Human Health Effects of Biomass Incinerators
Dioxins Damage Children and Adults

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Citations and Support Articles

Dioxins: an overview
Arnold Schecter, Linda Birnbaum, John J Ryan, John D Constable
University of Texas Health Science Center, School of Public Health, Dallas Campus, Dallas, TX 75390, USA.
Environmental Research (impact factor: 3.4). 08/2006; 101(3):419-28.
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"Dioxins did not exist prior to industrialization except in very small amounts due to natural combustions and geological processes (Czuczwa, et. al. 1984; Schecter et al. 1988; Ferrario and Byrne, 2000)."

Dioxins "Today are found in all humans, with higher levels commonly found in persons living in more industrialized countries (Scheter and Gasiewicz, 2003)."

"Although effects of small exposures to dioxins are unlikely to be detected by clinicians, the growing body of toxicological and epidemiological literature demonstrates that dioxins have had adverse impacts on our population."

"The Department of Health and Human Services recently concluded that 2,3,7,8 TCDD is carcinogenic to humans (National Toxicology Program, 2004). This is in agreement with both the IARC and the draft EPA position."

EPA's Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments, Volume 1 (PDF) (2012)
This document comprises the first of two EPA reports (*U.S. EPA's Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments Volumes 1 and 2 [Reanalysis Volumes 1 and 2]*) that, together, will respond to the recommendations and comments on 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) dose-response assessment included in the 2006 NAS report, *Health Risks from Dioxin and Related Compounds: Evaluation of the EPA Reassessment*. This document, Reanalysis Volume 1, includes (1) a systematic evaluation of the peer-reviewed epidemiologic studies and rodent bioassays

relevant to TCDD dose-response analysis; (2) dose-response analyses using a TCDD physiologically based pharmacokinetic model that simulates TCDD blood concentrations following oral intake; and (3) an oral reference dose (RfD) for TCDD.

http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=222203#Download

Cancer

The EPA report confirmed that dioxin is a cancer hazard to people. In 1997, the International Agency for Research on Cancer (IARC) -- part of the World Health Organization -- published their research into dioxins and furans and announced on February 14, 1997, that the most potent dioxin, 2,3,7,8-TCDD, is a now considered a Group 1 carcinogen, meaning that it's a known human carcinogen.

Also, in January 2001, the U.S. National Toxicology Program upgraded 2,3,7,8-TCDD from "Reasonably Anticipated to be a Human Carcinogen" to "Known to be a Human Carcinogen." See their reports on dioxins and furans from their *11th Report on Carcinogens* (find related documents under 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Furan). Finally, a 2003 re-analysis of the cancer risk from dioxin reaffirmed that there is no known "safe dose" or "threshold" below which dioxin will not cause cancer.
A July 2002 study shows dioxin to be related to increased incidence of breast cancer.

Dioxins & Furans: The Most Toxic Chemicals Known to Science

Full article at http://www.ejnet.org/dioxin/

Does dioxin cause cancer?

Yes. The EPA report confirmed that dioxin is a cancer hazard to people. In 1997, the International Agency for Research on Cancer (IARC) -- part of the World Health Organization -- published their research into dioxins and furans and announced on February 14, 1997, that the most potent dioxin, 2,3,7,8-TCDD, is a now considered a Group 1 carcinogen, meaning that it's a known human carcinogen.
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A July 2002 study shows dioxin to be related to increased incidence of breast cancer.

What other health problems are linked to dioxin exposure?

In addition to cancer, exposure to dioxin can also cause severe reproductive and developmental problems (at levels 100 times lower than those associated with its cancer causing effects). Dioxin is well-known for its ability to damage the immune system and interfere with hormonal systems.
Dioxin exposure has been linked to birth defects, inability to maintain pregnancy, decreased fertility, reduced sperm counts, endometriosis, diabetes, learning disabilities, immune system suppression, lung problems, skin disorders, lowered testosterone levels and much more.

Dioxins and Incineration

Dioxins are the most insidious pollutants released from incineration. Dioxin is considered to be the most toxic man-made substance.[1] Most dioxins are formed as unintended byproducts of combustion.[2] Trash, sewage sludge and biomass incinerators are each among the top sources of dioxin air pollution in the U.S.[3] Dioxin exposure is connected to a myriad of health problems including cancer, birth defects, diabetes, developmental disabilities, sexual reproductive disorders (including endometriosis, small penis, low sperm counts, delayed puberty, and malformed and mixed-sex genitalia), lowered testosterone levels, impaired immune system, allergies, low birth weight, dental defects, loss of intelligence and learning ability, ADHD and increased withdrawn/depressed behavior.[4]

After being emitted from various types of incinerators, dioxin falls out throughout the environment, avoiding water and seeking fat, rapidly climbing up the food chain.[5] Because dioxin bonds with fat so effectively, 93% of dioxin exposure comes through eating meat and dairy products.[6]

Dioxin emissions from incinerators, like other toxic pollutants, are measured only once per year at best; at worst, never. Air emissions are generally higher than normal when incinerators are starting up, shutting down, or experiencing malfunctions ("upset conditions"). This is especially true of dioxins.[7] Because annual emissions tests are done by the incinerator operator under the best operating conditions they can manage, they underestimate actual dioxin emissions by at least 30-50 times.[8] Continuous dioxin emissions monitoring technologies have existed for several years,[9] but the industry refuses to use them unless required by state environmental permitting agencies (which have yet to require them). This allows incinerators to claim their most toxic emissions are far lower than they really are.

[1] Mocarelli, et. al., "Paternal concentrations of dioxin and sex ratio of offspring," Lancet, 2000 May 27;355(9218):1838-9. http://www.ncbi.nlm.nih.gov/pubmed/10866441 "2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD or dioxin), is commonly considered the most toxic man-made substance." [can we cite a better source?; should we point out the difference

between 2,3,7,8-TCDD and dioxins are a broader category?]
[2] "An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the U.S. for the Years 1987, 1995, and 2000," Table ES-2, p. xlvi, U.S. Environmental Protection Agency, Nov 2006. http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=159286
[3] *Id.* MSW #3 [listed as 4th because sludge incineration is grouped with sludge land application in the final report; the draft report parses it out, making it clear that trash incineration is #3], industrial wood burning #7, SSIs #15, if you separate out sludge land application from incineration, applying the #s from the draft to do so.
[4] Center for Health, Environment & Justice, American People's Dioxin Report pp.15-20, 1999. http://chej.org/wp-content/uploads/Documents/American%20Peoples%20Dioxin%20Report.pdf (accessed 3/19/2011).
[5] 1994 EPA Dioxin Reassessment – Exposure Document. http://www.cqs.com/epa/exposure/part1_v1.htm
[6] Table 4-30. Estimated CDD/CDF Mean Background Exposures for Adults in the United States (National Academy of Sciences Review, Pt. 1, Vol. 2, Chap. 4, p. 4-110). http://www.epa.gov/ncea/pdfs/dioxin/nas-review/
[7] cite SSM emissions of dioxins? – Neil Carman's info?
[8] Wevers M. and De Fré R., "Underestimation of dioxin emission inventories," Organohalogen Compounds, Vol. 36, pp. 19-20 (1998). http://www.ejnet.org/toxics/cems/1998_DeFre_OrgComp98_Underest_DIoxin_Em_Inv_Amesa.pdf
[9] Dioxin Emission Monitoring Systems, Environmental Technology Verification Program, U.S. Environmental Protection Agency. http://www.epa.gov/etv/vt-ams.html#dems This page lists the four pieces of dioxin testing equipment that EPA tested and verified in 2006. The Amesa system (around since 1998) is one. It's a long-term sampler that can collect a sample of up to 30 days, allowing for year-round data collection if the operator resamples every 30 days. Others are semi-continuous or actual real-time dioxin emissions monitors. More on continuous emissions monitoring of dioxin here: http://www.ejnet.org/toxics/cems/dioxin.html

SECOND OPINION: THE MEDICAL PROFESSION DIAGNOSES **...**
File Format: PDF/Adobe Acrobat
Sep 6, 2011 **... Second Opinion**. PREFACE. This report is a compilation of statements from medical professionals and organizations throughout the United **...**
www.bredl.org/pdf3/**SecondOpinion**.pdf

For an detailed list of health problems related to dioxin, read the People's Report on Dioxin.

Here follows the report:

The American People's Dioxin Report
By The Center for Health, Environment & Justice

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Science

This report provides a summary of new scientific research on the toxic effects caused by orassociated with exposure to dioxin. The information in this report is drawn from a comprehensive assessment of the sources, fate, and health effects of dioxin contained in the Technical Support Document (TSD) to this report. The key points and conclusions of the TSD provide the basis for this report. Most of the research and studies discussed in this report have been published since a well publicized draft report on dioxin was released by the U.S. Environmental Protection Agency (EPA) in 1994. The American People's Dioxin Report is intended to inform the public and their representatives in government so appropriate action can be taken to safeguard the health of the American people. The scientific findings of this report make it clear that there is an extensive body of high quality scientific information describing the toxic effects of dioxin in people. This data indicates that dioxin is a potent chemical that produces a wide variety of toxic effects in animals and that some of these effects are occurring in people.

