Department of **Agriculture, Fisheries and Forestry**
Biosecurity Queensland

Fish kill testing summary: Catfish

Boyne River, Gladstone

As at 28 September 2012

Queensland Government

Contents

Background

Following reports of dead fish in early April 2012, Department of Environment and Heritage Protection (DEHP) staff inspected a section of the Boyne River. The staff found over 100 dead forktailed catfish (*Arius graeffei*) between the mouth of the Boyne River and the Benaraby Bridge, approximately 20km south of Gladstone. Many of the fish were noticeably decomposed.

Water quality monitoring undertaken on 4 April 2012 identified reduced salinity levels, due to freshwater inflow from heavy rainfall and subsequent overtopping of the Awoonga Dam on 22 and 23 March 2012. All other monitoring results have found water quality is consistent with those of a healthy waterway[i].

Biosecurity Queensland assists in investigations of fish kill events to exclude exotic disease or pathogens which may have the potential to cause significant infectious disease. Once these have been excluded, further investigation is undertaken to determine, if possible, the cause of a fish kill event.

Samples

On 5 April 2012, 10 dead forktailed catfish were collected by DEHP staff and submitted on ice to the Biosecurity Queensland laboratory for analysis. The fish had varying states of decomposition and preliminary observational assessments of the fish submitted indicated that more than half had poor body condition including a reduction of muscle mass. No general redness was observed.

Of the 10 fish submitted, nine were considered suitable for external examination and of these, only six were in suitable condition for detailed internal necropsy.

Samples from the six fish that were internally examined were submitted for organic chemical residue and metal testing pooled by tissue type for muscle, gill and liver tissue. Samples were analysed for:

- metals (aluminium, arsenic, barium, cadmium, chromium, copper, iron, lead, mercury, nickel, silver, zinc)
- organic pollutants including herbicide and pesticides, namely organophosphorus pesticides, organochlorine pesticides, synthetic pyrethroid pesticides, polychlorinated biphenyls (PCBs) and polynuclear aromatic hydrocarbons (PAHs).

Results summary

Due to the unavoidable deterioration of sample quality prior to examination, the necropsy, histological and bacteriological assessments performed were not able to distinguish subtle signs of disease from background tissue degeneration. The examinations were thus limited to detection of signs of obvious and severe parasitic or infectious disease, and no such signs were detected. More than half of the fish were in poor condition with reduced muscle mass and the fish had mostly empty stomachs. Some gill necrosis, gill fouling with sediment and low parasite loads were also observed. No cause of death was established.

Residue testing showed that metal or organic chemical contamination is unlikely to be the cause of the catfish kill. This is based on the results of comparing concentrations of these chemicals in the gill, muscle and liver tissue of fish from the fish kill with (1) data from the US Army Corp Engineers & United States Environmental Protection Agency (USEPA) Environmental Residue and Effect Database (ERED; US Army Corps of Engineers & USEPA, 2011) and (2) historical data on fish

[i] Department of Environment and Heritage Protection website: http://www.ehp.qld.gov.au/gladstone/fish-kills.html

from Queensland's east coast (Department of Science, Information Technology, Innovation and the Arts (DSITIA); unpublished data).

Significant findings

Veterinary diagnostic assessment

All examined fish had post mortem degeneration (PMD). In some samples, PMD was so severe that no comments were able to be made on the state of the tissues and organs. Even in those with less severe PMD, other than marked pathology or lesions, subtle changes in the organs could not be detected. Other lesions described must be interpreted with caution as they may just be artefacts of PMD.

As more than half of the fish showed weight loss and had mostly empty stomachs, there is an indication this population was not feeding normally. This may indicate compromised health prior to the fish kill. However, this weight loss alone does not explain the cause of the fish kill. Likewise, there was gross appearance of some gill necrosis in two of the fish. This may have been caused by biological, chemical or physical factors but this was unable to be determined at this examination. Since only two fish had this damage, the small amount of gill necrosis was not a common factor in all fish and does not explain the cause of the fish kill.

