

RECORDS OF FISH KILLS IN INLAND WATERS OF NSW & QUEENSLAND IN RELATION TO COTTON PESTICIDES

Gillian M. Napier¹, Peter G. Fairweather^{1*} & Anthony C. Scott²
CSIRO Land & Water

¹ Griffith Laboratory, Private Mail Bag 3 PO Griffith NSW 2680 Australia

² Canberra Laboratory, GPO Box 1666 Canberra ACT 2601 Australia

*Present Address: Deakin University, School of Aquatic Science and Natural Resource Management, PO Box 423 Warrnambool VIC 3280 Australia

ABSTRACT

Fish kills are highly visible events and media publicity of them is powerful in developing a perception that our rivers are highly degraded through the impact of pesticides or other human insults. Ecotoxicology data derived from laboratory experiments suggests that fish, compared with other aquatic species, are relatively sensitive to pesticides used extensively in cotton cultivation. For example, the organochlorine endosulfan yielded 96-hr LC50s ranging from about 0.1 µg/L for European carp to 0.2-2.4 µg/L for native species. However, there is anecdotal evidence that fish and other aquatic life are healthy in cotton tailwaters likely to be contaminated. We review database records of fish kills in northern NSW and southern Queensland kept by state governments, and report on fish kill frequencies in different river basins, their presumed or suspected causes, the species involved, and whether, and which, pesticides were detected in each incident. These databases showed that fish kills were reported more often from cotton-growing areas and during the cotton-growing season. More than half the 98 recorded fish kills were associated with pesticides by the investigators. However, many of the records gave only circumstantial evidence as to their cause, and only a few were actually investigated in detail. This makes it difficult to make a proper assessment of how pesticides may be implicated in fish kills or of the impact, more generally, that cotton pesticides are having on fish populations. We also present data on residues of endosulfan found in live fish in a lagoon near Wee Waa before and shortly after a fish kill. We conclude that simply measuring tissue concentrations of pesticide residues does not provide direct proof that a fish died from any pesticides detected. Overall we suggest that more careful scientific attention

needs to be paid to any fish kills occurring in irrigation areas before we can know the real extent of damage from pesticides and therefore what management is needed.

INTRODUCTION

Pesticides have been detected in many of our rivers that flow through cotton-growing regions (Whyte & Conlon, 1990; Preece *et al.* 1993; Cooper, 1994, 1995, 1996; Bowmer *et al.*, 1996). These measured concentrations were sufficiently high that we expect lethal effects upon fish populations (i.e. they exceed concentrations shown in laboratory toxicity tests to kill native and introduced fish, see Bowmer *et al.*, 1996 for review). Yet, reports of fish kills appear to be infrequent. Nonetheless catastrophic mortality of fish populations ("fish kills") or other vertebrates like birds is spectacular, will be widely covered by the media and can arouse public debate. This attention in turn brings intense pressure to bear on any industry associated with the presumed or suspected cause of a fish kill event. Opinion concerning such catastrophic mortality of fish is polarised between those people who contend that very few kills have occurred over the past 20 years and others who claim that kills are actually common but mostly go unreported. Such lack of reporting might arise because of the sparsity of our rural population, the action of predators or flow to remove dead fish before they are noticed, small dead fish going undetected, some people not reporting them (for whatever reason), etc.

Some anecdotal evidence from farmers and others suggests that cotton production tailwaters, expected to contain pesticides, do not cause fish kills. Indeed, on-farm storages of recycled water and wetland depressions filled with runoff from cotton fields often appear "healthy" and "contain fish and other aquatic life".

In Australian laboratory experiments (Sunderam *et al.* 1992), European carp (96-hr LC50 = 0.1 µg/L), bony herring (0.2 µg/L), golden perch (0.5 µg/L), eastern rainbowfish and silver perch (both 2.4 µg/L) exhibit some of the greatest susceptibilities to endosulfan, the major cotton pesticide (Bowmer *et al.*, 1996). Yet no fish kills have been reported coincident with high concentrations of endosulfan during monitoring in NSW cotton areas done by the NSW Department of Land & Water Conservation. For example, during 1991/92 (Preece & Whalley, 1993), endosulfan concentrations reached 0.3 µg/L in Barwon River at Mungindi, 0.8 µg/L in Gil-Gil Creek at Galloway, 1.3 µg/L in Pran Creek at Rossmore and 3.0 µg/L in the Gwydir River at Brageen. Similarly, fish have survived in lagoons in the Wee Waa district (Napier, 1992) when endosulfan concentrations measured in the water were close to the lethal values determined in the laboratory.

