

Short Summary

Response to Gladstone Ports Corporation scientist submissions

<http://www.environment.gov.au/coasts/gbr/gladstone/submissions/pubs/24gpc.pdf>

which critiqued the Future Fisheries Veterinary Service document:

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

Report for the Senate Committee considering the GBR Bill, 30 May 2013.

Dr Matt Landos

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Director, Future Fisheries Veterinary Service

Dr Batley CSIRO Review 1.

Dr Batley falsely claims the following:

- 1) A weight of evidence approach was not used in my report
- 2) Gladstone Harbour does not contain contaminated sediments of concern
- 3) Future Fisheries Veterinary Service reached unsupported conclusions
- 4) Future Fisheries Veterinary Service had not adequately considered the potential role of freshwater influx in December 2010-January 2011.

Dr Batley's claims are based on CSIRO water and sediment testing data collected well after the aquatic animals became diseased. CSIRO examined zero animals. The CSIRO study used inappropriate techniques for sediment sampling. Dr Batley did not mention the CSIRO and the Scientific Advisory Panel recommendations to test animals (biota). Dr Batley did not comment on the other reports (GHD EIS, Jones et al 2005, CRC study) which demonstrate high levels of metal/metalloid contamination in Gladstone Harbour sediments.

Dr Batley fails to acknowledge and/or explain the use of false baselines, the origin of all the metal/metalloid elevations in the green turtles sampled in Gladstone Harbour in July 2011, or diseases documented in pathology samples of animals in the FFVS project.

It must be noted that Dr Batley does not have veterinary training, nor training in pathology. Therefore he does not have the required qualifications to dismiss my conclusions or the findings of the highly qualified veterinary pathologist who examined the numerous animals sampled for my study.

The spatial (place) and temporal (timing) epidemiological evidence shows there is no link to a freshwater based stress trigger to the events observed. It also cannot explain any of the observed pathology, or the observed parasitic infestations which were all marine organisms. I note that a larger flood occurred in January 2013, nearly double the size of the 2010-11 flood. Yet there has been no repeat event in Gladstone, due to the dredging moving away from the heavily contaminated areas, the ocean dumping ceasing, the dirtiest dredges stopping, the bund wall leak being sealed and the absence of a major bund wall and sediment scouring. The necessary causes for the disease outbreak were an uncontrolled harbour development that caused massive resuspension of contaminated sediments.

Dr Nowak, University of Tasmania: Review 2.

Dr Nowak falsely claims FFVS misinterpreted papers on the relationship of parasites, contaminants and fish immunity.

Dr Nowak suggests that FFVS did not consider that contaminants have been known to decrease intensities of parasite infestations. Such an academic observation and extensive discussion is not relevant when the case at hand is one of elevated parasite intensities, not diminished parasite intensities. The literature clearly supports the FFVS hypothesis that contaminants can increase the intensities of the exact types of parasites seen in Gladstone fishes. Gladstone fishes were demonstrated in the FFVS study and in Biosecurity Qld studies to have elevated parasite intensities, associated with pathological skin lesions on sharks, barramundi (BQ) and queenfish.

This was the only criticism of the proposed causal pathway, which Dr Nowak otherwise found “logical and convincing”.

No alternative hypothesis was offered by Dr Nowak to explain this, or any of the lesions. No evidence to support any alternative hypothesis is offered.

No comment is offered as to whether Dr Nowak considers freshwater a potential cause of these skin lesions in fish.

Dr Nowak made numerous comments about how the study could have been improved, without any knowledge of the resources available. The budget was only ~\$55,000. I agree that more samples would have been better, more sampling sites would have been better, more laboratory experiments would have assisted, more tissue toxicology should have been done, more speciation of metals in

water, more immunological assays and more bioassays were appropriate. I also note that GPC, DERM and Biosecurity Queensland did not undertake all of these actions.

Dr Nowak suggested the crab prevalence survey data was statistically adequate, as was the queenfish parasite data.

Dr Nowak agreed that copper induced shell lesions were likely in Gladstone crabs, and that the prevalence study showed a marked elevation which was statistically significant.

Dr Nowak agreed with FFVS that the project had resulted in resuspension of sediments, increased risk of boat strike, increased noise.

Dr Nowak's claims regarding misinterpreting 2 other papers of the >200 in the reference list are also without basis. Details are provided in the full response paper provided to the Senate Committee.



Response to Gladstone Ports Corporation scientist submissions

<http://www.environment.gov.au/coasts/gbr/gladstone/submissions/pubs/24gpc.pdf>

reviewing the Future Fisheries Veterinary Service report:

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No payment was received from any source for the production of this report.

1: CSIRO Dr Batley report contained with Gladstone Ports Corporation submission to Minister Burke's Gladstone Review.

Executive summary

Dr Batley falsely claims the following:

- 1) A weight of evidence approach was not used in the report
- 2) Gladstone Harbour does not contain contaminated sediments of concern
- 3) Future Fisheries Veterinary Service reached unsupported conclusions

Dr Batley's claims are built around his assessment of largely irrelevant data, collected by CSIRO well after the aquatic animals of Gladstone had become sick. Indeed, recovery of surviving animals was underway by the time CSIRO tested sediments and water. The CSIRO data set is contradicted by more appropriately timed data collection which demonstrates significant metal and metalloid contamination of Gladstone Harbour sediments and water contrary to the opinion expressed by Dr Batley. There are gaps in the Government data due to the poor standard of the monitoring program, and the failure to implement recommendations from CSIRO and the Scientific Advisory Panel to test biota.

Dr Batley does not acknowledge the use of false baselines, which were created from data collected after the project had already commenced.

