An Independent Review of the February 2004 Lower Darling River Fish Deaths: Guidelines for Future Release Effects on Lower Darling River Fish Populations.

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A report prepared for NSW Department of Infrastructure, Planning and Natural Resources by the Murray-Darling Freshwater Research Centre.

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Cover photograph

Murray cod killed in the Darling River near Karoola station during the February 2004 fish kill event, courtesy of Wayne and Arleta Smith.

SUMMARY

In February 2004 an extensive kill of Murray cod (*Maccullochella peelii peelii*) occurred on the Lower Darling River between Menindee and Pooncarie. The deaths, which stretched for over 160 km of river, involved predominantly large Murray cod. Landowner and newspaper estimates of numbers of dead Murray cod ranged from 2 dead fish per kilometre of river, to 30 dead per kilometre. An adjusted estimate over the whole affected stretch of river produced a total of 3,000 Murray cod deaths (with minimum and maximum estimates of 1,000 and 5,000 deaths respectively).

Immediately prior to the deaths the Lower Darling had ceased flowing and was reduced to a series of remnant pools. During the week of the fish kill maximum air temperature exceeded 40 °C, with water temperatures over 30 °C. Initial reports indicated large Murray cod died several days after a front of water released from the Menindee Lakes passed down the Lower Darling River. Examination of water quality information, observations made by landholders, and expert opinion from a workshop held in July 2004, suggests the deaths were a consequence of oxygen depletion (due most likely to a combination of extreme temperature, high algal respiration and organic loading), with the possible added stress of sulfide and/or ammonia toxicity released from anoxic sediments in hypolimnetic pools. The ecological significance of this fish kill is discussed in this report, and management guidelines aimed at avoiding similar events are presented.

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INTRODUCTION

On the 17th of February 2004 an extensive Murray cod kill on the Lower Darling River at Harcourt station was reported to NSW Fisheries by a local landowner and the Sunraysia Daily newspaper. The deaths were of predominantly large Murray cod, with one report of up to 50 small Murray cod and Golden perch.

Initial reports indicated that the fish died several days after a front of water passed down the river, raising local concerns that the Murray cod population in the Lower Darling would be destroyed. The fish kill stretched for over 160 km of river between Menindee and Pooncarie, with the authors estimates of numbers of dead Murray cod of approximately 3,000. Dead Murray cod were observed at numerous points along the Lower Darling River from 14 February through to 19 February 2004.

The mass deaths were recorded by NSW Department of Infrastructure, Planning and Natural Resources (DIPNR) and NSW Fisheries staff, who responded by monitoring water quality in the affected section of river in the weeks after the deaths. DIPNR also increased the volume of flows to the river from the Menindee Lakes storage system for environmental relief. No samples of dead fish were collected at the time for pathological analysis.

Fish kills of this magnitude imply potentially important long term changes in ecosystem structure and function. The fish deaths have been the subject of significant media attention, particularly given that Murray cod is Australia's largest and most iconic freshwater species holding economic, cultural and recreational significance.

This report represents an independent enquiry into the Darling River fish deaths to determine the likely causes, comment on the potential significance of the event, and suggest future management actions that could be taken to reduce the likelihood of such a fish kill occurring in the future. Specifically the aims of this project were to:

- Define the magnitude of the event in terms of the area, numbers, species and size of fish affected.

- Comment on the ecological significance of the event in a local, regional and basin context.

- Review available information on the general circumstances and physical conditions leading to the event.

- Comment on the possible and most probable causes of the deaths.

- Define major risk factors for future fish kills in the Lower Darling River.

- Recommend management actions that can be used to reduce the risk of future fish deaths.

1   BACKGROUND

1.1   Recent Fish Kills in the Murray Darling Basin

Every year fisheries agencies record a number of fish kills of varying magnitude. Since the mid 1980s, there have been around 20 – 50 kills reported to NSW Fisheries each year, many of which are related to natural events and normal environmental cycles. Some kills are, however, caused by introduced disease, waterway pollution, or other human related activity. Relatively more kills occur in summer (40 – 50% of kills), with the main contributing factors determined to be high water temperature (and consequently lower dissolved oxygen) and low water levels in freshwater systems (NSW Fisheries, 2000). About 17% of kills are determined to be caused by low oxygen levels and temperature extremes, although no cause is determined for up to 50% of kills in NSW (NSW Fisheries, 2000).

In recent years, there have been a number of significant fish kills involving large Murray cod in the Murray-Darling Basin. Several of these are discussed briefly and summarised in Table 1.

Goulburn River January 2004 (EPA, 2004)

In January 2004 approximately 90 Murray cod died in the Goulburn River downstream of Nagambie Weir in Victoria. Numerous other fish species including Trout cod, Carp, Redfin, Goldfish and thousands of smaller fish also died in the event which stretched for over 10 km. The most likely cause of the deaths was low dissolved oxygen and associated effects, and possible sulfide toxicity, although sulfide toxicants were not detected in pathological analysis (EPA 2004).

There is a possibility that water with a high organic load, and hence low dissolved oxygen, from upstream of the Nagambie Weir system was released into the river channel (EPA, 2004). Organic loading depletes oxygen through the bacterial decomposition of carbon and nutrients washed into the system (Baldwin *et al*. 2001). More likely though is the possibility of poor quality water in the bottom of the weir pool mixing with surface water, transporting low dissolved oxygen water and sediment toxicants including sulfide into the main river channel. There is also speculation that a discharge of a toxic or oxygen sequestering chemical pollutant inadvertently entered the system (EPA, 2004). While a number of potential causes have been identified, no evidence is available to support any of the current hypotheses regarding the Goulburn River fish kill. This is in part due to the methods used and nature of the samples collected after the fish kill.

Goulburn-Murray Water attempted to improve water quality in the affected reach by releasing water from higher in the water column the day after the deaths were first reported. An Environmental Audit is currently underway to assist improvement to the management of the Goulburn River system.

Broken Creek November 2002 (Robinson, EPA 2003)

Approximately 150 large Murray cod and 6 Carp were recorded dead in the Broken Creek, a tributary of the Broken River, in November 2002. Again, low dissolved oxygen was determined to be the main cause of the fish kill, possibly as a consequence of the decomposition of a heavy infestation (large biomass) of the floating plant *Azolla* that had started to die off in the area at the time of the fish kill. Such a biomass would impede diffusion of oxygen across the air-water interface, and contribute a major carbon load to the system. Bacterial decomposition of this carbon load would deplete available oxygen (Robinson, EPA 2003).

An EPA report on the event suggested high ambient temperatures combined with low dissolved oxygen and organic loading may have resulted in the generation of high levels of mineral sulfides from the sediments (sulfide toxicity). Low pH readings taken at the time of the kill (from 5.3 to 6.1) may have been a consequence of the release of sulfides from anoxic sediments, though excessive sulfides (as would usually be indicated by high concentrations of sulfate as the sulfides were oxidised) were not picked up in EPA water quality testing. Such a scenario could affect larger fish more then smaller fish, which are possibly more able to find a micro-environment where toxin levels would be reduced, or oxygen levels higher. For example, efficient utilization of oxygen at the water-surface interface (aquatic surface respiration) by smaller individuals may account for their apparent greater survival (Sargent and Galat, 2002). It is possible the kill was caused by an unidentified pollutant or disease; however neither factor was recorded at the time of the kill (Robinson, EPA 2003).

Ovens River February 2003 (EPA, 2003)

A flash flood in the Buckland River following bushfire in the Dingo Creek catchment in northeast Victoria resulted in a major fish kill in the Ovens River. Large quantities of ash and unconsolidated soil were washed into the river following heavy rain, forming a slug of muddy water which was transported downstream. This slug created a rapid deterioration in water quality. Turbidity reached 70,000 NTU and dissolved oxygen dropped below 0.1 mg.L^{-1} for up to 24 hours. Species affected by the kill included Murray cod, Blackfish, Trout cod, Golden perch, Australian smelt, Carp, Catfish as well as Crayfish and Yabbies. It is estimated 98 -100% of fish were destroyed by the event in large sections of the Buckland and Ovens Rivers. The high turbidity made it difficult to locate and count dead fish, therefore the full extent of the kill was not determined (EPA, 2003).

Campaspe River 2001 (Streamline Research Pty. Ltd., 2001)

Inflow of deoxygenated 'blackwater' from Mt Pleasant and Forest Creek catchments in January 2001 following heavy localised rainfall resulted in a partial kill within the Campaspe River. The kill affected predominantly large Murray cod, Golden perch and Redfin. Low numbers of yabbies, shrimp and smaller fish including Australian smelt were also reported killed. Species that can tolerate low oxygen concentrations including Carp, Goldfish and Mosquitofish appeared unaffected (Streamline Research, 2001).

Table 1: Summary of other recent fish kills in the Murray Darling system.

Location	Numbers of Murray cod killed	Other species	Distance covered	Possible Causes
Goulburn River (January 2004)	Over 90	Trout-cod Carp Australian smelt Galaxias *sp*. Redfin Silver perch	Over 10 km	• Low DO (stratification, organic loading). • Possible sulfide or ammonia toxicity.
Broken Creek (November 2002)	Over 150	Carp	Not recorded	• Low DO (organic loading). • Possible sulfide or ammonia toxicity.
Ovens River (February 2003)	Up to 100% reduction in fish abundance. Only 6 Murray cod recorded due to the very high turbidity.	Blackfish Trout cod Golden perch Gudgeon *sp*. Australian smelt Carp Rainbow trout crayfish yabbies	Over 70 km in sections of Buckland and Ovens Rivers.	• Flash flood in Buckland River following a bush fire. • Low DO (high organic load). • High turbidity.
Campaspe River (January 2001)	Not recorded	Golden Perch Redfin Australian smelt yabbies shrimp	Not recorded	• Sudden decline in DO associated with floodwaters.
Darling River (February 2004)	Over 3,000 (estimates range from 1,000 to 5,000)	Golden Perch	Over 160 km	• High temperature. • Low DO (stratification, organic loading). • Possible sulfide or ammonia toxicity.

1.2   Description of Murray cod

Murray cod (*Maccullochella peelii peelii*) is considered by many as Australia's icon freshwater fish holding significance to Indigenous and European communities, and to recreational and commercial fisheries. They are able to live in a range of habitats from clear rocky streams in the upper slopes of the Murray-Darling Basin, to slow flowing turbid rivers. As a large-bodied and long lived top predator, they are a good indicator of the total health of the aquatic ecosystem, integrating the impacts of water resource development and habitat modification on lower trophic levels. Adult Murray cod prefer deeper water in sheltered areas containing cover in the form of snags or overhanging banks (Kearney and Kildea, 2001). Murray cod tend to reach sexual maturity at 4 to 5 years old (Rowland, 1998).