In this paper we analyse the records of fish kills held by various state authorities. Such observations come from unpublished records and state registers of fish kills, although it is unlikely that all fish kills are recorded. We further seek to derive how many are linked, at least anecdotally, to pesticides or other causes, such as poor water quality from river management operations or natural reasons. As a case study we present some data related to pesticide levels in fish from northern NSW cotton-growing areas around the times of some observed fish kills. Objective and critical analyses alone can provide the understanding necessary to assess the threat that pesticides pose to fish populations relative to other agents of mortality.

REGISTER OF FISH KILLS

A number of fish kills in the cotton growing areas of New South Wales and Queensland have been attributed to cotton pesticides and in a few cases the evidence has been strong enough to lead to a prosecution in the Land & Environment Court of NSW (Nowak *et al.*, 1995; J. Chapman, pers. comm. 1995). A registry of 'Fish Kills in New South Wales' has

been compiled by NSW Fisheries, with incidents dating back to the mid-1970s. The information was collated from a number of sources such as the NSW EPA (formerly the State Pollution Control Commission), Department of Land & Water Conservation (formerly Department of Water Resources), local councils, NSW Fisheries and articles in local newspapers. All the reported fish kills for the Gwydir, Namoi, Macquarie and Macintyre River systems up to early 1995 were tabulated (Table 1, Fig. 1). A similar list has been compiled by the Queensland Department of Environment (formerly Department of Environment and Heritage) for fish kills in the Border Rivers and other inland Queensland rivers. These fish kill databases overlap on only two occasions for a single border river, the Barwon at Mungindi, on 2/1/1989 and 7/1/1991. The first occasion led to a prosecution of a farmer by NSW EPA for endosulfan in water discharged to the river

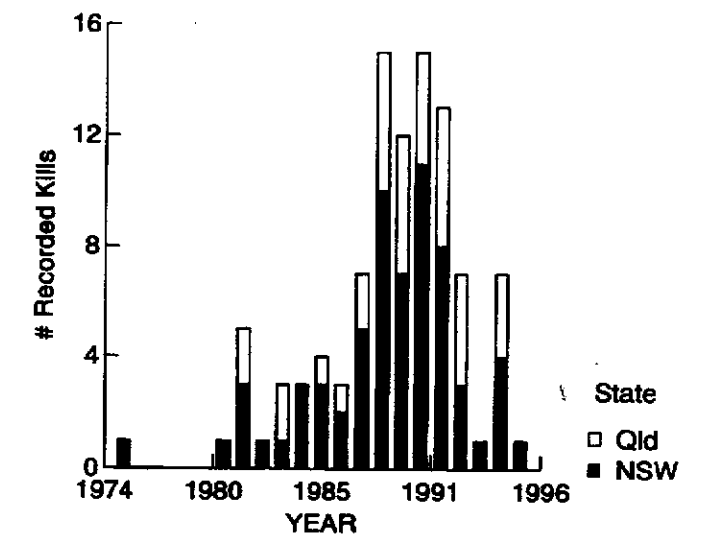


Figure 1: The frequency of occurrence of reported fish kills divided by state, from prior to 1980 to early 1995 in NSW and Queensland.

The records kept by these state authorities show a large number of reports from the late 1980s to the early 1990s (Figure 1) although the peak for Queensland lagged a few years behind NSW. Overall for the four river systems examined in each state, the NSW basins showed nearly twice as many records as those in Queensland

Table 1: Summary of database fish kills with their recorded (known or suspected) causes. DO stands for dissolved oxygen. * includes high temperature, ash from bushfires, infection after stress from snowmelt, angler discards, dam outlet being turned off, discharge from sewage works, "poor water quality" and "natural causes".

