ONTARIO Turfgrass SYMPOSIUM

Pesticide Exposure and Human Health (Part 2)

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In this article, we will address the concept of risk and the allegation that pesticide exposure is responsible for an increase in cancer incidence. Readers are encouraged to review Part One, which appeared in the June 2002 issue, for background information prior to reading this article. Part Three, which will appear in the next issue, will address the subjects of pesticide exposure and asthma, neurological affects in children, and endocrine disruption. Please note that references for the complete three part series are footnoted in the text and a detailed listing is available from the Sports Turf Association.

The Concept of Risk/Hazard

Unfortunately, a large portion of the problem we are facing today with cries from the general public for a ban on the use of pesticides for urban uses is related to misconceptions about risk. All activities in which we participate carry a certain element of risk. However, the public's perception of risk is distorted because people haven't been taught about risk. Risk perception has more to do with a combination of the frequency with which the risk is taken (familiarity with the activity), the level of control a person has over the risky situation, how much pleasure they derive from it, and an unconscious decision to accept certain risks because the benefit incurred outweighs the risk, than it does the magnitude of the risk. For example, many people feel perfectly safe driving a car but unsafe when flying; when in reality, the risk of being seriously or fatally injured in a car accident is far greater than being injured or dying in a plane crash.

Most of you reading this article probably drink coffee, drive cars, ride bicycles periodically, and enjoy the occasional alcoholic beverage. Some of you are smokers. Many of you enjoy being out in the sun and don't always wear sunscreen, and many have used a cell phone while driving. The purpose of presenting this lengthy list of activities is to point out that people take risks everyday, whether they are driving to work, crossing the street, riding a bike, smoking a cigarette or consuming alcoholic beverages. Each of these activities has a significant level of risk associated with it, and each bears a much greater health risk than you will ever incur from exposure to pesticides used on turf. Unfortunately, academia and the chemical industry have not effectively communicated the concept of risk to the general public. Putting risk into perspective is critical for the public to understand that pesticides can be used safely with minimal risk to human health.

In 1982, *Scientific American* published a paper that ranked various activities according to their annual contribution to the number of deaths in the US[1]. A listing of the top ten, in order of the most hazardous activities to the least hazardous, conveys just how distorted the public's perception of risk truly is. The most hazardous activity that a person can partake in is smoking. More people die of tobacco-related illnesses than any other cause. The remaining top 10 in order are: use of alcoholic beverages, motor vehicles, handguns, electrical power, motorcycles, swimming, surgery, x-rays and railroads. Cycling ranked 13th, fire fighting and police work ranked 16th and 17th respectively, use of contraceptives 18th, vaccinations 25th, and "pesticides" ranked 28th.

Certainly, each time a pesticide is handled there is some level of risk. "Risk" is

a function of the inherent toxicity of a substance and the exposure one has to it.

Risk = Toxicity x Exposure

Looking at this simple math equation, if the value for exposure is zero (no exposure), what happens to risk? It becomes zero. The most toxic substances known to human beings can be handled safely, with minimal risk, if there is *little or no exposure*. Alternatively, there can be considerable risk involved in handling a compound that *isn't very toxic* if exposure is high enough. By keeping one, or both, of these factors as close to zero as possible, the risk involved in handling and using pesticides can be minimized. Pesticide label directions are designed to do just that.

Occupational and Bystander Exposure

Scientists measure "exposure" and determine the amount of pesticide that is absorbed into the body, because this represents the internal dose. This internal dose is then compared to doses used in the animal studies discussed in Part One of this article. There are three main routes of exposure into the body:

- Ingestion (oral exposure)
- Contact (dermal exposure)
- Inhalation (respiratory exposure, what is breathed in)

Most occupational and bystander exposures to turf pesticides are a result of exposure through the skin (dermal exposure) or lungs (respiratory exposure).

