**Comments on**

**Proposed Acceptability for Continuing Registration (PACR Series) 2005**
**Re-evaluation of the Lawn and Turf Uses**
**of (2,4-Dichlorophenoxy)acetic Acid [2,4-D]**
**(PACR2005-01) February 21, 2005 - 555Kb**

**http://www.pmra-arla.gc.ca/english/pdf/pacr/pacr2005-01-e.pdf**

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## Introduction and general comments

The first point I would like to make is that it is completely inappropriate that the PMRA continually presume that a pesticide is harmless. The most glaring public example of this was seen with the proclamation of a conclusion rather than announcement of public consultation. Indeed such an announcement more closely resembles the PR from industry than the actions of an objective regulator.

Before I mention anything else about this document, it is good to mention some major changes taking place in the US EPA which have to be relevant in Canadian assessment. Some general failures of the Canadian licensing and re-evaluation system will also be addressed.

The first change is the EPA's review of their cancer risk assessment:

"*In the first such update in nearly 20 years, the EPA said children 2 years old and younger might be 10 times more vulnerable than adults to certain chemicals. Children between the ages of 2 and 16 might be three times more vulnerable to certain chemicals*."[1]

"*The EPA also said it is seeking new ways to gather scientific data on possible carcinogens. It said "the consideration of new, peer-reviewed scientific understanding and data in an assessment can always be consistent with the purposes of these cancer guidelines*." [1]

This review should change the balance of the evaluation of all cancer data in this document. Maybe it will even move cancer as one of the end-point considered?

The second is that the EPA proposes to update and revise its data requirements for the registration of conventional pesticide products. The inadequacy of the mandated tests, and how outdated they are have been brought forward for many years in Canada as well as in the US. The EPA states:

"*Since the data requirements …were first codified in 1984, information needed to support the registration of a pesticide chemical has evolved as the general scientific understanding of the potential hazards posed by pesticides has grown*."[2] Over the years, updated data requirements have been applied on a case-by-case basis to support

[1] Heliprin, John**;** March 30, 2005; *EPA Says Children May Be Vulnerable than Adults to Carcinogens*; Associated Press

[2] EPA ; March 11, 2005; *Pesticides; Data Requirement for Conventional Chemicals;* From the Federal Register Online via GPO Access [wais.access.gpo.gov] [DOCID:fr11mr05-29];[[Page 12277]]

individual applications, or imposed via Data Call-In (DCI) on all registrants of similar products. "*Although the data requirements imposed have progressed as scientific understanding and concerns have evolved, the codified data requirements have not been updated to keep pace. This proposal involves changes to the codified data requirements that pertain to product chemistry, toxicology, residue chemistry, applicator exposure, post-application exposure, nontarget terrestrial and aquatic organisms, nontarget plant protection, and environmental fate. Coupled with updating data requirements, EPA proposes to add a few new studies, reformat the requirements, and revise its general procedures and policies associated with data submission. By codifying existing data requirements which are currently applied on a case-by- case basis, the pesticide industry, along with other partners in the regulated community, attain a better understanding and are better prepared for the pesticide registration process. This proposed rule does not apply to the data requirements for the registration of antimicrobial pesticide products; inert ingredients for pesticide products; spray drift, product performance (efficacy); or biochemical, and microbial pesticides.*"[2]

In the 2,4-D assessment, there is mention of thyroid (one of the endocrine gland most commonly affected by pesticides, solvent and other chemicals) and immune problems among others, but they seem brushed aside when it comes time to choose relevant end-points to evaluate health effects.

There are still no or very few data to evaluate the effect of mixtures, even for the pesticides most commonly used together. (e.g. 2,4-D, mecoprop and dicamba) .Even the US EPA admits to floundering when evaluating mixtures.[3] Endocrine-disruption is no longer in question in science and governments,[4] although research protocols are still in development.[4] It is qualified as a daunting task to study complex interactions which often are species-specific, life-stage specific, and tissue-specific.[4] Unfortunately it seems that research will concentrate on only a few chemical groups first.[4] Low dose and mixture studies are also planned.[4] These bring up fundamental questions for the current regulatory approach also followed by the PMRA. *"The issue (of low dose) is an important one because the presence of such effects would challenge the validity of our current approaches to hazard identification and risk assessment for endocrine disruptors…EPA's entire chemical*

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[3] NATIONAL CENTER FOR ENVIRONMENTAL RESEARCH (U.S. Environmental Protection Agency, National Institute for Occupational Safety and Health, National Institute of Environmental Health Sciences); COMPLEX CHEMICAL MIXTURES; FY 2000 Science to Achieve Results (STAR) Program; Opening Date: April 10, 2000, Closing Date: July 10, 2000;
Location of quote: Under "Research Goals and Scope," then find "Exposure Assessment."

The EPA states clearly there are no methods for assessing the toxicity of mixtures of chemicals. "A major, long-term challenge for regulatory agencies is to develop defensible means
of combining exposure assessments in a manner that provides meaningful ways of determining potential health risks from total exposures to many chemicals. Lacking are methods to
characterize potential toxicological risk."

[4] SUBCOMMITTEE ON ENDOCRINE DISRUPTING CHEMICALS; March 4, 2005; Endocrine Disrupting Chemicals; (EDC) Research Program Review; Final Report of the Subcommittee on Endocrine Disrupting Chemicals; http://www.epa.gov/OSP/bosc/pdf/edc0503rpt.pdf

*regulation framework is based on the presumption that as dose increases, so does the prevalence and severity of adverse effects.”*[4]

Endocrine-disrupting, immune, low dose, and developmental neuro-toxicology studies are thus not yet part of mandated studies by regulatory agencies, and decisions are made on the old “presumed”[4] criteria of the dose makes the poison and a threshold below which there are no effects. Recent research also brought to the forefront the varying toxicity of isomers, mentioning that regulatory agencies are currently not paying enough attention to this problem. Add to that a series of recent articles on the synergistic combinations of two pesticides, interactions of pesticides with predation, disease and other stressors in frogs, as well as Rick Relyea[5] [6] illustrating the variation in response between various related species of frogs, synergy with some stresses (rendering some pesticides up to 46 times more lethal), and one can only realize how little we actually know about how a pesticide affects the real health and environment. (real is used in opposition to controlled experiments where everything is controlled and the same, except for the studied parameter). A recent study even identified a synergistic effect between a commonly prescribed drug and a subsequent exposure to chlorpyrifos.[7] The study authors used the following *“certain early drug or chemical exposures can predispose people to particular ailments.”*

Finally there is the issue of the chemically sensitive, to which I belong for the last 18 years. Surveys around the world identify around 12 % of citizens as chemically sensitive. “Approximately 4 million Canadians are affected by chemical sensitivity, half a million of whom are severe cases, with 5000 being disabled by this condition.”[8] All these people react negatively to chemicals; 80 % (3 million Canadians)[9] of them know what triggered their illness, and 60 % (1,800,000 Canadians) ascribe that to pesticide exposure. [9] These numbers are significant enough to indicate either that these people were not adequately

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[5] Relyea, Rick A.; 2003; Predator Cues and Pesticides: a Ddouble Dose of Danger for Amphibians; Ecological Applications 13(6); pp 1515-1531

[6] Relyea, Rick A. and Nathan Mills’ 2001; Predator-induced stress makes the pesticide carbaryl more deadly to grey treefrog tadpoles; PNAS; vol 98, no 5, pp2491-2496 . See references in Relyea’s papers and his website for more examples: http://www.pitt.edu/~relyea

[7] Duke University Press Release, March 30, 2004; Labor Drug Sensitizes Brain to Pesticide Injury;
Slotkin, T. et al; March 2004; *Toxicology and Applied Pharmacology*.
Chlorpyrifos-terbutalin
Both chemicals independently caused brain injuries not seen in the control rats, including the loss of brain cells and the nerve cell projections critical to communication among neurons. The effects persisted into adulthood. In addition to aggravation of the chemicals’ damaging effects on the brain, rats exposed to combined chemical treatment showed reduced nerve cell activity, and suffered significant loss of brain cells and nerve cell projections in portions of the brain central to learning and memory

[8] Ferrie, Helke; Oct. 2003; *Multiple Chemical Sensitivity* A Report prepared for the Ministry of Human Esources of the Government of Canada;

[9] Armstrong, Jennifer, MD; Oct 18, 2004; presentation at the *Body Burden* conference held at McGill University, Montreal, QC

protected by the regulatory system, that testing of pesticide effects on a few healthy animals should be questioned as to its relevance to people in varying states of health, and that the safety factors used are likely inadequate, or that most Canadians do not use pesticides according to label (challenging another one of your underlying assumptions). Whitewash statements don't have much sway on people who live this hell every day and have a long clinical history of linking their severe symptoms to pesticide exposures. And no, we are not basket cases. The definition of mental illness includes the absence of other explanations and a link between MCS and mental illness has been disproved over and over again by all serious researchers.

Let's remember that nothing in the world fits a controlled model, and nobody is 'average' for everything.

I will remind you that the fact that there is no consensus on how to do a study (one of industry's main arguments to discount endocrine effects[10]) does not mean that effects do not occur, or that they are considered unimportant by governments, contrarily to the statements from PMRA in past correspondence. It merely indicates that it is a new area of science for which standards have yet to be set, and illustrates better than anything else the inability of anyone to say any pesticide is "safe", whether used according to label or not.

## Comments on 2,4-D

Testing a technical form of 2,4-D and trying to apply the result to the toxicity of mixtures and formulas is a bit like trying to derive the properties of molecular 2,4-D by only testing some aspects of the toxicity of one element like carbon (C), hydrogen (H), Oxygen (O) or chlorine (Cl). If chlorine was chosen, we could say that chlorine gas is deadly, but table salt is much less toxic. This illustrates that it is not just the chlorine itself that may be the problem, but what it is mixed with. The same applies to purposeful mixtures and formulations of pesticides and chemicals, as it unfortunately applies to all the accidental chemical mixtures now ubiquitously identified in the human body and the environment. While this is happening and is finally being documented, the PMRA persists in only requesting a one to one evaluation of pesticides, interpreting the results as the whole 'science-based' truth, and denying Canadians the right to know what other substances such as formulants they are exposed to on a regular basis.