River	No. Kills	Pesticides		Low DO		Unknown		Other*
		No.	%	No.	%	No.	%	
NSW:								
Gwydir	14	7	50	1	7	6	43	0
Macquarie	27	9	33	1	4	14	52	5
Macintyre	12	4	33	1	8	6	50	1
Namoi	11	5	45	2	18	3	27	3
NSW sub-total	64	25	39	5	8	29	45	9
Queensland:								
Balonne/Condamine	14	10	71	3	21	1	7	1
Dawson	4	3	75	0	0	1	25	0
Macintyre/Barwon	16	15	94	1	6	0	0	0
Nogoa	0	-	-	-	-	-	-	-
Qld sub-total	34	28	82	4	9	2	6	1
TOTAL	98	53	54	9	8	31	32	10

(Table 1). Other rivers of NSW, with little or no irrigated cotton (e.g. the Darling below Bourke, Lachlan, Murrumbidgee and Murray Rivers), recorded another 50 fish kills (data not shown) and no other inland rivers of Queensland recorded any. The frequency of reported fish kills are associated with cotton-growing areas more strongly in Queensland than NSW (significant by χ^2 tests, $P < 0.001$). Of the 50 NSW kills in non-cotton areas, only 4% were associated with pesticides and both of these kills occurred on the Murrumbidgee River (which has extensive irrigation). Thus, cotton growing and pesticides as the likely cause were highly associated in these fish kill records (Fisher exact test, $P < 0.001$).

The timing of fish kills (Table 2) also suggests that kills are associated with cotton production. The frequency of recorded kills significantly increased over the cotton season (from approximately October through to March) more within the cotton areas than for non-cotton areas (by Fisher exact test, $P = 0.005$). Thus nearly 87% of reported kills from rivers with cotton production occurred during the six-

month cotton-growing season but only 66% did for non-cotton areas.

The records of which fish species were involved in kills are much more patchy; 58 of the NSW records and only one from Queensland included notes on fish identity. This difference may reflect the fact that in NSW the state fisheries department kept the record rather than an environmental agency (Qld). Of native species, golden perch and bony herring were the most often reported (Table 3). Of introduced fish, carp kills were the most common occurrence, reflecting the great abundance of carp in inland waters (Gehrke *et al.*, 1995) and perhaps their susceptibility to a cotton insecticide like endosulfan (Bowmer *et al.*, 1996). Large crustaceans were also observed as part of nine fish kills (Table 3). Unfortunately the sizes of the fish killed are not routinely recorded in these databases.

For each of the major rivers with cotton growing districts, a considerable proportion of the 98 reported fish kills have been attributed to pesticides: between 33% and 94% of reports implicated pesticides of some sort (Table 1). Queensland reports listed pesticides as a causative agent more

Table 2: Number of fish kills recorded per month for each river system. The six months shown in bold constitute the cotton-growing season in most years. Records of kills from non-cotton areas are from other inland rivers in NSW (see text).

Month	New South Wales				Queensland			Total	Non-Cotton Areas	
	Gwydir	Namoi	Macquarie	Macintyre	NSW Sub-Total	Condamine	Dawson			Barwon
January	6	3	6	4	19	6	1	4	30	15
February	3	2	1	2	8	3	1	2	14	5
March	2	2	0	0	4	2	1	1	8	1
April	0	0	1	2	3	0	0	1	4	4
May	0	0	0	0	0	0	0	0	0	3
June	0	0	0	0	0	1	0	0	1	1
July	0	0	1	0	1	0	0	0	1	2
August	1	0	3	1	5	0	0	0	5	4
September	0	0	1	1	2	0	0	0	2	3
October	1	1	2	0	4	1	0	0	5	7
November	0	1	2	0	3	0	1	3	7	1
December	1	2	10	2	15	1	0	5	21	4
Total	14	11	27	12	64	14	4	16	98	50

Table 3. The number of fish kill events ($n = 106$ records, all causes combined) where particular species of fish were recorded. # where fish species were noted in the record

River	No. Kills [#]	Native species						Exotic species				Other fauna		
		GP	BH	MC	CF	BF	SP	EC	T	GF	RF	Y	S	"fish"
Gwydir	13	4	5	1	0	0	0	4	0	0	0	2	2	3
Macquarie	26	6	1	3	3	1	0	13	6	2	2	1	0	1
Macintyre	10	7	5	4	1	0	1	6	0	0	1	1	1	0
Namoi	9	3	6	1	0	0	0	5	0	0	0	1	1	0
Dawson	1	0	1	0	1	0	0	0	0	0	0	0	0	0
TOTAL	59	20	18	9	5	1	1	28	6	2	3	5	4	4