Murray cod populations are estimated to have declined to 10% of pre-European settlement levels and are no longer common in parts of the Murray-Darling Basin (MDBC Native Fish Strategy 2003). Populations are still thought by many to be declining, although it is recognised that numbers can vary greatly in different areas within the Basin. Murray cod was listed as vulnerable on the national list of threatened species in 2003 (Environmental Protection and Biodiversity Act 1999). Murray cod are a slow growing, territorial and long lived apex predator integrating the impacts of water resource development and habitat modification on lower trophic levels. As such they are especially vulnerable to overfishing, habitat alteration, pollution and poor water quality (MDBC Workshop proceedings, 2004). This has been emphasised by a number of recent serious fish kills.

Anecdotal reports from some anglers and residents on the Lower Darling River suggest an increase in abundance of smaller Murray cod over the last four or five years. This supports recent fisheries data and numerous reports from anglers across the Murray-Darling Basin that there may have been a resurgence of Murray cod in parts of the basin, with small fish unusually abundant (Kearney and Kildea, 2001). Small resurgences in Murray cod populations in the Lower Darling River and downstream in South Australia appear to be back-correlated with recruitment events following floods in the late 1980s and early 1990s. However, the extent to which this observed resurgence is a result of artificial stocking is unclear. Data from recreational fisheries is circumstantial and refers to small sample sizes, thus limiting the precision of population estimates, and makes quantification of perceived resurgences in populations difficult (Kearney and Kildea, 2001).

1.3   The Lower Darling River System

The Menindee Lakes comprise nine natural ephemeral lakes adjacent to the Lower Darling River in far-west New South Wales. Prior to regulation the lakes filled during periods of high flow in the Darling River, and drained back into the river channel when flows receded, leaving a residual pool that frequently dried. Each lake was connected to the Darling River independently, except for Lake Cawndilla which is connected to Lake Menindee and not directly to the river.

During the 1960s the Menindee Lakes system was converted to the fourth largest water storage in the Murray-Darling Basin securing water supply for Broken Hill, Menindee and downstream water users. The main regulatory structure of the storage scheme is the Menindee Main Weir on the Darling River, just upstream from the town of Menindee. The Main Weir raises the water level of the Darling River to 12m above bed level, increasing the inundation frequency of lakes Tandure, Bijiji, Balaka and Malta. These lakes and the inundated floodplain behind the Main Weir are known collectively as Lake Wetherell. An inlet regulator on Lake Wetherell allows the transfer of water into Lake Pamamaroo and from there into Lakes Menindee and Cawndilla. Releases to the Darling River are measured from Weir 32, approximately 15 river kilometres downstream from the town of Menindee, and about 40 river kilometres from the Main Weir (Figure 1).
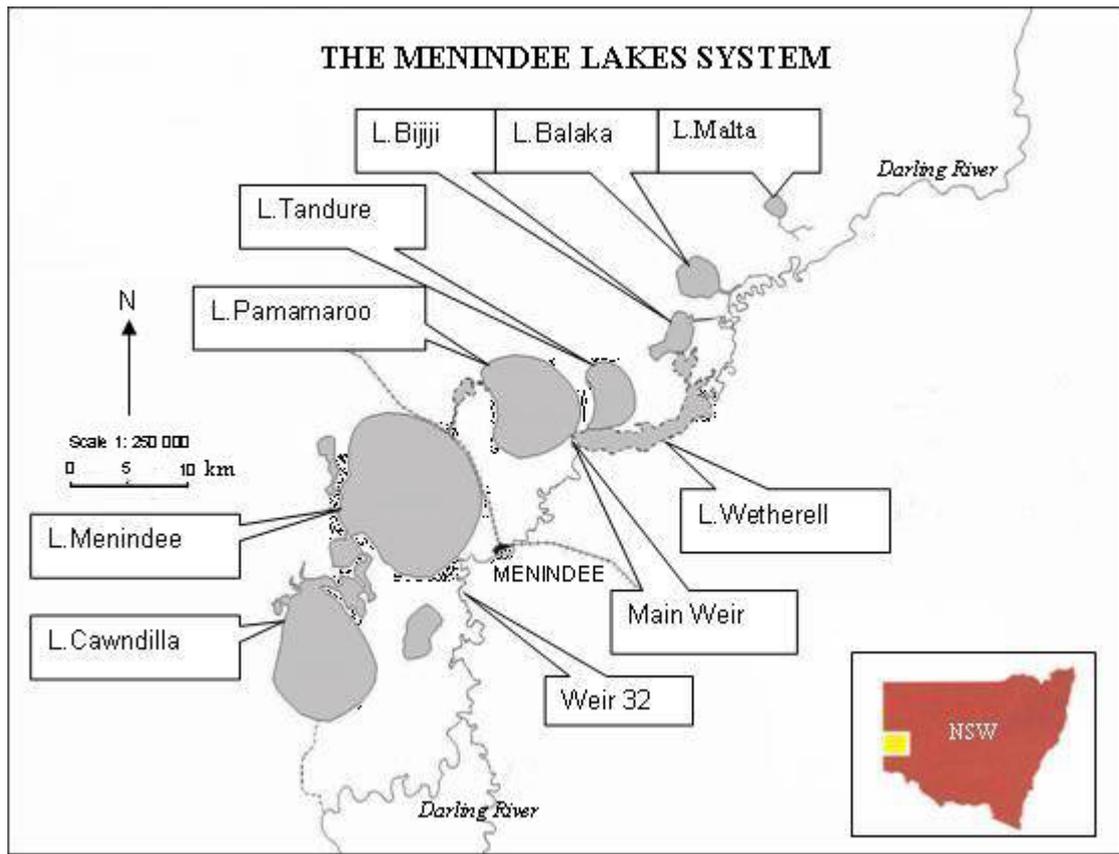
Figure 1: The Darling River showing the Menindee Lakes in far west New South Wales (Image courtesy of O. Scholz, MDFRC).

The surcharge capacity of the Menindee Lakes storage system is 1,999 GL. The Murray-Darling Basin Commission (MDBC) is responsible for managing the Menindee Lakes system and the Lower Darling River when the total storage volume within the Menindee Lakes storage scheme rises above 640 GL. The MDBC maintains control until the storage volume falls back below 480 GL, at which time all rights and management of the remaining water in storage revert to NSW State Water and NSW Department of Infrastructure, Planning and Natural Resources (DIPNR). This volume was assessed to provide security of supply to all adjacent and downstream users for one year with no inflows into the lakes system (DLWC, 1998).

In September 2001, the Lakes system was at 119% capacity. Successive years of drought and no major inflows reduced capacity to below 3% by November 2003. At this time the Darling River below the Menindee Lakes ceased to flow and was reduced to a series of residual pools.

The stretch of river between Weir 32 and Pooncarie is approximately 258 km long (see Figure 3 in section 2.2 of this report). DIPNR regularly monitors water quality at Pooncarie and Weir 32. Further monitoring is conducted irregularly during summer months at Tolarno station (82 km downstream of Weir 32), Karoola station (113 km), Harcourt station (122 km), Moorara station (191 km), Kinross station (211 km) and just upstream of the Pooncarie Weir (258 km downstream of Weir 32).

2 THE DARLING RIVER FISH KILL

2.1 Hydrological processes and releases in early 2004

From April 2003 to January 2004, only minor releases (150 to 190 ML.day^{-1}) were made by DIPNR from Lake Wetherell to meet minimum downstream user requirements. No water was available from the other lakes in the system due to the prolonged drought conditions. The Darling River upstream of Menindee had also ceased to flow. Releases to the Darling River below Weir 32 were ceased from 31 December 2003 to protect town water supply for Broken Hill and Menindee (DIPNR, Managing the Menindee Lakes Information Paper, December 2003). This was the first time the Lower Darling had ceased flowing since the construction of the Menindee Lakes scheme in the 1960s (MDBC, River Murray Water Media Release, 25 February 2004).

As a consequence, the Darling River downstream of Weir 32 was reduced to a series of isolated remnant pools. Irrigators were permitted to divert water from residual pools to supplement groundwater they were able to access. Water quality in the Menindee Lakes system and Lower Darling River was poor, with salinity reaching above 3,000 EC, and high levels of blue-green algae were recorded along the Lower Darling River (DIPNR, Managing the Menindee Lakes Information Paper, February 2004).

In January 2004 significant rain in southern Queensland and northern NSW resulted in flows of up to 400,000 ML expected to reach the Menindee Lakes by the end of March 2004. Small flows exceeding 1,000 ML.day^{-1} commenced into Lake Wetherell around 11 February, and were expected to peak mid March at about 17,000 ML.day^{-1}. The electrical conductivity of this floodwater was considered to potentially high, due to mixing with groundwater intrusion of water with conductivities recorded as high as 50,000 near Weir 19A, between Bourke and Louth (A. Hassett, DIPNR *pers. comm.*).

These inflows were expected to provide security for Broken Hill and Menindee town water supply, and to enable flows downstream of Weir 32 for stock and domestic use for at least 18 months. DIPNR announced environmental-dilution flows would commence from Lake Wetherell prior to the arrival of the northern floodwaters, to improve and protect water quality in residual pools and provide fish passage along the Lower Darling River (DIPNR, Managing the Menindee Lakes Information Paper, February 2004). While the water released was known to be of poor quality and high salinity, the flows were expected to provide relief along the river to the Pooncarie weir pool. The flows were however sufficiently small so as not to contaminate better quality water further downstream in lower parts of the Darling River behind Wentworth Weir (Schulz, 2004). In addition, the water released to the Lower Darling River was from the top of Weir 32, where the quality of the water was considered to be better than deeper water (A. Buchan DIPNR, *pers. comm.*).

On 26 January 2004, flows of between 150 to 200 ML.day^{-1} commenced from Weir 32. On 8 February 2004 the releases were increased, reaching approximately 300 ML.day^{-1} by 10 February, after it was realised the first environmental flow pulse would not be sufficient to reach Pooncarie (Figure 2). By that time it had become

apparent that significant flows from the Upper Darling River would soon flow into Lake Wetherell (NSW Fisheries briefing report, 19 February 2004).

