Penta treated wood should be aged three months after treatment and prior to submersion. This aging period will allow time for some of the carrier oil to evaporate and for the binding of penta with lignin in the middle lamella to begin, thus reducing the potential for higher rates of leaching when the treated wood is first installed...

For effective evaporation to occur during the aging, the treated wood needs to be stacked in such a way that air can circulate freely through the pile. The wood should not be stacked at a location where precipitation running off the wood could drain into a natural water body.

Pentachlorophenol exposed to sunlight on the surface of treated wood can form octachlorodibenzo-dioxin (OCDD) via a photolytic condensation reaction. After exposing small pieces of penta-treated wood for 20 days to natural and artificial light, measured OCDD concentrations of about 70 ppm (ng OCDD/mg PCP). The OCDD in turn degraded to HpCDD and HxCDD (hepta- and hexa-chlorodibenzodioxin). The final concentration of HxCDD was approximately 15 - 20 ppm. When P-9 oil was used as the solvent for penta, concentration of OCDD formed from penta ranged from 2 - 4 ppm. Of the three contaminants identified above, the most toxic is the hexachlorodibenzo-p-dioxin (HxCDD).

**OCTACHLORODIBENZO-P-DIOXIN**

CASRN: 3268-87-9

**Human Health Effects:**

**Evidence for Carcinogenicity:**

Overall evaluation: Other polychlorinated dibenzo-para-dioxins are not classifiable as to their...
carcinogenicity to humans (Group 3). /Polychlorinated dibenzo-para-dioxins/

**Human Toxicity Excerpts:**

/HUMAN EXPOSURE STUDIES/ All members of a Spanish family (father, mother and six children) developed chloracne. The causative agent was found to be the family's stock of olive oil, which had become contaminated with dibenzo-p-dioxins, dibenzofurans, pentachlorophenol, and hexachlorobenzene. The more highly chlorinated dibenzo-p-dioxins, in particular octachlorodibenzo-p-dioxin, were the predominant congeners in the oil. Three members of the family exhibited either an overt or a sub-clinical disturbance of kidney function. The father also had a chronic respiratory problem. These changes could not be unequivocally attributed to the dibenzo-p-dioxins. Experimental toxicity of the oil was limited to the development of the oil ceased, contained high levels of the dibenzo-p-dioxins and dibenzofurans. Extrapolation back to ingested dose was used to validate dosage estimates. The use of toxicity equivalence factors provided estimates of cumulative dosage to produce chloracne as 0.13-0.31 ug 2,3,7,8-tetrachlorodibenzo-p-dioxin/kg (using EPA toxicity equivalence factors) or 6.7-16 ug 2,3,7,8-tetrachlorodibenzo-p-dioxin/kg (using Nordic/NATO toxicity equivalence factors). This is the first incident in which human toxicity is related primarily to ingestion of dibenzo-p-dioxins and for which estimates of dosage can be made.


/HUMAN EXPOSURE STUDIES/ Workers exposed to dioxin had adipose tissue levels with a mean of 246 ppt. Unexposed workers had levels of 86 ppt. Nine workers with a history of exposure to PCDD's and chloracne in 1971 to 1973 had TCDD serum levels of 340 pg per gram blood lipid in 1990.


/HUMAN EXPOSURE STUDIES/ Due to their lipophilicity, /Chlorinated dibenzo-para-dioxins/ (CDDs) can concentrate in human breast milk and can be transferred to infants through nursing. In general, the amount of individual congeners in breast milk decreased as chlorination decreases. Excretion via milk is highest during the first weeks after delivery. Also, the concentration of CDDs in milk is higher in mothers breast-feeding their first child than in those breast-feeding their second child. CDDs transferred to infants through nursing are readily absorbed by the infants. A pharmacokinetic model predicted that the increased body burden in infants that results from breast-feeding does not translate into raised lifetime body burden. /Chlorinated dibenzo-p-dioxins/


/HUMAN EXPOSURE STUDIES/ Two studies of nursing infants suggest that ingestion of breast milk with a higher dioxin-furan TEQ value may alter thyroid function. Both studies had similar exposure groupings and some findings in common: both had significant increases in /thyroid-stimulating hormone (TSH)/ at about 3 months of age with higher TEQs, and in one report, significant increases at about 2 weeks of age and in the cord blood. ...These two developmental studies investigated relatively small numbers of infants with thyroid parameters in the normal range. However, the "high" group, at about 3 months of age, had increased TSH levels in comparison to the "low" group. Total T4 levels and total T4 to thyroid binding globulin (TBG) ratio were generally elevated in the high infants. /Polychlorinated dibenzo-p-dioxins and dibenzofurans/

