PALMER et al. v. NOVA SCOTIA FOREST INDUSTRIES

[1983] N.S.J. No. 534
60 N.S.R.(2d) 271; 128 A.P.R. 271*
S.SN.No. 02555

Nova Scotia Supreme Court Trial Division
Nunn, J.

September 15, 1983

¶ 1 **Nunn, J.**:— This is an application by the named plaintiffs in their individual capacities and as representatives of others for an injunction restraining the defendant, a company engaged in the forest industry in Nova Scotia, from spraying certain areas in the Province of Nova Scotia with phenoxy herbicides.

¶ 2 The action was originally brought in the late summer of 1982 when the plaintiffs obtained from this court per Burchell, J., an interim injunction restraining any spraying by the defendant pursuant to a license to spray during 1982 issued by the Nova Scotia Department of the Environment. The injunction was subsequently lifted by the Nova Scotia Court of Appeal in December, 1982 on the ground that no spraying could take place until the summer of 1983 and that there could be a full trial and hearing of the issues by that time.

¶ 3 The matter was then brought to trial in Sydney commencing May 2, 1983, with the relief claimed, in the statement of claim, to be a permanent injunction restraining the spraying of 2,4-D (Esteron 600), 2,4,5-T with its contaminant TCDD, and Esteron 3-3E, which is a mixture of 2,4-D and 2,4,5-T.

¶ 5 The trial consumed 21 days of taking evidence, 2 days of oral argument and further written briefs. The plaintiffs tendered 35 witnesses and the defendant 14. Aside from most of the named plaintiffs, all the rest of the witnesses with but a few exceptions were experts - highly educated in their various fields to which they gave evidence.

¶ 7 The subject of dioxins and chlorophenols has been widely disputed in many countries of the world both politically and before regulatory agencies. To my knowledge this is the first occasion where the dispute has reached the courts in Canada.

¶ 8 Because of the nature of this case, the notoriety it has received, the public interest it has generated and the volume of evidence presented to the court by both parties to this dispute it is necessary to set forth in some considerable detail the evidence presented.

[Partial list of plaintiffs]

¶ 14 Reverend Charles Mullendore, plaintiff, as an individual and as a representative of others in the Skye Mountain site area, Site E - Skye Mountain on the maps, testified that he has a residence and a 100-acre farm in the area. His farm extends to within one mile of the spray area as mapped for this site, and downhill from this site. He and his family, as well as others in his area, take their drinking water from a stream and use this stream to provide drinking water to livestock. The farmland is used for grazing and he, his family and neighbours use the general area for walking and berry picking. One stream in the area, Coalbrook Stream, is a breeding ground for trout and aquatic life. He joined as a plaintiff because of his concern over the use of chemicals where there is conflicting evidence as to their safety, possibly jeopardizing the health of he, his family and his neighbors. [*page275]

¶ 15 John Dan MacIntyre, retired, of West Bay Road, plaintiff and a representative of others at or near site D - MacIntyre Mountain, owns a 100 acre farm which adjoins the site. His residence is two miles below the site. He uses the farm for growing vegetables and potatoes and for pasture for a horse and some cows, and his family pick

blueberries and hunt in the area. He also cuts his firewood on his land. His family and some other neighbours live on the opposite side of the Trans Canada Highway from the site. He, his family and neighbours get their water from Rough Brook which adjoins his farm. He testified that this brook gets muddy after a rain storm which is indicative of some silting in this area. Rough Brook is actually on the site. His garden is located approximately one-half mile from the site. His concern was the possible harm the spray would create to his water and health.

¶ 21 Brian GooGoo, a full-blooded MicMac Indian, lives on the Whycocomagh Reserve which is 1 1/2 miles from Site E - Skye Mountain and located at the base of the mountain. He was formerly a chief and has been delegated by the [*page276] Whycocomagh Indian Band to represent the Band in a representative capacity in this action.

¶ 22 As part of life on the Reserve, the Indians hunt, fish, pick berries, medicinal bark, roots and herbs on the Reserve and on Crown lands. They fish eels, trout, salmon, flatfish, oysters and lobsters in the Bras D'Or Lakes into which a number of streams flow from Skye Mountain.

¶ 23 The concern here is that their way of life is being threatened by the effect the spray program will have on the area and to their health through their water and food supply. This witness asserted such effects are an interference with his people's aboriginal rights to carry out their life-style activities as they have done for centuries. He suggested a greater interference with those aboriginal rights by the very fact that the defendant has come upon some of those Crown lands, by virtue of leases, cut the timber and destroyed wild life without any consultation with the Indians. This latter aspect may very well be beyond the scope of the matters involved in this action.

¶ 24 Thomas Francis, another full-blooded MicMac living on the Afton Reserve, Antigonish County, and presently Chief of the Afton Reserve, is a plaintiff representing the Afton Reserve MicMac Band. This reserve constitutes two sections, both of which are adjacent to site C - Fraser's Grant. The Afton River flows from the site through the Reserve. There are 200 people living on the Reserve and they use the river as a source of fish, smelt and trout, medicinal roots, and for recreation. One person has cattle which drink from it and it is used for personal washing and washing of clothes. He lives 4 miles from the spray site.

¶ 25 The spray site itself is and has been used by the Band for hunting, fishing, the collection of roots, herbs and barks for herbal medicines and picking blueberries and raspberries.

¶ 26 His concern also is related to the safety of the chemicals and that this proposed sparying may be an interference with the Band's aboriginal rights.

¶ 29 Victoria Palmer, a plaintiff on her own behalf and on behalf of others in the vicinity of site B-Lochaber, resides on approximately 570 acres adjacent to the site. She and her husband use the land to grow all the food they eat, except for those things that cannot be produced in Nova Scotia. They have a large vegetable garden, dairy cows and beef cattle and from the milk they manufacture and sell yogurt. What they cannot use of the beef cattle, which incidentally are raised strictly on milk and grass, they sell. The land is unfenced, making it extremely likely she would often be on the lands of the defendant.

¶ 30 The Gusset Brook originates near the spray site and feeds a flood plain on her land where her cattle and a neighbour's graze and drink. One of her neighbours has a shallow well in this area from which he obtains his entire water supply. [*page277]

¶ 31 Wild life abounds in the area. There are other neighbours in the area and some 20 to 25 children. One of the neighbours raises sheep and presently has approximately 100 which pasture in the area.

¶ 66 The final group of witnesses offered by the plaintiffs were experts in various fields who gave opinion evidence to the court. A short summary of the evidence of each is as follows.

[most of the expert evidence is omitted]

¶ 107 Dr. Wulfman referred to TCDD as the second most toxic chemical known to man. Because of this, neither he nor any of his students actually worked with TCDD, but since dioxin is a catechol ether, the catechol molecule, a safe molecule, could be used to study the dioxins and this was the molecule used in any studies he performed.

¶ 108 Directing the court to the mobility of dioxins, he indicated that determining their mobility in the aquatic environment is not a simple matter and indicated several factors involved, namely:

1. TCDD has a very low solubility in water.
2. TCDD has a high affinity for soils and clay particles.
3. Low level detection presents severe analytical problems.

4. Low levels of pollutants in water frequently are concentrated by various organisms.

5. The biodegradability of dioxins.
6. Annual rainfall and timing.

7. Mean temperature, hours of sunlight, humidity and rainfall all play a role in biodegradation.

¶ 109 He suggests that the high affinity of TCDD for soil particles is grounds for concern over possible pollution of aquatic life, but it is highly unlikely that contaminated ground water will enter the aquifers. This affinity means TCDD will be found near the surface of the soil. This opens a danger of air borne dust in a dry forest floor condition and the run-off of soil particles in a heavy downpour.

¶ 110 He cited situations in Lake Ontario, the Tittabawassee River in Michigan and the Bayou Meto River in Arkansas as revealing that dioxins can be transported via water. He presented a series of slides, Exhibit 19, with his comments which illustrated his general testimony, elaborated on some points and revealed the sources of some of his comments. Some highlights include that 2, 4-D at 10% and 2,4,5-T at 2% of the total herbicides used do not constitute a large share of the [*page288] market; that there are other alternate herbicides; that 2% of 2,4-D is used in forestry out of approximately 60,000,000 pounds used annually in the United States; that Canadian use would be about 10% of that of the United States; that dioxin as a carcinogen is, compared to a number of other known carcinogens, in a class by itself, many times more toxic than those others. In this regard he referred to a table stated to be a U.S. Environmental Protection Agency table which showed dioxin to produce an increased risk of cancer of 100% to a 150 pound person who, over a lifespan of 70 years, breathed air containing one microgram of TCDD per cubic meter. Further, he disputed figures of Dow Chemical as to the presence of TCDD in fly ash in the quantity alleged and suggested that his conclusion was there was only a very small proportion of TCDD produced through fire. He further discounted the Dow Chemical Study of Wives (January 1981) concerning pregnancy risk.

¶ 111 Finally, he discussed the herbicide Esteron 3-3E, which is a mixture of 2,4-D and 2,4,5-T and the product proposed to be used to spray the Nova Scotia forests; the possibility of crystalization if stored for long periods, and/ or if subjected to low temperatures, as well the possibility of the compound changing with time. He concluded his direct testimony suggesting that some types of carcinogens require only one molecule to affect a living organism and that there is no threshold dose level, i.e. no safe dose limit.

¶ 112 On cross-examination, it was revealed that some of the information he relied on in his direct testimony and from which he drew conclusions were only working papers that he had obtained which were not included in the published report called A preliminary Summary Result, dated March 28, 1983 Dow Chemical Company - Midland Plant Wastewater Characterization Study done by the U.S. Environmental Protection Agency, and therefore had no force or effect.

¶ 113 His evidence concerning the Bayou Meto River was challenged and several of his statements on direct were discredited. With regard to the U.S. Environmental Protection Agency table, referred to earlier, he was asked the quantity of spray mix a 150 pound man would have to breathe per day to receive the quantity referred to in that table assuming the concentration of TCDD to be 10 ppb (parts per billion). The answer was 146 kgs. (322 pounds) of spray

mix every day for 70 years to incur the stipulated cancer risk of 100%. This 100% increased risk was further explained to mean that whatever risk a non-exposed person had to incur, cancer would be doubled to an exposed person.

¶ 114 Dr. Wulfman criticized the Dow Chemical Study, referred to earlier, suggesting a self-interest. He also criticized two National Research Council Studies which indicated in one that PCDD'S, including the 2378 isomer, were probably ubiquitous in the Canadian atmosphere, and in the other dealing with, among other things, the levels of phenoxy herbicides in the atmosphere during spraying on the praries, on the basis that representatives of Dow Chemical were involved on the panel in one and were an advisor on the other.