In 1992, Harris and Solomon investigated exposure to bystanders entering areas where the turf had been treated with the commonly used herbicide 2,4-D[2]. Exposure was measured by analyzing urine for residues of 2,4-D. Exposures occurring 1 hour after herbicide application were well below health protection guidelines and 24 hours after spraying, no chemical exposures were measurable. Dislodgeable residues from the treated turf fell from 8% to 1% during that period. The "rule of thumb" is that if treated surfaces are dry, exposure is reduced and is minimal. No detectable residues were found in the urine of 20 volunteers with the exception of 3 people who were barefoot, wearing shorts and contacted turf within one hour of application (which is against label directions). *No detectable residues were found in the urine of volunteers exposed to treated turf 24 hours after application.* At recommended application rates, exposure to turf sprayed with 2,4-D should present little risk to humans. Children should never contact treated turf until it is dry, and should never be in the vicinity during application.

A second study by the same lead authors examined exposure of homeowners making their own applications of 2,4-D, and exposure of household bystanders[3]. Residues were not detected in the urine of the bystanders. The only homeowner applicators that had 2,4-D in their urine were those who failed to wear protective gear and had experienced spills of the liquid concentrate or had excessive contact with the diluted mixture (residues ranged from non-detectable to 0.0071 mg/kg bodyweight, which is very low).

In 2001, Stephenson et al. measured homeowner applicator and bystander exposure to liquid and granular (plus fertilizer) formulations of chlorpyrifos (Dursban) applied to turf[4]. Urine was collected over a 96 hour period beginning immediately after application. Of 40 bystander study participants, only 4 had trace levels of chemical metabolites detected in their urine and only 1 had residues above the lowest quantifiable concentration (25 ug/L). (Note: The term "trace" means the metabolite was detected but the quantity was so low that an accurate measurement could not be obtained.) This was expected given the chemical properties of chlorpyrifos and the fact that only 1.5 to 3% of the chlropyrifos applied is dislodgeable immediately after application and less than 0.1% is dislodgeable 1 day after application[5,6].

Only 1 of 10 applicators who wore personal protective equipment (PPE) had trace levels of metabolites in his urine following application of the granular formulation, while 3 of 10 applicators who did not wear PPE had detectable residues in their urine (1 trace and 2 quantifiable). Of 11 volunteers wearing PPE and applying the liquid formulation, 2 had trace residues and 3 had quantifiable residues in their urine. Certainly the use of protective clothing reduced the extent of exposure. (Note: Dursban is no longer registered for domestic home and garden uses, the reasons for which are beyond the scope of this article; however, commercial formulations are available for use on golf courses, industrial sites, sod farms, ornamental plantings and highway medians.)

Many people are concerned about their pets contacting treated turf. In 1991, a widely publicized study suggested a relationship between canine malignant lymphoma (CML) and exposure to 2,4-D[7,8]. The study was highly criticized by experts for its design, as well as its analysis and interpretation of the data. Unfortunately, once incorrect information is released to the media, it is very difficult to refute or correct. The study data was reanalyzed by researchers at Michigan State Veterinary College who demonstrated that the data did not confirm a dose-response relationship between 2,4-D use and CML, or even a significant association between the two[9]. Studies are not available for all pesticides used on turf; therefore, owners should keep their pets indoors during pesticide application and until the turf is dry. As can be seen from the studies discussed, very little pesticide is dislodged from treated turf, particularly 24 hours after application.

Pesticides and Cancer

There has been a growing concern that exposure to pesticides, either through food residues or when applied to home interiors, turf and gardens, may be a major cause of various types of cancer. Concern has been fuelled by some epidemiology studies of pesticide manufacturers, applicators and farmers who have had high exposures and that are suggestive of an association with certain types of cancers such as prostate cancer and Non-Hodgkin's Lymphoma (NHL).

There are many studies suggesting pesticide exposure increases cancer risk in these populations and many indicating no effect. These studies have been plagued by small sample sizes (small numbers of

study participants) which reduces the statistical power of a study, and flaws inherent in using *questionnaires* to obtain exposure data rather than actual sample analysis because the studies are "retrospective" in nature; in other words, study participants who have already been diagnosed with cancer are asked to *recall what they were exposed to 15 to 20 years earlier*. The reason that exposures 15 to 20 years earlier are important is that there is a latency period between the time the causal exposure occurs and development of the disease. For many cancers, the latency period is close to 20 years. Obtaining accurate responses on a questionnaire is extremely difficult, if not impossible. In addition, few epidemiology studies have accounted for confounding exposures to other compounds, including medications, diesel fuel, etc.

