BC Centre for Disease Control
An agency of the Provincial Health Services Authority

Environmental Health Services
655 12th Avenue West
Vancouver, BC V5Z 4R4

Tel 604.707.2440
Fax 604.707.2441

www.bccdc.ca

Public Health Considerations on Cosmetic Use of Pesticides in BC

October 2013

Prepared by:

Environmental Health Services (EHS)
and
National Collaborating Centre for Environmental Health (NCCEH)

On behalf of:

Dr. Perry Kendall
Provincial Health Officer

Provincial Health Services Authority
Province-wide solutions. Better health.

A research and teaching centre affiliated with UBC

Contents

Definition of "Cosmetic" Use of Pesticides 1
Common Lawn and Garden Pesticide Ingredients 1
Risk Assessment Considerations 2
Exposure to Specific Cosmetic-use Pesticides 3
Toxicology and Epidemiology of Specific Cosmetic-use Pesticides 4
Effect of Bans on Cosmetic Use of Pesticides 6
Unintended Consequences of Pesticide Bans 6
Conclusions 7
References 8

Definition of "Cosmetic" Use of Pesticides

- Pesticides are intended to prevent, repel, limit, destroy, or mitigate unwanted species and include herbicides, insecticides, fungicides, algaecides, rodenticides and molluscicides.
- Approximately 5000 pesticide products have been evaluated and registered by the Canada Pest Management Regulatory Agency (PMRA) within Health Canada. The PMRA registers specific pesticide formulations for import and sale in Canada and labels pesticide products for intended use, which includes the class designations "domestic", "commercial" or "restricted". PMRA labeling requirements do not reference whether or not domestic class products are restricted for use as "cosmetic" pesticides.
- Provinces regulate where and to whom PMRA-approved pesticides can be sold, while, municipalities can regulate where and when such products can be used.
- BC Ministry of Environment defines "cosmetic use" of pesticides as "non-essential or optional use of pesticides for aesthetic purposes, such as improving the appearance of lawns, gardens, ornamental plants and other green spaces, or controlling unwanted organisms that do not pose an economic or health threat."[1]
- The *Cosmetic Pesticides Ban Act of Ontario* defines cosmetic as being "non-essential."[1] According to the Suzuki foundation, this term generally refers to pesticides used to improve the appearance of lawns, gardens, trees, and other aspects of landscaping.[2] Nova Scotia applies the *Non-essential Pesticide Control Act* to prohibit the sale of non-essential pesticides for use on lawns and ornamental plants, with some exemptions (e.g., home vegetable and fruit gardens).[3]

Common Lawn and Garden Pesticide Ingredients

- No comprehensive data on cosmetic use pesticides are available for BC since the Integrated Pest Management (IPM) Act, legislated in 2004, requires collection of information on Reportable Pesticides (having a Restricted or Commercial use label) and not Domestic class pesticides. A 2003 survey of a sample of vendors lists 56 active ingredients of domestic label pesticides sold in BC for home use (not including flea control products).[4]
- It is estimated that approximately 5% of the total annual sales of pesticides in Canada are for home and garden use.[5] Of the total volume of pesticide products registered with the PMRA, herbicides and insecticides accounted for 18.3% and 21.2%, respectively, of the domestic sector sales in 2008.[6]
- Based on 1997 US EPA data, the most common pesticide active ingredients for lawn and garden use by homeowners in the US by weight were the herbicides (1) 2,4-Dichlorophenoxyacetic acid (2,4 D) (an active ingredient of Weed 'n Feed) (2) glyphosate and other glycine derivatives (e.g., Roundup) (3) dicamba (benzoic acid) (4) mecoprop (chlorophenoxy) and the insecticides (5) diazinon (organophosphate) (6) chlorpyrifos (organophosphate) and (7) carbaryl (carbamate)[7] According to the PMRA, in 2000, the most common lawn-care chemicals in Canada (having the

largest number of end-use products) also included the phenoxy-herbicide, 2-Methyl-4-chlorophenoxyacetic acid (MCPA), and the insecticide malathion (an organophosphate).[8]