Key:
 GP = Golden or Murray perch, *Macquaria ambigua*
 BH = Bony herring, *Nematolosa erebi*
 MC = Murray cod, *Maccullochella peelii*
 CF = Catfish, *Tandanus tandanus*
 BF = River blackfish, *Gadopsis marmoratus*
 SP = Spangled perch, *Leiopotheron unicolor*
 EC = European carp, *Cyprinus carpio*
 T = Trout, *Oncorhynchus mykiss* or *Salmo trutta*
 GF = Goldfish, *Carassius auratus*
 RF = Redfin, *Perca fluviatilis*
 Y = Yabbies, probably *Cherax destructor*
 S = Shrimp, probably *Macrobrachium* sp.

often (82%) than NSW (39%), yielding 53 records of fish kills with pesticide involvement. In contrast, only nine of the 50 kills (18%, data not shown here) recorded for other inland rivers of NSW were associated with pesticides (these proportions are significantly different by χ^2 test, $P = 0.015$).

The second highest potential cause was low concentrations of dissolved oxygen (DO) such as occurs upon release of deep water from stratified reservoirs or from eutrophic conditions. Many cases were either ascribed to unknown causes (about a third to a half of all cases) or attributed to a wide range of other water conditions, some natural and some not (Table 1).

The most often cited pesticide was endosulfan, which was mentioned in 78% of cases (Table 4). The next largest category (14%) was unspecified "pesticides", probably reflecting no chemical analysis of waters or fish. Reports of pesticide in water were made in about half those kills implicating pesticides (26/53 = 49%). Only five reports from NSW indicated that pesticides were found in fish samples (Table 5), although the incidence of sampling and analysis without any detection is not indicated. Solitary reports were made of pesticide in sediments or freshwater mussels (Table 5).

Runoff was the most common source of pesticide contamination in Queensland but not reported from NSW (Table 5). This reflects the different ways each state handles used irrigation water, with disposal to waterways in Queensland but not in NSW. Overall the NSW records were less explicit about the possible sources of contamination (Table 5). In each state about 15% of incidents were linked to storms or floods breaching any water retention practices that were in place.

Despite our difficulties in assessing the evidence, it is still clear that cotton pesticides are at least implicated in the majority of those fish kills that have been reported, and that endosulfan is the pesticide most often implicated (Tables 1 & 3). For a small number of incidents in NSW, the evidence has been strong enough to be supported in court and has led to prosecutions (J. Chapman, pers. comm.).

FISH KILL TISSUE ANALYSIS

The main problem associated with interpreting fish kill records is determining the exact cause of death. Although chemical analyses of water and fish tissue might confirm that pesticide residues are present, this does not necessarily prove that the pesticides detected actually caused the fish kill. Methods are being developed to determine whether the residues accumulated in fish tissue are caused by sub-lethal background levels or by a recent lethal exposure (Nowak *et al.*, 1995).

The organochlorine insecticide endosulfan occurs in two isomers, alpha- and beta-endosulfan and the parent compound is a mixture of these in the ratio beta:alpha of 3:7. The alpha isomer is slightly more water soluble than beta, and Peterson & Batley (1991) modelled more beta isomer being taken up than alpha by fish. Both isomers are degraded in soil, water and in the livers of fish to a toxic degradation product (endosulfan sulfate), and other non-toxic metabolites (such as endosulfan diol). The toxic fractions (i.e. alpha, beta and the sulphate) are referred to as "total endosulfan" for toxicological purposes. Examining residues of these different forms of endosulfan have proved useful overseas (e.g. Matthiessen *et al.* 1982) for determining risks to wildlife. Matthiessen *et al.* (1982) suggested that the ratio of sulfate to parent isomers (i.e. alpha plus beta) can indicate how recent was any exposure, increasing with time since exposure.

Beta-endosulfan hydrolyses at a faster rate than alpha-endosulfan in water (Peterson and Batley, 1993). There is evidence of preferential metabolism of the beta isomer in fish liver (Nowak & Julli, 1991; Nowak *et al.*, 1995), which suggests again that the beta isomer metabolises at a faster rate than the alpha isomer. Therefore, the observed ratio of beta:alpha endosulfan, both in water and in fish livers, is an indicator of how recently the contamination occurred. Data on the endosulfan residues in fish livers presented in Nowak *et al.* (1995) suggested that fish killed by recent exposure to endosulfan will have a higher beta:alpha ratio than those exposed to sublethal levels. Nowak *et al.* (1995) also proposed other lines of evidence which can

