

Pectenotoxins in New Zealand bivalve molluscan shellfish, 2009-2019: risk assessment

New Zealand Food Safety Technical Paper No: 2020/12

Prepared for New Zealand Food Safety
by Mike Boundy (Cawthron), Tim Harwood (Cawthron), Sarah Finch (AgResearch), Andreas Kiermeier (Statistical Process Improvement Consulting), Cath McLeod (NZFSSRC) & Jeane Nicolas (NZFS)

ISBN No: 978-1-99-001761-2 (online)
ISSN No: 2624-022X (online)

April 2020

Disclaimer

While every effort has been made to ensure the information in this publication is accurate, the Ministry for Primary Industries does not accept any responsibility or liability for error of fact, omission, interpretation or opinion that may be present, nor for the consequences of any decisions based on this information.

This publication is available on the Ministry for Primary Industries website at <http://www.mpi.govt.nz/news-and-resources/publications/>

© Crown Copyright - Ministry for Primary Industries

Scientific Interpretative Summary

This SIS is prepared by New Zealand Food Safety (NZFS) risk assessors to provide context to the following report for MPI risk managers and external readers.

Report No. 3476 Pectenotoxins in New Zealand bivalve molluscan shellfish, 2009-2019: risk assessment

Pectenotoxins (PTXs) and their congeners are macrocyclic polyether-lactone compounds that are produced primarily by the marine dinoflagellate phytoplankton *Dinophysis* spp. During blooms of *Dinophysis* spp. filter feeding shellfish such as bivalve molluscs can accumulate the algae in their digestive glands and take up the lipophilic compounds produced by the algae. In addition to PTXs, *Dinophysis* spp. produce the diarrhetic shellfish poisoning (DSP) toxins, okadaic acid (OA), and dinophysistoxins (DTX1 and 2).

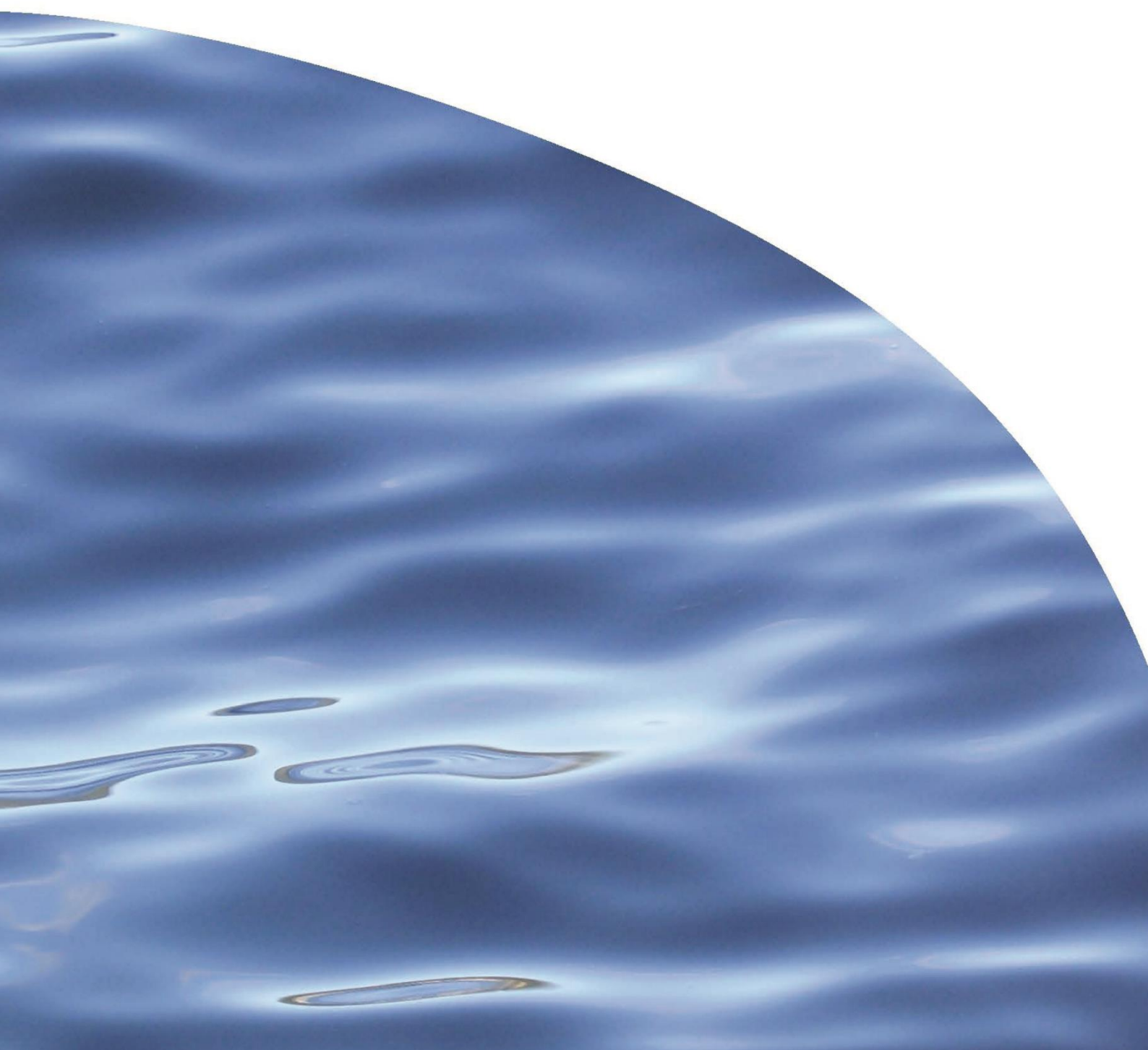
New Zealand Food Safety (NZFS) and the New Zealand shellfish industry have tested for biotoxins from bivalve molluscan shellfish over many years. NZFS contracted the Cawthron Institute to assess the food safety risk presented by PTX in New Zealand shellfish using data collected from 2009-2019. As part of this risk assessment, both PTX and DSP groups were reviewed, as they are currently regulated together in New Zealand.

The main PTX analogue observed in shellfish, PTX2, was detected in 1.3% of New Zealand shellfish samples analysed over the 2009-2019 period, with a maximum concentration of 0.079 mg/kg. However, over this time period there is no evidence that PTX has resulted in any human illness. DSP was detected in 4.2% of New Zealand shellfish samples, with a maximum concentration of 1.4 mg/kg, and 0.4% of samples over the current maximum permissible level of 0.16 mg OA eq/kg. Pre-dating the risk management programme of routine monitoring, a few historic cases of suspected DSP intoxication have been reported from non-commercial shellfish.

The risk assessment concludes that the food safety risk presented by PTX in New Zealand shellfish is low, and that the PTX-group should be removed from regulation in New Zealand. However, the risk assessment recommended that the current maximum permissible level of 0.16 mg OA eq/kg for DSP is retained. This evidence base will be used to reassess the current maximum permissible levels for DSP in our current monitoring programmes for shellfish.

REPORT NO. 3476

**PECTENOTOXINS AND OKADAIC ACID GROUP
TOXINS IN NEW ZEALAND BIVALVE MOLLUSCAN
SHELLFISH, 2009-2019: RISK ASSESSMENT**



PECTENOTOXINS AND OKADAIC ACID GROUP TOXINS IN NEW ZEALAND BIVALVE MOLLUSCAN SHELLFISH, 2009-2019: RISK ASSESSMENT

MIKE BOUNDY¹, TIM HARWOOD¹, SARAH FINCH², ANDREAS KIERMEIER³, CATH MCLEOD⁴

¹ CAWTHRON INSTITUTE

² AGRESEARCH

³ STATISTICAL PROCESS IMPROVEMENT CONSULTING AND TRAINING PTY LTD (AUSTRALIA)

⁴ NEW ZEALAND FOOD SAFETY SCIENCE & RESEARCH CENTRE (NZFSSRC)

Prepared for the Ministry for Primary Industries

Ministry for Primary Industries
Manatū Ahu Matua



CAWTHRON INSTITUTE

98 Halifax Street East, Nelson 7010 | Private Bag 2, Nelson 7042 | New Zealand

Ph. +64 3 548 2319 | Fax. +64 3 546 9464

www.cawthron.org.nz

REVIEWED BY:
Tom Wheeler

A handwritten signature in black ink, appearing to read 'Tom Wheeler'.

APPROVED FOR RELEASE BY:
Nico van Loon

A handwritten signature in black ink, appearing to read 'Nico van Loon'.

ISSUE DATE: 18 Feb 2020

RECOMMENDED CITATION: Boundy MJ, Harwood DT, Kiermeier A, McLeod C 2020. Pectenotoxins and Okadaic acid group toxins in New Zealand Bivalve Molluscan Shellfish, 2009-2019: Risk Assessment. Prepared for the Ministry for Primary Industries. Cawthron Report No. 3476. 89 p. plus appendices.

© **COPYRIGHT:** This publication must not be reproduced or distributed, electronically or otherwise, in whole or in part without the written permission of the Copyright Holder, which is the party that commissioned the report.

EXECUTIVE SUMMARY

New Zealand currently regulates the pectenotoxin (PTX) group as part of the diarrhetic shellfish poisoning (DSP) toxin group, which is inconsistent with the recommendations from the international guidance Codex Standard 292-2008. The origin of why or how the PTX-group analogues were included in the DSP regulation is unclear, although it is a historical decision that is now well understood to be inappropriate. Both PTX and DSP groups were reviewed as part of this risk assessment as they are currently regulated together in New Zealand.

Key findings for the pectenotoxins group:

- Including the PTX-group as part of the DSP-group is fundamentally flawed as they do not share the same toxicological mode of action.
- While PTX have been observed to be toxic to mice with i.p. administration, no toxicity is observed by oral administration when using authenticated material.
- The main PTX analogue observed in shellfish, PTX2, was detected in 1.3% of New Zealand shellfish samples analysed over the 2009-2019 period. A maximum concentration of 0.079 mg/kg was observed, which is lower than the European Food Safety Authority (EFSA) proposed limit of 0.12 mg/kg.
- The risk assessment of PTX in New Zealand shellfish during bloom events showed no instances of simulated shellfish meals exceeding the conservative acute reference dose recommended by the EFSA.
- There is no evidence that PTX has resulted in human illness worldwide, ever.

Key findings for the okadaic acid group (DSP):

- DSP was detected in 4.2% of New Zealand shellfish samples analysed over the 2009-2019 period with a maximum concentration of 1.4 mg/kg. There were 0.4% of samples with DSP over the current maximum permissible level of 0.16 mg OA eq/kg.
- There were 0.05% of samples where the PTX2 concentration caused the DSP result to exceed the maximum permissible level. These samples had DSP results at or near the maximum permissible level.
- A low risk of exposure above the acute reference dose was observed for the DSP group during bloom events. This risk is significantly reduced by the current regulatory limit of 0.16 mg OA eq/kg, to 0.9% for adult males and 1.4% for adult females.
- In New Zealand, only a few historic cases of suspected DSP intoxication have been reported, and these were from non-commercial shellfish pre-dating the risk management programme with routine monitoring using liquid chromatography tandem mass spectrometry.

Based on this risk assessment it is recommended that the PTX-group should be removed from the DSP-group in New Zealand and deregulated. The current DSP maximum permissible level of 0.16 mg OA eq/kg should be retained, as it is fit for purpose.

TABLE OF CONTENTS

1. INTRODUCTION	1
2. HAZARD IDENTIFICATION	3
2.1. Pectenotoxin group.....	3
2.1.1. Background.....	3
2.1.2. Production and Accumulation.....	3
2.1.3. Chemistry.....	3
2.1.4. Metabolism in shellfish	6
2.1.5. Methods of Analysis	7
2.2. Okadaic acid group.....	7
2.2.1. Background.....	7
2.2.2. Production and Accumulation.....	8
2.2.3. Chemistry.....	8
2.2.4. Metabolism in shellfish	9
2.2.5. Methods of Analysis	10
3. HAZARD CHARACTERISATION	11
3.1. Pectenotoxin group.....	11
3.1.1. Absorption, distribution, metabolism and excretion	11
3.1.2. Toxicity in animals.....	11
3.1.3. Toxicity Equivalency Factors.....	14
3.1.4. Mechanism of Action	14
3.1.5. Observations in humans.....	15
3.1.6. Evaluation of Hazard Characterisation.....	15
3.2. Okadaic acid group.....	16
3.2.1. Absorption, distribution, metabolism and excretion	16
3.2.2. Toxicity in animals.....	16
3.2.3. Toxicity Equivalency Factors.....	18
3.2.4. Mechanism of Action	18
3.2.5. Observations in humans.....	19
3.2.6. Evaluation of Hazard Characterisation.....	19
4. EXPOSURE ASSESSMENT	21
4.1. New Zealand 2009-2019	21
4.1.1. Method of Analysis.....	21
4.1.2. Raw Data	21
4.1.3. Data Clean-up	22
4.1.4. Data Exclusions	22
4.1.5. Location Grouping.....	22
4.1.6. Bloom Identification.....	23
4.1.7. Spatial Distribution of PTX in New Zealand.....	26
4.1.8. Temporal Distribution of PTX in New Zealand	33
4.1.9. Species distribution of PTX in New Zealand	37
4.1.10. Impact of PTX contribution to DSP regulation	38
4.1.11. Comparison of PTX2, PTX2SAs and DSP concentrations in shellfish	40
4.1.12. Pectenotoxin profiles.....	43
4.1.13. Comparison of PTX2 concentrations in New Zealand with Europe	48
4.1.14. Comparison of DSP concentrations in New Zealand with Europe.....	49
4.2. New Zealand Bivalve Consumption.....	50

5. RISK CHARACTERISATION	54
5.1. Pectenotoxin group.....	54
5.1.1. <i>Deterministic estimate of dietary exposure to PTX2</i>	54
5.1.2. <i>Probabilistic estimate of dietary exposure to PTX2</i>	55
5.1.2.1. <i>Methodology</i>	55
5.1.2.2. <i>Simulation model development</i>	55
5.1.2.3. <i>Consumption amount of shellfish</i>	56
5.1.2.4. <i>Relationship between DSP and PTX2</i>	56
5.1.2.5. <i>Distributions of PTX2</i>	57
5.1.2.6. <i>Estimating Exposure</i>	59
5.1.2.7. <i>Risk Characterisation</i>	60
5.2. Okadaic acid group.....	61
5.2.1. <i>Deterministic estimate of dietary exposure to DSP</i>	61
5.2.2. <i>Probabilistic estimate of dietary exposure to DSP</i>	62
5.2.2.1. <i>Methodology</i>	62
5.2.2.2. <i>Simulation model development</i>	62
5.2.2.3. <i>Consumption amount of shellfish</i>	63
5.2.2.4. <i>Distributions of DSP</i>	63
5.2.2.5. <i>Estimating Exposure</i>	64
5.2.2.6. <i>Risk Characterisation</i>	65
6. UNCERTAINTY AND GAPS	66
6.1. Pectenotoxin group.....	66
6.1.1. <i>Uncertainty</i>	66
6.1.1.1. <i>Scenario Uncertainty</i>	66
6.1.1.2. <i>Model Uncertainty</i>	66
6.1.1.3. <i>Parameter Uncertainty</i>	67
6.1.2. <i>Summary of Uncertainties</i>	67
6.1.3. <i>Data gaps</i>	68
6.2. Okadaic acid group.....	69
6.2.1. <i>Uncertainty</i>	69
6.2.1.1. <i>Scenario Uncertainty</i>	69
6.2.1.2. <i>Model Uncertainty</i>	70
6.2.1.3. <i>Parameter Uncertainty</i>	70
6.2.2. <i>Summary of Uncertainties</i>	71
6.2.3. <i>Data gaps</i>	71
7. RISK MANAGEMENT	72
7.1. Regulatory considerations.....	72
7.2. Risk Management Options for the Pectenotoxin group in New Zealand.....	76
7.3. Risk Management Options for the Okadaic acid group in New Zealand.....	77
8. CONCLUSION	79
8.1. Recommendations.....	81
9. REFERENCES	82

LIST OF FIGURES

Figure 1.	The structures of PTX-group analogues. A, B and C are different backbones, and the C-7 stereocenter can be in either the R or S configuration (Suzuki, 2014).	5
Figure 2.	Acid-catalysed inter-conversion of the pectenotoxins (Suzuki, 2014)	5
Figure 3.	The metabolism of pectenotoxin-2 in shellfish, red highlights changes in the structure during the metabolism step	6
Figure 4.	The structures of OA-group toxins	9
Figure 5.	Shellfish zones and subzones used for grouping shellfish sampling sites for bloom event identification.	25
Figure 6.	Number of samples analysed for biotoxins at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds. Circle size denotes number of samples tested	27
Figure 7.	Maximum concentration of pectenotoxin 2 at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.	28
Figure 8.	Maximum concentration of pectenotoxin 2 seco acids at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.....	29
Figure 9.	Maximum concentration of diarrhetic shellfish poisoning toxins at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.....	30
Figure 10.	Number of samples analysed for phytoplankton at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.	31
Figure 11.	Maximum concentration of <i>Dinophysis</i> spp. cells at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.	32
Figure 12.	Concentrations of PTX2, PTX2SAs, DSP and <i>Dinophysis</i> spp. throughout New Zealand over the 2009-2019 period.....	33
Figure 13.	Number of samples containing reportable levels for PTX2, PTX2SAs, and DSP by month over the 2009-2019 period.....	36
Figure 14.	Number of samples analysed by month over the 2009-2019 period	36
Figure 15.	Comparison of PTX2 contribution to DSP regulation in New Zealand over the 2009-2019 period on a logarithmic scale	38
Figure 16.	Comparison of DSP with PTX2+DSP in New Zealand over the 2009-2019 period on a logarithmic scale	39
Figure 17.	Comparison of PTX2 with Total DSP in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.	40
Figure 18.	Comparison of PTX2SAs with Total DSP in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.	41
Figure 19.	Comparison of PTX2 with PTX2SAs in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.	42
Figure 20.	PTX profile for Greenshell mussels (left), and Pacific oyster (right) based on the 97.5 th percentile concentrations of the PTX analogues excluding PTX2SAs in the bloom event C 201507-12.....	44
Figure 21.	PTX profile for Greenshell mussels (left), Pacific oyster (middle), and Scallops (right) based on the 97.5 th percentile concentrations of the PTX analogues including PTX2SAs in the bloom event C 201507-12	44
Figure 22.	Ratio of PTX-group analogues compared to PTX2 in different shellfish species during bloom event C 201507-12. Left: PTX2SAs, Right: PTX1 and PTX11.	45
Figure 23.	Ratio of PTX-group analogues compared to PTX2 in different shellfish species during bloom event A boi 201506-12. Left: PTX2SAs, Right: PTX1 and PTX11.....	46

Figure 24.	Comparison of chromatogram of PTX11 acquired with 5 MRM transitions in a) authentic PTX11 reference material, and b) concentrated Pacific oyster extract from site A015	47
Figure 25.	Scatter plot of the log ₁₀ PTX2 concentration versus the log ₁₀ DSP concentrations, showing only samples where both were detected; the blue line is the best fit regression line.....	57

LIST OF TABLES

Table 1.	Acute toxicities of PTX derivatives in mice by intraperitoneal injection	12
Table 2.	Acute toxicity of PTX derivatives in mice by oral administration (gavage)	13
Table 3.	Acute toxicities of OA-group toxins to mice by intraperitoneal injection	17
Table 4.	Results of toxicity testing of OA-group toxins to mice orally	18
Table 5.	List of shellfish sampling zones, subzones and descriptions of the region used for bloom event identification.	24
Table 6.	Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5 th percentile concentrations (mg/kg) of PTX2 in different years in New Zealand over the 2009-2019 period.....	34
Table 7.	Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5 th percentile concentrations (mg/kg) of PTX2 in different months of the year in New Zealand over the 2009-2019 period.....	35
Table 8.	Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5 th percentile concentrations (mg/kg) of PTX2 in different types of shellfish analysed in New Zealand over the 2009-2019 period	37
Table 9.	Concentrations of PTX2 measured by LC-MS/MS in shellfish samples grouped by DSP results in New Zealand over the 2009-2019 period (mg/kg)	48
Table 10.	Concentrations of PTX2 measured by LC-MS/MS in samples that were also analysed by the DSP mouse bioassay (EFSA, 2009a).....	49
Table 11.	Concentrations of DSP measured by LC-MS/MS in shellfish samples in New Zealand over the 2009-2019 period (mg/kg).....	50
Table 12.	Concentrations of DSP measured by LC-MS/MS in samples comparatively tested with the DSP mouse bioassay (EFSA, 2008).....	50
Table 13.	Summary of average and 97.5 th percentile portion sizes from the 2008 Adult Nutrition Survey (Parnell <i>et al.</i> , 2011)	52
Table 14.	Summary of marine food consumption by New Zealand children (5-14 years) and adults (15+ years) (Cressey <i>et al.</i> , 2019).....	52
Table 15.	Deterministic intake of PTX2 based on samples at or below the regulatory limit for DSP (excluding the PTX-group).....	54
Table 16.	Deterministic intake of PTX2 based on all samples, including those above the regulatory limit for DSP	55
Table 17.	Concentration of PTX2 simulated in shellfish meals using the two different model approaches (mg PTX2/kg).....	59
Table 18.	Simulated dietary exposure to PTX2 using the two different model approaches for a standard 60 kg adult (µg PTX2/kg bw).....	60
Table 19.	Deterministic intake of DSP based on samples at or below the regulatory limit for DSP (excluding the PTX-group)	61
Table 20.	Deterministic intake of DSP based on all samples, including those above the regulatory limit for DSP.....	62
Table 21.	Concentration of DSP simulated in shellfish meals using the two different model approaches (mg OA eq/kg).....	64
Table 22.	Simulated dietary exposure to DSP using the two different model approaches for a standard 60 kg adult (µg OA eq/kg bw).....	64
Table 23.	Summary of qualitative evaluation of the impact of uncertainties on the risk assessment of the dietary exposure of the PTX-group.....	68
Table 24.	Summary of qualitative evaluation of the impact of uncertainties on the risk assessment of the dietary exposure of OA-group toxins.....	71

Table 25.	Regulatory limits of DSPs and whether the DSP regulation includes the PTX-group in different countries and in the Codex standard	76
-----------	--	----

LIST OF APPENDICES

Appendix A. Bloom Identification	90
Appendix B. Bloom Summary Tables	112
Appendix C. Site Summary	147
Appendix D. Species Summary	162
Appendix E. Annual and Seasonal Summaries	173
Appendix F. Pectenotoxin Profiles	181
Appendix G. Exposure Assessment and Risk Characterisation	204

GLOSSARY

7-epi-PTX2SA	Abbreviation for the 7S isomer of pectenotoxin 2 seco acid
ARfD	The acute reference dose, an estimate of a substance in food or drinking water, expressed on a body weight basis, that can be ingested over a short period of time, usually during a meal or a day, without appreciable health risk to the consumer on the basis of all known facts at the time of evaluation
Dinoflagellate	Single-celled aquatic organism bearing two dissimilar flagella, a lash like appendage that protrudes from the cell body for motion and/or sensation, most are marine plankton
<i>Dinophysis</i>	A genus of dinoflagellates common in tropical, temperate, coastal and oceanic waters
Dinophysistoxin	Analogues of the okadaic acid group toxins produced by <i>Dinophysis</i> spp. which causes diarrhetic shellfish poisoning
DSP	Abbreviation for diarrhetic shellfish poisoning, a symptom of intoxication caused by the consumption of shellfish contaminated with okadaic acid group toxins
DTX	Abbreviation for dinophysistoxin
EFSA	European Food Safety Authority
FAO	Food and Agriculture Organization of the United Nations
FSANZ	Food Standards Australia New Zealand (FSANZ) is a statutory authority in the Australian Government Health portfolio
GI	Gastrointestinal tract, is an organ system with animals which takes in food, digests it to extract and absorb energy and nutrients, and expels the remaining waste as faeces.
HBGV	Health Based Guidance Value
i.p.	Intraperitoneal injection, i.p. administration is the injection of a substance into the peritoneum (body cavity)
IOC	Intergovernmental Oceanographic Commission of the United Nations Educational, Scientific and Cultural Organization (UNESCO)
LOAEL	Lowest-observed-adverse-effect level, the lowest concentration or amount of a substance found by experiment or observation that causes an adverse alteration of morphology, function, capacity, growth, development or lifespan of a target organism distinguished from normal organisms of the same species under defined conditions of exposure.

LoD	Limit of detection, the lowest quantity of a substance that can be distinguished from the absence of that substance with a stated confidence level.
LoQ	Lower limit of Quantitation, the lowest quantity of a substance that can be accurately quantified with a stated confidence level
LC-MS/MS	Liquid chromatography coupled to tandem mass spectrometry; an instrumental technique suitable for the analysis of chemical contaminants. Requires certified reference standards and robust relative toxicities for each compound of interest to be determined in order to be able to estimate sample toxicity
MBA	Mouse bioassay, a functional assay that detects biologically active toxin
<i>Mesodinium rubrum</i>	A species of ciliates, the most commonly attributed food source to <i>Dinophysis</i> through Myzocytosis
MPI	The Ministry for Primary Industries, department in charge of overseeing, managing and regulating the farming, fishing, food, animal welfare, biosecurity and forestry sectors of New Zealand's primary industries.
MRM	Multiple reaction monitoring, a highly sensitive and specific mode of operation for targeted acquisition and quantitation of target analytes using tandem quadrupole mass spectrometry
NOAEL	No-observed-adverse-effect level, denotes the level of exposure of an organism, found by experiment or observation, at which there is no biologically or statistically significant increase in the frequency or severity of any adverse effects of the tested protocol.
OA	Abbreviation for okadaic acid
OA-group	The family of variants of okadaic acid analogues (including dinophysistoxins and esters) which have a common structural backbone and similar chemical properties
Okadaic acid	A polyketide, polyether derivative of a C38 fatty acid toxin produced by <i>Dinophysis</i> and <i>Prorocentrum</i> spp. which causes diarrhetic shellfish poisoning
OMAR	Overseas Market Access Requirements, requirements negotiated by MPI with overseas officials that needs to be met prior to export to that market
PCTL	Percentile, a measure used in statistics indicating the value below which a given percentage of observations in a group of observations falls
Pectenotoxin	A compound produced by the dinoflagellate <i>Dinophysis</i> spp. that is toxic to mice by intraperitoneal injection
Phytoplankton	Microscopic marine algae
<i>Prorocentrum</i>	A genus of benthic dinoflagellates

PTX	Abbreviation for pectenotoxin
PTX-group	The family of variants of pectenotoxin analogues which have a common structural backbone and similar chemical properties
PTX2SA	Abbreviation for the hydrolysed seco acid of pectenotoxin 2
PTX2SAs	Abbreviation for the sum combination of PTX2SA and 7-epi-PTX2SA
Regulation	A rule or direction made and maintained by an authority. The Ministry for Primary Industries regulates marine toxins in shellfish based on the Regulated Control Scheme – Bivalve Molluscan Shellfish for Human Consumption to ensure the food is safe to consume.
sd	Standard deviation, is a measure of the amount of variation or dispersion of a set of values
spp.	Abbreviation for species (plural)
TDI	Tolerable Daily Intake, refers to the daily amount of an unintentional contaminant chemical that has been assessed safe for human beings on a long-term basis (usually whole lifetime).
TEF	Toxicity Equivalency Factors, the toxicity ratio of a compound from a chemical group that shares the same mode of action of a reference compound in the same group. The toxicity of the congener is expressed as a fraction of the toxicity of the reference compound in terms of potency, which is a pharmacological parameter that defines the amount of compound required for a certain effect
WHO	World Health Organisation
WSG-84	World Geodetic System, a standard used in cartography and satellite navigation including GPS, WSG-84 standard was established in 1984

1. INTRODUCTION

New Zealand currently regulates pectenotoxin (PTX) as part of the diarrhetic shellfish poisoning (DSP) toxin group. The New Zealand regulation for shellfish toxins is enforced by the Ministry for Primary Industries under the Animal Products Act 1999 to ensure that the food is safe to consume. The regulations are defined in the Animal Products Notice, Regulated Control Scheme – Bivalve Molluscan Shellfish for Human Consumption as issued by the Director-General (Ministry for Primary Industries, 2018). Samples which have testing results above the maximum permissible level, result in the closure of the shellfish harvesting area until levels of contaminants have returned to safe concentrations. Limited information has been reported on the presence of PTX in bivalve molluscan shellfish and their food safety risk in New Zealand. There is no evidence that PTX has caused human illness, that PTX and DSP can cause food poisoning in similar ways or that they interact with each other to cause more severe illness.

The origin of why or how the PTX-group analogues were included into the DSP regulation is unclear. Additionally, New Zealand was an early adopter of chemical analysis methods, which required these compound specific regulations. It is not known whether PTX was first included in the New Zealand DSP regulation and other countries followed, or if New Zealand included PTX to follow a specific overseas market access requirement (OMAR). Despite the uncertainty of why or how the PTX-group was incorporated into the DSP regulation, it is likely that this came about due to their co-occurrence as they are produced by the same dinoflagellate *Dinophysis* spp. rather than due to toxicological considerations. The mouse bioassay that was used historically for monitoring the DSP group was not specific to okadaic acid (OA) and its analogues, and resulted in positive detections due to the presence of any of a wide range of other compounds classes such as gymnodimine, spirolides, pectenotoxins, yessotoxins (YTX) and brevetoxins. In the light of further research it is now apparent that not all of these compound classes pose a risk to human health, and many were found to be demonstrably safe (e.g. gymnodimine). When analysis of toxins in bivalve molluscs was transitioned away from animal assays in favour of analytical chemistry methods, many compounds which were observed to result in mouse deaths such as YTX, OA and PTX were regulated by the Ministry for Primary Industries, New Zealand Food Safety (known at the time as New Zealand Food Safety Authority). More recently, regulation of YTX was reviewed by the Ministry for Primary Industries and while it has potent toxicity to mice by intraperitoneal injection, it was not observed to be toxic to mice orally and was not linked to any cases of human illness. YTX was deregulated in New Zealand in 2018 and now has relaxed regulation in many export markets.

During the 2018 revision of the Animal Products Notice Regulatory Control Scheme for Bivalve Molluscan Shellfish for Human Consumption (Ministry for Primary Industries, 2018), the inclusion of the PTX-group analogues within the DSP toxin

group regulation was raised as an issue that needed to be reviewed as PTX and DSP do not share the same mode of action and it is not scientifically supported that they should be regulated together. The DSP limit remained unchanged in the new 2018 notice with PTX still included in the regulation. The Ministry for Primary Industries (MPI) will determine whether it is appropriate to continue to regulate PTX in bivalve molluscan shellfish, and if so, what the best approach for regulation would be. Food Standards Australia New Zealand (FSANZ), a statutory authority for the Australian government, does not include PTX in the DSP regulation and has set a maximum level of 0.2 mg OA eq/kg rather than 0.16 mg OA eq/kg, which is the regulatory limit in New Zealand (FSANZ, 2017). A few other jurisdictions regulate for PTX such as Canada, Chile and the European Union. However, the European Food Safety Authority (EFSA) has issued an opinion that PTX should not be regulated with the DSP group (EFSA, 2009a).

A range of PTX analogues have been reported in bivalve molluscan shellfish. MPI and the New Zealand shellfish industry have tested for biotoxins from bivalve molluscan shellfish over many years and collated a large data set. The data from 2009-2019 has been used to fill knowledge gaps on the presence of PTX to provide an up-to-date exposure assessment for establishing the food safety risk presented by PTX in New Zealand shellfish. The concentrations of several PTX analogues have been reviewed along with their co-existence with the DSP toxins. Routinely the PTX analogue PTX2, as well as non-regulated metabolites PTX2SA and 7-epi-PTX2SA, collectively reported as pectenotoxin 2 seco acids (PTX2SAs), are monitored. Initially due to instrument limitations PTX1, PTX11 and PTX6 were not routinely monitored (McNabb *et al.*, 2005). With advancements in instrumentation, PTX1, PTX11, and PTX6 can now be acquired simultaneously by the liquid chromatograph-tandem mass spectrometry method used for regulatory monitoring in New Zealand without impacting performance. However, while these congeners have been added to the acquisition method they are not included in the routine processing and quantitation due to the additional time and cost required. For selected bloom events, including three bloom events with the highest detected PTX2 levels, the concentrations of PTX1, PTX11 and PTX6 were obtained by manually reprocessing the LCMS data acquired in order to retrospectively determine the PTX profiles within New Zealand shellfish.

Due to the interwoven nature of the regulation of PTX with the DSP group in New Zealand, a review of literature surrounding both PTX and OA groups was performed to identify and characterise the hazards they each may pose. This information was combined with the exposure assessment information in order to assess the risk of PTX and OA groups within New Zealand shellfish in order to provide robust scientific guidance on whether it is appropriate for the PTX-group to continue to be regulated in New Zealand, as well as review the suitability of the regulatory limits and the impacts of any regulatory changes.

2. HAZARD IDENTIFICATION

2.1. Pectenotoxin group

2.1.1. Background

There is no evidence that PTX has ever caused any harm to humans (FAO/IOC/WHO, 2004; Munday, 2017). The presence of PTX-group analogues in shellfish was discovered due to their acute toxicity in mice after intraperitoneal injections of lipophilic extracts. Animal studies indicate that they are much less potent via the oral route and that their effects do not induce diarrhoea (Miles *et al.*, 2004a). PTX-group analogues are exclusively produced from *Dinophysis* spp. and are co-produced with OA-group toxins. Analytical methods must reliably distinguish these compounds, and as they do not share the same mechanism of action they should not be regulated together (FAO/IOC/WHO, 2004).

2.1.2. Production and Accumulation

PTX congeners are macrocyclic polyether-lactone compounds that are produced primarily by the marine dinoflagellate phytoplankton *Dinophysis* spp. These dinoflagellates feed primarily on their prey ciliate *Mesodinium rubrum* (previously known as *Myrionecta rubra*) which in turn feeds on algal species such as *Teleaulax amphioxeia* (previously known as *Rhodomonas amphioxeia*) (Nishitani *et al.*, 2010). During blooms of *Dinophysis* spp. filter feeding shellfish such as bivalve molluscs can accumulate the algae in their digestive glands and take up the lipophilic compounds produced by the algae.

In addition to pectenotoxins *Dinophysis* spp. primarily produce the DSP toxins, which include okadaic acid (OA) and the dinophysistoxins (DTX1 and 2). The co-occurrence of PTX with these DSP toxins has been the driving force for investigation of the PTX congeners and appears to be the basis for combining PTX-group compounds and OA-group toxins in the food safety regulations.

Due to the complex nature of the *Dinophysis* spp. endosymbiosis and predator-prey relationship with other organisms in the environment they can be difficult to culture in the laboratory, making the study of the toxins they produce challenging.

2.1.3. Chemistry

The chemical structures of PTX-group analogues are shown in Figure 1. Pectenotoxins have similar molecular masses to OA/DTX analogues and are cyclic ethers. Most of the PTXs are a macrocyclic lactone (macrolide), while others exist as a ring-opened seco acid (Suzuki, 2014). PTX-group analogues are heat stable although are easily destroyed under strongly basic conditions such as those used to hydrolyse acyl esters for the analysis of DSP toxins (Vale and Sampayo, 2002).

PTX1 and PTX2 were originally isolated from Japanese scallops, *Mizuhopecten yessoensis* (previously known as *Patinopecten yessoensis*), and their structures were elucidated by single crystal X-ray diffraction techniques, NMR spectroscopy and mass spectrometry together with ultraviolet and infrared spectroscopy (Yasumoto *et al.*, 1984; Yasumoto *et al.*, 1985). More than 20 PTX analogues have now been discovered although the structures of PTX5 and PTX10 have not yet been elucidated. Treatment of 7R-PTX analogues under acidic conditions leads to an equilibrium mixture of spiroketal stereoisomers, 7R-, 7S- and 6-membered-B-ring-isomers as shown in Figure 2 (Sasaki *et al.*, 1998; Suzuki *et al.*, 2003). PTX4 and PTX7 are the spiroketal isomers of PTX1 and PTX6 respectively (Sasaki *et al.*, 1998), and equilibration between PTX6 and PTX7 and between PTX1 and PTX4 result in the formation of two additional isomeric products, PTX8 and PTX9. It has been suggested that PTX4 and PTX7 are naturally occurring compounds rather than artefacts of the extraction process, whereas PTX8 and PTX9 are artefacts obtained by acidic inter-conversions (Sasaki *et al.*, 1998). 7S and 6-membered-B-ring-isomers of PTX2 were named as PTX2b and PTX2c (Suzuki, 2014).

PTX2SA and its epimer 7-epi-PTX2SA, analogues of PTX2 in which the lactone ring had been hydrolysed, were identified in *Dinophysis acuta* from Ireland and Greenshell mussels, *Perna canaliculus*, from New Zealand (Daiguji *et al.*, 1998).

PTX11, an isomer of PTX1, along with spiroketal isomers PTX11b and PTX11c were detected by LC-MS/MS analysis of Greenshell mussels, *Perna canaliculus*, and *Dinophysis acuta* from New Zealand (MacKenzie *et al.*, 2002; Suzuki *et al.*, 2003; Suzuki *et al.*, 2006). PTX13 and PTX14 were isolated from *Dinophysis acuta* (Miles *et al.*, 2006a). PTX12, a pair of equilibrating 36-epimers of 38,37-dehydro-PTX2 was isolated from a *Dinophysis acuta* bloom in Norway (Miles *et al.*, 2004b).

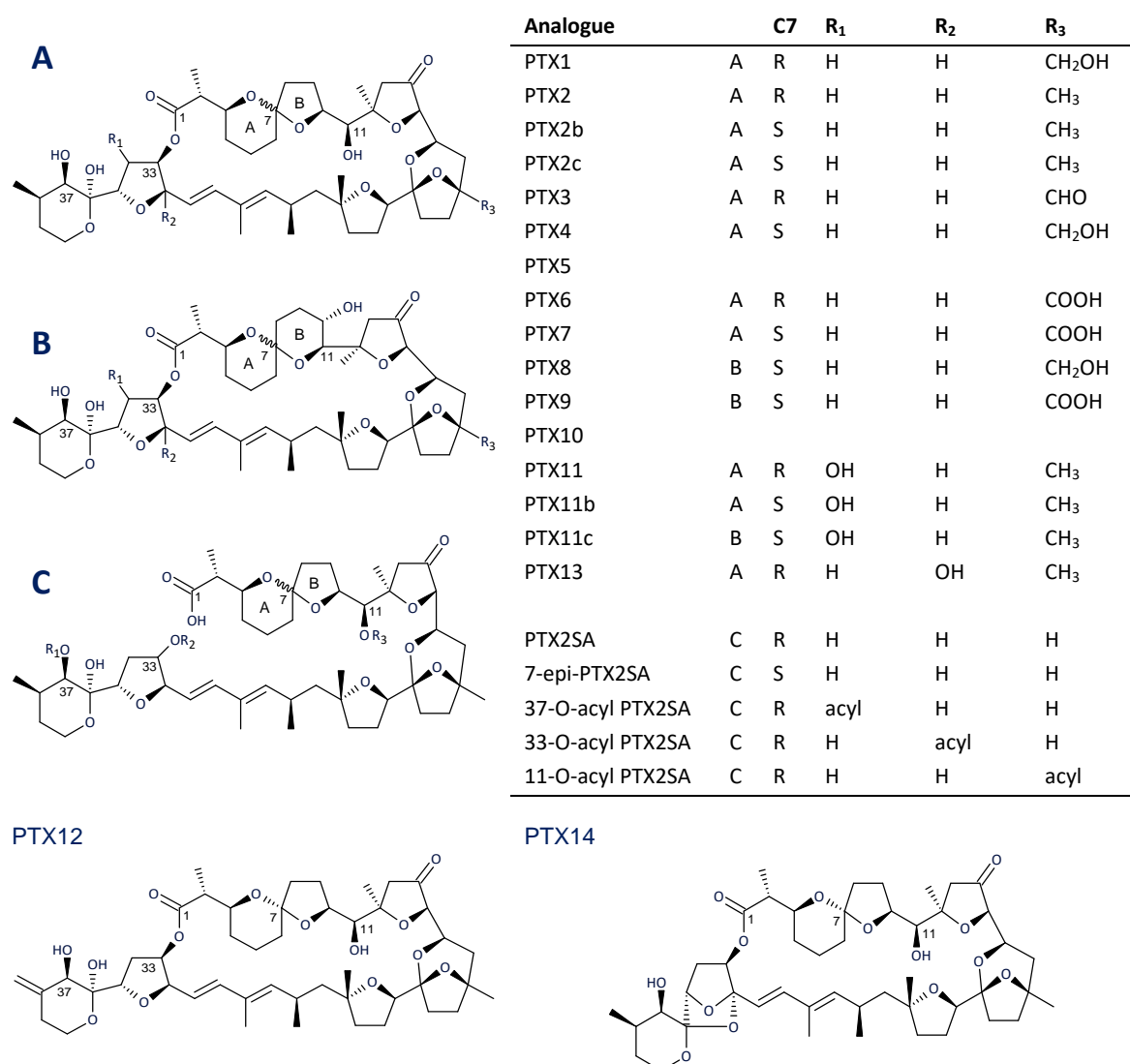


Figure 1. The structures of PTX-group analogues. A, B and C are different backbones, and the C-7 stereocenter can be in either the R or S configuration (Suzuki, 2014).

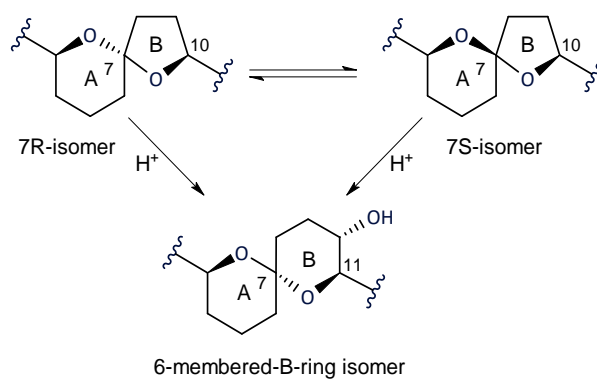


Figure 2. Acid-catalysed inter-conversion of the pectenotoxins (Suzuki, 2014)

2.1.4. Metabolism in shellfish

PTX2 is the major pectenotoxin congener produced by dinoflagellates and is typically the only congener monitored for regulatory purposes. Bivalves accumulate PTX2 by feeding on the algae and it may then be metabolised in the shellfish by two different processes as shown in Figure 3. In the Japanese scallop, *M. yessoensis*, PTX2 undergoes a stepwise oxidation of the methyl group attached to C-18 to an alcohol (PTX1), aldehyde (PTX3), or a carboxylic acid (PTX6) (Yasumoto *et al.*, 1989; Suzuki *et al.*, 1998). This bio-transformation of PTX2 in Japanese scallops was confirmed by the observation of PTX6 in scallops, which was found to be significantly higher than PTX2 despite *Dinophysis fortii* collected in the same location from the same event containing only PTX2 (Suzuki *et al.*, 1998).

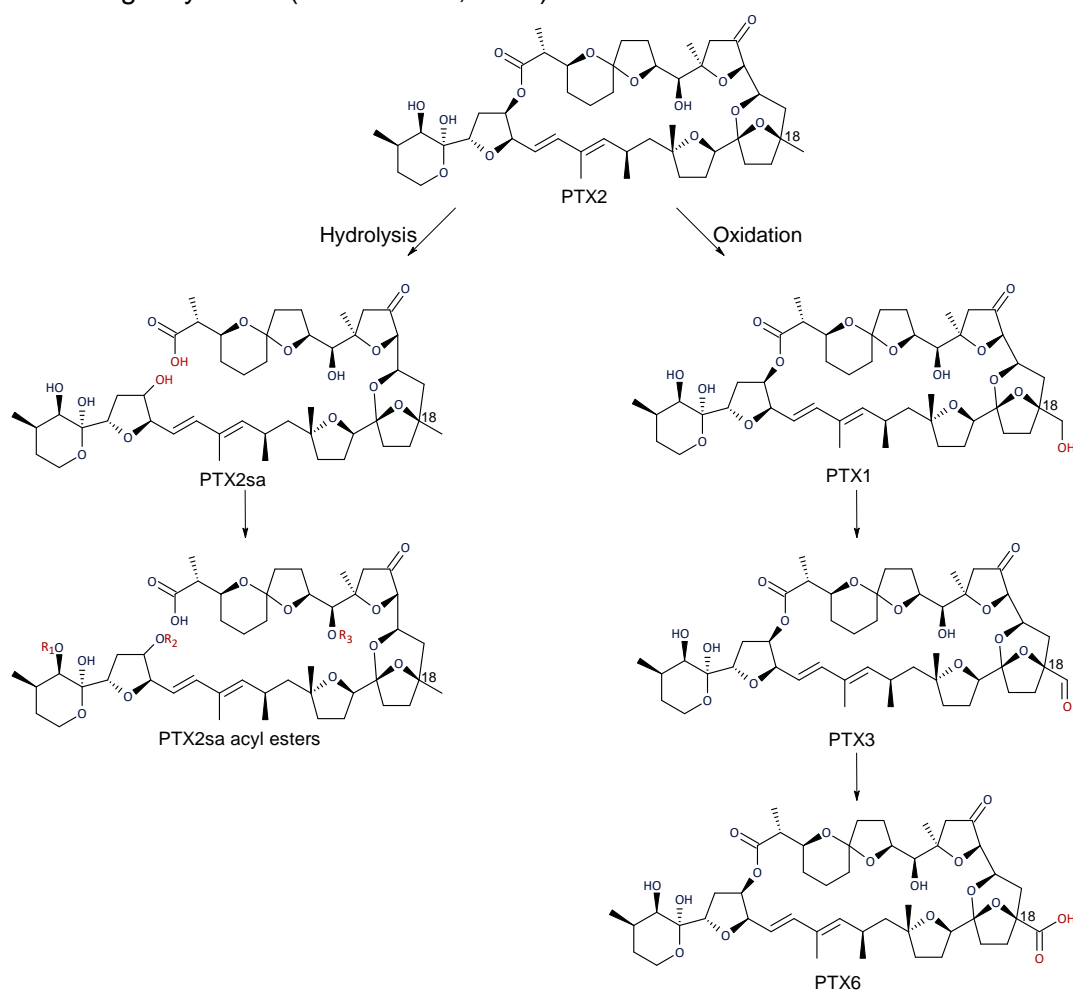


Figure 3. The metabolism of pectenotoxin-2 in shellfish, red highlights changes in the structure during the metabolism step

To further complicate matters the metabolism of PTX2 is species dependent, and a different metabolic process has been observed in shellfish from New Zealand (scallops, *Pecten novaezelandia*; mussels, *Perna canaliculus* and *Mytilus galloprovincialis*) and Norway (mussels, *Mytilus edulis*). In this case, the lactone moiety of PTX2 undergoes rapid enzymatic hydrolysis giving PTX2SA (Figure 3). This

biotransformation of PTX2 to PTX2SA was confirmed by *in vitro* experiments which showed that bivalve extracts converted PTX2 to PTX2SA (Suzuki *et al.*, 2001a; Suzuki *et al.*, 2001b; Miles *et al.*, 2004a). Although 7-epi-PTX2SA is detected in shellfish extracts, it appears that this is formed by non-enzymatic isomerisation (Sasaki *et al.*, 1998; Suzuki *et al.*, 2003).

2.1.5. Methods of Analysis

PTX-group analogues were first discovered as co-extractives while isolating OA-group toxins (Yasumoto *et al.*, 1985), and resulted in positive test results with the non-specific DSP screening mouse bioassay as they are toxic to mice by intraperitoneal administration. However, the development and validation of analytical methods for PTX-group analogues has been limited due to scarcity of reference materials, and the complexity of producing them in the laboratory. PTX2 is currently the only commercially available certified calibration solution on the market for this group and is available from both the National Research Council Canada (NRCC), as well as from Cifga in Spain. In the joint FAO/IOC/WHO expert consultation review it was stated that PTX2SA reference material was in development and the EFSA opinion stated in 2009 that PTX11 was expected to be released that year. Despite these intentions, as of 2019 neither of these materials have been released to market.

In New Zealand the PTX-group is monitored using liquid chromatography coupled to tandem mass spectrometry (LC-MS/MS). While this is a highly specific method of analysis, due to the absence of reference materials many of the PTX-group analogues are unable to be identified or quantified. Routinely, PTX2 as well as non-regulated metabolites PTX2SA and 7-epi-PTX2SA, collectively reported as pectenotoxin 2 seco acids (PTX2SAs), are monitored. In addition, PTX1, PTX11, and PTX6 can be identified by the method, but are not routinely processed and quantified. This is because only PTX2 and the seco acids could be analysed due to instrumental limitations. As technology improved PTX1, PTX11 and PTX6 could be detected and currently if high levels of PTX2 are observed then a retrospective reprocessing is conducted. As no reference materials are available for the other PTX-group analogues, the quantities are estimated using PTX2 as a reference standard with an assumed relative response factor of 1. A retention time quality control mixed extract is used to allow identification of peaks for PTX2, PTX2SA, 7-epi-PTX2SA, PTX1, PTX11 and PTX6.

2.2. Okadaic acid group

2.2.1. Background

Toxins from the OA group have been known to cause human illness since the late 1970s (Yasumoto *et al.*, 1978). The syndrome was named diarrhetic shellfish poisoning (DSP) due to the dominating symptom. The OA group has been detected in microalgae and/or bivalve molluscs globally. Analyses for this group have been a key

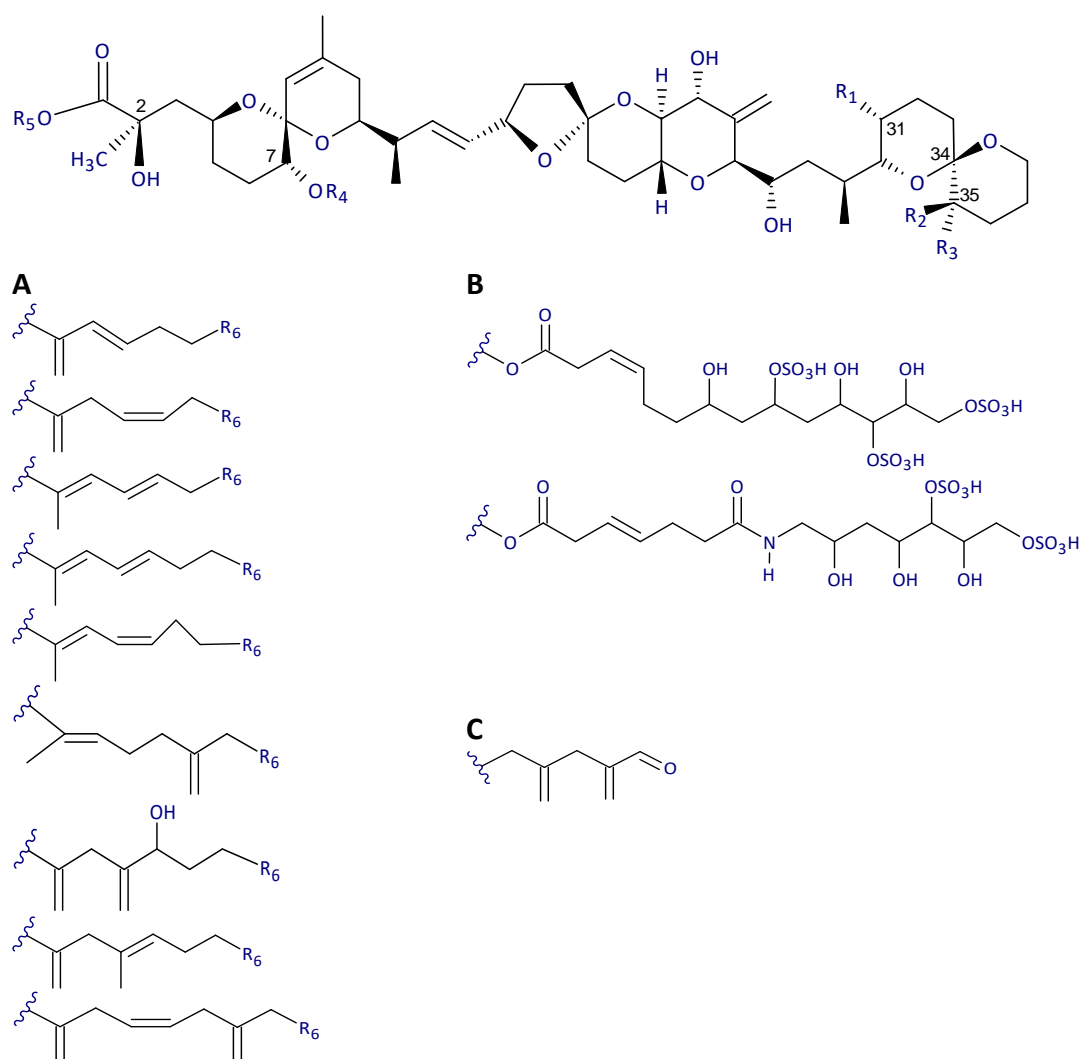
part of many biotoxin monitoring programmes. However, animal bioassays are unreliable for the quantitation of OA-group toxins as extracts are usually accompanied by other lipophilic compounds such as the PTX-group. This led to the development of multi-toxin methods based on LC-MS/MS to identify the different compounds so that the OA-group toxins can be regulated more accurately and quickly. OA-group toxins readily form esters with fatty acids and require hydrolysis prior to analysis by chemical methods. A regulatory level of 0.16 mg OA eq/kg has been established by Codex Alimentarius and has been adopted by New Zealand, Japan, USA, Mexico, Chile and Europe. However, some countries such as New Zealand and Europe currently include the PTX-group with the OA-group for DSP regulation.

2.2.2. Production and Accumulation

In addition to the PTX-group *Dinophysis* spp. produces the DSP toxins, OA, DTX1, and DTX2 (Yasumoto *et al.*, 1985). The co-occurrence of PTX with these DSP toxins has been the driving force for investigation of the PTX congeners and appears to be the basis for grouping PTX-group analogues and OA-group toxins in regulation. OA-group toxins are also produced by benthic dinoflagellates of the genera *Prorocentrum* (Dickey *et al.*, 1990; Quilliam *et al.*, 1996).

2.2.3. Chemistry

The chemical structures of OA-group toxins are shown in Figure 4. These toxins are lipophilic and heat stable. OA and DTX2 differ only by the position of one methyl group in the molecule and DTX1 has one additional methyl group. The C-1 carboxyl terminus and C-7 hydroxyl group are both commonly modified by esterification. The 7-O-acyl ester derivatives of OA, DTX1 and DTX2 are collectively known as DTX3. The C-1 carboxyl group can conjugate to different unsaturated diols forming allylic OA diol-esters. These OA diol-esters are produced by *Prorocentrum* (Yasumoto *et al.*, 1987) and *Dinophysis* spp. (Suzuki *et al.*, 2004). Water-soluble derivatives of these OA diol-esters have also been reported, named dinophysistoxin-4 (DTX4) and dinophysistoxin-5 (DTX5), in which the diol-esters are further conjugated to a polar side chain (Hu *et al.*, 1995; Macpherson *et al.*, 2003; Cruz *et al.*, 2006; Paz *et al.*, 2007).



Toxin	R ₁	R ₂	R ₃	R ₄	R ₅	R ₆
OA	CH ₃	H	H	H	H	
DTX1	CH ₃	CH ₃	H	H	H	
DTX2	H	H	CH ₃	H	H	
DTX3	H or CH ₃	H or CH ₃	H	Acyl	H	
Diol esters	CH ₃	CH ₃	H	H	A	OH
Sulfated DTX4/5	CH ₃	CH ₃	H	H	A	B
DTX6	CH ₃	CH ₃	H	H	C	

Figure 4. The structures of OA-group toxins

2.2.4. Metabolism in shellfish

When ingested by shellfish, a portion of the DSP toxins present in the dinoflagellates are acylated at the C-7 hydroxyl group with long-chain fatty acids, forming derivatives collectively known as DTX3 (Yasumoto *et al.*, 1989; Marr *et al.*, 1992).

2.2.5. Methods of Analysis

Currently in New Zealand some analogues of the OA-group are monitored using liquid chromatography coupled to tandem mass spectrometry (LC-MS/MS). This is a highly specific method of analysis but requires a chemical conversion step in order to hydrolyse the naturally produced esters of the OA-group toxins and allow determination of total OA-group toxins. Routinely OA, DTX1 and DTX2 are monitored, and analysed in the hydrolysed extract. OA, DTX1 and DTX2 can also be analysed without hydrolysis to determine how much of these compounds exist as the unesterified form, however only the total after hydrolysis is used for regulatory purposes. Reference materials are available for OA, DTX1 and DTX2, however DTX1 and DTX2 are only available in limited supply. OA is therefore used for quantification of DTX1 and DTX2, and relative response factors routinely experimentally determined using the DTX1 and DTX2 certified reference material. Certified reference materials must be authenticated and calibrated by an accredited reference material provider using traceable measures, for marine toxins this is typically performed by quantitative nuclear magnetic resonance (NMR). A naturally contaminated sample extract with well characterised detections of OA, DTX1 and DTX2 is included with each batch of samples analysed on the LC-MS/MS instrument to allow accurate identification of the congeners.

3. HAZARD CHARACTERISATION

3.1. Pectenotoxin group

3.1.1. Absorption, distribution, metabolism and excretion

Although not specifically designed to determine bioavailability of PTX toxins two studies have been conducted which provide some information. In the first study, Burgess (2003) reported after the dosing of a mixture of PTX2 and PTX2SA to mice by gavage, most of the toxins remained within the gastrointestinal tract and were almost fully excreted in the faeces without being absorbed. Traces of PTX-group analogues were detected in the livers of the animals after 6 hours, and 11% was present in the tissues of the gastrointestinal (GI) tract. None was excreted in the urine, and none could be detected in tissues after 24 hours. Following intraperitoneal injection (i.p.) administration of a similar mixture of PTX-group analogues these compounds could be detected in blood, internal organs and the GI tract. All detectable PTX was excreted in the faeces rather than in urine (Burgess, 2003). In the second study (unpublished), in Norway conducted by Espenes *et al.* (EFSA, 2009a) a single dose of PTX2 was administered by gavage at 5 mg/kg bw to mice. After 24 hours most of the PTX2 was found in the stomach (7 µg/g) with low amounts in the duodenum (0.27 µg/g), small intestine (0.13 µg/g) and colon (0.05 µg/g) and with trace quantities in the liver, kidney, heart and whole blood (<0.007 µg/g) (EFSA, 2009a). These experiments all suggest a low absorption of PTX-group analogues in the gut of mice following oral administration. There are no data on absorption, distribution, metabolism or excretion in humans.

3.1.2. Toxicity in animals

Pectenotoxins were first discovered as co-extractives while isolating OA-group toxins (Yasumoto *et al.*, 1985), and resulted in positive test results using the non-specific DSP mouse bioassay as they are toxic to mice by i.p.. The acute i.p. toxicities for the different PTX analogues are summarised in Table 1. However, information on feeding method, strain and sex of mice, is not documented in most of the available publications, which makes the interpretation and an accurate comparison of the data difficult. It is clear that PTX1, PTX2, PTX3 and PTX11 are of similar toxicity with lethal doses of between 192 and 411 µg/kg bw, PTX4 and PTX6 appear to be slightly less toxic with lethal doses of 770 and 500 µg/kg bw, respectively and PTX7, PTX8, PTX9, PTX2SA and 7-epi-PTX2SA are of low toxicity, with no deaths observed even at a dose rate of 5000 µg/kg bw.

Table 1. Acute toxicities of PTX derivatives in mice by intraperitoneal injection

Compound	Sex	Parameter ^a	Acute toxicity (µg/kg bw) ^b	Reference
PTX2	?	LD ₅₀	411	(Yoon and Kim, 1997a)
PTX2	Female	LD ₅₀	219 (183-257)	(Miles <i>et al.</i> , 2004a)
PTX11	Female	LD ₅₀	244 (214-277)	(Suzuki <i>et al.</i> , 2006)
PTX1	?	MLD	250	(Yasumoto <i>et al.</i> , 1985)
PTX2	?	MLD	260	(Yasumoto <i>et al.</i> , 1985)
PTX2	Female	MLD	192	(Miles <i>et al.</i> , 2004a)
PTX3	?	MLD	350	(Yasumoto <i>et al.</i> , 1989)
PTX4	?	MLD	770	(Yasumoto <i>et al.</i> , 1989)
PTX6	?	MLD	500	(Yasumoto <i>et al.</i> , 1989)
PTX7	?	MLD	>5000	(Sasaki <i>et al.</i> , 1998)
PTX8	?	MLD	>5000	(Sasaki <i>et al.</i> , 1998)
PTX9	?	MLD	>5000	(Sasaki <i>et al.</i> , 1998)
PTX2 seco acid	Female	MLD	>5000	(Miles <i>et al.</i> , 2004a)
7-epi-PTX2 seco acid	Female	MLD	>5000	(Miles <i>et al.</i> , 2006b)

^a MLD, minimum lethal dose; LD₅₀, median lethal dose

^b Brackets indicate 95% confidence limits

"?" indicates that this information was not provided in the cited reference

Mice injected with PTX2 quickly showed discomfort becoming hunched and lethargic soon after dosing. Over time, abdominal breathing was observed and respiration became laboured and progressively slowed. Cyanosis was observed before death which typically occurred 4 to 10 hours post-dosing (Yoon and Kim, 1997a; Miles *et al.*, 2004a). The symptoms of intoxication were identical for PTX11 (Suzuki *et al.*, 2006).

Histopathological studies have revealed that the major target for PTX2 is the liver. An i.p. injection of PTX2 induced a dose-dependent increase in alanine aminotransferase (ALT), aspartate aminotransferase (AST) and sorbitol dehydrogenase (SDH) (Yoon and Kim, 1997a). Consistent with this, the injection of PTX1 induced characteristic liver injuries (Terao *et al.*, 1986) and PTX6 has been shown to induce hepatic hemorrhage as well as injuries to the gastric organs and kidney (Ito *et al.*, 2008). In contrast, no histological changes were noted in the liver, kidneys, spleen, lung, heart, adrenals, thyroid, trachea, ovary, uterus, tongue, brain, pancreas or urinary bladder in mice killed 24 hours after i.p. injection of PTX2SA or 7-epi-PTX2SA at a dose rate of 5000 µg/kg (Miles *et al.*, 2006b).

