(27)

# ORAL HEARING INTO

# PROPOSED DECISION 167 – 1

# Carranstown WASTE MANAGEMENT FACILITY

# PROOF OF EVIDENCE

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Qualifications & Experience

My name is Paul Connett. I obtained my undergraduate degree from Cambridge University in the UK and my PhD in chemistry from Dartmouth College in the US. Since 1983 I have taught chemistry at St. Lawrence University in Canton, NY where I specialize in Environmental Chemistry and Toxicology. Over the past 21 years my research on waste management has taken me to 49 states in the US, and 47 other countries, where he has given approximately 2000 pro bono public presentations. I have co-authored 6 peer reviewed articles on dioxin and numerous other articles on waste management.My most recent publication on waste is a booklet "A Citizens' Agenda for Zero Waste" co-authored by Dr. Bill Sheehan of the Grass Roots Recycling Network (see http://www.grrn.org). I am currently documenting successful zero waste management programs and initiatives around the world in two series of videotapes entitled "On the Road to Zero Waste" and "Pieces of Zero."

I am going to present evidence on the following issues:

A) The dioxin issue.
B) A critique of Dr. Fergal Callaghan's submission on the projected dioxin doses from this project.
C) A critique of Mr. Ahearn's submission.



A) The dioxin issue.

I will begin my comments with the dioxin issue on which I have had some experience over the last 21 years.

Section 4.4.5 (pages 85-90) of the Indavar EIS largely trivializes this very serious issue. The summary of health effects as provided by the WHO (page 86) is dated and does little justice to recent findings. Many of these are readily available in the latest draft of the US EPA's reassessment of dioxin (see attachments).

There are three key issues on dioxin which are highly pertinent to this project.

1) The ability of dioxin to bioaccumulate and concentrate in the human food chain. The numbers here are quite staggering. Michael McLachlan at the Dioxin '97 conference in Indianapolis showed that in one day a freely grazing cow put into its body (from eating grass contaminated with gaseous and particulate dioxin) the

amount of dioxin equivalent to breathing the air next to the cow for 14 years!

2) The inability of the liver to convert these fat soluble contaminants into water soluble products, with the result that they accumulate in our (human) fat. The man has no way of getting rid of them, but a woman can: by having a baby! Thus dioxins which have accumulated in her fat for 20-30 years are passed to the fetus for nine months during pregnancy.
3) Dioxins are very potent disrupters of a number of hormonal systems, which are critically important in fetal development. Thus the human fetus gets exposed to the highest doses and is also the most vulnerable.
4) As a result of these concerns the Institute of Medicine on July 1 2003 issued a report recommending that parents of young girls encourage them to switch from whole milk to low fat milk and also to lower their animal fat intake, as early before pregnancy as possible.
5) The last place you want to build an incinerator is an area surrounded by farmland.
6) The last country you want to build incinerators is in Ireland which has the lowest levels of dioxin in cows milk in Europe and possibly any industrialized country in the world. This not only poses a direct health threat to people consuming the food, with possibility of increasing cancer and other health problems (see a recent paper from France indicating an increase in Non-Hodgkins Lymphoma near an incinerator, albeit an older one), but it also poses an economic threat to agriculture. It is much easier to persuade the middle men in agriculture that your dioxin levels in cows milk are low because you have no trash incinerators than trying to explain to them that you have trash incinerators but they are good ones!



B) Problems with Dr. Fergal Callaghan's (AWN Consulting Ltd) submission on solid PCDD/F concentrations and PCCD/F intake.

Dr. Callaghan trivializes the issue when he compares estimated dioxin TEQ inhalation doses from the proposed project with current background doses from food. He is comparing apples with oranges. Or rather a single apple with an orange orchard! It is well known that exposure via foodchains dwarfs exposure via inhalation, a subject on which I have published several articles. In fact as long ago as 1986, Tom Webster and I, in a paper presented to the Dioxin Symposia (Dioxin '86) held in Fukuoka, Japan, showed that one liter of cows milk would deliver as much dioxin as breathing the air next to the grazing cow for 8 months.

More importantly, Dr. Callaghan appears to have made a serious calculation error in the estimation of the dioxin dose to the "maximum at risk individual" (MARI). He defines a MARI as someone who lives "at the point of maximum PCDD/F deposition…and to be a subsistence farmer", who obtains "all of their food (vegetables, milk and meat) from a 100 m diameter site" (page 7).

On page 9, Dr. Callaghan tells us that the predicted increase dose to this MARI receptor would be 0.0027 pg/kg/day. However, previously on page 8, he indicates that the "annual average predicted ground level concentration of PCDD/F, from the proposed WTE

facility is 1 fg/m3" (note: 1 fg = 0.001 pg). In the next sentence he translates this into a daily dose from INHALATION of 0.015 pg or 0.0025 pg/kg/day. The latter figure was corrected at the hearing to 0.00025 pg/kg/day (i.e. 0.015 pg divided by 60 kg bodyweight).

There is something wrong here, if a subsistence farmer is getting a daily dose of 0.00025 pg/kg/day from inhaling air, he is going to get many times (at least 1000 times) more dioxin than this from ingesting food grown at the same point. Thus the total figure given by Callagahn of 0.0027 pg/kg/day for TOTAL dose (inhalation plus ingestion of locally produced food) is a gross underestimate of the dose to the MARI receptor.

If we assume that the dose from inhalation is correct (a very much simpler calculation than the food chain pathway calculations) and is equal to 0.00025 pg/kg/day, then based upon my experience with these kind of risk assessments the dose via food would be 1000 to 3000 times higher than this and would thus be 0.25 – 0.75 pg/kg/day.

If we now add this range to the baseline intake (i.e. current background exposure) calculation for the MARI of 0.575 pg/kg/day (see page 7) the total dose of baseline plus increment from the plant comes to 0.825 – 1.325 pg/kg/day. Thus we now have the potential to exceed the bottom end of the WHO allowable daily intake range of 1- 4 pg/kg/day. As it is believed that only the 1 pg/kg/day can be defended scientifically (the 4 pg/kg/day was added to give political wiggle room for those countries which did not want to tell their citizens that they were exceeding the ADI simply by consuming food!) then even by these weak standards the proposed facility should not be approved.



The latest draft of the EPA reassessment of dioxin is now using a daily dose of 0.001 pg/kg/day as indicating an incremental cancer risk of 1 in a million for lifetime exposure (70 years). Thus, in American terms, the corrected daily dose for the MARI translates into an incremental cancer risk of 825 –1325 per million or approximately 1 in 1000. No facility that predicts such a cancer risk would be permitted in the US. In my experience projects permitted by the US usually fall in the range of 1 to 100 in a million.

C) Overall problems with the proposal. A critique of Mr. Ahearn's submission.

The overall problems with the Indaver proposal can be best illustrated by reviewing the presentation by Mr. Ahearn. He covers 5 issues:

1. Waste policy
2. Incineration in Europe
3. Zero Waste
4. Company competency
5. Health Protection Systems

I will discuss each of these in turn.

1) Waste Policy

In this section Mr. Ahearn describes the waste heirarchy in which energy recovery comes next to bottom on the list of preferred options. He does not explain why his company is rushing the gun in Ireland, and pushing for this low priority option before very much progress has been made on higher priority options like reuse, repair, recycling and composting. However, he uses a familiar tactic used by the US incinerator industry of describing this heirarchy as an "integrated waste management system". This of course completely blurs the distinction of prioritizing your options and ensures that his preferred option gets maximum attention even while the heirarchy suggests otherwise. Note that he can only "officially" prioritize incineration over landfill, which he deftly does in this sentence, while putting it on a level platform with reuse and recycling: "If waste cannot be prevented we should try to minimize its production and if it is produced we should reuse it, recycle it, recover energy from it and only as a last resort should we dispose of it". Notice how differently his project would look, if we brought the words " and only as a last resort should we" a little closer to the beginning of the sentence so that it read: "If waste cannot be prevented we should try to minimize its production and if it is produced we should reuse it, recycle it, and only as a last resort should we recover energy from it and dispose of it".

And even better if we added composting, which is the key to changing the nature of landfilling in Ireland, to read

"If waste cannot be prevented we should try to minimize its production and if it is produced we should reuse it, compost it, recycle it and only as a last resort should we recover energy from it and dispose of it".



While Mr. Ahearn is anxious to distance incineration from disposal, by using the term "energy recovery" the amount of energy recovered from incineration is very small. Thus, from the point of view of moving towards sustainability, incineration has to be considered a disposal option, because once burned objects have to be replaced. At least two reports from the US have shown that about 3 times more energy is saved by reusing, recycling and composting the same materials burned in an incinerator. Hence the danger of allowing incineration to claim central stage before reuse, recycling and composting have been maximized. Most citizens recognize this but unfortunately the Irish government has not and continues to waste time. Ironically, if this project, and others like it go through, the high landfill charges will drive incineration and illegal dumping, rather than driving waste reduction, reuse, composting and recycling.

As far as the ash generated is concerned (about one ton for every three tons of trash burned) Mr. Ahearn waves his hands about finding uses for the bottom ash, which is not non-hazardous as he claims, and admits that Indaver may have to export the fly ash to Belgium. How viable is such a strategy, especially when Belgian activists hear that this is going to happen?

2. Incineration in Europe.

When Mr. Ahearn talks about incineration in Europe he fails to recognize that Europe has been trapped with incineration for many years. By and large they have much more densely populated countries. Sweden and Denmark have built district water heater systems around their incinerators. Huge amounts of money has been invested in incinerators in Germany, Switzerland and the Netherlands, making it difficult for them to abandon them. Most have tried to salvage what they can with retrofitted air pollution control equipment. Very few new incinerators have been built in these countries since the 80s.

It is a pity that Mr. Ahearn did not analyze more closely the situation in Flanders (which he illustrated in the Zero Waste section). Maybe the huge 70% diversion they are achieving has something to do with the moratorium that this region of Belgium had on incineration for at least 5 years. According to the figure he presents on page 8, Flanders is getting 70% diversion from both landfills and incinerators, presumably using waste reduction, recycling and composting. Clearly, Ireland should be seeing how they could get 70% diversion from landfills before committing to the costly, problematic and politicly divisive technology of incineration.

In my view, Irish agencies and decision makers should be looking at what is happening in countries not trapped by incinerators, particularly New Zealand, Australia, Canada and California. Todate many of the consultants giving advice in Ireland have drawn heavily from Europe and not from the rest of the world. I believe that New Zealand is particularly useful model because it has similar population density to Ireland as well as a strong agricultural economy to protect. Over 60% of municipalities in NZ have adopted a zero waste strategy.



3) Zero Waste

Clearly, Mr. Ahearn does not (or does not want) to understand the concept of zero waste. I have written on this subject and Mr. Ahearn has been present when I have talked about it, but he seems to have taken only what he needs to dismiss it as a viable option.

I am very surprised with his analysis of diversion rates in Canberra, as they are contrary to my impressions when I visited Canberra and videotaped the program three times over the last four years. I will be sending Mr. Ahearn's comments to program officials in Canberra to see if they concur with his analysis. I will forward their comments to this committee once I have heard from them. I would –and I am sure you would –like to know the truth about this.

Meanwhile, while it is true that the Canberra program began as a "No Waste to Landfill" law passed in 1996. I have heard no indication from the officials I have spoken with that they plan to build an incinerator there.

If the Zero Waste movement is channeled into a "Zero Waste to Landfill" movement it will completely subvert the philosophy of those leading this effort worldwide.

There are three key aspects about Zero Waste, which distinguish it from Mr. Ahearn's characterization. 1) The time frame, 2) the combination of industrial as well as community responsibility and 3) the role of the residuals to make that connection.

i) The time frame.There is no question that zero waste is an idealistic goal, but by declaring zero waste 2020, communities are putting this idealistic goal into a realistic time frame. 15 years is actually shorter than the lifespan of an incinerator, and an incinerator can only achieve a 70-75% reduction by mass.

ii) Community & Industrial responsibility. Practically everyone agrees that industry compounds the waste problem with overpackaging and poor design of products, but most of the pressure is going on communities to solve the problem. Governments need to be even handed because we will only get to zero waste ( or very close) if we get industrial responsibility as well as community responsibility (reusing, repairing, composting and recycling).

iii) These two can be made to connect if we send the residuals to a Residual Screening & Research facility, where the residuals are screened for more recyclables and more toxics and the dirty organic fraction is stabilized biologically in a second composting operation prior to an interim landfill. The point of the research is to communicate to industry, 'If we can't reuse it, recycle it or compost it, you shouldn't be making it". We need better industrial design for the twenty first century.



Mr. Ahearn's strategy of burning the residuals takes the pressure off industry as it hides the evidence of bad design and practice. It sadly takes away from an important pressure path towards a more sustainable future. In short, even if you made incineration safe you never would make it sensible. It simply does not make sense to spend so much money destroying resources we should be sharing with the future.

4) Company Competency

As far as the competency of Indaver is concerned, I have been more impressed with their PR than their substance. In a previous visit to Ireland I asked the Indaver Cork office to provide me with their dioxin testing data ( not averages or summaries but the full test results) for their facilities in Belgium. Despite repeated requests I have not received these and thus I have no way of knowing whether their claims about dioxin emissions are credible or not.

5) Health Systems.

In this section Ahearn lists the various Irish agencies which he claims will protect the public from this incinerator once it has been built. However, there are three critical things needed to protect the public from incinerator emissions:

a. Strong regulations.
b. Scientific monitoring
c. Aggressive government enforcement.

The 0.1 ng/M3 standard is a strong standard but it may not be adequate for an incinerator which is surrounded by agriculture and already has other dioxin sources (Cement kiln)

In the collection of baseline data there is little indication that the company plans to do an adequate scientific job of monitoring this facility. They have only collected soil and air samples. At the very least they need to be monitoring cows milk both upwind and downwind of the facility. I was pleased to see that the EPA is requiring two week sampling of dioxin (the AMESA system) in addition to the traditional six hour testing. However, this should be buttressed with monitoring of cows milk as this direct measurment is far more direct and meaningful in risk calculations on the one hand and reassuring the public on the other.

I am not very familiar with the track record of the Irish government's enforcement of regulations on polluting facilities, but in this case it might be very difficult for them to be too aggressive. This is a very costly project and it will not be easy to shut down if it proved necessary. Moreover, incineration is government policy and it is very difficult for scientists or citizens to get an open ear when there is policy at stake. The worst thing for any government to do is to admit they were wrong. One only has to look at the recent history of the Ministry of Health refusing to take a serious look at the dangers of fluoridation to see what I mean. After 4 years they have not been able to get a panel of experts to respond in writing to "50 Reasons to Oppose Fluoridation" which I presented to them in 2000 (see http://www.fluoridealert.50reasons.htm) even though they originally agreed to do so!

Thank you for your attention. I am happy to answer any questions you may have.

United States Environmental Protection Agency | Office of Research and Development Washington, DC 20460 | May 25, 2001 Update

# Information Sheet 1

## *Dioxin:* *Summary of the Dioxin Reassessment Science*

Scientists from the Environmental Protection Agency (EPA), other federal agencies and the general scientific community have conducted a reassessment of dioxin exposure and human health effects since 1991. This information sheet summarizes the draft reassessment, which is entitled *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds.* A more in-depth discussion can be found in the companion piece, *Dioxin: Scientific Highlights from Draft Reassessment (2000).*

The term "dioxin" refers to a group of chemical compounds that share certain similar chemical structures and mode-of-action biological characteristics. A total of 30 of these dioxin-like compounds exist and are members of three closely related families: the chlorinated dibenzo-*p*-dioxins (CDDs), chlorinated dibenzofurans (CDFs) and certain polychlorinated biphenyls (PCBs). The term dioxin is also used for the most well-studied and one of the most toxic dioxins, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). CDDs and CDFs are not created intentionally, but can be produced inadvertently in nature and by a number of human activities. Combustion, chlorine bleaching of pulp and paper, certain types of chemical manufacturing and processing, and other industrial processes all can create small quantities of dioxins. PCBs are no longer manufactured in the United States but formerly were widely used as coolants and lubricants in electrical equipment.



**Combining Risks from Dioxins - the Toxicity Equivalents Approach:**

Dioxins are believed to cause toxic effects in similar ways; that is, they share a "common mechanism of toxicity." As a result, EPA and others use an approach that adds together the toxicity of individual dioxins in order to evaluate complex environmental mixtures to which people are exposed. Because dioxins differ in their toxic potential, the toxicity of each component in the mixture must be accounted for in estimating the overall toxicity. To do so, international teams of scientists have developed Toxicity Equivalency Factors that compare the toxicity of different dioxins. Given these factors, the toxicity of a mixture can be expressed in terms of its Toxicity Equivalents (TEQ), which is the amount of TCDD it would take to equal the combined toxic effect of all the dioxins found in that mixture. The use of the TEQ approach represents a key assumption upon which many of the conclusions in the reassessment are based.

**Dioxin Toxicity:**

The reassessment finds that, based on all available information, dioxins are potent animal toxicants with potential to produce a broad spectrum of adverse effects in humans. Dioxins can alter the fundamental growth and development of cells in ways that have the potential to lead to many kinds of impacts. These include, for example, adverse effects upon reproduction and development; suppression of the immune system; chloracne (a severe acne-like condition that sometimes persists for many years); and cancer. EPA characterizes the complex mixtures of dioxin to which people are exposed as a "likely human carcinogen." This is based on the fact that individual components of this mixture could be characterized as "human carcinogens" or "likely human carcinogens" under EPA's draft cancer risk assessment guidelines (1996, 1999). In particular, TCDD, the most toxic of the dioxins, can be identified as a "human carcinogen" under the Agency's draft guidelines, based on the weight of the animal and human evidence, and the other dioxins as "likely human carcinogens."

**Dioxin Exposure:**

The reassessment proposes that most dioxin enters ecological food webs by being deposited from the atmosphere, either directly following air emissions or indirectly by processes that return dioxins already in the environment to the atmosphere. Once they reach the environment, dioxins are highly persistent and can accumulate in the tissues of animals. EPA estimates that most dioxin exposure occurs through the diet, with over 95% of dioxin intake for a typical person coming through dietary intake of animal fats. Small amounts of exposure occur from breathing air containing trace amounts of dioxin on particles and in vapor form, from inadvertent ingestion of soil containing dioxin, and from absorption through the skin contacting air, soil, or water containing minute levels. These processes result in widespread, low-level exposure of the general population to dioxins.

