples.

<sup>4</sup>Low = low exposure potential (groundwood mill/long log area, wood yard, Kraft mill)

<sup>5</sup>High = high exposure potential (bleach plant, dry end of the paper machines, rewind areas, finishing areas, effluent treatment plant)

**Table 21**  
**Toxic Equivalents (I-TEQ) in**  
**Mill Workers and Community Residents**  
**Boise Cascade: Rumford, Maine**  
**HETA 88-0140-2517**

	Exposure Group		
	Community (n=16)	Worker-Low Exposure (n=14)	Worker-High Exposure (n=32)
<b>PCDD Subtotal</b>	13.5 (9.5-19.1) <sup>1</sup>	15.9 (6.5-31.8)	13.3 (7.5-24.9)
<b>PCDF Subtotal</b>	5.0 (3.4-8.8)	5.9 (3.2-11.0)	4.7 (1.9-8.1)
<b>Total I-TEQ</b>	19.1 (12.9-25.9)	21.2 (9.8-41.6)	18.1 (10.7-32.9)

<sup>1</sup> Median and (range)

**Table 22**  
**Linear Regression Model for the Natural Logarithm of**  
**Total Toxic Equivalents (I-TEQ)**  
**Boise Cascade: Rumford, Maine**  
**HETA 88-0140-2517**

Variable	Parameter Estimate	Standard Error	p
Intercept	2.26	0.27	0.0001
Age (years)	0.009	0.003	0.001
Body mass index <sup>1</sup>	13.01	5.03	0.01
Fish consumption <sup>2</sup>	0.20	0.08	0.02
Cigarette smoking <sup>3</sup>	-0.17	0.08	0.05

<sup>1</sup> weight/(height)<sup>2</sup>

<sup>2</sup> consumption of fish from local rivers (ever yes/no)

<sup>2</sup> consumption of fish from local rivers (ever yes/no)



**Figure 1**  
Toxic Equivalents (I-TEQ) by Exposure Group  
Boise Cascade: Rumford, Maine  
HETA 88-0140-2517