[Schecter A, Gasiewicz TA; Dioxins and Health 2nd ed. p.778 (2003)] **PEER REVIEWED**

/SHORT AND SYMPTOMS/ Other less consistently reported effects from dioxin exposure in humans include /asthenia/, headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found
by others. /Polychlorodibenzodioxins/

/CASE REPORTS/ A case history of chloracne due to exposure to pentachlorophenol treated wood was described. A 32 yr old white male was seen at a university dermatology clinic complaining of an acneiform eruption of 6 mo duration. The patient was part owner of a firm that constructed piers for small boat marinas. The lumber used was pretreated with pentachlorophenol. Within about 9 mo after beginning work, he noted a papular acneform eruption that occurred over the entire body. The eruption was characterized by multiple, small yellow/white papules. Areas most involved included the malar regions of the face, post auricular area, the trunk, buttocks, thighs, and lower legs. Some of the papules were inflamed. A trephine punch biopsy of one of the papules showed a small epithelial lined cystic structure that communicated with the surface. The lining epithelium was composed of atrophic, but normal appearing, epidermis. Contained within the cyst was keratin like material. The condition was diagnosed as chloracne. The patient's condition improved after 6 wk oral treatment with isotretinoin. The patient remained asymptomatic for the ensuing 2 yr of observation. The patient returned to work wearing appropriate protective clothing. A sample of pentachlorophenol used by the firm and samples of treated wood were analyzed for OCDD. Samples from the surface of the lumber contained about ten to 40 times the amount of OCDD as did the wood itself. The undiluted pentachlorophenol contained 1600 ppm OCDD. It was concluded that the patient developed chloracne after exposure to pentachlorophenol treated lumber. The OCDD containing surface residue seemed to be the major source of the intoxication.

[Cole GW et al; Contact Dermatitis 15 (3): 164-8 (1986)] **PEER REVIEWED** PubMed Abstract

/EPIEMIOLOGY STUDIES/ To evaluate the association between exposure to pentachlorophenol and the occurrence of chloracne, we studied the medical and personnel records for individuals employed in the manufacturing of pentachlorophenol. Forty seven cases of chloracne were identified among 648 workers (7.0%) assigned to pentachlorophenol production at a single plant between 1953 and 1978. The annual incidence rate varied considerably, ranging from 0 (in 1953) to 1.46 (in 1978). No linear trend in the risk of chloracne was observed with the duration of employment in the pentachlorophenol department. Workers with a documented episode of direct skin contact with pentachlorophenol had a significantly increased risk of chloracne compared with workers who did not have a documented episode of direct skin contact (cumulative incidence ratio = 4.6; 95% confidence interval 2.6-8.1). Results confirm that chloracne is associated with exposure to pentachlorophenol contaminated with hexachlorinated, heptachlorinated, and octachlorinated dibenze-p-dioxins and dibenzofurans.


/EPIEMIOLOGY STUDIES/ One report has examined blood measures in 35 babies in Amsterdam. Four blood samples were taken: maternal blood around delivery, cord blood, and the infant's blood at 1 and 11 weeks af age. These samples were used to measure leucocytes (white blood cells), platelets, and differential (white cell count), along with plasma, activity of gamma-glutamyltransferase (GGT), /aspartate aminotransferase/ (AST), and /alanine aminotransferase/ (ALT), and levels of cholesterol and bilirubin. Dioxin and furan levels were measured in breast milk collected about 3 weeks postdelivery. None of the maternal blood measurements were outside the normal range. A statistically significant inverse correlation was observed in an uncorrected comparison of the number of polynuclear neutrophils and dioxin-furan levels in breast milk (r=-0.53, p=0.022); this disappeared when regression analysis compared these and controlled for gestational age. None of the other factors compared in the cord blood at 1 wk of age were statistically significant. The next set of analyses used the estimated cumulative dioxin-fin theun intake from breast feeding at 11 weeks of age; the PCDD/PCDF TEQs ranged from 5.7 to 123.7 pg TEQ/g fat, with a mean of 44.7 pg (significant correlations; ALT- correlation coefficient: 0.40 (p-value 0.02), AST- correlation coefficient: 0.44 (p-value 0.009), Platelets- correlation coefficient: -0.48 (p-value
These results are unadjusted, but remain significant after adjusting for maternal age, gestational age, and birth weight (regression coefficients were not presented). The authors proposed that changes in ALT and AST suggest an effect on the liver, associated with cumulative exposure to dioxins-furans, and note that all but three of the children had ALT and AST within normal ranges, but the distribution of some of these findings (e.g., an increase in platelets) did vary. From this study it is not possible to determine the reversibility or the clinical significance of these changes.  