The report's most striking finding is the impact of dioxin on the growth and development of children. Most of the new studies on dioxin address its effects on children, notably the effects on the development of the immune, reproductive, and nervous systems, in particular cognitive and learning abilities. While exposure of the general population occurs through ingestion of many common foods, children exposed *in utero* (in the womb) during critical periods of development appear to be

the most sensitive and vulnerable to the toxic effects of dioxin. In particular, dioxin has been associated with IQ deficits and increased susceptibility to infections in Dutch children exposed to "background" levels of dioxins. (These "background" levels are essentially the average daily intake of dioxin from food.) Studies in Finland have shown that dioxin interferes with normal tooth development in infants exposed to "background" levels. The Dutch studies have also shown an association between dioxin and a higher prevalence of withdrawn/depressed behavior in children. An association between PCBs and adverse effects on attentional processes and an increase in hyperactive behavior in children has also been reported in these studies.

This new evidence from human studies provides strong confirmation of the toxicity of dioxin and its impact on the general American public. With this in mind, Americans have a choice: take action to protect public health by eliminating dioxin creation or continue to allow dioxin to be created and not burden industry with the short term transition costs of elimination. Prudent public health policy would make every effort to eliminate environmental releases of dioxin and related compounds.

Description of Dioxin

Dioxin belongs to a family of chemicals with related properties and toxicity. There are 75 different dioxins, or polychlorinated dibenzodioxins (PCDDs), 135 different furans, or polychlorinated dibenzofurans (PCDFs), and 209 different polychlorinated biphenyls (PCBs). Each different form is called a "congener."

Not all of the "dioxin-like" chemicals have dioxin-like toxicity, and the toxic ones are not equally toxic. Only 7 of the 75 dioxins, 10 of the 135 furans, and 12 of the 209 PCBs have dioxin-like toxicity. These 29 different dioxins, furans, and PCBs all exhibit similar toxic effects caused by a common mechanism: binding to a particular molecule known as the aryl hydrocarbon or "Ah" receptor (see Chapter 5 of the TSD). It is believed that the tighter the binding to the Ah receptor, the more toxic the chemical. The most potent member of this family is 2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD, which also has the greatest affinity for the Ah receptor.

The word "dioxin" is often used imprecisely. Some people restrict its use only to 2,3,7,8-TCDD, the most toxic and most studied dioxin. Others extend its use to the whole class of chemicals with similar toxicity and whose effects are controlled or triggered by the Ah receptor. In this report, the terms "dioxin" and "dioxins" are used to refer to any of the dioxin family members that bind to the Ah receptor and elicit dioxin like effects.

Toxic Equivalents

Although all dioxin-like compounds are thought to act in the same way, they are not all equally toxic. Their different toxicities may be due to their unique properties of absorption, distribution, metabolism, and elimination in a body and/or strengths of binding to the Ah receptor. Therefore, the health risk of each congener is assessed by rating their toxicities relative to TCDD, the most potent of the dioxins. TCDD is assigned a value of "1" and each of the 17 toxic dioxins/furans and 12 PCBs is assigned a "toxicity factor" that estimates its toxicity relative to TCDD. The resulting

estimates are called toxic equivalency factors (TEFs), which have been recently updated by the World Health Organization.1 The toxic equivalency (TEQ) is determined by multiplying the concentration of a dioxin congener by its toxicity factor. The total TEQ in a sample is then derived by adding all of the TEQ values for each congener. While TCDD is the most toxic form of dioxin, 90% of the total TEQ value results from dioxin-like compounds other than TCDD.
The TEQ system is not perfect, but it is a reasonable way of estimating the toxicity of a mixture of dioxin-like compounds. There is good experimental support for the assumptions that underlie the TEQ system.1,2 TEQs make it possible to take toxicity data on TCDD, a compound about which our knowledge is vast, and estimate toxicity for other compounds about which much less is known.

Sources of Dioxins
Dioxin is found everywhere in the world - in water, air, soil, and sediment - even in places where dioxin or dioxin-containing products have never been used. This broad distribution is evidence that the sources are multiple and that dioxins can travel long distances. Unlike most chemicals, dioxins have no intended use or value. Dioxins are unintended by-products of many chemical and combustion processes which involve chlorine. They get into the environment from industrial air emissions, wastewater discharges, disposal activities, and from burning material that contains chlorine. The EPA estimates that 2,745 grams (gm) TEQ released into the air each year.3 Municipal solid waste incinerators, secondary copper smelting, and medical waste incinerators are identified as the top three sources of dioxin released into the air. Combustion sources account for nearly 80% of air sources. Dioxins are also released to water, soil and into consumer products, but these sources are poorly defined and only a few estimates have been made. A list of EPA's dioxin sources is shown in Table 1. In developing a "national inventory" of dioxin sources, EPA only made estimates for 20 of 54
identified air source categories, due to their lack of confidence in the available data.3 Preliminary estimates are made for 12 of the 34 poorly defined source categories, but these estimates are not included in the national inventory. EPA assigned "negligible" emissions to another 11 of these source categories and made no estimates for another 8 source categories even though there is some evidence of emissions. Overall, EPA's confidence in the data used to define dioxin releases to air, water, land, and products is weak and underestimates dioxin releases. Source categories that are left out of EPA's dioxin inventory include iron ore sintering, polyvinyl chloride (PVC) production, accidental/structural fires, landfill fires, backyard burning, releases from petroleum refineries, asphalt mixing plants, and contaminated sites and other "reservoirs" of dioxin. Regrettably, there are apparently no efforts to collect such data from these and other sources. Some of these source categories, if included, would contribute substantially to the national inventory and significantly increase the amount of dioxins estimated to be released into the environment.

Environmental Fate
Dioxins enter the atmosphere either directly from air emissions or indirectly from

volatilization from land or water, or from resuspension of particles. Depending on temperature and each congener's vapor pressure, dioxins are present in air as particulates or vapor. The more chlorinated compounds tend to bind to particulates and are protected from chemical and sunlight degradation. This protection may account for their relative abundance in the environment.

Table 1
Inventory of Sources of Dioxin in the United States
Reference Year 1995 Central estimate
(gm TEQ/year)

Air:
Municipal waste incineration 1,100
Secondary copper smelting 541
Medical waste incineration 477
Forest, brush and straw fires 208
Cement kilns(hazardous waste burning) 153
Coal combustion 72.8
Wood combustion –residential 62.8
Wood combustion –industrial 29.1
Vehicle fuel combustion – diesel 33.5
Cement kilns (nonhazardous wasteburning) 17.8
Secondary aluminum smelting 17
Oil combustion - industrial/utility 9.3
Sewage sludge incineration 6
Hazardous waste incineration 5.7
Vehicle fuel combustion – unleaded 6.3
Kraft recovery boilers 2.3
Secondary lead smelters 1.63
Cigarette combustion 0.81
Boilers/industrial furnaces 0.38
Crematoria 0.24
Total 2,745

Products:
Pentachlorophenoltreated wood
25,000
Bleached chemical wood pulp and paper mills 24.1
Dioxazine dyes and pigments 0.36
2,4-Dichlorophenoxy acetic acid 18.4
Non-incinerated municipal sludge 7
Total 25,050

Land:
Non-incinerated municipal sludge 207
Bleached chemical wood pulp and paper mills 1.4
Total 208

Water:
Bleached chemical wood pulp and paper mills **19.5**
Source: USEPA 3

Airborne dioxins can be carried large distances downwind from their sources as well as contribute significantly to local deposition.4,5 Eventually, airborne dioxins settle onto soil, plants, and water where they enter the food chain. Dioxin will fall out onto crops that are fed to dairy cows and beef cattle where it accumulates in the milk and meat of these animals. Dioxin is attracted to and accumulates in fat. People who consume the contaminated meat and dairy products ingest substantial amounts of dioxin. When dioxin falls out onto waterways, it settles in sediments or remains suspended in the water for long periods of time because dioxins generally do not dissolve in water. Here too, the dioxins move up the aquatic food chain to fish and then into people.

Dioxin in Food
Americans accumulate harmful levels of dioxins in their bodies mostly through the ingestion of food. Some segments of the population, such as nursing babies and people who eat a diet high in animal fat or foods contaminated because of their proximity to dioxin release sites, are exposed to higher than average levels of dioxin.6 Others, such as Vietnam veterans and some chemical plant workers, have accumulated additional dioxins because of their exposure to Agent Orange or other dioxin-contaminated chemicals in the workplace.7 Approximately 90%,6,7 and perhaps as much as 98%, 8 of the dioxin that average Americans are exposed to comes from the foods they regularly eat. Because dioxins accumulate in fatty tissue, they are found mostly in meat, fish, and dairy products. Consequently, when people consume these foods, they also consume dioxins. As Table 2 shows, ground beef has the highest dioxin content, with 1.5 picograms per gram (pg/gram) which is equivalent to 1.5 parts per trillion (ppt), of all meats consumed by Americans. Depending on what and how much people eat, the average daily intake of dioxins for Americans is approximately 2.2 pg TEQ/kg body weight (bw),9 ranging from 1 to 3 pg TEQ/kg bw.10 Daily intake increases to 3 to 6 pg TEQ/kg bw if dioxin like PCBs are included. The ingestion of dioxin in common foods has resulted in widespread low-level exposure of the general population.