Dr Batley does not explain the origin of all the metal/metalloid elevations in the green turtles sampled in Gladstone Harbour in July 2011. Nor the origin of copper and zinc elevations leading to shell disease in Gladstone mudcrabs.

Dr Batley offers no plausible explanation of the diseases observed in the animals sampled in the FFVS project.

If there were no problem with metals in Gladstone, why did the surrounding industries fund a multi-million dollar monitoring program which focuses on metals- the PCIMP?

Why did GPC spend millions on an EIS looking at sediment contaminants, if there was no potential problem?

I urge scientists and non-scientists to view Dr Batley's conclusions with extreme caution, as they do not approximate robust science nor do they add to understanding the widespread aquatic animal diseases which occurred in Gladstone Harbour.

Appropriateness of qualifications to review a veterinary investigation

Dr Batley does not have veterinary training, nor training in pathology. He acknowledges this in his introduction. Yet he goes on to dismiss the diagnosis reached in the investigation.

In the absence of appropriate qualifications and an absence of knowledge of pathology, which makes up the majority of the report, I contend, he does not have the required qualifications to dismiss the conclusions or the findings of the highly qualified veterinary pathologist who examined the numerous animals sampled for the study.

I am a registered Veterinary Surgeon in Queensland, and operate under the State legislation.

To reach a diagnosis of animal disease in Queensland is an Act of Veterinary Science under the Veterinary Surgeons Act 1936. To reach such a diagnosis, requires a veterinarian to be registered as the extract from the Act below notes.

“2A Meaning of *veterinary science*

(1) ***Veterinary science*** means the science of veterinary surgery or veterinary medicine.

(2) ***Veterinary science*** includes the following—

(a) diagnosing diseases in, and injuries to, animals, including, for example, testing animals for diagnostic purposes;

(b) giving advice based on a diagnosis under paragraph (a);”

The substance of the study which I undertook involved a veterinary investigation involving the use of a US Board Certified veterinary pathologist who has a PhD in epidemiology, Dr Michelle Dennis. A co-investigator of the report was Dr Ben Diggles who has a PhD in marine parasitology, and was involved in examining affected fish on board boats and in drafting the fish parasite section of the report. The report was also reviewed by Dr Brian Jones and Dr Rebecca McIntosh.

The examination of water science and environmental chemistry is a subset of a thorough veterinary investigation. On its own, it is inadequate to assess the likely cause of the sick and dead aquatic animals in Gladstone in 2011-2012.

My business has operated in all States of Australia and its core function is the diagnosis of the causes of sickness in aquatic animals. I have worked as an aquatic animal veterinarian full time for the past 13 years. My business services some of the largest aquaculture businesses in Australia with fish health veterinary expertise. They retain my services to accurately diagnose and resolve health problems as they arise.

Format of response

Page numbers refer to pages of the Scientific Reviews of Dr Batley and Dr Nowak.

Addressing specific claims made by Dr Batley

Page 2

Dr Batley claims in Section 2 paragraph 3 that the only water quality data I presented in the report was for turbidity. Unfortunately Dr Batley failed to notice the numerous references to the water quality documents produced by DEHP and the Gladstone Area Water Board. He also overlooks the presentation of copper data in the report. The report was already long, and to have included all of the data from the many DERM and EIS reports was not practicable. I did however reference it, so the

reader could go back to the source data and confirm that the conclusions which I drew were indeed accurate.

Page 7

Dr Batley detailed that he examined the executive summary- which was prepared for a non-science audience, and the conclusion lines of chapters. It is necessary to communicate the results of a publically funded project back to the public which includes non-scientists. The substantiating science is contained within all of the detail of the report, and the accompanying references, should readers chose to seek it.

Page 8

Dr Batley claims discussion is poorly structured and contains many generalisations. He states that “A more systematic discussion of the various issues would have been useful.”

In a report which brings together data from a range of different sources it is necessary to generalise, to avoid the report becoming unnecessarily long.

In response to Dr Batley, I note that no such thorough assessment of the issues has been produced by any Queensland Government Department, CSIRO, nor a Commonwealth Department. I also note that no independent review of any Government report on Gladstone has been published. So whilst the discussion may be imperfect in his view, it represents the largest literature review and aggregation of data analysis on the Gladstone project and its impacts. It presents the largest data set of detailed histological findings describing the disease status of aquatic animals in Gladstone. The reader needs to bear in mind the budget of the project was ~\$55,000. Compare this to the funding CSIRO received to undertake research in Gladstone recently of \$1.2 million.

Page 9

Dr Batley compares the weight of evidence veterinary investigation to an environmental assessment. Unfortunately The veterinary investigation of disease is not an environmental assessment. Hence to go on to describe what happens in an environmental assessment is irrelevant. The report provides details of a veterinary investigation.

Dr Batley suggests I did not apply a weight of evidence to the data describing the use of the + or – symbols to denote support or a lack of it for the evidence being considered.

Page 10

Dr Batley states “There is no weighing of evidence in the Landos Report.” Dr Batley appears to have overlooked the terminology which I did use to describe the weight of evidence which was uniformly: highly likely; likely; unlikely; highly unlikely.

Such terms are qualitative and appropriate for describing a weight of evidence approach. To apply such weightings requires knowledge of the fields of science used including epidemiology and pathology. These are not part of Dr Batley’s expertise by his admission. Dr Batley claims the weight of evidence assessment is based on a literature review, without comparison to field data. This is incorrect. The detailed reference list including the lengthy EIS documents all contain data used in the

report to support the statements made. Moreover the literature review is constantly referenced back to the clinical data gathered which was generated from gross pathology examinations and histopathology.

