NATIONAL ABORIGINAL AND ISLANDER HEALTH ORGANISATION

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HEALTH REPORT: N.S.W. COTTON

HEALTH PROBLEMS OF ABORIGINAL COTTON WORKERS IN NORTH-WEST NEW SOUTH WALES

REPORT OF NAIHO TEAM VISITING
THE NEW SOUTH WALES COTTON BELT

MAY 19, 1984

THIS REPORT IS BEING DISTRIBUTED TO ABORIGINAL PEOPLE AND OTHER CONCERNED PERSONS FOR COMMENTS

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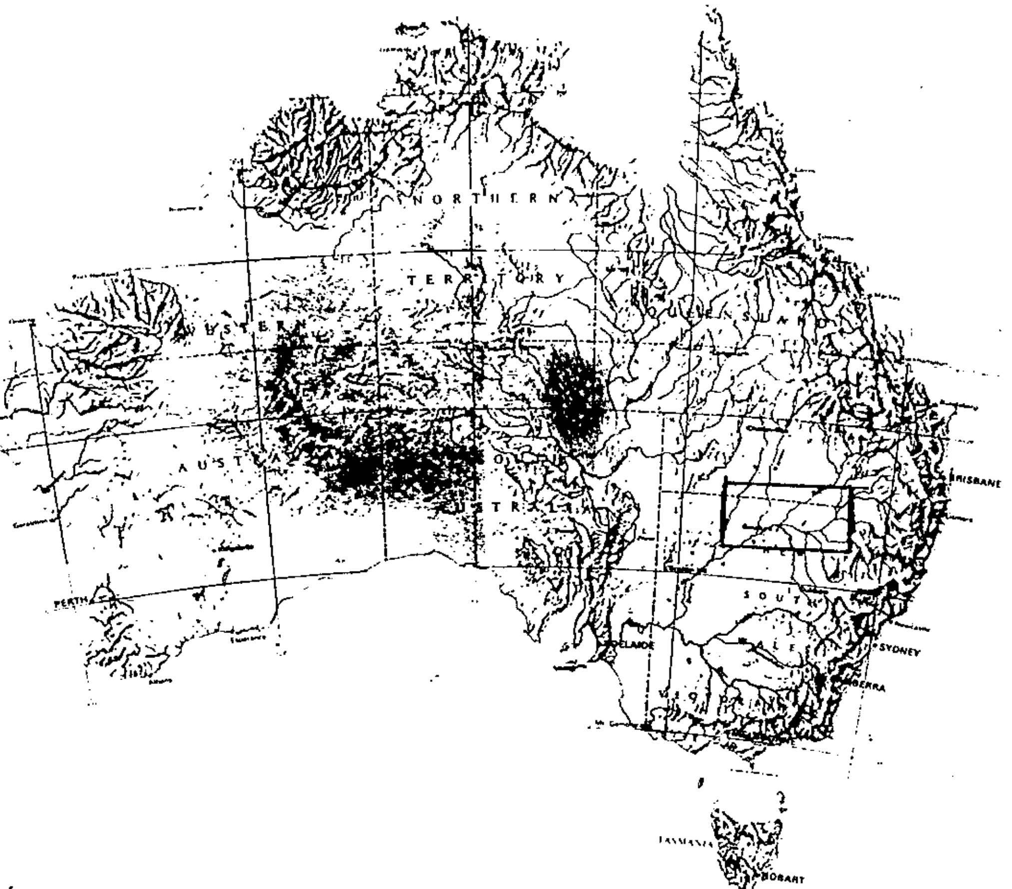
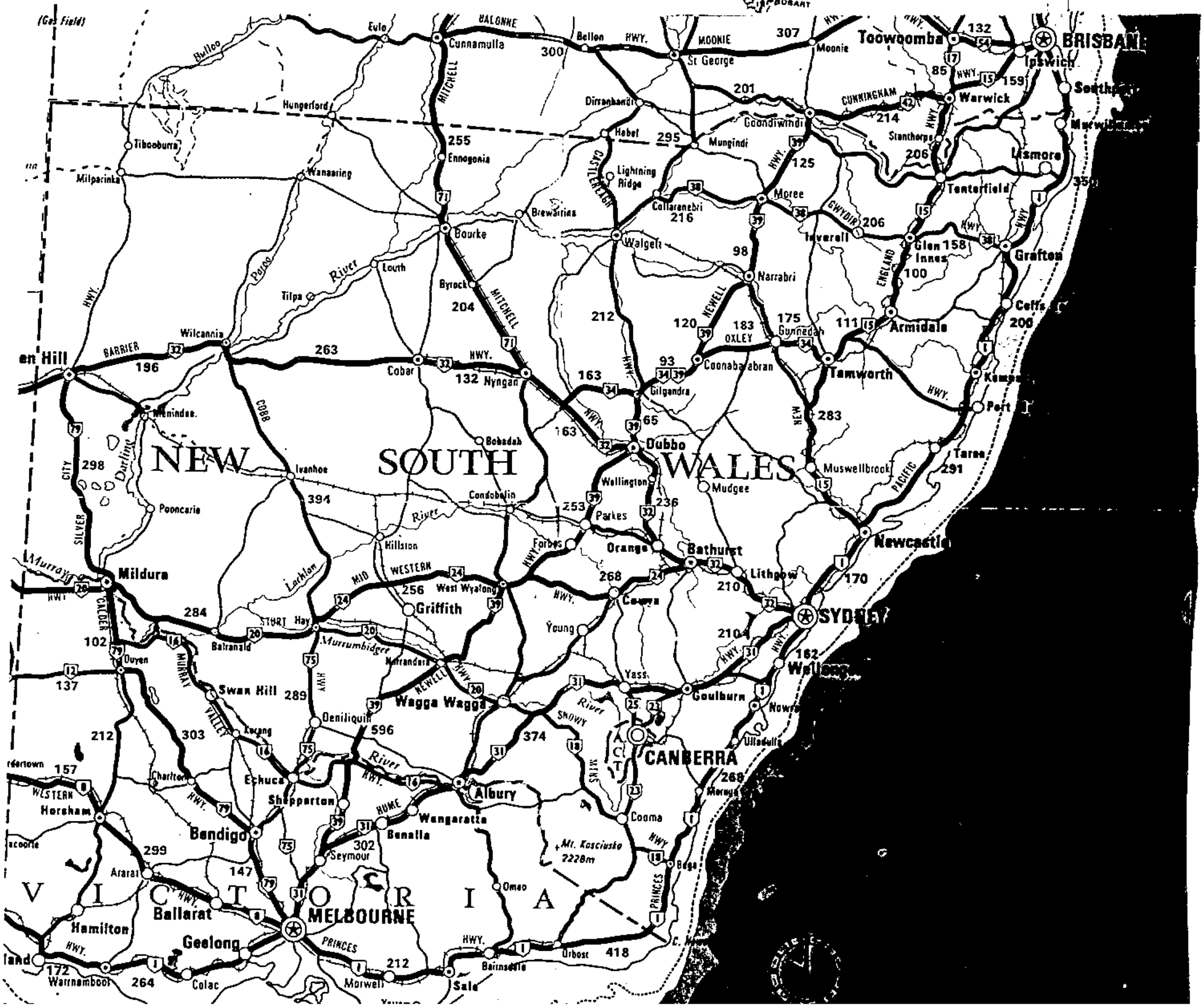




INTRODUCTION

In February 1984 there was a regional meeting of the National Aboriginal and Islander Health Organization (NAIHO) in Port Macquarie, New South Wales. Aboriginal representatives from Moree district of north-west N.S.W. requested NAIHO to help them investigate skin, breathing and other ailments which they felt were excessively frequent amongst cotton-workers. The people were afraid that these diseases were due to pesticides used on cotton crops.

A NAIHO team visited the area from May 2 - 13, 1984. The team included the NAIHO co-ordinators for Areas 16 and 17 (Tom Kellner of Taree and Noel Gillon of Bourke), Bob Davis of Taree and Dr Adrian Sleigh.

The team assembled in Moree with the more fortunate members driving across from Taree and Noel Gillon hitch-hiking from Bourke Funds were limited but the enquiry received generous assistance from local aboriginal people, the NAC office, the Aboriginal Legal Service and many others.

The team travelled throughout the cotton-belt from the Queensland border in the North to Bourke in the far West. At least 5000 km were covered and 1000 aboriginal people contacted. An illness/work-experience survey was performed and many professional people, including Government representatives, agronomists, farmers, union people and othere were contacted.

This report includes an account of pertinent aspects of the cotton industry, the spray chemicals, the plants, the aboriginal cotton-workers and the epidemiology of their complaints.

CONCLUSIONS AND RECOMMENDATIONS

General comments on pesticide poisoning

Allegations of pesticide poisoning must always be taken seriously. Sceptics should examine the following table, reproduced from a recently published book (Moses, M. Pesticides. Chapter 47 p. 563. In: Rom, William (Editor) Environmental and Occupational Medicine - Little-Brown - 1983.)

Epidemics of Poisoning by Pesticides

Pesticide	Source of Contamination	Number of Cases	Number of Deaths	Location	Year
Toxaphene	Collards	4	0	U.S.	1952
	Chard	3	0	U.S.	1952
Warfarin	Bait	14	2	Korea	1954
Endrin	Flour	159	0	Wales	1956
Parathion	Wheat	360	102	India	1958
	Flour	200	8	Egypt	1958
	Crops	79	0	U.S.	1958
Trithion	Crops	19	0	Trinidad	1960
Hexachlorobenzene	Seed grain	>3000	10%	Turkey	1960
Mercury	Seed grain	321	35	Iraq	1961
Parathion	Lice Rx	>17	15	Iran	1963
Mevinphos	Plants	6	0	U.S.	1963
Mercury	Seed grain	34	4	Pakistan	1963
Parathion	Crops	94	0	U.S.	1964
Metaisosystox	Crops	673	3	Egypt	1964
Nicotine	Mustard	11	0	U.S.	1965
Diazinon	Doughnut mix	20	0	U.S.	1965
Mercury	Seed grain	45	20	Guatemala	1966
Endrin	Flour	3	0	Egypt	1967
	Flour	691	24	Qatar	1967
	Flour	183	2	S. Arabia	1967
Parathion	Flour	600	88	Colombia	1967
	Sugar	300	17	Mexico	1969
Pentachlorophenol	Nursery linen	20	2	U.S.	1969
Parathion	Crops	23	0	U.S.	1969
Carbophenothion	Flour	7	0	U.S.	1969
Organophosphates (various)	Crops	55	0	U.S.	1970
Malathion	Malaria control	2800	5	Pakistan	1975
Monitor, Gusathion	Crops	3000	30	Egypt	1976
Dialiflor	Crops	85	0	U.S.	1976
Methomyl	Crops	4000	30	Egypt	1977
Parathion	Flour	79	17	Jamaica	1977
Aldicarb*	Cucumbers	14	0	U.S.	1979

* Suspected.

In her chapter on Pesticides Dr Moses points out that pesticide illness in field harvesters is greatly under-reported and that data on the long term effects of acute poisoning are very limited. The World Health Organization estimated that in 1973 there were 500,000 cases of pesticide poisonings world wide with a mortality rate of 1%. The N.S.W. Health Commission states in its publication POISONING BY PESTICIDES that occupational pesticide poisoning has resulted in hospitalization of 40-60 persons annually in New South Wales.

Specific findings: north-west New South Wales

(i) Relationship of aboriginal people to the cotton industry in N.S.W.

Cotton is a growing source of seasonal employment for aboriginal people in N.W. New South Wales. This is important for aboriginals are the most impoverished group within rural Australia. Cotton is unusual in that it is a high-tech chemically intensive field crop that is also a high human-contact crop because of the need for manual weeding.

(ii) Cotton- related diseases amongst aboriginal cotton workers.

Problems with rashes, blisters, vision, giddiness and asthma were all recalled with greater frequency by manual weeders (cotton chippers) than by those who had not done this type of work. Rash was the most frequent complaint: 56% (71/126) of those with cotton chipping experience remembered problems with rashes compared to 26% (9/34) of non-chippers. This difference in the rate of remembered rashes for chippers and non-chippers was statistically significant ($x^2 = 8.4$)

and the probability of such a finding by chance was less than 1 in a 100 ($p < 0.01$).

There are two plausible explanations for rashes associated with cotton chipping:

(a) exposure to toxic pesticide residues because of inappropriately rapid re-entry of workers into sprayed fields;

(b) exposure to toxic properties of plants which are being handled or brushed against.

The higher rate with which chippers experienced giddiness, visual troubles and asthma are suggestive of organophosphate/carbamate pesticide toxicity. This cannot be excluded as chippers have not been included in occupational safety programmes of blood testing for cholinesterase depression.

Chippers also frequently reported having been sprayed by a plane (30%) and having entered wet fields (72%). The spraying incidents apparently result either from poor co-ordination of spraying and chipping or from drift. The entry into wet fields in the early morning either results from a dew or from entry before sprays are dry. It could be quite hazardous to enter wet fields and is certainly hazardous to be sprayed.

Boils are epidemic in the Moree aboriginal community: 57% (72/126) of the chippers and 18% (6/34) of the non-chippers reported having had trouble with boils this year. The difference between the rates for chippers and non-chippers

is highly significant ($x^2 = 15.1$, $p < 0.001$). Presumably the boils result from skin trauma while cotton chipping.

No investigation was done on problems with gin workers. However, the noise and dust in some cotton gins warrants further investigation. Asthma and the cotton - specific disease of Byssinosis are known to affect cotton gin workers when dust is not controlled.

Accidental trauma associated with the machines used in the cotton industry seems to be excessively frequent and warrants further investigation.

(iii) Environmental contamination

There have been incidents of contamination of creeks and dams with pesticides that were associated with deaths of stock and wildlife. However we could not find any pattern of illness associated with river swimming.

Further work needed.

A. field studies of cotton workers.

More definitive data are required during a chipping season. Assuming co-operation from workers, chipping contractors and farmers it will then be possible to objectively classify and determine the incidence of rashes and blisters; it should also be possible to assess their relationship with

(i) chemicals used on the crop (chemical, formulation, diluent, spray technique, re-entry period);

(ii) evidence of pesticide absorption (blood and urine tests);

(iii) evidence of pesticide exposure (residue studies on clothes and crops, with and without a dew);

(iv) type of exposure (none, weeders, irrigation workers);

(v) type of weed and weeding technique (hoe only, no touching versus touching of the weeds).

With such a formidable list of variables it may only be possible to confirm that rashes and blisters are excessively frequent and that chippers are - or are not - contaminated with pesticides.

B. Consultation with botanists and toxicologists and other scientists.

C. Reference to the scientific literature.

Reforms needed

1. Those responsible for monitoring the use and impact of pesticides must communicate their findings to the public. Withholding information is not going to diminish fears regarding pesticides. Why not distribute the data through local government, "talk-back" chats on the radio and other means?

2. Occupational safety programs monitoring pesticide exposure need to be extended to cotton chippers. Neither the Department of Industrial Relations nor the chemical companies monitoring urinary chlordimeform are reaching the chippers.

3. All future incidents of spraying of field workers should be vigorously investigated. Aboriginals sprayed should sue for damages through the Aboriginal Legal Service.

4. Every effort should be made to unionize cotton chippers. First aid - particularly for abrasions, penetrating injuries, boils and organophosphate/carbamate poisoning - should be available at the cotton fields. The Union award should include first aid provisiona and detailed safety clauses; farmers and chipping contractors should be strongly encouraged to employ only unionized labour protected by workers' compensation insurances. (See Appendix 1 - a Union award in California).

5. All public health work with the aboriginal cotton workers will be enhanced by the development of Aboriginal Medical Services. Such a service was supported by 94% of 160 aboriginal people surveryed around Moree. An AMS would be the most effective means of reaching and involving the aboriginal people in solving their health problems.

6. The time that must elapse before cotton-workers re-enter a sprayed field needs re-evaluation. For most chemicals sprayed on cotton the "re-entry period" in N.S.W. is either not mentioned on the label or is stated to be "when dry". The rule of thumb used by many chippers and farmers is a re-entry period of one day for the few "bad" chemicals and "when dry" for the rest. This empirical approach could be quite hazardous for cotton chippers. Objective local data are needed and, in their absence, far greater caution than that exercised at present.

THE COTTON INDUSTRY

Perspective on Australian Production

Cotton is the most important natural fibre used in the world today. The cotton fibres surround the seed in the fruit of plants of the botanical genus Gossypium which have descended from plants used by the ancient civilizations of India, Egypt, Mexico and Peru. Once the seeds have been removed in a cotton gin, the fibre is referred to as cotton lint or raw cotton.

The cotton plant originally required a tropical environment. Today it is grown all over the world because the availability of irrigation water now permits cotton production in huge areas of low rainfall. Australian production has grown enormously over the last five years: 161.3 kilotonnes are expected from the 1984 harvest, compared to 53.0 kilotonnes in 1979. This increase in production is a result of the three-fold increase in the area planted. Some 135,500 hectares are being harvested in 1984 of which 76% is in New SouthWales and the balance in Queensland. Cotton production in north-west Australia ceased in 1975 due to overwhelming pest problems.

The cotton belt in N.S.W. is in the north-west of the state and extends south-west from Goondiwindi on the Queensland border, through the districts of Moree, Narrabri, Wee Waa and Walgett to Bourke on the Darling River. Thus the N.S.W. industry is based on the deep clay flood-plains of the tributaries of the Darling. The increase in production in this area of N.S.W. accounts for most of the growth in the industry nationally; factors include the rapid increase in the availability of irrigation water since the

Copeton Dam (near Inverell) was completed in 1976, the willingness of bankers to provide the very substantial finances necessary to plant and sustain an irrigated cotton crop, the excellent scientific support provided through the Cotton Research Station at Myall Vale near Narrabri and the realization by farmers that in these circumstances, cotton is an extremely profitable crop.

In the future, cotton will grow in importance nationally as a significant export. Most (87%) of this years crop is expected to be exported and earn AUS$262 millions in foreign exchange. The high quality of Australian cotton, the advantage of off-season production for exports to the Northern hemisphere and the proximity of the markets of Japan, Hong Kong, Taiwan and Korea should ensure that export performance is maintained.

Method used to produce Australian cotton

Cotton is grown in Australia by the high-technology methods pioneered in the U.S.A. Huge machines are used to clear, level and plough very large fields. Planting, fertilizing and harvesting is completely mechanized. In the irrigated areas, water is fed alongside the fields in deep wide ditches and then pumped or siphoned down each furrow between the rows of cotton plants to be collected as "tail-water" in run-off ditches and recycled. Chemical fertilizer is either injected into the soil, added to irrigation water or sprayed; weeds and pests are controlled by aerial and ground-spraying of herbicides and pesticides. Before cotton is harvested, chemical defoliants are aerially sprayed onto the crop in order to strip all leaves from the plants. After harvesting, the cotton is rolled onto semi-trailers and transported to the nearest gin where it is cleaned, dried, de-seeded and baled for sale. Production by this method in Australia is expensive but

leads to a very high yield. This years harvest will represent 1.04% of the world production but will be produced from a mere .41% of the totaly area harvested. Thus the yield per hectare in Australia is over twice the world average and is second only to that of Israel. The very high yield is achieved because of state-of-the-art soil and crop management, irrigation and very heavy use of chemicals.