Table 2 - Dioxin Levels in U.S. Foods

Food Type	**Total TEQ (pg/gram food) (ppt)**
Ground beef	1.5
Soft blue cheese	0.7
Beef rib steak	0.65
Lamb sirloin	0.4
Heavy cream	0.4
Soft cream cheese	0.3
American cheese sticks	0.3
Pork chops	0.3

Bologna 0.12
Cottage cheese 0.04
Beef rib/sirloin tip 0.04
Chicken drumstick 0.03
Haddock 0.03
Cooked ham 0.03
Perch 0.023
Cod 0.023
Source: Schecter 26

Dioxin Body Burden Levels

The average daily intake of dioxin results in an average dioxin tissue concentration in Americans that ranges from 28 to 41 nanograms (ng) TEQ/kg lipids (fat) and from 36 to 58 ng TEQ/kg lipids if dioxin-like PCBs are included.11,12 A single national average of 28 ng TEQ/kg or 28 ppt was estimated as part of the most extensive survey of dioxin in humans, the National Human Adipose Tissue Survey (NHATS). This survey was first conducted by the EPA in 1982.13 In 1987, the survey was repeated, and the results suggest some decreases in average dioxin body burdens, but the decreases may be due to improved analytical methods or to other issues involving methods of study. For most congeners, including TCDD, the differences between 1982 and 1987 tissue levels are not statistically significant.14

In addition to measuring exposure to dioxin by its daily intake, exposure can also be estimated by measuring how much of it builds up in the body. This is referred to as the "body burden" and is defined as the total accumulation of dioxin at any one time per kilogram of body weight.11 For a person this would be how much they have accumulated up to the time of the testing. Using the average tissue concentrations from the studies above, estimated average dioxin body burdens range from 6 to 9 ng TEQ/kg body weight. If dioxin-like PCBs are included, the average dioxin body burden ranges from 8 to 13 ng TEQ/kg body weight.11 In these estimates, TCDD contributes approximately 15% of the total TEQ. These estimates represent average body burdens for a middle-aged person. Approximately 10% of the population can be expected to have at least three times this level and others as much as seven times these levels. These high exposure groups include nursing infants, children, someworkers and farmers, people who rely on fish as a main staple of their diet such as some indigenous peoples and some fishermen, and people who live near dioxin-contaminated sites or dioxin-producing facilities. These groups have suffered a disproportionate share of dioxin exposure and many have already suffered the adverse health effects caused by these exposures. Indigenous peoples, for instance, who eat fish and sea mammals from the Arctic regions are exposed to dioxin at higher than average levels because dioxin and PCB levels are particularly high in these foods.15 Dairy cows, meat cattle, or other animals fed crops grown on soil contaminated with dioxin in the low part per trillion (ppt) levels accumulate significant amounts of dioxins.16 An incinerator in the Netherlands that emitted large amounts of dioxins contaminated milk from cows grazing nearby. This milk was so contaminated that it was declared to be hazardous waste by the Dutch

government.17 On the other hand, vegetarians, who consume less meat and dairy products, have below-average body burden levels of dioxin.18

Dioxin in Breast Milk

Dioxin accumulates in breast milk because it readily dissolves in the milk's rich fat content. During nursing, dioxin is transferred from mother to baby 19, 20, 21, 22, 23, 24 who may absorb as much as 95% of the dioxin in the milk.19, 20 Several studies reporting dioxin in human breast milk indicate that levels range from 20 to 30 ng TEQ /kg lipids in industrial countries and from 3 to 13 ng TEQ/kg lipids in less industrialized countries (Table 3).6, 7 The World Health Organization (WHO) reports a worldwide mean of 20 ng TEQ/kg lipids, with values ranging from a low of 3.1ng TEQ/kg lipids to a high of 110 ng TEQ/kg lipids.25

Table 3 - Dioxin Levels in Pooled Breast Milk Samples from Various Countries

Country	Total TEQ (ng/kg, lipid)
Vietnam - Da Nang	34
Japan	27
Germany	27
Canada	26
USA	20
Vietnam - Ho Chi Minh City	19
South Africa - White	13
Pakistan	13
Russia	12
South Africa - Black	9
Vietnam - Hanoi	9
Thailand	3
Cambodia	3

Source: Schecter, 1994 7

Nursing infants ingest considerably more dioxins each day than adults. Studies in the U.S. and in the Netherlands have estimated daily intake of dioxins according to infant age. The U.S. study found that nursing infants typically consume between 35 and 53 pg TEQ/kg body weight (bw) per day in breast milk.26 The more current Dutch study found that nursing infants typically consume about 112-118 pg TEQ/kg bw/day.27 If the Dutch study is correct and infants consume dioxin at the rate of about 112-118 pg TEQ/kg bw/day, and adults typically ingest between 3 and 6 pg TEQ/kg bw/day,10 then nursing infants consume about 50 times more dioxin per day than adults, confirming results from other studies.15, 26 It is estimated that approximately 10-14% of total lifetime exposure can occur via nursing.27, 28Breast-fed babies accumulate far more dioxins than do formula-fed babies. In one study, dioxin intake was 50 times greater in breast-fed infants than it was in formula-fed infants.23 In this same study, TEQ concentrations in blood from 11 month old formula-fed infants were less than one fourth the concentrations of the mother's blood and about 10 times less than the concentrations in infants that are

breast-fed for six to seven months. Although nursing infants are at increased risk because of their higher intake of dioxins, extensive studies in the Netherlands indicate that the benefits of nursing outweigh the risks. Breast milk contains all the nutrients in ideal proportion for optimum growth and development; the psychological benefits of nursing are invaluable; 29 and breast-fed babies have fewer respiratory illnesses, fewer skin problems, cry less, have fewer allergies, and are less constipated than other babies. For these and other reasons, despite the dioxin levels found in breast milk today, the World Health Organization (WHO) and the federal Agency for Toxic Substances and Disease Registry (ATSDR) both promote and support breast feeding.6, 10

A "Safe" Level of Dioxin

Three separate government agencies have established a "safe" or tolerable daily dose of dioxins.These guideline values are shown in Table 4. The table also shows how much dioxin exposure is "allowed" according to the guideline. This value is determined by multiplying the guidelinevalue, in picograms per kilogram (pg/kg) of body weight, by the body weight of an average person, which is typically 70 kilograms or about 150 pounds. For example, to convert ATSDR's Minimum Risk Level, multiply 1 pg/kg by 70 kg which results in 70 pg. This means that any daily intake greater than 70 pg would exceed the lowest measure of safety set by ATSDR. As thetable shows, the average daily dioxin intake of the American people exceeds the two federal government guidelines and is within the range of the international guideline. This average daily intake is more than 200 times higher than EPA's guideline, over twice ATSDR's guideline, and in the middle of the range of the WHO guideline.

Table 4 - Daily Intake of Dioxin (TEQ) Compared to Established Guidelines

Guideline (pg/kg bw/day) Equivalent Intake for 70 kg adult (pg/day)

USEPA Risk Specific Dose 30 0.01 0.70

ATSDR Minimal Risk Level 6 1.0 70.0

WHO Tolerable Daily Intake 10 1-4 70-280

Average Daily Intake of Dioxin in Food in the U.S. 2.2 154

Range of Daily Intake of 1-3

70-210 Dioxin in Food in the U.S.

Range of Daily Intake of Dioxin and Dioxin- Like PCBs in Food in the U.S.
3-6 210-420

According to the EPA, the American people's lifetime risk of getting cancer from exposure to dioxin is 1 in 10,000.30 The risk attributable to dioxin for highly exposed members of the population is 1 in 1,000. These risk estimates are based on ingesting a "risk specific dose" of 0.01 pg TEQ/kg bw/day over a 70-year lifetime. At this dose, there will be one additional cancer for every one million exposed people. One cancer per million is often considered an "acceptable risk" value.31 Since the average daily intake of dioxin ranges from 1 to 3 pg/kg bw/day (3-6 pg/kg bw/day if dioxin-like PCBs are included), everyday the general American public is exposed to a cancer risk that is 100 to 300 times higher than the one-in-a-million "acceptable"
cancer risk. Table 4 shows that the American people are already well above several federal and international guidelines for dioxin exposure as well as the typical "acceptable" cancer risk value."

Safe" Body Burdens

The biological effects of a toxin depend on the concentrations of that substance in a target organ over a critical period of time. These concentrations in turn depend on three important factors: the absorption, distribution and persistence of the toxin throughout the body. These factors help determine a person's lifetime accumulation, or body burden, of dioxin. As discussed earlier, body burden is the concentration of a substance in tissue or blood per kilogram of body weight. Because body burden measurements account for differences in absorption, distribution and persistence across species and between individuals, 32 they can be used to compare the doses needed to produce similar adverse effects in different species. Such a comparison was made by the World Health Organization which made a list of the most sensitive adverse health effects associated with exposure to dioxin in animals. These health effects, which are shown in Table 5, are primarily effects on the reproductive and immune systems. The WHO found that the lowest observed adverse effect levels (LOAELs), which ranged from 10 to 73 ng/kg, are all within a factor of 10 of the average body burden of 10 ng/kg in the human population. The USEPA made a similar comparison in their draft reassessment report in 1994.30 The EPA included sensitive adverse effects in people, which are included at the bottom of Table 5. This table shows that adverse effects are occurring in some people with body burden levels similar to those that produce adverse effects in animals. The table also shows that the average body burden levels in the general American population is just below the levels that are causing adverse effects in animals. Comparisons have also been made between the body burden levels of dioxin in animals and people that result in cancer. The body burden levels of dioxin at which exposed workers and experimental animals have higher numbers of cancers are similar. For both the workers and experimental animals, these body burden levels are also substantially higher than the body burden levels of dioxin in the general human population.12, 25

Table 5 - Animal Body Burden Levels Associated with Sensitive Adverse Effects
Body Burden (ng/kg)
Species Health effect (reference)
10 Mice Adult immune suppression 33

28 Rats Decrease in sperm count 34
42 Monkeys Endometriosis 35
42 Monkeys Object learning 36
50 Rats Immune suppression 37,38
73 Rats Genital malformations (females) 39
14 Humans Altered glucose tolerance 40
14 Humans Decreased testis size 41
83 Humans Decreased testosterone 42
10 Current average body burden in the general U.S. population
Source: WHO, 1998; 10 USEPA, 1994 30

Sensitive Non-Cancer Effects Observed in the General Population

The Dutch Studies - Neurodevelopmental and Immune Effects

Main findings: Four point deficit in IQ and increased susceptibility to infections in 42 month old children exposed to typical daily intake levels of dioxins/PCBs.

