



Gladstone Ports Corporation

Growth, Prosperity, Community.

Our Ref: #951905 JMS

5 June 2013

Senator Doug Cameron
Chair Environment and Communications Legislation Committee
P.O. Box 6100
Parliament House
Canberra ACT 2600

Dear Senator Cameron

Senate Hearing Committee Submissions of Drs Jeremijenko and Landos

Thank you for the opportunity to provide a response to the matters raised by Drs Jeremijenko and Landos in submissions to the Senate Hearing Committee on 23 May 2013.

From our experience Drs Jeremijenko and Landos have developed a particular point of view on dredging in Gladstone and are relentlessly pursuing their perceived concerns without any regard for providing any real evidence that supports their claims. It concerns GPC that these gentlemen continue to present information in the public arena that is either factually incorrect or are assumptions that are not substantiated with the support of any scientifically defensible evidence.

Due to concerns about the claims and conclusions included in Dr Landos' report GPC engaged leading scientists to undertake independent reviews of this paper. Copies of these reviews (by Dr Barbara Nowak from the University of Tasmania and Dr Graeme Batley from CSIRO) are provided for your reference. Both reviewers called into question the scientific rigour of the conclusions drawn by Dr Landos.

"While it is logical to expect that animals in the area of dredging could be stressed and there is a lot of evidence in scientific literature that this can lead to immunosuppression, the report does not provide any direct evidence of either

stress or immunosuppression in the animals from Gladstone as no stress or immune variables were measured". Barbara Nowak

"The weight of evidence was not ever evaluated, but a conclusion was drawn that supported the working hypothesis, based on the sheer number of flawed lines of evidence that were documented". Graeme Batley

A number of the points raised in these submissions relate to human health issues so your letter was forwarded to Queensland Health for review and comment. The response received from Dr James Smith, Director and Public Health Physician at the Central Queensland Public Health Unit is attached for your information. This response does not support Dr Jeremijenko's claims that illness in fishermen was caused by dredging activities. We strongly urge the Committee to contact Queensland Health's Environmental Health Unit for further comment on these issues.

An extensive environmental monitoring program has run since before dredging commenced and will continue well after the completion of dredging operations. Analysis of the data provided by this monitoring program has been undertaken by both independent scientists and government agencies. None of whom have identified any linkage between dredging activities and fish health or human health.

Several targeted investigations, notable, the Independent Panel Investigation into Fish Health and the CSIRO Investigation of Sediment Quality in Gladstone Harbour have drawn similar conclusions.

In their analysis of events in Gladstone both gentlemen have consistently down played the impacts caused by the significant flood events that occurred in Central Queensland in early 2011. These events have been shown to have dramatically affected the characteristics of the natural ecosystem of Gladstone Harbour and those effects persisted for a number of months.

Our detailed response to comments recorded in Hansard is attached to this letter. I trust this information satisfies your request. GPC is readily available to discuss issues raised in this letter should you require further comment.

Kind regards

LEO M ZUSSINO
CHIEF EXECUTIVE OFFICER

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Specific Responses from GPC regarding information provided by Drs Jeremijenko and Landos

Jeremijenko Submission

“the Gladstone Harbour was closed because of sick people”

1. This statement is incorrect; Queensland Fisheries closed the harbour on 16 September 2011 in response to concerns about sick fish. The action was a precautionary one to reduce the risk of exposure by fishers, and was taken after considerable lobbying by the Queensland Seafood Industry Association (QSIA).
2. The harbour was opened three weeks later after testing by Biosecurity Queensland and investigation by Queensland Health indicated no potential for adverse impacts on humans.

“there were over 40 sick fishermen who were exposed to dredge spoil”

1. No evidence has been provided which substantiates this claim. Reference is made to Queensland Health’s response which is attached.
2. At this time the dredging activity in the western basin was limited to a small backhoe dredger operating at low production levels.

Shewenella

1. A simple internet search shows that Shewenella isn’t a rare bacterium. Shewenella putrefaciens is a common bacteria found in fresh, brackish and marine water and mud. There are a number of different strains within the species, so it is referred to as a group. The bacteria are normally found in healthy marine animals and are commonly reported as food spoilage bacteria of fish in the seafood industry.
2. The linkage between Shewenella and a metal rich environment caused by dredging is unsubstantiated.

Skin Infections

1. This matter should be addressed with Queensland Health.
2. No evidence is provided to prove a link between the fisherman's ailment and dredging activities. An article printed in the Gladstone Observer dated 20 May 2011 (attached) outlines how a local man suffered an infection which resulted in the amputation of his leg. The infection was incurred in February 2011, two months before the western basin dredging program commenced. This suggests factors other than dredging may have been the cause of the reported illness.

"...dredge spoil is more dangerous than people are making out"

1. No scientific evidence is provided to substantiate this opinion.

"we should not be using the Great barrier reef as a dredge spoil dump zone."

1. Offshore disposal of dredged material from the Western Basin Dredging and Disposal Project is being disposed of at GPC's East Banks disposal site. This site is within the Port Limits and remote from the Great Barrier Reef.

"...especially the stuff in Gladstone, which is known to have high total metals associated with it"

1. Over 1,000 boreholes were drilled prior to dredging to ascertain the nature of dredge material. Sediment analyses demonstrated that the spoil generated by the dredging was not considered contaminated, and was consequently approved for unconfined offshore disposal. The area where dredging occurred is along the foreshore of Curtis Island, an area that has no existing industrial development nor point source discharges.
2. Ongoing monitoring of metals (both total and dissolved) has occurred throughout the project. Results show no evidence of dredging causing elevated trace metal concentrations in the Harbour waters. Metals in the harbour were found to be relatively low when compared to other industrial harbours from around the world and elevated metal concentrations in close proximity to the dredging were not found.
3. CSIRO conducted sediment quality studies in the harbour over 5 years ago and repeated the studies during dredging. CSIRO concluded that while some metals

levels were elevated they were naturally occurring, not from anthropogenic sources, and did not exceed guideline levels for environmental protection.

4. At the suggestion of the Department of Environment and Heritage Protection GPC commissioned Ecotox Services Australasia Pty Ltd to independently undertake Direct Toxicity Assessment on a number of water samples from Gladstone Harbour while dredgers were operating. One of the sampling sites was immediately adjacent to the dredge head of one of the dredgers. The results of this analysis showed no impact on any of the test species analysed. These included a copepod, a rock oyster, a marine worm, an amphipod, a barramundi and a sea urchin.

“I watched the turbidity carefully. I think you may all have seen these pictures.”

1. GPC has assumed that this reference is to photographs generated from Nearmap.com in July 2011. The photos in question were examined by GPC. Its analysis showed that if a person was to zoom out far enough on this image it became apparent that the image quality was inconsistent. GPC’s analysis is attached for the information of the Committee. We draw your attention to the edges of the images and the contrast between Gladstone Harbour and the outflow from the mouth of the Fitzroy River, north of Curtis Island.

“This limit is too hard, we cannot comply with it; we want to adjust it to a higher level”,

1. Dr Jeremijenko implies that limits could be changed without any due process. This was not the case.

Monitoring occurred prior to dredging to set baselines. This monitoring extended back for many months prior to any WBDDP works. However some additional monitoring sites were required by the Regulator prior to the commencement of any dredging. So, interim compliance triggers were developed based on the limited data set available to enable the project approval to be finalised.

It was always the intention to revise these ‘interim’ triggers once further data was gathered. The process for updating the triggers based on the additional scientific data took much longer than expected (9 months), extending well into the period when dredging works were being undertaken. Triggers at two of the four inshore compliance sites were increased after consideration of additional monitoring data. The Dredge Technical Reference Panel, including EHP and SEWPaC were intimately involved in this process. In fact, under instruction from

EHP some baseline data was actually removed from being considered to reduce the triggers.

Landos Submission

“Around 50 people became ill...”

1. see comments above.

“...satellite images show we have a 34 kilometre dredge plume...”

1. The claims made by Dr Landos of a plume extending 34 km out into the Coral Sea are disputable. This finding was offered in a report ‘Using satellite maps to document the extent of sediment plumes associated with dredging in Gladstone Ports Western Basin, Queensland’ Petus and Devlin (2012) which used MODIS imagery to try and differentiate the dredge plumes. GPC commissioned a review of this report by Australian Institute of Marine Science spatial analysis experts (Dr Richard Brinkman, Mr Craig Steinberg and Dr Lyndon Llewellyn) (copy attached). They concluded that the Petus and Devlin (2012) report suffered from a number of significant limitations. Notably, they concluded:

‘The major limitations found render much of the temporal analysis of TSM dynamics irrelevant, as the dominant natural processes controlling natural variability of suspended sediment concentrations were either omitted or represented incorrectly. Further, the analysis which was intended to establish correlations between TSM and dredging activities was almost entirely qualitative and did not possess the rigour of a thorough statistical analysis’.

2. As part of the EIS and WBDDP a huge investment has been made to understand and simulate the intensity and extent of the dredge plumes. These computer models have been extensively calibrated to provide scientific confidence for projections. The modelling has been undertaken by one of the country’s leading service providers in coastal hydrodynamic modelling (BMT WBM). These models demonstrate that the medium and high intensities of the plume are limited to the Harbour and do not extend into the Coral Sea.

“Harbour sediments contain nutrients.”

1. Dr Landos has ignored the impacts of nutrient contributions caused by major flood events occurring in Central Queensland in January 2011.

2. Excluding periods after major rainfall event, no significant increases in nutrient concentrations have been detected in the Harbour during the dredging work. Furthermore, there have been no significant increases in algal biomass (indicated by Chl a). The claims made by Dr Landos in relation to toxic algal blooms are unfounded.

Marine animal mortalities

1. Despite having comparative data, Dr Landos only used flood related deaths in turtles, etc in Gladstone and erroneously and deliberately misled the public that dredging is the cause of deaths.
2. The exact cause of fish health issue is unknown, however the collective opinion is the major flood event in the Boyne River introduced an enormous number (approx. 30,000) of Barramundi from Awoonga Dam into the harbour.
3. Only major marine deaths were:
 - Commercial fisherman netted from Boyne River and killed turtles
 - Flooding killed seagrass in 2011 and marine life from Townsville to Brisbane were affected

WBDDP Monitoring Program

1. The western basin dredging and disposal monitoring program was designed based on the detailed scientific assessments in the Environmental Impact Statement (EIS). The EIS process describes the dredging activity, locations, and characterises the type of spoil (including a large range of contaminants to meet the National Dredging Guidelines). From this process it identifies the risks to the environment. This formed the basis for the environmental monitoring, resulting in a focus on managing turbidity and maintaining seagrass beds as a key component of the marine ecosystem. The idea of developing a monitoring program to cover every single possible risk (even if it's consider low or no existent) is beyond the scope of any marine environmental monitoring program and is in contravention to the scientific process for managing environmental impacts

ATTACHMENTS:

1. Review of Landos report by Dr Barbara Nowak University of Tasmania
2. Review of Landos report by Dr Graham Batley – Chief Research Scientist at CSIRO.
3. Response to issues raised at the Senate Hearing from Dr James Smith, Director and Public Health Physician, Central Queensland Public Health Unit
4. Gladstone Observer 20 May 2010
5. GPC analysis of Nearmap image July 2011
6. Review of Petus & Devlin (2012) by Australian Institute of Marine Science.

**Scientific Review of:
“Investigation of the Causes of Aquatic Animal Health
Problems in the Gladstone Harbour and Nearshore
Waters”**

(Dr M. Landos, 1 October 2012)

**Prepared by Barbara F. Nowak
University of Tasmania**

This review has been prepared as a consultancy for Gladstone Ports Corporation. The scope of work was to review the Landos (2012) report (Landos, M. (2012) *Investigation of the Causes of Aquatic Animal Health Problems in the Gladstone Harbour and Nearshore Waters* (1 October 2012) Report commissioned by Gladstone Fishing Research Fund to investigate the causes of aquatic animal health problems in Gladstone Harbour and nearshore waters.) and provide specific comment on the suitability of the method to achieve the stated aim, the use of scientific literature, in particular suitability and context of interpretations and the validity of conclusions drawn. The review was prepared on the basis of the report as provided by Gladstone Ports Corporation and published literature. No additional field work or experimental work was undertaken.

Suitability of the method to achieve the stated aim

The aim of the investigation described in the report was to “To undertake a veterinary disease investigation of aquatic animals in and around Gladstone Harbour, with comparison to a remote reference site; and to assess the likely causes of observed diseased aquatic animals using field and laboratory diagnostic tools.”

The report was based on samples from Gladstone and one reference site. Most samples were from Gladstone. No prawn, scallop, coral or turtle samples were collected from the reference site. Most of the examinations were gross (visible to unaided eye - without using any magnification like a magnifying lens or a microscope), some histology was also undertaken. Parasites were identified, many to species level. Samples for microbiology were collected for selected samples (for example barramundi and catfish) and the presence of algae in water was also investigated. All those results were included (for example summary table for algae and full pathology reports for histopathology) in the report. Statistical analyses were used to test some of the data (for example for mud crabs), these analyses were appropriate and properly interpreted. Whenever possible the report refers to water quality and other environmental measurements. The report is detailed and it is well illustrated.

The sampling was limited and at times appeared to be opportunistic. Only two sites (Gladstone and a reference site) were mostly sampled. Some species were available only from Gladstone site. For some species numbers of individuals sampled were low. Ideally, a study to investigate potential environmental impacts on aquatic animal health, should be multidisciplinary, holistic and involve expertise from different areas. In particular an investigation of any fish mortalities is a complex procedure, which involves extensive observations, accurate recording of data, proper use of sampling procedures and use of a broad range of analytical and diagnostic methods (Meyer and Barclay, 1990). Adequate samples and environmental measurements collected as soon as possible are essential to allow broad investigation. Chemical analysis of fish organs, in particular gills can provide evidence confirming the effect of toxicants (Nowak et al 1995, Daglish and Nowak 2002, Daglish et al 2004). Lack of resources is often identified as one of the main constraints

when investigating fish mortalities (Nowak et al 2005). It is possible that most of the shortcomings, in particular limited sampling and analysis, were due to limited resources.

The methods appear to be suitable to achieve the broad aim stated in the report, in particular the veterinary disease investigation. The assessment of the likely causes of observed diseased aquatic animals using field and laboratory diagnostic tools could be improved, in particular better experimental design, larger number of samples (including additional samples from other control sites), more chemical analyses (including analysis of fish and shellfish organs) and more environmental data would improve the investigation. Ecotoxicology testing and laboratory experiments would be useful to provide more information, including interpretation of the results.

The use of scientific literature, in particular suitability and context of interpretations

The report cites 201 publications, most are scientific papers and a few textbooks, government reports and industry reports contribute more than a quarter and fewer than 10% are media items. Large part of the report consists of a literature review, including direct citations. Selected literature is suitable and most of the context of interpretation is appropriate.