The extensive mechanization of any crop would normally diminish direct human contact with the plant and decrease the number of agricultural workers exposed to hazards from the residue of any chemicals used. In this respect, cotton is very unusual. The weeds, Xanthium pungens (Noogoora burr), Xanthium spinosum (Bathurst burr) and Datura ferox (False Castor Oil), are very troublesome in the cotton growing areas. They must be removed by hand or by hoe, so creating the seasonal work known locally as "cotton chipping". This costs the farmer from $17 to $247 per hectare each year. The average cost is $74 per hectare; each cotton chipper is paid about $6.50/hr and works an eight hour day. Thus it can be calculated that the 135,500 hectares of cotton planted in 1983-84 in Australia, required 192,827 man-days of manual weeding. Cotton is planted in October and defoliated and harvested in April-May. Cotton chipping is required in December, January and February and most chippers work from a few days to six weeks or so. If the average number of days worked is thirty per chipper, some 6,500 people would be employed each year. Cotton is frequently sprayed with pesticides and occasionally with selective herbicides throughout the cotton chipping season. In fact cotton is one of the most heavily sprayed of all crops. It has been estimated that 47% of all pesticides used in agriculture in the U.S.A. are applied to cotton. Thus the high-tech, high-chemical crop of cotton is also a high-contact crop for a large number of

seasonal field workers in Australia and most of this contact occurs in north-west New South Wales. Therefore, any fears raised by cotton chippers concerning health hazards of their work merit serious evaluation.

In some areas of the world, herbicides have eradicated cotton chippers. This is not likely to occur in Australia for there are no herbicides selective enough to kill Noogoora burr, Bathurst burr and False Castor Oil without also killing the cotton plant. Thus cotton chipping is likely to be an important feature of rural Australia for a long time to come.

THE FINISHED CROP

THE ENEMY
(Heliothis larva)

THE COTTON WORKFORCE

Farmers

Cotton is not the major crop of the cotton belt. Far more land is devoted to grazing and wheat production than to cotton. Other crops include sorghum, barley, oats, peanuts and even pecan nuts. Many farmers include cotton as one of their activities but for certain large properties, cotton is the only crop. American expertise is contributed through some large U.S. owned properties and also via a few small farmers who stayed on after helping to introduce cotton into New South Wales in the early 1960's. Earlier attempts to introduce cotton around 1900 (at Moree) and also in the early 1920's were not sustained. However the crop has steadily gained acceptance and understanding by farmers after the N.S.W. Agricultural Department - C.S.I.R.O. Research Station, grew its first crop near Narrabri in 1958. Since the Copeton Dam was completed in 1976, the farmers around Moree have taken a rapidly increasing interest and this district is now a major producer.

Farm hands and irrigation workers

All cotton farms have a small permanent work-force to maintain and operate equipment and organize the irrigation. On some properties, workers spend many hours in the irrigation ditches priming or blocking off siphons that transfer water to the fields; other farms move the water with pumps that can be switched on or off without any need to get wet. Any workers who enter the ditches would be exposed to chemicals that have contaminated the irrigation dams via recycled run-off water. Chemicals could also contaminate a ditch during aerial spray operations of the crop or when ditches are directly sprayed with herbicides to control weeds.

Agricultural scientists

Cotton farming in Australia requires substantial scientific expertise and is the subject of continuing research. Spray companies, chemical and equipment manufactures, seed merchants, farmers and the Government all require the services of agricultural scientists. Some of these scientists frequently enter cotton crops to check on plants, soil and insects.

Aerial spray companies

There are many aerial spray companies involved with cotton. Some are efficiently organized and well financed - others less so. All spray companies must employ pilots, workers to mix and load the chemicals and others to mark the margin of each completed swathe as the pilot turns the plane for a return spray run. These are the "markers" or "flaggers". Some operate out of sealed cars in radio communication with the plane; others use flags and mark on foot. All the personel employed by a spray company are potentially exposed to lethal doses of spray chemicals. Companies must pass a strict test on safety procedures before being licensed to operate. Employees must use protective clothing and even masks. Chemicals must be stored in a leak proof and locked shelter and showers must be available at the storage and loading site. These guide lines can protect pilots, mixers and loaders well but markers are at considerable risk of being sprayed (directly or by drift) or even of being hit by planes which fly lower than the usual 10-15 feet above the crop. Companies who base their operations at urban airports are far easier to monitor for safeguards than those based on bush airstrips.

Cotton chippers

These workers are usually Aboriginals and are better employed directly by the farmer or through contractors who hire the chippers and organize the weeding from December to February each season. Up to 15 such contractors operate in Moree district annually. Some four or five of the contractors are stable and well organized but the rest are as transient as their workers. One large contractor in Moree has employed up to 500 people at one time. He often works several fields at once and organizes the work by radio. Each chipper usually moves along a row of cotton plants removing weeds by hand or by hoe. For every ten chippers, there is one "runner" who follows up on any missed weeds and reports poor performance to the contractor. Chippers are "hourly" employees and usually begin work at dawn and finish their eight hour day around 1-2 in the afternoon. Most contractors issue long handled hoes although a few use the cheaper, less robust and less healthy (for backs) short handled varieties. Hoes are issued each morning and returned that afternoon. It is hard work and a tough industry. Some small contractors are rumoured to start business by stealing hoes from others. Only some contractors pay workers compensation insurance and these that don't are able to pass on some of the "savings" by paying slightly higher wages. Most chippers earn about $50 a day.

Stick pickers

When a field is levelled for cotton someone must pick up the debris from small trees and bushes. This is a far smaller industry than chipping because stick-picking is only needed once when a new field is prepared. Stick pickers are organized in the

same way as chippers.

Cotton pickers

These workers drive huge machines that harvest several cotton rows simultaneously. Each time the machine passes over a row of well defoliated cotton plants it removes 70% of the cotton fibre. Each row is usually harvested two or three times. The pickers season is April-May and pickers are organized in the same way as as chippers and stick-pickers.

Gin workers

There are now cotton gins located throughout the cotton belt. Each gin serves many farms and usually operates 24 hours a day for 100 to 150 days a year. Gins are housed in very large, tall sheds. Cotton is sucked up from the load deposited by semi-trailers into long tubes and dried and fluffed as it moves rapidly towards the actual "gin". The gin is a rotating device in which small saws cut into the cotton to free the seeds, which are too large to fall through the spaces between the saws. Thus seeds and lint are separated. The lint is passed through more tubes, to a clamp that compresses it into bales ready for transport. The noise in a gin shed is very loud and some employees wear ear protectors. In some gins moving parts are well protected and the air is exhausted via a large dust chamber thus minimizing dust in the work place. Other gins are not so equipped.

ROLE OF THE AUSTRALIAN WORKERS UNION

Farm hands, equipment operators, gin workers, stick-pickers and cotton chippers who join the Australian Workers Union would then be covered by the Cotton Growing Employees (State) Award. The Award is presented in a seventeen page document and is primarily concerned with wages, leave, over-time, week-end penalty rates and meal hours. Worker safety is refered to in Clauses 18 and 23. Clause 18 gives employees the right to demand "rubber boots and other suitable protective clothing as considered reasonable" and Clause 23 incorporates the New South Wales Government Pesticides Act of 1978. Some contractors pay a lump sum to the A.W.U. to enroll all chippers they employ at $1.55 per person per week and provide A.W.U. - recommended toilet facilities on site. Such contractors also ensure their work force for workers compensation. Other contractors avoid union fees and do not insure their workers. This enables them to offer slightly higher wages. Many show little interest in joining the A.W.U. (Appendix 1)

An A.W.U. organizer is based at Narrabri. He reports relatively more success at unionizing sheep shearers than with cotton pickers. There are also problems in implementing the Award for those cotton chippers who are members of the A.W.U. For instance, few (if any) unionized workers actually receive week-end penalty rates.

CHEMICALS USED IN COTTON FARMING

General Comments

The chemicals used in cotton farming include fertilizers and soil additives, herbicides to control unwanted weeds, insecticides and acaricides to kill damaging insects and mites and defoliants to enable a trash-free harvest of cotton fibre. Herbicides, insecticides and acaricides are known collectively as pesticides.

Fertilizers and soil additives are usually applied before cotton chipping begins and defoliants after cotton chipping is finished. Insecticides and acaricides are sprayed frequently especially during the cotton chipping season when plants are growing rapidly. Herbicides are most useful before cotton plants emerge from the soil. Therefore cotton chippers are most likely to be exposed to those insecticides and acaricides applied to the crops during the chipping season.

Toxicity of the chemicals to humans

People may be exposed to the chemicals as follows:

1. by swallowing them in pure form or in contaminated drinking water;
2. by being splashed or sprayed;
3. by skin contacts with contaminated water or with sprayed plants;
4. by breathing the fumes of volatile chemicals;
5. by eating or smoking products directly contaminated;
6. by eating animals in which chemicals have accumulated via the food chain.

Chemicals may cause acute poisoning (if absorbed through the stomach, lungs or skin), irritation of the skin or eyes (as a liquid or as fumes) or lung damage (as fumes). Chronic poisoning may result from persistant low grade contamination. Certain chemicals can result in cancer (carcinogens), chromosonal damage (mutagens) or deformed children (teratogens). Some people may be allergic to a chemical and develop rashes, hives or asthma when exposed to this substance which may be innocuous to others.

Chemical manufacturers must provide detailed information on all of these potential hazards when seeking to register their porducts for agricultural use in N.S.W.

Insecticides and acaricides

(i) The pest problem

The main enemies of the cotton farmer are the Heliothis caterpillar, aphids, thrips and mites. Heliothis damages the cotton, aphids and mites damage the plant, and thrips secrete a sticky sugar which makes the lint difficult to spin. These pests must be controlled to maximize profits. In some areas (e.g. Ord River) pests were so troublesome that cotton farming was abandoned. In certain areas of Texas and Oklahoma cotton crops can be grown without much of an insect problem at all. In most parts of the world, including the irrigated cotton belt of N.S.W., farmers are winning the battle against insects by frequent use of insecticides.

The Cotton Research Station near Narrabri has recently helped to develop a computerized pest management plan called

SIRATAC. Farmers buy access to the SIRATAC "decision-tree" and watch their crops very closely for insects and mites. SIRATAC "tells" them the optimal time to spray and the most appropriate selective chemical to use. As more farmers join the scheme it is hoped that insecticide use will be optimized and minimized. This should lower costs, slow or even halt the development of insecticide resistance and decrease damage to harmless insect populations and to the environment.

(ii) Insecticide and acaricide toxicity to man

These chemicals are generaly divided into the following five classes:

(a) Chlorinated hydrocarbons or organo-chlorines (e.g. DDT, Gammexane);

(b) organophosphates (e.g. Malathion, Abate):

(c) Pyrethroids (e.g. Mortein);

(d) the carbamates (e.g. Baygon);

(e) a miscellaneous group (e.g. endosulphan, chlordimeform).

Some of the examples given above are in household use. Chemicals from all five groups are used by cotton farmers in N.S.W. A list of doses toxic to rodents by swallowing or by skin absorption is published in the N.S.W. Health Commission booklet "POISONING BY PESTICIDES" and is reproduced below. Chemicals underlined are used by cotton farmers.

TOXICITY TABLES

The amount of a toxix material, expressed in mg material per kg bodyweight of the animals, which causes the death of 50% of a test cage of animals, is called the LD $_{50}$ The oral LD $_{50}$ is obtained by feeding the material to the animal and the dermal LD $_{50}$ is obtained by applying it to their shaven backs. Thus, the smaller the LD $_{50}$ number the more toxic the pesticide may be; however, because the toxicity of a pesticide has been determined in animals the relationship of these figures to human poisoning is only approximate.

The concept of classification of pesticides by hazard is being discussed internationally to develop a general guide to the relationship of these LD $_{50}$ s to toxicity in man. A tentative scheme is shown:

Grading	LD50 mg/kg	
	Oral	Dermal
Extremely hazardous	5 or less	10 or less
Highly hazardous	5-50	10-100
Moderately hazardous	50-500	100-1000
Slightly hazardous	Over 500	Over 1000

Liquid preparations are considered in this system to be four times less hazardous, i.e., 10 mg/kg in a solid form rates the same as 40 mg/kg in liquid form.

ACUTE TOXICITY DATA FOR PESTICIDES

(1) INSECTICIDES AND ACARICIDES

(A) Organophosphorus Insecticides

Accepted Common Name	Some Common Trade Names	Oral LD50 mg/kg	Dermal LD50 mg/kg
Azinphos-ethyl	Gusathion A, Benzathion	9	280
Azinphos-methyl	Gusathion, Cothion	7	280
Carbophenothion	Trithion	7-30	800
Chlorfenvinphos	Birlane	10-155	30-108
Chlorpyrifos	Dursban, Lorsban	82-163	202
Demeton-S-Methyl	Metasystox (i)	57-106	303
Diazinon	Basudin, Gesapon	300-600	500->1200
Dichlofenthion		250	
Dichlorvos	Nuvan, Mafu	25	75-900
Dicrotophos	Bidrin	15	150-300
DDVP	See Dichlorvos		
Dimethoate	Rogor	200-300	700-1150
Dioxathion	Delnav	20	350
Disulfoton	Disyston	4	50
Ethion	"ethion"	13-34	1600
Ethoate-methyl		125	2000
Famphur	Warbex	35-62	1460-5093
Fenchlorphos	Lanokil, Ronnel	1000-3000	2000->5000
Fenitrothion	Folithion, Sumithion	130-200	700
Fensulfothion	Dasanit, Terracur P.	2	3
Fenthion	Lebaycid, Baytex	200	1300
Formothion	Anthio	400	400-1680
Haloxon	"haloxon"	900-2000	>6000
Iodofenphos	SC33, Nuvanol	2100	>2000
Maldison	Malathion	1400-1900	>4000
Mecarbam	Murfotox	15	380
Methamidophos	Tamaron	30	50-110
Methidathion	Supracide, Ultracide	20-48	25-400
Mevinphos	Phosdrin	3	90
Monocrotophos	Azodrin, Nuvacron	13	122
Naled	Dibrom	250-430	800-1100
Naphthalophos	Rametin	380	

Accepted Common Name	Some Common Trade Names	Oral LD_{50} mg/kg	Dermal LD_{50} mg/kg
Omethoate	Folimat, Le-mat	50	700
Parathion	Folidol E605	3	4-35
Parathion-methyl	Folidol M50	12	67
Phenamiphos	Nemacur	15	500
Phenkapton		50	>1000
Phorate	Thimet	2	70-300
Phosalone	Zolone	120	390
Phosmet	Imidan	113-245	2550
Phosphamidon	Dimecron	15	125
Pirimiphos-ethyl	Solgard	126	1000-2000
0,0,0,0-tetramethyl 0,0-thiodi-p-phenylene phosphorothioate	Abate	1000-8600	1370->4000
Thiometon	Ekatin	100	>200
Trichlorphon	Dipterex	650	>2800
Vamidothion	Kilval	64-100	1160
(B) Carbamate Insecticides			
Aminocarb	Matacil	30	275
Bendiocarb	Ficam, Multamat	179	>2500
Carbaryl	Sevin	400	>500
Methiocarb	Mesurol, Baysol	60-135	>2000
Methomyl	Lannate	27	>1600
Promecarb	Carbamult	35	450->2000
Propoxur	Baygon	80	>2400
(C) Chlorinated Hydrocarbon Insecticides			
Aldrin	"aldrin", Aldrex	40	>200
BHC (see Lindane)			
Camphechlor	Toxaphene	283	>1000
Chlordane	"chlordane"	283	>1600
DDT	"DDT"	300-500	2500
Dieldrin	"dieldrin"	40	>100
Endrin	"endrin"	3	60-120
Heptachlor	"heptachlor"	40	195-250
Isobenzan	Telodrin	5	5-30
Lindane	Gammexane	230	500-1000
Methoxychlor	"methoxychlor"	5000-7000	6000
(D) Other Insecticides			
Binapacryl	Morocide	58-225	720
Chloropropylate	Rospin	>5000	>150
Chlordimeform	Galecron, Acaron, Fundal	250-340	4000
Decachloro-octahydro-1,3,4-metheno-2H, 5H-cyclo buta (cd)-pentalen-2-one	Kepone	114	>2000
Derris (see Rotenone)			
Dicofol	Kelthane	575->2000	100-1230
Endosulfan	Thiodan	35	74-680
Fenazaflor	Lovozal	240	700->4000
Isopropyl 4,4-dibromobenzilate	Neoron	5000	>4600
Nicotine	"nicotine"	70	140
Oxythioquinox	Morestan	1100-3000	1000
Piperonyl butoxide		7500	
Pyrethrins	"pyrethrins"	570	>1350-5400
Rotenone	Derris	25-132	>940->3000

The organophospates sulprofos (Helothion) the synthetic pyrethroids cypermethrin (Ripcord, Nurelle) and deltamethrin (Decis) and the carbamate thiodicarb (Larvin) have been sprayed on the 83-84 crop but are not included on the 1979 Health Commission list.

(iii) Chemicals used frequently on cotton crops in N.S.W.

The most frequently used chemicals for cotton crops are tabulated on page 4 of the N.S.W. Agricultural Department cotton pesticides guide as reproduced below:

CONTROL STRATEGY FOR HELIOTHIS SPP. IN 1983/84

This strategy is proposed for cropping areas in northern N.S.W and Queensland with the exception of the Emerald area in Queensland (Clermont to Springsure), where synthetic pyrethroids are not recommended at any time. In that region, the same strategy, without the pyrethroid option in Stage 2, should be adopted.

(A) COTTON

STAGE 1 First Spray to Jan 9	STAGE 2 (42 days) Jan 10 to Feb 20	STAGE 3 Feb 21 to Last Spray
endosulfan BT/chlordimeform thiodicarb * acephate * methomyl sulprophos (Jan) plus mixtures with the ovicides chlordimeform and methomyl. If mite spray required: in January chlordimeform/mixture monocrotophos profenophos NO PYRETHROIDS * As yet unregistered	endosulfan BT/chlordimeform thiodicarb methomyl sulprophos profenophos parathion pyrethroid pyrethroid/mixtures * plus mixtures with the ovicides chlordimeform and methomyl. (If a pyrethroid spray fails in this stage and the cause is thought to be resistance, follow up with an insecticide from another chemical group. No more than THREE pyrethroid sprays should be applied in this stage).	methomyl thiodicarb parathion sulprophos profenophos plus mixtures with the ovicides chlordimeform and methomyl. NO PYRETHROIDS NO ENDOSULFAN

N.B. To help prevent resistance developing to other insecticides, use no less than THREE of the chemical groups listed below throughout the season.