The histopathology examination revealed a low number of several different metazoan parasites which is expected in wild fish. There were some lesions on the gills, including a hyperplasia (gross enlargement) of gill filament epithelium and, in some fish, an inflammatory cell infiltration or haemorrhage in filament epithelium. These changes have to be interpreted with caution due to the condition of the tissues. All the gills examined did have sediment on and between gill filaments. This fine granular material may have been acquired pre-mortem, at the time of death or post mortem. Nothing specific was detected in the histological examination that would explain this fish kill.

There were no consistent bacterial isolates detected across all fish sampled. This indicates that no bacterial disease was present in the dead catfish. The bacteriology examination identified bacterial isolates in some of the fish that are consistent with post mortem proliferation of these organisms. These results are consistent with the histological finding of bacterial post mortem proliferation seen in some of the fish at histology and therefore not considered significant. No evidence was found that would indicate that bacterial disease was a causal factor of the fish kill. Findings are summarised in Table 1.1.

Table 1.1: Summary of external and internal necropsy findings and histological and bacteriological examinations

Date received and case number	Sample type, number, condition	History	Findings
Date received: 5.4.12 **Case number:** P12-11528	**Sample type:** Whole forktailed catfish *(Arius graeffei)*. **Number:** 10 dead fish were received on ice. **Condition:** Due to varying states of decomposition, only nine were considered suitable for external examination. Of these, only six were in suitable condition for detailed internal necropsy.	**History:** Specimens were collected from a fish kill event observed at the mouth of the Boyne River, Gladstone.	• This species of fish is normally known to migrate between salt water and freshwater. • In general, there were only minor, superficial marks (linear lesions) on the flanks of some of the fish. • No general skin redness was observed. • More than half of the fish had poor body condition with reduced muscle mass. • All of the gills examined had sediment on and between gill filaments. • Two of the nine fish examined externally had localised gill filament necrosis in more than one gill arch. • None of the six fish examined internally had any evidence of normal feeding (e.g. stomachs were largely empty). • All specimens examined had post mortem degeneration. • Low numbers of several different metazoan parasites were observed in various tissues. • The absence of a consistent bacterial isolate in all fish sampled indicates no bacterial disease was present in the dead catfish. • *Vibrio*, *Stenotrophomonas*, *Shewanella* and *Photobacterium* species were isolated and considered to be post mortem growth.

Chemical residue analysis

The suite of organic chemicals analysed included organochlorine pesticides, organophosphate pesticides, pyrethroid pesticides, polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs). Of these only the organochlorine DDE pp was detected in the gill sample at

0.046 mg/kg. This is a breakdown product of DDT which has been phased out of use in Australia and it is likely that it is a residual from historic use. No data for gill concentrations of this breakdown product were available in the Environmental Residue and Effects Database (US Army Corp Engineers & USEPA, 2011) database or in the historical data available for Queensland's east coast (DSITIA, unpublished data).

Comparisons of metal residue results (Table 1.2) with the ERED database indicates that chromium, nickel, arsenic, silver, cadmium and barium concentrations measured in the catfish tissues were not associated with harmful effects (US Army Corp Engineers & USEPA, 2011). In contrast, copper, lead and zinc measured at concentrations that have been reported as harmful in overseas species. However, these metals have been measured at higher concentrations in healthy catfish collected from Queensland's east coast during surveys conducted by the State Government between 1977 and 1980 (this data is available on request from water.data@derm.qld.gov.au). Therefore, it is unlikely that these metal levels are the cause of death in these fish.

Iron was detected in gill tissue in catfish from the Boyne River at a concentration similar to that associated with mortality in laboratory-based experiments with an overseas fish species. The mortality in the overseas species was attributed to physical clogging of the gills by precipitated iron. However, in the Department of Agriculture Fisheries and Forestry pathology report (DAFF, 2012) gill necrosis was detected in only two of the nine fish examined, meaning that it is unlikely that iron contamination contributed to the fish kill.