It is notable Dr Batley did not include any reference to the Entox March 2012 Investigation of Contaminants in Green Turtles in Gladstone produced by Dr Caroline Gaus, this became available after the publication of the FFVS report. This included data showing high metal levels in the blood of turtles dying in Gladstone Harbour in July 2011. He offers no explanation as to where these metals/metalloids may have come from to be in the blood of these turtles in this location. I note that the exact compounds were identified in the GHD EIS Sediment assessment Appendix L to be above sediment guideline levels in several areas which were slated to be dredged. And further note that dredging and disposal had categorically resuspended massive quantities of this contaminated sediment due to the tidal conditions, dredging methods employed and absence of sediment controls used.

Dr Batley claims fish should/could have swum away from the toxic dredge spoil. This fails to acknowledge that some residual barramundi which had come over Awoonga dam and taken up temporary residence in the Boyne River, were living in freshwater, until the dam stopped overflowing, and tidal ingress brought marine water back into the river. By this stage in July 2011, all the water in front of them in the harbour was contaminated with resuspended dredge spoil, from the leaking bund wall, the dredging resuspension and the wash in from the ocean spoil dumping ground which was immediately to the east of the dumping ground. Those fish did not have the option of escape. Neither did the mudcrabs- which exactly as expected in response to increased metal uptake, showed increased rates of shell lesions, and massive drops in catch rates, at the time resuspension of contaminated sediments was occurring. Freshwater influxes have never been recorded to cause this shell disease in mudcrabs in Gladstone or anywhere else in the world. Dr Batley also did not comment on the fact that very low copper exposures can impact on the fishes sense of smell- thereby impairing its aversion responses. From the GAWB data and DERM data elevations in copper occurred sufficient to cause this effect.

Dr Batley claims that the stressor could be removed, thereby reducing or reversing impacts. This observation fails to take into account the reality of the turbidity monitoring which showed exceedances over trigger values unrelated to rainfall which were maintained for long periods of time, coincident with the harbour development. Dr Batley also failed to report on the rates of water movement out of the western basin, which is very slow to exchange. These details were in the report.

Dr Batley claims the significance of the area of dredging as a fraction of the greater area of the Harbour needed to be considered. I presented satellite data and referenced the JCU report showing a 34km dredge plume, which extended throughout the Harbour. I also witnessed the dredge plume from the ocean spoil dumping ground during sampling. I took first hand evidence from fishers who were on the Harbour every week about sudden and unprecedented changes to turbidity, well after floodwaters had cleared away. There were also rises in metals such as aluminium documented in Rodds Bay only after the commencement of the project. There were multiple lines of evidence which demonstrate the spatial extent of exposure. The area of impact was not limited to the footprint of dredging- it was denoted by the extent of sediment resuspension plumes.

A profound drop in catch rates of crabs, prawns and fish was documented and presented. This data demonstrates a trend which is the opposite of what was to be anticipated, as freshwater influxes are documented to increase productivity of inshore fisheries. An FRDC report showing this effect in Qld was referenced in the FFVS report as science based support.

Dr Batley claims measurements of copper and zinc in Gladstone Harbour were well below Australian sediment water quality guidelines. This statement contradicts the previous work of Jones et al 2005, and that of GHD in the EIS sediment assessment, where multiple exceedances for copper and zinc were reported adjacent to Curtis Island - in the location where DERM water testing subsequently found elevated copper levels. The tests in the following months were not available from DERM as strangely the samples were considered "contaminated" and not reported. No explanation was offered as to why more were not collected, or why only copper and zinc samples were contaminated. This location was also spatially where the FFVS prevalence survey of mudcrab lesions found the highest percentage of affected crabs.

Page 12

Dr Batley references Angel (2012), which used an unacceptable methodology of sample collection for sediment assessment. It only used grabbed samples which are inferior to the technique used by GHD in the EIS. Such sampling methods cannot predict the contaminant load of deeper sediments which were to be dredged. It is also notable that Angel 2012 results were collected after the dredging had commenced more than 14 months earlier. Thus the CSIRO December 2011 results could not be representative of what had been dredged months earlier when diseased animals first appeared. The study could not sample the same sediment from the already dredged areas, which were those which contained the contaminants which led to the diseased animals. CSIRO sampled after the main event was over. They also sampled on a very small tide, which leads to the lower water velocity and the least resuspension. At the time they sampled not all of the dredges were active.. CSIRO only sampled over 3 days, which severely limits their capacity to make statements about water quality outside of those 3 days. CSIRO did not sample for toxic algae, which the FFVS study did, finding toxic *Lyngbya* blooms.

The GHD sediment study contradicts the opinion of Dr Batley's assessment of sediments is.

http://www.westernbasinportdevelopment.com.au/eis_documentation

Appendix L.

These sediment studies show that areas of the western basin, those that were part of the dredging and dumping program were contaminated at levels that substantially exceeded the guidelines. They were only brought under the guideline by a process of dodgy averaging with sediments from the wider areas of the shipping channel which had lower levels of contaminants. True hotspots were abundant in the western basin and that material was dumped at sea, and also leaked out of the bund wall as it was dredged.

P31 of the GHD report shows manganese sediment concentrations 10 times over the guideline. And 127/396 samples were over guideline. It also shows arsenic in 20 samples over guideline. For many of the metals there is no NAGD guideline value eg vanadium, manganese, iron, aluminium.

P 64 gives the full summary of exceedances. And demonstrates how the use of averaging high results with lower results was done to bring levels under the guideline for manganese.