The U.S. EPA estimates that, on average, 2/3 of every pesticide product is made of formulants.[11] The PMRA does not have a clue.[12] Domestic pesticide

[10] http://www.lawnfacts.ca/article-0013.shtml
Landscape Ontario's Lawn Facts

[11] Northwest Coalition for Alternatives to Pesticides; 1998; "Toxic Secrets Inert Ingredients in Pesticides 1987-1997"; report by Californians for Pesticide Reform (exec summary and p.1)

formulations tend to contain much less active pesticide ingredient, and much more formulants than commercial ones, up to 99 % formulants.[13] Thirty-four 2,4-D formulants are known from the US, of which thirty are still licensed in Canada under the new March 2005 Formulants list. Of these, 7 are in list 2 and 3 are in list 4B. In the US, 70 % of these formulants are already recognized as chemically, biologically, or toxicologically active.[11] These chemicals are able to cause cancer, reproductive and nervous system harm, and other health and environmental problems. 43 % (13/30) are or have been used as pesticide active ingredients in the US.[11] Yet, 2,4-D formulants are summarily dealt with through a reference to the formulants regulations. This is absolutely unacceptable considering what we already know of the toxicity of many of these formulants, and what has never been looked at for many of the others. PACR2005-01 is still done with very little consideration given to formulations and mixtures. Please refer to separate documents *Fact sheet on 2,4-D Formulants,* and *2,4-D Formulants* for detailed analysis of potential 2,4-D formulants.

Furthermore, in the Canadian climate of perennial and almost total absence of essential data (individual pesticide sales and uses, water, air and other contamination, adverse effects, etc) it would indeed be a miracle if the PMRA gets even close to an adequate evaluation of exposure and health effects of any pesticide. Following the pattern amply documented for the US pesticide licensing and re-evaluations,[14] the value section of the risk-benefit assessment of 2,4-D is equally poorly documented (3 references and no consideration of alternatives in the assessment) and seemingly done without any science at all.

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[12] PMRA correspondence Date: Wed, 02 Mar 2005 16:32:36 -0500

[13] Vacco, Dennis C, Attorney General; revised 1994; The Secret Hazards of Pesticides: Inert Ingredients; New York State Dept of Law
Canadian label comparisons: the first 4 mixed amines on the list all contain less than .4% active ingredients (AI) total ( including dicamba and mecoprop) versus the first 3 commercial products listed which contain between 19.4 to 58.2 % AI. Most Weed-and-feed products contain less than 2 % active ingredients.

[14] Benbrook, Charles M, Ph.D.; 1996; *Pesticides at the Crossroads*; cconsumers Union; Yonkers, NY
"Despite the quantity and quality of intellectual effort applied to this task, risk assessments for pesticides remain hotly disputed, especially when debated within contested regulatory proceedings. Compared to risk assessment, benefits assessment methodology for pesticides has been developed through less intense, less openly participatory process, and has received less attention in the regulatory process. But benefits assessment is a structurally flawed and weal leg on the regulatory stool." P. 93
"A benefits assessment is generally carried out only when a pesticide is in regulatory trouble, and can serve as a registrant's last line of defense." P.93
"Non-chemical preventative practices or biointensive IPM options are generally not seriously considered. EPA does not require such data and registrants understandably pass up the chance to be so thorough. Lacking this information, the EPA has no basis on which to reach a judgment that non-chemical alternatives are effective and therefore has no basis to lower the estimate of benefits associated with use of a pesticide." P.94
"Registrants and others defending the use of a pesticide under EPA review have often predicted major crop and economic losses if products ere banned, frequently citing benefits assessment studies. In fact, we don't know of a single documented case in which such high losses actually materialized…The benefits assessment process is routinely biased in favor of chemical solutions and against biological and management-based pest management systems."
"Paradoxically, while high projected benefits have helped preserve high risk pesticide registrations, assessments showing low or negative benefits have rarely hastened cancellation of a pesticide." P.94

Consumer labels may be a legal document, but they are appended to whole formulations, on the basis of tests done on a single active ingredient (4th paragraph up). Anyone who knows that can draw their own conclusions about what a PMRA safety statement means. Please refer to the separate updated documents on *Forms of 2,4-D, Fact sheet on 2,4-D Formulants,* and *2,4-D Formulants* as well as the open letter I sent to Minister Dosanjh to document that there is absolutely no basis in fact for ever stating that 2,4-D is 'safe when used according to label', as neither 2,4-D or most other chemicals are used as the pure products tested.

In view of all this evidence, **I therefore request that the PMRA recant the statement of safety of 2,4-D published in the press release of February 21, 2005.**

Such a safety statement is disallowed under the PCP regulations and directive because people may misinterpret it as meaning personal safety. In view of the evidence presented in these comments, the PMRA is guilty of misleading the Canadian public by making such a statement in the February 21 media release.

If the more restrictive label directions relate to the value assessment for 2,4-D use on lawn and turf and are not a requirement for an acceptable human health risk assessment, then it brings the question of why it took 50 years of 2,4-D use to establish the lowest effective use rates and application methods in order to avoid unnecessary exposures. This interpretation, however, flies in the face of the PMRA statement in PACR2005-01 that if warranted the will more limitations when the 2,4-D diethanolamine studies have been properly evaluated. Correct me if I am wrong, are these not safety studies?

**I further request that 2,4-D be banned in Canada for use on lawns, by 2006 - just as it will be in Quebec**. It seems like there are so many data gaps and uncertainties for 2,4-D that they will never be filled up in my lifetime.

I request that 2,4-D be banned in Canada for use on lawns, by 2006 for the following reasons:

- 92 % of turf products currently contain DEA (as a form of 2,4-D mixed amines, by the definition in PACR2005-01)**,** and not as a formulant – I would have been unable to figure out formulants occurrence because of the secrecy clause)
- over 80 % are used in mixtures identified as synergistic but not evaluated as such,
- most mixtures contain racemic mecoprop for which the health data is incomplete,
- all turf use products contain formulants, 7 of which are on PMRA list 2 and 3 on list 4B, and about 70 % of which are already recognized as toxins in the US.

- 2,4-D was found to be a risk by the International Agency for Research on Cancer. The new PMRA document did not conclude that 2,4-D is not a carcinogen. Child cancer merited more study, but this was considered too difficult. Furthermore, the US EPA ‘s new cancer risk assessment concluded that children 2 years old and younger might be 10 times more vulnerable than adults to toxins. There is significant doubt, and the Precautionary Principle should prevail.
- The PMRA stated that 2,4-D is 'safe' IF 'label directions are followed'. This means that we and our children are at risk of harm when label directions are not followed. Hardly any monitoring takes place in Canada, and surveys and studies indicate that labels are mostly not followed. The Precautionary Principle would dictate that 2,4-D not be used.
- In the manufacture of 2,4-D, dioxins are potentially formed. Dioxins are persistent, bioaccumulative and carcinogenic. Since no check of 2,4-D products has been done since 1983, since the manufacturers have not yet submitted the mandated dioxin data, and since the data required by the PMRA do not include the dioxins most likely to be present (2 and 3 chlorine ones), there is no guarantee they are not present in consumer products without regular monitoring, which the PMRA indicates they are not willing to mandate.
This seems to be based on 2003 report to the Canadian Council of Ministers of the Environment, stating that all herbicide use in Canada accounts for 0.000006% of dioxin contamination in the environment and that there are many more significant sources of dioxin contamination, such as burning household garbage (11%) and commercial incineration (23%). Considering your own statement of the lack of monitoring for appropriate dioxins in herbicides, I am puzzled as to how that % was obtained.
- No testing is carried out for herbicide related dioxins (2 and 3 chlorine) in sediments in waterways adjacent to 2,4-D use.
- The stink that emanates from storage or use of lawn pesticides is the smell of the break-down product 2,4-dichlorophenol, which is very toxic. This chemicals is not even mentioned in the review of 2,4-D.
- Important gaps in relevant information exist, for example, reproduction and neurotoxicity studies required by the PMRA were not submitted, and cancer in children merited study but was not considered.
- 2,4-D has been found in blood, urine and semen. 2,4-D and has been linked to neurological impairment and to reproductive risk.
- From an environmental point of view, the use of 2,4-D on lawns is an ecological aberration because it favours monocultures that are very vulnerable to insect infestations. The CMHC identified much more sustainable practices.
- The value section of PACR2005-01 is not very informative of a scientific approach to estimate or calculate value.

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## Comments on Particular sections of PACR2005-01

***2.3 Regulatory history of 2,4-D***

*"The first Canadian re-evaluation of 2,4-D was announced in 1980 under the authority of Section 19 of the Pest Control Products Regulations. A re-evaluation of 2,4-D was undertaken in view of this product's broad range of applications and long history of use. It resulted in the identification of substantial data deficiencies that have since been filled by the key manufacturers of 2,4-D." A large number of more modern studies have been conducted, which have generated chemistry, mammalian toxicology, residue/metabolism, exposure, environmental toxicology and environmental fate data, to support the continued registration of 2,4-D in Canada and other countries, including the United States."*

This statement is not technically correct because it does not mention why the re-evaluation needed to be done at that time. It would be good to remember that 2,4-D, as well as many dozens of other pesticides (herbicides, insecticides etc) was on the list of chemicals for which the IBT laboratories had done all or most of the safety testing. The IBT testing[15], as well as that of many other labs at that time and since was found to be fraudulent. [15] [16]

What is important here is to notice the pattern that continues to this day: Canada as well as the US EPA knew that there were no or few valid studies on which to base a risk assessment and yet, **the chemical stayed on the market without any restriction knowing this important lack of information**[16] until the data is submitted and re-evaluated several decades later. The same is seen now. Studies on DEA (ingredient form) and developmental neurotoxicology not in but, instead of taking a precautionary approach, the note is made that 2,4-D may need further restrictions once these studies are received and analysed.

Furthermore, when the PMRA has determined to restrict some uses of a product, the pesticide continues to be sold with labels that are known to be inappropriate to protect health and environment for several more years.[17]

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[15] Novak, Roger A.; *The long arm of the lab laws*; Committing fraud in a chemical laboratory can be hazardous to your freedom; © 2001 American Chemical Society. (on their site)

[16] Van Strum, Carol; 1983; *A Bitter Fog Herbicides and Human Health;* Sierra Club Book, p.189-198 "
No registrations were suspended on the basis of falsified or shoddy IBT testing" p 188
p 192 "In April 1981, von Stackleberg files a Freedom of Information Act request for results of E.P.A. audits of other testing laboratories."
'The report they sent was on many more than eight or nine labs', von Stackleberg says. 'Of eighty-two labs audited, there were serious 'deficiencies' in twenty-five, and the routine destruction of laboratory reports and other documents made it impossible to audit the work of another twenty-two of the eighty-two labs.'

[17] Will the PMRA recall all unsold products with old labels more permissive than current guidelines? You have not in the past. A good example is racemic mecoprop which will be allowed for sale until Dec 31, 2009.