Dioxin levels in the environment have declined significantly since the 1970s following EPA regulatory controls and industry actions. EPA's best estimates of emissions from sources that can be reasonably quantified, indicate that dioxin emissions in the United States decreased by about 75% between 1987 and 1995, primarily due to reductions in air emissions from municipal and medical waste incinerators, and substantial further declines continue to be documented. Uncontrolled combustion such as burning of household waste is expected to become the largest quantified source of dioxin emissions to the environment. Dietary intake of dioxin also appears to be declining.

**Dioxin Effects in Human Populations**

EPA estimates that the amount of dioxin found in the tissues of the general human population (which is known as the "body burden") closely approaches (within a factor of 10) the levels at which adverse effects might be expected to occur, based on studies of animals and highly exposed human populations. Despite the potential risks, currently there is no clear indication of increased disease in the general population attributable to dioxin-like compounds. This may be due to limitations of current data and scientific tools rather than indicating that dioxin exposure is not causing adverse effects. For cancer, EPA estimates that the risks for the general population based on dioxin exposure may exceed 1 in 1,000 increased chance of experiencing cancer related to dioxin exposure.Actual risks are unlikely to exceed this value and may be substantially less. This range for cancer risk indicates an about 10-fold higher chance than estimated in EPA's earlier (1994) draft of this reassessment.



**Children and Other Groups of Concern**

Fetuses, infants, and children may be more sensitive to dioxin exposure because of their rapid growth and development. Data on risks to children are limited, however, and it is not known if the children in the general population are experiencing adverse effects from dioxin. Although breast milk appears to be a significant source of dioxin exposure for nursing infants, the overwhelming body of evidence supports the health benefits of breastfeeding despite the potential presence of dioxin. Other populations have experienced elevated exposures to dioxin as a result of food contamination incidents around the world, through the workplace or from industrial accidents, or from consumption of unusually high amounts of fish, meat, or dairy products containing elevated levels of dioxins. In some cases, such as U.S. Air Force personnel exposed to the herbicide Agent Orange contaminated with dioxin during the Vietnam War, dioxin exposure has been associated with adverse health effects.

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# Information Sheet 2

## *Dioxin: Scientific Highlights from Draft Reassessment (2000)*

Scientists from the Environmental Protection Agency (EPA), other federal agencies and the general scientific community have conducted a comprehensive reassessment of dioxin exposure and human health effects since 1991. See the discussion of the process in the companion document entitled, "Dioxin Reassessment Process: EPA is Moving Toward Completion of the Dioxin Reassessment." In the next few pages, the Agency summarizes the scientific highlights of the updated, draft reassessment of dioxin and related compounds, including the updated and revised "Dose Response" Chapter (Part II. Chapter 8), the new "Toxicity Equivalence (TEF)" Chapter (Part II. Chapter 9), and the updated, revised, and reformatted "Integrated Summary and Risk Characterization" (Part III).

Throughout this reassessment, concentrations of dioxin and related compounds are presented as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) equivalents (TEQs). One compound, TCDD is the best studied of this class of compounds and is the reference compound for assignment of toxicity equivalence factors (TEFs) for related congeners. The strengths and weaknesses as well as the uncertainties of the TEF/TEQ approach have been discussed in the report and, particularly, in a newly developed chapter (Part II. Chapter 9). Use of the TEQ approach is widely accepted in the international scientific community and is fundamental to the evaluation of this group of compounds which always exist in nature as complex mixtures of dioxins. The use of the TEQ approach represents a key assumption upon which many of the conclusions in this characterization hinge.



The reassessment finds that there is adequate evidence based on all available information, including studies in human populations as well as in laboratory animals and from ancillary experimental data, to suspect that humans may respond with a broad spectrum of effects from exposure to dioxin and related compounds. Research has highlighted certain prominent, biologically significant effects of TCDD. These biochemical, cellular, and organ-level endpoints have been shown to be affected by TCDD in experimental systems, but specific data on these endpoints do not generally exist for many of the other TCDD-like congeners. Despite this lack of congener specific data, there is reason to infer that these effects may occur for all dioxin-like compounds, as embodied in the concept of toxicity equivalence. A few of these effects have been observed under high exposure conditions in human populations; many others have not been investigated with well-designed human studies or in relevant populations. The mechanistic relationships of biochemical and cellular changes seen at very low levels of exposure in animals and humans to production of adverse effects generally detectible at higher levels remains uncertain and controversial. Based on the experience of the scientific community using animal models and evaluating a limited human data base, it is reasonable to infer that effects in the human population may span a wide range. These effects may range from changes in biology or biochemistry which may be judged by some to be adaptive (with little or no adverse impact), or which may arguably be considered by others to be adverse, at or near background levels of exposure to clearly adverse effects with increasing severity as exposure increases above background levels by orders of magnitude (10 to

100 times background). Enzyme induction, changes in levels of gene regulators or related receptors, and indicators of altered cellular function represent examples of biomarkers of exposure of unknown clinical significance which may or may not be early indicators of toxic response. Induction of activating/ metabolizing enzymes at or near background levels, for instance, may be adaptive or may be considered adverse since induction may lead to more rapid metabolism and elimination of potentially toxic compounds, or may lead to increases in reactive intermediates and may result in toxic effects. Demonstration of examples of both of these situations is available in the published animal literature. Other potentially adverse effects have been reported to be associated with exposure to dioxin and related compounds in human populations at or near average background population levels (within a factor of 10 of these levels). These include delay of developmental milestones, impacts on immune function, and, perhaps, increased incidence or susceptibility to disease, e.g., elevated incidence of adult onset diabetes. While potentially present in exposed populations, clearly adverse effects, including cancer, may not be detectable as increased incidence of disease until exposures exceed background by one or two orders of magnitude (10 or 100 times).

With regard to sensitivity, it is well known that individual species vary in their sensitivity to any particular dioxin effect. However, the evidence available to date indicates that humans may fall in the middle of the range of sensitivity for individual effects among animals rather than at either extreme. In other words, evaluation of the available data using comparable dose metrics suggests that humans, in general, are neither extremely sensitive nor insensitive to the individual effects of dioxin-like compounds as compared to other animals. Human data provide direct or indirect support for evaluation of likely effect levels for several of the endpoints discussed in the reassessment although the influence of variability among humans remains difficult to assess.



The scientific community has identified and described a series of common biological steps that are necessary for most if not all of the observed effects of dioxin and related compounds in vertebrates including humans. Binding of dioxin-like compounds to a cellular protein called the "Ah receptor" represents the first step in a series of events attributable to exposure to dioxin-like compounds including biochemical, cellular and tissue-level changes in normal biological processes. Binding to the Ah receptor appears to be necessary for all well-studied effects of dioxin but is not sufficient, in and of itself, to elicit these responses; further steps beyond receptor binding are required. The effects elicited by exposure to TCDD are shared by other chemicals which have a similar structure and Ah receptor binding characteristics. Consequently, it is reasonable to assume that the biological system responds to the cumulative exposure to other dioxin-like chemicals instead of exposure to any single dioxin-like compound. Based on our understanding of dioxin mode(s)-of-action to date, it is reasonable to conclude that interaction with the Ah receptor is necessary, that at comparable doses (e.g. similar body burdens) humans are likely to respond with many of the effects of dioxin demonstrable in laboratory animals, and that there is likely to be a variation among and within species and among tissues in individual species based on differential responses "down stream" from receptor binding.

Some of the effects of dioxin and related compounds such as enzyme induction, changes in hormone levels and indicators of altered cellular function have been observed in laboratory animals and humans at body burdens comparable to exposures at or near levels to which segments of the general population are exposed. Other effects are detectable only in highly exposed populations, and there may or may not be a likelihood of response in individuals experiencing lower levels of exposure. Adverse effects associated with temporary increases in dioxin blood levels based on short term high level exposures, such as those that might occur in an

industrial accident or in infrequent contact with highly contaminated environmental media, may be dependent on the impact of exposure on total body burden.

The exposure document (Part I) has been revised to reflect comments from the public and the Agency's Science Advisory Board (SAB). It presents an up-to-date (1999) and comprehensive emission inventory of dioxin and related compounds for the United States. A large variety of sources of dioxin have been identified, and characterized but others may exist. The available information suggests that the presence of dioxin-like compounds in the environment is primarily a result of formation of unintentional by-products of combustion or industrial practices and is likely to reflect changes in release over time. The principal identified sources of environmental release may be grouped into five types: Combustion and Incineration Sources; Metals Smelting, Refining and Processing; Chemical Manufacturing/Processing; Reservoir Sources; and Biological and Photochemical Processes. The Exposure Document provides "snapshots" of estimated emissions for the years 1987 and 1995. Because of the nature of the available data and the need to extrapolate national emission levels, confidence in these estimates varies. However, EPA's best estimates of releases of dioxin and related compounds (CDDs/CDFs) to air, water and land from reasonably quantifiable sources suggests an approximately 75% decrease between 1987 and 1995, due primarily to reductions in air emissions from municipal and medical waste incinerators. Regulations promulgated in 1995 for municipal waste combustors and 1997 for medical waste incinerators should result in a greater than 95% reduction in dioxin emissions from these two categories. Uncontrolled combustion such as burning of household waste is expected to become the largest quantified source of dioxin emissions to the environment. With the reduction in combustion and incineration sources, reservoir sources are likely to increase in importance.

Because dioxin-like chemicals are persistent and accumulate in biological tissues, particularly in animals, the major route of human exposure is through ingestion of foods containing minute quantities of dioxin-like compounds. This results in wide-spread exposure of the general population to dioxin-like compounds. It appears that daily intakes have come down since the 1970s and that, as of the mid-90s, adult daily intakes of dioxin and related compounds, including dioxin-like PCBs average 65 $pgTEQ_{DFP}WHO_{98}$/day. Certain segments of the population may be exposed to additional increments of exposure by being in proximity to point sources or because of dietary practices. The estimated levels of dioxin and related compounds in the environment and contributing to daily intakes in the U.S. are based on additional data collected since 1995. Further data collection is underway in studies by EPA, FDA and USDA scientists. Current estimated U.S. levels are consistent with levels reported for Western Europe and Canada, and support a conclusion that increased dioxin exposures are associated with industrialization. The consistency of U.S. levels with those of other industrialized countries also provides additional reassurance that the U.S. estimates are reasonable in the face of the limited data on U.S. levels, recognizing that some differences among countries will reflect national and international control efforts.



The reassessment presents the hypothesis that the primary mechanism by which dioxin-like compounds enter ecological food chains and human diet is via atmospheric deposition. Dioxin and related compounds enter the atmosphere directly through air emissions and are widely spread in the environment as a result of a number of physical and biological processes, for example, through erosion and run-off, volatilization from land or water, or from re-suspension of particles. Deposition can occur directly on to soil or plant surfaces. At present, it is unclear whether atmospheric deposition represents primarily current contributions of dioxin and related compounds from all media, or past emissions that persist and recycle in the environment. Understanding the relationship between these two scenarios will be particularly important in understanding the relative contributions

of individual point sources of these compounds to the food chain and assessing the effectiveness of control strategies focused on current or past emissions of dioxins in attempting to reduce dioxin exposures.

The term "background" exposure has been used throughout this reassessment to describe exposure of the general population, which is not exposed to readily identifiable point sources of dioxin-like compounds. Data on human tissue levels suggest that body burden among industrialized nations are reasonably similar. Average background exposure led to body burdens in the late 1980s ranged from 30-80 pg TEQ/g lipid (this equates to 30-80 ppt), with a mid-point of approximately 55 pg TEQ/g lipid, when all dioxins, furans and dioxin-like PCBs are included. High-end estimates of body burden of individuals in the general population (approximately the top 1% of the general population) may be more than 3 times higher, based on evaluation of blood-level data and on consumption of fat as a surrogate for dioxin intake. The average CDD/CDF/PCB tissue level for the general adult U.S. population appears to be declining and the best estimate of current (late 1990s) average body burden levels is 25 ppt ($TEQ_{DFP}$-$WHO_{98}$, lipid basis).

In addition to general population exposure, some individuals or groups may also be exposed to dioxin-like compounds from discrete sources or local pathways, including occupational exposures, direct or indirect exposure of local populations to discrete sources, exposure of nursing infants from mother's milk, or exposures of subsistence or recreational fishers. Daily exposures to these individuals may be significantly higher than among the general population. However, the differences in average body burden are expected to be much less than the differences in daily intake, particularly if these elevated exposures are periodic or for short duration. In addition, while it is often difficult, the health benefits of dietary components must factor into assessment of overall risk.



As described above, subtle changes in biochemistry and physiology such as enzyme induction, altered cellular function, and other potentially adverse effects have been detected in dioxin-exposed populations in a limited number of available studies. These findings, coupled with knowledge derived from animal experiments, suggest the potential for adverse impacts on human metabolism, and developmental and/or reproductive biology, and, perhaps, other effects in the range of current human exposures. Given the assumption that TEQ intake values represent a valid comparison with TCDD exposure, some of these adverse impacts may be occurring at or within one order of magnitude of average background TEQ intake or body burden levels. As body burdens increase within and above this range, the probability of occurrence, as well as the spectrum of human noncancer response, most likely increases. Because of the basic biological level at which dioxin and related compounds act, and because of the potential diversity of "down-stream" responses to a dioxin body burden, it is not currently possible to state exactly how or at what levels individuals in the population will respond. It is clear, that as recent data have developed, the margin of exposure (M-O-E)[1] between body burdens associated with background levels of exposure and levels where effects are detectable in humans, in terms of body burden

[1] The likelihood that noncancer effects may occur in the human population at environmental exposure levels is often evaluated using a "margin of exposure" (MOE) approach. A MOE is calculated by dividing the human, or human-equivalent animal, lowest observed adverse effect levels (LOAEL) or no observed adverse effect level (NOAEL) with the human exposure level of interest. MOEs in range of 100 -1000 are generally considered adequate to rule out the likelihood of significant effects in humans based on sensitive animal responses. The average intake levels of dioxin-like compounds in terms of TEQs in humans described above would be well within a factor of 100 of levels representing LOAELs in laboratory animals exposed to TCDD or TCDD equivalents. For several of the effects noted in animals, a MOE of less than a factor of ten, based on intake levels or body burdens, is likely to exist.

TEQs, is considerably smaller than previously estimated and, in some cases, may be 1 or even less. For certain effects, including subtle behavioral impacts, a "no effect level" has yet to be established.

These facts and assumptions lead to the inference that some members of the general population or more highly exposed, special populations may be at risk for a number of adverse effects. These may include, for instance, developmental toxicity based on the inherent sensitivity of the developing organism to changes in cellular biochemistry and/or physiology, impaired reproductive capacity based on structural or functional impacts, less ability to withstand an immunological challenge and others. This inference that more highly exposed members of the population may be at risk for various noncancer effects is supported by observations in animals, by human information, and by other scientific observations.

The deduction that humans are likely to respond with noncancer effects from exposure to dioxin-like compounds is based on the fundamental level at which these compounds impact cellular regulation and the broad range of species which have proven to respond adversely. Since, for example, developmental toxicity following exposure to TCDD-like congeners occurs in fish, birds, and mammals, it is likely to occur at some level in humans. It is impossible to state exactly how or at what levels individuals in the population will respond with adverse impacts on development or reproductive function, but some subtle effects on development have been noted in infants at near background exposures. Fortunately, there have been few human cohorts identified with TCDD exposures exceeding the high end of the background exposure range. When these cohorts have been examined, few clinically significant effects were detected. The focus of most currently available epidemiologic studies on occupationally TCDD-exposed adult males makes evaluation of noncancer effects in the general population difficult. It is important to note, however, that when exposures to very high levels of dioxin-like compounds have been studied, such as in the Yusho and Yu-Cheng cohorts, a spectrum of adverse effects have been detected in men, women and children. Some have argued that to deduce that a spectrum of noncancer effects will occur in humans in the absence of better human data overstates the science; most scientists in the reassessment as authors and reviewers have indicated that such an inference is reasonable given the weight-of-the-evidence from available data. As presented, this logical conclusion represents a testable hypothesis that may be evaluated by further data collection as more sensitive methods for evaluating human responses to dioxin exposure become available.



With regard to carcinogenicity, EPA characterizes the complex mixtures of dioxin to which people are exposed as a "likely human carcinogen."[2] This is based on the fact that individual components of this mixture could be characterized as " human carcinogens" or "likely human carcinogens" under EPA's draft cancer risk assessment guidelines (1996, 1999). In particular, TCDD, the most toxic of the dioxins, can be identified as a "human carcinogen" under the Agency's draft guidelines, based on the weight of the animal and human evidence, and the other dioxins as "likely human carcinogens." The epidemiological data alone are not yet deemed sufficient to characterize the cancer hazard of TCDD as being a "human carcinogen." However, combining consistent, suggestive evidence from epidemiology studies with the unequivocal evidence in animal studies and inferences drawn from mechanistic data supports the characterization of complex mixtures of dioxin and related compounds as "likely" cancer hazards. The confidence in this statement for specific environmental

[2] "Human carcinogen" and "likely" to present a cancer hazard to humans are descriptors which are consistent with the latest draft revised EPA Guidelines on Carcinogen Risk Assessment (1996, 1999). They are roughly equivalent to the terms "known" and "probable" human carcinogen which were contained in earlier (1986) EPA guidelines.

mixtures increases with the level of available congener-specific information. It is important to distinguish this statement of cancer hazard from the evaluation of cancer risk. While major uncertainties remain, efforts of this reassessment to bring more data into the evaluation of cancer potency have resulted in an estimate of 1 x $10^{-3}$ per pgTEQ/kgBW/day. This slope factor and resulting risk specific dose estimate represents a plausible upper bound on risk based on evaluation of human and animal data within the range of observation and at a minimally detectable response level ($ED_{01}$). These values are approximately 10 times higher than previous estimates (1985, 1994) which were based on fewer data. Considering the slope factors and current intake levels, upper bound (>95%-ile) risks for the general population may exceed $10^{-3}$ (1 in 1,000). "True" risks are not likely to exceed this value, are likely to be less, and may even be zero for some members of the population. The extent of cancer risk will depend on such parameters as route and level of exposure, overall body burden, dose to target tissues, individual sensitivity and hormonal status. This estimate of upper bound risk for the general population has increased from the risk described at background exposure levels based on EPA's earlier (1994) draft of this reassessment ($10^{-4}$-$10^{-3}$).