OTHER TOXICITY INFORMATION/ Toxic equivalency factors (TEF), indicate the toxicity of a compound relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin, which itself has been assigned a TEF of 1.0. Concentration data for polychlorinated dibenzo-p-dioxins are frequently reported in units of toxic equivalency which are equal to the measured concentration of substance multiplied by its TEF. The TEF for octachlorodibenzo-p-dioxin is 0.001.  


Skin, Eye and Respiratory Irritations:  
An eye irritant.  

Probable Routes of Human Exposure:  
Occupational exposure to octachlorodibenzo-p-dioxin may occur for workers involved in the production of pentachlorophenol(1), which typically is contaminated with octachlorodibenzo-p-dioxin and other dioxins and furans, through the inhalation of dust particles(SRC). A mean octachlorodibenzo-p-dioxin concn of 0.336 ug/cu m was measured in samples of breathing-zone air at a pentachlorophenol production site(1). A study was conducted in two office buildings in Boston to determine background contamination levels of octachlorodibenzo-p-dioxin in buildings with no known previous exposures(2); surface concns of 2.1-130 ng/sq m (mean of 22 ng/sq m) were detected in surface wipe samples(2); airborne concns of 0.0032-0.0076 ng/cu m (mean of 0.0048 ng/cu m) were detected in the office building air which was very similar to outdoor air concns in Boston(2). Monitoring data indicate that the general population may be exposed to octachlorodibenzo-p-dioxin via inhalation of ambient air and ingestion of food(3) containing octachlorodibenzo-p-dioxin(SRC).  


Body Burden:  
Breast milk from 9 women exposed to a PCB fire in Canada contained a mean octachlorodibenzo-p-dioxin concn of 193.44 ng/kg (fat basis) while the mean concn in milk from 16 control women was 131.70 ng/kg(1). Analysis of 30 human milk samples from Germany found levels of 0.185-1.217 (avg 0.530) ppb(2). Analysis of blood samples from a total of 300 humans in Vietnam, Germany and the US found levels of 0.087-1.655 ppb (lipid)(3). The following concns (ppb lipid) were found in human milk from various countries(3): Vietnam: 0.078-0.292; Cambodia: 0.059; Thailand: 0.068; Germany: 0.185; US: 0.233; Siberia: 0.0502(3). Autopsies of two humans from the US general population detected the
following octachlorodibenzo-p-dioxin concns (ppb whole wt basis)(4): adipose tissue: 0.428-0.695; liver: 0.224-0.350; muscle: 0.076-0.171; and kidney: 0.031-0.039(4). Based upon various monitoring studies, the background level of octachlorodibenzo-p-dioxin in human adipose tissue ranges from 0.050-1.500 ppb(5). Human adipose tissue obtained during autopsies in 5 Canadian municipalities had an avg concn of 1.0367 ng/g(6). An octachlorodibenzo-p-dioxin concn of 0.690 ppb was detected in the adipose tissue of a person exposed during a PCB transformer fire in Binghamton, NY(7); the concn in the adipose tissue of 4 control people was 0.428-0.695 ppb(7). Analysis of whole blood samples from 10 persons with no declared exposure found octachlorodibenzo-p-dioxin levels of 439-889 ng/kg fat basis(8). Results of the National Human Adipose Tissue Survey (NHATS) for fiscal year 1982 reported an octachlorodibenzo-p-dioxin concn range of 64-1250 ng/kg and a mean concn of 560 ng/kg(9).


Blood samples from several locations contained octachlorodibenzo-p-dioxin at mean concns as follows (location, concn in parts per trillion lipi wt): Baikalsk, Russia, 57 (whole, n=8); St. Petersburg, Russia, 89 (whole, n=60); North Vietnam, 132.3 (whole, n=82); South Vietnam, 696.4 (whole, n=383); Germany, 610.3 (whole, n=102); USA pooled, 1174 (plasma, n=100); Guam 748.6 (whole, n=10)(1). The concn of octachlorodibenzo-p-dioxin in individuals from Sweden with no, normal and high fish consumption were 357, 458, and 473 pg/g lipid, respectively(2). Whole blood from German mothers (collected in 1989) contained octachlorodibenzo-p-dioxin at a mean concn of 610 pg/g lipid (range, 145-1524 pg/g lipid)(3). Serum samples collected in 1993 from 11 donor with no known exposure to dioxins in Madrid, Spain contained octachlorodibenzo-p-dioxin at an avg concn of 397.03 (range 117.05-690) pg/g fat wt(4).