Effects of dioxins and PCBs on neurodevelopment, the immune system and thyroid hormones were observed in children from the general population of the Netherlands.43, 44 These studies found that prenatal (before birth) exposure to typical daily intake levels of dioxins/PCBs are associated with:

• Reduced birth weight and reduced growth from birth through 3 months of age; 45
• Delays in psychomotor development at 3 months; 46
• Neurodevelopmental delays at two weeks 47 and 18 months; 48
• Alterations in thyroid hormones at birth and at 3 months; 49 and
• Alterations in immune status from birth to 42 months.50, 51

The adverse neurological effects found at birth and at 18 months could not be detected at 42 months.52 However, a decrease in cognitive function as measured by a 4 point deficit in IQ was measured for the first time at 42 months.53 This difference may be explained by the different testing procedures used. Prenatal exposure to dioxins/PCBs were also found to be associated with other neurodevelopmental and behavioral effects at 42 months including a decrease in high level play, 54 an increase in non play activity, 54 and an increased prevalence of being withdrawn
and depressed.55 These Dutch studies also found that postnatal (after birth) exposure to typical daily levels of dioxins/PCBs was associated with:

• Delays in psychomotor development at 7 months; 46
• Alterations in thyroid hormones at 3 months; 49
• Alterations in immune status as indicated by an increased prevalence of recurrent middle ear infections and decreased prevalence of allergic reactions to food, pollen, dust and pets at 42 months; 51 and
• An increase in mean reaction times, a decrease in sustained attention, and an increase in hyperactive behavior at 42 months.54

The Finnish Study - Developmental Effects Main findings:

An association between dioxin exposure and hypo-mineralization defects of permanent teeth. A study of breast-fed Finnish children found an association between dioxin exposure and hypomineralization defects of permanent teeth.56, 57, 58 These findings suggest that the observed effects are primarily due to lactational

exposures. In contrast, the effects observed in the Dutch children were associated primarily with *in utero* exposure and not in children who were breast-fed. Teeth defects are also observed in the rice oil poisonings in both Japan 59 and Taiwan.60 There are some toxicological data in animals to support effects of dioxin on tooth development. Dioxin causes defects of dental hard tissues in rats, 61 perhaps by altering the action of epidermal growth factor receptor.62 Dental defects and changes in ameloblasts (enamel-forming cells) in rhesus monkeys exposed to PCBs have been reported.63

Miscellaneous Studies - Neurodevelopmental and Reproductive Effects

Two studies of children in the U.S. found similar neurodevelopmental effects associated with exposure to typical daily exposure levels of PCBs.64, 65 An ongoing German study also found neurodevelopmental effects associated with low-level PCB exposure.66 Some of the results differ among these studies. In a study of children from the general Japanese population, exposure to dioxin-like compounds are associated with adverse effects on thyroid hormones and the immune system.67, 68 Children of women exposed *in utero* to a complex mixture of PCDFs, PCBs and other compounds in the Taiwan rice oil poisoning incident of "Yu-cheng"(which translates to oil poisoning), suffered a number of effects including damage to the nervous and respiratory system;69 higher than normal incidence of middle ear infections; 70 and reduced penis size at adolescence.71

In Seveso, Italy, the site of a major plant explosion that sent a cloud of dioxin into the community, children who developed chloracne experienced transient changes in immune parameters, but no adverse immunological effects.72 Also, the sex ratio of children born (48 females to 26 males) in Seveso was not normal for several years following dioxin exposure, 73 but the same effect is not seen after dioxin exposure in the Yu-cheng children.74 Though a major study of women exposed to dioxin at Seveso is underway, the existing epidemiological evidence showing the effect of dioxin exposure on endometriosis is limited and mixed. One study in Israel found higher levels of dioxin in the blood of women with endometriosis than in controls.75 Workers with chloracne who worked at the Nitro, West Virginia trichlorophenol plant reported higher than expected sexual dysfunction and lower than normal libido.76 In summary, some evidence indicates that dioxin exposure interferes with normal growth and development in children from the general population. Developmental neurotoxicity associated with dioxin exposure includes cognitive deficits, behavioral alterations such as increased withdrawal/depression, hyperactive behavior, and attentional difficulties. Other effects that are transient are decreased neuro-optimality (nerve function) and decreased psychomotor ability. Developmental effects on the immune system include increased susceptibility to infections, altered lymphocyte subsets, and increased respiratory disease and otitis (inflammation of the ear) in highly exposed infants. Developmental and reproductive effects include altered sex ratio (more females born than males), small penis and endometriosis. Many of the effects on the development of the nervous system are more associated with *in utero* exposure than with breastfeeding.

The dental effects observed in the Finnish children are more strongly associated with dioxin exposure from breast milk, a finding consistent with the timing of tooth

mineralization in humans.

Hormonal Effects

Major findings: Decrease in testosterone in workers and an increased risk of diabetes associated with exposure to dioxin. Exposure to dioxin has a variety of effects on hormone function in animals and in people. In a group of U.S chemical plant workers (the NIOSH cohort), dioxin-exposed workers have lower than normal testosterone levels and higher than normal follicle-stimulating and luteinizing hormone levels, both of which can reduce sperm counts.42 Dioxin interferes with the hormone insulin and alters glucose tolerance which leads to diabetes. In one study of 55 exposed workers evaluated 10 years after exposure, 50% of the workers were diabetic or have abnormal glucose tolerance, an early indicator of diabetes.77 Since this striking finding, there have been mixed findings of diabetes or glucose tolerance in several studies. In the NIOSH workers, the risk of diabetes increased 12% for every 100 ppt dioxin in blood lipid.78 In a study of the Ranch Hand veterans, the soldiers who had the highest exposures to Agent Orange, those with blood dioxin greater than 33.3 pg/gm (ppt) have a relative risk of 2.5 for diabetes.41 A relative risk of 1.0 means that an exposed person is no more likely to develop the disease than an unexposed person. In a follow-up study, the veterans exposed to dioxin had a relative risk of 1.4 for glucose abnormalities, 1.5 for diabetes, and 2.3 for the use of oral medications to control diabetes.79 This study also found that Ranch Hand veterans exposed to dioxin develop diabetes at an earlier age than other veterans and that non-diabetic Ranch Hands exposed to dioxin have a relative risk of 3.4 for serum insulin abnormalities. In the ongoing study of the residents of Seveso, Italy, there is an increase in deaths from diabetes in females in the second highest exposure area and a slightly elevated increase (not statistically significant) in males.72 Deaths from diabetes in the highest exposed area showed a suggestive but not statistically significant increase, though the number of deaths are too few to draw any conclusions.

Cancer Effects

Epidemiological data from high exposure situations suggest that a number of the effects of dioxin exposure seen in animals also occur in humans . However, because studies in humans cannot be done under the same controlled conditions as studies in experimental animals, dioxin's effects on humans are not as clear cut as they are in animal models. Nevertheless, similarities between humans and experimental animals allow reasonable comparisons and projections from dioxin's effects in animals to its effects on humans: they both have the Ah receptor and associated factors; a number of biochemical responses are similar; and, on a body burden basis, many human responses to dioxin are reasonably comparable to the responses in animals.11, 12

Updates of ongoing studies indicate that dioxin exposure causes cancer in humans in a dose dependent fashion. The most important of these studies are the series of studies by Flesch-Janys and colleagues in Germany and by Bertazzi and colleagues in Italy. The studies of the German chemical plant workers attempt to quantify the dose-response relationship between estimated TCDD exposure and total

mortality.80,81 The Italian studies of mortality among those exposed to the Seveso plant accident also focus on cancer mortality in populations grouped by exposure level.82 Both research groups recognize limitations and uncertainties in their studies including estimating exposure and defining specific causes of death, among other limitations of epidemiologic studies. However, both series of studies strengthen the conclusion that dioxin exposure is related to cancer mortality in humans in a dose-related fashion.

Two additional important studies are the update of the NIOSH chemical workers in the U.S.83 and analysis of a group of Dutch workers 84 that is part of a larger international group of workers.85 The NIOSH update also shows a dose-response relation between dioxin exposure and cancer mortality. These studies together provide strong support for the decision by the World Health Organization's International Agency for Research on Cancer (IARC) to define TCDD as"carcinogenic to humans."25 In making an overall judgment of dioxin's carcinogenicity in humans, IARC now includes mechanistic information as well as human and animal data. For example, the importance of the Ah receptor in mediating dioxin's toxic effects and its presence in both humans and experimental animals is acknowledged. This decision is further supported by strong evidence in animal studies that show dioxin causes cancer in all studies that have been conducted. The U.S. National Toxicology Program (NTP) had upgraded dioxin from its status as "reasonably anticipated to be a human carcinogen" to "known to cause cancer in humans" in 1997,86 but reconsidered their decision based on procedural errors pointed out by industry. NTP has not decided whether they will upgrade dioxin or leave it as "reasonably anticipated to be a human carcinogen."
As discussed earlier, the lifetime risk of getting cancer from exposure to dioxin is 1 in 10,000 for the general American population and 1 in 1,000 for highly exposed members of the population.30 These risk estimates are based on ingesting a "risk specific dose" of 0.01 pg TEQ/kg bw/day over a 70-year lifetime. If these estimates are taken seriously, then the average exposure of the American people to dioxin poses an uncertain but potentially substantial risk, a point made at least a decade ago.87

Sensitive Non-Cancer Effects Observed in Animal Studies
Studies of dioxin's effects in experimental animals indicate that it causes a host of toxic effects including cancer; reproductive and developmental toxicity; damage to the immune system; neurotoxicity; endocrine disruption; liver and skin toxicity. Among the sensitive effects observed in animals are a number of biochemical and cellular effects that occur at body burden levels of about 10 ng/kg or less, levels comparable to those found in the average person.88 These effects include production of the liver enzymes CYP1A1 and CYP1A2; alterations in hormones, such as
epidermal growth factor (EGF), that affect growth and development; oxidative damage; and alterations in lymphocyte subsets,12 a measure of immune function. These observations suggest that dioxins cause biological effects at levels comparable to those found in the average American. At present, it is unclear if these effects are

adverse or not.

