To:
Chair Senator Michael Dembrow , and members of the Committee on Environment and Natural Resources

Testimony for SB 892 (Notification bill) and SB 500 (Report of Loss bill

From:
Ellen Saunders 47950 NWS Dingheiser Road Manning OR 97125
March 22, 2017.

I ask that you support SB 892 and SB 500. Though these bills do not go nearly far enough they must be passed to begin to limit the toxic poisoning.

I live in the Coast Range Mountains west of Banks Oregon in the timber area now owned by Weyerhaeuser. When Weyerhaeuser bought the forests surrounding my community a few years back they immediately clear cut large portions of the forests and hired Mike Applebee of Applebee Aviation to spray toxic herbicides. Mike Applebee has since had his spray license suspended for a year and has been fined. It was one of his herbicide truck drivers who blew the whistle and alerted the Department of Agriculture of the multiple illegal exposures of herbicide spray to Applebee's workers and across forest areas that should have been protected by buffers. This information is clearly documented by the Dep. of Ag. Weyerhaeuser is once again clear cutting near us and when they spray they will contaminate my organic orchard and garden because their aerial spray will drift as it did last time. The spray has sickened our wildlife and our deer population has shrunk to almost nothing since the last spraying.

Fortunately the harm that is caused by aerial spraying of 2,4-D, Roundup, Atrazine and similar chemicals (by many propriety names) is unnecessary. It is Unnecessary because fir trees grow in spite of what you do. They will choke out any underbrush in only a few years after planting. The health effect to water sheds, wildlife, adjacent farm and orchard land by herbicide aerial spray is devastating. The toxins drift not only when they are sprayed but again when they rise on the mists and descend to areas where they do not belong.

I am submitting the document:

Recognizing Illnesses Related to Forestry Herbicides
By Michael O'Malley, MD.,M.P.H. University of California, Davis

It is clear from reading this document that Oregon has been negligent in limiting the timber industries and the chemical companies. Repeated toxic poisoning has been reported from one end of the coast range to the other. I have a friend in Coos Bay who has suffered from a very fast growing and unexplained cancer just after a spraying incident. That same family also saw the deer dying in their yard. We can no longer ignored all the science that has been available for years about the serious toxic damage that is being caused to our people, land and wildlife.

Recognizing Illnesses Related to Forestry Herbicides

Recognizing Illnesses Related to Forestry Herbicides

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Supported by Contract # 98-0254 with funds provided by the United States Environmental Protection Agency

Introduction - Controversies regarding herbicide use in forestry management

I - Introduction - controversies regarding herbicide use in forestry management

California tribal groups and non-governmental environmental organizations have published material regarding use of herbicides in the national forests where California tribal members gather native plants for use in basketmaking.

An important The conflicts over herbicide use in the national forests in Northern California have been well summarized in online publications by members of the California Indian Basketweaver's Association :[1]

Why CIBA Exists

Weaving and use of baskets has been a core element of the spiritual and material culture of California Indian tribes for millennia. Continuation of the basketweaving tradition is essential to the continued survival of California Indian culture. CIBA's purpose is to preserve, promote, and perpetuate California Indian basketweaving traditions while providing a healthy physical, social, spiritual, and economic environment for basketweavers. We work to create and expand a functioning network of basketweavers who support one another in their gathering and weaving activities, and who pass their tradition to the next generation. Pesticides are a widespread threat to basketry plants. We work to halt their use on public and private lands while promoting alternative, environmentally sustainable forest management policies and practices. We strive also to improve safe, convenient access for basketweavers to gathering areas, while promoting a habitat stewardship role for native gatherers. To these ends, we educate government policy makers, resource managers, and the public, while supporting native communities in their efforts to influence local resource management decisions that affect their environment and health.

How CIBA Was Started

Research during the mid-1980s by CIBA's current executive director revealed concerns among some that California Indian basketweaving traditions could be at risk of dying out. Few younger weavers were learning to weave, and the mostly older women who continued to weave were finding it increasingly difficult to carry on their work. The demands of family life and the struggle to make a living, together with the destruction of plant habitats, pesticide use in gathering areas, and difficulty of obtaining access to gathering sites, were reducing the time and opportunity for plant tending, gathering, and basketweaving.

The need to support weavers and address these problems was confirmed at a statewide gathering of basketweavers in 1991. Following the gathering, a Basketweavers Council was formed to plan future gatherings, to establish a formal organization, and to begin solving the problems identified. At the next statewide gathering, in 1992, weavers formed the California Indian Basketweavers Association and members of the Council became its founding board of directors. Nonprofit status followed in 1993. The first, and current, executive director was hired in 1992. Since then, CIBA has employed various part-time administrative, program, newsletter, and grantwriting staff.

What CIBA Has Accomplished

In its brief history, CIBA has achieved much. Basketweaver attendance at the annual gathering has more than doubled since the first gathering in 1991. We hold the gathering at the same location for two years in a row, and move it to a new region of the state every other year in order to maximize attendance and communication among weavers. Through the sharing of stories of hardships and successes at the gathering, a sense of solidarity has replaced a feeling of isolation, and weavers have found new

Basket Linda L. Longshore (Gabrieleno/Tongva/Juaneno)

strength and renewed inspiration to continue their time-demanding and difficult work. Classes and "weaving circles" are being started throughout the state by experienced weavers. As a result, increasing numbers of native Californians are learning not only the skill of basketweaving, but the associated spiritual and cultural values of their tribal heritage.

Basket - Ruby Jean Vargas

CIBA has alerted state and federal land managers to the need to preserve access to gathering sites and has encouraged them to expand unrestricted access and native stewardship arrangements. We also have succeeded in getting the US Forest Service (USFS) to consult more closely with basketweavers and traditional practitioners on land and resource management issues. And although we haven't managed to get the USFS and private timber companies to stop using pesticides, we have made them aware of the dangers posed by their activities to weavers and native communities. As a result many public land managers attempt to minimize or avoid using pesticides near gathering areas. Our outreach to government officials in Washington DC has resulted in agency studies on forestry herbicide residues on plants of concern to basketweavers, and in an unprecedented government assessment of the concerns and desires of northern California native communities regarding pesticide use. CIBA has alerted land managers to the dangers of unregulated commercial collecting of basketry materials, medicinal, mushrooms and other "non-timber forest products". In response, the US Forest Service has begun holding meetings with basketweavers and others to develop policies for protecting native resources from overharvest and habitat damage by commercial collectors.

Ongoing Work

The use of pesticides on private and public lands is of utmost concern to California Indian basketweavers because of the harmful effects their use may have on the health of Native plant gatherers and communities, as well as the health and vitality of the environment. A weaver may be exposed to pesticides by making skin contact while gathering. In addition, most of the materials a weaver collects are passed through her mouth in preparing it for weaving. The plants that are eliminated by herbicide spraying because of their lack of commercial value are often the same plants that provide Native people with traditional foods and teas, are used in baskets and for healing, ceremonial and other traditional purposes.

Basket - Denise Davis (Mountain Maidu)

In Northwest California, there has been a history of herbicide use for many years. National forests in the area have suspended their spraying programs in response to public pressure, but were responsible for dousing many acres with 2,4,5-T in years past, with disastrous results on pregnant women. Private timber companies continue the aerial bombardment by herbicides, using such hazardous chemicals as 2,4-D, atrazine, sulfometuron methyl, and triclopyr. One private timber company owns 87% of lands within the boundaries of the Yurok Reservation, which borders both sides of the lower Klamath River. There have been reports of high incidence of cancer, respiratory ailments and heart disease among communities of the lower Klamath watershed. Native people have also reported an absence of honeybees, deformities in fish and lesions on the internal organs of deer. CIBA has continued to bring attention to this situation for several year, yet it is a very difficult one to deal with, as private landholders are not answerable to public concerns about pesticide spraying.

On public lands, CIBA has been active in trying to end the use of pesticides on national forests, which have resumed their use after a moratorium of several years. While some national forests choose not to use

herbicides, others such as the Stanislaus, Eldorado and Sierra National Forests are carrying out plans to spray many thousands of acres. On tribal lands, the Hoopa Tribe banned pesticide use on the Hoopa Reservation in 1978. Timber sales are an important source of income for the Tribe; timber sales run in the black, while manual removal of competing brush provides employment. The argument against such methods, however, is mainly an economic one. In terms of dollars, the cost of herbicide application costs less than "hand-grubbing" or mechanical methods. The Forest Service argues that it is necessary to use herbicides in areas where thousands of acres of trees have been burned and reforestation efforts are underway. CIBA has been active in the effort to stop pesticide use on forest lands, meeting with representatives of State and Federal agencies to express the importance of basketweaving to Indian culture and concerns about the health effects of possible exposure to pesticides in the course of gathering and processing basketry plants and other natural resources utilized by California Indians.

CIBA activities reported in the fall of 2000 include challenges to forest service herbicide projects near Nevada City California and in the Klamath River area:

Several herbicide spray projects proposed by the US Forest Service (FS) have been challenged by CIBA, resulting in delays or changes in final decisions. On the Tahoe NF, we joined with Forest Issues Group (FIG) and the South Yuba River Citizens League (SYRCL) of Nevada City to appeal the decision of the district ranger to implement the Cottonwood Fire herbicide project (see NL #30). That proposal, if implemented, would result in spraying herbicides (glyphosate and triclopyr) on 10,000-20,000 acres of forest lands to kill native shrubs and herbs allegedly competing with conifer trees. We believe In response to our written comments, and concerns expressed locally by the Klamath Forest Alliance and the Salmon River Council, we have been advised that the Klamath NF will forego for the time being the use of herbicides to control spotted knapweed, an invasive non-native plant, in the Salmon River drainage of northern California. Instead, they will give the local community an opportunity to continue their very effective program to control the plant by hand-weeding. We also learned that, if they consider herbicide use in the future, the highly dangerous herbicide 2,4-D would not be considered for use on this project. There are still several uncertainties involved with this proposal, however, and we will continue to monitor the project in order to keep herbicides out that the FS has failed to demonstrate that use of herbicides is essential. Evidence suggests that conifer survival and growth is good, and may even be aided by the beneficial effects of the associated species that share their habitat. Unfortunately, our appeal was denied in early August by the FS regional office in San Francisco. Consequently, we have joined forces with FIG and with Californians for Alternatives to Toxics (CATS), another appellant, to put the FS on notice of our intention to file a lawsuit in federal court against the project. The project is on hold pending the outcome of the litigation process. On the Modoc NF, the Long Damon Plantation Release project is another herbicide project designed to kill competing vegetation after a wildfire. The herbicide hexazinone would be applied in pelletized form from helicopters on more than 5,000 acres. Numerous culturally important plant species, such as chokecherry, Sierra plum, and sourberry, are at risk, as are wildlife species that occur in the numerous “lava reefs” and caves that exist throughout the area. In addition, some 87 different species of animals are dependent upon the sagebrush plant communities that occur throughout the area. We vigorously opposed the project in written comments to the FS of the Salmon River watershed. With the end of summer upon us, the period for application of herbicides is fast drawing to a close. We are hopeful that the year will pass without the national forests being subjected to any new large-scale herbicide spraying. For further information about these and other herbicide projects around the state, contact Vivian Parker, Resource Policy Analyst, at 530/622-8718.

Health concerns of individual basketmakers are also described in a publication entitled Cultural Traditions Endangered, written by the Ron Goode, chairman of the North Fork Mono Tribe: [2]

Interviews with Consultants (interviewed by ethnographic researcher: Ron W. Goode

Introduction - Controversies regarding herbicide use in forestry management

Rosalie Bethel: Is the North Fork Mono Tribes' spiritual leader, the head singer of the Coyote Moiety/Clan, a basket maker and is also a plant/food gatherer. In April at the Sierra Mono Museum Board of Directors meeting, Rosalie, gave a very disturbing account of her contact with chemical spraying. Her experience took place near Cold Springs Rancheria in Burrough Valley located in Fresno County. Rosalie, in her aging process, has knee problems and is not able to venture far from road accesses.

She was gathering redbud for making baskets and as is customary she immediately began cleaning and splitting some of the redbud shoots to check their quality. On her way home she noticed a tingling sensation around her mouth. Later, after arriving home she continued to split her sticks. This is when she noticed her mouth had become numb and she soon became very nauseated. Two weeks later when she returned to Cold Springs Rancheria she asked the folks there why none of the beautiful redbud shoots had not been gathered. She was informed that the County had sprayed with Round-up and some of the gatherers had known what the affects of chemical spraying caused.

Rosalie proceeded to say that her sister Frances Sherman had also been affected by chemical spraying while gathering basket material along roadways in both Fresno and Madera Counties. Both of these Elders had been affected during the time span between 1989-1990
(Rosalie Bethel, Personal Communication - April 1991).

In a follow-up interview with Rosalie, she criticized the Forest Service for poor logging management and the California Department of Forestry and County Governments for inadequate care of tree maintenance. Rosalie stated she cannot understand why the Forest shuts down logging operations when the weather is not a factor. She contends that the winter is a prime time to cut out the bug infested trees because during this time of the year the bark beetle is moving slowly but when spring and summer come this insect will have multiplied and will be working quite rapidly when the climate warms up. She went on to question why the State and County doesn't do anything about the mistletoe infection that our foothills and valley is suffering from. Rosalie stated that the mistletoe infection is like a cancer to the trees and that this cancer has infested all trees from the mountains to the valley floor
(Rosalie Bethel, Personal Communication -January 1992).

Meetings between tribal members and the California Department of Pesticide Regulation (CDPR)

Concerns about forestry herbicide use expressed by tribal members were also voiced in community meetings with the California Department of Pesticide Regulation at several sites between July 12 and October 18, 1997. These included Robinson Rancheria, Upper Lake, California, July 12, 1997; Fall River Mills, California, July 19, 1997; Karuk Tribal Community Center, Orleans, California, September 20, 1997; Yurok-Pecwan Community, Pecwan, California, October 16, 1997; Yurok-Klamath Community, Klamath, California, October 17, 1997. Community concerns were summarized by a Indian Dispute Resolution Services, a consultant firm engaged by the Department of Pesticide Regulation:[3]

HIGHLIGHTS OF PESTICIDE ISSUES COMMUNITY MEETINGS
I. Perceptions and beliefs about pesticides and pesticide use in their communities
expressed by Native Americans:
1. Pesticides are poison and harmful to all life.
2. Pesticides remain active in the environment forever (undetermined time).
3. Pesticides are the cause of many health problems.
4. Pesticides continue to be used widely within the areas we live in and gather from.
5. The specific sites we gather from and use for ceremonial purposes are sacred to us and we cannot simply relocate to another site.

6. We fear that disclosing specific gathering sites and sacred sites will result in more restrictions or damage to the sites.
7. We are concerned that full disclosure of our use of plants for medicinal and sacred purposes will result in exploitation. Disclosure of certain practices would violate our spiritual principles and beliefs.
8. We are not (or have not been) listened to or taken seriously, when we have attempted to express our concerns and fears about the use of pesticides.
9. The decline in many fish/animal/insect species is directly related to pesticide use.
10. Dwellings, schools, and water sources have been indiscriminately subjected to aerial spraying.
11. Aerial spraying continues to be a widespread practice by timber companies.
12. People feel that they have no "rights" as individuals or communities, all rights are vested with timber companies and government agencies.
13. Tribal members fear that the regulatory agencies will conduct a risk assessment, which will say that traditional food gathering practices and traditional basket weaving practices are not safe and should be discontinued.

II. Information Revealed or Confirmed by the Community Meetings

1. Native Americans in these communities live very close to nature.
2. Pesticide use has disrupted cultural patterns of life.
3. Many people rely upon untreated water sources originating in or adjacent to pesticide treated areas (surface water or springs).
4. A large percentage of their foodstuff is gathered from areas possibly impacted by pesticides. This is an economic necessity for many people, not just a matter of choice.
5. Acorns continue to be a part of the diet of many people (oaks are a pesticide targeted species).
6. Firewood from sprayed areas is gathered and burned.
7. People continue to visit (or camp in) traditional Tribal sites in areas owned and sprayed by timber companies despite postings and warnings.
8. Some timber companies provide access to traditional areas on company owned land (having locked gates) to certain Native Americans.
9. Many Native Americans are or have been employed in the forest products industry as loggers and mill workers, spending years in daily direct contact with products from sprayed areas.
10. Roadside gathering is a common practice, especially by the elderly and young.
11. Children of all ages accompany families while gathering and are exposed to whatever pesticides remain in the gathering environment.
12. Teas brewed from plant parts are widely used for many purposes.
13. Smoke/smudges from various plants are used for ceremonial and medicinal purposes.
14. Many communities are experiencing resurgence in traditional practices, resulting in an increased use of materials from the forest.
15. Elected Tribal government is not the only source of leadership in many native communities: in some groups, band or clan ties may have more influence.