No data was available from the ERED database (US Army Corp Engineers & USEPA, 2011) or the historical study (DSITIA, unpublished data) for aluminium concentrations in fish tissues, therefore the significance of the concentrations of aluminium detected cannot be determined.

A detailed comparison of tissue metal residue results with data from the ERED database and historical studies are presented in Appendix one.

Table 1.2: Summary of metal concentrations in gill, muscle and liver tissue of catfish from the Boyne River fish kill.

Metal (mg/kg)	Catfish gill	Catfish muscle	Catfish liver
Aluminium	385	3.2	5.9
Arsenic	0.31	0.23	0.15
Barium	9.4	0.2	0.2
Cadmium	<0.01	<0.01	0.05
Chromium	0.41	<0.05	<0.05
Copper	0.64	0.34	3.9
Iron	315	6.7	940
Lead	0.16	<0.05	0.08
Mercury	0.07	0.29	0.55
Nickel	0.17	<0.05	<0.05
Silver	<0.05	<0.05	<0.05
Zinc	100	6.3	330

Conclusion

No definitive cause has been found for the death of forktailed catfish (*A. graeffei*) in early April 2012 between the mouth of the Boyne River and the Benaraby Bridge.

The initial observation that the fish were in poor condition was confirmed with the examinations revealing fish with reduced muscle mass and mostly empty stomachs, indicating a possible absence of normal feeding.

There is no evidence of a bacterial, viral, fungal or parasitic cause of deaths, based on the samples examined and the necropsy and histological and bacteriological examinations performed.

Based on comparison with an international database (ERED) and previous survey data, it is unlikely that metal or organic chemicals measured are the cause of the catfish kill.

References

State of Queensland, Department of Agriculture, Fisheries and Forestry (DAFF), (2012). *Fish kill testing summary: Catfish, Boyne River, Gladstone, As at 15 May 2012.* Accessed 11 July 2012. http://www.daff.qld.gov.au/documents/AnimalIndustries_OtherAnimals/fish-kill-testing-summary-catfish.pdf

US Army Corp and USEPA. 2011. The Environmental Residue and Efffects Database. Available from: http://el.erdc.usace.army.mil/ered/. Last updated October 2011. Downloaded: 10 July 2012.

Appendix one:

Report and interpretation of the fish tissue analyses from catfish killed in the Boyne River

By Dr Susi Vardy and Dr Michael Warne, Catchment Water Science, Water Quality and Aquatic Ecosystem Health, Department of Science, Information Technology, Innovation and the Arts (DSITIA).

Summary

There is little evidence that metal or pesticide contamination is associated with the catfish kill in the Boyne River. This is based on comparing concentrations of these chemicals in gill, muscle and liver tissue from the fish with (1) data in the USEPA Environmental Residue and Effect Database (ERED; US Army Corp Engineers & USEPA, 2011) database and (2) historical data on fish from Queensland's east coast.

Comparisons of the concentrations of arsenic, cadmium, chromium, mercury, nickel and silver in tissues of the fish were either well below the concentrations associated with harmful effects or could not be quantified. It is therefore unlikely that these metals contributed to the fishkill. Comparisons to the ERED database indicate that copper, lead and zinc are at concentrations in fish tissue that have been reported to exert harmful effects in overseas species. However for all three metals, healthy catfish from Queensland's east coast have higher concentrations. Therefore, it is unlikely that these metals are associated with the Boyne River fish kill. Iron was detected in gill tissue in catfish from the Boyne River at a concentration similar to that associated with mortality in laboratory-based experiments with an overseas fish species. The mortality in the overseas species was attributed to physical clogging of the gills by precipitated iron. However, in the Department of Agriculture Fisheries and Forestry pathology report (DAFF, 2012) gill necrosis was detected in only two of the nine fish examined. Therefore, it is unlikely that iron contamination contributed to the fish kill.