¶ 115 Finally, on cross-examination he agreed that his theory of one molecule causing cancer is not generally held and there are other opinions.

¶ 116 This witness, though obviously competent in the field of his expertise, appeared to lose his objectivity and methods of scientific inquiry and took on the air of a protagonist when he discussed these chemicals with regard to their toxicity and effects in the environment. His analogy of "the fox guarding the chicken coop" is most revealing of his view. I could not help but feel, through a good deal of his evidence, that he was espousing a cause and his enthusiasm led him, at times, [*page289] outside the strictures of a scientific approach to data and other scientific opinion.

¶ 117 Dr. John Constable, a specialist in general surgery and plastic surgery and an associate professor at Harvard University Medical School, who had acquired an intimate knowledge of the whole health field in Vietnam during and following the war, was qualified as an expert entitled to give opinion evidence in the fields of health effects and the ecological effects of herbicide use in Vietnam, as well as an epidemologist to the extent of his epidemiological studies in Vietnam dealing with herbicides. His qualifications are fully set out in his expert report, Exhibit 6B, Tab E and his evidence on the record. However, it should be pointed out that his ecological expertise arose through his position as a director of American Wildlife, a division of World Wildlife. Because of this side interest, he studied as a side activity the effects of herbicides on wild life and the ecological system while in Vietnam. He had originally gone to Vietnam as a plastic surgeon and expert in burn therapy at the behest of the United States Committee of Responsibility to assist with the large number of children who had suffered napalm burns. During his early visits he became involved with Dr. M. S. Meselson, also of Harvard University, who was chairman of a committee studying the ecological, economic and medical effects of herbicide use in Vietnam. He was the medical expert on the committee and worked very closely with Dr. Meselson. In all he made six visits to Vietnam with four relating to herbicides.

¶ 118 He testified that, according to U.S. Government figures, 44 million litres of Agent Orange, a mixture of 2,4-D and 2,4,5,-T, 20 million litres of Agent White, 2,4-5T with pichloram (containing no known dioxims), and 8 million litres of Agent Blue, an arsenical acid (a different category of chemical compound), were sprayed starting in 1965 and increasing to maximum in the years 1967, 1968, 1969 and dropping off in 1970, stopping entirely by the U. S. in 1970. The considerable remaining stocks were then sprayed by the Vietnamese. The spraying was confined to South Vietnam and was known as Operation Ranch Hand.

¶ 119 The spraying resulted in destruction of almost 50 per cent of the mangrove forest, the source of fuel to the cities, as well as substantial crop destruction, resulting in the creation of large mud flats. In the tropical forest the high trees were killed. The result was the killing of these forest areas rather than defoliation. Animal habitats were destroyed and soil samples showed residual levels of dioxin.

¶ 120 Agent Orange, the mixture of 2,4-D and 2,4,5-T with its known contaminant TCDD, became the focus of his attention. Fish were studied and it was determined that some had residual quantities of dioxin. Samples of human milk were taken from mothers of heavily sprayed areas and areas where fish from the sprayed areas were eaten and a few of these showed dioxin or possible dioxin in the cream, although in less substantial quantities than in the fish. It was, however, impossible to determine the level of exposure or how any person actually received any dioxin as some were exposed to the spray, some breathed it, some ate sprayed materials and some had exposure through the food chain. Record keeping was extremely poor and the whole area was filled with uncertainties. According to the witness, two birth anomalies showed a striking increase, spina bifida and cleft palate, but these were really straws in the wind and

needed further research.

¶ 121 After the war ended he became involved with a Dr. Tung of Hanoi who was attempting epidemiological studies on the effects of the herbicides. Dr. [*page290] Tung prepared a total of three papers on studies he performed, the first two of which were not up to international standards, but the third was much better and was presented to a symposium held in Ho Chi Minh City, of which the witness was rapporteur. Dr. Tung's study was of 40,000 women. His idea was to study women in the North, totally unsprayed, who married men who served in the South who were exposed and compare them with those who married men from the North who were not exposed. By way of cohort analysis with as many controls as possible, the result, according to the witness, was a statistically valid difference in birth anomalies between the two groups. In other words, a northern woman who married a man from the south had a higher chance of birth anomaly than one married to a man from the north. This was a presumptive conclusion.

¶ 122 On the basis of his observations, knowledge of the literature and finally Dr. Tung's study, the witness offered the following opinions:

1. There is now sufficient evidence to state without question that the exposure of persons to phenoxy herbicides contaminated with TCDD poses a probability of harm to those persons and that it is the degree of probability, not the existence which is in dispute.

2. TCDD is persistent in the environment and is bio-accumulative (i.e. it bio-magnifies throughout the food chain).

3. Residues of TCDD were present in fish four years after cessation of the major use of the phenoxies in Vietnam and similarly in human breast milk.

4. Though the levels of TCDD which persist may be minute, one cannot say consumption of food animals from treated areas poses no measurable risk.

5. It is not currently possible to identify a no-effect level for TCDD for cancer reproductive effects.

6. That phenoxy herbicides contaminated with TCDD have been associated with spontaneous abortions, still births, hydatiform moles and congenital anomalies.

7. The deliberate broadcast of TCDD contaminated phenoxy herbicides should, for reasons of possible adverse health, be avoided unless there is no alternative.

¶ 123 On cross-examination he admitted that until January 1983, the date of the symposium, there was no scientifically documented study which would demonstrate any connection between the use of Agent Orange in Vietnam and birth defects. He was unaware of an Australian Case Control Study of Congenital Anomalies and Vietnam Service, January, 1983, which reached the opposite conclusion to the Tung study and explained a Canadian study by the Department of Health of the effects of spraying Agent Orange at Camp Gagetown, New Brunswick, which reached the same conclusion as the Australian study, as his own view until January 1983. When presented with statements from a number of the working papers presented to the symposium, the witness admitted that there were few conclusions, that most recommended further studies, that no specific association with dioxin has been conclusively proved and the results are presumptive only.

¶ 124 He confirmed the quantity of 2,4,5-T sprayed was 24 million litres and that contained 170 kg. of TCDD, or approximately 7 parts per million and agreed that modern production methods are able to control the amount of TCDD in commercial 2,4,5-T to less than .01 parts per million, which means the amount of TCDD contaminant in the 2,4,5-T to be sprayed in Nova Scotia is 1,000 times less than that in Agent [*page291] Orange.

¶ 125 He admitted the spray in Vietnam was a pure mixture of 2,4-D and 2,4,5-T and that the area was heavily sprayed, while he understood that in Nova Scotia the proposal is for a watered-down mixture which would produce a much lower concentration.

¶ 126 Theodore Sterling, Professor in the Faculty of Interdisciplinary Studies at Simon Fraser University, was qualified as an expert entitled to give opinion evidence in the fields of Epidemiology and Statistics. His opinion, as expressed in his expert report, Exhibit 6 B Tab F, is as follows:

"I find that a considerable body of evidence has accumulated by now showing that phenoxy herbicides, including the preparations of 2,4,5-T and 2,4-D are generally toxic, carcinogenic, teratogenic and mutagenic. Most of the toxicity and other health effects may be due to a variety of impurities and contaminants (especially dioxins) which are unavoidable byproducts of the manufacture of phenoxy herbicides. Existing evidence indicates that there may be no lower threshold to the health effects of some dioxins, especially TCDD which is an unavoidable byproduct in the manufacture of 2,4,5-T. However it is possible that phenoxy herbicide preparations are toxic, even if free of measurable quantities of dioxins."

¶ 127 In his direct testimony, after discussing the science of epidemiology's techiniques, including comparables and the importance of sample size, true comparisons, and proper weighing and balancing of all data, he indicated that epidemiological studies do not tell how a disease occurs or what the mechanism of disease is but rather indicate a trend that people exposed to a particular set of circumstances will have a higher probability of having a particular disease than others not so exposed. Further, it is almost impossible to have a clear cut epidemiological study. Any particular study gains weight through repetition with other studies of other population groups in other areas.

¶ 128 Interestingly, in his view, the study of phenoxy herbicides from the very beginning was an "adversary study". While phenoxy herbicides had been in use in agriculture and forestry since the early 1950's, they had only begun to be studied seriously in or about 1969, largely as a result of their use in Vietnam, and more particularly, epidemiological studies really did not start in earnest until 1978.

¶ 129 The witness was then directed to a number of studies upon which he based his earlier opinion, including:

(a) Alsea II, a study in Alsea County, Oregon, U.S.A., started because eight women in a heavily sprayed area had spontaneous abortions;

(b) Swedish studies of railroad workers exposed to 2,4,5-T;

(c) Dr. Tung's studies in Vietnam; and two other studies which appeared only later in the literature;

(d) Canadian study of agricultural workers on the prairies, and

(e) a German study of certain agricultural workers.

¶ 130 These last two he indicated were not well conducted, although he included them as adding weight to the former evidence. He included a number of his own articles with his expert opinion and these referred to a number of other studies done by various scientists, particularly on the effects of these substances on animals. Throughout his evidence, it was clear [*page292] that he put heavy reliance on the Alsea II study and the Swedish studies, both of which he considered to be valid epidemiological studies.

¶ 131 Essentially he says that while epidemiologists are loath to draw conclusions from a single study, conclusions may be drawn as there is a constant repetition of similar findings in different studies done in different circumstances. He considers there to be overwhelming evidence of an association between the herbicide and occurrences of cancer or malformations or spontaneous abortions of population, again placing great weight on the U.S., Swedish and Vietnam studies.

¶ 132 He criticized several other studies which had reached different conclusions, particularly with regard to "threshold levels" or "no response levels", mainly done on animals, largely on the basis that a sufficient number of animals were not used, though he did indicate that the actual numbers used were the standard number used by scientists the world over in conducting such tests.

¶ 133 On cross-examination the witness indicated he was a critic of the works of others. His attention was turned to

a quotation in a book, to which he contributed, entitled "Voodoo Science Twisted Consumerism" and attributed to him as follows, at p. 53:

"The Alsea II study is fundamentally sound from an epidemiological perspective. This study initiated by woman residents of Alsea County, Oregon, in desperation because of the failure of U.S. health agencies to attend to their problems has been done with the professionalism which is perfectly acceptable epidemiology. I for one find that the statistical analysis of that report quite adequate and convincing."

¶ 134 While the witness suggested those words were not direct quotes from him, he agreed with them. Counsel for the defendant then presented the witness with the following statements:

(a) New Zealand Report on the Alsea II Study (New Zealand Department of Health, Exhibit D 14, at p. 10.