Table 6. Endosulfan concentrations in whole fish from four lagoons near Wee Waa, NSW, after Napier (1992). *Gambusia* = *Gambusia holbrooki*. Entries for each month are the range (top line) & mean (bottom line) for the fish concentrations or duplicate samples for the water concentrations. *n* = number of fish samples analysed for that species/lagoon/month. Month 1 = 9-12th December 1988 with no fish kills observed but traces of endosulfan present in the water. Month 2 = January 1989, including fish surviving a kill at Jabiru Lagoon, 7th January 1989. * indicates fish kill, aa = as above

Species	Lagoon	Month	n	Fish Size Range mm	Water			Endosulfan In: Fish			Fish Lipids %	
					total µg/L	alpha mg/kg	beta mg/kg	β:α ratio	sulfate mg/kg	total mg/kg		SO ₂ :(α+β) ratio
Carp	Jabiru	1	2	38-45	0.319	0.11-0.32	0.21-0.65	1.90-2.03	0.04-0.34	0.36-1.31	0.13-0.35	0.88-2.16
		2*	7	30-72	0.557	0.22	0.43	1.97	0.19	0.84	0.24	1.52
Cudgewa	Cudgewa	1	2	65-72	0.216	0.06-0.78	0.07-0.72	0.60-1.88	0.11-1.07	0.29-2.39	0.28-6.92	0.62-2.73
		2	1	80	0.361	0.29	0.31	1.21	0.70	1.30	2.12	1.58
Weetawaa	Weetawaa	1	3	60-65	0.021	0.0-0.07	0.0-0.15	0.2-1.14	0.0-0.05	0.0-0.27	0.0-0.23	3.32-4.41
		2	1	80	0.023	0.04	0.08	1.07	0.03	0.14	0.12	3.87
Lowana	Lowana	1	4	35-70	0.003	0.05	0.05	1.00	0.04	0.14	0.40	2.52
		2	3	42-60	0.003	0.10-1.96	0.02-1.68	0.23-.86	0.03-0.17	0.15-3.74	0.03-0.82	0.74-1.49
Gambusia	Jabiru	1	2	24-38	0.036	0.73	0.59	0.57	0.10	1.43	0.35	1.04
		2*	4	25-36	0.003	0.02-.09	0.04-.060	0.41-3.00	0.0-0.10	0.08-0.23	0.0-0.77	1.14-2.85
Weetawaa	Weetawaa	1	2	23-32	0.004	0.06	0.05	1.21	0.06	0.17	0.48	1.97
		2	2	23-32	0.003	0.0-0.12	0.0-0.05	0.0-0.07	0.02-0.05	0.05-0.17	0.43-0.63	1.44-1.8
Gambusia	Jabiru	1	2	24-38	0.003	0.05	0.02	0.02	0.03	0.10	0.52	1.63
		2*	4	25-36	aa	0.80-0.95	2.48-5.10	3.19-5.36	2.73-6.09	5.99-12.14	0.84-1.01	2.62-2.71
Weetawaa	Weetawaa	1	2	23-32	aa	0.86	3.79	4.28	4.41	9.07	0.92	2.66
		2	2	23-32	aa	0.10-0.88	0.10-1.02	1.00-2.42	1.04-9.64	1.24-11.53	1.59-5.13	1.97-4.44
Weetawaa	Weetawaa	1	2	23-32	0.39	0.62	0.62	1.69	3.77	4.78	3.62	3.25
		2	2	23-32	0.08	0.0-0.16	0.0-0.10	0.0-0.66	0.36-0.57	0.57-0.62	0.1-1.37	2.02-3.52
Weetawaa	Weetawaa	1	2	23-32	0.11	0.08	0.05	0.33	0.46	0.59	0.68	2.77
		2	2	23-32	0.11	0.08	0.05	0.33	0.46	0.59	0.68	2.77

help to confirm the cause of the fish kill:

- an endosulfan residue in the gills at a concentration of more than 0.5 mg/kg indicates that the fish probably died from endosulfan poisoning; and
- endosulfan residues much greater than those found in live fish from the same area (or an area of similar endosulfan usage) indicate that the fish probably died from endosulfan. For example (see Bowmer *et al.* 1996), gill tissue analyses done by the NSW EPA for fish kills at Warren, Millie Creek and Gravelly Creek in 1991 showed high concentration of total endosulfan (alpha plus beta plus sulfate) residues in gill tissue (0.88 to 12.7 mg/kg) and relatively high beta:alpha ratios in livers (0.8 to 7.2), indicating that these fish probably died of endosulfan poisoning. Residue data from livers presented in Nowak *et al.* (1995) could also be interpreted by some readers such that lethal doses could be indicated by beta:alpha ratios > 0.4 and that lower doses or less recent exposure result in lower ratios.