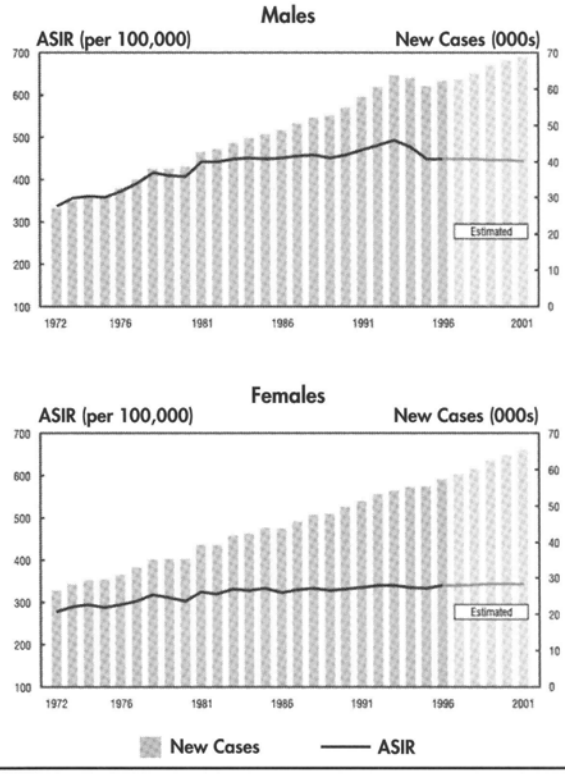
Prior to discussing the results of epidemiology studies on pesticide exposure and cancer, it is critical for the reader to understand what causes cancer and to become familiar with cancer incidence rates. Basically, cancer is caused by the failure of the body's immune system to repair mutations (damage or errors) in our DNA that in turn cause processes in the body's cells to go awry. DNA is the molecule we have in each of our cells that carries our own unique genetic code and also is responsible for cell division.

In a healthy individual, cells continually die and are replaced. Every time a cell divides and reproduces itself, there is an opportunity for an error to be made when the DNA duplicates. Every day, our bodies repair millions of mutations, most of which are naturally occurring. This is one of the roles of our amazing immune system. As we age, our DNA repair mechanisms start to falter and mutations that can lead to the growth of a tumour go uncorrected. Consequently, the *annual number of newly diagnosed cancer cases increases as the population ages*.

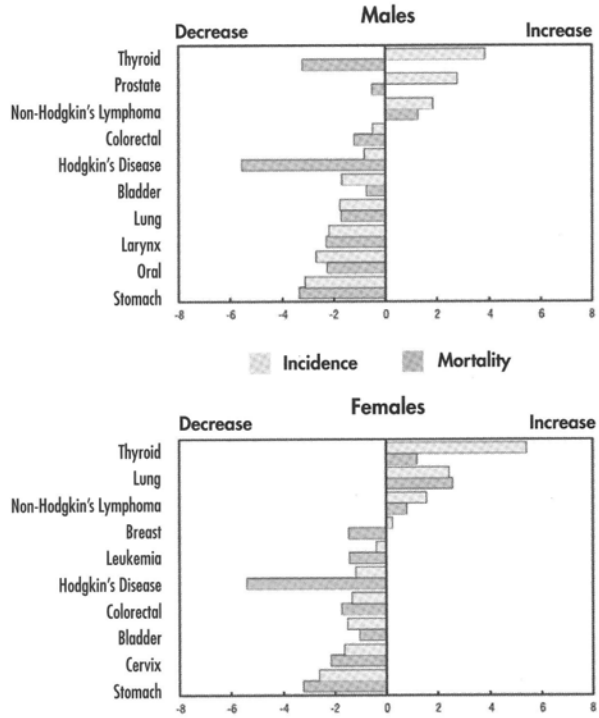
Cancer can also be caused as a result of exposure to an external stimulus that is extremely toxic to cells. If many cells are killed, the body increases the rate of cell division of the remaining cells to try and compensate for the loss. With an increase in the rate of cell division, comes an increased risk that an error will be made when duplicating the DNA. In the end, it all comes down to a failure of our immune system to repair damage to DNA, whether the mutations are caused by a chemical, UV radiation from the sun, exposure to cigarette smoke or cancer causing viruses etc.