"It is important from the foregoing that the report 'Alsea II' is grossly inadequate from a number of points of view, although this critique does not claim to have dealt with them all. Because the authors of 'Alsea II' have failed to consider and discount other more likely causes of the differences and correlations they obtained, it is felt that no weight whatsoever can be given to their conclusions."

[The court cited 8 more sources or bodies that had criticized the reliability of the Alsea II studies]

¶ 135 To all of these the witness indicated he disagreed with their conclusions so far as they relate to Alsea II although he appeared to waiver by saying:

"I think the Alsea Study is probably as good or as bad as most epidemiological studies are."

¶ 136 Although he claimed an intimate knowledge of the scientific works in this field he was not familiar with and had not read, a number of those from which the foregoing exerpts were taken.

¶ 137 Finally he testified that he presented a brief to a British Columbia Royal Commission on Herbicides and Pesticides. When it was suggested to him that his views were not accepted, he replied, "They were not the only ones."

¶ 138 On re-examination he then described the effect of the Alsea study as "another feather, you might call it, that adds to the total weight of it."

¶ 189 Dr. Mikael Eriksson testified, by agreement with counsel for the defendant, in substitution for Dr. Lennart Hardell who, through personal tragedy, was unable to attend at this trial. It was also agreed by counsel that the Hardell references, contained in Exhibits 6D and 6E, were admissible and could be used by the witness, as also Dr. Hardell's expert opinion contained in Exhibit 6B and Tab H.

¶ 190 Dr. Eriksson is a medical doctor, qualified to practice medicine in Sweden, and was associated with Dr. Hardell and participated in the studies which will be referred to as the Hardell studies. He is currently a consultant physician at the County hospital of Ostersund, Sweden, specializing in internal medicine and engaged in research concerning hematology from an epidemiological point of view.

¶ 191 He was qualified as an expert entitled to give opinion evidence in the fields of toxicology and epidemiology, particularly cancer epidemiology, as they may relate to phenoxy herbicides but limited to the work and studies he performed with Dr. Hardell.

¶ 192 He defined cancer as a malignant disease, probably of a one-cellular origin whereby that cell grows into a cell population called a tumor. The malignant tumor has two characteristic features. First, unlike with normal cells, it has an uncontrolled growth and second, it has an unsocial behavior, i.e., it does not accept any borders of tissues. It has an additional feature of being able to release "daughter tumors".

¶ 193 He stated that cancer is increasing in most parts of the world although the patterns of cancer may differ substantially from one country [*page304] to another. While cancer may to some degree be inherited, the main causal factors are environmental in the widest sense.

¶ 194 In discussing the procedures to determine whether a particular substance is carcinogenic, he indicated that animal testing is an acknowledged procedure although it is not conclusive because of the difference of species. Carcinogenic effects on humans of any substance may be revealed through epidemiological studies on human populations. These studies take two general forms, the "case control study" which begins with an incidence of disease with those having such disease being compared with a control group who do not have the disease as to earlier exposure of the substances under study, and, secondly, the "cohort study" which starts with an exposed population and the effects upon that population over given periods of time as that population marches through life.

¶ 195 To perform an acceptable case control study, the witness indicated there are many pitfalls and such a study is very complicated. There has to be a correct selection of cases. Control groups must be properly chosen. Confounding factors must be avoided and observational bias eliminated. This latter contains two elements, "recall bias" - a bias of the subject to recall, for example, his exposure to any substance, with cancer patients having a propensity to remember exposure to chemical substances to a higher degree than the control population; and "observer bias" - a tendency of the collector of information to overestimate the histories of exposures.

¶ 196 The witness related how the Hardell studies came about, as well as the procedures used. In 1977, Dr. Hardell observed three cases of a rare type of tumor - a soft tissue sarcoma - each person so afflicted reported rather extensive use of phenoxy-acetic herbicides during the 1950's and 1960's. In checking through the records of the Department of Oncology he found another four cases with similar exposure. This led to the epidemiological study which was a case control study. The study resulted in a conclusion of a relative risk for this kind of tumor of six compared to a relative risk of one in the control population, i.e., six times higher than the risk of the control population. The particular phenoxy herbicides used in Sweden were 2,4,5-T, 2,4-D and MCPA. In this study most of the people studied lived in northern Sweden, an area of large forests with a great forest industry. Apparently, prior to 1978, 2,4,5-T was the main herbicide used in the northern area.

¶ 197 A second study was performed in the southern part of Sweden, mainly an agricultural area, where 2,4-D and MCPA were the main herbicides used. This study related also to soft tissue sarcoma. This resulted in a determination of a relative risk for soft tissue sarcoma of 5.1.

¶ 198 Dr. Eriksson then explained that exposure to phenoxy acids alone was analyzed as part of the second study with a resulting relative risk of 6.8. Further, as another subdivision of this study, the relative risk relating to exposure of phenoxy acids free of 2,4,5-T i.e., 2,4-D MCPA, dichloroprop and mecaprop, was 4.2. This latter relative risk was described by Dr. Eriksson as not as highly significant but clearly significant.

¶ 199 The third study performed was again in northern-Sweden, but this time it related to malignant lymphoma, a disease two times more common than soft tissue sarcoma, and it resulted in a determination of a relative risk of 5.3. Here, again, exposure to phenoxy acids, excluding the chlorophenols, was analyzed and the relative risk was determined to be 4.8.

¶ 200 Dr. Eriksson outlined in detail, which I need not repeat, the methodology [*page305] of these studies. After initial selection of patients and controls by a matching process, a questionnaire was circulated with many questions, attempting to hide the real purpose of the questionnaire. The responses were studied and supplemented by a telephone interview. For deceased patients and controls, the same procedure was followed but with next-of-kin and relatives. With regard to exposure a questionnaire was sent to employers to confirm employment and exposure and the use of chlorophenols in their production process. Low levels of less than one day exposure and exposure within five years of diagnosis of the tumor were excluded from the study. Following determination of how many were exposed, statistical methods were applied to determine the results.

¶ 201 The studies performed were written up and published in various medical journals.

¶ 202 In discussing the concept of relative risk the witness explained that about one percent of all malignant

diseases are soft tissue sarcomas and about one out of four human beings will develop a malignant disease. Thus, the absolute risk of getting a soft tissue sarcoma may be stated as 0.25 percent. The relative risk is how many times more likely would it be to get the disease under study if exposed to a certain substance. The results of the first study, with a relative risk of 6, means that a person with the 0.25 absolute risk would now have a 1.50 risk. The same type of calculation could be made of the results of the other studies. In other words, considering that actually getting the disease gives a risk of 100 percent, this means that one's risk if so exposed would be 1.5 percent.

¶ 203 Throughout his evidence, however, the witness indicated that cancer is a "multi-factoral disease" and admitted that these chemicals might very well be only one of these many factors. Further, he told the court that there was no way of knowing the quantity of chemicals to which any of the subjects were exposed.

¶ 204 His view was that even if the risk was a relatively low one, it is tragic.

¶ 205 He then referred to four cohort studies conducted in the United States, resulting from accidents in chemical factories, which would indicate a relative risk of from 41 to 57, as studies which support and strengthen the Hardell findings. However, these were very small studies where there was very significant exposure.

¶ 206 Considerable evidence was given by the witness in support of methodology of the Hardell studies and it was his conviction that the pitfalls associated with a case control study were avoided in those studies.

¶ 207 The witness was then asked to comment on certain of the expert reports of witnesses to be produced by the defendant. He disagreed with any criticisms of the Hardell studies contained in these reports, dismissing parts of Dr. Steven Lamm's report as "irrelevant, unfair, unappreciative of Hardell's work, nonsense, so terrible, based upon stupid assumptions, contradictory with many vague assumptions, wrong conclusions and hardly believable estimations". Turning his attention to the report of Dr. Richard Wilson, he indicated astonishment that Dr. Wilson, Chairman of the Physics Department at Harvard University, could make such incorrect statements. He found one of Dr. Wilson's conclusions to be ridiculous. He referred to one chapter entitled "Comparative Risk" as so silly as to be unworthy of comment and to be a nonsense chapter.

¶ 208 He was not so hard when commenting on Dr. Robert Kilpatrick's report, [*page306] although he disagreed with the major points made in that report which were critical of the methodology of the Swedish studies and the validity of their results.

¶ 209 In the course of this part of his testimony, Dr. Eriksson did indicate that he did not accept the Vietnam studies as valuable as the possibilities of a good study there were very poor after the war.

¶ 210 Finally, in concluding his direct testimony, Dr. Eriksson claims there is a proven connection between exposure to phenoxy herbicide preparations, without closer knowledge of what substance and what combination of substances is the real cause, and the developing of at least two different types of malignant disease, namely, soft tissue sarcoma and malignant lymphoma. He claims this proven connection is widely accepted by the real authorities in the field. TCDD is at least one of the causes but their studies indicate the possibility of other contaminants or the phenoxy herbicides themselves as being responsible for the carcinogenic process. He also said one cannot make estimations from animal experiments because of the difference in species. Further, while he velieves the soft tissue sarcoma and malignant lymphoma are rare type cancers and can be studied easily by the case control method, other types of malignancies may result from exposure to these chemicals.

¶ 211 His concluding remark was that criticism of their studies was not valid and so regarded by independent researchers, while the main objections were levelled by industrially-connected researchers.

¶ 212 The witness was vigorously cross-examined, particularly with regard to criticisms by other scientists as to the validity of the Hardell Studies. For example, Dr. Philip Cole, a former Professor of Epidemiology, whom the witness acknowledged as an eminent epidemiologist, indicated in a letter, Exhibit D-43, stated that he believed it was the widely held view that TCDD was most likely the etiological agent, although the specific etiological agent was unknown, and massive exposures were involved in the cohort study he was referring to. Secondly, he considered the Hardell studies "unconvincing" and "minimally advanced" by the U. S. cohort studies and that the risk of soft tissue

sarcomas among phenoxy acid herbicide users "remains unknown". The witness did not agree with these observations.

¶ 213 Specific criticisms of methodology of the Hardell Studies made by Dr. Cole (Exhibit D-44) were suggested to the witness, each of which was rejected by him. Some of the criticisms suggested were the opportunity for selective recall because of the degree of publicity of exposure as a possible cause of cancer and the degree of publicity at the times of the studies was referred to. While the witness denied any substantial publicity, he was referred to Swedish newspaper articles as well as statements by Dr. Hardell that there was vigorous and sustained discussion in the press throughout the period of his studies. The sedond criticism was "interviewer bias" during the telephone inquiries as the interviewer knew the purpose of the study. Third, there was a difference between cases and controls in the validity and completeness of their histories with respect to their exposure and this was alleged to be the most serious limitation of the studies. Fourth, the instructions provided an opportunity for major recall bias as the purpose of the study would be obvious. Fifth, only "Yes" or "No" answers were called for an all questions were to be answered, i.e., at least there should have been a "don't know" category. Sixth, the questionnaire was to be returned to an oncological clinic, e.i., a cancer clinic. Seventh, the telephone interview increased the likelihood of recall bias. [*page307] Eighth, the results link exposure with sarcomas of numerous different histological types, namely 12, a unique fact as other chemical carcinogens have never been linked to more than three sites in humans. Eighth, the Akellson technique could not be used with good effect in a study of this kind. Ninth, the presence of recall bias is suggested by data presented in the report itself.