When metal/metalloid rich areas were dredged, the sediments did not automatically get their contaminant concentrations reduced, by the areas remote to the dredging that day with lower contaminant loads which had yet to be dredged. The resuspension plume from contaminated areas will contain the elevated contaminant loads at that time. It is not reduced by areas which were yet to be dredged.

Page 12

Dr Batley claims dissolved copper levels were similar before and after dredging, yet fails to examine the activities which were taking place in the western basin when so called “baseline” sampling was taking place. The construction dock was already underway with dredging and boat activity. Hence the GPC baseline was contaminated to begin with. Notably the only high readings in the “baseline”, were adjacent this dredging with the rest of the Harbour low. Dredge operations are recorded in the Mariners Notices regarding navigational hazards on the Harbour at that time in 2010.

Another study referenced in the report, http://www.ozcoasts.gov.au/pdf/CRC/25-PCcontaminants_summary.pdf shows:

“However, arsenic, chromium and nickel concentrations were consistently above the ANZECC low interim sediment quality guidelines at many sites”

Yet Dr Batley repeatedly claims:

“The sediments dumped from Gladstone Harbour could certainly not be considered as metal rich, based on the analytical data available.”

This statement is manifestly incorrect, in light of the evidence presented here.

Dr Batley states that the crab disease was present before this project, on page 12. This statement is true, but misses the point. The first time the disease was reported, was by commercial fishers after major capital dredging in the late 1990's. This led to a University Masters study investigating the cause which identified elevated metals (copper and zinc) exposure to be the cause. The crab population took around 10 years based on commercial crab catch data and observations of crabbers to recover from this insult, during which time, no further major capital dredging occurred. The crab fishery in Gladstone was booming prior to this project, when once again the first alert of disease came from the commercial sector, who observed crabs with holes in their shells and rust spot- identical to the late 90's. Dredging was involved both times, and on both occasions it is not the presence of disease which is critical, but the massive increase in prevalence in the crab population. The prevalence data from the FFVS study shows the largest prevalence of shell disease ever in Gladstone, well above that of the late 90's and well above a reference site.

Dr Batley contradicts himself presenting data showing oysters did experience increased copper aluminium and zinc uptake when dredging occurred in GHD 2009 document reference.

This leaves the question- if there were no elevated copper aluminium or zinc loads in sediments, where could these contaminants suddenly have come from? Even the authors he quotes from the

EIS considered that the bioavailable uptake by oysters of these metals was the result of dredging. However Dr Batley maintains that there is no significant elevated metals in sediments, therefore no problem.

Page 13

Dr Batley suggests there was no evidence that dredging was the cause of oyster mortality. The pathology of scallops showed clear evidence of disease in bivalves in the harbour. First hand observations of the Gladstone Area Water Board hatchery operator described oysters falling off pylons in May 2011.

Dr Batley makes no comment on the failure of the monitoring program to deploy any oysters during the project, or during the investigation of the sick aquatic animals.

Dr Batley quotes an inadequate study to refute claims of impacts on Facing Island corals- Ayling 2012. This study used observations by eye in highly turbid water to try and determine impacts. No coral was tagged for repeat examination, no coral was examined at the laboratory for disease status. The coral I sampled by pathology was suffering bacterial disease, entirely consistent with stress from contact with ocean dumped spoil. The GBRMPA identified in submissions to SEWPAC in 2009 that coral communities on Facing Island were likely to be exposed to spoil from the ocean dump site.

Dr Batley claims there were no links of mortalities in turtles to dredging. He dismisses the comment regarding arsenic claiming levels are low in sediment and water. Entox released results showing high arsenic levels in blood of turtles. And as outlined above arsenic was documented to be elevated in Gladstone sediments. This evidence contradicts the claims made by Dr Batley.

Dr Batley claimed that I presented no evidence that seagrasses had changed more than seasonally. I referenced multiple seagrass survey documents which demonstrated the failure of seagrass recovery in Gladstone after the project commenced in late 2010. Those documents also show the decline of the area closest to the initial dredging prior to the floods, but after the early construction work had began.

Dr Batley notes that seagrass has declined in other areas outside of Gladstone due to other reasons. Such information is irrelevant in understanding why the seagrass in Gladstone has declined. There is clear evidence from historical port development and dredging of time coincident seagrass loss. At least 550ha were buried in the spoil reclamation area at Fishermen's Landing. GBRMPA pointed this out to SEWPAC in 2010 emails.

Page 14

Dr Batley makes the statement that there is no evidence that the loss of habitat and food sources are the **sole** reason for decline in fisheries, or whether there will be a permanent loss of fisheries habitat.

In the FFVS report I did not claim that loss of food and habitat were the "sole" reason for the decline in fisheries, what I did point out was that ~550ha of seagrass and mudflat were buried under the reclamation area. Queensland Government policy agrees that 1 hectare of seagrass produces

\$47,800 worth of commercial seafood a year, based on Costanza modelling. This permanent loss, will have a permanent effect. However the wider documented loss of seagrass meadows in the western basin means the impact is greater again, due to changed hydrology of the harbour. Specifically it will contribute to impacts on the Universally Outstanding Value of the Great Barrier Reef World Heritage Area. Losses of EPBC listed species of dugongs and inshore dolphins during the project exceeded the PBR of these species, suggesting they may now be forced into localised extinction.

Page 14

Dr Batley suggested seasonal increases in turbidity might affect fish larvae already with naturally high turbidity. What Dr Batley failed to mention is that turbidity in the harbour was high when it should have been naturally low- ie in winter when low rainfall is common. Most species do not spawn all year round, so timing of turbidity is an important consideration. The other impact of dredging was that turbidity was elevated repeatedly for very long periods of time, at levels not recorded in any of the baseline work. This did not mimic natural turbidity events from rainfall.