Plasma samples from 20 non-occupationally exposed individuals living in Tarregona, Spain contain octachlorodibenzo-p-dioxin at a mean concn of 640.5 pg I-TEQ/g lipid (n=20; range, 137.0-993.0 pg I-TEQ/g lipid)(5). Blood samples from 50 normal Japanese women volunteers (approx 20 yo) collected between June 1993-1994 was found to contain octachlorodibenzo-p-dioxin at an avg concn of 310 (range, 110-1000) pg/g lipid wt(6). Adipose, spleen, and kidney tissue samples from 50 normal Japanese women volunteers (approx 20 yo) collected between June 1993-1994 was found to contain octachlorodibenzo-p-dioxin at an avg concns of 2,500 (range, 170-16000) pg/g lipid wt; 1,100 (range, 170-5,400) pg/g lipid wt; and 690 (range, 120-3,500) pg/g lipid wt, respectively(6). The mean concn of octachlorodibenzo-p-dioxin in 8 normal subjects from Japan collected in 1989 was 2,000 pg/g lipid (range, 160-13,000 pg/g lipid)(7). Human blood from individuals living in Germany were as follows (period, concn in pg/g lipid)(8): 1989, 625 (n=228); 1991, 446 (n=95); 1992, 462 (n=157); 1993, 340 (n=17); 1994, 231.0 (n=74); 1995, 293 (n=69); 1996, 257 (n=95); 1997/98, 320 (n=9); 1989-1998, 441 (n=744).


Adipose tissue from 17 subjects who died in Madrid, Spain from natural causes contained octachlorodibenzo-p-dioxin at concns ranging from 91-2847.5 pg/g fat wt (mean, 1318.1 pg/g fat wt)(1). Finnish human tissue samples (n=3) taken post-mortem exams in Helsinki contained octachlorodibenzo-p-dioxin at concns as follows (tissue, concn range in pg/g fresh wt)(2): testis, <5-9; liver, 230-700; adipose, 300-350. Placenta and fetus samples were collected in Binghamton, NY between 1984-1985; placenta (pool of 14 samples) had 282.1 ng/kg lipid of octachlorodibenzo-p-dioxin while fetus (8-14 week old, pool of 10) had 98.8 ng/kg lipid of octachlorodibenzo-p-dioxin(3). Tissue and fluid specimens were collected in 1995/96 from mothers from upstate New York; samples were analyzed for octachlorodibenzo-
p-dioxin as follows (tissue/liquid sample, concn in pg/g lipid)(4): adipose tissue, 214; predelivery blood, 374; placenta, 103; cord blood, 96; postpartum blood, 226; breast milk, 104. Levels of octachlorodibenzo-p-dioxin in adipose tissue samples from 15 autopsied subjects who lived in Tarragona, Spain (sampled between 1989-1999) ranged from 83.0 to 1,200 ng I-TEQ/kg fat (mean 472.53 ng I-TEQ/kg)(5). Levels of octachlorodibenzo-p-dioxin in adipose tissues from subjects living in Paris, France between Jan to May 1999 ranged from 0.01-0.09 pg I-TEQ/g lipid(6). The avg concn of octachlorodibenzo-p-dioxin in the blood of workers potentially exposed to dioxins in Finland between 1989-1990 were as follows (group, concn in pg/g lipid)(7): bleaching plant, 689 (n=14; range, 285-1471); paper mill, 625 (n=20; range, 288-1198); controls, 804 (range, 369-1745).