Group A (Organochlorines)	- endosulfan (Thiodan, Endosan)
Group B (Organophosphates)	- sulprophos (Helothion) - profenophos (Curacron) - acephate (Orthene) - parathion (Folidol)
Group C (Carbamates)	- methomyl (Lannate, Nudrin) - thiodicarb (Larvin)
Group D (PYRETHROIDS)	- fenvalerate (Sumicidin) - cypermethrin (Ripcord, Cymbush, Nurelle, Polytrin) - deltamethrin (Decis)
Group C (Miscellaneous)	- BT: Bacillus thuringiensis (Dipel)

It is worth noting that use of pyrethroids is now confined to the 42 day period January 10 to February 20. This is because Heliothis larvae rapidly developed resistance to the new synthetic pyrethroids used heavily from 1979 to 1982. The current strategy is to lower the "pyrethroid pressure" on Heliothis populations by only using these chemicals during one of the five generations of Heliothis that occur each season. (It takes 42 days for the eggs to develop through the five larval (caterpillar) stages, pupate and emerge as moths). This should retard the spread of pyrethroid resistance in Heliothis populations. It also means that cotton chippers will be potentially exposed to these chemicals during only half of the chipping season; this could prove helpful in identifying any health risks to chippers associated with cotton insecticides.

(iv) Medical aspects of poisoning

The effects of acute poisonings with these chemicals are generally well known. Organophosphate toxicity is a frequent reported cause of hospitalization. These chemicals inactivate the enzymes cholinesterase and pseudo-cholinesterase throughout the body. This "locks" on the parasympathetic nervous system causing headache, fatigue, giddiness, salivation, sweating, blurred vision, pin-point pupils, asthma and a slow pulse. Symptoms begin from minutes to hours after exposure, may worsen over 24 hours and can last for one week. A blood test will remain positive for up to a month or so. Death can be very rapid and the antidote is the drug atropine (tablets or injection) followed by the oxime 2-PAM. Theophylline should not be used. Most cases are a result of skin absorption; if the poison was swallowed

vomiting should be induced. Organophosphates can cause a mild depression of cholinesterase activity in the body without any symptom and such cases can be detected by a blood test. Methyl-parathion can be useful on cotton crops throughout the season and is one of the most toxic of all the organophosphates.

Carbamate insecticides have a similar effect to organophosphates when absorbed in toxic doses. The treatment is atropine but 2 -PAM should not be used. Victims recover quickly and blood tests return to normal within 48 hours. Anybody who absorbs a small non-toxic dose of a carbamate on top of a chronic asymptomatic low dose of organosphosphate may suddenly become dangerously ill. The carbamate methomyl is sprayed throughout the cotton season; it is very toxic and, because it is a volatile chemical, can easily be absorbed by inhalation.

Organochlorines can cause anxiety, restlessness, excitability and convulsions. All except DDT are well absorbed through the skin. The insecticide endosulphan is frequently used on cotton and has a toxicity similar to the organochlorines. Unlike DDT it does not accumulate in body tissues. DDT is no longer used in the cotton belt.

Pyrethroids are generally considered non-toxic. However the relatively new synthetic chemicals cypermethrin and deltamethrin have been reported to cause nasal stuffiness and numbing of the skin.

Chlordimeform is the most important of the miscellaneous group in the cotton area. It is not fully registered and only certain spray companies have a permit to use it. It is sprayed throughout the season. The manufacturer conducts constant urine checks on spray company personnel but not on other workers in the cotton industry. Chlordimeform is on a Department of Industrial Relations (Division of Occupational Health, N.S.W. Government) list of industrial substances of suspected carcinogenic potential. (Supplement to the Cotton Pesticides Guide 1983-84.) It has also been reported to cause acute haemorrhagic cystitis among employees of a chemical-packaging plant. (Appendix 2) Symptons included pain on passing urine, blood in the urine and rashes. The paper is included in this report as it is an excellent illustration of the difficulty in recognizing outbreaks of pesticide poisoning. Patients usually consult different Doctors and the symptoms are either non-specific or actually mimic other diseases.

Herbicides

The N.S.W. Agricultural Publication "COTTON PESTICIDES GUIDE 1983 - 84" lists 14 chemicals used as herbicides to control weeds in cotton fields. These are listed below together with the dose required to kill 50% of rats experimentally exposed to the chemical via the stomach (Oral LD_{50}) or the skin (Dermal LD_{50}). The LD_{50} figures were obtained from the N.S.W. Health Commission publication "POISONING BY PESTICIDES". The numbers listed in the "Use" column refer to the registration of the chemical for the following applications:

1. Control of weeds in irrigation ditches.
2. Control of weeds before cotton is planted.
3. Control of weeds after planting before cotton plants emerge.
4. Control of weeds once cotton plants are well established

COTTON FARMING HERBICIDES

Herbicide	Trade Name	Use	LD_{50} (mg/kg) oral	dermal
amitrole	Weedazol TL plus	1	1,100-5,000	10,000
amitrole-T	various names	1	-	-
atrazine	Gesuprim Primatol A	1	2,000	7,500
dinatramine	Cobex	2	-	-
diuron	Diurex, Karmex	1, 3, 4	3,400	no apparent toxicity
fluometuron	Cotoran	1, 2, 3, 4	8,900	10,000
fluometuron & prometryn combined	Cotogard	2, 3, 4	-	-
glyphosate	Roundup	1, 2	-	-
MSMA	"MSMA" Daconate 8	4	700-1,800	no apparent toxicity
nitralin	Planavin	2, 3	6,000	2,000
pendimethalin	Stomp	2	-	-
phenisophan	Verdinal	4	-	-
trifluralin	various names	2	3,700 - 10,000	5,000

The most frequently used herbicides are pendimethalin and trifluralin (before planting), fluometuron (at time of planting) and MSMA (after plants are at least 8 cm high). However, new herbicides are constantly being developed and farmers often change their preferences. There are 60 herbicides listed in the N.S.W. Health Commission publication referred to above,

but many of these would not be suitable for use with cotton as they would kill the cotton plants. MSMA is on the Department of Industrial Relations (Division of Occupational Health, N.S.W. Government) list of industrial substances of suspected carcinogenic potential. This list is reproduced below.

Industrial Substances suspect of carcinogenic potential

Suspect Carcinogenic Materials

The following are classified as an industrial substance suspect of carcinogenic potential for man:

Camphechlor	* Amino Triazole (Amitrole)
Chlordane	Arsenic Pentoxide
DDT	Arsenic Trioxide
Dieldrin	Sodium Arsenite
Heptachlor	DSMA
Lindane	Lead Arsenate
Carbon tetrachloride	MSMA
* Ethylene dibromide	Sulfallate
Ethylene oxide	CDEC
Copper, chrome, arsenate (CCA timber treatment)	Chlordimeform

* No assigned TLV.

Arsenic and chromium compounds have carcinogenic potential.

Although the acute toxicity of some of these compounds is low, exposure should be avoided or reduced to a low level because of suspect carcinogenic properties.

Ethylene dibromide used as an agricultural and horticultural fumigant for soil and plants must be used with extreme care.

Fertilizers and defoliants

In cotton farming fertilizers are used before, during, or soon after planting. Nitrogen is the key additive and is applied to the soil either by direct injection (as anhydrous

ammonium under pressure) or by mixing urea or nitram (NH_3 NO_3) in water. Sometimes organic phosphates are applied and occasionally Zinc as the sulphate or the oxide in sprays or a fertilizer mix. Fertilizers do not (or should not) get used during the cotton chipping season.

Defoliants are used in May immediately before harvesting. Many different chemicals are available as sprays to cause the leaves to crack away from the stem (abscission agents) and some of these have a strong bad smell that can often be detected a long way from the field. After leaf abscission has occurred a salt (Sodium nitrate) is sprayed on to dry out the leaves. The leaves then fall to the ground and the harvester can pick off the cotton without also picking leaves and stalks. Cotton chipping is finished well before defoliants are used although it may sometimes happen that chippers are working on a very late crop when defoliants are applied to a very early crop in a nearby property.

Defoliants sprayed onto a "nearby cotton crop" were recently reported to have caused a Wee Waa man to develop a severe blistering skin rash (Appendix 2A).

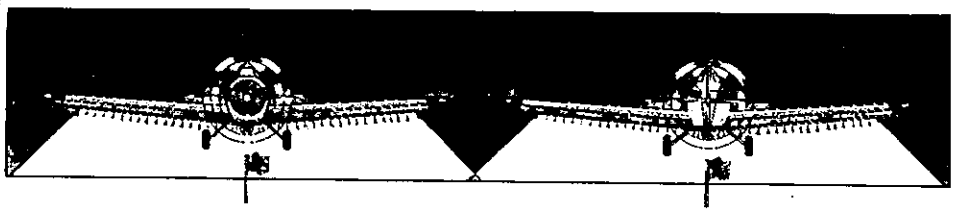
CHEMICAL APPLICATIONS: FORMULATIONS AND SPRAY TECHNIQUES

Sprays are prepared by emulsifing the chemicals in water or dissolving them in organic hydrocarbons or oils such as toluene, cottonseed oil or naptha oil. Some chemicals can be mixed together and the spray droplet size can vary. Generally ground sprays are applied from booms attached to vehicles that drive along the furrows. This permits direct application from a controlled height above the planted ridges and usually the droplets are quite large. It is not good for the soil to drive heavy vehicles through a cotton crop. The furrows are compacted so impairing self-mulching and penetration by rain or irrigation water. However, ground spraying enables a farmer to avoid the costs of spraying by air and to spray when planes are unavailable. Some large cotton growers never put vehicles in the crop once the cottonseeds have sprouted.

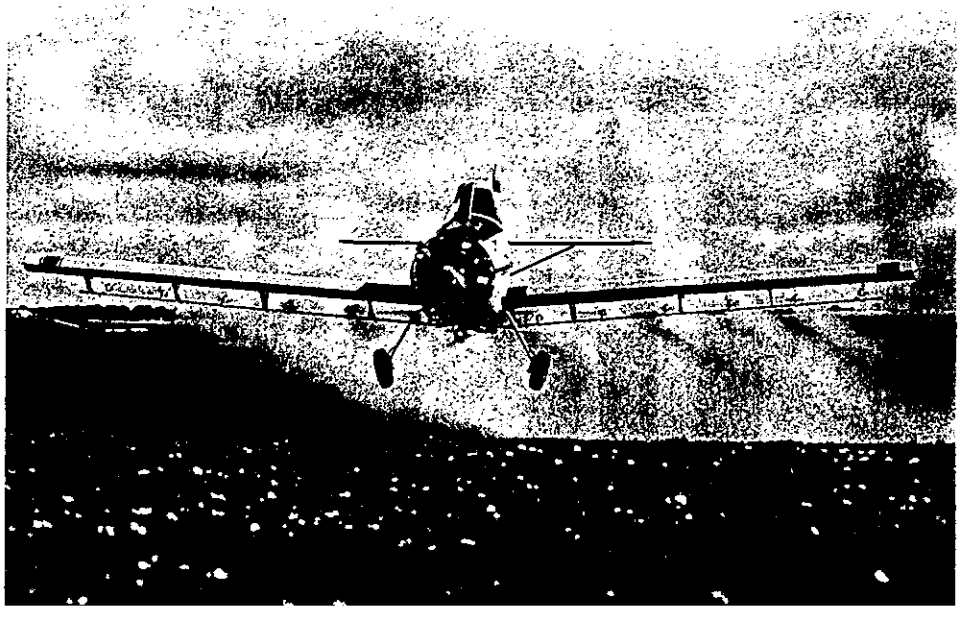
Aerial spraying is a complex science. The aim of course is to get the correct dose of chemical to the right place with minimum cost and maximum safety to operators and the environment. The various techniques were intensively reviewed by the N.S.W. Government in the 1970's. Large droplets would use more chemical and could fall on the crop in a stripe pattern. Small droplets would consume less chemical but may be more prone to drift. However, some drift is necessary to evenly cover the crop. Moreover small drops are more likely to be driven down by turbulence to the underside of leaves (where many pests are located) while spreading evenly over each plant in the crop.

Extrememly small drops of 50 microns in diameter are called Ultra Low Volume (ULV) and appear to be favoured in Australia at present. The chemicals are mixed in oils for this purpose and it seems that a light wind helps create turbulence at crop level and drives the spray down. However, on calm days, particularly when there is an inversion layer or thermal updrafts, the aerial ULV sprays can drift for miles and contaminate people, pastures, water supplies and other crops. This has caused many disputes. Safeguards against drift include avoiding ULV spraying on calm days, leaving a gap at the down wind edge of the crop and locating spray nozzles away from wing tips where vortices would carry the spray upwards. Non-selective herbicides should not be sprayed if adjacent fields contain crops that would be killed by the chemical. "Accidents" of this nature can lead to law suits (Appendix 2B). In the U.S.A. ULV spraying is more controversial than in Australia.

SPRAY TECHNIQUES



Effective swath overlap will result in a good job of weed control with no missed strips.



GOVERNMENT AGENCIES THAT REGULATE AND MONITOR THE USE AND EFFECTS OF PESTICIDES

The Pesticides Act

Pesticides are registered for agricultural use under the Pesticides Act, 1978, No 57 of the N.S.W. Government. This act continues a Register of Pesticides and Labels. The Registrar is empowered to approve and register products for use with specific crops if satisfactory data are provided as to the quality, efficacy and safety of the product and appropriateness of the label. The Registrar may also deregister the product or the label if new information makes this appropriate or impose use restrictions which must appear on the label. Such restrictions include:

(i) withholding periods: the minimum of days that must elapse between spraying a crop and submitting it for processing;

(ii) re-entry periods: the minimum number of days that must elapse before workers re-enter a crop after it has been sprayed.

Most labels include "withholding periods" which are especially important for food crops. Very few labels for cotton-approved pesticides (no more than 3 or 4) include "re-entry period" restrictions. (See Appendix 3 for examples of labels that do, and do not, include "re-entry periods")

Crop re-entry periods

California is far stricter than N.S.W. regarding re-entry periods. (Appendix 4) It must be noted that cotton fields

in California do not usually need chipping; and re-entry periods specified for cotton would probably be longer if manual weeding was still necessary. The Federal Environmental Protection Agency (EPA) in the U.S.A. is more liberal than the State of California in its recommendations on re-entry; none exceed 48 hours. Australia has devoted very little scientific research to the question of re-entry periods. Local data are obviously needed as climate, formulations, spray technique, spray frequency and crops may all differ somewhat between countries. Cotton chipping has been assumed to lead to little exposure to chemical resiudes. No consideration appears to have been given yet to the possible effects of a dew or of very early re-entry because of poor co-ordination between sprayers and chippers.

For many pesticides there is a residual effect on insects lasting several days after spraying the crop. This can often be a marketable feature of the chemical. The following table was taken from an advertising pamphlet distributed by a manufacturer.

Product	Rate L/ha	Days Residual Effect Under Continuous Pressure	Insects Controlled
Folimat	0.28	10	Thrips & Jassids
Folimat	0.14	7	Thrips & Jassids
Monocrotophos	1.8	5-8	Mites
Methomyl	0.40	4	Heliothis eggs
Methomyl	0.8	2	Heliothis larvae
Folidol M-50	1.4	2	Heliothis larvae
Metasystox (i)	0.30	7	Aphids
Helothion	2.0-2.8	5-6	Heliothis
Ripcord	2.0	7-12	Heliothis

NOTE: These approximate times will be extended if no Heliothis are present.

The residual effect of the spray chemical can be consequence of absorption by the plant or of persistance on the foliage. Any (ULV) spray which reaches the underside of a leaf is protected from ultra-violet radiation. Presumably the sprays with prolonged

effects on insects due to persistance on foliage would be the most likely to contaminate cotton chippers.

The Public Health Act (N.S.W.)

The Public Health Act, 1902 was last updated on January 22, 1982. It contains regulations concerning Hazardous Pesticides which are gazetted in Schedule 32 (see Appendix 5). These particular chemicals are subject to special controls additional to those of the Pesticides Act.

Inspectors

Both the Pesticide Act and the Public Health Act provide for inspectors with powers to investigate the use of Registered Pesticides and Hazardous Pesticides respectively.

Department of Industrial Relations (State)

The Division of Occupational Health in this Department helps to administer the Hazardous Pesticides Regulations of the Public Health Act. The Agricultural Laboratory performs chemical analysis for pesticides on people at risk or suspected of poisoning. (Appendix 6) A small team passes through the cotton belt each year and offers organophosphate tests to all those who volunteer. Very few (if any) aboriginal people participated at Moree or at Myall Vale this year. Of those that were tested (some 30 at Moree, 50 at Myall Vale) only 2 or 3 had a depression of cholinesterase levels. The testing team must cover a huge area and can only stay a few hours at each testing site. No tests are conducted at the cotton fields as the team does not

have a van equipped for such work.

The Poison Schedule Committee

This committee is chaired by Dr Allan Black of the Commonwealth Department of Health. Dr Black is a physician, pharmacist and toxicologist. All chemicals are assessed (including drugs) and ranked for their toxicity and other properties. It was not clear to us how the committee influences State regulations. Very comprehensive data packages are required by this body before any chemical is assessed. The committee does concern itself with crop re-entry periods for agricultural chemicals.

The N.S.W. Agriculture Department

The Registrar of Pesticides is located within the Head Office of the N.S.W. Agriculture Department. The Department takes a great interest in educating farmers to use pesticides responsibly. There is also a Division of Pesticides and Environmental Studies with extensive monitoring responsibilities and research entomologists are employed to help improve and evaluate the impact of pesticides. The Siratac computerized pest management program was developed jointly with the C.S.I.R.O.

Local Government

All municipalities employ professional health inspectors with considerable training on pesticides. They have wide ranging powers and responsibilities that interlock with all other agencies.

TOXIC PROPERTIES OF PLANTS ENCOUNTERED IN NEW SOUTH WALES COTTON FIELDS

Weeds

It was suggested to the team that Noogoora Burr could cause skin irritation. The scientific literature does contain references to toxic properties of Noogoora burr and also of Bathurst burr False Castor oil and cotton plants i.e. to all four of the plants to which cotton chippers are usually exposed (see Appendix 7). Scientists have focused research primarily on the toxicity to grazing animals that eat these plants or their products.

The False Castor oil belongs to the Datura species all of which contain potent tropane alkaloids, particularly scopolamine and hyoscyamine. These atropine-like substances can cause thirst, dilation of the pupils, flushing of the skin, extreme irritability, rapid pulse, convulsions, coma and death. Some of these symptoms are similar to those of organochlorine poisoning and opposite to those of organophosphate/cabamate toxicity. Humans are quite susceptible to tropane alkaloid poisoning. Poisoning results from sucking the nectar of flowers, from deliberate ingestion of seed or leaf concoctions for their hallucinogenic effect, from overdoses of medicinal drugs prepared from the plant or from contamination of food (e.g. flour) with Datura seeds. None of these exposures are likely to occur with cotton chippers. Fortunately no-one around N.W. N.S.W. appears to be aware of the (dangerous) potential of these plants for "tripping".