Page 15

Dr Batley claims ammonia toxicity is a low risk, based on sampling data. He does not comment on the adequacy of the location of the sampling sites, or the timing of the sampling in relation to tidal influences. Given the nature of the areas being dredged, there is a 100% probability of ammonia release. The fact that a large algal bloom was triggered is proof that large amounts of nutrient were released from dredged sediments.

Dr Batley quotes DERM claiming that at concentrations of dissolved oxygen over 50% that significant impacts on fish are unlikely. This statement is incorrect in relation to some marine fish species which will suffer impaired feeding, growth, immunocompromise and reproduction at such low levels. (WenWen, ZhenDong, & ShiJian, 2013) (Noga, 2000) (Remen, Oppedal, Torgersen, Imsland, & Olsen, 2012) (Cerezo & Garcia, 2004)

Dr Batley suggests that hydrogen sulphide will rapidly dissipate therefore it is not a risk. The problem is that it is also rapidly fatal to exposed animals. Fishes do not need to be bathing in hydrogen sulphide for weeks, for it to cause lethal effects. It is rapidly toxic to fishes, which is why there is an ANZECC value for ecosystem protection which is so low. Whilst hydrogen sulphide is rapidly depleted from marine water, in the case of this project it was being generated continuously for several months, with continuous dredging operations. This scenario of continuous release, was not the subject of the research Dr Batley quotes.

Dr Batley dismisses the toxicity of cyanide to marine fishes due to its rapid dissipation. Such a statement contradicts the knowledge of illegal use of cyanide by the ornamental fish trade to stun and capture reef species. The cyanide caught animals subsequently suffer from chronic toxicity and

die days to weeks after capture. The level reported at Gladstone was above ANZECC guidelines. Dr Batley does acknowledge the comments that due to its spatial input, it alone does not explain the observed diseased animals.

Page 16

Dr Batley suggests that due to seawater's buffer that pH drop is unlikely. In my experience, this depends on the amount of acid added. Numerous estuarine acid sulfate catchments on the east coast release sufficient acid to drop pH routinely in marine water to very low levels <pH 6. eg Richmond River- Tuckean Swamp; Manning River- Moto swamp; Trinity Beach-Cairns; Shoalhaven river. Studies in the shoalhaven demonstrate that marine biota in lowered pH water accumulate more metals due to greater mobilisation and bioavailability.

The issue is one of quantum. This project was unprecedented in scale in Gladstone, and mapping showed large areas of acid sulfate soils would be dredged. The risk was acknowledged in the EIS with an entire chapter of how to deal with this problem.

Dr Batley suggests the presence of acid sulfate soils is of no consequence given pH levels were not recorded to drop below 7.1. This does not make sense with the elevations of metals reported in oysters in the GHD 2009 study after dredging. Again there was no pH drop, yet metals were released in a bioavailable form, which were recorded to be taken up by oysters. So the data discredits the theory proposed that acid sulfate sediments will not contribute to metal releases. It is likely that due to poor sampling location and timing that low pH events were missed in the sampling.

Dr Batley claims that acidified porewater was unlikely to release any metals. Porewater is released with dredging so by definition must bring its contaminant load into the resuspension plume, whereby some of it will be in a bioavailable form. The activated metals do not all immediately become inert the moment the pore water is released into the marine water surrounding. Such claims by Dr Batley lack supportive science.

Page 17

Dr Batley does not acknowledge that the ANZECC guidelines systematically underestimate toxicity by not adding up mixture toxicity. That is, where aquatic animals are exposed to elevations of more than one metal at the same time. Such complex mixtures were what occurred in Gladstone during the dredging project. By failing to consider the actual mixture toxicity, a one by one approach can readily underestimate the true toxicity risks. The Entox Poulsen report he quotes raises this issue also.

Dr Batley suggested the FFVS criticism of the monitoring program was misplaced. I note other esteemed water quality scientists have deeply criticised the monitoring program for its inadequacies including Professor Jon Brodie and Professor Barry Hart. The failure of the monitoring to respond to masses of dying animals in April –September 2011 and undertake dissolved metal testing and testing for metal speciation is an omission which is not recommended in the ANZECC guideline. It was clear from April there was a major problem. The other failures of the monitoring program include: absence of use of sediment trays; sparse data collection only once monthly; no use of oysters as biomonitors; mysteriously missing data in key months for copper and zinc; no use of passive samplers; no testing in April to August 2011 when development was well underway.

Dr Batley suggests that copper's behaviour in marine waters would make it less bioavailable, inferring it was not responsible for problems observed. Dr Barbara Nowak's report contradicts this opinion and agrees with my interpretation of the crab disease. The crabs demonstrated disease which is known to be caused by increased exposure to copper and zinc. Subsequent testing I have done on affected mudcrabs has found massive elevations of copper in the affected crabs compared to the control site confirming they were taking up bioavailable copper (and zinc) up to January 2012. The question is then, what process has suddenly increased the bioavailability of these metals to crabs? In my opinion this can only have happened due to dredging- just as the GHD EIS documented in 2009.

Failing to understand that the pathology which the Gladstone crabs have, has only been described in association with increased metal exposure, is a key flaw in Dr Batley's assessment of copper.