***"2.4 External consultation for 2,4-D review***
*The PMRA convened an independent five-member expert Science Advisory Panel in June 2003 to comment on and provide input into the mammalian toxicology and exposure re-evaluations, the preliminary human health risk assessment as well as the environmental risk assessment of 2,4-D for lawn and turf use."*

The panel members were listen in App 1 : *"Dr. Tye Arbuckle, Healthy Environments and Consumer Safety Branch, Health Canada; Mr. Jeff Dawson, USEPA; Dr. Claire Infante-Rivard, Department of Epidemiology, Biostatistics and Occupational Health, McGill University; Dr. Leonard Ritter, Canadian Network of Toxicology Centres and Department of Environmental Biology, University of Guelph (Panel Chairperson); and Dr. Keith Solomon, Centre for Toxicology and Department of Environmental Biology, University of Guelph."*

The PMRA's definition of *"'independent'* indicates that *"the panel members of the Science Advisory Panel are independent of PMRA, in that none are PMRA staff members and that none took part in PMRA's assessment process."* [18]

Arbuckle and Infante-Rivard have published papers that lead me to think they are independent, I don't know anything about Jeff Dawson, but the presence of Ritter and Solomon on this committee is laughable and unacceptable.

If you bother reading publications by Crop Life Canada[19] and other pesticide industry publications, you will undoubtedly run into several articles a least from Solomon. Someone who is writing and presenting extensively and openly admits that "*concern for health effects…do not exist*," and compares believing in a cancer link to pesticides as "*believing in ghosts"* hardly seems to have the open mind required of an independent committee member.[19]

At the end of 2002, Solomon was also listed as a consultant with Ecorisk, which mostly has pesticide industry clients. Solomon also wrote to downplay the negative health effects of second hand smoke.[20] Anyone who has followed the

[18] "PMRA INFOSERV" PMRA_INFOSERV@hc-sc.gc.ca; Friday, April 01, 2005

[19] Groundswell; Crop Protection Institute vol 31 jan 2001 http://www.croplife.ca/english/pdf/newsletter0101.pdf where Solomon implies that anyone who believes that there is a link between pesticides and cancer is like *"some people believing in ghosts "in spite of countless years of study that have failed to show evidence in support of these phenomena." "I do not care one way or another if they choose not to use pesticides. I do care when this is done in the name of science and concern for health effects when, realistically, these do not exist*." Can someone making such statements really keep an open mind towards new evidence?

[20] 16 March 1997;Toronto Star supporting the tobacco companies. He is quoted as saying that gun-shot wounds were more of a problem than second-hand smoke. As referred to by http://www.electric-words.com/junk/carlo/carlo1.html; The following google entry unfortunately come up blank now:

pesticide bylaw debates across Canada will recognize the names as the two "independent expert" speakers most often brought in by industry to defeat the "forces of evil" (environmental and health activists). From articles they wrote, interviews they gave, to their presentations at hearings, there was absolutely nothing either one said that may imply the status of "independent". In fact, I would qualify their presentations as fitting the science of public relations a lot more than real science.

Solomon and Ritter are generally considered as industry "experts", which implies to many that they have lost their objectivity. Having these two "scientists" as panel members weakens any conclusions drawn by the PMRA about the health effects of any pesticide, including 2,4-D.

I believe that, in order to be able to appropriately assess studies, all PMRA staff and any outside contractors or experts hired by the PMRA should be familiar with the article "*HARLOT plc: an amalgamation of the world's two oldest professions*"[21] illustrating documented and commonly used ways of "adapting" scientific results to one's financial bottomline. Granted, this article deals with the drug industry, but many of the points apply equally well to pesticide testing, especially when one considers the same companies manufacture both. The mandatory disclosure of relationships to industry now commonplace in the world of medical publishing (because of the industry's documented tremendous abuses) has to make it to toxicological and environmental publications and data as well. The responses to the article indicate that the recorded behaviours are well-known and documented and very widespread.

It is no longer sufficient that any "industry-sponsored studies are required to adhere to internationally approved study protocols, which are subject to strict quality control and quality assurance scrutiny." The medical community has recognized that and risen to the challenge of objective data. Now it is time for toxicology to do the same.

***"2.6 Forms of 2,4-D***
*2,4-D is sold in a number of different amine salt or ester forms, all based on 2,4-D acid.*

***"Table 2.6.1 Forms of 2,4-D included in this assessment***
***Grouping Form***

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The Making of Peril by **... Keith Solomon**, Ph.D., Director of the Centre for Toxicology, University of Guelph. **...** Dangers of active and passive (**second hand**) smoking **...** www.uoguelph.ca/cntc/educat/peril/making.htm - 32k

[21] Sackett**,** David L *director*[1], Andrew D Oxman**,** *director*[2] on behalf of **HARLOT** plc; 20 Dec 2003; BMJ 2003;327:1442-1445 doi:10.1136/bmj.327.7429.1442; Snakes, ladders, and spin: *HARLOT* plc: an amalgamation of the world's two oldest professions; http://bmj.bmjjournals.com/cgi/eletters/327/7429/1442
Feel free to read the responses to absorb how knowledgeable people everywhere see the objectivity of industry testing and government regulations

## Forms of 2,4-D

Please see introduction and attachment on *Forms of 2,4-D* for comments on this section.

***"3.2 Physicochemical properties of 2,4-D acid and interpretation***

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|---|---|---|
| *Vapour pressure at 25°C* | *1.87 × 10$^{-2}$ mPa* | ***Low potential to*** volatilize ???? |
| *Henry's Law constant* | *1.32 × 10$^{-5}$ Pa m$^3$ mol$^{-1}$* | ***Non volatile from water or moist surfaces*** |

Thank God the PMRA realizes that 2,4-D is soluble in water. After the publication of Bernie Hill's solubility tables[22], it would have been a mistake not to recognize that.
I cannot figure out the statement that 2,4-D has "low potential to volatilize" and is "non volatile from water or moist surfaces". It is refuted by facts and many studies. If this statement is a technicality such as one form does not evaporate but is immediately transformed into one that does, then the form found in 2,4-D vapour should be considered for the exposure assessment, and not the original form which does not evaporate. Obviously when 2,4-D is regularly found in air and rain, it has to get there somehow, and people are exposed to it.

The report *Secondhand Pesticides Airborne Pesticide Drift in California*[23] defines total pesticide drift as droplets, dusts, volatilized vapor-phase and contaminated soil particles. It also mentions that the EPA 's definition of drift is exceedingly narrow and fails to include all forms of drift, and "in some cases includes less than 5 % of the total amount of off-site airborne pesticide drift." (p.4 Exec summary). "The present language on pesticide products does not adequately control spray drift that occurs during applications."[23] Furthermore, "the US EPA's approach to spray drift control focus on technical specifications such as spray droplets size and minimum and maximum allowable wind speed, most of which would be very difficult to enforce. The fact that acute poisonings still occur with disturbing regularity suggests that such minor technology enhancements simply will not suffice."[23]

17 % of 2,4-D is estimated to end up in the atmosphere by Environment Canada[24], with a good proportion falling back in rain. USGS data shows that 60

[22] Hill, B.D.; Leaching Potential (LP) Rankings for Herbicide Products Listed in the Alberta Crop Protection 2000 Handbook; Agriculture and Agri-food Canada; Research Branch; Lethbridge
[23] Kegley, Susan et al; 2003; *Secondhand Pesticides Airborne Pesticide Drift in California;* Californians for Pesticide Reform
[24] Environment Canada; Agricultural Pesticides and the Atmosphere; The Green Lane™, Environment Canada's World Wide Web site ;
http://www.ec.gc.ca/science/sandeoct01/article3_e.html
"Studies at agricultural research stations in the province estimated post-application volatilization rates for 2,4-D and triallate at about 18 per cent."

% of all air samples are contaminated with 2,4-D.[25] As is the case in the US[23], not only is there application drift in Canada, but there is also particle drift from wind and volatilization after application, neither of which seems to be adequately considered by the PMRA in this exposure assessment. At the very least, stating that 2,4-D "has low potential to volatilize" and is "non volatile from water or moist surfaces", must brings a significant underestimation of air exposure of bystanders to the skin, clothes and respiratory system. Exposure in air includes skin and clothes as well as respiratory. They can not be considered separately.

In section *5.1* ***Environmental fate,*** you recognize that ***"****Monitoring data indicate that 2,4-D is* ***prevalent in prairie rainfall at low concentrations (Donald et al. 2001; Hill et al. 1999; Waite et al. 2002)."*** Indeed, Grover [26] measured 20.8 % evaporation in 5 days (ester) in agricultural setting, concentrated when it is hot and sunny and when the soil is wet. Donald[27]'s latest article has a figure of the relationship between pesticide concentration in wetlands and their solubility in water. The most common pesticides in water are also the most common ones in rain.

**There is definitely a need to revisit the volatilization of 2,4-D and all exposure data as it considers exposure through air as negligible and is not considered in PAC22005-01. If EC estimates 18 % volatilization, then this % should be used in exposure calculations. Plug in real data in the models instead of theoretical contraptions.**

In another section of your draft assessment you mention that 8 % of all 2,4-D 's use was domestic in 1988 but that agricultural use has decreased 30 %. That has to proportionally increase the role of domestic use 2,4-D. It is therefore inappropriate to just defer this part of the assessment strictly to agriculture.[28]

Although most lawn use of 2,4-D is of the supposedly non-volatile amines, 2,4-D has nonetheless been measured 48 feet from treated areas, 1/6 of children whose lawn has been sprayed showed 2,4-D in body tissues[29] (the same as the CDC US population data[30]), and it has been measured indoors in concentrations up to 10 times higher than before lawn treatment.[31] I can assure you that 2,4-D

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[25] Cox, Caroline; 2,4-D:Exposure; Journal of Pesticide Reform; winter 1999 vol 19 no 4; p. 14-19, quoting Majewski, M.S.; and P.D. Capel. 1995. *Pesticides in the atmosphere; distribution, trends, and governing factors*. Pesticides in the hydraulic system. Vol 1. Chelsea, MI: Ann Arbor Press, Inc, p. 79

[26] Grover, r et al; 1985; *Fate of 2,4-D iso-octyl Ester after Application to a Wheat Field*; Environmental Quality 14 (2), April-June p. 203-210

[27] Donald, David B et al; 2005; *Mobilization of Pesticides on a Agricultural Landscape Flooded by a Torrential Storm*; Envir Toxic. and Chemistry 24(1) pp.2-10

[28] section 5.1 "However, this probably originates mainly with agricultural uses rather than turf and lawn uses, and will be addressed in the re-evaluation of 2,4-D agricultural uses."