If pesticide exposure is contributing to an increase in cancer, this should be reflected in age-adjusted cancer incidence rates over time. The graphs and data presented in this article are from "Canadian Cancer Statistics 2001" produced by the Canadian Cancer Society, the National Cancer Institute of Canada, Statistics Canada, Provincial/Territorial Cancer Registries and Health Canada[16]. You can review this information and more at www.cancer.ca. On entering the website, select Research and Statistics, then Statistics, Canadian Cancer Statistics 2001 report. (Note, the 2002 report was recently

Graphs 1 and 2. New cases and age-standardized incidence rates (ASIR) for all cancers, 1972-2001, males and females separately.



Graphs 5 and 6. Average annual percent change (AAPC) in age-standardized incidence (1989-1996) and mortality (1989-1996) rates for selected cancer sites, males and females separately.



added to the website; however, the summary in this article pertains to the 2001 report). More specific data was obtained from Cancer Surveillance On-Line http://cythera.ic.gc.ca/dsol/cancer/[17] (except where noted, see references provided).

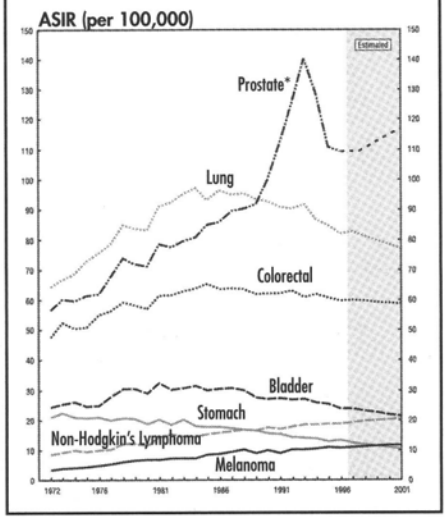
Canadian demographics are changing. The Canadian population is increasing and so is the average age of the population. As a result, the overall number of newly diagnosed cases is increasing because there are more people around to develop cancer and because the disease is more prevalent in older people.

In order to remove aging and population increases as confounding factors in cancer statistics, all cancer data is standardized for age and presented as the number of new cases (incidence), or deaths (mortality), per 100,000 of the population. This allows data to be compared from year to year without population increases and average age of the population complicating the issue. **Graphs 1 and 2** depict the effect of standardizing for age on the incidence numbers per 100,000 of the population in males and in females, respectively. These graphs also demonstrate that the age standardized incidence rate (ASIR) for all cancers combined has been relatively flat over the years.

While the data presented are from 1972 to 1996, the data prior to 1984 are not entirely accurate due to changing diagnostic criteria and inconsistencies in cancer registry reporting. Inclusion of these data gives the impression that cancer incidence was increasing during this time, which may not be true. The data from 1984 to 1996 are much more reliable. The data from 1997 to 2001 are estimated values as the actual numbers have not yet been published.

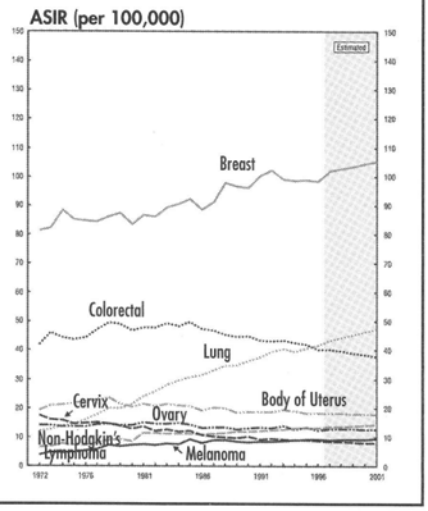
If we look at the age standardized incidence rates of various types of cancers individually (**Graphs 3 and 4**), we can see that, for most cancers, the incidence has been flat or decreasing since 1983. The only cancers for which increases appear to be occurring are thyroid (not shown on graph), lung, NHL and breast cancer in women, and NHL, thyroid (not shown on graph), and prostate cancer in men. (The increase in melanoma among older men and women will not be specifically addressed here as this is believed to be due to UV exposure.) These trends are also apparent in the average annual percent change in cancer incidence and mortality (1989 – 1996) for men and women

Graph 3. Age-standardized incidence rates (ASIR) for selected cancer sites, males, 1972-2001.