CASE STUDY: Jabiru Lagoon Fish Kill

It is useful to retrospectively examine some data collected after a known fish kill in Jabiru Lagoon near Wee Waa NSW (149°19'E:30°10'S) (reported by the landowner on 7/1/89) related to cotton growing. The sampling was conducted as part of a routine survey of biota in lagoons. This survey investigated the concentration of endosulfans in water and small whole fish that survived the kill. We ask the question, "is it possible to deduce that a fish kill occurred from these concentrations?"

For comparison, in December 1988, following cotton-field runoff after rain, Napier (1992) measured the total endosulfan concentration in duplicate water samples stored at 4°C between their collection and extraction on the same day. Water was extracted twice with 150 mL hexane:dichloromethane (1:1) in a 2 L separating funnel. The extracts were stored in 250 mL amber glass bottles with foil lined lids at 4°C. These stored samples were later concentrated to 5 mL using Kuderna-Danish evaporators (Chau & Lee

1982) and analysed using a Shimadzu GC-8A gas chromatograph with electron capture detector. The measured water concentration was above the 96-hour LC50 for carp (the main fish species in the lagoon) of 0.1 µg/L but there was no evidence of dead fish. Similar findings were obtained from monitoring in the NSW Central and North Western Water Quality Program (Preece *et al.* 1993; Cooper, 1994, 1995, 1996), where concentrations have often exceeded 0.1 µg/L without any evidence of fish kills.

On 7/1/89, there was a fish kill of carp in Jabiru Lagoon caused by overspray of an adjacent drainage channel. On 11/1/89 (i.e. four days after the fish kill), the total endosulfan concentration in the centre of the lagoon was still high at 0.3 µg/L. Dead, decomposing fish were seen but not collected. However, a large number of small fish (15-72 mm standard length) survived around the edges of the lagoon amongst dense vegetation (Table 6). There are many possible reasons for survival, including that this vegetation prevented complete mixing of contaminated water and hence provided safe havens (Bowmer *et al.* 1996).

Samples of these fish (carp and gambusia) were collected using a variety of nets & traps. Collected fish were frozen before analysis for endosulfan residues. Small specimens of similar size were grouped together and homogenised before weighing (approximately 3 g was required for analysis) and were prepared following the procedure outlined by Nowak & Julli (1991). The fish were too small to examine separately their different tissues such as livers or gills, so comparisons across different tissues should only be made with caution. Detection of endosulfan residues was performed on a Hewlett-Packard GC 5890A gas chromatograph with electron capture detector. These surviving fish showed a range of (whole fish) residues with total endosulfan concentrations between 0.29 and 11.54 mg/kg and beta:alpha ratios between 0.09 and 2.15 (Table 6). The ranges are comparable to values found from live fish sampled then and in the preceding December from three other lagoons that did not experience fish kills at these two times (Table 6). Earlier

River	Source:				Detected in:				
	Tailwaters	Runoff	Overspray, drift	Floods, storms	Water	Fish	Sediment	Mussels	
Gwydir	3	0	1	1	4	2	0	1	
Macquarie	1	0	0	0	1	1	0	0	
Macintyre	1	0	2	0	2	2	0	0	
Namoi	2	0	2	1	1	0	0	0	
Balonne/Condamine	0	6	4	3	7	0	0	0	
Dawson	0	1	2	1	0	0	0	0	
Macintyre/Barwon	10	10	5	3	11	0	1	0	
TOTAL	17	17	16	9	26	5	1	1	

Table 5. Sources of pesticides and where they were detected in recorded fish kill events.

Rivers	Endosulfan	Endrin	Chlorpyrifos	DDT	OP* & OC'	"pesticides"@
Gwydir	7	1	0	0	1	0
Macquarie	5	0	0	0	0	3
Macintyre	2	0	0	0	0	0
Namoi	2	0	1	0	0	1
Balonne/Condamine	8	0	0	1	0	1
Dawson	3	0	0	0	0	0
Macintyre/Barwon	13	0	0	0	0	2
TOTAL	40	1	1	1	1	7

* OP = unspecified organophosphates
OC = unspecified organochlorines
@ unspecified

Table 4. The incidence of different pesticides in recorded fish kills. No details were given of detection limits.

samples from Jabiru Lagoon also showed similar or more excessive ranges (Table 6). Interestingly, the samples taken after the fish kill show the highest SO₄:(α+β) ratios (Table 6) and these correspond to values that Matthiessen *et al.* (1982) found in fish viscera at the end of a spray season, indicating relatively advanced breakdown of the applied endosulfan in these whole fish. Unlike previous liver studies (Nowak *et al.* 1995), these whole fish concentrations showed beta:alpha ratios > 0.4 for both fish surviving the kill and *Gambusia* prior to it.