The current evidence suggests that both receptor binding and most early biochemical events such as enzyme induction are likely to demonstrate low-dose linearity. The mechanistic relationship of these early events to the complex process of carcinogenesis remains to be established. If these findings imply low-dose linearity in biologically-based cancer models under development, then the probability of cancer risk will be linearly related to exposure to TCDD at low doses. Until the mechanistic relationship between early cellular responses and the parameters in biologically based cancer models is better understood, the shape of the dose-response curve for cancer below the range of observation can only be inferred with uncertainty. Associations between exposure to dioxin and certain types of cancer have been noted in occupational cohorts with average body burdens of TCDD approximately 1-3 orders of magnitude (10 to 1,000 times) higher than average TCDD body burdens in the general population. In terms of total TEQ, the average body burden in these occupational cohorts level is within 1-2 orders of magnitude (10-100 times) of average background body burdens in the general population. Thus, there is no need for large scale low dose extrapolations to estimate upper bounds on general population cancer risk or to evaluate the impact of incremental exposures above background. Nonetheless, the relationship of apparent increases in cancer mortality in these populations to calculations of general population risk remains uncertain.



In summary, based on all of the data reviewed in this reassessment and scientific inference, a picture emerges of TCDD and related compounds as potent toxicants in animals with the potential to produce a spectrum of effects. Some of these effects may be occurring in humans at very low levels and some may be resulting in adverse impacts on human health. The potency and fundamental level at which these compounds act on biological systems appears to be analogous to several well studied hormones. Dioxin and related compounds have the ability to alter the pattern of growth and differentiation of a number of cellular targets by initiating a cascade of biochemical and biological events with the potential for a spectrum of responses in animals and humans. Despite this potential, and given the limited body of epidemiological evidence associating dioxin exposure with increases in various effects, there is currently no clear indication of increased disease in the general population attributable to dioxin-like compounds. The lack of a clear indication of disease in the general population should not be considered strong evidence for no effect of exposure to dioxin-like compounds. Rather, lack of a clear indication of disease is more likely a result of the inability of our current data and scientific tools to directly relate effects to dioxin exposure and related compounds at these levels of human exposure. Several factors suggest a need to further evaluate the impact of these chemicals on humans at or near current background levels. These are: the weight of the evidence on exposure and effects; an apparently low margin-of-exposure for noncancer effects; and potential for significant risks to some portion of the general population and

additivity to background processes related to carcinogenicity in the case of incremental exposures above background.

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# Dioxin Emissions from a Solid Waste Incinerator and Risk of Non-Hodgkin Lymphoma

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**Background:** It is not clear whether low environmental doses of dioxin affect the general population. We previously detected a cluster of patients with non-Hodgkin lymphoma around a French municipal solid waste incinerator with high dioxin emissions. To explore the environmental route suggested by these findings, we carried out a population-based case-control study in the same area.
**Methods:** We compared 222 incident cases of non-Hodgkin lymphoma diagnosed between 1980 and 1995 and controls randomly selected from the 1990 population census, using a 10-to-1 match. Dioxin ground-level concentrations were modeled with a second-generation Gaussian-type dispersion model, yielding four dioxin exposure categories. The latter were linked to individual places of residence, using Geographic Information System technology.
**Results:** The risk of developing non-Hodgkin lymphoma was 2.3 times higher (95% confidence interval = 1.4–3.8) among individuals living in the area with the highest dioxin concentration than among those living in the area with the lowest dioxin concentration. No increased risk was found for the intermediate dioxin exposure categories. Adjustment for a wide range of socioeconomic characteristics at the block group level did not alter the results.
**Conclusion:** Although emissions from incinerators are usually not regarded as an important source of exposure to dioxins compared with other background sources, our findings support the hypothesis that environmental dioxins increase the risk of non-Hodgkin lymphoma among the population living in the vicinity of a municipal solid waste incinerator.

**Key Words:** case/control study, dioxins, geographic information system, incineration, non-Hodgkin lymphoma, waste management

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Dioxin is the name given to two classes of organochlorine compounds, 75 polychlorinated dibenzo-*p*-dioxins (PCDD) and 135 polychlorinated dibenzofurans (PCDF). Seventeen tetrachloro-substituted congeners are toxic, with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) being the most potent. Environmental contamination by dioxin can happen by several routes: combustion (waste incineration, burning of various fuels, other high temperature sources such as cement kilns); metals smelting, refining and processing (iron ore sintering, steel production, scrap metal recovery); chemical manufacturing (chlorine bleached wood pulp, chlorinated phenols, chlorinated aliphatic compounds); biologic and photochemical processes (action of micro organisms on chlorinated phenolic compounds); and reservoir sources (soils, sediments, biota, water).

The U.S. Environmental Protection Agency[1] and the International Agency for Research on Cancer[2] have classified 2,3,7,8-TCDD as a human carcinogen. Non-Hodgkin lymphomas and soft-tissue sarcomas have been associated with occupational or accidental exposures to chemicals contaminated with dioxins.[3–7] Aside from the studies involving heavy exposures in industrial settings, few studies have looked at the impact of environmental exposure to dioxins on the health of the general population.[8] Dioxin emissions from municipal solid waste incinerators are one of the major sources of dioxins and therefore are an exposure source of public concern.

Our team recently examined the spatial distribution of non-Hodgkin lymphomas and soft-tissue sarcomas around a French solid waste incinerator with high dioxin emission levels.[9] Some legal guidelines for incinerator emissions had not been followed at this location. For example, in 1997, dust and hydrogen emission levels were higher than prescribed and exhaust gases were not maintained at temperatures of

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more than 850°C for the legal time (>2 seconds), allowing dioxins to be emitted. The first time that the dioxin concentration of an exhaust gas was ever measured (in December 1997) it was found to be 16.3 ng international toxic equivalency factor (I-TEQ)/$m^3$, whereas the European guide value is 0.1 ng I-TEQ/$m^3$.

Using a spatial scan statistic, we found evidence for clusters of non-Hodgkin lymphoma and soft-tissue sarcoma in the area that contains the solid waste incinerator. Standardized incidence ratios were 1.3 (95% confidence interval [CI] = 1.1–1.4) for non-Hodgkin lymphoma and 1.4 (CI = 1.1–1.9) for soft-tissue sarcoma.

These results suggested an airborne route of dioxin exposure, which is at variance with the common assumption that intake from food accounts for over 90% of the burden of dioxins in the general human population.[2] This assumption may not hold for people living in the vicinity of a solid waste incinerator, however.[1] Possible exposure pathways include direct exposure (vapor inhalation or dermal absorption) and, more likely, the consumption of plant products or poultry from contaminated areas.

To address this issue further, we carried out a population-based case-control study on the population living around this solid waste incinerator, focusing on non-Hodgkin lymphoma (which is more frequent than soft-tissue sarcoma, thus improving the precision of the estimates). We had information on non-Hodgkin lymphoma incidence and census data which could be geocoded. A dioxin dispersion model made it possible to link ground-level dioxin concentrations to individual addresses (using Geographic Information System [GIS] technology).



## METHODS

### Selection of Cases and Controls

The most likely cluster in our previous study consisted of the cantons of Audeux and Besançon. Detailed census data (needed to sample population controls) were available only for the city of Besançon, with an average population during the study period of 114,000 inhabitants. We therefore limited our study to this zone, excluding 29,000 inhabitants of Audeux. Besançon is the regional capital, with a stable urban population (113,000, 113,000 and 117,000 inhabitants in 1982, 1990 and 1999, respectively), spread over 65 $km^2$.

The municipal solid waste incinerator under investigation is located 4 km west of the city center. Combustion chambers 1 and 2 (each with a capacity of 2.1 metric tons per hour) were put into service in 1971. In 1976, a third combustion chamber was opened (with a capacity of 3 metric tons per hour). In 1998, approximately 67,000 metric tons of waste were processed. Combustion chamber 1 (the most polluting) was shut down on December 31, 1998.

We obtained non-Hodgkin lymphoma incidence data for 1980–1995 from the Doubs cancer registry. The period we studied is before the first public concern about putative effects of municipal solid waste incinerators. This registry was established in 1976 and is complete for non-Hodgkin lymphoma cases, as ascertained by the ratio of the number of deaths to the number of cases registered during 1983–1987, which at 47% (for the Doubs region) is very similar to those reported in other Western countries.[10] Virtually all cases were histologically verified (97% among men and 99% among women). We collected data concerning the patients' address at diagnosis, date of birth, gender, cancer diagnosis and age at diagnosis from their medical records. *International Classification of Disease for Oncology* (ICD-O) morphology codes were 9590/3-9595/3, 9670/3-9723/3 and 9761/3.

We selected controls from a reliable and accessible database, the population census. Because of French privacy laws and confidentiality requirements the only individual data available to researchers are sex, age categories (0–19, 20–39, 40–59, 60–74 and 75+ years), and residence in a given block. The block is the smallest level of geographic resolution in the French census database and is defined only in densely populated areas. Each block is typically a quadrangle bounded by four streets. First described in the 1990 census, there are 705 blocks in Besançon, averaging 161 inhabitants.

We randomly selected population-based controls, according to a 10-to-1 matching procedure. Matching criteria were sex and age, producing 10 strata. To adjust for differences between the index case diagnosis year (1980–1995) and 1990 (year of census), cases were matched to controls based on the age they would have been in 1990.

Risk factor data were limited to what was available through the census either on an individual level or on a block group level. The 705 blocks of the study area are combined into 52 groups for analysis of socioeconomic status measures (educational, occupational, household-based indicators).

### Data Analysis

We used residential address geocoding to pinpoint the location of case residences.[11] A municipal GIS analyst matched a file containing participants' addresses (street and number) against a street network file, using Star GIS software (Star Informatic, Liege, Belgium). The geographic coordinates of these exact locations were expressed in the Lambert two French-plane coordinate system.

To estimate dioxin exposure, we took advantage of a study performed in 1999 to support an environmental impact statement for a new combustion chamber. The work was carried out by Aria Technologies, Colombes, France, using APC3 software. APC3, a second-generation Gaussian-type dispersion model, allowed the three-dimensional modeling of the transport and dispersion of dioxin emissions. The model took into account meteorological data (5 years of data for

windspeed, wind direction, pressure, temperature and atmospheric stability), surface topography and obstacle descriptions, stack characteristics and dioxin emission rate from the solid waste incinerator. It assessed average concentrations in hundreds of meteorological conditions (one Gaussian plume for each particular meteorological condition). The respective contours of these modeled ground-level concentrations (0.0001 pg/m$^3$, 0.0002 pg/m$^3$, 0.0004 pg/m$^3$, 0.0016 pg/m$^3$) were digitized and transferred onto the surface of the map (Fig. 1).

This model was originally developed to predict the future impact of dioxin emissions, both from an old (but renewed) combustion chamber and from a new oven with up-to-date pollution controls. It was not possible to assess past exposure because past dioxin emission rates had not been collected. However, dispersion modeling is heavily influenced by factors that are stable over time (mean meteorological conditions, terrain elevations and stack height). Thus, we assumed that contour shapes, as derived from the prediction model, were reliable estimates of past dioxin deposition profiles and we used dioxin ground-level concentrations as relative figures rather than absolute figures to estimate past exposure. Hence, in the remaining part of this paper, the contours are classified as very low (modeled ground-level dioxin concentration <0.0001 pg/m$^3$ zone), low (modeled ground-level dioxin concentration 0.0001–0.0002 pg/m$^3$ zone), intermediate (modeled ground-level dioxin concentration 0.0002–0.0004 pg/m$^3$ zone) and high (modeled ground-level dioxin concentration 0.0004–0.0016 pg/m$^3$ zone) exposure areas.

We overlaid a map of case residences onto the digital dioxin concentration map to obtain a field—for risk—classification for each cancer patient. In the same way, we attributed a dioxin concentration category to each of the 705 city blocks and 52 block groups (provided half or more of their area was within a given contour).

We used conditional logistic regressions to calculate odds ratios (ORs) and 95% CIs for each level of dioxin

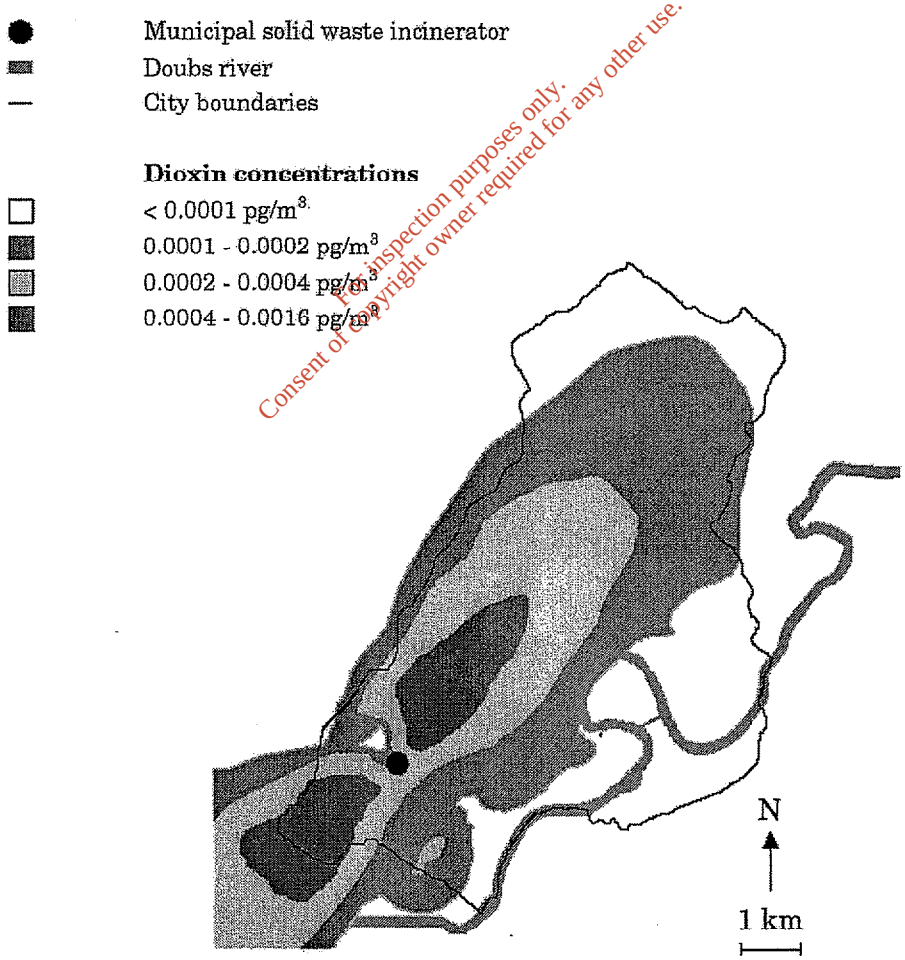




**FIGURE 1.** Modeled average ground-level dioxin concentrations around the municipal solid waste incinerator of Besançon, France.

**TABLE 1.** Socioeconomic Characteristics (Defined at the Block Group Level) of Dioxin Exposure Zones

| | Very Low (N = 11) | Low (N = 21) | Intermediate (N = 14) | High (N = 6) |
|---|---|---|---|---|
| Persons with a high school diploma (%) | 34 | 30 | 27 | 28 |
| Women in labor force (%) | 47 | 49 | 48 | 55 |
| Workers in labor force (%) | 17 | 24 | 26 | 23 |
| Unemployed in labor force (%) | 13 | 15 | 15 | 13 |
| Single woman as head of household (%) | 7 | 8 | 11 | 9 |
| Owner-occupied houses (%) | 35 | 32 | 29 | 36 |
| Number of persons per dwelling (mean) | 2.2 | 1.9 | 2.2 | 2.2 |
| Single-family houses (%) | 35 | 14 | 12 | 30 |

exposure estimated from the dispersion model. A set of dummy variables was generated for this categorical scale variable using the lowest category as the reference group. Models were run with Egret for Windows software (CYTEL Software Corporation, Cambridge, MA).

Multilevel models were run to explain the outcome (case/control status) defined at the individual level, while introducing risk factors at the individual level (dioxin exposure) and the block group level (socio-economic characteristics). MlwiN software (Institute of Education, London, United Kingdom) was used to carry out these analyzes.

## RESULTS

As expected, the risk of airborne dioxin exposure was not distributed evenly throughout the population. The distribution of dioxin fall-out was characterized by a skewed distance distribution, following a northeast to southwest direction, with peaks at varying distances on both sides of the incinerator (Fig. 1). The asymmetry of the distribution is caused by the foot-hills of the Jura mountains, which channel the wind preferentially in two directions. Socioeconomic characteristics (education, occupational social class and household-based indicators), defined at the block group level, did not vary with dioxin exposure category (Table 1).

During the 16-year study period, 225 non-Hodgkin lymphoma cases were diagnosed, corresponding to a mean age-standardized (world) incidence rate of 14.9 per 100,000 for the 1980–1995 time period. The age-standardized (world) incidence rate for France as a whole was estimated at 7.8 per 100,000 in 1995.[12] Address matching was successful for 222 cases (three medical records had incomplete address information). Eighty percent of non-Hodgkin lymphoma cases occurred within the 1990 ± 5 year time range and the proportion of males was 51%. The age distribution was slightly skewed toward young ages (lower quartile: 49 years; median: 66 years; upper quartile: 77 years).

The distribution of these cancer patients by dioxin exposure categories is displayed in Table 2. The conditional logistic regression analysis showed that individuals living in the highest exposed zone were 2.3 times more likely (CI = 1.4–3.8) to develop non-Hodgkin lymphoma than were individuals living in the very low emission area, with no increased risk for the other dioxin risk categories (Table 2).

The results of an analysis restricted to the non-Hodgkin lymphoma cases diagnosed between 1985 and 1995 were very similar: ORs were 1.3 (CI = 0.8–2.0), 1.0 (CI = 0.6–1.6) and 2.1 (CI = 1.1–3.7), for the low, intermediate and high dioxin exposure categories, respectively.

Adjustment for a wide range of block group characteristics (those reported in Table 1), introduced in turn in a 2-level hierarchical model, did not alter the results. Inclusion of socioeconomic status measures resulted in ORs ranging from 0.9 to 1.0, 0.9 to 1.0 and 2.1 to 2.4, for the low, intermediate and high dioxin exposure categories, respectively.