Flow from Lake Wetherell was increased again on 18 February. By 21 February significant flood-waters in the Upper Darling River had started to reach Lake Wetherell, and discharge from Menindee Main Weir was set at approximately 700 ML.day^{-1}. At this stage however, it was still unclear how the inflowing, relatively low salinity water would mix with halo-stratified water in the Lake Wetherell weir pool (Schulz, 2004). At an earlier DIPNR River Management Meeting, the decision was taken to flush saline water out of the system and provide an environmental flow to the Lower Darling River, with a release of greater magnitude (Schulz, 2004). This environmental flow was withheld until sufficient mixing of residual water and flood waters in Lake Wetherell had occurred, ensuring the salinity of the front of the environmental flow event was not excessively high. The "flushing flows" commenced on 23 February, peaking at 6,500 ML.day^{-1} on 3 March, and reducing to below 500 ML.day^{-1} by 14 March. Flows over Pooncarie Weir resumed around the 21 February (MDBC, River Murray Water Media Release, 25 February 2004).
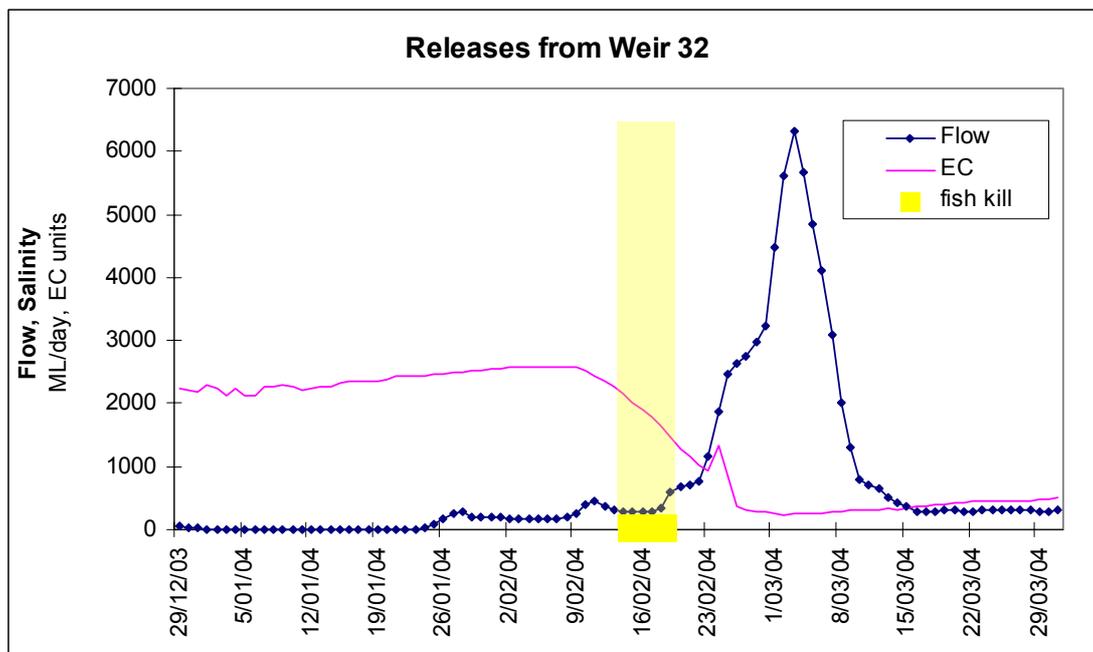

Figure 2: Releases to the Lower Darling River from Menindee in early 2004.

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2.2   The extent of the Darling River fish kill

On 17 February 2004, NSW Fisheries received reports from a local landowner and the Sunraysia Daily newspaper, of large numbers of dead Murray cod along the Darling River at Harcourt station (about 120 river kilometres downstream from Weir 32). Subsequent reports of dead Murray cod further upstream and downstream were made to NSW Fisheries, DIPNR and local newspapers over the following few days. One report of up to 50 dead Golden perch (*Macquaria ambigua*) at Wyarama station, about 15 km downstream of Harcourt, was made to the Sunraysia Daily Newspaper.

As with all fish kills, accurate estimates of the number of fish affected are hard to achieve (Strange, 1996). Discussions were held with 26 land owners, recreational anglers and departmental staff that visited the site at, or just after the event. Their observations indicated the fish deaths extended from above the northern border of Tandou station (about 70 km downstream of Weir 32), to upstream of the Pooncarie weir pool at Greenvale station (about 212 km downstream of Weir 32, and 46 km upstream of Pooncarie). This represents a stretch of river at least 160 km long.

Observations of numbers of dead fish would have been influenced by:

1.   The size and depth of residual pools (and hence the number of Murray cod occupying each pool), adjacent to and just upstream of each observation point prior to the releases of water from Weir 32 (which linked the pools).
2.   The presence of structures (eg. snags) that may 'catch' corpses as they float downstream.
3.   Removal of carcasses by observers and scavengers including foxes, pigs, pelicans and goannas.
4.   Double counting of corpses by different observers as they float downstream.
5.   The proportion of dead fish that float.

Because of the uncertainty in each of these factors, determination of an accurate estimate of the extent of the fish deaths is problematic. For example, estimates of the proportion of corpses that float quoted by land owners, fishermen and media sources, ranged from 25% to 75%. A pilot experiment conducted by a Murray cod farmer on the Murrumbidgee River in July 2004, found that of 20 adult Murray cod carcasses placed in a retaining pond, 60% floated and were removed within 3 days by scavengers, while the other 40% sank (B. Malcolm, Uarah Fish Hatchery *pers.comm.*). It should be noted that the float rate might be influenced by factors including temperature, size of the fish, and also what the fish had eaten prior to its death (B. O'Connor DSE, *pers.comm.*).

Despite this uncertainty, estimates of the number of dead Murray cod noted by land owners, anglers and other observers along the Darling River varied from between 2 fish per kilometre of river at Greenvale (at the lower end of the kill), and 30 fish per kilometre at the upper end of the kill (around Bindara and Whurlie stations). These estimates are presented in Figure 3 and in Appendix 1. Through extrapolation of observer estimates of dead Murray cod per kilometre along each station's river frontage, a figure of 1,900 dead Murray cod along the whole affected section of river was derived (Appendix 1). Applying a 60% float rate, this number increased to a total estimate of over 3,000 Murray cod killed in the event.

Figure 3: The Lower Darling River between Menindee and Pooncarie showing estimates of the number of Murray cod killed per kilometre at each station.

(Image courtesy of DIPNR, Buronga).

This estimate is based on the recollection of land owners and other attendees' observations of the fish kill. Given the remoteness of the affected stretch of river, the variability of Murray cod abundance between pools prior to the deaths, and the sparsity of observations made, it would not be unreasonable to expect the actual number of dead could be as low as 1,000 due to overestimation. For example, estimates of up to 20 dead Murray cod per kilometre along Willotia and Whurlie stations river frontage are based on observations made by landowners who only managed to access the river at several points over a long stretch of river, and on different days during and after the event.

Similarly, the time-lag between the actual deaths of the fish and their observation, and the removal of corpses from the river channel, could have underestimated the extent of the kill significantly, with the actual number of Murray cod killed potentially as high as 5,000. Conversion of observer's counts into an estimate of dead fish per kilometre is limited in accuracy; however it does allow a comparison of the relative affects of the kill along different reaches. In any case, it seems probable that the fish kill involved the death of Murray cod in the order of thousands, and not hundreds as initially reported.

Initial observations were mostly restricted to large Murray cod around 10 kg to 55 kg, with one observation of smaller fish dying at Wyarama station (including approximately 50 Golden perch, and Murray cod as small as 1 kg). While growth rates of Murray cod vary (depending on temperature, habitat and food availability), fish of 5 kg in weight are normally about 5 years old, and tend to grow at a rate of 1 to 2 kg each subsequent year (Rowland, 1988). Sexual maturity is generally reached at 4 or 5 years of age (around 5 kg), and larger specimens are generally more fecund (Rowland, 1998, Kearney and Kildea, 2001). This would indicate that most of the Murray cod killed by this event were breeding adults.

2.3   Review of water quality at the time of the fish kill

No water quality measurements were made by DIPNR in the Lower Darling River
between Weir 32 and Pooncarie in the two weeks prior to the deaths. Water quality
sampling was conducted weekly at Weir 32 and Burtundy (midway between
Pooncarie and Wentworth) before and after the fish kill. DIPNR measured EC,
temperature, turbidity and pH at six sites from Weir 32 to the Pooncarie Weir on
18/19 February, just after the fish deaths (Table 2). These sites were at Weir 32,
Tolarno station, Karoola station, Harcourt station, Kinross station and directly up
stream of the Pooncarie Weir. Dissolved oxygen (DO) was only recorded at Harcourt
and Kinross (in the mid-afternoon) on that sampling occasion. Water quality recorded
downstream of Pooncarie at Burtundy on 17 February is also presented in Table 2.

Table 2: Water quality recordings taken on 18/19 February 2004.

Site	Weir 32	Tolarno	Karoola	Harcourt	Kinross	Pooncarie	Pooncarie	Burtundy
	18/02/04	18/02/04	18/02/04	19/02/04	19/02/04	18/02/04	19/02/04	17/02/04
Time	-	18:00	18:30	15.00	14.00	19:45	13.00	-
E.C. ($\mu S.cm^{-1}$)	1495	2540	2730	3020	3060	1865	1920	4320
DO ($mg.L^{-1}$)	-	-	-	6.29	3.71	-	6.09	
pH	8.37	8.81	8.83	8.59	8.64	9.43	9.01	9.15
Turbidity (NTU)	11	-	-	24	21	-	39	11
Temp (°C)	29	31.6	31.5	30.9	29.6	27.9	27.1	29.3
Ammonia ($mg.L^{-1}$)	-	0.03	0.07	-	-	0.02	-	-
Flow ML.day-1	542	-	-	-	-	-	-	0

It was noted by landowners and DIPNR field staff that the colour of the water passing
downstream in the first two environmental releases from Weir 32 was very green, and
contained a lot of detritus in the form of detached water plants and leaf litter (Figure
4). The water was also observed to flow very slowly, especially in sections of river
where the channel was deeper.

Figure 4: The Darling River at Kinross station on 24 February showing the green colour of the first two releases from Weir 32.

Water quality was recorded again on 24 February about a week after the fish kill event at the same six sites (Weir 32 was sampled on 26 February), and also at Moorara Station. On this sampling occasion dissolved oxygen was recorded at all sites except Weir 32 (Table 3).

Table 3: Water quality recordings taken on 24 February 2004.

Site	Weir 32	Tolarno	Karoola	Harcourt	Moorara	Kinross	Pooncarie
	26/02/04	24/02/04	24/02/04	24/02/04	24/02/04	24/02/04	24/02/04
E.C. (μS.cm^{-1})	280	1218	1832	2120	2980	3070	2270
D.O (mg.L^{-1})	-	6.9	6.6	7.4	9.2	9.8	6.9
pH	7.17	8.42	8.45	8.53	8.64	8.84	8.63
Turbidity (NTU)	550	12	12	13	9	16	10
Temperature (°C)	27.4	26.3	26.3	27.3	26.5	25.8	26.3

On 24 February, the water sampled at Tolarno, Karoola and Harcourt would have been from the third release of water from Weir 32 (over 700 ML.day^{-1}) as demonstrated in Figure 7. This water was of greater volume and flow rate then the first two releases, and was noted to be a caramel brown colour (Figure 5). The third pulse appeared not to have reached the lower sites of Moorara, Kinross and Pooncarie

by February 24 (Figure 7); hence water tested at these three lower sites would have still been the green water of the first two releases (M. Chase DIPNR *pers. comm.*). By this time dilution and mixing in Lake Wetherell had significantly improved the quality of the water in storage, and environmental "flushing flows" had been increased to almost 2000 ML.day^{-1} (Schulz, 2004).