III. Health Concerns expressed by communities, perceived related to pesticide use

1. Pesticide exposed communities have a 25- 30 year history of problem pregnancies (some out- of- court settlements by chemical companies).
2. Perception of increased incidence of birth defects (e.g., cleft palates).
3. Anecdotal evidence of increased incidence of cancer; there is a perception that every family has experienced it.
4. Widespread uncertainty and fear about the safety of drinking water.
5. Personal accounts of physical symptoms of unknown origin i.e., blisters, skin rashes, nausea, respiratory problems, etc.

6. Concerns related to children's behavior patterns and school performance.

What the Communities Want

1. Right to know
a. Access to historical data documenting application of pesticides upon areas of concern.
b. Access to monitoring records and the results of any testing by monitoring agencies.
c. Notice of application plans impacting their area and advance notice of application dates.
d. The location of any roadside applications.
2. Involvement in the permit review process.
3. Representation on advisory groups related to pesticide use.
4. Ability to monitor the safety of drinking water, especially individual non-treated sources.
5. Effective and timely communication from all levels (government to government, isolated communities, etc.).
6. To be able to understand the complex, multi-layered pesticide regulation process.
7. Access to specific information on chemicals used, in a format and language that can be understood by a layperson.
8. Acceptance of our anecdotal evidence regarding the effects of pesticides on our health and environment.
9. Proof that we can be protected from harm.
10. Resources to undertake our own comprehensive health studies and surveys to determine the precise health status of community members; and to document the extent and nature of each community's health problems.
11. Training for health care providers to identify pesticide-related illness, document it, and report it.
12. Increased resources for treatment of illness and disease.
13. The Robinson Rancheria has unique needs (in addition to other pesticide applications) to understand the impact of the pesticides utilized in the Clear Lake hydrilla treatment project.

Specific concerns about cancer in the Klamath River area and residues of forestry herbicides in fish and game in the National forests were raised at meetings with the Yurok Environmental Group in September[4] and November 1999,[5] and January, 2000.[6] Additional concerns were also raised about the representativeness of plant residue and surface water sampling conducted by the Department of Pesticide Regulation. No conclusion was reached on a proposal to develop additional illness information by conducting a survey of Yurok tribal members (see appendix 2) potentially exposed to forestry herbicides. The occurrence of cancer in Klamath River communities was discussed with a representative of the California Cancer Registry. The data from that discussion are presented, along with comparison data on statewide cancer rates, in the discussion below.

Discussions with Indian Health Service and Tribal Clinics medical providers

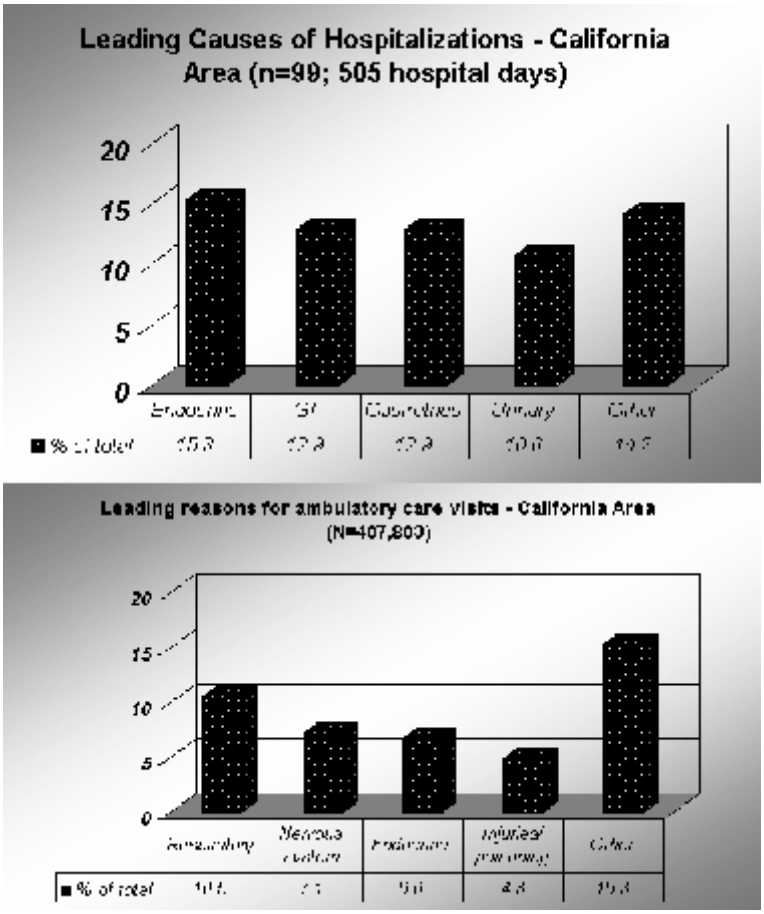
The concerns regarding illnesses related to the use of forestry herbicides were also discussed with medical providers at the June, 1999 California Clinician's Conference on Indian Health. Most providers were not previously aware of the forest herbicide controversy. Questions raised during the discussion dealt with the possible effects of pesticides used for structural pest control in clinics and pesticides used on alfalfa. Providers also asked questions about the relative toxicity of herbicides versus other classes of pesticides, especially insecticides. Specific questions regarding forestry herbicides included:

How do levels found in forest residue monitoring compare to food tolerance levels and residues found at food monitoring?

How much exposure is there from herbicides in stream runoff and ground water contamination?

Introduction - Controversies regarding herbicide use in forestry management

Other presentations at the June, 1999 meeting focused on care of diabetes and other chronic health problems commonly seen at California Indian Health clinics and hospitals (see figure).[7]



Reasons for hospitalization (above) and ambulatory care visits (below) California Indian Health Service and Tribal Clinics. **Source: Regional Variations in Indian Health, 1998-1999, U.S. Indian Health Service**

Non-governmental environmental organizations

Non-governmental organizations concerned about forestry herbicides, the use of herbicides in roadside spraying programs include **Californians for Alternatives to Toxics** (CATs), illustrated by this description of the organizations concerns about forestry herbicides.[8]

Forest Pesticide Use Investigated

CATs searched pesticide records available to the public in early 1997 and found that timber companies in northern California had again increased the amount of herbicide applications over the previous year. Crews of laborers hand sprayed over 13,500 Humboldt County acres, 5,000 acres each in Del Norte and Mendocino counties. Forests in Trinity, Siskiyou, Shasta and several other counties were also doused with chemicals to kill brush and weeds that compete with conifers replanted on clearcuts. Information indicating that timber companies are spraying thousands of gallons of highly toxic diesel fuel to kill unwanted vegetation turned up in the course of our investigation. We learned that not only is diesel spraying legal, the activity is not overseen by any government agency. Nor are surprise inspections possible; the agency in charge of regulating pesticide law, the county agricultural commissioner, cannot gain access to timberlands without contacting the owner in advance. And, water quality is not monitored by state regulators following ground applications as it is after aerial spraying.

Anti-Spray Activities

Other anti-forest spray activities in which CATs participated included: helping local groups learn about the chemicals used near their homes and how to organize their neighborhoods against herbicide applications. spotlighting illegal herbicide dumping and unsafe worker practices of spray contractor Winter Express, which, as a result, no longer works on the Northcoast. appearing before the regional water board to request a sample monitoring program for herbicide and diesel run-off from forest spray sites (no response yet from the agency). We also attended community and water district meetings. organizing a meeting in October of rural community groups to share information, strengthen ties and plan actions. coordinating with watershed groups to question the Department of Forestry about potential herbicide spraying on proposed timber harvests. CATs is the only organization that monitors, statewide, plans made by federal agencies to use pesticides on public lands.

Introduction - Controversies regarding herbicide use in forestry management

> One plan we were particularly concerned about involves using the highly toxic insecticide carbaryl in an effort to protect sugar pine trees which are genetically resistant to an imported disease expected to wipe out most of the species.
> Another proposal that also received special attention from CATs calls for killing gophers by planting strychnine on thousands of acres of national forest. We will join with other northern California organizations to oppose the plan in 1998.

CATS has also raised concerns about roadside use of herbicides throughout California, but particularly in the north coast section of the state[9]:

> California's Department of Transportation (Caltrans), a nearly $6 billion bureaucracy, discharges these dangerous chemicals onto state highways yet cannot provide even the most fundamental information regarding the size and extent of its herbicide spray program. In The Poisoning of Public Thoroughfares: How Herbicides Blight California's Roads, Californians for Alternatives to Toxics (CATs) describes for the first time ever how state and county agencies douse California roads - especially in the most populous regions - with huge quantities of chemical herbicides. Research for this report was conducted throughout a two year period beginning in early 1997 when a groundswell of public outrage over roadside spraying on the North Coast forced Caltrans to stop drenching local highways with toxic chemicals.

Conclusions

Tribal and community concerns include both acute and chronic effects of pesticides used in forestry management, especially cancer and birth defects. Pesticide of special concern include 2,4-D and other phenoxy herbicides. The potential contamination of water sources, acorns, and game animals are of concern in addition to the effects of herbicides on plants used in basket making. Right-of-way applications are of special concern to gatherers who collect materials from forest roadsides. Preliminary discussions with medical providers at the June, 1999 California Clinician's Conference on Indian Health indicated that many providers had not previously been aware of concerns about forestry herbicide use.

Basic Pesticide Toxicology

II - Basic pesticide toxicology

A perspective on the toxicity of herbicides used in forestry management is simplified by a basic understanding of the toxicology of the major classes pesticides (see inset box). The material is presented here includes a brief summary of the toxicology of organophosphates and carbamates; pyrethrins and pyrethroids; organochlorines; and the thiocarbamate phthalimido, and chlorobenzene fungicides. The material is based principally upon a review previously published in the Lancet, reprinted here with permission.[10] Additional material on the environmental fate and effect on wildlife for individual compounds is based on material from a coalition of agricultural extension services (EXTOXNET).[11] California illness data, based upon physician reports to the state pesticide illness registry are also discussed. Requirements for physician reporting and a pesticide illness report (PIR) are included in appendix III.

Use and chemical structural categories of pesticides

Users of pesticides naturally focus on the effect of individual compounds on the target organism. The effects of each compound on both target and other organism depend upon its chemical structure and important physical properties. The manner of exposure depends markedly on the vapor of individual compounds. Apart from fumigant compounds, most pesticides are designed to leave a residue on treated surfaces and have relatively low vapor pressures.

Insecticides
Organochlorines, organophosphates, carbamates, pyrethrins, synthetic pyrethroids, nicotine, rotenone, microbiological *(Bacillus thuringiensis)*

Herbicides
Trichloro/dichlorophenoxyherbicides, urea derivatives, carbamates, triazines, glyphosate

Fungicides
Thiocarbamates, miscellaneous compounds including captan, captofol, pentachlorophenol, iprodione, elemental sulphur

Antimicrobials
Triazine-S-triones, chlorine releasing agents, chlorine, dichloronitrobenzene

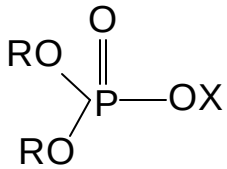
Rodenticides
Coumadin and derivatives, long-acting and short-acting anticoagulants, strychnine, sodium fluoroacetate

Organophosphates & N-methyl carbamates

Organophosphates (OPs) and N-methyl carbamates are the pesticides most frequently responsible for systemic illness. Specific materials most frequently encountered depend upon the nature of the application and local use patterns. Between 1982 - 1990, OPs most responsible for illnesses (n=2031) among non-occupational and non-agricultural workers in California were compounds of moderate toxicity: chlorpyrifos, DDVP, malathion, dimethoate, and propetamphos. Illnesses in agricultural workers over the same time period (n=1379) were most frequently associated with high toxicity OPs (mevinphos, methomyl, methamidophos, oxydemeton, and parathion), but also included moderately toxic compounds (dimethoate and phosalone).

Basic OP toxidrome

OPs poison the nervous system by inhibiting the breakdown of the transmitter acetylcholinesterase by the enzyme acetylcholinesterase. This results in overstimulation of portions of the nervous system which contain acetylcholine: muscarinic - post ganglionic fibers of the parasympathetic nervous system (control secretions of respiratory and GI tracts, heart rate, etc), sweat glands in the sympathetic nervous system, preganglionic fibers in the sympathetic nervous system, and skeletal muscle. The acronym MUDDLES (Miosis Urination Diarrhea Diaphoresis Lacrimation Excitation (of CNS) Salivation) is a helpful means of remembering the principal effects of cholinesterase inhibitors. Bradycardia is often severe and may be responsible for episodes of dizziness and syncope associated with organophosphate poisoning.



Basic organophosphate (OP) structure - R groups are usually methyl (CH3) or ethyl (CH3CH2) groups; X = leaving group

Basic Pesticide Toxicology

N-methyl-carbamates insecticides produce a toxidrome similar to the organophosphate but markedly briefer in duration. Although patients may exhibit symptoms following OP poisoning for 1-2 weeks, most recover from carbamate poisoning within 24 hours. except that it is more readily reversible since the complex between the cholinesterase enzyme and carbamate insecticides readily breaks down (see case example 2). It is not necessary for new enzyme to be synthesized for normal functioning to be restored. Consequently it is more difficult to detect cholinesterase depression due to carbamates.

Irritant effects

In addition to ChE-inhibition many organophosphates are associated with irritation of the skin and upper respiratory tract (see case example 1). The agents producing odor and irritant effects associated with most OPs are thought to be low molecular weight mercaptans and sulfides. Monitoring studies of communities near applications of OP cotton defoliant tributyl phosphorothioate (DEF) have demonstrated, for example, that the concentration of butyl mercaptan ranged from 0.29-9.93 ppb (well above the odor thresholds for mercaptans) while concentrations of the active ingredient were orders of magnitude less (0-0.034ppt). [12] Although the odors associated with OPs often give rise to characteristic irritant symptoms, they are also provoke non-specific systemic symptoms [13] such headache and nausea. Although most of the respiratory irritation is confined to the upper airways, occasional complaints of OP associated wheezing and chest-tightness are reported. These cases require careful evaluation, because bronchoconstriction sometimes results from systemic poisoning as well as airway irritation. Persistent reactive airways have occasionally been reported following exposure OPs independent of cholinesterase inhibition.[14]

Delayed neuropathy and neurobehavioral effects of OPs

The most serious non-ChE related effects of OPs are a delayed neuropathy (OPIDN) that presents 7-14 days after exposure. Historically associated OPIDN has been principally associated with a handful of OPs compounds that have a high propensity for inhibiting neuropathy target enzyme (NTE). These include compounds no longer used including leptophos and EPN. Most OPs have the capacity to produce OPIDN following massive intoxication, but none of the currently used OPs preferentially inhibit NTE at doses that do not also cause ChE inhibition.

The intermediate syndrome reported by Senanayake and Karalliedde is distinguished from OPIDN by onset within 24-96 hours after recovery from acute cholinergic crisis, tendency to affect the cranial nerves and proximal muscles, tetanic fade on EMG studies instead of denervation potentials. Recovery was also more rapid occurring over 4-18 days rather 6-12 months as is typical of OPIDN.[15]

The controversy regarding persistent neurobehavioral effects following recovery from OP poisoning dates back 30 years[16] and continues to be stimulated by recent clinical experience.[17] The study of a group of poisoned applicators reported by Savage in 1990 demonstrated deficits in memory and abstraction on test batteries, but normal neurological examinations.[18] Rosenstock demonstrated several deficits with the WHO test battery and also subclinical decreases in vibrotactile sensitivity, but also reported normal clinical examinations.[19] The test batteries conducted by Steenland [20]showed deficits in two neurobehavioral tests (sustained visual attention and mood scales) and decreased vibrotactile sensitivity in the toe, but neurological examinations were normal. Some studies of non-poisoned OP applicators have demonstrated similar types of subclinical neurobehavioral deficits and subclinical EEG abnormalities, [21, 22] while others have been largely negative. [23, 24]

Diagnostic and Laboratory Evaluations

Identification of the active ingredient involved in a pesticide exposure is essential to evaluating the exposure history and is the best done from a product label or material data safety sheet. Trade references such as the Farm Chemicals Handbook[25] provide a means of identifying agricultural pesticides from their trade names. (OPs and

carbamates can be identified from listing of atropine or 2-PAM as antidotes in the "Pesticide Dictionary" section of this reference). An online source of information is the pesticide label data base operated by the California Department of Pesticide Regulation.[a]

Diagnosis of the acute OP toxidrome is made based upon a history of exposure and a blood test of red cell and plasma cholinesterase. In situations where a high prevalence of OP poisoning is present or is strongly suspected (e.g. an illness outbreak or a group of applicators routinely handling OPs), a test within the laboratory normal range is not sufficient to rule out poisoning. given the wide range of normal values in the population. Appropriate diagnosis can be made by comparison with a baseline value, by doing serial followup tests, or by testing for regeneration of the native acetylcholinesterase enzyme following in vitro treatment of a blood sample with the cholinesterase antidote 2-PAM. There is no clear evidence as to what level of cholinesterase inhibition is necessary to produce symptomatic illness, although in the past claims have been made for various thresholds (e.g. 50% depression, or 80% depression) past based upon different case series. Variation in the degree of inhibition required to produce symptoms is related to the rate of inhibition.[26, 27]

Diagnosis of delayed neuropathy may be established by means of nerve conduction tests and characteristic onset of symptoms after significant exposure to an OP.[28] No specific tests are available to diagnose chronic neurobehavioral effects of OPs in individual cases. Reactive airways can be diagnosed with methacholine challenge tests,[29] but association with OP exposures in individual cases may be difficult to establish.