## DISCUSSION

We found a 2.3-fold risk for non-Hodgkin lymphoma associated with residence in areas classified as highly exposed to dioxin emitted from a municipal solid waste incinerator (as estimated by an airborne dispersion model), com-

**TABLE 2.** Association of Non-Hodgkin Lymphoma with Dioxin Exposure Categories,* City of Besançon, France, 1980–1995

| Dioxin Exposure | Non-Hodgkin Lymphoma Cases | Controls | OR (95% CI) |
|---|---|---|---|
| Very low† | 42 | 441 | 1.0 |
| Low | 91 | 952 | 1.0 (0.7–1.5) |
| Intermediate | 58 | 681 | 0.9 (0.6–1.4) |
| High | 31 | 146 | 2.3 (1.4–3.8) |

* From Aria Technologies modelling (with APC3 software).
† Reference category.

pared with very low exposure areas; the low and intermediate exposure categories did not exhibit an excess risk.

The strengths of this study are as follows. First, it was a population-based design. Cases were actively identified through multiple sources within a defined geographic area and controls were randomly selected from the same study area as the cases. The 10-to-1 matching procedure produced fairly precise relative risk estimates, as reflected by the narrowness of the corresponding confidence intervals (Table 2).

Second, we were able to use dioxin exposure data based on sophisticated methods for modeling of emissions.[13] The modeled ground-level concentrations represented the best available surrogates for past dioxin exposure measurements from the same source, given that no earlier measurements had been taken.

Third, this GIS-based case-control study improved upon the conventional case-control design. This study was based on a complete directory of Besançon city residents (census data), with a modest but relevant and reliable list of characteristics available at low cost. The amount of information does not increase proportionally with the size of the control group; a ratio of around 4 or 5 is usually considered to be a good trade-off. However, when the cost of additional information is negligible, a high control/case ratio is justified.[14] We decided a priori on a ratio of 10, which was kept constant across the strata. This considerably enhanced the precision and thereby improved the efficiency of the study.

Fourth, we carried out a sensitivity analysis based on multilevel modeling. More complex in theory and practice, it can demonstrate the independent effects of area characteristics and individual factors.

However, our methodology also presented some limitations. First of all, we lacked actual exposure data regarding biota in the contaminated area and exposed humans. In 1998, concentrations of dioxins in cow's milk from farms located within a 3-km radius of the incinerator were requested by public health authorities. Only four farms met the criterion, and one of these farms was not involved in cattle breeding. Dioxin concentrations (in ng I-TEQ/kg of fat) and distances between the farms and the plant were as follows: 1.03 (0.9 km), 0.59 (1.5 km) and 0.58 (2.0 km). However, the sampling frame was questionable as only one farm (with the highest dioxin level) was located under the plume of the incinerator's stacks.

To circumvent this lack of actual exposure data, we used dispersion modeling as a proxy for dioxin exposure, assuming that residents within a given contour were homogeneously exposed. When interpreting the results it is important to remember that this model was developed for regulatory purposes rather than as a means of assessing exposure to air pollution.[15] Furthermore, its representativeness of exposure over time had to be assumed in this study of long-term effects, because no data concerning dioxin emission levels are available for the period before 1997. Residence location as a surrogate of exposure cannot distinguish contributions from the direct and the indirect exposure pathways (*eg*, from air to soil and home-grown produce). We lacked the necessary household and soil measurements to confirm the validity of the dispersion model. Moreover pollutant-specific deposition modelings are interrelated and thus pollutant effects are difficult to separate. Emissions of dust and hydrogen chlorine, which were also above the legal limits, could be important if these exposures are associated with occurrence of non-Hodgkin lymphoma (but, to our knowledge, no such associations have been described).

This study is of mixed individual/ecological design with case residences linked to the dispersion map by exact address whereas control residences are at the block level. Thus, although census blocks have a limited area (decreasing the distance between actual and surrogate locations) and were assigned one of four exposure levels prior to control sampling, the possibility of some differential exposure misclassification cannot be ruled out.

Controls were residents in 1990, whereas cases were diagnosed between 1980 and 1995, introducing a time lag in the sampling for some matched sets. We believe that the shortness of this time lag did not affect the coverage of the target population. First, restricting the analysis to cases diagnosed between 1985 and 1995 did not alter the results. Second, the population of Besançon appears to be stable over time for the age groups considered; 86% of the people over 40 years of age who lived in Besançon in 1999 were already residing in the city in 1990. Third, if some housing development occurred during this short time period, there is no reason to believe that it was related to dioxin risk categories, for which modeling was performed in 1999. We conclude that the effect (if any) of such a short time lag would be to bias the odds ratios towards 1.0.

The lack of information pertaining to residence history and time-activity patterns limited our ability to ascertain the duration of exposure. Considering the long exposure-to-effect interval, some subjects might have lived elsewhere at the times of relevant exposure or have been lightly exposed to dioxins from the incinerator. However, this potential misclassification is likely to be random with respect to disease status, resulting in a bias of our risk estimates towards the null.[16]

Regarding other occupational or environmental sources of exposure to dioxins, there are no adjacent industrial sources of combustion-effluents; highly polluting industries were replaced 2 decades ago by small-scale advanced technologies. Before that time, the main factory (producing synthetic textiles) was located 5 km east of the city center, in the very low dioxin exposure area. No cement kilns, iron or steel works, or foundries were located in this area. Other potential thermal and combustion sources, such as automobile exhausts and home heating, result in diffuse, nonspatially organized

emissions. Alternatively, there is mounting evidence implicating phenoxy herbicides in the etiology of non-Hodgkin lymphomas. In general, but not consistently, positive associations have been found between occupational exposure to herbicides and non-Hodgkin lymphoma in case-control studies, whereas results from follow-up studies are less suggestive of an association.[17] In any case, Besançon is highly urbanized with few pastures (only four farms within a 3-km radius of the MSW incinerator). Thus, in our opinion, neither other factories nor farmland are likely to affect the interpretation of these results.

This study was also limited by the scarcity of covariates (only age and gender), which could potentially confound the relationship between dioxin exposure from the municipal solid waste incinerator and non-Hodgkin lymphomas. However, most reports of mortality and incidence data for lymphomas show no clear association with social class.[18] In our study, the similarities across block groups characterized by differing exposure levels are reassuring. Still, the possibility that there are other differences between subjects living in the highest exposure zone and those with lower exposures cannot be ruled out. For example, the percentage of subjects with a high school diploma is similar, but income may vary, or there may be differences at the high or low end of the education scale.

Thus, for all the above-mentioned reasons, we cannot firmly exclude the possibility that residual confounding affected the reported odds ratios.

We found that the risk of non-Hodgkin lymphoma incidence was elevated in the highest dioxin concentration category, suggesting a possible threshold effect. However, as we used a ranking system rather than actual measurements to classify exposure levels we cannot be more precise about this threshold level.

Other researchers have concluded that the health risks caused by dioxin emissions from incinerators are relatively unimportant compared with other background sources of dioxins.[19,20] Deml et al[21] found no indication of an enhanced body burden of dioxins and furans in nonoccupationally exposed persons living in the vicinity of a municipal waste incinerator. Dioxin and furan emissions in their study resulted in values of about 2 ng I-TEQ/m$^3$, which is much lower than levels from the Besançon incinerator (16.3 ng I-TEQ/m$^3$). Gonzales et al[22] showed that blood dioxin levels did not depend on the distance of residence from a Spanish incinerator with similarly low dioxin stack emissions (2.5 ng I-TEQ/m$^3$).

In a cancer risk assessment of dioxin and furan emissions from a municipal solid waste incinerator, Nessel et al[23] estimated the lifetime total cancer risk as $2.5 \times 10^{-6}$ in the highly exposed scenario, compared with $1.8 \times 10^{-7}$ in the common scenario (representative of the general population), which is a 10-fold difference. They nevertheless concluded that the relatively low magnitude of these risks suggests that the dioxin and furan emissions from this incinerator should not be of public health concern. Yoshida et al[24] showed that life-time cancer risk for residents living within 1 km of a municipal solid waste incinerator (and perhaps because of dioxin exposure), were twice as high as those of the general population, but considered these results sufficient to guarantee safety. In a recent quantitative risk assessment, Ma et al[25] found that the carcinogenic risk of dioxins (all cancers) ranged from $1 \times 10^{-6}$ (under the exposure scenario of insufficient local food production for residents' consumption) to $7.1 \times 10^{-5}$ (under the exposure scenario of sufficient local food production), for the most polluting of nine Taiwanese incinerators (6.67 ng I-TEQ/m$^3$).

However, our findings are in line with the results provided by Bertazzi et al[26] on the 20-year mortality of the Seveso population. People in the Seveso cohort had mean TCDD blood lipid concentration of 136 ng TCDD/kg, which falls between the typical occupational dioxin levels (> 1,000 ng TCDD/kg) and background levels (2–3 ng TCDD/kg).[27] Allowing for a latency time window of 15–20 years, results for non-Hodgkin lymphomas clearly did stand out, according to Bertazzi et al,[28] with a relative risk of 2.8 (CI = 1.1–7.0).

In summary, we find an increased risk of non-Hodgkin lymphoma in the highest exposure zone around a municipal solid waste incinerator that emitted high levels of dioxins. This finding, together with the non-Hodgkin lymphoma mortality excess reported by Bertazzi et al[26,28] around Seveso, lends support to the hypothesis that airborne dioxin exposure may be a public health concern.



## ACKNOWLEDGMENTS

We thank François Tourneux and Annibale Biggeri for their technical assistance. We are grateful to Robert Schwint (former mayor of Besançon) for allowing the support of the GIS municipal staff (Roland Draussin and Jean-Pierre Guivier) and for transmitting the dioxin diffusion model.

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# ORAL HEARING INTO

# PROPOSED DECISION 167 – 1

# Carranstown WASTE MANAGEMENT FACILITY

# PROOF OF EVIDENCE



Dr. Paul Connett
Professor of Chemistry
St. Lawrence University
Canton, NY
paul@fluoridealert.org

## County Meath,Ireland
## March 11, 2005.

A) The dioxin issue.

- B) A critique of Dr. Fergal Callaghan's submission on the projected dioxin doses from this project.
- C) A critique of Mr. Ahearn's submission.

# Dioxins -The biology

- Dioxins and dioxin-like compounds are fat soluble and easily cross membranes and enter cell
- Once in the cell they fit into a protein called the Ah receptor
- Another protein joins this combination
- This complex enters the nucleus and attaches to the DNA
- It doesn't cause mutations, but it does switch on genes
- Switching on genes results in the production of new proteins in the cell.
- In other words it functions like a fat soluble hormone.

# Two remarkable things about the Ah receptor

- 1) After 30 years of research scientists do not know what it is in the cell for. Have not identified its normal function.

- 2) The Ah receptor appears in evolution at the same time as the backbone appears in fish. Every species above invertebrates has the Ah receptor.

# The presence of dioxin can interfere with the levels of a number of key substances in a living cell

- **ENZYMES:**
- Cytochrome P4501A1, Cytochrome P4501A2, DT-Diaphorase, UDP-Glucuronyl Transferase,Glutathione-S-Transferase, Aldehyde Dehydrogenase, Ornithine Decarboxylase, Tyrosine Kinase,Thymidylate Transferase, Phosphoenolpyruvate Carboxykinase, Plasminogen Activator Inhibitor 2…
- **HORMONES & HORMONE RECEPTORS:**
- Androgens, Estrogens, Estrogen receptor, Glucocorticoid, Glucocorticoid receptor, Insulin, Insulin Growth Factor, Thyroid Hormones, Gastrin…
- **GROWTH & DIFFERENTIATION FACTORS:**
- Ras, Myc and Erb Oncogenes, EGF Receptor, TGF-alpha, TGF-beta 1, Beta 1, TNF-alpha, IL1-beta…

# Dioxins - Major Concerns

- **One liter of cows' milk gives the same dose of dioxin as breathing air next to the cows for EIGHT MONTHS (Connett and Webster, 1987).**
- **In one day a freely grazing cow puts the equivalent of 14 years of human breathing into its body (McLachlan, 1995)!**
- **The liver cannot convert dioxins to water soluble products thus they steadily accumulate in human body fat.**
- **The man cannot get rid of them BUT A woman can…**
- **By having a baby!**
- **Thus, the highest dose of dioxin goes to the fetus during pregnancy and then to the new born infant via breastfeeding.**

# 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)

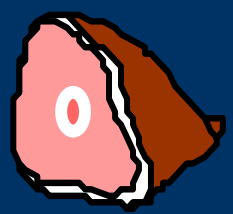
# Effect of Dioxins on Neonatal Thyroid Function after Low-exposure and High-exposure at various ages

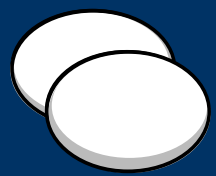
| | | Low-exposure (mean) | **High-exposure (mean)** | P* |
|---|---|---|---|---|
| At birth | T4 | 122.5 | 134.3 | 0.071 |
| | T4/TBG | 0.240 | 0.232 | 0.45 |
| | TSH | 10.4 | 11.9 | 0.58 |
| 1week | T4 | 154.5 | 178.7 | 0.006 |
| | T4/TBG | 0.291 | 0.332 | 0.006 |
| | TSH | 2.93 | 2.56 | 0.51 |
| 11weeks | T4 | 111.1 | 122.2 | 0.033 |
| | T4/TBG | 0.220 | 0.247 | 0.040 |
| | TSH | 1.81 | 2.50 | 0.044 |

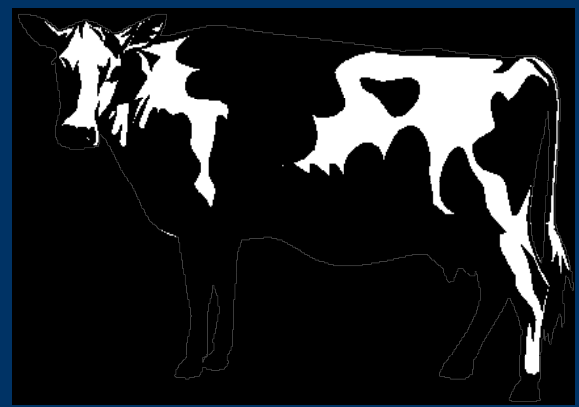
# DIOXINS IN OUR FOOD

- Dioxins are fat soluble and persistent and accumulate in the food chain, specially animal fats. Well over 90% of our dioxin intake comes from dairy products, meat,and fish.







# Dioxin in cow's milk

## pg I-TEQ/g fat (ppt)

- Denmark 2.6
- Finland 0.83 - 1.17
- France 1.81
- Germany 0.71 - 0.87
- Ireland 0.08 - 0.51 Average in Ireland = 0.2
- Netherlands 0.38 - 1.6
- Spain 1.2 - 2.0
- Sweden 0.93 - 2.0
- UK 1.01

Measurements reported in 1999, (IOM, 2003).

# Institute of Medicine, 2003

## Dioxins and Dioxin-like Compounds in the Food Supply



## Strategies to Decrease Exposure

July 1, 2003

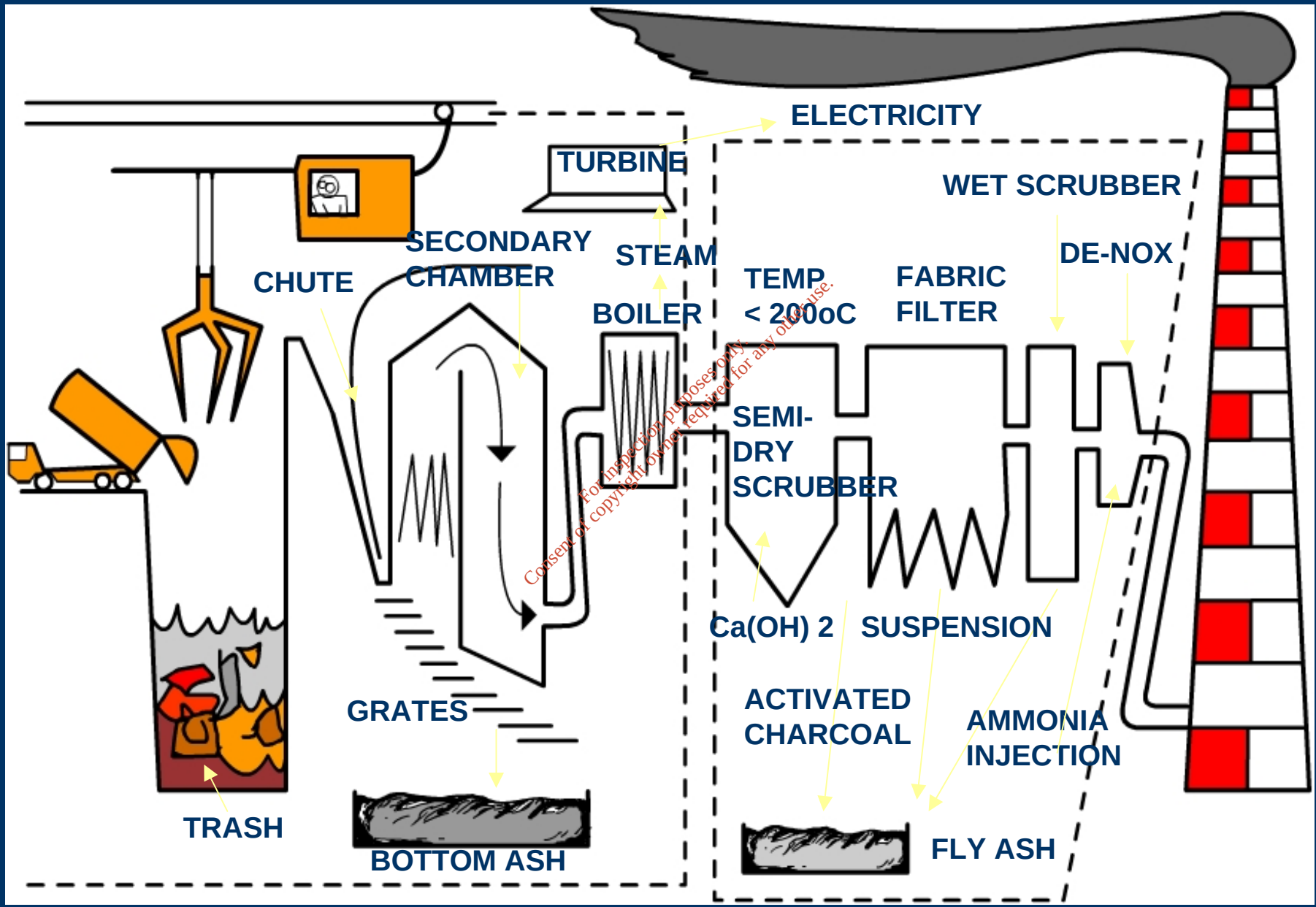
# Institute of Medicine, 2003

- Fetuses and breastfeeding infants may be at particular risk from exposure to dioxin like compounds (DLCs) due to their potential to cause adverse neurodevelopmental, neurobehavioral, and immune system effects in developing systems…

# Institute of Medicine, 2003

- …The committee recommends that the government place a high public health priority on reducing DLC intakes by girls and young women in the years well before pregnancy is likely to occur.

