Comments of Beyond Pesticides on Hazards of Pesticide Use
To Highland Park, IL Park Board
October 14, 2011

The support for strict adherence to natural organic turf management is driven by scientific findings of adverse effects associated with pesticide use as well as a high degree of uncertainty or unknown impacts related to chemical interactions, low-dose exposure, specific toxic endpoints or health effects, vulnerable population groups (such as children and those with existing illnesses like asthma), and synergistic effects of chemical mixtures. The identified hazards and uncertainties linked to pesticide use have formed the basis of the precautionary approach that is embodied in the historic approach that Highland Park has taken for turf management. I believe that it is irresponsible to suggest that product registration by EPA creates adequate assurance that the residents of Highland Park are adequately protected, given the known hazards and uncertainties, and in light of the availability of alternative organic practices. The vulnerability of children to pesticides is well-established, as are the problems of exposure to mixtures that are not assessed. This issue is discussed in detail in Beyond Pesticides' factsheet *Children and Pesticides Don't Mix,*[1] which cites peer-reviewed scientific literature on the health effects of pesticides.

With respect to the specific pesticides that are used, I have attached below a summary of chemical concerns among some of the pesticides that are or may be used:

Clopyralid
Clopyralid is a carboxylic acid herbicide classified by EPA in acute toxicity class III as slightly toxic. Laboratory studies have shown that clopyralid is a severe eye irritant,[2,3,4] and dermal irritation has also been noted which can lead to skin sensitization for prolonged skin exposures.[5] Some developmental and reproductive effects have been observed in laboratory animals. The livers and kidneys of rats as well as the livers of dogs were affected by changes in weight and decreased red blood cell counts.[6] Another study found that weights of rabbit fetuses decreased at both low and high doses of clopyralid. Skeletal abnormalities were also observed in these fetuses at all doses.[7] Clopyralid is persistent in soil, with a recorded half-life of 2-14 months,[8] and is degraded primarily by microbial activity. However, it does not readily bind to soil and is highly soluble in water. As a result, it is very mobile in soil and has the potential to move towards groundwater and contaminate surface water. Persistence of clopyralid in vegetation was evidenced by the contamination of composts from harvested lawn and grass clippings sprayed with clopyralid which severely damaged garden crops in Washington State in 2000. In 2002, the state banned the use of clopyralid on lawns and turf in order to keep the chemical from contaminating compost

[1] Beyond Pesticides, *Children and Pesticides Don't Mix*, http://www.beyondpesticides.org/lawn/factsheets/Pesticide.children.dontmix.pdf
[2] Cox, C. 1998. *Clopyralid - Herbicide Fact Sheet*. Journal of Pesticide Reform. 18(4).
[3] OSU Extension Service. 2002.*Clopyralid- Pesticide Fact Sheet: Forestry Use.*
[4] Tu, M., et al. 2001. *CLOPYRALID*. Weed Control Methods Handbook. The Nature Conservancy.
[5] Cox, C. 1998. *Clopyralid - Herbicide Fact Sheet*. Journal of Pesticide Reform. 18(4).
[6] *Ibid.*
[7] *Ibid.*
[8] Washington State University Cooperative Extension. *Clopyralid Herbicide and Compost*. Agriculture and Natural Resources Fact Sheet #538.

supplies.[9] That same year, California found that 65% of the composts samples tested positive for clopyralid, which led to the cancellation of residential uses for clopyralid in the state.[10]

Triclopyr

Triclopyr is a pyridine compound herbicide which mimics plant growth hormones called auxins, interfering with the normal plant growth response. It is readily absorbed through both roots and leaves, and is systemic, translocating throughout the plant. Triclopyr is classified as slightly toxic (class III) by EPA, though there are documented effects on reproduction, fetal development, and organ function, as well as irritation when exposed to high doses. The substance is acutely toxic to the eyes, causing severe irritation.[11] Subchronic and chronic feeding studies in dogs and rodents found damage to both the kidney and liver.[12] Data has also shown birth defects and adverse effects on reproduction associated with chronic exposure. Pregnant rodents exposed to the chemical had lower litter numbers and a higher incidence of birth abnormalities in offspring than did those which were not exposed.[13] Triclopyr is generally considered to be non-carcinogenic, but several laboratory tests have shown an increase in the incidents of breast cancer and genetic damage in rat embryos.[14] Triclopyr is also toxic to a range of wildlife, and its persistence can vary greatly depending on the application site, but some studies have shown it to persist longer than a year under certain conditions.[15]

2,4-D

2,4-D is one of the most widely used herbicides for the control of broadleaf weeds in commercial agriculture and residential landscapes in the U.S. About 46 million pounds of 2,4-D are used annually, with 16 million pounds used in non-agricultural settings, including parks, playing fields, and residential lawns[16]. Its health risks prompted a Special Review in 1985. A few years later in a unique move, several large pesticide companies with a common interest in keeping 2,4-D on the market formed a task force to keep the herbicide on the market. In 2007, EPA reversed its decision did not complete the Special Review[17]. In 2008, the Natural Resources Defense Council petitioned EPA to cancel all uses of 2,4-D. Once part of the deadly duo of chemicals that made up Agent Orange, can also be contaminated with several forms of dioxin, including 2,3,7,8-TCDD, a known carcinogen. Studies have also documented that once tracked indoors from lawns, 2,4-D can stay indoors (on carpets) for up to a year.[18,19]

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[9] WSDA. 2002. *Clopyralid in Compost Facts: Herbicide contamination raises problems for compost*.