Dr Batley mentions Aluminium very briefly, saying "apart from aluminium", but fails to touch on just how massive the increases were around the Harbour, and its potential for toxicity. Dr Batley fails to comment on the finding of high levels of aluminium inside barramundi gills tested by Biosecurity Queensland, indicating that fish were taking it up- ie it was bioavailable. He also avoids discussion of the aluminium results in the CSIRO report which were dismissed as outliers.

Dr Batley tries to diminish the facts contained in DERM's elevated water test results as somehow less valuable than those undertaken by the CSIRO scientists. Arguably Dr Batley has relied upon the least representative data to reach his conclusion that metals/metalloids played no role in causing the diseased aquatic animal outbreak in Gladstone. The data he relied upon was collected after all the events were triggered. And he relied on it, exclusively, without analysing the animals which were actually exposed, and demonstrating signs of disease. The 2012 CSIRO report suggested biota should be examined. Dr Batley did not mention this CSIRO recommendation, which remains unfulfilled by CSIRO/DERM.

Dr Batley uses an entirely invalid comparison of CSIRO sampling which took place well after animals were diseased in December 2011 to dismiss data from more time relevant points in September and October. Such a dismissal does not stand up to any level of epidemiological scrutiny.

Dr Batley makes no comment about the detection of 330µg/L of dissolved aluminium found in Graham's creek in CSIRO study, p18 of that report.

Dr Batley accuses me of the use of emotive language. Yet has failed to acknowledge the facts stated are demonstrably correct and do not invoke any emotion. I said animals would be exposed to elevated arsenic. The turtle bloods demonstrate that they were. The sediment studies in the past have shown the elevated arsenic loads in sediments. I stated that arsenic is known to be highly toxic depending on the form it is in. This statement is nothing but the scientific truth.

Dr Batley claims that a detection of elevated arsenic not immediately adjacent the dredges suggests a different source other than dredging. This statement fails to take account of tide, wind and timing of collection which are required to inform the meaning of the sample.

No such data was kept in the monitoring program. It is known from GAWB data that elevations in metals and turbidity coincided with massive elevations in the harbour- completely consistent with the harbour development being the cause, as at the time the bund wall was scouring out badly in big tides and dredging was underway, using dredges which are known to create the greatest plumes.

Dr Batley extends a quote from the report, suggesting further studies on the impacts of metals on animal health were required. I note that neither CSIRO, GPC, DERM nor Vision Environment undertook any such studies.

Dr Batley claims to quote examples of a “lack of detailed interpretation in the Landos Report that leads to the wrong conclusion” in relation to finding elevated aluminium higher at sites not immediately adjacent the dredging. Dr Batley fails to recognise the movement of the plume according to tide and wind and the degree of mixing which can occur at any site. The rise in aluminium from baseline levels measured is striking after the project commenced. Dr Batley offers no plausible explanation for what drove this increase.

Basic ecotoxicology principles were ignored in Gladstone. A whole of effluent toxicity test should have been done, but it was not. Dr Batley does not mention this fundamental tool to understand toxicity. He also does not comment on the striking absence of bioassay oysters throughout the project.

Page 20

Dr Batley suggests CSIRO research aluminium is not bioavailable. However the data from the few barramundi tested by the State Government showed levels inside the gills indicating bioavailable uptake had occurred. This data is not explained by Dr Batley.

Dr Batley’s mention of the typical origin of iron in marine waters are not relevant to apply to Gladstone where 26 million cubic metres of dredging was underway and a massive spill of dredge spoil through a leaking bund wall occurred. The relevant considerations for iron release, relate to the circumstances which actually took place at Gladstone. The EIS documented many areas of high iron in sediments. The Lyngbya toxic algae bloom did not occur after the freshwater influx in January 2011. It only took off in the later half of 2011 when dredging and the bund wall construction drove massive increases in turbidity at a time when there was no substantial rainfall.

Dr Batley makes a series of comments about exposures, stress and immunity which are all contradicted by the pathology evidence in the fish of elevated intensities of particular parasitic infections. The very parasitic infections which are known to be related to pollution based stressors in fish occurred in Gladstone fish and not elsewhere as documented at the reference site, and the absence of any similar event elsewhere on the Qld coast.

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Dr Batley was concerned that I had been selective in dismissing freshwater as a cause. The spatial and temporal epidemiological evidence shows there is no link to a freshwater based stress trigger to the events observed. It also cannot explain any of the observed pathology, or the observed parasitic infestations which were all marine organisms.

I note that a larger flood occurred in January 2013, nearly double the size of the 2010-11 flood. Yet there has been no repeat event in Gladstone. The reasons are evident on closer examination- there is no longer construction of a bund wall going on, it is no longer scouring in big tides due to repairs and engineering changes, and the leaks in the wall have been repaired. The bucket dredges which cause substantial sediment resuspension are all gone, and dredging is no longer in the most contaminated areas. I predicted that no repeat would occur unless the necessary causes were present, and it has not. The necessary cause was an uncontrolled harbour development that caused massive resuspension of contaminated sediments.

Comparing Gladstone to other harbour of the world is an invalid comparison. None of those other harbours contained a World Heritage listed area and its OUV.

2. University of Tasmania, Dr Barbara Nowak Scientific Review.

As noted for Dr Batley above Dr Nowak is not a veterinarian. The same comments apply to the applicability of the skill set of the reviewer, to adequately review a veterinary investigation.

I note her review, as with Dr Batley, was funded as a consultancy for Gladstone Ports Corporation.

Response to Dr Nowak report

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Dr Nowak notes sampling was limited and at times opportunistic. Later Dr Nowak states it is possible that the "limited sampling and analysis, were due to limited resources."

Dr Nowak states: "With few exceptions, only one reference site was sampled."