Treatment

For illness that does not require hospitalization (about 73% of definite-probable OP poisonings reported in California), decontamination of skin may be the principal treatment required. Return to work that does not involve OP exposure is appropriate in the absence of significant impairment. No re-exposure should be allowed until ChE levels have returned approximately to baseline levels.

Antidotal treatment is usually reserved for hospitalized cases. Atropine reverses muscarinic symptoms (respiratory and GI tract secretions, bradycardia, etc) of OP poisoning for relatively short periods (pharmacologic half-life = 70 minutes ± 30).[30] 1-2 mg IV doses may be used in place of the 0.5 - 1.0 mg doses used in treating symptomatic bradycardia associated with heart disease. Serious doses may be titrated to maintain clear breath sounds and a heart rate of 80-100 beats/minute. Protopam (2-PAM) breaks down the cholinesterase-OP complex when 1 g is given over 10-20 minutes intravenously (after taking diagnostic cholinesterase samples). It is effective against nicotinic, muscarinic and CNS poisoning symptoms, but is not generally recommended for treatment of carbamate poisoning because it may exacerbate the poisonings due to particular carbamates (carbaryl and propoxur). Case reports, however, have demonstrated that 2-PAM may occasionally be helpful in treating nicotinic effects of methomyl.[31]

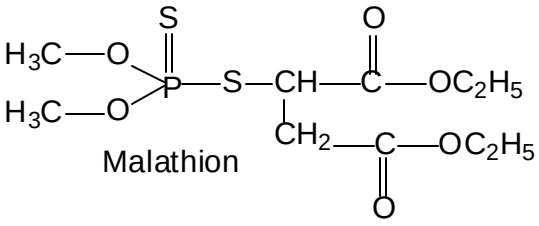
In the most severely ill patients, oxygen, clearance of secretions and artificial ventilation, may be required in addition to antidotal therapy. For these patients, use of morphine, aminophylline, and phenothiazines are contraindicated because of the increased risk of cardiac arrhythmias. Atropine should not be given until adequate ventilation has reversed hypoxia. These intensive care measures are more typically required following deliberate or accidental ingestion of organophosphates than for occupational illness. These cases are also more likely to develop delayed neuropathy and other complications. Treatment for these conditions is supportive and does not employ specific antidotes.

[a] (http://www.cdpr.ca.gov)

Basic Pesticide Toxicology

Environmental fate and effect on wildlife

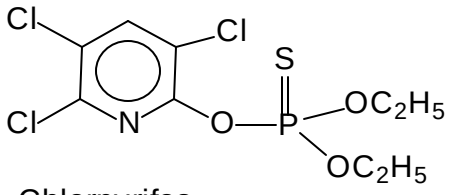
The environmental fate of typical OPs is illustrated by data on malathion. The half-life of malathion in soil - breaking down by hydrolysis and biological action - varies from 1 to 25 days, depending upon how tightly it binds to the soil matrix. In river water, the half-life is less than a week, but has a half-life up to 3 weeks in distilled water.[32]



Malathion

The toxicity of malathion for birds is variable. The acute oral LD50 for mallards, 1485 mg/kg is similar to the LD50 for rodents, but the LD50 for pheasants, is markedly less, 167 mg/kg. It also has a wide range of toxicity for fish - the LC50 is 0.06 mg/L for walleye, for brown trout 0.1 mg/L, and for carp (goldfish) 10.7 mg/L. Aquatic inverterbrates are sensitive to levels as low as 1 µg/L. It is highly toxic to aquatic stage of amphibians and also to honeybees.[32]

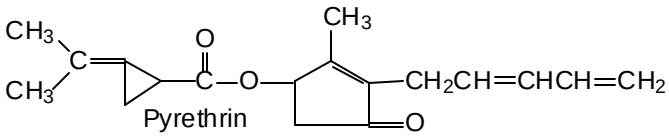
Another typical OP compound, of moderate toxicity, is chlorpyrifos. It has a half-life in soil markedly longer than malathion - typically 60 to 120 days, but may be more 1 year in selected conditions. Dissipation from water has a half-life varying from 3.5 to 20 days. The rate of hydrolysis breakdown is fastest in alkaline waters. In water at pH 7.0 and 25 C, it had a half-life of 35 to 78 days. Residues remain on plant surfaces for approximately 10 to 14 days.[32]



Chlorpyrifos

Aquatic and general agricultural uses of chlorpyrifos pose a serious hazard to wildlife and honeybees. Chlorpyrifos is generally more toxic to birds - mallards (LD50 - 112 mg/kg), house sparrows (LD50 - 21.0 mg/kg), chickens (LD50 - 32 mg/kg) - than to mammals. For fish the 96-hour LC50 for chlorpyrifos ranges from 0.009 mg/L in mature rainbow trout to 0.806 mg/L in carp (goldfish).[33]

Pyrethrin and Synthetic Pyrethroid Insecticides



Pyrethrin

Pyrethrum is a partially refined extract of the chrysanthemum flower that has been used as an insecticide for more than 60 years. In addition to the insecticidal pyrethrin compounds they may contain sequiterpene lactones that are well recognized causes of allergic rhinitis and contact dermatitis. The large majority of current U.S. registrations contained purified pyrethrins rather than pyrethrum extract, but clusters of allergic rhinitis are still occasionally reported in offices where pyrethrin insecticides have been applied. This tendency is particularly unfortunate because rapid hydrolysis in mammalian liver prevents the nervous system effects responsible for their toxicity to insects (oral LD50 for pyrethrum = 1500 mg/kg in rats) are not seen in mammals. Synthetic pyrethrins (pyrethroids) are based structurally on the pyrethrin molecule but modified to improve stability. Some pyrethroids have been reported to cause systemic occupational poisoning in China,[34] but have been recognized only sporadically as a cause of occupational poisonings elsewhere.[35] Nevertheless, some pyrethroids have markedly higher acute oral toxicity than pyrethrum. For example, the oral LD50 for deltamethrin in 81 mk/kg in adult rodents. It shows even higher toxicity in immature animals (weanling rats -oral LD50 = 5.1 mg/kg).[36] In adult humans, 10 mg/kg oral doses have been reported to cause seizures,[26 36] and an estimated 2 mg/kg dose was associated with short term systemic effects in a pediatric ingestion case reported in California.

Treatment

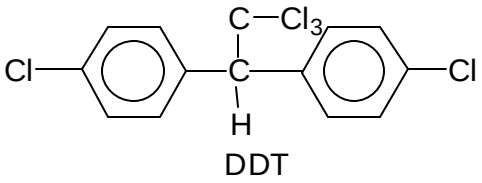
The key element of treatment of pyrethrin/pyrethrum allergy is removal from exposure. Apart from allergen avoidance, allergic contact dermatitis may be treated with appropriate systemic or topical steroids, depending upon severity, allergic rhinitis with antihistamines, decongestants, and a steroid nasal sprays; and asthma with O2 agonists and inhaled steroids. Cutaneous paresthesias may also be treated with topical vitamin E,[37] which appears to act by blocking the effect of pyrethroids on the sodium channel in neurons on the skin. Treatment of acute systemic pyrethroid poisoning is supportive.[38]

Environmental fate and effect on wildlife

Pyrethrins alone provide limited crop protection because they are not stable, broken down in water to nontoxic products and also decomposed by exposure to light, air and mild acids and alkalis.[39] Synthetic compounds, show much longer half-lives. For example, the pyrethroid compound cyfluthrin is sensitive to breakdown has a half-life of 56-63 days in loam and sandy loam soils by sunlight. On soil surfaces, its half-life is markedly less, approximately 48-72 hours. Because of its low polarity, it forms an oily surface film on water, but breaks down quickly (usually 1 day or less) on exposure to sunlight.[40]

Pyrethrin is toxic to fish and otheraquatic life, but minimally toxic to mallards and otherbird species, such as mallards.The compounds are fat soluble, but do not bioaccumulate becauase of their metabolic lability. [39] The model synthetic compound, cyfluthrin, has low toxicity to birds and waterfowl (LD50 range from >2,000 mg/kg). It is nevertheless toxic to aquatic organisms (LC50 for rainbow trout 0.00068 mg/l and for freshwater invertebrate Daphnia magna (LC50 = 0.14 ng/l or .00000014 mg/l). Cyfluthrin is highly toxic to bees, with an LD50 of 0.037 mg/bee.

Organochlorine Insecticides



DDT

DDT is the prototype organochlorine. It has low acute toxicity compared to the most toxic organophosphates (oral LD_{50} in the rat is 113 mg/kg; LD_{50} for parathion of 4-13 mg/kg). Symptoms of overexposure are most common after ingestion and include hyperexcitability, tremulousness, and in extreme cases, seizures. Most organochlorines are extremely persistent in the environment. They have an equally long half life in the body and can be measured several months to several years after a significant exposure. A notable exception is endrin, which is much more readily metabolized (by oxidation) than the other organochlorines and is not detectable in the blood stream more than two weeks after an exposure. Endrin is two-four times more toxic than DDT, with an LD_{50} of 16-43 mg/kg in the rat. Food contamination with endrin has been responsible for a number of illness clusters worldwide, as illustrated by an outbreak reported from Pakistan in the 1980's. Because the symptoms resembled those of encephalitis, the cause of the illnesses was not initially apparent.[41]

Cancer and chronic effects

Chronic neurotoxicity associated with low level exposures to the organochlorine compound chlordane has been reported by Kilburn and Thornton. This study compared residents of a Houston apartment complex contaminated with chlordane to a comparison group recruited from networking contacts and newspaper advertisements in a series of neurobehavioral tests. Identified deficits included tests of balance, reaction time, and verbal recall.[42]

Many organochlorines produce liver tumors in rodent bioassays.[43] Although a study of a cohort of industrial workers exposed to chlordane showed no cancer excess, a study of four organochlorine plants conducted by Brown showed an excess of biliary tract and liver cancer in one plant (manufacturing aldrin, dieldrin, and endrin).[44] A recent

prospective study based upon the New York University Women's Health Study demonstrated a significant association between body stores of the DDT metabolite DDE and breast cancer,[45] but the finding was not reproduced in a similar prospective study conducted in California.[46]

Environmental fate and effect on wildlife

DDT has a half life of between 2-15 years in soil and in water markedly less, approximately 28-56 days.[47] It has low acute toxicity to birds (e.g., LD50 = 2,240 mg/kg in mallard), but the compound bioaccumulates and has been associated with chronic exposure of predatory bird species and effects on reproduction. It is very toxic to many aquatic invertebrate species. The LD50 for DDT in bees is 27 ug/bee.[47]

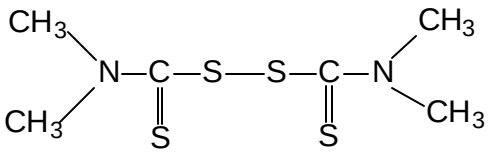
The organochlorine compound dicofol is still in use. It has a soil half-life of 16-60 days. Its half-life in aqueous solution at pH 5 is 47 to 85 days. Its effect on wildlife is comparable to that of DDT with a low-toxicity for birds and a high toxicity to organism.

Fungicides

Fungicides represent a broad range of chemical classes, including the thiocarbamates, phthalimido compounds, and chlorobenzenes.

Thiocarbamates

Thiram is the prototypical thiocarbamate compound. Reported oral LD50 values for thiram are 620 to over 1900 mg/kg in rats; 1500 to 2000 mg/kg in mice; and 210 mg/kg in rabbits. The dermal LD50 is greater than 1000 mg/kg in rabbits and in rats.[48] It is irritating to the eyes, skin, and respiratory tract and is a skin sensitizer. It also has an Antabuse®-like effect when taken together with alcohol.



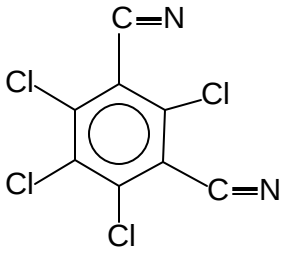
Thiram

Environmental fate and effect on wildlife

The soil half-life for thiram is reported as 15days. In water, thiram is rapidly broken down by hydrolysis and photodegradation, especially under acidic conditions. Thiram has low toxicity to birds, but highly toxic to fish (LC50 for the compound is 0.23 mg/L in bluegill sunfish, 0.13 mg/L in trout, and 4 mg/L in carp).

Chlorobenzenes

Chlorothalonil is a representative chlorobenze compound. It is slightly toxic to mammals, but it can cause severe eye and skin irritation in certain formulations. The oral LD50 is greater than 10,000 mg/kg in rats and 6000 mg/kg in mice. The acute dermal LD50 in both albino rabbits and albino rats is 10,000 mg/kg.[49] Allergic skin responses have also been reported by multiple investigators.



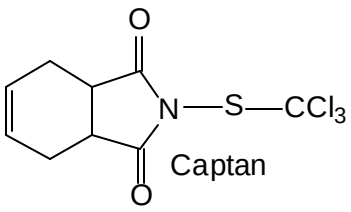
Chlorothalonil

Environmental fate and effect on wildlife

In soil, the half-life is from 1 to 3 months, with increased soil moisture or temperature increases degradation. In very basic water (pH 9.0), about 65% of the chlorothalonil was degraded into two major metabolites after 10 weeks. [49] It has low avian toxicity (LD50 mallards -5000 mg/kg) but a high toxicity to to fish (LC50 0.25 mg/L - rainbow trout; 0.3 mg/L - bluegills, 0.43 mg/L - channel catfish), aquatic invertebrates, and marine organisms.

Basic Pesticide Toxicology

Phthalimido compounds



Captan, the prototypical phthalimido compound has a low acute toxicity (rat oral LD50 - 8400 to 15,000 mg/kg; mouse LD50 - 7000 mg/kg). Workers exposed to high concentrations of captan in air (6 mg/m3) experienced eye irritation including burning, itching, and tearing. Skin irritation also occurred in some cases.[50]

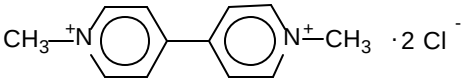
Environmental fate and effect on wildlife

Captan has soil half-life of 1 to 10 days. In water, it has a half-life of 1 to 54 hours, depending upon environmental acidity and temperature. Effective fungitoxicity persists for 23 days after application. It has low avian toxicity (e.g., 5000 mg/kg in mallard ducks and pheasants). It is very toxic to fish (96-hour LC50 - 0.056 mg/L in cutthroat trout and chinook salmon), but only moderate toxicity for aquatic invertebrates (96-hour LC50 for Daphnia magna - 7 to 10 mg/L).[50]

Herbicides

Herbicides with relatively specific effects on plant biochemistry (such as inhibiting photosynthetic electron transport or mimic the effect of plant growth hormones) typically have low mammalian toxicity. Others, such as the bipiridyls diquat and paraquat, are reactive chemicals that interact in a non-specific way with many types of living tissue. The section below reviews the toxicology of individual compounds.

Paraquat



Paraquat dichloride

Although not used frequently in California forestry, paraquat represents a broad array of older herbicide compounds with nonselective mechanisms of action. Paraquat is notorious for the delayed onset pulmonary fibrosis that it produces after systemic absorption (reported oral LD50 values of 110 to 150 mg/kg in rats, 50 mg/kg in monkeys, 48 mg/kg in cats, and 50 to 70 mg/kg in cows).[51] Ingestion of either compound produces Ingestion produces oral and abdominal pain, nausea, vomiting, diarrhea, followed by liver and renal injury. Topical irritation from paraquat and diquat are also very similar: irritation and fissuring of skin of hands, cracking and discoloration of fingernails, conjunctivitis following ocular exposure; respiratory exposure produces sore throat and coughing. The occurrence of systemic poisoning following dermal exposure[52] is controversial. Documentation of the illness may be aided by urine or serum levels of paraquat.[43]

California experience

80 (81.6%) of the 98 possible, probable and definite illnesses reported to the California illness registry between 1982 and 1995 with paraquat identified as the primary pesticide involved pesticide handlers (mixers/loaders/applicators/flaggers).