While in general the literature is cited correctly, at times the statements from literature are generalised, taken out of context, for example it is stated in the report that: "Palm (2011), Khan (1990; 1991; 2011), Mackenzie (1999), Marcogliese (2005) and Palm and Ruckert (2009) discuss and demonstrate that in wild fish the normal host-parasite relationship can be disturbed by exposure to contaminants. The result is increased prevalence and increased intensity of parasitic infections." However Palm (2011) says: "Consequently, the occurrence of heteroxenous (multi host life cycle) parasites in an area affected by pollution can be related to the number of intermediate hosts at the studied sites (Xinghua 1987; Overstreet et al. 1996). The intermediate hosts may be more sensitive to environmental changes than the parasite, which in the case of endoparasites is buffered from the environment by the host physiology (Paperna and Overstreet 1981)." This means that Palm (2011) recognised that contrary to the statement in the report not all parasitic infections would increase in prevalence and intensity as a result of exposure to contaminants. However, this is a minor problem as the sentence "The result is increased prevalence and increased intensity of parasitic infections" could be accepted as the statement of the author not supported by the literature (there is no reference at the end of that sentence). At the same time it is obvious that the interpretation of the author of the review is not consistent here with the interpretation of the cited reference.

Another example of potential use of inappropriate reference is: "Exposure to metals can also increase skin mucus secretion, encouraging growth of ectoparasitic monogeneans and copepods, which feed on mucous (Bols, 2001)."

The reference cited in the Reference list is: "Bols, N. B. (2001). Ecotoxicology and innate immunity in fish. *Developmental and Comparative Immunology*, 25, 853-873."

This should be:

Bols, N. B., Brubachea, J.L., Ganassin, R.C., Lee, L.E.J. (2001). Ecotoxicology and innate immunity in fish. *Developmental and Comparative Immunology*, 25, 853-873.

The cited review (Bols et al, 2001) does not mention that ectoparasitic monogeneans and copepods feed on mucus, so it should not be used to support this sentence. Furthermore, the cited review mentions complexity of mucus response, including changes in composition and reduction in numbers of goblet cells in some cases (Bols et al., 2001) so this generalised statement does not really reflect content of the cited publication, which states "Toxicants can have complex effects on skin mucus, including effects on its amount, release, physical state, and possibly composition" (Bols et al., 2001).

Validity of conclusions drawn

The conclusions of the report state:

“In considering the weight of available evidence on the potential causes of observed aquatic animal health disorders in Gladstone, FFVS considers these disorders are most likely to be a direct consequence of the Western Basin Dredging and Disposal Project. Specific mechanisms include:

- resuspension and mobilisation of contaminants (metals and metalloids) from sediments;
- increased parasitism due to stress, immunosuppression and external irritation from poor water quality and toxicosis;
- increased boat traffic vessel strike risk;
- noise and
- generation of toxic algal blooms due to disturbance of sediments and release of nitrogen, iron and other nutrients.”

There is no doubt that Western Basin Dredging and Disposal Project resulted in resuspension of sediments, increased boat traffic, increasing risk of boat strikes to turtles and mammals and increased noise from increased boat traffic, pile driving and dredging.

Skin lesions in marine animals, which are mentioned in the report, are likely a result of resuspension of sediment and either physical damage or exposure to contaminants. For example, greenback flounder, *Rhombosolea tapirina* had dermal erosion when exposed for 6 weeks to sediments from a contaminated site in Tasmania, with 67% of fish affected in disturbed contaminated sediment/normal diet treatment and 33 % in contaminated sediment/contaminated diet treatment, fish exposed to the sediment but without physical contact were not affected (Mondon et al., 2001). There is a potential of infections being involved in skin lesions, but this is most likely species-specific (different for different fish species). For example queenfish from Gladstone were reported to show erythema associated with *Lepeophthirus spinifer* (sea lice) with infection intensities of up to 46 copepods and a mean intensity of 21.2 copepods/fish. The relationship between the number of those parasites and lesion intensity in queenfish in Gladstone area shown in Figure 10 in the report is convincing and suggests that skin lesions in this species were associated with sea lice infection. Also skin damage in bull sharks and whaler sharks was reported to be associated with parasitism (*Dermophthirius* sp. *Pandarus* sp.)

Statistical analyses showed that the mud crabs from Gladstone area had significantly greater prevalence of shell disease than the mud crabs from a reference site and that there was a significant relationship between the disease and distance from the dredging site (Figure 11 in the report – although the units on Y axis should be 100 times higher as they are supposed to be presented as percentage). The prevalence reported (37.8%) was greater than observed before (14.3% - Andersen 2003), however the prevalence of shell disease at the reference site was greater (13.7%) than the baseline reported in the literature (5% - Andersen 2003). While copper and zinc were elevated (two to three times greater) in mud crab hepatopancreas from Gladstone compared to Ayr in two consecutive years (1999-2000), the source of copper and zinc could not be determined as water and sediment from crab burrows had low concentrations of metals (Andersen 2003). Most dissolved metal concentrations were significantly elevated in Port Curtis compared to the concentrations measured in the adjacent coastal waters, however these concentrations are at the lower end of the concentration range of these metals measured in industrialised harbours around the world (Angel et al 2010). Experimental exposure to sublethal levels of copper suggested that the pathology was most likely caused by copper exposure (Andersen 2003). It is therefore likely that this disease is caused by exposure to elevated levels of copper.

The other conclusions in the report are generalised and extrapolated from literature but not necessarily based on the results of this investigation. For example, Figure 8 in the report shows working hypothesis of causal pathways, which in general is logical and convincing,

however it is assumed that increased prevalence/intensity of parasitic infections is directly related to immunosuppression. While it is logical to expect that animals in the area of dredging could be stressed and there is a lot of evidence in scientific literature that this can lead to immunosuppression, the report does not provide any direct evidence of either stress or immunosuppression in the animals from Gladstone as no stress or immune variables were measured. Ideally, blood samples should be taken and tested for cortisol level to measure stress and for immune function to measure immunosuppression. The author suggests that parasitism is a secondary measure of immunosuppression and that the resuspension of the sediments resulted first in immunosuppression of the aquatic animals and that caused increased parasitism as a result of exposure to contaminated sediments. It is true in some cases. For example Pacific sand lance, *Ammodytes hexapterus*, exposed for 5 weeks to sediment contaminated by oil had lower phagocytic function of blood cells (indicating immunosuppression) and increased infection with monogenean *Gyrodactylus* sp. (indicating increased parasitism, Moles and Wade 2001). However, anthropogenic changes can have different outcomes depending on the parasite (Table 1) and the change (type of disturbance and pollution).

Table 1. Generalised outcomes of pollution effects on parasitism

Effect of pollution	Outcome	Examples of parasites	Reference
Immunosuppression of host	Increase in parasitism	Monogeneans	Khan and Tullin 1991, Moles and Wade 2001
Altered host density	Reduction in parasitism	Tapeworms, Digeneans (for example blood flukes), nematodes, acanthocephalans	Hechinger and Lafferty, 2005
Direct beneficial effect on parasite	Increase in parasitism	Ciliates	Ogut and Palm 2005
Direct toxic effect on parasite	Reduction in parasitism	Monogeneans	Gheorghiu et al 2007

The report states that increased parasitism is an outcome of exposure to contaminants. However, parasite numbers can decrease, increase, or remain unaffected by anthropogenic pressures on the oceans (Lafferty 2003). For example, significantly higher prevalence and mean intensity of cestode *Lacistorhynchus dollfusi* and nematode *Anisakis* sp. were reported in Pacific sanddab *Citharichthys sordidus* at the non-outfall stations compared to outfall stations in Santa Monica Bay, most likely due to changes in invertebrate population densities (intermediate hosts for both these groups of parasites are invertebrates) in response to the presence of contaminants (Hogue and Swig 2007). The outcome of the presence of contaminants can be affected by the parasite species and its life cycle. Meta-analysis of published data showed that aquatic parasites differed in their susceptibility to specific types of pollution, and that in general freshwater parasites appeared to be more responsive to contaminants than those in marine environment, possibly due to greater complexity of marine environment (Blonar et al 2009). The same analyses showed that in general parasitism decreases at polluted sites, particularly for metals and acidification and for

Acanthocephala and Digenea among the parasite taxa (Blanar et al 2009). For example, prevalence and intensity of parasitic infections were lower in oil-treated (through contaminated sediment or water soluble fraction) winter flounder (*Pseudopleuronectes americanus*) naturally infected with a digenetic trematode (*Steringophorus furciger*), and Atlantic cod (*Gadus morhua*) infected with an acanthocephalan (*Echinorhynchus gadi*), (see Khan and Kiceniuk, 1983). The meta-analysis also showed that parasitism increased at polluted sites in cases of eutrophication and for microparasites, including unicellular parasites such as Apicomplexa, Ciliophora, Mastigophora, as well as Microspora, and Myxozoa (Blanar et al 2009).

The report assumes that all parasites benefit from exposure to contaminants. The effect of contaminants on the parasite is partly determined by the parasite's life cycle. Different parasites have different life cycles and while some parasites can benefit from toxic and eutrophic environment, others will be adversely affected by pollution and environmental disturbance, sometimes through the effect on their hosts (Table 1). In some cases the intermediate or final host can be removed from the system and result in reduced parasitism. This is confirmed by the report on page 40 stating "Exposure of fish to pollutants is known to generate an increased infection pressure of monoxenous parasites (species with only one host, such as monogeneans like *Dermophthirus* sp. and copepods like *L. spinifer*), and a decreased infection pressure of heteroxenous parasites with complex life cycles (Khan, 1991; Diamant, 1999)." The monoxenous taxa responded to more types of pollution, with generally stronger effect sizes, than the heteroxenous taxa in meta-analysis of pollution related studies on parasites (Blanar et al. 2009). However, the statement about monoxenous parasites is only partly true as some parasites are more sensitive to contaminants than their host (MacKenzie et al., 1995). The numbers of those sensitive parasites would decrease if the water was contaminated with that particular chemical. In fact, most parasites appear to be sensitive to toxicants (Lafferty et al., 1997; Lafferty et al., 2008).

Another scenario not considered by the report is the implication of direct effects of contaminants on host populations. If it is the host which is affected by anthropogenic change and its populations decrease it may have adverse effects on parasitism. For example fishing can reduce parasitism, either simplifying food webs or reducing availability of hosts required by parasites, particularly those with complex life cycles (Amundsen and Kristoffersen, 1990; Kuris and Lafferty, 1992; Ward and Lafferty, 2004; Lafferty et al., 2008). This is due to reduced parasite transmission efficiency due to the decline in host abundance (Dobson and May, 1987), or the decrease of the diversity of host reducing diversity of parasites with complex life cycles (Hechinger and Lafferty, 2005) or the shift to younger, smaller individuals in the fished populations (Sala and Knowlton, 2006), which usually have fewer parasites (Combes, 2001).

Even if we assume as the report suggests that the animals were stressed, we still cannot use the stress as the basis for prediction of the effects of stress and infectious disease at the population level (Lafferty & Holt 2003). Although many interactions between stress and disease are possible, based on simulation models, stress seems most likely to reduce the impact of infectious diseases as long as transmission is closed (infections tend to be transmitted between members of the same population) and host specificity is high. Stress will generally increase the impact of infectious diseases not specific to a host or that have large (often relatively resistant) reservoir populations (Lafferty & Holt 2003). Stress usually increases the impact of noninfectious diseases. There is also a relationship between transmission and host density. Usually transmission increases as host density increases, allowing for higher contact rates between infected and uninfected individuals (Anderson & May 1986). Thus, dense populations are more likely to have more parasites (Arneberg et al. 1998), meaning that some epizootics could be due to increasing host density, not due to the outside stressors.

Many of the histological changes described in the animals from Gladstone area are mild and affecting half of the examined animals or fewer than 50% (Table 4 in the report). Most of them could have other aetiology than proposed by the report, those other possible causes of these changes and parasitic infections were not considered by the author of the report. Very few individuals were examined histologically from the reference site. Fish collected at the reference site are not pathology free and some of the changes are the same as in fish from Gladstone area. For example, histologically examined sharks from both Gladstone and reference site had mild non-suppurative gastritis with intralesional coccidian or barramundi from both sites had mild mixed to granulomatous gastritis/enteritis with intralesional larval nematodes and mild to moderate visceral and lamellar granulomas within intralesional sanguinicolid eggs or mullet from both sites had visceral myxozoan cysts (Table 4 in the report). While this is expected in the wild fish populations, it is not really acknowledged by the report, which appears to assume that all pathology and the presence of parasites is caused by activities in Gladstone Harbour. The main difference is the presence of skin lesions present only in fish from Gladstone. These skin lesions (dermatitis) were present in just under half of the fish and then just about half of them had ectoparasites associated with the dermatitis (Page 56 of the report – “Histology of Gladstone fish and sharks sampled by FFVS, revealed the presence of dermatitis in 20/42 fish sampled, with 8/20 associated with ectoparasites”). Statements of high parasites intensities grossly and in histology (Table 1 in the report) are generalised and not supported by results for most species sampled. While this is stated in some sections of the report (for example page 23 “coincident with appearance of marine parasites on some, but not all affected fish”), more generalised statements are made elsewhere (for example Table 1 in the report).

Some of the conclusions presented in this report are valid, for example there is no doubt that Western Basin Dredging and Disposal Project resulted in resuspension of sediments, increased boat traffic, increasing risk of boat strikes to turtles and mammals and increased noise from increased boat traffic, pile driving and dredging. However, there are some oversimplifications and generalisations in the report. That includes an assumption that contamination of marine environment results in an increase of parasitism. With a few exceptions only one reference site was sampled, which means that it is possible that any differences between the two sites may be a result of other differences than the dredging and disposal project.

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Scientific Review of:
**Investigation of the Causes of
Aquatic Animal Health Problems in
the Gladstone Harbour and
Nearshore Waters** by Matt Landos

Graeme Batley

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Prepared for Gladstone Ports Corporation

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Executive summary

In 2012, Dr Matt Landos, a veterinary pathologist and Director of Future Fisheries Veterinary Service Pty Ltd (FFVS) Future Fisheries Veterinary Service (FFVS), produced a report for the Gladstone Fishing Research Fund entitled '*Investigation of the Causes of Aquatic Animal Health Problems in the Gladstone Harbour and Nearshore Waters*'. In March 2013, I was asked by Gladstone Ports Corporation to undertake a review of this report.

The working hypothesis of the author was that causal pathways all stemmed from '*dredging and disposal resuspension of contaminated sediments*'. The report relied upon a weight-of-evidence approach to draw conclusions in support of this hypothesis. The conventional applications of multiple lines of evidence in a weight-of-evidence assessment use measured data to support each line of evidence. The approach adopted in the Landos Report was to present literature data in support of observed impacts on particular aquatic biota, often obtained under conditions that differed from those in Gladstone Harbour and mostly at higher contaminant concentrations than those reported for the dredging site, in an approach that falsely implied cause and effect. The weight of evidence was not ever evaluated, but a conclusion was drawn that supported the working hypothesis, based on the sheer number of flawed lines of evidence that were documented.

All of the reported data on dissolved metal and metalloid concentrations in the waters and sediments of Gladstone Harbour indicate only marginal exceedances of the Australian and New Zealand water quality guideline trigger values, and there were definitely no concentrations that would result in chronic toxicity effects on fish. Concentrations of metals and metalloids in the dredged sediments were well below sediment quality guidelines and typical of slightly-to-moderately disturbed environments. In terms of contaminant concentrations, Gladstone Harbour would not rank as a site of environmental concern compared to a great many harbours worldwide.