The interpretation of the results is hampered by a number of limitations:

- by pooling tissue from several fish into one sample for gills, one for liver and another for flesh, variability in the data is not available
- no statistical analyses can be conducted
- information on the size, age or sex of the fish is not available.

The pooling of tissue to generate a single sample for each tissue type severely limits the usefulness of such data and such practices should be avoided, wherever possible, in the future. Pooling may be required to provide enough sample but if this is the case then more than one pooled sample is required.

Background

In response to a catfish kill in the Boyne River, on 5 April 2012, Queensland Government scientists collected samples of the dead fish and submitted them for a pathology analysis. A report was released in on 15 May, 2012 by the Department of Agriculture, Fisheries and Forestry (DAFF), 2012). At the same time as fish were collected for pathology, samples were sent to the NATA accredited Queensland Forensic and Scientific Services to determine the concentrations of

contaminants in fish organs and tissue. The current report interprets such data and determines the potential for contaminants to have contributed to the fish deaths.

Methods

Six freshly dead fish were collected for contaminant analysis and gill, flesh and liver samples were dissected and pooled. The following contaminants were analysed for in the fish tissue:

- metals (aluminium, arsenic, barium, cadmium, chromium, copper, iron, lead, mercury, nickel, silver, zinc)
- organic pollutants including polychlorinated biphenyls (PCBs), polynuclear aromatic hydrocarbons (PAHs), herbicides, and pesticides including organophosphorus, organochlorine, and synthetic pyrethroid pesticides.

All chemical contaminants concentrations were reported on a wet weight basis.

The results from the tissue analysis were compared to two data sources in order to assess whether the fish deaths may have arisen from exposure to either pesticide or metal exposure. The data sources included:

- The US Army Corp Engineers & United States Environmental Protection Agency (USEPA) Environmental Residue and Effect Database (ERED; US Army Corps of Engineers & USEPA, 2011) (http://el.erdc.usace.army.mil/ered/). This database is sourced from the literature. It contains data from studies where biological effects and tissue contaminant concentrations were simultaneously measured in the same organism. Data for each contaminant were scanned and data which would not be of use for this assessment were not included in the assessment (i.e. no observed effects concentrations (NOECs) and Effective Dose (ED) data relating to biochemical changes). These biochemical effects often indicate exposure to a contaminant only, and are not necessarily related to fish illness, or chronic effects in fish.
- Results from a survey between 1977 and 1980 undertaken by the then Environmental Protection Agency where metal (cadmium, copper, zinc and mercury) and pesticides concentrations were measured in gill, flesh and liver samples from catfish (species unknown) collected in 12 catchments and subcatchments in Queensland (Barnes, Bakers, Fitzroy, Burnett-Mary, Noosa, North Pine, Cabbage Tree Creek, Brisbane River (mouth), Brisbane River (Yerongpilly), Bremer, Logan, Norman and Tallebudgera). Results were sampled from healthy catfish. This data is available upon request from water.data@derm.qld.gov.au.

Results and Discussion

Metals

Aluminium

No data was available from the ERED database or the historical study for aluminium concentrations in fish tissues. Therefore it is not possible to comment on the concentrations of aluminium found in the pooled catfish samples (385 mg/kg in gill tissue, 3.2 mg/kg in muscle tissue and 5.9 mg/kg in liver tissue).

Arsenic

Arsenic was detected in gill, muscle and liver tissue in the catfish from the Boyne River. Arsenic was detected in gill tissue at a concentration of up to 0.31 mg/kg. LD10 values (concentrations lethal to 10 per cent of the test organisms) of arsenic that correspond to gill tissue concentrations for Mozambique Tilapia (*Oreochromis mossambicus*) of 7.27 mg/kg, and LC50 values

(concentrations lethal to 50 per cent of the test organisms) correspond to a gill concentration of 13.47 mg/kg to the same species (US Army Corp Engineers & USEPA, 2011). These concentrations are more than an order of magnitude higher than that detected in the gill tissue of the catfish in the Boyne River. No chronic effects data are available.