- The PMRA recently re-evaluated commonly available lawn and turf pesticides in Canada. Herbicides accepted for continued registration for domestic use are 2-4-D, MCPA, dicamba (benzoic acid) and bensulide (organophosphate). Commonly used pesticides that were discontinued for residential use as of December 2012 include the herbicide mecoprop and the insecticides carbaryl, chlorpyrifos and malathion. Diazinon was phased out in 2009.
- The PMRA's decision on glyphosate herbicide use is expected in 2014. As well, the insecticides pyrethrin (a natural product extracted from chrysanthemum flowers), pyrethroids (synthetic analogues), and their synergists are being re-evaluated but decisions on their continued registration are not anticipated until 2016.
- In Ontario, 82 active ingredients of pesticides are prohibited for lawn and garden use (Class 9) along with the sale of 295 products containing these ingredients (class 8). Ingredients banned for cosmetic use in Ontario include pesticides currently registered by the PMRA for domestic use (the herbicides 2-4-D, MCPA, dicamba, bensulide and glyphosate and the insecticides pyrethrin and some pyrethroids).[9] However, some of the active ingredients of formulations prohibited in Ontario for lawn and garden use are approved for agriculture and other commercial applications.
- 2,4-D was introduced for agricultural use in the 1940s and is formulated as a mixture of acids, salts and esters. Fertilizer-pesticide combination products for lawn and turf, which often contain 2,4-D, are no longer registered by the PMRA as of December 31, 2012.

Risk Assessment Considerations

- Determining the hazard of a particular pesticide is complex, with inherent toxicity determined by chemical properties (e.g., water solubility and field half-life which affects persistence), formulation ingredients (such as surfactants and solvents) and pharmacokinetics (absorption, distribution, metabolism and excretion in humans).
- The PMRA evaluates toxicology data submitted by manufacturers to assess long term health risks and biological mechanisms based on experimental studies on animals and cell cultures. Limitations of risk management based on toxicology include difficulties in approximating real-life exposure conditions (such as interactions with other chemicals) and extrapolation of effects observed in experimental animals treated with high doses to humans.
- Epidemiological studies are usually based on observations of occupational groups such as pesticide manufacturers, agricultural workers and professional pesticide applicators and the findings may not be transferable for residential exposures to lawn and plant pesticides. The lack of accurate information on specific pesticides and frequency and duration of exposures is a further limitation.

- Exposures to pesticides are affected by the mode of application, type of protective gear worn (for domestic purposes no protective wear is presumed) during handling, application and clean-up, as well as the amount absorbed through the skin and weather conditions.
- Routes of exposure include inhalation, ingestion and dermal contact during application or container handling and by ingestion or dermal contact with residues. Exposure to household residents also may occur through track-in and take-home exposures from parks and neighboring lawns by residents (particularly when not removing shoes) and their pets and by drift from outdoors.
- An important concern is the vulnerability of children at all stages of development to adverse health effects from pesticides due to behavioural factors affecting exposure (hand and mouth behaviour and playing close to the ground) and developmental factors (e.g., immature immune and nervous systems) affecting toxicity.[10]
- A prospective epidemiological study is now underway in the US to assess environmental influences, including pesticide exposure, for 100,000 children followed from early pregnancy to age 21.[11] Health Canada is mounting a 5 year study titled "Maternal-Infant Research on Environmental Chemicals (MIREC)" involving 2000 pregnant women to determine the extent of exposure during pregnancy and the post-natal period through bio-monitoring for organophosphate insecticides, among other chemicals.[12]

Exposure to Specific Cosmetic-use Pesticides

- Overall, it is exceedingly difficult to determine from existing research what the contribution of cosmetic use of pesticides is to the population's aggregate exposure to multiple pesticides from indoor residential use and agriculture and commercial applications. Food intake is regarded as perhaps the most important contributor to population pesticide exposure overall, particularly for children.[13]
- Lu and colleagues (2009) collected spot urine samples for the detection of pyrethroid metabolites among 23 children over a one year period and found that dietary intake, residential use of pyrethroid insecticides and seasonal differences were predictors of children's exposure.[14] Exposure to pyrethroids was dependent on dietary sources throughout the year, with periodic elevations in exposure related to recent residential use, such as treatment of head lice and gardening activities.[14]
- Another study by Lu and colleagues (2001) found significantly higher levels of organophosphate metabolites in the urine of children whose parents used that class of insecticides in their flower and vegetable gardens.[15]
- Studying the effect of a particular pesticide in isolation of all other pesticides (and other contaminants) in epidemiological studies is problematic. For instance, 17 pesticide residues were analyzed in carpet, floor and tabletops of New York state homes in 2001.[16] The highest levels in urban homes were for malathion and chlorpyrifos (no longer registered by the PMRA), but low levels of the pyrethroids tetramethrin and resmethrin, as well as 2,4-D were detected.