Potential effects from hydrogen sulfide, acid sulfate soils, ammonia and cyanide highlighted in the Report could be easily dismissed as minor environmental concerns, with concentrations unlikely to reach those quoted in the studies referenced in the Report.

While there is evidence in the literature of immunosuppression in aquatic organisms caused by metals and metalloids, the data are sparse and the concentrations at which effects were observed are above those measured in Gladstone Harbour waters.

Surprisingly the author chose to dismiss freshwater inputs as a cause of fish disease, despite greater than usual rainfall at the time of disease outbreaks. Often the cause of environmental effects is the result of multiple stressors of which freshwater and associated turbidity would rank highly, along with stormwater runoff of contaminants, licensed discharges, excessive fish stocks, food scarcity. Dredging activities may be an additional stressor, but on the basis of monitoring evidence, would not appear to be a major causal factor. More research generating sound cause and effect evidence is clearly needed.

1 Introduction

Gladstone Ports Corporation (GPC) is currently undertaking a substantial dredging project in Gladstone Harbour (Western Basin Dredging Project). The purpose of the dredging project is to deepen and widen existing shipping channels and swing basins and create new shipping channels, swing basins and berth pockets. During the course of the project, a number of independent parties have raised concerns about the project and its potential impact on the Western Basin. In late 2011, incidences of disease, including bulging/red eyes, blindness, severe skin lesions and skin discolouration were reported in barramundi and other fish species being caught in Gladstone Harbour, leading to an independent investigation into its causes by the Queensland Government (Gladstone Fish Health Scientific Advisory Panel, 2012). As early as 2003, Andersen (2003) had identified shell disease in crabs from the Harbour.

In 2012, Dr Matt Landos was commissioned by Gladstone Fishing Research Fund to investigate the causes of aquatic animal health problems in Gladstone Harbour and near-shore waters. The report produced from this study *'Investigation of the Causes of Aquatic Animal Health Problems in the Gladstone Harbour and Nearshore Waters'* (Landos, 2012) claims to present a scientifically based weight-of-evidence assessment. The stated objective of this report was to undertake a veterinary disease investigation of aquatic animals in and around Gladstone Harbour and to assess the likely causes of observed diseased animals using field and laboratory diagnostic tools. The author's working hypothesis was that causal pathways all stemmed from *'dredging and disposal resuspension of contaminated sediments'*.

In March, 2013, I was contacted by GPC and requested to carry out an independent review of the Landos Report to establish the scientific credibility of the assessment approach and the conclusions drawn. The focus of this review will be on water science and environmental chemistry.

As expected, the report contains considerable detail on the pathology of the observed impacted aquatic animals, together with discussion of potential causes. While veterinary pathology is outside of my area of expertise, I have over 40 years' experience in the study of contaminant fate and impacts in aquatic systems, and am an internationally recognised expert in the area, so am able to authoritatively comment on the conclusions drawn with respect to contaminants. I understand that a second reviewer will be addressing the issues to do with fish pathology.

1.1 Scope of work

The specific scope of the review as stated by GPC was to:

1. Review the Landos (2012) Report and provide specific comment on:
 - Suitability of the method to achieve the stated aim
 - The use of scientific literature, in particular suitability and context of interpretations
 - Validity of conclusions drawn
2. Review the report database provided by GPC and identify additional reports required for supporting the review.
3. Write a plain English concise, but comprehensive, review report that addresses the key requirement outlined in Part 1.

2 Overview of the Report and the Approach to the Review

The Landos Report contains the results of veterinary pathological investigations conducted by Future Fisheries Veterinary Service (FFVS) into disease and lesions in aquatic organisms in and around Gladstone Harbour. The aim was to not only undertake these investigations but to also assess the likely causes of the observed diseases using field and laboratory diagnostic tools.

The report covers 287 pages, the first 105 pages containing results and discussion sections, with 201 references to reports and other scientific publications. Greater details on pathological investigations are contained in 11 appendices.

The results section (termed '*Results and Weight of Evidence Assessment*') reports data on water quality, algal analysis, gross pathology and histopathology. No water quality data were measured, but reference is made to Gladstone Area Water Board (GAWB) water testing results for 2011 (GAWB, 2011) of turbidity, suspended solids, nitrogen, and phosphorus which 'all peaked at levels which were above historical data'. For these analytes, the only data presented in the Report were for turbidity (Landos 2012, Appendix 10).

In the discussion of water quality, there is also reference to Department of Environment and Resource Management (DERM) data for metals which according to the Landos Report demonstrate that '*numerous dissolved metals exceeding the ecosystem ANZECC guidelines in Gladstone Harbour*'. Data are not provided in this section but a short table is presented much later in the Discussion Section containing maximum concentrations only.

The '*Discussion – weight of evidence*' section then proceeds to discuss a series of lines of evidence, based on a sequence of considerations of each of the target aquatic organisms: fish; mud crabs – evidence of impacts; shellfish – scallops and oysters; coral; turtles, dolphins and dugongs; algae; and seagrass – evidence of impacts. Separate sections deal with: Foreseeable effects of dredging and Harbour development; Historical contaminants in Gladstone Harbour; Predictable acid sulfate soil disturbance consequences; Post-dredging changes to metal levels in Gladstone Harbour; Specific metal elevations in Gladstone Harbour: potential impacts; Aggregate effects of some metals; and Noise – underwater acoustic impacts. Conclusions were reached within each discussion section, and these are shown in bold in the Report after the evidence had been discussed. I have underlined the language used to reflect the author's interpretation of the certainty of the evidence. The list is as follows:

1. **Increased rates of disease and lesions observed in fish are highly likely to be due to exposure to sub-optimal environmental conditions including increased exposure to toxicants, elevated suspended sediment loads and increased noise-based stressors. The parasitic infestations are likely to be secondary sequelae to the stress and immunosuppression of the host from exposure to poor water quality.**
2. **Increased exposure to metals including copper and zinc are likely to be responsible for the increase in shell disease in mud crabs. It is highly likely that future crab production will be seriously impacted by the sustained poor water quality generated by the dredging and disposal project'**
3. **Shellfish populations exposed to the increased sediment load generated by sediment dredging are likely to have been severely impacted. The timing of mortality of oysters suggests that**

sediment resuspension and increased metal exposures from the dredging and disposal project has contributed to oyster mortalities.

4. Ocean disposal of fine sediments is likely to have contributed to disease in corals through deposition of organically enriched sediments onto coral in addition to the toxicological effects of released metals and metalloids.
5. The sharp increase in turtle, dolphin and dugong mortalities was a direct consequence of the dredging and disposal through toxicoses and secondary immunological compromise and secondary diseases.
6. Deterioration of water quality due to sediment resuspension is likely to be a significant factor in the decline and hindering the recovery of seagrasses.
7. The loss of habitat and its associated food sources and shelter are likely to have generated stress in aquatic biota which relied upon those areas. Permanent loss of fishery productivity will predictably follow this loss of habitat.
8. The already observed declines in fish, prawns and crabs observed in Gladstone, are due to a combination of increased disease and mortality rates, in part recruitment failure, also entrainment in hydraulic dredging activities, and movement away from the noise and poor water quality. Given the overlay of the dredging plume over key nursery area of the Western Basin, the Narrows, South Trees Inlet, Colosseum Inlet, 7 mile inlet and Rodds Bay and its extended duration, FFVS expects serious deleterious impacts on fishery recruitment as a consequence of sediment resuspension.
9. There is considered to be a high risk from ammonia release from the dredging activities
10. Even mild reduction in dissolved oxygen observed post dredging may be biologically significant to exposed biota, as gill function may be compromised from increased suspended sediments and compounding impacts of contaminants such as aluminium, arsenic, zinc and copper.
11. Biota in the area of dredging may have been exposed to stressful or lethal levels of hydrogen sulfide released from dredged sediments, and potentially toxic levels of cyanide from Orica releases above their licence limits.
12. There is insufficient evidence to implicate PAHs or PCBs with a primary role in observed aquatic animal diseases
13. The resuspension of sediments, in particular potential acid sulfate soils, has facilitated the release of large loads of metals and metalloid compounds into more bioavailable dissolved forms. The measured elevations of metals in late 2011 by DERM, compared to historic levels, provide evidence of these elevations taking place after the commencement of dredging.
14. In some areas of the Harbour very substantial concentrations of metals and metalloids were available to be resuspended and depending on a range of other conditions, leading to mobilisation in association with dredge plumes (Petus and Devlin, 2012). DERM/GPC monitoring for metals was infrequent. Hence numerous time points were not sampled. As such, peak values around the times when aquatic animals were reported to be sick, may not have been documented. The DERM monitoring did not use the appropriate metal analysis method for dissolved metals, until after aquatic animals were already being reported to be sick. As soon as monitoring commenced, elevations over ANZECC guidelines in several dissolved metals were reported.
15. There were significant levels of contamination of metals and metalloids detected in the Western Basin Dredging and Disposal EIS sediment assessment in some areas of the harbour. Substantial historical test data are available to compare results from sampling after September 2011 when aquatic animal disease events were occurring. It is the opinion of FFVS that the

origin of some metals and metalloids has not been adequately studied to determine the contribution, if any, from local industry compared to natural geological sources.

16. FFVS does not agree with the DERM/DEHP interpretation that the recorded exceedances are unimportant in terms of causation of observation aquatic animal health problems. Poulsen and Escher (2012) and Berntssen, Kroglund, Rosseland and Wendelaar Bonga (1997) demonstrate that the levels of metals observed in Gladstone Harbour are sufficient to be biologically active and stressful to fish. The increased general disease rate, and increased parasitic intensities observed by FFVS and Fisheries Queensland provide substantial evidence to support this hypothesis.
17. It is the opinion of FFVS that animals exposed to elevations in dissolved copper levels, particularly those above the ANZECC guideline, are likely to have incurred a physiological stress. In combination with other stressors outlined, this is likely to have led to immunosuppression, and can explain the increased intensity of parasitic infestations observed. It is also likely to have contributed (in addition to other metals like zinc) to the increased rust spot disease observed in mud crabs.
18. It is the opinion of FFVS that substantial elevations in dissolved aluminium are likely to be the result of resuspension and release of aluminium from Harbour sediments due primarily to dredging and spoil disposal, with contributions also from local industry. This has likely resulted in significant impacts on the skin and gills of affected fish, causing direct effects such as increased mucous secretion, olfactory cell damage, as well as osmoregulatory and respiratory stress. This stress has contributed to immunosuppression of fish, leading to increased intensities of parasitism, and may also be contributing to failure of some fish to feed.
19. Fish exposed to elevated levels of arsenic are likely to experience sub-lethal stress, which in turn elicits increases in oxidative stress, rendering the exposed animals less capable of defending themselves against pathogens such as parasites. Exposure to arsenic may also have contributed to the increased mucous production on the fish, in addition to elevated aluminium and toxic algal blooms.
20. Increased exposure of mud crabs to dissolved zinc, which has been mobilised by dredging and resuspension of sediment, is likely to have contributed, in combination with other stressors, to the increased rates of shell disease (rust spot) in Gladstone crabs.
21. Increased availability of iron, together with elevated nutrient levels in the harbour, are likely to have provided conditions suitable for blooms of the cyanobacterium *Lyngbya*, which has been associated with skin lesions on fishermen (Figure 10), and other toxic phytoplankton blooms.
22. As highlighted in this FFVS report, a multiplicity of stressors have been generated in Gladstone Harbour, secondary to this Harbour development project which are likely to have a cumulative impact on the biota. One of the potential consequences of immunosuppression is increased intensities of parasitic infections, with concomitant increased pathological impacts. Such changes were observed in samples collected and examined by FFVS from Gladstone compared to the reference site.
23. The aggregate effect of metals, contaminants, turbidity and noise are likely to have contributed to the alterations in fish/other aquatic animals (cetaceans) behaviour in the Gladstone area, with animals either moving away, or not entering many areas they usually inhabit.
24. FFVS concludes that the available evidence does not support the hypothesis that the freshwater influx, or the influx of barramundi, caused the widespread aquatic animal disease

event in Gladstone Harbour and nearby waters. Both were contemporaneous, rather than causal.

In deriving the above list, references have been made to other studies where potential impacts have been suggested, and sometimes proven for similar environmental circumstances. These need to be carefully assessed to determine whether possible explanations in the literature have been transposed as positive evidence to strengthen a case for likely impacts in Gladstone Harbour.

A summary of the assessment provided in tabular form in the Executive Summary of the Landos Report, is shown in Table 1.

Table 1. Weight of evidence assessment of the plausibility and likelihood of various risk factors in disease induction in Gladstone Harbour (from Landos, 2012)

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as casual	Likelihood of significant causal role
Freshwater	Nil. Based on the lesions observed in fish and crabs detailed in histological reports which cannot be caused by freshwater exposure.	Nil. Timing does not fit with outbreak. Areas remote to Gladstone with similar or larger volume of freshwater influx had no similar aquatic animal disease outbreaks. Previous freshwater influxes into Gladstone have not precipitated any similar events. Freshwater alone does not cause rust spot lesions in crabs, nor dermatitis in fish. Freshwater is lethal to the marine parasites observed on barramundi and bull shark skin, yet the parasite intensities were high.	Very low.
Metals exposure immuno-suppression	Yes. Exposure and uptake can trigger immunosuppression.	Yes. Multiple exceedances of ANZECC guideline of multiple metals over extended periods of time coincident with dredging. Elevated metals levels detected in some tested biota (low sample numbers limit power of analysis). Dissolved metal levels in water were lower prior to dredging. High parasite intensities identified grossly and in histology are consistent with fish, turtle and scallops that are immunosuppressed.	Very high.
Metals exposure direct tissue effects	Yes. Exposure to elevated levels of dissolved metals can cause acute effects on fish skin and impairment of olfactory function.	Yes. High levels (inc aluminium, zinc, arsenic and copper) recorded by DERM/DEHP, sufficient to cause observed symptoms in fish sharks and rays, including increased mucous secretion, skin lesions, and thin body condition due to failure to feed.	Very high.