[29] Samuel, O. and M. Valcke, "Study on Body Contamination of Children in Quebec," presented at the "Pesticides in Our Bodies: A Toxic Legacy," October 18th, 2004;

[30] United States Center for Disease Control; 2003; "Second National Report on Human Exposure to Environmental Chemicals"

[31] Nishioka, Marcia G.; 2001; Distribution of 2,4-D in Air and on Surfaces inside Residences **after Lawn Applications**: Comparing Exposure Estimates from Various Media for Young Children; Environmental Health Perspectives Volume 109, Number 11, November 2001

formulation vapour is most concentrated in the area where it is used, as I can still pick up every treated lawn because of my severe pain reactions, even when I am driving in the street in a car with the vents shut off. I used to have reactions from any treated lawn for at least 2 weeks, which indicates that there is still evaporation at that time. Many thousands of environmentally sensitive and asthmatic people across Canada can identify sprayed lawns equally well from their own severe reactions. Whatever is evaporating reaches levels high enough to cause illness in municipalities at peak use time (late May-June and Aug-Sept here in Saskatchewan, although since many large lawn companies are expanding their business, I hit sprayed lawns 2 times out of 3 from the first week of May to the end of September).

In addition, when I stay in town at those times, the result is asthma and respiratory problems, as well as fatigue, pain and weakness, while I can avoid these negative side effects by going away for 7-10 days and asking my neighbours to spray or weed-and-feed while I am gone. I may call it a holiday, but it not always convenient to leave home for such a reason. Who should compensate me for the expensed I incur from leaving town?

The contribution of local domestic use of 2,4-D cannot be ignored when measuring exposure.

### 7.2 Impurities, byproducts and contaminants
### 2,4-dichlorophenol ignored

By industry's own admission, 2,4-dichlorophenol is released with the application of lawn care products containing 2,4-D.[32] I find it interesting that industry mentions it as being of "extremely low toxicity as a vapour"[32] while Health Canada mentions that "*Chlorophenols are readily absorbed when administered by the oral, inhalation or dermal routes. They accumulate mostly in the liver and kidney of experimental animals and to a lesser degree in the brain, muscle and fat*."[33] 2,4-dichlorophenol (CAS: 120-83-2) is now monitored by the US Toxic Release Inventory (2002)[34], is on the drinking water Contaminant Candidate List

[32] Landscape Ontario's Lawn Facts; #6 ; http://www.lawnfacts.ca/article-0013.shtml

*"Exposure to the odour of 2,4-D is hazardous"*

Pure 2,4-D is odourless. **Odour associated with the application of lawn care products comes from a small amount of the primary breakdown product, 2,4-dichlorophenol (DCP).** Another odour may come from a small amount of dimethylamine, a neutralizing agent of the 2,4-D formulation. Both of these compounds are considered extremely low toxicity as a vapour, although low levels are quite odorous.

[33] **PDF]** Chlorophenols; February 1987, (edited October 1987); www.hc-sc.gc.ca/hecs-sesc/water/pdf/dwg/chlorop.pdf

[34] http://toxmap.nlm.nih.gov/toxmap/main/mapIt.do?registryNumber=120-83-2

(EPA),[35] is listed in the Domestic Substance List[36], the EXSD - Pilot Phase for Screening Assessment; He Canada[37], and List B3 of the ARET Substance List; Environment Canada[38] in Canada. The government of Quebec has criteria for remediation[39] and at least one company listed by Industry Canada has developed a process to clean chlorophenols including 2,4-dichlorophenold from the environment.[40]

Yet, there is no mention of it in this 2,4-D assessment, while you mention potential contamination by dioxins and NDMA in the *7.2 Impurities, byproducts and contaminants* section. Some background research on 2,4-dichlorophenol indicates a 1/2 life of 5.3 days in air[41]. 2,4-dichlorophenol may be released to the environment in effluents from its manufacture and use as a chemical intermediate and from chlorination processes involving water treatment and wood pulp bleaching. Releases can also occur from various incineration processes or from metabolism of various pesticides in soil.[41] The EPA set a RfD of 3E-3 mg/kg/day for decreased delayed hypersensitivity response, and a LOAEL of 30

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[35] Chemical Regulation Reporter: Current Reports Index – summary; **INDEX**
**Vol. 29, Nos. 1-13, pp. 1-352 Jan. 3 -- March 28, 2005; http://www.bna.com/current/che/topd.htm**

2,4-Dichlorophenol *(120-83-2)*
– Drinking water, Contaminant Candidate List, EPA includes in final second list, 222

[36] http://www.ec.gc.ca/substances/cgi-bin/cas_e.exe?requete_cas=120-83-2

[37] EXSD - Pilot Phase for Screening Assessment; He Canada ; Last Modified 2002-04-22
http://www.hc-sc.gc.ca/hecs-sesc/exsd/pilot_phase.htm

**[38] ARET Substance List; Environment Canada; Last updated: 2004-01-05, Last reviewed: 2004-01-05 List B-3 (meet or exceed toxicity criterion);**
**http://www.ec.gc.ca/nopp/aret/en/list.cfm**

[39] Quebec Government; Soil Protection and Contaminated Sites Rehabilitation Policy; APPENDIX 2 / Generic criteria for soils and groundwater lists 2,4-DCP as .3 microgram in ground water, and 100 microgram under seepage into surface water or infiltration into sewers.
http://www.menv.gouv.qc.ca/sol/terrains/politique-en/appendix2-criteria.htm

[40] -- Industry Canada; Biotechnology research Institute NRC Last Updated: 2004-10-04;
http://strategis.ic.gc.ca/app/ccc/search/navigate.do?language=eng&portal=1&estblmntNo=234567037732&profile=completeProfile

**"Chlorophenols are a major group of pollutants of environmental concern because of their** toxicity and widespread uses**. Among the 19 congeners of chlorophenols, 2-chlorophenol (2-CP),** 2,4-dichlorophenol (2,4-DCP), **2,4,6-trichlorophenol (2,4,6-TCP), and pentachlorophenol (PCP) are listed in the** Priority Pollutant List of the U.S. Environmental Protection Agency (USEPA**)."**

[41] Spectrum Laboratories: Chemical Fact Sheet – CAS # 120832;
http://www.speclab.com/compound/c120832.htm

ppm for subchronic toxicity to rats.[42] I question the reasoning behind the low confidence in this oral RfD[42]. It implies that evidence is discounted for no better reason than 'we don't usually look at that', a non-scientific statement if there is one. The same document mentions under section 1.B that the reference Concentration for Chronic Inhalation Exposure (RfC) is not available at this time. This does not mean it's safe but that it has not been determined, which should lead to caution instead of an inappropriate statement such as "safe when used according to label". Furthermore, 2,4-dichlorophenol "accumulates in annelids, fish and insects" and is a "suspected endocrine disruptor"[43]

The issue of diffuse pollution such as would arise from pollutants being released from the use of products commonly available on the market is very poorly assessed by any regulatory agencies. It currently tends to be discounted because it is so hard to estimate a quantity. Regulatory agencies focus our regulations in point of origin such as your statement that there is very little in the 2,4-D, while microbial breakdown may locally create a large amount in turf treated with 2,4-D.

## 4.0 Effects having relevance to human health

### 4.1 Toxicology summary

*p.13 "The **DEA** form of 2,4-D had a different toxicological profile compared to the other forms listed above. Available studies and foreign review summaries showed both a qualitative and quantitative difference in the toxicological effects that occurred after oral and dermal administration of the test article.* ***Liver effects observed in a three-week dermal study in rabbits were not noted with the other forms of 2,4-D, and dietary studies indicated that 2,4-D DEA induced more severe thyroid and reproductive organ toxicity at lower dose levels when compared to all other forms of 2,4-D****."*

*"In addition, short-term oral and dermal studies indicate that pure DEA causes brain and spinal cord demyelination in rats and is immunotoxic in rats and mice (NTP 1992a, 1992b, 1994). DEA is also classified as a List 2 formulant: potentially toxic*

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[42] IRIS; File First On-Line 01/31/1987; 2,4-dichlorophenol; US EPA; http://www.epa.gov/iris/subst/0041.htm

**I.A.5. Confidence in the Oral RfD;** ***Study – Low; Database – Low; RfD – Low;***

"The study (Exon and Koller, 1985) used an adequate number of animals and measured very sensitive endpoints (immunological functions) in an appropriate manner. As these **endpoints are not commonly used in derivation of human health risk evaluations, confidence in the study is rated low**. Additional published studies did not look for the critical effects and did not support the magnitude of the NOEL/LOAEL. Therefore, confidence in the database is rated low. Low confidence in the RfD follows".

[43] PANNA database; 2,4-dichlorophenol; http://www.pesticideinfo.org/Detail_Chemical.jsp?Rec_Id=PC34811

*formulants, with a high priority for testing (USEPA 2002b). Accordingly, the database for 2,4-D acid and the DMA, EHE and BEE forms of 2,4-D* ***does not support the DEA form of 2,4-D****. The registrant has submitted additional information regarding the toxicity of DEA on its own (as opposed to the DEA form of 2,4-D) to the PMRA for consideration.* ***Mitigation measures for the DEA form of 2,4-D may be proposed depending on the outcome of the current review of this additional information****."*

See the separate document on *Forms of 2,4-D* for the number of products currently on the market containing DEA: 92 % including weed-and-feed products. This document includes the calculations and explanations. PACR2005-01 therefore has value for only 8 % of the products currently on the Canadian market. How can you justify your statement of "safe when used according to label" under these circumstance?

***4.1.1 Toxicology profile of 2,4-D acid, BEE, EHE and DMA***

*Available data indicated that all tested forms of 2,4-D were* ***readily absorbed and excreted after oral administration. Peak plasma levels were attained four hours after dosing****. Urine was the main route of excretion and tissue residues were low. The acid and amine forms were excreted unchanged, and 2,4-D esters (BEE and EHE) were rapidly hydrolysed to 2,4-D acid, which was excreted, unchanged, in the urine and, to a lesser extent, in the feces. Volatile metabolites of the esters were eliminated via expired air. Other metabolites of 2,4-D esters were recovered in the excreta. Despite the formation of other metabolites, 2,4-D esters and amine salts did not appear to impart higher toxic potentials or show different target organ toxicity relative to 2,4-D acid in acute and short-term toxicity studies."*

*p.14 "allometric scaling of data from mice, rats, dogs and humans indicated that* ***renal clearance of 2,4-D was approximately 30-fold slower in dogs compared to humans****, making the dog less relevant as an indicator species for human toxicity. For this reason, the PMRA did not utilize the dog studies in the 2,4-D risk assessment."*

*"****In all species, the primary target organ for toxicity was the kidney.*** *Short- and long-term exposures via dietary administration induced similar effects and levels of toxicity in mice and rats, whereas* ***dogs exhibited toxic effects at lower doses than rodents****."*

Considering that the kidney is a known target organ for toxicity, and that 2,4-D is excreted unchanged in the urine, it would indicate that the urinary system is exceptionally exposed to 2,4.-D. Perhaps then an increase of 4-fold in bladder cancer in dogs may not be surprising.[44] A follow-up study will also be conducted in dogs and children.[45]

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[44] Glickman LT, Raghavan M, Knapp DW, Bonney PL, Dawson MH. Herbicide Exposure and the Risk of Transitional Cell Carcinoma of the Urinary Bladder in Scottish Terrier Dogs. J Am Vet Med Assoc 2004; 24:1290-1297.