In comparison to the i.p. route of administration there have been few studies conducted to investigate the acute oral toxicity of the PTX-group analogues. The results available are summarised in Table 2. Although focussed on yessotoxins, a study in the 1990s (Ogino *et al.*, 1997), reported what appeared to be oral acute toxicity of PTX2. In this study the oral toxicity of PTX2 was reported to be similar to that generated by i.p. injection. However, the results reported appear dubious as no

dose-dependency was observed. The mortality recorded at a dose of 25 µg/kg bw (25%) was higher than that seen in mice given 100 µg/kg bw (0%) or 200 µg/kg bw (20%) while that observed at a dose of 400 µg/kg bw (25%) was lower than that recorded at 300 µg/kg bw (40%). In contrast, the study by Miles *et al.* (2004a) showed no signs of toxicity in any of the 5 mice dosed PTX2 at a dose rate of 5000 µg/kg. It is unclear why the two studies gave conflicting results but it should be noted that there can be a high incidence of gavage-associated deaths and that the administration technique can impact on the results (Rao *et al.*, 2001; Damsch *et al.*, 2011; Munday, 2014). The acute oral toxicity of PTX2SA (Miles *et al.*, 2004a) and PTX11 (Suzuki *et al.*, 2006) was found to be equally low with no signs of toxicity observed in any of 5 mice dosed with either compound at a dose rate of 5000 µg/kg bw.

Table 2. Acute toxicity of PTX derivatives in mice by oral administration (gavage)

Compound	Sex	Parameter	Acute toxicity (µg/kg bw)	Reference
PTX2	Male	LD ₅₀	~200*	(Ogino <i>et al.</i> , 1997)
PTX2	Female	MLD	>5000	(Miles <i>et al.</i> , 2004a)
PTX11	Female	MLD	>5000	(Suzuki <i>et al.</i> , 2006)
PTX2 seco acid	Female	MLD	>5000	(Miles <i>et al.</i> , 2004a)

* This estimate is questionable because of the absence of a dose-response in this study.

In summary, the study shows that although PTX2 and PTX11 are among the most toxic PTX-group analogues by i.p. injection both show no toxicity at a dose rate of 5000 µg/kg bw when administered orally. This difference is likely to be due to a low level of uptake from the gastrointestinal tract. Consistent with this assumption, after an oral administration of a mixture of PTX2 and PTX2SA the majority of the toxins remained within the gastrointestinal tract and were excreted in the faeces (Burgess, 2003). PTX7, PTX8, PTX9, PTX2SA and 7-epi-PTX2SA were not toxic even at 5000 µg/kg bw by i.p. injection and PTX2SA was also not toxic at this dose rate orally.

Two sub-chronic toxicity studies have been reported using i.p. administration. At a dose rate of 20 or 100 µg/kg bw of PTX2 (daily) in mice over a 1 or 2 week period did not cause deaths or changes in clinical chemistry indicative of liver or kidney toxicity, whilst at 200 µg/kg bw 50 % of the animals died (Yoon and Kim, 1997b).

Administration of PTX2 i.p. at 100 µg/kg bw for 20 consecutive days to nude mice inoculated with tumour cells had no effect on body weight (Chae *et al.*, 2005). There is no information available regarding the oral chronic toxicity or genotoxicity of the PTX-group analogues.

The question as to whether or not the PTX-group analogues induce diarrhoea is of great importance as it underpins the validity of whether the PTX-group should be included in the DSP class of toxins. This question has been complicated by the fact that *Dinophysis* spp. produces not only PTX-group analogues but also okadaic acid

and its derivatives which are well known for their diarrhetic effect. It is therefore inevitable that algal extracts and shellfish extracts contain both groups of toxins. Further compounding this difficulty, the PTX and DSP group toxins are hard to separate chromatographically, such that pure PTX-group analogues are hard to achieve. Preparations used for toxicological evaluation must be proved to be free of contaminating DSP toxins. PTX1 did not cause diarrhoea when injected i.p. into suckling mice at a dose rate of 1000 µg/kg bw (Terao *et al.*, 1986), or when given by gavage (Hamano *et al.*, 1986). Using intestinal models Hamano *et al.* (1986) showed that PTX1, unlike OA or the DTXs, caused no fluid accumulation in rabbit or mouse intestinal loops. In contrast, Ishige *et al.* (1988) dosed PTX2 of unspecified purity to mice by gavage and noted diarrhoea and fluid accumulation in the intestine of 1/1 mouse dosed at 250 µg/kg bw. Diarrhoea also occurred in 1/5 mice dosed at 1000 µg/kg bw and in 1/1 and 1/1 mice dosed at 2000 and 2500 µg/kg bw, respectively. Ito *et al.* (2008) used intestinal weight as an indicator of diarrheagenicity and showed that PTX6 did not induce increased intestinal weight in rats at a dose rate of 2000 µg/kg bw. In contrast, these authors reported that rats dosed with PTX2 (of unspecified purity) at 1500 µg/kg bw showed a diarrhetic effect. However, no diarrhoea was observed in mice dosed with fully authenticated PTX2, PTX11 or PTX2SA at 5000 µg/kg bw orally (Miles *et al.*, 2004a; Suzuki *et al.*, 2006). It is possible that the Ishige and Ito studies may have used PTX2 contaminated with DSP toxins. If so, these results would be consistent with the PTX-group compounds not inducing diarrhoea.

3.1.3. Toxicity Equivalency Factors

For risk assessment and management, knowledge of the amount of toxin congeners in the shellfish is not sufficient. There is also the need to know the relative toxicity of each of the congeners, so that the total toxicity of the material in the extract can be estimated. This requires the determination of Toxicity Equivalency Factors (TEFs) (FAO/WHO, 2016). In the EFSA (2009a) review, a provisional TEF of 1 was proposed for PTX1, PTX2, PTX3, PTX4, PTX6 and PTX11 until more robust data becomes available. PTX7, PTX8, PTX9, PTX2SA and 7-epi-PTX2SA were not assigned TEFs due to their low toxicity in the animal studies available. In the 2016 Joint FAO/WHO review of Toxicity Equivalency Factors for Marine Biotoxins Associated with Bivalve Molluscs, the i.p. toxicity of pectenotoxins were discussed but no TEFs for this group were recommended.

3.1.4. Mechanism of Action

Unlike OA and its derivatives PTX1 showed no inhibitory activity in the protein phosphatase 2A assay (Fladmark *et al.*, 1998). Instead, PTX-group analogues interact with F-actin, causing changes in the structure of the cellular cytoskeleton, and there is evidence that such interaction is involved in the toxicity of these substances to cells *in vitro* (Chae *et al.*, 2005; Rossini and Hess, 2010). The PTX-group analogues that show acute toxicity *in vivo* have also been shown to interact with actin in primary hepatocytes or neuroblastoma cells *in vitro*, while little or no effect was seen with the

relatively non-toxic PTX9 and PTX2SA (Ares *et al.*, 2007; Espina *et al.*, 2008; Espina *et al.*, 2010). PTX2 has been shown to cause a concentration-dependent decrease in both rate and yield of skeletal muscle actin polymerisation and this inhibitory effect was also noted for all other actin isoforms (cardiac, smooth muscle and non-muscle actin). In contrast, PTX2SA showed no effect on the polymerisation of any of the actin isoforms (Butler *et al.*, 2012). It is still unclear whether the interactions with actin are involved with the toxicity of PTX-group analogues *in vivo*, and the pathway or pathways whereby interactions could cause tissue damage or death is presently unknown (Munday, 2014).

3.1.5. Observations in humans

Significant poisoning events have occurred in Australia which were initially attributed to the presence of PTX2SA. In one event in 1997, 100 people were poisoned following consumption of pipis (*Plebidonax deltoides*) harvested off the NSW coastline which resulted in 56 cases of hospitalisation. Symptoms of this poisoning included nausea, vomiting and diarrhoea (Burgess and Shaw, 2001). This result seemed contradictory to the toxicity and lack of diarrheagenicity of the PTX-group analogues in mice and it was later found that the toxic shellfish contained esters of OA-group toxins. These were not initially detected at the time of the event as they require a hydrolysis step to be performed during sample preparation to allow detection (Burgess, 2003). These compounds are now believed to be the cause of the poisoning event in Australia and currently it is widely accepted that there is no evidence of any adverse effects of PTX-group analogues in humans (FAO/IOC/WHO, 2004; Munday, 2017).

3.1.6. Evaluation of Hazard Characterisation

In 2004 the FAO expert consultation considered that the data available was insufficient to establish an acute reference dose (ARfD) for the PTX-group and determined human exposure was >8300 (Canada) and >3100 (Norway) times lower than the LD₅₀ by gavage in mice (FAO/IOC/WHO, 2004). In 2006, the Codex Alimentarius Commission recommended that no action level for PTX2 should be set in the Codex standard and that they should not be regulated. In the 292-2008 standard that was revised in 2015 there is no regulation of the PTX-group (CX/FFP 06/28/6 Add.1 2006; Codex Standard 292-2008, 2015).

The EFSA CONTAM Panel derived an ARfD of 0.8 µg PTX2 eq/kg from the reported lowest-observed-adverse-effect level (LOAEL) for PTX2 of 250 µg/kg bw based on a single mouse dosed by gavage with PTX2 of unspecified purity (Ishige *et al.*, 1988; EFSA, 2009a). The effects observed in this study involved fluid accumulation in the intestine and damage to intestinal villi. Such changes are seen with okadaic acid derivatives, and since PTX-group analogues coexist with these substances, from which they are difficult to separate, this result must be viewed with caution, particularly since a dose of 5000 µg/kg of a certified sample of PTX2 caused no toxic effects in

mice (Munday, 2014). It is therefore likely that the ARfD suggested by the CONTAM Panel is set too low.

3.2. Okadaic acid group

3.2.1. Absorption, distribution, metabolism and excretion

Experiments with tritium labelled OA orally administered to mice showed that OA was present in all tissues of the mice. However, after 24 hours the highest amount of OA was observed in the intestinal contents, significant quantities had been excreted in urine and faeces and only very small amounts were present in organs (brain, lung, spleen, heart, liver, kidney) (Matias *et al.*, 1999). When dosed with 50 µg/kg bw more than 59% of the recovered OA was accounted for in the stomach, liver and gall bladder, intestinal content and intestinal tissue, and faeces. In mice dosed with 90 µg/kg bw, the intestinal tissue, content and stomach accounted for 77% of the total OA recovered. This is in accord with the symptomology of diarrhoea, which was observed at the higher dose rate (90 µg/kg bw) and this study indicated that the diarrhoea is due to an increase of OA in intestinal cells.

In a case of human intoxication from shellfish containing acyl derivatives of DTX1, only free DTX1 was observed in the faeces, suggesting that hydrolysis of OA-group esters can occur within the human gastrointestinal tract (García *et al.*, 2005).

In vitro, incubation with human cytochromes has been observed to oxidise OA to yield 11-hydroxy-OA, 43-hydroxy-OA and 36-hydroxy-OA. The inhibition of PP2A by these metabolites is only slightly less potent than OA, suggesting that these transformations do not significantly detoxify OA. However, given the potency of OA and relatively low abundance of these metabolites it is unlikely that these metabolites play a significant role in the toxicity of OA (Guo *et al.*, 2010; Liu *et al.*, 2012).

3.2.2. Toxicity in animals

The acute toxicities of okadaic acid and its derivatives to mice by intraperitoneal injection are summarised in Table 3 (Munday, 2014). The acute toxicity of OA determined in different studies was reasonably consistent, with LD₅₀ values of between 192 and 225 µg/kg bw, although survival times were dependent on the strain of mouse used (Suzuki, 2012). The symptoms of toxicity included lethargy and cyanosis and were associated with fluid accumulation in the intestinal lumen and damage to the GI tract (Tubaro *et al.*, 2003). The toxicity of DTX1 (MLD of 160 µg/kg bw) was similar to that of OA but DTX2, DTX3 and DTX4 were all of lower toxicity. On the basis of the results of Aune *et al.* (2007), DTX2 was assigned a relative toxicity of 0.6 compared to OA. The i.p. injection of DTX1 caused intestinal injury whereas DTX3 did not and hepatic effects have also been noted by some authors (Ito and Terao, 1994).

Table 3. Acute toxicities of OA-group toxins to mice by intraperitoneal injection

Toxin ^a	Strain	Sex	State of Alimentation	Parameter	Acute Toxicity (µg/kg bw) ^b	Reference
OA	CD-1	Female	?	LD ₅₀	204	(Aune <i>et al.</i> , 2007)
OA	HLA:(SW)BR	Female	?	LD ₅₀	210	(Dickey <i>et al.</i> , 1990)
OA	CD-1	Female	Fed	LD ₅₀	225	(Tubaro <i>et al.</i> , 2003)
					(176-275)	
OA	?	?	?	LD ₅₀	192	(Tachibana <i>et al.</i> , 1981)
DTX1	?	?	?	LD ₉₉	160	(Murata <i>et al.</i> , 1982)
DTX2	CD-1	Female	?	LD ₅₀	352	(Aune <i>et al.</i> , 2007)
DTX4	?	?	?	LD ₅₀	610	(Hu <i>et al.</i> , 1995)
OA	ddY	Male	?	MLD	200	(Yanagi <i>et al.</i> , 1989)
DTX3	?	?	?	MLD	500	(Yasumoto <i>et al.</i> , 1985)
7-O-(16:0)-OA	ddY	Male	?	MLD	5550	(Yanagi <i>et al.</i> , 1989)
7-O-(18:2)-OA	ddY	Male	?	MLD	5550	(Yanagi <i>et al.</i> , 1989)
7-O-(22:6)-OA	ddY	Male	?	MLD	550	(Yanagi <i>et al.</i> , 1989)

^a(16:0) Palmitoyl fatty acid ester

(18:2) Linoleoyl fatty acid ester

(22:6) Docosahexaenoyl fatty acid ester

? Information not provided in the cited reference

^bFigures in brackets indicate 95 % confidence limits.

In contrast to the i.p. route, the information available regarding the oral toxicity of OA and its analogues is limited and is also much less consistent. To highlight this inconsistency, the results of various studies are summarised in Table 4. In one study OA was found to be toxic orally at a dose rate of 400 µg/kg bw while in another no deaths were observed at a dose rate of 1000 µg/kg bw. Some variation may be expected due to the strain of mouse but in both studies CD-1 mice were used although they were of different genders. Furthermore, inconsistencies were found even between different experiments of the same study. Le Hégarat *et al.* (2006) reported mortality of 5/5, 0/3, 5/5 and 0/3 mice at dose rates of 770, 610, 575 and 525 µg/kg bw, respectively. No explanation for this lack of consistency was suggested by the authors other than to say that inconsistent results have previously been reported for the oral toxicity of OA. However, diarrhoea was consistently noted in all studies and histopathological injuries were observed in the small intestines of dosed mice. The toxicity of DTX1 and DTX3 fell within the range of toxicities observed for OA and these compounds also induced diarrhoea as well as intestinal and hepatic injuries.

Table 4. Results of toxicity testing of OA-group toxins to mice orally

Toxin	Strain	Sex	Mortality	Dose rate (µg/kg bw)	Reference
OA	CD-1	Male	LD ₅₀	400	(Ito <i>et al.</i> , 2002)
OA	NMRI	Female	LD ₅₀	880	(Aune <i>et al.</i> , 2012)
DTX1	ddY	Male	LD ₅₀	200-300	(Ogino <i>et al.</i> , 1997)
DTX3	ICR	Male	MLD	750	(Ito and Terao, 1994)
OA	ICR	Male	0/3	750	(Terao <i>et al.</i> , 1993)
OA	ICR	Male	0/5	500	(Yuasa <i>et al.</i> , 1994)
OA	CD-1	Female	0/5	1000	(Tubaro <i>et al.</i> , 2003)
OA	CD-1	Female	4/5	2000	(Tubaro <i>et al.</i> , 2003)
OA	Swiss	Female	5/5	770	(Le Hégarat <i>et al.</i> , 2006)
OA	Swiss	Female	0/3	610	(Le Hégarat <i>et al.</i> , 2006)
OA	Swiss	Female	5/5	575	(Le Hégarat <i>et al.</i> , 2006)
OA	Swiss	Female	0/3	525	(Le Hégarat <i>et al.</i> , 2006)

There is no information available regarding the chronic toxicity and genotoxicity of the OA-group analogues. One sub-acute study has been conducted whereby an oral administration of OA was given daily for 7 days at 1000 µg/kg bw (Tubaro *et al.*, 2004). Two of the five mice died after 5 days and over the experiment mice showed diarrhoea, bodyweight loss and reduced food intake. At necropsy, dark areas were seen on the liver surface and the small intestines were full of fluid. No haematological changes were seen although an increase in ALT and AST were observed which is indicative of liver damage. Histology showed ulceration and submucosal inflammation of the forestomach. The daily dose rate given in this experiment was high compared to the acute oral LD₅₀.

3.2.3. Toxicity Equivalency Factors

In the EFSA (2008) review, a TEF of 1 was assigned to OA and DTX1, and a TEF of 0.6 was assigned to DTX2, with esterified forms treated as equal to the corresponding unesterified toxins (OA, DTX1, DTX2). In the 2016 Joint FAO/WHO review of Toxicity Equivalency Factors for Marine Biotoxins Associated with Bivalve Molluscs, the TEF of 1 was kept for OA and DTX1, and DTX2 was recommended to be changed to 0.5 (FAO/WHO, 2016).

3.2.4. Mechanism of Action

OA-group toxins are potent inhibitors of the serine/threonine protein phosphatases PP1 and PP2A (Bialojan and Takai, 1988). Results from a PP2A assay show that the relative activity of DTX2 and OA corresponds well to the toxicity of these compounds by i.p. injection into mice (Aune *et al.*, 2007). Consistent with this, the relatively non-toxic 7-O-palmitoyl OA derivative is only a weak inhibitor of PP1 and PP2A (Takai *et al.*, 1992). However, this relationship between i.p. toxicity and protein phosphatase

inhibition is less correlated for DTX4 which is 500 times less effective than OA as a PP inhibitor but 300 times less toxic than OA by i.p. injection. Furthermore, 7-O-docosahexaenoyl-OA is highly toxic to mice but has only a mild effect on the protein phosphatases (Nishiwaki *et al.*, 1990). The correlation between diarrheagenic activity and potency toward protein phosphatases is even less correlated illustrating that the mechanism by which the diarrhetic activity is induced by OAs is not yet well understood (Munday, 2014). Injection of OA causes vessel congestion and extravasation into the lamina propria (Hamano *et al.*, 1986; Terao *et al.*, 1986). Based on the available evidence the EFSA panel concluded that the mechanism by which OA induces diarrhoea in animals and humans includes submucosal fluid accumulation in the intestine wall, the fluid then crosses the epithelial barrier by paracellular pathway and is eventually secreted into the intestinal lumen (EFSA, 2008).

3.2.5. Observations in humans

Toxins from the OA-group have been known to cause human illness since the late 1970s and it is a problem of worldwide significance. The predominant symptoms associated with DSP are nausea, vomiting, diarrhoea and abdominal pain which are observed soon after ingestion of contaminated shellfish. Symptoms generally resolve within 2-3 days and no deaths attributable to DSP have been reported (Munday, 2014). It has been suggested that the OA-group toxins could be associated with digestive cancers (Cordier *et al.*, 2000). The prevalence of cancer in various coastal areas of France was surveyed to investigate possible correlations between cancer and the incidence and duration of shellfish harvesting closures due to the presence of OA. After considering alcohol consumption as a confounding factor, a significant correlation between harvest closures and colonic cancer was observed in men, although no such association was observed in women. No measurements of shellfish intake were made in the different areas, and toxin levels in the shellfish that were consumed were not assayed (Cordier *et al.*, 2000). Following on from this study, Lopez-Rodas *et al.* (2006) investigated the association between colorectal cancer and consumption of shellfish in a Spanish population. In this study a statistically significant positive association was observed. However, it should be noted that this study also found a highly significant association between shellfish consumption and meat consumption, the latter of which is a well-known risk factor for colorectal cancer.

3.2.6. Evaluation of Hazard Characterisation

In 2004 the joint FAO/IOC/WHO expert consultation established an acute reference dose (ARfD) of 0.33 µg OA eq/kg (FAO/IOC/WHO, 2004). In 2006, the Codex Alimentarius Commission expert consultation drew conclusions based on real cases of human illnesses from both Japanese and Norwegian data when assessing the OA group. They concluded that the current level of 0.16 mg OA eq/kg provides adequate protection to consumers (CX/FFP 06/28/6 Add.1 2006). The working group noted that most procedures at the time include hydrolysis of naturally occurring esters of OA-group toxins in order to obtain a total DSP toxicity. It has been proven that esters of

OA-group toxins in many cases are the dominant component to total DSP toxicity. When analysing the OA-group toxins using analytical chemistry methods, hydrolysis of the naturally occurring esters is an essential part of the methodology for regulatory monitoring. The EFSA review of OA-group toxins proposed an ARfD of 0.3 µg OA eq/kg (EFSA, 2008), and that in order for a 60 kg adult to not exceed the ARfD based on a 400 g large portion, shellfish should not contain more than 0.045 mg OA eq/kg.

4. EXPOSURE ASSESSMENT

4.1. New Zealand 2009-2019

4.1.1. *Method of Analysis*

Biotoxin testing performed on commercial and non-commercial samples in New Zealand uses liquid chromatography-tandem mass spectrometry (LC-MS/MS). Several changes have occurred with the implementation of this method of analysis over the years with improvements to the technology resulting in improved performance (e.g. limit of detection). Routinely a fixed limit of reporting is established which is reliably able to be achieved by the instrumentation.

For the original single laboratory validation of the method performed in 2002, PTX2 had a limit of detection of 0.01 mg/kg, a limit of quantitation of 0.03 mg/kg and a reporting limit of 0.05 mg/kg. The reporting limit was later reduced to 0.01 mg/kg sometime before 2008 (Holland, 2002). In 2008, new instrumentation allowed the method to be optimised for higher throughput testing (McNabb and van Ginkel, 2008). In 2013, new instrumentation and improvements to the method of analysis allowed the limit of detection for PTX2 to be reduced to 0.007 mg/kg, and the lower limit of quantitation to be reduced to 0.02 mg/kg (Boundy and McNabb, 2013). During the 2009-2019 period all the PTX2 results were reported with a reporting limit of 0.01 mg/kg.

The changes to the method over the years also resulted in changes in performance for the DSP group. Due to poor availability of certified reference materials for the DSP group, only OA was assessed for limit of detection and quantitation, and the performance of DTX1 and DTX2 was assumed to be the same. Free OA was initially assessed with a limit of detection of 0.016 mg/kg. The performance of total OA (after hydrolysis) was not assessed at this time, however the sample preparation results in a more dilute extract which would be expected to have a raised reporting limit. In 2008, total OA (after hydrolysis) was assessed with a limit of detection of 0.041 mg/kg, and a reporting limit of 0.05 mg/kg (McNabb and van Ginkel, 2008). In 2013, total OA was reassessed following method improvements, with a limit of detection of 0.01 mg/kg, and limit of quantitation of 0.05 mg/kg. Between 2009 and June 2015 the reporting limit for OA, DTX1 and DTX2 was 0.05 mg/kg. It was then reduced to 0.01 mg/kg.

4.1.2. *Raw Data*

Biotoxin testing and phytoplankton raw data for 2009-2019 was sourced from the Cawthron laboratory information management system (LIMS) database excluding samples with null entries to either site code or results. For each result data were exported including identifiers, site code, site description, sample ID, sample type, sampled date, received date, analysis method, reported name, reported result, and unit. Results for PTX2, PTX2SAs (sum of PTX2SA and 7-epi-PTX2SA), total OA, total

DTX1 and total DTX2 were extracted for each sample. DSP was calculated by adding the total OA and DTXs toxins following hydrolysis, i.e. excluding the PTX-group. As PTX1, PTX11 and PTX6 were not processed and quantified as part of the monitoring programme no data were available for these congeners to be exported from the LIMS database. Samples from five bloom events (C|201507-12, I|bpk|201607-201703, I|bpk|200904-201005, B|201509-12 and A|boi|201506-12) were reprocessed to quantify PTX1, PTX11 and PTX6 which are acquired in the LC-MS/MS method of analysis although not processed as part of the routine monitoring programme. Raw data for the reprocessed batches (including trace results below the reporting limit) were exported directly from the TargetLynx processing software (Waters Corporation Milford, MA, US).

4.1.3. Data Clean-up

Due to changes in the methods of analysis, analyte reported names, and site codes over this time period, several exports were required to obtain a complete dataset. The raw data from all data exports was pooled together. For both the biotoxin and phytoplankton data sets, where changes to reporting names and site codes had been made over the data set, each of the unique identifiers was normalised to the currently accepted value allowing a coherent dataset to be generated.

4.1.4. Data Exclusions

For the biotoxin data, data from unclassified site locations such as overseas product testing (n=55), imported products (n=12), and Chatham Island (n=5) were removed. This yielded a total of 18947 sample results with sampling dates spanning 4th January 2009 to 2nd September 2019.

For phytoplankton data, data from unidentifiable sites (n=1173) were removed. This yielded a total of 35277 sample results with sampling dates spanning 4th January 2009 to 9th September 2019.

4.1.5. Location Grouping

Each site was classified into their sample zone based on the shellfish sampling zone (leading letter in the site code), and data were plotted over time. For each site, where possible, an approximate decimal degrees latitude, longitude coordinate in the World Geodetic System 1984 (WGS-84) reference frame for the sampling area was retrieved or assigned manually based on its site description. Sample results were plotted over a map of New Zealand using the free and open-source cross-platform geographic information system QGIS.

Where large numbers of positive samples were observed, or where distinct bloom events were isolated to smaller regions within these zones was observed, a subzone was assigned to each of the sites.

4.1.6. Bloom Identification

Bloom events were classified for shellfish sites within New Zealand from 2009-2019 by first grouping the sites by their sampling zone from the leading letter of the site code. Where many samples with overlapping blooms were detected the zones were separated into subzones by identifying natural barriers which isolate the different regions within the shellfish zones. Zones and subzones used for identifying bloom events are listed in Table 5 and their locations shown in Figure 5. Concentrations of PTX2, PTX2SAs, and DSP toxins alongside *Dinophysis* cell counts were plotted over the 10-year period for each subzone.

Blooms were characterised by visually looking at accumulation/depuration patterns in the concentrations over time. Bloom events were assigned if any of the below conditions were observed in at least one sample within the event:

- a) PTX2 was at or above reportable levels (0.01 mg/kg)
- b) DSP toxins were at or above reportable levels (0.05 mg/kg until June 2015, 0.01 mg/kg after June 2015).
- c) PTX2SAs (sum of PTX2SA and 7-epi-PTX2SA) was at or above 0.1 mg/kg (10-fold higher than the reporting limit of 0.01 mg/kg)

The bloom event was determined to start at the first detection of any of the above groups, and end at the last detection of any of the above groups. In several cases, if a new bloom had started prior to the previous bloom depurating and the blooms were decided to be far enough apart to be considered as separate events, then the lowest concentration point was used to divide the two events.

All samples within the zone or subzone were assigned as part of the bloom event over the time period established. Each bloom event was then reviewed, and any sites that were observed to not have had any toxin detections were excluded.

Concentrations of PTX2, PTX2SAs, and DSP toxins alongside *Dinophysis* cell counts for each subzone from 2009-2019 are plotted in Appendix A, with bloom event identifications assigned.

Bloom event identifications were assigned a unique identifier in the following format:

[Zone] | [Subzone] | [StartYYYYMM] - [EndYYYYMM]

Where: [Zone] is the zone in which the sample was analysed from (leading letter of the site code). [Subzone] was the subzone of the sample, as listed in Table 5. The Subzone was only included for bloom events in zones that were divided into subzones. [StartYYYYMM] was the starting date of the bloom in year then month numerical format. And [EndYYYYMM] was the end date of the bloom in year then month in numerical format. For example: I|bpbk|201607-201703 is a bloom event which

is in Zone I, from the Banks Peninsula/Kaikoura subzone (bpk), that started in July 2016 and ended March 2017.

Where the bloom start year and end year were the same, the end year was omitted, and when the bloom had the same start and end month and year the bloom event omitted the end date and “-” separator (e.g. bloom event I|ota|201611). Bloom events which were still ongoing at the time of exporting the raw data from the database were not assigned an end date although terminated with the “-” (e.g. bloom event I|bpk|201904-)

Table 5. List of shellfish sampling zones, subzones and descriptions of the region used for bloom event identification.

Zone	Subzone	Description
A		Northland
A	boi	Bay of Islands
B		Hauraki gulf/Auckland
C		Firth of Thames
D		Bay of Plenty
E		Hawkes bay
F		Northwest coast, north island
G	tas	Tasman bay
G	for	Forsyth island
G	pel	Pelorus Sounds
G	qc	Queen Charlotte Sounds
G	eb	East bay
G	tc	Tory channel
G	ptU	Port Underwood
G	clo	Cloudy bay
H		South Taranaki Bight
I	bpk	Banks Peninsula/Kaikoura
I	ota	Otago
J	wc	West coast
J	Fio ¹	Fiordland
J	Fov	Foveaux Strait

1 – No shellfish samples were observed from Fordland, however some phytoplankton samples from this area were tested.

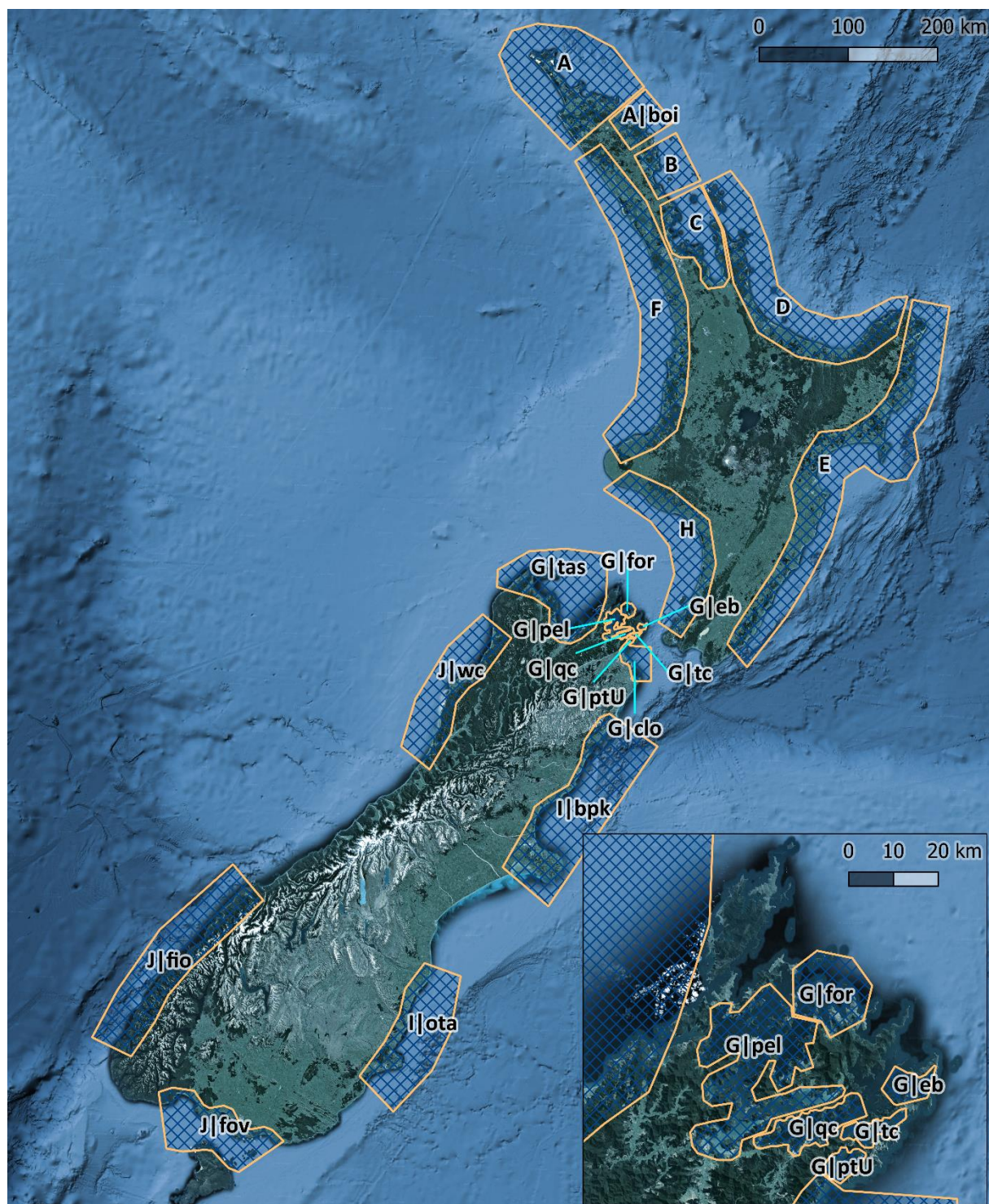


Figure 5. Shellfish zones and subzones used for grouping shellfish sampling sites for bloom event identification.

4.1.7. *Spatial Distribution of PTX in New Zealand*

The number of samples analysed from each testing site in New Zealand over the 2009-2019 period are shown in Figure 6. The maximum concentrations at each shellfish sampling site were plotted for PTX2 (Figure 7), PTX2SAs (sum of PTX2SA and 7-epi-PTX2SA, Figure 8), and DSP (Figure 9). The number of samples, number of detections (observations above reporting limit), percent detections, min, max, mean, median and 97.5th percentile (PCTL) concentrations for PTX2, PTX2SAs and DSP for each shellfish testing site are summarised in Appendix Table C-1.

The approximate number of samples analysed for phytoplankton from each testing site in New Zealand over the 2009-2019 period are shown in Figure 10. The maximum concentrations of *Dinophysis* spp. at each shellfish samples site in New Zealand over the 2009-2019 period were plotted (Figure 11).

PTX2 and *Dinophysis* spp. were both detected throughout the country with notably elevated concentrations and occurrence observed in Banks Peninsula, the Firth of Thames, and Port Underwood. Relative concentrations of PTX2, PTX2SAs and DSP were similar across the different regions, with typically PTX2SAs > DSP > PTX2. However, in some bloom events there was notably relatively less PTX2 and PTX2SAs compared to DSP.

The locations of *Dinophysis* spp. detection were similarly consistent with the observations of PTX2, PTX2SAs and DSP. However, no phytoplankton samples from the West Coast where a PTX2/DSP bloom was detected has been tested. This suggests that some revision of phytoplankton monitoring sites may be advisable. However, the reason for absence of phytoplankton sampling from this area is likely due to the difficulty imposed from the location and environment. The concentrations of *Dinophysis* spp. cell counts did not correlate to detections of PTX2, PTX2SAs and DSP with some higher cell counts not resulting in higher concentrations of PTX2, PTX2SAs and DSP. This is likely due to differences in the production of algal species, and potentially non-producing strains.

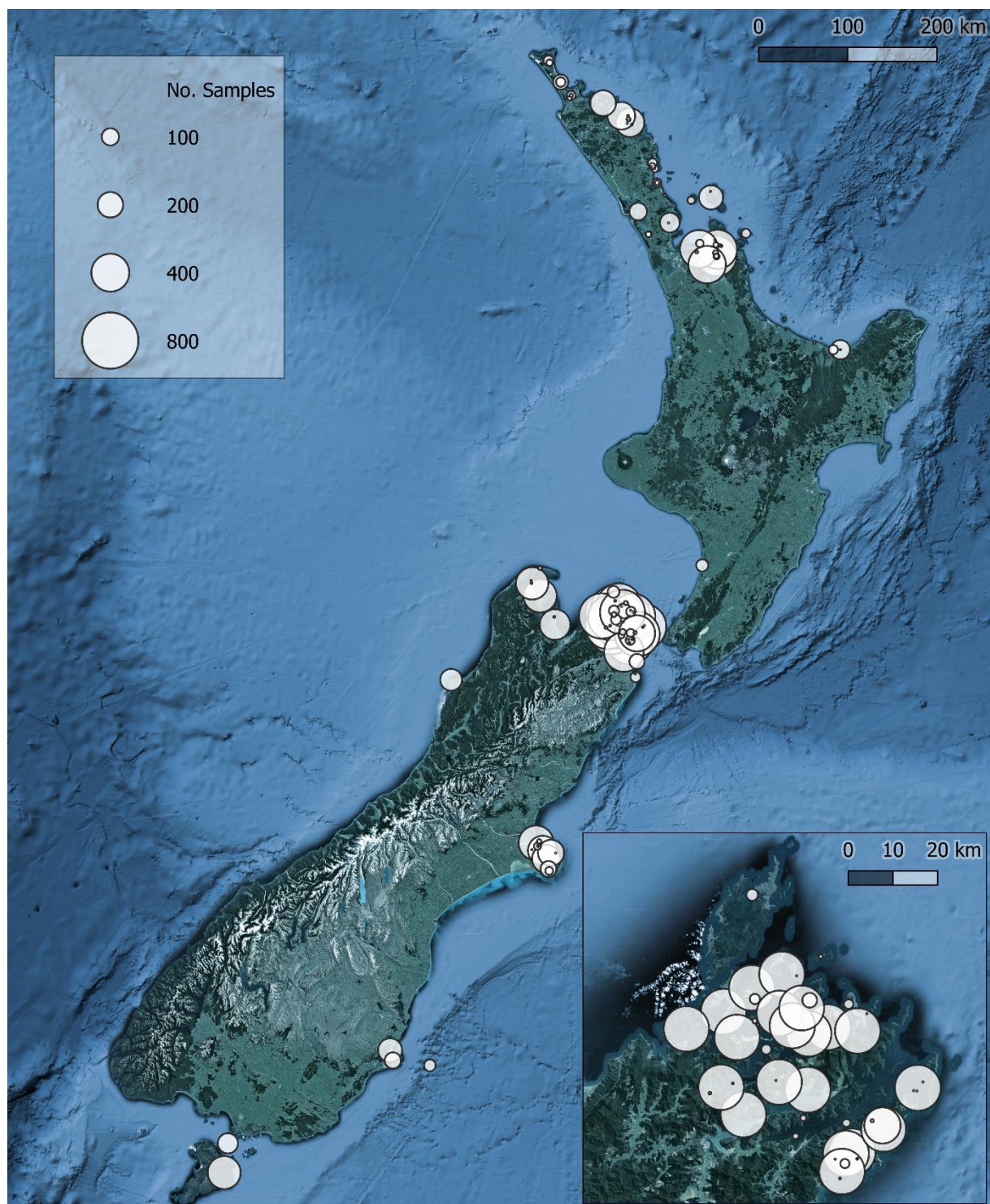


Figure 6. Number of samples analysed for biotoxins at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds. Circle size denotes number of samples tested.

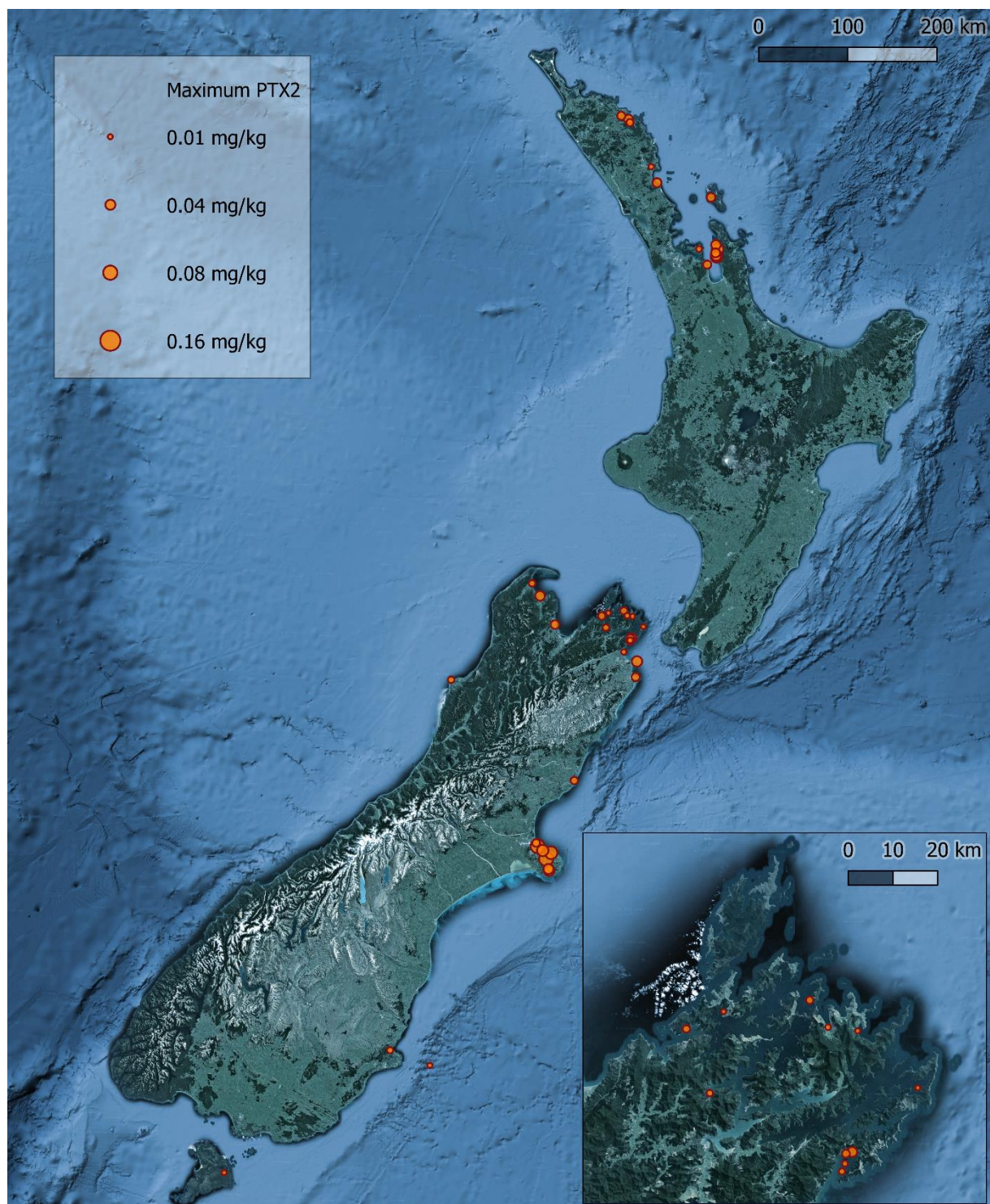


Figure 7. Maximum concentration of pectenotoxin 2 at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.

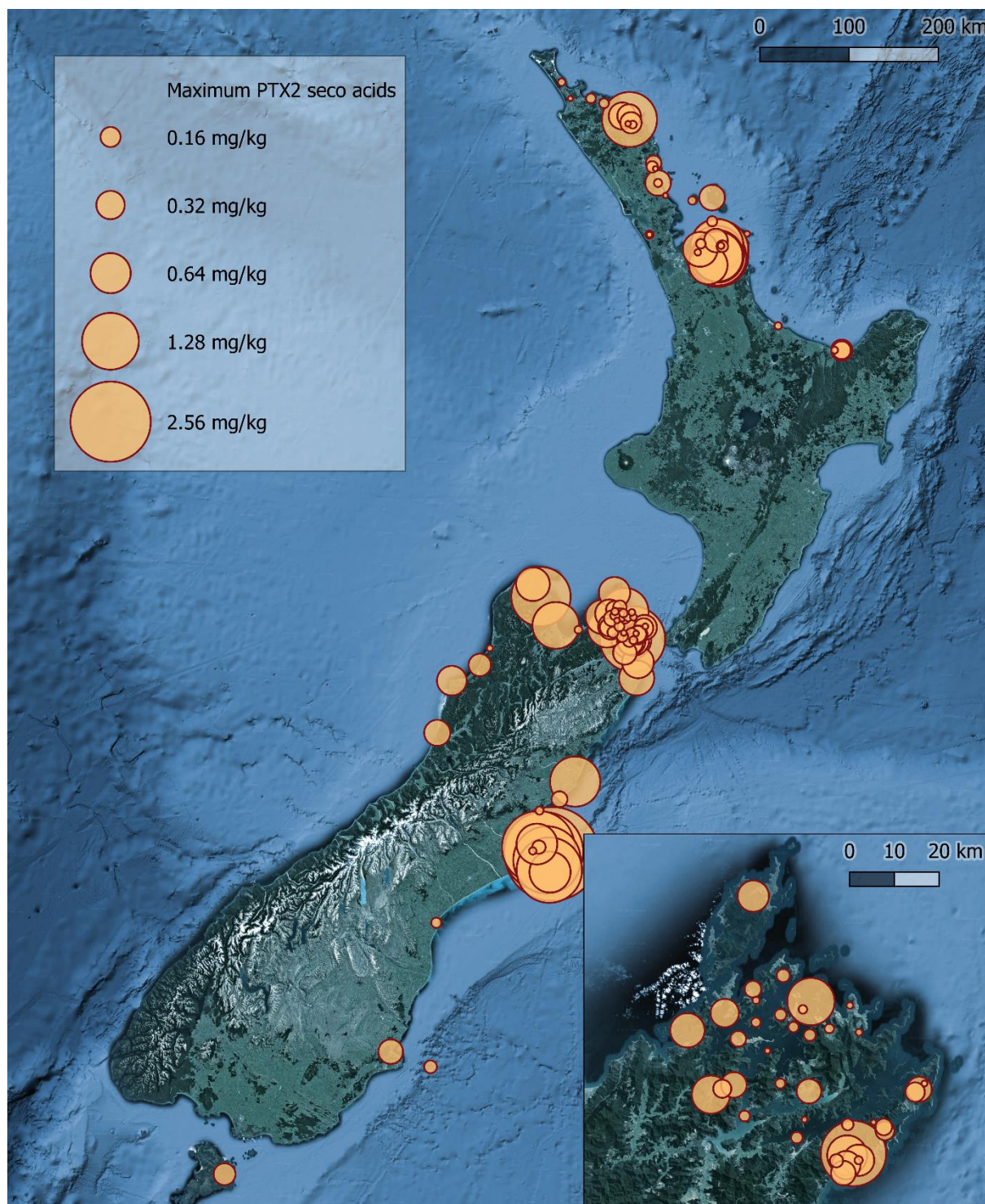


Figure 8. Maximum concentration of pectenotoxin 2 seco acids at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.

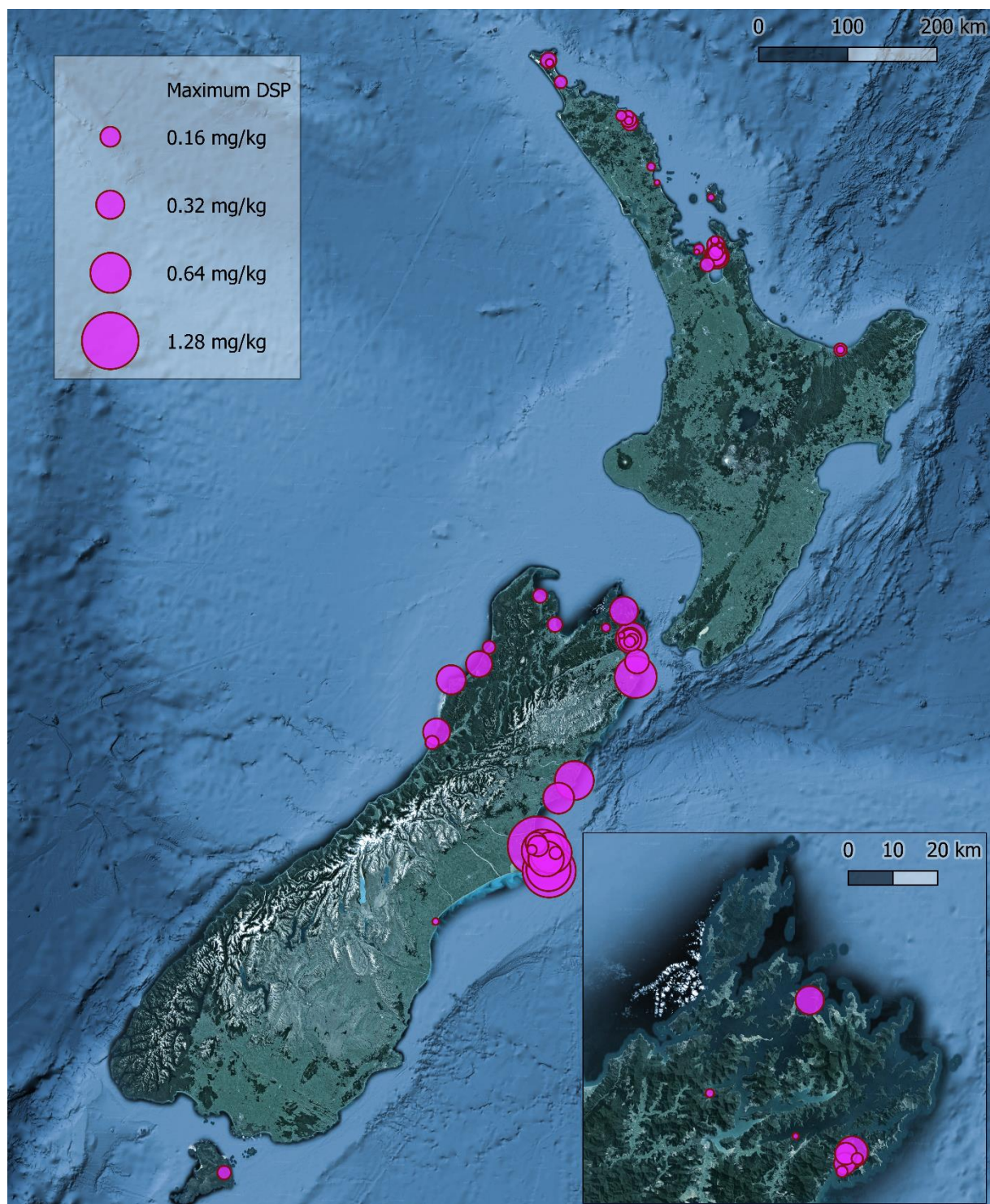


Figure 9. Maximum concentration of diarrhetic shellfish poisoning toxins at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.

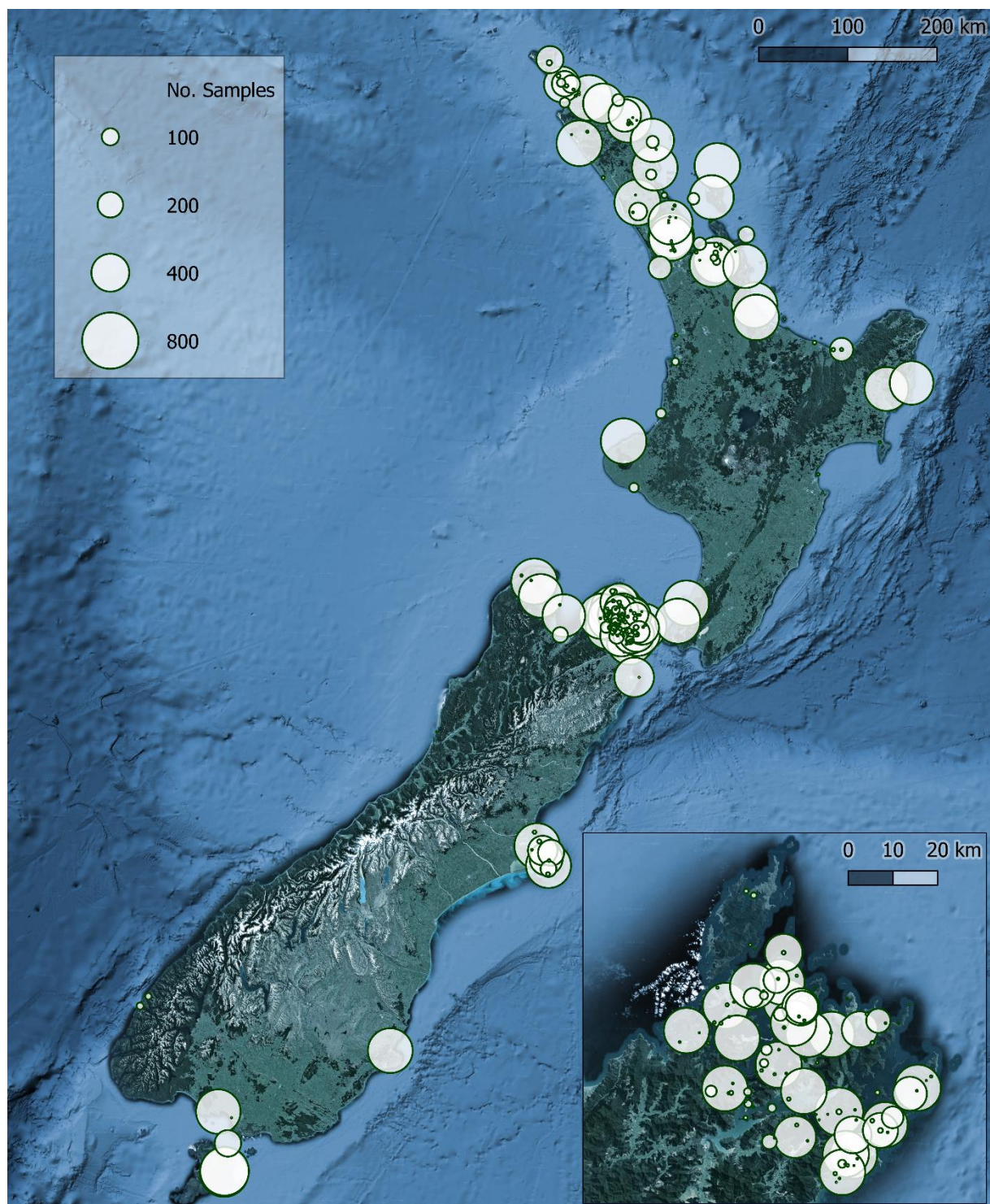


Figure 10. Number of samples analysed for phytoplankton at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.

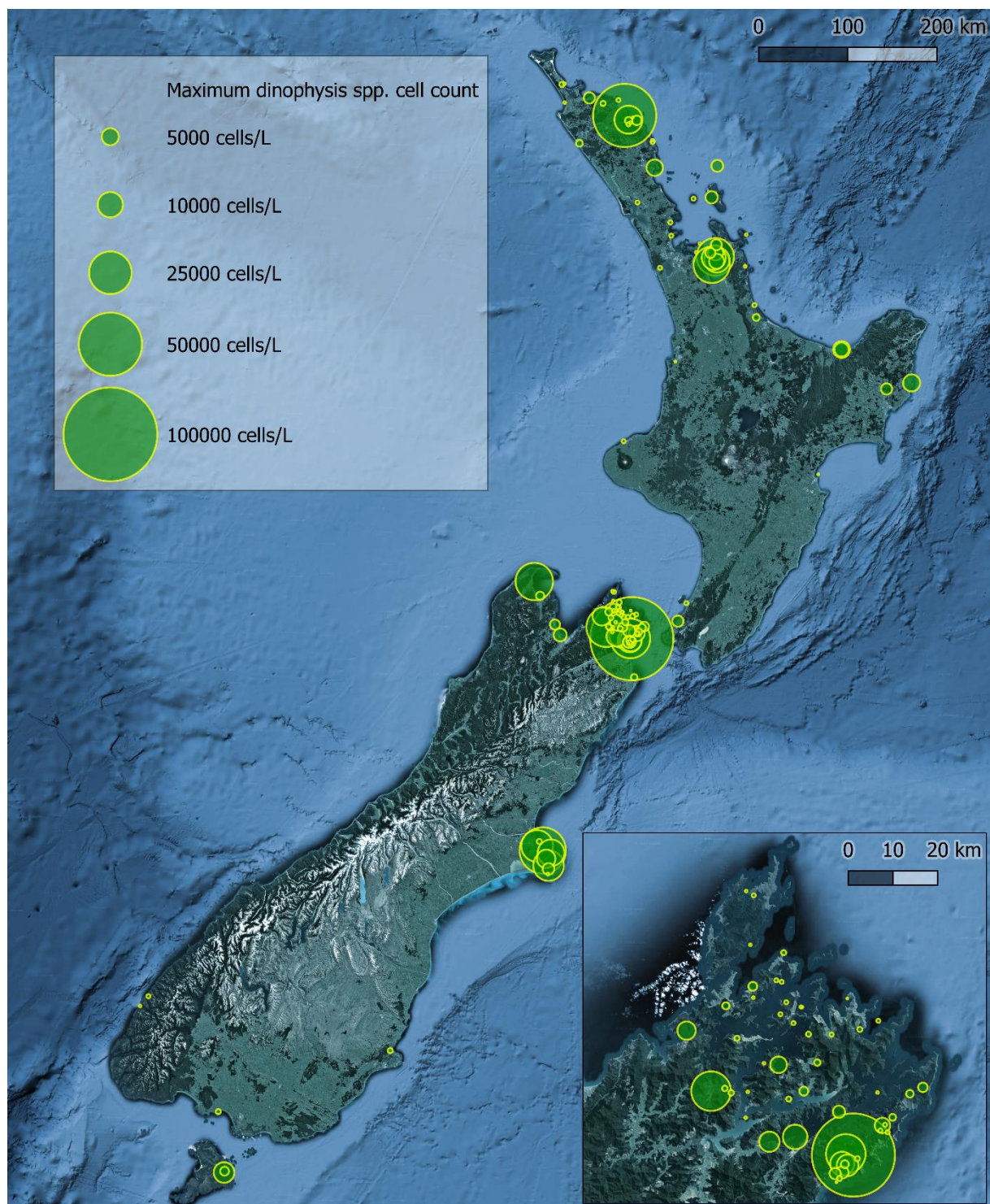


Figure 11. Maximum concentration of *Dinophysis* spp. cells at sample testing sites throughout New Zealand over the 2009-2019 period, with enlarged section for the Marlborough Sounds.

4.1.8. Temporal Distribution of PTX in New Zealand

The concentrations of PTX2, PTX2SAs (sum of PTX2SA and 7-epi-PTX2SA) and DSP in New Zealand (independent of sample site) over the 2009-2019 period were plotted over time, together with the *Dinophysis* spp. cell concentrations (Figure 12). In June 2015 the reporting limit of DSP toxins was reduced from 0.05 mg/kg to 0.01 mg/kg due to acquisition of a more sensitive LC-MS/MS system. This resulted in an increased number of detections of DSP and better visualisation of the data.

Elevated levels of *Dinophysis* spp. were typically observed during periods of elevated PTX2, PTX2SAs and DSP. Differences in the relative amounts of these compounds were observed between bloom events.

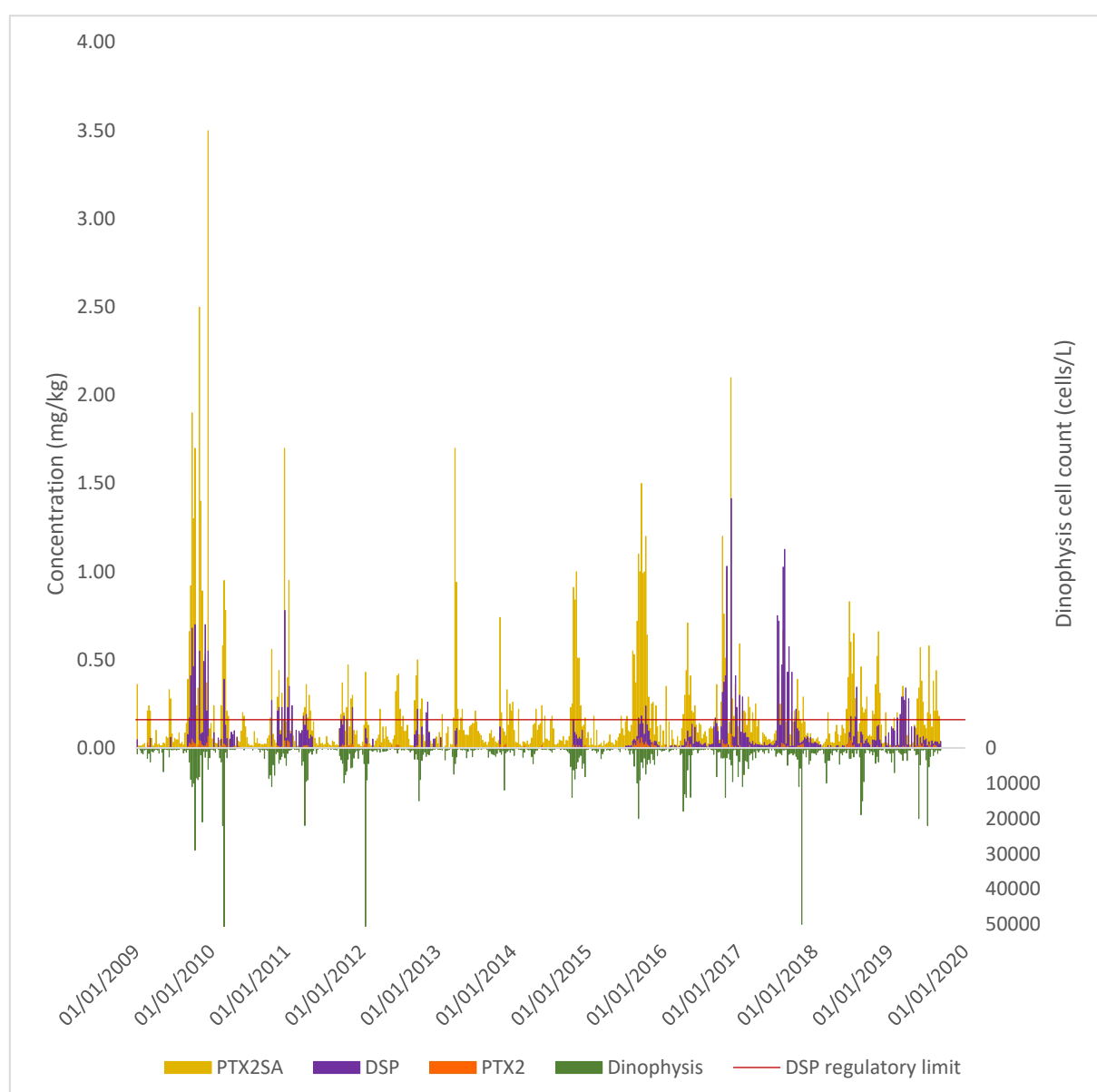


Figure 12. Concentrations of PTX2, PTX2SAs, DSP and *Dinophysis* spp. throughout New Zealand over the 2009-2019 period

Results were grouped by year in order to assess potential change in occurrence over the 2009-2019 period, PTX results are summarised in Table 6, and a comprehensive summary of PTX2, PTX2SAs and DSP is in Appendix Table E-1.

Table 6. Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5th percentile concentrations (mg/kg) of PTX2 in different years in New Zealand over the 2009-2019 period

Year	No. Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
2009	1688	56	3.3%	0.010	0.063	0.019	0.015	0.048
2010	1618	14	0.9%	0.010	0.041	0.014	0.011	0.035
2011	1684	21	1.2%	0.010	0.043	0.016	0.014	0.038
2012	1647	13	0.8%	0.011	0.025	0.015	0.013	0.024
2013	1723	5	0.3%	0.010	0.021	0.017	0.019	0.021
2014	1776	10	0.6%	0.010	0.016	0.013	0.014	0.016
2015	1871	66	3.5%	0.010	0.059	0.021	0.017	0.053
2016	1836	21	1.1%	0.010	0.079	0.026	0.021	0.078
2017	1924	14	0.7%	0.010	0.027	0.017	0.017	0.026
2018	1857	12	0.6%	0.011	0.058	0.023	0.017	0.054
2019	1323	19	1.4%	0.010	0.024	0.014	0.012	0.023
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052

Both 2009 and 2015 showed elevated bloom occurrence with 3.3-3.5% of samples having detectable PTX2 compared to the other years where only 0.6-1.4% of the samples had detectable PTX2.