LD50s associated with liver concentrations of arsenic between 5.69 mg/kg (Mozambique tilapia - *Oreochromis mossambicus*) and 122.8 mg/kg (Green sunfish - *Lepomis cyanellus*) are reported in the ERED database (US Army Corp Engineers & USEPA, 2011). The concentration measured in the liver of the Boyne River catfish was 0.15 mg/kg – at least an order of magnitude lower than the ERED values. Similarly, the concentration in catfish muscle was over an order of magnitude lower (0.23 mg/kg) than in the concentrations in muscle tissue associated with LD50s for the Mozambique tilapia (ranged between 3.1 and 5.32 mg/kg). No chronic effects data are available for these tissue types.

No data was available for arsenic in the historical study of catfish tissue concentrations collected from Queensland's east coast.

Barium

No data was available from the ERED database (US Army Corp Engineers & USEPA, 2011) or the historical study for barium concentrations in fish tissues. Therefore, it is not possible to comment on the measured concentrations of barium (9.4 mg/kg in gill tissue, 0.2 mg/kg in muscle tissue and 0.2 mg/kg in liver tissue).

Cadmium

Cadmium was not detected in either the gill or the muscle of the catfish (i.e was <0.01 mg/kg).

The measured concentration of cadmium in the pooled catfish liver sample was 0.05 mg/kg. The lowest concentrations reported in liver tissue in the ERED database (US Army Corp Engineers & USEPA, 2011) was a LOED (the lowest dose that exerts a statistically significant effect) associated with a lethal dose of 0.175 mg/kg in juvenile Brook trout (*Salvelinus fontinalis*). This result was 3.5 times the magnitude of the concentrations of cadmium found in the liver of the catfish in the Boyne River.

The concentrations of cadmium in liver tissue of catfish reported in the historical study ranged between <0.025 and 1.61 mg/kg which are similar or higher to those in the catfish from the Boyne River indicating that cadmium was unlikely to be the cause of the fish kill.

Comparisons to both the ERED database and Queensland's east coast fish tissue study indicate that cadmium is unlikely to have caused the fish kill.

Chromium

Chromium was not detected above the limit of reporting (LOR) of 0.05 mg/kg, in the pooled muscle and liver tissues.

Chromium was detected in gill tissue at a concentration of 0.41 mg/kg. A concentration of 5.88 mg/kg of chromium in gill tissue was associated with mortality and growth effects in juvenile Chinook Salmon (*Oncorhynchus tshawytscha*) (US Army Corp Engineers & USEPA, 2011). This concentration was an order of magnitude above the measured concentration of chromium in the catfish gill tissue from the Boyne River. No gill tissue data was available for chromium in the historical study.

The available comparisons indicate it is unlikely that chromium was responsible for the fish kill in the Boyne River.

Copper
Copper was measured at concentrations of 0.64 mg/kg in the gill tissue of the Boyne River catfish. This is similar to the lowest reported effect of 0.65 mg/kg for a LD11 in juvenile rainbow trout (US Army Corp Engineers & USEPA, 2011).

The concentration of copper measured in the muscle of the Boyne River catfish was 0.34 mg/kg. The lowest recorded muscle concentrations associated with an effect were an ED40 growth effect at 0.02 mg/kg (US Army Corp Engineers & USEPA, 2011). The highest measured effect was an LD50 associated with a muscle concentration of 1.64 mg/kg (US Army Corp Engineers & USEPA, 2011). Historical data collected from Queensland's east coast ranged from <0.05 to 9 mg/kg copper in catfish muscle. The copper concentrations in tissue from the Boyne River catfish were at the lower range of this range.

The copper concentration measured in the catfish liver from the Boyne River was 3.9 mg/kg. This was lower than the range of 5.4-80 mg/kg measured in healthy catfish in the historical surveys from Queensland's east coast. Copper concentrations in liver samples associated with effects ranged from 38.85 mg/kg (ED64 for fry of the Common carp) to 480 mg/kg which was the LOED for immature Bluegill in growth, mortality and reproduction.