[45] Hively, Suzanne;Thursday, March 03, 2005; Study finds higher rates of bladder cancer among dogs exposed to herbicides; Plain Dealer Columnist
http://www.cleveland.com/living/plaindealer/index.ssf?/base/living/1109759743127880.xml

I understand that humans have a 2,4-D retention rate between the dog and the rat. I fail to understand how eliminating the dog data from the assessment will serve any kind of safety purpose.

*"In adult rats,* ***neurotoxic effects were evident after a single high-dose exposure****. The observed incoordination and slight gait abnormality were no longer evident four days later.* ***Repeated*** *high doses also* ***affected forelimb grip strength*** *and* ***induced retinal degeneration****. Published studies involving intraperitoneal and subcutaneous administration of 2,4-D acid to pregnant rats as well as studies focusing on oral exposure of pups through mother's milk during postnatal days 15–25, resulted* ***in myelin deficiency in the central nervous system of pups****. Another study using a combination of prenatal and postnatal exposures showed a delay in the development of the surface righting reflex, geotaxic response and hindlimb support in rat pups, which correlated with* ***alterations in the development of the monoamine systems in the brains of these rats as adults*** *(Bortolozzi et al. 1999, 2003; Duffard et al. 1995, 1996; Rosso et al. 1997, 2000; Sturtz et al. 2000). Although these effects were observed at much higher dose levels relative to the doses causing the primary target effects in the short- and long-term studies, these findings* ***may be an indication of offspring sensitivity after exposure to 2,4-D during prenatal and postnatal development****."*

It is interesting that induced retinal degeneration is mentioned here. Japanese studies of epidemics of myopia and astigmatism linked them to the use of organophosphates in certain areas.[46] The scientist involved replicated the air concentrations of OPs, in particular malathion, and found damage to the optic nerve in the rats exposed at the level of exposure of people. This is not a mandated end point for pesticide registration, even though the cost of glasses and eye care has to be very high. 2,4-D has recorded neurotoxic effects so it may be appropriate to check such effects for it.

Surely, if a link is confirmed between 2,4-D and vision problems, or skin and respiratory or other health problems, the treatment cost of those conditions should be taken into consideration in the risk-assessment. After all, in Canada, the government of Canada pays for medical treatment, and therefore Canadians pay through our taxes. Surely, the cost of treating conditions resulting from pesticide poisoning can no longer be ignored in risk-benefits assessment.

*"Although a preliminary study reported fewer fetal implantations in 2,4-D treated rats,* ***errors in the study design negated the study authors' interpretation*** *(Cavieres et al. 2002). In addition, a study using a coformulation of 2,4-D and picloram, which is not*

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A follow-up study at Purdue will examine exposure to lawn chemicals in dogs and children.

[46] Saku Disease; http://www.mapperleyplains.co.uk/oprus/saku.htm

*registered for turf in Canada, was negative for male mediated birth defects (Oakes et al. 2002).”*

Cavieres’s paper[47] has never been recalled and still stands. Like all peer-reviewed papers it has been discussed.

While I have not found a formula of 2.4-D and picloram registered in Canada, my check of picloram formulations last year indicated that one did not have specific restrictions for lawns. I don’t believe it could be so far fetched to think that it may be used on lawns considering what is known about use outside of label.

**Myth of Pesticides used according to label**

You have to get it out of your mind that people use pesticides products according to label. It is nothing but a false assumption. Where are your studies to show that this is the case? On the other hand, there are many surveys and studies in the US and Canada showing the opposite for everyone, from farmers, to professional applicators and individuals.( refs available on request) Even cities: last fall saw a local city using *Escort* on Sept 29, 2004, hardly an active growing period for any weed. Aerial applicators regularly spray people, houses and gardens in Saskatchewan and nobody can do a thing about it because Federal laws are so weak. I just read a paper indicating regular illegal widespread use of OPs and carbamates for baiting predators.[48]

Only dealing with labeled use puts anyone exposed to widespread non-labeled use at risk. While non-labeled use users can in theory pay fines, the Saskatchewan complaint mechanism to Sask Ag and Foods is clearly not trying to collect information but suggesting that people deal with problems themselves by hiring an agronomist, who may be able to recognize crop damage from herbicides, but is hardly qualified to deal with health effects to livestock or people. Furthermore, SAF can only be contacted for “licensed” uses, not domestic ones. This is emphasizing again the usual lack of any consequences for most people not using pesticides according to label. This is hardly a situation conducive to following label use.

*P16 ( or 9 of doc) “Reference doses for various population subgroups have been set* ***based on no observed adverse effect levels (NOAELs) for the most relevant endpoints, namely effects on body weight, renal toxicity, neurotoxicity and maternal mortality****. These reference doses incorporate various uncertainty factors to account for*

[47] Cavieres, et al. 2002. Developmental toxicity of a commercial herbicide mixture in mice and effects on embryo implantation and litter size. *Environmental Health Perspectives*. 110(11): 1081–1085.

[48] Wobeser, G et al; 2004; *Secondary Poisoning of Eagles Following Intentional Poisoning of Coyotes with Anticholonesterase Pesticides in Western Canada*; *Journal of Wildlife Diseases,* 40(2), 2004, pp. 163–172

*extrapolating between rats and humans, for variability within human populations and for data uncertainties. Additional safety factors have also been applied, where warranted, to protect pregnant females and their unborn children as well as nursing children from identified endpoints of concern."*

NOTE: The original recommendation for safety factors for pregnant females and their unborn children was supposed to be an additional 10 for a total of 1000. However, the factor of 10 is not applied across the board as you mention here ("where warranted"). In fact you use 3.

It is obvious that if the end points considered were different, the NOAELS may also be quite different and likely more restrictive. For instance, consideration of endocrine-disruption, low dose and immune parameters, even respiratory irritation, etc might show the NOAELS you accept as "the ultimate truth" to be not even close to the protection needed.

I notice that cancer is not one of the end-points under consideration for NOAELS, and therefore final assessment. Again, as the EPA published their new risk assessment for children under 2 years old as being 10 times higher than adults, the cancer risk should be re-evaluated with proper studies instead of meta-analysis. [1] Hopefully this should put it into the end-point considered.

*Cancer*
*"Because of the inconsistent epidemiological associations, the recognition that there are many other factors that may have contributed to the weakly positive associations and the fact that the animal studies designed to show causality were consistently negative, the PMRA concurs that 2,4-D cannot be classified as to its human carcinogenicity on the basis of all available and relevant data."*

1. As neither 2,4-D or any other chemical is used alone in the real world (other than malathion for ULV spraying), the studies on isolated pure ingredients are totally irrelevant to the toxicity of pesticide formulations which we encounter every day. ( see Introduction and general comments) This indeed is the main problem in comparing "regulatory" studies with epidemiological or environmental data. I contend that the fact that we are all still in ignorance regarding the 'secret" formulants in pesticide formulations is in large part what is gumming up the results. As many formulants are added to increase permeability, their effect would be at the least additive, and at the worst synergistic with the active ingredient(s). "Regulatory" studies cannot and do not account for reality. And studying each formulant separately would not give the whole story either any more than studying gthe toxicity of carbon or chlorine can give an ideal of the toxicity of the final molecule. Only full disclosure of ingredients and studies of the toxicity of formulation product such as done by Cavieres[47] can give a true view of health effects related to real exposures.

Remember that epidemiology is a recognized branch of medicine and, like all other sciences, it should be judged according to its own scientific criteria, not consistently compared to “regulatory toxicology”, which is like comparing bananas with earthworms.

2. The PMRA still does not mandate low dose studies. Because any such study fall outside you narrow area of expertise (regulatory toxicology), I believe the PMRA may lack the expertise to know what criteria to use to evaluate the low dose studies you have to analyse. Hopefully, the expertise will come in time. However, in the meantime, your lack of expertise and requirements for these studies is putting people at risk.

**Dioxins**

*Page 11*

*“Interpretation of epidemiological results for potential cancer or developmental and reproductive effects were often confounded by factors such as the general grouping of 2,4-D with other pesticides (2,4,5-T[4], 2,4-DB, MCPA, MCPB, mecoprop, atrazine etc.), and, in older studies, pesticide contamination with TCDD[5]. This highlights the need for more precise epidemiological study designs with proper exposure characterizations to identify any specific associations between 2,4-D exposure and human health effects.*

*[4] Dioxin (TCDD) contamination of 2,4,5,-T was identified in the early 1970s and the manufacturing process was then improved to reduce this contaminant significantly (<0.5 ppm). 2,4,5-T, once registered in Canada for forestry use (not lawn and garden), has not been used in Canada since 1982. Registration was discontinued in 1985.”*

As you note, there used to be contamination of 2,4-D by dioxins. I understand that no product has been checked since 1983, just on the word of industry that the problem was solved. I contend that this is a bad regulatory decision. As in everything else, people only do what they have to, and we all need to get a parking or speeding ticket once in a while as a reminder. This indeed is why I believe that there are so many pesticide applications not respecting label specifications. No one is checking.

I understand that the 2 and 3 chlorine dioxins form the bulk of the dioxins formed in the manufacture of chlorophenoxy herbicides. Yet you only ask for 5 samples, likely carefully chosen by industry, and only to be analysed for dioxins containing 4 chlorines or more, and for which you have yet not received data.

If 2 and 3 chlorines are currently listed as toxic and worthy of attention, they should be taken into consideration as well, and tests requested for them.

Please remember that industry is picking the samples, not only for the 5 dioxin tests you are asking for, but for all the studies they hire people to perform for them. This gives them the opportunity of picking samples low in contamination for such studies compared to what is usually available on the market. In that case, the mandated studies could not be compared to what is available on the market

regarding contamination with dioxins and therefore negative health effects they may cause.

***4.2 Residential risk assessment***

*"Residential risk is estimated by* ***calculating a margin of exposure (MOE) based on comparing the potential exposure to the most relevant endpoints from toxicology studies****.*
*The calculated MOE is then compared to a target MOE, which incorporates safety factors protective of the most sensitive subpopulations.* ***If the MOE is less than this target MOE, it does not necessarily mean that exposure will result in adverse effects, rather the absence of adverse effects is less certain****. Mitigation measures are necessary to reduce exposure if MOEs are less than the target MOE."*

Again, the question of what end points are chosen for calculations will determine the end results. For instance, respiratory problems are excluded form the end-point studied although inhalation is considered 100% absorbed.[49] The assumed end-point are body weights effects from an oral study. In fact PACR2005-01 notes that there is an "*absence of repeat-dose inhalation data*".