- 2,4-D is the most common cosmetic pesticide in use. Low levels of 2,4-D were detected in more than 80% of carpet dust samples obtained in the homes of 135 preschool children in two US states.[17] After lawn application, 2,4-D was detected in indoor air and all surfaces in all 13 homes. Important factors for indoor contamination were track-in by dogs as well as by children and homeowner applicators (particularly when shoes were not removed). Re-suspension of floor dust was a major source of 2-4-D on tables and window sills and was a determinant of indoor air levels.[18]

Toxicology and Epidemiology of Specific Cosmetic-use Pesticides

Acute reactions

- The acute toxicity of some of the common herbicides (e.g., 2,4-D and glyphosate) includes irritation of the skin and eyes, gastrointestinal symptoms, and corrosive injury if swallowed. Most pesticide poisonings result from home uses and children are at greatest risk of accidental exposures which, in extreme cases, can result in seizures, coma and death particularly from organophosphate poisonings.[19]
- Exposure to pyrethrin or pyrethroid insecticides, now commonly used in homes and gardens, has been associated with contact dermatitis and allergic respiratory reactions including rhinitis and asthma.[19]

Chronic effects

- In general, there is a lack of epidemiological studies which provide evidence as to adverse health effects associated with exposure to residents from use of specific “cosmetic” pesticides.
- Chronic health effects associated with pesticides are complex and multi-factorial in aetiology and include cancer (leukemias and lymphomas), reproductive, neurological, immune system, endocrine, and respiratory effects.

Mutagenic, reproductive and developmental effects

- The majority of epidemiological studies on the effects of glyphosate exposure on reproductive health have been negative.[20] Examples of specific assessments: MCPA is not regarded as a reproductive toxicant[21] and bensulide was not found to be mutagenic based on microbial testing.[22]
- A review of 2,4-D epidemiology and toxicology concluded that there is no human evidence of associated adverse reproductive outcomes.[23] However, reproductive risks associated with exposure to 2,4,-D are suggested by some Canadian studies. An Ontario cohort study of farm women found 20 to 40% increased risks of early spontaneous abortion associated with preconception exposure to glyphosate, carbaryl and 2,4,D.[24] Of relevance to real world conditions, is that exposure to more than one type of pesticide increased the risk substantially. For example, exposure to both carbaryl and 2,4-D increased the risk of spontaneous abortion 27 times compared to the risk from carbaryl alone; however levels of exposure were higher than would be

expected from householder use.[24] Another study by these investigators found detectable levels of 2,4-D in the semen of half of a small sample of farmers who had recently used the herbicide.[25] Ecological studies have found patterns of higher birth defect rates in infants born to residents living adjacent to farms which were using high volumes of 2,4-D and MCPA; although this type of study design is weak, the results suggest the need for further research.[26] The US EPA summarized the results of animal studies assessing teratogenic effects of prenatal 2,4-D exposure by ingestion. While positive effects were shown in some rat exposure studies, those involving mice and rabbits were negative.[27]

- 2,4-D is considered to be hormonally active and associated with endocrine disrupting ability as demonstrated in occupational health studies. For example, elevated luteinizing hormone which increases testosterone levels was found in the serum of male forest pesticide applicators after 2,4-D spraying.[28] An indication of human immunosuppressive effects was the temporary reduction in various immune cell populations and diminished proliferation of lymphocytes upon mitogen stimulation in a small study of farmers exposed to commercial formulations of 2,4-D and MCPA.[29]
- Exposure to pyrethrins and piperonyl butoxide (PBO), a synergist used in most pyrethroid formulations, was evaluated for developmental effects. Higher prenatal exposure to PBO (pyrethrins were too difficult to measure) was associated with lower Bayley scales of infant development.[30]