Developmental neurotoxicity: Subtle deficits in object learning are observed in the offspring of rhesus monkeys chronically exposed to dioxin *in utero* and from breast milk.36 Similar exposure to dioxin also adversely affects long-lasting learning and memory in rats.89 In this study, deficits in exposed animals of both sexes for different learning tasks were observed. Some of these tasks may represent a response strategy rather than improvement in learning or memory.

Endometriosis: The incidence and severity of endometriosis in rhesus monkeys chronically exposed to dioxin rises as the dose increases.35 Surgically-induced endometriosis has been enhanced in dioxin-exposed monkeys 90 and in rats and mice.91 In human endometrial tissue, the Ah receptor is expressed, suggesting that it is involved during the reproductive phase of this tissue.92

Effects on the Developing Reproductive System: Pregnant rats exposed to a single dose of dioxin during the development of fetus' organs give birth to both male and female offspring with permanent damage to their reproductive systems.34, 39
Immunotoxicity: Pregnant female rats exposed to dioxin give birth to offspring with an immune system problem called "delayed type hypersensitivity" 37, 38 which renders the animals more susceptible to viral infections. Captive harbor seals fed Baltic fish with 210 ng TEQ/kg lipid in their blubber develop delayed type hypersensitivity relative to controls which were fed cleaner Atlantic fish with only 62 ng TEQ/kg lipid in their blubber.93 The seals fed the contaminated fish were less able to mount a normal immune response. Eight week old mice treated with 10 ng/kg of dioxin die more frequently than controls when exposed to influenza virus.33 This viral susceptibility occurs at the lowest level of any effect observed in animals. This represents the most sensitive adverse effect of dioxin exposure on record.

Conclusion: The American People are at Serious Risk from their Daily Intake of Dioxin in Food

This report integrates all the information including the newest studies on dioxins' effects on human health and comes to the following conclusions:

- All American children are born with dioxin in their bodies. The greatest impact appears to be on the growth and development of children. Disrupted sexual development, birth defects and damage to the immune system may result.
- Dioxin exposure has been associated with IQ deficits, increased prevalence of withdrawn/depressed behavior, adverse effects on attentional processes, and an increase in hyperactive behavior in children. These effects have been reported in 42-month old Dutch children whose exposure to dioxins/PCBs came primarily before birth.
- Dioxin exposure has been associated with alterations in immune function including increased susceptibility to infections and changes in T-cell lymphocyte populations.

These effects have been reported in 42-month old Dutch children whose exposure

todioxins/PCBs came primarily before birth. Altered immune function, reported at birth, 3, and 18 months of age, persists to 42 months of age in these children. Reported immune effects include an increase in middle ear infections and chicken pox, and a decrease in allergic reactions.

• There is evidence of both developmental and reproductive effects in children exposed to dioxin. These effects include defects in permanent teeth, adverse effects on thyroid hormones, altered sex ratio (more females than males), and increased respiratory disease.

• The average daily intake of dioxin in food poses a substantial cancer risk to the general American population. The lifetime risk of getting cancer from exposure to dioxin is 1 in 10,000 for the general American population and 1 in 1,000 for highly exposed members of the population. These risks are 100 and 1,000 times higher, respectively, than the onein- a-million "acceptable" cancer risk.

• Nearly all Americans are exposed to dioxin through ingestion of common food, mostly meat and dairy products. Dairy cows and beef cattle absorb dioxin by eating dioxin contaminated feed crops. The crops become contaminated by airborne dioxins that settle onto soil and plants. Dioxins enter the air from thousands of sources including incinerators that burn medical, municipal, and hazardous waste.

• The average daily intake of the American people is already well above several federal guidelines and at mid-range of international guidelines for dioxin exposure. The average daily intake of the American people is more than 200 times higher than EPA's cancer risk guideline, over twice ATSDR's lowest adverse effect level, and in the middle of the range of the World Health Organizations's tolerable food intake.

• At higher risk of exposure to dioxin are children, nursing infants, some workers and farmers, people who eat fish as a main staple of their diet such as some indigenouspeoples and fishermen, and people who live near dioxin release sites. These groups of people are likely exposed to at least 10 times as much dioxin as the general population. Dioxin is an ubiquitous poison that is in our food and that causes many toxic effects in people and animals. The neurodevelopmental and reproductive effects observed in children may be the most disturbing new evidence. The small shifts in cognitive ability or thyroid levels may be the tip of the iceberg of the impact of dioxin on the general American public. We know that the daily dioxin intake of Americans is already too high, and exceeds several federal risk guidelines. We also know that some members of the general population are particularly sensitive to exposure to dioxin and others are exposed to higher than average daily levels. These are infants and children, people who live near contaminated sites, fishermen and indigenous people who rely on fish as a main staple of their diet, workers, and others with high exposures. These groups have suffered a disproportionate share of dioxin exposure and many have already suffered the adverse health effects caused by these exposures. Every effort should be made to eliminate environmental releases of dioxin and related compounds. Americans have a choice: take action to protect public health by eliminating dioxin creation or continue to allow dioxin to be created and not burden industry with the short term transition costs of elimination and related compounds.

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Perinatal Exposure to Low Doses of Dioxin Can Permanently Impair Human Semen Quality

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ELEVATED DIOXINS IN PROXIMITY TO INCINERATOR

Air emissions from waste incinerators have been positively identified as a cause of cancer and other health damage in humans.
The incineration of solid waste leads to air emissions containing heavy metals, dioxins, and other volatile organic compounds [39-41].
Although many waste incinerators studies of communities living in the vicinity (i.e: 0.5km to 5km) of municipal solid waste incinerators have shown elevated levels of dioxins in blood samples, compared to background population levels have now been equipped or updated with improved air pollution control techniques, toxic emissions are still being released to the atmosphere. At optimum operating levels, these emissions are small, but incinerators rarely perform to optimum, or even required, standards [39-41].

39. Carman, N., Incinerators for combustion, C. officials, Editor. 1995, Sierra Club:Austin,Texas.

40. Brady,T., Proposed new incinerators, K.E.G.I. Mr Rob Lindsay, Editor. 1999, Environment and Business Group: Auckland, New Zealand.

41. Connett, P. and E. Connett, Municipal Waste Incineration - Wrong Question,Wrong Answer.The Ecologist, 1994. 24(1): p. 14-19.

These pollutants may be transported for considerable distances downwind from incinerators, with significant pollutant levels being measured within several kilometres of a facility [42].

42. Greenpeace International, Incineration and Human Health: State of knowledge of the impacts of waste incinerators on human health.

Emission violations and malfunctions are common even at new, state-of-the-art incinerators due to mechanical and operational problems, and it is "technically remote to achieve even 80% continuous compliance" with air emission regulations [39].

39. Carman, N., Incinerators for combustion, C. officials, Editor. 1995, Sierra

Club:Austin,Texas.

Dioxins are the most toxic man-made substances and are "formed from precursors that are either constituents of the waste or are also formed by chemical recombination of materials in the waste" [42].

42. Greenpeace International, Incineration and Human Health: State of knowledge of the impacts of waste incinerators on human health. 2001:Amsterdam.

The predominant source of dioxins is products containing chlorine, such as PVC plastic. Dioxins can be destroyed during combustion in an incinerator but can also be regenerated by processes in the post-combustion zone. It has been shown that the total amount of dioxins exiting an incinerator in various forms can exceed the amount entering as raw waste [41,42].

Even small quantities of pollutants such as dioxins, furans, and mercury can be detrimental to human health and the environment. Many of these substances (dioxins in particular) can be carried long distances from their emission sources, persist for decades in the environment without breaking down into less harmful compounds, and accumulate in soil, water, and food sources [43].

43. N.R.C., Waste Incineration and Public Health. 2000, National Academy Press: Washington, D.C.

Small amounts of toxic substances can gradually build-up in the tissues of organisms to reach critical and fatal levels. Therefore, even tiny emission levels of these substances are unacceptable and slowly but surely lead to the eventual poisoning of communities and ecosystems.
Incinerator workers are exposed to high concentrations of dioxins and other toxic substances resulting from in-plant waste combustion emissions, regardless of the standard protective equipment worn [42-45].