(**Graphs 5 and 6**). One trend not apparent from these graphs is the increase in the incidence of testicular cancer in men aged 20 – 49 years. Looking at these specific cancers individually, several comments can be made. (Note: Lung cancer in women will not be specifically addressed as it is widely acknowledged that tobacco use is responsible for the increased incidence of this disease.)

Graph 4. Age-standardized incidence rates (ASIR) for selected cancer sites, females, 1972-2001.



Thyroid Cancer

Thyroid cancer is more prevalent in women than men. An increased incidence of thyroid cancer between 1984 and 1998 has indeed been observed in women 20 – 49 years of age. The magnitude of this increase has not, however, been observed in males of similar age; the increase in males has been small during the same time period.

It is interesting to note that the incidence of this type of cancer took a jump between 1991 and 1995 in both sexes and most age groups *suggesting* improved detection of this type of lesion; however, this does not explain the dramatic increase among women compared to men.

In the early 1990s, the increased use of fine-needle aspiration biopsy may account for a portion of this increase. Incidence of thyroid cancer rises slowly with age. Many studies have linked exposure to radiotherapy directed to the neck region during childhood with a significantly increased risk of thyroid cancer. External exposure during adulthood and internal exposure to therapeutic or diagnostic doses of radioactive iodine, however, do not appear to increase risk. Changes in iodine intake may increase the incidence of some types of thyroid cancer and decrease the incidence of others. Diet may play a role, with consumption of vegetables (e.g. cruciferous) conveying some level of protection. Due to the difference in incidence between men and women, hormonal factors may be responsible. While studies have been conducted to assess a possible relationship between thyroid and breast cancers, the associations demonstrated have been weak, study sample sizes small and the conclusions not always consistent.

Non-Hodgkin's lymphoma (NHL)

The incidence of non-Hodgkin's lymphoma (NHL) has increased in both sexes of the 20 – 49 year old age group between 1984 and 1998, with the incidence, as well as the percentage increase, being greater in males than females.

The risk of NHL increases with age. Patients treated with radiation therapy for other cancers are at increased risk of developing NHL, and those treated with both radiation and chemotherapy are at even greater risk. Epstein-Barr virus has been associated with some uncommon types of NHL. HIV is a risk factor for NHL and the incidence among AIDs patients is much higher than in the general public; consequently, any increase in the incidence of HIV and AIDS will result in a concomitant increase in the incidence of NHL. Since the incidence of HIV and AIDS is rising more rapidly among men than women, it would be expected that a greater increase in the incidence of NHL in men would be observed.

Several epidemiology studies have concluded associations between exposure to phenoxy herbicides such as 2,4-D and MCPA, which are commonly used in agriculture and on turf, and the development of NHL[18, 19, 20, 21]. The majority of these studies have not measured exposure directly and failed to account for concomitant exposures to potential carcinogens (e.g. diesel fuel, prescription drugs) and exposure to oncogenic viruses found or suspected to play a role. In some studies, associations were found with certain occupations only; however, more research is required on this subject because definitive conclusions cannot be drawn from the epidemiology studies currently available.

Breast Cancer

The increased incidence of breast cancer in women may be due to lifetime exposure to estrogen which stimulates both normal and abnormal breast cell development[22]. Lifestyle changes such as having fewer children, giving birth at a later age and a reduction in the duration of breast-feeding or not breast-feeding at all, increase lifetime exposure to endogenous estrogen. High fat diets and genetics (BRCA1 and BRCA2 genes) also play a role. The use of oral contraceptives and hormone replacement therapy have been implicated as causal factors; the former by allowing women to delay pregnancy until a later age and the extent of the latter being dependent on the duration of treatment in addition to other factors. A portion, but not all, of the increase in incidence can be attributed to improved diagnostic techniques (the increased use of mammography).

Apparent from Graph 4, is that the increase in the incidence of breast cancer seems to be paralleling an increase in the incidence of lung cancer in women suggesting an association between the two diseases. While the incidence of lung cancer in males has declined due to a reduction in smoking among men, the incidence of lung cancer in women is still on the rise. The number of smokers in the female population has not declined to the same extent as among males, which would explain this statistic.