Carcinogenicity

- A recent review of cohort and case-control studies evaluating the carcinogenic potential of glyphosate concluded that the associations between any site-specific cancer and exposure to glyphosate were inconsistent.[31] The majority of cohort studies involved pesticide applicators, while case-control studies were either in agricultural or residential settings. Among the few positive studies was the finding of an increased risk of non-Hodgkin's lymphoma (NHL) among Swedish residents exposed to glyphosate; further statistical adjustment in this study for additional exposure to other pesticides reduced the association.[32]
- The US EPA categorizes 2,4-D as "Group D", which is applied when the assessment of the evidence is inadequate and cannot be interpreted as indicating the presence or absence of a cancer effect.[27]
- A recent analysis of the case-control study of male residents from 6 Canadian provinces found that the odds ratio of 2,4-D exposure with NHL was slightly elevated, although not statistically significant (OR 1.2, 0.98-1.65); whereas NHL was increased in relation to exposure to phenoxy herbicides in general (OR 1.45, 1.13-1.87) and to mecoprop in particular (OR 2.26, 1.54-3.31). No relationship was found with MCPA.[33]
- On the contrary, a US population-based case-control study found no excess risk of NHL from herbicide use on the lawn and garden (adjusted OR 1.02, 95% CI 0.84-1.23) and no relationship with greater duration, frequency or total number of applications, as obtained by interview. Carpet dust levels of 2,4-D were detected equally in 78% of homes of cases and of controls, while dicamba was detected in 15% of cases and even a greater percentage (20%) of controls.[34]

- Maternal exposure during pregnancy to residential pesticides (particularly insecticides, but also herbicides) has been associated with childhood leukemia.[35] Although a review of pesticides and childhood cancers found the majority of studies to be negative, in one study an increased risk of childhood brain tumours was associated with exposure to garden insecticides and herbicides during infancy.[36]

Effect of Bans on Cosmetic Use of Pesticides

- Cosmetic pesticide bans can be regarded as an initial step to minimize exposure to pesticides as there are other and perhaps more influential sources of pesticide exposure to the population, such as indoor applications to control pests and consumption of food containing residual pesticides.
- Reductions in use of pesticide products or changes in pest control practices have been demonstrated after a pesticide ban.[37,38] The implementation of sale restrictions in the province of Quebec in 2006 was followed by a consistent decline in household use of pesticides in lawns and gardens.[39] According to the Statistics Canada "Household and the Environment Survey" the percentage of households with a lawn or garden that used chemical pesticides in Quebec decreased from 15% in 2006 to 4% in 2007.[40,41] This is in contrast to respective yearly values of 29% and 25% for British Columbia.[40,41]
- An analysis of Ontario water samples just prior to and after the 2009 cosmetic pesticides ban showed a significant reduction in median concentrations averaging 65% for 2,4-D, dicamba and MCPP in at least one half of the ten urban streams sampled.[42]
- Demonstrating changes in bio-indicators of pesticide exposure (e.g., urine or blood measurements of residues) associated with changes in patterns of cosmetic pesticide use would provide better evidence of reduction in exposure attributable to the ban.
- The recent report by the BC Special Committee on Cosmetic Pesticides concluded there was insufficient scientific evidence to support a provincial ban on the sale of domestic label active pesticide ingredients and compounds for cosmetic use.[5]

Unintended Consequences of Pesticide Bans

- Concerns have been expressed regarding economic repercussions (especially to the lawn-care industry). Contrary to the concerns expressed by lawn care companies in reaction to Toronto's 2003 bylaw to reduce cosmetic pesticides, the percentage of landscaping and lawn care sector businesses increased in Toronto by 30% between 2001 and 2006, a rate similar to the rest of Ontario.[36]
- An unintended consequence would be the use of unregulated toxic pesticides obtained from other jurisdictions or by using pesticides registered for farming or other non-domestic uses. Anecdotally, the CBC reported that a New York farm supply owner commonly sold pesticides to Canadian cross-border shoppers interested in maintaining weed-free lawns.[43]

- A lack of control of invasive weeds is a concern, especially where these weeds migrate from pesticide restricted areas to farms. In recognition of this potential problem, three provinces with pesticide bans (Quebec, Ontario and Nova Scotia) allow exceptions for specific pesticides used for the control of invasive pests and weeds in residential areas.[5]
- Cultural practices (i.e. mowing, fertilizing, irrigation, cultivation, planting and selection of grass varieties) have been shown to be cost-effective in controlling weeds; particularly as many varieties have become resistant to herbicides.[44]
- Gardening activities are considered to be therapeutic, offering a sense of achievement, satisfaction and aesthetic pleasure particularly for older people.[45] It is possible that physical shortcomings may affect the pleasure derived from gardening, particularly for manual weeding. Although there are no estimates of the risk of injury to residents from specific domestic garden work activities such as manual removal of weeds, occupational health studies of nursery workers have identified pruning of plants and weeding as activities involving excess trunk flexion and subsequent lower back pain.[46]
- Violation of powerful societal norms of well-kept neighborhoods (characterized by the "industrial lawn" which is weedless, green, with conventional grass species only)[47] may result in social conflict and reduced property values. However, a widespread provincial ban would mitigate individual responsibility for the aesthetics of lawn care.[48]