Figure 5: The Darling River at Tolarno on 24 February 2004 showing the brown colour of the third release from Weir 32.

Water temperature in the stretch of river between Weir 32 and Pooncarie on February 18 and 19 reached over 30°C at several sites (Table 2). After the third and larger pulse passed downstream, temperature dropped to between 26 and 27 °C (Table 3) as a consequence of a drop in ambient temperature and mixing within the larger volume of water. Figures 6 and 7 illustrate the high ambient and water temperatures recorded at the time dead Murray cod were observed in the river.
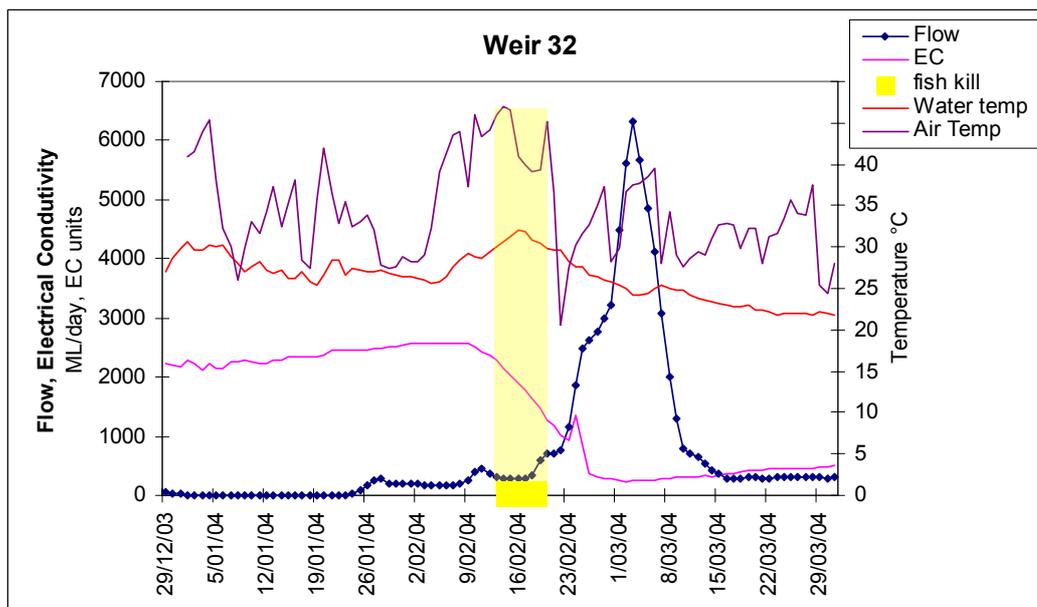
Figure 6: Air and water temperature, flow and salinity upstream of Weir 32 at the time of the Murray cod deaths.

Dissolved oxygen concentrations after the first two pulses on February 18 ranged from between 3 to 6 mg.L^{-1} (Table 2). Dissolved oxygen levels appear to have increased over the following week prior to the third pulses arrival, to above 8 mg.L^{-1} (Kinross and Moorara in Table 3). Dissolved oxygen levels decreased again after the passing of the third larger pulse (Tolarno, Karoola and Harcourt in Table 3), though concentrations below 6.5 mg.L^{-1} were not recorded. The larger volume of water in the third pulse released from Weir 32 appear to have been sufficient to maintain dissolved oxygen concentrations in the Darling River above levels stressful to fish.

The salinity in the residual pools along the river would most likely have been higher before water released from Weir 32 reached each pool, and it appears that fish did not die until after the second released passed each pool. Further to this, salinity levels at Burtundy, downstream of Pooncarie, were over 4,000 EC during the week the fish kill occurred (Table 2), yet no dead Murray cod were reported below Pooncarie. While the high salinity levels may be a minor stress to Murray cod, they are unlikely to have contributed directly to the fish deaths in the Darling River.

In the section of the Darling River from Weir 32 to Pooncarie, pH ranged from 8.4 to 9.4 on February 18 and 19, and from 8.4 to 8.8 on February 24 (Table's 2 and 3). High pH values, if caused by alkaline toxins like ammonia, can have adverse effects on fish and will be discussed in sections to follow. While pH values below 6 are usually stressful to most fish species (NSW Fisheries 2000), all recorded pH values in the Darling River around the time of the kill were higher then 8.3. This indicates that acidic (low) pH were unlikely to have factored in the kill event. However, high levels of sulfate (97 mg.L^{-1}) were recorded in the pool behind Weir 32 on 27 January; just days after gradual release of water from Lake Wetherell had begun. This high sulfate concentration could indicate the oxidation of toxic sulfide from disturbed anoxic

sediment, which would normally be expected to lower the pH. Possible pH buffering of acidic addition (and thus a maintenance of higher pH), by high bicarbonate concentrations also recorded behind Weir 32 on 27 January (180 mg.L^{-1}) is discussed in the sections to follow.

Figure 7 shows a time line including releases from Lake Wetherell, observation dates of dead Murray cod, algal peaks, and dissolved oxygen recordings around the time of the mass deaths.

Figure 7: Time line showing releases from Lake Wetherell, observation dates of dead Murray cod, maximum daily air and water temperatures, algal peaks and dissolved oxygen recordings.

		23-Jan	24-Jan	25-Jan	26-Jan	27-Jan	28-Jan	29-Jan	30-Jan	31-Jan	1-Feb	2-Feb	3-Feb	4-Feb	5-Feb	6-Feb	7-Feb	8-Feb	9-Feb	10-Feb	11-Feb	12-Feb	13-Feb	14-Feb	15-Feb	16-Feb	17-Feb	18-Feb	19-Feb	20-Feb	21-Feb	22-Feb	23-Feb	24-Feb	25-Feb	26-Feb	27-Feb
Max Air Temp.		35.4	32.5	33.0	33.9	32.0	27.8	27.5	27.6	28.9	28.2	28.3	29.0	32.3	39.2	41.4	43.6	44.0	37.2	46.0	43.3	44.3	46.0	46.9	46.5	40.9	40.0	39.2	39.3	45.3	36.7	20.6	27.6	30.2	31.8	32.8	35.2
Max Water Temp.		26.7	27.3	27.1	27.0	27.1	27.2	26.6	26.2	25.9	26.0	25.9	26.0	25.6	25.8	26.5	27.6	28.5	29.2	28.8	28.7	29.4	30.0	30.6	31.2	32.1	31.8	30.9	30.4	29.9	29.7	29.6	28.2	27.0	26.6	26.1	26.2

km from Weir 32	Station	Notes
0	Weir 32	150 ML/day; 300 ML/day; 700 ML/day; > 1000 ML / day
28	Wanda	
39	Bono	
50	Tandou	
75	Bindara	
82	Tolarno	DO = 6.9
90	Willaba	
93	Willotia	
113	Karoola	DO = 6.6
122	Harcourt	DO = 6.3; DO = 7.4
130	Whurlie	
133	Wyarama	
160	Selma	
177	Polia	
191	Moorara	DO = 9.2
200	Court Nareen	
202	Aston	
212	Kinross	DO = 3.7; DO = 9.8
212	Greenvale	
232	Mullingar	
258	Pooncarie	estimated flow - flow logger broken; DO = 6.9

Key:
- 1st release from Lake Wetherell - over 150ML /day (estimated flow as logger broken)
- 2nd release - over 300 ML/day
- 3rd pulse - over 700 ML/day
- Environmental dilution flows (1000 - 6000 ML/day)
- Maximum daily water temperature over 30°C (average of Menindee and Pooncarie recordings)
- Algal peak (around 1000,000 cells / ml)
- Definite observations of dead Murray cod
- Estimated dates of observation's of fish deaths
- DO Recorded dissolved oxygen concentration (mg/L)

3 POSSIBLE CAUSES OF THE FISH KILL

A workshop attended by DIPNR staff and Murray-Darling Freshwater Research scientists was held in July 2004 to discuss the details of the Darling River fish deaths. At the workshop four main factors considered likely to have caused the fish kill were discussed. These factors are:

- Temperature
- Dissolved oxygen
- Chemical toxicity
- Turbidity (specifically organic turbidity).

The mechanisms of each causal factor are discussed below, including the potential for extremes in water quality parameters to contribute to Murray cod mortality. Flow diagrams illustrating important determinants and effects for each causal factor are included in Appendix 2.

Note: Discussion with NSW State Water in Menindee, and with land owners and operators adjacent to and directly upstream of the Darling River fish kill, indicate that no addition or spillage of an agricultural toxin in to the river had been identified or reported at the time of the deaths. The possible involvement of a toxic contaminant in the Darling River fish deaths cannot be ruled out, though testing for such pollutants would need to target specific chemicals suspected to be in the system. As no spillage or seepage into the Lower Darling River was reported prior to the event, this report has focused on other possible causes for the fish kill.

3.1 Temperature

The average daily maximum air temperature recorded at Pooncarie and Menindee from 10 to 20 February was 43.4°C (Bureau of Meteorology, 2004). The average maximum daily water temperature at Weir 32 during the same period was 30.3°C, with maximum daily water temperature exceeding 31°C from February 15 to 17, and peaking over 32°C (Figure 7) (DIPNR unpublished data). The logging station at Pooncarie malfunctioned from February 2 to 25, consequently only water temperatures for Weir 32 are quoted on these dates. It is assumed that similar temperatures would have been experienced along the section of Darling River between Weir 32 and Pooncarie.

It is reasonable to assume even higher water temperatures could have been experienced in shallower pools where the released water heated up as it passed over the hot river bed. Murray cod will tolerate water temperatures up to 33°C (Koehn and O'Conner, 1990), though temperature tolerance can be influenced by variation in other water quality characteristics.

It is likely that at least the deeper pools along the Darling River prior to the releases from Weir 32 would have been temperature stratified. This occurs when surface water heats up faster than deeper water, and the warmer surface waters do not mix with the colder bottom waters (Rees *et al*. 2003). Mitrovic *et al*. (2003) demonstrated stratification in the Darling River at Bourke, and also at Wilcannia when discharge and flow was low. Discharge of less then 200 ML.day^{-1} at Wilcannia (upstream of the Menindee Lakes) during the hotter months, combined with persistent high temperatures resulted in more persistent stratification (Mitrovic *et al*., 2003).

Stratification can lead to depletion of dissolved oxygen (anoxia) in the cooler, deeper waters and sediments of the pool, the consequences of which are discussed in the following sections. Although water temperature had previously reached 30°C for three consecutive days earlier in January of 2004, flows from Weir 32 had ceased just days before, and stratification in pools along the Lower Darling River may not have been intensive.

3.2   Dissolved Oxygen (DO)

3.2.1 Oxygen depletion

Few fish species can tolerate prolonged exposure to dissolved oxygen levels below 3 mg.L^{-1}, especially large bodied or more active fish with a higher oxygen demand (NSW Fisheries 2000).