42. Greenpeace International, Incineration and Human Health: State of knowledge of the impacts of waste incinerators on human health. 2001:Amsterdam.
43. N.R.C., Waste Incineration and Public Health. 2000, National Academy Press: Washington, D.C.
44. Pilspanen,W.H., J.M. Czuczwa, and I.M. Sobeih,Work area air monitoring for chlorinated dioxins and furans at a municipal waste power boiler facility. Environmental Science and Technology, 1992. 26: p. 1841-1843.
45. National Institute for Occupational Safety and Health (NIOSH), NIOSH

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Populations living near incinerators are also at risk of health impacts from toxic air emissions, particularly those living downwind who receive the most toxic "fallout". Although emissions may be diluted and dispersed over space and time, the chronic exposure to low-level doses of environmentally persistent, toxic substances has the potential to cause human health issues after a long latency period.
Studies of communities living in the vicinity (ie: 0.5km to 5km) of municipal solid waste incinerators have shown elevated levels of dioxins in blood samples, compared to background population levels [42]. High concentrations of dioxins have also been found in dairy products and vegetable crops originating from agricultural areas near incinerators [42], and intake of these food items contributes to increased dioxin levels in humans. Therefore, the impact of an incinerator may be far-reaching if toxin-laden produce is exported outside the local community.
Polyaromatic hydrocarbons (PAHs), polychlorinated biphenyls, dioxin, and other commonly emitted substances from incinerator stacks have been classified as human carcinogens or likely/possible human carcinogens [46]. In particular, soft tissue sarcoma, non-Hodgkin's lymphoma, lung cancer, liver cancer, and cancer of the larynx have been positively linked to exposure to incinerator emissions [42, 47].

42. Greenpeace International, Incineration and Human Health: State of knowledge of the impacts of waste incinerators on human health. 2001:Amsterdam.
47. Viel, J.-F., et al., Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around a municipal solid waste incinerator with high dioxin emission levels.American Journal of Epidemiology, 2000. 152: p. 13-19.

Children are particularly vulnerable to toxic reuse and develop cancers after only short exposure times [48].

48. Knox, E.G., Childhood cancers, birthplaces, incinerators and landfill sites. International Journal of Epidemiology, 2000. 29: p. 391-397.

Increased congenital abnormalities, such as orofacial clefts, spina bifida, and genital malformation in infants have also been attributed to incinerator emissions, particularly to dioxin [42].

42. Greenpeace International, Incineration and Human Health: State of knowledge of the impacts of waste incinerators on human health. 2001:Amsterdam.

Ash is a product of incineration. Bottom ash consists of post-combustion waste residues and non-combusted materials, including heavy metals, and fly ash is composed of particles captured in air filters. Reducing toxins in air emissions results in increasing levels of toxins captured in fly ash, which will eventually leach into soil and water from landfill ash deposits. Attempts have been made to divert ash from landfill by incorporating it in roading and cement block construction, with incinerator operators claiming that the ash consequently becomes inert. Research has shown that this is not the case, and heavy metals in particular are leaching from roading material and cement blocks incorporating incinerator ash, endangering local ecosystems and communities [49]. In one case in Newcastle, UK, where ash from a local incinerator had been applied from 1994-1999 on local allotments and paths, hazardous levels of dioxins and heavy metals were found. Amongst other warnings, residents were advised to keep infants off the allotments and refrain from eating egg and animal produce from the area [50].

49. Ryder, R.E., Incinerator Ash is Inert.ToxCat, 2000. 3(1).
50. ENDS, Regulatory foul-ups contributed to Byker ash affair. 2000. p. 17-18.

13.3. Health Effects from Living Near an Incinerator: A UK study found that there was an increased risk of lethal congenital anomaly such as spina bifida and heart defects for women living near incinerators(72a). Two other studies (72bc) showed a significant increase in the risk of sarcoma, correlated both with the level and the length of environmental modeled exposure to dioxin-like substances. The risk excess is also evident in females, and, for both sexes taken together, for cancers of the connective and other soft tissue. Another study found that exposure to high levels of dioxin was correlated to significantly lower boy to girl birth ratio(72d). An ATSDs investigation found an increased pattern of respiratory problems in community residents living near an incinerator(72e). In addition, residents complained about irritation of the airways and poor motor coordination. Another study's results indicated a significantly increased risk of mortality among women living in the vicinity of the incinerators compared with those living far away, for all causes, colon and breast cancer, diabetes, and cardiovascular diseases(72f).

(72) (a) Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, north west England, 1956–93 ; T J B Dummer, H O Dickinson, L Parker; J Epidemiol Community Health 2003;57:456-461; & (b) Sarcoma risk and dioxin emissions from incinerators and industrial plants: a population-based case-control study (Italy), P. Zambon et al, *Environmental Health* 2007, **6:**19, http://www.ehjournal.net/content/6/1/19; & (c) Risk of soft tissue

sarcomas and residence in the neighbourhood of an incinerator of industrial wastes, P. Comba et al, *Occup Environ Med* 2003;60:680-683; & (d) Birth Sex Ratio and Dioxin exposure, P. Mocarelli et al, The Lancet, May, 2000; (e) & Agency for Toxic Substances and Disease Registry, U.S. DOH, 1994, http://www.atsdr.cdc.gov/testimony/testimony-1994-07-08.html; & (f) Incinerator and Spatial Exposure Distribution: An Example of Small Area Study in Italy, A Ranzi et al, Epidemiology: November 2006 - Volume 17 - Issue 6 - pp S114-S115

Dioxins: An overview . . . Birnbaum

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- J Toxicol Environ Health A. 2002 Dec 13;65(23):1937-43.

- **Dioxins in commercial United States baby food.**

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- **Abstract**
- This is the first known study of dioxins, dibenzofurans, and polychlorinated biphenyls (PCBs) in commercial American bottled baby foods purchased in the United States. Dioxins, persistent chlorinated organics, are inadvertent by-products of chemical synthesis or combustion and are toxic to humans and other animals. Almost all dioxins enter the body through food consumption, specifically from food products containing animal fat. Major-brand bottled baby food containing meat was purchased at U.S. supermarkets and 12 pooled samples were analyzed for dioxins using high-resolution gas chromatography with high-resolution mass spectrometry. Low levels of dioxins were found in these products. The range was from 28 to 226 parts per quadrillion (ppq) dioxin toxic equivalents (TEQ). This is reported on a whole or wet weight (as eaten) basis. As a comparison, findings of dioxins in U.S. supermarket meat ranged from 28 to 540 ppq. Although

dioxin levels are generally lower in these baby foods than in meat or poultry, the presence of dioxins in commercial baby food containing meat is cause for concern.

J Toxicol Clin Toxicol. 2002;40(4):449-56.

Chronic effects of toxic environmental exposures on children's health.

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Source

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Abstract

Children have unusual patterns of exposure to environmental chemicals, and they have vulnerabilities that are quite distinct from those of adults. Increasingly, children's exposures to chemicals in the environment are understood to contribute to the causation and exacerbation of certain chronic, disabling diseases in children including asthma, cancer, birth defects, and neurobehavioral dysfunction. The protection of children against environmental toxins is a major challenge to modern society.

Dioxins in organic eggs: a review

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Abstract

Eggs contribute for about 4% to the daily dioxin intake of humans. Research among layer farms in the Netherlands and other EU countries has shown that organic eggs contain more dioxin than conventional

ones and that a significant number of organic farms produce eggs with a dioxin content that exceeds the EU standard. The hens' intake of dioxins from various sources leads to an increase in the dioxin content of organic eggs. These sources include plants, feed, soil, worms and insects, and compared with hens on conventional and free-range farms, organic hens make more use of these sources due to better access to the outdoor run. Plants appear to be relatively unimportant as a source of dioxins. Also commercial organic feed generally has very low dioxin contents, but not much is known about non-commercial feed. Consumption of worms and insects and particularly ingestion of soil are important causes of high dioxin levels in eggs. Management interventions, like a reduction of the time the

hens spend outside,
may decrease the dioxin levels in organic eggs but at the same time may interfere with the image of the organic production system.

*** Steven Lester, Science Director for The Center for Health, Environment & Justice, points out "that the average backgrond exposure of the American public to dioxin in food is very close to or above he EPA new reference dose."

April 19, 2010

The Honorable Dee Freeman
Secretary
N.C. Department of Environment and Natural Resources
1601 Mail Service Center
Raleigh, NC 27699-1601

Dear Secretary Freeman:
In recognition of the numerous and serious adverse health consequences that can result from human exposure to the components of emissions of biomass burning, the North Carolina Academy of Family Physicians (NCAFP) is issuing a letter of concern regarding the development of biomass burning plants in the State of North Carolina.

Biomass burning of poultry litter and wood wastes creates emissions of particulate matter that research has shown increase the risk of premature death, asthma, chronic bronchitis, and heart disease. (1, 2) This burning process also creates numerous byproducts, including nitrogen oxides and volatile organic compounds that increase smog and ozone, which are known to increase lung disease and mortality (3); sulfur dioxides which also contribute to respiratory disease (4); arsenic which can increase the risk of cancer (5); mercury which can increase the risk of brain and kidney disease and affect the developing fetus (6); and dioxins which may increase the risk of cancer, heart disease, diabetes mellitus, developmental delays in children, neurotoxicity, and thyroid disease (7). These health effects would increase disability and death in all age groups, but particularly in the most vulnerable—developing fetuses, newborns, children, those with chronic illness, and the elderly. As a result of this increased disability and disease, medical costs in the state will increase.

One of the reasons for encouraging renewable energy through legislation like the North Carolina Clean Smokestack law was to provide cleaner air for citizens. However, there is concern that burning of poultry litter may result in similar or greater emissions of nitrogen oxides, particulate matter, carbon monoxide, and carbon dioxide to coal- burning plants (8).