Conclusions

- The impact on population health of exposure to pesticides used specifically for cosmetic purposes is difficult to quantify.
- There is poor quality of evidence pertaining to the direct health impacts associated with exposure of residents to pesticides used for cosmetic purposes.
- Acute and long-term toxicity has been demonstrated for many of the common pesticides used for cosmetic purposes, acutely in documented poisonings, and long-term, typically in studies of experimental animals, applicators or farm families exposed at levels well above those associated with cosmetic applications.
- Relative exposure to the active ingredients of cosmetic pesticides used in lawns and gardens compared to exposure to the same agents used indoors, in agriculture and commercially, is not well characterized, but likely is small.
- Possible harms resulting from a provincial ban of cosmetic pesticides may be the illegal use of toxic pesticides and musculoskeletal injuries among householders using manual methods to remove weeds.
- With regard to provincial public health actions, children are particularly vulnerable to exposure and effects of toxins at all stages of development and would most likely benefit from measures to reduce exposures to pesticides from any source.

References

1. Ontario Ministry of Environment. Bill 64. Cosmetic Pesticides Ban Act. Chapter 11. An Act to amend the Pesticides Act to prohibit the use and sale of pesticides that may be used for cosmetic purposes. MOE.(2008). Available from: http://www.e-laws.gov.on.ca/html/source/statutes/english/2008/elaws_src_s08011_e.htm.
2. David Suzuki Foundation staff. Highlights of Ontario's cosmetic pesticide ban. Vancouver, BC: David Suzuki Foundation; 2012. Available from: http://www.davidsuzuki.org/issues/health/science/pesticides/highlights-of-ontarios-cosmetic-pesticide-ban/.
3. Nova Scotia Environment. Lawns and ornamental gardens - non-essential pesticides. Halifax, NS: Government of Nova Scotia; [cited 2012 Dec 4]; Available from: http://www.gov.ns.ca/nse/pests/non-essential.pesticides.as6YDU789p\.
4. ENKON Environmental Limited. Survey of pesticide use in British Columbia: 2003. Surrey, BC: Prepared for Environment Canada and the British Columbia Ministry of the Environment; 2005 Oct. Available from: http://www.env.gov.bc.ca/epd/ipmp/publications/tech_reports/pest_survey_03.pdf.
5. British Columbia Special Committee on Cosmetic Pesticides. Report of the Special Committee on Cosmetic Pesticides. Victoria, BC: Legislative Assembly, Province of British Columbia; 2012 May. Available from: http://www.leg.bc.ca/cmt/39thparl/session-4/cp/reports/PDF/Rpt-CP-39-4-Report-2012-MAY-17.pdf.
6. Health Canada. Pest control products sales report 2007-2008. Ottawa, ON: Health Canada; 2008 [cited 2013 Oct 27]; Available from: http://www.hc-sc.gc.ca/cps-spc/pubs/pest/_corp-plan/sales-2007-2008-ventes/index-eng.php.
7. Aspelin A, Grube A. Pesticides industry sales and usage: 1996 and 1997 market estimates. Washington, DC: U.S. Environmental Protection Agency, Office of Pesticide Programs; 2000. Available from: http://www.epa.gov/opp00001/pestsales/97pestsales/market_estimates1997.pdf.
8. Health Canada. Re-evaluation note: re-evaluation of lawn and turf uses of pesticides. Ottawa, ON: Health Canada, Consumer Product Safety; 2000 Jun. Available from: http://www.hc-sc.gc.ca/cps-spc/pubs/pest/_decisions/rev2000-04/index-eng.php.
9. Ontario Ministry of Environment. Pesticides Act. Toronto, ON: MOE; 2012 [updated May]; Available from: http://www.ene.gov.on.ca/environment/en/legislation/pesticides_act/index.htm.
10. Landrigan PJ, Goldman LR. Protecting children from pesticides and other toxic chemicals. J Expo Sci Environ Epidemiol. 2011 Mar-Apr;21(2):119-20.
11. Landrigan PJ, Trasande L, Thorpe LE, Gwynn C, Lioy PJ, D'Alton ME, et al. The National Children's Study: a 21-year prospective study of 100,000 American children. Pediatrics. 2006 Nov;118(5):2173-86.
12. Health Canada. Maternal-Infant Research on Environmental Chemicals (The MIREC Study). Ottawa, ON: Health Canada; 2010 [cited 2012 Nov 22]; Available from: http://www.hc-sc.gc.ca/ewh-semt/contaminants/human-humaine/mirec-eng.php.
13. Roberts JR, Karr CJ, Council On Environmental Health. Pesticide exposure in children. Pediatrics. 2012 Dec;130(6):e1765–e88.
14. Lu C, Barr DB, Pearson MA, Walker LA, Bravo R. The attribution of urban and suburban children's exposure to synthetic pyrethroid insecticides: a longitudinal assessment. J Expo Sci Environ Epidemiol. 2009 Jan;19(1):69-78.