There was a marked increase in DSP detections from 2015 onwards due to a decrease in reporting limit from 0.05 mg/kg to 0.01 mg/kg. Before 2015, 0.3-3.2% of samples had detectable DSP, and 5.2-9.0% of samples having detectable DSP after 2015. There was no change in the reporting limit of 0.01 mg/kg for PTX2 over this time period. The larger number of low-level detections for DSP in this period also resulted in a decrease in the median concentration (Appendix Table E-1, Figure E-3).

Results were grouped by month in order to assess potential seasonality, PTX results are summarised in Table 7, and a comprehensive summary of PTX2, PTX2SAs and DSP is in Appendix Table E-2. Detections of PTX2 were observed in all months of the year with the largest number of detections in August-November, with elevated detections also in February.

Table 7. Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5th percentile concentrations (mg/kg) of PTX2 in different months of the year in New Zealand over the 2009-2019 period

Month	No. Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
January	1615	10	0.6%	0.011	0.043	0.020	0.016	0.041
February	1617	30	1.9%	0.010	0.027	0.014	0.012	0.026
March	1679	10	0.6%	0.010	0.023	0.013	0.011	0.021
April	1594	11	0.7%	0.010	0.039	0.016	0.014	0.035
May	1634	10	0.6%	0.011	0.022	0.015	0.015	0.022
June	1574	15	1.0%	0.010	0.058	0.020	0.016	0.052
July	1594	9	0.6%	0.011	0.027	0.016	0.015	0.026
August	1563	21	1.3%	0.010	0.052	0.022	0.018	0.047
September	1514	47	3.1%	0.010	0.059	0.021	0.018	0.054
October	1593	50	3.1%	0.010	0.046	0.017	0.015	0.034
November	1542	28	1.8%	0.010	0.079	0.024	0.017	0.078
December	1428	10	0.7%	0.010	0.063	0.021	0.013	0.058
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052

The number of samples containing reportable levels of PTX2, PTX2SAs and DSP by month over the 2009-2019 period are shown in Figure 13. The number of samples analysed per month is shown in Figure 14. The number of samples was generally consistent with a gradual increase over time and an outlier in October 2015. Increased sampling is most likely in response to the Firth of Thames bloom event C|201507-12. The final sample point showed a low number of samples as this was an incomplete month.

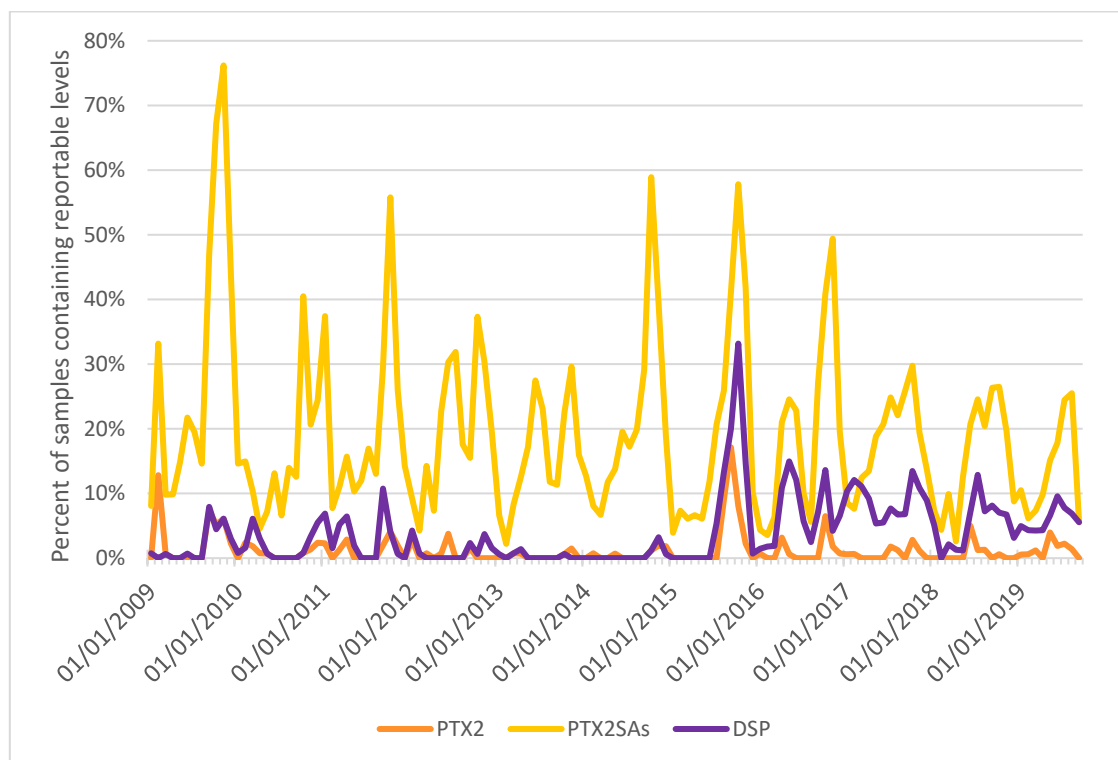


Figure 13. Number of samples containing reportable levels for PTX2, PTX2SAs, and DSP by month over the 2009-2019 period

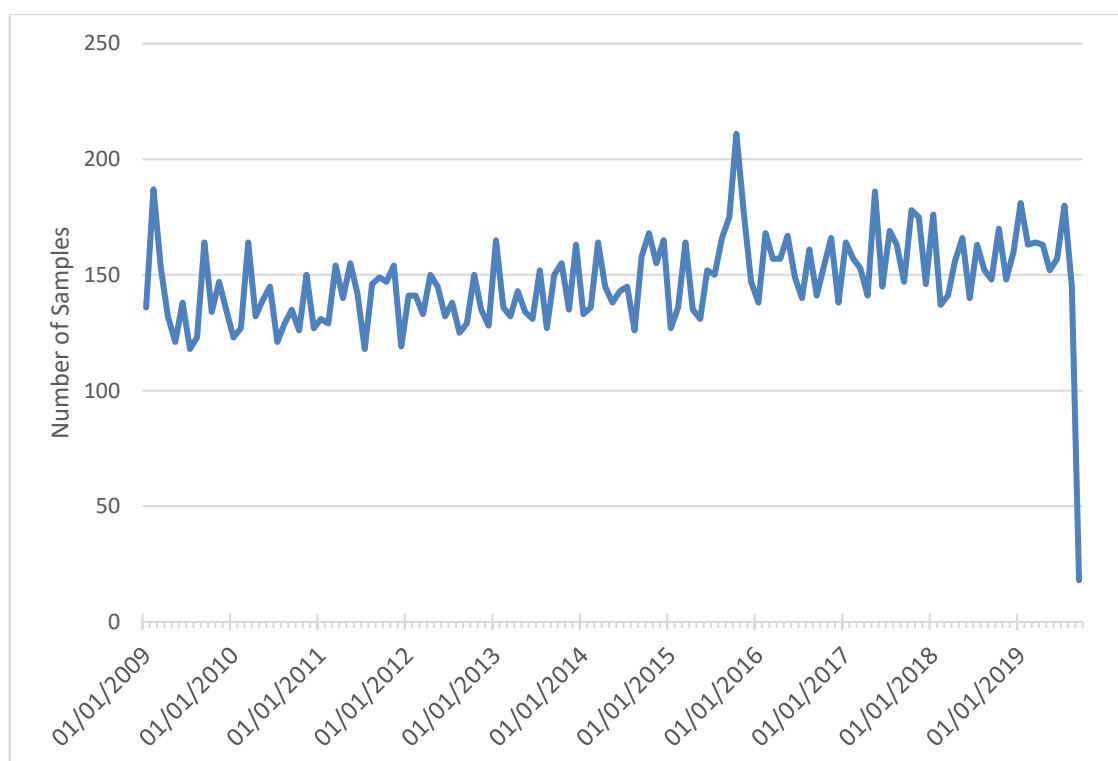


Figure 14. Number of samples analysed by month over the 2009-2019 period

4.1.9. Species distribution of PTX in New Zealand

Sample results were sorted by type of shellfish, results for PTX2 are summarised in Table 8. For comprehensive results summarising PTX2, PTX2SAs and DSP refer to Appendix D. The data available from the LIMS database only identified species by a common name.

The most commonly tested type of shellfish was Greenshell mussels (84%), followed by Pacific oyster (6%), Clams (5%), Scallops (2%) and Dredge oyster (1%). Small numbers of other species (<1% each) were also analysed. The highest percent detection rate for any type was Blueshell mussels. This observation is likely a result of sampling bias as typically Blueshell mussels are not analysed as part of commercial testing and are instead taken from areas in response to a bloom event.

Table 8. Summary of the number of samples analysed, detections, and minimum, maximum, mean, median and 97.5th percentile concentrations (mg/kg) of PTX2 in different types of shellfish analysed in New Zealand over the 2009-2019 period

Organism ¹	Sites	No. Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
Greenshell mussel	83	15947	186	1.2%	0.010	0.079	0.019	0.015	0.056
Pacific oyster	22	1141	40	3.5%	0.010	0.027	0.015	0.015	0.026
Clam	11	1042	6	0.6%	0.013	0.027	0.018	0.016	0.026
Scallop	20	298	4	1.3%	0.012	0.032	0.020	0.017	0.031
Dredge oyster	8	228	1	0.4%	0.043	0.043	0.043	0.043	0.043
Surf Clam	6	97	5	5.2%	0.010	0.024	0.015	0.012	0.023
Blueshell mussel	12	56	7	12.5%	0.011	0.042	0.021	0.020	0.039
Queen Scallop	2	52	2	3.8%	0.010	0.011	0.011	0.011	0.011
Tuatua	5	28	0						
Pipi	2	19	0						
Cockle	3	17	0						
Oyster	5	9	0						
Abalone	3	8	0						
Geoduck	3	5	0						
Total	144	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052

1 – Organism as identified in the LIMS database

4.1.10. Impact of PTX contribution to DSP regulation

In New Zealand and Europe the PTX-group is currently regulated as part of the DSP group. However, in this report the PTX-group has been excluded from the DSP group. To compare the impact of the inclusion of the PTX-group in regulatory monitoring the DSP concentration and DSP+PTX2 concentration were calculated for each sample. As PTX2 is the only PTX-group congener which is routinely monitored (apart from the non-regulated seco acids), PTX2 was used as a surrogate for the PTX-group. These results were then compared against the current regulatory limit (Figure 15).

Samples were grouped by either:

- DSP+PTX2 below the regulatory limit
- DSP above the regulatory limit
- DSP at or below regulatory limit, DSP+PTX2 above regulatory limit

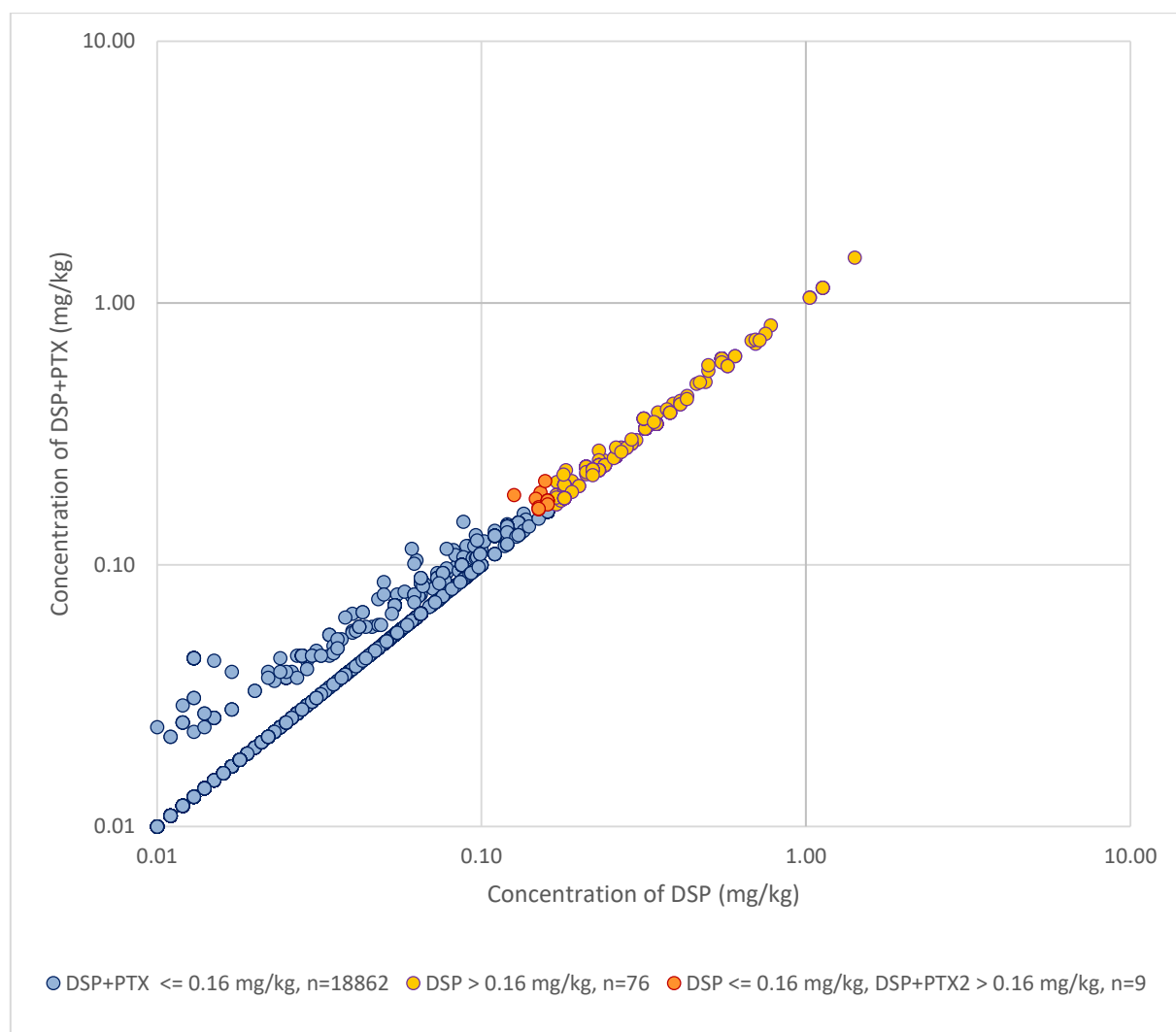


Figure 15. Comparison of PTX2 contribution to DSP regulation in New Zealand over the 2009-2019 period on a logarithmic scale

Of the 18947 samples analysed, a total of 85 samples were above the regulatory limit for DSP when PTX2 was included. Excluding PTX2 only 76 samples would have been considered above the DSP regulatory limit. As observed in Figure 16, two trend groups were observed in the samples, those samples where PTX2 was detected (orange), and samples where PTX2 was not detected (blue). Samples where only DSP is detected are either due to PTX2 being below the detection limit, or blooms from dinoflagellate species that do not produce PTX-group analogues, such as *Prorocentrum* spp..

Where PTX2 was detected there was a relatively higher contribution of PTX2 at lower concentrations of DSP. There is a reasonable explanation for this based on the known metabolism of PTX2 to PTX2SA in New Zealand shellfish. As the PTX2 is accumulated by the shellfish relatively more PTX2 would be metabolised to PTX2SA over time resulting in relatively lower PTX2 concentrations compared to DSP as the bloom progresses and DSP accumulates.

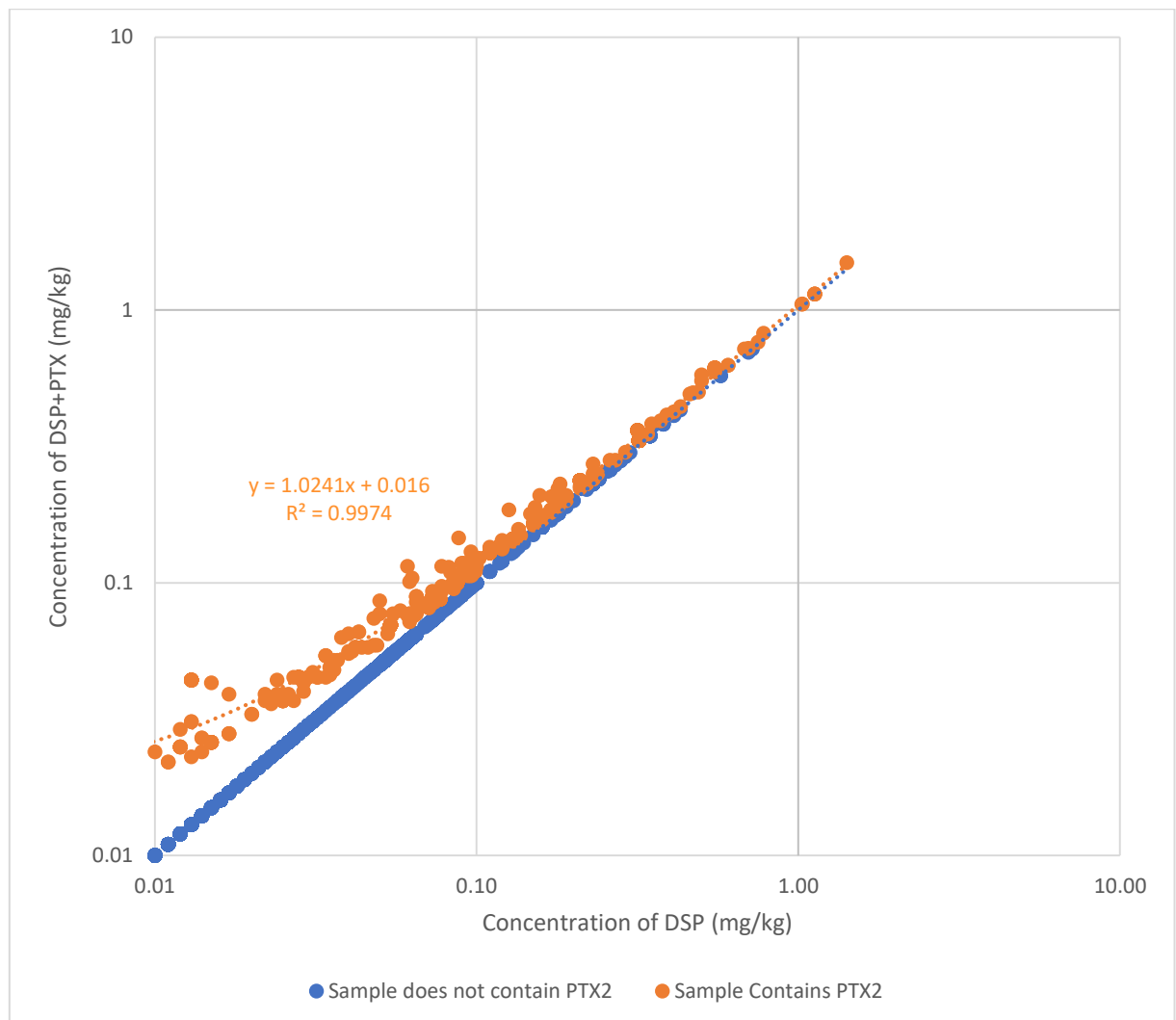


Figure 16. Comparison of DSP with PTX2+DSP in New Zealand over the 2009-2019 period on a logarithmic scale

Of the nine samples in the 2009-2019 period that were pushed above the regulatory limit (maximum permissible value) by the PTX2 concentration, three of the DSP concentrations were at the regulatory limit (0.16 mg/kg), five were at 0.15 mg/kg, and one at 0.13 mg/kg. There were 75 samples that contained reportable levels of PTX2 and no reportable DSP, with 90% of these samples analysed prior to July 2015 when the limit of reporting for the DSP analogues was 0.05 mg/kg rather than 0.01 mg/kg.

4.1.11. Comparison of PTX2, PTX2SAs and DSP concentrations in shellfish

PTX2 concentrations were plotted against DSP concentrations in all samples (Figure 17). Of the 18947 samples analysed, 176 contained both DSP and PTX2, 615 contained DSP although no PTX2, 75 contained PTX2 although no DSP, and 18081 contained neither DSP nor PTX2.

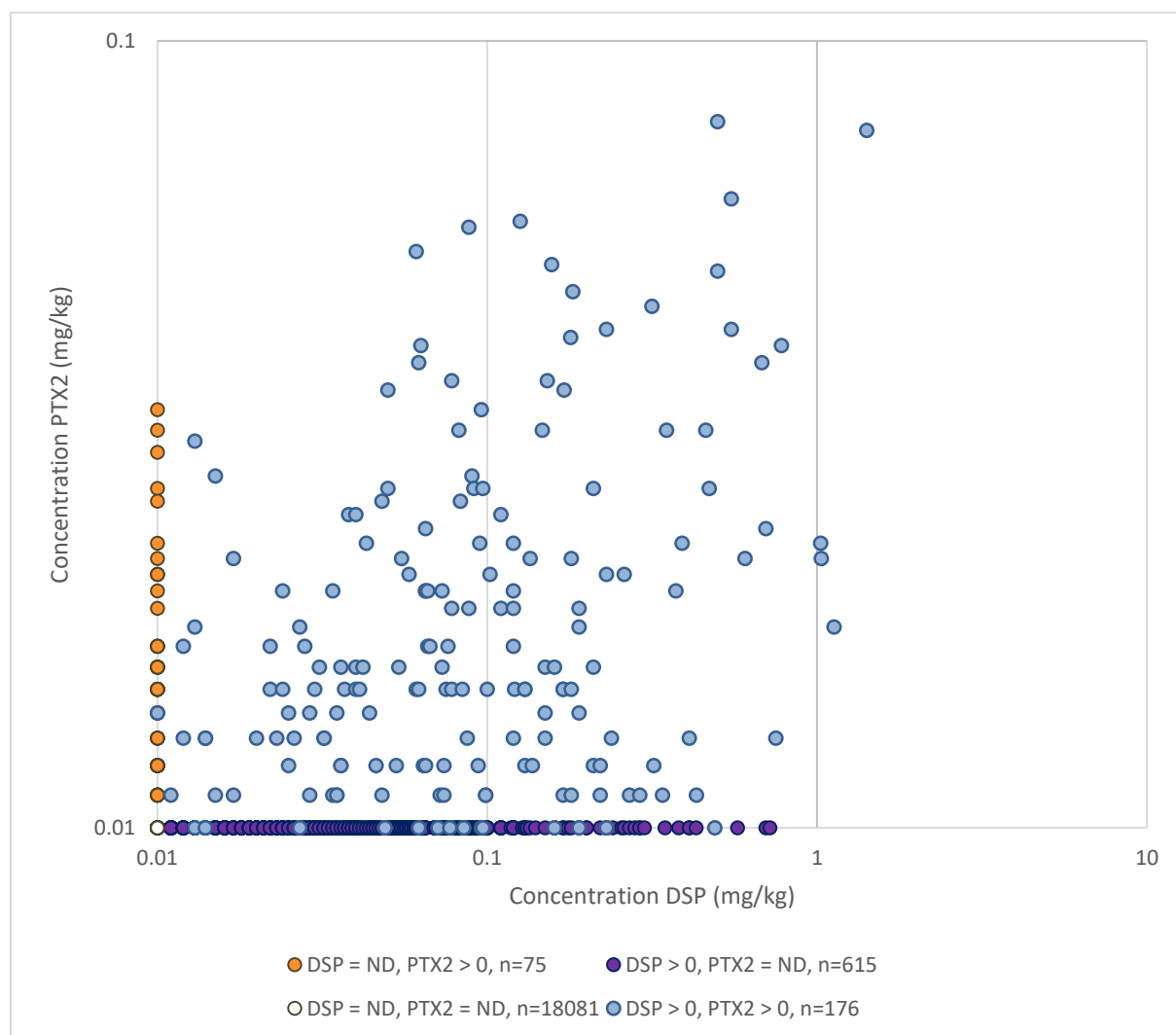


Figure 17. Comparison of PTX2 with Total DSP in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.

The PTX2 concentrations plotted against the DSP concentrations by species in New Zealand over the 2009-2019 period is shown in Appendix Figure D-7.

The PTX2SAs concentrations were plotted against the DSP concentrations in the samples (Figure 18). Of the 18947 samples analysed, 681 had both PTX2SAs and DSP detected, 3042 contained PTX2SAs although no DSP, 110 contained DSP although no PTX2SAs and 15114 contained neither PTX2SAs nor DSP.

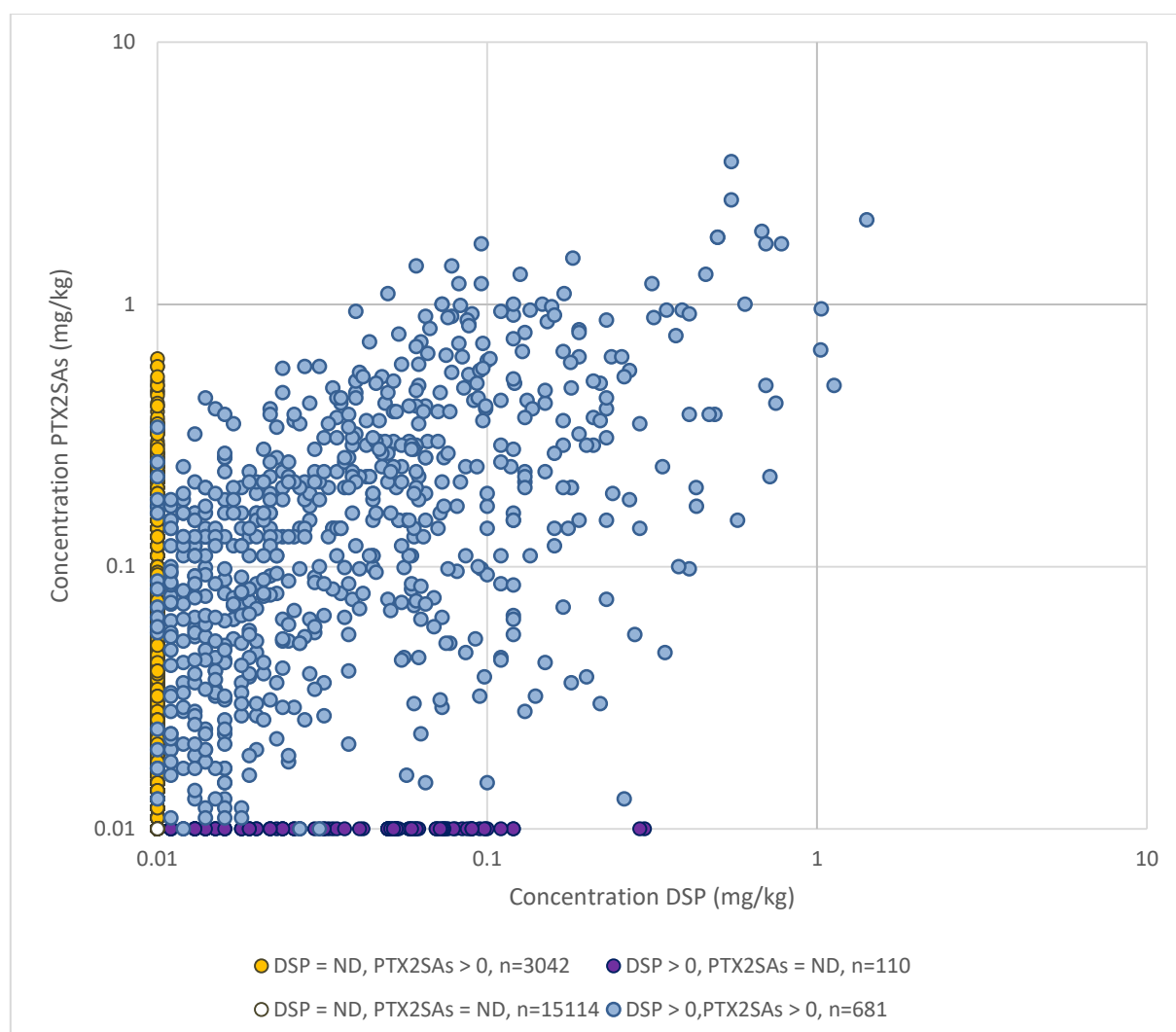


Figure 18. Comparison of PTX2SAs with Total DSP in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.

The PTX2SAs concentrations plotted against the DSP concentrations by species in New Zealand over the 2009-2019 period is shown in Appendix Figure D-8.

The PTX2 concentrations were plotted against the PTX2SAs concentrations in the samples (Figure 19). Of the 18947 samples analysed, 250 had both PTX2 and PTX2SAs detected, 3473 contained PTX2SAs although no PTX2, 1 contained PTX2 although no PTX2SAs and 15223 contained neither PTX2 nor PTX2SAs.

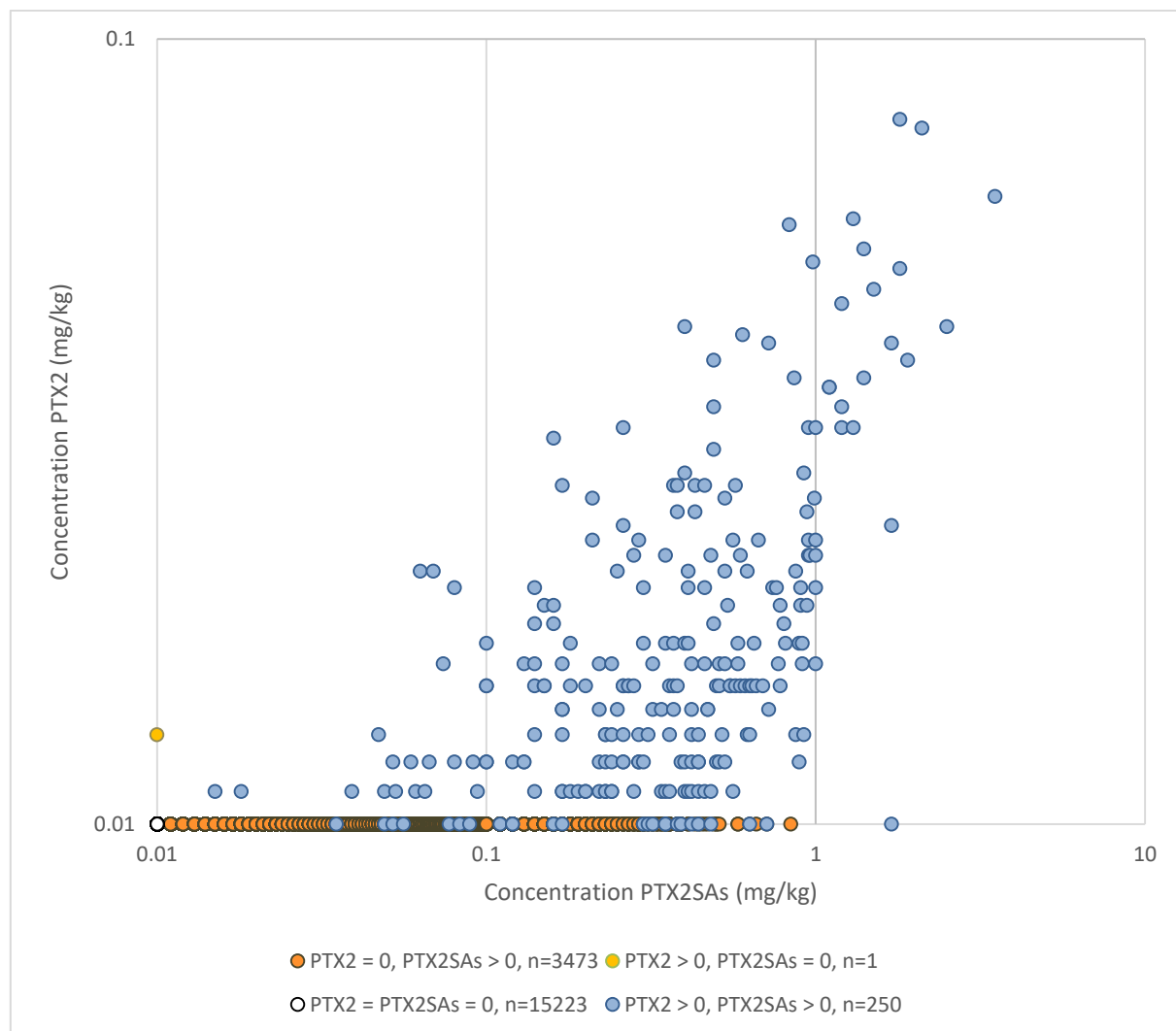


Figure 19. Comparison of PTX2 with PTX2SAs in New Zealand over the 2009-2019 period. Note: Due to the logarithmic scale non-detect results were displayed at 0.01 mg/kg. ND = not detected.

The PTX2 concentrations plotted against the PTX2SAs concentrations by species in New Zealand over the 2009-2019 period is shown in Appendix Figure D-9.

4.1.12. Pectenotoxin profiles

Samples from five bloom events as classified in section 4.1.6 (C|201507-12, I|bpk|201607-201703, I|bpk|200904-201005, B|201509-12 and A|boi|201506-12) were reprocessed to quantify PTX1, PTX11 and PTX6 which are acquired in the LC-MS/MS method of analysis although not processed as part of the routine monitoring programme. As reference materials are unavailable for these compounds, they were unable to be directly quantified, and were instead semi-quantified using PTX2 as a reference standard with an assumed relative response factor of 1 (Holland *et al.*, 2003). A retention time quality control extract was used which contained PTX2, PTX2SA, 7-epi-PTX2SA, PTX1, PTX11 and PTX6.

No detections of PTX1, PTX11 or PTX6 were observed above the 0.01 mg/kg reporting limit in any of the samples reprocessed in these bloom events. Trace detections were observed for PTX1 and PTX11 in some samples, and no detectable PTX6 was observed in any samples. As only trace detections were observed, PTX profiles were assessed including all trace detections including those below the quantitation and reporting limits.

A comprehensive summary of the numbers of samples, number of detections, percent detections, mean, 97.5th percentile (PCTL) and max concentrations for PTX2, PTX1, PTX11 or PTX6 for each site and species in the bloom events are summarised in Appendix F.

Three blooms containing the highest levels of PTX2 (C|201507-12, I|bpk|201607-201703 and I|bpk|200904-201005) were reprocessed to include PTX1, PTX11 and PTX6 to assess PTX profiles in New Zealand shellfish.

Bloom event C|201507-12 contained the third highest concentration of PTX2, although had substantially more samples (195) than I|bpk|201607-201703 (76) and I|bpk|200904-201005 (69) giving it the richest data set of these three blooms to examine for PTX profiles. PTX profiles excluding PTX2SAs based on the 97.5th percentile concentrations for the bloom event C|201507-12 are shown for Greenshell mussels and Pacific oysters in Figure 20. PTX profiles including PTX2SAs based on the 97.5th percentile concentrations for the bloom event C|201507-12 are shown for Greenshell mussels, Pacific oysters and Scallops in Figure 21.

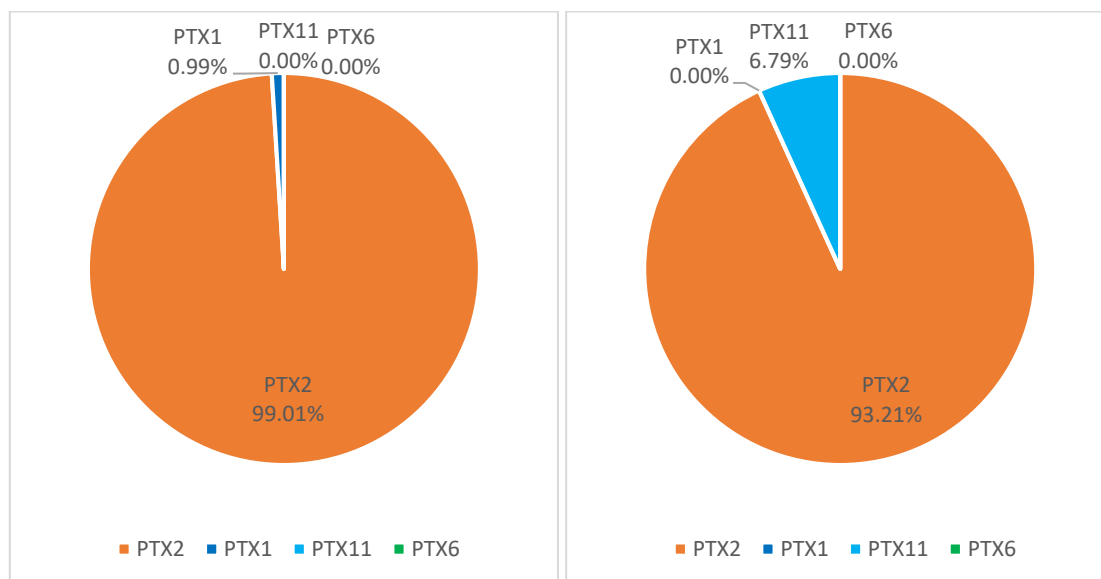


Figure 20. PTX profile for Greenshell mussels (left), and Pacific oyster (right) based on the 97.5th percentile concentrations of the PTX analogues excluding PTX2SAs in the bloom event C|201507-12

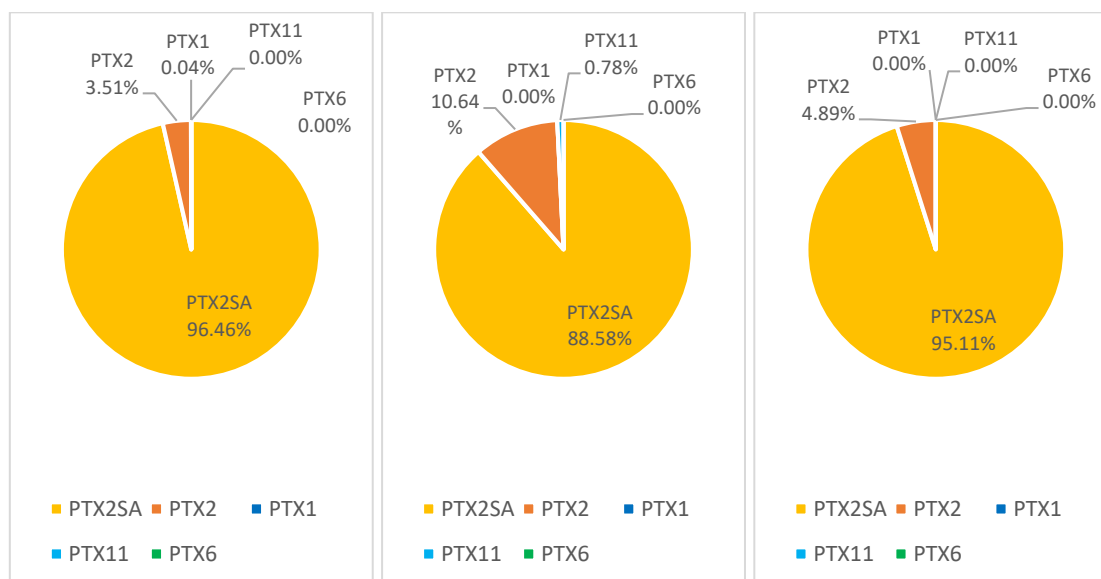


Figure 21. PTX profile for Greenshell mussels (left), Pacific oyster (middle), and Scallops (right) based on the 97.5th percentile concentrations of the PTX analogues including PTX2SAs in the bloom event C|201507-12

Greenshell mussels showed a trace detection of PTX1 with an average of 1.1% of the concentration of PTX2. The PTX1 detections showed a similar accumulation and depuration trend to PTX2. PTX1 and PTX6 have previously been reported to form via metabolism in Japanese Scallops (*M. yessoensis*) (Yasumoto *et al.*, 1989; Suzuki *et al.*, 1998). However, due to the very low abundance of PTX1 observed in the Greenshell mussels it is unlikely to be from metabolic conversion from PTX2, in contrast to that observed in *M. yessoensis*. Therefore, the detection of trace PTX1 in the Greenshell mussels suggests that either only a trace of PTX1 is produced within

the algae, or that most of it is converted either via enzymatic metabolism within the mussels, or by chemical means. Ring-opened seco acids of PTX1 are not monitored in this method.

Pacific oysters showed a trace detection of PTX11 with an average of 13.1% of the concentration of PTX2 (Figure 22). To illustrate the minor contribution of the other PTX analogues they are displayed as a ratio to PTX2 which is the routinely monitored congener. As there were only three Pacific oyster samples analysed during the bloom, there was not enough information to observe an accumulation and depuration trend. As PTX11 is observed only in the Pacific oyster samples, it suggests that this congener is produced via metabolism within this shellfish species. The site of oxidation in PTX11 is in close proximity to the lactone and has been reported to prevent enzymatic hydrolysis to form ring-opened seco acids (MacKenzie *et al.*, 2012). As PTX11 does not enzymatically metabolise to seco acids in the manner of PTX2, this could explain the relatively higher abundance of PTX11 in the Pacific oysters. The Pacific oysters also showed much lower abundance of PTX2SAs, this could be due to different binding of the compounds within the flesh, a weaker metabolism of PTX2 than in Greenshell mussels, or due to competing metabolism to form other congeners such as PTX11.

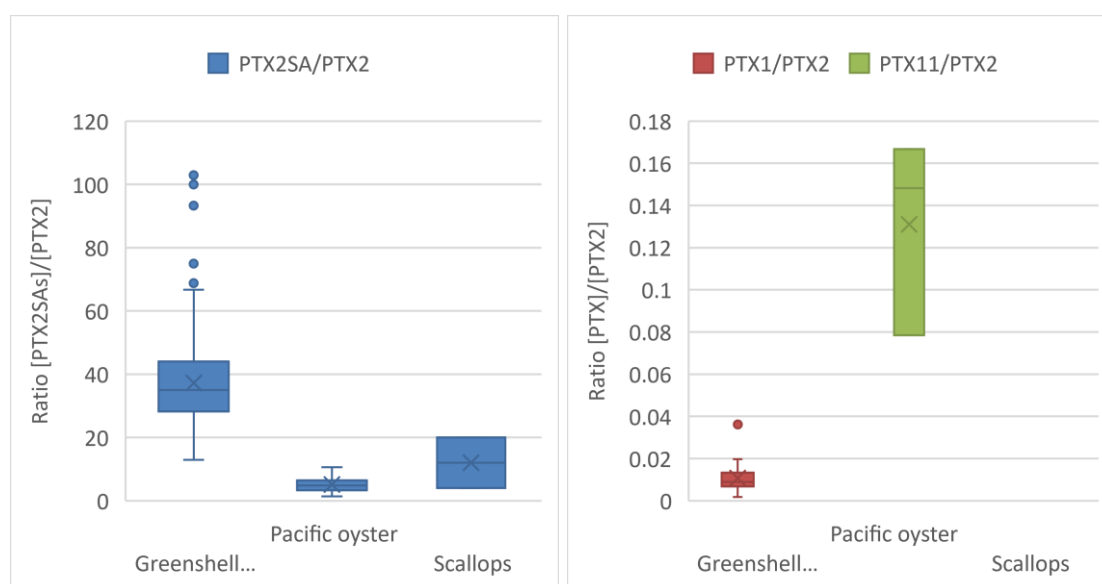


Figure 22. Ratio of PTX-group analogues compared to PTX2 in different shellfish species during bloom event C|201507-12. Left: PTX2SAs, Right: PTX1 and PTX11.

Of the 76 samples analysed in bloom event I|bpk|201607-201703 only 4 samples (5%) contained detectable PTX1, and no detections were observed for PTX11. Bloom event I|bpk|200904-201005 was analysed on an older generation, less sensitive LC-MS/MS instrument, and as a result no detections of any trace PTX1 or 11 were able to be observed in those samples. Due to the limited value of the data obtained from these two bloom events, two additional blooms (B|201509-12 and A|boi|201506-12)

were selected for reprocessing, targeting blooms which contained potential shellfish species of interest.

Bloom event B|201509-12 was selected for reprocessing as most of the samples present within the bloom were New Zealand scallops. Japanese scallops, *M. yessoensis*, have been reported to have a very different metabolism to New Zealand scallops, with oxidation to PTX1, PTX3 and PTX6 (Yasumoto *et al.*, 1989; Suzuki *et al.*, 1998). In the New Zealand scallops, PTX2SAs was observed but PTX1 and PTX6 were not detected, confirming that the species of scallops analysed in the B201509-12 bloom are dissimilar to the Japanese scallops.

Bloom event A|201506-12 was selected for reprocessing as most of the samples present within the bloom were Pacific oyster (Appendix Figure F-13). As with bloom event C|201507-12, the Pacific oysters showed traces of the peak assigned as PTX11, with 82% having detectable PTX11. The observation of PTX11 in the Pacific oysters did not follow the same accumulation/depuration as PTX2. The reason for this is unknown. PTX11 was observed at up to 3-times higher concentration than PTX2 (Figure 23), although it only reached a maximum of approximately 0.0010 mg/kg, one tenth of the limit of quantitation and reporting. It is unclear why PTX11 is observed preferentially in Pacific oysters, or why relatively higher concentrations were observed at the beginning of the bloom event prior to PTX2 accumulation. One possible explanation is that there is a matrix interference in Pacific oyster that interferes with the identification and quantitation of PTX11.

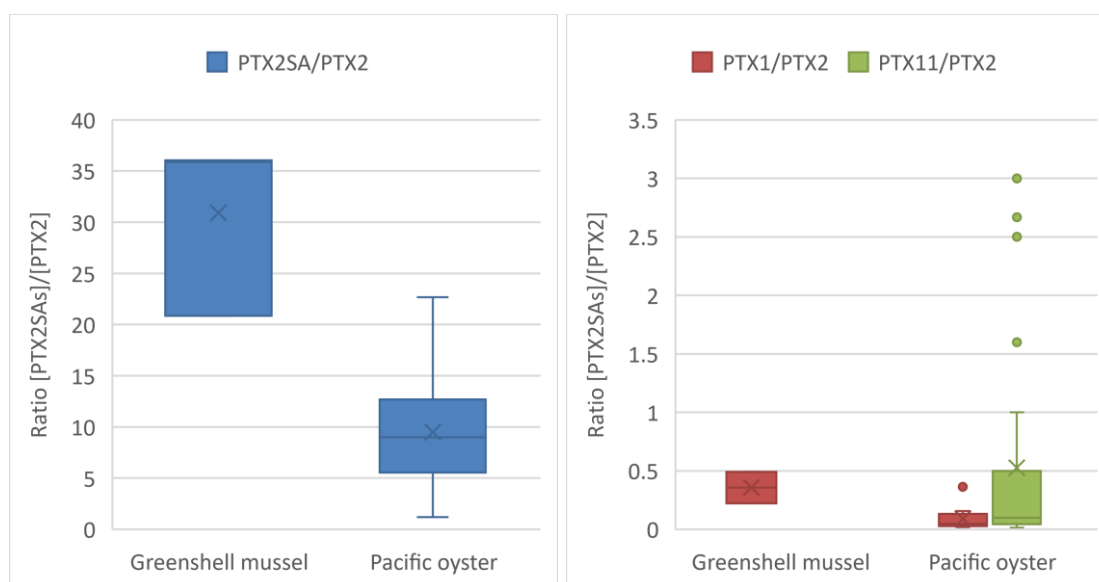


Figure 23. Ratio of PTX-group analogues compared to PTX2 in different shellfish species during bloom event A|201506-12. Left: PTX2SAs, Right: PTX1 and PTX11.

In order to confirm the occurrence of PTX11 in oysters, a Pacific oyster sample from site A015 sampled on 26/09/19 was taken during a bloom event and analysed in

conjunction with an authentic sample of PTX11 that was obtained from Cawthron Natural Compounds (New Zealand).

After product ion scans on the authentic PTX11 material, five multiple reaction monitoring (MRM) transitions were configured and used to compare the authentic PTX11 to the PTX11 observed in the Pacific oyster. The relative ratios of all five MRM transitions were in good agreement supporting that the trace of PTX11 observed in Pacific oysters is genuine (Figure 24).

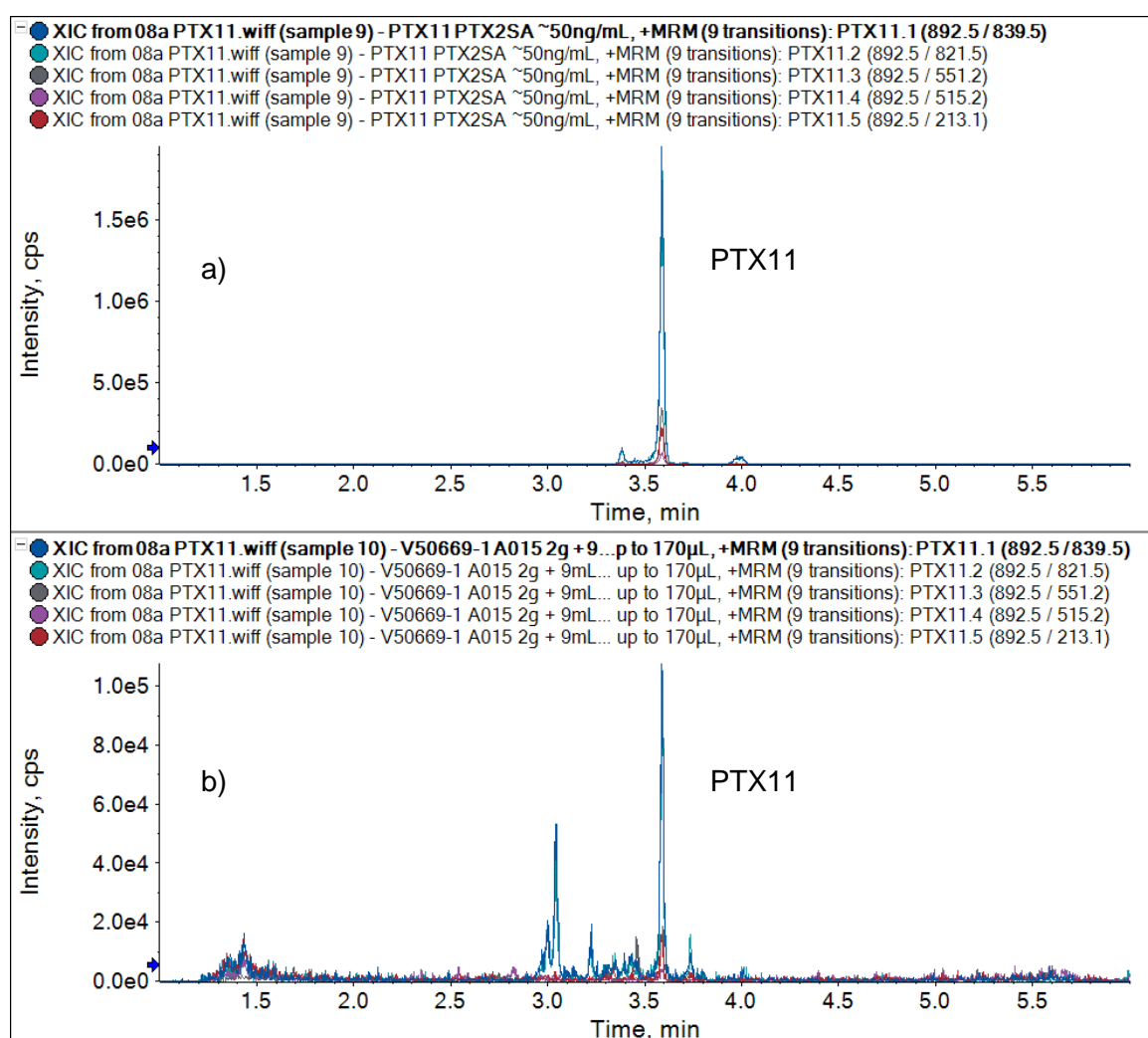


Figure 24. Comparison of chromatogram of PTX11 acquired with 5 MRM transitions in a) authentic PTX11 reference material, and b) concentrated Pacific oyster extract from site A015

In all shellfish, PTX2SAs were the most dominant analogues. PTX2SAs are non-regulated metabolites observed within many shellfish species. All species analysed for toxin profiles had proportionately high levels of these seco acids, suggesting they were all able to metabolise PTX2 in this manner. This metabolism may be part of the reason why PTX2 concentrations are much lower in New Zealand compared to that

observed in overseas studies, despite a similar prevalence of algal PTX2 producing species.

4.1.13. Comparison of PTX2 concentrations in New Zealand with Europe

In the EFSA review of pectenotoxins (EFSA, 2009a), a total of 487 samples from Italy, Norway, Spain and the United Kingdom were tested with both the DSP mouse bioassay and LC-MS/MS in order to assess the exposure risk of pectenotoxins to European consumers. Samples that were positive by the DSP mouse bioassay were considered by the CONTAM panel to be non-accessible to consumers when assessing the upper limit of PTX2 with dietary exposure and excluded them from the risk assessment. PTX2 was the only PTX-group analogue monitored in the European study.

A total of 18947 samples were assessed in this study in New Zealand over the 2009-2019 period. As only 389 of the 18947 samples were reprocessed to obtain results for PTX1, PTX11 and PTX6, there were insufficient data to assess the exposure including these congeners. These 389 samples reprocessed for PTX1, PTX11 and PTX6 represented the blooms of the highest concentrations of PTX2, and there were no results for these congeners above the reporting limit of 0.01 mg/kg. Therefore, it is not expected that excluding PTX1, PTX11 and PTX6 from the assessment would have a significant impact. As PTX-group analogues are produced by organisms which also produce DSP, samples above the regulatory limit for DSP would not be expected to be available to consumers. Results were assessed separately for samples with DSP above the regulatory limit (n=76), and for samples at or below the regulatory limit (n=18871). The reporting limit for the LC-MS/MS method used for the analysis of the samples was 0.01 mg/kg. Most of the samples were below this threshold and were not reported. A bounding approach was applied where the mean, median and 97.5th percentile was calculated both for the data set with non-reported results assigned a maximum value and for the data set with non-reported results assigned their minimum value. To assess the lower bound (LB), all values below the reporting limit were assigned 0, the minimum possible value. To assess the upper bound (UB), all values in the data set that were below the reporting limit were assigned 0.01, the maximum possible value of a non-reportable result. The mean, median and 97.5th percentile was calculated from both the lower bound and upper bound datasets (Table 9).

Table 9. Concentrations of PTX2 measured by LC-MS/MS in shellfish samples grouped by DSP results in New Zealand over the 2009-2019 period (mg/kg)

DSP result	N	Mean (LB/UB)	Median (LB/UB)	97.5 PCTL (LB/UB)	Maximum	% NQ	% > 0.16 mg PTX2/kg
> 0.16	76	0.0120/0.0120	0.0164/0.0198	0.0772/0.0772	0.0790	34.2	0
≤ 0.16	18871	0.0002/0.0101	0.0000/0.0100	0.0000/0.0100	0.0590	98.9	0

N = Number of samples, 97.5 PCTL = 97.5th percentile, UB = Upper bound, LB = Lower bound
LB is calculated by substituting results below reporting limit with 0, and UB is calculated by substituting results below the reporting limit with the reporting limit.

Due to the low level of contamination and the large amount of non-detect results, mean, median and 97.5th percentile results are influenced by the choice of upper or lower bound approach.

%NQ – Percentage of samples with result below the reporting limit

Most samples did not contain any reportable PTX2, as a result, the mean, median and 97.5th percentile for samples where DSP was at or below the regulatory limit was the reporting limit of the method. No samples exceeded the regulatory limit of 0.16 mg PTX2/kg. In contrast in the European study (EFSA, 2009a), 0.6% of the mouse bioassay negative samples and 4.9% of the mouse bioassay positive samples exceeded 0.16 mg PTX2/kg (Table 10). The highest detection was 0.079 mg/kg, and the highest detection where the sample was not above the DSP (excluding PTX-group) regulatory limit was 0.059 mg/kg. This maximum result is similar to the 95th percentile of 0.079 mg/kg determined by EFSA for samples which tested negative by the DSP mouse bioassay, although significantly lower than the maximum levels of PTX2 reported in Europe, 0.418 mg/kg where the mouse bioassay was positive, and 0.183 mg/kg where the mouse bioassay was negative (EFSA, 2009a).

Table 10. Concentrations of PTX2 measured by LC-MS/MS in samples that were also analysed by the DSP mouse bioassay (EFSA, 2009a).

Mouse Bioassay	N	Mean (LB/UB)	Median (LB/UB)	95 PCTL (LB/UB)	Maximum	% NQ	% > 0.16 mg PTX2/kg
Positive	164	0.000/0.039	0.032/0.042	0.130	0.418	50.6	4.9
Negative	323	0.000/0.002	0.014/0.016	0.079	0.183	62.2	0.6

N = Number of samples, 95 PCTL = 95th percentile, UB = Upper bound, LB = Lower bound

LB is calculated by substituting results below reporting limit with 0, and UB is calculated by substituting results below reporting limit with the reporting limit.

Due to the low level of contamination and the large amount of non-detect results, mean, median and 97.5th percentile results are influenced by the choice of upper or lower bound approach.

%NQ – Percentage of samples with result below the reporting limit

4.1.14. Comparison of DSP concentrations in New Zealand with Europe

In the EFSA review of okadaic acid and its analogues (EFSA, 2008), a total of 1210 samples from Ireland, United Kingdom, France and the Netherlands were tested with both the mouse bioassay and LC-MS/MS in order to assess the exposure risk of DSP to European consumers. Samples that were positive by the DSP mouse bioassay were considered by the CONTAM panel to be non-accessible to consumers when assessing the upper limit of DSP with dietary exposure and excluded them from the risk assessment.

A total of 18947 samples were assessed in this study in New Zealand over the 2009-2019 period. The reporting limit for total okadaic acid, and total dinophysistoxins 1 and 2 was 0.05 mg/kg until July 2015, and since then has been 0.01 mg/kg. Most of the samples were below this threshold and were not reported (Table 11).

Table 11. Concentrations of DSP measured by LC-MS/MS in shellfish samples in New Zealand over the 2009-2019 period (mg/kg)

N	Mean	Median	97.5 PCTL	Maximum	% NQ	% > 0.16 mg OA eq/kg
18947	0.003	0.000	0.023	1.415	95.8	0.40

A total of 76 samples (0.40%) exceeded the regulatory limit of 0.16 mg OA eq/kg. In contrast in the European study (EFSA, 2008), 425 out of the 1210 samples (35%) were above the regulatory limit of 0.16 mg OA eq/kg. Of these samples 100 (13%) of the mouse bioassay negative samples and 325 (71%) of the mouse bioassay positive samples exceeded 0.16 mg OA eq/kg (Table 12). The highest detection in New Zealand was 1.4 mg/kg. This maximum result is lower than the maximum observed by EFSA for samples which tested negative by the DSP mouse bioassay, and substantially lower than the maximum observed by EFSA.

Table 12. Concentrations of DSP measured by LC-MS/MS in samples comparatively tested with the DSP mouse bioassay (EFSA, 2008).

Mouse Bioassay	N	Mean	Median	97.5 PCTL	Maximum	% NQ	% > 0.16 mg OA eq/kg
Negative	755	0.022	0.066	0.240	2.240	44	13
Positive	455	0.240	0.486	1.810	8.864	11	71

4.2. New Zealand Bivalve Consumption

In order to assess the risk associated with consuming contaminated shellfish, data on meal sizes are required. Unfortunately, most consumption surveys are targeted to obtain data on consumption over time, which is best suited to chronic toxicity risk assessments. Because consumption surveys are often summarised as “average amount of a food consumed over the survey period” it is usually impossible to discern the frequency and amount per serving. Knowing only the average amount consumed (e.g. 50 g/day) does not provide information whether a consumer eats consistent portions daily throughout the week, or whether larger portions (e.g. 175 g/meal) are consumed on average a couple of times per week.

In the EFSA review a probabilistic estimate of dietary exposure to PTX2 was performed using both the occurrence data and consumption data (EFSA, 2009a). As insufficient information was available for the distribution of portion sizes, the CONTAM Panel decided to use a triangular distribution as a simple and pragmatic approach. The distribution was characterised by three values, the minimum, the most probable and the maximum. 0 g was used as the minimum, 100 g was used as the “most probable” value (although it was noted that there was no evidence that it is the most frequently consumed portion size), and the large portion size of 400 g was used to represent the maximum. The 400 g large portion size represents the 95th percentile in Germany and the Netherlands (EFSA, 2009a). This value is in the higher end of the range of 95th percentile reported by the EU members states and is therefore likely to

cover a higher percentile for the entire EU. This is also in good agreement with the risk assessment from the joint FAO/IOC/WHO expert consultation where three values were chosen, 100 g which is the standard portion size, 250 g which covers the 97.5th percentile of the consumers from most countries for which data were available, and 380 g which was the highest 97.5th percentile of the reported countries portion size for consumers, reported by the Netherlands (FAO/IOC/WHO, 2004). This upper value was conservative as the 97.5th percentile for shellfish portion sizes for adults was 133 g in Japan, 181 g in Australia, 225 g in the USA, and 263 g in New Zealand.

Because of the large seasonal variations, the frequency of consumption and the number of consumers should be determined on a one-year basis (FAO/IOC/WHO, 2004). Within the whole population, 35% consume bivalve molluscs both in Norway, and France. In Norway, 33% consume bivalve molluscs between 1 and 11 times per year, and 2% consumer between 1 and 8 times per month. With shorter surveys the number of consumers was 11% in France (7 days), 8% in Italy (7 days), 4% in the US (2 days), 3% in New Zealand (1 day) and 2% in Australia (1 day) (FAO/IOC/WHO, 2004).

In a 1999 survey entitled NZ Food: NZ People (Russell *et al.*, 1999) from a qualitative food frequency questionnaire of 4576 respondents aged 15 or over, 6% of the respondents reported that they consumed shellfish at least once per week. A higher portion of NZ Māori and Pacific people reported consuming shellfish at least once per week, with 15% males and 14% females of NZ Māori reported consuming shellfish at least once per week, and 26% males and 31% females of Pacific people reported consuming shellfish at least once per week. In the 2002 survey entitled NZ Food: NZ Children (Parnell *et al.*, 2003), of 3275 New Zealand children aged 5-14, 9% reported that they consumed shellfish at least once per week. Similar to the adult survey, a higher proportion of Māori and Pacific people reported consuming shellfish at least once per week, and 15% males and 18% females of NZ Māori reported consuming shellfish at least once per week, and 26% males and 23% females of Pacific people reported consuming shellfish at least once per week. From these surveys no portion size information was able to be obtained and it is unclear what portion of this shellfish is relevant. This is because shellfish in the survey included other non-bivalve seafood (e.g. crab meat) which are not expected to accumulate the natural toxins commonly attributed to bivalve molluscs.