Prostate Cancer

The incidence of prostate cancer among men rose very slowly from 1984 to 1988. The dramatic increase in the incidence of this cancer between 1989 and 1993 can be explained by improved diagnostic techniques – primarily the use of Prostate Specific Antigen (PSA) testing. The increase in incidence occurred just after this new technique was introduced.

There has been a subsequent decline in incidence since 1993, as existing cases were diagnosed. This is truly indicative of an increase due to improved diagnostics. Risk factors include a family history of prostate cancer, high fat diet and vitamin D deficiency. Findings in epidemiology studies of occupation and prostate cancer risk have suggested a slightly increased risk among farmers, athletes, power plant workers, firefighters, workers in leather processing industries and soap/perfume manufacturing; however, the casual risk factors have not been confirmed[23, 24, 25].

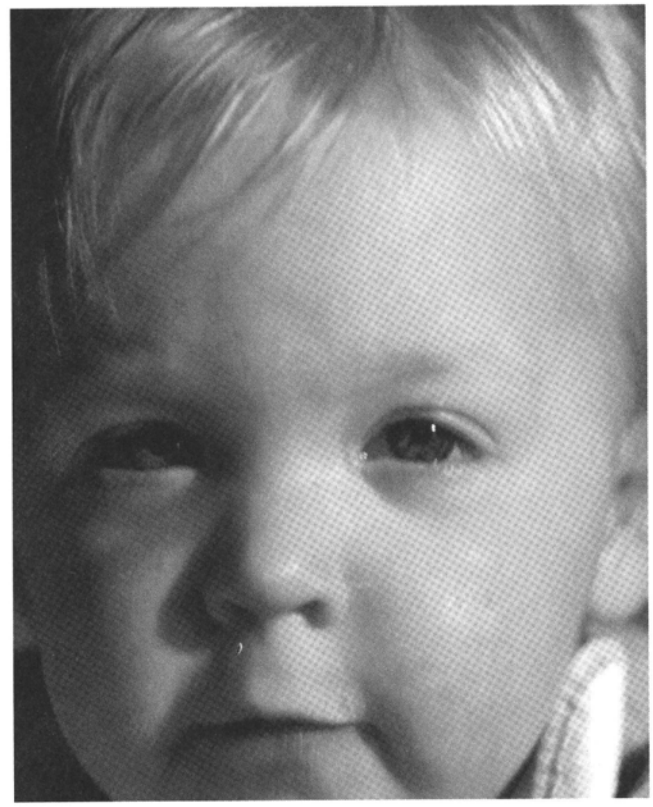
Testicular Cancer

The incidence of testicular cancer has increased steadily in men aged 20 – 49 from approximately 6 cases per 100,000 of the population in 1984 to 8.5 cases in 1998. Incidence among men aged 50 and over has been flat to slightly declining (actual number of cases is low at approximately 1 – 2 per 100,000 each year).

The main risk factors for testicular cancer are cryptorchidism or undescended testicle(s) and a family history of the disease, suggesting a genetic component. Approximately 14% of the diagnosed cases occur in men with cryptorchidism. There is an increased incidence among men with white collar or professional occupations as opposed to those who would be involved in manufacturing or spraying pesticides. This observation suggests socioeconomic status or lifestyle may be associated with the disease. It does not suggest that pesticide exposure is responsible. Exposure to "endocrine-disrupting chemicals" has been suggested as a possible contributing factor; however, it has not yet been demonstrated that the level of exposure the average public incurs to such chemicals originating from a variety of sources, including those that are natural, is sufficient to cause such a response.

Childhood Cancers

There has been a great deal of publicity suggesting that the incidence of childhood leukemia is increasing and that pesticides are responsible. Actually, the ***incidence of leukemia in children is not increasing in Canada*** or in the US. Incidence of leukemia in Canadian boys has remained relatively flat around a mean of 4.6 cases per 100,000 during the period from 1984 to 1998. Incidence has also been relatively stable among girls (approximately 3.9 cases per 100,000) during the same period. The incidence of leukemia peaks in children 1 to 4 years of age at approximately 8 cases per 100,000 and declines afterwards to approximately 2.5 cases per 100,000 in children 10 to 14 years of age.