The Xanthium species (Noogoora burr and Bathurst burr) can cause convulsions, colic, liver and kidney damage and death

to animals that feed on the plant. The poison is thought to be carboxy atractyloside or sesquiterpene lactones (mostly xanthumin); some other substances of unknown toxicity have also been isolated. As cotton chippers do not eat the plant it seems unlikely that they would be poisoned by Xanthium weeds. However, it was noted that contact with Bathurst burr can produce a skin rash "in some people". This, of course, is true of virtually all plants but it could be that this quality is more pronounced for Bathurst burr.

Cotton plants

Cotton-seed meal may be toxic for animals, especially non-ruminants such as pigs. The main toxin is the substance gossypol, a polyphenolic sesquiterpene pigment with insecticide properties that is contained within small purple spots in the leaves, stems and seeds of cotton plants. Cotton has evolved many terpenoids as tannins which are currently under study by Drs Peter Twyne and Bob Nobel (Department of Primary Industry) in Queensland. Gossypol-free cotton plants have been bred but are especially susceptible to insects. It is considered unlikely that cottonplant toxins would be found on the plant surface in an unbound form that could contaminate people who contacted the plant. It is worth noting that gossypol has recently attracted considerable interest, especially in the Peoples Republic of China, because of its properties as a male contraceptive.

ANALYSIS OF COMPLAINTS THAT ABORIGINAL COTTON WORKERS DEVELOP SKIN, BREATHING AND OTHER AILMENTS

Initial Enquiries

Before the investigation commenced the only information available was that cotton workers were getting "funny boils" and "rashes". As boils and scabies are prominent problems in aboriginal communities all over Australia assessing the complaints was not going to be easy. We assembled in Moree and soon learned that "blisters", "asthma", "peculiar weaknesses" and other problems were on the list. We also learned that aboriginals frequently work with cotton as chippers and that the chipping season had finished two months earlier. The itinerant workers had left but most aboriginals that remained in the area had experience as cotton chippers.

Other complaints were that swimming in the river was causing "sore eyes" and an "itchy rash" and that many fish were caught with "sores" so bad that aboriginal fisherman threw them back. There had also been instances of dead fish floating in large numbers in some of the rivers and people complained that towards the end of summer the stench of agricultural sprays was noticeable in townships throughout the cotton belt.

Over the first two days we contacted as many aboriginal people as possible. We discovered that the complaints were widespread and that many had concluded that the problems were a consequence of poisoning and environmental contamination by chemicals sprayed onto cotton fields. It was also apparent that cotton farming on a large scale was new to the Moree area and was a much valued

source of employment at Christmas time. There was also considerable disquiet amongst the non-aboriginal townsfolk and a feeling of sudden encirclement by the strange, chemically intensive and highly mechanized industry of cotton farming.

Dr Sleigh contacted the Moree Doctors and, together with Noel Gillon, spoke with the Catholic nuns who operate a clinic at Mehi Crescent aboriginal village. The nuns had noted that boils seemed to be excessively frequent nowadays and both the Doctors and the nuns quickly pointed out the poor hygiene and crowding evident at Mehi Crescent and Stanley village - the two aboriginal settlements on the outskirts of Moree. The Doctors had not encountered any proven cases of florid poisoning with agricultural chemicals for some years. There was a recent case of apparent organophosphate poisoning but the blood test failed to confirm the diagnosis. There had been two children born with neural tube defects in the past year (one with anencephaly and one with meningomyelocoele); but no-one was aware of an excessive rate of such malformations over the past few years. Some Doctors had noted a "contact dermatitis" in patients who were cotton chippers which did not appear to be serious as it resolved after a day or two away from the fields.

The overall impression of the Moree Doctors was that poor hygiene, poor diet, cigarettes and alcohol posed a far greater threat to the health of aboriginals than a few scratches, the odd rash, blisters or skin infections acquired by chipping cotton. They also stressed that they were concerned about the excessive frequency of mechanical trauma amongst those who worked with cotton gins and harvesters. Most of these people are working very long hours and fatigue could be contributing to the many

accidents reported in the local press. (See appendix 8)

After two days in Moree the team split up to explore the whole cotton belt with one group travelling west through to Bourke and the other north and east. The operations of the cotton industry were examined, the Cotton Research Station visited, and aboriginal people were contacted throughout the area. The complaints were repeated in other areas.

The team reconvened in Moree four days later and compared notes. It was obviously necessary to attempt to classify and quanitify the complaints and assess their relationship to cotton work applying the information on cotton farming we had now acquired. Moree has the largest aboriginal population in the cotton belt so this was the logical focus for further enquiries. Local people felt themselves to be the "most surveyed people on earth" and were reluctant to endure yet another formal survey. Eventually we agreed on a very simple questionnaire which was devised so they could conduct it themselves. (See appendix 9) The survey was pre-tested and the aboriginal team members, together with some respected local aboriginal people, were trained not to lead answers to any of the questions. Most of the interviews were at Moree but some were conducted at Toomelah and Boggabilla to the north and a few at Wee Waa in the south west.

Time and money limited the survey to two days and a total of one hundred and sixty (160) aboriginal people were interviewed. The team then broke up leaving Dr Sleigh to continue enquiries in Moree and Sydney over the next four days into some technical aspects of cotton farming.

The aboriginal people of Moree

The aboriginal population of Moree township is estimated by the Department of Aboriginal Affairs to be 1,750 or 16.7% of the town population. Approximately five hundred live in Stanley village or Mehi Crescent village (with an average of 10.6 persons per house) and the rest live elsewhere in the town itself. The aboriginal population is substantially younger than the non-aboriginal residents and the aboriginal workforce is estimated at seven hundred and twenty (720) persons.

Results of the survey on health complaints and cotton work

The sex, place of residence and chipping experience of those interviewed were as follows:

	Chippers			Non-chippers		
	M	F	T	M	F	T
Moree	67	38	105	9	21	30
Toomelah	9	2	11	0	0	0
Wee Waa	6	1	7	0	0	0
Boggabilla	2	1	3	1	3	4
Total	84	42	126	10	24	34
%	67	33	100	29	71	100

Overall 79% (126/160) of those interviewed had been cotton chipping and 67% of all cotton chippers were males. All of those who had worked with cotton had been chipping. Some of the 126 cotton chippers had also done other cotton-related jobs as follows:

Stick picking	70
Cleaning ditches	23
Cotton picking	21
Cotton gin work	11
Flagging planes	9
Mixing spray	6

As stick picking is a once only job for each area of land cleared for cotton it is obvious that cotton chipping is the most frequent type of exposure to the cotton industry amongst those aboriginals interviewed.

The male chippers interviewed tended to have had more experience (exposure) as cotton chippers than did the women. This was estimated by asking people the number of seasons they had worked with cotton.

		Seasons worked			
		1	2	3	>3
Sex	M	11	17	19	37
	F	24	5	4	9
	T	35	22	23	46

A compatible trend was noted when people who had worked with cotton were asked if they had become sick at or after work: those most experienced (i.e. males) reported relatively more illness.

		Sick at or after work		
		Yes	No	%sick
Sex	M	26	58	31
	F	10	32	24
	T	36	90	29

However this association between sex and illness rate at or after work did not reach statistical significance ($p > 0.05$). When cotton work experience was tabulated directly with the rate with which people reported sick at or after work the results were as follows:

	Seasons worked			
	1	2	3	> 3
T	35	22	23	46
No sick	5	8	6	17
% sick	14	36	26	37

The accuracy of the above data are obviously dependent on memory; experiences in past years could be less remembered than those of the most recent season.

The type of illnesses described by those who became sick at or after work were relatively few. Some people described more than one complaint.

		Sick at/after work
Complaints	Rash	22
	Blisters	6
	Sore eyes	5
	Headache & nausea	5
	Vomiting	2

Blisters on the hands were excluded from the analysis as they would be expected to result from handling a hoe. Overall

29% (36/126) of these who worked with cotton complained of being sick at or after work and 61% (22/36) of those people reported developing a rash.

An alarming proportion of people (30%) stated they had been sprayed by a plane directly or by drift. There was an association between degree of experience of cotton work and being sprayed.

	Seasons worked				
	1	2	3	> 3	
T	35	22	23	46	126
Sprayed by a plane	4	6	9	19	38
% sprayed	11	27	39	41	30

Most (72%) of cotton workers stated they had chipped in a field wet from a spray. The chance of having worked in such a field tended to rise with increasing chipping experience.

	Seasons worked			
	1	2	3	> 3
T	35	22	23	46
Worked in wet field	17	13	21	40
% in wet field	49	59	91	87

Farmers, spray companies and some agronomists told us a heavy dew was common in cotton fields even in December and January during the chipping season. A senior Government agronomist and a

meteorologist doubted a dew would form, even in the microclimate of an irrigated cotton field, before late February when chipping is finished. Chippers start work at dawn and Moree weather records show 6 a.m. wet and dry bulb temperatures close enough to form a dew occasionally during December-January. However, such temperatures are always so high ($\geq 15^{\circ}C$) that evaporation would be substantial and theoretically the dew would never "hit the ground". If fields are wet because of an early morning dew then emulsifiable chemical residues left from sprays could be remixed by the dew and so pose a risk to those who enter the crop. If fields are wet in the morning because they were sprayed during the night the hazard to chippers is obvious.

Only 8% (10/126) of cotton workers reported having had a blood or urine test for spray chemicals. Given that six people had worked mixing sprays this suggests that cotton chippers as a group are almost never tested for organophosphate toxicity or for other chemicals.

People were also asked if they had been troubled with any boils this year. Boils were reported by 57% (72/126) of the chippers and 18% (6/34) of the non-chippers. This difference between chippers and non-chippers is statistically highly significant ($x^2 = 15.1$, $p < 0.001$) and the rates for both groups are extraordinarily high. Boils were mostly described on arms, legs and buttocks and occasionally on the face.

It should be noted that all ages were not equally represented amongst the chipper and non-chipper groups.

	No of persons in age group (yrs)		
	15-25	26-45	46+
Chippers	60	50	16
Non-chippers	16	5	13

Thus the age group 26-45 years was relatively less represented and older (46+) persons more represented in the non-chipper group. However, the median ages (chippers 26 years, non chippers 25 years) were similar.

People were also asked whether they had ever had a rash, blister, giddiness, visual trouble or asthma. This question was placed below that concerning illness after work in a cotton crop to avoid suggesting answers to the "after work" question. The checklist question was designed to ascertain the absolute frequency with which people remembered these complaints and to allow comparison of chippers and non-chippers. The results were as follows:

Complaint checklist	Positive	
	Chippers No (%)	Non-chippers No (%)
Rash	71 (56)	9 (26)
Blister	15 (12)	1 (3)
Vision trouble	30 (24)	5 (15)
Giddiness	33 (26)	4 (12)
Asthma	22 (17)	2 (6)

Rash is a frequently remembered complaint in this community and was usually "itchy". All complaints are substantially more common amongst chippers. It is disturbing to see "old people's diseases" of visual trouble and giddiness reported more frequently in the

younger group - the chippers.

Despite all these complaints only 64 of the 135 Moree residents interviewed stated they had seen a Doctor this year. Those that had seen a Doctor went to the nun's clinic, the hospital or to private surgeries with approximately equal frequency.

There were 93 people who had swum in a river and 14 complained of feeling sick afterwards. The post-swimming illnesses described were:

Ear infection	1	Nausea & vomiting	3
Rash	1	Eyes sore	3
Boils	2	Virus	1
Coughing	1	No explanation	2

Discussion of survey results

The data support the contention that rashes, blisters and boils are related to cotton chipping. The cross consistencies and the recurring trend of increasing complaints with longer exposures to chipping would be hard to "manufacture". Boils are epidemic and probably represent a septic complication of skin trauma suffered when handling the weeds while chipping. The high rate in non-chippers presumably has resulted from contact with chippers.

The frequency with which people enter wet fields and/or get sprayed must raise strong suspicions that skin contamination with pesticides is currently an occupational hazard of cotton chipping. It would be a hazard for which very few cotton chippers are tested.

The higher rate of giddiness, asthma and visual troubles in the (younger) chipper group compared to non-chippers could be a result of organophosphate or carbamate poisoning (or both). Alternatively the asthma could be an allergic phenomenon to sprays (chemicals or diluents), dust, plants or cotton fibres. Rashes and blisters could also result from widespread allergy to vegetable matter.

The data do not support the contention that river swimming has produced unexpected ailments.

COMPLAINTS REGARDING ENVIRONMENTAL CONTAMINATION

Fish deaths

The team visited the place where many fish had suddenly died earlier this year (Gil Gil creek, near Moree). We noted that a cotton field was very close to the creek bank and that many trees in the creek were dead. We subsequently learned that two water samples had been taken, one at the bridge where the deaths were first noted and one a mile to the west. The first sample contained endosulphan (0.9 µg/l) as did the second 1.5 µg/l); neither sample contained measurable quantities of heptachlor, HCB, DDT, Deildrin, organophosphates or "herbicides". Obviously the creek was contaminated with the insecticide endosulphan which is known (and labelled) to be very toxic to fish. An episode of fish poisoning in the Rhine river in Germany some years ago was associated with endosulphan levels of 0.7 µg/l. The EPA guidelines in the U.S.A. for safe levels for fresh water aquatic life are 0.003 µg/l. It seems most likely that endosulphan killed the fish at Gil Gil creek.

We learned of a similar episode at a dam on Boonydoon farm near Mungindi on the Queensland border. Wildlife and one cow died suddenly in or near the dam. Endosulphan residues were found in cotton plants (11 mg/kg), weeds (14 mg/kg), a dead wood-duck (1mg/kg), a dead fish (0.2mg/kg), a dead crayfish (0.6mg/kg) and in the dam water ("0.0006 mg/l" = 0.6 µg/l). Again this seems to be a case of wildlife death associated with environmental contamination with endosulphan but should not be considered as "proven"; apparently the case is legally sub judice in Queensland at present.

Fish diseases

People reported that many fish had bad sores. We learned of a condition known on the coast as "Bundaberg fish disease" affecting fish in river estuaries from Sydney to West Irian. The disease causes skin ulcers considered pathologically to be "phycomycotic granulomas" (chronic fungal infections of the skin). The disease appears to be also known as "Red Spot"; it afflicts many species and is thought to be occurring with inland fish as well. (See appendix 10)

We did not discover what relationship, if any, the "sores" on Moree fish have to "Bundaberg fish disease" or "Red Spot".

Other complaints

People also thought the drinking water was contaminated with chemicals, that wildlife were dying and that a mouse plague was present in town.

Drinking water supplies are monitored by the Moree Plains Shire Council and general environmental studies are conducted through the Office of Pesticides and Environmental Studies of the N.S.W. Department of Agriculture. Unfortunately key people with information on drinking water studies and wildlife problems were away from their offices during our investigation and we have no objective information on these problems.

We did establish that there is a mouse plague, both in Moree and the surrounding rural areas. In fact we actually obtained some hard figures on this problem. Recently, in the kitchen of an important building in Moree some 22 mice were caught in a mere 20 minutes! Mice were also "bad" two years ago during the drought. As this season has had good rains it is not easy to relate mice plagues to season. Nor is there any obvious link to pesticide use. Some people suggested that the storage of wheat in open silos after bumper harvests may be causing the mouse plagues.

Finally, people noted that there were no snakes in the fields at all and believed this meant the fields were too poisoned for snakes to survive. These observations especially during the mouse plague are interesting. However, we did not learn of any studies elsewhere where snakes were used in sprayed fields as sentinel animals to monitor the hazards of chemical residues. It is possible that it was snake prey (e.g. mice) rather than snakes themselves, that could not survive in the fields. Outside the fields venomous snakes are a serious problem around Moree. One tourist pamphlet describes certain areas too dangerous to go walking because of snakes. Unfortunately we could not get any objective data on "the snake problem".

United Farm Workers contract with cotton grower California

ARTICLE 14: HEALTH AND SAFETY

A. The Union and the Company are concerned with the health of the employees and the working conditions provided such employees. Therefore, the Company expressly agrees to strictly abide by and strictly comply with all applicable Federal and State laws, rules and regulations promulgated for the health and safety of employees. Upon notification by the Union of any alleged violation of this Section by the Company, the Company's designated representative shall immediately meet with the Union to discuss the matter to mutual resolution of the alleged violation. This Article shall include but not be limited to the use of machinery, vehicles, and dangerous chemicals and sprays, and any provision of food, drinking water, housing and sanitary facilities.

B. It is understood and agreed that it is necessary in the sophisticated farming practices of today that certain agricultural chemicals must be used for the control of pests and growth of the product. The Company shall notify the Union as soon as possble prior to the application of herbicides and pesticides. One baseline and other additional cholinesterace test shall be taken on any employees applying oranophosphates at the Company's expense, and if requested, results of said test(s) shall be given immediately to an authorized Union representative. The following records shall be kept at the ranch office and made available to any authorized Union representative:

1. A plan showing the size and location of fields and a list of crops or plants being grown.

2. Pesticides and herbicides used, including brand names plus active ingredients, registration number on the label and

manufacturers batch or lot number.

3. Date and time applied or to be applied.
4. Location of crops or plants treated or to be treated.
5. Amount of each application.
6. Formula and concentration.
7. Method of application.
8. Person who applied the pesticide and address.
9. Date of harvest.

C. No employee shall be required to work in any work situatio which would immediately endanger his health or safety. An employee shall make a reasonable effort to notify the Company of the existence of such condition.

D. In accordance with law, there shall be adequate toilet facilities, seperate for men and women, in the field accessible workers, that will be maintained by the Company in a clean and sanitary manner.

E. Each place where there is work being performed shall be provided with suitable, cool, potable drinking water convenient to workers. Individual paper drinking cups shall be provided.

F. Tools and equipment and protective garments necessary to perform the work and/or to safequard the health of or to prevent injury to a worker's person shall be provided and maintained by the Company, including but not limited to boots and rain gear for irrigators, wrenches for equipment operators, tools for service person, and protective garments for sprayers. The service person shall be provided with overalls. Employee(s) shall be responsible

cont.

for returning all such equipment or gear that was checked out to them, but shall not be responsible for normal wear and tear including breakage or disintegration due to normal wear and tear. Employee(s) shall be charged actual cost for equipment that is not returned. Receipts for returned equipment shall be given to the employee(s) by the Company.

G. Adequate first-aid supplies shall be provided and kept in clean and sanitary dust-proof containers.

COLLECTIVE BARGAINING AGREEMENT

BETWEEN

KLEEN SEED FARMS (Cotton Farmer)

AND

UNITED FARM WORKERS OF AMERICA, AFL-CIO

shall remain in full force and effect.