In the 2008/09 New Zealand Adult Nutrition Survey (Parnell *et al.*, 2011), carried out from October 2008 to October 2009, a 24-hour recall of 4721 adults aged 15+, including 1040 Maori and 757 Pacific peoples was used (Table 13). It was not stated if people consumed more than one type of the seafood listed so a total mollusc consumption cannot be determined. It was also not clear how the average portion size was determined.

Table 13. Summary of average and 97.5th percentile portion sizes from the 2008 Adult Nutrition Survey (Parnell *et al.*, 2011)

Commodity	Number of respondents consumed	Average portion size (g).	97.5 th percentile portion size (g)
Abalone (Paua)	3	114	268
Mussels	65	82	256
Oysters	234	6	94
Scallops	9	51	91
Tuatua	1	240	240

In a risk assessment of Ciguatoxins in seafood in New Zealand (Cressey *et al.*, 2019), information on food consumption in New Zealand from the previous New Zealand consumption surveys were reviewed and is summarised in **Error! Reference source not found.** (Russell *et al.*, 1999; Parnell *et al.*, 2003; Parnell *et al.*, 2011). A lower consumption of molluscs was observed for children (5-14 years) compared to adults (15+ years), however it is unclear on consumption frequency as well as how the portion sizes would compare, and more importantly how portion size relative to consumer body weight would compare between children and adults.

Table 14. Summary of marine food consumption by New Zealand children (5-14 years) and adults (15+ years) (Cressey *et al.*, 2019)

Metric	Children (5-14 years)	Adults (15+)	
	2002	1997	2009
	<i>Finfish</i>		
Consumers (% of total respondents)	14.9	18.0	20.3
Consumer mean (g/person/day)	89	99	134
Population mean (g/person/day)	13.3	17.8	27.2
	<i>Crustaceans</i>		
Consumers (% of total respondents)	0.8	0.9	1.2
Consumer mean (g/person/day)	78	130	82
Population mean (g/person/day)	0.6	1.2	1.0
	<i>Shellfish (Molluscs)</i>		
Consumers (% of total respondents)	0.5	2.4	1.5
Consumer mean (g/person/day)	49	106	85
Population mean (g/person/day)	0.5	2.5	1.2

In a review of New Zealand bivalve availability to consumers in 2011 (King and Lake, 2013), it was estimated that bivalve consumption was 8 g/person/day for the total New

Zealand population or 407 g/person/day for shellfish consumers. This does not take account of factors such as weight loss from cooking or wastage during preparation.

In a 2011-2012 Australian National Nutrition and Physical Activity Survey (Australian Bureau of Statistics, 2014; Williamstown Contamination Expert Panel, 2015), which used a 24-hour recall of 12153 people aged 2 years and over, 76 respondents reported that they consumed molluscs. The mean daily consumption for all respondents was 0.5 g/day, and 79 g/day for consumers only. The 50th, 90th, 95th and 97th percentiles for consumers only was 63, 146, 180, and 248 g/day respectively.

While insufficient data are available to create a robust meal size distribution for risk modelling, an approximation can be made using simulations such as using a triangular distribution as was performed by EFSA (EFSA, 2009a). This simulation performed by EFSA was applied to a 'standard' 60 kg adult. As children are typically of smaller size, they would be at greater risk of exposure from consuming the same amount of a contaminant. The consumption summary from Cressey *et al.* (2019) indicates that children typically consume less molluscs than adults. However, it is unclear if the portion sizes consumed by children are relatively smaller or larger than the portion sizes consumed by adults when compared to body weight.

5. RISK CHARACTERISATION

5.1. Pectenotoxin group

5.1.1. Deterministic estimate of dietary exposure to PTX2

Based on the assumption that products above the regulatory limit for DSP (0.16 mg OA eq/kg), excluding the PTX-group (Table 9) do not reach the market, the dietary exposure can be estimated as in Table 15. Three portion sizes were used to assess the exposure of PTX2: 100 g, the standard portion size; 268 g, the highest 97.5th percentile portion size of shellfish species by New Zealand consumers; and 400 g, the large portion size adopted by EFSA for risk assessment. The upper bound dataset was used for the 97.5th percentile concentration (Table 9).

Table 15. Deterministic intake of PTX2 based on samples at or below the regulatory limit for DSP (excluding the PTX-group)

	Units	97.5 th Percentile	Maximum
Concentration PTX2	mg PTX2/kg	0.01	0.059
Exposure by eating 100 g	µg PTX2/person	1.0	5.9
	µg PTX2/kg bw	0.02	0.10
Exposure by eating 268 g	µg PTX2/person	2.7	15.8
	µg PTX2/kg bw	0.04	0.26
Exposure by eating 400 g	µg PTX2/person	4.0	23.6
	µg PTX2/kg bw	0.07	0.39

bw = body weight, based on 60 kg person.

The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated with the 97.5th percentile of occurrence in samples at or below the regulatory limit for DSP is 0.07 µg PTX2/kg bw. This is substantially lower than the acute reference dose (ARfD) proposed by EFSA (2009a) of 0.8 µg PTX2/kg bw. As the 97.5th percentile was represented by the limit of quantitation as 98.9% of the samples analysed were below the reporting limit, the maximum value was also used to assess the potential risk of PTX2. The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated with the maximum occurrence in samples at or below the regulatory limit for DSP is 0.39 µg PTX2/kg bw. This is less than half of the ARfD proposed by EFSA (2009a) of 0.8 µg PTX2/kg bw. A 60 kg person would have to consume approximately 814 g of shellfish at 0.059 mg PTX2/kg to reach the conservative ARfD proposed by EFSA.

The dietary exposure of PTX2 was also estimated for all samples, including those that were above the regulatory limit for DSP, despite these samples not expected to be accessible to consumers (Table 16).

Table 16. Deterministic intake of PTX2 based on all samples, including those above the regulatory limit for DSP

	Units	97.5 th Percentile	Maximum
Concentration PTX2	mg PTX2/kg	0.01	0.079
Exposure by eating 100 g	µg PTX2/person	1.0	7.9
	µg PTX2/kg bw	0.02	0.13
Exposure by eating 268 g	µg PTX2/person	2.7	21.2
	µg PTX2/kg bw	0.04	0.35
Exposure by eating 400 g	µg PTX2/person	4.0	31.6
	µg PTX2/kg bw	0.07	0.53

bw = body weight, based on 60kg person.

The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated with the maximum concentration of PTX2 observed from all samples over the 2009-2019 period is 0.53 µg PTX2/kg bw. This still represents less than the ARfD proposed by EFSA (2009a) of 0.8 µg PTX2/kg bw. A 60 kg person would have to consume approximately 608 g of shellfish at 0.079 mg PTX2/kg to reach the conservative ARfD proposed by EFSA.

As the portion sizes that have been used for this study are based on consumption for adults, they are not appropriate for assessing the exposure to children. Children are more susceptible to exposure of toxins as they typically weigh less than adults, however their portion sizes may also be smaller. Without appropriate data on portion sizes with respect to body weight it is not possible to evaluate the risk exposure to children.

5.1.2. Probabilistic estimate of dietary exposure to PTX2

5.1.2.1. Methodology

An excel spreadsheet containing PTX2 and DSP data, for New Zealand sites/zone and different bivalve species was loaded into the statistical software R 3.6.1. (R Core Team, 2019) for analysis and the risk characterisation simulation.

The mc2d package (version 0.1-18) for R was used in the development of the simulation and risk characterisation (Pouillot and Delignette-Muller, 2010).

A detailed summary of the exposure assessment and risk characterisation for PTX2 can be found in Appendix G.1.

5.1.2.2. Simulation model development

The main components of the exposure assessment and risk characterisation were the consumption amount of bivalve mollusc and the distribution of PTX2 concentrations in bivalve molluscs. A probabilistic estimate of dietary exposure to PTX2 was performed

by a Monte Carlo simulation to generate the amount of PTX2 consumed in a sitting (adjusted by kg body weight), using a total of 1,000,000 iterations, i.e. consumed shellfish meals. The focus of the risk assessment was on the acute exposure of the consumption of PTX2.

5.1.2.3. Consumption amount of shellfish

No New Zealand specific data on the consumption of bivalve shellfish could be obtained and various data sources have been compared and discussed in Section 4.2. Similarly, overseas data on consumption of bivalve shellfish for acute toxicity assessment were also insufficient. Consequently, the approach used by EFSA was replicated here. In the EFSA (2009a) review of the PTX-group, a triangular distribution was used for the portion sizes because insufficient information was available. This distribution was defined by the minimum value of 0 g; most likely value (mode) of 100 g; and maximum value of 400 g. The 400 g large portion is likely an over-estimate and hence the likely exposure to PTX2 would also likely be over-estimated.

5.1.2.4. Relationship between DSP and PTX2

The relationship between DSP and PTX2 (on the log-log scale) is shown in Figure 17. Similarly, Figure 25 was generated to allow best fit regression, although the concentrations were \log_{10} transformed rather than the axes scaled, and the least squares regression line was fitted to the \log_{10} transformed values. This \log_{10} transformation excluded results that were below the limit of reporting.

The regression model indicates that there is a statistically significant relationship between \log_{10} DSP and \log_{10} PTX2 (P -value < 0.001). However, while the relationship is statistically significant, the utility of using this relationship is minimal due to the small amount of variability that is explained by the model, i.e. $R^2=0.103$ or 10.3%. This is reflected in the large residual standard error of 0.1976. Consequently, using this model at the limit of detection for DSP (0.01 mg/kg) the prediction for PTX2 in shellfish is 0.013 mg/kg with an approximate 95% prediction interval of 0.005-0.032. At the upper range of DSP (1 mg/kg) the predicted PTX2 is 0.025 with an approximate 95% prediction interval of 0.010-0.063 mg/kg. Based on these large prediction intervals and high overlap, there is little to be gained from including a relationship between DSP and PTX2 when assessing the exposure of PTX2. Hence, results where DSP was above the regulatory limit were not excluded when simulating the exposure.

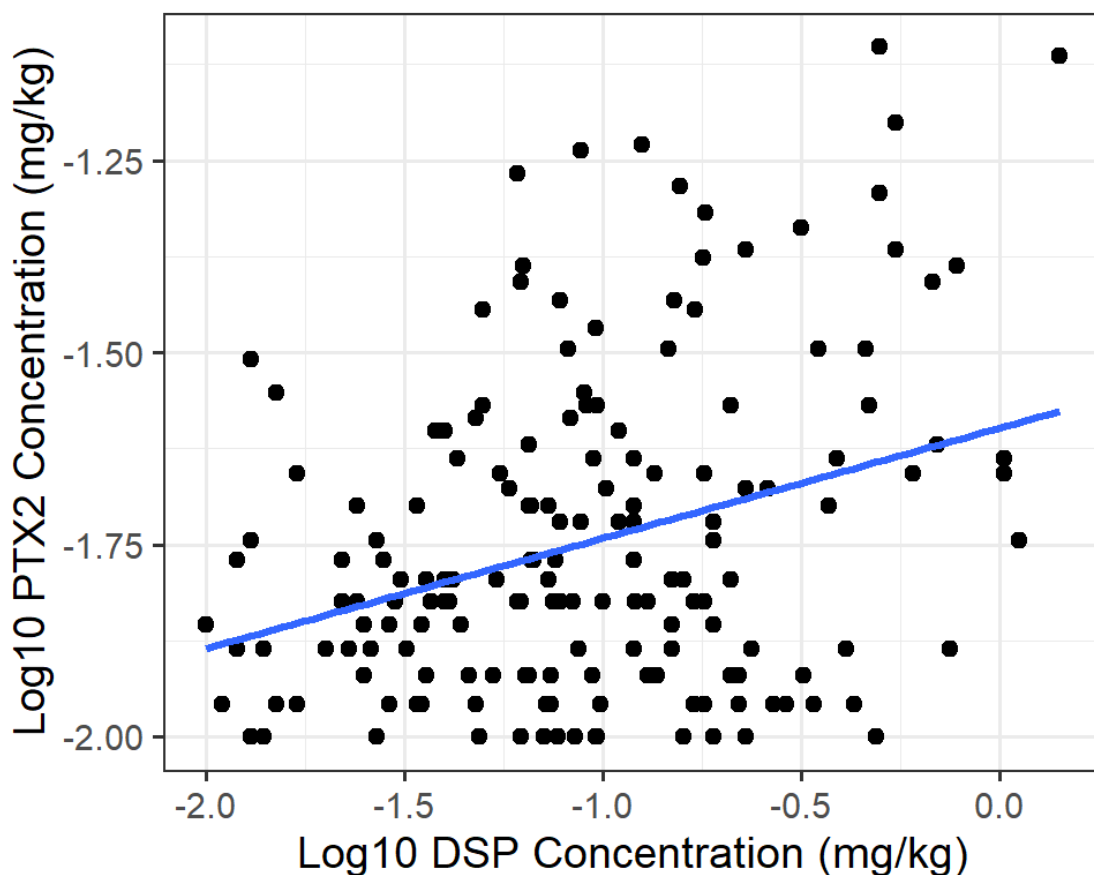


Figure 25. Scatter plot of the \log_{10} PTX2 concentration versus the \log_{10} DSP concentrations, showing only samples where both were detected; the blue line is the best fit regression line.

5.1.2.5. Distributions of PTX2

With respect to assessing exposure, and hence the potential risk, from consumption of bivalve shellfish containing PTX2 it is worthwhile to focus on time periods when exposure is likely to occur. This is especially pertinent since most test results in the data set are non-detections, many of which relate to times when algal blooms were not present. For example, there were only 251 (1.3%) detections of PTX2 in New Zealand shellfish from 18947 tests (Table 6). Hence, if random sampling would occur across all those test results, 98.7% of exposures would be zero (or very low if some non-detects were not true zeros).

Focusing on bloom events appears to provide a more focussed approach, especially in relation to potential risk management actions. Bloom events have been defined as detections of either PTX2, PTX2SA or DSP (Section 4.1.6), the exposure assessment is based on the subset of test results where these conditions are met.

There are two approaches that could be used for the simulation of exposure in relation to the distribution of PTX2 from which realisations are drawn. The first approach is to

fit a suitable parametric distribution to the observed PTX2 values, and hence this approach can result in realisations of PTX2 that were not directly observed. The second is to draw realisations from the empirical distribution of PTX2 concentration, and hence only concentrations that were actually observed can be sampled.

Irrespective of the approaches used, a difficulty arises due to the large number of non-detects in the data set, as only 1.32% of the 18947 test results had detectable levels of PTX2. While analysis methods exist to deal with non-detects, they assume that all non-detects have an actual, but unobserved, non-zero concentration of the analyte in the sample. This assumption is unlikely to be true, as it is more likely that some sample concentrations are non-zero (e.g. those in the early stages or late stages of a bloom), while most are true zeros. Consequently, the non-detect results could be modelled using a zero-inflated or hurdle approach, where a mixture of two distributions, a fixed zero value, which occurs with some probability p , and another non-zero, continuous distribution which occurs with a probability of $1 - p$. However, in the case of continuous distributions, such fitting functions are not readily available. It should be noted that this is unlikely to be problematic, as any values below the limit of detection (0.01 mg/kg) are unlikely to contribute substantially to the dietary exposure (see below).

EFSA (2009a) also included a binomial distribution model whether a serving contained PTX2, or not. This approach was further complicated by the assumption that not all non-detects were true zero PTX2 concentrations, but that 20% of these did contain PTX2 but below the limit of quantification. It should be noted that how non-detects, i.e. those below the limit of detection or quantification, are dealt within the probabilistic assessment is of limited value. If the PTX2 concentration is less than or equal to the LoD of 0.01 mg/kg, then this implies that a 60 kg consumer would have to consume 4.8 kg or more, of bivalve shellfish in order to reach the conservative EFSA ARfD. For this reason, the simplifying assumption is made that non-detects are assigned a PTX2 concentration of 0.01 mg/kg.

The results from the PTX2 detections were best fitted by a log-normal distribution (Appendix G.1.2.). The results have a very high mode (most probable value) at the very low detectable concentrations, higher than the best fitting distribution. This also affects the rest of the distribution over the range from about 0.013 to 0.023 mg PTX2/kg, where the parametric distribution 'over-predicts' the probabilities while for larger values it 'under-predicts' the probabilities with which PTX2 concentrations have been observed.

Two approaches were undertaken to estimate distributions of PTX2, during bloom events only, for the exposure modelling:

Model 1:

- Binomial distribution with probabilities of a detection/non-detection are equal to those in the bloom data set (i.e. 6.55% and 93.45% respectively).
- Detects are generated from a log-normal distribution (variables: meanlog = -4.098, sdlog = 0.445) that was the best fit to the detections, and this was left truncated at the limit of reporting of 0.01 mg/kg.
- Non-detects are assigned a PTX2 concentration of 0.01 mg/kg.

Model 2:

- Using the empirical distribution of PTX2 concentrations from the bloom data set
- Non-detects are assigned a PTX2 concentration of 0.01 mg/kg.

The mean, median, 95th percentile, 97.5th percentile, 99th percentile and maximum concentrations of simulated shellfish meals, consumed during bloom events, generated from the two model approaches are shown in Table 17. These clearly indicate that the choice of approach bears little on the results obtained – the biggest effect is on the maximum, because a great maximum PTX2 concentration can be obtained under the parametric model.

Table 17. Concentration of PTX2 simulated in shellfish meals using the two different model approaches (mg PTX2/kg).

Distribution	Mean	Median	95 th PCTL	97.5 th PCTL	99 th PCTL	Max
Model 1	0.0106	0.0100	0.0138	0.0201	0.0272	0.127
Model 2	0.0106	0.0100	0.0120	0.0170	0.0270	0.079

5.1.2.6. Estimating Exposure

The final step in this model is to combine the consumption and concentrations data to estimate the amount of PTX2 consumed in a single sitting, adjusting for the weight of an adult and converting the µg PTX2/kg bw. As there were not suitable portion size data to create an accurate distribution model to simulate intake portions, the triangular distribution used by EFSA for simulating consumption of an adult was used. The risk of exposure of contaminants to children is important to consider with risk assessments. However, as there are insufficient data to estimate the consumption of shellfish for a child, this risk characterisation is not able to estimate the exposure in children. As children typically have smaller body mass than adults they would be at greater risk from the same amount of exposure of harmful substances. On the contrary, it is also possible that children would consume smaller portion sizes reducing the amount of any contaminant they would be exposed to. It is unclear how these factors would balance out.

The output of the subsequent exposure to PTX2 for each of the two models for simulating PTX2 concentrations are shown in Table 18. The two models used for the concentration of PTX2 result in similar exposures, hence the approach used has little effect on the results. The 99th percentile was approximately one tenth of the conservative EFSA ARfD and the maximum exposure for a standard 60 kg adult was approximately half the conservative EFSA ARfD. The average mass of an adult male in New Zealand is 86.7 kg, and an average adult female is 73.3 kg (Pearson *et al.*, 2018). The larger body masses would result in an even lower dietary exposure.

Table 18. Simulated dietary exposure to PTX2 using the two different model approaches for a standard 60 kg adult ($\mu\text{g PTX2/kg bw}$).

Distribution	Mean	Median	95 th PCTL	97.5 th PCTL	99 th PCTL	Max
Model 1	0.0296	0.0266	0.0572	0.0630	0.0892	0.533
Model 2	0.0294	0.0265	0.0564	0.0616	0.0814	0.505

5.1.2.7. Risk Characterisation

The risk characterisation in the current context is a simple matter of comparing the exposure distributions to the corresponding Health Based Guidance Value, which for PTX2 is the conservative ARfD of 0.8 $\mu\text{g/kg bw}$ proposed by EFSA (2009a).

From the dietary exposure to PTX2 from bivalve shellfish calculated above, none of the 1,000,000 iterations resulted in an exposure exceeding the ARfD (based on the maximum). This is consistent with the absence of reported human illnesses.

5.2. Okadaic acid group

5.2.1. Deterministic estimate of dietary exposure to DSP

Based on the assumption that products above the regulatory limit for DSP, excluding the PTX-group (Table 9) do not reach the market, the dietary exposure can be estimated as in Table 19. Three portion sizes were used to assess the exposure of PTX2, 100 g, the standard portion size; 268 g, the highest 97.5th percentile portion size of shellfish species by New Zealand consumers; and 400 g, the large portion size adopted by EFSA for risk assessment.

Table 19. Deterministic intake of DSP based on samples at or below the regulatory limit for DSP (excluding the PTX-group)

	Units	97.5 th Percentile	Maximum
Concentration DSP	mg OA eq/kg	0.019	0.160
Exposure by eating 100 g	µg OA eq/person	1.9	16.0
	µg OA eq/kg bw	0.03	0.27
Exposure by eating 268 g	µg OA eq/person	5.1	42.9
	µg OA eq/kg bw	0.08	0.71
Exposure by eating 400 g	µg OA eq/person	7.6	64.0
	µg OA eq/kg bw	0.13	1.07

bw = body weight, based on 60 kg person.

The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated with the 97.5th percentile of occurrence in samples at or below the regulatory limit for DSP is 0.13 µg OA eq/kg bw. This is substantially lower than the acute reference dose (ARfD) proposed by EFSA (2008) of 0.3 µg OA eq./kg bw. As results above the regulatory limit were excluded, the maximum of this data set was the regulatory limit (0.16 mg OA eq/kg). The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated at the regulatory limit for DSP is 64 µg OA eq/person or 1.07 µg OA eq/kg bw. This is higher than the ARfD proposed by EFSA (2008) of 0.3 µg PTX2/kg bw. A 60 kg person would only have to consume approximately 113 g of shellfish at the regulatory limit to reach the ARfD proposed by EFSA.

The dietary exposure of DSP was also estimated for all samples, including those that were above the regulatory limit for DSP, despite these samples not expected to be accessible to consumers (Table 20).

Table 20. Deterministic intake of DSP based on all samples, including those above the regulatory limit for DSP.

	Units	97.5 th Percentile	Maximum
Concentration DSP	mg OA eq/kg	0.0230	1.4150
Exposure by eating 100 g	µg OA eq/person	2.3	141.5
	µg OA eq/kg bw	0.04	2.36
Exposure by eating 268 g	µg OA eq/person	6.2	379.2
	µg OA eq/kg bw	0.10	6.32
Exposure by eating 400 g	µg OA eq/person	9.2	566.0
	µg OA eq/kg bw	0.15	9.43

bw = body weight, based on 60kg person.

The exposure for a New Zealand consumer of a large (400 g) portion of shellfish meat contaminated with the 97.5th percentile concentration of DSP in all samples over the 2009-2019 period is 0.15 µg OA eq/kg bw. This is less than the ARfD proposed by EFSA (2008) of 0.3 µg OA eq/kg bw. At the maximum concentration of 1.415 mg OA eq/kg a 60 kg person would have to only consume approximately 13 g of the shellfish to reach the ARfD proposed by EFSA. Samples that are above the regulatory limit may pose a risk to shellfish consumers, and it is important that the regulation of the DSP group continues to ensure protection to consumers.

5.2.2. Probabilistic estimate of dietary exposure to DSP

5.2.2.1. Methodology

An excel spreadsheet containing PTX2 and DSP data, for NZ sites/zone and different bivalve species was loaded into the statistical software R 3.6.1. (R Core Team, 2019) for analysis and the risk characterisation simulation.

The mc2d package (version 0.1-18) for R was used in the development of the simulation and risk characterisation (Pouillot and Delignette-Muller, 2010).

A detailed summary of the exposure assessment and risk characterisation for PTX2 can be found in Appendix G.2.

5.2.2.2. Simulation model development

The main components of the exposure assessment and risk characterisation were the consumption amount of bivalve mollusc and the distribution of DSP concentrations in bivalve molluscs. A probabilistic estimate of dietary exposure to PTX2 was performed by a Monte Carlo simulation to generate the amount of DSP consumed in a sitting (adjusted by kg body weight). The focus of the assessment was on the acute effects of the consumption of DSP.

5.2.2.3. Consumption amount of shellfish

No specific data on the consumption of bivalve shellfish could be obtained and various data sources have been compared and discussed in Section 4.2. Consequently, the approach used by EFSA was used here. In the EFSA (2008) review of Okadaic acid group analogues a triangular distribution was used for the portion sizes because insufficient information was available. This distribution was defined by the minimum value of 0 g; most likely value (mode) of 100 g; and maximum value of 400 g. The large (400 g) portion is likely an over-estimation and hence the exposure to DSP would also likely be over-estimated.

5.2.2.4. Distributions of DSP

For the DSP concentration a similar approach as for PTX2 was used.

The results from the DSP detections were best fitted by a log-normal distribution (Appendix G.2.2.). While some lack of fit was again evident, the deviations do not appear as large as those for PTX2.

Two approaches were undertaken to estimate distributions of DSP, during bloom events only, for the exposure modelling:

Model 1:

- Binomial distribution with probabilities of a detection/non-detection are equal to those in the bloom data set (i.e. 20.63% and 79.37% respectively).
- Detects are generated from a log-normal distribution (variables: meanlog = -3.257, sdlog = 1.017) that was the best fit to the detections, and this was left truncated at the limit of reporting of 0.01 mg/kg as 371 of the 791 detections were between 0.01 and 0.03 (which relates to how the three analogues are analysed and combined for reporting).
- Non-detects are assigned a DSP concentration of 0.03 mg/kg (the sum of the three analogues).

Model 2:

- Using the empirical distribution of DSP concentrations from the bloom data set
- Non-detects are assigned a DSP concentration of 0.03 mg/kg (the sum of the three analogues).

Each of these two models were fitted to data, as well as to the data excluding results above applying the regulatory limit.

The mean, median, 95th percentile, 97.5th percentile, 99th percentile and maximum concentrations of simulated shellfish meals, consumed during bloom events, generated from the two model approaches are shown in Table 21.

Table 21. Concentration of DSP simulated in shellfish meals using the two different model approaches (mg OA eq/kg).

Distribution	Mean	Median	95 th PCTL	97.5 th PCTL	99 th PCTL	Max
Excluding samples above the regulatory limit						
Model 1	0.0341	0.0300	0.0695	0.0974	0.127	0.16
Model 2	0.0305	0.0280	0.0570	0.0850	0.100	0.16
Including samples above the regulatory limit						
Model 1	0.0383	0.0300	0.0843	0.1340	0.219	3.06
Model 2	0.0389	0.0300	0.0740	0.1300	0.270	1.41

5.2.2.5. Estimating Exposure

The final step in this model is to combine the consumption and concentrations data to estimate the amount of DSP consumed in a single sitting, adjusting for the weight of an adult and converting the µg OA eq/kg bw.

The output of the subsequent exposure to DSP during bloom events for each of the two models for simulating DSP concentrations are shown in Table 22. The two models used for the concentration of DSP result in similar exposures, hence the approach used has little effect on the results. When samples exceeding the regulatory limit for DSP are excluded, then the 97.5th percentile was below the EFSA ARfD of 0.3 µg OA eq/kg bw. The 99th percentile for a standard 60 kg adult was just above the EFSA ARfD. The average mass of an adult male in New Zealand is 86.7 kg, and an average adult female is 73.3 kg (Pearson *et al.*, 2018). The larger body masses would result in a lower dietary exposure with the 99th percentile being closer to or below the ARfD. Clearly, from Table 22 it can also be seen that including all DSP samples results in the 97.5th percentile exceeding the EFSA ARfD for DSP (suggesting between 2.5 and 5% of exposures would exceed the ARfD, see section 5.2.2.6 for details).

Table 22. Simulated dietary exposure to DSP using the two different model approaches for a standard 60 kg adult (µg OA eq/kg bw).

Distribution	Mean	Median	95 th PCTL	97.5 th PCTL	99 th PCTL	Max
Excluding samples above the regulatory limit						
Model 1	0.0948	0.0798	0.192	0.283	0.418	1.03
Model 2	0.0848	0.0720	0.169	0.235	0.359	1.04
Including samples above the regulatory limit						
Model 1	0.1060	0.0808	0.229	0.385	0.662	14.10
Model 2	0.1080	0.0779	0.203	0.373	0.767	9.23

5.2.2.6. Risk Characterisation

The risk characterisation in the current context is again a simple matter of comparing the exposure distributions to the corresponding Health Based Guidance Value (HBGV). For DSP a value of 0.33 µg OA eq/kg bw has been used by the World Health Organization and a value of 0.3 µg OA eq/kg bw by EFSA (2008).

When no regulatory limit is applied for DSP, 3.58% of the estimated exposures exceed the HBGV of 0.3 µg OA eq/kg bw assuming a 60 kg adult when using the log-normal distribution to model the DSP concentrations. A slightly lower percentage of 3.26% is obtained when the DSP concentrations are sampled from the empirical DSP distribution during blooms (see Appendix G).

Applying the regulatory limit of 0.16 mg OA eq/kg for DSP results in a reduction in the percentage of exposures exceeding the HBGV, irrespective of which distribution is used for DSP, with 2.23% using the log-normal distribution and 1.55% when the DSP concentrations are sampled from the empirical DSP distribution during blooms. Using the log-normal distribution results in more values in the tail area, even though these are restricted to <0.16 mg OA eq/kg. Consequently, even with the regulatory limit in place it is estimated that 0.90% and 1.42% of exposures during bloom events exceed the DSP HBGV for adult males and females respectively. Despite the low calculated risk of exposure during bloom events, there are only a few historical cases of human poisoning suspected from DSP-toxins in New Zealand from recreational shellfish (MacKenzie *et al.*, 2002).

6. UNCERTAINTY AND GAPS

6.1. Pectenotoxin group

6.1.1. *Uncertainty*

The evaluation of inherent uncertainties in the assessment of exposure to the PTX-group has been performed considering the report on Characterising and Communicating Uncertainty in Exposure Assessment (WHO/IPCS, 2008).

6.1.1.1. *Scenario Uncertainty*

Appropriate calibration standards for PTX-group toxins are only available for PTX2, as such PTX2 is the only PTX-group analogue (excluding seco acids) that was routinely monitored over the 2009-2019 period, and this analogue was used for exposure assessment. Other PTX analogues (PTX1 and PTX11) have also been observed at trace levels but have not been routinely monitored, so insufficient data are available for them. Samples from the blooms containing the highest concentrations of PTX2 were reprocessed to assess the occurrence of PTX1, PTX11 and PTX6 (Section 4.1.12). In all of those results the other PTX analogues that were monitored never exceeded the reporting limit of 0.01 mg/kg. While there is a possibility that other PTX analogues would contribute to the total risk, this is likely to be small.

The impact of processing or cooking was not included in the risk assessment. The uncertainty that this may pose is considered negligible as these toxins are heat stable. Cooking will likely reduce the water content and may concentrate the lipophilic toxins with the flesh. However, it is unknown whether consumption data that were used to base the portion size estimates on are for raw or cooked product.

6.1.1.2. *Model Uncertainty*

The extremely high number of samples having levels below the limit of reporting of 0.01 mg/kg may introduce uncertainty to the overall estimate. It was assumed that samples that were reported as less than the limit of reporting were at the reporting limit to give a conservative assessment. At this concentration there is no possibility of reaching the ARfD based on a large (400 g) portion size, which was the largest portion size that was modelled.

All results were based on routine monitoring results during periods of bloom events as defined in section 4.1.6 where risk would be present to the consumer. This is a conservative estimate of risk. As the analysis was from pre-market routine monitoring, this may not reflect the real range of occurrence of PTX-group analogues in shellfish that reaches the market.

6.1.1.3. Parameter Uncertainty

Due to insufficient data available on portion sizes for shellfish intake for acute risk assessment, a triangular distribution was used to simulate portion sizes. This includes a 400 g large portion size. This distribution is unlikely to accurately represent what occurs in reality. It is expected to over-estimate the consumption of shellfish and as such the estimated exposure and the food safety risk. As the risk assessment was performed based on a 60 kg 'standard' adult body mass, the risk for children is not assessed. As there is insufficient information on consumption data for children this is unable to be evaluated.

As the highest concentration of PTX2 observed in New Zealand shellfish was lower than the concentration required for a 400 g large portion size to reach the conservative ARfD proposed by EFSA, the analysis indicated no risk to NZ public. Best practice dictates that if risk is identified in the deterministic risk assessment, a probabilistic risk assessment is performed. If no risk has been identified, it is optional to perform the probabilistic assessment. Despite a lack of evidence of risk in the analysis presented here, the probabilistic risk assessment was performed. The accuracy of the best fit probability distribution that was developed may introduce an unknown level of uncertainty. The risk characterisation was performed both using the fitted distribution model, as well as the empirical distribution of the data set.

There is no evidence of acute oral toxicity of PTX2, as studies which used authenticated PTX2, PTX11 or PTX2SA material did not show any toxicity at the highest dose (5000 µg/kg). The ARfD of 0.8 µg PTX2/kg proposed by EFSA was used. This is based on older toxicity data which is questionable as this was a single mouse dosed by PTX2 of unknown purity. In this study the mouse showed diarrhetic toxin symptoms suggesting that the toxicity observed may have been caused by contamination of the materials with the OA-group toxins which coexist and are difficult to separate. It is therefore likely that the ARfD suggested by the EFSA CONTAM Panel (EFSA, 2009a) is overly conservative. If the more recent studies with no observable effects at 5000 µg/kg of authenticated material are used as a basis of establishing an ARfD, taking into account a 10-fold safety factor for difference in species, and 10-fold safety factor for variation within species an ARfD of 50 µg/kg bw would be appropriate. In the light of this, the use of the EFSA ARfD of 0.8 µg/kg bw appears to be a significant over-estimation of the food safety risk of the PTX-group.

6.1.2. Summary of Uncertainties

Table 23 summarises the uncertainty evaluation and highlights the main sources of uncertainty and indicates an estimate of whether the respective source of uncertainty might have led to over- or under-estimation of the exposure or resulting risk.

Table 23. Summary of qualitative evaluation of the impact of uncertainties on the risk assessment of the dietary exposure of the PTX-group

Source of Uncertainty	Impact ^a
Exposure during bloom periods was used for a conservative risk assessment	+
Incomplete information on bivalve consumption in New Zealand, no data for children	+/-
Influence of non-detects on exposure estimate	+
Product which would not be expected to be available to consumers due to routine pre-market monitoring and closures for related toxin classes (DSP) were not excluded	+
Dietary exposure estimate was only based on PTX2 and did not include other PTX analogues	-
Conservative ARfD used for assessing food safety risk	+

^a + = uncertainty with potential to cause over-estimation of exposure and human health risk

- = uncertainty with potential to cause under-estimation of exposure and human health risk

6.1.3. Data gaps

- There is no evidence that the PTX-group has ever caused any harm to humans (FAO/IOC/WHO, 2004; Munday, 2017). The FAO/IOC/WHO expert consultation on biotoxins in bivalve molluscs concluded that more data on the subchronic oral toxicity are needed before an ARfD can be established (FAO/IOC/WHO, 2004). However, EFSA proposed an ARfD of 0.8 µg PTX2/kg bw. This value is considered conservative, as it is based on data that are likely to be inaccurate. This is because the study utilised PTX2 of unknown purity and observed diarrhoea which is accepted to be absent in the symptomology of the PTX-group. It is therefore likely that the early studies of Ishige and Ito used PTX2 contaminated with OA and these studies should be discounted (Ishige *et al.*, 1988; Ito, 2006).
- Data on long-term/carcinogenicity, genotoxicity and reproductive toxicity are needed. However, the large difference observed between the intraperitoneal and oral toxicity of PTX-group analogues in mice highlights the low absorption of these toxins. Furthermore, PTX-group analogues are not accumulated within animals when consumed and are excreted rapidly meaning that these toxins are unlikely to be any more toxic when consumed over time. The likelihood of significant toxicity with long-term exposure is also reduced as no toxicity is observed on acute oral exposure even at 5000 µg/kg PTX2 (Miles *et al.*, 2004a).
- Shellfish consumption data should be extended to include portion size and frequency of individual and total shellfish species. For robust accurate

toxicological assessment, portion size data should be linked to body mass and cover both children and adults.

- Further reference materials are required to analyse the range of PTX analogues that EFSA has proposed toxicity equivalency factors (TEF) values for (PTX1, PTX3, PTX4, PTX6 and PTX11), and certified tissue reference materials for PTX-group analogues are required to allow laboratories to quantitate these analogues to better understand the risk posed by their occurrence and make enforcement of regulations possible.
- Robust TEF values are required in order to accurately quantify this group of compounds, and these should be generated via the oral route of administration. However, the absence of observed toxicity via the oral route will make this challenging.

Despite these data gaps, unless new evidence is presented which demonstrates that the PTX-group poses a genuine human health food safety risk then the costly studies required to investigate these gaps seem to be not warranted or necessary.

6.2. Okadaic acid group

6.2.1. *Uncertainty*

The evaluation of inherent uncertainties in the assessment of exposure to DSP has been performed considering the report on Characterising and Communicating Uncertainty in Exposure Assessment (WHO/IPCS, 2008).

6.2.1.1. *Scenario Uncertainty*

The concentration of DSP is determined by the sum of OA, DTX1 and DTX2 that are analysed after hydrolysis to determine the total DSP present within the shellfish. The application of the individual reporting limits for each of these congeners results in a reporting limit for DSP that cannot be readily defined. All of the non-detect samples were assumed to have a concentration of the sum reporting limit of 0.03 mg/kg. The method of analysis has improved over the years. When DSP monitoring first began the reporting limit for OA and DTXs were each 0.05 mg/kg, resulting in a sum reporting limit close to the regulatory limit of 0.16 mg/kg. Monitoring of these congeners is now possible with an individual toxin reporting limit of 0.01 mg/kg, which has resulted in a significant increase in low level detections. Only higher-level detections were observed in the historic samples and as a result the distribution model may be biased towards the high end. However, 0.03 mg/kg was chosen to be applied to all non-detect results so as to not over-estimate the risk of DSP. This may result in a small under-estimation of the low-level detections DSP in the historic samples.

The impact of processing or cooking was not included in the risk assessment. The uncertainty that this may pose is considered negligible as these toxins are heat stable.

Cooking will likely reduce the water content and may concentrate the lipophilic toxins with the flesh. However, it is unknown whether consumption data that were used to base the portion size estimates on are for raw or cooked product.

6.2.1.2. Model Uncertainty

The extremely high number of samples having levels below the limit of reporting may introduce uncertainty to the overall estimate. It was assumed that samples that were reported as non-detects were at the sum reporting limit to give a conservative assessment. At the 0.03 mg/kg concentration assumed for the non-detect samples there is no possibility of reaching the ARfD based on a 400 g large portion size, which was the largest portion size that was modelled.

All results were based on routine monitoring results during periods of bloom events as defined in section 4.1.6 where risk would be present to the consumer. This is likely to over-estimate the risk to consumers as only periods where the risk was greatest were evaluated. As the analysis was from pre-market routine monitoring, this may not reflect the real range of occurrence of OA-group toxins in shellfish that reaches the market.

6.2.1.3. Parameter Uncertainty

Due to insufficient data available on portion sizes for shellfish intake for acute risk assessment, a triangular distribution was used to simulate portion sizes. This includes a 400 g large portion size. This distribution is unlikely to accurately represent the real situation, although it is expected to over-estimate the consumption of shellfish and as such the exposure and resulting risk.

As the highest concentration of OA-group toxins observed in New Zealand shellfish was above the concentration required for a 400 g large portion size to reach the conservative ARfD proposed by EFSA, a potential risk was identified and best practise dictates that a probabilistic risk assessment is required. The accuracy of the best fit probability distribution that was developed may introduce an unknown level of uncertainty. The risk characterisation was performed using both the fitted distribution model, as well as the empirical distribution of the data set.

Regarding the human case studies used for the derivation of the ARfD there is uncertainty with respect to the ingested amount of OA-group toxins. On the other hand, these studies cover a wide range of shellfish consumers. As such the overall uncertainty in the ARfD proposed by EFSA is considered to be low.

6.2.2. Summary of Uncertainties

In Table 24 a summary of the uncertainty evaluation highlights the main sources of uncertainty and indicates an estimate of whether the respective source of uncertainty might have led to over- or under-estimation of the exposure or resulting risk.

Table 24. Summary of qualitative evaluation of the impact of uncertainties on the risk assessment of the dietary exposure of OA-group toxins

Source of Uncertainty	Impact ^a
Exposure during bloom periods was used for a conservative risk assessment	+
Poor sensitivity of the method of analysis in historic samples	+/-
Incomplete information on bivalve consumption in New Zealand, no data for children	+/-
Influence of non-detects on exposure estimate	+
Limitations on the proposed ARfD	+/-

^a + = uncertainty with potential to cause over-estimation of exposure and human health risk

- = uncertainty with potential to cause under-estimation of exposure and human health risk

6.2.3. Data gaps

- Data on long-term/carcinogenicity and further studies on genotoxicity (i.e. clastogenicity) and reproductive toxicity are needed. Further data on absorption, excretion and metabolism are also required (FAO/IOC/WHO, 2004).
- Detailed reports on foodborne illnesses during harmful algal blooms, and reliable data on toxin content in the event of outbreaks of DSP should be provided in order to reduce uncertainty in the ARfD for OA-group toxins. However, this requires outbreaks, which is undesirable. If the regulatory control system continues to mitigate the risk, then it is expected that acquiring such data will be unlikely.
- Effects of shellfish processing (e.g. storage, cooking and freezing) on toxin levels should be investigated.
- Shellfish consumption data should be extended to include portion size and frequency of individual and total shellfish species. For robust acute toxicological assessment, portion size data should be linked to body mass and cover both children and adults.
- Only limited reference material is available for DTX1 and DTX2. As such they currently cannot be routinely incorporated into the regulatory monitoring programme and are only used for infrequently calibrating relative response factors. Isolation of bulk DTXs for use as reference materials is needed.

7. RISK MANAGEMENT

7.1. Regulatory considerations

The history of the regulation of OA derivatives and the PTX group is described in multiple reports of various working groups, scientific opinions and Codex Committee reports. A summary is provided below:

Report 1:

In 2003 the Codex Committee on Fish and Fishery Products (CCFFP) asked for scientific evidence to enable the establishment of maximum levels in shellfish for shellfish toxins (PSP, DSP, ASP, NSP, YTX-group and PTX-group) (FAO/WHO, 2004).

The FAO, WHO and IOC held a joint *ad hoc* expert consultation on biotoxins in bivalve molluscs in Oslo, Norway in 2004. The report (FAO/IOC/WHO, 2004) noted:

- A regulatory level of 0.16 mg OA eq/kg shellfish is implemented in some countries.
- The importance of ester forms of OA was recognised.
- Established an ARfD of 0.33 µg OA eq/kg bw based on a LOAEL of 1 µg OA/kg and a safety factor of 3 to convert to a no-observed-adverse-effect level (NOAEL). The consumption of 250 or 380 g shellfish by a 60 kg adult would yield a derived guidance level of 0.08 or 0.05 mg OA eq/kg shellfish meat, respectively.
- PTX-group analogues do not induce diarrhoea and they should be regulated separately from OA.

Report 2:

In 2006 the joint FAO/WHO food standards programme Codex Committee on Fish and Fishery Products met in Beijing. They produced a “Report of the working group meeting to assess the advice from the Joint FAO/WHO/IOC Ad hoc expert consultation on biotoxins and bivalve molluscs” In this document (FAO/WHO/IOC, 2006):

- The working group concluded that the current standard of 0.16 mg OA eq/kg provides adequate protection for consumers.
- The working group recommended that the codex standard not identify any action for the PTX group. At this time, they should not be regulated.
- The working group recommended that, should data/evidence become available, the potential for adverse effects of PTX to humans will be reassessed.

Report 3:

The European Commission on marine biotoxins in shellfish made a request to consider okadaic acid and analogues. As a result an “Opinion of the Scientific Panel

on Contaminants in the Food Chain on a request from the European Commission on marine biotoxins in shellfish – okadaic acid and analogues (Question No EFSA-Q-2006-065A)” was generated in 2007 (EFSA, 2008). This paper stated:

- Regulation (EC) No 853/2004 states that molluscs should not exceed 160 µg of OA equivalents/kg for OA, DTX and PTXs in combination. The fact that these toxins are grouped together appears to be based on possible co-occurrence of OA-group toxins and PTX-group rather than on toxicological considerations, since the PTX-group does not share the same mechanism of action as OA-group toxins.
- Considering the ARfD of 0.3 µg OA eq/kg bw. In order for a 60 kg adult to avoid exceeding the ARfD, a 400 g portion of shellfish should contain no more than 18 µg OA = 0.045 mg OA eq/kg shellfish meat. This is lower than the current regulation of 0.16 mg OA eq/kg. Based on consumption and occurrence data there is a chance of around 20% to exceed the ARfD of 0.3 µg OA eq/kg bw when consuming shellfish currently available on the European market.
- TEFs were established by the panel: OA = 1, DTX1 = 1, DTX2 = 0.6
- Because the PTX-group does not share the same mechanism of action as OA-group toxins they must not be included in the regulatory limit for OA-group toxins.

Report 4:

The European Commission on marine biotoxins in shellfish made a request to consider the PTX group. As a result a “Scientific opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on marine biotoxins in shellfish – pectenotoxin group (Question No EFSA-Q-2006-0065C)” was produced on 27 May 2009 (EFSA, 2009a). This paper stated:

- The CONTAM panel recognised that the oral data on the PTX group toxins were limited and partly conflicting but to be prudent decided to take into consideration the intestinal toxicity of PTX2 observed in mice and rats at the LOAEL of 250 µg/kg bw (Ishige *et al.*, 1988) and 300 µg/kg bw (Ito, 2006), respectively. To convert to a NOAEL a factor of 3 was applied as well as the factor used for the uncertainty over a different species of 100. This gave an ARfD of 0.8 µg PTX2/kg bw. It is assumed that this ARfD is conservative.
- Using consumption and distribution data the panel estimated that a 60 kg person consuming 400 g of shellfish meat had a 0.8% chance of exceeding the ARfD of 0.8 µg PTX2/kg bw.
- Based on i.p. toxicity the CONTAM panel assigned a TEF of 1 to PTX1, PTX2, PTX3, PTX4, PTX6 and PTX11. The analogues PTX7, PTX8, PTX9, PTX2SA and 7-epi-PTX2SA are much less toxic and were not assigned TEFs.
- There are no data on adverse effects of PTX-group toxins in humans.

Report 5:

The European Commission requested that EFSA summarise the outcome of the adopted opinions on marine biotoxins that are currently regulated in the European Union legislation. In August 2009 a document was produced “Scientific opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on marine biotoxins in shellfish – summary on regulated marine biotoxins (Question No EFSA-Q-2009-00685)”. This report (EFSA, 2009b) states:

- Current EU limits specify 0.16 mg OA eq/kg shellfish meat and that this is a combination of OA, DTXs and PTXs; however, the CONTAM panel concluded that PTX should be considered separately.
- Based on limited information available the CONTAM panel concluded that processing of shellfish could lead to an approximate 2-fold increase in the concentration of lipophilic marine biotoxins (OA-, AZA-, PTX- and YTX-group toxins) in shellfish meat.
- TEFs adopted by the CONTAM panel were as above (7.1 Report 4).
- Based on current data the current EU regulatory limit values for OA are not sufficiently protective for consumers.
- For PTX-group toxins the EU limit values appear to be sufficiently protective for consumers.

In 2011 the regulation EC No 2074/2005 as regards recognised testing methods for detecting marine biotoxins in live bivalve molluscs was amended (Commission Regulation (EU), 2011). The EU-RL LC-MS/MS method shall be the reference method for the detection of marine toxins OA-group toxins (OA, DTX1, DTX2, DTX3 including their esters) and PTX-group toxins (PTX1 and PTX2).

Report 6:

In the report of the 32nd session of the Codex Committee on Fish and Fishery Products (Bali, Indonesia - 2012) (FAO/WHO, 2013):

- This report gave a table of toxin analogues to consider. On this list the OA-group consisted of OA, DTX1, DTX2 and the esters of OA, DTX1 and DTX2 (fatty acid-esters).
- The European Union comments at this meeting (CX/FFP 12/32/7) say that “*the EU suggests the introduction of PTXs, together with OA and DTXs in the list of biotoxins maintaining the established limit of <0.16 mg of OA equivalents. This position is justified because, although the toxicity of this family of toxins is not completely clear, it is demonstrated the conversion of these toxins in toxic compounds when associated with OA and its derivatives. Data of TEFs, Minimum applicable range, Precision (RSDR) and recovery percent can be provided in a later stage if appropriate*”. This is quoted as written in the document, and it is not clear what the justification was that they intended to convey. This suggestion from the EU delegation is further confounded since the EFSA review a few years prior had concluded that PTX and OA groups should not be regulated together. The revised table suggested by the EU

appears not to be adopted by the Codex Committee and was not included in the revised Codex Standard 292-2008 which they were reviewing (Codex Alimentarius, 2015). A note in the report says that *“the EU had wanted PTXs and YTXs to be included in the Table 2 but that there was no consensus in the working group on this”*.

Report 7:

In the report of the 37th session of the Codex Committee on Fish and Fishery Products (FAO/WHO, 2014):

- This report gave a table which included only OA, DTX1 and DTX2 in the OA-group of toxins.

Report 8:

In 2016, in Rome, a technical paper was produced by FAO/WHO “Toxicity equivalence factors for marine biotoxins associated with bivalve molluscs” In this report (FAO/WHO, 2016):

- The expert group recommended TEFs for OA and analogues. The TEF for OA and DTX1 was recommended to be 1 which is the same as the previous EFSA proposed TEFs. The TEF for DTX2 was recommended to be 0.5 which is a slightly lower than the EFSA proposed TEF of 0.6 for this analogue. No TEF is recommended for DTX3 as this compound represents a mixture of ester derivatives of OA, DTX1 and DTX2 and during analysis by LC-MS/MS a hydrolysis step yields the parental toxins.
- The expert group stated that *“In general, where the presence of contamination with OA has been ruled out, indications are that PTXs are not toxic orally”*. There was no mention of the PTX-group with regard to regulatory limits either with the OA group or separately.

Report 9:

In the Codex standard for live and raw bivalve molluscs STAN 292-2008 (Codex Alimentarius, 2015) the maximum level/kg of mollusc flesh for the OA group of biotoxins was not more than 0.16 mg of OA equivalents. The OA group contained OA, DTX1 and DTX2. The PTX-group was not mentioned in this document. There have not been any revisions to this Codex standard since 2015.

Report 10:

In the Fish and Fishery Products Hazards and Controls Guidance document (Fourth Edition, August 2019) (Department of Health & Human Services USA, 2019):

- Table A-5 “FDA and EPA safety levels in regulations and guidance” gives the levels of Okadaic acid (Diarrhetic Shellfish Poisoning (DSP)) as above 0.16 mg/kg total OA acid equivalents (ie, combined free OA, DTX1, DTX2 and their acyl-esters)
- The only mention of the PTX-group in the document was under “Additional toxins found in molluscan shellfish” where it was stated that “A number of

toxins identified in molluscan shellfish have shown toxicity in mouse studies but have not been linked to human illness. These toxins are as follows: Pectenotoxins (PTX) have been detected in phytoplankton and/or molluscan shellfish in Australia, Italy, Japan, New Zealand, Norway, Portugal, Spain and the U.S.” This list included cyclic imines and YTXs as well. There was then a note “PTX and YTX have been found to co-occur with DSP toxins (OA and DTXs) in shellfish” “At this time, FDA makes no recommendations in this guidance document and has no specific expectations with regards to controls for PTX, YTX, and cyclic imines for processors Hazard Analysis Critical Control Point (HACCP) plans”.

For the majority of the toxin classes most countries’ current regulations follow the Codex standards. However, for some toxin classes, including the PTX-group, some countries regulate them while others consider that there is not enough evidence to demonstrate that they are a hazard to human health as no human illness has ever been linked to their consumption and for this reason they do not regulate them. A summary of current regulatory limits is given in Table 25 (Vilariño *et al.*, 2015). Whether the PTX-group forms part of the OA equivalents/kg limit is also highlighted in the table which demonstrates the inconsistencies globally in the way that this toxin class is regulated.

Table 25. Regulatory limits of DSPs and whether the DSP regulation includes the PTX-group in different countries and in the Codex standard

Toxin	Codex	NZ	AUS	Japan	Canada	USA	Mexico	Chile	EU
Max limit (µg OA eq/kg)	160	160	200	160	200	160	160	160	160
Includes PTXs	No	Yes	No	No	Yes	No	No	Yes	Yes

7.2. Risk Management Options for the Pectenotoxin group in New Zealand

There are 4 possible options for future management of the PTX-group in New Zealand.

1. *Status quo*, continue to regulate the PTX-group in New Zealand within the DSP regulation
2. Remove the PTX-group from the DSP regulation, and do not regulate the PTX-group
3. Regulate the PTX-group separately at the current DSP level of 0.16 mg PTX2 eq/kg
4. Regulate the PTX-group separately at the EFSA conservative level of 0.12 mg PTX2 eq/kg

As PTX and OA groups do not share the same mode of action, we suggest there is no justification to continue regulating the PTX-group within the DSP regulation.

There is no evidence that PTX has ever caused any human illness and oral dosing with authenticated PTX2 using mice showed no toxicity at 5000 µg/kg bw. It is therefore unlikely that the PTX-group poses any realistic threat to human health. Comparing possible PTX levels to the conservative (likely based on inaccurate data) EFSA ARfD also shows that the level of potential risk observed in New Zealand from the PTX-group is extremely small. Considering the occurrence of the PTX-group on its own, independently of DSP over the 2009-2019 period, no samples had exceeded the PTX2 concentrations at or above the 0.12 mg PTX2 eq/kg level let alone the existing DSP regulatory limit of 0.16 mg/kg in which PTX2 is currently included. Therefore, based on the current available data it is unlikely that there will be any meaningful or measurable impact between not regulating or regulating PTX2 at the current DSP or proposed EFSA level. However, as Codex does not recommend any regulation of the PTX group, and there is no evidence that it poses a health risk to humans, we suggest it would be appropriate to deregulate this toxin class.

As a large portion of shellfish aquaculture in New Zealand is for the export market and in some export nations PTX-group is regulated as part of the DSP group, continued monitoring of PTX-group should be performed to allow industry to make informed decisions prior to export and to maintain market access.

7.3. Risk Management Options for the Okadaic acid group in New Zealand

While considering the removal of the PTX-group from the DSP regulation, it is appropriate to review the risk and recommendations for regulation of the DSP group. There are 3 possible options for management of the OA-group in New Zealand.

1. *Status quo*, continue to regulate the PTX-group in New Zealand within the DSP regulation
2. Remove the PTX-group from the DSP regulation, regulate DSP at current 0.16 mg OA eq/kg
3. Remove the PTX-group from the DSP regulation, regulate DSP at EFSA maximum safe level of 0.045 mg OA eq/kg
4. Remove the PTX-group from the DSP regulation, regulate DSP at FSANZ maximum safe level of 0.20 mg OA eq/kg

As we have stated earlier, the PTX and OA groups do not share the same mode of action, therefore there is no justification to continue regulating the PTX-group within the DSP regulation.

The level of potential risk of exposure to DSP observed in New Zealand is small. The regulatory limit in most countries is 0.16 mg OA eq/kg, based on Codex recommendations. In the EFSA scientific opinion an ARfD of 0.3 µg/kg bw was established, which would equate to 0.045 mg OA eq/kg in shellfish for a 60 kg person

with a 400 g large portion size. Since this recommendation, no country has adopted a lower value as a regulatory limit. Approximately 0.4% of all samples analysed in New Zealand over the 2009-2019 period exceeded the current Codex regulatory limit for DSP, and if the regulatory limit were to be decreased to the EFSA recommended limit of 0.045 mg OA eq/kg then 1.7% of samples would expect to result in harvest closures, more than 4 times the current number of closures. If the current regulatory limit were to be increased to 0.2 mg OA eq/kg, then approximately 0.3% of samples would expect to result in harvest closures, an insignificant difference compared to the current regulatory limit.

When evaluating the risk during bloom events, it was estimated that 3.58% of exposures using the log-normal theoretical distribution and 3.26% when the DSP concentrations were sampled from the empirical distribution exceeded the EFSA ARfD of 0.3 µg OA eq/kg bw assuming a 60 kg 'standard' adult. Applying the current regulatory limit of 0.16 mg OA eq/kg for DSP resulted in a reduction in the percentage of exposures exceeding the EFSA ARfD, irrespective of which distribution was used for DSP, with 2.23% using the log-normal theoretical distribution and 1.55% when the DSP concentrations are sampled from the empirical distribution. The log-normal theoretical distribution results in more values in the tail area, even though these are restricted to <0.16 mg OA eq/kg. Consequently, even with the regulatory limit in place it is estimated that 0.90% and 1.42% of exposures during bloom events exceed the ARfD for adult New Zealand males and females, respectively. In comparison, the EFSA review determined the probabilistic estimate of dietary exposure of DSP in Europe would result in approximately a 20% risk of a single meal exceeding the ARfD.

In the Joint FAO/WHO/IOC *ad hoc* expert consultation on biotoxins and bivalve molluscs (FAO/WHO/IOC, 2006) the working group concluded that the current standard of 0.16 mg OA eq/kg "*provides adequate protection for consumers*". As a large portion of shellfish aquaculture in New Zealand is for the export market, it is important to remain informed about any changes to the regulation of this toxin group. EFSA proposed a lower level of 0.045 mg OA eq/kg as safe for human consumption based on a 400 g large portion size. While this recommendation has not been adopted by any country including Europe, if any regulatory body implements a reduced regulatory limit such as the EFSA recommended limit, then there could be a significant impact to potential export.

8. CONCLUSION

The grouping of related toxins for the assessment of human exposure is essential as toxicity is generally not due to one individual compound but rather a mixture of related structural analogues. Since the mouse bioassay (MBA) has been proven to be inaccurate and is considered by many countries to be unethical for routine screening, this is now handled by instrumental chemical analysis of shellfish samples for all known analogues of the toxin class. Since analogues will have different toxicities, to translate this into an estimate of overall toxicity the relative toxicities of the individual components must be applied. To determine TEFs, toxicity data is considered with the following order of importance; data from human cases (outbreaks) > oral LD₅₀ in animals > intraperitoneal (i.p.) LD₅₀ in animals > MBA and *in vitro* data (Botana *et al.*, 2017). The fundamental principle of a shared mechanism of action for the application of TEFs is met for OA and the DTXs as both are active on protein phosphatases. However, PTX-group analogues are inactive on protein phosphatases and instead exert their effect by action on F-actin. In our view, including the PTX-group as part of the DSP group of toxins is therefore not justified. This position is consistent with the view expressed by numerous scientific opinions and FAO/WHO/IOC committees as detailed in the report summaries (section 7.1).

Scientifically it is a straightforward decision to remove the PTX-group from the DSP group of toxins, but a harder question is whether the PTX-group should be regulated at all. There is no evidence to suggest that the PTX-group has caused any human illness, a fact that is acknowledged and accepted in the modern literature and various EFSA and WHO documents.

From the deterministic risk assessment of PTX2 the highest concentration observed in shellfish over the 2009-2019 period would require a large 608 g portion size to meet the conservative ARfD proposed by EFSA. With the probabilistic risk assessment of PTX2, there were no simulated cases exceeding the conservative ARfD. To provide an estimate of the acute risk to human health the most relevant parameter is the toxic dose by oral administration. It is now well-recognized that PTX-group analogues do not induce diarrhoea. The early studies of Ishige and Ito that were used as a basis of the conservative ARfD (Ishige *et al.*, 1988; Ito, 2006), which used PTX2 of unknown purity, should be discounted due to the observation of diarrhoea and the likelihood of contamination with OA-group toxins. More recent studies with fully authenticated material showed oral PTX2 toxicity in mice to be >5000 µg/kg bw. Similarly, PTX11 and PTX2SA showed an equally low toxicity and there is no reason to predict that other PTX analogues would be of greater toxicity. The 5000 µg/kg bw is the highest dose tested in the toxicity studies and represents a NOAEL rather than LOAEL or LD₅₀ which may be considerably higher. If the more recent studies with no observable effects at 5000 µg/kg of authenticated material are used as a basis of establishing an ARfD, taking into account a 10-fold safety factor for difference in species, and 10-fold safety factor for variation within species an ARfD of 50 µg/kg bw would be

appropriate. For a 60 kg 'standard' adult human applying the large portion size for a meal proposed by EFSA of 400 g, the level of PTX2 equivalents which would be considered safe is 7.5 mg/kg mollusc flesh. This is approximately 100-fold higher than the maximum observed occurrence of PTX2 in New Zealand shellfish over the 2009-2019 period.

As is the case for most shellfish toxin classes there are no data available on the chronic toxicity of the PTX-group analogues. However, since experiments have shown that absorption of the PTX-group analogues appears to be low then it is considered that the risk of cumulative toxicity with repeated exposure to the PTX-group is small.

From the deterministic risk assessment of OA-group toxins the maximum concentrations observed in New Zealand shellfish exceeded the ARfD suggesting that there is a potential risk to consumers from DSP toxins. There is low risk of exceeding the DSP ARfD during bloom events as determined by the Monte Carlo simulation, and this was significantly reduced with the application of the current regulatory limit. Despite the low calculated risk of exposure during bloom events there are only a few historical cases of human poisoning suspected from DSP toxins in New Zealand, and this was from recreationally gathered shellfish. The absence of outbreaks supports the conclusion that the existing regulatory limit is fit for purpose. This is further supported by the 2006 the joint FAO/WHO food standards programme Codex Committee on Fish and Fishery Products working group meeting which concluded that the current standard of 0.16 mg OA eq/kg provides adequate protection for consumers (FAO/WHO/IOC, 2006).

8.1. Recommendations

Based on the scientific consensus that the PTX-group should not be regulated with the DSP group as they do not share the same mode of action, and there is no evidence that the PTX-group have any impact on human health, the following recommendations are made:

- Remove pectenotoxins from the Diarrhetic Shellfish Poison (DSP) regulation in New Zealand. This will require amending the footnote to table 3 in the New Zealand Animal Products Notice Regulatory Control Scheme for Bivalve Molluscan Shellfish from:

“¹ Okadaic acid includes okadaic acid (OA), dinophysistoxins (DTX1 and DTX2) and pectenotoxins (PTX1 and PTX2), where for okadaic acid and dinophysistoxins a hydrolysis step is required in order to detect the presence of esterified okadaic acid group toxins.”

to:

“¹ Okadaic acid includes okadaic acid (OA), dinophysistoxins (DTX1 and DTX2), where a hydrolysis step is required in order to detect the presence of esterified okadaic acid group toxins.”

- Maintain regulation of Diarrhetic Shellfish Poison (DSP) at the current maximum permissible level of 0.16 mg OA eq/kg of mollusc flesh.
- Consistent with the Codex Standard 292-2008, no action levels should be set for the PTX-group, and they should not be regulated.