Activity	Topical	Respiratory/systemic	Total
Handler	50	30	**80**
Drift	1	8	**9**
Other	**3**	**6**	**9**

Total	**54**	**44**	**98**

A listing of some typical individual cases, including both systemic and topical effects of paraquat, is given below:

Selected paraquat cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Exposure circumstances and symptoms
Primarily topical effects			
82- 85	1/29/82	Kern	A hose blew off can being rinsed, spraying him in the face and resulting in chemical burns.
83- 480	4/29/83	Kern	A gust of wind blew material onto arms which had previous abrasions. He suffered from increased discomfort at the site of the previous injuries.
83- 614	5/10/83	Kern	He was spraying field edges with material, pump leaked onto legs, causing burning sensation on leg.
84-2486	10/9/84	Kern	An applicator brushed his unprotected hands against recently treated plants; he developed burning sensation and swelling at the site of contact.
86-1089	6/4/86	Kern	While a mixer/loader poured paraquat by hand, some of the material splashed into unprotected eye, causing conjunctivitis.
88-2057	7/12/88	Tulare	While a work applied paraquat, a hose or coupling broke, spraying him in the face and left eye, causing acute conjunctivitis.
90- 919	3/12/90	Kern	Driving closed cab tractor wearing only coveralls as protective gear, applicator opened the door to turn a valve on when a hose blew off and sprayed him in the face; he developed conjunctivitis and blepharitis.
90-1088	5/8/90	Madera	He removed a glove for a short time while applying paraquat with a hand wand sprayer in a vineyard, then developed a rash and burning sensation in the exposed area.
90-71	1/26/90	San Joaquin	Worker mix/loading herbicide and splashed on to pre-existing cut which subsequently developed cellulitis.
Systemic/respiratory			
87- 105	2/10/87	Kern	Mix tank hose broke, spraying supervising foreman in the face. He developed chest pain that evening and was treated for bronchitis two days later.
87- 239	2/27/87	Solano	Mixed and applied paraquat without training or protective equipment. He subsequently developed nausea, vomiting, possible syncope, and diarrhea. His pulmonary function was unaffected.
87- 371	4/8/87	Butte	A grower sprayed paraquat without wearing goggles and a defective nozzle allowed spray to hit him in the face. He developed eye irritation, weakness, and shortness of breath.
87- 863	5/21/87	Los Angeles	A mechanic replacing a seal on a spray rig without wearing goggles. Paraquat splashed on the face and torso of. He was not wearing goggles. He developed headache, lost of appetite, tiredness, dizziness and was hospitalized overnight.
87- 984	6/12/87	Fresno	Spot spraying weeds in orchards without a respirator worn. He developed nausea, vomiting, diarrhea, dizziness and a nose bleed.
88- 579	4/26/88	Yolo	After 5 hours of applying paraquat while wearing a poorly fitting respirator, he developed acute vomiting, sore throat, and nasal irritation. A paraquat screen was negative.

Selected paraquat cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Exposure circumstances and symptoms
89-2411	11/3/89	San	3 workers felt mist from a paraquat application in an adjoining field. Symptoms
89-2412		Joaquin	included headache, dizziness, nausea/vomiting, sore throat, blurred vision, blistering
89 -2413			rash on arms, lower legs, and face

Environmental fate and effect on wildlife

Paraquat binds tightly to soil and is consequently persistent (half-life greater than 1000 days), although the bound material has a low biological activity. It has a markedly variable aqueous persitence(in a laboratory stream water column, the half-life was 13.1 hours, but in other study; at low levels in water, however, paraquat had a half-life of 23 weeks).[51]

The compound is less toxic to birds (acute oral LD50 values of 981 mg/kg and 970 mg/kg in bobwhite and Japanese quail,) than to mammals.[51] Toxicity to aquatic life is moderate (96-hour LC50 for paraquat is 32 mg/L in rainbow trout, and 13 mg/L in brown trout; LC50 for the aquatic invertebrate Daphnia pulex is 1.2 to 4.0 mg/L).

Neurotoxicity

The structural analogy between paraquat and 1-methyl-4-phenylpyridinium (MPP+) has given rise to concern that exposures to paraquat might lead to early onset of Parkinson's disease similar to that associated with the use of the meperidine analog MPTP as a street drug in the U.S. during the early 1980's.[53] Although paraquat can penetrate the blood brain barrier when administered to animals at high doses,[54] it does so to a very limited extent[55] and principally in areas of the brain with a diminished barrier.[56] [57] The hypothetical concern raised by the structural analogy to MPP+ has not been supported by clinical evaluation of the sequelae of near-fatal poisoing cases.[58] Pathologic findings in the brain in fatal poisonings are likely secondary to anoxia.[59]

Triclopyr

Triclopyr is a selective systemic herbicide - absorbed by foliage and roots and translocated throughout the plant. Susceptible species of broad leaf weeds treat the compound as a growth hormone or auxin. Grasses are unaffected at normal application rates.[60]

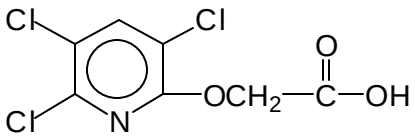
It has a low acute mammalian toxicity (rat oral LD50 of triclopyr - 630 to 729 mg/kg and over 2000 mg/kg for various amine and ester formulated products). The level of irritation produced depends upon the formulation.[61] Chronic toxicity studies in Fischer 344 rats using doses of triclopyr (70/sex/group) at 0 (vehicle = acetone), 3, 12 or 36 mg/kg/day. 10/sex/group were sacrificed at 6 months and 12 months showed decreased kidney weight but no oncogenic effect. Studies in albino rats at 0, 3, 10 or 30 mg/kg body weight, 50/sex/group whowed increased kidney weights but no oncogenic effect.

Environmental effect of triclopyr on understory plants - it characteristically causes treated foliage to turn red and the effect is therefore very noticeable. Photograph - Paul Holzberger - Humboldt County Department of Agriculture

Basic Pesticide Toxicology

California experience

There were 8 cases involving possible, probable and definite illnesses reported to the California illness registry between 1982 and 1995 with triclopyr identified as the primary pesticide. These included 6 cases involving exposure to handlers (mixers/loaders/applicators/flaggers), usually involving topical symptoms.



Triclopyr

Activity	Topical	Respiratory/Systemic	Total
Handlers	5	1	**6**
Drift	1	0	**1**
Other	1	0	**1**
Total	**7**	**1**	**8**

Individual cases involving triclopyr and other forestry herbicides are shown in the table below.

Selected triclopyr cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Exposure circumstances and symptoms
92- 445	3/18/92	Contra costa	While using backpack sprayer, a hose failed, spilling material onto his buttocks and lower back. It soaked through his coveralls causing rash on the exposed area.
92-1279	4/5/92	Santa Clara	A worker mowed lawns treated with triclopyr 3 days earlier. He developed irritation and blisters of the forearms within a few hours of handling clippings from the treated lawn.
91- 509	3/6/91	Glenn	Worker was sprayed in the left eye when he opened a pressurized spray tank, while his goggles were on his forehead. He developed injected sclera and conjunctivitis.
90-1593	6/27/90	Sonoma	A worker sprayed herbicide on lawn and developed dizziness, nausea, slurred speech and weakness.
89-1503	6/30/89	Santa Clara	Accidental discharge from a spray gun shot herbicide in a worker's eye and he developed conjunctivitis.
88-2832	10/27/88	Imperial	The worker was spraying weeds along the roadside when a big rig drove by and blew spray back in her face. She developed mild erythema on her face.
87-2579	11/6/87	Del Norte	Forestry applicator developed eye irritation after it was scratched by brush possibly contaminated with triclopyr.
87-2723	11/23/87	Del Norte	Forestry worker developed eye irritation after he set an open container of triclopyr down hard and some of the material splashed into his eye.

Environmental fate and effect on wildlife

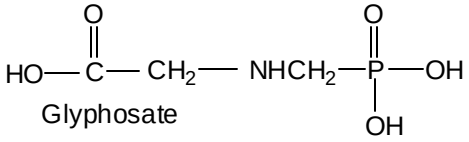
The half-life in soil ranges from 30 to 90 days, depending on soil type and environmental conditions, with an average of 46 days. Reported half-lives in water are 2.8 to 14.1 hours, depending on season and depth of water.[61] The compound is translocated by plant after uptake by roots or foliage. The estimated half-life after forest application is is 2 to 3 months.

Basic Pesticide Toxicology

Triclopyr has minimal aviantoxicity (LD50 in the mallard duck, for example, is 1698 mg/kg; the 8 days LC50 in bobwhite quail and Japanese quail fed triclopyr for are 2935 ppm and 3278 ppm). It also has a low toxicity for fish (96 hour LC50 117 mg/L in rainbow trout and 148 mg/L in bluegill sunfish) and aquatic invertebrate Daphnia magna (LC50 for the amine salt of 1170 mg/L).

Basic Pesticide Toxicology

Glyphosate



Glyphosate

Glyphosate isopropylamine salt (Roundup®) (CAS number: 38641), commonly formulated with a surfactant or spreading agent to enhance leaf surface contact, inhibits 5-enolpyruvylshikimate-3-phosphate-synthase (EPSPS), an enzyme involved in the synthesis of the aromatic amino acids, tyrosine and phenylalanine - needed for manufacture of proteins.[60] Mammals obtain aromatic amino acids from dietary sources, so that glyphosate has very low systemic toxicity for humans. Glyphosate is largely non-selective in its effect on plant biochemistry - so that it has a broad range of action similar to that of paraquat. Ivy-like plants with thick, waxy leaf surfaces are relatively resistant to glyphosate because they do not permit translocation of salts or polar compounds. This limitation can be overcome by adding a detergent to the mixture for spraying. Glyphosate is used on an extremely widescale in the United States for both agricultural and non-agricultural weed control, but paraquat is still preferred in many areas of the world because of its lower cost.

The systemic toxicity of glyphosate is low (acute oral LD50 of 5600 mg/kg in the rat). The active ingredient also causes minimal irritation of the eye and skin. Nevertheless, formulations of glyphosate are frequently associated with irritation, probably due to the surfactant component. Allergic dermatitis due to an isothiazoline preservative in the formulated product has also been reported.[62] In animal tests, glyphosate is not a carcinogen or a teratogen.[63]

California Experience

There were 557 cases involving possible, probable and definite illnesses reported to the California illness registry between 1982 and 1995 with glyphosate identified as the primary pesticide. These included 478 cases (85.8% of the reported total involving exposure to handlers (mixers/loaders/applicators/flaggers), and typically involving topical symptoms.

Activity	Topical	Respiratory/Systemic	Total
Handlers	407	71	**478**
Drift	25	16	**41**
Clean/fix/repair	9	0	**9**
Field residue	2	1	**3**
Other exposures	17	9	**26**
Total	**460**	**97**	**557**

Some typical individual cases included:

Selected glyphosate cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Exposure circumstances and symptoms
82- 502	3/18/82	Riverside	Was spraying weeds, and mist irritated his eyes.
82-1050	6/7/82	Riverside	Was spraying tall weeds, brushed up against some that were still wet from the application, and got some material in her eye.

Selected glyphosate cases reported to the California pesticide illness registry, 1982-1995

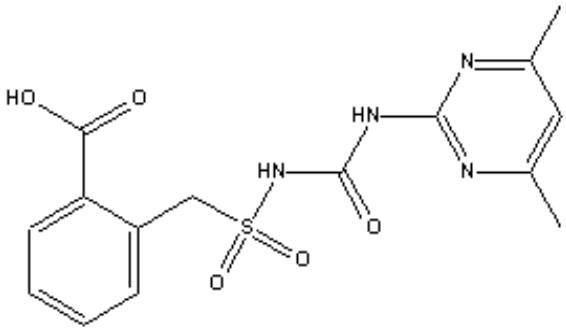
ID	DOI	County	Exposure circumstances and symptoms
82-1137	6/7/82	Santa Cruz	Spraying glyphosate when the wind blew spray on her face. She developed itchy, burning eyes and a sore throat.
94- 566	4/6/94	Fresno	Worker was spraying the grounds of a cotton gin with glyphosate when some of the material dripped onto his left arm and leg. He developed a red, itchy rash on the left arm and thigh.
95-1823	9/19/95	Alameda	A landscaper bumped a sprayer while loading it into his trailer and glyphosate sprayed into his face. He flushed his eyes, but still developed eye irritation.
95-1846	6/23/95	Fresno	A worker was spraying weeds when the wand separated from the rubber hose; glyphosate splashed onto his face and eyes. He developed erythema of the cheeks, injected sclera of both eyes.

Environmental fate and effect on wildlife

The average soil half-life is 47 days and it is strongly adsorbed to most soils. Because of this property, it does not leach to groundwater, even though it is highly soluble. The aqueous half-life ranges from 12 days to 10 weeks. [64] On contact with vegetation, it is translocated and does not usually leave a surface residue.

Glyphosate has a low avian toxicity (dietary LC50 in both mallards and bobwhite quail is greater than 4500 ppm). It also has a low aquatic toxicity (The 96-hour LC50 is 120 mg/L in bluegill sunfish, 168 mg/L in harlequin, and 86 mg/L in rainbow trout; greater than 10 mg/L in Atlantic oysters, 934 mg/L in fiddler crab, and 281 mg/L in shrimp).[64]

Sulfometuron



Sulfometuron-methyl is used as either post-emergent or pre-emergent spray for control of annual and perennial grasses and broad leaved weeds. In forestry applications, it is used to control woody tree species. It functions by blocking cell division in the stem and root tips. Its systemic toxicity is low (rat LD50 > 5000 mg/kg). It is not a skin irritant or a skin sensitizer, but does cause mild irritation in the Draize eye test. At doses of 25 mg/kg/day in chronic feeding studies, it causes reduced red-blood cell counts and increased liver weight in dogs. Increased white cell counts were observed in a chronic rat feeding study.[65]

Environmental fate and effects on wildlife

Sulfometuron-methyl has a low avian toxicity (oral LD50 in mallards > 5000 mg/kg) but is somewhat toxic to fish (the LC50 in rainbow trout and in bluegil sunfish is greater than 12.5 mg/L) and to the water flea, Daphnia magna (LC50 is 125 mg/L for the technical material and greater than 1000 mg/L for dispersible granules).

The soil half-life ranges from 20 to 28 days. It breaks down in water with a half-life ranging from 1 day to more than 2 months. [65]

California experience

Basic Pesticide Toxicology

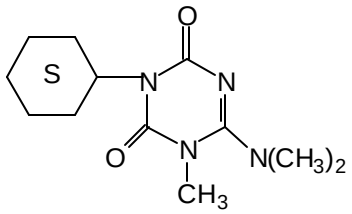
There were 4 cases of possible, probable, or definite illness reported to the California illness registry between 1982 and 1995, including 3 in handlers and 1 case of accidental drift exposure:

Sulfometuron cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Comments and Symptoms
88-1945	8/10/88	San Diego	Individual was sprayed in the face with a sulfometuron methyl when a hose burst. He developed chemical irritation of the left eye.
89- 40	1/24/89	Santa Clara	A worker was mixing and loading sulfometuron methyl without safety gear; he developed a cough and irritated throat.
89-2410	10/17/89	Sacramento	A worker drove a vehicle while someone else sprayed, but was exposed to sulfometuron methyl when he stepped outside. He developed tingling of skin on arms and face, headache and sore throat.
91- 74	12/21/90	Santa Clara	A worker noticed a broken pipe on rig while spraying sulfuron methyl. He did not have any recorded direct exposure, but the following day developed swelling and redness of the face and ears.

Hexazinone

Hexazinone is a contact and residual herbicide - controls woody plants in reforestation - site preparation for conifer release - available as a water soluble powder, liquid applied undiluted from hand sprayer. Mouse oncogenicity study showed an increase in hepatocellular adenomas and carcinomas in females at 10,000 ppm daily in the diet. Studies in the dog and hamster did not show any oncogenic effects.



Hexazinone

California Experience

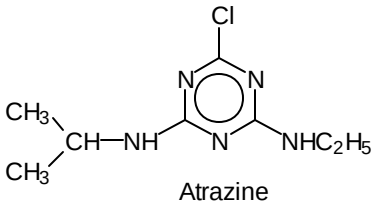
Only 2 cases reported to the state illness registry between 1982 and 1995, as described below.

Hexazinone cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Symptoms and Comments
88- 479	16-Mar-88	Tuolumne	Worker developed breathing difficulties after applications of hexazinone. His doctor diagnosed bronchitis and slight asthma probably exacerbated by work environment.
90- 604	24-Feb-90	Kern	Ground applicator spraying hexazinone and diuron wearing coveralls, goggles, rubber gloves, boots and respirator developed hives all over his body. He did not have any recorded accidental exposure.

Atrazine

Atrazine is a pre-emergent herbicide used for control of broadleaf and grassy weeds in field crops such as corn and sorghum, for forestry plantings, and as a non-selective pre-emergent for road side right-of-way and other non-crop applications. Over 64 million acres of cropland were treated with atrazine in the U.S. in 1990.