The above comparisons indicate that copper could have contributed to the catfish kill observed in the Boyne River. However, the likelihood of this occurring is low given that healthy catfish from Queensland's east coast often have higher tissue concentrations.

Iron
No data was available in the ERED database (US Army Corp Engineers & USEPA, 2011) for muscle and liver tissue. The concentrations of iron detected in muscle and liver tissue in catfish collected in the Boyne River were 6.7 mg/kg and 940 mg/kg, respectively.

Iron was detected in gill tissue in catfish from the Boyne River at a concentration of 315 mg/kg. Concentrations between 320 and 1050 mg/kg of iron in gill tissue was associated with mortality LD50 (lethal dose to 50 per cent of the test specimens) in juvenile Brown Trout (*Salmo trutta*) (US Army Corp Engineers & USEPA, 2011). The authors of the study associated the mortality with physical clogging of the gills (Dazall and MacFarlane 1999). In the pathology report released by DAFF (2012) gill necrosis was detected in two of the nine fish submitted for pathology. The authors of the report state 'as only two fish had this damage, the small amount of gill necrosis is not a common factor and does not explain the cause of the fish kill.' Given the lack of gill damage in the fish from the Boyne River fish kill, it is unlikely that iron contamination contributed to the fish kill.

No data was available for iron in the historical study of contaminant levels in catfish from Queensland's east coast.

Lead
Lead was measured at concentrations of 0.16 mg/kg in the gill tissue of the Boyne River catfish. This is around two orders of magnitude lower than the lowest reported effect of 18 mg/kg associated with morphology and development LOED for the Brook Trout (*Salvelinus fontinalis*) (US Army Corp Engineers & USEPA, 2011).

Lead was not detected above the LOR (0.05 mg/kg) in the pooled muscle tissue.

The lead concentration measured in the catfish liver from the Boyne River was 0.08 mg/kg. This compares with the range of <0.1-5 mg/kg measured in healthy catfish in previous surveys of

waterways of Queensland's east coast. Lead concentrations in liver samples associated with effects ranged from 0.393 mg/kg (a growth ED89 for adult bluegill) to 16 mg/kg liver tissue concentration associated with a LD50 for the juvenile Fathead minnow (*Pimephales promelas*) (US Army Corp Engineers & USEPA, 2011).

Overall, the above comparisons indicate that it is unlikely that lead contributed to any significant degree to the fish kill in the Boyne River.

Mercury

Mercury was measured at concentrations of 0.07 mg/kg in the gill tissue of the Boyne River catfish. No data was available for gill concentrations in the ERED database (US Army Corp Engineers & USEPA, 2011) or the historical study of contaminant levels in catfish from Queensland's east coast.

The concentration of mercury measured in the muscle of the Boyne River catfish was 0.29 mg/kg. The lowest recorded muscle concentrations associated with an effect were an ED45 growth effect at 0.8 mg/kg in Chum salmon (*Oncorhynchus keta* in fry muscle tissue) (US Army Corp Engineers & USEPA, 2011). The highest measured effect was an LD25 associated with a muscle concentration of 13.4 mg/kg in the European eel *Anguilla anguilla*. Historical data in healthy catfish ranged between <0.02 mg/kg and 0.5 mg/kg in samples collected throughout Queensland's east coast.

The mercury concentration measured in the catfish liver from the Boyne River was 0.55 mg/kg. This is an order of magnitude lower than the lowest reported concentration of mercury in gill tissue associated with effects (an LOED for growth in Chum salmon (*Oncorhynchus keta*) at a concentration of 0.96 mg/kg in fry liver tissue) (US Army Corp Engineers & USEPA, 2011). This compares with the range of <0.2-1.6 mg/kg measured in healthy catfish in previous surveys.

Given the above comparisons, it is unlikely that mercury contributed to the fish kill in the Boyne River.