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as casual	Likelihood of significant causal role
Metals exposure shell disease in crabs	Yes. Uptake of elevated levels of copper and zinc can induce rust spot lesions in crabs.	Yes. Prevalence survey indicates elevated prevalence of rust spot in Gladstone mud crabs compared to reference site. FFVS pathology demonstrated lesions in Gladstone crabs are morphologically consistent with metal induced endocuticle shell disease. Post dredging elevations in dissolved copper and zinc reported by DEHP.	Very high.
Toxic algae (fish impact)	Yes, plausible cause of fish skin and gill damage, and elevated blooms can trigger irritation/stress/death.	Limited, details of reports of algal blooms have not been released by GPC/DERM. Yes, elevated Chlorophyll monitoring levels. Blooms not detected in all locations where FFVS identified sick fishes. Toxic algae not known to cause shell lesions in crabs.	Uncertain (likely only partial role)
Toxic algae (human impact)	Yes. <i>Lyngbya</i> known for its dermatotoxins.	Yes. <i>Lyngbya</i> identified in association with sick fishermen, and in algal monitoring study by FFVS. Lesions in many people consistent with <i>Lyngbya</i> exposure after the commencement of major dredging and never before in Gladstone Harbour fishers	
Turbidity-increased suspended sediment directly causing stress	Yes. Exposure and uptake of fine sediment can change behaviour and impact on fish and mollusc immune response. Yes, elevated turbidity can smother seagrass with sediment and reduce its health. Yes, elevated turbidity can reduce light available to	Yes. Numerous exceedances of permit conditions for turbidity levels, in the absence of significant rainfall. When dredges broke down prior to UNESCO March 2012 visit, turbidity in Harbour improved. When dredging increased to seal bund wall in late July early August 2012, turbidity had a sustained increase. Leaking bund wall acknowledged by GPC to be contributing to elevated Harbour turbidity.	High (however contaminants associated with this resuspended sediment likely to be much more important).
Noise	Yes. Documented to impact on communications between fish, and potentially impact reproductive behaviours. Affects mollusc feeding.	Yes. Massive increase in boat movements across Harbour to Port Curtis estimated to have exceeded 20,000 per month since Curtis Island development commenced.	Very high (however contaminants likely to be much more widespread impact, extending up estuaries eg Colosseum, Boyne and 7 mile.

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as causal	Likelihood of significant causal role
Habitat/food loss	Yes. Nutritional stress can cause immunosuppression.	Yes. Large area of seagrass documented to have declined and large benthic area lost to reclamation area, and mangroves cleared for wharf development. Loss of seagrass reduces marine ecosystem productivity through diminishment of food web contribution	High (however contaminants likely to be much more important in short term acute diseases). Long term loss of productivity highly likely.
Parasitism	Yes. Can cause lesions observed, but typically requires a concurrent environmental/ husbandry stressor.	Yes. High parasite loads on some, but not all diseased animals. Higher intensities and prevalence of infestations compared to reference site. Indicating elevated parasite intensity may have been secondary to primary immunosuppression in Gladstone.	Very high – but only as a secondary factor to immunosuppression and skin damage.
Other sediment based toxicants	Yes. Ammonia, hydrogen sulfide and hydrocarbons can all cause similar disease conditions to those observed. Yes, ammonia and hydrogen sulfide known to occur in intertidal sediments in particular, and legacy hydrocarbons from spills and local shale	Insufficient monitoring of these parameters has taken place to determine if exposure has taken place or not. Many of the monitoring sites are too remote to the site of dredging and development on Curtis Island to detect volatile compounds.	High – but effects likely to be more localised.
Primary bacterial disease	Yes. Streptococcus agalactiae association with wild Qld Groper mortalities – but unlikely to affect so many different species at the same time. Yes, bacterial disease in molluscs can result in high level mortalities.	No. Negative bacterial cultures on numerous sick animals, and pathology does not indicate the presence of primary bacterial infections in diseased animals. Where present, infections are considered to be secondary opportunists on compromised or damaged hosts (scallops, and mud crabs).	Very unlikely that primary bacterial infection can explain the observed sickness and mortalities.
Viral disease	Yes. Can cause wild fish disease outbreaks, but unlikely in fish, turtles, dolphins and crabs simultaneously.	A few sharks had pancreatitis of unknown cause (potentially viral). However the bulk of the animals examined had no evidence from large number of histological samples of viral disease.	Very unlikely to be the primary cause of observed sickness and mortality.

The approach taken in this review was to examine each of the 24 bolded conclusion statements. This entailed reviewing the preceding 'evidence' including checking the cited references to ensure that they were relevant to the issue being addressed, and this was not always the case. Finally, I have reviewed the summary in Table 1 above, and the extent to which the conclusions re 'likelihood of significant causal role' can be supported.

As a general observation, the Report has a very large reference list and the discussions draw heavily on these references to support the author's hypotheses. The Discussion section is poorly structured and contains many generalisations from the literature without discussion of the evidence. A more systematic discussion of the various issues would have been useful.

3 Overview of the Weight-of-Evidence Approach

The author has adopted what he terms a 'weight of evidence' (WOE) assessment to support his hypothesis. Traditionally, a WOE approach to ecosystem assessment brings together a set of lines of evidence obtained from detailed studies on a system of concern and draws conclusions based on the reinforcement of the strength of agreement between several different lines of evidence. Early users of WOE apply best professional judgement of harm to a range of lines of evidence (e.g. Fuchsman et al., 2000). The process should incorporate judgments about the quality, extent, and congruence of data. Increasingly it is being recognised that in environmental assessments, effective WOE evaluations must include both observational (e.g. ecologically based) and investigative components (e.g. toxicological determinations of cause and effect in relation to chemical and/or physical stressors) (Burton et al., 2002). I have had a major involvement in the adoption of weight-of-evidence assessments of sediment quality both internationally (Burton et al., 2002), and nationally for the current revision of the Australian and New Zealand sediment and water quality guidelines (Simpson et al., 2008; Batley and Simpson, 2009), so I am very familiar with the intended application of this approach in environmental assessments.

Suter and Cormier (2011) have discussed in more detail the assembly of multiple disparate lines of evidence in support of causal assessments that is flexible, transparent and defensible and is intended to balance the need for rigor and discipline with the need for sufficient flexibility to accept all relevant evidence and generate creative solutions to difficult environmental problems. The approach is summarised in Figure 1. Of critical importance is an assessment of the strength and quality of the evidence, and this is determined by giving each piece of evidence a weight. That is, does it support or weaken an apparent impairment in a condition assessment, a hypothesized cause in a causal assessment, or a hypothesized effect in a risk assessment? Conventionally, this aspect of weight is represented by a + or – symbol. As discussed by Suter and Cormier (2011): *'if the evidence is weighted, the judgment is guided by the weights as well as how many criteria or types of evidence support a hypothesis. Hence, the weight of the body of evidence is the aggregate weight of the relevant pieces and categories of evidence. However, characteristics of the body of evidence as a whole also influence its quality. A body of evidence has greater weight if it has the following qualities.*

- *Credibility — the body or evidence is based on relevant and high quality information weighted by relevance, and quality of study design and execution.*
- *Coherence — the body of evidence is internally consistent, consistent with scientific knowledge and theory, and together logically explains the facts in the case as judged by internal consistency and consistency with theory.*
- *Strength — the body of evidence includes pieces of evidence that are logically compelling (e.g. the effect occurred before the cause) or that present quantitatively strong relationships (e.g., high correlation coefficients or relative likelihoods) (see section above). Strength is weighted based on logical implication and by the independence, quantitative strength, and specificity of the evidence.*
- *Diversity — many sources of evidence and characteristics of causation are represented in the body of evidence including a variety of types of evidence and evidence from different datasets.'*

It is appropriate to assess how the various lines of evidence are used in the Landos Report. From my reading, the weight of evidence appears to be based on a series of literature references to effects seen on aquatic organisms as a result of dredging and disposal activities largely without detailed comparisons with field measurements in Gladstone Harbour. The WOE result is a summation of possible impacts which is then used to attribute a probable cause.

As noted by Burton et al (2002), 'the term WOE suggests that a level of certainty exists with the assessment's conclusion when, in fact, there may continue to be significant uncertainty in the conclusions. This misconception can create significant erosion of the decision-making process linking assessment and remediation, resulting in incorrect management decisions that may be over- or under-protective of human and wildlife health. In recent years, improvements have been made to the WOE process, such as by defining a "consensus-based" approach or through the use of a variety of quantitative methods for improved integration of multiple lines-of-evidence (LOE).'

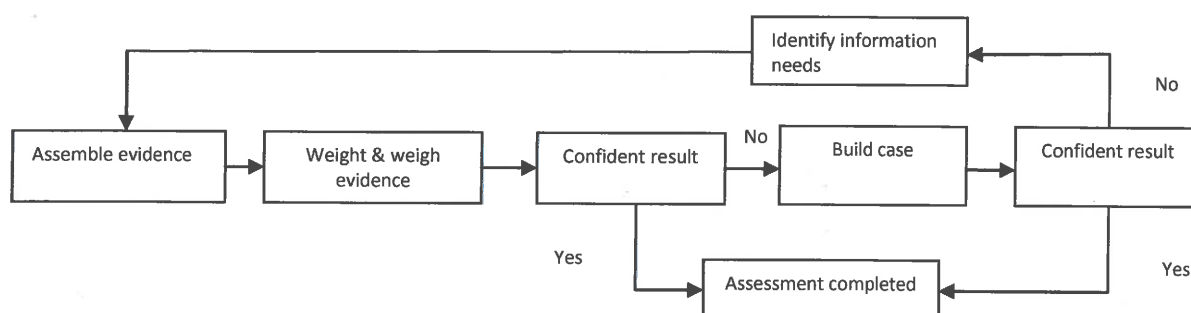


Figure 1. Diagram of a general approach for weighing evidence in the synthesis stage of an environmental assessment (Suter and Courmier, 2011)

There is no weighing of evidence in the Landos Report. We are simply supplied on page 86 with the following Conclusion:

'In considering the weight of available evidence on the potential causes of observed aquatic animal health disorders in Gladstone, FFVS considers these disorders are most likely to be a direct consequence of the Western Basin Dredging and Disposal Project. Specific mechanisms include:

- **resuspension and mobilisation of contaminants (metals and metalloids) from sediments;**
- **increased parasitism due to stress, immunosuppression and external irritation from poor water quality and toxicosis;**
- **increased boat traffic vessel strike risk;**
- **noise; and,**
- **generation of toxic algal blooms due to disturbance of sediments and release of nitrogen, iron and other nutrients.'**

4 Detailed Discussion of the Lines of Evidence

4.1 Disease and lesions due to exposure to toxicants and suspended sediments etc

The summary finding on page 41 of the Landos report is ***'FFVS concludes that the increased rates of disease and lesions observed in Gladstone fish, in comparison with the reference site, are highly likely to be due to exposure to sub-optimal environmental conditions in Gladstone Harbour including: increased exposure to toxicants (primarily metals/metalloids); elevated suspended sediment loads; and increased noise based stressors which are primarily the result of the Western Basin Dredging and Disposal project. The parasitic infestations observed are likely to be a secondary sequelae to the stress and immunosuppression of the host from exposure to poor water quality'***. This follows a general discussion of the incidence of parasitic infections and a general reference (Zeilokoff, 1993) to *'dredging as an activity which is highly disturbing to the aquatic environment, due to the remobilisation of legacy pollutants (see also Rice, 1987; Bonnet, 2000; Nayar, 2004; Esslemont, Russell, and Maher, 2004; Burton, 2010; and Hedge, Knott, and Johnston, 2009). The resuspension of sediment increases the exposure of aquatic life to mixtures of bound pollutants such as polycyclic hydrocarbons and metals by altering their bioavailability.'* There are no cause and effect linkages between the effects and the exposure at this stage of the Report. I accept that elevated concentrations of metals and suspended sediments can be stressors, but the key questions are under what conditions and how do these conditions compare to what is found at Gladstone. These are discussed again later in the Landos Report.

A secondary question relates to the period over which organisms are exposed to elevated contaminant concentrations and suspended sediments. As noted by Wilber and Clark (2001) (a reference used by the author to substantiate comments on the effects of dredging on metal bioavailability (page 40)), fish have the ability to swim away from suspended sediment associated with dredging, and any associated elevated metal concentrations. *'Under most (dredging) scenarios, fishes and other motile organisms encounter localized suspended sediment plumes for exposures of minutes to hours, unless the organism is attracted to the plume or unless individuals follow the plume or are confined to an area with restricted circulation'*. This possibility offers a different interpretation.

Chronic toxicity effects usually require exposures of many hours and removal of a stressor can often reverse or at least reduce any impacts, although recovery of organisms from exposures has generally been poorly studied.

The Report presents a number of references to document impacts of suspended sediments on larval fish and disturbed host parasite relationships that may indicate impacts on fish recruitment in areas impacted by the dredging plume. The significance of this area compared as a fraction of the greater area of the Harbour needs to be considered.

4.2 Copper and zinc cause shell disease in crabs

A number of conclusions were made with respect to disease in mud crabs, based largely on the research by Andersen and co-workers. As part of her MSc research, Andersen (2003) found that *'copper and zinc were elevated in mud crab hepatopancreas from Gladstone compared to a reference site at Ayr in two consecutive years. Mean copper concentrations of Gladstone crabs were two to*

three times greater than for mud crabs sampled from other locations in Queensland. Total metal burdens were also elevated in the Gladstone mud crabs, with a high variation in the diseased mud crabs suggesting that this group may be unable to regulate metal levels.' Copper exposure experiments indicated that calcium uptake into juvenile crabs was inhibited by sub-lethal copper exposure. *'The copper exposure experiments supported the pathology findings that rust spot shell lesions are likely to be caused by a defect in the manufacturing process of the endocuticle.*

Attempts to identify the sources of copper to the crabs were inconclusive, with analyses of water and sediments collected from mud crab burrows finding very low concentrations of metals. It was surmised that diet may be a source of copper to Gladstone mud crabs through biomagnification along the food chain.

Later studies (Andersen et al., 2005) examined bioaccumulation and food chain pathways in more detail, and found that mud crabs accumulated marginally higher copper and zinc from a site near Boat Creek than at a reference site or other sites in Gladstone Harbour. The food source of the copper was not identified as mud crabs had a varied diet.

Earlier in the Landos report it is stated (page 41) that *'benthic (bottom dwelling) animals are the most exposed to contaminated sediments (Gerbersdorf et al., 2011). As such, and given the conditions described in Gladstone Harbour, it is not surprising that mud crabs were suffering from serious metals-related diseases after the commencement of dredging.'* However, measurements of sediment copper and zinc concentrations in Gladstone Harbour (Angel et al., 2012) revealed that concentrations were well below Australian sediment quality guidelines. Measurements by DEHP (2012a) confirmed that metal and metalloid concentrations in sediments being dredged were below sediment quality guidelines (ANZECC/ARMCANZ 2000), and unlikely to be a source of dissolved metals.

The link between dissolved copper and zinc concentrations and disease is equally tenuous. Dissolved copper and zinc concentrations quoted in the report are only marginally above the guideline trigger values as will be discussed later, and were the similar both before and after dredging. The disease in mud crabs was present long before dredging commenced.

The strength of this line of evidence is therefore poor.

4.3 Shellfish populations severely impacted by sediment load and metal exposure causing oyster mortality

The Report makes reference to reduced shellfish numbers and quotes a personal communication as support. No scientific data are presented. Reference is made to a paper showing that shellfish can accumulate metals during dredging (Knott et al., 2009). This study referred to Port Kembla Harbour (NSW) and the effect on recruitment. No bioaccumulation was studied. The Port Kembla sediments contained very high concentrations of zinc (>1000 mg/kg) and lead concentrations of around 200 mg/kg, orders of magnitude higher than those found in the Gladstone Harbour sediments (Angel et al., 2012). The Knott et al. reference is clearly of little relevance to the Gladstone Harbour dredging.