Atrazine

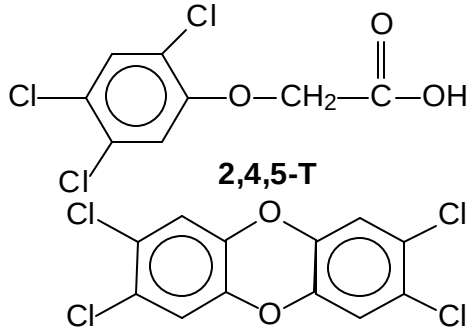
Atrazine has a low systemic toxicity (LD50 - 3090 mg/kg in rats), but has been

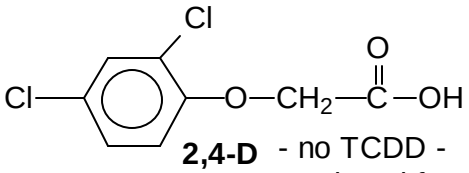
identified as mild irritant.[66] Severe contact dermatitis to atrazine has been reported in a farmer with a history of dermatitis caused by propachlor. This patient had a positive patch-test reaction to 1:1000 dilution of a commercial atrazine formulation.[66]

Similar to other triazine compounds, atrazine causes mammary tumors in rodent feeding studies.[66] In lifetime feeding studies, the tumors occurred in association with an atrazine concentration of 1000 ppm, but not with a concentration of 500 ppm in the diet.[67]

California experience

There was one case involving possible, probable and definite illnesses reported to the California illness registry between 1982 and 1995 with atrazine identified as the primary pesticide. This was a case of eye irritation (**82-1198**) associated with mixing and loading of atrazine.





2,4-D - no TCDD - produced from direct chlorination of phenol

Dibenzodioxins -contaminants derived from hydroxylation of tetrachlorobenzene in manufacture of 2,4,5-T

Phenoxy compounds

Salts of the chlorophenoxyacetic acids (2,4-D, 2,4,5-T, MCPA) are readily absorbed by the roots of broadleaf plants and the numerous ester deriviatives are readily absorbed by the foliage and are translocated to the plant growth zones and function as hormonal growth inhibitor (auxins).Tomlin, 1997 #2] The selective effect of plants on broadleaf plants has made them popular for use on grain crops and they are used extensively on turf as well. 2,4,5-T is no longer on the market because of the tendency of the manufacturing process (hydroxylation of tetrachlorobenzene) to run out of control, producing contamination with dioxin and catastrophic process breakdowns. 2,4-D, produced by chlorination of phenol, is not subject to contamination with the dioxins present in formulations made from 2,4,5-T.[62]

2,4-dichlorophenoxy acetic acid and its derivatives can provoke mild to moderate acute skin and respiratory irritation. Systemic poisoning after ingestion produces myotonia, nausea, vomiting, abdominal pain, and diarrhoea followed by muscle twitching, myotonia, metabolic acidosis, and a hypermetabolic state with fever, tachycardia, hypertension, sweating, convulsions, and coma.[43] Cases of peripheral neuropathy have been reported following relatively large dermal exposures to 2,4-D over the course of a few days and following cases of ingestion.[68, 69 70, 71 43]

Basic Pesticide Toxicology

California experience

There were 28 cases of possible, probable, or definite illness reported to the California illness registry between 1982 and 1995, with 18 (64.3%) involving handlers and 8 (25.8%) related to a single episode of accidental drift exposure:

Activity code and description	Topical	Systemic	Total
Handlers	16	2	**18**
Drift	1	7	**8**
Exposure to concentrate	0	1	**1**
Other	0	1	**1**
Total	**17**	**11**	**28**

Typical cases included:

Selected 2,4-D cases reported to the California pesticide illness registry, 1982-1995

ID	DOI	County	Symptoms and Comments
89- 469 and related cases	3/27/89	Alameda	Tightness in chest, headache, dizziness and topical symptoms following drift exposure to 2,4-D in employees of a company producing electricity by wind power.
91-3202	4/26/91	San Diego	Itching and rash on the arms, neck and around the eyes in an applicator employed by the county.
92- 416	3/31/92	Contra Costa	Headache, shortness of breath and nose, throat and lung irritation. Occupation = 1999
93- 419	2/3/93	Fresno	An applicator developed a macular rash on neck and chin.
94-1067	4/20/94	Calaveras	Redness and severe pain in the right eye in an applicator.

Cancer

Bioassays have shown limited evidence for carcinogenicity of 2,4-D in animals (astrocytomas in male rats at high doses only in one study).[72] A recent repeat of the chronic rat study did not show an increase in astrocytomas.[73] A 1977 review by the International Agency for Research on Cancer (IARC) did not find evidence of 2,4-D related tumors in a mouse study involving doses of 323 mg/kg in the diet. Similar studies of the isopropyl, butyl, and isooctyl esters of 2,4-D were also negative, and administration by subcutaneous injections did not increase tumor incidence.[74] Evaluation of 0.05% 2,4-D as a promoting agent for rodent liver tumors induced by 2-acetylaminofluorene (2-AAF) and diethylnitrosamine (DEN)was negative. In contrast, 0.1% nafenopin (NAF), 0.015% perfluorooctanoic acid (PFOA), and 0.05% 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) were associated with hepatocellular carcinoma in 83, 33 and 16% of the treated animals, respectively. Tests with the Salmonella mutagenicity assay, the micronuclei assay, and the mouse liver assay for unscheduled DNA synthesis.

Case-control studies in the USA have however, found associations between frequent use of 2,4-D. The initial study, based upon the Kansas tumor registry, identified a six-fold risk of non-Hodgkin lymphoma (NHL) associated with handling 2,4-D more than 20 days/year.[75] A case-control study in Nebraska[76] identified a non-significant 50% increase in risk of NHL among 2,4-D handlers and an 80% increase in risk among those exposed more than 20 days per year. Adjustment for the possible effects of other pesticides, fungicides and organophosphates, yielded a 3 fold risk for NHL among frequent 2,4-D users. Atrazine also appeared to be a risk for NHL in this study, but this apparent association disappeared after adjustment for exposure to 2,4-D.[77] A similar study in Iowa and Minnesota case-control study by Cantor[78] identified small increase in risk for 2,4-D, but the risks did not increase with latency or failure to use protective equipment. Similar to the Nebraska study, association was noted for several insecticides

(carbaryl, chlordane, dichlorodiphenyltrichloroethane, diazinon, dichlorvos, lindane, malathion, nicotine, and toxaphene), especially with a history of first use prior to the year 1965 and failure to wear protective equipment. The OP's were also identified as an NHL risk factor for women applicators in Nebraska.[79]

The of findings of the various 2,4-D studies were criticized as inconsistent by Bond,[80] who also described the mortality in 2,4-D manufacturing employees at Dow Chemical. An initial study showed 2 deaths due to NHL.[81, 82] A follow-up study on the same cohort reported recently showed 3 cases of NHL compared to 1 expected.[83] The study authors reported that the cases occurred in employees with relatively low exposure and with short-latency. Zahm reported a cohort study on employees of a lawn service company who frequently handled 2,4-D, along with other lawn care chemical products. There were four deaths due to NHL (SMR = 1.14, CI = 0.31, 2.91), including 3 in male lawn applicators (SMR = 1.63, CI = 0.33, 4.77), and 2 in applicators employed for three or more years (SMR = 7.11, CI = 1.78, 28.42). There was also an excess of bladder cancer in the cohort, based on 3 cases. Two of the three bladder cancer cases did not have any direct contact with pesticides.[84] A study of phenoxy manufacturing workers in Denmark, did not show any association between exposure and risk of NHL, but did identify a non-significant risk for soft-tissue sarcoma.[85] A study of Swedish forestry workers - whose principal exposure was to MCPA - did not show any association between herbicide exposure and NHL. Employment in animal husbandry - raising minks - was associated with a significant risk of Hodgkin's disease.[86]

An interesting side-line in research on 2,4-D has been a series of studies on exposure to 2,4-D as a risk factor for lymphoma in animals. In 1991, Hayes[87] reported on a hospital based case-control study that used questionnaires given to the owners of 491 animals with canine lymphoma, 466 animals with non-cancerous conditions, and 479 controls with other types of tumors (to limit potential response bias). Owners of dogs with developed malignant lymphoma reported using 2,4-D lawn treatments 2,4-D significantly more frequently than owners of control animals (odds ratio = 1.3). The risk increased to twofold excess with four or more yearly owner applications of 2,4-D. The degree of animal exposure to 2,4-D following yard treatments was subsequently evaluated by Reynolds.[88] In 44 dogs exposed to 2,4-D-treated lawns an average of 10.9 days after application, 2,4-D concentrations greater than or equal to 10.0 mug/l were found in 33 dogs (75%) and concentrations of : 50 mug/l were found in 17 (39%). The highest concentrations of 2,4-D in urine (21.3 mg/l) was found in animals sampled within 2 days after application.

The suggestion from the Hayes study that 2,4-D causes lymphoma in dogs is contradicted by the results of an experimental 2,4-D chronic feeding study of beagles involving doses of 1-10 mg/kg/day.[89] Some non-malignant effects on the kidney and liver were observed at the highest doses, but no malignancies were observed. As with experimental other studies of chronic effects in animals, the high doses administered were offset by small numbers of animals (5/dose group). The results of the experimental study do indicate that if 2,4-D does cause canine lymphoma, it occurs too infrequently to be recognized in a study with such small numbers. (Typical rodent carcinogenicity studies, for example, have 50 or 70 animals in each dose group.)

Forest herbicide use data and environmental monitoring

III - Exposures to forestry herbicides and environmental monitoring

Forestry herbicides are used for control of native plants (e.g., manzanita and bear-berry) that germinate in the aftermath of fires and after clear-cuts and compete for growth resources with evergreen seedlings. Depending upon accessibility and other circumstances, by aerial applications (figure) or ground (figure). Unsurprisingly, monitoring studies indicate a markedly greater dispersion of active ingredients and a larger percentage of positive samples with aerial compared to ground applications.[90] Typical herbicides applied to forestry acreage in California include triazines, phenoxy herbicides and related auxin compounds, carboxylic acids, and urea-aniline compounds:.

Reported applications to forestry acreage in 1995

Commodity / chemical	Applications	Pounds	Acres
Atrazine	44	3,830.49	1,873.50
2,4-D,butoxyethanol ester, dimethylamine salt, isooctyl ester	154	18,381.70	8,765.50
Glyphosate, isopropylamine salt	243	30,775.70	25,640.75
Hexazinone	218	47,022.37	11,783.08
Octylphenoxypoly ethoxyethanol (surfactant)	110	14,453.68	16,440.00
Paraquat dichloride	2	0.99	0.40
Petroleum hydrocarbons + Polyoxyethylene mixed fatty acid ester	93	26,833.50	6,874.50
Sulfometuron methyl	52	325.33	3,447.00
Triclopyr + butoxyethyl ester	469	81,016.20	37,413.30
Reported forestry total	1,908	230,947.9104	Not specified

Environmental Monitoring for Herbicides

For monitoring studies DPR scientists selected specific plant species and plant parts to sample based on discussions with basket makers and site observations. In 1997, for example, sampling for glyphosate, triclopyr and hexazinone was conducted in 17 treatment units in the Lassen, Eldorado, Stanislaus, and Sierra National Forests. The plants selected included stem or shoot samples (bitter cherry, buckbrush, deer brush, dogwood, willow), root samples (bracken fern, soaproot), leaf or stalk samples (deergrass, golden fleece, pearly everlasting), and fruit or nut food samples (elderberry, manzanita, oak). For 13 species, staff collected 211 samples, including 92 in treatment areas; 49% contained detectable residues of herbicides. Of the 119 samples collected outside treatment areas, 3% contained detectable residues of herbicides. The lowest concentration that was reliably detected was 0.10 parts per million (ppm) for glyphosate, 0.05 to 0.10 ppm for hexazinone, and 0.01 to 0.07 ppm for triclopyr in various plants.[90]

Forest application by helicopter - photo from Environmental Monitoring and Pest Management Branch, California Department of Pesticide Regulation

Forest herbicide use data and environmental monitoring

Forest herbicide use data and environmental monitoring

	Inside Treatment Areas			Outside Treatment Areas		
Herbicide/method	**no. positive**	**no. samples**	**range[a] concentration (ppm)**	**no. positive**	**no. samples**	**range[a] concentration (ppm)**
Glyphosate/ground	16	31	0->10	2	44	0-10
Hexazinone[b]	20	43	0-10	1	46	0-0.5
Pronone®/air	16	17	0-10	0	17	ND
Pronone®/ground	1	10	0-0.5	0	19	ND
Velpar®/ground	3	16	0-1.0	1	10	0-0.5
Triclopyr/ground	9	18	0-0.7	1	29	0-10
TOTAL	**45**	**92**	**0->10**	**4**	**119**	**0-10**

[a] ND - none detected. Detection limits: glyphosate 0.1 ppm, hexazinone 0.05-0.2 ppm and triclopyr 0.01-0.07 ppm

[b]Pronone® - granular formulation; Velpar® - liquid formulation

The residues remained detectable in scattered areas for several months after application.

Herbicide	1-8 wk	9-16 wk	17-24wk	25-32 wk	33-40 wk	41-48 wk
Glyphosate	7/10	7/14	0/5	2/2	not sampled	not sampled
Hexazinone	11/14	5/8	3/11	0/1	1/3	0/6
Triclopyr	5/7	3/10	1/1	not sampled	not sampled	not sampled
Totals	23/31	15/32	4/17	2/3	1/3	0/6

Dissipation studies

An additional dissipation study was conducted in National Forest sites in the Central Sierras. A total of 209 plant samples from 41 treatment sites were collected among the Eldorado, Stanislaus and Sierra National Forests.[91] Samples consisted of the four plant species (bracken fern roots *(Pteridium aquilinum var. pubescen*s) for basket weaving, buckbrush or deerbrush shoots *(Ceanothus intergerrimu*s, *Ceanothus cuneatus*, respectively) for basket weaving, golden fleece foliage *(Ericameria arborescen*s) for medicinal purposes; and manzanita berries *(Arctostaphylos* spp.) for food) treated with glyphosate, triclopyr, or hexazinone. Because of the differences in application rates and application methods (ground broadcast with granules versus direct liquid spray), and plant parts sampled (aerial parts versus underground rhizomes) herbicides concentration in plants varied greatly. Immediate post-application concentrations varied from 122 ug/g in golden-fleece foliage for hexazinone liquid spray to non-detectable in bracken fern rhizomes in site treated with granular hexazinone. Glyphosate and triclopyr were remained detectable at concentrations of about 1 ppm in dead or decayed plant material sampled 36 weeks after treatment. At this time, most of the plant materials were dead and decayed.

Ground application crew in previously burned forest hill side - photo from Environmental Monitoring and Pest Management Branch, California Department of Pesticide Regulation

Forest herbicide use data and environmental monitoring

Hexazinone residues between 0.1 and 1 ppm were also detected in buckbrush shoots at the application site 60 weeks after treatment. [91, 92] Offsite movement was evaluated by taking a set of 6 samples at varying distances from the application site. At 5-15 feet from the application site, 2/6 samples had herbicide residue detected, compared with 1/6 samples collected at 20-40 feet, 50-70 feet and and 80-100 feet. [91, 92]

Surface water monitoring

Contamination of surface water has also been evaluated by CDPR in samples collected in the Klamath River area in Humboldt and Del Norte counties.[93] Ground application sites studied included the (A) Terwer Creek Tributary (9 treated acres with 32 pounds of atrazine on 4/20/99), (B) Blue Creek (36 acres treated with 130 lb. atrazine and 20 lb. triclopyr on 4/21/99), and (C) Pecwan Creek Tributary (25 acres treated with 91 lb of atrazine on 4/26/99). A Terwer Creek site (D) was also monitored after an aerial application (360 acres treated with 360 lb. of triclopyr and 338 lb of 2,4-D. The monitoring conducted at site A on 5/1/99, site B and site C on 5/2/99, and on site D 5/14/99, in each case more than a week after application, did not show any peristent residues. Samples on the day of application confirmed the presence of active ingredients in the tank mixes in concentrations ranging from 0.26% (tricoplyr) to 1.13% (2,4-D) and to 2.9% (atrazine).

Monitoring studies were also conducted in Del Norte County of aerial application sites in the fall of 1999. Sites monitored included site E - the Hoppaw Creek Tributary (40 acres treated on 9/16/99 with 60 lbs of triclopyr), site F - Hunter Creek Tributary (105 acres treated on 9/14/99 with 157.5 lbs of triclopyr), and site G Wilson Creek Tributary (13 acres treated 9/20/99 with 13 lbs of glyphosate). Monitoring at sites F and G, beginning on the day of application, did not show detectable levels of herbicide in any of the runoff samples collected. At site E, no samples were positive for triclopyr on the day of application (9/16/99), but surface water samples collected 10/27/99 showed triclopyr at concentrations ranging from 0.174 to 0.430 ppb. Reference values for triclopyr in water include an operational standard of 10 ppb used by the North Coast Regional Water Quality Board and a chronic toxicity threshold of 30 ppb used by the California Department of Fish and Game.[94]

Manner of exposure in production of baskets

Exposure to residues on plant materials usually comes from skin contact. However, weavers often place plant materials in the mouth while making baskets. Residues on plants may be absorbed in the process. Direct measurement of exposures to basketweavers, comparable to those performed on forestry application workershandling glyphosate[95] and hexazinone [96]were not available.