¶ 214 To all of these the witness disagreed and maintained throughout the validity of the Hardell studies.

¶ 215 He did not agree with the suggestion of counsel that there was a dose-response relationship with these chemicals. Although later he did admit that there was a "trend" of a dose-response relationship found in all these studies and he was not prepared to indicate how significant this was.

¶ 216 He indicated that they had no knowledge of the degree of TCDD to which the studied subjects had been exposed, nor did they know if or how much TCDD contaminant was contained in the 2,4,5-T to which the subjects were exposed. As well, though 2,4-D and mecaprop were chiefly used in Southern Sweden, they had no knowledge as to whether and what quantity of 2,4,5-T might also have been used there. The witness maintained that while the specific etiological agent is unknown, the possibility of other contaminants as well as the herbicides themselves must be considered.

¶ 217 The witness agreed that the seven cases which were involved in the first Hardell study were massively exposed and in all cases studied, except one, diseased and control persons were all occupationally exposed. The second study also involved solely occupationally exposed persons. Again, there was no actual exposure data.

¶ 218 Counsel suggested that Dr. Hardell's public position was that his studies pose only a concern for those occupationally exposed, but the witness disagreed, despite suggestions otherwise (Exhibits D-48, and 6-E Tab 66).

¶ 219 The witness was then referred to a number of the articles submitted as part of the Hardell report (Exhibit 6) again suggesting all cases reported in the various studies were cases of occupational exposure. Several passages in some of the articles by other Swedish scientists suggesting that it was reasonable to predict safe levels of use and that the concern was to develop methods to reduce exposure of those actively engaged in using these chemicals is the prime concern were put to the witness. He did not agree with either.

¶ 220 A number of times the witness indicated that some of the scientists who criticized the Hardell studies were biased because they were industrially related, although he admitted that there were very eminent scientists critical of Hardell's studies who certainly were not biased. It did appear that he felt this bias more than he admitted in his testimony.

¶ 221 When he was advised of Finnish studies of herbicide sprayers in the railway and forestry industries where no soft tissue sarcomas were found, he indicated the study was too small although it was of 1,971 workers. He was not aware of the developments in the Ranch Hard studies in the United States.

¶ 222 A final article entitled Risk Evaluation of Some Chemical Pesticides published in 1982 in Sweden (Exhibit

D-51) was referred to the witness. This was a committee study where 15 scientists were hired who did the bulk of the study. Among other things, this report, which studied all the registered cancer material from 1961 to 1979 [*page308] including the Hardell study period, 1974 to 1979, concluded that the results were not in accordance with the Hardell studies. In comparing male farmers, woods workers and horticultural workers with the general population, no increased risk between the two groups could be proven regarding Hodgkin's disease, non-Hodgkin's lymphoma and soft tissue sarcoma. The report further suggested that Hardell's estimates were impaired by major chance mistakes which contribute to his higher relative risk. The report stated further that there is no hint of an upward trend in the incidence of soft tissue sarcoma despite the fact that the handling of phenoxy acids has increased substantially from 1948 to 1975. Considering the latency period, an upward trend ought to have developed. Further, the report concluded that the recorded studies (presumably including the Hardell studies) do not cause any reason for concern regarding the dangers of passive exposure.

¶ 223 The witness disagreed with the main contents of this report. On being questioned by the court he indicated that these compounds have a very low acute toxicity, i.e., a low immediate toxic effect, while it is the chronic toxicity, which is long term, that he is concerned about. He also indicated that it could not be proved in any single case that a person suffered a soft-tissue sarcoma as a result to 2,4-D or 2,4,5-T or, indeed by TCDD, for it may be some other thing in society which causes soft tissue sarcoma.

¶ 231 The defendant then called a group of witnesses …

¶ 257 Dr. Dieter Riedel, head of the Reassessment Section of the Pesticides Division of Health and Welfare Canada, with a doctorate in Teratology, was called by the defendant to outline the work of his department which involves surveying the literature and doing their own laboratory work. He testified that 2,4-D was registered for use in Canada [*page312] in 1947 and 2,4,5-T in 1952 and both are still registered. Normally there is a reevaluation of a registration every five years and certainly when new information indicating new concerns becomes available.

¶ 258 His department through the World Health Organization has access to virtually all the literature in the field. When the Hardell studies were made public, he visited Hardell in Sweden as part of his work. With all of this together with their own laboratory work, the conclusions of the Department of Health and Welfare Canada are that the levels would be low and aside from misuse or illegal use should not cause any concern as far as the general public is concerned. His department has not recommended any suspension or deregistration of 2,4-D or 2,4,5-T for forestry use.

¶ 267 Dr. Michael Newton, a professor of Forest Ecology at Oregon State University, was qualified as an expert, entitled to give opinion evidence in the areas of forest ecology, silviculture, forest management, environmental effects of herbicides and the application of herbicides in the forest environment. He has done extensive work and study in these fields in Oregon, as well as on a large woodlot that he owns in Vermont and another in Oregon. He has published some 130 research publications and has done extensive consulting work. In all he has spent 22 years in tree farming.

¶ 268 His opinion, offered to the court, is as follows:

1) The risk of using phenoxy herbicides on forest lands is virtually non-existent.

2) Such risk as there is is borne entirely by applicators, who, in accepting this risk, reduce risks that would have to be borne by someone else, and at a greater intensity.

3) Risks off-site owing to action of herbicides are limited to sensitive crops, notably beans and potatoes in Nova Scotia. The incidence of such damage is very rare, minor in extent, and is of no health concern whatever.

4) Considering the array of methods useful for protecting forest regeneration from weeds, herbicides, especially

phenoxys, are uniquely conducive to maximum wildlife production with negligible effects on watersheds and fisheries.

5) Weeding in the boreal forests is necessary as the complement of harvesting to maintain spruces and balsam fir as predominant components in a sustained harvest cycle.

6) There is excellent evidence that chemical weeding methods provide positive and consistent growth responses in Canadian Boreal forests.

7) Evidence is widespread that use of herbicides, especially phenoxys, can be a valuable tool in maintaining both productivity and amenity values in forests.

8) Failure for a forested region to pursue vigorous reforestation and management programs will result in widespread ecological and social disruption.

¶ 269 In his direct testimony he covered most, if not all, of the points mentioned in his 42 page report filed as Exhibit D-26A, Tab 1. To set the stage for his evidence, he first gave certain definitions.

1. Forest Management - the planning administration and execution of forest operations involved in the overseeing and management of a resource area that is dominated by trees with the purpose of preserving the resource in perpetuity.

2. Silviculture - that part of forest management that deals with the manipulation of the trees and the things that depend on the trees to achieve the goals of the owner.

3. Forest Ecology - that part of the science of the relation between plants and organisms and their environment which deals with the forest system.

¶ 270 He then discussed the concept of weeding as it applies to the forest [*page314] and the techniques of carrying it out, i.e., manual release of conifers and herbicidal release. He indicated that herbicidal release is the more effective and it is probably safer and certainly less costly. In Oregon approximately 98 per cent of the conifer release is done by the application of herbicides, normally by aerial spray. The remaining 2 per cent is done by a combination of manual release and hand spraying. He indicated that in five studies in Oregon it was observed that in every case the sudden change of environment occasioned by hand release techniques led to a short-term reduction in growth rather than the desired increase while there was no such decrease in growth with chemical release. He concluded that manual release was not desirable on any substantial scale nor was it effective over a term of years while chemical release with one spray was generally effective for the whole period of growth of the trees, 40 to 50 years.

¶ 271 He testified he had visited a number of the sites in question here just before the trial. He was taken to them by helicopter with Hugh Ross of the defendant and Logan Norris and Frank Dost, all witnesses in this action. Pictures were taken at the locations visited which were introduced as Ex. D-28 and commented on by the witness. Before making these comments, he made the general observation that the state of the forests, conditioned by the spruce bud worm were an "unmitigated disaster". The significance of this is that there is now a tremendous demand on the current inventory of trees and this, coupled with the salvage operations on the dead trees, gives an incentive for immediate and effective reforestation techniques to prevent a future short supply.

¶ 272 In reviewing the photographs he pointed out problem areas, explained the competing growth, largely aspen, raspberry, maple and birch and indicated what he would recommend on each site - some 2,4-D and some a combination of 2,4-D and 2,4,5-T and a few acres where no release at all is necessary.

¶ 273 With regard to the growth of aspen, it was his opinion, that aspen has no value as a resource to a grower because of the large quantity of it and its low value for pulp mills.

¶ 274 One of the photographs of Ex. D-28 was of a site at Fraser Mills which was sprayed in 1982. This photograph showed the effects of the spray and, according to the witness, clearly defined the buffer zones on either side of a water course with the growth indicating that these zones had, indeed, not been sprayed. He commented that buffering is an effective method of keeping biologically important amounts of chemical away from a stream. The U.S.

Environmental Protection Agency recommends a one-half swath width as a buffer zone which permits spraying to the edge of a water way while some states call for a one-swath width which is 50 to 60 feet. Contrasted to this the Nova Scotia requirement is 30 meters which he described as a large and effective buffer which more than meets the requirements of the table of recommended concentrations set out in the Table at p. 13 of his report (U.S.E.P.A. Table of Recommended Concentration Maxima for Silvicultural Chemicals).

¶ 275 With regard to the Skye Mountain area and the adjacent Indian Reservation, it was his opinion that both air drainage, which he explained, and water drainage would have to travel approximately 10 miles, during which there would be perhaps a hundred-fold dilution, before reaching the Reservation, and it would present no risk whatever. He fortified this opinion by indicating he had made a number of studies of the effects of these chemicals in water.