The use of oysters as biomonitors for metals was examined in 2002 by Andersen et al. (2002) at sites associated with dredging activity near the RG Tanna coal terminal site and at reference sites. Not all metals accumulated at the same rates but there were indications of increased concentrations of copper, aluminium and zinc in oysters sampled at sites closest to the dredging. The transplanted oysters were sampled and analysed before dredging commenced after a one month deployment as well as one and two months after dredging had commenced. As noted by GHD (2009a), *'sampling was undertaken four weeks after dredging commenced, so increases may not have resulted from dredging. However, concentrations at two months of dredging were assumed to reflect the effects of dredging activities.'* A subsequent study in 2005 of oyster bioaccumulation at three sites in

Gladstone Harbour and at a control site (Andersen et al, 2005) showed tissue copper at 80-130 mg/kg wet weight at all sites with the control at 100 mg/kg. Zinc ranged from 170-270 mg/kg, but 220 mg/kg in controls. These oysters were collected off rocks so may have reflected near-shore impacts. In all studies, no evidence was produced to support the statement that dredging was a cause of oyster mortality.

4.4 Ocean disposal of fine sediments contributes to disease in corals with effects from metals and metalloids

The reported negative effects of metal-rich fine sediment from dredging activities on coral exposed to suspended sediment plumes from dredging activities in Townsville (on page 49), is used in the Report to implicate the effects of dumped sediments on corals in reefs on the oceanic side of Facing Island. Recent studies by Ayling et al. (2012) of these reefs concluded that there was no suggestion from *'that the benthic community on Facing Island reefs has been impacted by Gladstone Port dredging operations over the past 12 months'*. They further noted that *'with the exception of Curtis Island, these reefs appear to be typical of inshore reefs in the southern Great Barrier Reef region. Algal cover was relatively high and coral cover low to moderate. The extremely turbid water mass on the outside of Curtis Island that was evident on all three visits to this location does not appear to allow coral growth'*.

The sediments dumped from Gladstone Harbour could certainly not be considered as metal rich, based on the analytical data available (Angel et al., 2012; DEHP, 2012a). There are valid concerns generally for suspended sediments near coral reefs, but no data are provided to indicate the presence of either high suspended sediments or high dissolved metals. Further research is needed before this line of evidence can be accepted.

4.5 Turtle, dolphin and dugong mortalities a direct consequence of the dredging and disposal

While there is clear evidence of mortalities in turtles, dolphins and dugongs, there is nothing that directly links this to dredging activities. The comment that arsenic might be a causative agent is difficult to support because of the low arsenic concentrations measured in both waters and sediments.

I am not qualified to comment on the effect of *Lyngbia* blooms raised in the report as a possible toxin to turtles.

4.6 Deterioration of water quality due to sediment resuspension is a factor in the decline of seagrasses

Seagrass decline can be the result of many factors including such things as turbidity, and freshwater input. No evidence is produced to show that seagrasses have changed more than seasonally, or that the level of turbidity has impacted on seagrass health. There are many areas of Australia where seagrasses have been declining, largely due to increased development, increased nutrient inputs, and sediment instability (Fox et al., 2007). To justify the conclusions reached in the Landos Report, a more thorough assessment is warranted.

4.7 Loss of habitat and its associated food sources and shelter are likely to have generated stress in aquatic biota

Loss of habitat, food sources and shelter in the local areas impacted by dredging will certainly be potential stressors to some aquatic biota. There is no evidence that this is the sole reason for any decline in fisheries across Port Curtis, or whether there will be permanent loss of fisheries habitat.

4.8 Declines in fish, prawns and crabs due to increased disease and mortality rates, recruitment failure, also entrainment in hydraulic dredging activities, and movement away from the noise and poor water quality

It is acknowledged that dredging has clearly had an impact on turbidity in some regions of the Harbour. The Report cites references that indicate that suspended sediment can have effects on larval species of fish. The ANZECC/ARMCANZ (2000) guideline for turbidity in estuaries is 1-20 NTU. Baseline data collected by GHD (2009b,c) pre-dredging showed that turbidity was naturally higher in the wet season in areas slated for dredging with median values from 10-23 NTU and 95th percentile as high as 127-176 NTU, compared with a dry season median of 9 NTU and 95th percentile of 30-90 NTU. These values are well above turbidity values affecting fish larvae (4 mg/L or near 2 NTU) (Partridge and Michael, 2010), indicating that in the regular wet season in the absence of dredging effects on fish larvae might be expected. There was no discussion in the Report of what turbidity levels might be during dredging.

The Report presents a number of references to document impacts of suspended sediments on larval fish and disturbed host parasite relationship that may indicate impacts on fish recruitment in areas impacted by the dredging plume. The significance of this area as a fraction of the greater area of the Harbour needs to be considered.

4.9 High risk from ammonia release from the dredging activities

The Report correctly identifies ammonia as a constituent of sediment pore waters. In a recent paper (Batley and Simpson, 2009), we discussed the potential impacts from dredging of released ammonia: *'In a detailed examination of porewater ammonia release during a dredging and dumping operation at a Queensland harbour site, sampling at the dumpsite after the dumping operation found that concentrations in the pore water of the dumped sediments had declined from 30–40 mg/L total NH₃-N/L in samples from the barge prior to dumping, to 22 µg/L within 10 min of disposal and <2 µg/L within an hour (Richardson, private communication).'*

'For water impacts, short-term effects could be considered as those occurring in the order of hours from disposal time, whereas long-term effects would occur after as much as 24 h post disposal (or dredging). As shown above, water quality monitoring during dredging and disposal of sediment did not result in exceedances of the proposed revised guideline value of 460 µg total NH₃-N/L. For toxic effects occurring over time frames of a few hours or less, it would be appropriate to use guidelines based on acute NOECs, rather than the more conservative PC95 values. A value of 1550 µg total NH₃-N/L, calculated from the PC95 value based on acute LC50s (Table 2) divided by 5 to convert to a NOEC, is recommended.'

The above paper used recent toxicity data to update the existing ANZECC/ARMCANZ (2000) guideline for ammonia in marine waters to 460 µg total NH₃-N/L. At the pH of seawater, some 50% of the ammonia is present as unionised ammonia. This has low solubility in seawater and can be

easily lost by volatilisation. This is the major reason that ammonia concentrations fall rapidly after dredging. It is noted in the Report that DERM monitoring '*did not detect levels of ammonia nitrogen above 17 µg/L*'. Concentrations measured by DERM (2012) in the dredge plume were between 2 and 9 µ/L as N. The concluded 'high risk' from ammonia is therefore a weak line of evidence not supported by literature data.

4.10 Low dissolved oxygen post dredging significant to exposed biota, compounding impacts of contaminants such as aluminium, arsenic, zinc and copper

There are possible instances of reduced oxygen concentration in a dredged sediment plume, however, the waters are well mixed and the likelihood that this of itself will cause toxicity to exposed biota is low. Measurements in 2011 by DERM (DERM, 2012) showed that subsurface dissolved oxygen concentrations in Gladstone Harbour were greater than 85% saturation during the November and December sampling, with lower concentrations being found in the estuaries. Here the lowest concentration was 70%, but significant significant impacts on fish are unlikely at DO concentrations greater than 50% saturation (DERM, 2009). The potential for compounding impacts from aluminium, arsenic, zinc and copper is extremely low given the concentrations of these contaminants as will be discussed later.

4.11 Biota exposed to hydrogen sulfide released from dredged sediments, and cyanide releases

Hydrogen sulfide is produced by bacterial reduction of sulfate in anoxic sediments. A lot of the sulfide is complexed by iron as iron monosulfide, but free hydrogen sulfide is often present in pore waters in equilibrium with the HS⁻ ion. A paper from the US Army Corps of Engineers (Sims and Moore, 1995) notes that '*hydrogen sulfide in sediment pore water is not treated as a contaminant of concern for the regulatory evaluation of dredged material since it undergoes rapid oxidation and dilution during dredging and disposal.*'

Contrary to the comments in the Landos report, there is a conservative low reliability ANZECC/ARMCANZ guideline for sulfide in marine waters of 1 µg/L (ANZECC/ARMCANZ, 2000), however, the only acute toxicity data for fish show effects over 500 µg/L (Sims and Moore, 1995). Given its short half life in oxygenated seawater (reported by Ostland and Alexander (1963) to be as low as 20 minutes), hydrogen sulfide is not considered a threat to fish toxicity.

Cyanide is also included (on page 49) as a potential toxicant, with a report that discharges by Orica '*were more than double, their discharge licence for cyanide (1 mg/L) release into Gladstone Harbour in January and February 2012 (Stitt, 2012).*' The toxicity of cyanide is dependent on whether it is free or complexed. The ANZECC/ARMCANZ guideline for free cyanide in marine waters is 4 µg/L (ANZECC/ARMCANZ, 2000), with acute effects on fish seen at 70 and 109 µg/L, respectively for black bream and Australian bass, (Pablo et al., 1996). Free cyanide is readily lost from seawater by volatilisation as HCN (Dzombak et al., 2006). This volatilisation has been a major problem in the toxicity testing of cyanide in seawater, with the estimated half life at around 24 h. In well-mixed waters in a tropical marine environment at higher temperatures (28-30°C), the losses are expected to be even greater. Coupled with the likely dilution of the discharge, cyanide is not seen as a major source of toxicity.

The Landos Report suggests that '*cyanide may have contributed to local impacts on aquatic animal health,*' but acknowledges that '*the cyanide spill is highly unlikely to be causal of fish health problems*

observed on the oceanic side of Facing Island, and >30 km to the south in 7 mile estuary, where FFVS sampled diseased fish, due to the substantial dilution effects. Hence the cyanide spill is insufficient on its own to be the factor responsible for the common disease issues across the wider Gladstone area'.

Loss of habitat, food sources and shelter in the areas impacted by dredging will certainly be potential stressors to some aquatic biota. It is not proven that this is the sole reason for any decline in fisheries, or whether there will be permanent loss of fisheries habitat.

4.12 PAHs or PCBs not implicated in aquatic animal diseases

The Report concludes there is insufficient evidence to implicate PAHs and PCBs with a primary role in observed animal stresses. I agree with this conclusion. Such compounds have very low water solubility and are more likely to partition to suspended sediments and ultimately accumulate in bottom sediments. Those already present in dredged sediments will largely be undetectable in pore waters.

4.13 Potential acid sulfate soils, has facilitated the release of metals and metalloid compounds into more bioavailable dissolved forms

The potential risks from acid sulfate soil remobilisation were raised on page 71 of the Report. Pyrite (FeS_2) the major constituent of acid sulfate soils, is not rapidly oxidised, with 3- 17% of very fine pyrite suspended in seawater oxidised in 1 day slowing to 20% after 1 week (Morse et al., 1991). Oxidation of coarser sedimentary pyrite would take appreciably longer. Iron monosulfide (FeS) (so-called acid-volatile sulfide), also present in anoxic sediments is quite rapidly oxidised on exposure to oxygenated seawater (<1 hour) (Simpson et al., 1998).

Oxidation of both pyrite and FeS generates acid, however, as seawater is well buffered, it is unlikely that the pH would be significantly affected. In seawater, metals released as a consequence of any acidification are readily scavenged by the iron oxyhydroxide products of the oxidation that precipitate and settle to the sediments. The incidence of acidification and increased metal bioavailability referenced on page 71 of the Report are only significant in freshwater systems. In Australian estuaries, the incidence of acidification and impacts on oysters are largely limited to low salinity regions where the pH buffering from seawater is reduced. Monitoring data (DERM, 2012) show a minimal impact from acidification with pH values measured in 2011 in the dredging zone between 7.6 and 8.0 compared to 7.7 to 8.1 at other sites and 7.7 to 8.2 at the spoil grounds. The Report has therefore misinterpreted the problems associated with acid sulfate soils.

A quote is made on page 69 of the Report from a report (GHD, 2009d) saying that the sediments containing acid sulfate soils do not have the capacity to self-neutralise. The quote has no accompanying comment, and it could be assumed that it is referring to seawater, however, on reading the reference, it was found to be referring to the presence of calcium carbonate in the sediments and its ability to neutralise acidity in pore waters in sediments near the bund wall. This will only affect organisms in the sediments that might be exposed at low tide, but not in the water column. There is unlikely therefore to be any release of metals as a consequence of pore water acidification.

4.14 Metals and metalloids issues with dredging

Metals and metalloids are raised throughout the Report as the major stressor, together with turbidity due to suspended sediments. This section of my report discusses items 14-16 of the list presented above that specifically deals with metals.

It is well known that dissolved metals at concentrations exceeding the water quality guideline trigger values may be toxic to aquatic biota. In the Report, it is implied that exceedance of any guideline is taken as evidence that there are likely to be toxic impacts. This is not the case, and it is important to understand the application of the guidelines and how they are derived to appreciate this.

Australian water quality guideline trigger values (ANZECC/ARMCANZ, 2000) are derived from observed chronic toxicity data. The chronic no observed effects concentrations (NOECs) for particular test organisms are fitted to a species sensitivity distribution plot (Figure 2). The input data must include tests on at least 5 species from four different taxonomic groups. Typically the most sensitive organisms comprise algae and small invertebrates, appearing at the lowest concentrations at the bottom end of the curve, with less sensitive organism such as fish showing responses at much higher concentrations. From the distribution, a concentration is read off the x axis that protects a given percentage of species, and for slightly-to-moderately disturbed water bodies such as Gladstone Harbour, this would be 95% species protection. The response curve typically covers a concentration range from 1 to over 1000 $\mu\text{g/L}$, and fish might only be impacted at concentrations that are 10 times the guideline values (e.g. Figure 2 for copper in seawater).

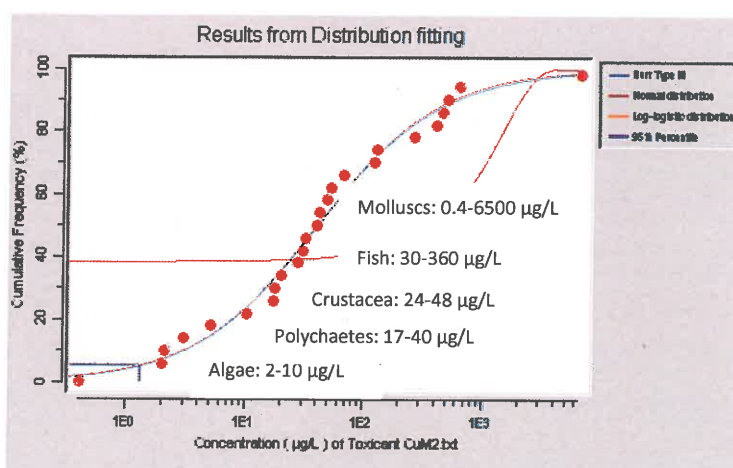


Figure 2. A species sensitivity distribution for copper in estuarine and marine waters. Each point represents a chronic NOEC for a particular aquatic species. Sensitive organisms such as algae fall at the bottom of the curve followed by invertebrates and fish. The blue line represents the statistical fit using a Burr Type III distribution. The 95% species protection number is indicated by the line at 5% of species on the y axis.