Comparison to residue tolerances

In terms of the question regarding residue tolerances at the California Clinician's Conference (above), the peak residue levels measured were in the range of the EPA commodity tolerances for the monitored herbicides. The ranges were typically wide, with the high end of the range derived from tolerances on forage crops and the low end of the range from tolerances meat, milk and other animal products. For example, the peak measured residue of glyphosate was 10 ppm, compared to allowable residue tolerances ranging from 0.1(sunflower seeds) to 200 ppm (alfalfa hay). The level was above the 0.5 ppm tolerance for glyphosate on typical food commodities (citrus and tree nuts). For triclopyr, the peak residue level measured was 0.7 ppm. This was also in the range, 0.01(milk) - 500 ppm (grass forage)of the EPA commodity tolerances and somewhat above the 0.3 ppm tolerance for triclopyr on rice.

Forest herbicide use data and environmental monitoring

Herbicide Commodity Tolerances

Commodity / chemical	Peak residue (ppm)	Tolerance Range (ppm)	Representative Commodities	Tolerance (ppm)
Atrazine	NA	0.02-15	Wheat	0.25
2,4-D	NA	0.05 - 1000	Fruits/berries	0.1
			Apples	5.0
			Grapes	0.5
Glyphosate	10	0.1-200	Citrus	0.5
			Tree nuts	1.0
Hexazinone	10	0.1-10.0	Blueberries	0.2
			Pineapples	0.5
Sulfometuron methyl	NA	0.05 - 20.0	Wheat	0.1
Triclopyr	0.7	0.01-500	Rice	0.3

NA - not available

Exposure estimates made from modeling programs

Estimates of exposure to forestry herbicides during basketmaking and other tribal cultural activities is currently being performed using the CalTOX modeling program.[97] A description of the modeling procedure has recently been published and included a comparison between CalTOX residue estimates for plants, ground-surface soil, root-zone soil, vadose-zone (shallow soil below the root zone and above the water table) and residue measurements reported in the literature. A preliminary estimate performed with CalTOX for granular hexazinone (Pronone® 5G and 25G), indicates that exposure is to be 1,000 - 10,000 times lower than the Food and Drug Administration's acceptable daily intake (ADI) level of 0.033mg/kg/day. A more complete evaluation, including estimated exposures to liquid formulations of hexazinone, glyphosate and triclopyr is still in progress.

North Coast Cancer Data

V - North Coast Cancer Data

The California pesticide illness registry collects principally data on acute illnesses. Questions regarding possible environmental causes of cancer can however be addressed by evaluating data from the California Cancer Registry (CCR). It collects data about all cancers diagnosed in California (except basal and squam0ous cell carcinoma of the skin and carcinoma in situ of the cervix). The Registry is a collaborative effort involving the California Department of Health Services, ten regional registries, hospitals, cancer researchers throughout the nation and the Public Health Institute. The database includes information on demographics, cancer type, extent of disease at diagnosis, treatment, and survival. As in other states, the lifetime population incidence of cancer in California is high, affecting 2 of every 5 residents. In Region 6 (16 Northern California Counties - Butte, Colusa, Del Norte, Glenn, Humboldt, Lake, Lassen, Mendocino, Modoc, Napa, Plumas, Shasta, Siskiyou, Sonoma, Teharna, Trinity), the annual incidence of new cancer cases is 387.2/100,000 population - approximately 3,300 cases in the region annually in Humboldt and Del Norte county and 36,000 in registry region 6 as a whole (Table 1).

Table 1. All Sites, by gender/age - Region 6, Humboldt/Del Norte - 1993-1997
Average-annual age-adjusted incidence rate most recent 5-year period (per 100,000)

	Region 6		Humboldt/Del Norte	
	New Cases	Age-Adjusted-Rate (AAR) Cases/per 100,000	New Cases	AAR
Total	36,276	395.1 (CI*= 390.9-399.4)	3,397	387.2 (CI = 373.9-400.9)
Male	18,769	447.3 (CI = 440.7-453.9)	1,705	424.6 (CI =404.3-445.7)
Female	17,507	357.4 (CI = 351.8-363.1)	1,692	361.1 (CI =343.4-379.7)

* 95% confidence interval

Comparison of age-adjusted site-specific cancer rates (AAR) for Region 6 and for Del Norte and Humboldt Counties with those for California as a whole indicate relatively few differences. The data in Table 2 and Table 2b indicate that there was a 2/3 lower rate, compared with the state as a whole, in Region 6 and in Humboldt/Del Norte Counties for liver cancer. For lung and bronchial cancer, the rates were 15-20% higher in Region 6 and in Humboldt/Del Norte Counties compared with the statewide rates. Rates of kidney and renal pelvis cancers were also elevated for females, in Humboldt and Del Norte counties, based upon relatively small number of total cases.

Figure 1 shows time-trends in the incidence of Non-Hodgkin's lymphoma (NHL) in California Cancer Registry Region 6 and in the subregion encompassing Humboldt and Del Norte Counties between 1988 and 1997. Although the rate for the subregion was elevated above the statewide rate (15.1 cases/100,000 population) between 1988 and 1995, the rate fell to a level equivalent to the statewide rate for 1996 and 1997. The rate of lung cancer, by contrast, was consistently elevated above statewide rates for both Region 6 and the Humbold/Del Norte subregion throughout the 1993-1997 period. The rate for males in the subregion varied between between 80-110 cases/100,000 population compared to the statewide rate of 66.5 cases/100,000. The rate for females was approximately 60 cases/100,000 compared to the statewide rate of 42.5 cases/100,000.

Although the excess of lung cancer in the region may be attributable to differences between smoking habits in the Region 6 versus other 5 Cancer Registry regions, the cause of the temporary elevation of rates of NHL between 1988 and 1995 in the subregion is less clear. No single cause for this condition has previously been described. Whether any of the cases had exposure to 2,4-D comparable to the cases in the investigation reported by Hoar[75] cannot be ascertained from the available information.

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Table 2. Average-annual age-adjusted incidence rates per 100,000 population, by site and sex, all races combined - 1993-1997

Site	Region 6				Humboldt/Del Norte				State	
	Male		Female		Male		Female		Male	Female
	Cases	AAR	Cases	AAR	Cases	AAR	Cases	AAR	AAR	AAR
Oral Cavity & Pharynx	611	**15.3**	312	**6.4**	40	**10.3**	25	**5.2**	**14.0**	**6.0**
Esophagus	240	**5.7**	79	**1.5**	24	**5.8**	10	--	**5.6**	**1.6**
Stomach	349	**8.2**	214	**3.9**	37	**9.0**	19	**3.3**	**10.5**	**4.9**
Colon & Rectum	2,129	**49.1**	1,870	**33.3**	214	**52.9**	166	**32.7**	**47.8**	**33.7**
Colon	1,484	**33.7**	1,417	**24.7**	151	**36.6**	129	**24.8**	**33.1**	**24.8**
Rectum & Rectosigmoid	645	**15.5**	453	**8.6**	63	**16.3**	37	**7.9**	**14.7**	**8.9**
Liver	160	**3.9**	70	**1.4**	16	**3.8**	7	--	**5.9**	**2.1**
Pancreas	436	**10.3**	444	**7.8**	31	**7.6**	44	**8.4**	**9.6**	**7.5**
Larynx	253	**6.3**	79	**1.8**	26	**6.6**	6	--	**5.9**	**1.3**
Lung & Bronchus	3,230	**77.4**	2,697	**54.6**	323	**81.2**	257	**55.5**	**66.5**	**42.5**
Melanoma of the Skin	654	**16.0**	498	**11.1**	85	**21.1**	54	**11.8**	**15.9**	**10.3**
Breast	49	**1.2**	5,239	**112.0**	4	--	486	**105.4**	**0.9**	**106.8**
Cervix Uteri			364	**8.4**			37	**7.8**		**9.2**
Corpus Uteri			1,062	**22.2**			107	**24.4**		**19.9**
Ovary			725	**15.4**			72	**16.3**		**14.1**
Prostate Gland	5,259	**125.2**	--	--	429	**109.2 #**	--	--	**134.7**	
Testis	198	**5.0**	--	--	23	**5.1**	--	--	**4.2**	
Urinary Bladder	1,366	**31.3**	444	**8.0**	120	**29.3**	40	**7.3**	**27.1**	**6.8**
Kidney & Renal Pelvis	493	**12.1**	315	**6.4**	46	**11.7**	36	**7.9**	**11.2**	**5.7**
Brain & Nervous System	290	**7.6**	225	**5.4**	27	**6.7**	30	**6.8**	**6.7**	**4.8**
Thyroid Gland	78	**1.9**	267	**6.4**	9	--	37	**8.5**	**2.6**	**7.1**
Hodgkin's Disease	100	**2.7**	89	**2.3**	13	--	7	--	**2.6**	**2.1**
Non-Hodgkins Lymphoma	768	**18.4**	645	**12.7**	66	**16.6**	83	**17.0**	**19.0**	**11.7**
Multiple Myeloma	211	**4.8**	190	**3.4**	18	**4.3**	20	**3.6**	**5.1**	**3.4**
Leukemia	506	**12.4**	380	**7.9**	41	**10.2**	26	**5.6**	**12.5**	**7.7**
Acute Lymphocytic	46	**1.5**	52	**1.7**	4	–	1	--	**2.0**	**1.5**
Chronic Lymphocytic	149	**3.6**	95	**1.7**	15	**3.9**	6	--	**3.4**	**1.5**
Acute Myelocytic	145	**3.3**	113	**2.3**	5	--	10	--	**3.4**	**2.2**
Chronic Myelocytic	65	**1.6**	60	**1.2**	7	--	5	--	**1.7**	**1.0**

North Coast Cancer Data

Table 2b - Calculation of Ratios of Cancer Rates in Region 6 and Humboldt/Del Norte County versus Cancer Rates for California as a Whole 1993-1997

Site	California		Region 6				Humboldt/Del Norte			
	Male AAR	Female AAR	Male AAR	Ratio	Female AAR	Ratio	Male AAR	Ratio	Female AAR	Ratio
Oral Cavity & Pharynx	14	6	15.3	**1.09**	6.4	**1.07**	10.3	**0.74**	5.2	**0.87**
Esophagus	5.6	1.6	5.7	**1.02**	1.5	**0.94**	5.8	**1.04**	--	
Stomach	10.5	4.9	8.2	**0.78**	3.9	**0.80**	9	**0.86**	3.3	**0.67**
Colon & Rectum	47.8	33.7	49.1	**1.03**	33.3	**0.99**	52.9	**1.11**	32.7	**0.97**
Colon	33.1	24.8	33.7	**1.02**	24.7	**1.00**	36.6	**1.11**	24.8	**1.00**
Rectum & Rectosigmoid	14.7	8.9	15.5	**1.05**	8.6	**0.97**	16.3	**1.11**	7.9	**0.89**
Liver	5.9	2.1	3.9	**0.66**	1.4	**0.67**	3.8	**0.64**	--	
Pancreas	9.6	7.5	10.3	**1.07**	7.8	**1.04**	7.6	**0.79**	8.4	**1.12**
Larynx	5.9	1.3	6.3	**1.07**	1.8	**1.38**	6.6	**1.12**	--	
Lung & Bronchus	66.5	42.5	77.4	**1.16**	54.6	**1.28**	81.2	**1.22**	55.5	**1.31**
Melanoma of the Skin	15.9	10.3	16	**1.01**	11.1	**1.08**	21.1	**1.33**	11.8	**1.15**
Breast	0.9	106.8	1.2	**1.33**	112	**1.05**	--		105.4	**0.99**
Cervix Uteri		9.2			8.4	**0.91**			7.8	**0.85**
Corpus Uteri		19.9			22.2	**1.12**			24.4	**1.23**
Ovary		14.1			15.4	**1.09**			16.3	**1.16**
Prostate Gland	134.7		125.2	**0.93**	--		109.2 #		--	
Testis	4.2		5	**1.19**	--		5.1	**1.21**	--	
Urinary Bladder	27.1	6.8	31.3	**1.15**	8	**1.18**	29.3	**1.08**	7.3	**1.07**
Kidney & Renal Pelvis	11.2	5.7	12.1	**1.08**	6.4	**1.12**	11.7	**1.04**	7.9	**1.39**
Brain & Nervous System	6.7	4.8	7.6	**1.13**	5.4	**1.13**	6.7	**1.00**	6.8	**1.42**
Thyroid Gland	2.6	7.1	1.9	**0.73**	6.4	**0.90**	--		8.5	**1.20**
Hodgkin's Disease	2.6	2.1	2.7	**1.04**	2.3	**1.10**	--		--	
Non-Hodgkins Lymphoma	19	11.7	18.4	**0.97**	12.7	**1.09**	16.6	**0.87**	17	**1.45**
Multiple Myeloma	5.1	3.4	4.8	**0.94**	3.4	**1.00**	4.3	**0.84**	3.6	**1.06**
Leukemia	12.5	7.7	12.4	**0.99**	7.9	**1.03**	10.2	**0.82**	5.6	**0.73**
Acute Lymphocytic	2	1.5	1.5	**0.75**	1.7	**1.13**	–		--	
Chronic Lymphocytic	3.4	1.5	3.6	**1.06**	1.7	**1.13**	3.9	**1.15**	--	
Acute Myelocytic	3.4	2.2	3.3	**0.97**	2.3	**1.05**	--		--	
Chronic Myelocytic	1.7	1	1.6	**0.94**	1.2	**1.20**	--		--	

North Coast Cancer Data

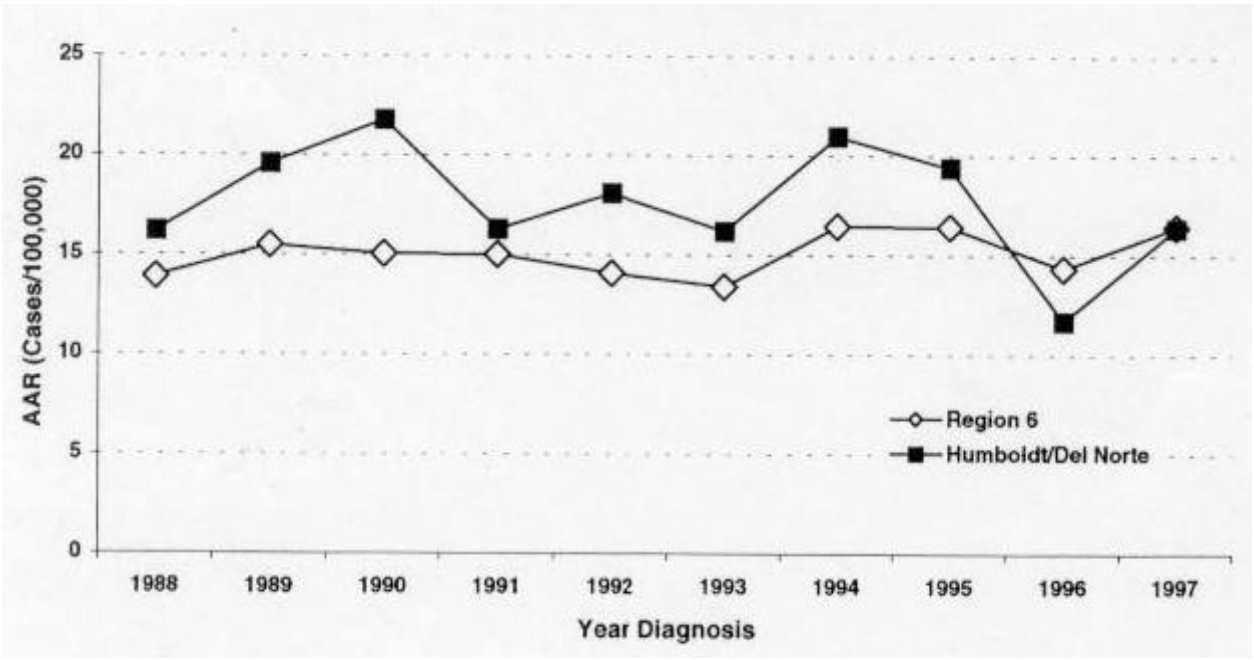

Figure 1 - time trends in the incidence of Non-Hodgkin's lymphoma (NHL) in California Cancer Registry Region 6 and in Humboldt and Del Norte Counties. For comparison purposes, the statewide AAR for NHL from 1993-1997 was 15.1cases/100,000 population , combined rate for males and females.