Several potential export markets currently regulate pectenotoxins as part of the DSP group (e.g. EU). PTX2 should continue to be monitored to meet overseas market access requirements (OMAR). While PTX2SA has been observed to have only a weak relationship to DSP, it still has a useful role in the routine DSP monitoring as an informal biomarker to aid in the early detection of presence of dinoflagellates which also produce the DSP toxins.

9. REFERENCES

- Ares, I., C. Louzao, B. Espiña, M. Vieytes, C. Miles, T. Yasumoto and L. Botana (2007). "Lactone ring of pectenotoxins: a key factor for their activity on cytoskeletal dynamics." Cellular Physiology and Biochemistry 19(5-6): 283-292.
- Aune, T., A. Espenes, J. A. B. Aasen, M. A. Quilliam, P. Hess and S. Larsen (2012). "Study of possible combined toxic effects of azaspiracid-1 and okadaic acid in mice via the oral route." Toxicon 60(5): 895-906.
- Aune, T., S. Larsen, J. A. Aasen, N. Rehmann, M. Satake and P. Hess (2007). "Relative toxicity of dinophysistoxin-2 (DTX-2) compared with okadaic acid, based on acute intraperitoneal toxicity in mice." Toxicon 49(1): 1-7.
- Australian Bureau of Statistics (2014). Australian Health Survey: Nutrition First Results – Food and Nutrients, 2011-12: 78.
- Bialojan, C. and A. Takai (1988). "Inhibitory effect of a marine-sponge toxin, okadaic acid, on protein phosphatases. Specificity and kinetics." The Biochemical journal 256: 283-290.
- Botana, L. M., P. Hess, R. Munday, A. Nathalie, S. L. DeGrasse, M. Feeley, T. Suzuki, M. Van Den Berg, V. Fattori, E. G. J. T. i. f. s. Gamarro and technology (2017). "Derivation of toxicity equivalency factors for marine biotoxins associated with Bivalve Molluscs." 59: 15-24.
- Boundy, M. and P. McNabb (2013). Update Method Validation - Cawthron Method 40.105 Determination of Marine Toxins in Shellfish by LC-MS/MS. Internal Report, Cawthron: 29.
- Burgess, V. and G. Shaw (2001). "Pectenotoxins—an issue for public health: a review of their comparative toxicology and metabolism." Environment international 27(4): 275-283.
- Burgess, V. A. (2003). Toxicology investigations with the pectenotoxin-2 seco acids, Griffith University.
- Butler, S. C., C. O. Miles, A. Karim and M. J. Twiner (2012). "Inhibitory effects of pectenotoxins from marine algae on the polymerization of various actin isoforms." Toxicology in Vitro 26(3): 493-499.
- Chae, H.-D., T.-S. Choi, B.-M. Kim, J. H. Jung, Y.-J. Bang and D. Y. Shin (2005). "Oocyte-based screening of cytokinesis inhibitors and identification of pectenotoxin-2 that induces Bim/Bax-mediated apoptosis in p53-deficient tumors." Oncogene 24(30): 4813.
- Codex Alimentarius (2006). Twenty-Eighth Session, Beijing, China, September 18–22, 2006 CX/FFP 06/28/6 Add.1. Codex Committee on Fish and Fishery Products.
- Codex Alimentarius (2015). "Standard for live and raw bivalve molluscs." Codex Standard 292-2008.
- Commission Regulation (EU) (2011). Commission Regulation (EU) No 15/2011 of 10 January 2011 amending Regulation (EC) No 2074/2005 as regards recognised testing methods for detecting marine biotoxins in live bivalve molluscs. Official Journal of the European Union. L6: 3-6.

Cordier, S., C. Monfort, L. Miossec, S. Richardson and C. Belin (2000). "Ecological analysis of digestive cancer mortality related to contamination by diarrhetic shellfish poisoning toxins along the coasts of France." Environmental Research 84(2): 145-150.

Cressey, P., A. Bolton, O. Pantos and J. Nicolas (2019). Risk Profile: Ciguatoxins in seafood, New Zealand Food Safety: 57.

Cruz, P. G., A. H. Daranas, J. J. Fernández, M. L. Souto and M. Norte (2006). "DTX5c, a new OA sulphate ester derivative from cultures of *Prorocentrum belizeanum*." Toxicon 47(8): 920-924.

Daiguji, M., M. Satake, K. J. James, A. Bishop, L. MacKenzie, H. Naoki and T. Yasumoto (1998). "Structures of new pectenotoxin analogs, pectenotoxin-2 seco acid and 7-epi-pectenotoxin-2 seco acid, isolated from a dinoflagellate and greenshell mussels." Chemistry Letters(7): 653-654.

Damsch, S., G. Eichenbaum, A. Tonelli, L. Lammens, K. Van den Bulck, B. Feyen, J. Vandenberghe, A. Megens, E. Knight and M. Kelley (2011). "Gavage-related reflux in rats: identification, pathogenesis, and toxicological implications (review)." Toxicol Pathol 39(2): 348-360.

Department of Health & Human Services USA (2019). Fish and Fishery Products Hazards and Controls Guidance, Fourth Edition.

Dickey, R. W., S. C. Bobzin, D. J. Faulkner, F. A. Bencsath and D. Andrzejewski (1990). "Identification of okadaic acid from a Caribbean dinoflagellate, *Prorocentrum concavum*." Toxicon 28(4): 371-377.

EFSA (2008). "Opinion of the Scientific Panel on Contaminants in the Food chain on a request from the European Commission on marine biotoxins in shellfish - okadaic acid and analogues." The EFSA Journal 589: 1-62.

EFSA (2009a). "Scientific Opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on marine biotoxins in shellfish - pectenotoxin group." 1109: 1-47.

EFSA (2009b). "Scientific Opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on Marine Biotoxins in Shellfish - Summary on regulated marine biotoxins." The EFSA Journal 1306: 1-23.

Espina, B., M. Louzao, I. Ares, E. Cagide, M. Vieytes, F. Vega, J. Rubiolo, C. Miles, T. Suzuki and T. Yasumoto (2008). "Cytoskeletal toxicity of pectenotoxins in hepatic cells." British journal of pharmacology 155(6): 934-944.

Espina, B., M. C. Louzao, I. R. Ares, E. S. Fonfría, N. Vilariño, M. R. Vieytes, T. Yasumoto and L. M. Botana (2010). "Impact of the pectenotoxin C-43 oxidation degree on its cytotoxic effect on rat hepatocytes." Chemical research in toxicology 23(3): 504-515.

FAO/IOC/WHO (2004). Report of the Joint FAO/IOC/WHO ad hoc Expert Consultation on Biotoxins in Bivalve Molluscs. Food and Agriculture Organization of the United Nations: 31.

FAO/WHO (2004). Report of the twenty-sixth session of the Codex Committee on Fish and Fishery Products, Aalesund, Norway, 13-17 October 2003.

FAO/WHO (2013). Report of the thirty-second session of the Codex Committee on Fish and Fishery Products. Bali, Indonesia.

FAO/WHO (2014). Report of the thirty-third session of the Codex Committee on Fish and Fishery Products. Bergen, Norway.

FAO/WHO (2016). Technical paper on Toxicity Equivalency Factors for Marine Biotoxins Associated with Bivalve Molluscs. Rome. 108 pp.

FAO/WHO/IOC (2006). Report of the working group meeting to assess the advice from the joint FAO/WHO/IOC ad hoc expert consultation on biotoxins in bivalve molluscs, CX/FFP 06/28/6-Add.1. Beijing, China.

Fladmark, K. E., M. H. Serres, N. L. Larsen, T. Yasumoto, T. Aune and S. O. Døskeland (1998). "Sensitive detection of apoptogenic toxins in suspension cultures of rat and salmon hepatocytes." Toxicon 36(8): 1101-1114.

FSANZ (2017). Maximum levels of non-metal contaminants. F2017C00333 S19-5. FSANZ. Australia New Zealand Food Standards Code - Schedule 19.

García, C., D. Truan, M. Lagos, J. P. Santelices, J. C. Díaz and N. Lagos (2005). "Metabolic transformation of dinophysistoxin-3 into dinophysistoxin-1 causes human intoxication by consumption of O-acyl-derivatives dinophysistoxins contaminated shellfish." The Journal of toxicological sciences 30(4): 287-296.

Guo, F., T. An and K. S. Rein (2010). "The algal hepatotoxin okadaic acid is a substrate for human cytochromes CYP3A4 and CYP3A5." Toxicon 55(2-3): 325-332.

Hamano, Y., Y. Kinoshita and T. Yasumoto (1986). "Enteropathogenicity of diarrhetic shellfish toxins in intestinal models." Journal of the Food Hygiene Society of Japan 27(4): 375-379.

Holland, P. (2002). Method 40.105 ASP and DSP toxins in shellfish by LC-MS. Internal Report, Cawthron Institute: 74.

Holland, P. T., P. McNabb, A. L. Selwood, L. MacKenzie and V. Beuzenberg (2003). LC-MS methods for marine biotoxins and their introduction into the New Zealand shellfish regulatory programme. HABTech 2003. P. Holland, L. Rhodes and L. Brown. Nelson, New Zealand, Cawthron Report No. 906, Cawthron Institute: 10-17.

Hu, T., J. M. Curtis, J. A. Walter and J. L. C. Wright (1995). "Identification of DTX-4, a new water-soluble phosphatase inhibitor from the toxic dinoflagellate *Prorocentrum lima*." J. Chem. Soc., Chem. Commun.: 597-599.

Ishige, M., N. Satoh and T. Yasumoto (1988). "Pathological studies on mice administered with the causative agent of diarrhetic shellfish poisoning (okadaic acid and pectenotoxin-2)." Hokkaidoritsu Eisei Kenkyushoho(38): 15-18.

Ito, E. (2006). Verification of diarrhetic activities of PTX-2 and okadaic acid in vivo. 12th International Conference on Harmful Algae.

Ito, E., T. Suzuki, Y. Oshima and T. Yasumoto (2008). "Studies of diarrhetic activity on pectenotoxin-6 in the mouse and rat." Toxicon 51(4): 707-716.

Ito, E. and K. Terao (1994). "Injury and recovery process of intestine caused by okadaic acid and related compounds." Natural Toxins 2(6): 371-377.

Ito, E., T. Yasumoto, A. Takai, S. Imanishi and K. Harada (2002). "Investigation of the distribution and excretion of okadaic acid in mice using immunostaining method." Toxicon 40(2): 159-165.

King, N. and R. Lake (2013). "Bivalve shellfish harvesting and consumption in New Zealand, 2011: data for exposure assessment." New Zealand Journal of Marine and Freshwater Research 47(1): 62-72.

Le Hégarat, L., A.-G. Jacquin, E. Bazin and V. Fessard (2006). "Genotoxicity of the marine toxin okadaic acid, in human Caco-2 cells and in mice gut cells." Environmental Toxicology 21(1): 55-64.

Liu, L., F. Guo, S. Crain, M. A. Quilliam, X. Wang and K. S. Rein (2012). "The structures of three metabolites of the algal hepatotoxin okadaic acid produced by oxidation with human cytochrome P450." Bioorganic & medicinal chemistry 20(12): 3742-3745.

Lopez-Rodas, V., E. Maneiro, J. Martinez, M. Navarro and E. Costas (2006). "Harmful algal blooms, red tides and human health: Diarrhetic shellfish poisoning and colorectal cancer." Anales de la Real Academia Nacional de Farmacia 72: 391-408.

MacKenzie, L., P. Holland, P. McNabb, V. Beuzenberg, A. Selwood and T. Suzuki (2002). "Complex toxin profiles in phytoplankton and Greenshell mussels (*Perna canaliculus*), revealed by LC-MS/MS analysis." Toxicon 40(9): 1321-1330.

MacKenzie, L. A., A. I. Selwood and C. Marshall (2012). "Isolation and characterization of an enzyme from the Greenshell™ mussel *Perna canaliculus* that hydrolyses pectenotoxins and esters of okadaic acid." Toxicon 60(3): 406-419.

Macpherson, G. R., I. W. Burton, P. LeBlanc, J. A. Walter and J. L. C. Wright (2003). "Studies of the biosynthesis of DTX-5a and DTX-5b by the dinoflagellate *Prorocentrum maculosum*: regiospecificity of the putative Baeyer-Villigerase and insertion of a single amino acid in a polyketide chain." J. Org. Chem. 68(5): 1659–1664.

Marr, J. C., T. Hu, S. Pleasance, M. A. Quilliam and J. L. C. Wright (1992). "Detection of new 7-O-acyl derivatives of diarrhetic shellfish poisoning toxins by liquid chromatography-mass spectrometry." Toxicon 30(12): 1621-1630.

Matias, W., A. Traore and E. Creppy (1999). "Variations in the distribution of okadaic acid in organs and biological fluids of mice related to diarrhoeic syndrome." Human & experimental toxicology 18(5): 345-350.

McNabb, P., A. I. Selwood and P. T. Holland (2005). "Multiresidue method for determination of algal toxins in shellfish: single-laboratory validation and interlaboratory study." Journal of AOAC International 88(3): 761-772.

McNabb, P. and R. van Ginkel (2008). Updated Method Validation - Cawthron Method 40.105 Determination of marine toxins in shellfish by LC-MS/MS. Internal Report, Cawthron: 26.

Miles, C. O., A. L. Wilkins, A. D. Hawkes, D. J. Jensen, A. I. Selwood, V. Beuzenberg, A. L. MacKenzie, J. M. Cooney and P. T. Holland (2006a). "Isolation and identification of pectenotoxins-13 and -14 from *Dinophysis acuta* in New Zealand." Toxicon 48(2): 152-159.

Miles, C. O., A. L. Wilkins, J. S. Munday, R. Munday, A. D. Hawkes, D. J. Jensen, J. M. Cooney and V. Beuzenberg (2006b). "Production of 7-epi-pectenotoxin-2 seco acid and assessment of its acute toxicity to mice." Journal of Agricultural and Food Chemistry 54(4): 1530-1534.

Miles, C. O., A. L. Wilkins, R. Munday, M. H. Dines, A. D. Hawkes, L. R. Briggs, M. Sandvik, D. J. Jensen, J. M. Cooney, P. T. Holland, M. A. Quilliam, A. L. MacKenzie, V. Beuzenberg and N. R. Towers (2004a). "Isolation of pectenotoxin-2 from *Dinophysis acuta* and its conversion to pectenotoxin-2 seco acid, and preliminary assessment of their acute toxicities." Toxicon 43(1): 1-9.

Miles, C. O., A. L. Wilkins, I. A. Samdal, M. Sandvik, D. Petersen, M. A. Quilliam, L. J. Naustvoll, T. Rundberget, T. Torgersen, P. Hovgaard, D. J. Jensen, B. Lundve and O. Lindahl (2004b). PTX-12, a new pectenotoxin from *Dinophysis* species and mussels in Scandinavia. 5th International Conference on Molluscan Shellfish Safety, Galway, Ireland.

Ministry for Primary Industries (2018). Regulated Control Scheme - Bivalve Molluscan Shellfish for Human Consumption.

Munday, R. (2014). Toxicology of Seafood Toxins: A Critical Review. Seafood and Freshwater Toxins: Pharmacology, Physiology, and Detection. L. M. Botana, CRC Press: 197-290.

Munday, R. (2017). "Toxicology of Seafood Toxins: Animal Studies and Mechanisms of Action." Recent Advances in the Analysis of Marine Toxins 78: 211.

Murata, M., M. Shimatani, H. Sugitani, Y. Oshima and T. Yasumoto (1982). "Isolation and structural elucidation of the causative toxin of the diarrhetic shellfish poisoning." Bulletin of the Japanese Society of Scientific Fisheries 48(4): 549-552.

Nishitani, G., S. Nagai, K. Baba, S. Kiyokawa, Y. Kosaka, K. Miyamura, T. Nishikawa, K. Sakurada, A. Shinada and T. Kamiyama (2010). "High-level congruence of *Myrionecta rubra* prey and *Dinophysis* species plastid identities as revealed by genetic analyses of isolates from Japanese coastal waters." Appl. Environ. Microbiol. 76(9): 2791-2798.

Nishiwaki, S., H. Fujiki, M. Suganuma, H. Furuya-Suguri, R. Matsushima, Y. Iida, M. Ojika, K. Yamada, D. Uemura, T. Yasumoto, F. J. Schmitz and T. Sugimura (1990). "Structure-activity relationship within a series of okadaic acid derivatives." Carcinogenesis 11(10): 1837-1841.

Ogino, H., M. Kumagai and T. Yasumoto (1997). "Toxicologic evaluation of yessotoxin." Natural Toxins 5: 255-259.

Parnell, W., R. Scragg, N. Wilson, D. Schaaf and E. Fitzgerald (2003). "NZ food: NZ children." Key Results of the 2002 National Children's Nutrition Survey.

Parnell, W., N. Wilson, C. Thomson, S. Mackay and N. Stefanogiannis (2011). "A focus on nutrition: key findings of the 2008/09 New Zealand Adult Nutrition Survey." Ministry of Health: Wellington, New Zealand.

Paz, B., A. H. Daranas, P. G. Cruz, J. M. Franco, J. G. Napolitano, M. Norte and J. J. Fernandez (2007). "Identification and characterization of DTX-5c and 7-hydroxymethyl-2-methylene-octa-4,7-dienyl okadaate from *Prorocentrum belizeanum* cultures by LC-MS." Toxicon 50(4): 470-478.

Pearson, A., M. Gibbs, K. Lau, J. Edmonds, D. Alexander and J. Nicolas (2018). 2016 New Zealand total diet study, Ministry for Primary Industries, Wellington.

Pouillot, R. and M. L. Delignette-Muller (2010). "Evaluating variability and uncertainty separately in microbial quantitative risk assessment using two R packages." International journal of food microbiology 142(3): 330-340.

Quilliam, M. A., W. R. Hardstaff, N. Ishida, J. L. McLachlan, A. R. Reeves, N. W. Ross and A. J. Windust (1996). Production of diarrhetic shellfish poisoning (DSP) toxins by *Prorocentrum lima* in culture and development of analytical methods. Seventh International Conference on Toxic Phytoplankton, Sendai, Japan, International Oceanographic Commission of UNESCO, Paris.

R Core Team (2019). "A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing; 2012." URL <https://www.R-project.org>.

Rao, G., T. Peace and D. Hoskins (2001). "Training could prevent deaths due to rodent gavage procedure." Contemporary topics in laboratory animal science 40(4): 7.

Rossini, G. P. and P. Hess (2010). Phycotoxins: chemistry, mechanisms of action and shellfish poisoning. Molecular, clinical and environmental toxicology, Springer: 65-122.

Russell, D., W. Parnell, N. Wilson, J. Faed, E. Ferguson, P. Herbison, C. Horwath, T. Nye, P. Reid and R. Walker (1999). "NZ food: NZ people." Key results of the 1997 national nutrition survey. Wellington: Ministry of Health: p71.

Sasaki, K., J. L. Wright and T. Yasumoto (1998). "Identification and characterization of pectenotoxin (PTX) 4 and PTX7 as spiroketal stereoisomers of two previously reported pectenotoxins." Journal of Organic Chemistry 63(8): 2475–2480.

Suzuki, H. (2012). "Susceptibility of different mice strains to okadaic acid, a diarrhetic shellfish poisoning toxin." Food Additives & Contaminants: Part A 29(8): 1307-1310.

Suzuki, T. (2014). "Chemistry and Detection of Okadaic Acid/Dinophysistoxins, Pectenotoxins and Yessotoxins." Toxins and Biologically Active Compound from Microalgae 1: 99-152.

Suzuki, T., V. Beuzenberg, L. Mackenzie and M. A. Quilliam (2003). "Liquid chromatography-mass spectrometry of spiroketal stereoisomers of pectenotoxins and the analysis of novel pectenotoxin isomers in the toxic dinoflagellate *Dinophysis acuta* from New Zealand." Journal of Chromatography A 992(1-2): 141-150.

Suzuki, T., V. Beuzenberg, L. Mackenzie and M. A. Quilliam (2004). "Discovery of okadaic acid esters in the toxic dinoflagellate *Dinophysis acuta* from New Zealand using liquid chromatography tandem mass spectrometry." Rapid Communications in Mass Spectrometry 18(10): 1131-1138.

Suzuki, T., L. Mackenzie, D. Stirling and J. Adamson (2001a). "Conversion of pectenotoxin-2 to pectenotoxin-2 seco acid in the New Zealand scallop, *Pecten novaezelandiae*." Fisheries Science 67(3): 506-510.

Suzuki, T., L. Mackenzie, D. Stirling and J. Adamson (2001b). "Pectenotoxin-2 seco acid: a toxin converted from pectenotoxin-2 by the New Zealand Greenshell mussel, *Perna canaliculus*." Toxicon 39(4): 507–514.

Suzuki, T., T. Mitsuya, H. Matsubara and M. Yamasaki (1998). "Determination of pectenotoxin-2 after solid-phase extraction from seawater and from the dinoflagellate *Dinophysis fortii* by liquid chromatography with electrospray mass spectrometry and ultraviolet detection. Evidence of oxidation of pectenotoxin-2 to pectenotoxin-6 in scallops." Journal of Chromatography A 815(1): 155–160.

Suzuki, T., J. A. Walter, P. LeBlanc, S. MacKinnon, C. O. Miles, A. L. Wilkins, R. Munday, V. Beuzenberg, A. L. MacKenzie, D. J. Jensen, J. M. Cooney and M. A. Quilliam (2006). "Identification of pectenotoxin-11 as 34S-hydroxypectenotoxin-2, a new pectenotoxin analogue in the toxic dinoflagellate *Dinophysis acuta* from New Zealand." Chemical Research in Toxicology 19(2): 310-318.

Tachibana, K., P. J. Scheuer, Y. Tsukitani, H. Kikuchi, D. Van Engen, J. Clardy, Y. Gopichand and F. J. Schmitz (1981). "Okadaic acid, a cytotoxic polyether from two marine sponges of the genus *Halichondria*." Journal of the American Chemical Society 103(9): 2469–2471.

Takai, A., M. Murata, M. Isobe, G. Mieskes and T. Yasumoto (1992). "Inhibitory effect of okadaic acid derivatives on protein phosphatases. A study on structure–affinity relationship." Biochemical Journal 284(2): 539–544.

Terao, K., E. Ito, M. Ohkusu and T. Yasumoto (1993). A comparative study of the effects of DSP-toxins on mice and rats. Toxic Phytoplankton Blooms in the Sea: Proceedings of the Fifth International Conference on Toxic Marine Phytoplankton, Newport, Rhode Island, U.S.A., 28 October-1 November 1991. Y. S. Theodore J. Smayda, Elsevier: 581-587.

Terao, K., E. Ito, T. Yanagi and T. Yasumoto (1986). "Histopathological studies on experimental marine toxin poisoning. I. Ultrastructural changes in the small intestine and liver of suckling mice induced by dinophysistoxin-1 and pectenotoxin-1." Toxicon 24(11-12): 1141–1151.

Tubaro, A., S. Sosa, G. Altinier, M. R. Soranzo, M. Satake, R. Della Loggia and T. Yasumoto (2004). "Short-term oral toxicity of homoyessotoxins, yessotoxin and okadaic acid in mice." Toxicon 43(4): 439-445.

Tubaro, A., S. Sosa, M. Carbonatto, G. Altinier, F. Vita, M. Melato, M. Satake and T. Yasumoto (2003). "Oral and intraperitoneal acute toxicity studies of yessotoxin and homoyessotoxins in mice." Toxicon 41(7): 783-792.

Vale, P. and M. A. d. M. Sampayo (2002). "Pectenotoxin-2 seco acid, 7-epi-pectenotoxin-2 seco acid and pectenotoxin-2 in shellfish and plankton from Portugal." Toxicon 40(7): 979-987.

Vilariño, N., M. C. Louzao, M. Fraga and L. M. Botana (2015). From science to policy: dynamic adaptation of legal regulations on aquatic biotoxins. Climate Change and Marine and Freshwater Toxins. L. M. Botana, M. C. Louzao and N. Vilariño, De Gruyter, Inc.

Williamstown Contamination Expert Panel (2015). Preliminary Dietary Exposure Assessment – Seafood – Tilligerry Creek and Fullerton Cove, Williamstown NSW, New South Wales Government: 17.

World Health Organization International Programme on Chemical Safety (WHO/IPCS) (2008). Uncertainty and data quality in exposure assessment, World Health Organization.

Yanagi, T., M. Murata, K. Torigoe and T. Yasumoto (1989). "Biological activities of semisynthetic analogs of dinophysistoxin-3, the major diarrhetic shellfish toxin." Agricultural and Biological Chemistry 53(2): 525-529.

Yasumoto, T., M. Murata, J.-S. Lee and K. Torigoe (1989). Polyether toxins produced by dinoflagellates. Mycotoxins and Phycotoxins '88. S. Natori, K. Hashimoto and Y. Ueno. Amsterdam, Elsevier: 375–382.

Yasumoto, T., M. Murata, Y. Oshima, G. K. Matsumoto and J. Clardy (1984). Diarrhetic shellfish poisoning. Seafood Toxins, ACS ACS Symposium Series 262: 207–214.

Yasumoto, T., M. Murata, Y. Oshima, M. Sano, G. K. Matsumoto and J. Clardy (1985). "Diarrhetic shellfish toxins." Tetrahedron 41(6): 1019–1025.

Yasumoto, T., Y. Oshima and M. Yamaguchi (1978). "Occurrence of a new type of shellfish poisoning in the Tohoku district." Bulletin of the Japanese Society of Scientific Fisheries 44(11): 1249-1255.

Yasumoto, T., N. Seino, Y. Murakami and M. Murata (1987). "Toxins Produced by Benthic Dinoflagellates." The Biological Bulletin 172(1): 128-131.

Yoon, M. Y. and Y. C. Kim (1997a). "[Acute toxicity of pectenotoxin-2 and its effects on hepatic metabolising enzyme system in mice]." Korean Journal of Toxicology 13: 183–186 [in Korean].

Yoon, M. Y. and Y. C. Kim (1997b). "Toxicity and changes in hepatic metabolizing enzyme system induced by repeated administration of pectenotoxin 2 isolated from marine sponges." Korean Journal of Pharmacognosy 28(4): 280–285 [in Korean].

Yuasa, H., K. Yoshida, H. Iwata, H. Nakanishi, M. Suganuma and M. Tatematsu (1994). "Increase of labeling indices in gastrointestinal mucosae of mice and rats by compounds of the okadaic acid type." Journal of Cancer Research and Clinical Oncology 120: 208-212.

APPENDIX A. BLOOM IDENTIFICATION

Table A-1. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins in shellfish zones within New Zealand 2009-2019

		PTX2							PTX2SAs							DSP						
Zone	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A	916	42	4.6%	0.010	0.034	0.016	0.015	0.027	227	24.8%	0.010	1.200	0.061	0.029	0.214	29	3.2%	0.010	0.100	0.035	0.026	0.099
B	116	5	4.3%	0.012	0.032	0.018	0.013	0.031	17	14.7%	0.010	0.260	0.089	0.052	0.256	2	1.7%	0.012	0.025	0.019	0.019	0.025
C	1916	58	3.0%	0.010	0.059	0.021	0.017	0.053	317	16.5%	0.010	1.500	0.189	0.075	1.000	208	10.9%	0.010	0.238	0.041	0.030	0.145
D	384	1	0.3%	0.031	0.031	0.031	0.031	0.031	34	8.9%	0.010	0.260	0.057	0.023	0.194	12	3.1%	0.011	0.065	0.024	0.017	0.064
F	150	0							5	3.3%	0.010	0.026	0.015	0.013	0.025	0						
G	13278	63	0.5%	0.010	0.043	0.016	0.014	0.038	2475	18.6%	0.010	1.700	0.051	0.021	0.300	138	1.0%	0.010	0.700	0.066	0.050	0.282
H	54	0							0							0						
I	1525	77	5.0%	0.010	0.079	0.021	0.015	0.064	563	36.9%	0.010	3.500	0.153	0.058	0.828	368	24.1%	0.010	1.415	0.095	0.032	0.600
J	608	5	0.8%	0.011	0.017	0.012	0.011	0.016	85	14.0%	0.010	0.350	0.063	0.027	0.267	34	5.6%	0.011	0.340	0.116	0.092	0.299
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

Table A-2. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins in shellfish subzones within New Zealand 2009-2019

Zone Subzone	Sites	No Samples	PTX2							PTX2SAs							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A	9	426	0							14	3.3%	0.011	0.038	0.019	0.017	0.038	9	2.1%	0.014	0.099	0.031	0.018	0.092
A boi	7	490	42	8.6%	0.010	0.034	0.016	0.015	0.027	213	43.5%	0.010	1.200	0.064	0.031	0.217	20	4.1%	0.010	0.100	0.037	0.028	0.098
B	8	116	5	4.3%	0.012	0.032	0.018	0.013	0.031	17	14.7%	0.010	0.260	0.089	0.052	0.256	2	1.7%	0.012	0.025	0.019	0.019	0.025
C	18	1916	58	3.0%	0.010	0.059	0.021	0.017	0.053	317	16.5%	0.010	1.500	0.189	0.075	1.000	208	10.9%	0.010	0.238	0.041	0.030	0.145
D	11	384	1	0.3%	0.031	0.031	0.031	0.031	0.031	34	8.9%	0.010	0.260	0.057	0.023	0.194	12	3.1%	0.011	0.065	0.024	0.017	0.064
F	5	150	0							5	3.3%	0.010	0.026	0.015	0.013	0.025	0						
G clo	4	556	4	0.7%	0.010	0.043	0.023	0.020	0.042	69	12.4%	0.010	0.490	0.066	0.027	0.386	14	2.5%	0.050	0.700	0.177	0.100	0.632
G eb	4	563	1	0.2%	0.010	0.010	0.010	0.010	0.010	159	28.2%	0.010	0.250	0.032	0.019	0.151	0						
G for	8	1151	2	0.2%	0.011	0.013	0.012	0.012	0.013	56	4.9%	0.010	0.035	0.015	0.013	0.033	0						
G pel	23	7387	9	0.1%	0.010	0.022	0.015	0.014	0.022	972	13.2%	0.010	0.890	0.032	0.017	0.157	12	0.2%	0.028	0.320	0.115	0.083	0.295
G ptU	47	11393	50	0.4%	0.010	0.043	0.016	0.014	0.036	2151	18.9%	0.010	1.700	0.050	0.021	0.290	132	1.2%	0.010	0.700	0.066	0.048	0.295
G qc	2	32	0							11	34.4%	0.012	0.053	0.022	0.015	0.051	1	3.1%	0.016	0.016	0.016	0.016	0.016
G tas	8	913	13	1.4%	0.010	0.037	0.018	0.016	0.036	164	18.0%	0.010	1.400	0.082	0.023	0.576	5	0.5%	0.054	0.087	0.074	0.077	0.086
G tc	4	940	0							149	15.9%	0.010	0.140	0.029	0.019	0.095	0						
H	1	54	0							0							0						
I bpk	13	1204	74	6.1%	0.010	0.079	0.021	0.016	0.065	554	46.0%	0.010	3.500	0.154	0.059	0.846	367	30.5%	0.010	1.415	0.095	0.032	0.600
I ota	4	321	3	0.9%	0.010	0.016	0.012	0.011	0.016	9	2.8%	0.013	0.240	0.088	0.058	0.228	1	0.3%	0.018	0.018	0.018	0.018	0.018
J fov	2	446	1	0.2%	0.011	0.011	0.011	0.011	0.011	42	9.4%	0.010	0.190	0.036	0.021	0.169	3	0.7%	0.011	0.074	0.033	0.013	0.071
J wc	5	162	4	2.5%	0.011	0.017	0.013	0.011	0.017	43	26.5%	0.010	0.350	0.089	0.055	0.346	31	19.1%	0.022	0.340	0.124	0.110	0.303
Total	144	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

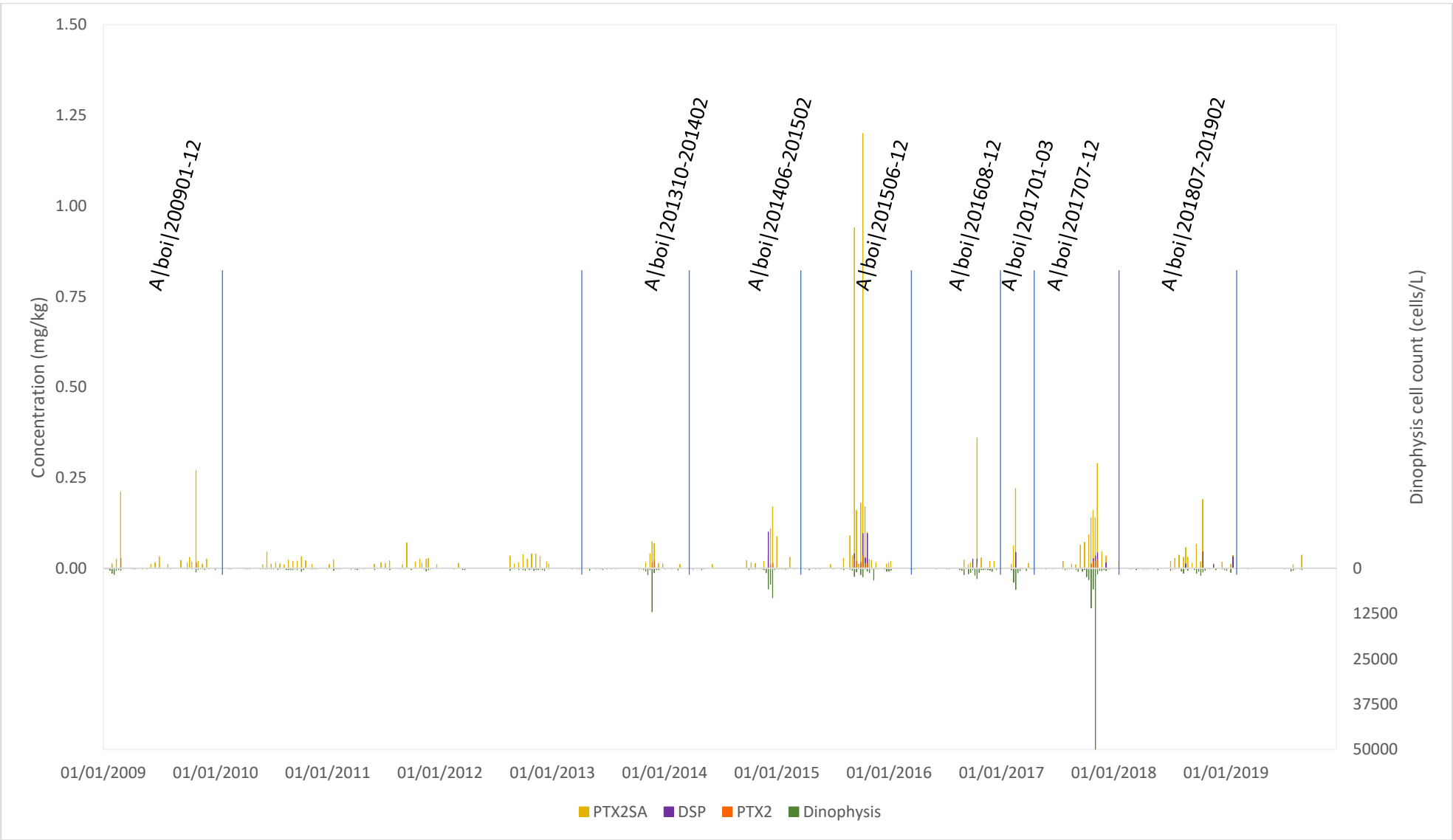


Figure A-1. Classification of bloom event(s) at Zone A|boi, Bay of Island shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

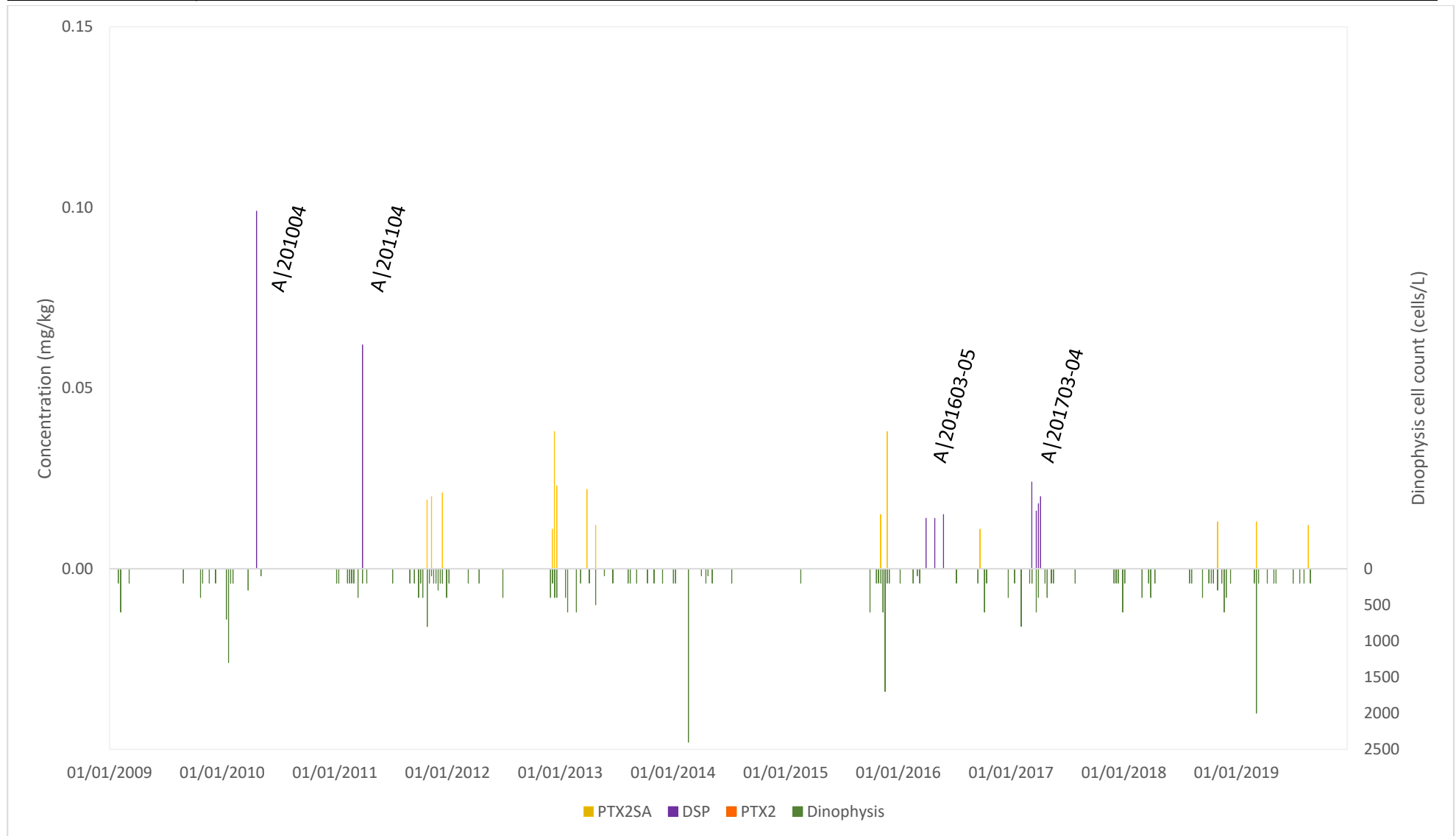


Figure A-2. Classification of bloom event(s) at remaining Zone A shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

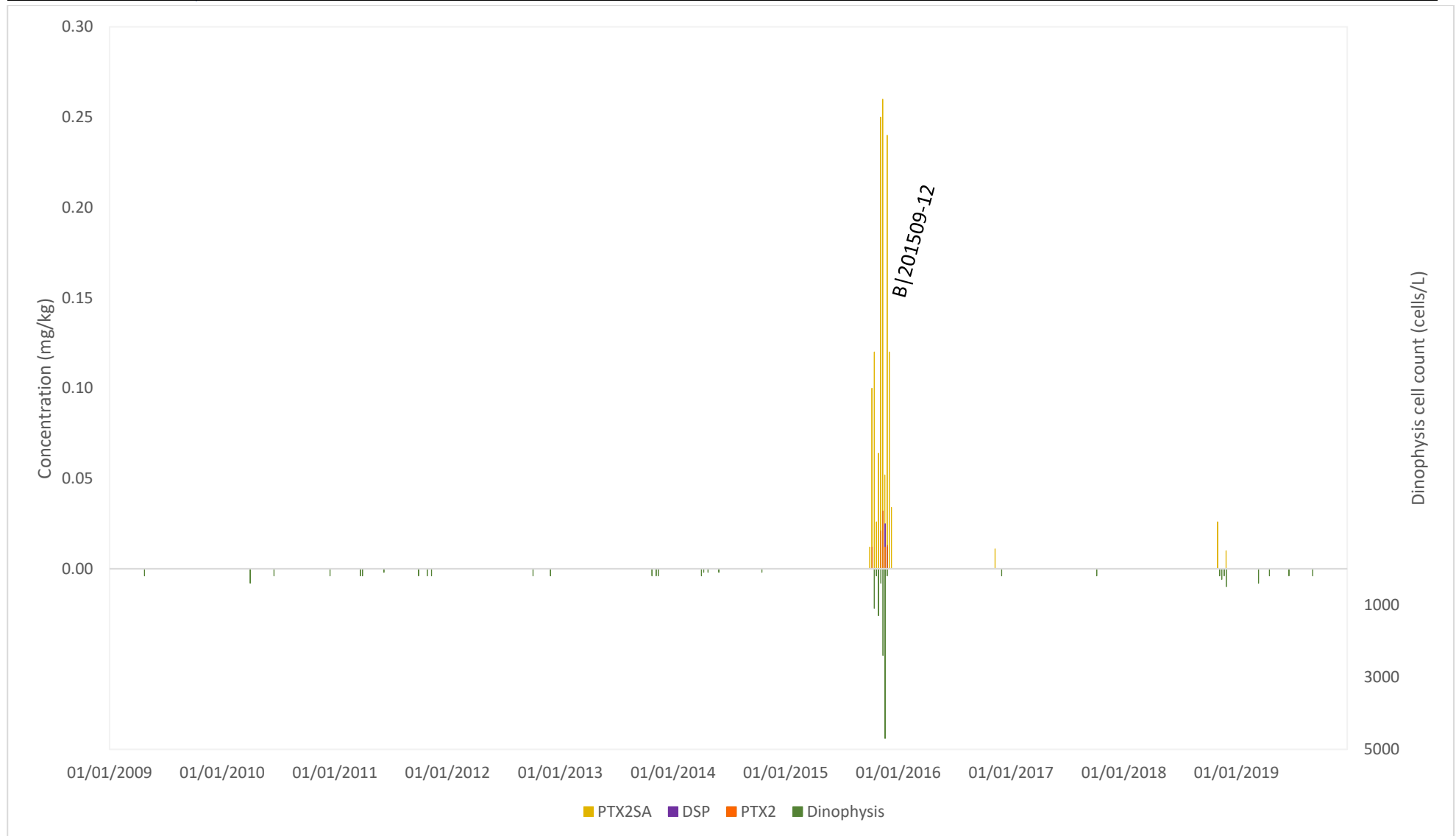


Figure A-3. Classification of bloom event(s) at Zone B shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

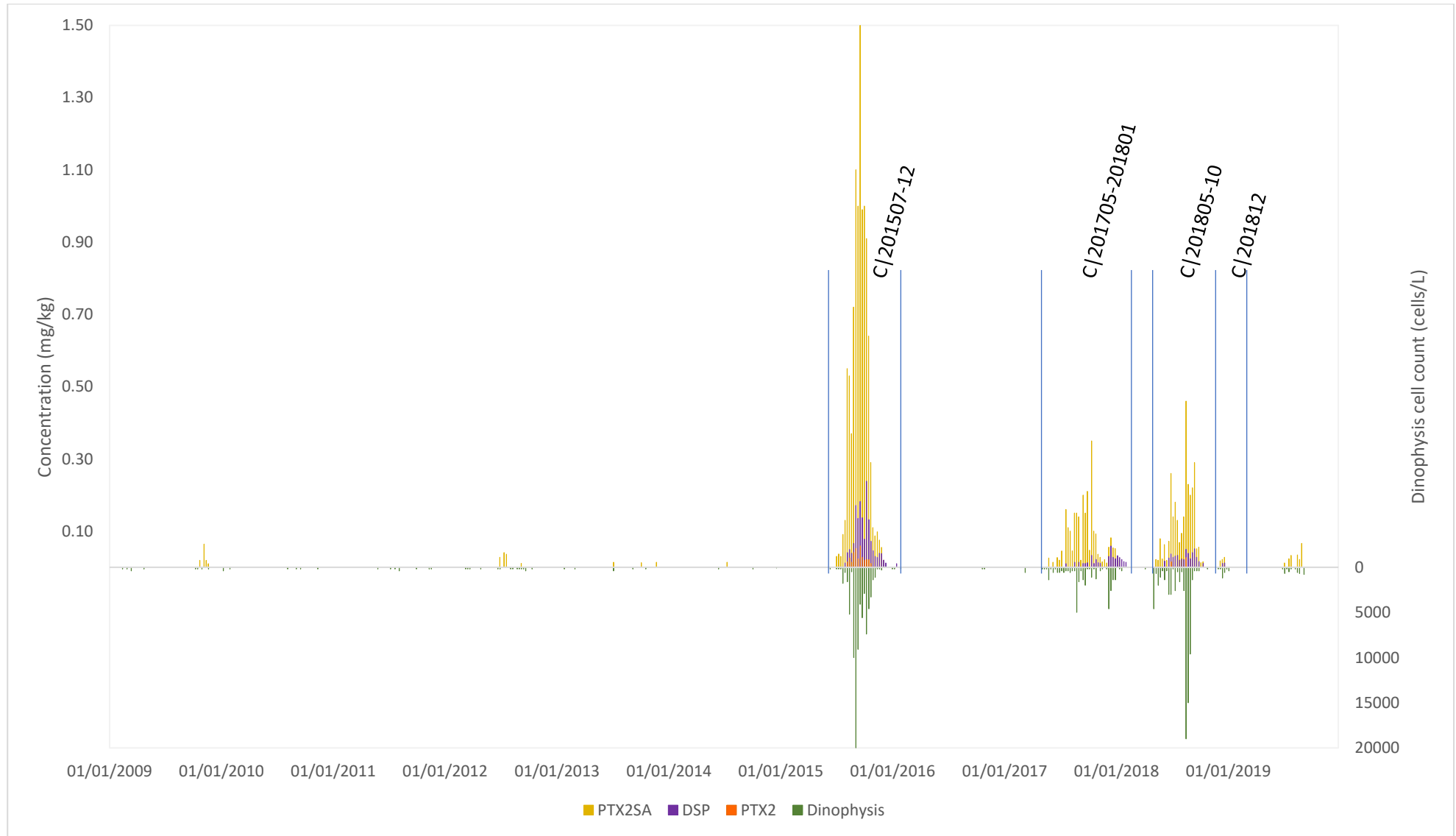


Figure A-4. Classification of bloom event(s) at Zone C shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

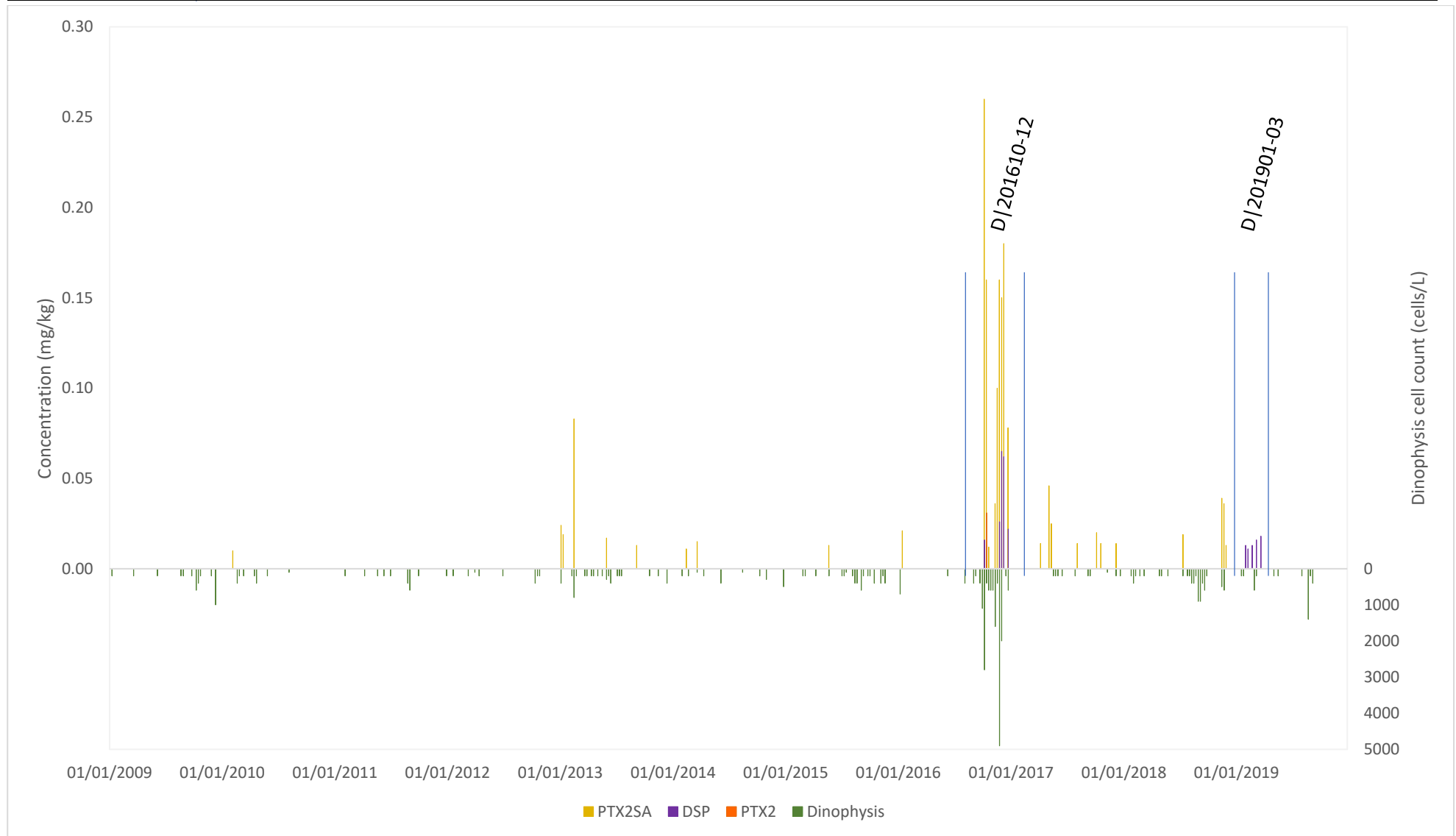


Figure A-5. Classification of bloom event(s) at Zone D shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

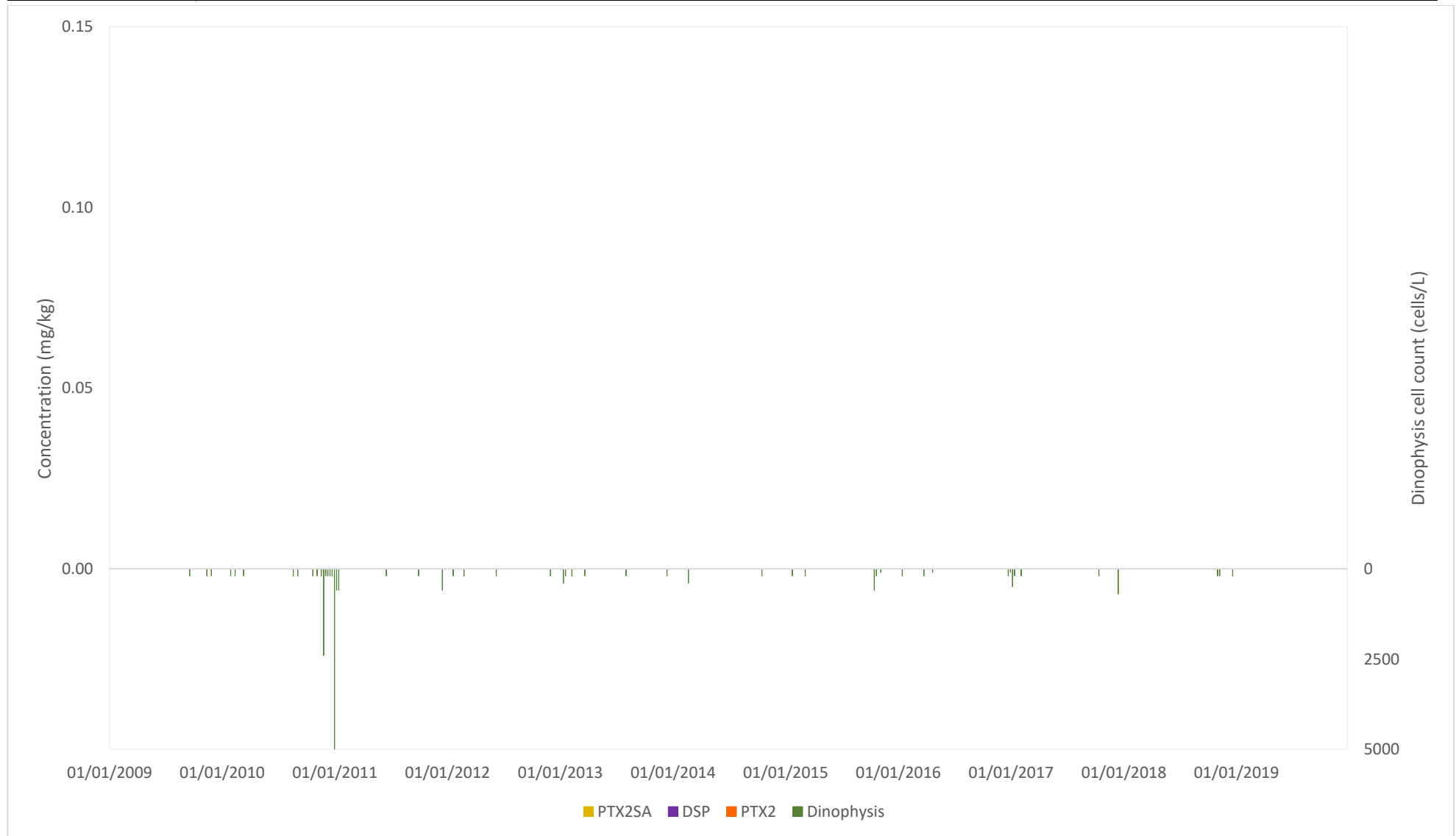


Figure A-6. Classification of bloom event(s) at Zone E shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

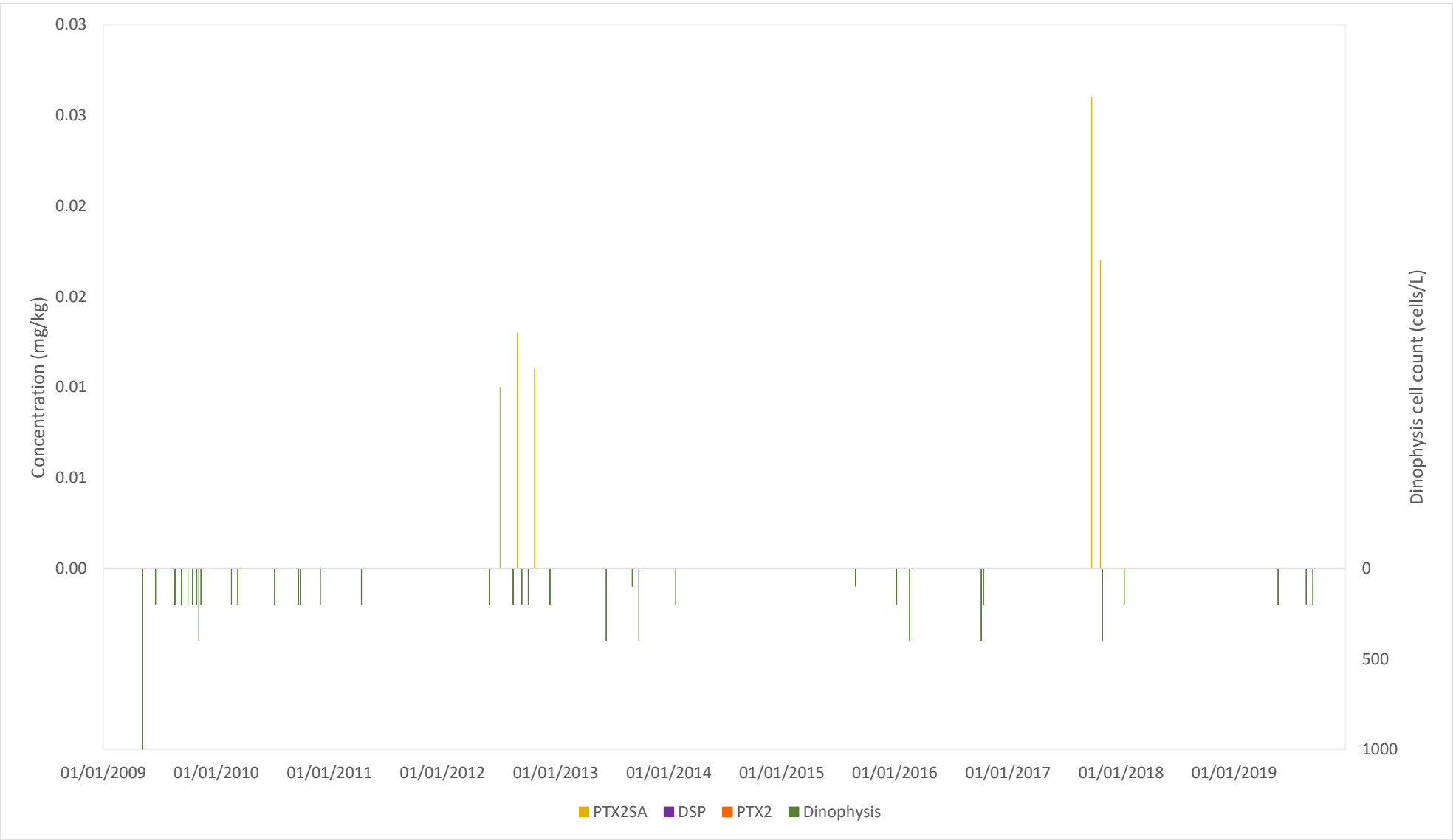


Figure A-7. Classification of bloom event(s) at Zone F shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

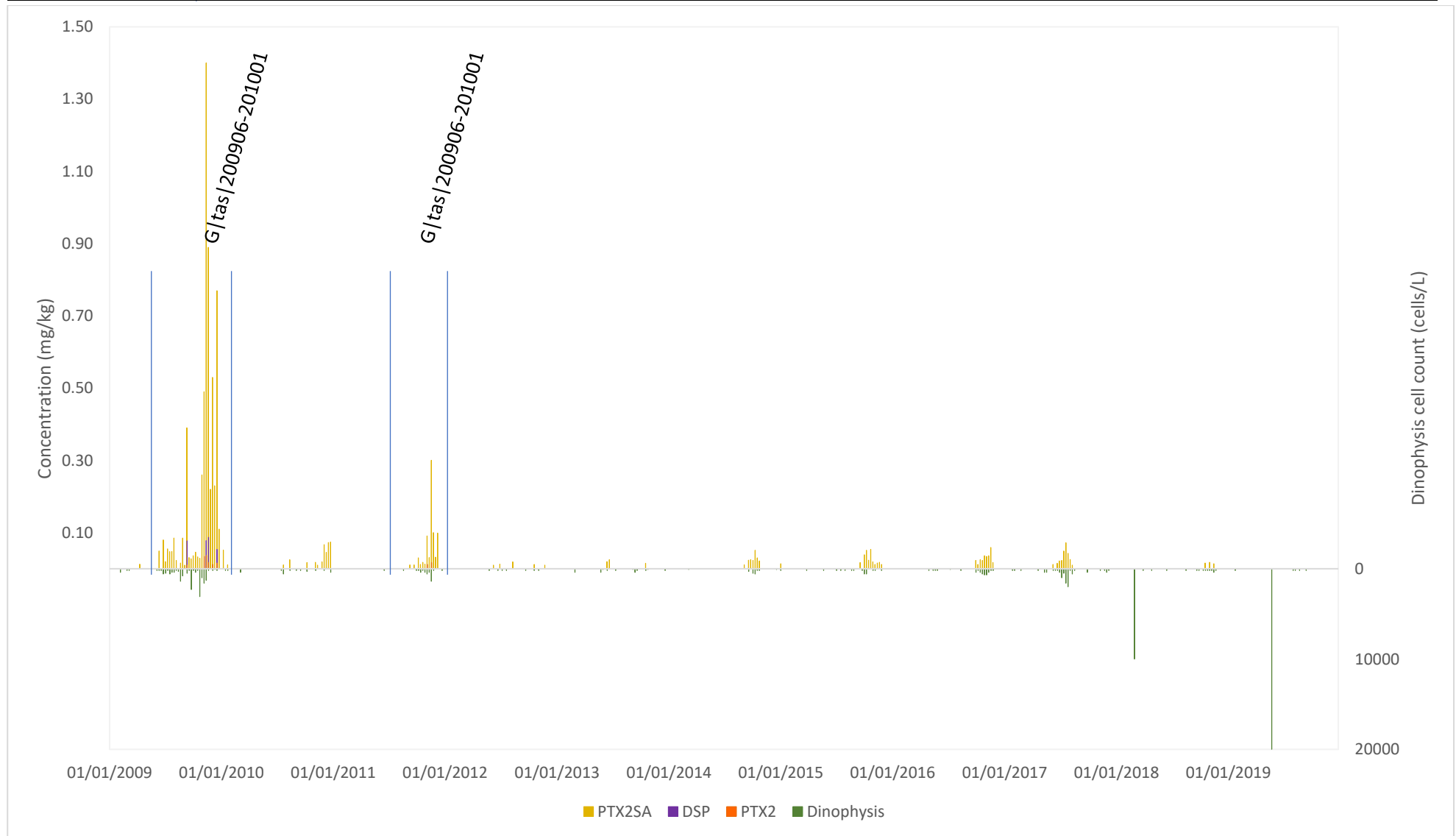


Figure A-8. Classification of bloom event(s) at Zone G|tas, Tasman Bay shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