The concn of octachlorodibenzo-p-dioxin in human milk samples were determined for several locations around the world as follows (all ng/kg fat wt): USA Tennesse, 234; South Africa (Black), 196; South Africa (White), 254; Karachi, Pakistan, 180; Binh Long, Vietnam, 146; Vung Tau, Vietnam, 181; Tay Ninh, Vietnam, 415; Song Be province, 79.6(1). In a German study, a decrease in the concn of octachlorodibenzo-p-dioxin was found with the number of breast feeding periods(2); for example, the mean concn of octachlorodibenzo-p-dioxin in the first, sixth, and twelfth week of breast feeding was 292 (n=34), 252 (n=23), and 187 (n=6) pg/g fat, respectively(2). For human milk samples collected in 1985/86 at 3-geographic regions of Norway, the avg concn of octachlorodibenzo-p-dioxin ranged from 149.9 to 156.0 pg/g fat basis(3). Breast milk of Intuit women and women from southern Quebec had concns of octachlorodibenzo-p-dioxin of 292.3 (n=40) and 131.7 (n=96) ng/kg fat wt, respectively, for samples collected between 1989-1990(4). 526 human milk samples collected between 1986 and 1991 from mothers living in North Rhine-Westphalia, Germany contain octachlorodibenzo-p-dioxin at an avg concn of 1.4 pg/g fat (range, 0.2-14.0 pg/g fat)(5). Breast milk from Auckland (urban, n=20) and Christchurch (rural, n=17) sampled between Oct 1987 and May 1988 had avg concns of octachlorodibenzo-p-dioxin of 9.0 and 5.5 ng/kg in whole milk, respectively, and 220 and 200 ng/kg in milk fat, respectively(6). 26 human milk samples collected in late 1993-1994 from several locations in Japan contained octachlorodibenzo-p-dioxin at a mean concn of 4.9 pg/g whole milk (range, 1.4 to 14 pg/g whole milk)(7). Octachlorodibenzo-p-dioxin avg levels in human milk from Paris, France and Madrid, Spain were 290.0 (n=15) and 234.0 (n=13) pg/g fat wt collected in 1990(8). Thirty individual human milk samples from 5 main towns in Jordan contain octachlorodibenzo-p-dioxin at concns ranging from 29.0 to 147 ng/kg fat in 1994(9). Human milk samples taken from mothers living on the Kola Peninsula (Russia), a highly contaminated industrialized region, were analyzed for chemical contaminants(10); in human milk samples from the cities of Murmansk, Monchegorsk, and Tromso in April 1993, the concn of octachlorodibenzo-p-dioxin was 49.94 (n=15), 84.95 (n=15), and 106.48 ng/kg milk fat, respectively(10). [(1) Schecter A et al; Chemosphere 20:919-25 (1990) (2) Beck H et al; Chemosphere 25: 1015-20 (1992) (3) Clench-Aas J et al; J Tox Env Health 37: 73-83 (1992) (4) Dewailly E et al; Chemosphere 25: 1245-49 (1992) (5) Fuerst P et al; Chemosphere 25: 1029-38 (1992) (6) Bates MN et al; Environ Health Perspective 102 (Suppl 1): 211-17 (1994) (7) Hashimoto S et al; Chemosphere 31: 4067-75 (1995) (8) Gonzalez MJ et al; Bull Environ Contam Toxicol 56: 197-204 (1996) (9) Alawi MA et al; Chemosphere 33: 2469-74 (1996) (10) Polder A et al; Chemosphere 37: 1795-806 (1998)] **PEER REVIEWED**

Levels of octachlorodibenzo-p-dioxin in pooled samples of breast milk collected in 1996 from mothers living in the Tarrogona area, Spain ranged from 53.0 to 234.00 pg/g fat (mean, 145.67 pg/g fat)(1). Human milk samples were collected from mother's living in Helsinki (urban) and Kuopio (rural), Finland; samples collected between 1992-1994 were found to contain octachlorodibenzo-p-dioxin at mean concns of 230 (n=20; range, 58.0-349) and 126 (n=64; range, 62.7-310) pg/g fat, respectively(2). A pooled
A sample of human milk (40 mothers) from Rio de Janeiro, Brazil in 1992 contained 420 pg octachlorodibenzo-p-dioxin per g milk fat (3). The concn of octachlorodibenzo-p-dioxin in Swedish mother's milk was determined as follows (year, concn in TEQ pg/g lipid where TEF=0.0001): 1972, 0.06; 1976, 0.04; 1980, 0.03; 1984/85, 0.02; 1988/89, 0.03; 1990, 0.03; 1991, 0.02; 1992, 0.02; 1997, 0.01 (4).