- Substituting low-fat or skim milk, for whole milk… coupled with other substitution of foods lower in animal fat by girls and young women in the crucial years before pregnancy…



# Calculating dioxin dose to MARI

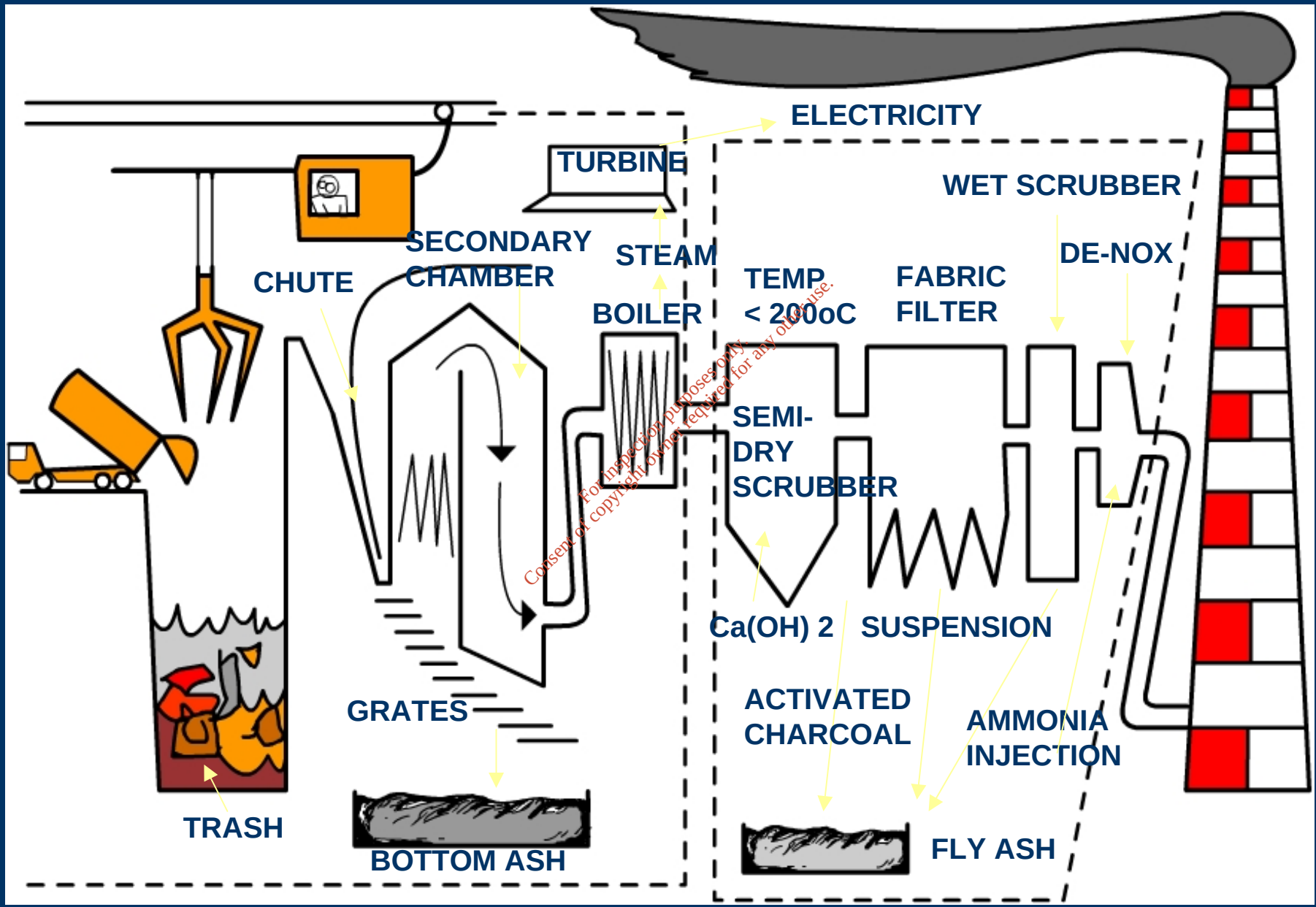
- The predicted increase dose to the “maximum at risk individual” (MARI) receptor would be 0.0027 pg/kg/day (p.9). This cannot be correct.
- On page 8, the “annual average predicted ground level concentration of PCDD/F, from the proposed WTE facility is 1 fg/m3” (note: 1 fg = 0.001 pg).
- This translates into a daily dose from INHALATION of 0.015 pg or 0.00025 pg/kg/day.
-

# Calculating dioxin dose to MARI

- However the dose for a subsistence farmer from ingestion exceeds dose from inhalation by three orders of magnitude say 1000 - 3000 times
- Thus if inhalation dose is 0.00025 pg/kg/day, the ingestion dose will be 1000 - 3000 X 0.00025 i.e. 0.25 - 0.75 pg/kg/day.
- If we add this dose from the facility to the background dose for the MARI of 0.575 pg/kg/day, then the total dose becomes 0.825 - 1.325 pg/kg/day.
- This figure could thus exceed the bottom figure in the WHO ADI of 1-4 pg/kg/day. In my view the 1 pg/kg/day figure is the only one defendable scientifically.
-

# Calculating dioxin dose to MARI

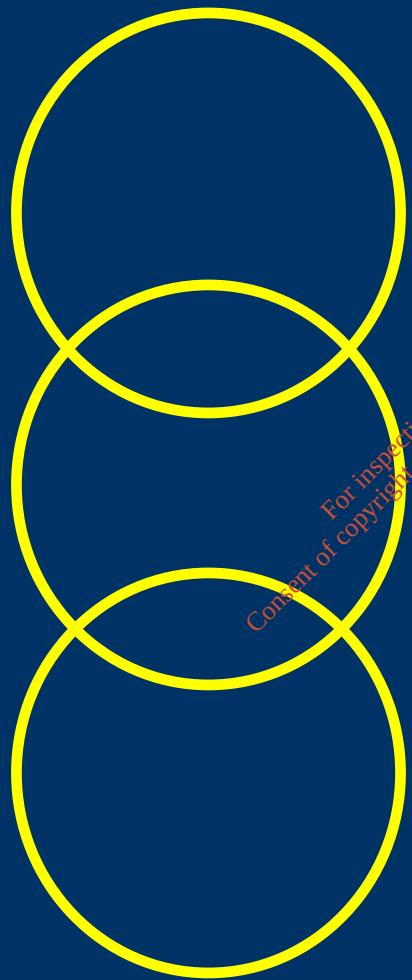
- In American terms, the dose of 0.825 - 1.325 pg/kg/day would translate into an incremental lifetime cancer risk of 825-1325 in a million.
- The US EPA does not permit facilities with a cancer projection rate of over 100 in a million.
- Thus, it is very unlikely that this facility would not be permitted in the US.



# When it comes to dioxin and incinerators

- Governments always say to the citizens
- You don't have to worry
- Because we have tough new air emission standards
- But…

# THE CHAIN OF PUBLIC HEALTH PROTECTION HAS THREE LINKS.



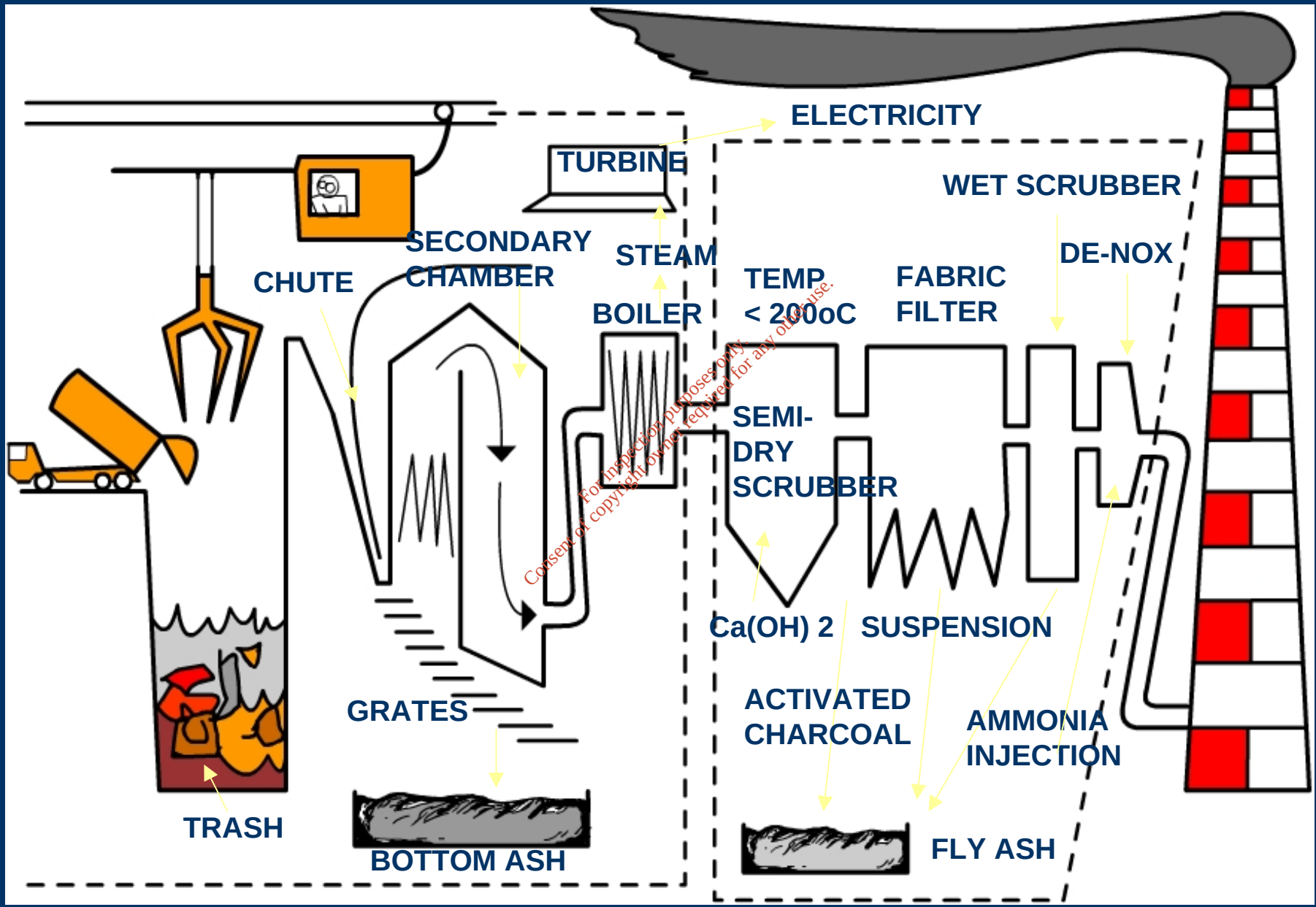
STRONG REGULATIONS

ADEQUATE MONITORING

TOUGH ENFORCEMENT



**IF ANY LINK IS WEAK THE PUBLIC IS NOT PROTECTED**



ZERO WASTE

**NO TO INCINERATION**

**NO TO LANDFILL**

**MOVING TOWARDS THE FRONT END -we need to design waste out of the industrial system**



**NO TO A THROWAWAY SOCIETY**

**ZERO WASTE BY 2020**

**YES TO A SUSTAINABLE SOCIETY**

# To achieve Zero Waste

We need three things:

1) COMMUNITY RESPONSIBILITY (at back end)

2) INDUSTRIAL RESPONSIBILITY (at front end)

3) GOOD LEADERSHIP

# End of Presentation to EPA on March 11, 2005

# Community Responsibility

- Begins with separation of compostables, recyclables and residuals (e.g. San Francisco).
- Drop off centers for household toxics (e.g. Nova Scotia)
- Centers for reuse & repair (and retraining) of appliances and furniture etc (e.g. Burlington, Vermont)
- Deconstruction -not demolition- of old buildings (e.g. Canberra, Australia)
- Residual screening facilities (e.g. Nova Scotia).

# COMMUNITY RESPONSIBILITY BEGINS WITH SEPARATION

1. COMPOSTABLES

2. RECYCLABLES

3. RESIDUALS

LOCAL USE ?

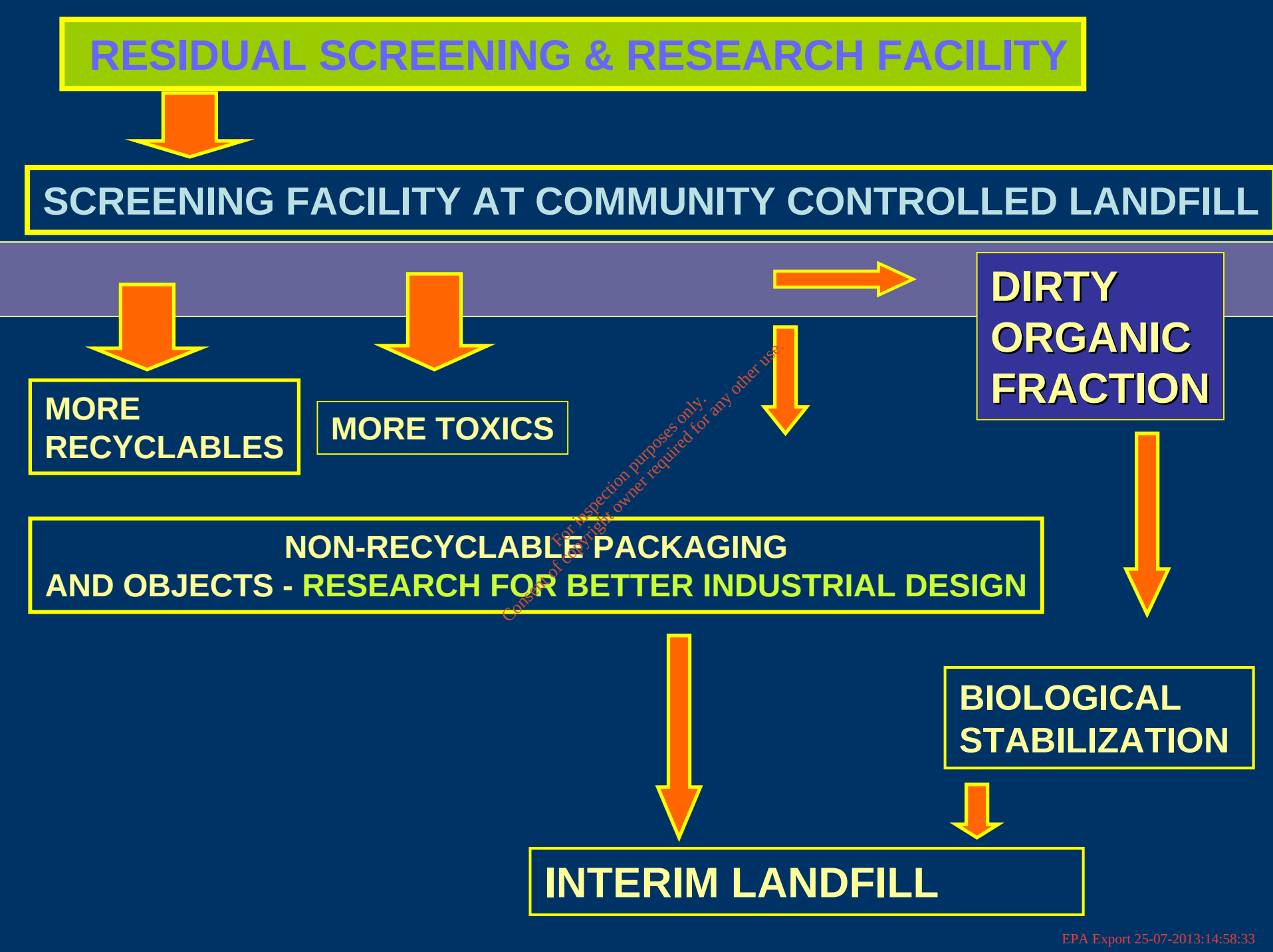
Pay by bag



1. COMPOSTING FACILITY

2. MATERIALS RECOVERY FACILITY & RE-MANUFACTORING

3.RESIDUAL SCREENING & RESEARCH FACILITY



- If we can't re-use it, recycle it or compost
  - industry shouldn't be making it.
- We need better industrial design for the 21st Century.

# A comparison

- With incineration
- You convert three tons of trash to
- one ton of ash
- that nobody wants!

# With a zero waste strategy

- You convert three tons of trash into:
  - One ton of recyclables
  - One ton of compostables,
  - and
  - One ton of education!

- Remember you don’t have to get down to zero to beat incineration because only reduce the amount of waste going to landfill by 70%. 30% is left as toxic ash.
- Canberra has reached 70% diversion in less than 10 years!

# INDUSTRIAL RESPONSIBILITY

1) Better industrial design

2) Extended Producer Responsibility

3) Clean up manufacturing process

# INDUSTRIAL RESPONSIBILITY IN ACTION

## 1) THE BEER INDUSTRY, ONTARIO, CANADA

- Uses refillable glass bottles
- 98% recovered
- Reusable glass bottles 11 cents cheaper per serving than disposable bottles.
- 2000 jobs in collection and cleaning
- No cost to municipality
- Packaging costs internalized

# INDUSTRIAL RESPONSIBILITY IN ACTION

## 2) XEROX CORPORATION EUROPE

- Recovering old copying machines from 16 countries
- Over 95% of materials reused or recycled!
- $76 million saved in 2000 !!