The NCAFP requests that the North Carolina Department of Environment and Natural Resources strongly consider the potentially harmful consequences to the health and wellbeing of North Carolina citizens when contemplating the permitting of biomass burning plants in the state.

With best regards,
R.W. (Chip) Watkins, MD, MPH
President, NC Academy of Family Physicians

cc: Jeffrey P. Engle, MD, North Carolina State Health Director
Jennifer L. Mullendore, MD, Co-Chair, NCAFP Health of the Public Council
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AMERICAN ACADEMY OF
FAMILY PHYSICIANS

STRONG MEDICINE FOR AMERICA

April 23, 2010

The Honorable Dee Freeman
Secretary
N.C. Department of Environment and Natural Resources
1601 Mail Service Center
Raleigh, NC 27699-1601

Dear Secretary Freeman:

As the Executive Vice President and Chief Executive Officer of the American Academy of Family Physicians (AAFP), and as a North Carolina native, I am writing today to echo the concerns raised recently with your office by the North Carolina Academy of Family Physicians (NCAFP) in their letter of April 19 (enclosed). The AAFP, representing over 94,700 family physiciansand medical students, supports strong action to limit and correct the pollution of our land, atmosphere and water that threatens the health of our patients.

We believe that the proposed biomass burning facilities pose a serious risk to the health of patients. Current research, as cited in the NCAFP's letter, indicates that the burning of poultry litter and wood wastes, as would be done in the proposed facilities, leads to increased risk of premature death and serious chronic illnesses. The plants additionally will have a negative impact on the health of our patients through emissions of nitrogen oxides, sulfur dioxides, arsenic, mercury and dioxins, all of which link directly to respiratory, brain, kidney, heart and thyroid diseases; cancer; diabetes mellitus; neurotoxicity; developmental delays in children and disruptions in fetal development. These emissions will have an adverse effect on the health of the most vulnerable North Carolinians: developing fetuses, newborns, children, those with chronic illness and the elderly. The result of bringing these biomass burning facilities online will be increased disability and disease, which will lead to increased medical costs.

A key reason for encouraging renewable energy through the North Carolina Clean Smokestack law was to provide cleaner air for its citizens. The evidence offered by the NCAFP indicates that the burning of poultry litter and wood waste does not comply with the spirit of that legislation. The AAFP requests that the North Carolina Department of Environment and Natural Resources consider the potentially harmful consequences to the health and well-being of the citizens of North Carolina—our patients—when contemplating the permitting of biomass burning plants in the state.

Sincerely,

Douglas E. Henley, MD, FAAFP
Executive Vice President and Chief Executive Officer

cc: Jeffrey P. Engle, MD, North Carolina State Health Director

Enclosure: North Carolina Academy of Family Physicians Letter of Concern Regarding Biomass Burning (April 19, 2010)

Executive Vice President, Douglas E. Henley, MD, FAAFP
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•Five out of six studies have found elevated dioxin levels in blood in communities near incinerators.

•Staessen et al. (2001); Gonzales (2000); Miyata (1998); Deml et al. (1996); Van den Hazel and Frankort (1996); Startin et al. (1994)

•Dioxin levels in the blood of people living near a new incinerator increased by 10-25 percent during the two years following the startup of the incinerator. Gonzalez et al. (2000)

+++

Effects of dioxins on thyroid function of new born babies
•H.J. Pluim et al., The Lancet, May 23, 1992. (Volume 339, 1303)
•Examined 38 new born babies, divided them into 2 groups:
•Low-exposed (mothers had average 18.6 ppt dioxins in milk fat, range 8.7 - 28)
•High-exposed ((mothers had average 37.5 ppt dioxins in milk fat, range 29 - 63)

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Dioxin Tied to Endometriosis
Science, 262, 1373,
26 November 1993

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Developmental Effects of Dioxins
Linda S. Birnbaum
Health Effects Research Laboratory, US EPA
Environmental Health Perspectives,
103: 89-94, 1995

++++

Exposure to dioxin and dioxin-like compounds as a potential factor in developmental disabilities
Tom Gasiewicz et al.
Mental Retardation & Developmental Disabilities Research Reviews,
3: 230-238, 1997

+++

Dioxins in cows milk
•1989 Dioxins in cow's milk fat in Netherlands very high downwind of incinerators = 12 ppt. Result: 16 Farmers not allowed to sell milk for 5 years.
•German law:

1) cannot sell milk > 5 ppt.
2) 3-5 ppt, have to reduce source
3) goal: <0.9 ppt.

In 1996, cow's milk in Ireland average 0.23 ppt Ireland has no municipal waste incinerators.

In 1998, cow's milk downwind of incinerators in France = 15 ppt.Result: Three incinerators closed.

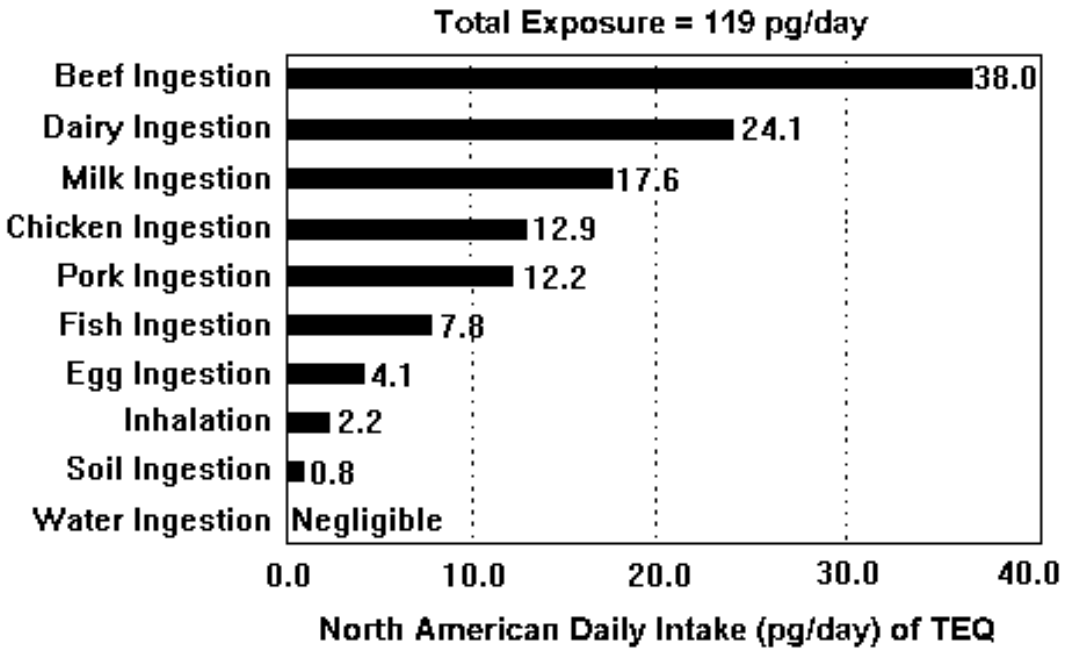
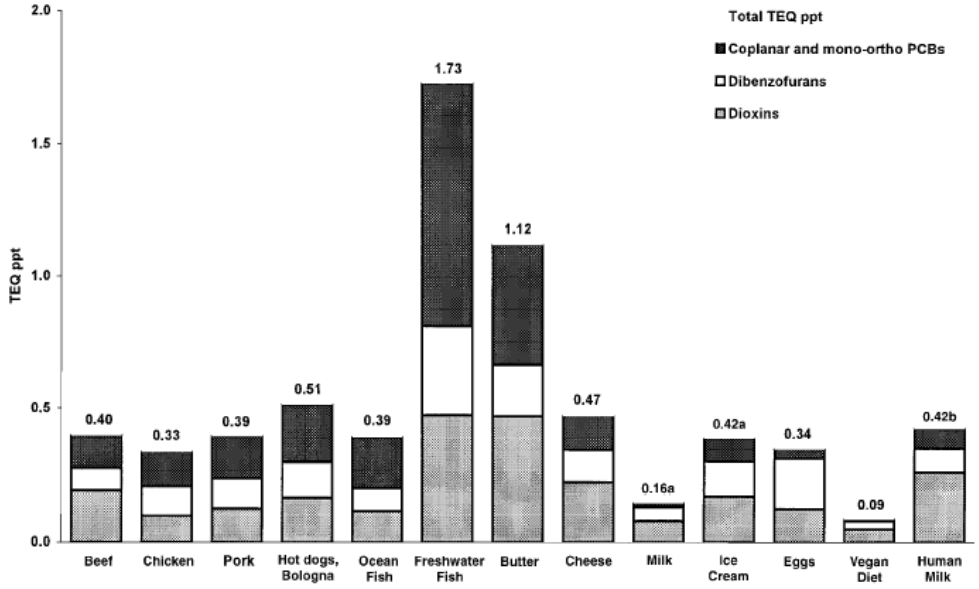


Chart from EPA Dioxin Reassessment Summary 4/94 - Vol. 1, p. 37

Figure II-5. Background TEQ exposures for North America by pathway)

Levels of Dioxin in U.S. Food Supply (1995):



ırt from May 2001 study by Arnold Schecter et. al., Journal of Toxicology and Environmental Health, Part A, 63:1–18]

In Response to engineers contradictions regarding dioxins:

* Themelis and Castaldi contradict one another, when one admits that dioxins are formed "during all combustion processes, in the presence of chlorine" and the other claims that no dioxins will be formed if they "operate in a temperature regime that will not produce the dioxins in the first place."