¶ 276 Given the hypothetical that Esteron 3-3E in a mixture of one part Esteron 3-3E to 72 parts water, was to [*page315] be sprayed in August, 1983 in Nova Scotia by aerial, helicopter or ground spray, with a thirty meter buffer zone on each side of any water course, the witness was asked his opinion as to what effect this would have on wild life in the area. His answer was the effect would be absolutely restricted to change of habitat. Under all his studies, the dose picked up by wildlife was a small fraction of the doses recorded by laboratory studies as a maximum safe level, and none was retained in the edible portions of animals consumed by humans. With three pounds per acre of herbicide to be used in Nova Scotia, he would not expect to get more than three parts per million in the stomach of any animal, and again, in his opinion this would be quickly passed through the animal and out into the urine for there is no apparent tendency for the animals to retain the chemical. He concludes from his studies that these chemicals neither bio-accumulate nor biomagnify.

¶ 277 According to his observations, 2,4,5-T and 2,4-D degrade very quickly and in Nova Scotia the conditions between May and October are very favourable for degradation.

¶ 278 On the same hypothetical as set forth above, the witness was asked what would be the effect the spray would have on the quality of the drinking water for persons living adjacent to the spray sites. He answered the effect would be psychological as there would be no biological effect because there simply is not enough material in the water. In his future opinion a person living between one-half mile and four miles from the spray site would have to drink trainloads of water to pick up a maximum safe dose of 2,4,5-T.

¶ 279 The witness and several others did a test on themselves to study skin absorption by strapping a one square foot cloth, saturated with a normally used concentration of spray, to their thighs and leaving it for two hours. The result was that the skin is a very effective barrier for just over two milligrams of the chemical migrated into the body and this amount is one-thousandth of a maximum reported safe level of 2,4,5-T on a daily basis for life. This led him to the conclusion that even the applicators of the spray are under no risk unless they are spraying over their heads with their mouths open. He also testified that because of its binding character even clothing is an effective barrier.

¶ 280 With regard to fish, he would expect a non-detectable concentration and nothing which would be injurious to them. He illustrated this by reference to an accidental spillage of the equivalent of a 55 gallon drum of chemical (200 plus pounds of 2,4,5-T) into a stream some distance upstream of a salmon fish hatchery. There was a sufficient quantity that it made the water froth and smell and the fish were in their most sensitive stage, yet there was no observable effect on the fish.

¶ 281 When asked about plants and berries, the witness indicated that plants immediately respond to the chemical and anyone would know there is something wrong with them and while berries remain edible for 2 or 3 days, one would have to eat extremely large quantities, beyond what one's digestive tract could tolerate, before reaching the maximum recommended safe dose.

¶ 282 In his opinion these chemicals degrade quickly in the soil.

¶ 283 The witness then discussed the various methods of spraying, particularly aerial spray, the opportunity for drift and concluded that there is ample technological information now to avoid essentially all problems in spraying. [*page316]

¶ 284 With regard to manual release, the witness discounted this as an effective method. It would not permit the yield goal, would require more acres to produce a result similar to a sprayed area, would require a large labour force with a high incidence of injuries, and would result in a reduction of the benefits, employment and otherwise, throughout the production system.

¶ 287 The witness admitted that he had testified on behalf of Dow Chemical Company on several occasions as an expert witness although he indicated that any fee was turned over to a research foundation and not retained personally by him.

¶ 288 He indicated that TCDD is more persistent than 2,4-D or 2,4,5-T although its persistence was relative and not terribly persistent in animals. Particularly, it was not very persistent in daylight and quite persistent in a dark place with a half-life of approximately one year in microorganisms and a biological half-life of 17 days in some of the animals studied.

¶ 289 With regard to toxicity he said he was familiar with the literature and used all the information to determine the safety factors set forth at p. 344 of the article by himself and Dr. Norris appended to his expert report. He defined the safety dosage factor as the number of doses tolerated before some effect is produced and identified based upon the most sensitive indicator.

¶ 290 It was his view that the concentration of TCDD in 2,4,5-T is overwhelmed by the 2,4,5-T itself in its effects and the preponderance of the literature makes it abundantly clear that TCDD does not contribute to the toxicity of 2,4,5-T until the contamination level exceeds several parts per million and the current product commercially prepared contains only several parts per billion.

¶ 291 When directed to breathing these chemicals, the witness testified that inhalation does not contribute significantly to exposure.

¶ 292 Dr. Newton then explained the detection level itself, particularly with regard to the deer he had studied, and explained that with a detection level of .006 milligrams per kilogram, anything indicated to be below that level was non-detectable and could in fact be zero, but because of the inability of science to measure to zero, it was merely indicated to be below .006, i.e. non-detectable.

¶ 293 Dr. Newton was an impressive witness, obviously well qualified and extremely knowledgeable in his area of expertise. He was professional in his manner, and competent and scientific in his approach to the evidence he was giving. I found his evidence to be quite helpful.

¶ 403 Dr. Steven Lamm, a consultant in epidemiology, was qualified as an expert entitled to give opinion evidence in the field of medical epidemiology with particular reference to methods and interpretation, all with regard to 2,4-D and 2,4,5-T and their contaminants. His expert report is filed as D-26-D Tab 9.

¶ 404 He concluded, after a review of the germane toxicological and epidemiological literature relating to carcinogenic and reproductive (including teratogenic) risk potentials for 2,4-D, 2,4,5-T and TCDD, that there would no significant adverse human health effects from the proper use of these chemicals as proposed. Those environmentally exposed would be risk free and those occupationally exposed would have a minimal risk well within acceptable standards giving a wide margin of safety.

¶ 405 It was his view that there was sufficient studies both as to teratology and reproductive loss and carcinogenity from these chemicals that judgmental assessments and estimates of magnitude of potential risk from exposure can be developed.

¶ 406 He then made three judgmental assessments.

1. There is no evidence that TCDD or phenoxy herbicide causes human birth defects. The NOEL (No Effect Level) for

teratologic effect for these chemicals is:

2,4-D - 25 mg./kg. bw/day

2,4,5-T - 20 mg.kg bw/day

TCDD - 0.03 ug./kg. bw/day

2. There is no evidence that the use of phenoxy herbicides has caused human pregnancy loss. The NOEL for reproductive effects is the same as that for teratologic effect, while that for TCDD is 0.001 ug./kg. bw/day.

3. There has been some human epidemiological evidence that soft tissue sarcomas may be caused by TCDD at least at levels sufficient to cause chloracne. These observations have only been made in workers occupationally exposed in the 1950's to 1960's to these chemicals with concentrations of TCDD far greater than that found in today's products. Animal studies give a NOEL for cancer of 0.001 ug./kg. bw/day.

¶ 407 There is no evidence of weight from epidemiology or toxicology that 2,4-D or 2,4,5-T cause any cancer.

¶ 408 From these conclusions he then developed a quantitative risk assessment of carcinogenic risk and a safety estimate for the margin of safety for teratogenic or reproductive effect.

¶ 409 For purposes of assessment he assumed the validity of the Hardell study and that TCDD was the active ingredient. He then calculated the annual risk of developing soft tissue sarcoma on the basis of exposure similar to that of the 1950-1960 period as 3.3 x 10 -8 /year, i.e. 1 in 30 million. He said this risk is a conservative estimate and is still decidedly small and falling well below that level which regulatory agencies choose to regulate. If 20 persons were involved in doing the spraying, there would be an estimated risk of one soft tissue sarcoma every 1.5 million years. Persons environmentally exposed would have markedly less exposure (usually about one-thousandth) and an annual risk of 1 in 30 billion (3.3 x 10 -11).

¶ 410 Taking the lower NOEL for reproductive effect of TCDD, and the NOEL for 2,4-D and 2,4,5-T referred to in his judgmental assessments, he determines that for the herbicide applicator [*page331] the annual carcinogenic risk is 3.3 x 10 -8 , the teratogenic potential has a safety factor of 300 to 50,000, and the reproductive potential has a safety factor of over 1,600. For those environmentally exposed the carcinogenic risk becomes 10 -11 ; the teratogenic potential has a safety factor of over 300,000; and the reproductive safety factor is 1,600,000.

¶ 411 His occupational safety factors were based on the assumption that all workers were pregnant females and that exposure occurred during the period of maximum fetosensitivity to birth defect injury.

¶ 412 to illustrate the margin of safety here he indicated that safety values of 100 are usually considered more than sufficiently protective.

¶ 413 He concluded his report by stating that all the risks he considered - occupational and environmental - are well below the levels at which regulatory agencies choose to concern themselves.

¶ 414 In his direct testimony he reviewed the contents of his written report and commented on the literature he used to make his calculations as well as commented upon a number of other studies referred to him. He also criticized the Hardell study, and its methodology. He answered a number of criticisms which Dr. Eriksson had made of his report. One of the significant points made was that there was so much similarity in the Hardell results that the methodology becomes suspect. Normally a specific chemical will have certain specific effects according to cancer epidemiology, yet Hardell's study comes up with essentially the same relative risk for malignant lymphomas, nasal carcinomas and soft tissue sarcomas. There are at least a dozen different cancers within the malignant lymphoma group and up to 50 different types of cancer in the soft tissue sarcoma group. His point in this regard is that the experience has been that one or at most two cancers appear as the high risk cancer for a specific relative exposure and when a study shows a specific chemical to appear to cause everything, it becomes questionable.

¶ 415 In looking at cohort studies to test the Hardell hypothesis, it was his view that these studies showed no significant excess of cancer. In his view these cohort studies are the best studies in that there are high exposures (generally factory accidents) and well defined cohorts. From these he indicated that he found no evidence supporting the lymphoma hypothesis, some suggestion to support the soft tissue sarcoma (but only in the group with massive exposure due to accident) and, among the user group, no support for soft tissue sarcoma.

¶ 416 With regard to birth defects he indicated no changes have been found in the United States in the incidence of various birth defects both nationwide and geographical despite the fact that phenoxy herbicides have been banned in forest use since 1979. If it were causal, then a drop should have been noticed in the Pacific Northwest, yet none has been observed.

¶ 417 In cross-examination the witness was referred to numerous statements and reports and he indicated that while there is some suggestion of a causal relationship between phenoxy herbicides and soft tissue sarcoma, he does not think that the data at this time warrants a conclusion of causal relationship. He further stated that the more he looks at these studies (suggesting a causal relationship) and the way they were done, he is less impressed with the support for a causal relationship.

¶ 443 Dr. Robert Kilpatrick, a practicing physician in England, Dean of Medicine at the University of Leicester, specializing in internal medicine and clinical pharmacology, and Chairman of the Advisory Committee on Pesticides and other Toxic Chemicals in the United Kingdom, was qualified as an expert witness entitled to give opinion evidence in the field of internal medicine, pharmacology, toxicology, the regulatory process and the safety of the herbicides 2,4-D and 2,4,5-T if used in the prescribed manner. His expert report was filed as Exhibit D-26-E.