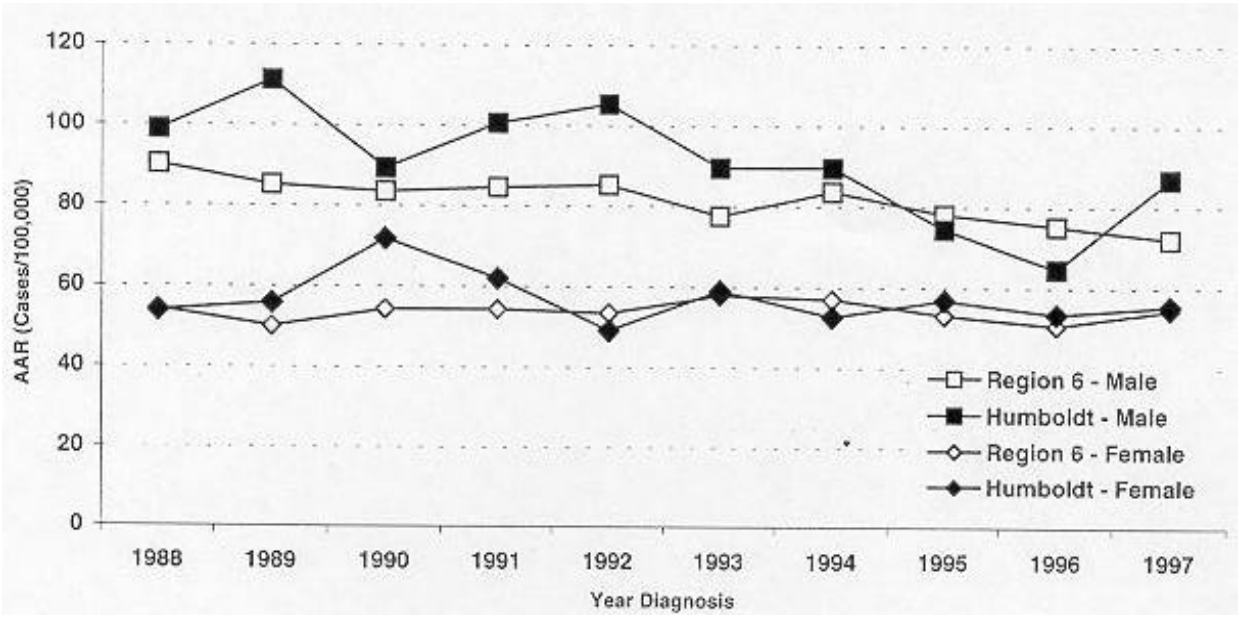

Figure 2 - time trends in the incidence of lung and bronchial cancer in California Cancer Registry Region 6 and in Humboldt and Del Norte Counties. For comparison purposes, the statewide AAR for lung and bronchial cancer from 1993-1997 was 66.5 cases/100,000 population for males and 42.5 cases/100,000 population for females.

Continuing Medical Education - Post Test

Concerns have been raised regarding

a) 2,4-D
b) sulfometuron-methyl
c) atrazine
d) triclopyr
e) glyphosate
f) plant residues
g) residues in water
h) residues in wildlife causing decline of fish and animal populations
i) exposures to children
j) all of the above

Common, or prevalent, health problems seen at Indian Health Service, Tribal Clinics and hospitals include all except):

a) diabetes
b) hepatitis C
c) respiratory conditions
d) nervous system complaints
e) Q fever
f) injuries/poisoning

Exposure and toxicity principals

Of the following, the least important predictor(s) of pesticide toxicity is :

a) use category
b) chemical class
c) manner of use

The most important physical/chemical property for predicting inhalation exposure to pesticide:

a) solubility in organic solvents
b) solubility in water
c) vapor pressure

Insecticides

OP questions

Appproximate range of toxicity, as measured by single dose rodent oral LD50's

a) 3 mg/kg - 1500 mg/kg
b) 25 mg/kg - 550 mg/kg
c) 75 mg/kg - 925 mg/kg
d) 100 mg/kg - 1200 mg/kg

Non-specific symptoms of OP poisoning include all except:

a) nausea
b) headache
c) vomiting
d)diarrhea
e)excessive salivation
f) confusion

g) malaise
h) dyspnea (shortness of breath)

Relatively specific symptoms of OP poisoning include all except:

a) miosis - narrow pupils
b) seizures
e) excessive salivation
g) bradycardia (slow heart rate)
i) excessive bronchial secretions
j) excessive salivary secretions

Specific symptoms of poisoning are most likely to occur following:
a) acute exposure (e.g. ingestion or sudden accidental exposure)
b) subacute
c) chronic

Non-specific symptoms of poisoning **most likely to occur** as the **only** symptoms with:
a) acute exposure (e.g. ingestion or accidental)
b) subacute
c) chronic

Carbamate questions
Appproximate range of toxicity, as measured by single dose rodent oral LD50's
a) 0.5 mg/kg - 308 mg/kg
b) 25 mg/kg - 550 mg/kg
c) 75 mg/kg - 925 mg/kg
d) 100 mg/kg - 1200 mg/kg

Similarities between OP and carbamate toxicity include:
a) carbamates have both acute and subacute effects
b) OPs and carbamates have only acute effects
c) both OPs and carbamates have similar toxidromes
d) regeneration of RBC cholinesterase following OPs poisoning requires no longer than 30 days
e) regeneration of plasma cholinesterase poisoning may require 90-100 days
f) both OPs and carbamates cause delayed neuropathic effects

The degree of ChE inhibition required to cause symptomatic illness is:
a) 30%
b) 50%
c) 75%
d) Symptomatic illness depends upon the rate, as well as the degree, of ChE inhibition

Continuing Medical Education - Post Test

Non-cholinesterase effects of OPs include all of the following except:

a) irritation of the eyes
b) irritation of the upper respiratory tract
c) irritation of lower respiratory tract
d) delayed neuropathy
e) hypersalivation

True statements include all of the following except:

a) The reported environmental half-life for malathion depends upon how tightly it binds to soil
b) The breakdown chlorpyrifos in water is independent of pH
c) chlorpyrifos residues remain on plants for approximately 10-14 days

Pyrethrins

True statements include all of the following except:
a) Acute oral LD50 for pyrethrum in rodents is 1500 mg/kg
b) Chrysanthemum extract formulations (called pyrethrum) may contain allergenic sesquiterpen lactones
c) Rapid breakdown in the mammalian liver occurs by hydrolysis
d) Environmental half-life is 25 days
e) Symptoms of topical irritation or paresthesia are sometimes relieved by Vitamin E

Pyrethroids

Appproximate range of toxicity, as measured by single dose rodent oral LD50's
a) 0.1 mg/kg - 1500 mg/kg
b) 25 mg/kg - 550 mg/kg
c) 52-480mg/kg
d) 75 mg/kg - 925 mg/kg
e) 100 mg/kg - 1200 mg/kg

True statements include
a) Topical paresthesias are a common effect
b) Toxidrome includes seizures and coma following ingestion
c) Rapid breakdown in the mammalian liver occurs by hydrolysis
d) Half-life in water is short compared to half-life in soil
e) Symptoms of topical irritation or paresthesia are sometimes relieved by Vitamin E

Organochlorines

The single dose rodent oral LD50 for DDT is:
a) 0.1 mg/kg
b) 25 mg/kg
c) 113 mg/kg
d) 500 mg/kg
e) 1200 mg/kg

Symptoms of organochlorine overexposure include all of the following except:
a) hyperexcitability
b) tremors
c) seizures after accidental food contamination
d) hemiparesis

e) reports of persistent neurobehavioral effects (balance, reaction time, verbal recall) in a cohort study

The biological half-life of DDT is
a) a few days
b) a few weeks
c) several years

Increased risk of breast cancer associated with DDT (check the incorrect answer)
a) was identified in a case-control study nested within a prospective cohort study
b) exposure determination was based upon prospectively measured levels of DDT and its metabolites in blood
c) was confirmed in a similarly designed study conducted in San Francisco

Fungicides

Thiocarbamate compounds

The single dose LD50 for thiram fed to mice is:
a) 0.1 mg/kg
b) 25 mg/kg
c) 113 mg/kg
d) 500 mg/kg
e) 1200 mg/kg
f) 1500-2000 mg/kg

Reported human health effects of thiram and related compounds include all of the following except:
a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization
c) Antabuse effect (inhibition of intermediate alcohol metabolism)
d) Thyrotoxicosis

The soil half-life of thiram is
a) 2-5 days
b) 15 days
c) 30 days
d) several months to several years

Chlorobenzene compounds

The single dose rodent oral LD50 for chlorothalonil is:
a) 0.1 -10 mg/kg
b) 25 - 200 mg/kg
c) 100 - 500 mg/kg
d) >1,000 mg/kg
e)>10,000 mg/kg
f)>20,000 mg/kg

Human health effects of chlorothalonil include all of the following except:
a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization

c) Antabuse effect (inhibition of intermediate alcohol metabolism)
d) Asthma

The single dose LD50 for chlorothalonil fed to mice is:
a) 0.1 mg/kg
b) 25 mg/kg
c) 113 mg/kg
d) 500 mg/kg
e) 1200 mg/kg
f) 6000 mg/kg

Phthalimido compounds

The single dose LD50 for captan fed to mice is:
a) 0.1 mg/kg
b) 25 mg/kg
c) 113 mg/kg
d) 500 mg/kg
e) 1200 mg/kg
f) 7000 mg/kg

Reported human health effects of captan:
a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization
c) Antabuse effect (inhibition of intermediate alcohol metabolism)

Herbicides

Herbicide mechanisms include all of the following except:
a) inhibition of the synthesis of the aromatic amino acids tyrosine and phenylalanine
b) interference with plant hormones (auxins)
c) inhibition of cholinesterase
d) direct tissue injury
e) interference with electron transpor
f) interference with stem and root tip cell division

Paraquat

The single dose LD50 for paraquat fed to rats is:
a) 0.1 mg/kg
b) 25 mg/kg
c) 110-150 mg/kg
d) 500 mg/kg
e) 1200 mg/kg
f) 7000 mg/kg

Common adverse effects or properties of occupational exposure include all of the except:
a) burns of the skin
b) epistaxis
c) eye irritation or conjunctivitis
d) pulmonary fibrosis

Continuing Medical Education - Post Test

True statement regarding concerns regarding paraquat as a cause of Parkinson's disease include all of the following except:

a) they are based upon structural analogy to 1-methyl-4-phenylpyridinium (MPP+)

b) they are diminished by the limited ability of paraquat to cross the blood brain barrier

c) they are supported to some degree by associations found in case control and descriptive epidemiologic studies

d) they are supported by clinical observation of Parkinson's disease in survivors of acute poisoning

Triclopyr

Appproximate range of toxicity, as measured by the single dose rodent oral LD50

a) 1.0 -15 mg/kg

b) 25 - 200 mg/kg

c) 110 to 150 mg/kg

d) 250 - 500 mg/kg

e) 630 - 729 mg/kg

f) >3000 mg/kg for amine and ester derivatives

Human health effects of triclopyr include (check all that apply):

a) Irritation of the skin, eyes and respiratory tract

b) Skin sensitization

c) Antabuse effect (inhibition of intermediate alcohol metabolism)

d) Asthma

The environmental half-life in soil is

a) 3-5 days

b) 6-10 days

c) 15-25 days

d) 30-90 days

Glyphosate

Appproximate range of toxicity, as measured by the single dose rodent oral LD50

a) 1.0 -15 mg/kg

b) 25 - 200 mg/kg

c) 110 to 150 mg/kg

d) 250 - 500 mg/kg

e) 1000- 1500 mg/kg

f) 5600 mg/kg

Human health effects of **glyphosate** formulations include all of the following except:

a) Irritation of the skin and eyes due to a surfactant compound

b) Skin sensitization associated with a preservative compound

c) cholinesterase inhibition

Sulfometuron

Appproximate range of toxicity, as measured by the single dose rodent oral LD50

a) 1.0 -15 mg/kg

b) 25 - 200 mg/kg

c) 110 to 150 mg/kg

d) 250 - 500 mg/kg
e) 1000- 1500 mg/kg
f) >5000 mg/kg

Human health effects of **sulfometuron** identified from California illness data include (check all that apply):
a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization
c) Antabuse effect (inhibition of intermediate alcohol metabolism)
d) Asthma

Hexazinone

Appproximate range of toxicity, as measured by the single dose rodent oral LD50
a) 1.0 -15 mg/kg
b) 25 - 200 mg/kg
c) 110 to 150 mg/kg
d) 250 - 500 mg/kg
e) 1000- 1500 mg/kg
f) 1690 mg/kg
g) >5000 mg/kg

Human health effects of **hexazinone** include identified from limited California illness data include all of the following except:
a) Irritation of the skin, eyes and respiratory tract
b) Urticaria following exposure to mixture with diuron
c) Antabuse effect (inhibition of intermediate alcohol metabolism)
d) A possibly associated case of asthma

Atrazine

Appproximate range of toxicity, as measured by the single dose rodent oral LD50
a) 1.0 -15 mg/kg
b) 25 - 200 mg/kg
c) 110 to 150 mg/kg
d) 250 - 500 mg/kg
e) 1000- 1500 mg/kg
f) 3090 mg/kg
g) >5000 mg/kg

Animal carcinogenicity studies with atrazine and other triazine compounds show:
a) hepatomas
b) gastric cancer
c) brain cancer
d) lymphomas
e) mammary tumors

Human health effects of **atrazine** include (check all that apply):
a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization
c) Antabuse effect (inhibition of intermediate alcohol metabolism)
d) Asthma

Continuing Medical Education - Post Test

Phenoxy herbicides

The single dose LD50 for 2,4-D fed to rats is:

a) 0.1 mg/kg
b) 25 mg/kg
c) 110-150 mg/kg
d) 500 mg/kg
e) 1200 mg/kg
f) 7000 mg/kg

Human health effects of **2,4-D** include all of the following except:

a) Irritation of the skin, eyes and respiratory tract
b) Skin sensitization
c) Myotonia (muscle weakness)

True statements regarding phenoxy herbicide contamination with dioxin (TCDD) include all of the following except:

a) TCDD derives from the chlorination of phenol to produce dichlorophenol
b) TCDD derives from hydroxylation of tetrachlorobenzene to produce trichlorophenol
c) Contamination with TCDD caused 2,4,5-T to be removed from the market

True statements regarding 2,4-D and cancer include:

a) 2,4-D is a potent experimental carcinogen, producing tumors at multiple sites, mutliple doses, and multiple species.
b) Handling 2,4-D once or twice a year is associated with an increased risk of non-Hodgkin's lymphoma
c) Handling 2,4-D 20 or more times a a year is associated with an increased risk of non-Hodgkin's lymphoma
d) The association with non-Hodgkin's lymphoma is seen in case control studies but not in any of the cohort studies reported
e) Environmental residues have been documented to cause non-Hodgkin's lymphoma

Forest herbicide usage and environmental monitoring

Based upon 1995 data, the most frequently used herbicide was:

a) hexazinone
b) atrazine
c) triclopyr
d) glyphosate
e) 2,4-D

From dissipation studies

a) most applied herbicides dissipate completely within days of application
b) most dissipate complete within 2 weeks of application
c) low level residues are detectable in plant tissues for several months after application

True statements regarding surface water sampling in National Forest lands include all of the following except:

a) limited sampling in May, 1999 in the Terwer Creek and Blue Creek watersheds (more than a week post application of atrazine and triclopyr) did not show any persistent residues
b) sampling one month after application of triclopyr showed levels below 1 ppb in the Hoppaw Creek Tributary

Continuing Medical Education - Post Test

Exposures in production of baskets include all of the following except:
a) monitoring has been conducted using measurements from skin wipes and clothing
b) estimates of exposure are calculated using modeling software
c) residues identified in forest plant tissues are in the range of residue tolerances

North Coast Region 6 and Humboldt/Del Norte cancer data show (indicate the incorrect answer):
a) The data were derived from a case-control study
b) The data are descriptive in nature
c) A consistent excess of smoking related cancers was identified
d) the cause of the excess of lymphomas in early 1990's was not defined

Concepts in epidemiology include all of the following except:
a) Case reports and descriptive studies can be classified as hypothesis-generating study designs
b) Cause and effect relationships can sometimes be inferred from case-control studies and cohort studies
c) Rules for inferring cause and effect depend upon evaluating the strength and specificity of associations
d) Rules for inferring cause and effect depend upon consistent associations from study to study
e) Multiple comparisons in case control studies involve multiple disease outcomes and in cohort studies involve multiple exposures variables

Examples of descriptive epidemiologic methods include all of the following except:
a) pesticide illness surveillance
b) cancer surveillance
c) birth defect surveillance
d) time trends in cancer incidence
e) proposed survey regarding the effects of herbicides
f) case-control studies

Pesticide illness reporting requirements include all of the following except:
a) Report to the local health officer within 24 hours
b) Skin irritation cases
c) Systemic poisoning cases
d) Eye injury cases
e) Home exposure cases
f) Occupational injuries
g) Suicides or suicide attempts
h) Penalties for reporting cases that prove unrelated to pesticide exposure on further investigation

Appendix I - Concepts in Epidemiology

Basic concepts in epidemiology

To evaluate the information available about the effects pesticides on of humans, it is necessary to understand some of the basic concepts of epidemiology. This is similar to necessity of understanding the design of basic animal tests used to test the toxic effects of pesticides. Particular attention will be paid to the quality of various kinds of information and how particular kinds of evidence can be used to infer cause and effect relationships.