[10] de la Fuente, M. *Clopyralid and Compost in California*. University of California Cooperative Extension.

[11] C. Cox. 2000. *Triclopyr: Herbicide Fact Sheet.* Journal of Pesticide Reform 20(4).

[12] U.S. EPA. Prevention, Pesticides and Toxic Substances. 1998. Reregistration eligibility decision (RED): Triclopyr. Washington, D.C.

[13] *Ibid*.

[14] U.S. EPA. Office of Prevention, Pesticides, and Toxic Substances. 1996. Carcinogenicity peer review of triclopyr. Memo from McMahon, T.F., and E. Rinde, Health Effects Div., to R. Taylor, Registration Div. and T. Luminello, Special Review and Reregistration Div. Washington, D.C.

[15] Norris, L.A., M.L. Montgomery, and L.E. Warren. 1987. Triclopyr persistence in western Oregon hill pastures. Bull. Environ. Contam. Toxicol. 39: 134-141.

[16] USEPA. 2005 Reregistration Eligibility Decision (RED) 2,4-D Factsheet. Office of Prevention, Pesticides and Toxic Substances. Washington DC http://www.epa.gov/oppsrrd1/REDs/factsheets/24d_fs.htm

[17] Beyond Pesticides Daily News. 2007 http://www.beyondpesticides.org/dailynewsblog/?p=161

[18] Nishioka MG, Burkholder HM, Brinkman MC, Gordon SM. 1996. Measuring lawn transport of lawn applied herbicide acids from turf to home: Correlation of dislodgeable 2,4-D turf residues with carpets dust and carpet surface residues. *Environmental Sci and Tech*. 30:3313-3320.

[19] Rudel, Ruthann, et al. "Phthalates, Alkylphenols, Pesticides, Polybrominated Diphenyl Ethers, and Other Endocrine-Disrupting Compounds in Indoor Air and Dust." *Environmental Science and Technology* 37(20):4543-4553.

Several scientific studies have been presented to the agency that point to 2,4-D's association with cancer, for example non-Hodgkin's lymphoma.[20],[21] Nevertheless, the agency concluded that "the data are not sufficient to conclude that there is a cause and effect relationship between exposure to 2,4-D and non- Hodgkin's lymphoma."[22] 2,4-D was then classified as a Group D, not classifiable as to human carcinogenicity. Studies from the National Cancer Institute and other sources have reported an association between exposure to lawn chemicals, like 2,4-D, and adverse impacts in dogs.

There is also a wealth of relevant scientific information available indicating that 2,4-D has endocrine disrupting activity. A study by Garry et al. found a direct correlation of urinary levels of 2,4-D with serum levels of luteinizing hormone (LH) and high testosterone levels at the time of highest exposure suggest a direct effect on hormonal levels by the chlorophenoxy herbicide.[23] LH, produced by the pituitary gland, stimulates the production of testosterone and helps regulate the menstrual cycle and ovulation. Fluctuations in these hormones may affect human fertility. Others found that abnormal sperm[24] and higher rates of birth defects[25] were observed in farmers with long-time exposure to 2,4-D. Animal studies have also observed the hormone effects of 2,4-D exposure. Xie et al. (2005) observed estrogenic activity in rainbow trout[26] exposed to 2,4-D, while another study found the thyroid glands of laboratory rats were sensitive to 2,4-D as decreases in the thyroid gland transport and production functions, and impairment of hormone iodination in the thyroid were observed after acute exposure.[27] Other studies have found that 2,4-D promotes the proliferation of androgen-sensitive cells by acting synergistically with its main metabolite, 2,4-dichlorophenol (DCP), also known for its endocrine disrupting effects.[28,29]

Dicamba

[20] Lennart Hardell, and Eriksson, M. (1999) A case-control study of non-Hodgkin lymphoma and exposure to pesticides. *Cancer* 85, 1353-1360.

[21] Ibrahim, M.A., Bond, G.G., Burke, T.A., Cole, P., Dost, F.N., Enterline, P.E., Gough, M., Greenberg, R.S., Halperin, W.E., McConnell, E., and et al.1991. Weight of the evidence on the human carcinogenicity of 2,4-D*. Environmental Health Perspectives* 96, 213-222.

[22] U.S. EPA. 2005. Reregistration Eligibility Decision for 2,4-D. Office of Prevention Pesticides and Toxic Substances. Washington DC

[23] Garry, V.F., Tarone, R.E., Kirsch, I.R., Abdallah, J.M., Lombardi, D.P., Long, L.K., Burroughs, B.L., Barr, D.B., and Kesner, J.S. 2001. Biomarker correlations of urinary 2,4-D levels in foresters: genomic instability and endocrine disruption. *Environmental Health Perspectives* 109, 495-500.