¶ 444 He first described dose-response curves and how they are determined, as well as "no-effect levels" and their importance in the regulatory process. Then he was directed to the three reports contained in his report, Exhibit D-26-E and he testified as to each one.

¶ 445 First was the March 1979 review which arose from allegations of possible harm and an increased concern, worldwide, about 2,4,5-T and its contaminant TCDD. The Committee had previously approved its use as prescribed over a number of years. By March 1979 the [*page335] detectable limit was 100 parts per billion (the present level of detection in the U.K. is 10 parts per billion).

¶ 446 Four questions were posed and answered in the review:

1. Is pure 2,4,5-T safe (i) for spraying and (ii) if treated material is burnt? (Pure meant below the level of detection).

2. Can any TCDD (as an impurity) be tolerated in 2,4,5-T and if so, how much?

3. Do any products on the U.K. market exceed this tolerance?

4. Can any of the allegations about the risks attendant on the use of 2,4,5-T herbicide be substantiated?

¶ 447 To answer these questions, a complete review of the literature from the time of the last review was made and allegations of harm to individuals were investigated.

¶ 448 The answers given were as follows:

Question 1. No harm to humans and wildlife at that level of 100 parts per billion and no perceptible additional risk when treated material burnt.

Question 2. 2,4,5-T contaminated with TCDD to the level of 100 parts per billion can continue to be used with risk.

Question 3. No.

Question 4. Such allegations were not sufficiently documented or, where they have been thoroughly investigated, canont be substantiated.

¶ 449 The conclusion of this review was 2,4,5-T could continue to be safely used, even with a TCDD contaminant to a level of 100 parts per billion.

¶ 450 In 1980 there were a very large number of individuals alleged to have been harmed by 2,4,5-T and because of this and trade union pressure another review was made. Everyone alleged to have been harmed was investigated. As well, all new literature was reviewed. The Alsea II study was considered and the review concluded that "the data reported in this study were not valid in either scientific or statistical terms".

¶ 451 With regard to the cases studied, the alleged harm did not stand up to critical investigation. While most of the cases related to miscarriages and birth deformities, they were found, on investigation, not to be related to or could not have been caused by 2,4,5-T.

¶ 452 The conclusion of the committee in this review, set forth at page 25 of the Review was that they could find no sound medical or scientific evidence that humans or other living creatures, or our environment, would come to any harm if cleared 2,4,5-T herbicide continued to be used in the U.K. for the recommended purposes and in the recommended way.

¶ 453 Following this the Committee was asked to consider the matter again in 1982 and a report, by way of letter to the Minister of Agriculture, Fisheries and Food, was filed. The letter was dated December 13, 1982, but was not released until January 1983. In it the Hardell studies are referred to and criticized in some detail saying that the procedures used for establishing the study groups and some aspects of the collection of the exposure data "were not reliable". [*page336]

¶ 454 The witness in his viva voce evidence further outlined what he considered to be defects or flaws in the Hardell studies, such as recall bias, the controls, the failure to consider socio-economic groups, the lack of any indication of exposure related to duration of disease and the exposure of one day as his starting level. He also expressed surprise that, with a relative risk factor of five as Hardell found, there was not an obvious increased incidence of soft tissue sarcoma.

¶ 455 Returning to the letter, the Committee indicated that it found no reason to alter its previous recommendations. It added that it has been unable to find one case of a softtissue sarcoma having developed from the use of 2,4,5-T in the U. K. Further, in the studies considered, particularly Hardell's, the deficiencies cause an artificial raise in the relative risk factor. In fact, when these are taken into account Hardell's corrected risk factor would not be out of line.

¶ 456 He did not agree with Dr. Eriksson's opinion that cancer was on the increase and stated that when the apparent risk is corrected for population structure, it usually shows that cancer is not on the increase. By changing population structure, he means that people are living longer and other diseases have changed or reduced. He did agree that about 80% of cancer is environmentally caused but qualified "environmental" to mean all environmental factors, including sunlight, radiation, nutrition, etc.

¶ 457 Dr. Kilpatrick stated definitely that the position of the Advisory Committee as of the date of his giving evidence was that these chemicals are safe when used in accordance with the directions on the label.

¶ 458 From a toxicological point of view, the witness indicated that it is generally accepted that there is a doseresponse relationship for cancer. He was not able to state that the dose response goes through zero, but he did indicate that as you get towards zero the response produced gets less and less.

¶ 459 When asked the hypothetical question based on the chemicals 2,4-D and 2,4,5-T and their quantity, as well as the TCDD contaminant of .1 part per million, the mixture to be used and the area to be sprayed in Nova Scotia together with the nearness of water courses and residences, the witness indicated that there was no hazard.

¶ 460 With the same hypothetical but based on 2,4-D only, the witness answered there would be, in his opinion, no

health hazard.

¶ 461 One of the final points made by this witness was that the literature indicates there are many sources of TCDD in the environment other than from phenoxy herbicides.

¶ 462 On cross-examination the witness indicated that it was his committee's view that every encouragement should be given to an effective and acceptable alternative to 2,4,5-T for forestry use basically because the TCDD is a contaminant with no useful purpose.

¶ 463 He maintained that the Advisory Committee was truly independent and free of any conflicting interests.

¶ 464 He was cross-examined at great length on such matters as related to the method of spray application in the U.K., the U.K. medical system, no-effect labels, the production of chlorinated dioxins by incineration of refuse and fossil fuels, TCDD accumulation and its half-life, some conflicting reports, the Hardell studies and the like, but he did not retreat in any way from his direct testimony and written expert report. He did accept that it was their position that there is a case to answer [*page337] and further evidence was required.

¶ 465 When questioned about dose response, the witness made the interesting point that in dealing with a carcinogen, when dosage is decreased, it may take longer than a lifetime to produce the tumor which would make it of statistical interest only. It gets more and more difficult to prove a chemical harmful as one gets lower down a dose-response relationship. He suggested that when considering all the laboratory work done on these chemicals and the adverse results shown, you must always consider that in relation to the amount used. His position was clearly they were safe for use in the recommended way and for the recommended purposes.

¶ 466 He also agreed that 2,4,5-T was banned in a number of countries as well as by a number of the local authorities in the U.K., but insisted that none of these banned its use on medical scientific evidence with the possible exception of Sweden. Incidentally, other evidence indicates Sweden first restricted its use before the Hardell studies were published.

¶ 467 Three main issues were presented to **the court**:

(1) The representative action was not a proper action, under the Civil Procedure Rules and the case law;

(2) Whether the native Indians have aboriginal rights on lands outside their reservations; and

(3) Whether an injunction should be granted to restrain the proposed spraying.

[The Court’s reasoning on the first two issues is omitted]

¶ 510 Finally, I now turn to the third issue, indeed the main issue, whether or not the plaintiffs are entitled to injunctive relief.

¶ 511 The relief claimed is set forth in the statement of claim as follows:

(b) a permanent injunction enjoining [*page343] the defendant from spraying the phenoxy herbicides 2,4-D and 2,4,5-T at the sites,

(c) a declaration that the plaintiffs have the right to be free of exposure to the phenoxy herbicides 2,4-D and 2,4,5-T.

(d) the costs of this action,

(e) such other relief as this Honourable Court thinks just.

¶ 512 The legal causes of action on which the relief is claimed are alleged to arise from the proposed spraying by the defendant and fall within the following categories:

(i) private nuisance;
(ii) trespass to land;
(iii) the rule in Rylands v. Fletcher;

(iv) the right of riparian owners to water undiminished in quality;

¶ 522 With regard to these causes of action alleged by the plaintiff, I have been referred to volumes of cases by both sides for assistance as to what constitutes the various causes and what each includes, including some cases from American jurisprudence. However, it is unnecessary for me to review the whole of the law in these areas.

¶ 523 The essence of private nuisance is explained by Salmond and Heuston on The Law of Torts (18th Ed.), at p. 48:

"The generic conception involved in private nuisance may really be found in the fact that liability in nuisance flows from an act or omission whereby a person is annoyed, prejudiced or disturbed in the enjoyment of land, whether by physical damage to the land or by interference with the enjoyment of the land or with his exercise of an easement, profit or other similar right or with his health, comfort or convenience as occupier of such land."

¶ 524 Blackstone's Commentaries, Book III, c. 13, p. 1202 defines private nuisance as:

"...anything done to hurt or annoyance of the lands, tenements or hereditaments of another."

¶ 525 Street on Torts (5th Ed.), at p. 212 states:

"The essence of the tort of nuisance is interference with the enjoyment of land."

¶ 526 In Fairview v. City of Dartmouth, 40 N.S.R.(2d) 313; 73 A.P.R. 313, Pace, J.A., in considering the tort of nuisance states at p. 323:

"In my opinion the tort of nuisance is complete when there is an unreasonable interference with the enjoyment of land and it need not be accompanied by negligence. Nuisance by its very nature is concerned with the intrusion or invasion of the interest in land, while in negligence the conduct of the party complained of is of vital importance. A person may create a nuisance to his neighbour without any negligence, or again, as is most frequently found in nuisance cases, the person creating the nuisance may act intentionally to carry out a laudable undertaking and without any negligence be liable to his neighbour."

¶ 527 The law is clear, however, that only some substantial interference with a person's enjoyment of property gives rise to an action in nuisance. Turpin v. Halifax-Dartmouth Bridge Commission (1960), 21 D.L.R.(2d) 623.

¶ 528 Equally clear is the requirement that there must be proof of damage. In Halsbury's Laws of England (4th Ed.), vol. 34, p. 105 this proposition is put as follows:

"The damage need not consist of pecuniary loss but it must be material or substantial, that is, it must not be merely sentimental, speculative or trifling, or damage that is merely temporary, fleeting or evanescent ...."

¶ 529 In the present case the allegation is that these offending chemicals, if they get to the plaintiffs' land, will interfere with the health of the plaintiffs thereby interfering with their enjoyment of their lands. Clearly such an interference, if proved, would fall within the essence of nuisance. As a serious risk of health, if proved, there is no doubt that such an interference would be substantial. In other words, the grounds for the cause of action in nuisance

exist here provided that the plaintiffs prove the defendant will actually cause it, i.e. that the chemicals will come to the plaintiffs' lands and that it will actually create a risk to their health. With this, I will deal later. [*page346]

¶ 530 Trespass to land, on the other hand, does not require proof of damage and is actionable per se. As stated in Salmond and Heuston on The Law of Torts, supra:

"It is a trespass [to land] to place anything upon the plaintiff's land, or to cause any physical object or noxious substance to cross the boundary of the plaintiff's land, or even simply to come into physical contact with the land ... ."