There is no reference at all to a NOAEL for respiratory problems or any suggestion that respiratory problems have ever been considered. (I admit I have not read all the references - no time) although severe respiratory effects are commonly reported from exposure to lawn chemicals and have also been part of my personal experience.

A commercial label [50] [51] warns of severe eye injury, the domestic one checked simply gives general precautionary statements, while health sites indicate potential respiratory problems as well as skin sensitization problems which may lead to chronic conditions. [52] PACR2005-01 is asking for a respiratory warning on

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[49] section 4.4.2 of PACR2005-01: "Despite the absence of repeat-dose inhalation data, it was assumed that body weight effects would also be a critical endpoint by this route of exposure. Thus, the most relevant study to assess short-term aggregate exposure was the repeat-dose developmental toxicity study in rats, which established a NOAEL of 12.5 mg/kg bw/day based on decreased body weight gain.
"Inhalation exposure and oral ingestion through dietary and non-dietary pathways are considered to be 100% absorbed."

[50] GREEN CROSS KILLEX 500 TURF HERBICIDE LIQUID CONCENTRATE – commercial- http://eddenet.pmra-arla.gc.ca/4.0/4.1.2.asp?regn=16971%2E00&page=1&uniqueid=4%2F22%2F2005+1%3A30%3A44+PM
**"May cause severe eye irritation and reversible eye damage**." No ref to respir problems (accessed Apr 22, 04)

[51] SCOTTS CANADA LTD.* KILLEX WEED CONTROL IN LAWNS;- domestic- http://eddenet.pmra-arla.gc.ca/4.0/4.1.2.asp?regn=23948%2E00&page=1&uniqueid=4%2F22%2F2005+1%3A30%3A44+PM (accessed apr 22, 04) :: Avoid contacting skin, eyes, and clothing.

[52] Metropolitan Emergency Response and Logistical Information Network ; May, 1989;
http://www.ndcrt.org/data/EPA_Chemical_Fact_Sheets/2_4-D.html
HAZARD SUMMARY

commercial labels only,[53] although many commercial products can be used on turf. Therefore neither people exposed to the vapors or their MDs would know that their respiratory problems may be due to exposure to the formulation.

The 1984 Who 2,4-D assessment identified a number of missing data for 2.4-/d including skin sensitization and acute inhalation LC50 among others, and that "None of the available studies on 2,4-D-induced myotonia were designed to establish no-observed-adverse-effect levels for the various myotoxic effects in intact animals".[54] Nothing in PACR2005-01 indicates any consideration of myotonia or myotoxic effects, which are common in my experience.

*Page 13*

***"4.2.1 Relevant toxicological endpoints and target margins of exposure for acute and shortterm exposures to homeowners and children"***

*…" **The most relevant endpoint for acute risk assessment was considered to be increased skeletal variations in rat fetuses noted in a rat developmental study...** The target MOE was 300 based on standard uncertainty factors (10× for interspecies variation, 10× for intraspecies variation) as well as an extra 3× safety factor to protect for potential sensitivity to the young noted in a series of published neurotoxicity studies."*

*"A separate acute exposure and risk assessment was conducted for children…The **endpoint of concern was increased incoordination and slight gait abnormalities** noted in the acute neurotoxicity study in rats. In this study, the **NOAEL was 75 mg/kg bw/day** based on acute neurotoxic effects occurring at the LOAEL of 250 mg/kg bw/day."*

*"To assess the risk to **toddlers...** The rat developmental study was chosen, which had a **NOAEL of 12.5 mg/kg bw/day** based on a decrease in body-weight gain at the LOAEL of 50 mg/kg bw/day."*

The latter was an oral study adapted with a 10% factor for dermal absorption?

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* 2,4-D can affect you when breathed in and by passing through your skin.
* Skin or eye contact can cause irritation.
* Breathing exposure can irritate the throat and airways.
* Exposure may also lead to a skin allergy, with rash and itching. If allergy develops, even low future exposures can trigger rash.

REASON FOR CITATION

* 2,4-D is on the Hazardous Substance List because it is regulated by OSHA and cited by ACGIH, DOT, DEP and EPA.

[53] Section **8.2.2 Label statements relating to health**

The label text of **Commercial Class** products containing 2,4-D must include the following text:

**Toxicological Information**

2,4-D may cause severe irritation to the eyes. Prolonged breathing of 2,4-D may cause coughing, burning, dizziness or temporary loss of muscle coordination. Other possible effects include fatigue, muscle weakness or nausea. Treat symptomatically.

Nothing for domestic labels.

[54] World Health Organization; Geneva, **1984;** http://www.inchem.org/documents/ehc/ehc/ehc29.htm#SectionNumber:6.5

In here you only use rats NOAEL while we know that humans retain 2,4-D longer and are in between rats and dogs in health effects of 2,4-D. The appropriateness of this decision must be questioned.

There is no consideration of 2,4-dichlorophenol here. If it is the form that industry identifies as vaporizing from applications, it would be essential to include NOAELS for 2,4-dichlorophenol in the assessment.

It would also be valuable as one finds in EPA and WHO documents to mention what is and is not known. For instance, is there an acute or chronic respiratory NOAEL for either chemical or both? What is the NOAEL for skin and eye problems? Etc A table indicating what has been actually measured and not measured would be very useful.

Your calculations of aggregate risk assessment are based on industry statement that 2,4-D is not volatile, which was discussed earlier does not fit at all with the measurements that 18 % of all 2,4-D applied evaporates. The assessment would be more accurate if it used measurements.

**Dietary exposures**

There is no public record of whether 2,4-D is found in Canadian or other foods, other than the one study showing it at high levels in wheat products when used pre-harvest.[55] Indeed, a recent review[56] indicates that "*recently, the PDP has not tested fruits and vegetables for 2,4-D."* So again your assessment seems to be based on estimates and replacement values instead of real data.

**Drinking Water**

More and more Canadian studies show widespread contamination of drinking water with 2,4-D in mixture with MCPA, chlopyralid, dichlorprop and several other pesticides. Similarly, Cessna's study of dugouts also found contamination, much worsened at turnover time when sediment mixes in with the water column.[57] This means, drinking, cooking, washing, clothes washing etc water. People are getting exposed to a mixture of pesticides every day all the time. It can't be more chronic than that.

**Aggregate exposure**

Add to that the CDC data[30] that one out of every 6 Americans carries 2,4-D in his body and the similar Quebec children study[29] showing one out of 6 children has body levels of 2,4-D after their lawn is sprayed, and the 10 times higher levels in

[55] Cessna, A.J. and F.A. Holm; 1994; *Residues of 2,4-D in wheat following application after heading*. Can. J. Plant Science

[56] Cox, Caroline; winter 1999; *2,4-D Exposure;* Journal of Pesticide Reform vol 19 no 4 pp 14-19

[57] Cessna, A.J. and J.A. Elliott; October 1999; *Seasonal Variation of Concentrations of Herbicides and Major Inorganic Ions in Farm Dugouts*; *Rural Health and Safety in a Changing World Symposium Proceedings*; Saskatoon;

house dust after spraying and that it remains in house dust for up to 1 year.( Nishioka- see refs 57 58)

***4.2.3 Exposure and risk assessment for persons entering a treated area***
*"Postapplication exposure and risk were estimated for children and adults contacting treated residential lawns and golf courses,* ***based on assumptions outlined*** *in the USEPA's draft Standard Operating Procedures (SOPs) for Residential Exposure Assessments and the recommended revisions by the USEPA's Science Advisory Council (USEPA 1997b, 2001)."*

*"Acute and short-term risk assessments were conducted as there is potential for relatively* ***higher exposures to children and adults on the day of application, and for repeated lower exposures over a short-term period (1 to 7 days), as 2,4-D residues dissipate****. Based on TTR data generated by the Broadleaf Turf Herbicide Transferable Foliar Residue task force, peak TTR levels were 2.63% of the applied rate and 7–day average TTR levels were 0.35% of the applied rate."*

*"New postapplication exposure data relevant to estimating dermal exposure from contact with treated turf were received from the ORETF in February 2004. The PMRA, the USEPA and the California Department of Pesticide Regulation are* ***currently evaluating*** *these data. Preliminary calculations suggest that, while exposure estimates might increaseslightly, target MOEs would still be met for all postapplication scenarios.* ***If necessary, the PMRA will publish a revised risk assessment after a full review of the new data****."*

*"The contribution of inhalation exposure to overall exposure in postapplication scenarios is considered to be negligible, due to the low volatility of 2,4-D and the dilution effect of outdoor use. This rationale is supported by evidence from several published studies."*

One of many assumptions is finally recognized here, as well as a reference to dissipation over 1-7 days. If 2,4-D still dissipates for 7 days, **should the re-entry interval not be extended ?**

Exposure seems to be calculated strictly on the basis of transferable residues (TTR). Before I can accept results from the *Broadleaf Turf Herbicide Transferable Foliar Residue task force*, it would be useful to know who was on it. The comments in the section about the independence of the advisory committee apply here. That new data have not yet been evaluated by EPA should make the PMRA play safe instead of having to reduce exposure again later.

Inhalation exposure cannot be discounted because18 % of 2,4-D evaporates after application, 2,4-dichlorophenol is an integral part of the evaporative products according to industry[32] (see previous section on 2.4-dichlorophenol),

with dimethylamine[32], which can cause skin burns as well as wheezing and shortness of breath.[58]

If it's not the 2,4-D itself that is found in the air, whatever it is should be measured and taken into consideration in calculating human exposure, as well as water and indoor chronic persistent exposures.