The criticism of the monitoring (page 73 in the Report) that labile metal species had not been measured in the initial monitoring program is misplaced. The measurement hierarchy provided in the ANZECC/ARMCANZ Australian and New Zealand Guidelines for Fresh and Marine Water Quality (2000) recommends that a hierarchy of measurements be followed, namely total metals measured first, then if the guideline trigger value is exceeded measure the dissolved metals, then only if these concentrations exceed the trigger values should labile and potentially bioavailable metals measured. In this context, while measurements of labile copper were not undertaken, there is extensive literature to support the fact that in estuarine and marine waters, labile copper are typically considerably below the dissolved concentration (as low as 50%) (e.g. Batley, 1987). This is largely

because copper complexation by dissolved organic matter forms compounds that are less bioavailable.

Table 7 in the Report lists water quality exceedances for aluminium, copper, zinc, chromium and arsenic. Only dissolved concentrations are relevant because the total concentrations include metals in suspended particulates that are not bioavailable. The maximum recorded dissolved copper concentration was 3 µg/L compared to a trigger value of 1.3 µg/L. Apart from the uncertainties around the trigger value, a measured dissolved copper concentration that is 2.3 x the trigger value would trigger a measure of bioavailable metals, that would be expected to be well below 3 µg/L. Copper is therefore not an issue in terms of bioavailability to biota in Gladstone Harbour.

Because fish appear higher on the species sensitivity distribution (Figure 2), the concentration at which fish would be affected by copper is likely to be above 30 µg/L. This means that there would be no impact on chronic fish toxicity at the measured maximum dissolved copper concentrations. Similarly, the maximum measured dissolved zinc concentration of 25 µg/L would have no effects on fish.

Dissolved chromium in seawater can be present either as chromium (III) or (VI), but chromium (VI) is highly reactive and readily reduced to chromium (III). The quoted chromium concentrations do not exceed the guideline trigger value for chromium (III), the likely oxidation state, and are only marginally above the chromium (VI) guideline, again of little consequence for fish.

The quoted maximum arsenic concentration of 2.6 µg/L is not unusual for estuarine and marine waters (Port Phillip Bay waters are as high as 3 µg As/L). The guideline value is, however, low reliability, termed an environmental concern level, and is likely to be extremely conservative (over-protective).

Overall, apart from aluminium, it can be confidently stated that the maximum measured concentrations of other metals pose no particular toxic threat to fish or to some 90% of species.

It is worth noting that the DERM data referred to in Table 7 of the Report are at odds with the measurements by CSIRO in 2012 (Angel et al., 2012), where the findings were:

- (i) *'The concentrations of dissolved metals were below the available ANZECC/ARMCANZ marine water quality guideline trigger values (95% protection, high or moderate reliability) that apply in Australia at all 21 sites sampled and the concentrations were relatively low compared to other industrialised harbours.'*
- (ii) *'Dissolved aluminium concentrations were above the ANZECC/ARMCANZ (2000) environmental concern level of 0.5 µg/L at the majority of sites sampled. It should be noted that there is no reliable guideline value for aluminium in marine waters in Australia and the ECL value is a highly conservative value based on very limited toxicity data. There are no water quality guidelines that apply for aluminium in marine waters in Europe or North America. From the current data set, it was not possible to attribute a specific source of the dissolved aluminium.'*
- (iii) *'The study did not detect any 'hot spots' of metal concentrations. There was no detectable elevation of metal concentrations at sites where dredging was being conducted.'*
- (iv) *'The dissolved copper concentrations measured in Port Curtis in December 2011 were lower than the ANZECC/ARMCANZ guideline value of 1.3 µg/L. However, the dissolved copper concentrations were noticeably higher than the concentrations measured in the CSIRO surveys in December 2003 and 2004, indicating increased inputs of these metals from various sources, and the concentrations at some sites were only marginally lower than the ANZECC/ARMCANZ guideline value. Dissolved cadmium and zinc concentrations were comparable to those measured in 2003/2004 by CSIRO.'*

It has been well documented that the sampling and analysis of trace metals in marine waters is a highly specialised exercise that requires extreme care in the preparation of sample containers and in the collection of samples to avoid contamination. The CSIRO scientists were highly experienced with this type of sampling, so their results are of high quality and not readily dismissed.

4.14.1 ARSENIC CAUSES SUB-LETHAL STRESS IN FISH

The reference to potential impacts of 'elevated arsenic' on page 53 of the Report is typical of the emotive extrapolations throughout the report: *'it is expected that exposed animals would have elevated arsenic levels. Arsenic is known to be a highly toxic element depending on the form it is in.'* Concentrations are not reported, nor is the form of arsenic in the organisms tested. It is a fact that most aquatic organisms have elevated arsenic concentrations, but the inorganic arsenic is metabolised to non-toxic organoarsenic compounds (e.g. arsenobetaine) so does not represent a threat to the fish or human consumers.

Dissolved arsenic concentrations sampled in Gladstone Harbour by DERM had a maximum concentration of 2.6 µg/L, as already noted (Table 7 in the Report). Concentrations of 16 µg/L in Auckland Creek referred to on page 76 of the Report, imply a different source to dredging which has been the primary causative factor implicated in the Report.

4.14.2 BIOACCUMULATION

Bioaccumulation of metals is an accepted line of evidence indicating that metals are present in a bioavailable form and are able to be taken up by organisms. As noted in the Landos Report (page 69), previous studies by Apte et al. (2005) had indicated that concentrations of aluminium, arsenic, copper, chromium, iron, mercury, nickel, selenium and zinc were significantly enriched in marine biota within Port Curtis compared to a reference site. The Apte et al. report went on to say (and not quoted in the Landos Report) *'this does not necessarily imply adverse effects resulting from exposure to elevated concentrations. Further studies are required to investigate whether organism health is impaired by these increased body burdens of metals.'*

4.15 Dissolved aluminium released by dredging and spoil disposal

Special consideration is given in the Landos Report to aluminium. It quotes DERM data for dissolved aluminium with a maximum of 80 µg/L. This was for a site in the Calliope River, 6.4 km from the Harbour (DERM, 2011). A second site had 70 µg/L, but well north of the dredging area. In the dredge plume 20 µg/L was measured. CSIRO data from 2004, prior to any dredging, also showed elevated aluminium (DEHP, 2012b). The implication of this is that aluminium is not associated necessarily with dredging, but with other sources, such as licensed discharges, or natural mineralisation. This is an example of the lack of detailed interpretation in the Landos Report that leads to the wrong conclusions.

The guideline value for aluminium of 0.5 µg/L is an environmental concern level of low reliability, owing to the lack of published toxicity data for marine biota. Current research by CSIRO indicates that a high reliability trigger value is likely to be closer to 10 µg/L. In our studies, no toxicity was observed to fish species at concentrations below 1000 µg/L, so a dissolved aluminium concentration of 80 µg/L is unlikely to be toxic.

As in all assessments of metal impacts, an exceedance of a guideline requires further investigation of metal speciation before the potential for impact can be eliminated. It is the bioavailable fraction, often appreciably less than the total dissolved concentration that controls toxicity.

The Landos Report commented that *'the valency of aluminium has not been reported from water samples in Gladstone'*. Aluminium in solution has a valency of 3 (unless it is metallic aluminium of valency 0). Clearly what Dr Landos meant to say was that the speciation of aluminium has not been reported. This is not easily done. Our recent research has shown that dissolved aluminium is present as $\text{Al}(\text{OH})_4^-$ in seawater and, at concentrations in excess of around 500 $\mu\text{g}/\text{L}$, starts to form insoluble $\text{Al}(\text{OH})_3$ forms. Toxic effects are possibly then due to gill clogging rather than aluminium uptake.

4.16 Iron, elevated nutrient levels in the harbour, causes *Lyngbya* blooms

Increased iron concentrations were reported by Landos to have been detected in the Harbour by DERM (2012) in 2011. The presence of *Lyngbya* has also been reported. As noted on page 78 of the Landos Report, increased *Lyngbya* blooms are promoted by increased bioavailability of iron and elevated nutrient concentrations. Typically high iron concentrations in seawater are associated with a freshwater input, with iron precipitating in the low salinity areas of the mixing zone, termed the turbidity maximum. The highest dissolved iron (possibly colloidal) was 145 $\mu\text{g}/\text{L}$ at the reference site Rodds Bay (DERM, 2012). Total iron was variable between samplings (as was dissolved iron) and was highest at Boat Creek, a freshwater input. Here is a link between iron and *Lyngbya* blooms, but it would seem that the input of iron is natural rather than associated with dredging.

4.17 Multiple stressors and immunosuppression

The Australian and New Zealand water and sediment quality guidelines are based on chronic toxicity data (ANZECC/ARMCANZ, 2000). They are not predictors of disease effects. It is known that biomarkers of stressor responses in organisms from metals can be seen at concentrations below guideline trigger values. There is a growing research area looking at genotoxicity and immune responses of organisms to stressors. A comprehensive review of chemically induced immunosuppression was recently prepared by Poulsen and Escher (2012). The interpretation of their findings in the Landos Report (page 74) was that, along with other references, Paulsen and Escher *'demonstrated that sub-lethal copper exposure has led to immunosuppression in fish, and thereby increased susceptibility to disease'*, leading to a more sweeping conclusion that *'animals exposed to elevations in dissolved copper levels, particularly those above the ANZECC guideline, are likely to have incurred a physiological stress. In combination with other stressors outlined, this is likely to have led to immunosuppression, and can explain the increased intensity of parasitic infestations observed.'*

My reading of the Poulsen and Escher report was that there was evidence (albeit largely qualitative) that metals can suppress the function of the immune system in a wide range of marine organisms, but at concentrations above those found in Gladstone Harbour, however, the number of studies demonstrating exposure concentrations causing immunosuppression in seawater are limited. The authors concluded that there is a correlation between immunosuppression and susceptibility to pathogens and parasites.

Copper was shown by Poulsen and Escher to affect immune response in a number of organisms, but the lowest copper concentration at which effects were observed was 20 $\mu\text{g}/\text{L}$ for a marine blue mussel, well above the maximum measured dissolved concentration of 3 $\mu\text{g}/\text{L}$ (and of which a significant percentage is likely to be complexed by natural organic matter and not bioavailable). The only listed response for a marine fish was for rainbow trout and that occurred at 250 $\mu\text{g}/\text{L}$ Cu/L (Poulsen and Escher, 2012), which was 83 times the maximum dissolved concentration. Furthermore, the additional references (Mirza et al., 2009 and Tierney et al., 2010) quoted in

support of the statement on page 74 of the Landos Report that *'copper induced olfactory toxicity is also known to occur in fishes at levels similar to those detected in Gladstone Harbour'* both refer to freshwater fish and at concentrations well in excess of those detected in Gladstone Harbour. These are further examples of the author being loose with the truth.

4.18 Freshwater not implicated

I was concerned at the selectivity of evidence that began in the Executive Summary with the statement: *'The contrary view that a freshwater influx led to the observed impacts on aquatic animal health is scientifically unsupportable. The reality is that Queensland coastal estuaries have for centuries received large freshwater influxes. The types of diseases observed recently in Gladstone have not been a feature of previous freshwater influxes.'* It is highly likely that the impacts on aquatic biota are the result of multiple stressors. Freshwater is a known stressor to many marine organisms (MacGinitie, 1939). The fact that freshwater effects are absent in other sites does not negate the possibility that freshwater could significantly contribute, with other co-stressors, to the observed impacts. Similarly effects such as high populations and food scarcity could be further stressors that may warrant discussion. The former is mentioned briefly on page 40 of the Report.

5 Summary of the Evidence and Conclusions

In Table 2, I have summarised the findings of my review of the lines of evidence presented in the Landos Report as an additional column to the Report findings reproduced in Table 1.

Table 2. Comments on the weight of evidence findings from Table 1

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as casual	Likelihood of significant causal role	Reviewer comments on evidence
Freshwater	Nil. Based on the lesions observed in fish and crabs detailed in histological reports which cannot be caused by freshwater exposure.	Nil. Timing does not fit with outbreak. Areas remote from Gladstone with similar or larger volume of freshwater influx had no similar aquatic animal disease outbreaks. Previous freshwater influxes into Gladstone have not precipitated any similar events. Freshwater alone does not cause rust spot lesions in crabs, nor dermatitis in fish. Freshwater is lethal to the marine parasites observed on barramundi and bull shark skin, yet the parasite intensities were high.	Very low.	Disagree. Freshwater is likely to be a significant co-stressor
Metals exposure immuno-suppression	Yes. Exposure and uptake can trigger immunosuppression.	Yes. Multiple exceedances of ANZECC guideline of multiple metals over extended periods of time coincident with dredging. Elevated metals levels detected in some tested biota (low sample numbers limit power of analysis). Dissolved metal levels in water were lower prior to dredging. High parasite intensities identified grossly and in histology are consistent with fish, turtle and scallops that are immunosuppressed.	Very high.	Exceedence of guidelines is not significant for key metals and metalloids. Evidence for concentrations producing immunosuppression is limited and semi-quantitative
Metals exposure direct tissue effects	Yes. Exposure to elevated levels of dissolved metals can cause acute effects on fish skin and impairment of olfactory function.	Yes. High levels (inc aluminium, zinc, arsenic and copper) recorded by DERM/DEHP, sufficient to cause observed symptoms in fish sharks and rays, including increased mucous secretion, skin lesions, and thin body condition due to failure to feed.	Very high.	Misinterpreted. There is no evidence for concentrations of bioavailable metals and metalloids above guidelines
Metals exposure shell disease in crabs	Yes. Uptake of elevated levels of copper and zinc can induce rust spot lesions in crabs.	Yes. Prevalence survey indicates elevated prevalence of rust spot in Gladstone mud crabs compared to reference site. FFVS pathology demonstrated lesions in Gladstone crabs are morphologically consistent with metal induced endocuticle shell disease. Post dredging elevations in dissolved copper and zinc reported by DEHP.	Very high.	There is no evidence of 'elevated' concentrations of copper and zinc

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as casual	Likelihood of significant causal role	Reviewer comments on evidence
Toxic algae (fish impact)	Yes, plausible cause of fish skin and gill damage, and elevated blooms can trigger irritation/stress/ death.	Limited, details of reports of algal blooms have not been released by GPC/DERM. Yes, elevated Chlorophyll monitoring levels. Blooms not detected in all locations where FFVS identified sick fishes. Toxic algae not known to cause shell lesions in crabs.	Uncertain (likely only partial role)	Agreed
Toxic algae (human impact)	Yes. <i>Lyngbya</i> known for its dermatotoxins.	Yes. <i>Lyngbya</i> identified in association with sick fishermen, and in algal monitoring study by FFVS. Lesions in many people consistent with <i>Lyngbya</i> exposure after the commencement of major dredging and never before in Gladstone Harbour fishers		Agreed
Turbidity-increased suspended sediment directly causing stress	Yes. Exposure and uptake of fine sediment can change behaviour and impact on fish and mollusc immune response. Yes, elevated turbidity can smother seagrass with sediment and reduce its health. Yes, elevated turbidity	Yes. Numerous exceedances of permit conditions for turbidity levels, in the absence of significant rainfall. When dredges broke down prior to UNESCO March 2012 visit, turbidity in Harbour improved. When dredging increased to seal bund wall in late July early August 2012, turbidity had a sustained increase. Leaking bund wall acknowledged by GPC to be contributing to elevated Harbour turbidity. JCU satellite study suggests dredge	High (however contaminants associated with this resuspended sediment likely to be much more important).	Increases in turbidity occur regularly after heavy rain. Contaminants associated with suspended sediment have not been shown to be at high concentrations
Noise	Yes. Documented to impact on communications between fish, and potentially impact reproductive behaviours. Affects mollusc feeding.	Yes. Massive increase in boat movements across Harbour to Port Curtis estimated to have exceeded 20,000 per month since Curtis Island development commenced.	Very high (however contaminants likely to be much more widespread impact, extending up estuaries eg Colosseum, Boyne and 7 mile.	Possibly – not my area
Habitat/food loss	Yes. Nutritional stress can cause immunosuppression.	Yes. Large area of seagrass documented to have declined and large benthic area lost to reclamation area, and mangroves cleared for wharf development. Loss of seagrass reduces marine ecosystem productivity through diminishment of food web contribution	High (however contaminants likely to be much more important in short term acute diseases). Long term loss of productivity highly likely.	There is no evidence of high contaminant concentrations in waters or sediments