More research is required; however, in the meantime, we should reduce the exposure of children to pesticides, not reduce the use of pesticides. The leading cause of death among Canadian children is not cancer – it's injuries.

Epidemiology studies suggesting an association between pesticide exposure and childhood leukemia are flawed due to small sample sizes and lack of statistical power, recall bias (asking a mother after her child has been diagnosed with cancer to remember what she was exposed to during her pregnancy and what her baby was exposed to after birth), failure to quantitatively measure exposure to pesticides and the identity of those pesticides, estimating exposure from birth certificate data or parental occupational title instead of actual sample measurements, and failure to control for confounding factors such as other exposures, just to name a few.

In 1998, Hoar Zahm and Ward of the US National Cancer Institute published a review paper summarizing data in the literature on pesticide exposure and cancer risk in children[26]. Their paper states that, while the studies reviewed were limited by a lack of pesticide exposure information, small sample sizes and the risk of recall bias (plagued by memory and other complicating factors), the risks reported were greater than those reported for pesticide exposed adults suggesting that children may be more sensitive to the carcinogenic effects of pesticides. (Note: The results could also indicate that parents more readily implicate pesticides when questioned about their child's exposure, compared to their response regarding their own exposure.)

The authors concluded that future research must include better methods for quantifying exposure to pesticides, investigation of the possibility of genetic-environmental interactions, etc. These are reasonable suggestions. The authors also concluded that reducing or eliminating pesticide exposure has the potential to prevent at least some childhood cancers. This is one conclusion that many scientists believe is a "leap" considering the weaknesses of the studies cited.

A year later, 5 researchers from the same institution published a review paper that concluded that increases in childhood cancer can be explained by improved diagnostic techniques[27]. Linet et al. examined incidence and mortality patterns among 14,540 children under the age of 15 years that were diagnosed with cancer from 1975 to 1995. They concluded that

there was no substantial change in incidence for the major pediatric cancers and rates have remained relatively stable since the mid-1980s. The modest increases that were observed for brain/central nervous system (CNS) cancers, leukemia and infant neuroblastoma, were confined to the mid-1980s. This pattern suggests that increases likely reflect diagnostic improvements or reporting changes that occurred during that time.

The subject of pesticide exposure and children's cancer risk is an emotional one and determining whether or not a relationship exists is quite complicated[28]. Even researchers from the US National Cancer Institute have differing opinions on the subject; however, those who believe that there is a connection concede that exposure has not been well defined and that the available studies investigating pesticide use and the development of pediatric cancers have many additional flaws including small sample sizes and case-control bias.

More research is required; however, in the meantime we should reduce the exposure of children to pesticides, not reduce the use of pesticides. As was seen from the Harris and Solomon (1992), and Stephenson et al. (2001) data, applying the correct application rate of a turf pesticide, and restricting contact with treated turf for a 24 hour period will result in non-detectable residues among bystanders, which translates to no detectable exposure to individuals contacting treated turf[2,4].

(Note: The leading cause of death among Canadian children is not cancer – it is injuries[29]. Many of these "accidental" deaths are preventable. Injury mortality statistics include deaths due to unintentional injuries such as motor vehicle accidents and falls, in addition to deaths due to suicide and assault (including child abuse). In 1996, 16 deaths per 100,000 occurred in people under age 20 due to injuries. This is equivalent to 30.5% of all deaths in this age group.)

Conclusions of Scientific Review Panels and Authors of Review Papers on the Subject of Pesticides and Cancer

In 1997, the National Cancer Institute of Canada's Advisory Committee on Cancer Control (ACOCC) addressed the issue of public exposure to pest control products to determine whether a significant level of risk existed that would necessitate the Canadian Cancer Society changing its priorities which are currently focused on tobacco control strategies. ACOCC established an Ad Hoc panel on pesticides and cancer[30]. While the Ad Hoc panel concerned itself primarily with pesticides used in the agricultural scenario, the published conclusions of this panel were that: no association was found between pesticide use and cancer and several factors may reduce cancer rates including:

- Reduction in smoking
- Increased consumption of fruits and vegetables
- Control of infections
- Avoiding intense exposure to sunlight
- Increasing physical activity
- Reducing alcohol consumption

The following year, world-renowned epidemiologist, Sir Richard Doll reviewed the published literature on potential causes of cancer and drew conclusions very similar to those of the Ad Hoc panel[31]. He concluded that smoking, alcohol, pharmaceutical products, infection, electromagnetic radiation (ionizing, UV, lower frequency), occupation, industrial products, pollution (air, water, food), physical inactivity, reproductive hormones and dietary factors (not pesticide related) were all causes of cancer.