**Average Daily Intake:**

**WATER:** Based upon monitoring of Canadian drinking water, the avg daily intake of octachlorodibenzo-p-dioxin has been estimated to be 0.0001 ng/adult/day (1). **FOOD:** The daily intake of all polychlorinated dibenzodioxin isomers has been estimated to be 0.1397 ng/adult/day, the majority of which is octachlorodibenzo-p-dioxin (1); food contribution comes from most food groups with meat, eggs and dairy products the major contributors (1). Based upon a Japanese market basket survey, the avg daily intake for octachlorodibenzo-p-dioxin has been estimated as 0.690 ng/adult/day (2). Based on food samples collected in Madrid, Spain during April 1995, the avg daily intake of octachlorodibenzo-p-dioxin has been estimated as 16,144.96 pg/person/day (3).


**Emergency Medical Treatment:**

**Emergency Medical Treatment:**

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The following Overview, *** DIOXINS ***, is relevant for this HSDB record chemical.

**Life Support:**

- This overview assumes that basic life support measures have been instituted.

**Clinical Effects:**

**0.2.1 SUMMARY OF EXPOSURE**

**0.2.1.1 ACUTE EXPOSURE**

A) **USES:** Dioxins have no intended commercial use; exposure is through their presence as a byproduct or contaminant of certain herbicides.
Increased concentrations of octachlorodibenzo-p-dioxin in cases with breast cancer--results from a case-control study.

Hardell L¹, Lindström G, Liljegren G, Dahl P, Magnuson A.

Author information

Abstract

Organochlorines are persistent and highly lipophilic environmental contaminants which bioaccumulate in the food chain. Some of these chemicals, 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane (DDT) and polychlorinated biphenyls (PCBs), have been suggested to be of significance in the aetiology of breast cancer. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is an anti-oestrogen in animal studies and should be thus lower the risk of breast cancer. The other isomers of polychlorinated dibenzo-p-dioxins (PCDDs) or the chemically related polychlorinated dibenzofurans (PCDFs) have not been tested regarding carcinogenesis of the breast. The purpose of this study was to investigate whether PCDDs or PCDFs influence the risk for breast cancer. Consecutive patients who underwent surgery for a breast disease between 1993 and 1995 were recruited for the study. Cases were 22 patients with infiltrative breast cancer and controls were 19 patients operated for a benign breast disease during the same time period. Approximately 10 g of breast tissue free from tumour was taken from the specimen and frozen until analysis. Fat was extracted, cleaned and analysed with a high-resolution gas chromatograph coupled to a high-resolution mass spectrometer. Median concentrations of octachlorinated dibenzo-p-dioxin (OCDD) were 598 (170-14,880) and 396 (103-1,847) pg/g lipid in the cases and in the controls, respectively. In a multivariate logistic regression analysis controlling for other risk factors for breast cancer increased odds ratio (OR) was obtained for OCDD: 401-1000 pg/g lipid yielded OR 3.8, 95% confidence interval (CI) 0.4-39, > 1000 pg/g lipid gave OR 5.2, CI 0.4-72. When the lipid OCDD variable was examined as a continuous risk factor there was a 1.09 (9%), CI 0.95-1.25, increase in the adjusted OR for breast cancer per 100 unit (pg/g lipid) increase in OCDD. No differences were found between cases and controls for the other six tested PCDDs. Mean concentration of TCDD was in the cases 3.6 (1.0-7.9) and in the controls 3.3 (1.1-6.3) pg/g lipid. For PCDFs no significant differences were found between cases and controls. The results were not changed if oestrogen or progesterone receptor status, S-phase fraction and DNA ploidy were considered. Breast tissue concentration of OCDD was increased in cancer patients, whereas the concentrations of other PCDDs and PCDFs were equal in cases and controls.

http://en.wikipedia.org/wiki/Chloracne

Chloracne is an acne-like eruption of blackheads, cysts, and pustules associated with over-exposure to certain halogenated aromatic compounds, such as chlorinated dioxins and dibenzofurans. The lesions are most frequently found on the cheeks, behind the ears, in the armpits and groin region.

The condition was first described in German industrial workers in 1897 by von Bettman, and was initially believed to be caused by exposure to chlorine(hence the name "chloracne"). It was only in the mid-1950s that chloracne was associated with aromatic hydrocarbons.[1] The substances that may cause chloracne are now collectively known as chloracnegens.

Chloracne is particularly linked to toxic exposure to dioxins (byproducts of many chemical processes, including the manufacture of herbicides such as Agent Orange)—so much so that it is considered a clinical sign of dioxin exposure. The severity and onset of chloracne may follow a typical asymptotic dose-response relationship curve.