Dissolved oxygen depletion can result from a diversity of factors. Throughout diurnal cycles, all aerobic organisms consume oxygen through respiration. During the daytime, algae and aquatic plants contribute dissolved oxygen to the system during photosynthesis. The night-time combination of oxygen depletion through respiration and reduced photosynthetic re-oxygenation means that dissolved oxygen concentrations can fall, and are often lowest at dawn (Baldwin *pers. comm.*). Microbial breakdown of organic matter (entering the river from flooding or rain events) can also lead to oxygen depletion, as in the Broken Creek fish kill of November 2002 (Robinson, EPA 2003).

Dissolved oxygen was 3.7 mg.L^{-1} at Kinross station on 19 February (DIPNR unpublished data), a few days after the passing of the second pulse of water released from Weir 32. This measurement was taken at 14:00 hours when maximum diurnal levels of dissolved oxygen would be likely due to photosynthetic addition. As noted above, concentrations would have been lower during the night. Dissolved oxygen was 6.3 mg.L^{-1} at Harcourt on 19 February (where the second pulse had passed several days earlier), and 6.1 mg.L^{-1} in the Pooncarie weir pool. Dissolved oxygen on 24 February ranged from 6.6 to 9.8 mg.L^{-1} at sites between Weir 32 and Pooncarie, and was lower at the three most upstream sites (where the third released pulse had recently passed). This indicates that dissolved oxygen concentration increased in the days following the passing of the second pulse front, before decreasing again following the passing of the third front.

Fish killed by oxygen depletion often exhibit a wide open mouth, flared gills and a bent back head (NSW Fisheries 2000). These symptoms were commonly displayed by the fish observed in the Darling River fish kill (Figure 8)

Figure 8: Murray cod killed in the Darling River during the February 2004 fish deaths, with gaping mouths and bent back heads suggesting oxygen stress.

(Image courtesy of the Sunraysia Daily)

3.2.2   Organic loading

Breakdown of organic matter by aerobic bacteria in aquatic systems consumes oxygen. Large increases of organic matter in the water column (known as organic loading), can cause dissolved oxygen concentrations to drop to levels lethal to fish. Soon after flooding, a large pulse of carbon and nutrients is released from sediments and organic litter into the overlying water, which is used by aerobic bacteria for growth. If the consumption of oxygen occurs at a greater rate than oxygen replenishment from the atmosphere and photosynthesis, the water column can become anoxic (Baldwin *et al*. 2001).

It was noted by DIPNR field staff and local landowners that the water flowing down the Darling River at the time of the fish deaths contained an abundance of leaf litter, detritus and aquatic plants picked up by the flood pulse. Under the hot conditions experienced in the river around the time of the kill, this material would begin breaking down once inundated. The first two pulses of water released from Weir 32 could have been sufficiently small and slow flowing that organic loading of the pools may have occurred.

In addition, stratification in a pool would reduce the chance of aerated surface water mixing with anoxic deeper water (Howitt and Baldwin 2003). Further, oxygen has a lower solubility in warmer water, reducing the atmospheric re-aeration of anoxic water that may have existed in the water column during the fish kill.

3.2.3   Blue-green algae bloom

Algal blooms have high rates of respiration and can interfere with light transmission at the air-water interface. Algal abundance in a system is largely dependant on the availability of nutrients and sunlight (Haider et al. 2003), and is inversely related to flow rate, with higher abundance at lower flow rate (Sherman *et al*. 1994, Mitrovic *et al.* 2003). Consequently higher algal numbers and greater respiration demand are more likely during extended hot and/or low flow periods. Large algal blooms can significantly reduce dissolved oxygen in the water column to concentrations stressful and even lethal to many fish species (Codd 1995).

The NSW Algal Co-ordinating Committee has set an 'indicative' high alert blue green algal cell density level of 15,000 cells.ml $^{-1}$. In late December 2003 blue-green algal counts in water behind Weir 32 were greater then 85,000 cells.ml $^{-1}$. On 20 January 2004 blue-green algal counts in the Darling River at Karoola station were almost 400,000 cells.ml^{-1} (Figure 9). Of these 25,700 were considered by DIPNR to be potentially toxic species (*Anabaena spp*. and *Microcystis sp*). Two weeks later on 3 February at Karoola station, coinciding with the estimated arrival of the first release from Weir 32 (Figure 7) , blue-green algal counts peaked over 1,000,000 cells.ml $^{-1}$, of which 84,900 were considered potentially toxic (DIPNR algal data).
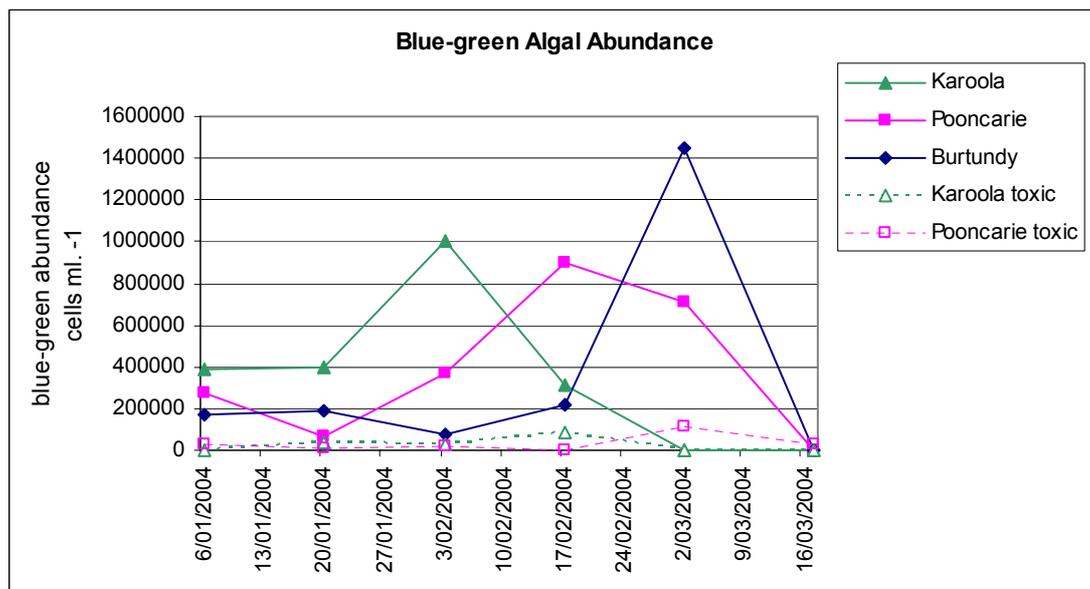

Figure 9 - Blue-green algal abundance in the Lower Darling River.

The peak in blue-green algal densities was recorded at Pooncarie two weeks later on 17 February, with total blue-green counts of greater then 900,000 cells.ml^{-1} of which about 118,000 cells.ml^{-1} were considered potentially toxic (Figure 9). This recording coincided with the expected arrival of the first and second pulses of water released from Weir 32 (Figure 7). (NB; as the DIPNR flow recorder at Pooncarie was out of service from February 2 to 25, travels times for the first pulse of water released from Weir 32 are estimates).

As shown in Figure 9, the bloom appeared to die-off considerably within two weeks of peaking at each sample collection site. This die-off occurred over the same period of time that fish were reported dead in the Lower Darling River. Many fish kills reported in eutrophic water bodies at times of blue-green algal blooms are traditionally ascribed to oxygen depletion due to bloom respiration, high microbial oxygen demand during bloom die-off, physical gill blockage by algal cells and colonies, high pH levels, and high ammonia during bloom senescence (Codd, 1995).

It therefore seems reasonable that the die-off of substantial numbers of algal cells (a crash in numbers of over 600,000 cells.ml^{-1}), would considerably raise the microbial oxygen demand in warm water already low in dissolved oxygen, with possibly lethal effects on fish. Efficient utilization of oxygen at the water-surface interface (aquatic surface respiration) by smaller fish (Sargent and Galat, 2002) may account for the kill event involving predominantly large Murray cod. Additionally, large Murray cod were likely to have established territories within the residual pools prior to the deaths, and may have been reluctant to move to better quality water then smaller fish (Humphries *pers. comm.*).

Although the potentially toxic component of the blue-green algal counts only represent a small proportion of the overall total cell count, the numbers of potentially toxic cells still exceed 'high alert' cell density levels. Factors that trigger algal toxin formation are not precisely known (Haider *et al.* 2003). However, given these blue-green cells were contained in a relatively shallow and small volume of water in the river channel (restricting the possibility of fish escape), their toxic potential to fish could be considered significant. Cyanobacteria (blue-green algae) blooms have often been associated with fish kills, though the cause of death has usually been difficult to ascertain. Exposure of fish to the cell contents of cyanobacteria can promote osmoregulatory imbalance, resulting from stimulation of the drinking response, increased volume of fluid in the gut and inability to remove excess water, increasing the opportunity for absorption of toxins (Best *et al.* 2002). Cyanobacterial neurotoxins can also induce paralysis of respiratory muscles, causing death by suffocation (Haider *et al.* 2003). Unfortunately no samples of dead fish from the February 2004 event were collected and tested for blue-green related toxins.

3.3 Chemical toxicity

If the rate of microbial consumption of oxygen in sediments is sufficiently high, the sediments and the overlying water column can become anoxic, especially if thermal and salt stratification occurs in the water body. In anoxic conditions, anaerobic bacteria can use compounds other then oxygen for respiration, including sulfate and nitrate. Sulfate (SO_4^{2-}) is converted to sulfide (S^{2-}), and nitrate (NO_3) is converted to either nitrogen gas (N_2) or ammonia (NH_3). Both ammonia and sulfides are toxic to fish at relatively low concentrations.

The ammonia and sulfide levels at which there exists some toxic effect would not normally appear in a flowing system. However, toxic levels could potentially occur in stratified waters where microbial activity at depth has a major impact on water quality. If stratification rapidly breaks down, or if bottom water is released, fish may be exposed to water that is both anoxic and contains toxic concentrations of ammonia or sulfide (Rees *et al.* 2003).

3.3.1 Sulfide toxicity

Under anoxic conditions, sulfur compounds in the sediments remain in a reduced state and can be evidenced by the smell of hydrogen sulfide (H_2S, rotten egg gas), or by black sediment (Baldwin *et al.* 2002). If these sediments are then mixed through the water column, rapid oxidation of the reduced sulfur compounds can occur, releasing sulfate (SO_4^{2-}) into the water (McCarthy *et al.* 2003). For example,

$$HS^- + 4H_2O \leftrightarrow SO_4^{2-} + 9H^+ + 8e^-$$

Sulfate measurements (SO_4^{2-}) at Weir 32 on 27 January 2004 were 97 mg.L^{-1}, two days after a gradual release of water from Weir 32 had begun. This concentration is very high and could indicate the oxidation of toxic H_2S from disturbed anoxic sediments or hypolimnetic water. Oxidation of sulfide produces acid, thus normally lowering pH, yet low pHs were not observed in the system at the time this high sulfate recording was made. However, the measured alkalinity (bicarbonate, HCO_3^-) in the water behind Weir 32 was high (180 mg.L^{-1}). This high concentration of bicarbonate would react with (or buffer) any acid produced, in the oxidation of sulphide, limiting any drop in pH.