Executed this 19th day of September, 1983.

UNITED FARM WORKERS OF AMERICA, AFL-CIO

KLEEN SEED FARMS

Appendix 1A

A.W.U. AWARD
PAY SCALE

COTTON GROWING EMPLOYEES ETC (STATE) AWARD

BASIC WAGE $92.90

ALL RATES ONE AS FROM OCTOBER 6, 1983. April 16th 1984

RATES OF PAY	RATE PER WEEK	RATE PER HOUR
General Farm Hand	$ 223.50 237.90	$ 5.7125 5.9475
Field Equipment Operator	$ 230.90 246.30	$ 5.915 6.1575
Mechanical Equipment Operator	$ 250.20 260.50	$ 6.2550 6.5125

LEADING HANDS $12.00 per week for all purposes of the Award

CASUAL EMPLOYEES (work less than forty hours, including 15% loading and holiday pay)

General Farm Hand	$ 7.1276 p.h.
Field Equipment Operator	$ 7.3303 p.h.
Mechanical Equipment Operator	$ 7.8046 p.h.

Stick Picker and Chipper	$ 5.97 p.h. 6.21
Stick Picker and Chipper	$ 6.4775 p.h. with holiday pay 6.73
Stick Picker and Chipper per 8 hour day	$ 51.82
Less tax of 10%	$ 5.18
Net pay per day	$ 46.64

A.W.U. dues for Stick Picker and Chipper is $1.55 per week, to be taken out of the first day's pay of each week.

Each week consists of five days, Monday to Friday. Appropriate overtime rates for all employees on Saturday, Sunday and Public holidays.

E C ECOB
SECRETARY

C NEWTON
ORGANISER
PHONE : (067) 922-102

BE:RS
26/1/84

Appendix 2

Acute Hemorrhagic Cystitis

Industrial Exposure to the Pesticide Chlordimeform

David S. Folland, MD; Renate D. Kimbrough, MD; Richard E. Cline, PhD; Richard C. Swiggart; William Schaffner, MD

• **An outbreak of hematuria occurred from May 20 to May 23, 1975, among employees of a chemical packaging plant. Nine of 22 workers who packaged the insecticide chlordimeform in a separate shed became severely ill with abdominal pain, dysuria, urgency to void, or hematuria. None of 18 persons who worked in other areas of the plant were affected. Four additional workers who had packaged the chemical during the previous year had a history of similar symptoms. Bladder biopsy specimens from three affected persons showed severe hemorrhagic cystitis; chlordimeform and 2-methyl-4-chloroaniline, a metabolite of chlordimeform, were present in urine specimens collected three days after exposure. The illness lasted from one week to two months; the workers recovered completely. Chlordimeform that was injected subcutaneously into three cats produced similar, though less severe, changes in the bladders of two animals.**

(*JAMA* 239:1052-1055, 1978)

THE SEARCH for effective insecticides that have a limited environmental impact has led to the production of a wide variety of new agents.

For editorial comment see p 1072.

The health hazard to workers involved in the production and handling of some of these pesticides is not sufficiently appreciated. Chlordimeform hydrochloride is one of a group of new pesticides called formamidines.[1,2] It has been used primarily to control mites and insects on cotton, but is also used on some fruit and vegetable crops.

Chlordimeform is manufactured in Switzerland, is distributed to at least 20 countries,[3] and was marketed in the United States from 1971 to 1976 under the trade names Galecron and Fundal. The chemical was shipped to this country in bulk and was repackaged for retail sale. This article reports an outbreak of acute toxic hemorrhagic cystitis among workers in a chlordimeform packaging plant.

From the Bureaus of Epidemiology (Dr Folland) and Laboratories (Drs Kimbrough and Cline), Center for Disease Control, Atlanta; the Tennessee Department of Public Health (Dr Folland and Mr Swiggart), and the Department of Medicine, Vanderbilt University School of Medicine (Dr Schaffner), Nashville, Tenn.

The use of trade names is for identification only and does not imply endorsement by the Public Health Service or the US Department of Health, Education, and Welfare.

Reprint requests to Department of Medicine, Vanderbilt University School of Medicine, Nashville, TN 37232 (Dr Schaffner).

BACKGROUND

For three years, a packaging plant in Tennessee had received 74-kg drums of chlordimeform powder, which was then repackaged into 1.19-kg bags. It was packaged intermittently on a demand basis; approximately 5,400 kg of chlordimeform could be packaged daily. In 1975, the ch dimeform packaging apparatus was m to a shed that was separate from the pl other packaging operations. The shed made of curved metal and measured app imately 8×33 m. Ventilation was prov only through a side door and a si window at the end of the shed. Some pr tive clothing was made available to workers, but most wore only street clo and a cotton mask.

Because the metal shed was reporte be considerably hotter than the buildi which the packaging line had been loc previously, workers avoided using the tective garb. They also did not use available shower facilities, and manage did not insist on their use. The plant been inspected by the state health de ment's Division of Occupational and R logical Health early in May 1975, and tions were issued including one concer the inadequacy of workers' protective c ing. Finally, on May 20, a number of employees began work, and they ran repackaging machine much faster than customary, creating a fine, airborne mi the chemical.

On May 23 and May 24, 1975, a phys in a rural Tennessee town saw three y men who had gross hematuria and s toms of severe bladder irritation. Sin three had worked on the chlordimeform of the packaging plant during the prec week, he notified the Tennessee Depart of Public Health, and an investigatio undertaken.

METHODS

Medical histories were obtained fr (91%) of 48 employees who had work the plant during the week of May 20, and from four additional workers in

cont

hematuria had developed during the year before May 1975. Hematuria was assessed by dipstick in 38 of the 44 available employees. A case was defined as an employee of the packaging plant who had gross or microscopic hematuria, symptoms of bladder irritation (urgency, increased urinary frequency, and dysuria), or both.

A history of urinary tract symptoms was also sought from ten farmers who had recently sprayed their crops with chlordimeform. Preexposure and postexposure urine specimens were obtained from five farmers for amine analysis and screening for hematuria by dipstick.

Total urine hexane-soluble amine concentration was measured in 15 of 44 employees; urine concentrations of chlordimeform, 2-methyl-4-chloroaniline, and amine conjugates were measured in three hospitalized employees.

Total amine concentrations were determined by adding 3 ml of 10N sodium hydroxide to 10 ml of urine; the mixture was heated under nitrogen in a capped centrifuge tube for two hours at 80 to 82 °C. This process hydrolyzed chlordimeform and most of its metabolites to 2-methyl-4-chloroaniline. The amine was extracted with hexane and concentrated under a stream of nitrogen. The concentrate was analyzed by gas-liquid chromatography (GLC) at 120 °C on a column packed with 3% OV-1 attached to a Coulson detector specific for halogen. Recovery of total amine by this procedure was 75% ±7% in the concentration range of chlordimeform found in the patient's urine specimens. Starting with a 10-ml urine sample, the limit of detection was approximately 10 parts per billion of chlordimeform.

To determine the concentration of the chlordimeform and its metabolites, urine was made alkaline by adding 0.5 ml of 10N sodium hydroxide to 10 ml of urine; the mixture was then extracted three times with 20% ether in hexane. The concentrated extracts were analyzed by GLC at 120 °C for the amines and at 160 °C for chlordimeform. Water-soluble biological conjugates of chlordimeform and its metabolites remaining in the water layer after extraction were then hydrolyzed and heated, as in the procedure for total hexane-soluble amines; the liberated amines were extracted and determined by GLC.

To confirm the identity of chlordimeform and 2-methyl-4-chloroaniline in urine, a number of different liquid phases of varying polarities were used in GLC columns. Confirmation was also obtained by gas-chromatography–mass spectroscopy using an LKB 9000 instrument interfaced with a system 150 computer.

Animal Experiments.—An attempt was made to reproduce the bladder lesion in cats. Nine adult cats were divided into groups and were given doses for five days subcutaneously, with 50 mg/kg chlordimeform, 2-methyl-4-chloroaniline, or the suspension vehicle. Autopsies were performed on all cats that died or were killed. Tissues were fixed in buffered 4% formaldehyde and stained with hematoxylin and eosin.

RESULTS

Symptoms developed in nine (41%) of 22 persons who worked on the chlordimeform packaging line from May 20 to May 23, 1975. In contrast, none of the 18 workers in other areas of the plant where chlordimeform was not processed had experienced either hematuria or symptoms ($P<.003$). All affected persons were male; their median age was 21 years. Three men were hospitalized.

The nine symptomatic men reported symptoms suggesting bladder and urethral inflammation, ie, dysuria (nine), urgency to void (seven), increased urinary frequency (seven), nocturia (six), gross hematuria (six), penile discharge (six), abdominal pain (seven), and back pain (four). A peculiar symptom of "feeling hot" was reported by six workers. Two parents spontaneously recounted how their affected son left his bed and slept in front of the air conditioner, which he turned up, while the rest of the family was uncomfortably cool. Workers also reported sleepiness (nine), a skin rash (five), a sweet taste (four), and anorexia (four). No photophobia, cyanosis, fever, or dyspnea was noted. While most workers recovered within seven to 18 days, symptoms persisted in three workers for one to two months. Four additional workers who had packaged the chemical during the previous year had a history of similar symptoms. A case report of one person who was affected in the outbreak is presented.

Report of a Case

A 17-year-old boy worked on the chlordimeform line for three days beginning on May 20, 1975, as a packaging-machine operator on the 4:30 PM to 12 AM shift. While working he wore jeans, a short-sleeved shirt, cap, and a cotton mask, but no gloves. After working for 2½ hours on the third night, he felt hot and nauseated; he left the shed and vomited once. He slept until noon the next day and, when he awoke, experienced urgency, dysuria, and hematuria with blood clots. He urinated every five to ten minutes during the next day and ten to 15 times at night. He also complained of a penile discharge, abdominal pain, and drowsiness. His regular bowel movements ceased. He noted a rash on his arms, which began as fine papules; his skin exfoliated after two to three days.

Over the next two weeks, dysuria, urgency, and intermittent gross hematuria persisted. He consulted his physician four times. The following observations were noted: normal blood pressure, gross hematuria; RBCs (4+), proteinuria (4+), and an occasional undefined cast on urinalysis; and normal serum chemistry values (including serum electrolytes, BUN, and creatinine), except for SGOT level, which was elevated to 75 units/liter (normal, 0 to 40). His urine contained 15.2 μg/ml (ppm) of total amines, 2.16 μg/ml of chlordimeform, and 4.16 μg/ml of 2-methyl-4-chloroaniline.

Two weeks after the onset of symptoms, the patient was hospitalized. Findings from the general physical examination were normal; his blood pressure was 120/70 mm Hg. Laboratory studies included a WBC count of 6,500/cu mm, and a hematocrit reading of 43%. A centrifuged urine specimen contained 60 to 70 RBCs and 0 to 3 WBCs per high-powered microscopic field. Two urine cultures were sterile. The SGOT level remained elevated to 85 units/liter, and total serum protein level was 8.4 mg/dl. The serum creatinine level was 1.5 mg/dl and the creatinine clearance was 86 ml/min. Sulfobromophthalein sodium (BSP) retention was 9.0% and 7.8% in 45 minutes on two occasions. A cystogram showed low-pressure bilateral reflux and a contracted urinary bladder with a capacity of only 200 ml. Cystoscopic findings showed diffuse hyperemic inflammation and edema of the bladder wall. A bladder biopsy specimen demonstrated changes of acute hemorrhagic cystitis.

During the five weeks after hospitalization, symptoms of urgency and dysuria gradually subsided. On examination nine weeks after onset, symptoms had ceased, and urinalysis findings and bladder capacity were normal.

Hospitalized Patients

The following abnormalities were noted in the three hospitalized workers: microscopic hematuria and pyuria in all three, proteinuria in two patients, low creatinine clearance (86 ml/min in one patient), decreased serum complement level (68 to 78 units/liter in three patients), elevated SGOT level (49 and 85 units/liter in two patients), prolonged BSP retention (9% at 45 minutes in one patient), elevated serum amylase level (115 and 152 units/liter in two patients), small bladder capacity (75 to 200 ml in all three patients), ureteral reflux (two patients), and an intense inflammatory reaction in three bladder biopsy specimens (Fig 1). Findings from cystoscopy performed nine weeks after exposure showed normal-appearing bladders and normal bladder capacities. Findings from urinalyses

cont.

Fig 1.—Section of bladder biopsy specimen from patient with hemorrhagic cystitis. Epithelium shows local ulceration with some areas of complete slough. Moderate submucosal chronic inflammation and conspicuous congestion and dilation of submucosal vascular plexus. No cytologically atypical epithelial cells (hematoxylin-eosin ×100).

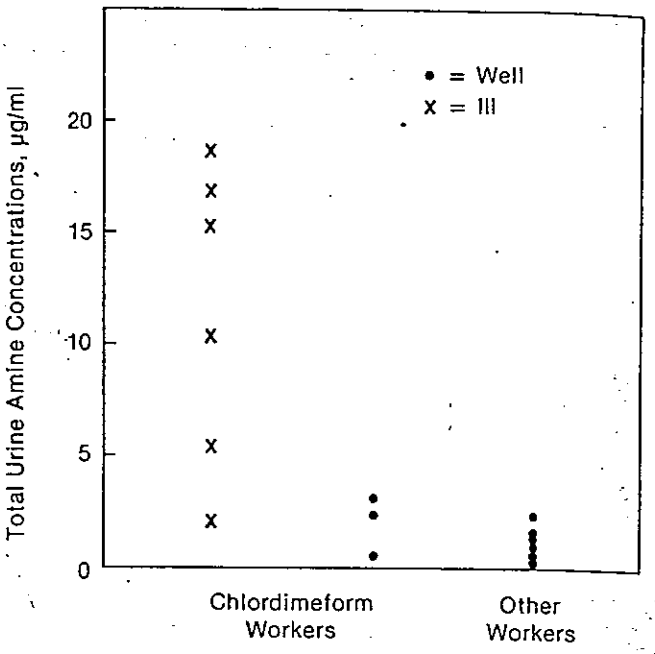


Fig 2.—Concentrations of total hexane-extractable amine in aqueous basic fraction of urine from chemical packaging plant workers.

had also returned to normal.

Attempts to isolate viruses (especially adenovirus type 11) from urine specimens of three acutely ill workers were unsuccessful. Testing of serum specimens from acutely ill and convalescent patients demonstrated no evidence of recent adenoviral infection.

Toxicologic Studies

The highest concentrations of total amines were found in the urine of workers who had packaged chlordimeform and who had been ill (Fig 2). Low, but measurable, amounts were found in the urine of three workers who packaged chlordimeform and were not ill and in the urine of six employees who did not package chlordimeform. While some of the chlordimeform was excreted unchanged in the urine, more was excreted as a metabolite, 2-methyl-4-chloroaniline, or as conjugates (Table).

Farm Users of Chlordimeform

The survey of ten farmers who used chlordimeform showed that none had experienced gross hematuria, and only one complained of dysuria. Microscopic hematuria was found in one of six farmers tested. The hematuria resolved three weeks after the farmer stopped spraying with chlordimeform. The amount of total hexane-extractable amine in four of the farmers' urine specimens was higher after they had sprayed, but concentrations were lower than in all but one symptomatic employee of the packaging plant.

Toxicologic Studies in Aqueous Basic Fraction of Urine*

Case	Days Between Exposure and Urine Collection	Total Amine, µg/ml (ppm)	Chlordimeform, µg/ml (ppm)	2-Methyl-4-chloroaniline, µg/ml (ppm)	Conjugates, µg/ml (ppm)
1	3	11.0	1.10	3.75	6.25
2	3	15.2	2.16	4.16	8.67
3	3	2.6	0.04	1.25	1.17

*Studies performed on three hospitalized workers.

Animal Studies

Two cats injected with chlordimeform died. Autopsy findings showed that the bladder of one cat contained a large blood clot. Microscopically, the two cats had hemolyzed blood in the kidney tubules, degeneration and sloughing of the transitional epithelium of the bladder, and bile stasis and vascular degeneration of hepatocytes.

The remaining seven cats were killed three days after the last dose. The bladders of the three cats that were given either the metabolite or the parent compound appeared edematous or congested on gross inspection. The kidneys, bladders, and livers of these animals were normal by light microscopic examination.

The bladders of the four control cats were normal.

COMMENT

To our knowledge, this is the first reported outbreak of human chlordimeform poisoning. The only previously reported toxic exposure occurred as a successful suicide in Japan in 1969,[4] the victim having ingested 30 ml of a 50% chlordimeform solution. The medical report made no note of a urinalysis and no autopsy was done.

The epidemiologic evidence indicates that short-term chlordimeform exposure produced this outbreak of hemorrhagic cystitis among workers in a chemical packaging plant. The occurrence of gross hematuria, an uncommon symptom, among young men whose single gathering place was at work, suggested a common source of exposure. The absence of fever and secondary cases in household contacts made an infectious cause unlikely. The high attack rate among workers who packaged chlordimeform and the absence of illness among workers in other parts of the plant implicated the pesticide as the toxic agent.

Many circumstances contributed to the outbreak. The chlordimeform packaging line had been moved to a small building with inadequate ventilation. The company did not provide adequate protective garb for the workers nor supervision sufficient to ensure the

safe operation of the packaging machine. New employees ran the machine in a manner that exposed them and others to excessive amounts of the pesticide. The early summer heat and the poor ventilation probably accentuated the hazard. The workers wore lighter clothing, increasing the risk of chemical absorption through the skin. Chlordimeform and its metabolites are excreted largely in the urine,[5] and relative dehydration would result in more concentrated urine, exposing the bladder to greater concentrations of the pesticide and its metabolites. The relative importance of dermal, respiratory, and alimentary absorption of chlordimeform could not be estimated, but all of these routes may have been important.

Workers may expose others to toxic agents that they carry away from the workplace on themselves and their clothes.[6] Hemorragic cystitis developed in a young woman during the outbreak; she was a romantic contact of one of the affected new employees. Although she never entered the plant building, she rode home in the same car with the workers who had chlordimeform on their bodies and clothing, particularly in their trouser cuffs. The car smelled of the chemical. While the precise nature of the young woman's physical exposure to her friend could not be established with certainty, it is likely that her illness was related to such secondary exposure to the pesticide.