Nickel

Nickel was not detected above the limit of reporting (0.05 mg/kg) in the pooled muscle and liver tissues.

Nickel was detected in gill tissue at a concentration of 0.17 mg/kg. Concentration ranges of nickel between 39.09 and 122.02 mg/kg in gill tissue have been reported as being associated with LD50 values for Rainbow Trout (*Oncorhynchus mykiss*) and 202.8 mg/kg as the LD50 for the Common Carp (*Cyprinus carpio*). These results are two to three orders of magnitude higher than those found in the gill tissue of catfish from the Boyne River.

No data was available for nickel in the historical study.

Given the above comparisons, it is unlikely that nickel contributed to the fish kill in the Boyne River.

Silver

Silver was not detected above the limit of reporting (0.05 mg/kg) in the pooled gill, muscle and liver tissues.

No data was available for silver in the historical study.

Given the lack of detectable silver, it is unlikely that silver contributed to the fish kill in the Boyne River.

Zinc

The zinc concentration in the catfish liver was 330 mg/kg. Historical data for healthy catfish livers from Queensland's east coast was between 7.2 and 1000 mg/kg, illustrating the variation between fish and between sites. In contrast, the Boyne River catfish liver zinc concentrations were higher than the lowest growth ED reported (an ED4 – the dose that exerts a 4 per cent reduction in growth) associated with a liver concentration of 34.44 mg/kg in yellow perch (*Perca flavescens*). Measured zinc concentrations in the adult liver tissue of Bluegill was 207 mg/kg, associated with a growth ED89 (the dose that exerts an 89 per cent reduction in growth).

The zinc concentration in the catfish muscle was 6.3 mg/kg. Historical data for healthy Queensland catfish ranged from 0.1-300 mg/kg in muscle tissue. No muscle data for zinc was available in the ERED database (US Army Corp Engineers & USEPA, 2011).

The measured zinc concentration in the catfish gill from the Boyne River was 100 mg/kg, two orders of magnitude higher than the lowest concentrations associated with ecological effects from the ERED database (US Army Corp Engineers & USEPA, 2011) (0.27 mg/kg in gill tissues associated with LD50 in juvenile rainbow trout (*Oncorhynchus mykiss*). No historical gill data was available.

Comparisons to data in the ERED database (US Army Corp Engineers & USEPA, 2011) indicate that zinc could exert some negative effects on the catfish from the Boyne River. However, the data for Queensland's east coast indicates otherwise. Given, the historical data are for healthy catfish from Queensland, greater reliance should be placed on this data. It is therefore, unlikely that zinc contributed in any major degree to the fish kill in the Boyne River.

Pesticides

The suite of chemicals analysed by QHFSS included organochlorine pesticides (OCs), organophosphate pesticides (OPs), and pyrethroid pesticides. Of these only the OC DDE pp was detected in the gill sample at 0.046 mg/kg. This is a breakdown product of DDT which has been phased out of use in Australia and it is likely that it is a residual from historic use. No data for gill concentrations of this breakdown product was available in the ERED database (US Army Corp Engineers & USEPA, 2011) or in the historical data available for Queensland.

Overall, it is unlikely that pesticides were responsible for the fish deaths in the Boyne River.

References

Dalzell, DJB and Macfarlane NAA (1999). The toxicity of iron to brown trout and effects on the gills: a comparison of two grades of iron sulphate. *Journal of Fish Biology.* Volume 55, Issue 2, pages 301–315.

State of Queensland, Department of Agriculture, Fisheries and Forestry (DAFF), (2012). *Fish kill testing summary: Catfish, Boyne River, Gladstone, As at 15 May 2012.* Accessed 11 July 2012. http://www.daff.qld.gov.au/documents/AnimalIndustries_OtherAnimals/fish-kill-testing-summary-catfish.pdf

US Army Corp and USEPA. 2011. The Environmental Residue and Efffects Database. Available from: http://el.erdc.usace.army.mil/ered/. Last updated October 2011. Downloaded: 10 July 2012.