Current guidelines suggest a moderate reliability trigger level of 0.001 mg.L^{-1} of sulfide expressed as un-ionised H_2S for aquatic systems (Australian Water Quality Guidelines for Fresh and Marine Waters: ANZECC, 1992). ANZECC guideline values for individual chemicals are an estimate of the maximum concentration unlikely to cause adverse environmental effects. Acute sulfide concentrations (lethal concentration that kills 50% of test organisms in a given time, usually after 96 hours for fish) from 0.007 - 0.041 mg.L^{-1} have been demonstrated on freshwater fish species. However, toxicity testing for Murray cod has not been performed, and extrapolation from laboratory specimens to relevant species in the field and to whole ecosystem effects introduces large uncertainties in the estimation of risks (ANZECC,

1992). Any sulfide present in the water at the time of the Darling River fish deaths would have been rapidly oxidised to sulfate (though is not necessarily the only source of sulfate), and testing for sulfide was not performed.

Increased temperature may increase the sensitivity of fish to hydrogen sulfide. Additionally, the concentration of hydrogen sulfide dissolved in solution is inversely correlated to the concentration of dissolved oxygen, i.e. a greater H_2S concentration is available to aquatic organisms when less oxygen is present (ANZECC, 1992). Given the high sulfate readings in the water behind Weir 32 prior to the fish kill, and the warm and possibly oxygen-depleted water conditions at the time of the deaths, the possibility that high levels of sulfide were released into the system before being oxidised cannot be excluded. The turbulence created by the release water from Menindee Main Weir into the Lower Darling River may have been sufficient to "turn-over" stratified water behind Weir 32, (and possibly in stratified pools along the Lower Darling River), causing a mixing of toxic sulfide in to the water column.

It is of note that on 2 March 2004, five weeks after releases began from Weir 32, (and large volumes of water from the Upper Darling had reached Lake Wetherell), sulfate levels at Weir 32 had dropped to 9 mg.L^{-1}.

3.3.2   Ammonia toxicity

Anaerobic conditions can lead to the production of ammonia (NH_3) through decomposition of organic nitrogen compounds. In aqueous solution, ammonia acts as a base, acquiring hydrogen ions from H_2O to yield ammonium (NH_4^+) and hydroxide ions (OH $^-$).

$$NH_4^+ \text{ (aq)} + OH^- \text{ (aq)} \leftrightarrow NH_3 \text{ (aq)} + H_2O \text{ (aq)}$$
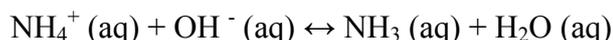
The pH where half of the ammonia is in the form of ammonium ions and the other half as ammonia is 9.24. When the pH is below approximately 8.5, all the ammonia is in the form of ammonium, while at a pH above approximately 10, all the ammonia is as dissolved ammonia gas. Ammonia is significantly more toxic to fish than ammonium ions, and the toxicity of ammonia is greater in more alkaline waters at higher temperatures. Ammonia is also more toxic under conditions of decreased oxygen concentrations (DEH, 2004). Sargent and Galat (2002) suggest low dissolved oxygen and ammonia act synergistically in fish kills.

Even low levels of ammonia (< 0.1 mg.L^{-1}) can act as a strong irritant to fish, especially to the gills. Prolonged exposure to sub-lethal levels can lead to gill hyperplasia – the swelling and clumping of gill filaments. This can result in respiratory stress and create conditions for opportunistic bacteria and parasites to proliferate. Even short exposure to higher concentrations (> 0.1 mg.L^{-1}) can lead to skin, eye, and gill damage. Elevated levels can also cause ammonia poisoning by suppressing normal ammonia excretion from the gills. If fish are unable to excrete this metabolic waste product there is a rise in blood-ammonia level resulting in damage to internal organs (www.fishdoc.co.uk).

Under most natural conditions of pH and temperature, ammonia has moderate chronic toxicity to aquatic life (DEH, 2004). Chronic (stressful) effects to fish can be seen long after first exposure to a toxic chemical. Current guidelines suggest a trigger level of 0.9 mg.L^{-1} of ammonia at pH of 8 for freshwater systems (Australian Water Quality Guidelines for Fresh and Marine Waters: ANZECC, 1992). This figure is potentially reduced at higher temperatures and pHs. Acute or lethal effects of ammonia have been recorded for freshwater fish species from 0.88 - 169 mg.L^{-1} (ANZECC, 1992). No data are available on the short-term acute (or lethal) effects of ammonia to Murray cod.

A strong smell of ammonia was noticed by DIPNR staff in Lake Wetherell on 17 February (G. Schulz, personal observation). On 18 February, just after the fish deaths, ammonia concentrations were measured at three sites along the Darling River between Weir 32 and Pooncarie. In all cases, the ammonia concentration was lower than 0.1 mg.L^{-1}. The highest recording of 0.07 mg.L^{-1} was made at Karoola at which time the water temperature was over 31°C and the pH was over 8.8. A measurement of 0.02 mg.L^{-1} of ammonia was recorded at Tolarno where the water temperature was also over 31°C and pH was over 8.8; and 0.03 mg.L^{-1} of ammonia was recorded at Pooncarie where pH was over 9.4 and the water temperature was almost 28°C.

Although these concentrations are below the suggested trigger levels, when combined with high temperatures and pH, and low dissolved oxygen concentrations, the concentrations of ammonia recorded in the water column could be toxic to fish. Such toxicity could have a more severe affect on larger fish less able to escape to micro-environments lower in toxin concentration. Efficient utilization of oxygen at the water-surface interface (aquatic surface respiration) by smaller individuals may account for their apparent greater survival (Sargent and Galat, 2002). It is also possible that large Murray cod were likely to have established territories within a reach of the river, and would be more reluctant to move to better quality water then smaller fish (Humphries, *pers comm.*).

3.4   Turbidity

Inorganic turbidity readings taken at the time of the fish deaths in the Lower Darling River were not high, and would be unlikely to have affected Murray cod either through gill clogging or through a reduction of prey visibility. There was however very high blue-green algal cell densities recorded in the river at the time of the fish deaths, which can be considered organic turbidity. There is a possibility that so many cells in the water could have had a gill clogging effect, though it is unlikely considering that only Murray cod appear to have been affected. More likely is the affect of such huge numbers of algal cells on dissolved oxygen concentrations in the water (as discussed above). High salinity levels such as those recorded in the Lower Darling River prior to the kill can have a flocculating effect on suspended solids, thus increasing light penetration in the water column. This in turn would enhance the potential for algal growth (Oliver *et al*. 1999).

4. DISCUSSION OF MECHANISMS RESPONSIBLE FOR THE FISH DEATHS

The aim of this section is to determine from the information presented in previous sections the most likely causes of the fish deaths in the Lower Darling River in February 2004. The conclusions described here were discussed at the expert workshop attended by DIPNR staff and Murray-Darling Freshwater Research scientists held in July 2004, and the justification for these conclusions is presented below.

The simplest explanation for the deaths is that, irrespective of the water releases from the Menindee Lakes, combined high temperatures and reduced dissolved oxygen concentrations in residual pools along the Lower Darling River could have been sufficient to cause the deaths of large Murray cod. The extended period of very hot weather just prior to and during the fish kill would have increased the oxygen demand of fish in the remnant pools which already contained water of poor quality. At higher temperatures, the oxygen saturation concentration of water is lower and larger fish have greater oxygen requirements than smaller fish, thus explaining the presence of only larger bodied fish in the kill.

It could, therefore, be argued that these factors alone were responsible for the fish deaths. If this was the case, it would be expected that fish would also have died downstream of Pooncarie, where the river was also reduced to a series of remnant pools containing water of poor quality. Fish deaths were, however, only reported in the river above Pooncarie, strongly implicating a fatal effect of the two pulses of water released from the Menindee Lakes in late January and early February (which re-established flow between Weir 32 and Pooncarie).

A suggestion was made that dense, salty water in the pulses released from Weir 32 sank, taking the heat to the bottom of the remnant pools. This is unlikely, as salinity readings in the Darling River at Burtundy (downstream of Pooncarie) prior to the kill were equal, and perhaps even higher then the water released from Weir 32. Although Burtundy is outside the stretch affected by the kill, it would be expected similar salinity levels would have existed upstream of Pooncarie prior the fish deaths. That is, the water in the pools along the Darling River was saline before releases were made from Weir 32, so high salinity is unlikely to have directly contributed to the Murray cod deaths.

Workshop participants agreed that the fish deaths were more likely caused by temperature induced oxygen stress (as described above), and further oxygen depletion resulting from bacterial breakdown of the high organic loads present in the first two water pulses from Weir 32. The front of a flood pulse will experience significant dissolved oxygen depletion as organic matter is picked up and broken down. Because the volume of water in the first two pulses released from Weir 32 was relatively small, the actual concentration of carbon and nutrients in the water would likely have been very high. This, combined with high water temperatures exacerbated by water moving across hot river bed, would stimulate high microbial activity and thus an increased chance of anoxia (Baldwin *et al.* 2001). A further reduction in dissolved oxygen concentrations would have resulted from the microbial breakdown of the large algal

bloom in the Lower Darling River that crashed concurrently with observations of dead Murray cod.

The pulses released from Weir 32 were moving slowly, with an estimated flow velocity of approximately 0.34 m/s, or 1.2 km/hour. Consequently, atmospheric re-aeration through turbulence or flow might have been less then the oxygen depletion through organic loading, algal respiration and microbial breakdown of the large algal bloom. As previously stated, larger fish have greater oxygen requirements and are affected more severely by oxygen depletion then smaller fish. It would also be reasonable to presume smaller fish are more able to move to areas in the system where oxygen depletion would be less severe (such as upstream behind the pulse front, or into surface water where the dissolved oxygen concentration might be elevated through re-aeration). Larger fish may also have established territories within the system, and hence be reluctant to move to better environments. These factors assist further in explaining why predominantly large Murray cod died during the February 2004 event.

It was also agreed by workshop participants that one additional component may have further contributed to the Murray cod deaths; the release of chemical toxins into the water column. The residual pools behind Weir 32 and along the Lower Darling River may have been stratified prior to the resumption of flows from Lake Wetherell (though no data was collected prior to the fish deaths to confirm this). The release of water to the Lower Darling River could have been sufficient to "turn-over" these stratified pools, exposing fish to anoxic water that potentially contained toxic concentrations of ammonia and sulfide.