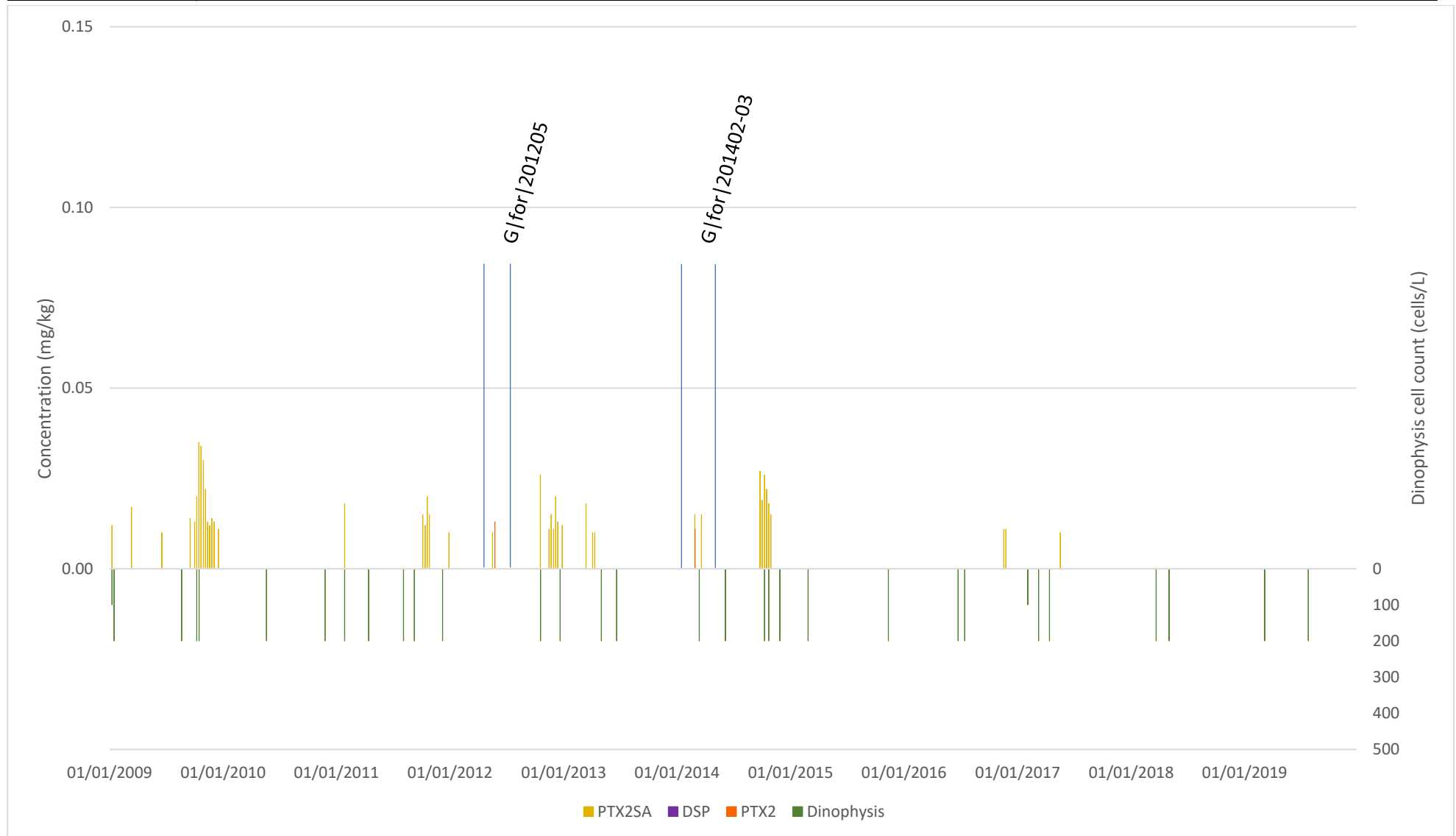


Figure A-9. Classification of bloom event(s) at Zone G|for, Forsyth Island shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

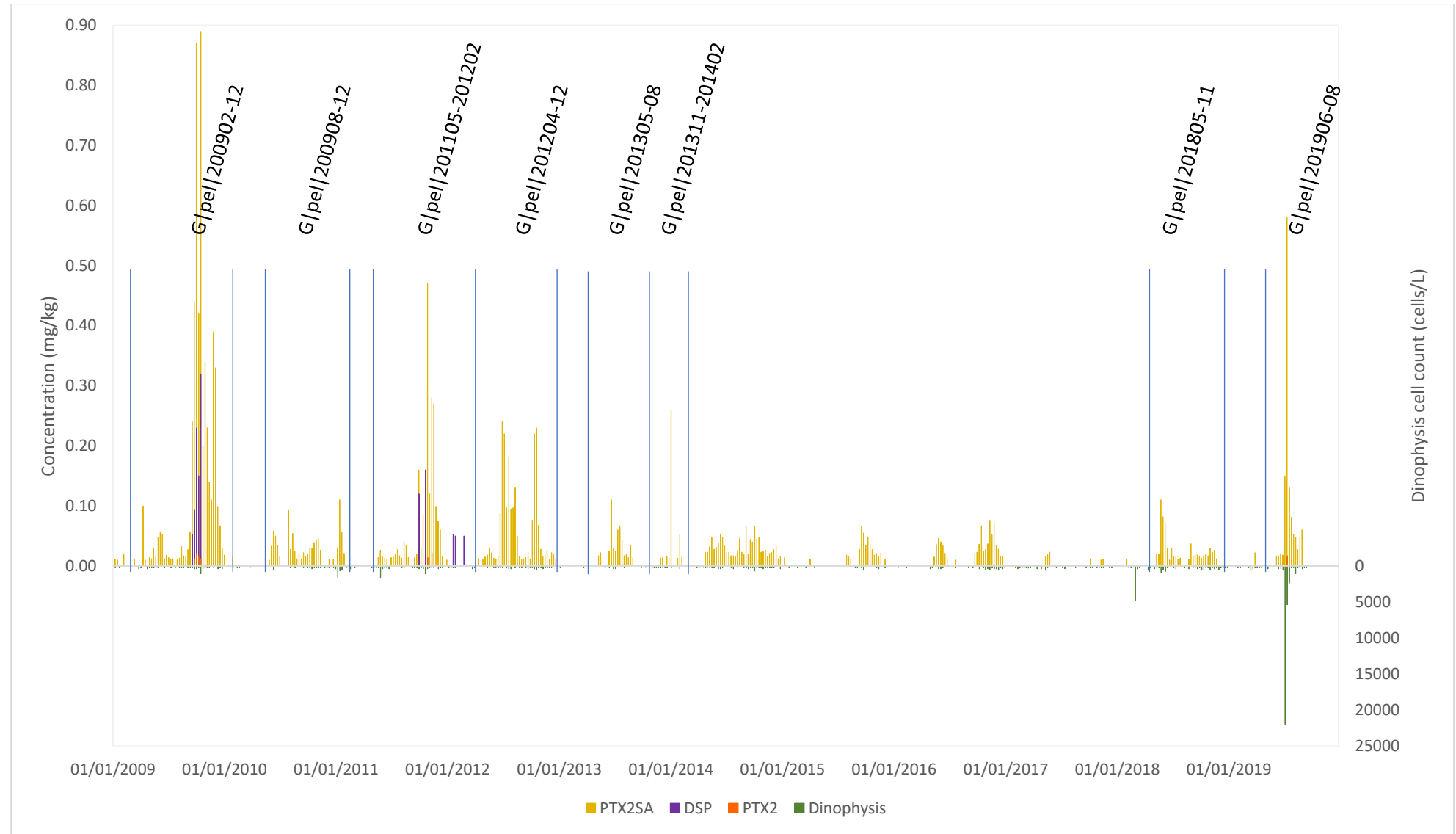


Figure A-10. Classification of bloom event(s) at Zone G|pel, Pelorus Sounds shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

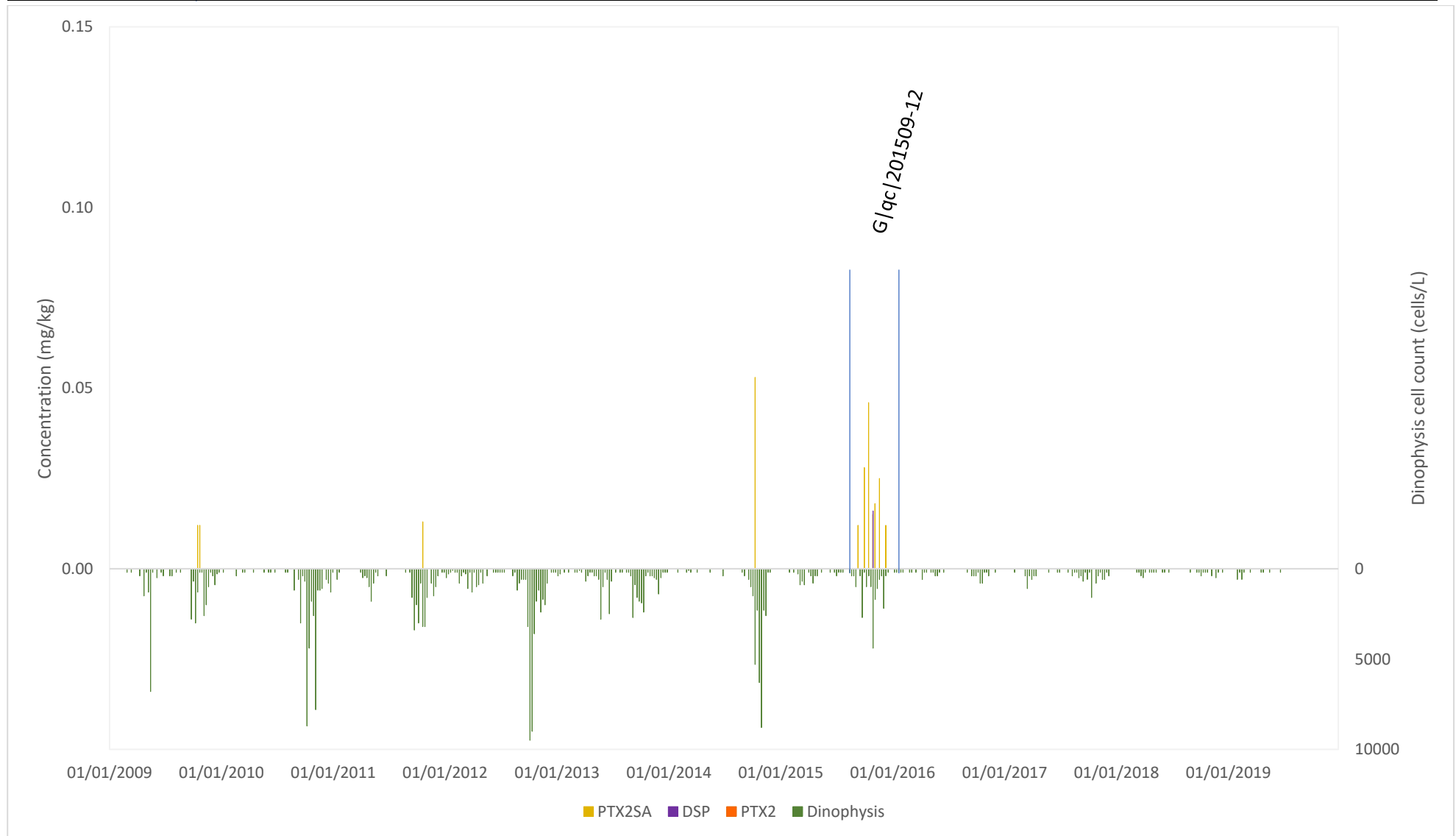


Figure A-11. Classification of bloom event(s) at Zone G|qc, Queen Charlotte Sounds shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

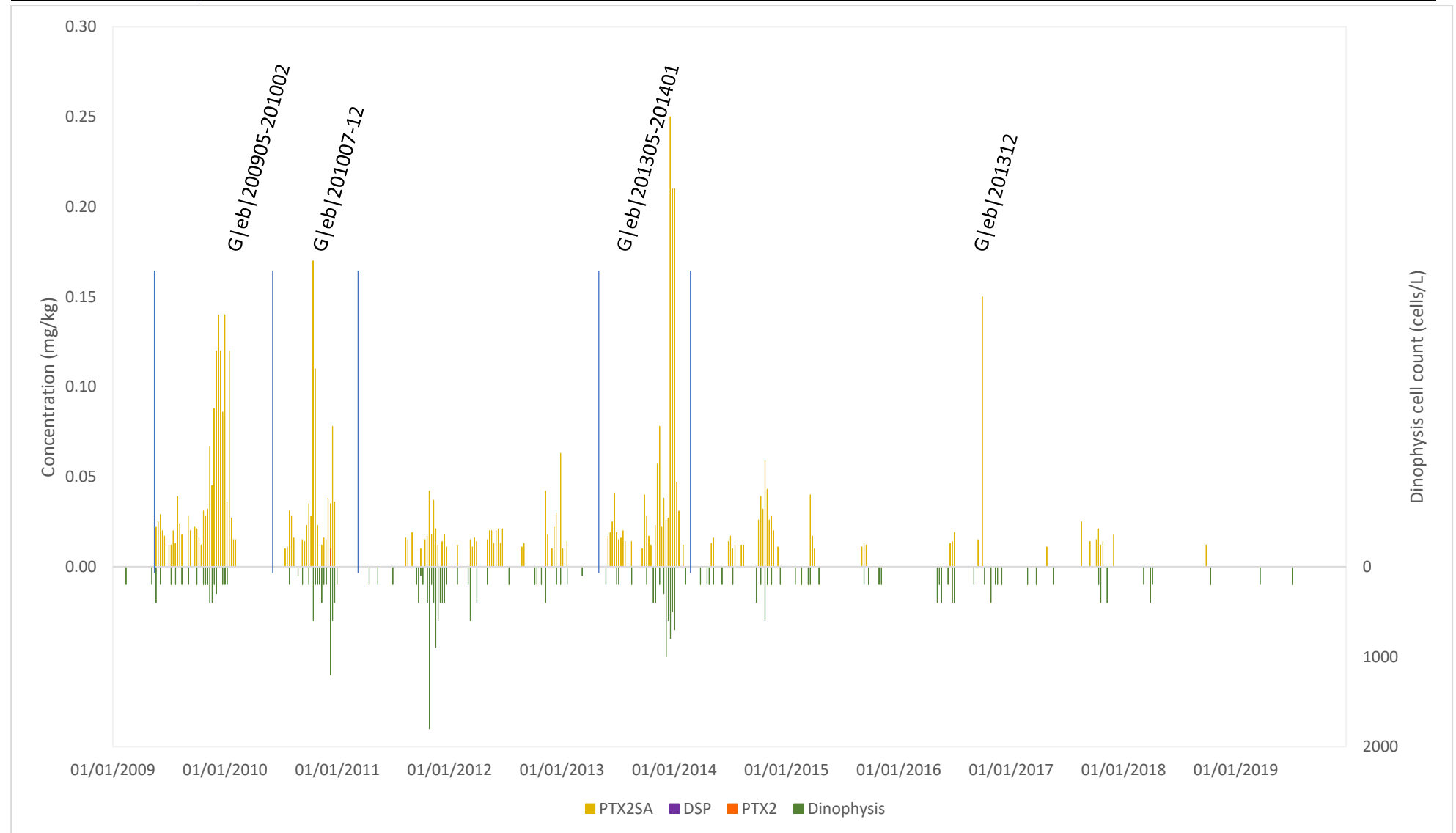


Figure A-12. Classification of bloom event(s) at Zone G|eb, East bay shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

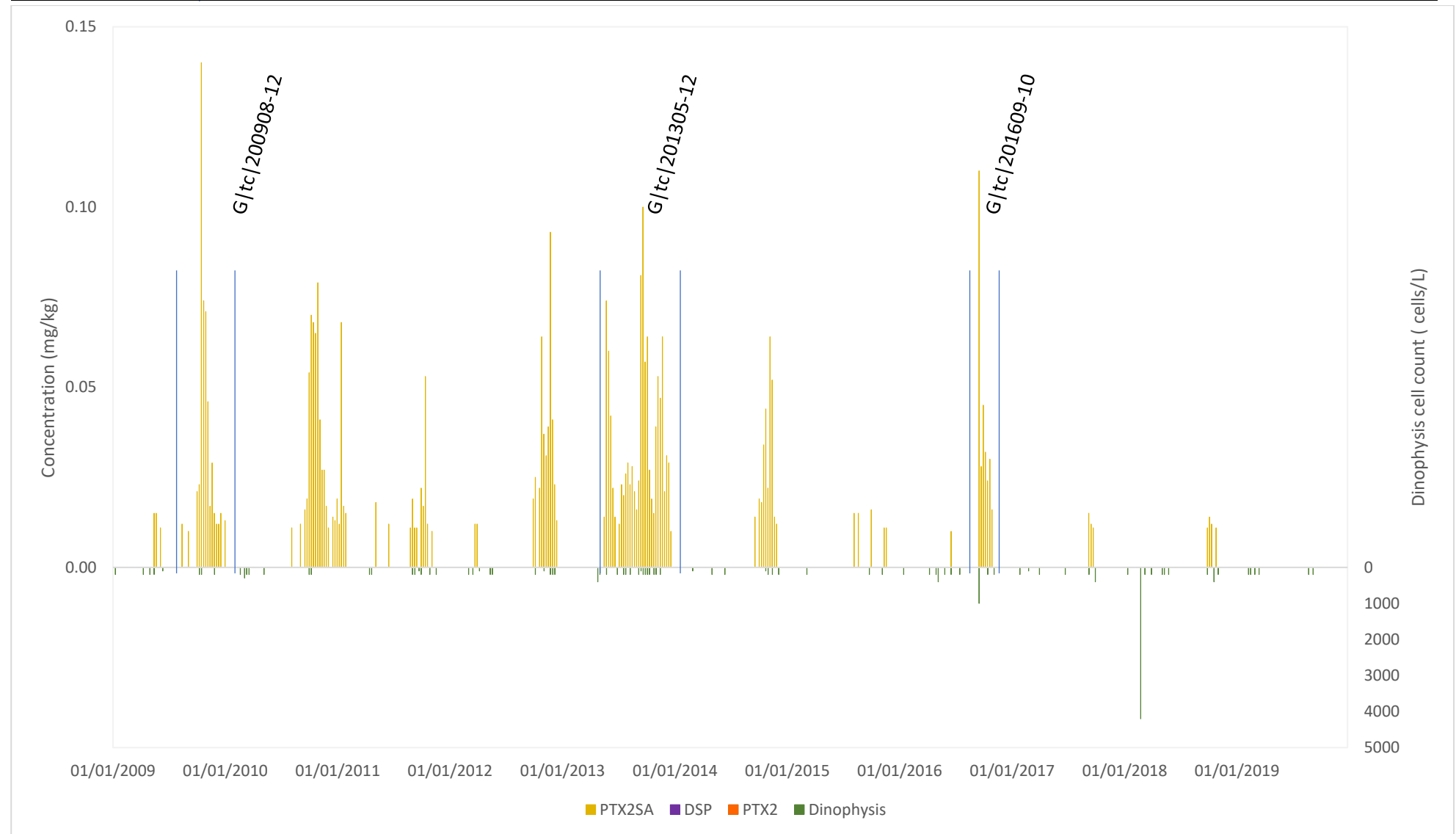


Figure A-13. Classification of bloom event(s) at Zone G|tc, Tory Channel shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

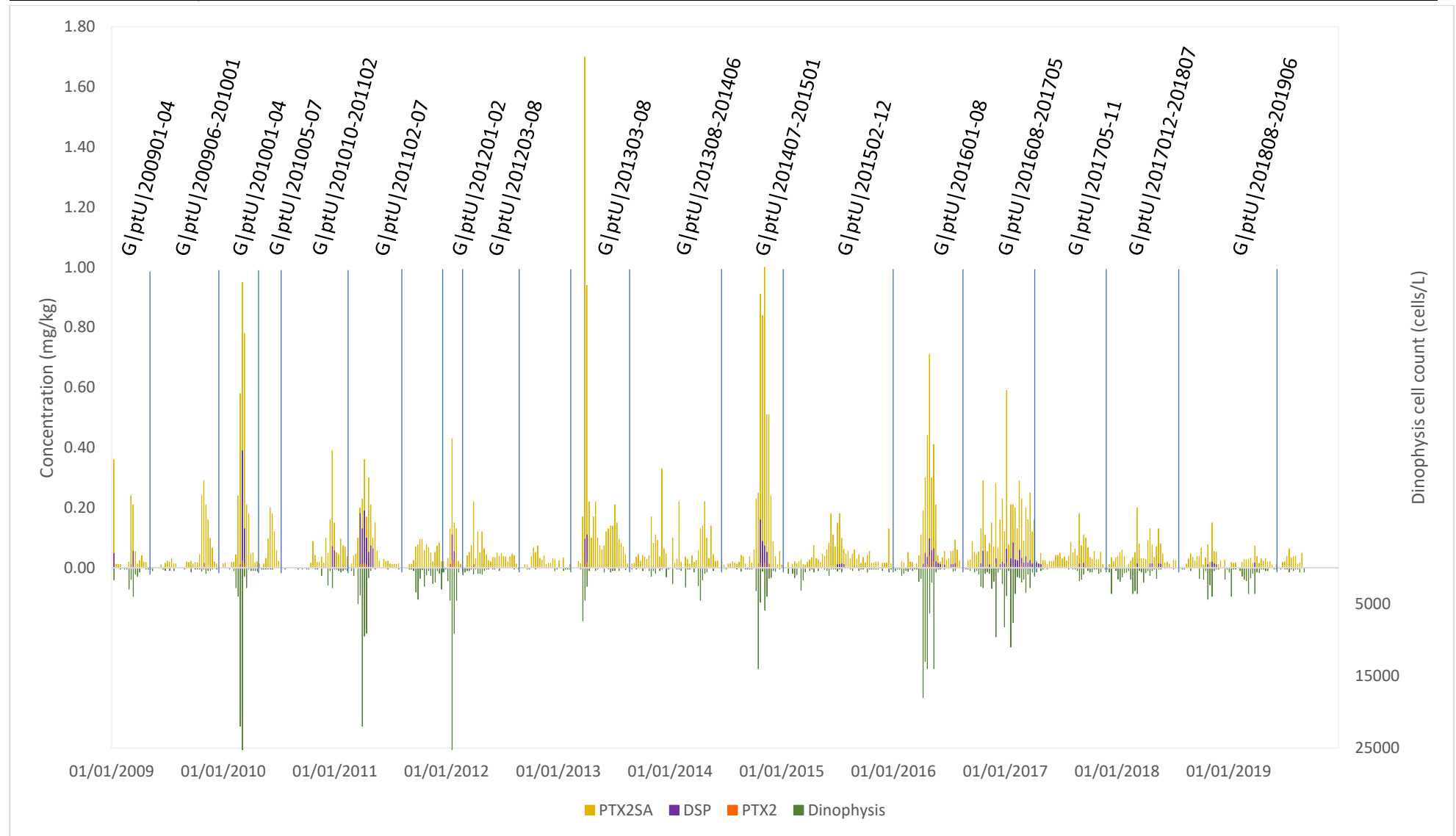


Figure A-14. Classification of bloom event(s) at Zone G|ptU, Port Underwood shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

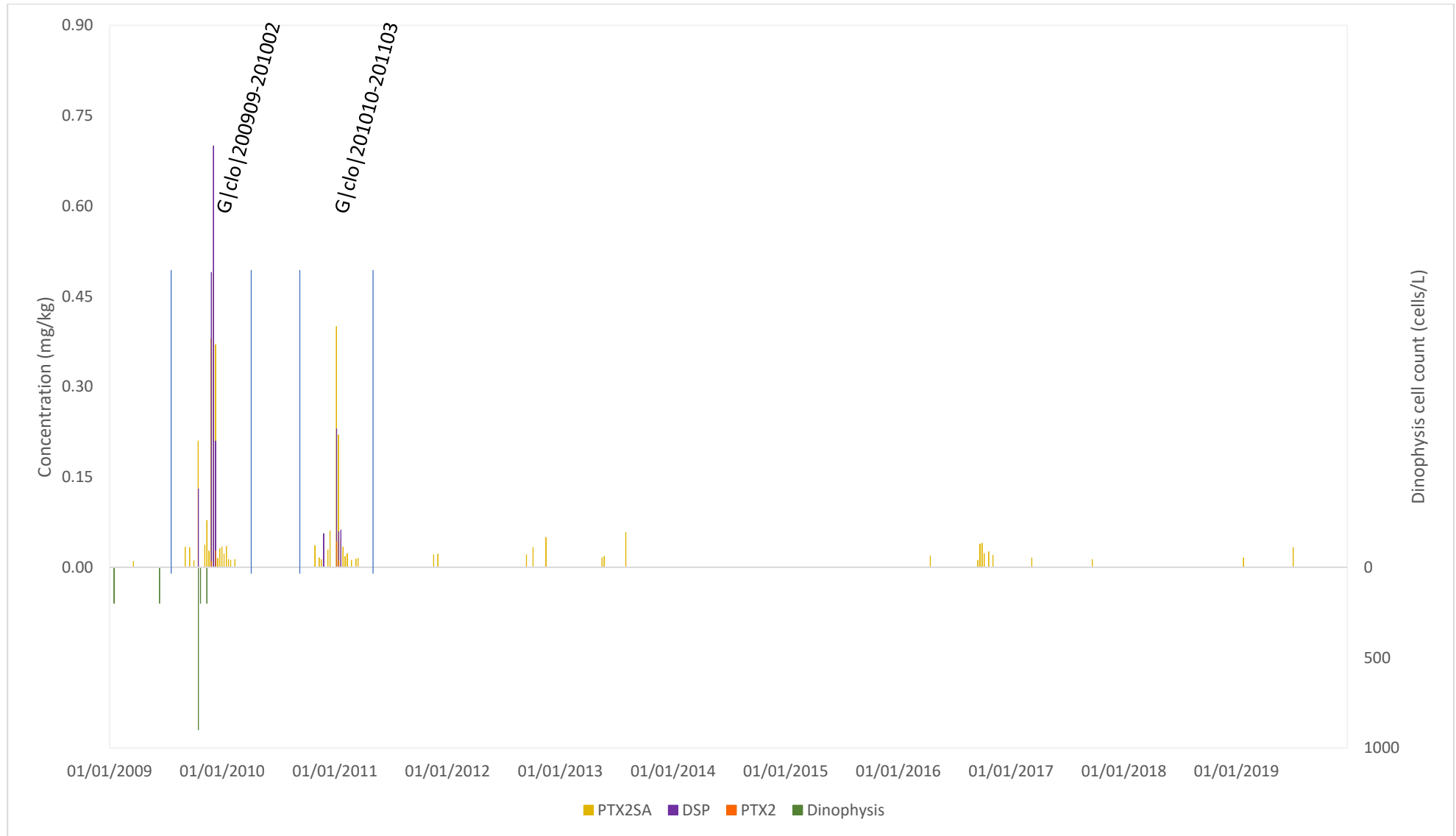


Figure A-15. Classification of bloom event(s) at Zone G|clo, Cloudy bay shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

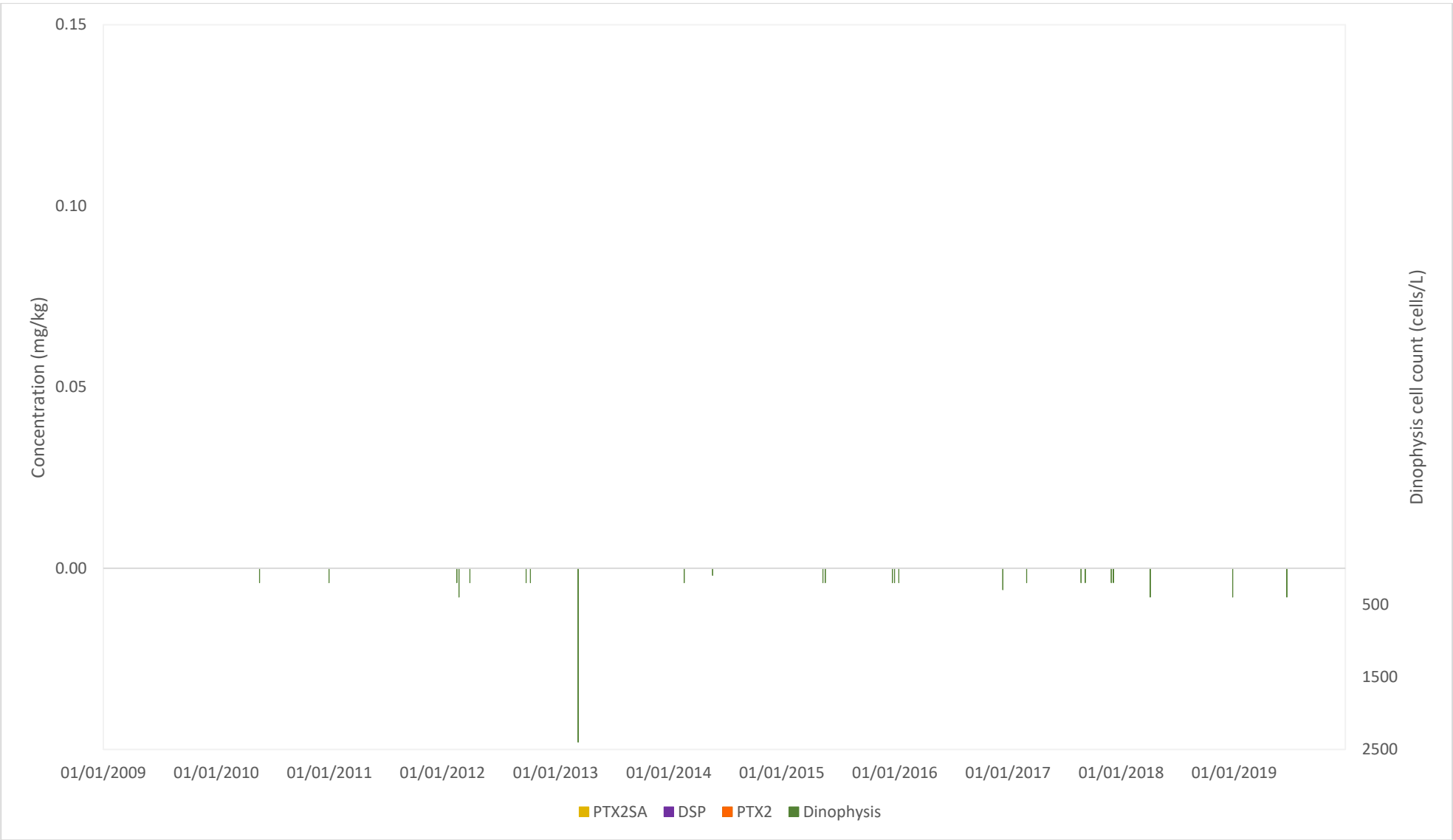


Figure A-16. Classification of bloom event(s) at Zone H, South Taranaki Bight shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

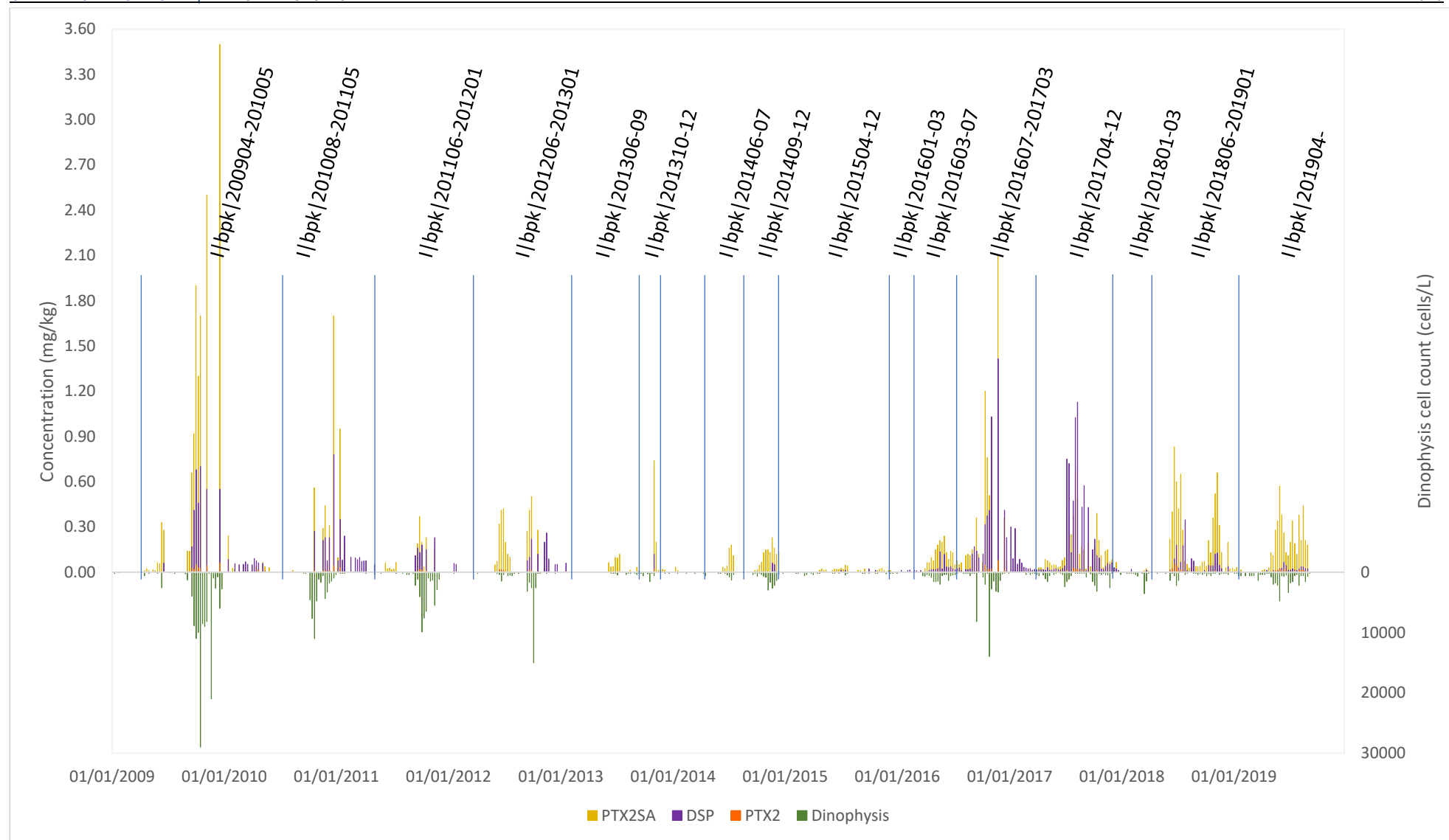


Figure A-17. Classification of bloom event(s) at Zone I|pbk, Banks Peninsula and Kaikoura shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

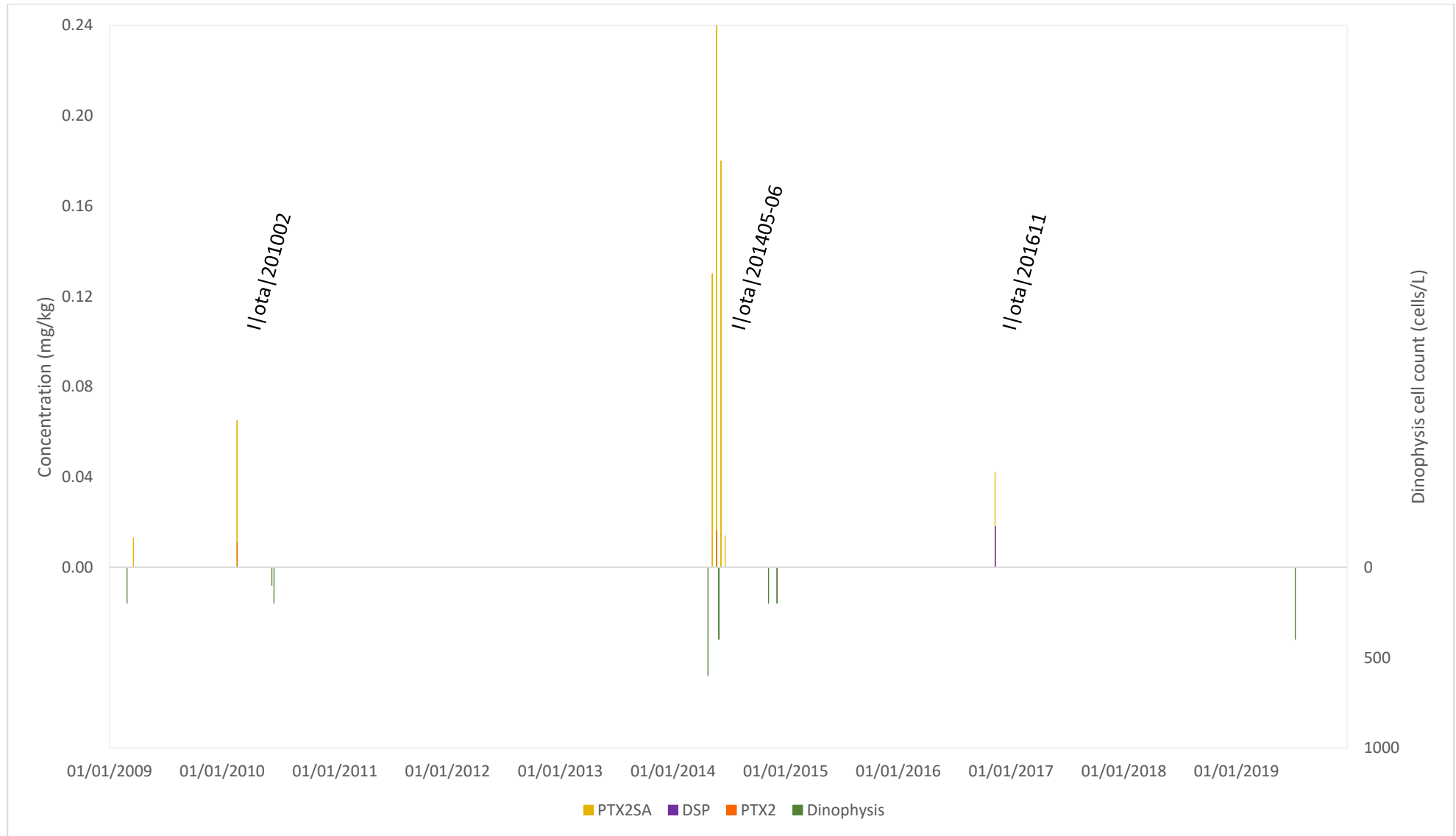


Figure A-18. Classification of bloom event(s) at Zone I/ota, Otago shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

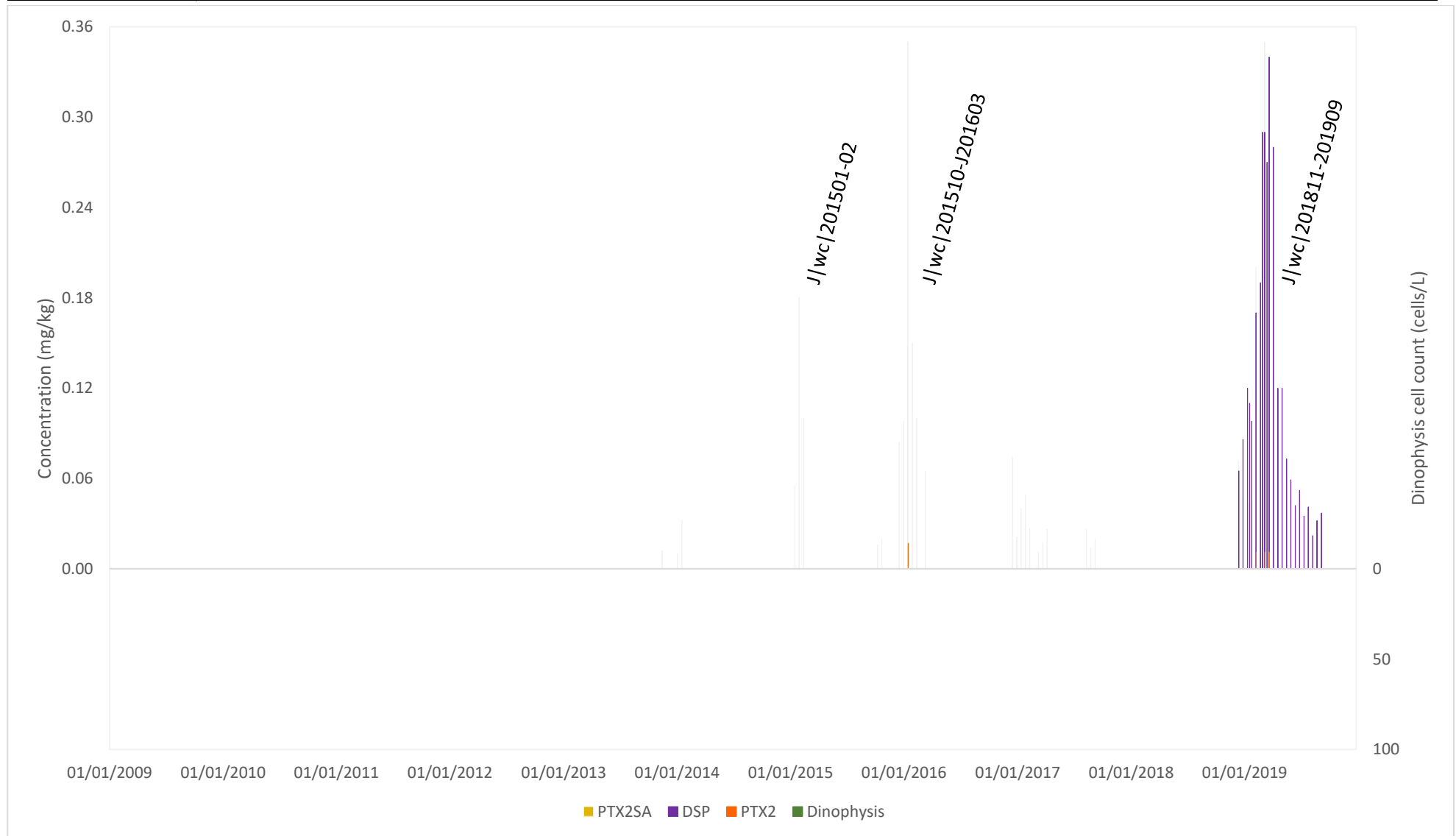


Figure A-19. Classification of bloom event(s) at Zone J|wc, West Coast shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

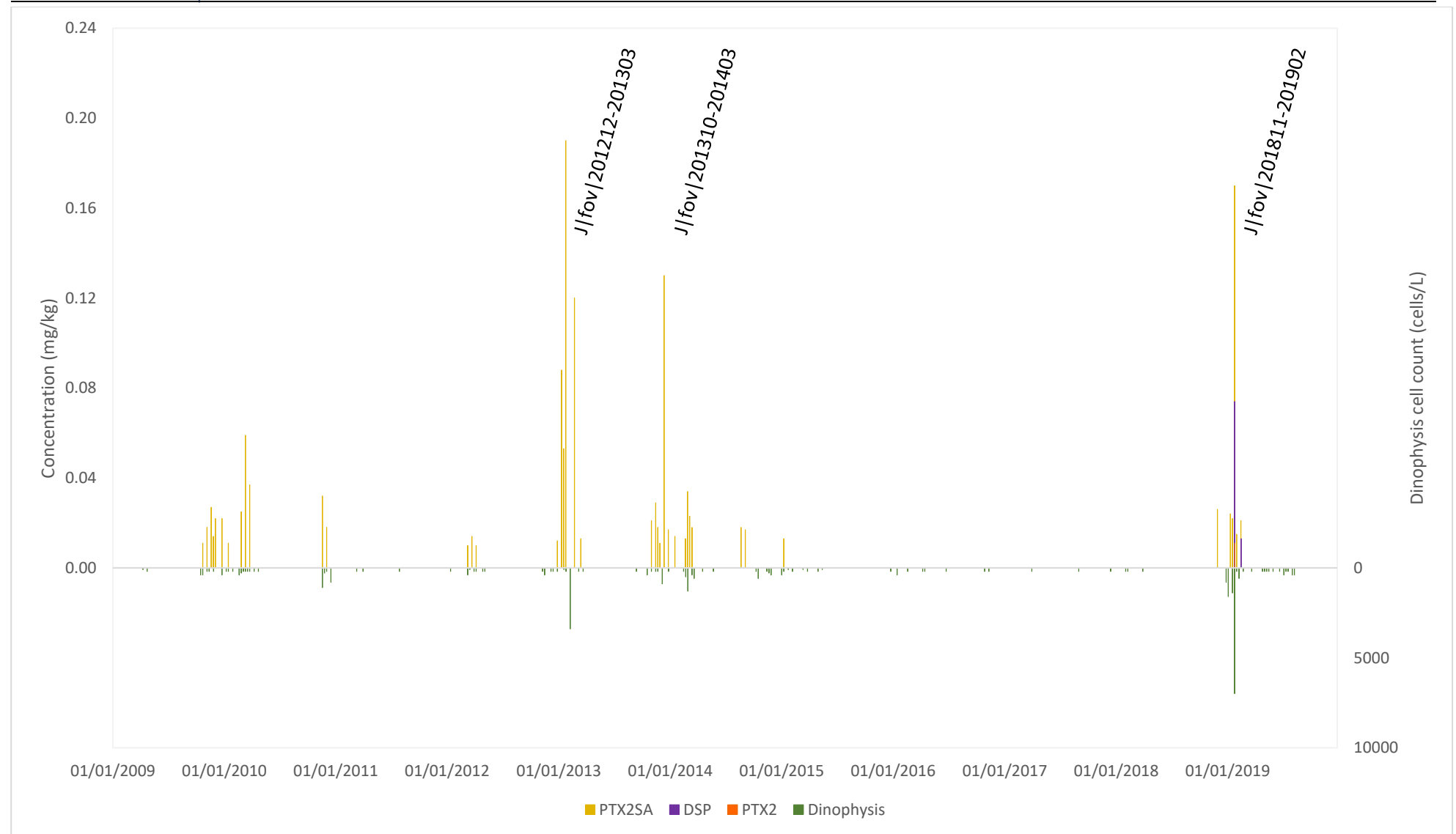


Figure A-20. Classification of bloom event(s) at Zone J/fov, Foveaux Strait shellfish sites. Pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish toxins (top) and dinophysis phytoplankton cell counts (bottom)

APPENDIX B. BLOOM SUMMARY

The number of samples, number of detections, percent detections, min, max, mean, median, 97.5th percentile (PCTL) concentrations each for pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish poisoning toxins for each bloom event are summarised in Table B-1. Summaries detailing the sites affected for each bloom event are summarised in Tables B-2 to B-83.

LIST OF TABLES

Table B-1.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in classified bloom events (Appendix A) within New Zealand 2009-2019.	115
Table B-2.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A 201004 .	119
Table B-3.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A 201104 .	119
Table B-4.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A 201603-05 .	119
Table B-5.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A 201703-04 .	119
Table B-6.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 200901-12 .	119
Table B-7.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201310-201402 .	120
Table B-8.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201406-201502 .	120
Table B-9.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201506-12 .	120
Table B-10.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201608-12 .	121
Table B-11.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201701-03 .	121
Table B-12.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201707-12 .	121
Table B-13.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A boi 201807-201902 .	122
Table B-14.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event B 201509-12 .	122
Table B-15.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event C 201507-12 .	123
Table B-16.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event C 201705-201801 .	123
Table B-17.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event C 201805-10 .	124
Table B-18.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event C 201812 .	124
Table B-19.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event D 201610-12 .	124
Table B-20.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event D 201901-03 .	124
Table B-21.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G clo 200909-201002 .	125
Table B-22.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G clo 201010-201103 .	125
Table B-23.	Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G eb 200905-201002 .	125

Table B-24. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G eb 201007-12	126
Table B-25. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G eb 201305-201401	126
Table B-26. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G for 201205	126
Table B-27. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G for 201402-03	126
Table B-28. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 200902-12	127
Table B-29. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201005-201101	128
Table B-30. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201105-201202	129
Table B-31. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201204-12	130
Table B-32. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201311-201402	130
Table B-33. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201311-201402	131
Table B-34. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201805-11	131
Table B-35. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G pel 201906-08	132
Table B-36. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 200901-04	132
Table B-37. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 200906-201001	132
Table B-38. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201001-04	133
Table B-39. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201005-07	133
Table B-40. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201010-201102	133
Table B-41. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201102-07	134
Table B-42. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201201-02	134
Table B-43. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201203-08	134
Table B-44. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201303-08	135
Table B-45. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201308-201406	135
Table B-46. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201407-201501	135
Table B-47. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201502-12	136
Table B-48. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201601-08	136
Table B-49. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201608-201705	136
Table B-50. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 2017012-201807	137
Table B-51. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201705-11	137
Table B-52. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G ptU 201808-201906	137
Table B-53. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G qc 201509-12	137

Table B-54. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G tas 200906-201001	138
Table B-55. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G tas 201109-12	138
Table B-56. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G tc 200908-12	138
Table B-57. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G tc 201305-12	138
Table B-58. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event G tc 201609-10	139
Table B-59. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 200904-201005	139
Table B-60. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201008-201105	139
Table B-61. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201106-201201	140
Table B-62. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201206-201301	140
Table B-63. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201306-09	140
Table B-64. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201310-12	141
Table B-65. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201406-07	141
Table B-66. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201409-12	141
Table B-67. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201504-12	142
Table B-68. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201601-03	142
Table B-69. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201603-07	142
Table B-70. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201607-201703	143
Table B-71. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201704-12	143
Table B-72. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201801-03	144
Table B-73. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201806-201901	144
Table B-74. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I bpk 201904-	144
Table B-75. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I ota 201002	145
Table B-76. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I ota 201405-06	145
Table B-77. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I ota 201611	145
Table B-78. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J fov 201212-201303	145
Table B-79. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J fov 201310-201403	145
Table B-80. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J fov 201811-201902	146
Table B-81. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J wc 201501-02	146
Table B-82. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J wc 201510-J201603	146
Table B-83. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event J wc 201811-201909	146

Table B-1. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in classified bloom events (Appendix A) within New Zealand 2009-2019.

Bloom	Sites	No Samples	PTX2							PTX2SAs							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A 201004	1	1	0							0							1	100.0%	0.099	0.099	0.099	0.099	0.099
A 201104	1	1	0							0							1	100.0%	0.062	0.062	0.062	0.062	0.062
A 201603-05	1	4	0							0							3	75.0%	0.014	0.015	0.014	0.014	0.015
A 201703-04	1	4	0							0							4	100.0%	0.016	0.024	0.020	0.019	0.024
A boi 200901-12	2	83	25	30.1%	0.010	0.027	0.015	0.015	0.026	71	85.5%	0.011	0.270	0.077	0.067	0.210	0						
A boi 201310-201402	2	13	2	15.4%	0.016	0.021	0.019	0.019	0.021	9	69.2%	0.011	0.074	0.029	0.014	0.073	0						
A boi 201406-201502	3	32	3	9.4%	0.010	0.014	0.012	0.012	0.014	13	40.6%	0.011	0.170	0.048	0.024	0.152	1	3.1%	0.100	0.100	0.100	0.100	0.100
A boi 201506-12	3	33	6	18.2%	0.013	0.034	0.020	0.018	0.033	21	63.6%	0.011	1.200	0.155	0.035	1.070	6	18.2%	0.012	0.096	0.049	0.035	0.096
A boi 201608-12	4	19	1	5.3%	0.013	0.013	0.013	0.013	0.013	11	57.9%	0.010	0.360	0.053	0.020	0.285	2	10.5%	0.026	0.026	0.026	0.026	0.026
A boi 201701-03	2	8	0							6	75.0%	0.011	0.220	0.068	0.045	0.201	2	25.0%	0.017	0.044	0.031	0.031	0.043
A boi 201707-12	2	32	5	15.6%	0.013	0.023	0.018	0.018	0.023	22	68.8%	0.010	0.290	0.064	0.029	0.222	5	15.6%	0.013	0.043	0.026	0.027	0.042
A boi 201807-201902	4	38	0							18	47.4%	0.011	0.190	0.035	0.020	0.137	4	10.5%	0.010	0.045	0.025	0.023	0.044
B 201509-12	3	16	5	31.3%	0.012	0.032	0.018	0.013	0.031	14	87.5%	0.012	0.260	0.105	0.081	0.257	2	12.5%	0.012	0.025	0.019	0.019	0.025
C 201507-12	14	195	55	28.2%	0.010	0.059	0.021	0.016	0.053	168	86.2%	0.010	1.500	0.294	0.165	1.100	145	74.4%	0.010	0.238	0.049	0.038	0.154
C 201705-201801	4	107	0							62	57.9%	0.010	0.350	0.068	0.043	0.205	28	26.2%	0.010	0.059	0.020	0.016	0.041
C 201805-10	4	65	3	4.6%	0.015	0.027	0.022	0.025	0.027	58	89.2%	0.010	0.460	0.096	0.060	0.342	33	50.8%	0.011	0.051	0.025	0.022	0.050
C 201812	3	6	0							4	66.7%	0.018	0.028	0.022	0.022	0.028	2	33.3%	0.011	0.012	0.012	0.012	0.012
D 201610-12	3	18	1	5.6%	0.031	0.031	0.031	0.031	0.031	13	72.2%	0.012	0.260	0.112	0.100	0.236	7	38.9%	0.013	0.065	0.032	0.022	0.065
D 201901-03	1	5	0							0							5	100.0%	0.011	0.018	0.014	0.013	0.018
G clo 200909-201002	3	33	2	6.1%	0.010	0.027	0.019	0.019	0.027	28	84.8%	0.011	0.490	0.100	0.035	0.416	9	27.3%	0.059	0.700	0.224	0.130	0.658

Bloom	Sites	No Samples	PTX2							PTX2SAs							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G clo 201010-201103	2	35	2	5.7%	0.012	0.043	0.028	0.028	0.042	20	57.1%	0.012	0.400	0.060	0.027	0.315	5	14.3%	0.050	0.230	0.091	0.059	0.213
G eb 200905-201002	1	38	0							34	89.5%	0.012	0.140	0.044	0.026	0.140	0						
G eb 201007-12	1	24	1	4.2%	0.010	0.010	0.010	0.010	0.010	20	83.3%	0.010	0.170	0.037	0.026	0.142	0						
G eb 201305-201401	2	37	0							29	78.4%	0.010	0.250	0.051	0.025	0.222	0						
G for 201205	1	2	1	50.0%	0.013	0.013	0.013	0.013	0.013	1	50.0%	0.010	0.010	0.010	0.010	0.010	0						
G for 201402-03	1	3	1	33.3%	0.011	0.011	0.011	0.011	0.011	2	66.7%	0.015	0.015	0.015	0.015	0.015	0						
G pel 200902-12	16	444	5	1.1%	0.010	0.021	0.014	0.012	0.021	205	46.2%	0.010	0.890	0.055	0.019	0.339	6	1.4%	0.052	0.320	0.153	0.122	0.309
G pel 201005-201101	12	391	0							82	21.0%	0.010	0.110	0.022	0.016	0.058	0						
G pel 201105-201202	16	528	3	0.6%	0.010	0.022	0.015	0.014	0.022	105	19.9%	0.010	0.470	0.036	0.017	0.204	5	0.9%	0.050	0.160	0.087	0.053	0.156
G pel 201204-12	14	470	0							138	29.4%	0.010	0.240	0.031	0.016	0.203	0						
G pel 201305-08	5	83	0							24	28.9%	0.010	0.110	0.028	0.019	0.084	0						
G pel 201311-201402	4	31	0							9	29.0%	0.012	0.260	0.045	0.014	0.218	0						
G pel 201805-11	8	215	0							52	24.2%	0.010	0.110	0.022	0.015	0.079	0						
G pel 201906-08	7	64	1	1.6%	0.017	0.017	0.017	0.017	0.017	39	60.9%	0.010	0.580	0.049	0.022	0.171	1	1.6%	0.028	0.028	0.028	0.028	0.028
G ptU 200901-04	3	46	1	2.2%	0.012	0.012	0.012	0.012	0.012	23	50.0%	0.010	0.360	0.066	0.020	0.294	2	4.3%	0.047	0.055	0.051	0.051	0.055
G ptU 200906-201001	3	93	2	2.2%	0.013	0.016	0.015	0.015	0.016	46	49.5%	0.010	0.290	0.057	0.020	0.236	0						
G ptU 201001-04	3	41	4	9.8%	0.010	0.023	0.015	0.013	0.022	29	70.7%	0.011	0.950	0.175	0.053	0.831	5	12.2%	0.050	0.390	0.164	0.130	0.370
G ptU 201005-07	3	25	0							16	64.0%	0.010	0.200	0.053	0.022	0.193	0						
G ptU 201010-201102	3	53	0							38	71.7%	0.010	0.390	0.062	0.038	0.279	4	7.5%	0.050	0.071	0.058	0.056	0.070
G ptU 201102-07	4	82	6	7.3%	0.010	0.015	0.012	0.012	0.015	57	69.5%	0.011	0.360	0.077	0.040	0.312	11	13.4%	0.052	0.190	0.105	0.081	0.188
G ptU 201201-02	3	23	4	17.4%	0.012	0.025	0.018	0.018	0.025	17	73.9%	0.012	0.430	0.095	0.040	0.366	2	8.7%	0.054	0.110	0.082	0.082	0.109
G ptU 201203-08	3	70	1	1.4%	0.011	0.011	0.011	0.011	0.011	45	64.3%	0.010	0.220	0.042	0.034	0.147	0						

Bloom	Sites	No Samples	PTX2							PTX2SAs							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G ptU 201303-08	5	75	2	2.7%	0.010	0.019	0.015	0.015	0.019	54	72.0%	0.010	1.700	0.123	0.053	0.761	3	4.0%	0.052	0.110	0.086	0.096	0.109
G ptU 201308-201406	3	127	0							76	59.8%	0.010	0.330	0.047	0.028	0.220	0						
G ptU 201407-201501	5	104	5	4.8%	0.012	0.016	0.014	0.014	0.016	61	58.7%	0.010	1.000	0.163	0.060	0.875	4	3.8%	0.052	0.160	0.093	0.081	0.155
G ptU 201502-12	3	141	0							71	50.4%	0.010	0.180	0.037	0.020	0.158	5	3.5%	0.010	0.014	0.011	0.011	0.014
G ptU 201601-08	4	94	6	6.4%	0.010	0.039	0.020	0.018	0.037	63	67.0%	0.010	0.710	0.115	0.043	0.522	26	27.7%	0.010	0.097	0.032	0.021	0.096
G ptU 201608-201705	3	113	3	2.7%	0.013	0.015	0.014	0.015	0.015	83	73.5%	0.011	0.590	0.086	0.053	0.290	29	25.7%	0.010	0.083	0.025	0.018	0.068
G ptU 2017012-201807	3	93	0							43	46.2%	0.011	0.200	0.042	0.030	0.130	6	6.5%	0.012	0.014	0.013	0.014	0.014
G ptU 201705-11	3	76	0							45	59.2%	0.011	0.180	0.041	0.029	0.109	4	5.3%	0.010	0.015	0.013	0.013	0.015
G ptU 201808-201906	3	96	0							54	56.3%	0.010	0.150	0.029	0.022	0.075	5	5.2%	0.010	0.020	0.014	0.014	0.020
G qc 201509-12	1	7	0							7	100.0%	0.012	0.046	0.022	0.018	0.043	1	14.3%	0.016	0.016	0.016	0.016	0.016
G tas 200906-201001	3	58	9	15.5%	0.010	0.037	0.021	0.017	0.036	48	82.8%	0.010	1.400	0.203	0.050	0.887	5	8.6%	0.054	0.087	0.074	0.077	0.086
G tas 201109-12	3	21	4	19.0%	0.010	0.017	0.014	0.014	0.017	19	90.5%	0.011	0.300	0.067	0.030	0.282	0						
G tc 200908-12	2	24	0							16	66.7%	0.010	0.140	0.033	0.016	0.115	0						
G tc 201305-12	3	68	0							47	69.1%	0.010	0.100	0.031	0.026	0.080	0						
G tc 201609-10	2	13	0							10	76.9%	0.012	0.110	0.042	0.029	0.108	0						
I bpbk 200904-201005	4	70	14	20.0%	0.011	0.063	0.027	0.022	0.059	38	54.3%	0.010	3.500	0.494	0.070	2.575	25	35.7%	0.050	0.700	0.216	0.090	0.688
I bpbk 201008-201105	4	39	6	15.4%	0.010	0.041	0.019	0.012	0.040	13	33.3%	0.011	1.700	0.386	0.290	1.475	23	59.0%	0.050	0.780	0.161	0.095	0.544
I bpbk 201106-201201	3	33	5	15.2%	0.010	0.019	0.014	0.015	0.019	31	93.9%	0.017	0.370	0.103	0.074	0.310	23	69.7%	0.053	0.230	0.096	0.073	0.203
I bpbk 201206-201301	5	36	7	19.4%	0.011	0.017	0.013	0.012	0.017	21	58.3%	0.013	0.500	0.204	0.200	0.460	12	33.3%	0.051	0.260	0.113	0.083	0.249
I bpbk 201306-09	5	31	0							19	61.3%	0.010	0.120	0.041	0.034	0.110	0						
I bpbk 201310-12	4	18	1	5.6%	0.020	0.020	0.020	0.020	0.020	10	55.6%	0.011	0.740	0.112	0.017	0.619	1	5.6%	0.120	0.120	0.120	0.120	0.120
I bpbk 201406-07	3	15	0							10	66.7%	0.017	0.180	0.067	0.037	0.176	0						

Bloom	Sites	No Samples	PTX2							PTX2SAs							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I bpk 201409-12	4	33	0							28	84.8%	0.012	0.230	0.086	0.069	0.183	3	9.1%	0.051	0.060	0.054	0.051	0.060
I bpk 201504-12	4	74	0							36	48.6%	0.010	0.047	0.018	0.015	0.041	4	5.4%	0.011	0.023	0.015	0.012	0.022
I bpk 201601-03	1	10	0							0							6	60.0%	0.010	0.015	0.012	0.012	0.015
I bpk 201603-07	6	57	0							51	89.5%	0.015	0.240	0.089	0.077	0.218	38	66.7%	0.010	0.135	0.026	0.019	0.119
I bpk 201607-201703	11	76	11	14.5%	0.011	0.079	0.032	0.022	0.079	44	57.9%	0.011	2.100	0.305	0.099	1.755	66	86.8%	0.010	1.415	0.148	0.054	0.764
I bpk 201704-12	7	139	7	5.0%	0.010	0.027	0.016	0.013	0.026	113	81.3%	0.010	0.670	0.077	0.033	0.396	87	62.6%	0.010	1.126	0.092	0.019	0.746
I bpk 201801-03	3	15	0							6	40.0%	0.010	0.024	0.015	0.013	0.023	2	13.3%	0.013	0.019	0.016	0.016	0.019
I bpk 201806-201901	5	87	9	10.3%	0.011	0.058	0.024	0.016	0.055	65	74.7%	0.013	0.830	0.174	0.064	0.654	37	42.5%	0.010	0.346	0.060	0.042	0.196
I bpk 201904-	4	82	14	17.1%	0.010	0.024	0.014	0.014	0.023	66	80.5%	0.011	0.570	0.157	0.125	0.415	40	48.8%	0.010	0.065	0.019	0.016	0.045
I ota 201002	1	3	2	66.7%	0.010	0.011	0.011	0.011	0.011	3	100.0%	0.052	0.065	0.058	0.058	0.065	0						
I ota 201405-06	1	4	1	25.0%	0.016	0.016	0.016	0.016	0.016	4	100.0%	0.014	0.240	0.141	0.155	0.236	0						
I ota 201611	1	1	0							1	100.0%	0.042	0.042	0.042	0.042	0.042	1	100.0%	0.018	0.018	0.018	0.018	0.018
J fov 201212-201303	1	8	0							6	75.0%	0.012	0.190	0.079	0.071	0.181	0						
J fov 201310-201403	1	14	0							11	78.6%	0.011	0.130	0.030	0.018	0.106	0						
J fov 201811-201902	1	11	1	9.1%	0.011	0.011	0.011	0.011	0.011	7	63.6%	0.015	0.170	0.044	0.024	0.149	3	27.3%	0.011	0.074	0.033	0.013	0.071
J wc 201501-02	1	3	0							3	100.0%	0.055	0.180	0.112	0.100	0.176	0						
J wc 201510-J201603	1	12	1	8.3%	0.017	0.017	0.017	0.017	0.017	8	66.7%	0.016	0.350	0.110	0.091	0.315	0						
J wc 201811-201909	5	31	3	9.7%	0.011	0.011	0.011	0.011	0.011	18	58.1%	0.015	0.350	0.124	0.086	0.316	31	100.0%	0.022	0.340	0.124	0.110	0.303
Unclassified	114	13424	0							865	6.4%	0.010	0.095	0.022	0.017	0.067	0						
Total	144	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

Table B-2. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|201004**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A001	1	0							0							1	100.0%	0.099	0.099	0.099	0.099	0.099
Subtotal	1	0							0							1	100.0%	0.099	0.099	0.099	0.099	0.099

Table B-3. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|201104**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A002	1	0							0							1	100.0%	0.062	0.062	0.062	0.062	0.062
Subtotal	1	0							0							1	100.0%	0.062	0.062	0.062	0.062	0.062

Table B-4. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|201603-05**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A002	4	0							0							3	75.0%	0.014	0.015	0.014	0.014	0.015
Subtotal	4	0							0							3	75.0%	0.014	0.015	0.014	0.014	0.015

Table B-5. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|201703-04**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A346	4	0							0							4	100.0%	0.016	0.024	0.020	0.019	0.024
Subtotal	4	0							0							4	100.0%	0.016	0.024	0.020	0.019	0.024

Table B-6. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|boi|200901-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	82	25	30.5%	0.010	0.027	0.015	0.015	0.026	70	85.4%	0.011	0.270	0.078	0.068	0.210	0						
A015	1	0							1	100.0%	0.017	0.017	0.017	0.017	0.017	0						
Subtotal	83	25	30.5%	0.010	0.027	0.015	0.015	0.026	71	85.5%	0.011	0.270	0.077	0.067	0.210	0						

Table B-7. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201310-201402.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	2	0							2	100.0%	0.011	0.016	0.014	0.014	0.016	0						
A015	11	2	18.2%	0.016	0.021	0.019	0.019	0.021	7	63.6%	0.011	0.074	0.033	0.014	0.073	0						
Subtotal	13	2	15.4%	0.016	0.021	0.019	0.019	0.021	9	69.2%	0.011	0.074	0.029	0.014	0.073	0						

Table B-8. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201406-201502.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	10	1	10.0%	0.014	0.014	0.014	0.014	0.014	5	50.0%	0.014	0.170	0.051	0.019	0.157	0						
A015	21	2	9.5%	0.010	0.012	0.011	0.011	0.012	7	33.3%	0.011	0.110	0.049	0.031	0.107	1	4.8%	0.100	0.100	0.100	0.100	0.100
A016	1	0							1	100.0%	0.024	0.024	0.024	0.024	0.024	0						
Subtotal	32	3	9.4%	0.010	0.014	0.012	0.012	0.014	13	40.6%	0.011	0.170	0.048	0.024	0.152	1	3.1%	0.100	0.100	0.100	0.100	0.100

Table B-9. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201506-12.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	14	0							8	57.1%	0.012	0.100	0.042	0.025	0.098	0						
A015	16	4	25.0%	0.013	0.019	0.016	0.016	0.019	10	62.5%	0.011	0.180	0.069	0.032	0.178	3	18.8%	0.012	0.029	0.020	0.020	0.029
A030	3	2	66.7%	0.025	0.034	0.030	0.030	0.034	3	100.0%	0.098	1.200	0.746	0.940	1.187	3	100.0%	0.040	0.096	0.077	0.096	0.096
Subtotal	33	6	18.2%	0.013	0.034	0.020	0.018	0.033	21	63.6%	0.011	1.200	0.155	0.035	1.070	6	18.2%	0.012	0.096	0.049	0.035	0.096

Table B-10. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201608-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	8	0							4	50.0%	0.020	0.059	0.031	0.022	0.056	0						
A015	9	0							6	66.7%	0.010	0.029	0.017	0.015	0.028	0						
A030	1	1	100.0%	0.013	0.013	0.013	0.013	0.013	1	100.0%	0.360	0.360	0.360	0.360	0.360	1	100.0%	0.026	0.026	0.026	0.026	0.026
A277	1	0							0							1	100.0%	0.026	0.026	0.026	0.026	0.026
Subtotal	19	1	5.3%	0.013	0.013	0.013	0.013	0.013	11	57.9%	0.010	0.360	0.053	0.020	0.285	2	10.5%	0.026	0.026	0.026	0.026	0.026

Table B-11. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201701-03.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A015	6	0							4	66.7%	0.011	0.071	0.031	0.021	0.068	1	16.7%	0.017	0.017	0.017	0.017	0.017
A040	2	0							2	100.0%	0.062	0.220	0.141	0.141	0.216	1	50.0%	0.044	0.044	0.044	0.044	0.044
Subtotal	8	0							6	75.0%	0.011	0.220	0.068	0.045	0.201	2	25.0%	0.017	0.044	0.031	0.031	0.043

Table B-12. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event A|boi|201707-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	15	2	13%	0.018	0.023	0.021	0.021	0.023	9	60.0%	0.015	0.290	0.072	0.029	0.264	2	13.3%	0.013	0.043	0.028	0.028	0.042
A015	17	3	18%	0.013	0.020	0.017	0.018	0.020	13	76.5%	0.010	0.140	0.058	0.034	0.140	3	17.6%	0.015	0.034	0.025	0.027	0.034
Subtotal	32	5	16%	0.013	0.023	0.018	0.018	0.023	22	68.8%	0.010	0.290	0.064	0.029	0.222	5	15.6%	0.013	0.043	0.026	0.027	0.042

Table B-13. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **A|boi|201807-201902**.