# The San Francisco Program



**Robert Haley**

**Recycling Program Manager**

**Department of the Environment**

**City and County of San Francisco**

# San Francisco

- Population = 850,000
- Little space
- Education has to be done in three languages
- Over 50% diversion reached by 2000
- 63% diversion reached by 2004
- 75% diversion by 2010 (goal)
- 100% diversion by 2020 i.e. Zero Waste

# The “Fantastic 3”

# ALL FOOD SCRAPS, YARD TRIMMINGS AND COMPOSTABLE PAPER GO IN THE GREEN CART

# ALL BOTTLES, CANS AND RECYCLABLE PAPER GO IN THE BLUE CART

# WHAT CAN'T BE RECYCLED OR COMPOSTED GOES IN THE BLACK CART

# PROGRAM BROCHURE OUTSIDE

## The new program

Welcome to the Fantastic 3, the new program designed to make recycling easier, reduce waste and control litter. Now it is easier to recycle by combining all bottles, cans, cardboard and paper in the blue cart. The green cart is for all compostables, such as food scraps and yard trimmings. This program will allow San Francisco to achieve the state-mandated 50% recycling goal.

## Beginning next week

Your new program will start next week. Please note that your collection day might have changed — see attached flyer. Roll your carts curbside the night before your collection day for pick-up.

**Call Sunset Scavenger at 415-330-1300 or visit www.sunsetscavenger.com for more details.**


Printed on Recycled Paper

## Space-saving tips

Recycle your old containers. Make room for your new carts by recycling your old garbage can and recycling bins. Place your old can and bins on the curb the night before the first collection day of the new program. Please be sure they are empty. We will pick them up for recycling at no additional charge. Your old can and bins cannot be used with the new program.

Share with your neighbors. Neighbors with separate accounts can share carts. One 64-gallon cart takes up less room than two 32-gallon carts. Call 415-330-1300 to arrange a cart sharing option.

Got big stuff? Schedule a Bulky Item Pickup. Call customer service at 415-330-1300 to schedule removal of bulky items. Limit two pickups per year.

## Helpful hints

Recycle more and you may save money! Generating less garbage may allow you to use a lower-capacity black cart, reducing your monthly rate. Minimum service is one 20-gallon black cart.

Larger carts are available. Larger BLUE and GREEN carts are available and will be serviced at no additional charge. Larger BLACK carts are available for an additional monthly charge. For more information or to arrange for a different size cart, call customer service at 415-330-1300 or visit our website at www.sunsetscavenger.com.



# Recycling is now as easy as 1, 2, 3 ...

**Recyclables**
Paper, cardboard, bottles and cans...in the SAME cart!
[redacted]
Todo lo reciclable

**Compostables**
All food scraps, food-soiled paper and yard trimmings!
[redacted]
Todo lo que sirve para hacer abono

**Garbage**
Anything NOT recyclable.
No hazardous materials, please.
[redacted]
Basura

**For more information, call 415-330-1300 or visit www.sunsetscavenger.com**

[redacted]
[redacted] 330-1300 • *

*Si desea una copia de este folleto en español, llame al 330-1300.*

sfrecycles.org
(415)554-RECYCle

People • Service • Environment
SUNSET SCAVENGER COMPANY
Your Local Recycling Company
100 Percent Employee-Owned

# BUS SHELTER AD


# SPLIT COMPACTING SIDE-LOADERS FOR RECYCLABLES AND REFUSE

# Recycle Central

@ Pier 96


# DEDICATED COMPACTING SIDE-LOADERS FOR COMPOSTABLES

# SMALL BUSINESSES ARE COLLECTED WITH RESIDENCES


# Compost Facility

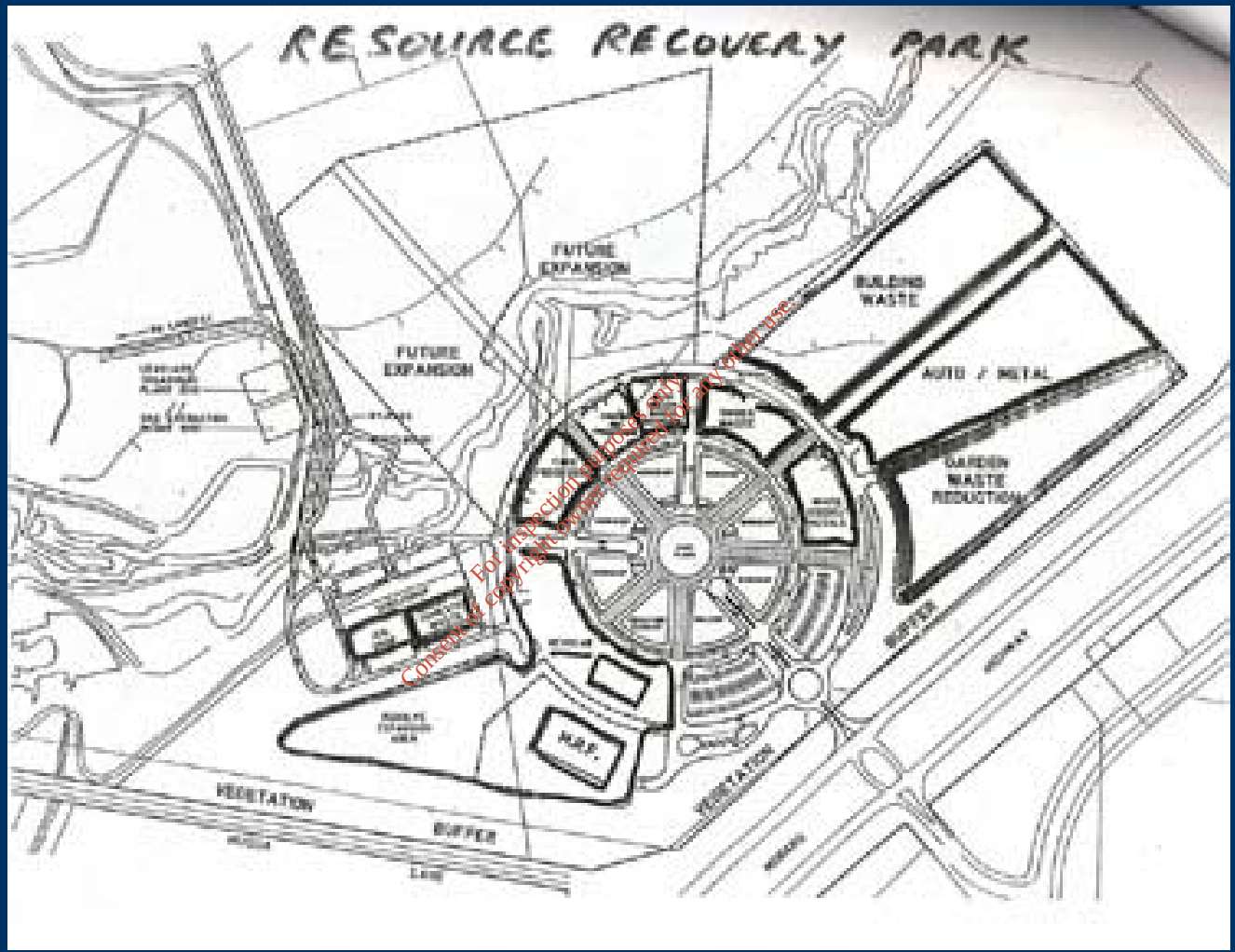
# RICH COMPOST READY FOR MARKET

# ORGANIC PRODUCE RETURNS TO SF MARKETS & RESTAURANTS

# Canberra, Australia.

- Canberra (the capitol of Australia) was the first city worldwide to declare a zero waste goal.

- Law passed in 1996: “No Waste by 2010”



# NEW ZEALAND

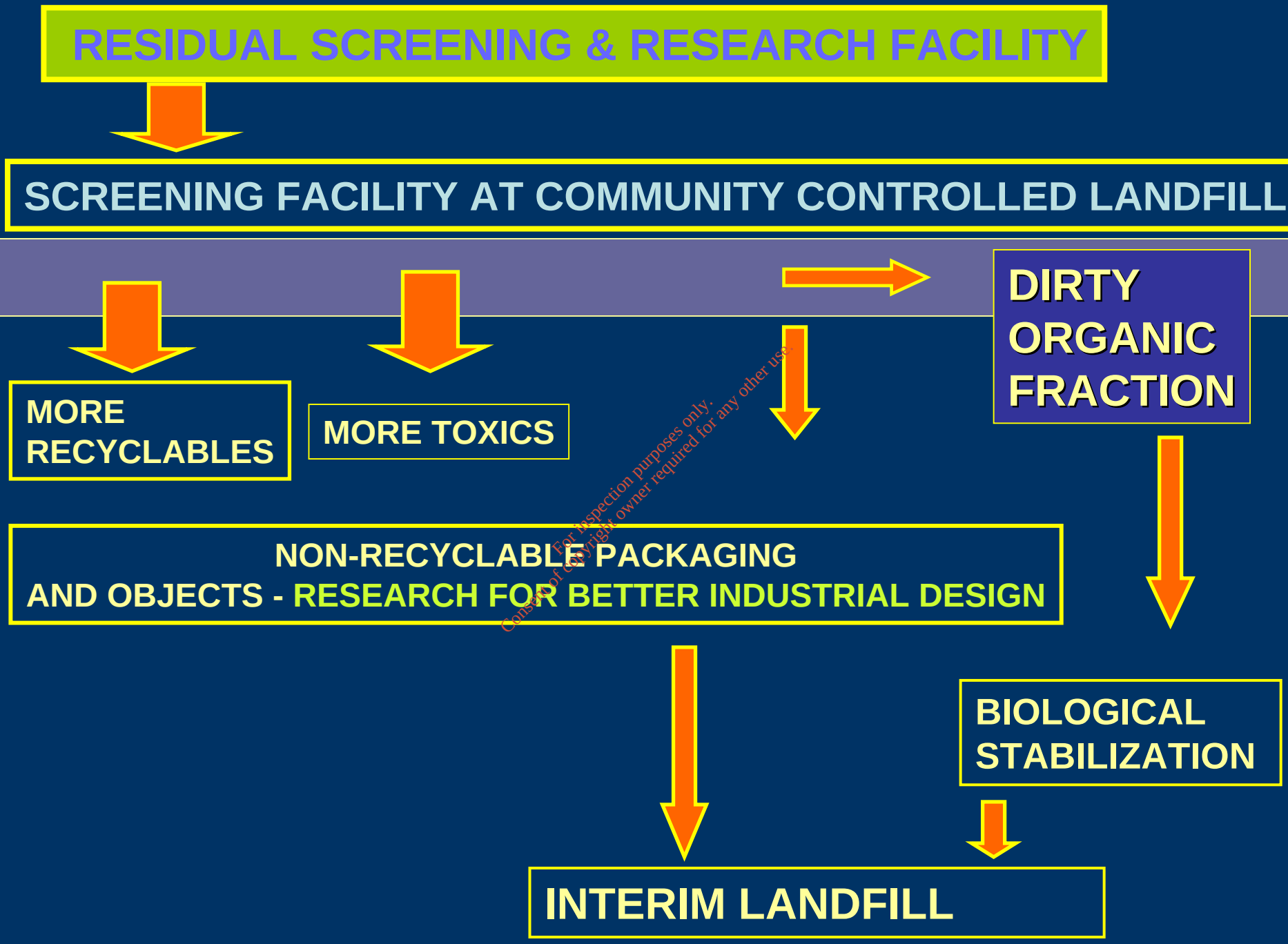
- By 2004 over 60% of the municipalities in NZ had declared a Zero Waste goal by 2020.

# Nova Scotia

- 50% waste diverted from landfills in 5 years (Halifax ~ 60%)
- 1000 jobs created since April 1996
- Another 2000 jobs created in industries using separated materials

# Materials re-used in manufacturing in Nova Scotia:

- All corrugated cardboard
- All newsprint, magazines, & other paper
- Most plastic containers and some plastic film
- All waste paint
- All organic material
- All glass
- All tires
- Steel goes to Quebec



- If we can't re-use it, recycle it or compost
  - industry shouldn't be making it.
- We need better industrial design for the 21st Century.

# A comparison

- With incineration
- You convert three tons of trash to
- one ton of ash
- that nobody wants!

# With a zero waste strategy

- You convert three tons of trash into:
  - One ton of recyclables
  - One ton of compostables,
  - and
  - One ton of education!

- Remember you don’t have to get down to zero to beat incineration because only reduce the amount of waste going to landfill by 70%. 30% is left as toxic ash.
- Canberra has reached 70% diversion in less than 10 years!

# INDUSTRIAL RESPONSIBILITY

1) Better industrial design

2) Extended Producer Responsibility

3) Clean up manufacturing process

# INDUSTRIAL RESPONSIBILITY IN ACTION

## 1) THE BEER INDUSTRY, ONTARIO, CANADA

- Uses refillable glass bottles
- 98% recovered
- Reusable glass bottles 11 cents cheaper per serving than disposable bottles.
- 2000 jobs in collection and cleaning
- No cost to municipality
- Packaging costs internalized

# INDUSTRIAL RESPONSIBILITY IN ACTION

## 2) XEROX CORPORATION EUROPE

- Recovering old copying machines from 16 countries
- Over 95% of materials reused or recycled!
- $76 million saved in 2000 !!

# Pieces of Zero

# A piece of zero

- In 2003, Cole's Bay, Tasmania, became the first town in Australia to ban plastic shopping bags -since then 80 more towns have followed suit.

# A piece of zero

Irish Plastic Bag Tax:

- It is estimated that the introduction of a 15-cent environmental levy on plastic bags has reduced… consumption of these bags by about 92 %…Receipts from the levy (in 2003) …amounted to over 12.7 million Euros. …proceeds are used to support waste management and other environmental initiatives… (A survey) indicated that reusable shopping bags are now being used by 90 % of shoppers.

- Ref: Sustainable Consumption and Production in the European Union, 2004.

# Other initiatives

- The COOP supermarket chain near Florence allows customers to refill their own containers with shampoo, detergent etc.

# GOOD LEADERSHIP

We need political and industrial leaders who are

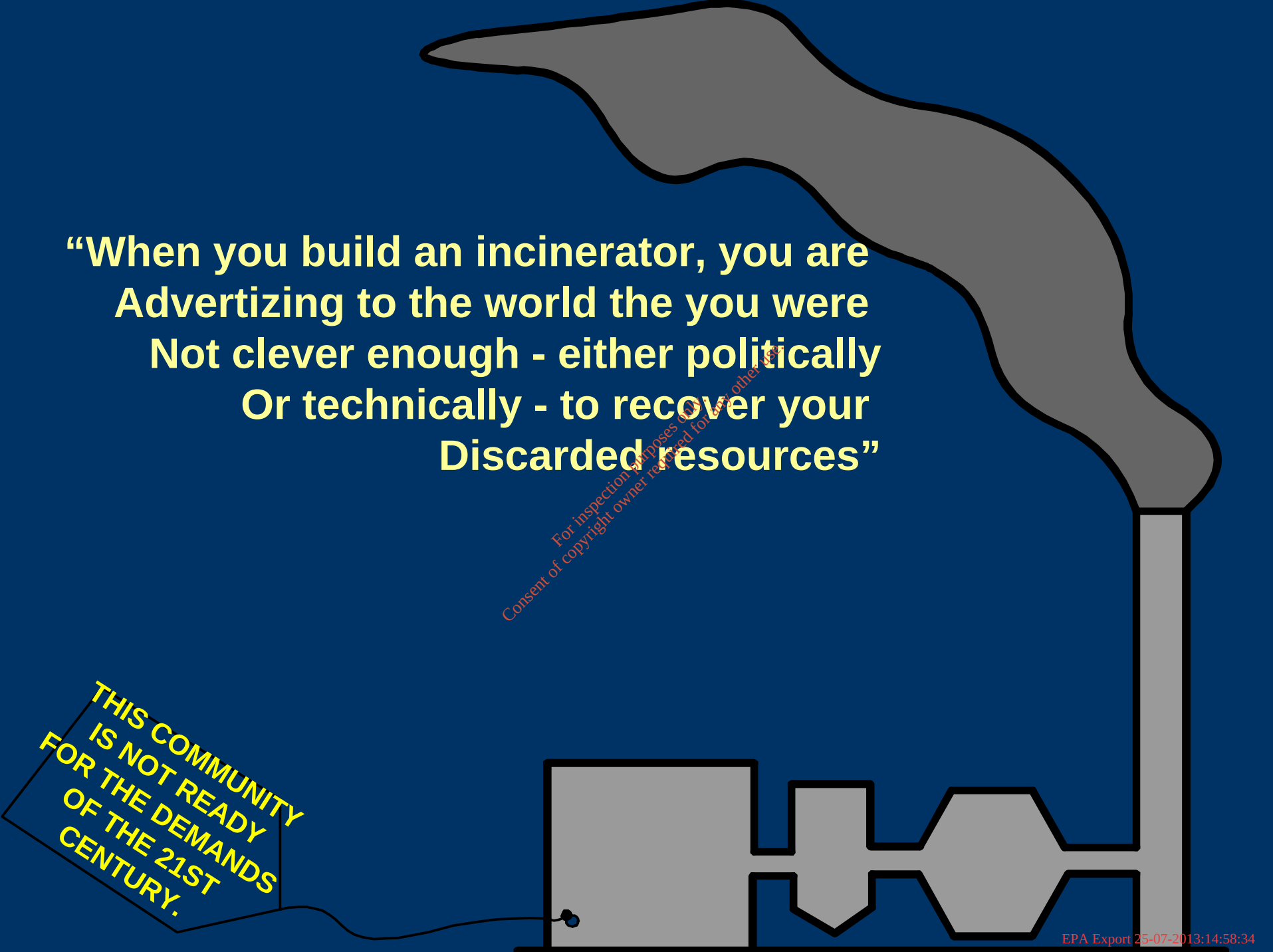
visionary

imaginative

creative

and

WHO ARE NOT BORING



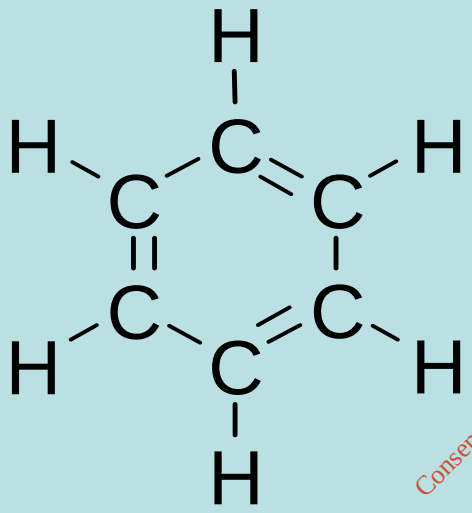
# MUNICIPAL WASTE INCINERATION: A POOR SOLUTION FOR THE TWENTY FIRST CENTURY

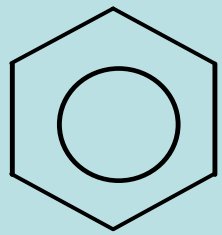
www.no-burn.org

Remember you don’t have to get down to zero to beat incineration

Incinerators only reduce the amount of waste landfilled by 70%

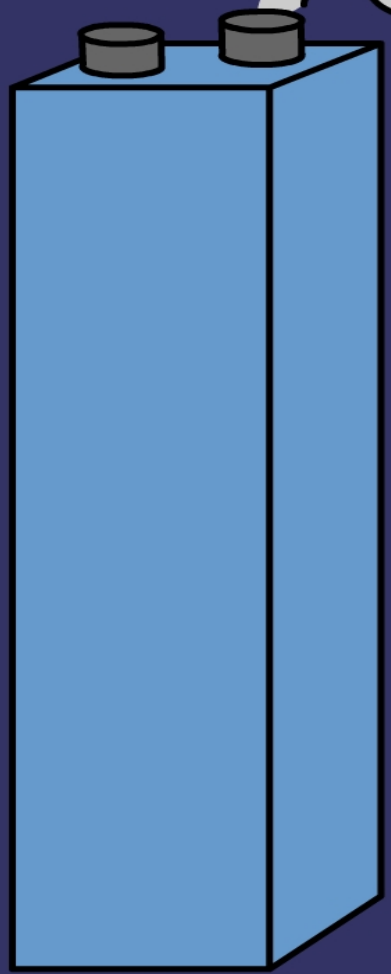
# Benzene Depictions





$C_6H_6$

# AIR EMISSIONS



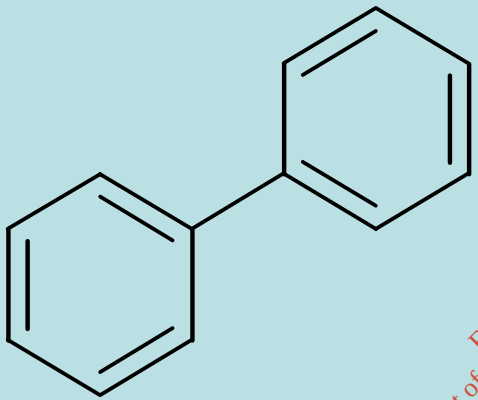
- $CO_2 + H_2O$
- **ACID GASES:**
  HCl, HF, $SO_2$
  $NO_x$
- **TOXIC METALS:**
  Pb, Cd, Hg, As, Cr etc
- **NEW COMPOUNDS:**
  PCB's
  PCDDs (DIOXINS)
  PCDFs (FURANS)
  CHLORINATED BENZENES
  PHENOLS, NAPTHALENES
  ETC