Proposed risk factor for observed diseases	Plausibility of risk factor to cause the observed lesions based on peer reviewed literature	Available evidence to support proposed risk factor as casual	Likelihood of significant causal role	Reviewer comments on evidence
Parasitism	Yes. Can cause lesions observed, but typically requires a concurrent environmental/husbandry stressor.	Yes. High parasite loads on some, but not all diseased animals. Higher intensities and prevalence of infestations compared to reference site. Indicating elevated parasite intensity may have been secondary to primary immunosuppression in Gladstone.	Very high – but only as a secondary factor to immunosuppression and skin damage.	Possibly, but not at the measured concentrations of metals
Other sediment based toxicants	Yes. Ammonia, hydrogen sulfide and hydrocarbons can all cause similar disease conditions to those observed. Yes, ammonia and hydrogen sulfide known to occur in intertidal sediments in particular, and legacy hydrocarbons from spills and local shale oil deposits known to be present.	Insufficient monitoring of these parameters has taken place to determine if exposure has taken place or not. Many of the monitoring sites are too remote to the site of dredging and development on Curtis Island to detect volatile compounds.	High – but effects likely to be more localised.	Ammonia and hydrogen sulfide unlikely to be a concern based on their known fate in seawater.
Primary bacterial disease	Yes. Streptococcus agalactiae association with wild Qld Groper mortalities – but unlikely to affect so many different species at the same time. Yes, bacterial disease in molluscs can result in high level mortalities.	No. Negative bacterial cultures on numerous sick animals, and pathology does not indicate the presence of primary bacterial infections in diseased animals. Where present, infections are considered to be secondary opportunists on compromised or damaged hosts (scallops, and mud crabs).	Very unlikely that primary bacterial infection can explain the observed sickness and mortalities.	Not my area
Viral disease	Yes. Can cause wild fish disease outbreaks, but unlikely in fish, turtles, dolphins and crabs simultaneously.	A few sharks had pancreatitis of unknown cause (potentially viral). However the bulk of the animals examined had no evidence from large number of histological samples of viral disease.	Very unlikely to be the primary cause of observed sickness and mortality.	Not my area

A major concern is that the weight-of-evidence approach adopted in the Landos Report is inconsistent with accepted applications in ecological risk assessment. Rather than basing lines of evidence on measured data, observed biological effects are linked in the Report to possible causes in what can best be termed a ‘guilt by association’ approach. Because there are a large number of such ‘lines of evidence’ produced, a conclusion is reached that *‘the observed aquatic animal health disorders in Gladstone, FFVS considers these disorders are most likely to be a direct consequence of the Western Basin Dredging and Disposal Project’*.

My finding is that the available data do not support this conclusion. In particular, metal and metalloid concentrations in the dredged sediment and in the water column are not at

concentrations of environmental concern. Gladstone Harbour could be considered relatively uncontaminated by comparison with the majority of the world's harbours and these do not have evidence of fish disease.

More research is needed to identify the causes of the observed disorders, however, it is likely that it is the result of multiple stressors. These stressors could include, freshwater, fish stocks, availability of food, licensed discharges, and stormwater runoff. To already stressed biota, dredging might represent a further stress, but clearly it is not the primary cause of impacts as the Landos Report claims, and given the low measured concentrations of metals in both dredged sediments and waters in Gladstone Harbour, its contribution as a stressor is likely to be minor. Similar multi-stressor impacts were concluded in the investigation of oyster mortality in the George Bay in Tasmania (Batley et al., 2010).

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04 June 2013

Central Queensland
Hospital and Health Service

Mr John Sherriff
Gladstone Ports Corporation Ltd
PO Box 259
Gladstone Qld 4680

Dear Mr Sherriff

Re: Enquiry from Senate Environment and Communication Legislation Committee

Thank you for your email sent on 30 May 2013. Paul Florian, Manager of Environmental Health and I have taken the opportunity to review both the letter from Senator Cameron and associated transcripts. We disagree with the assertion made by Drs Jeremijenko and Landos that a communicable disease outbreak occurred in relation to human exposure to either fish or seawater in Gladstone Harbour, Queensland in 2011.

Central Queensland Public Health Unit (CQPHU) received reports from a total of 43 people who were concerned they might have been unwell or had infections or other skin conditions as a result of contact with diseased fish or seawater. Most of these people were interviewed during an initial investigation or subsequent follow up, to establish whether there was any clear pattern of illness among interviewees and to identify possible links between diseased fish and risks to human health.

A human health risk assessment was completed on 06 October 2011, including findings from 24 people who initially reported health concerns. This investigation identified no link between the conditions found in fish and human health issues. CQPHU continued to investigate reports over following months. No new information was identified which may have changed the findings of the October 2011 report.

An independent scientific panel reviewed the investigation undertaken by Queensland Health and other Queensland Government agencies and reported on this review in January 2012.¹ The reviewers concluded on page 10 of the report that:

¹ Gladstone Fish Health Scientific Advisory Panel (2012). Final Report. Brisbane: Gladstone Fish Health Scientific Advisory Panel. Available at: <http://www.ehp.qld.gov.au/gladstone/pdf/gladstone-sap-report.pdf>

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"Queensland Health had conducted an appropriate and adequate investigation of the fishers. The Panel agreed with Queensland Health that the cases described did not form a single outbreak of one disease. The Panel agreed that there was no indication of an outbreak of disease in fishers that could be linked with disease in fish in Gladstone Harbour and agreed that additional investigations by Queensland Health of this group of fishers was not warranted."

During the investigation, a number of fishers described infected injuries and skin infections such as boils. Laboratory testing had not been undertaken by health care providers in many cases. The cause of infections, where it was known, was *Staphylococcus aureus*. In particular, community-acquired MRSA or non multi-resistant Methicillin Resistant *Staphylococcus aureus* (nmMRSA) was identified in five individuals.

Staphylococcus is spread through person-to-person direct contact, especially in crowded situations. It also spreads when people share personal items like towels and razors. Breaks in the skin such as cuts and scratches also increase the risk of infection. These staphylococcal infections were not considered to be related to handling diseased fish.

Thank you again for your email. Paul Florian and I are very happy to discuss this letter if you have any additional questions and would welcome any further enquiries in relation to this matter.

Should it wish to do so, GPCL is also welcome to attach this letter with its response to the Senate Committee.

Yours sincerely

Dr James Smith
Director and Public Health Physician
Central Queensland Public Health Unit

Calliope River bug takes man's leg

Simon Crase | 20th May 2010 2:04 PM |

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Fisherman Jim

almost lost his after picking up vibrio vulnificus in the Calliope River in February
A RARE flesh-destroying bug has cost a man his leg and almost his life after being struck down by devastating bacteria in the Calliope River.

Fisherman Jim, as he's known to his mates, was dubbed the "the miracle man" by Rockhampton medical staff, because he survived the horrific ordeal.

In late February, Fisherman Jim, 75, and his son went fishing at the start of the Calliope River anabranch for a peaceful afternoon's fishing, unaware of the tragic saga to unfold over the next few days.

Jim had got a small cut on his leg sometime during the fishing trip, during which they caught a couple of crabs, and at day's end, he climbed out of the boat into the river at the boat ramp to make the short trip to dry land.

"That's when we're pretty sure the bacteria got into him," he said.

Fishermans Jim's health deteriorated in no time, and his family .

"At midday the following day, Dad was restless, and the cut had a blister on the side of it," he said

Later that afternoon, Jim's son got a phone call from his step-mother to say his father's calf muscle had gone numb.

"He'd gone downhill, and his calf muscle had gone a blacky, purple with massive blisters through it," Jim's son recalled.

"I got a shock, because I'd never seen him so sick. He was a tough man."

Fisherman Jim was then taken to Gladstone Hospital by ambulance after a 000 call was placed.

"By 4pm he was incoherent. The thing spread like a bushfire," Jim's son explained.

Within an hour he became more confused and local doctors had no idea of Fisherman Jim's ailment.

"No-one had ever seen anything like it," Jim's son said.

What it was turned out to be a deadly bacteria, *Vibrio vulnificus*.

Queensland Health Central Queensland Public Health physician Dr Margaret Young said the germ occurs naturally in tropical and temperate coastal marine waters.

"It flourishes in water temperatures of 180C and over," she said.

"The germ does not eat flesh, but it releases toxins that can progressively destroy skin and muscles.

"Such infections may occur when a skin wound is contaminated by coastal seawater."

Infections have occurred from Cape York to the Gold Coast, and the germ is not a notifiable disease.

Fisherman Jim's condition worsened and he was heavily sedated as a preventative measure to stop him

"They virtually had to knock him out because he was so confused and kept pulling the instruments on him off," he said.

At 11pm, the decision to send the stricken man to Rockhampton was made, as nothing could be done to assuage the man's suffering.

"His body swelled up, the veins in his eyes stuck out, his eyes bulged, and his head swelled to another third of its size," Jim's son said.

"They were running around watching him die, virtually.

"The (Gladstone) doctors were doing the best they could, and tried everything.

"One of them told me Dad's survival rate was 10 per cent, according to one of the doctors when I asked."

Fisherman Jim arrived at Rockhampton ICU about 2am, with his son following close behind.

"I got a thousand and one questions (about it) and I was sure it had come off the ocean," he said.

"They were excellent up there, I take my hat off to them."

In a fortuitous turn of events, a Rockhampton doctor had encountered the bacteria in Darwin and ordered a concoction of antibiotics be immediately administered.

"The leg was going blacker and had spread above his knee," he said.

At 7am, Fisherman Jim's son had the unenviable task of authorising doctors to remove his father's leg.

The surgery stabilised Fisherman Jim, however the chances of him living were exceptionally slim.

"The doctor was dead set sure he'd die," he said.

"His organs started shutting down, and if they hadn't taken his leg off he would have been dead within an hour."

Within four days, the bacteria had been isolated and Jim's son was told its name, vibrio vulnificus.

"It travels just underneath your skin, and all the tissue behind it is dead," Jim's son said, after doing his own amateur research online.

In a coma for three weeks, Fisherman Jim was on dialysis for four weeks, the swelling taking that length of time to subside.

"He was probably supposed to die, so no one ever said anything until he woke up," Jim's son said.

"I thought he was dead and it was the machines keeping him alive.

"He's a tough old bastard, very fit, and I've seen him get hit by box jellyfish and not even blink."

When Jim awoke, for the first day there was no movement, and Jim's son feared the family would be left with a shell. Then he slowly came around.

"He was very confused and couldn't understand why the family was there and we'd say 'how are you?' and he'd say, 'I'm alright, how are you?' and here he is lying there missing a leg," his son said.

Jim's son suspects bilge water from a large vessel may have been the cause, however this has not been confirmed.

"(Rockhampton doctors) told us what it was and where it came from, and they said it was the likely scenario," he said.

"They couldn't be sure but it was the one that made sense."

A short conversation after the ordeal gave an insight into Fisherman Jim's thoughts on his recent loss of limb, when an intern asked him about only having one leg.

"He replied, 'You don't really worry about s**t like that'," Jim's son said.

"(The intern said) 'No mate you don't do you,' and lifted up his pants and said, 'Look you can get them in titanium in any colour you like'."

As for the medical team in Rockhampton who managed to save a man's life when he was three parts way to being dead, Jim's son believes it was their skill and knowledge, as much as his father's tenacity and spirit, that kept him from dying.

"They were brilliant, I've never seen a team like it, the way they worked together, and Central Queensland is lucky to have them," he said honestly.

Every time Fisherman Jim's son now gets a cut, he's paranoid.

"I want the awareness spread that this thing is out there," he said.

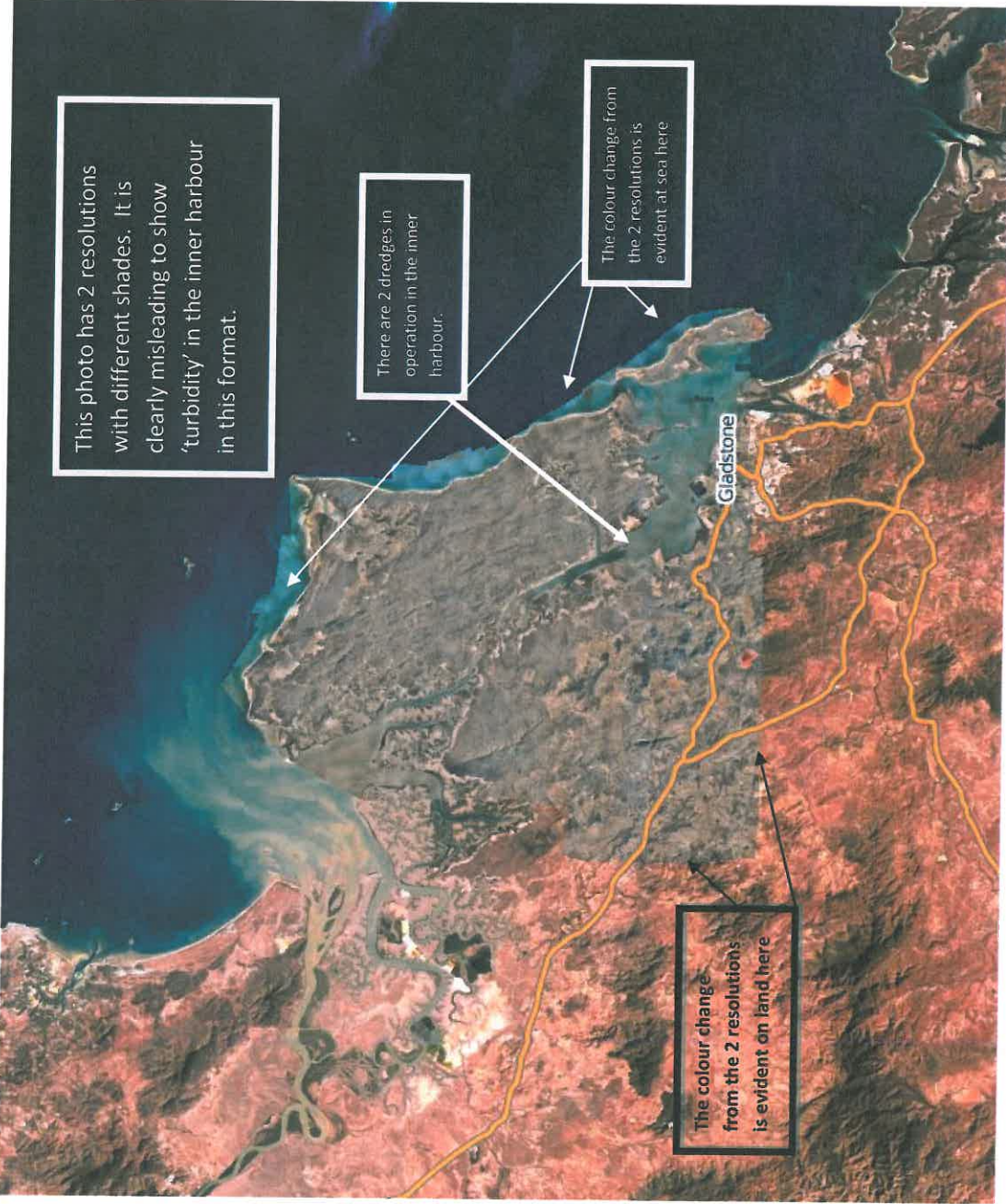
"Carry spray and if you get a cut, spray it and gurney the cut.