Elevated levels of sulfate were recorded behind Weir 32 just after releases to the Lower Darling resumed on January 27, 2004, which could support this hypothesis (as discussed earlier in section 3.3.1). This sulfide would have been transported downstream and, as the volume of the pulse was relatively small and slow flowing, may have been sufficiently high to poison large fish. Also, studies have shown that ammonia and low oxygen act synergistically to kill fish (Sargent and Galat, 2002). The toxicity of the low levels of ammonia recorded in the Lower Darling River may therefore have been significantly increased as a result of de-oxygenation.

The fish deaths occurred just after the passing of the second pulse of water (300 ML.day^{-1}). Hence it would appear that the first pulse (150 ML.day^{-1}) was not sufficiently large to turn the pools over. Alternatively, the first pulse may have "turned-over" stratified pools in the Darling River below Weir 32, and the second pulse transported the released toxins downstream.

Even if the concentrations of these toxins were not sufficient to be lethal on their own, the combined physiological stress of sub-lethal toxicity, heat stress and oxygen depletion would appear to have been sufficient to kill large numbers of big Murray cod.

5. ECOLOGICAL SIGNIFICANCE OF THE KILL

Despite the deaths of such a large number of Murray cod being of major concern, the ecological significance of the deaths is difficult to ascertain. Among the many potential ramifications, the reduced predation on smaller fish within the Darling River system could conceivably lead to an increase in the abundance and distribution of carp (*Cyprinus carpio*) (Kearney and Kildea, 2001). Alternatively, feeding of Murray cod on smaller planktivorous fish may decrease the frequency and severity of algal blooms through trophic biomanipulation pathways (Shapiro *et al.,* 1975).

The size of the Murray cod population in the affected stretch of river prior to the fish kill is unknown; indeed most landowners were surprised that so many large cod had actually occupied the remnant pools along the river before the fish deaths. The size of the population in the Lower Darling River subsequent to the deaths is also as yet unknown, so we cannot comment as to what proportion of the resident population was destroyed by this event.

Despite fishing clubs in the Broken Hill and Lower Darling area have reporting low catch rates since the fish kill (M. Hanley, Western Darling Fishing Club *pers. comm.*), there have been several reports by landowners and the Sunraysia Daily newspaper of large fish being caught in the affected stretch of river since the fish deaths. Further to this, NSW Fisheries officers caught Murray cod at a number of sites between Weir 32 and Pooncarie as part of their Integrated Fish Monitoring project (NSW Fisheries 2004, unpublished data). These included 4 Murray cod at Tolarno, 7 at Karoola, 3 at Moorara and 1 at Pooncarie, and indicate that populations of Murray cod still exist in the Lower Darling River.

There is speculation that fish kill events of this magnitude would have occurred naturally in pre-regulated river conditions, as evidenced by the fact that the Darling River historically underwent periods where it dried out completely. In dryland rivers, periods of low flow are just as important as periods of high flow for maintaining ecological processes (Thoms and Sheldon 2002). In the confined space of each remnant pool prior to the Darling River fish kill, it can be presumed that populations of smaller fish may have been greatly reduced due to predation by Murray cod. As such, the Murray cod deaths may have provided a window of opportunity for populations of smaller native fish species (including smaller size classes of Murray cod) to rebuild while the adult cod populations slowly return.

This is not to discount the potential devastation to the Murray cod population arising from the mass deaths. Murray cod numbers in the Murray-Darling Basin have been estimated to be as low as 10% of pre-European populations (MDBC Native Fish Strategy 2003). Density-dependent recruitment issues aside, the death of such a large number of adult (breeding) Murray cod will likely further impact the stocks of this iconic fish within the Darling River for many years. Without determination of population size before and after the fish kill, however, we are unable to estimate the nature and extent of this impact.

There are also knowledge gaps in our understanding of aspects of Murray cod biology. For example we do not have sufficient information with regard to the structure and dynamics of Murray cod populations (including larval and juvenile fish),

and the habitat requirements for successful recruitment of early life stages (MDBC Workshop proceedings, 2004). We cannot, therefore, assume re-population of Murray cod in the Lower Darling River will occur unimpeded by regulatory structures that restrict up-stream migration and recruitment, or loss of key habitat areas due to flow regulation.

Murray cod reach sexual maturity at approximately 4 to 5 years of age (Rowland, 1998). If fish passage, flow and food supply conditions for successful recruitment were met, cod spawned in the 2004/05 breeding season by surviving adults in the Darling River would (at the earliest) reach sexual maturity in 2008/09. This will almost certainly not replace the breeding population lost in the February 2000 deaths. Rather, it serves to demonstrate the ecological issues behind successful recruitment, and the time lag between spawning and successful establishment of future breeding generations. Given these possible impediments, the impact of the 2004 fish kill on the size and future recruitment of Murray cod populations in the lower Darling River could last decades.

Estimates of population replacement through stocking of Murray cod in the Lower Darling River were generated by Dr John Koehn of the Department of Sustainability and the Environment, using ESSENTIAL population modelling software (Todd *et al.* 2001). The program generated probabilities and time scales for the replacement and re-establishment of 1500 female Murray cod for three different stocking regimes (1,500 females being half of the estimated 3,000 Murray cod killed in the February 2004 event, assuming a 50:50 sex ratio). A stocking regime of 200,000 fingerlings yearly for 10 years, gives an 80% chance after 46 years of the establishment of 1,500 15 year old females. Such a stocking program would cost an estimated $1.36 million. Stocking of 50,000 fingerlings yearly for 40 years gave an 80% chance of replacement of 1,500 females after 52 years (estimated cost of $1.9 million). A one-off stocking of 2 million fingerlings gave an 80% chance that 1,500 females would be established after 49 years, at a cost of $1.2 million (J. Koehn , *pers. comm.*).

This model does not take into account any 'natural' recruitment from existing cod stocks that might occur in the system, nor does it consider the possibility that the population of Murray cod in the Lower Darling River was only fractionally reduced by the February 2004 fish deaths (as suggested by the NSW Fisheries Integrated Monitoring Data discussed above). It does however demonstrate that the time taken for the replacement of the estimated 1,500 female Murray cod killed in the event could be in the order of decades, even if artificial restocking is incorporated to supplement re-establishment. Additionally, it should be recognised that artificial restocking of Murray cod from non-local brood stock can pose a serious threat to the genetic integrity of wild populations. Adaptation to local conditions may be disrupted by the introduction of stock from distant sources, and could potentially reduce the overall viability of natural stocks (Kearney and Kildea, 2001).There is a great need to monitor the Murray cod population size and structure in the Lower Darling River, in order to determine the effect of kills of this magnitude on the ecology of this system.

6. FUTURE MANAGEMENT GUIDELINES

The available evidence indicates that the fish deaths occurred as a consequence of depletion of dissolved oxygen, with the possible added stress of chemical toxins released from anoxic sediments or anoxic hypolimnetic waters. Major risk factors for future oxygen depletion-related fish kills, as described above, include:

- high temperatures in periods of low or no flow.
- high organic loading and "black water" events.
- algal blooms.
- chemical toxicity resulting from hypolimnetic stratification.

If future fish kills are to be avoided, decisions in regard to the future management and monitoring of the Menindee Lakes and the Lower Darling River system should be made within the context of the likelihood of similar events of this magnitude recurring. As such, the following recommendations are suggested as a guide to reduce the risk of significant fish deaths in the future.

1. Collection of baseline data

Collection of baseline thermal stratification, water quality and sediment quality data from a range of sites above Weir 32, above Menindee Main Weir (in Lake Wetherell and the Darling River Channel), and along the Lower Darling River is recommended. Data should include collection from a range of temporal (seasonal) and flow conditions. This would assist identification of possible causes of low dissolved oxygen and poor water quality in the Darling River at different times of year, and under a range of flow conditions.

2. Continuous and real-time water quality monitoring

Continuous and real-time monitoring of water quality and algal numbers above and below storage and release points at Weir 32 and Menindee Main Weir, and along the Lower Darling River particularly in warmer months and periods of low or no flow is also recommended. This would ideally monitor dissolved oxygen levels (early morning when levels are usually at their lowest), water temperature, pH, sulfide, and ammonia. More frequent and rapid processing of blue-green algal abundance data during warmer periods, to provide early determination of bloom existence, should also be incorporated in the monitoring program.

Although extensive routine monitoring is impractical, the importance of some form of continuous monitoring is highlighted by the short-lived nature of the fish kill event. Infrequent monitoring creates the possibility of detecting major events too late to change operating systems and minimise the effect on downstream aquatic organisms.

As a minimum, a more frequent dissolved oxygen monitoring program should be considered to provide early warning of potential events and allow time for management responses to minimise potential impacts. It would be preferable to record dissolved oxygen levels at several depths, but at the least, the collection depth should correspond to the depth at which water is drawn from each weir and transferred downstream to the Darling River under normal operating procedures. 'Trigger levels' could be developed which initiate an investigation if breached.

Any investigation should comprise more detailed monitoring to determine the source of the low dissolved oxygen concentrations and, based on that information, identify possible remedial actions. Automatic water quality loggers could be deployed at strategic points in the Menindee Lakes and Lower Darling should future funding permit.

3. Water quality of released water

Monitoring flood pulses as they progress downstream with regular water quality testing for dissolved oxygen depletion, water temperature, algal levels and anoxia related toxins is also suggested. Monitoring should focus at and behind the pulse front to provide early warning of potential events, and allow time for management responses to minimise potential impacts. One suggested strategy for reducing the depletion of oxygen in the pulse front would be to aerate water coming downstream through instillation of aerators. Several locals suggested that a coordinated synchronisation of landowner's irrigation pumps in reverse as the pulse passed could have the effect of aerating the pulse as it passes down stream (D. Freeman personal communication). It is not clear if such an action can significantly increase water column dissolved oxygen levels above critical levels, and future work in this area may yield important short-term solutions for minimising fish kill impacts.

4. Water storage volumes

Where practical, fish kills may be avoided by increasing water storage levels in the Menindee Lakes to secure water supply for Menindee and Broken Hill, and preserve water quality for downstream environmental releases at volumes large enough to minimise oxygen depletion in flow pulse fronts. In December 2003, releases to the Darling River from Menindee ceased when total capacity of the lakes fell below 2.3%. Where possible, water use restrictions could be implemented to maintain the residual volume in the Menindee Lakes at a considerably higher level. This would in turn allow environmental releases downstream, when required, to be large enough to provide sufficient mixing and dilution of poor water in remnant pools.

Larger release volumes would also dilute the concentration of toxins released in and transported from hypolimnetic pools behind the Menindee Main Weir, Weir 32, or along the Lower Darling River, that might be "turned-over" as the pulse passes. Smaller releases may be possible in cooler months of the year where

microbial breakdown of organic matter would be slower and less likely to produce anoxia in the flood pulse.

This recommendation recognises the possibly conflicting requirement of DIPNR to ensure the release of potentially saline water from the Lower Darling River, does not compromise water quality in the Darling and Murray Rivers upstream of Wentworth weir pool.