¶ 531 Again there is no doubt in my mind that, if it is proved that the defendant permits any of these substances on the plaintiffs' lands, it would constitute a trespass and be actionable. This very question was considered by Dickson, J., in Friesen et al. v. Forest Protection Limited, supra, at pp. 161-2, and I fully adopt these comments as applicable to the present case:

"Trespass may be described as a wrongful act done in disturbance of the possession or property of another or against the person of another, against his will (38 Halsbury (3rd Ed.) at p. 734). And again, every unlawful action by one person on land in possession of another is a trespass for which an action lies although no actual damage is done (ibid, p. 739). And that a person does not know an act to be wrongful makes him no less a trespasser. To throw a foreign substance on the property of another, and particularly in doing so to disturb his enjoyment of his property, is an unlawful act. The spray deposited here must be considered such a foreign substance, and its deposit unquestionably amounted to a disturbance, however slight it may have been, of the owner's enjoyment of their property. I therefore must conclude that the defendant, in depositing the spray did in fact commit what would, in the absence of statutory authority, be considered a trespass. This of course does not involve any question of whether or not the spray may have been toxic or nontoxic, because even to have thrown water or garbage or snow or earth tippings, or any substance on the property would equally have amounted to an act of trespass."

¶ 532 Again, entitlement to a remedy, will be based upon proof as to whether such substances will be deposited on the lands of the plaintiff.

¶ 533 While it is out of place in considering the various alleged causes of action, I shall deal with the defence that the depositing of spray, even if some is deposited on the lands of others, is justified by statutory authority, namely the Nova Scotia Pulp Limited Agreement Act, S.N.S. 1958, c. 9, which Act provides for a forest management plan by which the defendant undertakes and is committed to perform certain silvicultural activities on Crown lands. Without referring in any detail to the provisions of that Act, it is sufficient to state that nothing in that Act permits the committing of nuisance or trespass to others nor does it in any way purport to remove any of the common law rights of adjacent property owners. For this defence to succeed the legislature would have to expressly sanction the spraying activity and, after proof that it was used for the purpose authorized with every precaution observed to prevent injury, then for any damage resulting, independently of negligence, the party causing the damage would not be responsible. For a full discussion of this defence see Friesen et al. v. Forest Protection Limited, supra, pp. 162-169.

¶ 534 As a result there is no merit to this defence in the present action.

¶ 535 The rule in Rylands v. Fletcher (1868), L.R. 3 H.R. 330, is a simple one, of long standing in English jurisprudence. The rule is stated in Salmond and Heuston on The Law of Torts (18th Ed.), supra, at p. 297: [*page347]

"This principle is one of the most important examples of absolute or strict liability recognized by our law - one of the chief instances in which a man acts at his peril and is responsible for accidental harm, independently of the existence of either wrongful intent or negligence. The rule may be formulated thus: The occupier of land who brings and keeps upon it anything likely to do damage if it escapes is bound at his peril to prevent its escape, and is liable for all the direct consequences of its escape, even if he has been guilty of no negligence."

¶ 536 No elaboration of the rule or applications of it are necessary. It is for the plaintiffs to prove the constituent elements - its likelihood to do damage, its escape and the direct consequences.

¶ 537 I shall reserve the matters of riparian rights and groundwater rights until after I deal with the burden of proof.

¶ 538 The complete burden of proof, of course, rests upon the plaintiffs throughout for all issues asserted by them. If the spraying had actually occurred, they would have to prove by a preponderance of probabilities the essential elements of either or all of the alleged causes of action as I have set them out. However, the spraying has not occurred and this application is for a "quia timet" injunction. This can be translated as "which he fears". In other words, a plaintiff does not have to wait until actual damage occurs. Where such damage is apprehended, an application for a "quia timet" injunction is an appropriate avenue to obtain a rremedy which will prevent the occurrence of the harm. That remedy also, however, is not without its limitations.

¶ 539 In Attorney General v. Corporation of Manchester, [1893] 2 Ch. 87, Chitty, J., states at p. 92:

"The principle which I think may be properly and safely extracted from the quia timet authorities is, that the plaintiff must show a strong case of probability that the apprehended mischief will, in fact, arise."

¶ 540 This passage was approved by Anglin, J., in the Supreme Court of Canada in Matthew v. Guardian Insurance Company (1918), 58 S.C.R. 47, at p. 61 and is still the proper principle to consider in an application of this kind.

¶ 541 It was argued by the plaintiffs that the principle as expressed in Salmond and Heuston on The Law of Torts, supra, is more appropriate. At p. 555 they state as follows:

"In all cases, however, it seems necessary that there shall be a sufficient degree of probability that the injury will be substantial and will be continued, repeated, or committed at no remote period, and damages will not be a sufficient or adequate remedy."

¶ 542 It was suggested that this demonstrates that there is no set standard of proof that has to be met - simply that the risk that the plaintiff's right will be breached be significantly great in all the circumstances.

¶ 543 I fail to see the difference suggested. A "strong case of probability" and "a sufficient degree of probability" create only a semantic difference and not a difference in substance. I prefer the former as more clearly setting forth the proper principle. This does not impose an impossible burden nor does it deprive the court of its ability to consider the balance of convenience or inconvenience or hardship between the parties nor the size or amount of the injury or distress which might occur. All of these [*page348] are factors which are woven into the fabric of "a strong probability" and are considered in determining whether the burden or proof has been met.

¶ 544 The plaintiffs must, however, prove the essential elements of a regular injunction, namely irreparable harm and that damages are not an adequate remedy as they are also essential elements of the "quia timet" injunction.

¶ 545 Finally, any injunction is a discretionary remedy and sufficient grounds must be established to warrant the exersice by the court of its discretion.

¶ 546 I am satisfied that a serious risk to health, if proved, would constitute irreparable harm and that damages would not be an adequate remedy. Further, recognizing the great width and elasticity of equitable principles, I would have no hesitation in deciding that such a situation would be one of the strongest which would warrant the exercise of the Court's discretion to restrain the activity which would create the risk.

¶ 547 This matter thus reduces itself now to the single question. Have the plaintiffs offered sufficient proof that there is a serious risk of health and that such serious risk of health will occur if the spraying of the substances here is permitted to take place?

¶ 548 Before answering this question, there are a number of matters which must be considered.

¶ 549 This action is to restrain the spraying of two particular phenoxy herbicides, namely 2,4-D and 2,4,5-T and perhaps a third, if a mixture of 2,4-D and 2,4,5-T known under the trade name Esteron 3-3E is considered to be a separate herbicide. More particularly, the evidence submitted by the defendant indicated a specific quantity and mix of these substances which it intended to use and it is this evidence which is of relevance to the issues here.

¶ 550 Because of the nature of the issues in dispute, the witnesses produced and the testimony given, the enormous publicity attached to the trial and the public interest involved, the evidence went far beyond the particular substances involved and related to all the phenoxy herbicides and their derivatives. The whole trial took on the aura of a scientific inquiry as to whether the world should be exposed to dioxins. Scientists from all over North America, as well as from Sweden were called and testified. Scientific reports and studies from scientists the world over were filed as part of the evidence. In order to give both sides full opportunity to present their cases fully, it was necessary to grant this latitude although both parties were aware that the final decision would have to relate to the particular facts between the parties before the court.

¶ 551 As to the wider issues relating to the dioxin issue, it hardly seems necessary to state that a court of law is no forum for the determination of matters of science. Those are for science to determine, as facts, following the traditionally accepted methods of scientific inquiry. A substance neither does nor does not create a risk to health by court decree and it would be foolhardy for a court to enter such an enquiry. If science itself is not certain, a court cannot resolve the conflict and make the thing certain.

¶ 552 Essentially a court is engaged in the resolution of private disputes between parties and in the process follows certain time-honoured and well-established procedures and applies equally well-established principles of law, varying and altering them to adjust to an ever-changing society. Part of the process is the determination of facts and another part the application of the law to those facts, once determined, [*page349] and designing the remedy. As to the occurrence of events, the court is concerned with "probability" and not with "possibility".

¶ 553 I have, as I have said, set out a reasonably complete summary of the evidence heard by me, as well as some of the documentary evidence and I have done so specifically because of the nature of this trial, recognizing that it may be somewhat unorthodox to set out the evidence in such detail when there is so much. I believe it to be important in this case, that it be obvious just what the evidence was so that the approach and reasoning of the court be more easily understood.

¶ 554 While this dispute is between the parties to the action and will depend upon the facts and their relevance as I find them, this case is unique by its nature and the world-wide interest in its result. Apparently it is the first time that the leading scientists of opposing views regarding dioxin have met in court where the chemicals involved are at the very centre of the dispute.

¶ 555 As a result, this is one of those very rare cases where, I believe, the court owes a duty which goes beyond the parties, to make clear its findings so as to give some assistance, one way or another, which will help the understanding of those so vitally interested in this substance.

¶ 556 By way of background, the phenoxy herbicides are a group of herbicides which have been widely used in many parts of the world since the late 1940's and early 1950's. They are selective in that they affect only certain types of vegetation, namely broad-leaved plants, and have no, or little effect on conifers. They are a family of compounds having similar chemical and biological properties but each member compound differs in effect on individual plants. The two family members concerned here are 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid).

¶ 557 2,4,5-T and all of its derivative herbicides contain a chemical contaminant formed in the manufacturing process, unavoidably to the present time and of no value, known as 2,3,7,8-tetrachlorodibenzo-p-dioxin and referred to as TCDD. Originally the quantity of TCDD in the manufactured product was in the range of 80 parts per billion but, over the years, improvements in manufacturing techniques have reduced this amount to less than 0.1 parts per million. In fact the present supply of 2,4,5-T held by the defendant has been formulated with a TCDD content indicated as "non-detectible" at 0.01 parts per million.

¶ 558 The herbicide 2,4-D is free of any TCDD contamination.

¶ 559 Both herbicides have been widely used in Canada, and elsewhere, upon agricultural crops, forests, roadside and railroad rights-of-way in vast quantities and, only until recently, without too much precaution. In comparison to other uses, forestry has used only a small percentage of the total used.

¶ 560 Phenoxy herbicides are used to discriminate between the unwanted plants and those desired to be retained or encouraged. In a forestry site they are used to release the young conifers from competition. In Nova Scotia those competitors are the hardwoods, such as aspen, birch and maple, and raspberry. Their use is not designed to kill all competition but to permit light to reach the young conifers and decrease competition for soil nutrients and moisture. After a few years the young conifers will outgrow the competition and be permanently released. In Nova Scotia the evidence is, and I accept it, that the situation requires one treatment, and perhaps a second [*page350] after 3 to 5 years, over a forty-year period.

¶ 561 Treatment is usually applied in early summer when susceptibility is greatest but it can be applied in late summer or early fall.