* Castaldi is misleading when he talks about the temperature range. He correctly states: "Dioxins are produced in high temperature regimes where chlorine is present along side hydrocarbons." However, the implication is that dioxins are formed at high temperatures. They're not. They're formed in the 200°C (392°F) to 400°C (752°F) range as the exhaust gases and ash are cooling down.[1] What matters most is not the high temperature they reach, but the SPEED at which the exhaust gases and ash drop through that temperature range. This used to be made worse by incinerators that used "hot-side" electrostatic precipitators (ESPs) which allowed the exhaust gases into the ESPs (which are a type of pollution control device) above 400° and which retained the gases in the dioxin formation temperature range longer, massively increasing dioxin emissions.[2] Better incinerators use quench systems to drop the gases through that temperature range more quickly, though this doesn't prevent all dioxin formation... it just reduces it. Most dioxins are formed on the surface of the ash (both the bottom ash and the fly ash particles, some of which escape to the air, some of which are captured in the

pollution controls, becoming "fly ash"). I don't think any incinerators try to quench the bottom ash, where most of the dioxin will be present. It's also worth noting that most of the dioxins are formed on the ash because of the presence of metals that serve as dioxin formation catalysts -- most notably copper, iron and zinc.[3]

* Their claim that activated carbon injection reduces dioxin emissions is false. It may reduce the emissions to the air, but it increases total dioxin formation by increasing the amount in the fly ash. The dioxin concentration in fly ash with carbon injection is 4-5 times higher than without carbon injection.[4]

* The claims about massive reductions in dioxins from MSW incinerators are also wrong and misleading. First, most of the reductions of dioxin emissions were from the shut down of so many trash incinerators (thanks to grassroots organizing in many cases). A lot of the reductions are from the closure or upgrading of incinerators that used hot-sided ESPs, like the one in Harrisburg, PA. The reductions don't count any increases due to carbon injection because they don't measure dioxin emissions in the ash. Most importantly, in terms of air emissions, the issue is that they do NOT continuously test for dioxins. By only testing once per year (under best operating conditions), they fail to capture the data on the excessive dioxin emissions during startup, shutdown and malfunction times, when the emissions limits don't tend to apply anyway (even if they were monitoring). Annual stack tests underestimate actual dioxin emissions by 30-50 times.[5] Real-time dioxin emissions monitoring equipment exists and has been tested and verified by EPA,[6] but I only know of one place in the U.S. that requires it so far -- a small local government in Pennsylvania where I wrote a local air pollution law that they used to stop a crematorium from being built there by subjecting it to continual testing and reporting requirements for mercury and dioxins.[7]

REFERENCES:

[1] 1994 EPA Dioxin Reassessment, Estimating Exposure to Dioxin-Like Compounds, Volume 2, Chapter 3. http://www.cqs.com/epa/exposure/v2chap3.htm (full report available at http://www.cqs.com/epa/exposure/) Some relevant quotes from the chapter include:

In this investigation, significant increases in total concentration of dioxin TEQ occurred between temperatures of 280-400° C, and concentrations declined at temperatures above 400° C. This is in agreement with the experimental evidence of the temperature range defined as the "window of opportunity" for catalytic formation of CDDs/CDFs on the surfaces of fly ash particles.

Formation kinetics are most favored at temperatures between 200 to 350° C.

Facilities of particular concern are those that use ESPs which operate in a

temperature range of 200° - 400° C. As discussed in Section 3.5 these conditions can promote the formation of CDDs/CDFs.

Moreover, formation occurs outside and downstream of the combustion zone of a furnace to a combustion source in regions where the temperature of the combustion offgases has cooled to between 200 and 400° C (Vogg et al., 1987; Bruce et al., 1991; Cleverly et al., 1991; Gullet et al., 1990a; Commoner et al., 1987; Dickson and Karasek, 1987; Dickson et al., 1992).

[2] See http://www.ejnet.org/dioxin/esp.html as well as references to "hot-sided ESPs" in the EPA dioxin reassessment document (http://www.cqs.com/epa/exposure/v2chap3.htm).

[3] "Metals as Catalysts for Dioxin Formation," http://www.ejnet.org/dioxin/catalysts.html. I've compiled and made available several scientific research papers on the topic here.

[4] Chang, M., Lin, J., 2001. Memory effect on the dioxin emissions from municipal waste incinerator in Taiwan. Chemosphere 45: 1151-1157. [see also the separate email I'll forward with the abstract and thread discussing it]

[5] Wevers M. and De Fré R., "Underestimation of dioxin emission inventories," Organohalogen Compounds, Vol. 36, pp. 19-20 (1998). http://www.ejnet.org/toxics/cems/1998_DeFre_OrgComp98_Underest_DIoxin_Em_Inv_Amesa.pdf

"The Amesa system [a long-term dioxin sampler tested and verified by U.S. EPA in 2007] was used for continuous sampling during periods of 15 days. The analysis was carried out in double by 2 laboratories, VITO and GfA. They show that a standard emission measurement according to the European standard method EN 1948 during a period of 6 hours resulted in an emission concentration of 0.25 ng TEQ/Nm3, while the average over 2 weeks in the same period was 8.2 to 12.9 ng TEG/Nm3. This illustrates that the standard measurement underestimated the avereage emission by a factor 30 to 50. [Note: it's actually 33 to 52 times higher.] As a result of these findings doubts have risen over the real emission of the incinerators, and the special commission on incineration has asked from all incinerators in the Flemish region to use the continuous sampling system in order to demonstrate their compliance with the emission limit."

More on continuous emissions monitoring of dioxin here: http://www.ejnet.org/toxics/cems/dioxin.html

[6] Dioxin Emission Monitoring Systems, Environmental Technology Verification Program, U.S. Environmental Protection Agency. http://www.epa.gov/etv/vt-ams.html#dems This page lists the four pieces of dioxin testing equipment that EPA tested and verified in 2006. The Amesa system is one (it's a long-term sampler that

can collect a sample of up to 30 days). Others are semi-continuous or actual real-time dioxin emissions monitors. Their "Technology Brief" on Dioxin Emission Monitoring Systems (http://www.epa.gov/etv/pubs/600s07002.pdf) states:

"The four verified technologies fall under one of two categories: automated isokinetic sampling systems of flue gas with laboratory analysis, or semi-continuous laser-based systems that produce ions which are typically detected using a time-of-flight mass spectrometer (TOFMS). Long-term continuous samplers collect samples over time periods up to several weeks to obtain a cumulative record of source emissions and provide evidence of emission levels. Real or semi-real-time continuous monitors, with a frequency of measurement at real time or up to an hour, provide quick feed back to the plant operator by measuring dioxin emission levels on-site."

[7] See http://www.actionpa.org/ordinances/ for the Kulpmont Borough, Pennsylvania ordinance.

FROM EPA Website . . . .

[PDF] 2010 TRI National Analysis Qs and As 05-23-2012
... Electric utilities accounted for 35% of all releases to air of **dioxins** in 2010 and reported an **increase** of 5% from 2009 to 2010. **...**
http://www.epa.gov/tri/tridata/tri10/nationalanalysis/qanda/2010_TRI_N...

2010 TRI National Analysis Qs and As
Table of Contents
Overview of the 2010 Data
.....

Dioxin and Dioxin-like Compounds

Total disposal or other releases of dioxins increased 18% from 2009 to 2010.

Air releases of dioxins increased by 10%.

• Chemical manufacturers accounted for almost 64% of total disposal or other releases of dioxins in 2010. They reported a 7% decrease from 2009 to 2010.

• Hazardous waste management facilities and primary metals sector reported the largest increase in total disposal or other releases of dioxins from 2009 to 2010, primarily as on-site land disposal.

• Electric utilities accounted for 35% of all releases to air of dioxins in 2010 and reported an increase of 5% from 2009 to 2010.

Emissions to air of hazardous substances and particulate matter, 1990-2010
Increased emissions of hazardous substances to air

Netherlands: The emissions to air of several hazardous substances increased in 2010. The increase was partly due to higher activity in parts of industry.
The emissions of arsenic, some heavy metals, particulate matter and dioxins to air have increased in 2010. The increase was due to increased activity in parts of industry, and a higher content of contaminants in raw materials and reducing agents used in metal production. The emissions to air of several hazardous substances including PAHs (polycyclic aromatic hydrocarbons) have been considerably reduced since 1990.
The calculations of emissions to air for the period 1990-2010 are performed by Statistics Norway in collaboration with the Climate and Pollution Agency.

Emissions of heavy metals, PAH-4 and dioxins. Per cent change 1990-2010 and 2009-2010 and harmful effects

Dioxins 80 % reduction 1990 to 2010
Dioxins 19% increase from 2009 to 2020

Dioxins from biomass and other incinerators are not adequately monitored, but EPA estimates that – while most sources of dioxin pollution, including residential wood burning, declined from 1987 through 2000 (the latest national inventory) – dioxin from industrial wood burning increased 56% in that time. [1] This industry-wide total emissions inventory found such an increase because the industry expanded quite a bit in those 13 years, with at least 70 new biomass burning units going online in that time. At least 20 more have gone online since 2000, and dioxins from biomass will continue to rise as long as the industry is growing.[2]

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[1] "An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the United States for the Years 1987, 1995, and 2000," U.S. EPA, November 2006, Figure 1-5 and Table 1-17.
http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=159286
[2] eGRID 2012 Database, U.S. Environmental Protection Agency, 2009 data released on 5/10/2012. http://www.epa.gov/cleanenergy/energy-resources/egrid/