Table D-16: Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diamethoc shellfish poisoning toxins (mg/kg) in bloom event A1501201007-201502.																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A014	14	0							5	35.7%	0.011	0.037	0.026	0.031	0.036	0						
A015	19	0							8	42.1%	0.011	0.066	0.023	0.018	0.059	0						
A018	1	0							1	100.0%	0.012	0.012	0.012	0.012	0.012	0						
A030	4	0							4	100.0%	0.013	0.190	0.074	0.047	0.180	4	100.0%	0.010	0.045	0.025	0.023	0.044
Subtotal	38	0							18	47.4%	0.011	0.190	0.035	0.020	0.137	4	10.5%	0.010	0.045	0.025	0.023	0.044

Table B-14. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **B|201509-12**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
B007	4	0							2	50.0%	0.046	0.097	0.072	0.072	0.096	0						
B015B	1	1	100.0%	0.012	0.012	0.012	0.012	0.012	1	100.0%	0.052	0.052	0.052	0.052	0.052	1	100.0%	0.025	0.025	0.025	0.025	0.025
B024B	11	4	36.4%	0.012	0.032	0.020	0.017	0.031	11	100.0%	0.012	0.260	0.115	0.100	0.258	1	9.1%	0.012	0.012	0.012	0.012	0.012
Subtotal	16	5	31.3%	0.012	0.032	0.018	0.013	0.031	14	87.5%	0.012	0.260	0.105	0.081	0.257	2	12.5%	0.012	0.025	0.019	0.019	0.025

Table B-15. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **C|201507-12**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
C002	12	5	41.7%	0.010	0.036	0.019	0.016	0.034	12	100.0%	0.020	1.100	0.325	0.190	1.048	9	75.0%	0.010	0.120	0.037	0.028	0.106
C003	3	0							2	66.7%	0.017	0.017	0.017	0.017	0.017	1	33.3%	0.010	0.010	0.010	0.010	0.010
C004	3	0							3	100.0%	0.011	0.054	0.025	0.011	0.052	1	33.3%	0.013	0.013	0.013	0.013	0.013
C009	31	8	25.8%	0.011	0.022	0.015	0.014	0.022	27	87.1%	0.010	0.590	0.197	0.200	0.564	25	80.6%	0.011	0.078	0.037	0.037	0.071
C029	33	11	33.3%	0.010	0.041	0.022	0.020	0.040	27	81.8%	0.020	1.000	0.375	0.230	0.968	27	81.8%	0.010	0.152	0.059	0.048	0.149
C038	29	5	17.2%	0.010	0.014	0.012	0.012	0.014	22	75.9%	0.016	0.480	0.158	0.091	0.449	16	55.2%	0.012	0.039	0.024	0.025	0.038
C041	4	0							3	75.0%	0.010	0.016	0.013	0.013	0.016	1	25.0%	0.011	0.011	0.011	0.011	0.011
C056	3	0							1	33.3%	0.022	0.022	0.022	0.022	0.022	0						
C059	11	4	36.4%	0.011	0.036	0.021	0.019	0.035	11	100.0%	0.063	1.100	0.377	0.290	1.075	11	100.0%	0.019	0.171	0.069	0.061	0.159
C060	15	6	40.0%	0.012	0.059	0.024	0.017	0.055	15	100.0%	0.060	1.300	0.341	0.160	1.195	15	100.0%	0.017	0.238	0.072	0.046	0.203
C061	12	5	41.7%	0.014	0.032	0.021	0.017	0.031	12	100.0%	0.036	1.200	0.452	0.380	1.142	10	83.3%	0.012	0.083	0.045	0.041	0.083
C063	25	4	16.0%	0.015	0.054	0.029	0.024	0.052	19	76.0%	0.012	1.400	0.268	0.066	1.184	15	60.0%	0.011	0.090	0.033	0.022	0.080
C065	1	0							1	100.0%	0.220	0.220	0.220	0.220	0.220	1	100.0%	0.025	0.025	0.025	0.025	0.025
C323	13	7	53.8%	0.010	0.052	0.026	0.019	0.051	13	100.0%	0.063	1.500	0.489	0.480	1.344	13	100.0%	0.020	0.182	0.080	0.078	0.175
Subtotal	195	55	28.2%	0.010	0.059	0.021	0.016	0.053	168	86.2%	0.010	1.500	0.294	0.165	1.100	145	74.4%	0.010	0.238	0.049	0.038	0.154

Table B-16. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **C|201705-201801**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
C009	27	0							20	74.1%	0.017	0.210	0.087	0.073	0.196	13	48.1%	0.011	0.059	0.020	0.014	0.050
C029	29	0							20	69.0%	0.010	0.350	0.084	0.053	0.265	15	51.7%	0.010	0.033	0.020	0.021	0.033
C038	28	0							13	46.4%	0.012	0.200	0.042	0.027	0.154	0						
C063	23	0							9	39.1%	0.011	0.066	0.031	0.029	0.063	0						
Subtotal	107	0							62	57.9%	0.010	0.350	0.068	0.043	0.205	28	26.2%	0.010	0.059	0.020	0.016	0.041

Table B-17. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **C|201805-10**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
C009	20	2	10.0%	0.015	0.027	0.021	0.021	0.027	18	90.0%	0.017	0.460	0.126	0.101	0.375	15	75.0%	0.011	0.050	0.026	0.022	0.048
C029	17	1	5.9%	0.025	0.025	0.025	0.025	0.025	17	100.0%	0.015	0.380	0.133	0.094	0.344	15	88.2%	0.013	0.051	0.026	0.024	0.047
C038	18	0							17	94.4%	0.010	0.190	0.043	0.028	0.146	2	11.1%	0.018	0.020	0.019	0.019	0.020
C063	10	0							6	60.0%	0.010	0.180	0.046	0.022	0.161	1	10.0%	0.016	0.016	0.016	0.016	0.016
Subtotal	65	3	4.6%	0.015	0.027	0.022	0.025	0.027	58	89.2%	0.010	0.460	0.096	0.060	0.342	33	50.8%	0.011	0.051	0.025	0.022	0.050

Table B-18. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **C|201812**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
C009	2	0							1	50.0%	0.018	0.018	0.018	0.018	0.018	0						
C029	2	0							2	100.0%	0.021	0.022	0.022	0.022	0.022	2	100.0%	0.011	0.012	0.012	0.012	0.012
C063	2	0							1	50.0%	0.028	0.028	0.028	0.028	0.028	0						
Subtotal	6	0							4	66.7%	0.018	0.028	0.022	0.022	0.028	2	33.3%	0.011	0.012	0.012	0.012	0.012

Table B-19. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **D|201610-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
D001	7	1	14.3%	0.031	0.031	0.031	0.031	0.031	6	85.7%	0.015	0.260	0.119	0.100	0.248	2	28.6%	0.013	0.016	0.015	0.015	0.016
D009	10	0							6	60.0%	0.012	0.180	0.103	0.114	0.178	4	40.0%	0.018	0.065	0.042	0.042	0.065
D010	1	0							1	100.0%	0.130	0.130	0.130	0.130	0.130	1	100.0%	0.026	0.026	0.026	0.026	0.026
Subtotal	18	1	5.6%	0.031	0.031	0.031	0.031	0.031	13	72.2%	0.012	0.260	0.112	0.100	0.236	7	38.9%	0.013	0.065	0.032	0.022	0.065

Table B-20. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **D|201901-03**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
D001	5	0							0							5	100.0%	0.011	0.018	0.014	0.013	0.018
Subtotal	5	0							0							5	100.0%	0.011	0.018	0.014	0.013	0.018

Table B-21. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|clo|200909-201002**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G020	18	2	11.1%	0.010	0.027	0.019	0.019	0.027	14	77.8%	0.011	0.490	0.175	0.125	0.454	9	50.0%	0.059	0.700	0.224	0.130	0.658
G030	1	0							1	100.0%	0.015	0.015	0.015	0.015	0.015	0						
G115	14	0							13	92.9%	0.012	0.040	0.026	0.027	0.039	0						
Subtotal	33	2	6.1%	0.010	0.027	0.019	0.019	0.027	28	84.8%	0.011	0.490	0.100	0.035	0.416	9	27.3%	0.059	0.700	0.224	0.130	0.658

Table B-22. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|clo|201010-201103**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G030	16	1	6.3%	0.012	0.012	0.012	0.012	0.012	3	18.8%	0.015	0.220	0.086	0.024	0.210	0						
G115	19	1	5.3%	0.043	0.043	0.043	0.043	0.043	17	89.5%	0.012	0.400	0.055	0.029	0.274	5	26.3%	0.050	0.230	0.091	0.059	0.213
Subtotal	35	2	5.7%	0.012	0.043	0.028	0.028	0.042	20	57.1%	0.012	0.400	0.060	0.027	0.315	5	14.3%	0.050	0.230	0.091	0.059	0.213

Table B-23. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|eb|200905-201002**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G019	38	0							34	89.5%	0.012	0.140	0.044	0.026	0.140	0						
Subtotal	38	0							34	89.5%	0.012	0.140	0.044	0.026	0.140	0						

Table B-24. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|eb|201007-12**.

Table D-24: Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diametric shellfish poisoning toxins (mg/kg) in bloom event 0125/2010/12.																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G019	24	1	4.2%	0.010	0.010	0.010	0.010	0.010	20	83.3%	0.010	0.170	0.037	0.026	0.142	0						
Subtotal	24	1	4.2%	0.010	0.010	0.010	0.010	0.010	20	83.3%	0.010	0.170	0.037	0.026	0.142	0						

Table B-25. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|eb|201305-201401**.

Table D-28: Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diametric shenish poisoning toxins (mg/kg) in bloom event 0125/201000-201401.																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G019	36	0							28	77.8%	0.010	0.250	0.048	0.024	0.223	0						
G110	1	0							1	100.0%	0.150	0.150	0.150	0.150	0.150	0						
Subtotal	37	0							29	78.4%	0.010	0.250	0.051	0.025	0.222	0						

Table B-26. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|for|201205**.

Table D-26: Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diametric shellfish poisoning toxins (mg/kg) in bloom event 01/01/2012-05/01/2012																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G017	2	1	50.0%	0.013	0.013	0.013	0.013	0.013	1	50.0%	0.010	0.010	0.010	0.010	0.010	0						
Subtotal	2	1	50.0%	0.013	0.013	0.013	0.013	0.013	1	50.0%	0.010	0.010	0.010	0.010	0.010	0						

Table B-27. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|for|201402-03**.

Table B-27. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diametric shellfish poisoning toxins (mg/kg) in bloom event 0101201402-03.																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G050	3	1	33.3%	0.011	0.011	0.011	0.011	0.011	2	66.7%	0.015	0.015	0.015	0.015	0.015	0						
Subtotal	3	1	33.3%	0.011	0.011	0.011	0.011	0.011	2	66.7%	0.015	0.015	0.015	0.015	0.015	0						

Table B-28. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|200902-12**.

Table B-26. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarmidic sherman poisoning toxins (mg/kg) in bloom event 01pet200502-12.																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	21	0							10	47.6%	0.010	0.035	0.016	0.013	0.033	0						
G009	46	0							8	17.4%	0.010	0.100	0.028	0.018	0.087	0						
G010	47	0							29	61.7%	0.010	0.320	0.061	0.029	0.285	0						
G013	46	0							18	39.1%	0.011	0.340	0.101	0.083	0.293	0						
G014	21	0							9	42.9%	0.011	0.023	0.017	0.018	0.023	0						
G015	47	0							15	31.9%	0.010	0.039	0.020	0.017	0.039	0						
G016	21	0							12	57.1%	0.011	0.021	0.015	0.015	0.020	0						
G018	21	0							13	61.9%	0.015	0.110	0.050	0.039	0.103	0						
G027	46	0							19	41.3%	0.010	0.056	0.022	0.018	0.048	0						
G028	21	0							13	61.9%	0.012	0.024	0.018	0.019	0.024	0						
G031	20	0							10	50.0%	0.010	0.025	0.017	0.018	0.024	0						
G037	21	0							20	95.2%	0.011	0.055	0.024	0.020	0.054	0						
G039	26	0							8	30.8%	0.012	0.024	0.018	0.017	0.024	0						
G043	9	0							2	22.2%	0.014	0.018	0.016	0.016	0.018	0						
G067	26	5	19.2%	0.010	0.021	0.014	0.012	0.021	14	53.8%	0.011	0.890	0.250	0.089	0.884	6	23.1%	0.052	0.320	0.153	0.122	0.309
G085	5	0							5	100.0%	0.065	0.390	0.190	0.099	0.384	0						
Subtotal	444	5	1.1%	0.010	0.021	0.014	0.012	0.021	205	46.2%	0.010	0.890	0.055	0.019	0.339	6	1.4%	0.052	0.320	0.153	0.122	0.309

Table B-29. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201005-201101**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	35	0							9	25.7%	0.011	0.035	0.017	0.015	0.032	0						
G009	36	0							12	33.3%	0.010	0.110	0.039	0.026	0.105	0						
G010	36	0							20	55.6%	0.010	0.058	0.022	0.017	0.054	0						
G014	35	0							10	28.6%	0.013	0.046	0.024	0.021	0.046	0						
G015	35	0							7	20.0%	0.010	0.028	0.017	0.017	0.027	0						
G018	34	0							1	2.9%	0.012	0.012	0.012	0.012	0.012	0						
G027	36	0							11	30.6%	0.010	0.039	0.017	0.014	0.036	0						
G028	35	0							1	2.9%	0.012	0.012	0.012	0.012	0.012	0						
G031	35	0							2	5.7%	0.011	0.012	0.012	0.012	0.012	0						
G037	35	0							6	17.1%	0.011	0.029	0.016	0.012	0.028	0						
G039	36	0							1	2.8%	0.011	0.011	0.011	0.011	0.011	0						
G085	3	0							2	66.7%	0.017	0.030	0.024	0.024	0.030	0						
Subtotal	391	0							82	21.0%	0.010	0.110	0.022	0.016	0.058	0						

Table B-30. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201105-201202**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	39	0							12	30.8%	0.010	0.022	0.014	0.015	0.021	0						
G009	40	0							13	32.5%	0.012	0.054	0.025	0.019	0.050	0						
G010	40	1	2.5%	0.010	0.010	0.010	0.010	0.010	10	25.0%	0.010	0.077	0.028	0.017	0.075	0						
G013	38	2	5.3%	0.014	0.022	0.018	0.018	0.022	11	28.9%	0.010	0.470	0.137	0.075	0.423	0						
G014	40	0							10	25.0%	0.010	0.026	0.015	0.014	0.026	0						
G015	39	0							3	7.7%	0.014	0.017	0.015	0.014	0.017	0						
G016	40	0							6	15.0%	0.010	0.028	0.017	0.015	0.028	0						
G018	39	0							5	12.8%	0.017	0.059	0.032	0.018	0.058	0						
G027	40	0							9	22.5%	0.011	0.093	0.034	0.020	0.091	0						
G028	39	0							7	17.9%	0.012	0.054	0.022	0.015	0.050	0						
G031	39	0							1	2.6%	0.012	0.012	0.012	0.012	0.012	0						
G037	39	0							6	15.4%	0.011	0.017	0.013	0.013	0.017	0						
G039	38	0							6	15.8%	0.011	0.022	0.016	0.017	0.022	0						
G042	5	0							2	40.0%	0.011	0.035	0.023	0.023	0.034	0						
G043	5	0							2	40.0%	0.014	0.014	0.014	0.014	0.014	0						
G067	8	0							2	25.0%	0.140	0.160	0.150	0.150	0.160	5	62.5%	0.050	0.160	0.087	0.053	0.156
Subtotal	528	3	0.6%	0.010	0.022	0.015	0.014	0.022	105	19.9%	0.010	0.470	0.036	0.017	0.204	5	0.9%	0.050	0.160	0.087	0.053	0.156

Table B-31. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201204-12**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	36	0							13	36.1%	0.010	0.047	0.023	0.017	0.046	0						
G009	36	0							6	16.7%	0.010	0.017	0.013	0.012	0.017	0						
G010	37	0							28	75.7%	0.010	0.230	0.038	0.020	0.223	0						
G013	35	0							1	2.9%	0.014	0.014	0.014	0.014	0.014	0						
G014	36	0							14	38.9%	0.010	0.240	0.090	0.091	0.234	0						
G015	36	0							5	13.9%	0.013	0.037	0.021	0.019	0.035	0						
G016	36	0							14	38.9%	0.010	0.069	0.018	0.013	0.054	0						
G018	36	0							4	11.1%	0.011	0.018	0.015	0.015	0.018	0						
G027	36	0							18	50.0%	0.011	0.098	0.031	0.017	0.089	0						
G028	36	0							9	25.0%	0.011	0.022	0.014	0.013	0.021	0						
G031	35	0							7	20.0%	0.010	0.042	0.018	0.013	0.040	0						
G037	36	0							8	22.2%	0.010	0.045	0.016	0.011	0.040	0						
G039	36	0							8	22.2%	0.010	0.017	0.012	0.012	0.016	0						
G043	3	0							3	100.0%	0.011	0.021	0.014	0.011	0.021	0						
Subtotal	470	0							138	29.4%	0.010	0.240	0.031	0.016	0.203	0						

Table B-32. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201311-201402**.

Table 2-32. Summary of postdetoxin 2, predetoxin 2 fecal data, and diarrhetic shellfish poisoning toxins (mg/kg) in brown shrimp, 2010-11 to 2016-17																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G010	10	0							4	40.0%	0.013	0.016	0.014	0.014	0.016	0						
G014	10	0							3	30.0%	0.013	0.052	0.027	0.015	0.050	0						
G027	10	0							1	10.0%	0.012	0.012	0.012	0.012	0.012	0						
G121	1	0							1	100.0%	0.260	0.260	0.260	0.260	0.260	0						
Subtotal	31	0							9	29.0%	0.012	0.260	0.045	0.014	0.218	0						

Table B-33. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201311-201402**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G010	10	0							4	40.0%	0.013	0.016	0.014	0.014	0.016	0						
G014	10	0							3	30.0%	0.013	0.052	0.027	0.015	0.050	0						
G027	10	0							1	10.0%	0.012	0.012	0.012	0.012	0.012	0						
G121	1	0							1	100.0%	0.260	0.260	0.260	0.260	0.260	0						
Subtotal	31	0							9	29.0%	0.012	0.260	0.045	0.014	0.218	0						

Table B-34. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201805-11**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	27	0							5	18.5%	0.010	0.026	0.015	0.011	0.025	0						
G009	26	0							13	50.0%	0.011	0.060	0.023	0.016	0.055	0						
G010	29	0							10	34.5%	0.012	0.030	0.018	0.017	0.029	0						
G013	29	0							5	17.2%	0.010	0.014	0.012	0.011	0.014	0						
G014	28	0							11	39.3%	0.011	0.110	0.038	0.021	0.103	0						
G015	26	0							2	7.7%	0.012	0.015	0.014	0.014	0.015	0						
G027	25	0							2	8.0%	0.011	0.011	0.011	0.011	0.011	0						
G037	25	0							4	16.0%	0.011	0.024	0.016	0.014	0.023	0						
Subtotal	215	0							52	24.2%	0.010	0.110	0.022	0.015	0.079	0						

Table B-35. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|pel|201906-08**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	11	0							11	100.0%	0.011	0.028	0.015	0.012	0.027	0						
G009	13	0							9	69.2%	0.011	0.130	0.055	0.048	0.122	0						
G010	12	0							1	8.3%	0.011	0.011	0.011	0.011	0.011	0						
G014	12	0							11	91.7%	0.010	0.064	0.033	0.031	0.063	0						
G073	3	1	33.3%	0.017	0.017	0.017	0.017	0.017	3	100.0%	0.043	0.580	0.258	0.150	0.559	1	33.3%	0.028	0.028	0.028	0.028	0.028
G121	1	0							1	100.0%	0.060	0.060	0.060	0.060	0.060	0						
G135	12	0							3	25.0%	0.010	0.013	0.011	0.011	0.013	0						
Subtotal	64	1	1.6%	0.017	0.017	0.017	0.017	0.017	39	60.9%	0.010	0.580	0.049	0.022	0.171	1	1.6%	0.028	0.028	0.028	0.028	0.028

Table B-36. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|200901-04**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	16	1	6.3%	0.012	0.012	0.012	0.012	0.012	11	68.8%	0.011	0.360	0.092	0.025	0.330	2	12.5%	0.047	0.055	0.051	0.051	0.055
G012	15	0							4	26.7%	0.010	0.055	0.027	0.021	0.052	0						
G040	15	0							8	53.3%	0.011	0.170	0.051	0.017	0.158	0						
Subtotal	46	1	2.2%	0.012	0.012	0.012	0.012	0.012	23	50.0%	0.010	0.360	0.066	0.020	0.294	2	4.3%	0.047	0.055	0.051	0.051	0.055

Table B-37. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|200906-201001**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	31	1	3.2%	0.013	0.013	0.013	0.013	0.013	19	61.3%	0.011	0.290	0.062	0.022	0.254	0						
G012	31	1	3.2%	0.016	0.016	0.016	0.016	0.016	13	41.9%	0.011	0.240	0.073	0.044	0.219	0						
G040	31	0							14	45.2%	0.010	0.130	0.035	0.018	0.115	0						
Subtotal	93	2	2.2%	0.013	0.016	0.015	0.015	0.016	46	49.5%	0.010	0.290	0.057	0.020	0.236	0						

Table B-38. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201001-04**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	14	2	14.3%	0.015	0.023	0.019	0.019	0.023	13	92.9%	0.011	0.950	0.235	0.050	0.899	2	14.3%	0.130	0.390	0.260	0.260	0.384
G012	14	0							9	64.3%	0.011	0.210	0.055	0.021	0.187	1	7.1%	0.050	0.050	0.050	0.050	0.050
G040	13	2	15.4%	0.010	0.011	0.011	0.011	0.011	7	53.8%	0.035	0.630	0.216	0.130	0.587	2	15.4%	0.060	0.190	0.125	0.125	0.187
Subtotal	41	4	9.8%	0.010	0.023	0.015	0.013	0.022	29	70.7%	0.011	0.950	0.175	0.053	0.831	5	12.2%	0.050	0.390	0.164	0.130	0.370

Table B-39. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201005-07**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	9	0							8	88.9%	0.012	0.200	0.089	0.077	0.197	0						
G012	8	0							6	75.0%	0.010	0.024	0.017	0.018	0.024	0						
G040	8	0							2	25.0%	0.011	0.014	0.013	0.013	0.014	0						
Subtotal	25	0							16	64.0%	0.010	0.200	0.053	0.022	0.193	0						

Table B-40. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201010-201102**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	18	0							17	94.4%	0.014	0.270	0.056	0.037	0.222	2	11.1%	0.050	0.054	0.052	0.052	0.054
G012	17	0							4	23.5%	0.010	0.027	0.016	0.013	0.026	0						
G040	18	0							17	94.4%	0.012	0.390	0.078	0.048	0.298	2	11.1%	0.057	0.071	0.064	0.064	0.071
Subtotal	53	0							38	71.7%	0.010	0.390	0.062	0.038	0.279	4	7.5%	0.050	0.071	0.058	0.056	0.070

Table B-41. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201102-07**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	27	2	7.4%	0.010	0.012	0.011	0.011	0.012	26	96.3%	0.011	0.300	0.083	0.050	0.244	4	14.8%	0.052	0.073	0.061	0.060	0.072
G012	21	0							11	52.4%	0.011	0.039	0.021	0.017	0.038	0						
G021	9	1	11.1%	0.014	0.014	0.014	0.014	0.014	6	66.7%	0.016	0.320	0.086	0.031	0.292	1	11.1%	0.190	0.190	0.190	0.190	0.190
G040	25	3	12.0%	0.011	0.015	0.013	0.012	0.015	14	56.0%	0.013	0.360	0.106	0.069	0.318	6	24.0%	0.063	0.180	0.121	0.115	0.179
Subtotal	82	6	7.3%	0.010	0.015	0.012	0.012	0.015	57	69.5%	0.011	0.360	0.077	0.040	0.312	11	13.4%	0.052	0.190	0.105	0.081	0.188

Table B-42. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201201-02**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	9	2	22.2%	0.015	0.020	0.018	0.018	0.020	8	88.9%	0.012	0.270	0.073	0.024	0.246	0						
G012	7	0							3	42.9%	0.032	0.070	0.047	0.040	0.069	0						
G040	7	2	28.6%	0.012	0.025	0.019	0.019	0.025	6	85.7%	0.012	0.430	0.147	0.130	0.395	2	28.6%	0.054	0.110	0.082	0.082	0.109
Subtotal	23	4	17.4%	0.012	0.025	0.018	0.018	0.025	17	73.9%	0.012	0.430	0.095	0.040	0.366	2	8.7%	0.054	0.110	0.082	0.082	0.109

Table B-43. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201203-08**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	24	1	4.2%	0.011	0.011	0.011	0.011	0.011	24	100.0%	0.013	0.220	0.053	0.040	0.163	0						
G012	23	0							6	26.1%	0.010	0.014	0.012	0.013	0.014	0						
G040	23	0							15	65.2%	0.011	0.150	0.036	0.028	0.118	0						
Subtotal	70	1	1.4%	0.011	0.011	0.011	0.011	0.011	45	64.3%	0.010	0.220	0.042	0.034	0.147	0						

Table B-44. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201303-08**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	24	2	8.3%	0.010	0.019	0.015	0.015	0.019	24	100.0%	0.014	1.700	0.211	0.110	1.263	2	8.3%	0.096	0.110	0.103	0.103	0.110
G012	23	0							8	34.8%	0.010	0.045	0.018	0.015	0.040	0						
G021	3	0							3	100.0%	0.024	0.039	0.033	0.037	0.039	0						
G040	23	0							17	73.9%	0.012	0.150	0.046	0.021	0.134	0						
G122	2	0							2	100.0%	0.150	0.390	0.270	0.270	0.384	1	50.0%	0.052	0.052	0.052	0.052	0.052
Subtotal	75	2	2.7%	0.010	0.019	0.015	0.015	0.019	54	72.0%	0.010	1.700	0.123	0.053	0.761	3	4.0%	0.052	0.110	0.086	0.096	0.109

Table B-45. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201308-201406**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	43	0							38	88.4%	0.010	0.330	0.062	0.038	0.228	0						
G012	42	0							11	26.2%	0.010	0.031	0.016	0.015	0.029	0						
G040	42	0							27	64.3%	0.010	0.220	0.038	0.026	0.137	0						
Subtotal	127	0							76	59.8%	0.010	0.330	0.047	0.028	0.220	0						

Table B-46. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201407-201501**.

		PTX2							PTX2SAs							DSP						
Site	No Samples	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	38	4	10.5%	0.012	0.016	0.015	0.015	0.016	31	81.6%	0.010	1.000	0.229	0.064	0.933	4	10.5%	0.052	0.160	0.093	0.081	0.155
G012	28	0							7	25.0%	0.011	0.070	0.024	0.019	0.063	0						
G021	6	0							3	50.0%	0.016	0.079	0.040	0.025	0.076	0						
G040	31	1	3.2%	0.013	0.013	0.013	0.013	0.013	19	61.3%	0.010	0.620	0.130	0.087	0.503	0						
G125	1	0							1	100.0%	0.069	0.069	0.069	0.069	0.069	0						
Subtotal	104	5	4.8%	0.012	0.016	0.014	0.014	0.016	61	58.7%	0.010	1.000	0.163	0.060	0.875	4	3.8%	0.052	0.160	0.093	0.081	0.155

Table B-47. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201502-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	48	0							45	93.8%	0.010	0.180	0.047	0.031	0.177	5	10.4%	0.010	0.014	0.011	0.011	0.014
G012	46	0							4	8.7%	0.010	0.025	0.014	0.011	0.024	0						
G040	47	0							22	46.8%	0.010	0.130	0.022	0.015	0.087	0						
Subtotal	141	0							71	50.4%	0.010	0.180	0.037	0.020	0.158	5	3.5%	0.010	0.014	0.011	0.011	0.014

Table B-48. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201601-08**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	36	6	16.7%	0.010	0.039	0.020	0.018	0.037	32	88.9%	0.011	0.710	0.182	0.102	0.594	20	55.6%	0.010	0.097	0.037	0.024	0.096
G012	27	0							8	29.6%	0.010	0.050	0.022	0.019	0.047	0						
G021	3	0							3	100.0%	0.014	0.060	0.038	0.039	0.059	0						
G040	28	0							20	71.4%	0.013	0.190	0.057	0.033	0.176	6	21.4%	0.012	0.022	0.015	0.015	0.021
Subtotal	94	6	6.4%	0.010	0.039	0.020	0.018	0.037	63	67.0%	0.010	0.710	0.115	0.043	0.522	26	27.7%	0.010	0.097	0.032	0.021	0.096

Table B-49. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201608-201705**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	39	3	7.7%	0.013	0.015	0.014	0.015	0.015	39	100.0%	0.011	0.590	0.126	0.110	0.305	22	56.4%	0.011	0.083	0.028	0.021	0.072
G012	37	0							13	35.1%	0.012	0.053	0.026	0.022	0.050	0						
G040	37	0							31	83.8%	0.011	0.230	0.061	0.054	0.178	7	18.9%	0.010	0.019	0.015	0.016	0.019
Subtotal	113	3	2.7%	0.013	0.015	0.014	0.015	0.015	83	73.5%	0.011	0.590	0.086	0.053	0.290	29	25.7%	0.010	0.083	0.025	0.018	0.068

Table B-50. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201712-201807**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	31	0							27	87.1%	0.011	0.200	0.052	0.034	0.155	5	16.1%	0.012	0.014	0.013	0.014	0.014
G012	31	0							3	9.7%	0.013	0.024	0.020	0.023	0.024	0						
G040	31	0							13	41.9%	0.011	0.072	0.028	0.023	0.066	1	3.2%	0.012	0.012	0.012	0.012	0.012
Subtotal	93	0							43	46.2%	0.011	0.200	0.042	0.030	0.130	6	6.5%	0.012	0.014	0.013	0.014	0.014

Table B-51. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201705-11**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	26	0							26	100.0%	0.011	0.092	0.042	0.041	0.088	2	7.7%	0.013	0.015	0.014	0.014	0.015
G012	25	0							3	12.0%	0.012	0.018	0.014	0.013	0.018	0						
G040	25	0							16	64.0%	0.011	0.180	0.045	0.027	0.154	2	8.0%	0.010	0.013	0.012	0.012	0.013
Subtotal	76	0							45	59.2%	0.011	0.180	0.041	0.029	0.109	4	5.3%	0.010	0.015	0.013	0.013	0.015

Table B-52. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|ptU|201808-201906**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G011	38	0							30	78.9%	0.010	0.150	0.034	0.025	0.097	5	13.2%	0.010	0.020	0.014	0.014	0.020
G012	21	0							1	4.8%	0.016	0.016	0.016	0.016	0.016	0						
G040	37	0							23	62.2%	0.010	0.064	0.023	0.019	0.058	0						
Subtotal	96	0							54	56.3%	0.010	0.150	0.029	0.022	0.075	5	5.2%	0.010	0.020	0.014	0.014	0.020

Table B-53. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|qc|201509-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G023	7	0							7	100.0%	0.012	0.046	0.022	0.018	0.043	1	14.3%	0.016	0.016	0.016	0.016	0.016
Subtotal	7	0							7	100.0%	0.012	0.046	0.022	0.018	0.043	1	14.3%	0.016	0.016	0.016	0.016	0.016

Table B-54. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|tas|200906-201001**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G001	20	1	5.0%	0.017	0.017	0.017	0.017	0.017	20	100.0%	0.010	0.450	0.107	0.050	0.412	0						
G005	10	3	30.0%	0.010	0.030	0.018	0.013	0.029	10	100.0%	0.020	0.870	0.297	0.238	0.805	2	20.0%	0.077	0.087	0.082	0.082	0.087
G007	28	5	17.9%	0.012	0.037	0.023	0.017	0.037	18	64.3%	0.012	1.400	0.256	0.033	1.183	3	10.7%	0.054	0.078	0.069	0.076	0.078
Subtotal	58	9	15.5%	0.010	0.037	0.021	0.017	0.036	48	82.8%	0.010	1.400	0.203	0.050	0.887	5	8.6%	0.054	0.087	0.074	0.077	0.086

Table B-55. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|tas|201109-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G001	8	0							6	75.0%	0.012	0.160	0.069	0.065	0.153	0						
G005	7	2	28.6%	0.012	0.015	0.014	0.014	0.015	7	100.0%	0.011	0.260	0.065	0.030	0.235	0						
G007	6	2	33.3%	0.010	0.017	0.014	0.014	0.017	6	100.0%	0.011	0.300	0.068	0.018	0.269	0						
Subtotal	21	4	19.0%	0.010	0.017	0.014	0.014	0.017	19	90.5%	0.011	0.300	0.067	0.030	0.282	0						

Table B-56. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|tc|200908-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G029	3	0							1	33.3%	0.011	0.011	0.011	0.011	0.011	0						
G091	21	0							15	71.4%	0.010	0.140	0.034	0.017	0.117	0						
Subtotal	24	0							16	66.7%	0.010	0.140	0.033	0.016	0.115	0						

Table B-57. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|tc|201305-12**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G022	32	0							31	96.9%	0.010	0.100	0.035	0.027	0.086	0						
G029	5	0							1	20.0%	0.016	0.016	0.016	0.016	0.016	0						
G091	31	0							15	48.4%	0.012	0.044	0.024	0.021	0.042	0						
Subtotal	68	0							47	69.1%	0.010	0.100	0.031	0.026	0.080	0						

Table B-58. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **G|tc|201609-10**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G022	7	0							7	100.0%	0.016	0.110	0.040	0.030	0.100	0						
G091	6	0							3	50.0%	0.012	0.099	0.046	0.028	0.095	0						
Subtotal	13	0							10	76.9%	0.012	0.110	0.042	0.029	0.108	0						

Table B-59. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **I|bpk|200904-201005**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	8	2	25.0%	0.011	0.011	0.011	0.011	0.011	7	87.5%	0.010	0.063	0.031	0.031	0.060	0						
I032	24	3	12.5%	0.013	0.063	0.038	0.039	0.062	12	50.0%	0.010	3.500	0.586	0.060	3.060	11	45.8%	0.050	0.680	0.195	0.070	0.648
I036	37	9	24.3%	0.011	0.051	0.027	0.024	0.049	18	48.6%	0.016	2.500	0.640	0.340	2.203	14	37.8%	0.050	0.700	0.233	0.135	0.651
I108	1	0							1	100.0%	0.026	0.026	0.026	0.026	0.026	0						
Subtotal	70	14	20.0%	0.011	0.063	0.027	0.022	0.059	38	54.3%	0.010	3.500	0.494	0.070	2.575	25	35.7%	0.050	0.700	0.216	0.090	0.688

Table B-60. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **I|bpk|201008-201105**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	2	0							1	50.0%	0.011	0.011	0.011	0.011	0.011	0						
I032	16	1	6.3%	0.012	0.012	0.012	0.012	0.012	6	37.5%	0.051	0.290	0.143	0.085	0.290	8	50.0%	0.053	0.210	0.110	0.079	0.208
I036	10	2	20.0%	0.010	0.011	0.011	0.011	0.011	2	20.0%	0.440	0.560	0.500	0.500	0.557	4	40.0%	0.050	0.270	0.157	0.153	0.267
I038	11	3	27.3%	0.010	0.041	0.028	0.032	0.041	4	36.4%	0.190	1.700	0.788	0.630	1.644	11	100.0%	0.070	0.780	0.199	0.098	0.673
Subtotal	39	6	15.4%	0.010	0.041	0.019	0.012	0.040	13	33.3%	0.011	1.700	0.386	0.290	1.475	23	59.0%	0.050	0.780	0.161	0.095	0.544

Table B-61. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201106-201201.

Table D-5. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diastereoisomeric pectenotoxins (mg/L) in brown shrimp (Appendix D-1)																						
Site	No Samples	PTX2							PTX2SAs						DSP							
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I032	8	3	37.5%	0.015	0.019	0.016	0.015	0.019	8	100.0%	0.030	0.370	0.171	0.170	0.356	7	87.5%	0.060	0.180	0.110	0.110	0.173
I036	12	2	16.7%	0.010	0.013	0.012	0.012	0.013	10	83.3%	0.017	0.230	0.081	0.055	0.212	6	50.0%	0.053	0.230	0.127	0.130	0.221
I038	13	0							13	100.0%	0.037	0.140	0.078	0.064	0.134	10	76.9%	0.055	0.086	0.067	0.066	0.084
Subtotal	33	5	15.2%	0.010	0.019	0.014	0.015	0.019	31	93.9%	0.017	0.370	0.103	0.074	0.310	23	69.7%	0.053	0.230	0.096	0.073	0.203

Table B-62. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201206-201301.

Table D-02: Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diastereoisomeric pectenotoxins (mg/kg) in brown shrimp (spike to 100 to 1000)																						
Site	No Samples	PTX2							PTX2SAs						DSP							
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	8	0							2	25.0%	0.029	0.280	0.155	0.155	0.274	3	37.5%	0.058	0.120	0.084	0.073	0.118
I032	10	3	30.0%	0.011	0.014	0.012	0.012	0.014	8	80.0%	0.013	0.500	0.242	0.270	0.486	6	60.0%	0.060	0.260	0.153	0.150	0.255
I035	12	2	16.7%	0.012	0.017	0.015	0.015	0.017	5	41.7%	0.100	0.410	0.224	0.200	0.398	3	25.0%	0.051	0.089	0.064	0.052	0.087
I036	2	0							2	100.0%	0.075	0.170	0.123	0.123	0.168	0						
I038	4	2	50.0%	0.011	0.016	0.014	0.014	0.016	4	100.0%	0.049	0.320	0.168	0.151	0.313	0						
Subtotal	36	7	19.4%	0.011	0.017	0.013	0.012	0.017	21	58.3%	0.013	0.500	0.204	0.200	0.460	12	33.3%	0.051	0.260	0.113	0.083	0.249

Table B-63. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201306-09.

Table 2-36. Summary of postenotoxin E, preenotoxin E fecal debris, and diarrhetic shellfish poisoning toxins (mg/kg) in brown shrimp (Litopenaeus setiferus)																						
Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	13	0							3	23.1%	0.010	0.033	0.019	0.015	0.032	0						
I032	2	0							1	50.0%	0.011	0.011	0.011	0.011	0.011	0						
I035	6	0							6	100.0%	0.015	0.064	0.036	0.030	0.063	0						
I036	6	0							6	100.0%	0.015	0.120	0.059	0.046	0.117	0						
I038	4	0							3	75.0%	0.014	0.098	0.049	0.035	0.095	0						
Subtotal	31	0							19	61.3%	0.010	0.120	0.041	0.034	0.110	0						

Table B-64. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201310-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	5	0							2	40.0%	0.014	0.058	0.036	0.036	0.057	0						
I032	2	0							1	50.0%	0.015	0.015	0.015	0.015	0.015	0						
I035	6	1	16.7%	0.020	0.020	0.020	0.020	0.020	5	83.3%	0.011	0.740	0.197	0.019	0.686	1	16.7%	0.120	0.120	0.120	0.120	0.120
I036	5	0							2	40.0%	0.011	0.039	0.025	0.025	0.038	0						
Subtotal	18	1	5.6%	0.020	0.020	0.020	0.020	0.020	10	55.6%	0.011	0.740	0.112	0.017	0.619	1	5.6%	0.120	0.120	0.120	0.120	0.120

Table B-65. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201406-07.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	6	0							1	16.7%	0.028	0.028	0.028	0.028	0.028	0						
I032	3	0							3	100.0%	0.017	0.062	0.033	0.020	0.060	0						
I036	6	0							6	100.0%	0.023	0.180	0.091	0.076	0.178	0						
Subtotal	15	0							10	66.7%	0.017	0.180	0.067	0.037	0.176	0						

Table B-66. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201409-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	10	0							5	50.0%	0.014	0.048	0.023	0.018	0.045	0						
I032	8	0							8	100.0%	0.012	0.160	0.101	0.120	0.158	1	12.5%	0.051	0.051	0.051	0.051	0.051
I035	7	0							7	100.0%	0.049	0.230	0.101	0.068	0.218	1	14.3%	0.051	0.051	0.051	0.051	0.051
I036	8	0							8	100.0%	0.057	0.140	0.098	0.103	0.138	1	12.5%	0.060	0.060	0.060	0.060	0.060
Subtotal	33	0							28	84.8%	0.012	0.230	0.086	0.069	0.183	3	9.1%	0.051	0.060	0.054	0.051	0.060

Table B-67. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201504-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	32	0							12	37.5%	0.010	0.047	0.019	0.013	0.045	4	12.5%	0.011	0.023	0.015	0.012	0.022
I032	13	0							6	46.2%	0.012	0.028	0.019	0.018	0.027	0						
I035	19	0							12	63.2%	0.011	0.026	0.017	0.015	0.025	0						
I036	10	0							6	60.0%	0.011	0.022	0.017	0.018	0.022	0						
Subtotal	74	0							36	48.6%	0.010	0.047	0.018	0.015	0.041	4	5.4%	0.011	0.023	0.015	0.012	0.022

Table B-68. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201601-03.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	10	0							0							6	60.0%	0.010	0.015	0.012	0.012	0.015
Subtotal	10	0							0							6	60.0%	0.010	0.015	0.012	0.012	0.015

Table B-69. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201603-07.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I007	4	0							4	100.0%	0.082	0.240	0.130	0.098	0.230	4	100.0%	0.019	0.135	0.078	0.078	0.134
I008A	15	0							10	66.7%	0.015	0.077	0.030	0.025	0.069	2	13.3%	0.013	0.015	0.014	0.014	0.015
I032	4	0							4	100.0%	0.049	0.180	0.122	0.130	0.176	3	75.0%	0.016	0.024	0.020	0.020	0.024
I035	11	0							11	100.0%	0.033	0.160	0.083	0.070	0.153	11	100.0%	0.010	0.031	0.020	0.019	0.031
I036	16	0							16	100.0%	0.025	0.210	0.115	0.110	0.206	12	75.0%	0.012	0.030	0.018	0.018	0.028
I038	7	0							6	85.7%	0.030	0.220	0.084	0.055	0.206	6	85.7%	0.014	0.039	0.026	0.025	0.039
Subtotal	57	0							51	89.5%	0.015	0.240	0.089	0.077	0.218	38	66.7%	0.010	0.135	0.026	0.019	0.119

Table B-70. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201607-201703.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I002	3	2	66.7%	0.016	0.022	0.019	0.019	0.022	2	66.7%	0.510	1.000	0.755	0.755	0.988	3	100.0%	0.027	0.605	0.281	0.210	0.585
I004	1	0							1	100.0%	0.630	0.630	0.630	0.630	0.630	1	100.0%	0.256	0.256	0.256	0.256	0.256
I007	15	4	26.7%	0.020	0.077	0.035	0.022	0.073	11	73.3%	0.028	2.100	0.474	0.150	1.815	15	100.0%	0.061	1.415	0.357	0.260	1.280
I008A	24	2	8.3%	0.014	0.022	0.018	0.018	0.022	14	58.3%	0.027	0.480	0.151	0.088	0.477	16	66.7%	0.010	0.180	0.054	0.022	0.169
I021	2	0							2	100.0%	0.063	0.070	0.067	0.067	0.070	2	100.0%	0.120	0.170	0.145	0.145	0.169
I026	1	0							1	100.0%	0.021	0.021	0.021	0.021	0.021	1	100.0%	0.038	0.038	0.038	0.038	0.038
I031	1	0							1	100.0%	0.100	0.100	0.100	0.100	0.100	1	100.0%	0.381	0.381	0.381	0.381	0.381
I032	6	0							1	16.7%	0.022	0.022	0.022	0.022	0.022	4	66.7%	0.010	0.030	0.022	0.024	0.030
I035	14	2	14.3%	0.011	0.079	0.045	0.045	0.077	7	50.0%	0.011	1.800	0.317	0.013	1.584	14	100.0%	0.013	0.500	0.083	0.033	0.409
I036	8	1	12.5%	0.046	0.046	0.046	0.046	0.046	3	37.5%	0.011	1.200	0.467	0.190	1.150	8	100.0%	0.014	0.316	0.062	0.022	0.271
I038	1	0							1	100.0%	0.051	0.051	0.051	0.051	0.051	1	100.0%	0.027	0.027	0.027	0.027	0.027
Subtotal	76	11	14.5%	0.011	0.079	0.032	0.022	0.079	44	57.9%	0.011	2.100	0.305	0.099	1.755	66	86.8%	0.010	1.415	0.148	0.054	0.764

Table B-71. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201704-12.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I004	17	5	29.4%	0.011	0.027	0.018	0.018	0.027	13	76.5%	0.030	0.670	0.236	0.200	0.616	13	76.5%	0.095	1.126	0.479	0.431	1.096
I007	3	0							3	100.0%	0.059	0.270	0.166	0.170	0.265	3	100.0%	0.030	0.081	0.053	0.048	0.079
I008A	36	0							21	58.3%	0.010	0.210	0.047	0.031	0.165	10	27.8%	0.010	0.038	0.021	0.021	0.035
I032	13	0							12	92.3%	0.013	0.054	0.028	0.022	0.054	6	46.2%	0.012	0.024	0.017	0.017	0.024
I035	36	1	2.8%	0.012	0.012	0.012	0.012	0.012	31	86.1%	0.010	0.390	0.052	0.027	0.203	24	66.7%	0.010	0.053	0.017	0.014	0.043
I036	11	0							11	100.0%	0.015	0.086	0.042	0.031	0.084	9	81.8%	0.011	0.024	0.015	0.015	0.022
I038	23	1	4.3%	0.010	0.010	0.010	0.010	0.010	22	95.7%	0.010	0.350	0.082	0.048	0.298	22	95.7%	0.014	0.110	0.035	0.020	0.101
Subtotal	139	7	5.0%	0.010	0.027	0.016	0.013	0.026	113	81.3%	0.010	0.670	0.077	0.033	0.396	87	62.6%	0.010	1.126	0.092	0.019	0.746

Table B-72. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201801-03.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I004	2	0							1	50.0%	0.017	0.017	0.017	0.017	0.017	2	100.0%	0.013	0.019	0.016	0.016	0.019
I032	5	0							3	60.0%	0.010	0.024	0.015	0.011	0.023	0						
I036	8	0							2	25.0%	0.011	0.015	0.013	0.013	0.015	0						
Subtotal	15	0							6	40.0%	0.010	0.024	0.015	0.013	0.023	2	13.3%	0.013	0.019	0.016	0.016	0.019

Table B-73. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201806-201901.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I004	5	1	20.0%	0.042	0.042	0.042	0.042	0.042	3	60.0%	0.047	0.600	0.262	0.140	0.577	5	100.0%	0.074	0.346	0.173	0.176	0.329
I008A	38	1	2.6%	0.013	0.013	0.013	0.013	0.013	20	52.6%	0.023	0.660	0.213	0.160	0.594	12	31.6%	0.025	0.128	0.062	0.046	0.126
I032	4	4	100.0%	0.016	0.058	0.030	0.022	0.056	4	100.0%	0.220	0.830	0.495	0.465	0.808	3	75.0%	0.015	0.088	0.048	0.042	0.086
I035	28	1	3.6%	0.017	0.017	0.017	0.017	0.017	26	92.9%	0.013	0.650	0.100	0.040	0.419	11	39.3%	0.010	0.066	0.029	0.021	0.063
I036	12	2	16.7%	0.011	0.013	0.012	0.012	0.013	12	100.0%	0.013	0.440	0.139	0.054	0.435	6	50.0%	0.011	0.038	0.025	0.025	0.038
Subtotal	87	9	10.3%	0.011	0.058	0.024	0.016	0.055	65	74.7%	0.013	0.830	0.174	0.064	0.654	37	42.5%	0.010	0.346	0.060	0.042	0.196

Table B-74. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event I|bpk|201904-.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I008A	26	3	11.5%	0.016	0.024	0.019	0.017	0.024	15	57.7%	0.011	0.400	0.113	0.079	0.355	10	38.5%	0.011	0.065	0.026	0.018	0.060
I032	15	7	46.7%	0.010	0.022	0.013	0.011	0.021	13	86.7%	0.014	0.350	0.178	0.180	0.347	8	53.3%	0.010	0.023	0.015	0.015	0.022
I035	19	1	5.3%	0.015	0.015	0.015	0.015	0.015	17	89.5%	0.031	0.380	0.161	0.140	0.380	9	47.4%	0.010	0.022	0.015	0.012	0.022
I036	22	3	13.6%	0.012	0.015	0.013	0.013	0.015	21	95.5%	0.012	0.570	0.172	0.130	0.505	13	59.1%	0.011	0.036	0.019	0.016	0.033
Subtotal	82	14	17.1%	0.010	0.024	0.014	0.014	0.023	66	80.5%	0.011	0.570	0.157	0.125	0.415	40	48.8%	0.010	0.065	0.019	0.016	0.045

Table B-75. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **I|ota|201002**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I020	3	2	66.7%	0.010	0.011	0.011	0.011	0.011	3	100.0%	0.052	0.065	0.058	0.058	0.065	0						
Subtotal	3	2	66.7%	0.010	0.011	0.011	0.011	0.011	3	100.0%	0.052	0.065	0.058	0.058	0.065	0						

Table B-76. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **I|ota|201405-06**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I013	4	1	25.0%	0.016	0.016	0.016	0.016	0.016	4	100.0%	0.014	0.240	0.141	0.155	0.236	0						
Subtotal	4	1	25.0%	0.016	0.016	0.016	0.016	0.016	4	100.0%	0.014	0.240	0.141	0.155	0.236	0						

Table B-77. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **I|ota|201611**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I009	1	0							1	100.0%	0.042	0.042	0.042	0.042	0.042	1	100.0%	0.018	0.018	0.018	0.018	0.018
Subtotal	1	0							1	100.0%	0.042	0.042	0.042	0.042	0.042	1	100.0%	0.018	0.018	0.018	0.018	0.018

Table B-78. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|fov|201212-201303**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J013	8	0							6	75.0%	0.012	0.190	0.079	0.071	0.181	0						
Subtotal	8	0							6	75.0%	0.012	0.190	0.079	0.071	0.181	0						

Table B-79. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|fov|201310-201403**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J013	14	0							11	78.6%	0.011	0.130	0.030	0.018	0.106	0						
Subtotal	14	0							11	78.6%	0.011	0.130	0.030	0.018	0.106	0						

Table B-80. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|fov|201811-201902**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J013	11	1	9.1%	0.011	0.011	0.011	0.011	0.011	7	63.6%	0.015	0.170	0.044	0.024	0.149	3	27.3%	0.011	0.074	0.033	0.013	0.071
Subtotal	11	1	9.1%	0.011	0.011	0.011	0.011	0.011	7	63.6%	0.015	0.170	0.044	0.024	0.149	3	27.3%	0.011	0.074	0.033	0.013	0.071

Table B-81. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|wc|201501-02**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J004	3	0							3	100.0%	0.055	0.180	0.112	0.100	0.176	0						
Subtotal	3	0							3	100.0%	0.055	0.180	0.112	0.100	0.176	0						

Table B-82. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|wc|201510-J201603**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J004	12	1	8.3%	0.017	0.017	0.017	0.017	0.017	8	66.7%	0.016	0.350	0.110	0.091	0.315	0						
Subtotal	12	1	8.3%	0.017	0.017	0.017	0.017	0.017	8	66.7%	0.016	0.350	0.110	0.091	0.315	0						

Table B-83. Summary of pectenotoxin 2, pectenotoxin 2 seco acids, and diarrhetic shellfish poisoning toxins (mg/kg) in bloom event **J|wc|201811-201909**.

Site	No Samples	PTX2							PTX2SAs							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
J001	4	0							3	75.0%	0.055	0.270	0.155	0.140	0.264	4	100.0%	0.120	0.290	0.213	0.220	0.289
J003	4	0							4	100.0%	0.031	0.200	0.124	0.133	0.199	4	100.0%	0.072	0.270	0.146	0.120	0.260
J004	21	3	14.3%	0.011	0.011	0.011	0.011	0.011	10	47.6%	0.036	0.350	0.126	0.079	0.325	21	100.0%	0.022	0.340	0.108	0.086	0.315
J024	1	0							1	100.0%	0.015	0.015	0.015	0.015	0.015	1	100.0%	0.065	0.065	0.065	0.065	0.065
J279	1	0							0							1	100.0%	0.072	0.072	0.072	0.072	0.072
Subtotal	31	3	9.7%	0.011	0.011	0.011	0.011	0.011	18	58.1%	0.015	0.350	0.124	0.086	0.316	31	100.0%	0.022	0.340	0.124	0.110	0.303

APPENDIX C. SITE SUMMARY

Table C-1. Summary of PTX 2, PTX2SA, and DSP (mg/kg) in by shellfish site within New Zealand 2009-2019.