FINE PARTICULATE (SUB MICRON PARTICLES)

# PART 1
# DIOXINS

# Chemical structures

# Biphenyl



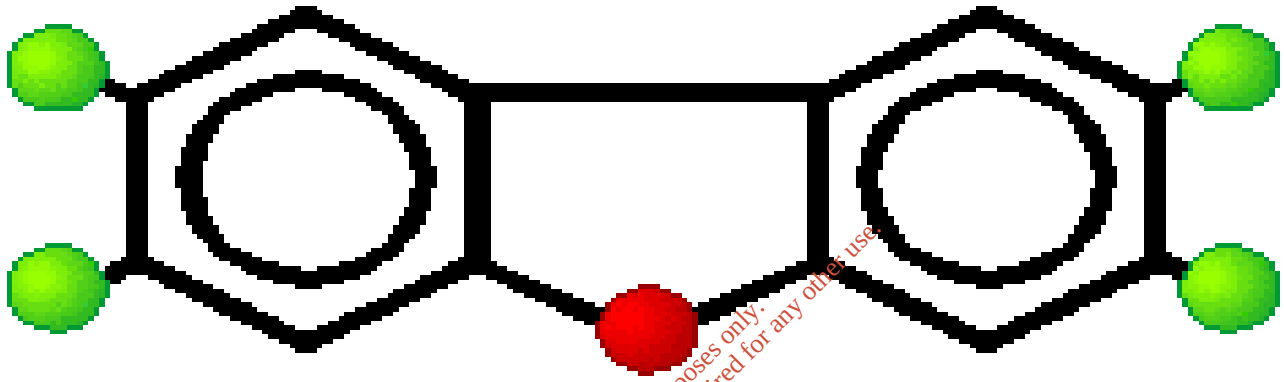
**Biphenyl**

# Polychlorinated biphenyls

- PCBs consist of two benzene rings joined together (biphenyl) with chlorine substituted for hydrogen at 1 to 10 positions. There are 209 PCBs.

# PCDFs or FURANS

- Furans (or PCDFs) have an oxygen atom forming a five membered ring (the furan) between the two benzenes of PCBs. There are 135 furans.



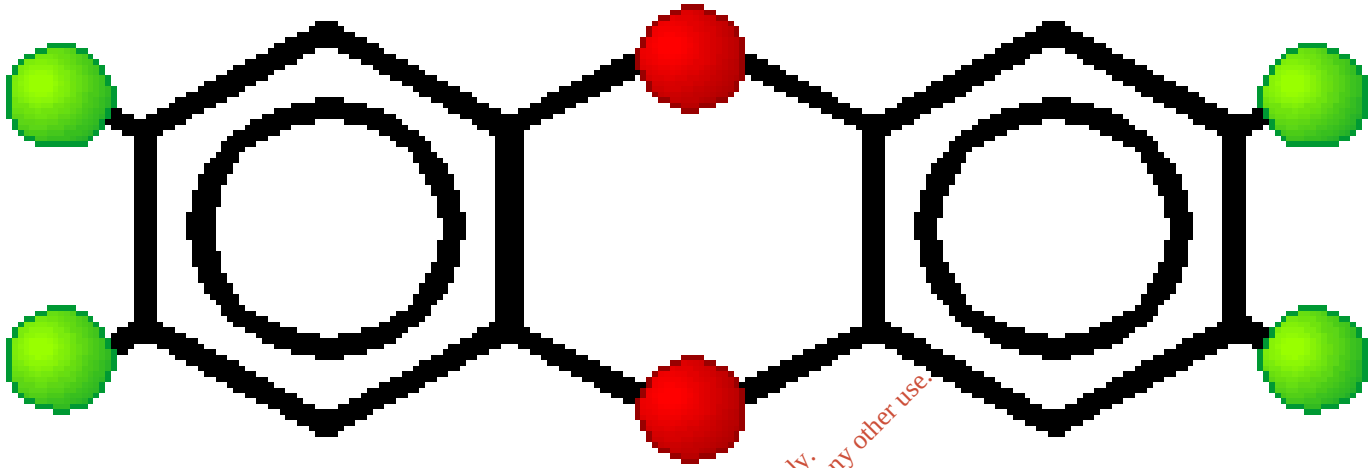


2,3,7,8-TetraCDF

# 2,3,7,8-TETRA CHLORO DIBENZO FURAN

# PCDDs or dioxins

- Dioxins (or PCDDs) have two oxygen atoms linking the two benzene rings, forming the dioxin ring. There are 75 dioxins.



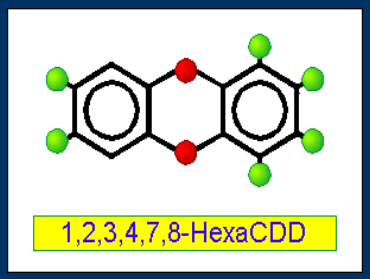


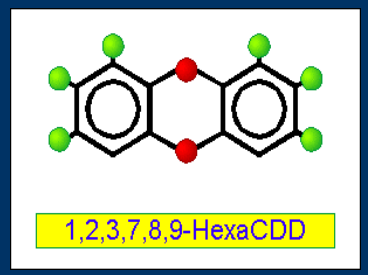
**2,3,7,8-TetraCDD**

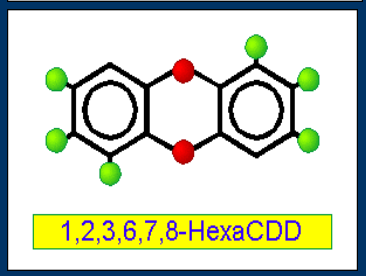
# 2,3,7,8-TETRA CHLORO DIBENZO DIOXIN

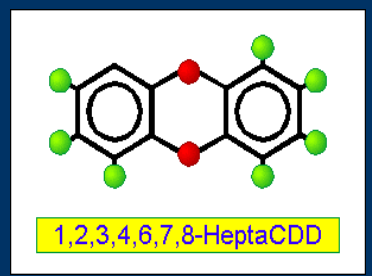
# There are 17 extremely toxic dioxins and furans. They have chlorine at the 2,3,7 and 8 positions:

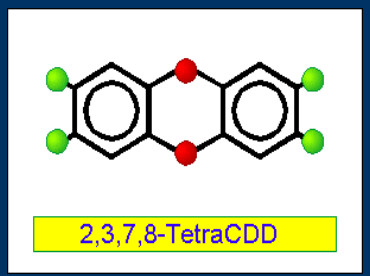
**7 Dioxins and 10 Furans**

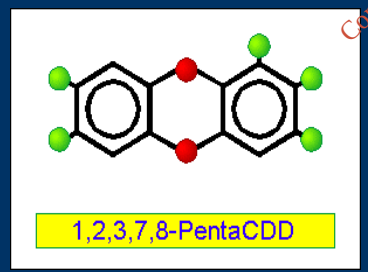


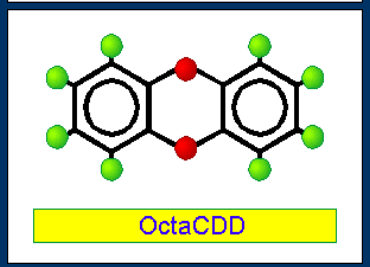



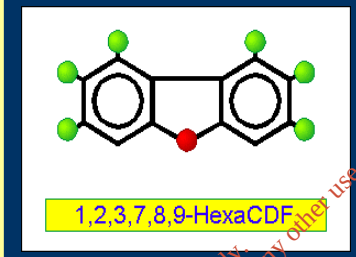



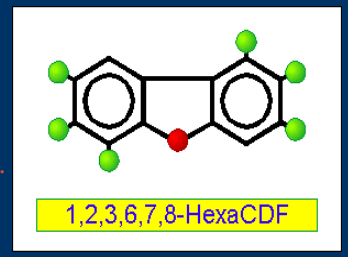



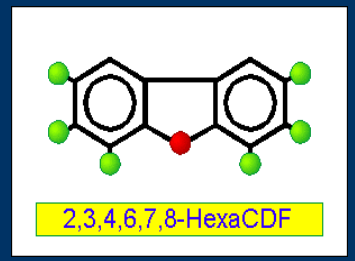



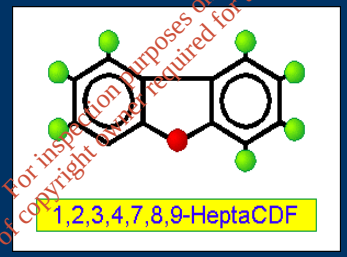



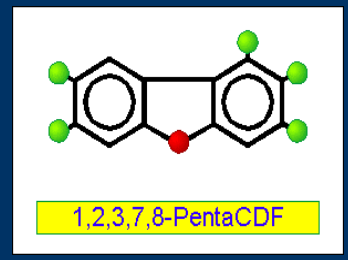



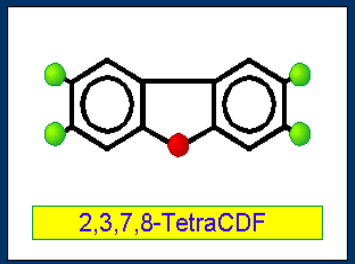



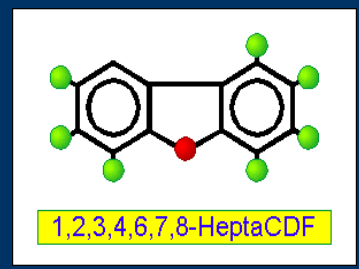



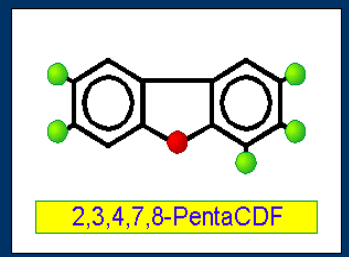



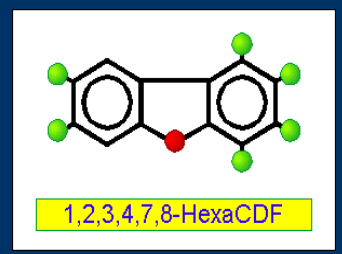



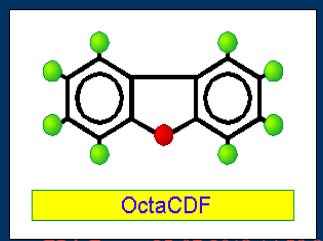



# The biology

- Dioxins and dioxin-like compounds are fat soluble and easily cross membranes and enter cell
- Once in the cell they fit into a protein called the Ah receptor
- Another protein joins this combination
- This complex enters the nucleus and attaches to the DNA
- It doesn't cause mutations, but it does switch on genes
- Switching on genes results in the production of new proteins in the cell.
- In other words it functions like a fat soluble hormone.

# Two remarkable things about the Ah receptor

- 1) After 30 years of research scientists do not know what it is in the cell for. Have not identified its normal function.

- 2) The Ah receptor appears in evolution at the same time as the backbone appears in fish. Every species above invertebrates has the Ah receptor.

# The presence of dioxin can interfere with the levels of a number of key substances in a living cell

- **ENZYMES:**
- Cytochrome P4501A1, Cytochrome P4501A2, DT-Diaphorase, UDP-Glucuronyl Transferase,Glutathione-S-Transferase, Aldehyde Dehydrogenase, Ornithine Decarboxylase, Tyrosine Kinase,Thymidylate Transferase, Phosphoenolpyruvate Carboxykinase, Plasminogen Activator Inhibitor 2…
- **HORMONES & HORMONE RECEPTORS:**
- Androgens, Estrogens, Estrogen receptor, Glucocorticoid, Glucocorticoid receptor, Insulin, Insulin Growth Factor, Thyroid Hormones, Gastrin…
- **GROWTH & DIFFERENTIATION FACTORS:**
- Ras, Myc and Erb Oncogenes, EGF Receptor, TGF-alpha, TGF-beta 1, Beta 1, TNF-alpha, IL1-beta…

# The Politics

# Keynote speaker at Dioxin Symposium in Toronto, 1989, says dioxin not a problem for humans

- “Dioxins have never been shown to kill one human, nor induce birth defects or cancer in humans, a US researcher says.
- “The widespread dread of the compounds could be a false alarm of historic proportions, said Curtis Travis, director of the office of risk analysis at the Oak Ridge National laboratory…
- “PCBs and furans…are shown by human health records to be equally harmless, despite widespread worry about them…
- “Hundreds of millions of dollars in public money are being wasted on dioxin research…”
  - Toronto Star, Sept 18, 1989.

# A status briefing for the Deputy Administrator of the US EPA, 2-14-92

- Dioxin does cause cancer in humans.
- Cancer may not be the most sensitive toxic response…
- Recent evidence strengthens the conclusion that the sensitivity of humans is similar to that of experimental animals…
- Current exposure levels to dioxin…appear to place people at or near a body burden where sensitive responses may occur, especially for…nursing infants, recreational and subsistence anglers…

# 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)

# Effect of Dioxins on Neonatal Thyroid Function after Low-exposure and High-exposure at various ages

| | | Low-exposure (mean) | **High-exposure (mean)** | P* |
|---|---|---|---|---|
| At birth | T4 | 122.5 | 134.3 | 0.071 |
| | T4/TBG | 0.240 | 0.232 | 0.45 |
| | TSH | 10.4 | 11.9 | 0.58 |
| 1week | T4 | 154.5 | 178.7 | 0.006 |
| | T4/TBG | 0.291 | 0.332 | 0.006 |
| | TSH | 2.93 | 2.56 | 0.51 |
| 11weeks | T4 | 111.1 | 122.2 | 0.033 |
| | T4/TBG | 0.220 | 0.247 | 0.040 |
| | TSH | 1.81 | 2.50 | 0.044 |

# Politics versus Science

- In 1993, it was discovered that one trash incinerator in Columbus, Ohio, was putting out 984 grams of Dioxin TEQ per year
- This was more than the whole of Germany, twice as much as the Netherlands and three times as much as Sweden - for all sources.
- The Ohio EPA did a health risk assessment and declared that “there are no substantial health risks posed”

# Politics versus Science (cont.)

- The Columbus Health Department put out a Dioxin fact Sheet for citizens
- They explained that one part per trillion was equal to taking a 1 second vacation after working 31,700 years.
- They converted the maximum emission rate from the incinerator to 1341.9 ppt**
- ** One part per trillion (ppt) is equivalent to taking a 1 second vacation after working 31,700 years. The maximum emission is equal to 1,342 seconds or a 22.4 minute vacation taken in 31,700 years (if a person worked all year)
- or if a person worked 40 hours per week it would take 133,567 years to earn the 22.4 minute vacation.

# Politics versus Science (cont.)

- Prior to a second dioxin test of the incinerator (March 94), the following excerpts appeared in the plant operator's log:

- "We lost our north end trash last weekend…Remember the tests are very important and is our future" 2-14-94

- "We must have a 'good source' of trash for the test" 2-15-94.

- "It appears the second shift crane operator used the good, dry material on Sunday…" 2-18-94

- "This test is our future and I would think everyone would be extremely interested in helping out if possible." 2-21-94

- "Continue to hold the M.R. trash…we will fill the area up with M.R.trash in preparation for our testing." 2-22-94

# Politics versus Science (cont.)

- The Columbus Health Department brought in a consultant (Dr. Greg Rigo) to put the dioxin problem into perspective.At a press conference (which got front page coverage) he presented an inventory of dioxin emissions in the US:

- 33,000 grams Dioxin enters the US environment annually
- 62% = from unknown sources (possibly volcanoes and rotting wood)
- 20.5% = from motor vehicles
- 4.0% = from the production of herbicides & pesticides
- 2.6% = from ALL US MUNICIPAL WASTE INCINERATORS
- combined (approx. 130 at that time)
- 2.0% = from chlorine bleaching of paper

- PROBLEM - 2.6% of 33,000 grams is 850 grams per year, which was less than the output from the incinerator (984 grams per year) he was supposedly investigating!