The metabolism of chlordimeform has been studied in dogs, rats, and goats.[5] One of its major metabolic products is 2-methyl-4-chloroaniline, and this compound was identified in the urine of three hospitalized workers. It has long been known that the bladder is a sensitive end organ to the toxic effects of aniline compounds. Acute toxic effects caused by aniline and 2-methyl-4-chloroaniline (4-chloro-orthotoluidine) were first recognized among workers in the German dye industry at the turn of the century,[7,8] and were well studied there and in England in the 1920s.[9-12] Gross hematuria and "strangury" (severe dysuria due to bladder irritation) were common complaints. Findings from cystoscopic examination showed hemorrhagic cystitis and exfoliation of the bladder epithelium.[9-12] Similar findings were produced in cats that were exposed to 2-methyl-4-chloroaniline by applying it to intact skin.[11] While the character and severity of the urinary symptoms described previously are similar to those experienced by the workers exposed to chlordimeform in this outbreak, our toxicologic analyses do not specifically implicate the pesticide or any single metabolite as the toxic agent.

The workers experienced systemic effects as well as local irritation of the bladder. All were unusually somnolent, a symptom mentioned in an early report of 2-methyl-4-chloroaniline toxicity.[11] There also may have been liver, kidney, or pancreatic toxicity, suggested by elevated SGOT level and prolonged BSP retention, low creatinine clearance, decreased serum complement level, and elevated serum amylase values. The more serious features of acute aniline intoxication, cyanosis and coma, were not experienced in this outbreak. The affected workers recovered completely.

This outbreak is one of a growing number of serious industrial pesticide exposures, the most dramatic in the United States involving kepone.[13] Clearly, such incidents can be prevented only by meticulous attention to safety standards at the workplace. The author[11] of one of the first studies of acute aniline toxicity strongly cautioned against allowing workers to breathe a warm, damp mist of the chemical, and especially to avoid extensive skin contact. Unfortunately, since many workers engaged in unskilled jobs are recruited from an itinerant, lower socioeconomic group, employers seemingly devote less-than-adequate attention to safety instruction and supervision. For various reasons, regulatory agencies do not stringently enforce existing safety standards.[13]

This outbreak illustrates some of the difficulties in identifying toxic events caused by chemical agents in humans. Sporadic exposures may result in illnesses that are not recognized, partly because work-related disease is neglected in medical school teaching. In this episode, six individual physicians saw one or two affected workers without suspecting a toxin. Their diagnoses were either urinary tract infection or gonorrhea. This incident was investigated only because three patients went to the same physician, who suspected and *reported* a possible common-source exposure. With the introduction of many new chemical agents into the workplace and our environment, alert physicians should suspect toxic exposures if unusual or severe symptoms are present, especially if more than one patient is affected.

John Armstrong, MD, first suspected and reported a toxic exposure. Oliver Graves, MD, performed the clinical evaluations. A. L Middleton, Jr, MD, supplied the biopsy specimens. John A. Liddle, PhD, provided technical assistance. John C. Hierholzer, PhD, performed the virologic and serologic studies.

References

1. Sanders, HJ: New weapons against insects. *Chem Engineering News* 53:18-31, 1975.
2. Dittrich V: Systemic action of N-(2-methyl-4-chlorophenyl)-N, N′, N′-dimethylformanidine (C-8514, Schering 16268). *J Econ Entomol* 60:558-569, 1967.
3. Pesticide residues in food. *WHO Tech Rep Ser* 502:1-46, 1972.
4. 1971 evaluations of some pesticide residues in food: The monographs: Chlordimeform. *WHO Pesticide Residues Ser* 1:1-45, 1972.
5. Knowles CO: Metabolism of two acaricidal chemicals. *J Agric Food Chem* 18:1038-1047, 1970.
6. Baker EL Jr, Folland DS, Taylor TA, et al: Lead poisoning in children of lead workers: Home contamination with industrial dust. *N Engl J Med* 296:260-261, 1977.
7. Starck M: Ein seltener Fall von Anilinvergiftung. *Ther Monatshefte* 6:376-377, 1892.
8. Leichtenstern O: Uber Harnblasenentzündung und Harnblasengeschwülste bei Arbeitern in Farbfabriken. *Dtsch Med Wochenschr* 24:709-713, 1898.
9. Scheele K: The action of acute and chronic aniline poisoning upon the bladder. *Urol Cutan Rev* 31:281-285, 1927.
10. Scheele K, Stolze M: Das Cystoskopische Bild der Stangurie bei akuter Anilinvergiftung. *Z Urol* 21:161-167, 1927.
11. Lehmann KB: Studien über die Wirkung der Chloraniline und Chlortoluidine und des salzauren 5-Chlor-2-toluidins. *Arch Hyg Bakteriol* 110:12-32, 1933.
12. Currie AN: Chemical haematuria from handling 5-chloro-ortho-toluidine. *J Indust Hyg* 15:205-213, 1933.
13. Sterrett FS, Boss CA: Careless kepone. *Environment* 19:30-37, 1977.

NSW GRAIN FARMER Wednesday April 4 1984

Wee Waa cotton spray: chemical gives violent allergic reaction

At Easter time last year, Wee Waa agricultural implement importer, Michael Ciesolka, suffered a violent allergic reaction resulting in a severe skin rash, the loss of the skin on the palms of his hands and the soles of his feet, and the bursting of some of his smaller blood vessels.

After consultations with medical practitioners and a dermatologist at the University of Newcastle, Mr Ciesolka and his family believe they have traced the cause of the problem—chemicals used as cotton defoliants.

When the spraying starts this season, Mr Ciesolka has no alternative other than to leave his home and family, returning when the chemical application stops.

The rashes and skin loss Mr Ciesolka can contend with, but his concern is that the allergic reaction could damage his larger blood vessels.

"Last season, two weeks before Easter, I was working at a shed on my father's irrigation property at Wee Waa. At the time I had no shirt on.

"I was aware at the time of a plane flying around a nearby cotton crop."

He left Wee Waa later that day with a visiting American for a trip in connection with his agricultural equipment importing business. They drove to Toowoomba, Surfers Paradise, Lismore, Maitland, Bathurst, and Griffith.

"My skin had broken out in spots the day we left. By the time we reached Griffith I had developed a severe rash on my shoulders and face—so much so that I could not smile.

"A doctor at Griffith was unable to help me. He thought I was on drugs, and gave me some tablets. The tablets knocked me around, and I moped about for a week before deciding to see a general practitioner in Narrabri."

The Narrabri doctor referred Mr Ciesolka to a dermatologist. "I had lost a lot of skin. The skin on my hands and palms had lifted off, and the hard skin on the soles of my feet also lifted.

"It was not until weeks after the cotton spraying had been completed that we worked out beyond reasonable doubt what the cause of the problem was."

According to Mr Ciesolka, members of his family have always disliked chemicals such as DDT that are used on cotton. "Salt does just as good a job as a defoliant," he said.

But he bears no animosity towards the chemical companies or the spraying contractors. "They are very good to me," he said.

"They keep me posted about their spraying activities. The industry has got to use chemicals to make a dollar. I'm not a 'greenie'. After all, I am the odd one out. So I should move, rather than expect people to stop spraying."

However, Mr Ciesolka believes the chemicals should be used in moderation. "Defoliants are poisons, after all."

Mr Ciesolka imports Adams agricultural tillage tools from the Adams company, Oklahoma. He has been casting around in the importing and commercial sector for suitable lines since he left St Stannislaus College, Bathurst, in 1982.

"My father and I originally started looking at irrigation equipment," he said. "We are the agents for Raincat centre pivots, and Pierce side roller irrigation systems. But strong competition has led to a price war, and we are not in it to be fighting people over prices."

He opted instead for tillage tools—"anything from a mouldboard plough shear to a disc".

The Ciesolka range of imported products includes "a few lines unique to Australia".

"Our people don't make knock-on points like Connor Shea's yet," he said. "But we are looking at trash worker and scarifier points."

Eventually, the enterprising young businessman plans "to make my own stuff here in Australia".

"The only problem is that the steel here is not as good."

Another alternative is import part-processed steel, and attend to the hard facing in Australia.

Mr Michael Ciesolka, Wee Waa agricultural implement importer, at the

Aviation insurers move to shed chemical responsibility

By ANTHONY HOY

The aviation industry has taken steps to push the responsibility for chemical damage to crops from aerial spraying operators onto their farmer clients.

Mr Bernard Ashby, NSW manager for the Australian Aviation Underwriting Pool Pty Ltd...Aerial spray operators would be forced out of business if claims by farmers before the courts come to a head.

The NSW manager for the Australian Aviation Underwriting Pool Pty Ltd, Mr Bernard Ashby, said once chemicals had been properly applied, farmers would become liable for inversion problems and problems with volatile chemicals.

Mr Ashby, speaking at the "Farming to Survive" symposium at Moree, claimed aerial operators had last season succumbed to pressure from farmers to undertake spraying operations in unsuitable conditions.

Most of the operators would be forced out of business if the claims by farmers presently before the courts came to a head, he said.

He warned that aerial-spraying insurances could be cancelled completely within 12 months, forcing operators out of business in States such as Victoria and Queensland, where insurance was compulsory.

"You might wonder what aviation insurance has to do with your business. Well, until a few seasons ago, probably nothing," Mr Ashby said.

"I presume most of the farmers attending this seminar are growers from the Moree-Narrabri area. You would know, then, that last season was the worst season ever for chemical damage to crops, as a result of adjoining land being sprayed.

"The problem has become so bad, that over the last four to five years, for every dollar in insurance premium that our company has taken on this type of business, we have paid out four dollars in claims.

Farmers in the crowd Mr Ashby was addressing at this point of his speech broke into laughter. "Now, that is the attitude of the general public to insurance companies," Mr Ashby said.

"It is thought that we own big buildings, and that we are big multi-million dollar corporations. This is all fantasy.

"The buildings are rented, and we pay to put our names on them."

Mr Ashby said that in Victoria and Queensland the aerial operators cannot operate without an insurance policy. "We have got to the stage where we have given them 12 more months before we cancel the insurance completely," he said.

"They then cannot operate these aircraft to spray your crop. They will not be able to spray your weeds when it is too wet for you to get on.

"My eyes boggled today when I saw the tonnages of wheat that growers in the Moree district, for instance, do get, and when I related these tonnages to claim figures that were forwarded from this area.

"I am standing up here to be shot at. But the claims are exaggerated, and this worsens the figures.

"Most of the aerial operators have got limited insurance. Most of them will go out of business if these claims come to a head in court, because they just cannot get sufficient insurance for it."

Mr Ashby said the answer to the problem with a lot of the chemicals—once they had been properly applied—was to make the farmer liable for herbicide damage on adjoining properties.

"All the inversion problems and the problems with volatile chemicals that escape from your land once the chemicals have been applied correctly by the operator is the farmer's problem, just as if it was the farmer's bull that had got out and savaged the neighbor's cow.

When questioned as to where the operator's liability stopped, and the owner's liability started, Mr Ashby said inversion problems had until now been picked up by insurance companies. In the short term, however, it would become the farming community's responsibility.

"It is a bit difficult to say what [illegible] the inversion occurs. The simple answer to this is that we will either be putting a limit of distance on the property, or from the property being sprayed.

"Now, if any of you do order an aerial spray operator, and he damages any of your crops, he is not insured for that. It is only insurance against the neighbor's crop.

"If we start putting a distance on it, if the operator is working in accordance with proper regulations with regard to wind and temperature

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"All this vaporising of chemicals that goes on for miles around afterwards is going to be the farmers' problem in future, because we will know how far the drift can go from a faulty operation.

"There will be the odd case when a pilot does have a faulty nozzle in his aircraft, but the pilot will have a meter in his aircraft which will tell him immediately if he has got a leaky nozzle—something that farmers do not have on their ground rigs.

"We feel that a lot of the problems attributed to the aircraft operators have in fact come from the farmer's ground rigs."

Mr Jeff Esdaile, manager of Livingstone Farms, asked Mr Ashby to comment on his own practice of getting together with the aerial operator to make a decision on whether spraying would take place on the day in question, and together nominating the fields that could be sprayed and those that could not be sprayed.

"It is more or less a joint decision," Mr Esdaile said. "We rely to a certain extent on the aerial operator's previous experience as to whether or not his aircraft is going to get drift under those conditions.

"I am not talking so much about vapor drift for miles, but rather, I am talking about a knockdown herbicide's droplet drift for 300 to 400 metres into your own crop or a neighbor's crop."

Mr Ashby said one of the problems last season had been that, after several years of drought, all farmers had the opportunity to get a decent crop. "Last year the same happened to the aerial operators," he said.

"Because you have had lean seasons, they have had lean seasons. And last year they were as interested to make money as you were.

"You fellas did put a lot of pressure on them, which they did succumb to, and often last year they operated in conditions in which they should not have operated.

"If they operate under the correct conditions of wind and everything else, provided you are prepared to have a small margin left around your crop, you should not have these problems with spray drift from an aircraft."

Appendix 3

®FOLIDOL M50 BL SPRAY

®Trademark of BAYER GERMANY

ACTIVE CONSTITUENT:	500 g/L parathion-methyl (O,O-diethyl (4-nitrophenyl) phosphorothioate)

NOT TO BE USED FOR ANY OTHER PURPOSE
WITHHOLDING PERIOD: DO NOT APPLY LATER THAN 14 DAYS BEFORE HARVEST
FLAMMABLE: KEEP AWAY FROM NAKED FLAME
Workers should not handle crops for one day after spraying unless wearing protective clothing.
This produce is too hazardous to be recommended for use in the home garden.
Harmful to bees. Do not apply when bees are actively foraging.

UNION CARBIDE

Larvin® 375

Thiodicarb Insecticide

Aqueous Flowable

ACTIVE CONSTITUENT: 375 g/L 3,7,9,13-tetramethyl-5,11-dioxa-2,8,14-trithia-4,7,9,12-tetra-azapentadeca-3,12-diene-6,10-dione (An anti-cholinesterase compound)

FOR CONTROL OF *HELIOTHIS* SPP. IN COTTON

NOT TO BE USED FOR ANY PURPOSE, OR IN ANY MANNER CONTRARY TO THIS LABEL UNLESS AUTHORISED UNDER APPROPRIATE LEGISLATION.

WITHHOLDING PERIOD: Do NOT apply less than 21 days before harvest.
Do NOT allow livestock to graze cotton crops treated with LARVIN 375.

RE-ENTRY PERIOD: Workers may re-enter treated fields once spray deposits are dry.

Thiodan ULV INSECTICIDE

Active Constituent 240 g/L endosulfan Solvent: 295 g/L liquid hydrocarbon

HOECHST

For the control of Heliothis (Cotton Bollworm, Native Budworm), Rough Bollworm, Thrips, Aphids, Green Vegetable Bug and other pests as recommended on cotton.

NOT TO BE USED FOR ANY PURPOSE, OR IN ANY MANNER, CONTRARY TO THIS LABEL UNLESS AUTHORISED UNDER APPROPRIATE LEGISLATION.

WITHHOLDING PERIOD

NOT TO BE APPLIED LATER THAN 28 DAYS BEFORE HARVEST.

Appendix 14

TO: Adrian Sleigh

5/10/84

RE-ENTRY PERIODS

(California cotton)

Memo

RE: Pesticides used in the cotton industry – according to the Agricultural Commissioner's Office – Kern County, Calif.

Pesticide	Re-entry Period
Kelthane	less than 24 hours. (as soon as it dries)
Azodrine	48 hrs.
Parathion	48 hrs.
Disiston	24 hours
Methomyl	24 hours
Monitor	24 hours
Sevin	as soon as it dries.
Systox	48 hours
Superside	48 hours
Temik	24 hours.
Thimet	48 hours
DD (used prior to planting)	3 days

Herbicide

Round Up	as soon it dries
Dynitro	24 hours
Paraquat	24 hours

Pesticides that are Organo-phosphates are: Parathion and Temik.

Pesticide that are a carbanate (cholinastrate-inhibitor) is Methomyl.

+ Treflan less than 24 hrs.

per. Mr. F. Ortiz
UFW
Crops Dept
KEENE Ca. 93531

California Re-entry Periods
(all crops)

NOTE: Authority: Sections 407 and 12981, Food and Agricultural Code.

Reference: Sections 12980 and 12981, Food and Agricultural Code.

6858. Reentry Intervals In Days. (a) The following reentry intervals, in days, apply to the crops indicated:

	Corn	Citrus	Peaches & Nectarines	Grapes	Apples	All Other Crops
Azinphosmethyl (Guthion)	-	30	14	21	14	1
Carbophenthion (Trithion)	2	14	14	14	2	2
Carbosulfan (Advantage)	-	7	-	-	-	-
Chlorpyrifos	-	2	-	-	-	-
Demeton (Systox)	2	5	7	7	2	2
Dialifor (Torak)	-	-	-	75	-	-
Diazinon	-	5	5	5	-	-
Dicrotophos (Bidrin)	-	-	-	-	-	2
Dimecron (Phosphamidon)	-	14	-	-	2	2
Dimethoate (Cygon)	-	4	-	4	-	-
Dioxathion (Delnav)	-	30	30	30	1	1
Disulfoton	2	-	-	-	2	2
Endosulfan (Thiodan)	2	2	2	2	2	2
EPN	-	14	14	14	14	2(e)
Endrin	-	-	-	-	-	2
Ethion	2	30	14	14	2	2
Imidan	2	2	5	5	2	2
Malathion	-	1	1	1	-	-
Metasystox (R)	2	2	2	2	2	2
Methidathion (Supracide)	-	30	2	-	2	2
Methiocarb (Mesurol)	-	-	7	-	-	-

	Corn	Citrus	Peaches & Nectarines	Grapes	Apples	All Other Crops
Methomyl (Lannate, Nudrin)	2	2	2	2	2	1
Mevinphos (Phosdrin)	2	4	4	4	2	2
Monocrotophos (Azodrin)	-	2	-	-	-	2
Naled (Dibrom)	-	1	1	1	-	-
Oxamyl (Vydate)	-	2	2	-	2	1
Parathion-ethyl	-	30(a) 45(b) 60(c)	21	21	14	2(e)
Parathion-methyl	2	2	21	14(d) 21(f)	14	2(e)
Phorate (Thimet)	2	-	-	-	-	2
Phosalone (Zolone)	-	7	7	7	-	-
Propargite (Omite)	-	-	-	7	-	-
Sulfur	-	1	1	1	-	-
TEPP	-	4	4	-	2	2

Footnotes:

(a) For all applications with spray mixtures containing 2 pounds or less of actual parathion per 100 gallons, with rates of 8 pounds or less actual parathion per acre, and a total of no more than 10 pounds per acre in the previous 12 months.

(b) For all applications with spray mixtures containing 2 pounds or less of actual parathion per 100 gallons, with rates of more than 8 pounds actual parathion per acre, or more than 10 pounds per acre in the previous 12 months.

(c) For all applications with spray mixtures containing more than 2 pounds of actual parathion per 100 gallons.

(d) The reentry interval for methyl parathion on grapes in Monterey County is six days.

Update to Table 47.1 From:
Moses, M. Pesticides. -10-
Chapter 47 In: Rom, William. (Editor)
Environmental & Occupational Medicine
1983 Little-Brown

SCHEDULE 32.

(Reg. 123A (1).)