Smoking and dietary factors are the most important, responsible for approximately 30%, and 20 to 50% of fatal cancers, respectively. Occupation, industrial products and pollution (including pesticides) combined, were thought to be responsible for a total of 3 to 4% of all fatal cancers.

In addition to other benefits, increased consumption of fruits and vegetables may reduce cancer rates.

Doll stated that there is no sound, scientific evidence to suggest that pollution from all sources, including pesticides, is a significant cause of cancer.The 9th Report on Carcinogens, published in 2000 by the US Dept. of Health and Human Services/National Toxicology Program, listed over 50 compounds *known to be human carcinogens*[32]. Not one pesticide was included on this list. The criteria used to define "known" were that "there is sufficient evidence of carcinogenicity from studies in humans which indicates a causal relationship between exposure to the agent, substance or mixture and human cancer...."

Table 1. HERP Percentage Values for Common Substances

Daily Exposure	Carcinogenic Component	HERP %
Beer (257 g)	Ethyl alcohol	2.8
Coffee (13.3 g)	Caffieic acid	0.1
Bacon (100 g)	Diethylnitrosamine	0.003
Lindane, daily dietary intake	Lindane	0.000001
Chlorothalonil (Daconil), daily dietary intake	Chlorothalonil	0.00000001

On the list are items such as aflatoxins produced by a fungus that grows on nuts, alcohol, asbestos, arsenic, coal tar, diethylstilbesterol (DES), tobacco smoking, environmental tobacco smoke, smokeless tobacco, exposure to UV light from solar radiation, sun lamps and tanning booths, crystalline silica and tamoxifen. Tamoxifen is a drug used very successfully to combat breast cancers that grow in response to estrogen. Tamoxifen also increases the risk of endometrial cancer, a form of uterine cancer, which is why it is listed; however, the risk of developing endometrial cancer is so small in comparison to the benefit gained among women with breast cancer that the drug is widely used and will not be banned. Things come full circle to risk versus benefit. The major causes of cancer listed in the "9th Report" were:

- Smoking
- Dietary imbalances (insufficiency of many micronutrients, insufficient consumption of fruits and vegetables)
- Hormonal factors, primarily influenced by lifestyle
- Chronic infections, mostly in developing nations
- Inflammation
- Genetic factors

In 1987, Ames et al. developed a ranking of carcinogenic substances to provide insight into the real risks that threaten our quality and length of life[33]. Often the threat to our health is not from rigorously tested products like pesticides, but from other substances to which we have unconsciously accepted the risks involved for the benefits obtained.

Table 1 (on the previous page) lists a few of the substances Dr. Ames has ranked using his Human Exposure/Rodent Potency (HERP) Index.

We test carcinogens on animals not humans and measurements are expressed as the rodent carcinogenic potency. To relate a product's carcinogenic potential in rodents to its carcinogenic potential in humans, the Rodent Carcinogenic Potency values are converted to HERP values. The higher the HERP % the greater the carcinogenic risk to humans. As you can see, the carcinogenic potential of beer and coffee are far greater than that of Lindane, an organochlorine insecticide or chlorothalonil, a fungicide that is commonly used on turf.

Conclusions

Reviews of sound, scientific, peer-reviewed data, indicate that allegations suggesting occupational and bystander exposure to pesticides is associated with increased cancer incidence, is currently *unfounded*. Unfortunately, the media's presentation of possible associations has created an irrational fear about pesticides among the general public.

As mentioned in Part One of this article, any pesticide ban approved by a municipality is a political decision based on emotion and not one based on sound science. This fact should be clearly communicated to the constituents of the municipalities involved. ♦