Thus live fish showed a similar range of (whole fish) endosulfan concentrations whether or not a fish kill had occurred. If these fish had then died from natural or other causes unrelated to pesticides but were analysed for endosulfan, then death may have been erroneously attributed to contamination with that pesticide. Unfortunately we do not know whether the fish that died during the kill had higher concentrations in their tissues. This requires collecting specimens immediately after a kill has occurred (O'Sullivan 1996).

DISCUSSION

Reliability of Fish Kill Observations

Several aspects of reliability need to be considered in relation to the link between pesticides and fish kills:

- the number of reported fish kills may under-estimate actual kills, simply because they may occur in remote locations, or are not officially reported, or because the fish may be rapidly removed by birds or other scavengers;
- there are other possible causes of fish kills, apart from pesticides (Nowak, 1996; O'Sullivan, 1996). These include extreme pH values of the water, oxygen depletion, ammonia, metals, pathogens and naturally occurring organic materials; and
- increased drainage of water to rivers may increase pesticide input coincidentally with some of the above factors as stagnant water is displaced from the landscape to river systems, resulting in the compounding effects of several causes.

Fish kills attributable to pesticides might be under-estimated through missing evidence such as not knowing of exposure to pesticides experienced by the fish due to the time lag between the fish kill and obtaining water samples for analysis.

The total number of fish kills officially recorded is probably substantially less than those that actually occur. As an example, Napier (1992) reported three fish kills during a four-week survey of the Wee Waa district in the 1988-89 season, and none of these incidents were recorded in the NSW Fisheries register of fish kills. Another example of how these incidents can go unanalysed is the massive fish kill (estimated as 10,000 large fish) that occurred on the Namoi River in April 1995, and was not reported for over a week, by which time the samples were too badly decomposed to analyse (J.C. Chapman, pers comm.).

One of the important factors in investigating fish kills is timeliness of sampling. Freshly dead or dying fish will give more information about recent exposures (especially from concentrations in their livers), and water quality results taken soon after the incident will provide a better picture of conditions at the time of the kill (O'Sullivan, 1996). Obtaining recent data is especially important if pesticides with short half-lives in water are implicated; the endosulfan isomers, for instance, have a half-life in water of only a few days (Peterson & Batley, 1993) versus approx. 2 weeks for the toxic sulfate metabolite. Testing for pesticides in sediments could give an indication of longer term exposure.

One of the difficulties in such an assessment of recorded fish kills is the quality of the observations. These vary considerably across: the experience and interest of the observer; how soon they are made after the fish kill; the resources available to the people doing the investigation; and the range of possible "suspects" regarding causes. For example, if pesticides or low DO are suspected but the dead fish (and their waters) cannot be reached for sampling purposes very quickly, the worth of such a scientific study becomes dubious (O'Sullivan, 1996). Consequently we have been reasonably liberal here in our acceptance of what are

listed in the record as either proven or suspected causes of kills (often no distinction is made). This bias means that our data about causes (Table 1) should be accepted as indicating the number of occasions that fish kills have been associated with particular potential causal agents. The standard of proof *per se* is unknown from these registers but is probably less than most scientific studies.

Therefore our results indicate that more effort should be put into investigating pesticides as causal agents of fish kills in inland rivers of NSW & Queensland. The large number of reported kills and that endosulfan is associated with many of them is cause enough for concern. The otherwise excellent "Protocol for Investigating and Reporting Fish Kills" of NSW Fisheries/NSW EPA does not explicitly request testing for pesticides in water or sediments. We believe that the results here mean such an emphasis is warranted.