HAZARDOUS PESTICIDES.

PART 1.—*Organic Phosphate.*

azinphos-ethyl
azinphos-methyl
carbophenothion
chlorfenvinphos
coumaphos
demeton-O
demeton-O-methyl
demeton-S
demeton-S-methyl
demeton-S-methyl sulphone
dialifos
dichlorvos
dicrotophos
dimefox
1,3-di(methoxy carbonyl)-1-propen-2-yl dimethyl phosphate
dioxathion
disulfoton
ethion
ethoprophos
famphur
fenamiphos
fenitrothion
fensulfothion
fenthion-ethyl
hexa-ethyl tetraphosphate
isocarbophos
leptophos
mecarbam
methamidophos
methidathion
mevinphos
mipafox
monocrotophos
naphthalophos
omethoate
parathion
parathion-methyl
phenkapton
phorate
phosfolan
phosphamidon
schradan

sulfotep
TEPP
terbufos

PART 2.—*Carbamate.*

aldicarb
aminocarb
bendiocarb
carbofuran
formetanate
methomyl
promecarb.
propoxur (except in aerosol packs of less than 1 kilogram net contents)
thiofanox

PART 3.—*Chlorinated Hydrocarbon.*

aldrin
camphechlor
chlordane
dieldrin
endosulfan
endrin
heptachlor
mirex
pentachlorphenol
sodium pentachlorphenate

PART 4.—*Miscellaneous.*

acrolein
arsenic, inorganic compounds thereof
chlordimeform
5-chloro-3-methyl-4-nitropyrazole
decamethrin
dinitro ortho cresol
dinoseb
diquat
ethylene dibromide
nicotine sulphate
Oxamyl
paraquat
phenyl mercuric acetate
phenyl mercuric chloride

Appendix 6

DEPARTMENT OF INDUSTRIAL RELATIONS
DIVISION OF OCCUPATIONAL HEALTH
REQUEST FOR PESTICIDE AND WEEDICIDE ANALYSIS IN BLOOD AND URINE

PLEASE ENTER ALL DETAILS BELOW

Postal Address for return of report .....
.....
.....
Post Code.....

Forward specimens to:

DIVISION OF OCCUPATIONAL AND ENVIRONMENTAL HEALTH
Agricultural Laboratory - Room 119
Joseph Street, LIDCOMBE 2141
Telephone (02) 6460344 or 6460222

Patients Name .... Date of Birth.....
Surname *Christian Name*

Address ..... Post Code .....

Occupation ..... Employer .....

Clinical Symptoms .....

PLEASE SEE OVER FOR THE TYPE OF TEST REQUIRED, AND COLLECTION PROCEDURE

REQUEST Please tick

- ☐ 1. Organophosphates & Carbamates (Cholinesterase activity)
- ☐ 2. Organochlorines
- ☐ 3. Weedicides
- ☐ 4. Miscellaneous

Date of last exposure ..... Date specimen collected .....

Name & Address of Patient's Doctor .....

..... Telephone .....

I HEREBY CONSENT TO MY EMPLOYER BEING INFORMED OF THE RESULTS OF THE ABOVE TESTS

Signed ..... (Patient)

RESULT (OFFICIAL USE ONLY)

				REMARKS
Organophosphates & Carbamates (Cholinesterase activity)	Whole Blood	N.R.	80-120% Normal	.....
	Plasma	"	0.64-1.03 Δ Ph/hr	.....
	Red Blood Cells	"	0.42-1.22 Δ Ph/hr	.....
Organochlorines in blood (Parts per billion) = ppb	Hexachlorobenzene	200	M.R.L.	.....
	DDT	100	"	.....
	Dieldrin	50	"	.....
	Heptachlorepoxide	20	"	.....
	.....			.....
Weedicides in urine (ppb)	Pentachlorphenol	100	M.R.L.	
	2,4-D	100	"	
	2,4,5-T	100	"	
Miscellaneous	.....			

ABBREVIATIONS

N.D. = Not detected
N.R. = Normal range
M.R.L. = Maximum recommended level

☐ Results satisfactory Signed .....

☐ Results unsatisfactory Signed ..... (Medical Officer)

RECOMMENDATIONS Results indicate overexposure to.....Remove from exposure until results reach acceptable limits. A further blood/urine specimen is required

cont.

COLLECTION PROCEDURE

a) ALL specimens should be labelled, cooled (cooler bricks) and forwarded without delay to this Division.

b) Glass bottles with foil lined lids available from this Division.

c) This Division can provide medical advice on the treatment of poisonings.

PLEASE TICK PESTICIDES USED

1) ORGANOPHOSPHATES AND CARBAMATES (Cholinesterase activity)

Ten (10) mL of heparinized WHOLE BLOOD. Some common examples of these pesticides are listed below

ORGANOPHOSPHATE PESTICIDES

Common Name	Trade Name
☐ Azinphos	Gusathion
☐ Demeton-S-Methyl	Metasystox
☐ Diazinon	Basudin
☐ Dichlorvos (DDVP)	Nuvan
☐ Dimethoate	Rogor
☐ Fenitrothion	Folithion
☐ Fenthion-ethyl	Lucijet
☐ Maldison	Malathion
☐ Mevinphos	Phosdrin
☐ Monocrotophos	Azodrin
☐ Napthalophos	Rametin
☐ Parathion	Folidol

CARBAMATE PESTICIDES

Common Name	Trade Name
☐ Bendiocarb	Ficam
☐ Carbaryl	Sevin
☐ Methomyl	Lannate
☐ Propoxur	Baygon

Symptoms of Organophosphate and Carbamate poisonings usually occur within 12 hours of contact

2) ORGANOCHLORINE PESTICIDES

Ten (10) mL of heparinized WHOLE BLOOD Collected in an approved plastic syringe (STERIMED) and placed in a glass bottle with an aluminium foil lined lid. The blood must not come in contact with plastic or rubber. Some common examples are listed below

☐ Chlordane
☐ D.D.T.
☐ Dieldrin
☐ Aldrin
☐ Heptachlor

3) WEEDICIDES

Ten (10) mL of URINE collected as follows: Urine specimens must be collected in a glass bottle with an aluminium foil lined lid. The urine must not come in contact with plastic or rubber. Care must be taken to prevent contamination from hands and clothing during collection. Some common examples of weedicides are listed below.

☐ 2,4-D
☐ 2,4,5-T
☐ Pentachlorophenol
☐ Picloram

4) MISCELLANEOUS

Occupational exposure to compounds containing: Arsenic, Cadmium, Chromium, Mercury, Thallium.
Urine Specimen: Please contact this laboratory as each of the above has a specific requirement for testing.

5) OTHERS: Please specify:.....

S.O. 4295 D. West, Government Prin ...

Appendix 7

From: G.M. Cunningham (et al.)
Plants of Western New South Wales
Publ. NSW Govt. Printing Office, 1981.

NOOGOORA BURR

**Xanthium occidentale* Bertol. = (X. pungens) Asteraceae

Clotbur, cockleburr, large cockleburr.

Erect coarse annual forb, to 2 m or more high, the stems rough-hairy and often purplish-tinged. *Leaves* alternate, large, ovate-triangular, heart-shaped at the base, to 15 cm wide, dissected into 3 or 5 large lobes, darker green on the upper surface, the margins coarsely toothed, the stalks long and often purplish. *Flowerheads* inconspicuous, borne in the leaf axils, male and female heads separate, the male heads globular, the female heads ovoid, both subtended by bracts, the upper bracts of the female heads developing into a spiny burr. *Fruit* an oblong woody dark-brown burr, 16–18 mm long, covered with numerous hooked spines and bearing 2 longer stout and almost straight spines at the tip; seeds 2 in each burr, one larger than the other. *Flowering* summer-autumn.

Similar to californian burr *(Xanthium orientale)*; see that species for differences.

HABITAT: Low-lying areas subject to occasional inundation, on a wide range of soils; most frequent in black box, river red gum, coolibah and bimble box communities but also occurs in many others.

DISTRIBUTION: Throughout the region; the frequency and abundance of this weed in southern districts has increased markedly during the past 15 years. (N,Q,S,W;N.Amer.)

NOTES: Very dense and extensive stands of noogoora burr occur along river and creek floodplains and flats after late-spring or summer flooding, with masses of seedlings establishing and crowding out all other species of plants. Elsewhere, occurrences may be reduced to isolated plants or to small colonies along channel banks or roadsides. At maturity the burrs readily attach to the coats and tails of passing animals and are widely dispersed in this manner. The burrs can cause considerable matting of wool, thereby lowering fleece values and causing discomfort to the animals. Dispersal is also assisted by water as the plants grow right to the edges of streams and can tolerate shallow flooding for long periods. Noogoora burr is grazed by stock only when no other feed is available. Grazed in quantity it can poison stock, the seedlings being much more toxic than more mature plants. Sheep, cattle and pigs are known to be affected, the symptoms including nervousness, trembling, excitement and a trembling gait. Death can be quite rapid and, according to McBarron (1978), painful. However, despite the undoubted toxic properties of the plant cases of poisoning in this region have not been reported. It would, however, appear prudent to keep stock out of paddocks in which forage is otherwise scarce and major infestations of noogoora burr are known to be present; this particularly applies at the time germination is likely to occur as the cotyledons (first leaves) are very toxic.

In addition to its effect on grazing animals noogoora burr is also reported to reduce egg production in poultry presented with feed contaminated with parts of the plant. It can also cause skin irritation to people who handle it. Infestations can be controlled by hand-pulling of plants before seeding, by chemical spraying or by cultivation when young. Eradication, however, is often a long-term project and may not be achieved for several years as the burrs each contain 2 seeds, one of which may not germinate for several seasons after the first seed has germinated.

Noogoora burr is a proclaimed noxious weed in all Australian states.

**Xanthium spinosum* L. Asteraceae

Spiny cocklebur, common cocklebur, spiny clotbur.

Much-branched, erect, often bushy annual forb, to 1 m or more in height and diameter, with round stout and often yellowish stems. *Leaves* stalked, lanceolate, entire or 3- to 5-lobed, shiny dark-green above with a light-green midrib, dull whitish-green below, with one or two 3-branched yellow spines at the base of each stalk. *Flowerheads* inconspicuous, the male flowers borne in spikes at the ends of the branches, the female flowers borne in the leaf axils. *Fruit* an ovoid-oblong burr, 10–12 mm long, covered by numerous yellow slender hooked spines and 2 small straight spines at the top, each burr containing 2 seeds. *Flowering* summer-autumn.

HABITAT: Natural pastures generally, disturbed or flooded soils along roadsides, channel banks, dry creek channels; on most soil types and in most plant communities.

DISTRIBUTION: Throughout the region. (A,T;S.Amer.)

NOTES: Bathurst burr is one of the best known weeds of the region and prolific stands may occur after summer rains or flooding. Growth is rapid, and flowering and seeding continue from mid-summer until the plants die in the winter months. The hooked spines on the burrs cling to the coats and tails of animals and the burrs are mostly spread in this way, although some are spread by water. Of the two seeds contained in each burr usually only one germinates in a particular season, the other seed remaining dormant until the second or third year, with some seeds retaining viability for considerably longer. Stock rarely eat bathurst burr (deterred no doubt by the sharp spines of the plant if for no other reason) and if forced to do so can develop gastroenteritis and diarrhoea. McBarron (1976) cites reports of loss of egg production and early moult in poultry provided with feed contaminated with bathurst burr material. Dense stands of the weed inhibit growth of more desirable species and may prevent stock movement to some degree, but its main nuisance value lies in the burrs, which can be a major contaminant of wool. The spines borne along the stems can also become attached to the fleeces of sheep and cause problems at shearing. In some people, contact with the plant can cause a skin rash.

Control of bathurst burr can be achieved by timely cultivation, by hand hoeing when the plants are young, or by spraying with appropriate herbicides. In particular seasons a proportion of some populations of bathurst burr plants may die following defoliation by a caterpillar. A fungus affecting seed production of the weed has also shown some promise as a means of biological control, but occurrences of the fungus appear to be too infrequent to be of much consequence. Bathurst burr is a proclaimed noxious weed in all states of Australia. P 728

FIERCE THORNAPPLE

**Datura ferox* L. Solanaceae

Long-spined thornapple, long-spurred thornapple, false castor oil.

Stout bushy annual forb, to 1 m high, the branches repeatedly forked and green or purplish towards the base, the whole plant moderately hairy except for the more mature parts, on which the hairs may be sparse or absent. *Leaves* with an unpleasant odour when crushed, alternate, on long slender stalks, broad-ovate to rounded-triangular in outline, mostly to 13 cm long and 7–10 cm wide, the margins variously toothed or lobed. *Flowers* white, borne singly on short stalks in the forks of the branches, trumpet-shaped when fully open, to 6 cm long, 5-lobed, each lobe ending in a fine point. *Fruit* an erect capsule about 3 cm long and as wide, encircled at base by a stiff apron, beset with 40–60 stout conical spines (Fig. 59a), at first green, becoming brown when mature and splitting into 4 uniform parts; seeds black or grey, 4–5 mm long, tapered towards the base. *Flowering* late spring-autumn.

HABITAT: Disturbed ground, waste areas, roadsides and alluvial flats, usually near areas of habitation.

DISTRIBUTION: Widespread, but not recorded for the far north-west of the region. (A,T;China)

NOTES: A vigorous summer-growing plant which is a troublesome weed of summer crops in regions of higher rainfall. Although regarded as toxic, no cases of livestock poisoning specifically due to this species have been recorded in Australian literature (Everist, 1974). It is apparently unacceptable to stock and is not known to be grazed, but caution should be exercised when confining stock to areas in which this plant is common as all thornapples contain alkaloids which are potentially toxic. Proclaimed a noxious weed for part or all of the mainland

Noogoora burr = *Xanthium pungens*

From: Everest L. Selwyn. Poisonous Plants of Australia, Sydney, Angus & Robertson, 1981.

Xanthium pungens Widder: NOOGOORA BURR OR CAT'S EGGS (Plate 14). Some authors include this species under *X. chinense* Mill. It is included by some botanists under one variable species, *X. strumarium* L., which occurs in Europe and North America.

Description: Annual herb with coarse, upright, branched stems; leaves alternate, rough to the touch, dull green, 3–5 lobed and irregularly toothed, 5–15 cm long and almost as wide, abruptly narrowed at the base into a stiff stalk; male flowers in small globular heads near the top of short stalks in the forks of the upper leaves; female flowers in clusters in the forks of the leaves, developing into the characteristic burrs; mature burrs oval to oblong in shape, 2–2.5 cm long, brown, covered with hooked spines and with two rigid straight spines at the apex; each burr containing two seeds (achenes); seedling plants at first consisting of two narrow, thick, smooth, tender, dull-green seed-leaves (cotyledons) spreading out from the top of a soft, sappy stalk, cotyledons about 2.5 cm long, growing rapidly to about 5 cm, 6–12 mm wide, blunt at the tip, broadest below the middle and tapering to a thick, flattened stalk at the base; true leaves emerging from between the cotyledons, thinner in texture and covered with short bristly hairs; edges toothed.

Distribution and Habitat: Probably native to North America and the West Indies. It is widespread in Queensland and northern New South Wales and in parts of the Northern Territory, rare in South Australia. It grows most abundantly on creek banks and flooded flats, but commonly occurs also as a weed of summer fallows and row crops.

Poisonous Principle: American workers (Cole et al. 1980) identified the toxic principle in *Xanthium* spp. as *carboxyatractyloside* and showed that the pure compound produced identical clinical signs and lesions in pigs. Earlier American workers (Egley and Rogers 1959) postulated that the toxic compound has a primary amine group and an aromatic ring but the Australian workers (Sutherland 1978) found no evidence to support this hypothesis.

Sesquiterpene lactones were reported from *X. pungens* (*X. chinense*) and related species but there was no indication as to whether or not these compounds were toxic. The major component was *xanthumin* in *X. pungens*, *X. californicum* and *X. cavinillesii* and *xanthimin* in *X. italicum* (McMillan 1973, 1974, 1975).

Earlier claims that the toxic principle is a glycoside (*xanthostrumarin*) or the phenol *hydroquinone* (see Hurst 1942; Kingsbury 1964; Cole et al. 1980) have not been confirmed. No hydroquinone could be detected in either *X. pungens* from Queensland or a sample of *X. californicum* from the U.S.A. (McGrav 1971; Sutherland 1978).

Conditions of Poisoning: In the field, most cases occur in cattle or pigs which have access to young seedlings, particularly in spring and early summer when there has been sufficient rain to germinate the burrs but not enough to produce useful grass (Kenny et al. 1950). [illegible] and seeds extracted from burrs (Hall 1976). Small quantities of toxin have also been isolated from leaves of advanced seedlings and mature plants (Sutherland 1978) but no field cases have been reported from ingestion of either intact burrs or mature leaves.

Toxicity, Symptoms and Lesions: In the field, animals frequently die overnight without symptoms being observed. In feeding tests (Seddon and King 1938) the cotyledons were toxic to pigs, cattle and sheep but the true leaves were not poisonous. The lethal dose for pigs was about 2% of body weight, for calves 1·8% and for sheep considerably higher. In feeding tests with hammer-milled burrs (Hall 1964b) two sheep died less than 18 hours after drenching with a watery suspension of 225 g and two calves died in 21 and 60 hours respectively after drenching with a watery suspension of 450 g of hammer-milled burrs.

Affected animals usually show weak and rapid pulse and symptoms of acute gastro-intestinal pain; they often die in convulsions.

At autopsy there is usually accumulation of clear, straw-coloured fluid in abdominal and chest cavities and excess fluid in the pericardial cavity. Frequently there are subendocardial haemorrhages of the left heart, gelatinous or fibrinous material in the abdominal cavity and evidence of liver and/or kidney damage (Kenny et al. 1950).

Histopathological changes are chiefly in the liver, mainly necrosis and congestion. Sometimes there is fatty degeneration in the myocardium and slight patchy necrosis of uriniferous tubules in the kidney. The kidney lesions are vacuolation and loss of cytoplasmic contents of tubular cells with pyknosis of nuclei (Seawright 1978).

Three other species in the "*X. strumarium*" complex have been recorded as naturalized in Australia. These are *Xanthium californicum* Greene, CALIFORNIAN BURR (ascribed by some authors to *X. orientale* L.), *X. cavanillesii* Schouw. and *X. italicum* Mor. *X. italicum* and *X. cavanillesii* occur in eastern New South Wales, the latter restricted to a small area north-west of Sydney. Both grow with *X. pungens*. *X. californicum* is recorded only from eastern border regions of South Australia and adjacent areas in south-west New South Wales and north-west Victoria (McMillan 1975).

In feeding tests in Hungary (Horvath and Modor 1970), cotyledons of *X. italicum* produced typical "Noogoora burr poisoning" in young pigs.