5. Distribution of remnant pools

It is further suggested that surveys be conducted to map remnant pool distribution, size and volume along the Lower Darling River during low or no flow conditions, to provide better understanding of hydrology and water quality variability in the system. This would identify sites susceptible to stratification and potential fish kill events in future periods of low flow, and allow for management responses to minimise potential impacts at these susceptible sites.

6. Murray cod populations

It is recommended that surveys be conducted to assess the distribution, size and structure of remaining populations of Murray cod in the Lower Darling River system. This knowledge would both assist in the understanding of the significance of the February 2004 event, and be a useful management tool in minimising the potential impacts of future management decisions.

7. Fish kill response

If, despite all efforts, similar fish kills occur in the future, it is recommended that appropriately trained departmental representatives attend and investigate fish kills swiftly when they are first reported. Representatives would be required to:

- Discuss the fish kill with appropriate NSW Fisheries managers at the earliest opportunity,
- Locate pulse fronts that may have contributed to a kill event so that downstream implications can be minimised,
- Collect water quality and blue green algal data and analyse and report the results to appropriate managers immediately, and
- Collect samples of dead fish for pathologiczal analysis, as prescribed in the NSW Fisheries Protocol for Investigating and Reporting Fish Kills (NSW Fisheries, 2000).

Conclusion

The Murray cod is Australia's largest and most recognised native freshwater fish, significant both as a top order predator in aquatic ecosystems, and to Indigenous and European communities. Given that Murray cod populations basin wide have declined dramatically since European settlement, the significance of the Lower Darling River fish deaths in February 2004, involving thousands of adult fish over a 160 kilometre stretch of river, is magnified. Such an event is particularly significant to local communities for whom Murray cod hold considerable social value, and also contribute to the attraction of visiting anglers and tourists to the area.

The fish deaths were most likely a consequence of oxygen depletion (due to a combination of extreme temperature, high algal respiration and organic loading), with the possible added stress of sulfide and/or ammonia toxicity released from anoxic sediments in hypolimnetic pools. Given this, the implementation of a management plan centred on fish stock assessment, fish kill response and seasonal and event based water quality monitoring focusing on trigger levels is recommended. Such a program would reduce the risk of similar events in future periods of low or no flow, and would allow for management responses to minimise potential impacts on the aquatic ecosystem downstream should conditions conducive to fish deaths re-occur.

Appendix 1 Estimates of numbers of Murray cod killed

Estimates of number of Murray cod killed in the February 2004 event, based on observer's accounts of number of dead fish per kilometre at each station along the affected stretch of river.

Station	Distance from Weir 32 (km)	Estimated number of dead Murray cod per km in each reach	Approximate length of reach (km)	Number of dead Murray cod in reach
Weir 32 - Wanda	36	0	36	0
Bono	50	0	14	0
Tandou	60	4	10	40
Willotia	69	20	9	180
Bindara	80	30	11	330
Tolarno	88	20	8	160
Willotia	96	20	8	160
Harcourt	123	15	27	405
Whurlie	131	15	8	120
Wyarama	153	10	22	220
Polia	178	6	25	150
Court Nareen	200	4	22	88
Aston	211	3	11	33
Greenvale	215	2	4	8
			total	1894
			Correction for 60% float rate	3157

Appendix 2   Determinants and effects of the main causal factors of the 2004 Darling River fish deaths.

Effects of high temperature with relevance to the Darling River fish deaths.

TEMPERATURE

Determinants

Effects

- Temperature and condition of pools
- In stream temperature
- Shading
- Release water
- **SUN**
- **High Water Temperature**
- Stratification
- Hot dry river bed
- Surface area and volume
- Wind
- Colour and turbidity
- ↑ Microbial and algal respiration
- ↓ DO concentration
- ↑ Fish respiration
- ↑ Oxygen requirement
- ↓ Oxygen solubility
- Physiological stress
- ↑ Pathogen growth abundance
- **Fish suffocation**
- Species specificity
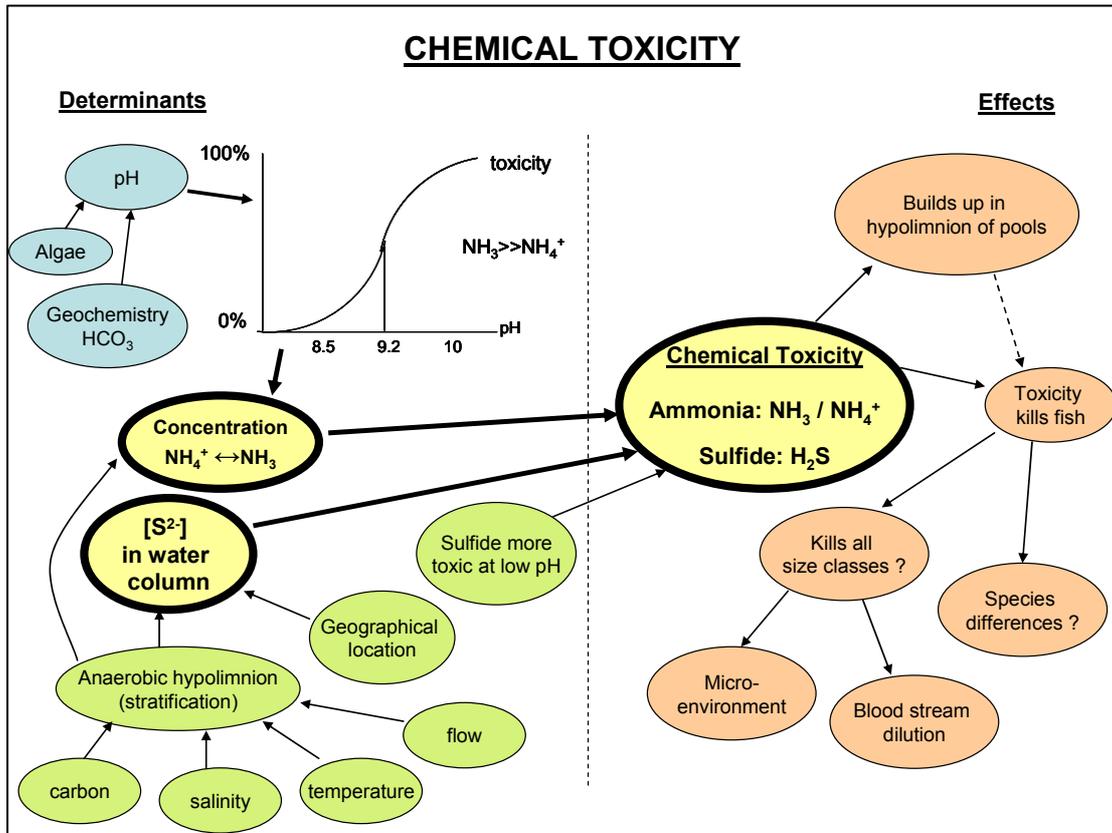- Recent breeding events
- Larger fish die first

Effects of low dissolved oxygen with relevance to the Darling River fish deaths.

DISSOLVED OXYGEN

Determinants

Effects

- Available organic matter
- Bacterial breakdown of organic matter
- Temperature
- **Chemistry:** ↑ NH_3 / NH_4^+, ↓ NO_x, ↑ Fe^{2+} and Mn^{2+} ↓ SO_4^{2-}, ↑ S^{2-}
- See Chemical Toxicity flow diagram (overleaf)
- Respiratory stress
- Stratification (anoxic hypolimnion)
- Salinity (density)
- diurnal
- Available nutrients
- Photosynthetic oxygenation
- Algal abundance
- Wind
- **Low DO**
- **Symptoms:** • open mouth • flared gills • head tilted back
- **Species differences:** • carp more tolerant • yabbies evacuating?
- **Large Fish:** Gill surface area vs tissue weight
- **Large Fish:** Reluctance to leave territory
- **Large Fish:** Unable to access surface water or upstream of front where DO higher
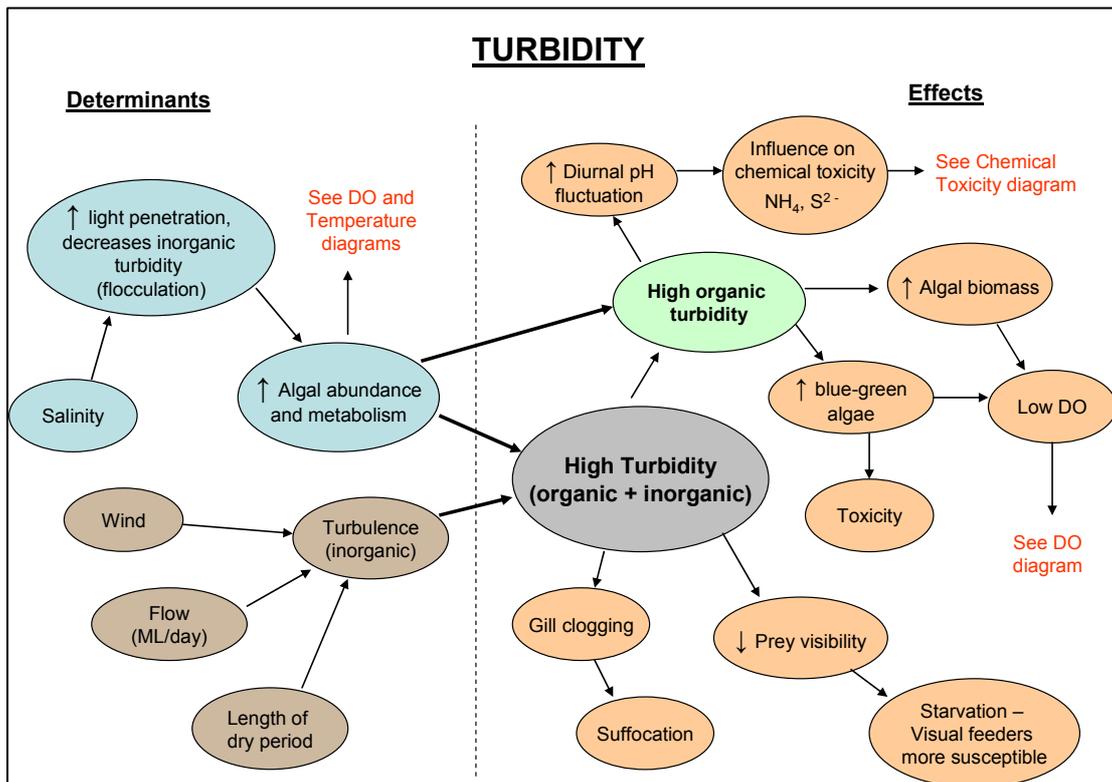- Large fish die first

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The effects of chemical toxicity with relevance to the Darling River fish deaths.

The effects of high turbidity with relevance to the Darling River fish deaths.

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