I see no mention of other recent studies measuring 2,4-D in dust, carpets, air in houses, or from Samuel's study[29] of 2,.4-D in 1/6 of the children's urine.[59] [60]

*P 27* ***4.4.2 Short-term aggregate risk assessment***
*Short-term aggregate exposure to 2,4-D was* ***estimated*** *based on contributions from food, drinking water and residential exposure (dermal, inhalation and oral components). With respect to route of exposure, there was no systemic toxicity in short-term dermal exposure studies. However, the oral route of exposure (rat and rabbit developmental studies) confirmed that decreases in body weight and/or body-weight gain were consistent endpoints of concern.* ***Despite the absence of repeat-dose inhalation data[61], it was assumed that body weight effects would also be a critical endpoint by this route of exposure.*** *Thus, the most relevant study to assess short-term aggregate exposure was the repeat-dose developmental toxicity study in rats, which established a NOAEL of 12.5 mg/kg bw/day based on decreased body weight gain.*

*Short-term aggregate exposure* ***estimates*** *from food, residential exposure (dermal, inhalation and incidental oral components) and drinking water did not indicate any unacceptable risk. The calculated DWLOCs ranged from 170 to 910 µg/L, with the most sensitive population subgroup being females 13–50 years re-entering areas treated with 2,4-D BEE. These were compared to the chronic estimate of 2,4-D residues in drinking water, which is 0.3 µg/L. This is* ***lower than the calculated DWLOCs for all populations and, therefore, is below the PMRA's level of concern.***

This statement is based on one stated assumption, and 2 estimates of questionable value (because of little basis in measurements).It is also based on 2 assumptions inherent to the current regulatory process: the dose makes the poison, and there is a threshold below which there are no effects. Endocrine-disruption and low dose studies evidence have put these 2 assumptions into question [2] [4] as the only principle guiding toxicity and yet, all the mandated data

[58] see accompanying document on formulants in 2.4-D formulations
[59] Nishioka MG, Lewis RG, Brinkman MC, Burkholder HM, HinesCE, Menkedick JR. Distribution of 2,4-D in Air and on Surfaces inside Residences after Lawn Applications: Comparing Exposure Estimates from Various Media for Young Children. Environ Health Perspect.2001;109:1185–1191

[60] Nishioka MG, Burkholder HM, Brinkman MC, Gordon SM. Measuring Transport of Lawn-Applied Herbicide Acids from Turf to Home: Correlation of Dislodgeable 2,4-D Turf Residues with Carpet Dust and Carpet Surface Residues. Environ. Sci. Technol., 1996;30 (11), 3313 -3320.
[61] The absence of this data was identified by WHO in 1984 in WHO http://www.inchem.org/documents/ehc/ehc/ehc29.htm#SectionNumber:6.5

are based on them. This is one major illustration of why the EPA states that "*information needed to support the registration of a pesticide chemical has evolved as the general scientific understanding of the potential hazards posed by pesticides has grown*"[2]

There is yet another hidden assumption, that what is currently considered the acceptable levels in water and food are protective enough. We have seen these "acceptable" values being brought down for many toxins in the past (lead, mercury, PCBs) and now a recognition that many of these toxins affect health at all levels therefore do not have an effects threshold. These numbers were likely achieved looking only at one exposure pathway and one chemical at a time. When one seed the CDC data and realizes that the average American carries 13 out of the 38 pesticides tested, plus a lot more other foreign chemicals in their bodies, one has not only a right but an obligation to question whether the established guidelines are appropriate for current conditions and exposures. The recent statement by Ms Carter-Phillips, head of Health Canada's cosmetics program, that the burden of testing is put on the companies and that *"What may be acceptable today, based on the science, may not necessarily be acceptable in the future"*[62] applies equally to pesticides. You cannot get any meaningful public protection from standards that mostly ignore the last 20 years of science discoveries.

## *5.0 Environmental assessment*

*"Environmental risks from the use of 2,4-D on lawns and turf were assessed. The standard deterministic approach (tier 1) was used, and risk was characterized by the quotient method, the ratio of the estimated environmental concentration to the effects endpoint of concern for the most sensitive species.*
***Risk quotient values less than one are considered indicative of a low risk of non-target effects occurring,*** *whereas values greater than one are considered to indicate that some degree of risk exists for effects on non-target organisms."*

### *5.1 Environmental fate*

*"Therefore,* ***all of these forms are considered to be highly mobile and susceptible to run off from the treatment site****. There may be a potential for leaching to groundwater, particularly in areas where the soils are permeable (e.g., sandy soil) and where the depth to the water table is shallow, provided there is rainfall soon after application.*

In spite of the recognition of this potential serious problem of groundwater contamination, all you request is a warning on the label. 2,4-D has a long 1/2 life in groundwater. A restriction from use in permeable soils would be more appropriate. [63]

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[62] Page, Shelley and S. Allan; April 16, 2005; *Not so pretty: Most beauty routines include the use of carcinogens, allergens and other harmful substances*; The Ottawa Citizen

[63] section **8.2.3 Label statements relating to the environment**

*2,4-D acid and the amines are* ***non volatile owing to their low vapour pressures*** *(WHO 1998).* ***2,4-D EHE and 2,4-D BEE esters are more volatile*** *than the amines or the acid owing to their higher vapour pressure. Monitoring data indicate that 2,4-D is* ***prevalent in prairie rainfall at low concentrations (Donald et al. 2001; Hill et al. 1999; Waite et al. 2002). However, this probably originates mainly with agricultural uses rather than turf and lawn uses, and will be addressed in the re-evaluation of 2,4-D agricultural uses."***

I already dealt with the inadequately treated issue of volatility under ***"3.2 Physicochemical properties of 2,4-D acid and interpretation.*** Please refer to that section for discussion.

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*P 32 "Aerobic biotransformation is the major route of transformation of 2,4-D as well as its amine and ester forms in soil and the aquatic environment."*
*classified as non-persistent to slightly persistent in soil and natural water because of aerobic biotransformation, with* ***half-lives of 1.7 to 31 days in soil and 4.5 to 29 days in water****. Biotransformation of the EHE and BEE esters in soil*
*and natural water occurs rapidly with half-lives of less than two hours (WHO 1998). The biotransformation rate is reduced under anaerobic conditions, and the* ***amine form (DMA) is persistent under anaerobic conditions with a half-life exceeding one year in sediments****.*

Please see the section of *2,4-dichlorophenol ignored* for products of transformation.
There seem to be many other degradation products equally undealt with here such as 4-chlorophenoxy acid, which degrades into 4-chlorophenol and the rest of that cascade and maybe other I am not aware of at present. I did not have time to look up their ½ life or what I could find on them, but ignoring them cannot serve human or environmental health. When degradation products and their particular characteristics and toxicity are an integral part of using a product, they have to be taken into consideration when establishing exposure and environmental assessment.

## p.32 environmental toxicology

*"Granular formulations provide a unique exposure scenario, because birds use grit to aid in digestion of food. The number of granules required to reach the LD50 for a particular size of bird and the number of granules available per metre square were compared to determine risk. For the granular products of 2,4-D, the assessment indicated some* ***potential risk of acute effects to very small birds (sparrow size, less than 15 g), assuming the birds will consume the granules.*** *However, the risk to small birds*

"The use of this chemical may result in contamination of groundwater particularly in areas where soils are permeable (e.g., sandy soil) and/or the depth to the water table is shallow."

*from granular formulations is limited because the* ***granulars are much larger than their preferred grit size****.*"

The direct risk to birds of granular formulations is therefore considered unavoidable and acceptable, as was stated to a public meeting by a local PMRA inspector. Pierre Mineau[64] gave an estimate of 68 million birds/year dying form pesticide use in North America.

Weed and Feed type combination fertiliser / pesticide products are very popular, (203 products of which 142 contain 2.4-D)

As well as being a danger to birds who may eat it as grit, this type of product should be prohibited because:
• Granular "weed and feed" products are slow release therefore designed to persist longer in the environment
They out-gas over extended periods of time, so neighbours who have to leave their homes don't know when it is safe to return.
• Granular "weed and feed" products are over applied to the entire lawn, not merely to areas of weeds, so are counter to pesticide reduction strategies. The mixture of fertiliser and herbicide is incompatible because one ingredient should be applied to the entire lawn, and one is intended for problem spots. Indeed, unfinished packages of the product are commonly used even when there are no weeds, and the homeowner merely wants some fertiliser. Combination products are responsible for untold excessive use of pesticide.
• Granular "weed and feed" products stick on shoes and children's hands and are very mobile. Dust is carried by the wind and tracked indoors. As noted above, children's exposures from dust may greatly exceed expected exposure from food.

As has been documented by Rachel Carson in Silent Spring (1962) and since, a major effect of herbicides is indirect, by disturbing their habitat and food supply.**Error! Bookmark not defined.** A uniform lawn obviously is less diverse, and likely contributes to the absence of many species of birds in urban areas. 2,4-D is very toxic to some earthworms, **Error! Bookmark not defined.** which could impact Robin populations.

I did not see anything in your assessment (Proposed Acceptability for Continuing Registration - PACR2005-01) to even attempt to take into consideration indirect effects of 2,4-D on wildlife.

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"However, in a real life situation the risk would be less than predicted because in natural water, biotransformation of the esters to the acid occurs rapidly (i.e., in less than two hours). This was supported by monitoring data showing that 2,4-D in runoff presents

[64] Mineau, Pierre; March 103, 2000; *Pesticides and Birds – A Practical Approach*; Wildlife toxicology workshop; Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon; SK

little or no potential risk to freshwater and estuarine/marine fish. Monitoring data also confirmed that there was no risk to aquatic invertebrates, except from the ester form (EHE). **The EHE form presents a moderate risk owing to its higher toxicity than the other forms."**

This implies that you consider a short ½ life to be of no consequence to organisms or ecosystems. This is another questionable assumption. Please look at recent research dealing with pulse exposures. It finds that effects on a species or an ecosystem are often worse because, without constant pressure from a particular chemical, the system goes back to forms and species not well adapted to a constant onslaught of chemical, and is therefore somewhat taken by surprise every time, increasing the negative effects.

Intermittent doses, even short-lived, at above aquatic life guidelines have the potential to severely disrupt an aquatic ecosystem. Aquatic organisms, just like humans, can only die once. I understand from researchers in the field that above guidelines pulses are a regular occurrence in many systems, including in Saskatchewan.

Simple mortality for "little or no potential risk" is not a good enough measure. Growth of fish has been reduced by levels of .6 ppm, .3 ppm, and the ability of rainbow trout to capture food was reduced by 5 ppm.**Error! Bookmark not defined.** I do not have any contamination data to compare these levels to.

Pulses are harder to document and would result in effects being found without being able to identify the cause.

"In a 1988 report commissioned by Agriculture and Agri-Food Canada, it was estimated that **342 000 kg of 2,4-D a.e. were used for broadleaf weed control on turf in Canada per year.** This represented **7% of the total estimated** 2,4-D use in Canada at that time. Since the publication of that report, the total 2,4-D consumption in Canada is estimated to have decreased by 30%. The decrease in total 2,4-D use was due to the recent registration of alternatives to 2,4-D for agricultural uses.

These data are 17 years old! Mandate the industry to give you more recent ones and please make them public! As mentioned earlier, the proportional increase in turf use has to be accounted for. Furthermore, the fact that 18% of 2,4-D applied evaporates, and local concentrations certainly regularly reach levels of health effects for asthmatics and chemically sensitive people indicate that turf use contributes to air and rain concentrations. Turf use cannot be discounted as a source of pollution.

*"A **total of 69 products** (37 commercial and 32 domestic class products) containing 2,4-D are registered under the Pest Control Products Act for use on fine turf in Canada, including 35 coformulations of 2,4-D, mecoprop and dicamba; 10 coformulations of*

*2,4-D and mecoprop; 1 coformulation of 2,4-D and dicamba; and 23 products that contain only 2,4-D (as of 12 February 2004). All products are applied as sprays, except for two domestic class products that are solid formulations (bar and stick).*
*In addition to these products, there are numerous* ***fertilizer/herbicide products*** *registered for use on fine turf in Canada that contain 2,4-D. These fertilizer/herbicide products are* ***registered with the Canadian Food Inspection Agency under the Fertilizers Act and not with the PMRA under the Pest Control Products Act****. However, the conditions for use of these products must reflect the registered uses for the pesticide under the Pest Control Products Act*."