Prevention and Treatment: Cattle and pigs should be kept out of paddocks where large amounts of Noogoora burr have germinated and other feed is scarce. Administration of fatty substances such as milk, lard or vegetable oils to animals which have eaten the seedlings may prevent poisoning (Kingsbury 1964).

The plant itself is easily killed by spraying with 2,4-D or MCPA in water at the rate of 1 kg per 10,000 m². Seed dormancy makes it necessary to treat infested areas for two successive seasons after every year in which the plant is allowed to produce ripe burrs.

Appendix 7 (cont)

Datura: 627

A cosmopolitan genus, mainly American in origin but containing some species native to Asia and one native to Australia.

There are two fairly well-marked groups:

(a) shrubby species which some botanists maintain in the separate genus *Brugmansia*
(b) herbaceous species belonging to the genus *Datura* in the restricted sense.

One native and 5 naturalized species of herbaceous *Datura* have been recorded from Australia (Haegi 1976).

Poisonous Principle: All species have similar poisonous principles. These are tropane alkaloids, mainly *scopolamine* (*hyoscine*) and *hyoscyamine*. At least 8 other related alkaloids have been reported, usually as minor components, the most common being *meteloidine*, *atropine*, *norscopolamine* and *norhyoscine*. Differences amongst species are chiefly in the relative concentration of these alkaloids. They also vary in amount and concentration in different parts of individual plants and are influenced by stage of growth and environmental conditions (see Evans and Wellendorf 1959; Evans et al. 1965; Kingsbury 1964; Schultes and Hofmann 1973; Schultes 1976).

Toxicity, Symptoms and Lesions: Humans appear to be most susceptible to poisoning by these plants but cases have been recorded in cattle, horses, sheep, pigs, dogs, fowls, mules and ostriches. Seeds are generally regarded as the most poisonous parts of the plants. Decoctions of leaves of some species, particularly *D. stramonium*, have been used for relief of asthma and this and other preparations have also been used as hallucinogenic drugs (see Schultes and Hofmann 1973; Schultes 1976).

In humans, symptoms vary according to the concentration of different alkaloids in the particular sample and to the reaction of individual patients (Kingsbury 1964). When decoctions of the plant are taken, symptoms may appear within a few minutes but onset of symptoms may be delayed for several hours if the leaves or seeds are eaten.

At first, there is intense thirst and dilatation of pupils, followed by flushing of the skin and extreme irritability. Later, there is delirium and a tendency to pick at imaginary objects. Sometimes there is elevation of temperature and weak, rapid pulse. Some patients become violent. Excessive consumption of water and failure of the parasympathetic nervous system usually result in distension of the bladder. Finally, there may be convulsions, followed by coma and death. In some people, coma follows shortly after the appearance of the first symptoms.

With sublethal doses or in cases where treatment is applied successfully, the symptoms may continue for 12–48 hours and then diminish but the pupils may remain dilated for up to two weeks.

Animals most commonly poisoned in the field are pigs and poultry, most cases being due to consumption of crushed grain containing *Datura* seeds. The feeding of whole grain rarely produces symptoms. Cattle and sheep are poisoned more rarely and most feeding tests with these animals have given negative results.

Symptoms are similar to those observed in humans and there are no characteristic lesions on post-mortem.

From: Everest L. Selwyn, op.cit.

Conditions of Poisoning: Overdoses of preparations used for medicinal purposes or as hallucinogenic drugs commonly cause poisoning in humans. Children are frequently poisoned by eating or sucking nectar from newly opened flowers, especially of the shrubby ornamental species. In some countries, cases of poisoning have been reported in people who have eaten flour made from grain contaminated with *Datura* seeds.

Poisoning of cattle, sheep and horses rarely occurs except when the animals are forced to eat large quantities, usually because of shortage of other feed. Pigs and poultry are sometimes poisoned by eating crushed contaminated grain.

Prevention and Treatment: Feeding to pigs or poultry of crushed grain contaminated with *Datura* should be avoided.

Distribution and Habitat: Native to South America, it is the most widely cultivated species of this group in Australia. Most of the records of *Datura arborea* and *D. suaveolens* in Australian literature apply to this species (Haegi 1976).

Poisonous Principle: From cultivated specimens of a peach-flowered form of this species, Griffin (1966) isolated 9 known tropane alkaloids and two unidentified bases. *Scopolamine* was the principal alkaloid in all parts of the plant and *meteloidine* was also a major alkaloid of leaf and stem.

Toxicity: Many cases of poisoning in humans are on record, chiefly in children who have eaten seeds or fragments of flowers. Consumption of portion of one flower has been known to produce non-fatal poisoning in a 4-year-old boy. Symptoms were delirium and hallucinations for about 36 hours, followed by sleep and eventually complete recovery (Queensland Herbarium Records 1957). In another case, a boy 8 years old ate the pith and bark from about 10 cm of the stem of this species. Shortly afterwards he developed hallucinations and exhibited intermittent periods of violence with a tendency to take off his clothes and run about. Hallucinations recurred 26 hours after development of the original symptoms but the boy recovered completely in about 3 days (Queensland Herbarium Records 1970).

(B) HERBACEOUS SPECIES (*Datura* in the restricted sense).

Datura ferox L.: LONG-SPINED THORNAPPLE, LONG-SPURRED THORNAPPLE, FIERCE THORNAPPLE OR FALSE CASTOR OIL (Plate 38).

Description: Annual herb: stems stout, erect, repeatedly forked, up to 1 m high but usually smaller and often flowering at a height of 30 cm or less: leaves soft, dark green, with an unpleasant odour when crushed, 5–15 cm long, with a slender, long stalk and a broad blade, narrowed at the tip to a blunt point; the margins irregularly toothed: flowers with a 5-lobed green calyx and a funnel-shaped, white corolla which spreads out at the end into 5 lobes, each lobe ending in a fine point: fruits erect in the forks of the branches, 4–5 cm long, oval in shape, covered with coarse prickles of irregular length: seeds dark grey, rough.

Distribution and Habitat: Native to southern Asia. It is naturalized in southern and central Queensland, New South Wales, Victoria, South Australia and Western Australia, mainly on waste land or in ploughed paddocks. In some areas it is a troublesome weed in summer and early winter crops and has been recorded as a rare weed in Tasmania.

Toxicity: Although the plant is regarded as toxic, no cases of livestock poisoning specifically due to this species have been recorded in Australian literature.

In feeding tests with ground fresh seeds of this species, concentrations equivalent to 10% and 20% of the whole ration significantly depressed growth rate of chickens but at concentrations of 5% and less there was no effect on the birds after 3 weeks of feeding (Springhall and Seawright 1972). *Hyoscine* was the only alkaloid found on analysis in amounts of 0·009%–0·024% in rations containing 2·5%–20% *Datura* seed meal.

In several tests with cattle, feeding of rations containing *Datura ferox* seeds, ground and unground, at rates of 2 kg per head per day for 3 days produced no toxic symptoms. Two different samples were used, one of pure *Datura* seed with a total alkaloid content of 0·07%, the other sorghum grain containing 60% *Datura* seeds. The total alkaloid content of the whole ration in the latter sample was also 0·07% (Hall 1972).

Appendix 7 (cont)

COTTON PLANTS.

From: Everest L. Selwyn, op.cit.

MALVACEAE

Gossypium: COTTON. Four species, *G. barbadense* L., SEA-ISLAND COTTON, *G. hirsutum* L., UPLAND COTTON, *G. arboreum* L. and *G. herbaceum* L. are the sources of most of the cultivated varieties of cotton. In Australia, virtually all the cotton grown commercially is derived from *G. hirsutum*.

Cotton seeds, and cottonseed meal which is obtained from the residue of crushed seeds after expression of the oil, can be toxic under some circumstances.

Poisonous Principles: The major toxic principle is *gossypol*, a polyphenolic pigment of somewhat unusual composition. It is contained in special glands that can be seen as small black dots in the seeds. The amount of gossypol present in cottonseed meal depends to some extent upon the degree to which these glands are ruptured during processing or storage and the degree of subsequent inactivation of gossypol (Kingsbury 1964).

The toxic principle responsible for the development of abnormalities in eggs during storage is *malvalic acid*, a cyclopropenoid fatty acid present in the seeds (Shenstone and Vickery 1959).

Toxicity, Symptoms and Lesions: Kingsbury (1964) discussed the toxicity of cotton seed and cottonseed meal at some length.

The raw seed can be toxic if eaten by livestock in sufficient quantity. If the seeds are eaten by laying hens, the eggs can deteriorate in storage, exhibiting pink discoloration of the whites and mottling and a putty-like consistency of the yolks. This condition is discussed in more detail under *Malva parviflora*.

The poisonous properties of the meal are determined mainly by the content of free gossypol but it is possible that shortage or imbalance of protein and/or vitamins in the whole ration may be contributing factors in reducing growth rates and producing symptoms noted in some cases.

The free gossypol content of cottonseed meal varies considerably with the variety of cotton involved, locality and seasonal conditions under which it is grown, the method by which the meal is prepared and the conditions under which it is stored.

Free gossypol is inactivated in time by reactions which occur spontaneously in the meal, converting it to non-toxic "bound gossypol". The amount of toxic gossypol present at any time depends upon the degree of rupture of the pigment glands and of subsequent reactions leading to destruction or "binding" of the gossypol.

No information on the free gossypol content of Australian grown and prepared cottonseed meals has been noted in the literature.

Most commercial milling procedures that leave a meal low in gossypol content (such as cooking at high temperatures prior to pressing) also cause a large reduction in the protein value of the meal. This appears to be due to a chemical reaction between free gossypol and lysine. Cottonseed meals of high protein content usually contain the greatest amounts of free gossypol unless they have been prepared by mechanical separation of the pigment glands prior to crushing.

Different species of animals differ in their susceptibility to gossypol poisoning. Pigs, rabbits and guinea pigs are most susceptible, poultry are somewhat less susceptible and ruminants such as cattle and sheep are most tolerant.

For pigs, cottonseed meals with a free gossypol content greater than 0·01% are potentially poisonous. Kingsbury (1964) noted that American cottonseed meals with the usual levels of gossypol (percentage not stated) could be added safely to pig rations up to about 9% but at this level the meal would provide insufficient protein to meet nutritional requirements when maize was the principal foodstuff. He also noted that there were some commercially produced cottonseed meals that could safely be added to pig feed in amounts up to 25% of the total ration.

In cattle and sheep, gossypol is partly detoxicated in the rumen but there is evidence that different breeds of dairy cattle differ in their capacity to detoxify this compound.

Poultry are intermediate in susceptibility between pigs and ruminants but amounts of gossypol less than those needed to produce death can cause depression of growth rate, lowered hatchability, discoloured yolks and whites or whites of abnormal consistency. According to Kingsbury (1964) the amount of free gossypol in the diet of poultry should not exceed 0·01%. Symptoms are similar in pigs and in ruminants. They usually appear abruptly after animals have been on cottonseed meal for periods varying from 4 weeks to about 12 months. Death usually follows 2–6 days after the appearance of symptoms but occasionally animals may linger for about a month. The most prominent symptoms are gasping, difficult breathing and occasionally frothing at the mouth, sometimes with blood staining. There may be loss of weight, emaciation and weakness without loss of appetite. Sometimes convulsions occur immediately prior to death. Immediately after death mucous membranes may show bluish dis-

At autopsy there is evidence of progressive heart failure—widespread congestion and oedema, large quantities of straw-coloured or blood-stained fluid in the body cavities, congestion and swelling of lungs, kidneys, mesenteric lymph glands and other organs. Sometimes there is ulceration and irritation in the gastro-intestinal tract. The liver is congested and shows evidence of degenerative changes. Areas of skeletal muscle appear white and the heart is flabby and oedematous.

Pathological examination of the liver shows disappearance of most parenchymal cells, leaving a thin zone of intact cells around the periphery of each lobule surrounding a blood-filled cavity. The heart shows areas of necrosis.

Kingsbury (1964) noted that gossypol produces death by preventing the release of oxygen from haemoglobin, thus reducing the oxygen-carrying capacity of the blood.

Prevention and Treatment: In rations being fed to pigs or poultry, the amount of cottonseed meal should not exceed about 9% of the total ration unless meals of low gossypol content are used. Higher percentages can be tolerated by sheep and cattle. The use of cottonseed meal for supplementary feeding presents little hazard of toxicity.

Kingsbury (1964) noted that the feeding of iron salts is said to protect animals against gossypol poisoning and to prevent abnormalities in stored eggs due to the effects of malvalic acid.

Xanthium spinosum L.: BATHURST BURR.

The cotyledons are also reputed to be toxic in the same way as those of Noogoora burr but there is little field evidence of poisoning by this species.

The plant is a low-growing, much-branched annual herb with narrowly 3-lobed leaves which are bright green on the upper surface and very pale underneath: at the base of each leaf-stalk there is a pair of conspicuous, bright yellow, 3-branched spines: the burrs are small, yellowish in colour and with numerous short, slender, hooked spines over the body of the burr as well as two straight spines at the apex.

It is widely distributed in Queensland, New South Wales, Victoria, South Australia, Northern Territory and Western Australia and generally comes up on disturbed ground. It can grow at almost any time of the year. It is believed to be native to South America.

Appendix 7 (cont)

From:

G.M. Cunningham (et al.) op.cit.

Weeds in a N.S.W. cotton field.

noogoora burr

bathurst burr

fierce thornapple

Appendix 8

MOREE CHAMPION
Vol. 17. No. 36 — Thursday, May 4, 1984 — 25c TELEPHONE 52 1455 — P.O. Box 397 — Telex AA66649 Registered by Australia Post - publication No. NAC1173

Woman, caught in cotton picker, flown to Sydney

A 45-year-old Collarenebri woman, caught by one leg in the picking head of a cotton picker at 9.30am yesterday, was flown to Sydney later in the day for treatment at St Vincent's Hospital.

She is Mrs Beryl Hicks, who was working on a picker operator for Colly Farms Pty Ltd on its Collymongle Station.

Sources in the area said Mrs Hicks was seeking to clear a blockage from the picker head when one leg became caught in the mechanism.

Dr Mahoney, of Collarenebri, attended the case and later called in Moree surgeon Dr Peter Whitnall, in case urgent surgery was needed on the spot.

Dr Whitnall flew to the scene of the accident.

Mrs Hicks spent four hours trapped in the machine, after which she was flown direct from Collymongle to Sydney.

Dr Whitnall said yesterday that Mrs Hicks suffered multiple penetrating-type wounds, other abrasions and injuries commensurate with crushing to one leg.

The other leg, he said, had been severely twisted.

The machine's picking head was virtually pulled apart by cutting it, after which it was dragged open by manual efforts and tractors pulling from either side.

Dr Whitnall added that word from St Vincent's last yesterday afternoon was that Mrs Hicks condition was satisfactory — but he felt she might have a lengthy convalescence.

HEALTH COMPLAINTS/COTTON WORK-EXPERIENCE

NAME AGE SEX

Appendix 9

Survey conducted in May 1984

1. HAVE YOU WORKED WITH COTTON? YES NO

- cotton chipping (yes / no)
- stick picking
- flagging planes
- cleaning ditches
- mixing sprays
- cotton picking
- cotton gin work

How many seasons (years)? 0 1 2 3 >3

Did you ever get sick when working or after work? yes no
(explain) _____

Have you ever been sprayed? (yes / no)

Have you ever worked in a field still wet from a spray? (yes / no)

Did you ever have a special blood or urine test for spray chemicals? (yes / no)

2. HAVE YOU EVER HAD TROUBLE

- with vision (yes / no)
- with giddiness (yes / no)
- with a blister or a rash (yes / no) explain _____
- itchyness (yes / no)
- asthma or wheeze (yes / no)

3. DO YOU SWIM IN THE RIVER? (yes / no)

do you get sick after swimming? (yes / no) explain _____

4. HAVE YOU HAD ANY BOILS THIS YEAR? (yes / no) where? _____

5. HAVE YOU SEEN A DOCTOR THIS YEAR? (yes / no) where? _____

6. WOULD YOU SUPPORT AN ABORIGINAL MEDICAL SERVICE IN MOREE? (yes / no)

OTHER COMMENTS? _____

Primary cause of 'Bundaberg disease' found

REFERENCE was made in the September issue of *Australian Fisheries* to an investigation into the so-called 'Bundaberg fish disease' by veterinary pathologists of the Queensland Animal Research Institute.

This disease was first reported in 1972 from the Burnett River and is characterised by the presence of large unsightly sores upon the skin of many commercial and non-commercial estuarine fish. With each successive year, the apparent range of this disease has extended both north and south of the original outbreak.

A major problem besetting early investigations was that all the samples examined exhibited large, well progressed ulcers. Such ulcers typically yielded fungal and bacterial agents which were essentially secondary invaders. That is to say, they had not actually caused the original wound but merely invaded a small existing sore and subsequently expanded it to form the large ulcers characteristic of this disease.

John Burke and Les Rodgers of the Queensland Fisheries Service, who have been actively investigating this problem since 1972, managed last winter to catch some fish which had only just become infected and were exhibiting very early lesions.

Results from the examination of these fish indicate that the principal agent responsible for such disease outbreaks is a bacterium, *Vibrio anguillarum*, which has caused similar problems in other parts of the world.

The Queensland investigators are also looking at the possibility that, for some species, skin parasites may be causing sufficient damage to assist the bacterium to penetrate the skin and subsequently form a small sore.

It is known that conditions of reduced salinity and temperature, such as accompanie substantial rainfall, will favour the invasion and growth of the secondary invading agents. Under these river conditions a small sore may become a large ulcer very rapidly.

The study is being funded by a grant from the Commonwealth Government's Fishing Industry Research Trust Account.

Investigation of 'Bundaberg fish disease'

IN 1972, a disease occurred in estuarine fish in the Bundaberg area of Queensland and caused concern among local fishermen. A common finding in these fish was reported to be skin ulcers.

In the two subsequent years, fish with lesions similar to those of the so-called 'Bundaberg fish disease' were seen in other parts of south-east Queensland.

In 'Dermal Ulceration of Mullet *(Mugil cephalus)*' *(Aust. Veterinary J. Vol. 52, May 1976)*, R. A. McKenzie and W. T. K. Hall, of the Animal Research Institute, Queensland Department of Primary Industry, report the pathological findings in some diseased fish from these outbreaks.

In their summary they say: 'Phycomycotic granulomas are described in the skin of seven mullet from extensive outbreaks of dermal ulceration. The fungus involved was probably a member of the Family *Saprolegniaceae* . . . The primary cause of the disease is uncertain.'

Sending a copy of the paper to *Australian Fisheries*, Mr McKenzie, a veterinary pathologist, says: 'Since publishing this, I have examined several fish from the inland rivers of Queensland with a very similar disease.

'These were bony bream and golden perch. One golden perch was parasited by a *copepod* which is a possible initiator of these skin ulcers.

'I hope this information may be of some interest to you in the light of recurring interest in this syndrome among the fishermen on the coast and inland.'