The budget of the project was extremely limited with a total of \$55,000, 37% of which was raised from public donations with donation tins placed around Gladstone. 7% came from commercial fishers, and the remainder from seafood processors throughout Queensland. Further substantial in-kind contributions were provided by all fishing and crabbing boats, otherwise the project could not have achieved the spatial coverage that it did. There were also substantial in-kind contributions from several scientists who assisted in the sampling and drafting of the report. My thanks to all those who donated and participated to create the only data independent of financially conflicted sources in Gladstone.

The sampling was limited by money. I'd argue the quantity of sampling, travel costs and time spent in the field, in addition to the extensive report represent value for money unable to be matched by CSIRO or University of Tasmania. I personally contributed over \$80,000 in professional time gratis to complete this work. The work was not undertaken for profit. Critically the numbers of animals sampled are epidemiologically robust. The prevalence survey of crabs sampled more than 600

animals, which allows considerable statistical confidence in the results. The spatial coverage of the experimental design was also thorough. The numbers sampled for pathology were also statistically significant, and offered thorough coverage of the study area.

Dr Nowak states ideally the study should be multi-disciplinary.

I agree- and it was. The study involved a marine parasitologist with a PhD, another marine scientist and a marine ecologist. The draft report was also reviewed by a Professor with a water quality background, and a senior fish pathologist.

Dr Nowak states that chemical analysis of gills should have been undertaken.

I agree, and note that no such analysis was undertaken on any epidemiologically significant scale by CSIRO, DERM or Biosecurity Queensland at any stage. DERM were the lead agency for the fish kill investigation. They did not follow their own fish kill investigation manual, nor the National Fish Kill Protocol developed by Fisheries Research Development Corporation (FRDC).

I approached FRDC on multiple occasions, seeking supplemental funding to undertake these key analyses, but was unable to get funding as the project was deemed “too political”.

Subsequently I obtained sufficient funding from other public donors to process a limited number of crab tissues for metal analysis and confirmed the diagnosis of metal-induced shell lesions in Gladstone crabs, with high levels of copper and zinc. This report is to be released soon.

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Dr Nowak states that ecotoxicity testing and laboratory experiments would be useful to provide more information, and enable a more thorough interpretation of results.

I agree, and note that no such testing was undertaken by GPC, DERM or Biosecurity Queensland at any time. Had funding permitted I would have undertaken at least a whole of effluent toxicity study. It is the State Government’s responsibility for undertaking such investigations, not that of a private veterinarian.

Dr Nowak selectively extracts quotations from the report, removing their context. Dr Nowak suggested I had taken scientific literature out of context, in relation to contaminant effects on fish parasite infestations.

Dr Nowak claimed that I suggested all parasites increased with contaminant induced immunosuppression of fishes. However when the relevant paragraph is read in its entirety, it is clear that the discussion highlighted that a specific groups of parasites were identified at higher intensities on Gladstone fishes: all were monoxenous parasites. The literature clearly states that monoxenous parasites are the group of parasites likely to increase in intensity of infestation in contaminant induced immunocompromised fish.

So the Gladstone data points very clearly to an analogous situation to that reported in fishes elsewhere in the world in association with contaminated environments.

The quotation offered by Dr Nowak which indicated that for some parasites certain pollutants could decrease parasite infestations is not relevant to the data being considered in the FFVS report. The Gladstone data showed increased intensities of infestation relative to the control site. Moreover the histological data showed that the parasites were likely to be causing some of the tissue damage observed, due to their increased intensities of infection.

I am unsure why Dr Nowak is discussing depression of parasites by pollutants when this has no relevance to the actual circumstances occurring in Gladstone.

Monogenean parasites were reported to be increased on sharks and previously on barramundi by Biosecurity Queensland. Increased parasitic copepods were documented on queenfish, and coccidian parasites in barramundi and other species of fishes. All have direct lifecycles.

Dr Nowak failed to highlight that the parasite infestations which are documented in the scientific literature to rise with pollutant exposure, were the exact same parasite groups which were detected at elevated intensities of infestation, in association with tissue damage in Gladstone fishes. Importantly the same parasite fauna were detected on the reference site fishes, but intensities of infestation were very low, and they were not in association with tissue damage.

The conclusions of the chapter on parasites are entirely supported by the literature quoted.

Dr Nowak claims I misinterpreted the Patterson et al 2007 reference on *Pfiesteria*. To quote directly from the conclusion of the paper:

“This study suggests that CPE (the *pfiesteria* toxin) is cytotoxic to keratinocytes at high concentrations,..” –parentheses added for meaning.

I said in the report “Patterson et al (2007) identified that the toxins could damage and kill skin cells. The term cytotoxicity refers to damage and death of a cell. A keratinocyte is a skin cell.

The report FFVS does not go further to say this took place in fishes. Skin sensitisation is a different pathological condition to cytotoxicity. I did not discuss sensitisation which Dr Nowak refers to.

Dr Nowak suggests another example of inappropriate use of reference in the report is in relation to Bols et al (2001). Again Dr Nowak, fails to place the paper in the context of the clinical data from Gladstone. Commercial fishers reported profuse mucous secretions from affected fishes. Bols (2001) does demonstrate, as well as the other references offered, that this increased secretion can be a consequence of exposures to metals.

Bols et al (2001) state:

“Excessive mucus secretion is a common response, which has been seen after exposure to heavy metals,....)

So this paper does support the contention that water based contaminants can increase mucous secretion. Moreover it makes the link between pollutant impacts on mucous, and impacts on

immunity. Within the context of the chapter where immunity had already been linked to parasite infestation intensity, the use of Bols (2001) is not out of place.