See separate document of *Forms of 2,4-D* for details and the weed-and-weed list.
I suggest that you append the list of products the PMRA licenses to the review document on 2,4-D as my own count, in February 2005, came to 83 products: 40 domestic (sale to the public without license) and 43 commercial instead of your 69. An appended list would have avoided that, as we would all have known what products were being talked about.

I had no difficulty obtaining a list of weed and feed products from the CFIA. I am sure it would have equally easy or even easier for the PMRA to request such a list. How can the PMRA make proper estimates of use and exposure without even knowing how many products are involved or what they contain? I find that absolutely unacceptable. There are 203 products listed, 142 containing 2,4-D. All but 2 (140/142) were mixed with mecoprop or mecoprop-dicamba. As the form of the products was not listed, I had to deduct it from the manufacturing concentrates. I concluded that the 140 mixture products are all mixed amines and that most were mixed with racemic mecoprop.

92 % of all 2,4-D products licensed for turf are composed of mixed amines, meaning they all contain DEA, which was specifically excluded form the current assessment. I would like to note that, although some of your licensed manufacturing concentrates indicate DMA, the associated CAS number is the one for the mixed products and not the DMA CAS. All PMRA end-use products also indicated mixed amines for the composition, and not DMA. I therefore used that data to conclude that all amine products licensed for turf currently on the market contain DEA as well. If this is not correct, then the CAS number should be changed.

Furthermore, 86.6% (195/225) of all 2,4-D products registered for turf are mixed with mecoprop, and 78.2 % of all 2,4-D turf care products on the Canadian market are mixed with racemic mecoprop, which industry chose not to re-register last year because of the lack of safety data. It will in time be replaced by mecoprop-p.

Considering that DEA is specifically excluded form this assessment, and that most products are mixed with racemic mecoprop, which does not have safety

data, and which was also implicated in the IBT scandal[16], the PMRA is erring in making a statement of safety in your Feb 21st, 2005 media release. You have no basis in fact to make it.

*"Combinations of 2,4-D, mecoprop and dicamba, especially at a ratio of 2:1:0.1, have been shown to be synergistic" ( p. 36, value)*

Here you clearly note that this pesticide mixture is synergistic, and yet there is not a word about, or even a theoretical attempt at evaluating increased health and environmental risk resulting from that synergy. Neither is there a safety factor added to account for a potential synergy.

This is no more acceptable than not considering aggregate exposure to organophosphates when we know that human body burden includes many of them at once.

This kind of data is essential , especially for products used in combination.

**p.36 (30) Value**

"With respect to weed control spectrum, there is no real benefit in replacing 2,4-D with another phenoxy herbicide like MCPA [(4-chloro-2-methylphenoxy) acetic acid, CAS chemical name], by virtue of the fact that their properties are so similar. Aside from MCPA, which is not coformulated with 2,4-D, no domestic or commercial class herbicide that has a weed control spectrum equivalent to 2,4-D is currently registered in Canada for use on fine turf."

"2,4-D is efficacious against certain problematic broadleaf weeds on turf and there are no alternative herbicides to phenoxyalkanoic and benzoic acid herbicides on turf."

That's it? Very poor value section, and only 3 references… Why not assess the potential of corn gluten and other alternatives approaches in replacing 2,4-D? Benbrook explains that it has not been a habit to do so.[14] Is it even your mandate? Are you still working under the old Act or the new 2002 one?

In spite of close to 1/3 of the Canadian population now being protected by pesticide bylaws, the PMRA does not even attempt to question the hidden assumption that a water, fertilizer and pesticide hungry lawn is what Canadians want, or is what is desirable under a new sustainability framework.

*"Considering that weed control on turf is important, it is concluded that 2,4-D on turf has value."* P. 37

This quote says it all. What science can compete with such a value judgment? . Why can you not analyse the benefits like you do the risks?[14]

A lawn is not a crop field producing food. Industry's allegations that a lawn prevents injuries has been investigated and the only reference found to injury

rates was one study of a lawn compared to a bare and maybe rocky as well, area. If industry truly has studies indicating a link between weeds and injuries, demand that they make it public so we can all judge it. Similarly, industry claims that grass cleans the air, and produces oxygen equally apply to any vegetation, even poison ivy. These claims should have no value in judging whether 2,4-D is necessary, until the "advantages" of grass are adequately compared to other common lawn species. For all I know, we may conclude that lawn is wanting. A recent CMHC study certainly concludes that it is easy to spend as much as 50 per cent less time, 85 per cent less money and 100 per cent less water and pesticides with a natural lawn or other landscaping options than with a conventional lawn.[65]

*"The PMRA announced the launch of a label improvement program for all 2,4-D products in 1994. At that time, the maximum label rate for turf was adjusted to 2.24 kg a.e./ha under the PMRA's efforts to harmonize rates with the USEPA. A review of the recommendations for the application rates for turf and lawn on the labels of currently registered products containing 2,4-D indicated that the minimum effective rate is lower than the current maximum of 2.24 kg a.e./ha. The Industry Task Force II on 2,4-D Research Data supports an overall rate reduction from 2.24 to 1.75 kg a.e./ha for all applications to turf including granular and liquid formulations."*

So the PMRA already decreased the application rate in 1994, which mean that the 'safe' application rate recommended before was no longer 'safe" after. Now you decrease again, which makes yesterday's application rate 'unsafe" by definition. When you get the DEA data, today's application rate may also be become 'unsafe" as you may have to mitigate some more.

And how long will it take before the labels are changed to the new application rates and other information? Will people be using 2,4-D 'safely' in the meantime?

How can you feel justified using the illegal term 'safe' when the reason your regulations prohibit it is so people do not interpret it as meaning product safety?

The PMRA has to recant the safety statement made on February 21, 2005.

### 8.2.2 Label statements relating to health

The label text of **Commercial Class** products containing 2,4-D must include the following text:

**Toxicological Information**

2,4-D may cause severe irritation to the eyes. Prolonged breathing of 2,4-D may cause coughing, burning, dizziness or temporary loss of muscle coordination. Other possible effects include fatigue, muscle weakness or nausea. Treat symptomatically.

[65] CMHC; About Your House fact sheet on low-maintenance lawns; www.cmhc.ca

In my extensive experience, the first step in treating any effects is to strip and wash. I usually do it twice with cold soapy water, then have a hot bath.

The label should include remove all clothing as soon as possible, launder or throw away, and wash body entirely as a first step. Exposure continues, with worsening symptoms, until the clothing is removed and the body is washed.

**Table 9.1.1.1 Chemistry data requirements**
**Registration number Required chemistry data—Data codes (DACOs)**

The fact that these studies have not yet been evaluated should preclude any sfety statement, unless you do not intend to consider the results in your assessment.

By all means, these studies have to be submitted, but there are many others that also need to be done and that the PMRA is not yet mandating such as endocrine- disruption, immune, mixtures, etc)

**9.3 Data requirements relating to occupational and bystander exposure**
All registrants must either gain access to data of the Broadleaf Turf Herbicide Task Force and the ORETF, or provide equivalent data.

As mentioned earlier in PACR2005-01, the ORETF data has not yet been evaluated by the EPA. The exposure data is therefore still incomplete, which leads to more assumptions to plug into equations, instead of measurements, resulting in potentially inaccurate exposure standards.

"The PMRA has carried out an assessment of the available information and has concluded that the use of 2,4-D and associated EPs to treat lawns and turf **does not entail an unacceptable risk of harm to human health or the environment, provided the mitigation measure recommended in this document is adopted**. Standard precautionary statements and label improvements are also recommended."

I strongly disagree with this statement for all the reasons mentioned above including that 92 % of turf products contain DEA, that over 80 % are used in mixtures identified as synergistic but not evaluated as such, that most mixtures contain racemic mecoprop for which the health data is incomplete, that all turf use products contain formulants, 7 of which are on PMRA list 2 and 3 on list 4B, and about 70 % of which are already recognized as toxins in the US.

As safety statements are disallowed under the PCP regulations and directive **because people may misinterpret it as meaning personal safety**, the PMRA

is guilty of misleading the Canadian public by making such a statement in the February 21 media release.

Add to these arguments the following:

1. that 2,4-D was found to be a risk by the International Agency for Research on Cancer. The new PMRA document did not conclude that 2,4-D is not a carcinogen. Child cancer merited more study, but this was considered too difficult. Furthermore, the US EPA ‘s new cancer risk assessment concluded that children 2 years old and younger might be 10 times more vulnerable than adults to toxins. There is significant doubt, and the Precautionary Principle should prevail. The Canadian Cancer Society is also recognizing that pesticides cause some cancers and is calling for a cosmetic ban.

2. The PMRA has stated that 2,4-D is 'safe' IF 'label directions are followed'. This means that we and our children are at risk of harm when label directions are not followed. Hardly any monitoring takes place in Canada, and surveys and studies indicate that labels are mostly not followed. The Precautionary Principle would dictate that 2,4-D not be used. We have already seen that most professional applicators do not protect themselves adequately while applying pesticides on lawns.

3. In the manufacture of 2,4-D, dioxins are potentially formed. Dioxins are persistent, bioaccumulative and carcinogenic. Since no check of 2,4-D products has been done since 1983, since the manufacturers have not yet submitted the mandated dioxin data, and since the data required by the PMRA do not include the dioxins most likely to be present (2 and 3 chlorine ones), there is no guarantee they are not present in consumer products without regular monitoring, which the PMRA indicates they are not willing to mandate.
4. No testing is carried out for herbicide related dioxins in sediments in waterways adjacent to 2,4-D use.
5. The stink that emanates from storage or use of lawn pesticides is the smell of the break-down product 2,4-dichlorophenol, which is very toxic. This chemicals is not even mentioned in the review of 2,4-D.
6. Important gaps in relevant information exist, for example, reproduction and neurotoxicity studies required by the PMRA were not submitted, and cancer in children merited study but was not considered.
7. 2,4-D has been found in blood, urine and semen. 2,4-D and has been linked to neurological impairment and to reproductive risk. It is also found in 1/6 of the children whose parents treated their lawns, and in 1/6 of the US population.
8. From an environmental point of view, the use of 2,4-D on lawns is an ecological aberration because it favours monocultures that are very vulnerable to insect infestations. The CMHC identified much more sustainable practices.

I suggest that 2,4-D be banned in Canada for use on lawns, by 2006 - just as it will be in Quebec.