[24] Lerda, D., and Rizzi, R. 1991. Study of Reproductive Function in Persons Occupationally Exposed to 2,4-Dichlorophenoxyacetic Acid (2,4-D). *Mutation Research* 262, 47-50.

[25] Garry, V.F., Schreinemachers, D., Harkins, M.E., and Griffith, J. 1996. Pesticide Appliers, Biocides, and Birth Defects in Rural Minnesota. *Environmental Health Perspectives* 104, 394-399.

[26] Xie, L.T., Thrippleton, K., Irwin, M.A., Siemering, G.S., Mekebri, A., Crane, D., Berry, K., and Schlenk, D. 2005. Evaluation of estrogenic activities of aquatic herbicides and surfactants using an rainbow trout vitellogenin assay. *Toxicol. Sci*. 87, 391-398.

[27] Malysheva, L.N., and Zhavoronkov, A.A. 1997. Morphological and histochemical changes in the thyroid gland after a single exposure to 2,4-DA herbicide. *Bull. Exp. Biol. Med.* 124, 1223-1224.

[28] Kim, H.-J., Park, Y.I., and Dong, M.S. 2005. Effects of 2,4-D and DCP on the DHT-Induced Androgenic Action in Human Prostate Cancer Cells. *Toxicological Sciences*. 88(1), 52–59 pp. 52-59.

[29] McKinlay, R., Plant, J.A., Bell, J.N.B., and Voulvoulis, N. 2008. Endocrine disrupting pesticides: Implications for risk assessment. *Environment International* 34, 168-183.

Dicamba, originally registered in 1967, is a benzoic acid herbicide used in agricultural, industrial and residential settings. This neurotoxic pesticide is linked to organ damage and reproductive effects.[30] A 1992 study of farmers by the National Cancer Institute found that exposure to dicamba approximately doubled the farmers' risk of contracting the cancer non-Hodgkin's lymphoma two decades after exposure.[31] Symptoms of poisoning include muscle cramps, shortness of breath, nausea, vomiting, skin rashes, loss of voice, and swollen glands. It has attracted attention because of the toxicity of its contaminants, both dioxin and nitrosamines, and its propensity to leach through soil.

Glyphosate

Glyphosate is also cause for continued concern, as it has been linked to a number of serious human health effects, including increased cancer risk,[32] neurotoxicity, and birth defects,[33] as well as eye, skin, and respiratory irritation. One of the inert ingredients in product formulations of Roundup, polyoxyethyleneamine (POEA), kills human embryonic cells.[34]

It is also of particular concern due to its toxicity to aquatic species as well as instances of serious human health effects from acute exposure. EPA acknowledges that glyphosate has the potential to contaminate surface water because it does not readily break down in water or sunlight. Due to glyphosate's potential for water contamination, the agency has established a maximum contaminant level (MCL) for glyphosate (0.7ppm).[35] The agency lists the short- and long-term health effects for drinking water exposures: for relatively short periods of time, congestion of the lungs and increased breathing rate; for lifetime exposure at levels above the MCL kidney damage and reproductive effects.

Conclusion

We cannot be dismissive of pesticide use, especially in a country where children suffer from elevated rates of asthma, learning disabilities and other health problems that can be caused or exacerbated by pesticide exposure. Beyond Pesticides' Pesticide-Induced Diseases Database[36] shows nearly 400 entries of peer-reviewed epidemiologic and laboratory studies based on real world exposure scenarios that link public health effects to pesticides. As the site says, "The scientific literature documents elevated rates of chronic diseases among people exposed to pesticides, with increasing numbers of studies associated with both specific illnesses and a range of illnesses." With some of these diseases at very high and, perhaps, epidemic proportions, there is an ongoing need for public policy in Highland Park that eliminates dependency on pesticides.

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[30] Cox, Caroline. 1994. Northwest Coalition for Alternatives to Pesticides (NCAP), Pesticide Factsheets. http://www.pesticide.org/get-the-facts/pesticide-factsheets/factsheets/dicamba

[31] Cantor, K.P. 1992. Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota. *Cancer Res.* 52:2447-2455.

[32] M. Eriksson, et. al. 2008. Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis. *International Journal of Cancer.* 123(7): 647-1663 http://onlinelibrary.wiley.com/doi/10.1002/ijc.23589/abstract

[33] M. Antoniou, et. al. 2011. "Roundup and birth defects: Is the public being kept in the dark?" *Earth Open Source.* *http://www.scribd.com/doc/57277946/RoundupandBirthDefectsv5*

[34] Benachour, et. al. 2009. "Glyphosate Forumlations Induce Apoptosis and Necrosis in Human Umbilical, Embryonic, and Placental Cells. 22(1): 97-105 http://pubs.acs.org/doi/abs/10.1021/tx800218n

[35] USEPA. 1993. Reregistration Eligibility Decision (RED) Glyphosate. Office of Prevention, Pesticides and Toxic Substances. Washington DC

[36] Beyond Pesticides, Pesticide-Induced Diseases Database, http://www.beyondpesticides.org/health/