The observation that increased mucous may provide a greater feed source for mucous eating parasites was provided by the editing marine parasitologist Dr Ben Diggles. Such a proposition is plausible. I acknowledge that this linkage was not made in the Bols 2001 paper.

However, when the paragraph is read in its entirety the numerous references form a coherent supporting explanation for the clinical signs and pathology evidence from Gladstone fishes.

Moreover there is no other potential explanation which can explain the signs. The flood hypothesis put forward by Government is not a plausible explanation. It is incoherent with the temporal and spatial epidemiology of the case. It is incoherent with the observed pathology of the fishes and crabs. It is incoherent with the history of commercial fish harvest.

There is no evidence at all, to support the flood hypothesis.

Dr Nowak does not offer comment on the likelihood of floods playing any role at all.

Dr Nowak claims that the causal hypothesis presented in the report assumes that increased prevalence/intensity of parasitic infections is directly related to immunosuppression.

This assumption by Dr Nowak, does not represent the analysis in the chapter on parasitic infections. The chapter mentions not only immunosuppression but also changes in mucous secretions can affect the vulnerability of skin. And it makes the association based on the actual observed increased parasitic infections, not specifying all parasites in general.

There were insufficient funds to undertake blood based assays to demonstrate immunosuppression. I agree with Dr Nowak this would be ideal for a project. I note that GPC/DERM and Biosecurity Queensland undertook no such testing at any time on fish. And that GPC withheld the Entox turtle blood report for 9 months prior to public release.

Where water quality has clearly deteriorated, as was documented in Gladstone Harbour based on metals testing, turbidity testing, ambient noise levels and toxic algal bloom detection, it is not speculative to suggest that resident aquatic animals may be experiencing elevated stressors.

I note that no scientist has claimed the ecosystem was not under stress.

The Gladstone Scientific Advisory Panel came to the same conclusion as FFVS on this point. All of the pathology, across all of the species of animals sampled needs to be considered, when determining if stressful conditions may have prevailed in the harbour.

The Entox turtle contaminant study indicated that levels of metals in the bloods of turtles may be sufficient to cause toxicity, hence stressful.

Dr Nowak accepts that the pathology demonstrated in mudcrabs is consistent with elevated exposures to copper. Dr Nowak accepts that the prevalence study demonstrated a massive increase in disease rates in Gladstone mudcrabs.

Combine this with the fish and scallop data, and a coherent picture of immunosuppression driven disease emerges.

The exact types of parasites which increased in Gladstone, were those expected to do so, should fish be suffering immunosuppression from contaminant exposure. The academic discussion by Dr Nowak that contaminants could cause reduction in parasites is not relevant, given the elevations on Gladstone fishes reported, and the contaminant distribution was reported.

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Dr Nowak falsely claims that the report claims “all parasites benefit from exposure to contaminants.” The report does not make this claim anywhere. It does claim that the specific types of increased parasite intensities observed were likely due to multiple stressors, including contaminants, impacting on the immune function of the exposed fishes. And such a conclusion is broadly supported by the scientific literature.

In a report, which was published for a broad scientific and non-scientist background, which ran over 100 pages it is both necessary and acceptable to generalise. The fact that a minority of monoxenous parasites may be diminished by contaminants is unimportant in the context of the Gladstone investigation. As very clearly multiple different parasite intensities were substantially increased.

Dr Nowak’s numerous references to this effect, have no relevance to Gladstone, as parasite numbers were high, not low.

Veterinary pathology is a complex discipline requiring substantial training to attain a high level of competence. The veterinary pathologist used for this project is a registered specialist and a Diplomate of the American College of Veterinary Pathologists, she has an undergraduate degree in Veterinary Science, and a PhD in epidemiology.

Dr Nowak is not similarly qualified.

Dr Nowak claimed that lesions observed on pathology could have other causes.

Such a general statement is true. However, Dr Nowak did not go on to list any such causes, nor test any of these other proposed causes, against the evidence which was presented for this case. These steps are essential to ascribe likelihood and to reach a presumptive diagnosis.

Dr Nowak states that the report assumes that all pathology is due to human activities in Gladstone Harbour. This is another incorrect assumption.

The report did not assume this was the case. The report clearly considered the reference site fishes, and presented all of its data also. The report noted that the intensity of the infestations and severity of tissue changes was greater in Gladstone fish.

Dr Nowak suggested “very few” individuals were examined from the reference site. Yet the pathology report details 27 individuals were examined by histopathology and more than 150 mudcrabs were examined by gross pathology from the reference site. Numerous other fishes were examined grossly, but were not sampled due to their normal clinical appearance.

Dr Nowak agreed that one of the major differences between the reference site and Gladstone was the presence of skin lesions in Gladstone. Photographs from interim reports illustrate the elevated parasite loads on affected fishes. The pathology reports also made comments about parasite intensities where they were observed histologically.

Dr Nowak criticises the use of a generalisation in an executive summary of a report which assembles a substantial amount of data from a wide variety of sources including that generated within the project. With respect, it is essential to use generalisations to communicate a complex scientific investigation to a broad, often non-scientific audience. The content of the FFVS report, as Dr Nowak acknowledges does provide the detail, which is specific and accurately represents the data collected, and the observations made. None of the generalisations contradict the substance of the report.

Post script

I would be interested to read the critique of Dr Batley and Dr Nowak of the methods, reporting, discussion and conclusions reached by GPC, DERM and Biosecurity Queensland based on their investigation. There has been no independent review of the conclusions of each of those groups.

In my opinion the standard of those reports and investigation in comparison to the FFVS work is poor.