15. Lu C, Knutson DE, Fisker-Andersen J, Fenske RA. Biological monitoring survey of organophosphorus pesticide exposure among pre-school children in the Seattle metropolitan area. Environ Health Perspect. 2001 Mar;109(3):299-303.
16. Obendorf SK, Lemley AT, Hedge A, Kline AA, Tan K, Dokuchayeva T. Distribution of pesticide residues within homes in central New York State. Arch Environ Contam Toxicol. 2006 Jan;50(1):31-44.
17. Morgan MK, Sheldon LS, Thomas KW, Egeghy PP, Croghan CW, Jones PA, et al. Adult and children's exposure to 2,4-D from multiple sources and pathways. J Expo Sci Environ Epidemiol. 2008 Sep;18(5):486-94.
18. Nishioka MG, Burkholder HM, Brinkman MC, Lewis RG. Distribution of 2,4-D acid in floor dust throughout homes following homeowner and commercial lawn applications: Quantitative effects of children, pets, and shoes. Environ Sci Technol. 1999;33:1359-65.
19. Karr CJ, Solomon GM, Brock-Utne AC. Health effects of common home, lawn, and garden pesticides. Pediatr Clin North Am. 2007 Feb;54(1):63.
20. Mink PJ, Mandel JS, Lundin JI, Sceurman BK. Epidemiologic studies of glyphosate and non-cancer health outcomes: a review. Regul Toxicol Pharmacol. 2011;61(2):172-84.
21. Bellet EM, van Ravenzwaay B, Hellwig J, Pigott G. Reproductive toxicity of MCPA (4-chloro-2-methylphenoxyacetic acid) in the rat. Int J Toxicol. 2001;20(1):29-38.
22. Anderson KJ, Cutaia AJ. Evaluation of bensulide for mutagenic properties in microbial test system. J Agric Food Chem. 1972;20(3):656-8.
23. Garabrant DH, Philbert MA. Review of 2,4-dichlorophenoxyacetic acid (2,4-D) epidemiology and toxicology. Crit Rev Toxicol. 2002 Jul;32(4):233-57.
24. Arbuckle TE, Lin Z, Mery LS. An exploratory analysis of the effect of pesticide exposure on the risk of spontaneous abortion in an Ontario farm population. Environ Health Perspect. 2001 Aug;109(8):851-7.
25. Arbuckle TE, Schrader SM, Cole D, Hall JC, Bancej CM, Turner LA, et al. 2,4-Dichlorophenoxyacetic acid residues in semen of Ontario farmers. Reprod Toxicol. 1999 Nov-Dec;13(6):421-9.
26. Garry VF, Schreinemachers D, Harkins ME, Griffith J. Pesticide appliers, biocides, and birth defects in rural Minnesota. Environ Health Perspect. 1996 Apr;104(4):394-9.
27. U.S. Environmental Protection Agency. 2,4-Dichlorophenoxyacetic Acid (2,4-D). Chemical summary. Toxicity and Exposure Assessment for Children's Health. Washington, DC: EPA; 2007. Available from: http://www.epa.gov/teach/chem_summ/24D_summary.pdf.
28. Garry VF, Tarone RE, Kirsch IR, Abdallah JM, Lombardi DP, Long LK, et al. Biomarker correlations of urinary 2,4-D levels in foresters: genomic instability and endocrine disruption. Environ Health Perspect. 2001 May;109(5):495-500.
29. Faustini A, Settimi L, Pacifici R, Fano V, Zuccaro P, Forastiere F. Immunological changes among farmers exposed to phenoxy herbicides: preliminary observations. Occup Environ Med. 1996 Sep;53(9):583-5.
30. Horton MK, Rundle A, Camann DE, Boyd Barr D, Rauh VA, Whyatt RM. Impact of prenatal exposure to piperonyl butoxide and permethrin on 36-month neurodevelopment. Pediatrics. 2011 Mar;127(3):e699-706.
31. Mink PJ, Mandel JS, Sceurman BK, Lundin JI. Epidemiologic studies of glyphosate and cancer: a review. Regul Toxicol Pharmacol. 2012;63(3):440-52.