¶ 562 The benefits, of course, are a greater yield over a shorter period of time of the conifer forest.

¶ 563 The contaminant TCDD is one of the most toxic chemicals known to man. One witness described it as "exquisitely toxic". Much has been written on TCDD and its effects. It has been indicated that there are upwards of 40,000 different articles on the subject. A great number of those were submitted to the Court. It is not my intention to summarize those, nor is it necessary, as almost every scientific witness as well as the regulatory agencies have already done that. While there are opposing views, and the whole field is not without some uncertainty, there is no dearth of writings. So there can be no doubt that everything submitted was considered by me, I have read every article submitted to me, a formidable task in itself, and I now join the group who has reviewed all the relevant literature, although I am far from convinced that this volume of documents was necessary for this case.

¶ 564 Having mentioned regulatory agencies, it is appropriate to indicate that most countries, including Canada, have regulatory agencies, whose function it is to regulate and control the use of new chemical compounds before they are exposed to the environment. In Canada this is done through various divisions of Health and Welfare Canada. Drs. Riedel and Krewski, two of the defendant's witnesses, testified in this area. In the United Kingdom there is the Advisory Committee on Pesticides and other Toxic Chemicals of which Dr. Kilpatrick, another of the defendant's witnesses, is Chairman. In the United States, their legislation in this field and the approach taken is so different that little help can be gained by further considering the E.P.A.

¶ 565 As to Canada and the United Kingdom, both have registered 2,4-D and 2,4,5-T for forestry use with a maximum TCDD level of 0.1 parts per million. Registration for use in Canada for 2,4-D was in 1947 and 2,4,5-T in 1952. In both jurisdictions reviews are made periodicially after reviews of the literature and independent study by highly trained and competent scientists. The evidence indicates this to be an ongoing process. In both countries registration for use is still in effect and neither jurisdiction has accepted that there are valid studies which would cause them to cancel the registration.

¶ 566 The provincial Department of Environment is also involved in this ongoing process as it relates to Nova Scotia and that department has not registered the use of these herbicides.

¶ 567 I do not mention regulatory agencies of other countries but there are some countries, notably Sweden, where 2,4,5-T is either restricted or prohibited. However, I have no evidence before me indicating that any such restriction or prohibition is the result of a scientific inquiry. All seem to be political decisions made for whatever reason. Even in the United States no such inquiry has been made and completed. Those decisions, therefore, are of no help to me.

¶ 568 To some extent this case takes on the nature of an appeal from the decision of the regulatory agency and any such approach through the courts ought to be discouraged in its infancy. Opponents to a particular chemical ought to direct their activities towards the regulatory agencies or, indeed, to government itself where broad [*page351] areas of social policy are involved. It is not for the courts to become a regulatory agency of this type. It has neither the training nor the staff to perform this function. Suffice it to say that this decision will relate to, and be limited to, the dispute between these parties.

¶ 573 The spray containers contain directions regarding the use of the chemicals and I am satisfied that the defendant is entitled to ask the court to assume that any spraying will be done in accordance with those directions. See

Attorney General v. Corporation of Nottingham, [1904] 1 Ch. 673. The defendant is further entitled to ask for the assumption that its conifer release program will be properly managed and that all scientific skill and knowledge usually associated with such a program will be used.

¶ 574 Indeed, I make both of those assumptions. Clearly the quia timet injunction would not be available if the grounds were only an allegation of possible misuse or anticipated unauthorized or illegal use.

¶ 575 Having accepted Mr. Ross' testimony and accepting the evidence of Donald Freer and Exhibit D-70 that the defendant's supply of 2,4,5-T is formulated with a TCDD content of "non detectible" at 0.01 parts per million, it is obvious that the amount of TCDD to be sprayed in Nova Scotia by the defendant is infinitesimally small. After hearing Lt. Col. Thalken I do not accept Dr. Wulfman's suggestion that storage may affect the product in any way.

¶ 576 It is, therefore, in the light of this concentration of TCDD that I must consider whether the plaintiffs have met the burden of proof.

¶ 577 The plaintiffs, I should add, also claimed a second family of evil contaminants of both 2,4-D and 2,4,5-T besides the dioxins and those were the family of "furans" or, more properly, the dibenzofurans. They also alleged that there was still the possibility of risk to health from, if I might use the term, pure 2,4-D and 2,4,5-T. However, almost the whole of the evidence related to TCDD. I am satisfied that I have not heard sufficient evidence of a probable risk to health of the furans or pure 2,4-D or pure 2,4,5-T to warrant any fear of risk to health, particularly when considering the quantity of these substances to be used here. The totality of evidence in this regard does not even come close to establishing any probability, let alone a strong probability, of risk to health to warrant the granting of quia timet injunctive relief.

¶ 578 A great deal of the evidence submitted related to animal studies where TCDD were reported to have caused [*page352] various effects indicating it to be, among other things, fetotoxic, teratagenic, carcinogenic and to cause immunological deficiencies, enzymatic changes, liver problems and the like. Also it is alleged to bioaccumulate and be persistent both in soil and in tissue. I do not pretend to have included all of its effects, but those are the most major. I was asked to make findings of fact in all of these areas, but I decline to do so. Nothing would be added to the body of scientific fact by any such determination by this court. That TCDD has had all of these effects is undoubtedly true in the experiments described, but, in every case, the effect must be related to dose. In the animal studies the doses are extremely high and, in all cases, many, many thousands of times greater than any dose which could be received in Nova Scotia.

¶ 579 Human studies are a different matter because actual testing is not an acceptable process in our society. I do note that Dr. Newton, another of the defence witnesses, participated in some actual testing on himself and several others, without any apparent harmful result. The human information comes from a number of studies made in various countries of the world. Some resulted from industrial accidents, some from the Vietnam experiences and some from massive industrial exposure. It was in this area that a great deal of the evidence was directed. I am satisfied that in all these cases the exposure was massive, either through accident or industrial exposure or the Vietnam War.

¶ 580 The use of these products in Vietnam bears some comment. They are defoliants and their use in Vietnam was to defoliate areas as completely and as quickly as possible. As a result, Agent Orange, a mixture of 2,4-D and 2,4,5-T, was sprayed without dilution in massive quantities as were Agent White, 2,4-D with pichloran (this latter contained no known dioxins). Between 1967 and 1970, 44 million litres of Agent Orange and 20 million litres of Agent White were sprayed on South Vietnam. After this the Vietnamese sprayed the considerable remaining stocks. This is the famous operation Ranch Hand.

¶ 581 While these amounts were sprayed pure and without dilution and without too much care, there is no scientifically acceptable proof of risk to health. There are studies, particularly Dr. Long's, which suggest some effects but these studies are widely accepted as flawed and therefore prove nothing. There are other contradictory studies suggesting no evidence of risk to health. Therefore, despite the quantity of these substances released into the Vietnam environment, there are, at present, no conclusions which can be drawn. One outstanding point, however, is that with the quantity used and the toxicity alleged, one would have expected that, at least, some of the alleged effects would have been obvious.

¶ 582 The studies from which the opponents of dioxin gained the most support and entrenched their position were the Hardell studies in Sweden. They are amply referred to by many of the witnesses and I do not intend to repeat them or discuss them in detail. They claim to prove an increased carcinogenic risk from exposure. It is not for the court to accept or reject their validity, but despite the defence of the validity of the studies by Dr. Erikkson I am satisfied that a substantial group of informed scientists, including those who testified on behalf of the defendant, consider them to be flawed and unacceptable as proof of the results claimed. I do not deny their importance nor the zeal of the scientists involved in them, but I accept the evidence which indicates that they cannot be taken at face value. I am not alone in this regard as the United Kingdom Advisory Committee has not seen fit to alter its course as a result of these studies nor has the [*page353] defendant's witnesses as representing the generally accepted view of responsible scientists, and also as indicative of the risks involved. Each of them categorically states that neither 2,4-D nor 2,4,5-T, nor the concentration of TCDD presently in 2,4,5-T, nor the mixture of 2,4-D and 2,4,5-T in the concentrations to be sprayed on Nova Scotia forests pose any health hazard whatsoever. I am unable to accept that the plaintiffs have proved any strong probability or a sufficient degree of probability of risk to health to warrant the granting of the remedy sought, a quia timet injunction.

¶ 597 Having made this finding, it is unnecessary for me to consider the matter of riparian rights or groundwater rights. Since I have accepted that no risk to health has been proved, I need not consider these areas. Were I required to do so, and perhaps to allay public fears, I will add that the strongest evidence indicates that these substances sprayed in the Nova Scotia environment will not get into or travel through the rivers or streams, nor will they travel via groundwater to any lands of the plaintiffs who are adjacent to or near the sites to be sprayed.

¶ 598 Further, if any did the amount would be so insignificant that there would be no risk.

¶ 599 I need not consider whether any particular area need be sprayed, whether other substances should be used, or whether manual release is a better approach. While considerable evidence was adduced in this regard, it is not the court's function to direct how the defendant should manage its affairs or carry out its activities. My only concern is whether or not the defendant should be restrained from the proposed activity. While those factors may have been considered in the wide discretionary area if an injunction were to be granted, they do not arise when the plaintiffs have not proved the grounds for an injunction.

¶ 600 There is, accordingly, no nuisance, real or probable. As to trespass, none has been proved as probable to occur. Possibilities do not constitute proof. Similarly, there has been no basis established for the application of the rule in Rylands v. Fletcher, as neither the danger of the substance nor the likelihood of its escape to the plaintiffs' lands has been proved.

¶ 601 Therefore, the answer to the single remaining question I posed earlier which has two parts - have the plaintiffs offered sufficient proof that there is a serious risk of health and that such serious risk of health will occur if the spraying of the substances here is permitted to take place - is, for each part, in the negative.

¶ 602 One final comment is warranted. This decision could have been much shorter and, I am sure, the parties would have accepted that it was on the basis of the evidence presented, and that all was considered. It was obvious throughout that the subject is of vital interest to the public. It still is, as is evidenced by events in this province after the trial itself. For this reason I felt it incumbent upon me to set forth this detail of fact and my own observations so as to make clear that all the evidence available has been presented by the parties, and that, based on this evidence, fully weighed and considered, this court is of the opinion that these spraying operations can be carried out in safety and without risk to the health of the citizens of this province.

¶ 603 The plaintiffs, therefore, fail in this action and the defendant is entitled to its costs, to be taxed.

¶ 604 Since the defendant has claimed damages and the parties agreed to set aside the matter of damages until after the decision of the main issue, I shall [*page354] hear the parties as to damages at their convenience.

¶ 605 Judgment to be entered accordingly.

Case Name:

Application dismissed.