# Politics versus Science (cont.)

- Dr. Greg Rigo was hired by the Vinyl Institute to investigate the relationship between the chlorine content in trash and dioxin emissions. In a memo they described Rigo as “user friendly”.

- Rigo found that there was no relationship between chlorine content and dioxin emissions!

# Dioxin Tied to Endometriosis



*Science*, 262, 1373,
26 November 1993

# Our Stolen Future

## How Man-made Chemicals are Threatening our Fertility, Intelligence and Survival



Theo Colborn
John Peterson Myers
Dianne Dumanoski

1994

# 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

# Regulatory levels

- Outside US, Allowable Daily Intakes (ADI) range from 1 to 10 pg/kg bodyweight/day. WHO = 1-4 pg/kg/day.
- In US there is no safe level for a suspected carcinogen; the US EPA uses health risk assessment instead.
- In 1985 US EPA estimated that 0.006 pg/kg/day would yield a lifetime cancer risk of 1 in a million.
- Industry has fought this standard for 20 years!
- The latest draft from US EPA has lowered the level to 0.001 pg/kg/day
- Canada's ADI is still at 10 pg/kg/day!!!!!!!

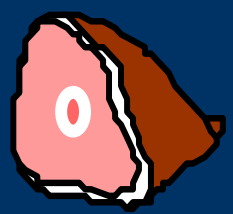
# In most industrialized countries

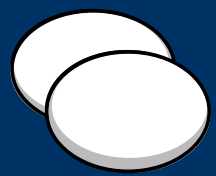
- Citizens are getting between 1 and 2 picograms of dioxin TEQ/ kg bodyweight/ per day

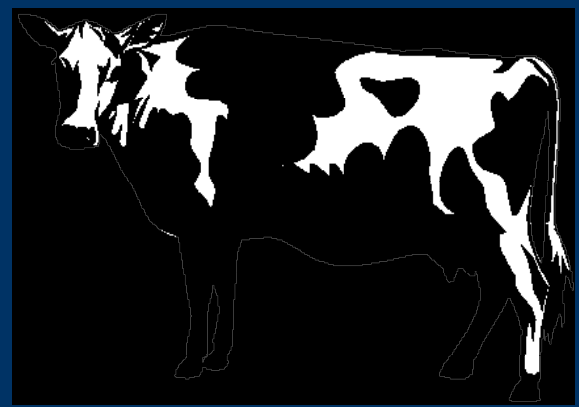
# DIOXINS IN OUR FOOD

- Dioxins are fat soluble and persistent and accumulate in the food chain, specially animal fats. Well over 90% of our dioxin intake comes from dairy products, meat,and fish.







# Dioxins: chemically stable but extremely biologically active

- Dioxins switch on genes
- Produce different proteins, including enzymes and growth factors
- Disrupt at least six different hormonal systems: male and female sex hormones; thyroid hormones; insulin; gastrin and gluocorticoid.

# What are PCBs,dioxins and furans?

.

# Dioxins - major concerns

**One liter of cows' milk gives the same dose of dioxin as breathing air next to the cows for EIGHT MONTHS (Connett and Webster, 1987). In one day a freely grazing cow puts the equivalent of 14 years of human breathing into its body (McLachlan, 1995)!**

**The liver cannot convert dioxins to water soluble products thus they steadily accumulate in human body fat. The man cannot get rid of them BUT A woman can…**
**…by having a baby!**

**The highest dose of dioxin goes to the fetus during pregnancy and then to the new born infant via breastfeeding.**

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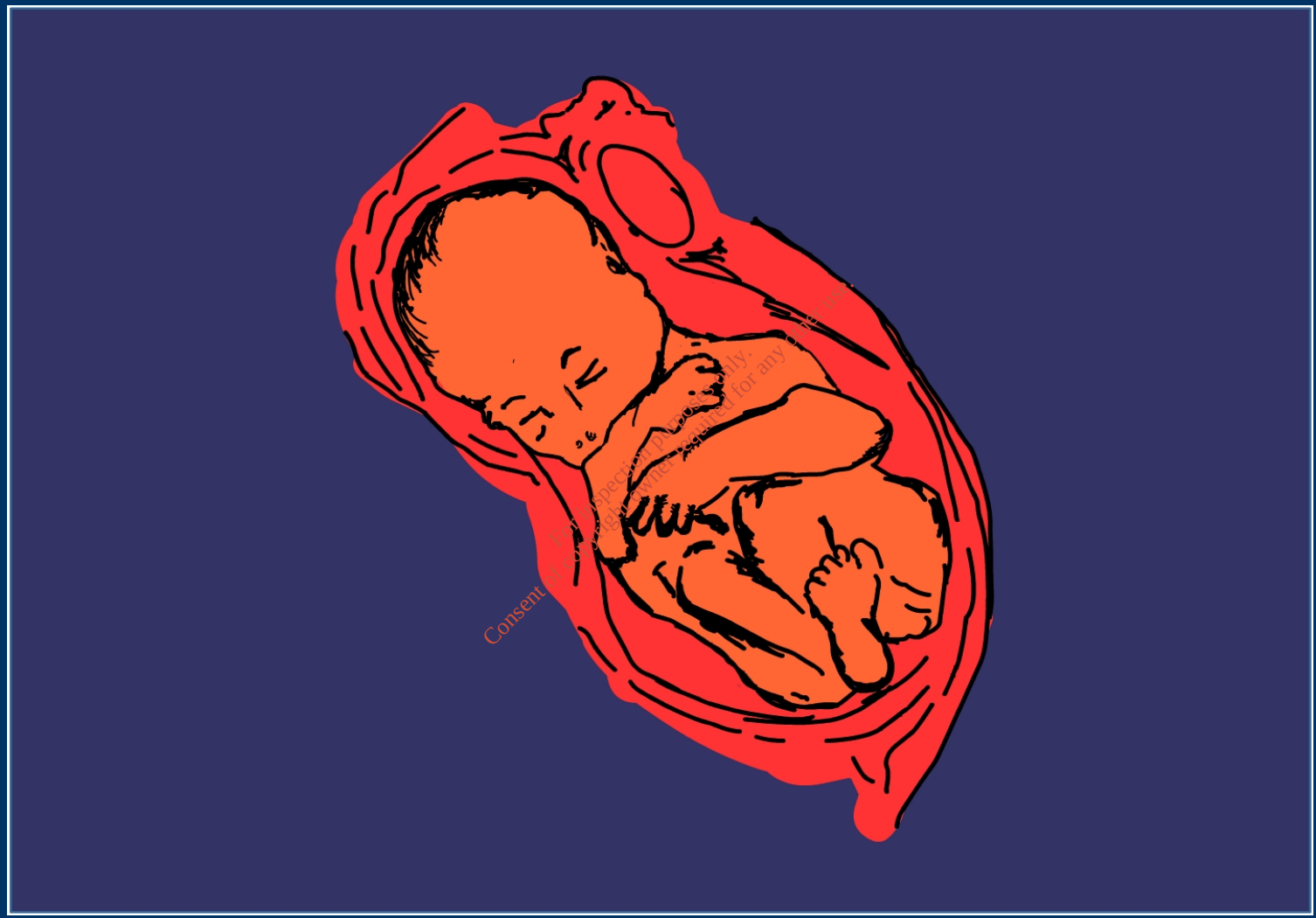
- **The man cannot get rid of them BUT A woman can…**
- **By having a baby!**
- **Thus, the highest dose of dioxin goes to the fetus during pregnancy and then to the new born infant via breastfeeding.**

# Citizen involvement

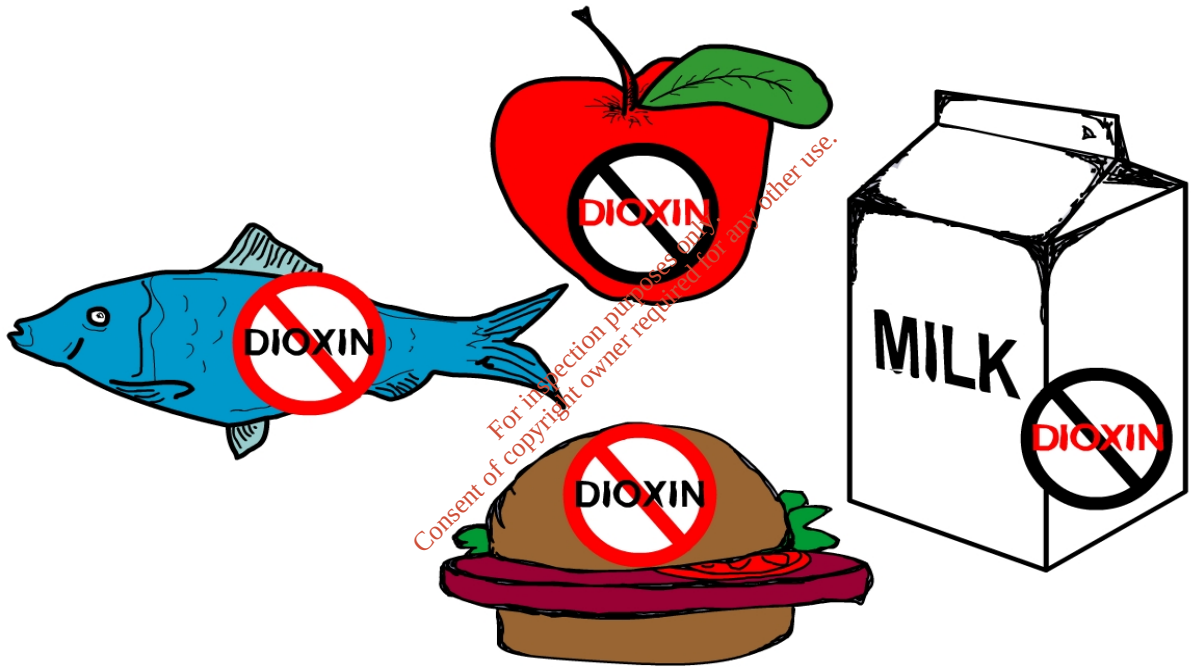
- Greenpeace involvement from the 1980s.
- First Citizens' Conference on Dioxin, Chapel Hill, NC, 1991 (transcript and 10-part video series).
- Second Citizens' Conference on Dioxin, St. Louis, Missouri, 1992.
- Third Citizens' Conference on Dioxin, Baton Rouge, Louisiana, 1994.
- Formation of "Health Care Without Harm", 1994.
- "Dying from Dioxin", Lois Gibbs et al 1996.
- Formation of the Global Alliance for Incineration Alternatives (GAIA) in South Africa, 2000 (no-burn.org and grrn.org)

# WE WANT DIOXIN



# OUT OF OUR BABIES!

# WE WANT DIOXIN






# OUT OF OUR FOOD!

# Dioxins in cow's milk - history

- 1989 Dioxins in cow's milk 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.

# Dioxin in cow's milk

## pg I-TEQ/g fat (ppt)

- Denmark 2.6
- Finland 0.83 - 1.17
- France 1.81
- Germany 0.71 - 0.87
- Ireland 0.08 - 0.51 Average in Ireland = 0.2
- Netherlands 0.38 - 1.6
- Spain 1.2 - 2.0
- Sweden 0.93 - 2.0
- UK 1.01

Measurements reported in 1999, (IOM, 2003).

# The politics again!

- The US EPA published its draft reassessment of dioxin in 1994 (which was virtually complete).
- We are still waiting for the final version ten years later!

# Institute of Medicine, 2003

## Dioxins and Dioxin-like Compounds in the Food Supply



## Strategies to Decrease Exposure

July 1, 2003

# Institute of Medicine, 2003

- Fetuses and breastfeeding infants may be at particular risk from exposure to dioxin like compounds (DLCs) due to their potential to cause adverse neurodevelopmental, neurobehavioral, and immune system effects in developing systems…

# Institute of Medicine, 2003

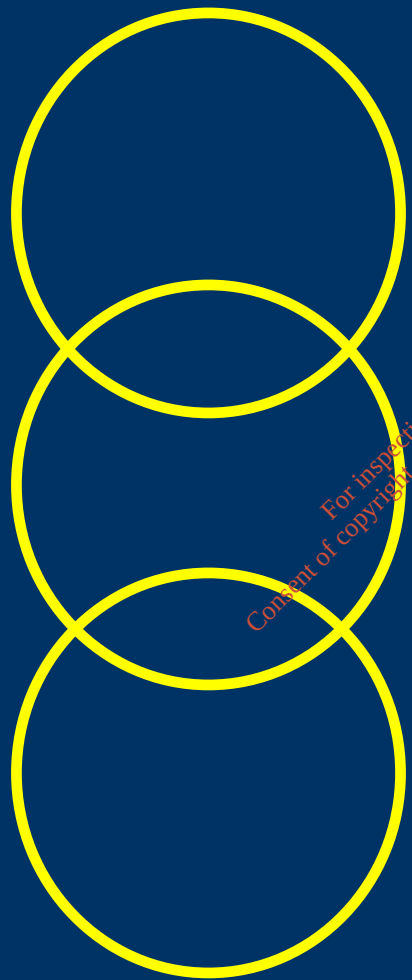
- …The committee recommends that the government place a high public health priority on reducing DLC intakes by girls and young women in the years well before pregnancy is likely to occur.

- Substituting low-fat or skim milk, for whole milk… coupled with other substitution of foods lower in animal fat by girls and young women in the crucial years before pregnancy…

# When it comes to dioxin and incinerators

- Governments always say to the citizens
- You don’t have to worry
- Because we have tough new air emission standards
- But…

# THE CHAIN OF PUBLIC HEALTH PROTECTION HAS THREE LINKS.



**STRONG REGULATIONS**

**ADEQUATE MONITORING**

**TOUGH ENFORCEMENT**



**IF ANY LINK IS WEAK THE PUBLIC IS NOT PROTECTED**

# De Fre and Wevers (1998)

- De Fre and Wevers compared 6 hour testing for dioxins with 2 week testing (on same incinerator)
- 
- They found 30-50 times higher concentration (mass divided by total volume of flue gas) in the 2 week test compared to 6 hour test.

- Reason: 2 week test picks up upset conditions as well as start up and shut down.

# Regulatory agencies have enormous power when it comes to permitting incinerators

But little political will when it comes to protecting the public once the facility is built.

Health Risk Assessment

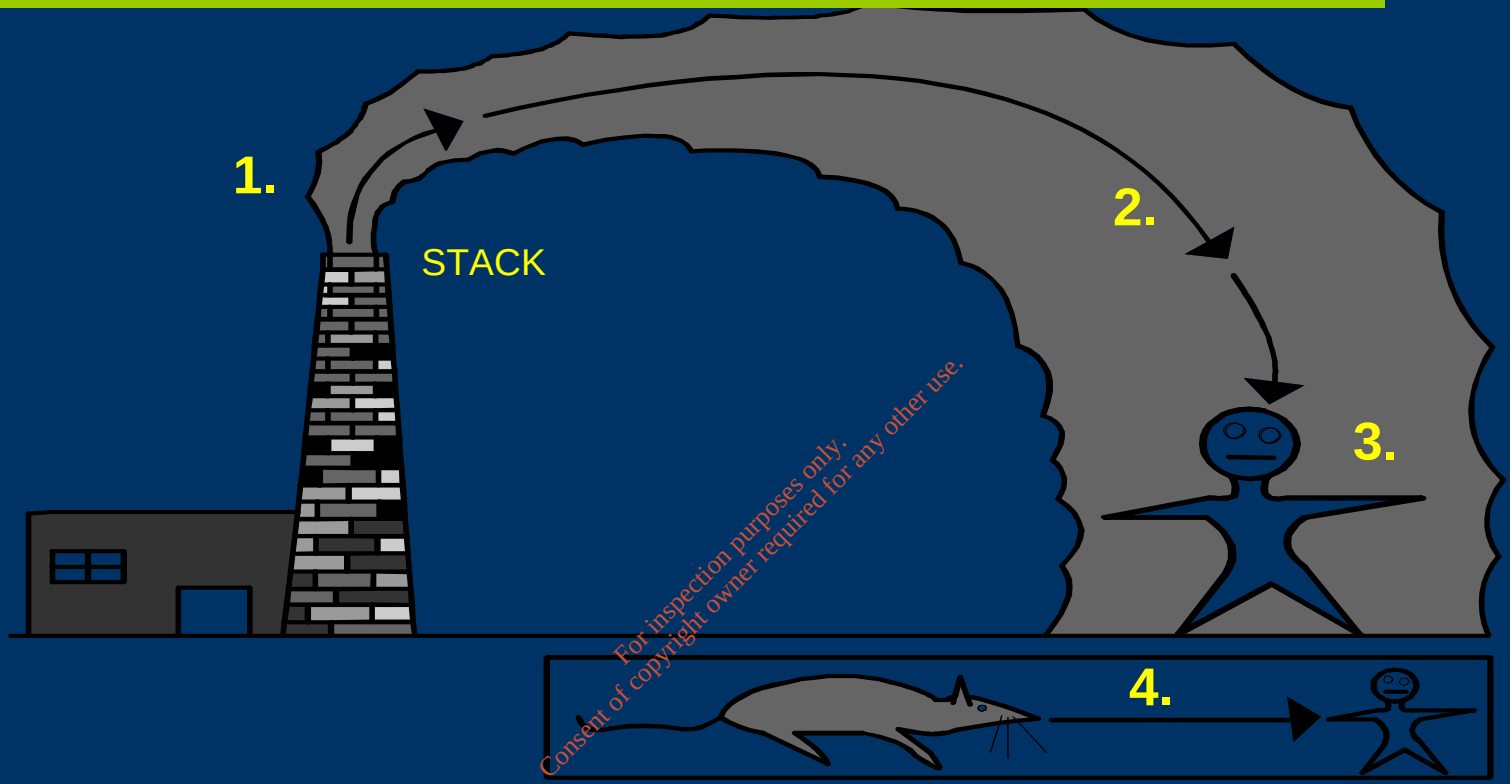
replaces

Public Health Protection

The people lose their health, their property values, even their homes, while

Consultants make a fortune!

# 4 COMPONENTS OF RISK ANALYSES.






1. ESTIMATION OF EMISSIONS.
2. CALCULATION OF DISPERTION USING COMPUTER MODEL. GIVES GROUND LEVEL CONCENTRATIONS.
3. CALCULATION OF HUMAN UPTAKE.
4. EXTRAPOLATION OF HUMAN RISK FROM ANIMAL STUDIES.