"It could be last cut you ever get."

July 2011 (Nearmap.com) - Gladstone zoom



July 2011 (Nearmap.com) - Gladstone region





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Review of Petus and Devlin (2012)

Using satellite maps to document the extent of sediment plumes associated with dredging activity in Gladstone Port's western basin, Queensland

A report prepared for the
Gladstone Ports Corporation

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SCOPE

Provision of an independent review of Petus and Devlin (2012) *Using satellite maps to document the extent of sediment plumes associated with dredging activity in Gladstone Port's western basin, Queensland*

Executive summary

The study by Petus and Devlin (2012) to use MODIS imagery to attempt to differentiate the acute suspended sediment loads associated with dredging activities from suspended sediment loads due to other natural processes has a number of significant limitations. These reduce the applicability for quantitative assessment of the extent of sediment plumes associated with dredging activities in the Port of Gladstone. The major limitations of the study are:

- The use of an uncalibrated algorithm to infer Total Suspended Matter concentration (mg l^{-1})
- The inappropriate treatment of tidal data in the analysis of natural processes contributing to elevated TSM.
- The omission of wind forcing in the analysis of natural processes contributing to elevated TSM.
- Sub-optimal approach for the detection and removal of cloud contamination of remote sensing imagery.

The major limitations listed above render much of the temporal analysis of TSM dynamics irrelevant, as the dominant natural processes controlling natural variability of suspended sediment concentrations were either omitted or represented incorrectly. Further, the analysis which was intended to establish correlations between TSM and dredging activities was almost entirely qualitative and did not possess the rigour of a thorough statistical analysis.

Remote sensing algorithms to determine suspended sediment loads are not generic. However, methodologies that utilise comprehensive in-situ data across the range of TSM representative of real-world conditions to inform the development of an empirical algorithm specific to the location in question are valid and could be successfully applied to Gladstone Harbour.

Background

The Australian Institute of Marine Science was commissioned by Gladstone Ports Corporation to provide an independent review of the report entitled “Using satellite maps to document the extent of sediment plumes associated with dredging activity in Gladstone Port's western basin, Queensland”, authored by Drs. Caroline Petus and Michelle Devlin of the Catchment to Reef Research Group, Australian Centre for Tropical Freshwater Research (TROPWATER), James Cook University (ACTFR Research Publication 12/02).

Introduction

The application of remote sensing techniques for coastal management is well established. Remote sensing techniques, either satellite or aircraft based, provide a cost effective means to map spatially heterogeneous features over broad areas, with imagery often available in near real-time and at temporal frequency sufficient to support cause/effect studies (see, for example, Klemas, 2011). In the context of coastal development along the Queensland coast, remote sensing techniques have been previously employed for operational monitoring of dredging activities and associated sediment plume at Hay Point, North Queensland (Islam et. al., 2007). The use of remote sensing to derive quantitative estimates of suspended sediment concentrations typically requires the development of empirical relationships between remotely sensed reflectance and in-situ observation of the parameters of interest.

The aim of the Petus and Devlin (2012) study was to use MODIS imagery to attempt to differentiate the acute suspended sediment loads associated with dredging activities from suspended sediment loads due to other natural processes such as tides and river flows. The study made several conclusions, including:

- Rrs(645) values increased after dredging in July 2011 and variability was not related to tidal currents or river discharge;
- Rrs(645) values during the 2011 spring are above historical values for the same season, including the spring of 2010 when river discharges were higher;
- measurable Rrs(645) levels were observed up to 34 km from the main dredging site during the 2011 spring.

Nomenclature

The following acronyms and abbreviations are used throughout this review:

TSM: total suspended matters concentration (mg l^{-1})

Rsr: remote sensing reflectance

Rsr(645): remote sensing reflectance of MODIS band 1 (centred on 645 nm)

Review of methods and their limitations

Derivation of TSM from MODIS Imagery

Petus and Devlin (2012) estimate total suspended matter (TSM) from MODIS Band 1 reflectance (Rsr(645)) using an algorithm developed to map turbidity and TSM in the coastal waters influenced by the Adour River plume, in the Bay of Biscay, and based on empirical relationship between *in situ* TSM concentrations and Rsr(645). This algorithm is specific to the characteristics of the particular suspended sediment in the region of the original study, and therefore not quantitatively applicable to regions with differing sediment characteristics, and additionally, not applicable to the case of dredging where sediment characteristics may change over the duration of the dredging activity as different material is dredged and disposed offshore. Such algorithms are very site-dependent which is evident when compared to similar studies such as that of Moreno-Madrinan et. al. (2010).

Application of the Petus et .al. (2010) algorithm to another region, as has been done in the present study, will yield a value that is an uncalibrated proxy for TSM. The authors acknowledge this significant limitation of their approach, but argue that the application of the algorithm will indicate qualitative changes in TSM. However, the relationship between TSM and Rsr(645) as given in Petus et .al. (2010) is a quadratic polynomial, and as such assuming qualitative changes in TSM from measured changes in Rsr may prove misleading without calibration to the reflective characteristics of the specific suspended sediment. Inferring qualitative changes from changes in Rsr would require the range of variability of Rsr to be similar in both cases (Adour River and Gladstone), and this is not clear from the report.

Setting specific thresholds to delineate clear or turbid water (e.g. 5 and 10 mg^l⁻¹, see section 4.3.2) in order to calculate % occurrence is irrelevant, given that the TSM calculation is uncalibrated for the region of the Port of Gladstone, and therefore the estimated TSM concentrations are not quantitative and do not represent real-world TSM values.

It should also be noted that the equation given in Section 4.3.1 of the report is incorrect in describing the polynomial relationship between TSM and Rsr(645) as given in Petus et. al., 2010 – but this is most likely a simple textual error.

The analysis uses only 1 spectral band of the MODIS data, and is therefore a simplified view of the spectral reflectance of the water. In shallow coastal areas, the satellites are able to ‘see’ the seabed under some conditions (clear water) and in order to accurately distinguish the seabed from a turbidity signal, you must first determine the minimum threshold value from a history of observations, matched with bathymetry.

The above listed limitations in applying the uncalibrated algorithm of Petus et. al. (2010) could be significantly reduced if the study included calibration of the parameters derived from remote sensing imagery, through development of an empirical algorithm based on in-situ observations of suspended sediment concentration, over the full range of variability, as was done for the case of the Adour River plume (Petus et. al., 2010). This need for local validation is acknowledged by the authors.

Section 3.2 of the report suggests that satellite images will be able to differentiate between dredge sediment and algal blooms, but there is no further discussion of the determination of algal concentrations using remote sensing techniques.

Cloud detection

The authors acknowledge that clouds present a problem by contaminating the MODIS band 1 imagery, and that the problems posed by cloud contamination are increased when clouds are located above turbid coastal areas. In addition, during winter, low cloud can be of similar temperature to the underlying water and cloud contamination could be at the sub-pixel level (smaller than 250m in size), and as such, removing cloud contamination is not trivial. Contamination of the Rsr signal by clouds will lead to significant 'noise' in the remotely sensed image and propagate through the processing to contaminate the derived parameters and products (TSM, % occurrence and the like). The authors point out that clouds are "identifiable with expert knowledge by their particular shapes and locations" and explain that cloud detection was done manually. There is however contradiction in the definition of non-cloudy days: "*Non-cloudy* days were defined as days when less than 90% of clouds cover the Gladstone inshore area" on page 12, but then on page 35, "*Selection of non-cloudy days was done manually and based on the acceptance of 10 % of cloud cover over the Port area*".

Approaches that use a combination of MODIS bands are able to improve confidence and accuracy in cloud identification, and although these methods may present particular issues related to differing spatial resolution between bands, they should be considered in place of manual selection.

Determination of the role of tidal currents and river flood plumes in controlling natural variability in TSM.

The report attempts to differentiate the acute suspended sediment loads associated with dredging activities from suspended sediment loads due to other natural processes such as tides and river flows. As such, an attempt is made to infer the contribution of tidal flows and river discharges to the TSM as estimated from MODIS imagery. There is no discussion of the contribution of wave driven resuspension and its impact on TSM.

The Boyne and Calliope Rivers discharge into the vicinity of the Port of Gladstone, and both of these rivers are gauged with data provided via the Australian Bureau of Meteorology. It is appropriate to use river flow data as a proxy for the enhanced delivery of suspended matter through river discharges. The contribution to the offshore turbidity from other rivers in the vicinity of Gladstone (e.g. Fitzroy, Burnett, Mary) should have also been included in the analysis, especially given the significance of the river flow in this region over the last few years.

There is significant problem with the approach taken to estimate tidal range as a proxy for tidal currents. The report states that "*tide amplitude is calculated as the difference between the high and low tide levels (m) measured at mid-day*". This approach is entirely incorrect, and this is reflected in the tidal amplitude chart plotted in Figure 5-2, which does not show the sinusoidal nature of the various tidal harmonics that should show a fortnightly envelope of spring to neap tides in a plot of tidal sea-level data. The inappropriateness of the method for determining tidal range is highlighted on page 16 with the statement that "*Tidal amplitudes range from 2.5 to 4.3 meters (Figure 5.2) and*

are considered representatives to tidal current speeds". In reality, maximum daily tidal range at Gladstone range between ~1.2m and ~4.0m, during neap and spring tides, respectively. This error in estimating tidal range as a proxy for tidal current is a major flaw of the approach taken in this study and it impacts the conclusions regarding the attribution of elevated TSM to either natural causes or dredging. Further, the report indicates that daily tidal range was then smoothed using a 20-day running average (see page 20) which would remove any tidal signal from the data and render the tidal data useless.

Of equal concern is the failure of the study to take into account the effects of wave driven resuspension. Waves are a key factor controlling coastal turbidity, in both exposed and semi enclosed embayments such as Gladstone Harbour, and in shallow coastal areas, wind speed and direction can be used as a reliable proxy for wind driven waves and the concomitant resuspension of sediment. Wind data for the period covered by this study are available from the Australian Bureau of Meteorology. It is surprising that such an obvious process that contributes to coastal turbidity was not included in the analysis of natural processes that impact the suspended sediment concentrations in the region of Gladstone Harbour. The omission of wave resuspension is another significant flaw in the approach adopted in this study, and again it impacts the conclusions regarding the attribution of elevated TSM to either natural causes or dredging.

Temporal analysis of satellite derived TSM and potential correlations with dredging activities and natural processes

The approach to correlate historical observations of Rsr/TSM with tidal currents and river discharges is entirely qualitative even though there appears to be sufficient remote sensing and environmental data to enable a more robust statistical analysis. The lack of statistical analysis limits the conclusions that can be made.

Further, the analysis of the Rsr values at selected pixels within the study domain to determine long term trends is again limited by an approach that is almost entirely qualitative. An attempt is made to fit a linear relationship between Rsr/TSM and time (see Figure 5-3, page 18), but this approach is completely inappropriate for a situation where the TSM displays significant temporal variability, as it does in Gladstone harbour.

Review of conclusions

The authors begin their conclusion by listing several of the limitations of study, including:

- acknowledging the potential contamination of some Rsr data by clouds.
- acknowledging that radiance reflected from adjacent land can contaminate the MODIS signal in near shore locations;
- recognising that bottom influence can increase the Rrs(645) signal recorded in shallow regions;
- acknowledging that atmospheric corrections of the remote sensing product used are only adapted to turbid environments;
- acknowledging that the TSM algorithm employed to convert Rrs(645) to TSM values was not validated in the Great Barrier Reef.

Other significant shortcomings of the approach include:

- the uncertainty of extracting even representative qualitative changes from the non-linear relationship between Rsr and TSM;
- the omission of waves from the analysis;
- the inappropriate calculation and use of tidal data;

The three shortcomings listed above render much of the temporal analysis irrelevant, as the dominant natural processes controlling natural variability of suspended sediment concentrations were either omitted or represented incorrectly. Further, the temporal analysis and the authors' attempts to show correlations between TSM and dredging activities are entirely qualitative and do not possess the rigour of a thorough statistical analysis.

The conclusion that there is *“an increase of the Rrs(645) (proxy for sediment concentrations) values after the dredging resumed in 2011 above the observed values of previous years, including 2010, which was influenced by the Fitzroy floods”* does not acknowledge the limitations listed above and is based only on qualitative analysis.

The statement that *“Rrs(645) values over 0.0117 (i.e. TSM values over $\sim 5\text{mg l}^{-1}$) are observed as far as 35 km from the main dredging site”* is misleading due primarily to the uncalibrated algorithm used in this study and therefore the arbitrary nature of the Rsr threshold values chosen to delineate turbid and clear water.

Requirements to robustly apply remote sensing technique to Gladstone Harbour

Remote sensing techniques to determine coastal turbidity have been successfully applied in other geographical regions (see for example Petus et. al., 2010; Moreno-Madrinan et. al., 2010). These studies utilised extensive in-situ observations to develop empirical algorithms specific for the locations in question. The algorithms developed are not generic due to the strong dependence of the turbidity and spectral reflectance on physical, biological, and mineral characteristics of particles and dissolved materials, and therefore should not be applied under different geographical and oceanographic conditions. Algorithms for the GBR inshore lagoon have been developed and are applied by CSIRO (Brando, pers. comm.; Brando et. al., 2012) but are not yet publicly available for wider application. However, methodologies that utilises comprehensive in-situ data across the range of TSM representative of real-world conditions to inform the development of an empirical algorithm specific to the location in question are valid and could be successfully applied to Gladstone harbour.

Other comments

The report is not well written with many grammatical and spelling errors.

The report lacks a good map of the study site showing all the areas referenced in the report, including specific areas of concern and the East Bank dredge disposal area.

The figure labelling and referencing throughout the document is generally incorrect and inconsistent making it extremely hard for the reader. Examples: Reference (on Page 9) to Figure 1 in fact refers to Figure 4.1; Reference (on page 21) to Figure 9 presumably relates to Figure 5-7, and the reference to Figure 10 leaves the reader guessing.

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