32. Eriksson M, Hardell L, Carlberg M, Akerman M. Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis. Int J Cancer. 2008 Oct 1;123(7):1657-63.

33. Pahwa M, Harris SA, Hohenadel K, McLaughlin JR, Spinelli JJ, Pahwa P, et al. Pesticide use, immunologic conditions, and risk of non-Hodgkin lymphoma in Canadian men in six provinces. Int J Cancer. 2012 Dec 1;131(11):2650-9.

34. Arya N. Pesticides and human health. Can J Public Health. 2005 MAR-APR;96(2):89-92.

35. Turner MC, Wigle DT, Krewski D. Residential pesticides and childhood leukemia: a systematic review and meta-analysis. Environ Health Perspect. 2010 Jan;118(1):33-41.

36. Daniels JL, Olshan AF, Savitz DA. Pesticides and childhood cancers. Environ Health Perspect. 1997 Oct;105(10):1068-77.

37. Cole DC, Vanderlinden L, Leah J, Whate R, Mee C, Bienefeld M, et al. Municipal bylaw to reduce cosmetic/non-essential pesticide use on household lawns - a policy implementation evaluation. Environ Health. 2011;10:74.

38. Whyatt RM, Garfinkel R, Hoepner LA, Holmes D, Borjas M, Williams MK, et al. Within- and between-home variability in indoor-air insecticide levels during pregnancy among an inner-city cohort from New York City. Environ Health Perspect. 2007 Mar;115(3):383-9.

39. Environment Canada. Household use of chemical pesticides and fertilizers. Gatineau, QC: Environment Canada; 2012. Available from: http://www.ec.gc.ca/indicateurs-indicators/default.asp?lang=en&n=258BC62B-1#wq_fertpest_en.

40. Statistics Canada. Households and the environment 2006. Ottawa, ON: Statistics Canada; 2006 Feb. Available from: http://www.allianceforwaterefficiency.org/WorkArea/DownloadAsset.aspx?id=2530.

41. Statistics Canada. Households and the environment 2007. Ottawa, ON: Statistics Canada; 2009 Jul. Available from: http://www.statcan.gc.ca/pub/11-526-x/11-526-x2009001-eng.pdf.

42. Todd AK. Changes in urban stream water pesticide concentrations one year after a cosmetic pesticides ban. Toronto, ON: Ontario Ministry of the Environment, Environmental Monitoring and Reporting Branch; 2010 Nov. Available from: http://www.ene.gov.on.ca/stdprodconsume/groups/lr/@ene/@resources/documents/resource/stdprod_080108.pdf.

43. Paris M. Gardeners cross-border shop for illegal pesticides. CBC News, Environment Unit. 2011 May 19. Available from: http://www.cbc.ca/news/politics/story/2011/05/19/pol-pesticides-sneaky.html.

44. Hernke MT, Podein RJ. Sustainability, health and precautionary perspectives on lawn pesticides, and alternatives. Ecohealth. 2011 Jun;8(2):223-32.

45. Milligan C, Gatrell A, Bingley A. "Cultivating health": therapeutic landscapes and older people in northern England. Soc Sci Med. 2004;58(9):1781-93.

46. Singh S, Arora R. Ergonomic intervention for preventing musculoskeletal disorders among farm women. J Agric Sci. 2010;1(2):61-71.

47. Tekle AM. Lawns and the new watershed law. Marquette Law Review. 2012;95:213-42.

48. Hirsch R, Baxter J. The look of the lawn: pesticide policy preference and health-risk perception in context. Environ Plann C: Government Pol. 2009;27(3):468-90.