Case reports and case-series

Cases reports and case series often give clues to the distribution of cases within a population, and frequently give rise to follow-up studies that can evaluate causality. For example, the initial case reports of soft-tissue-sarcoma (STS) in Swedish workers exposed to phenoxy-acetic acids[98] gave rise to a series of case control studies showing a strong association between STS and exposure to phenoxy-acetic acids.[99]

The care with which both exposures and illness is documented is critical. For example, in some reported cases associating childhood aplastic anemia with exposure to organochlorines, there is minimal documentation of exposure.[100] [101] The reports would be correspondingly more compelling if the cases were demonstrated to have elevated blood levels of chlordane or other organochlorine compounds. Laboratory evidence of exposure adds compelling weight to both case-reports and to follow up studies designed to evaluate cause and effect relationships.[45]

Disease frequency

Disease frequency measures depend both upon ascertaining cases and upon measuring the size of a study population. In large populations accurately ascertaining cases may be difficult and involve substantial trade-offs between sensitivity and specificity. For example, a telephone or mail questionnaire might allow may allow for rapid survey of a population. But a phone questionnaire would have a lower specificity than review of cases ascertained by physical examination, or review of medical records.

In addition to case ascertainment, defining disease frequency depends upon identifying the number of individuals exposed to the agent of interest. This may not be possible to do with any accuracy. Many descriptive studies used proxy measures such as census or employment data. Although descriptive information does not usually allow researchers to make causal inferences, it is often invaluable in developing new research hypotheses that can be tested in analytical studies.

Incidence and prevalence

Incidence is defined as the number of new cases of illness or injury in a study population within a specified time period - which day, month, year, decade , et cetera. Prevalence, by contrast, is the number of cases present in the population at a specified point in time - e.g., the time of an examination survey. The two measures of disease frequency are related by the following simple equation:

equation 1 **Prevalence = Incidence x Duration**

Both prevalence and incidence can be addressed in a single study. For example, in a survey of workers employed by a particular employer, all contact dermatitis cases identified by physical examination in the survey participants define the prevalence of that condition in the study group. In the hypothetical study of 100 workers, 4 were found to have contact dermatitis at the time of the exam (Prevalence= 4/100). A questionnaire administered on the day of the examination is also used to evaluate the occurrence of new rashes on the forearms since the beginning of the current

agricultural season (5 months). This determines that 30% of the workers report having a new rash, of an unspecified type, since the onset of the current agricultural season. The estimated duration of the rash from

equation 1 = **Duration** = incidence/prevalence = (4/100)*(100*5 months/30)= 2/3 month = **20 days**.

The above analysis assumes that all of the cases of contact dermatitis found on examination are due to the exposure of interest. This may not be the case, as the hypothetical study described does not include a comparison group and examination surveys of the general U.S. population have shown a mean contact dermatitis prevalence of approximately 4%. A further premise is that all of the rashes reported on the questionnaire are due to the same hypothetical exposure, an assumption even less likely to be true.

Estimating population incidence from surveillance

Population incidence may also be estimated from illness registry data, ultimately derived from cases seeking medical treatment. The completeness of reporting depends upon a) the number of ill persons who seek treatment, b) the number of persons seeking treatment who are properly diagnosed, and c) the percentage of those who are diagnosed who are actually reported to the registry. These problems are illustrated by a survey conducted by Discher who evaluated reporting from multiple sources on the same study population:

Evaluation and classification of surveillance information

Procedures for evaluating pesticide illnesses necessarily differ from those used in evaluating cancer. While the latter depend upon tissue samples and their pathologic interpretations, evaluation of pesticide illnesses depends upon an interpretation of information on the degree of reported exposure and the coherence of the reported symptoms with those expected from the recognized toxidrome (or symptom pattern) of individual pesticide compounds. Rather than a tissue diagnosis, the registry records a degree of probability (possible, probable or definite) in the relationship between the exposure and the reported signs and symptoms.

Evaluation of Cause and Effect

As in other types of science, the most desirable type of information derives from experimental studies of human subjects. In medicine, the model for this type of study is the randomized clinical trial. It is not usually possible to do this type of study with pesticides.

Approximation of the experimental method is sometimes possible by means of well conducted epidemiologic research. This may be the case, for example, where accurate means of assessing both the exposure variables and the disease outcome are readily available and both the disease and the exposures of interest have a significant degree of variability in the study population. Because such ideal study conditions do not occur very frequently by chance, researchers, in some fields of medicine, endeavor to create them using clinical trials and other experimental studies.

Although experimental studies often have ideal conditions for evaluating the relationship between exposure and outcome, they do have predictable limitations. For example, a common question in interpreting data from experimental studies is how well the study group represents the population group of interest. Use of paid volunteers, for example, may make various systematic experimental studies possible, but still leave a question about whether the results can readily be generalized to the general population.

Whether the data derives from experimental or observational studies, the rules for identifying causal associations in are agreed upon by all epidemiologists, even though their application in practice is the frequently the subject of

great controversy. In brief, a causal relationship between an exposure factor and the subsequent occurrence of disease may be inferred to the degree that:

1) The association is consistent from study to study;

2) The association is statistically strong;
3) There is a dose-response relationship;

4) The association is biologically plausible - in terms of pesticide related skin disease, this means, is the reported association consistent with existing human literature and animal literature.

5) The association is highly specific - e.g. as with vinyl chloride and angiosarcoma of the liver, or asbestos and pleural mesotheliomas.

Exposure evaluation

The limiting problem with most epidemiologic studies is an inability to accurately assess the level of exposure. The reliability of the exposure information in epidemiologic studies may vary from extremely reliable information generated from biologic markers measured systematically in a study population to possibly inaccurate information generated from questionnaires. The greatest difficulty arises from questionnaires attempting to identify the nature and extent of exposures that occurred many years previously. The use of-such questionnaires has been a principal weakness in many case control-studies. Bias from selective recall of ill individuals or in relatives of deceased patients may be extremely difficult to control.

Control selection

Careful selection of control populations is a principal means of minimizing bias. In a randomized clinical trial, this is achieved by randomly assigning subjects from a uniform group to treatment or non-treatment status. In a cohort study the comparison group may simply be the population at-large, if data exists for the distribution of the outcome of interest within the overall population. The population-at-large is commonly the comparison group for the cohort mortality study. For disease outbreak investigations, the comparison group is usually an at-Unfortunately, there is objective means of measuring the extent of bias, except by comparing the difference in odds ratios obtained using different control populations. For example, in a study of childhood brain cancer and chemical exposures, controls were selected from the general population by random digit dialing and also from a list of children with non-brain cancers. Even when this is done there is no a priori means of knowing which control population should be considered the "gold standard" in terms of recall bias. When controls are selected from among patients with another type of illness, the studies may be prone to another type of negative statistical bias, if there is an unrecognized association between the exposure of interest and the "control" illness.

Multiple comparisons

A final methodologic consideration is the problem of multiple comparisons. In case control studies, it is possible to record on a questionnaire many exposures and evaluate their statistical association with the illness in question. In this situation it is possible that some exposures will be statistically associated with the occurrence of illness purely by chance. It is therefore important in evaluating case control studies to consider which exposures have truly been identified a priori as concerns. For cohort studies, the problem of multiple comparisons arises not from the possibility of evaluating the difference in incidence between the exposed and control populations for multiple illness outcomes.

Proposed questionnaire

Proposed questionnaire

YUROK ENVIRONMENTAL MONITORING WORK GROUP

HERBICIDES SURVEY FORM
September 1999

The purpose of this survey is to document general information about illnesses, deaths, cancer, birth defects, or other effects that you feel may be related to the application of herbicides on the Yurok Reservation. Please return your comments to either the Yurok Tribal Environmental Program in Eureka or Bertha Peters at the Weitchpec Community Center.

Do you know of anyone that has been affected (health-wise) by the spraying of herbicides on the Reservation?

If so, who was it?

Where does the person live (general location)?

Where (general location) was the person exposed to the herbicide?

How was the person exposed to the herbicide?

How many times and over what time period was the person exposed to the herbicide?

What type of herbicide was it?

How did the herbicide affect the person?

Did the person get sick?

What type of sickness did the person get?

Did the person see a doctor about it?

What type of treatment did the doctor give the person?

How long did the person see a doctor about his/her illness?

Did the person develop cancer?

When did the person get sick?

How long was the person sick?

Did the person die from his/her illness?

How long after the exposure did the person die from his/her illness?

Were there birth defects related to the illness?

Proposed questionnaire

What other sickness was there related to the illness?

Pesticide illness report (PIR) form and physician reporting requirements

State of California
Environmental Protection Agency (Cal/EPA)

Office of Environmental
Health Hazard Assessment

PESTICIDE ILLNESS REPORT

(For illnesses caused by pesticides--including sanitizers and disinfectants)

PATIENT:

Name: Age: Sex: [ ] 1 M [ ] 2 F

Address: City: County:

Phone No.:( ) Social Security Number

Occupation: Language [ ] 1 English [ ] 2 Spanish [ ] 3 Other

PHYSICIAN FILING REPORT:

Physician's name:

Physician's address:

INJURY:

At Address: City County:

Was injury: [ ] l At Home [ ] 2 At Work--agriculture [ ] 3 At Work--nonagriculture [ ] 4 Other exposure

If at work: a) Employer's name:

Employer's address:

b) Manager or Supervisor:

Date of exposure: / / Time of exposure: [:] a.m. [: J p.m.

Date of illness: / / Date of death: / /

Is there reason to believe others were exposed? [ ] 1 No [ ] 2 Yes

PATIENT'S DESCRIPTION OF EXPOSURE:

Activity at time of exposure:

[ ] 1 Applying pesticides [ ] 2 Manufacturing pesticides [ ] 3 Mixing pesticides [ ] 4 Entering pesticide areas

[ ] 5 Disposing of pesticides or their containers [ ] 6 Eating contaminated food

Other exposure (explain):

Name of pesticide(s): Ingredient(s) of pesticide(s):

Primary route of exposure: [ ] 1 Oral [ ] 2 Dermal [ ] 3 Eye [ ] 4 Inhalation [ ] 5 Unknown

PHYSICIAN'S DESCRIPTION OF EXPOSURE:

Date first seen / / Time first seen:

Major signs, symptoms, adverse reactions:

Hospitalized? [ ] 1 No [ ] 2 Yes If Yes, hospital name: City:

Emergency room only? [ ] 1 No [ ] 2 Yes

Physician's office only? [ ] l No [ ] 2 Yes

Diagnostic studies ordered? [ ] l No [ ] 2 Yes If Yes, which studies?

Diagnosis:

Treatment:

Brief description of incident (if female, indicate if pregnant):

AGENCY COMPLETING FORM:

Agency/County: By whom:

Address:

Phone no.:

Form OEH-PETS 004 (Rev. 5/99)(PIR_R99.doc)

Pesticide illness report (PIR) form and physician reporting requirements

AUTHORITY
Part 1. Physician Responsibility

The Health and Safety Code (Section 105200) requires that a physician who knows, or has reasonable cause to believe, that a patient has a pesticide-related illness must report that case to the local health officer by telephone within 24 hours. The reporting requirement includes all types of pesticide cases: skin and eye injuries, systemic poisonings, suicides, homicides, home cases, and occupational cases. **Failure to comply with the foregoing reporting requirement renders the physician liable for a civil penalty of $250.00.**

A case seen as a pesticide poisoning, or suspected as a pesticide poisoning, may not be categorized as "first-aid" and must be reported (Health and Safety Code, Section 105200).

For occupational cases, there is the additional requirement to send a copy of the "Doctor's First Report of Occupational Injury or Illness" (DFR) to the local health officer within seven days and also to send the DFR to the State Department of Industrial Relations.

Part 2. Responsibility of the Local Health Department Regarding Pesticide Illness Reporting

Each local health officer shall immediately notify the county agricultural commissioner and shall report to the Director, Department of Pesticide Regulation, the Director, Office of Environmental Health Hazard Assessment, and, for occupational cases, the Director, Department of Industrial Relations, on a form prescribed by the Director, Office of Environmental Health Hazard Assessment, each case reported to him or her pursuant to this section within seven days after receipt of any such report (Health and Safety Code, Section 105200).

Addresses and phone numbers

Department of Pesticide Regulation,
Worker Health and Safety Branch,
830 K Street
Sacramento, CA 95814-3510
(916) 445-4222 (voice); (916) 445-4280 FAX

Department of Industrial Relations,
Division of Labor Statistics and Research
P.O.Box 420603
San Francisco, CA 94142-0603
(415) 703-3020 (Voice); (415) 703-3029 (FAX)

Office of Environmental Health Hazard Assessment
Pesticide and Environmental Toxicology Section (PETS)
1515 Clay Street, 16th Floor
Oakland, CA 94612
(510) 622-3170 (Voice); (510) 622-3218 (FAX)

Part 3.Definitions (abridged) of "economic poison" (pesticide) and "pest:"

Food and Agriculture Code, Section 12753 defines an "economic poison" (pesticide) as: "Any substance, or mixture of substances which is intended to be used for defoliating plants, regulating plant growth, or for preventing, destroying, or mitigating any pest, as defined in Section 12754.5, Which may infest or be detrimental to vegetation, man, animal, or households, or be present in any agricultural or nonagricultural environment whatsoever."

Food and Agriculture Code, Section 12754.5 defines "pest" as: "Any of the following that is, or is liable to become, dangerous or detrimental to the agricultural or nonagricultural environment of the state:
(a) Any insect, predatory animal, rodent, nematode, or weed;
(b) Any form of terrestrial, aquatic, or aerial plant or animal, virus, fungus, bacteria, or other microorganism (except

viruses, fungi, bacteria, or other microorganism on or in living man or other living animals);
(c) Anything that the director, by regulation, declares to be a pest."

AVAILABILITY OF THIS FORM
Additional copies of this form are available from the Office of Environmental Health Hazard Assessment, PETS, 1515 Clay Street, 16th Floor, Oakland, CA 94612. Telephone (510) 622-3170 (Voice) or (510) 622-3218 (FAX).

Superfund sites neighboring California National Forest or Tribal Lands

Members of Tribal Staff working in Hoopa Valley raised issues regarding potential exposures to residues of heavy metals, documented as present in soil samples taken by U.S. EPA, including lead and arsenic. Samples for most volatile organics were negative. Cleanup involved removing contaminated soil from the site. A review of publicly available information from the U.S. EPA identified several other waste sites investigated and closed by U.S. EPA in the Hoopa Valley.

Archived Sites - investigated and closed by US EPA

Hoopa Valley		
	California Pacific Lumber Mill	12/21/89
	Chromite Float Deposit	3/27/1990
	Football Field Area Dump Site	6/1/1988
	Hailstone Allotment Campbell Fld Hoopa	6/1/1986
	Hoopa Shopping Ctr Area Mill Site	6/1/1988
	Old Airport Mill (Abandoned)	7/1/1987
	PG&E Hoopa Transformer Substation	4/1/1986
	Rhd Veneer	10/1/1986
	Risling Lumber Mill (Abandoned)	5/1/1988
	Running Silver Mine (And Others)	7/1/1987
	Supply Creek Ldfl	7/1/1987
Clearlake		
	Eastlake Landfill	12/13/89
Pala Indian Reservation		
	Pala Indian Reservation	

Source: **http://www.epa.gov/superfund/sites/arcsites/caacity.htm**

Additional site information available from The Comprehensive Environmental Response, Compensation, and Liability Information System (CERCLIS), sponsored by the EPA's Office of Emergency and Remedial Response, Information Management Center. The database contains information on site inspections, preliminary assessments, and remediation activities at hazardous waste sites.

Contamination of Museum Artifacts with Arsenic or Mercury Preservatives

Because of potential return of museum artifacts (including woven baskets) to tribes under the under the 1990 Native American Graves Protection and Repatriation Act, museum practices preserving artifacts deserve attention. Historically, materials used include salts of mercury and arsenic and organic materials. Other materials applied include organochlorine and organophosphate insecticides. In more recent years, museums have used fumigant gases, less likely to leave a residue on artifacts. The list below, based on materials published by a curator from the Smithsonian (Catharine Hawks, Historical Survey of the Sources of Contamination of Ethnographic Materials in Museum Collections. **Collections Forum** 2001; 16(1-2):2-11), describes both current and past materials used:

Materials

Alcohol
Aldrin
Bendiocarb (Ficam)
Benzene hexachlorides (e.g., Lindane)
Borax
Boric acid
Carbaryl
Carbolic acid (phenol)
Carbon disulfide
Carbon tetrachloride/ethylene dichloride
Chlordane
Chlorpicrin
Chlorpyrifos (Dursban)
Diatomaceous earth
Diazinon
Dichlorodiphenyltrichoroethane (DDT)
Dichlorvos (Vapona)
Dieldrin
Edolan U
Endrin aldehyde
Endosulfan II
Ethylene dibromide
Ethylene oxide
Formaldehyde
Heptachlor
Hydrogen cyanide gas
Hydrogen phosphide
Lauryl pentachlorophenate
Malathion
Methyl bromide
Methyoxychlor
Naphthalene
Orthodichlorobenzene
Paradichlorobenzene
Pentachlorophenol
Propoxur (Baygon)
Pyrethrins (synthetic)
Silica gel
Sodium aluminum fluorosilicate
Sodium fluorosilicate

Contamination of Museum Artifacts with Arsenic or Mercury Preservatives

Sulfuryl fluoride
1,2,4-trichlorobenzene