ACKNOWLEDGMENTS

We thank: LWRRDC for funding, and Dr Kath Bowmer (now of Charles Sturt University) for leading the consultancy that initially began this analysis; Allan Lugg (NSW Fisheries) and Pauline Semple (Queensland Department of Environment) for access to the fish kill databases; and Richard Whyte, David Greenhalgh, John Chapman, Angela Arthington, Andrew Brooks and the audience at the 1995 LWRRDC cotton research workshop for comments on various stages of this study. Gary Jones, Barbara Nowak and two anonymous reviewers helpfully criticised earlier drafts of this manuscript. Thanks especially to NSW Fisheries and Queensland Department of Environment for keeping these databases.

REFERENCES

Bowmer, K.H., P.G. Fairweather, G.M. Napier, and A.C. Scott. (1996). *Biological Impacts of Cotton Pesticides* LWRRDC Occasional Paper No. 03/96, Land & Water Resources Research & Development Corporation, Canberra.

Chau, A.S.Y., and H.B. Lee. (1982). Basic principles and practices on the analysis of pesticides. Pages 27-81 in A.S.Y. Chau, B.K. Afghan, and J.W. Robinson, eds. *Analysis of Pesticides in Water*. CRC Press, Boca Raton, Florida

Cooper, B. (1994). *Central & North West Regions Water Quality Program, 1993/94 Report on Pesticide Monitoring*. NSW Department of Water Resources TS94.087, Parramatta.

Cooper, B. (1995). *Central & North West Regions Water Quality Program: 1994/95 Report on Pesticides Monitoring*. Water Quality Services Unit, Department of Land and Water Conservation, TS95.087, Parramatta.

Cooper, B. (1996). *Central & North West Regions Water Quality Program: 1995/96 Report on Pesticides Monitoring*. Water Quality Services Unit, NSW Department of Land and Water Conservation, TS96.048, Parramatta.

Gehrke, P.C., P. Brown, C.B. Schiller, D.B. Moffatt, and A.M. Bruce. (1995). River regulation and fish communities in the Murray-Darling River system, Australia. *Regulated Rivers: Research & Management* 11: 363-375.

Matthiessen, P., P.J. Fox, R.J. Douthwaite, and A.B. Wood. (1982). Accumulation of endosulfan residues in fish and their predators after aerial spraying for the control of tsetse fly in Botswana. *Pesticide Science* 13: 39-48.

Napier, G.M. (1992). *Application of Laboratory-derived Data to Natural Aquatic Ecosystems*. Unpublished PhD thesis, Macquarie University.

Nowak, B. (1996). Toxicants as causes of fish kills. Pages 31-34 in D. O'Sullivan, ed. *Fish Kills: Causes and investigation in Australia*. Key Centre for Aquaculture Workshop Series, Aquaculture Sourcebook 13, Turtle Press, Sandy Bay, Tasmania.

Nowak, B., and M. Julli. (1991). Residues of endosulfan in wild fish

from cotton growing areas in New South Wales. *Toxicology and Environmental Chemistry* 33:151-167.

Nowak, B., A. Goodsell, and M. Julli. (1995). Residues of endosulfan in carp as an indicator of exposure conditions. *Ecotoxicology* 4:363-371.

O'Sullivan, D. (ed.) (1996). *Fish Kills: Causes and investigation in Australia*. Key Centre for Aquaculture Workshop Series, Aquaculture Sourcebook 13, Turtle Press, Sandy Bay, Tasmania.

Peterson, S.M., and G.E. Batley. (1991). *Fate and transport of endosulfan and diuron in aquatic ecosystems*. Centre for Advanced Analytical Chemistry, Investigation Report CET/LH/IR013, CSIRO Division of Coal & Energy Technology.

Peterson, S.M., and G.E. Batley. (1993). Fate of endosulfan in aquatic ecosystems. *Environmental Pollution Series A* 82:143-152.

Preece, R., and P. Whalley. (1993). *Central & North West Regions Water Quality Program, 1991/92 Report on Pesticide Monitoring*. NSW Department of Water Resources TS 93.094, Parramatta.

Preece, R., P. Whalley, and B. Cooper. (1993). *Central & North West Regions Water Quality Program, 1992/93 Report on Pesticide Monitoring*. Water Quality Services Unit, NSW Department Water Resources, TS93.081, Parramatta.

Sunderam, R.I.M., D.M.H. Cheng, and G.B. Thompson. (1992). Toxicity of endosulfan to native and introduced fish in Australia. *Environmental Toxicology & Chemistry* 11: 1469-1476.

Whyte, R.J., and M.L. Conlon. (1990). *The NSW Cotton Industry and the Environment*. State Pollution Control Commission TR90/9, Sydney.