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
A001	40	0							0							1	2.5%	0.099	0.099	0.099	0.099	0.099
A002	80	0							2	2.5%	0.012	0.022	0.017	0.017	0.022	4	5.0%	0.014	0.062	0.026	0.015	0.058
A003	35	0							0							0						
A005	32	0							0							0						
A005A	6	0							0							0						
A006	3	0							2	66.7%	0.013	0.038	0.026	0.026	0.037	0						
A008	203	0							9	4.4%	0.011	0.038	0.019	0.019	0.035	0						
A014	230	28	12.2%	0.010	0.027	0.015	0.015	0.026	126	54.8%	0.010	0.290	0.059	0.042	0.206	2	0.9%	0.013	0.043	0.028	0.028	0.042
A015	246	11	4.5%	0.010	0.021	0.016	0.016	0.021	75	30.5%	0.010	0.180	0.039	0.020	0.162	8	3.3%	0.012	0.100	0.032	0.024	0.088
A016	2	0							1	50.0%	0.024	0.024	0.024	0.024	0.024	0						
A018	1	0							1	100.0%	0.012	0.012	0.012	0.012	0.012	0						
A027	9	0							1	11.1%	0.011	0.011	0.011	0.011	0.011	0						
A030	8	3	37.5%	0.013	0.034	0.024	0.025	0.034	8	100.0%	0.013	1.200	0.362	0.144	1.155	8	100.0%	0.010	0.096	0.045	0.036	0.096
A040	2	0							2	100.0%	0.062	0.220	0.141	0.141	0.216	1	50.0%	0.044	0.044	0.044	0.044	0.044
A277	1	0							0							1	100.0%	0.026	0.026	0.026	0.026	0.026
A346	18	0							0							4	22.2%	0.016	0.024	0.020	0.019	0.024
B007	34	0							2	5.9%	0.046	0.097	0.072	0.072	0.096	0						
B013	20	0							0							0						
B014	16	0							0							0						

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
B015B	1	1	100.0%	0.012	0.012	0.012	0.012	0.012	1	100.0%	0.052	0.052	0.052	0.052	0.052	1	100.0%	0.025	0.025	0.025	0.025	0.025
B021	1	0							1	100.0%	0.010	0.010	0.010	0.010	0.010	0						
B024	10	0							1	10.0%	0.026	0.026	0.026	0.026	0.026	0						
B024B	30	4	13.3%	0.012	0.032	0.020	0.017	0.031	11	36.7%	0.012	0.260	0.115	0.100	0.258	1	3.3%	0.012	0.012	0.012	0.012	0.012
B027	4	0							1	25.0%	0.011	0.011	0.011	0.011	0.011	0						
C002	12	5	41.7%	0.010	0.036	0.019	0.016	0.034	12	100.0%	0.020	1.100	0.325	0.190	1.048	9	75.0%	0.010	0.120	0.037	0.028	0.106
C003	3	0							2	66.7%	0.017	0.017	0.017	0.017	0.017	1	33.3%	0.010	0.010	0.010	0.010	0.010
C004	3	0							3	100.0%	0.011	0.054	0.025	0.011	0.052	1	33.3%	0.013	0.013	0.013	0.013	0.013
C009	392	10	2.6%	0.011	0.027	0.017	0.015	0.026	72	18.4%	0.010	0.590	0.132	0.096	0.480	53	13.5%	0.011	0.078	0.030	0.025	0.066
C029	445	12	2.7%	0.010	0.041	0.023	0.021	0.040	71	16.0%	0.010	1.000	0.200	0.091	0.913	59	13.3%	0.010	0.152	0.039	0.027	0.142
C033	128	0							0							0						
C038	402	5	1.2%	0.010	0.014	0.012	0.012	0.014	53	13.2%	0.010	0.480	0.090	0.034	0.405	18	4.5%	0.012	0.039	0.024	0.021	0.038
C041	4	0							3	75.0%	0.010	0.016	0.013	0.013	0.016	1	25.0%	0.011	0.011	0.011	0.011	0.011
C048	23	0							2	8.7%	0.014	0.027	0.021	0.021	0.027	0						
C055	3	0							2	66.7%	0.028	0.041	0.035	0.035	0.041	0						
C056	24	0							5	20.8%	0.013	0.036	0.020	0.014	0.035	0						
C059	11	4	36.4%	0.011	0.036	0.021	0.019	0.035	11	100.0%	0.063	1.100	0.377	0.290	1.075	11	100.0%	0.019	0.171	0.069	0.061	0.159
C060	15	6	40.0%	0.012	0.059	0.024	0.017	0.055	15	100.0%	0.060	1.300	0.341	0.160	1.195	15	100.0%	0.017	0.238	0.072	0.046	0.203
C061	12	5	41.7%	0.014	0.032	0.021	0.017	0.031	12	100.0%	0.036	1.200	0.452	0.380	1.142	10	83.3%	0.012	0.083	0.045	0.041	0.083
C063	424	4	0.9%	0.015	0.054	0.029	0.024	0.052	40	9.4%	0.010	1.400	0.145	0.031	0.932	16	3.8%	0.011	0.090	0.032	0.020	0.079
C065	1	0							1	100.0%	0.220	0.220	0.220	0.220	0.220	1	100.0%	0.025	0.025	0.025	0.025	0.025
C066	1	0							0							0						

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
C323	13	7	53.8%	0.010	0.052	0.026	0.019	0.051	13	100.0%	0.063	1.500	0.489	0.480	1.344	13	100.0%	0.020	0.182	0.080	0.078	0.175
D001	182	1	0.5%	0.031	0.031	0.031	0.031	0.031	16	8.8%	0.010	0.260	0.059	0.020	0.223	7	3.8%	0.011	0.018	0.014	0.013	0.018
D001A	1	0							0							0						
D005	33	0							1	3.0%	0.013	0.013	0.013	0.013	0.013	0						
D009	123	0							14	11.4%	0.012	0.180	0.058	0.036	0.174	4	3.3%	0.018	0.065	0.042	0.042	0.065
D010	1	0							1	100.0%	0.130	0.130	0.130	0.130	0.130	1	100.0%	0.026	0.026	0.026	0.026	0.026
D017	2	0							0							0						
D025	1	0							1	100.0%	0.021	0.021	0.021	0.021	0.021	0						
D026	2	0							0							0						
D029	1	0							0							0						
D033	34	0							1	2.9%	0.019	0.019	0.019	0.019	0.019	0						
D047	4	0							0							0						
F003	16	0							0							0						
F017	15	0							2	13.3%	0.017	0.026	0.022	0.022	0.026	0						
F025	14	0							3	21.4%	0.010	0.013	0.011	0.011	0.013	0						
F029	1	0							0							0						
F042	104	0							0							0						
G001	304	1	0.3%	0.017	0.017	0.017	0.017	0.017	55	18.1%	0.010	0.450	0.062	0.031	0.332	0						
G003	3	0							2	66.7%	0.019	0.024	0.022	0.022	0.024	0						
G005	275	5	1.8%	0.010	0.030	0.016	0.013	0.029	50	18.2%	0.010	0.870	0.085	0.024	0.560	2	0.7%	0.077	0.087	0.082	0.082	0.087
G006	2	0							0							0						
G007	319	7	2.2%	0.010	0.037	0.020	0.017	0.037	57	17.9%	0.010	1.400	0.102	0.021	0.842	3	0.9%	0.054	0.078	0.069	0.076	0.078

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G008	553	0							85	15.4%	0.010	0.047	0.017	0.015	0.042	0						
G009	550	0							83	15.1%	0.010	0.130	0.028	0.017	0.100	0						
G010	554	1	0.2%	0.010	0.010	0.010	0.010	0.010	171	30.9%	0.010	0.320	0.034	0.022	0.180	0						
G011	580	24	4.1%	0.010	0.039	0.016	0.015	0.030	459	79.1%	0.010	1.700	0.094	0.041	0.510	75	12.9%	0.010	0.390	0.042	0.022	0.135
G012	554	1	0.2%	0.016	0.016	0.016	0.016	0.016	117	21.1%	0.010	0.240	0.029	0.017	0.170	1	0.2%	0.050	0.050	0.050	0.050	0.050
G013	548	2	0.4%	0.014	0.022	0.018	0.018	0.022	50	9.1%	0.010	0.470	0.074	0.026	0.327	0						
G014	556	0							93	16.7%	0.010	0.240	0.033	0.017	0.165	0						
G015	552	0							50	9.1%	0.010	0.039	0.018	0.017	0.038	0						
G016	552	0							47	8.5%	0.010	0.069	0.015	0.013	0.027	0						
G017	555	1	0.2%	0.013	0.013	0.013	0.013	0.013	32	5.8%	0.010	0.035	0.018	0.015	0.034	0						
G018	554	0							44	7.9%	0.010	0.110	0.029	0.020	0.085	0						
G019	560	1	0.2%	0.010	0.010	0.010	0.010	0.010	157	28.0%	0.010	0.250	0.032	0.019	0.143	0						
G020	41	2	4.9%	0.010	0.027	0.019	0.019	0.027	14	34.1%	0.011	0.490	0.175	0.125	0.454	9	22.0%	0.059	0.700	0.224	0.130	0.658
G021	33	1	3.0%	0.014	0.014	0.014	0.014	0.014	15	45.5%	0.014	0.320	0.057	0.037	0.242	1	3.0%	0.190	0.190	0.190	0.190	0.190
G022	369	0							74	20.1%	0.010	0.110	0.030	0.021	0.094	0						
G023	16	0							8	50.0%	0.012	0.053	0.026	0.022	0.052	1	6.3%	0.016	0.016	0.016	0.016	0.016
G027	550	0							116	21.1%	0.010	0.098	0.024	0.018	0.077	0						
G028	550	0							55	10.0%	0.010	0.054	0.017	0.016	0.034	0						
G029	26	0							3	11.5%	0.011	0.053	0.027	0.016	0.051	0						
G030	433	1	0.2%	0.012	0.012	0.012	0.012	0.012	21	4.8%	0.012	0.220	0.035	0.021	0.139	0						
G031	550	0							40	7.3%	0.010	0.042	0.016	0.013	0.031	0						
G037	552	0							68	12.3%	0.010	0.055	0.018	0.016	0.047	0						

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G039	539	0							29	5.4%	0.010	0.029	0.016	0.014	0.028	0						
G040	562	8	1.4%	0.010	0.025	0.014	0.012	0.023	299	53.2%	0.010	0.630	0.056	0.027	0.236	28	5.0%	0.010	0.190	0.054	0.019	0.183
G042	11	0							2	18.2%	0.011	0.035	0.023	0.023	0.034	0						
G043	40	0							8	20.0%	0.010	0.021	0.014	0.014	0.020	0						
G045	16	0							3	18.8%	0.012	0.013	0.012	0.012	0.013	0						
G046	35	0							3	8.6%	0.011	0.017	0.013	0.012	0.017	0						
G050	549	1	0.2%	0.011	0.011	0.011	0.011	0.011	21	3.8%	0.010	0.018	0.012	0.011	0.018	0						
G062	2	0							0							0						
G067	76	5	6.6%	0.010	0.021	0.014	0.012	0.021	16	21.1%	0.011	0.890	0.237	0.130	0.883	11	14.5%	0.050	0.320	0.123	0.094	0.298
G070	1	0							1	100.0%	0.014	0.014	0.014	0.014	0.014	0						
G073	4	1	25.0%	0.017	0.017	0.017	0.017	0.017	3	75.0%	0.043	0.580	0.258	0.150	0.559	1	25.0%	0.028	0.028	0.028	0.028	0.028
G075	1	0							0							0						
G076	1	0							0							0						
G085	51	0							7	13.7%	0.017	0.390	0.143	0.067	0.381	0						
G089	1	0							0							0						
G091	540	0							71	13.1%	0.010	0.140	0.028	0.019	0.084	0						
G100	8	0							0							0						
G103	2	0							2	100.0%	0.019	0.026	0.023	0.023	0.026	0						
G110	1	0							1	100.0%	0.150	0.150	0.150	0.150	0.150	0						
G113	2	0							0							0						
G114	7	0							0							0						
G115	80	1	1.3%	0.043	0.043	0.043	0.043	0.043	34	42.5%	0.010	0.400	0.040	0.023	0.141	5	6.3%	0.050	0.230	0.091	0.059	0.213

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
G119	5	0							1	20.0%	0.012	0.012	0.012	0.012	0.012	0						
G121	2	0							2	100.0%	0.060	0.260	0.160	0.160	0.255	0						
G122	2	0							2	100.0%	0.150	0.390	0.270	0.270	0.384	1	50.0%	0.052	0.052	0.052	0.052	0.052
G125	1	0							1	100.0%	0.069	0.069	0.069	0.069	0.069	0						
G135	39	0							3	7.7%	0.010	0.013	0.011	0.011	0.013	0						
G140	2	0							0							0						
G226	1	0							0							0						
G238	1	0							0							0						
G256	2	0							0							0						
G311	1	0							0							0						
G312	1	0							0							0						
G324	1	0							0							0						
H002	54	0							0							0						
I002	3	2	66.7%	0.016	0.022	0.019	0.019	0.022	2	66.7%	0.510	1.000	0.755	0.755	0.988	3	100.0%	0.027	0.605	0.281	0.210	0.585
I004	25	6	24.0%	0.011	0.042	0.022	0.021	0.040	18	72.0%	0.017	0.670	0.250	0.185	0.653	21	84.0%	0.013	1.126	0.352	0.220	1.076
I007	22	4	18.2%	0.020	0.077	0.035	0.022	0.073	18	81.8%	0.028	2.100	0.346	0.130	1.616	22	100.0%	0.019	1.415	0.265	0.133	1.213
I008A	346	8	2.3%	0.011	0.024	0.016	0.015	0.024	114	32.9%	0.010	0.660	0.091	0.036	0.484	63	18.2%	0.010	0.180	0.039	0.022	0.138
I009	1	0							1	100.0%	0.042	0.042	0.042	0.042	0.042	1	100.0%	0.018	0.018	0.018	0.018	0.018
I013	179	1	0.6%	0.016	0.016	0.016	0.016	0.016	5	2.8%	0.013	0.240	0.115	0.130	0.234	0						
I018	9	0							0							0						
I020	51	2	3.9%	0.010	0.011	0.011	0.011	0.011	3	5.9%	0.052	0.065	0.058	0.058	0.065	0						
I021	3	0							2	66.7%	0.063	0.070	0.067	0.067	0.070	2	66.7%	0.120	0.170	0.145	0.145	0.169

Sites	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
I026	1	0							1	100.0%	0.021	0.021	0.021	0.021	0.021	1	100.0%	0.038	0.038	0.038	0.038	0.038
I031	1	0							1	100.0%	0.100	0.100	0.100	0.100	0.100	1	100.0%	0.381	0.381	0.381	0.381	0.381
I032	209	21	10.0%	0.010	0.063	0.020	0.014	0.061	92	44.0%	0.010	3.500	0.190	0.059	0.895	57	27.3%	0.010	0.680	0.092	0.053	0.494
I035	238	8	3.4%	0.011	0.079	0.023	0.016	0.069	127	53.4%	0.010	1.800	0.105	0.040	0.407	74	31.1%	0.010	0.500	0.035	0.018	0.138
I036	258	19	7.4%	0.010	0.051	0.021	0.015	0.049	125	48.4%	0.011	2.500	0.195	0.086	1.290	73	28.3%	0.011	0.700	0.082	0.024	0.510
I037	90	0							0							0						
I038	88	6	6.8%	0.010	0.041	0.020	0.014	0.040	53	60.2%	0.010	1.700	0.138	0.063	0.770	50	56.8%	0.014	0.780	0.076	0.055	0.325
I108	1	0							1	100.0%	0.026	0.026	0.026	0.026	0.026	0						
J001	4	0							3	75.0%	0.055	0.270	0.155	0.140	0.264	4	100.0%	0.120	0.290	0.213	0.220	0.289
J003	4	0							4	100.0%	0.031	0.200	0.124	0.133	0.199	4	100.0%	0.072	0.270	0.146	0.120	0.260
J004	152	4	2.6%	0.011	0.017	0.013	0.011	0.017	35	23.0%	0.010	0.350	0.082	0.047	0.350	21	13.8%	0.022	0.340	0.108	0.086	0.315
J006	133	0							0							0						
J013	313	1	0.3%	0.011	0.011	0.011	0.011	0.011	42	13.4%	0.010	0.190	0.036	0.021	0.169	3	1.0%	0.011	0.074	0.033	0.013	0.071
J024	1	0							1	100.0%	0.015	0.015	0.015	0.015	0.015	1	100.0%	0.065	0.065	0.065	0.065	0.065
J279	1	0							0							1	100.0%	0.072	0.072	0.072	0.072	0.072
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

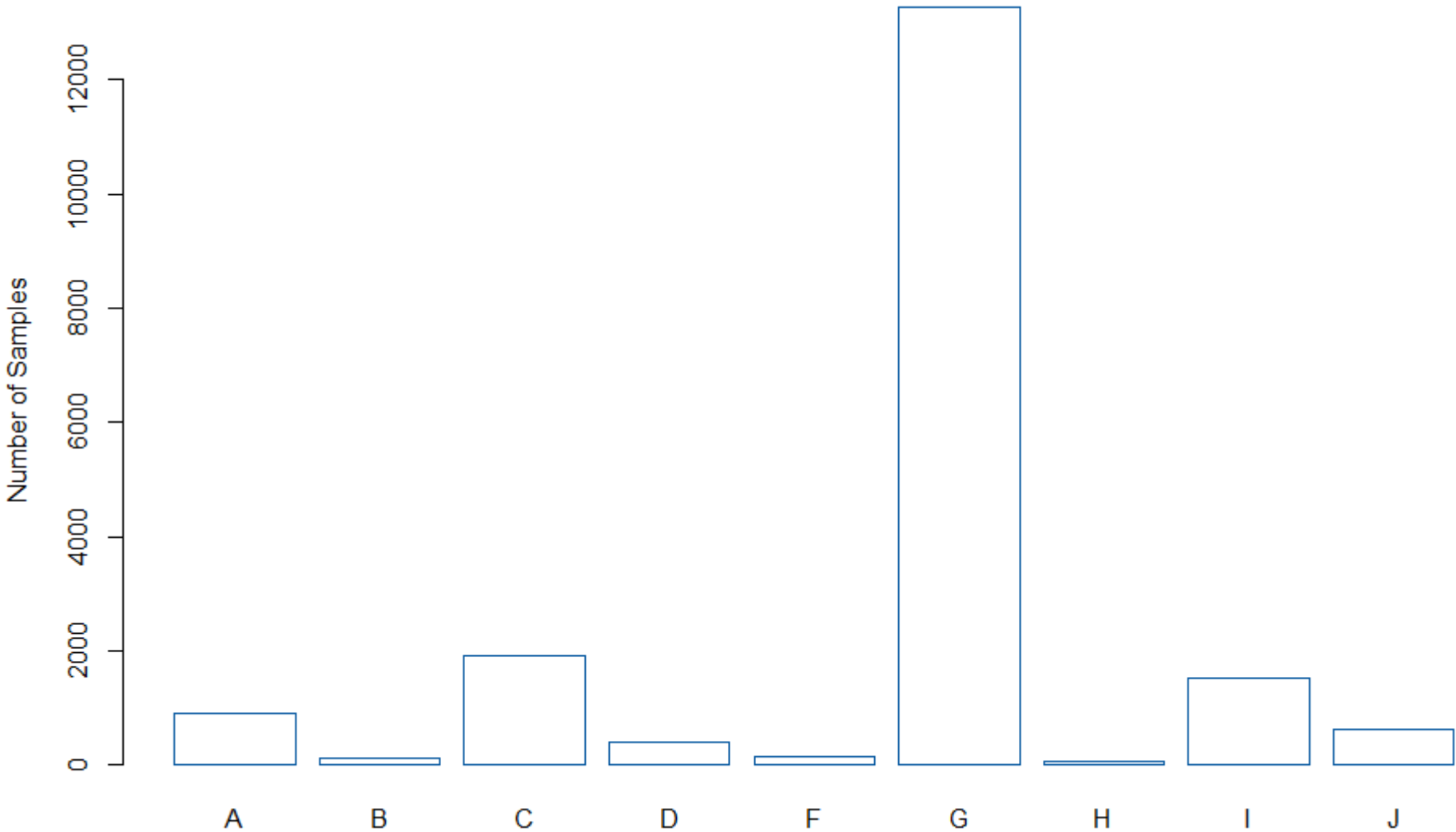


Figure C-1. Samples analysed for shellfish toxins by Zone in New Zealand over the 2009-2019 period

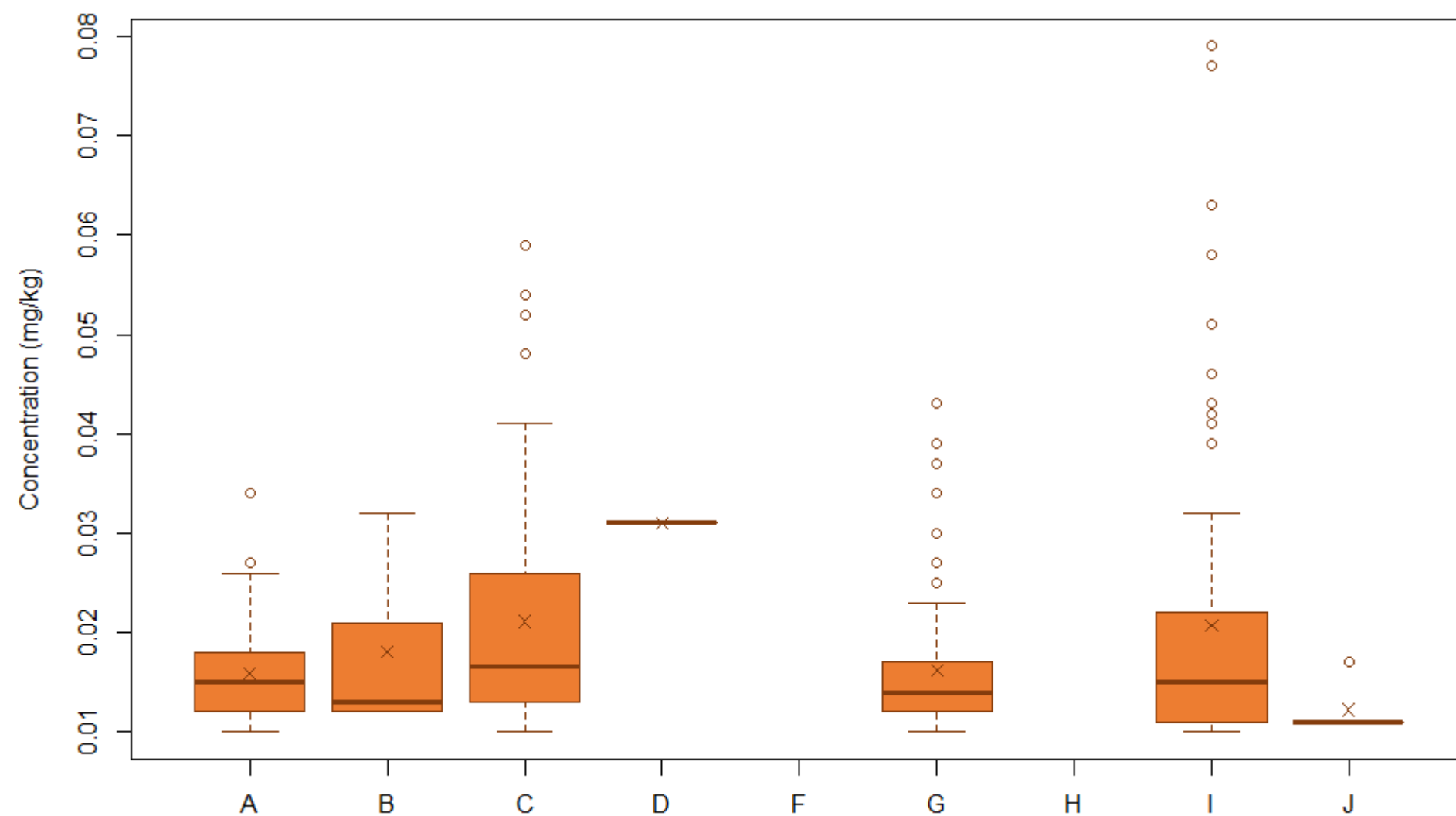


Figure C-2. Box and whisker plot of PTX2 concentrations for each Zone in New Zealand over the 2009-2019 period

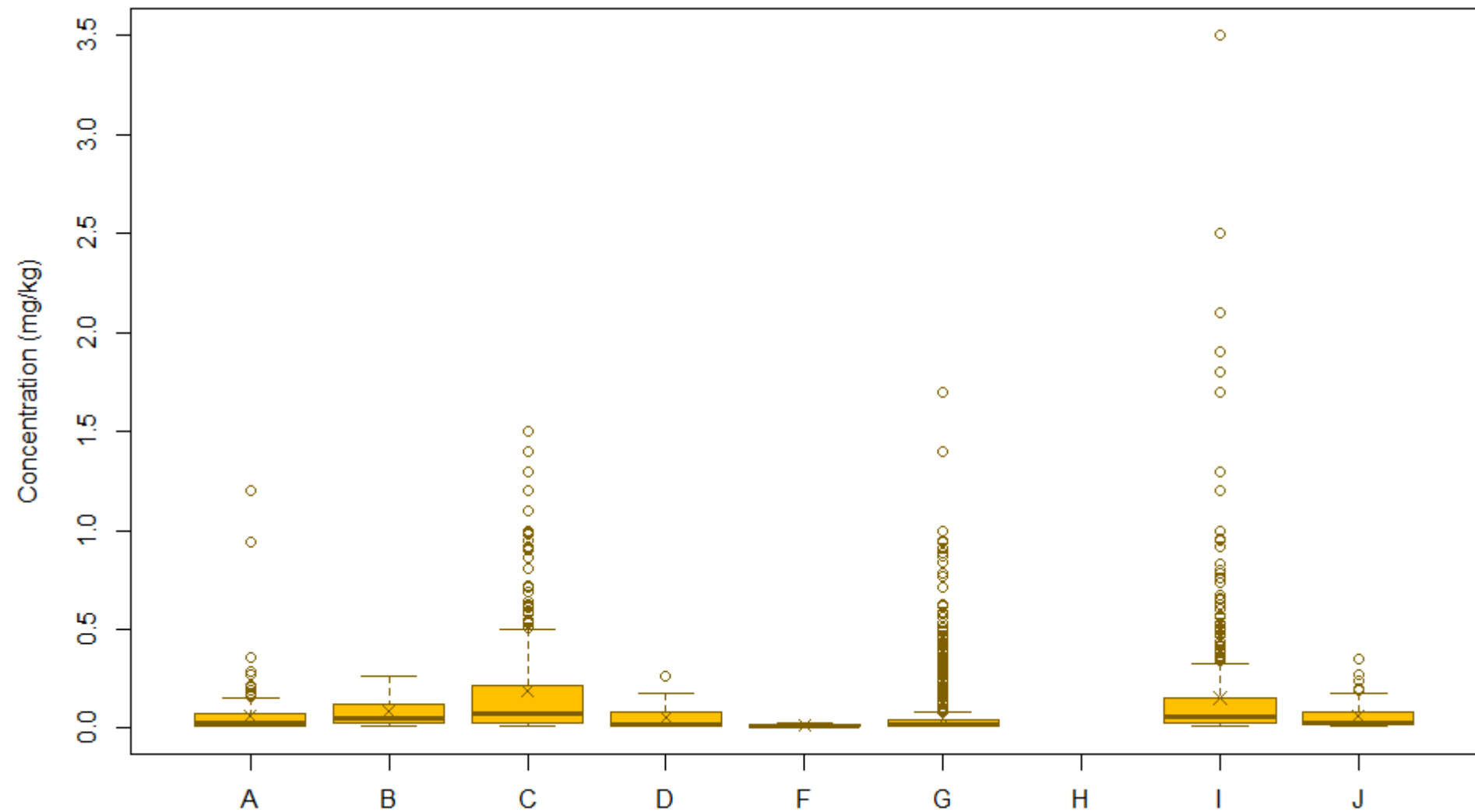


Figure C-3. Box and whisker plot of PTX2SA concentrations for each Zone in New Zealand over the 2009-2019 period

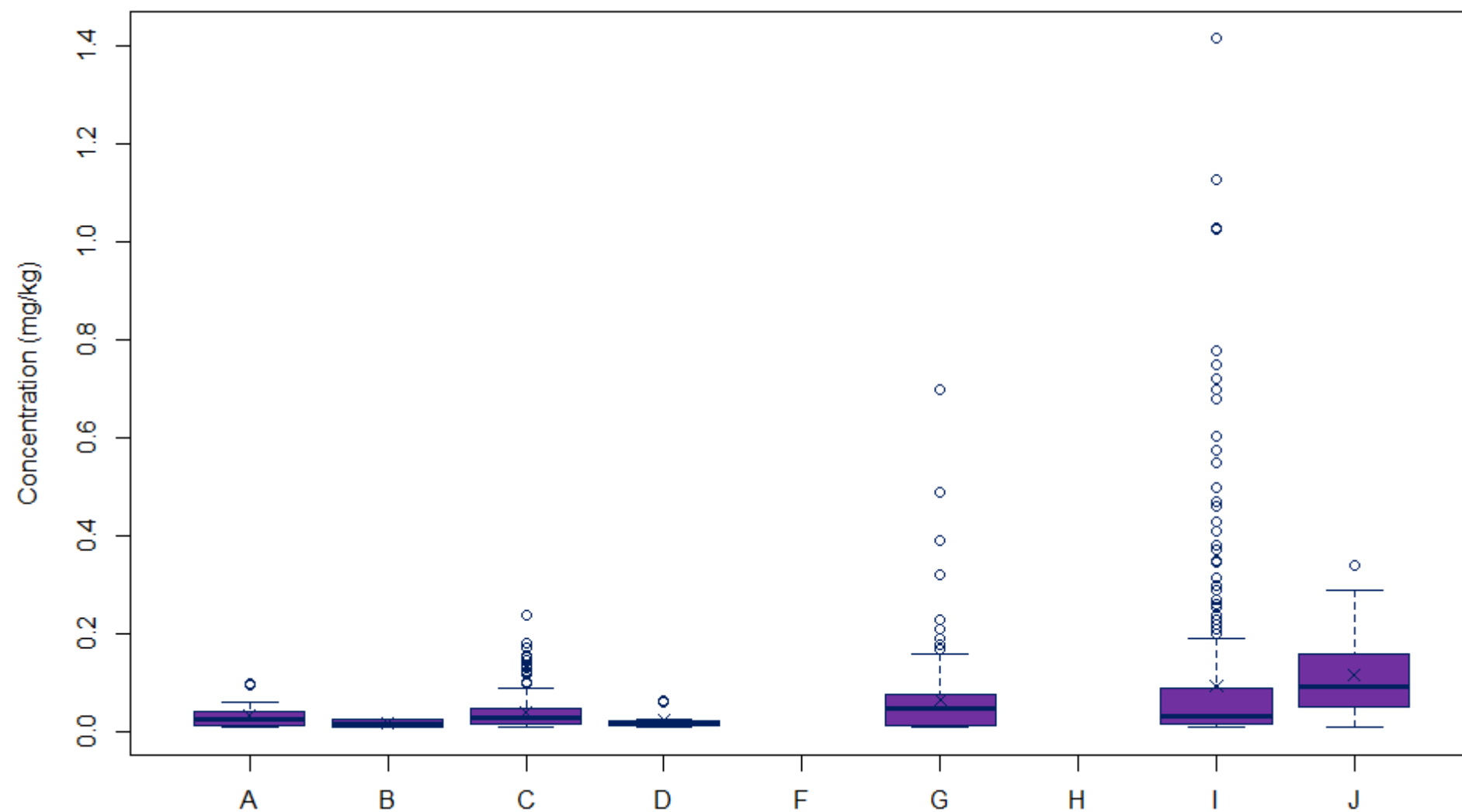


Figure C-4. Box and whisker plot of DSP concentrations for each Zone in New Zealand over the 2009-2019 period

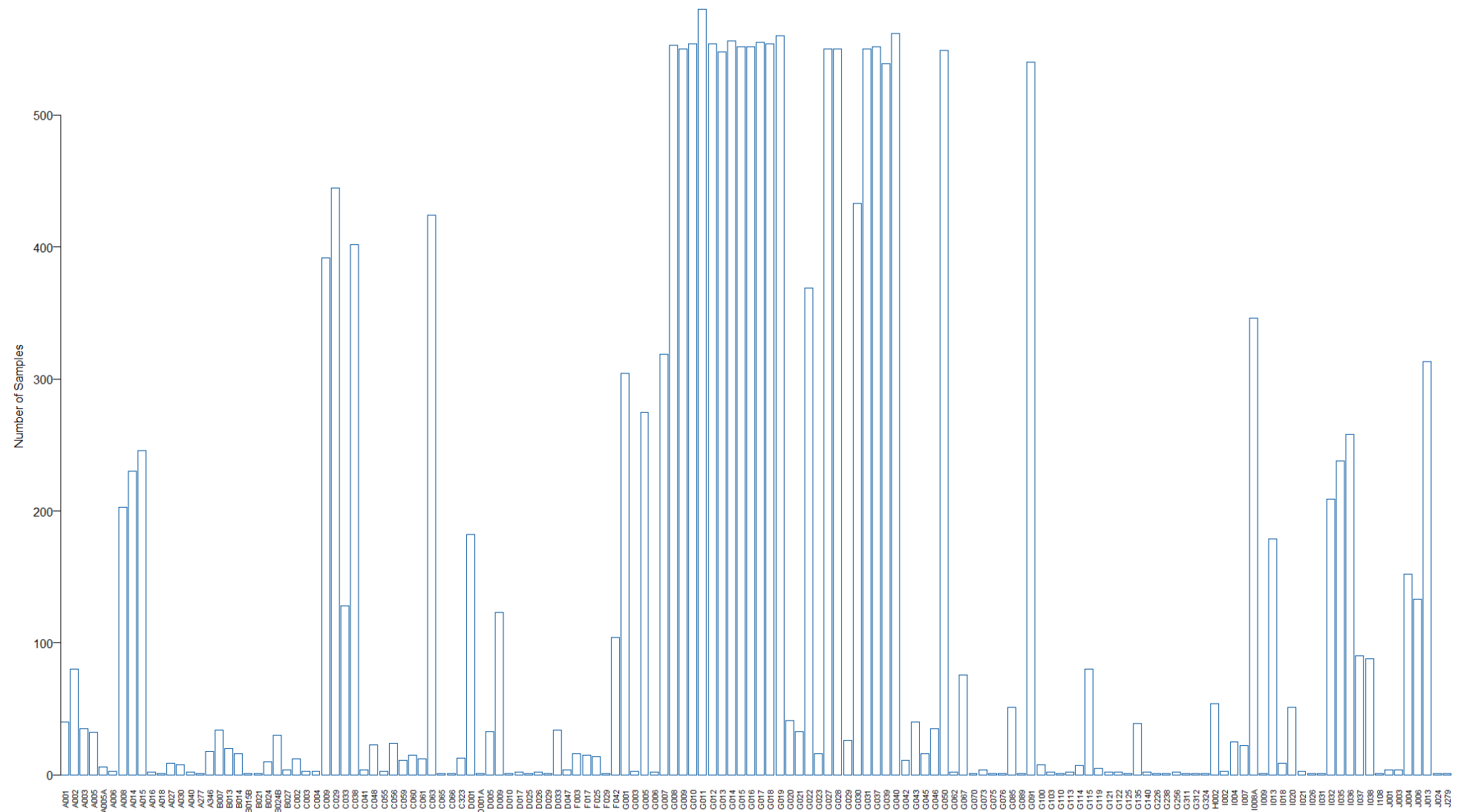


Figure C-5. Samples analysed for shellfish toxins by Site in New Zealand over the 2009-2019 period

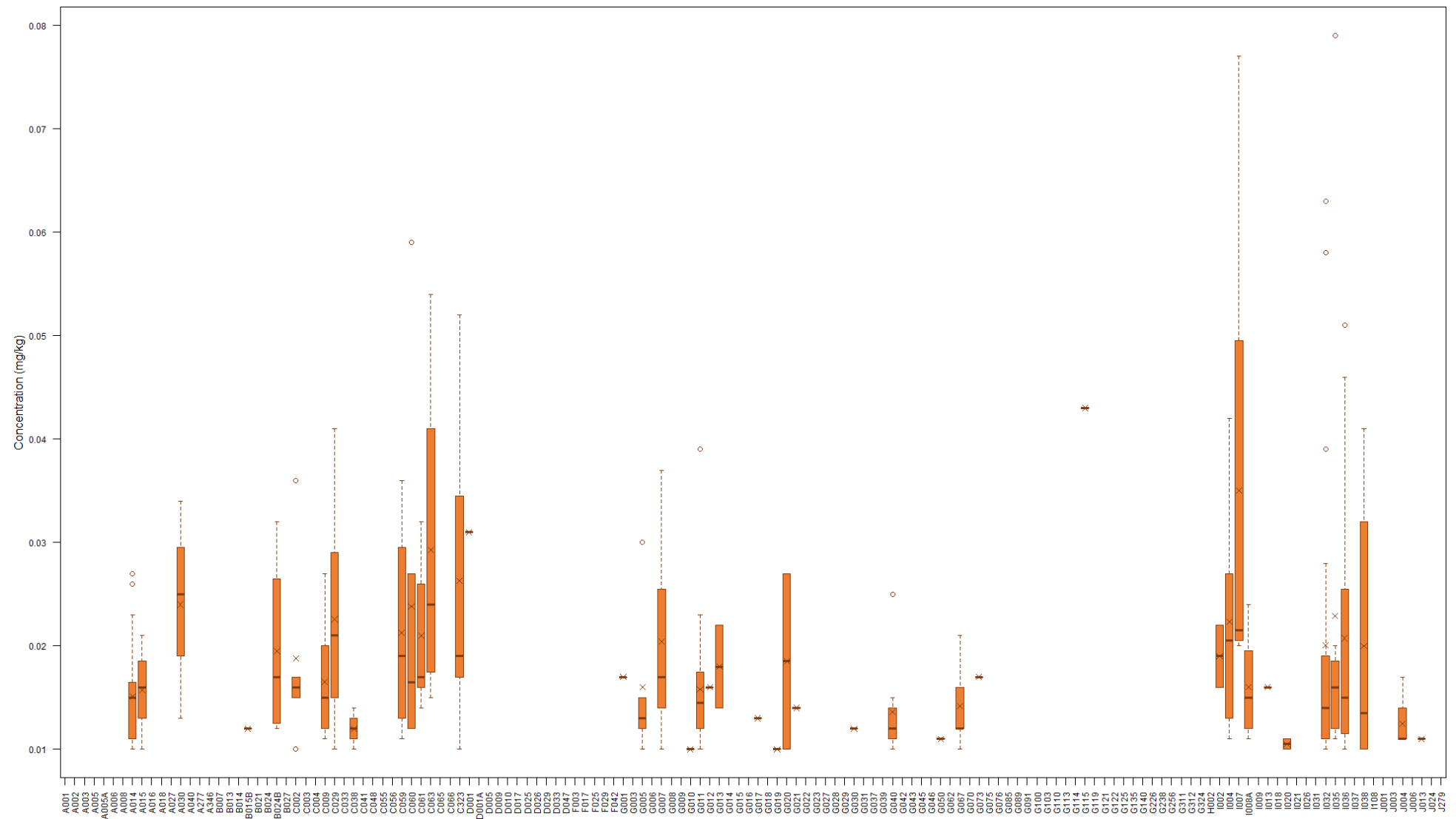


Figure C-6. Box and whisker plot of PTX2 concentrations for each Site in New Zealand over the 2009-2019 period

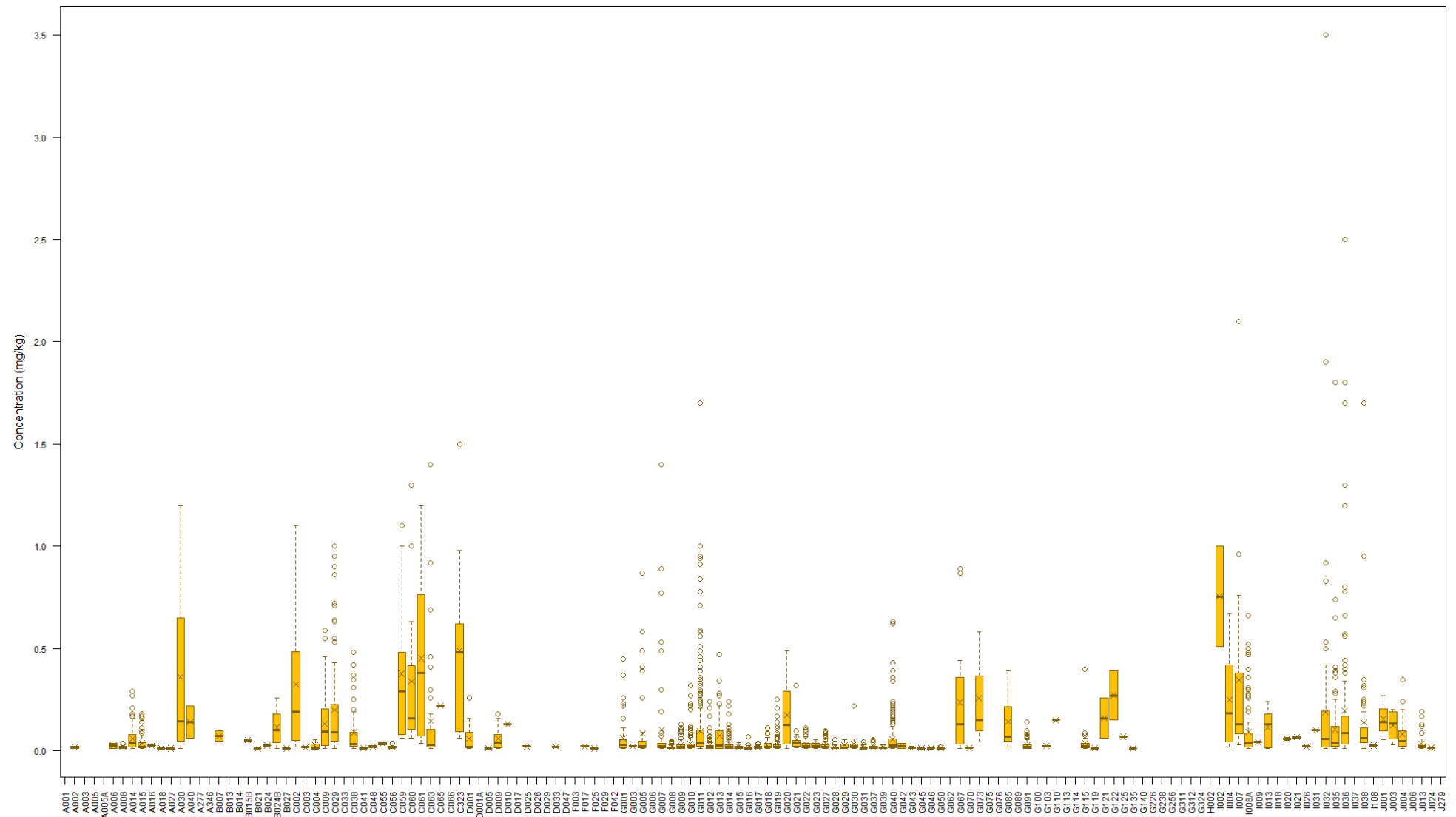


Figure C-7. Box and whisker plot of PTX2SAs concentrations for each Site in New Zealand over the 2009-2019 period

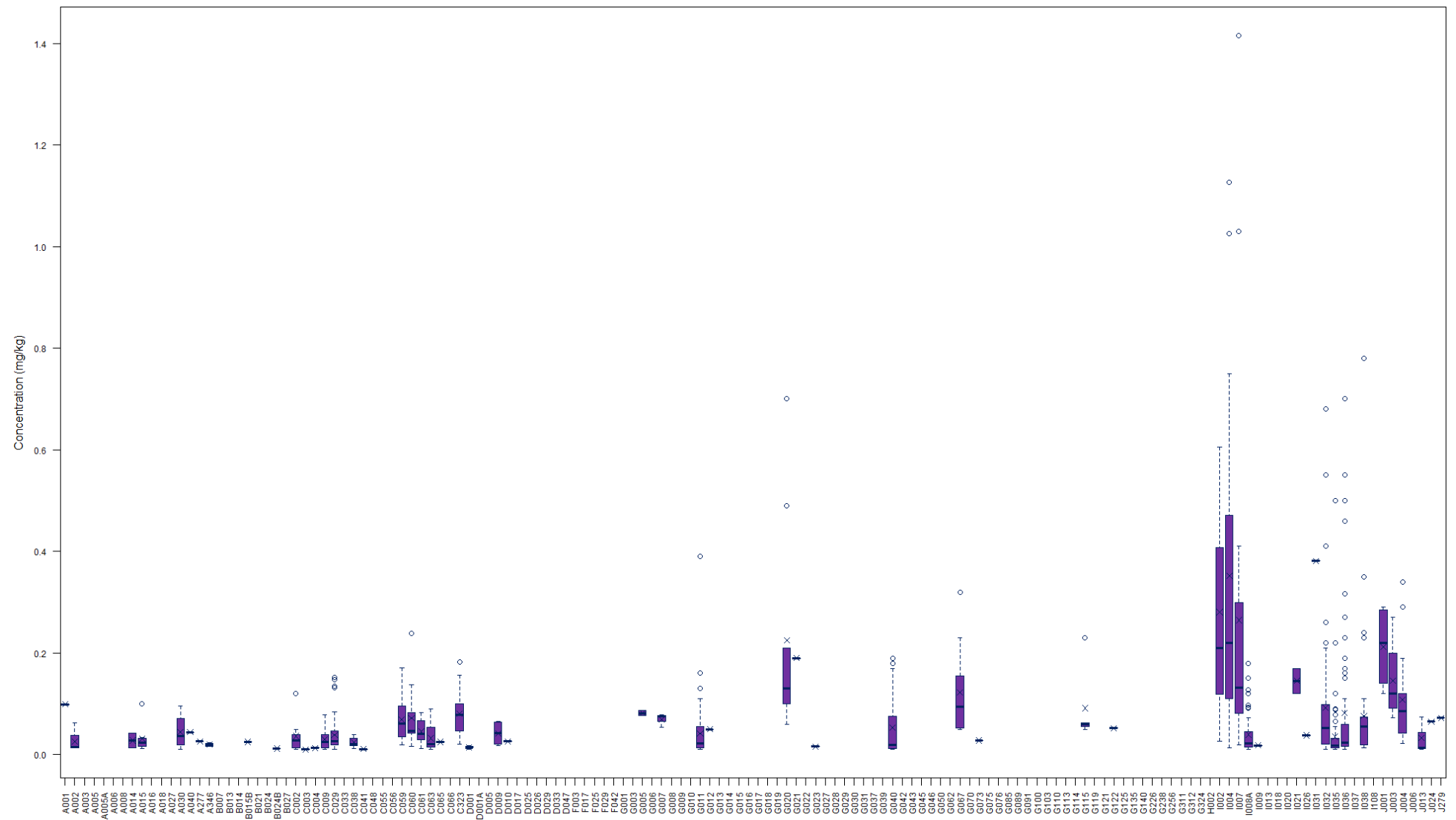


Figure C-8. Box and whisker plot of DSP concentrations for each Site in New Zealand over the 2009-2019 period

APPENDIX D. SPECIES SUMMARY

The number of samples, number of detections, percent detections, min, max, mean, median, 97.5th percentile (PCTL) concentrations each for pectenotoxin 2, pectenotoxin 2 seco acids and diarrhetic shellfish poisoning toxins for each species are summarised in Table D-1.

Concentrations of PTX2, PTX2SAs, and DSP in shellfish species in New Zealand over the 2009-2019 period are summarised in Figure D-2.

Comparisons between the concentrations of pectenotoxin 2 seco acids against pectenotoxin 2 for each shellfish matrix is shown in Figure D-5, and comparisons between the concentrations of pectenotoxin 2 seco acids against diarrhetic shellfish poisoning toxins for each shellfish matrix is shown in Figure D-6.

Table D-1. Summary of PTX2, PTX2SA, and DSP (mg/kg) in by species within New Zealand 2009-2019.

Species	Sites	No Samples	PTX2							PTX2SA							DSP						
			Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
Greenshell Mussel	83	15947	186	1.2%	0.010	0.079	0.019	0.015	0.056	3215	20.2%	0.010	3.500	0.080	0.024	0.523	655	4.1%	0.010	1.415	0.064	0.031	0.333
Pacific Oyster	22	1141	40	3.5%	0.010	0.027	0.015	0.015	0.026	225	19.7%	0.010	0.290	0.049	0.028	0.174	19	1.7%	0.010	0.100	0.029	0.020	0.100
Clam	11	1042	6	0.6%	0.013	0.027	0.018	0.016	0.026	119	11.4%	0.010	0.660	0.095	0.033	0.491	62	6.0%	0.010	0.700	0.059	0.024	0.194
Scallop	20	298	4	1.3%	0.012	0.032	0.020	0.017	0.031	45	15.1%	0.010	0.260	0.042	0.018	0.249	1	0.3%	0.012	0.012	0.012	0.012	0.012
Dredge oyster	8	228	1	0.4%	0.043	0.043	0.043	0.043	0.043	33	14.5%	0.010	0.400	0.037	0.022	0.129	3	1.3%	0.056	0.230	0.116	0.062	0.222
Surf Clam	6	97	5	5.2%	0.010	0.024	0.015	0.012	0.023	29	29.9%	0.010	0.400	0.094	0.041	0.386	10	10.3%	0.011	0.490	0.087	0.033	0.409
Blueshell Mussel	12	56	7	12.5%	0.011	0.042	0.021	0.020	0.039	40	71.4%	0.012	1.000	0.178	0.059	0.766	39	69.6%	0.013	1.126	0.267	0.150	1.031
Queen Scallop	2	52	2	3.8%	0.010	0.011	0.011	0.011	0.011	3	5.8%	0.052	0.065	0.058	0.058	0.065	0						
Tuatua	5	28	0							5	17.9%	0.010	0.021	0.013	0.011	0.020	0						
Pipi	2	19	0							0							0						
Cockle	3	17	0							2	11.8%	0.012	0.013	0.013	0.013	0.013	0						
Oyster	5	9	0							6	66.7%	0.010	0.086	0.040	0.028	0.085	2	22.2%	0.050	0.059	0.055	0.055	0.059
Abalone	3	8	0							0							0						
Geoduck	3	5	0							1	20.0%	0.014	0.014	0.014	0.014	0.014	0						
Total	144	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

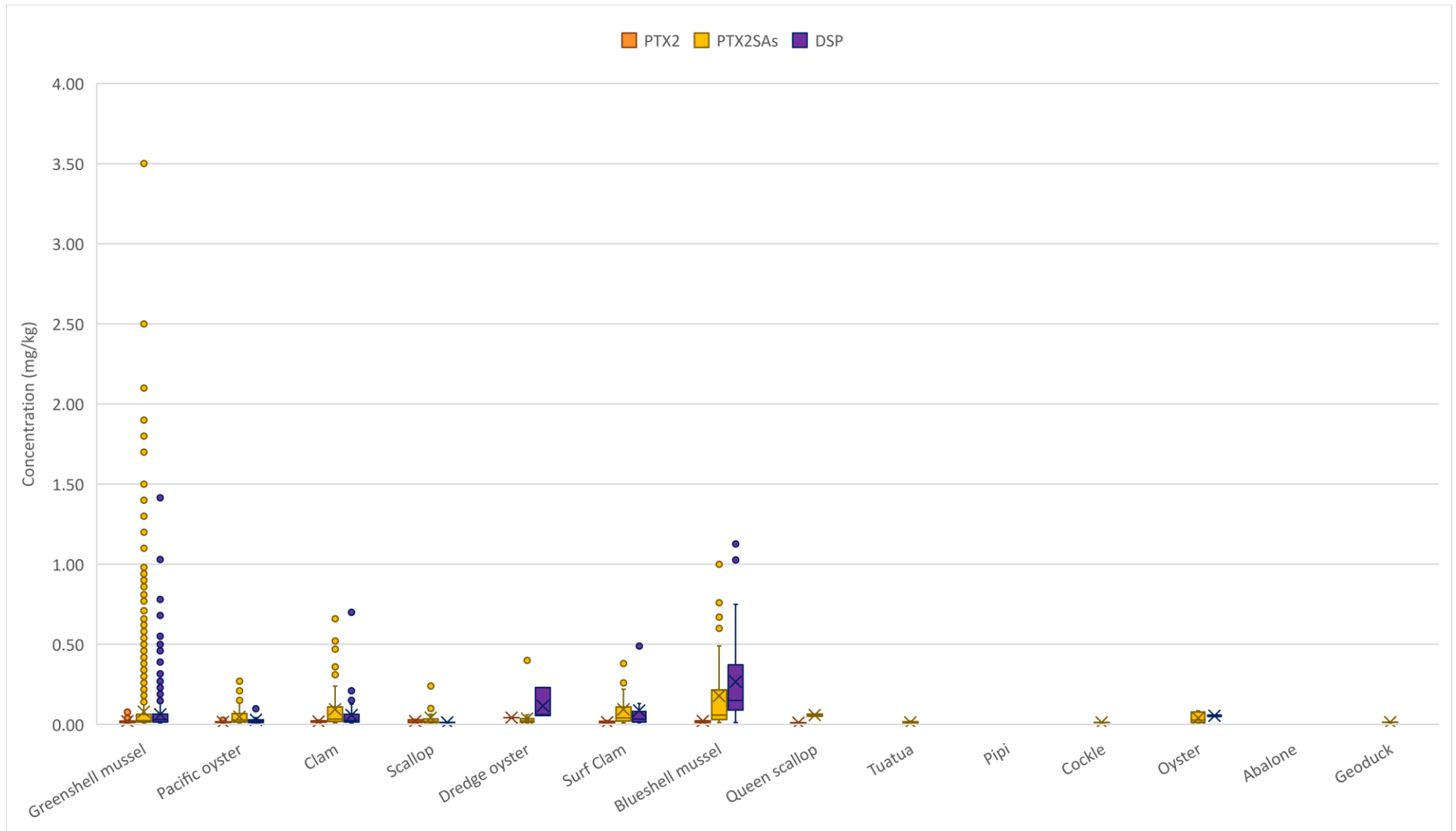


Figure D-1. Box and whisker plot of concentrations of PTX2, PTX2SAs, and DSP in shellfish species in New Zealand over the 2009-2019 period

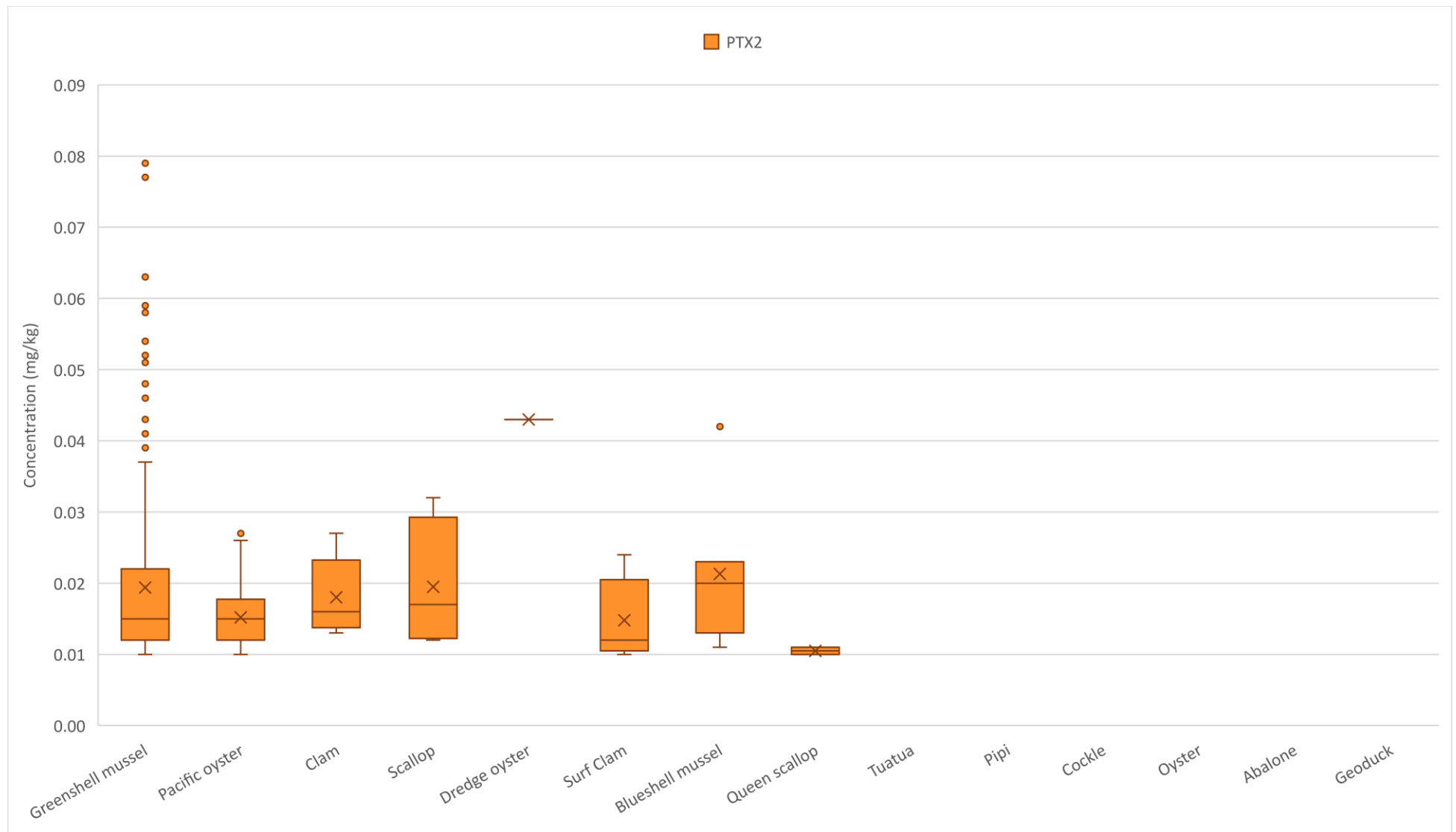


Figure D-2. Box and whisker plot of concentrations of PTX2 in shellfish species in New Zealand over the 2009-2019 period

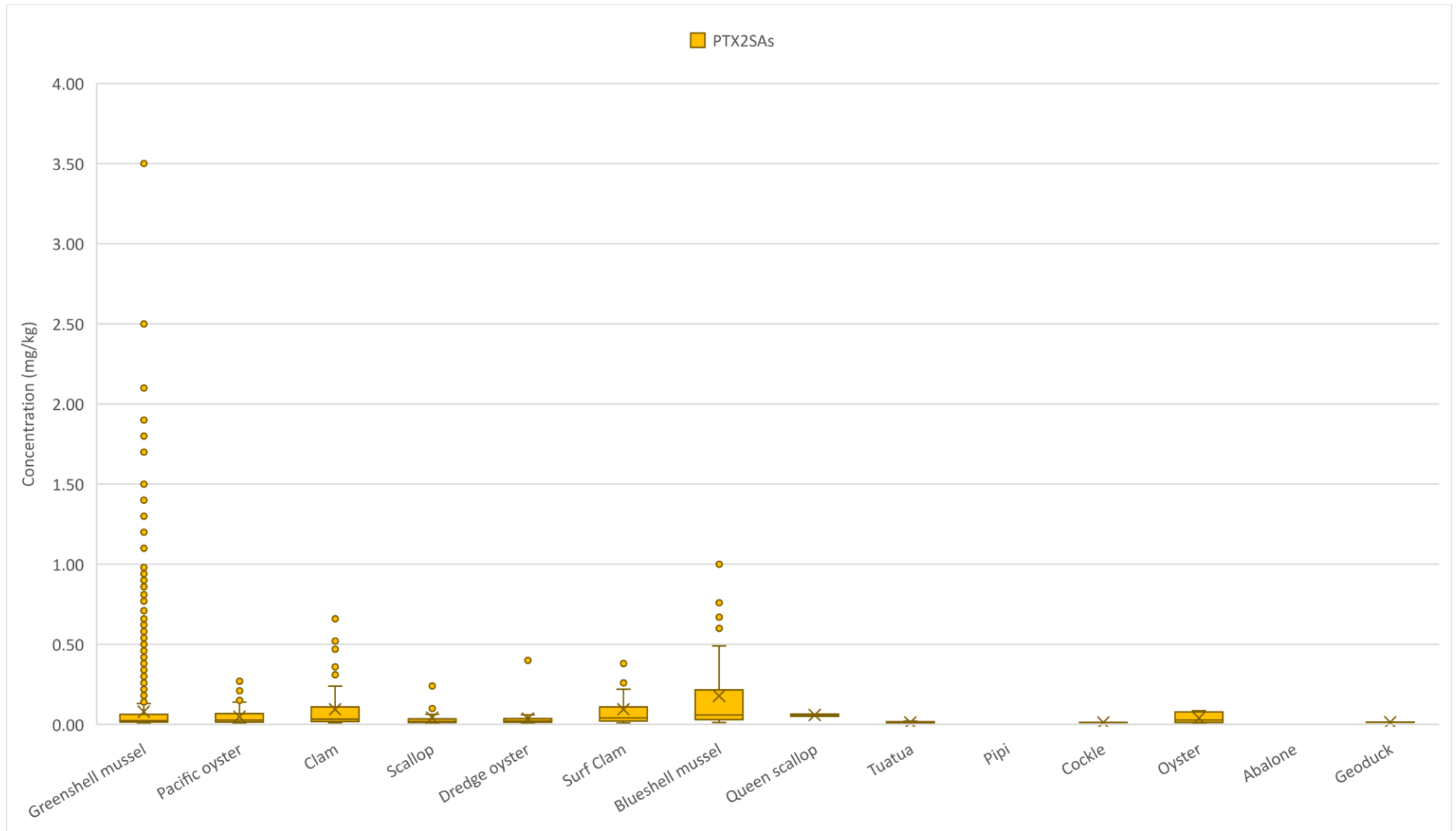


Figure D-3. Box and whisker plot of concentrations of PTX2SAs in shellfish species in New Zealand over the 2009-2019 period

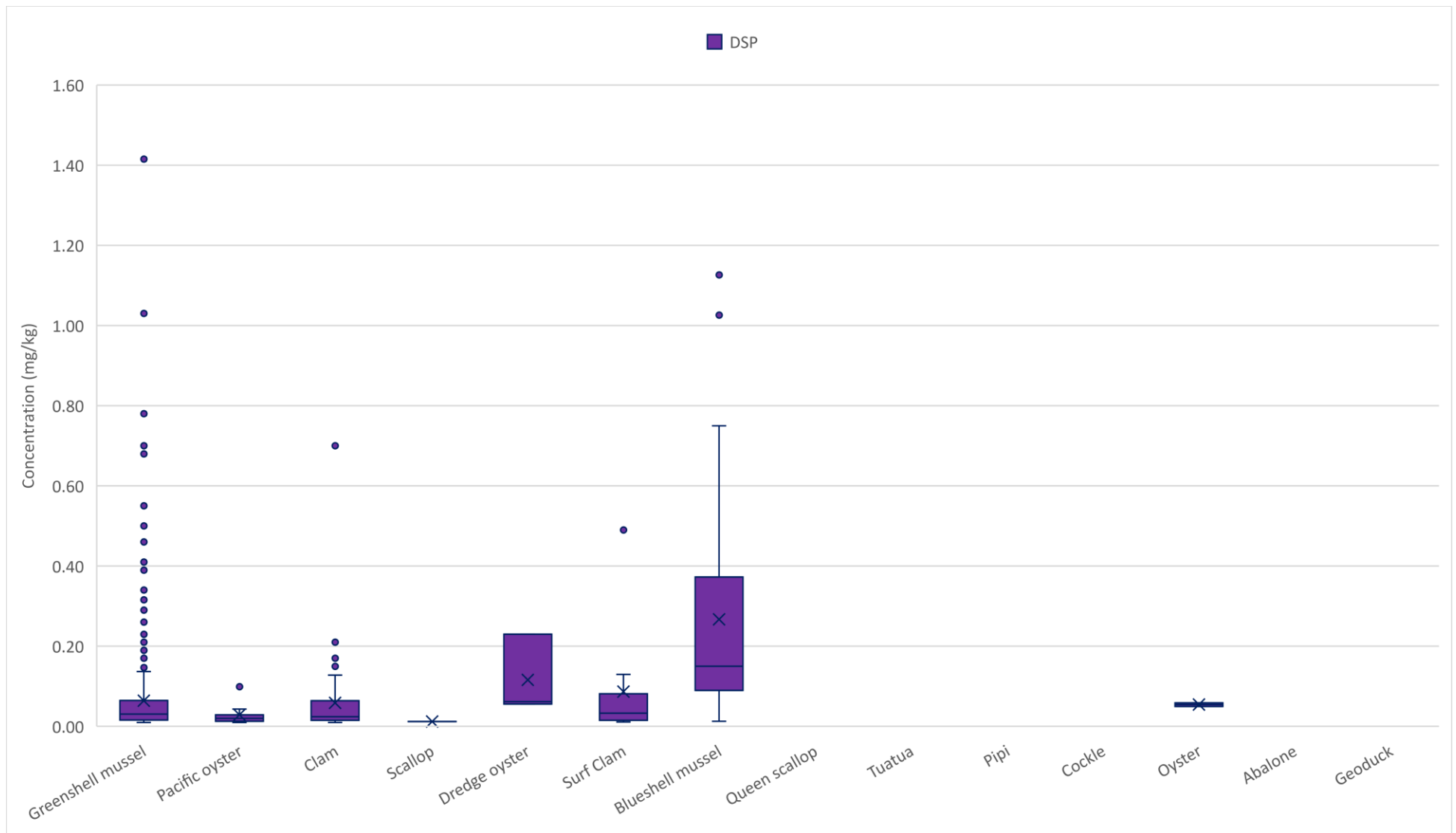


Figure D-4. Box and whisker plot of concentrations of DSP in shellfish species in New Zealand over the 2009-2019 period

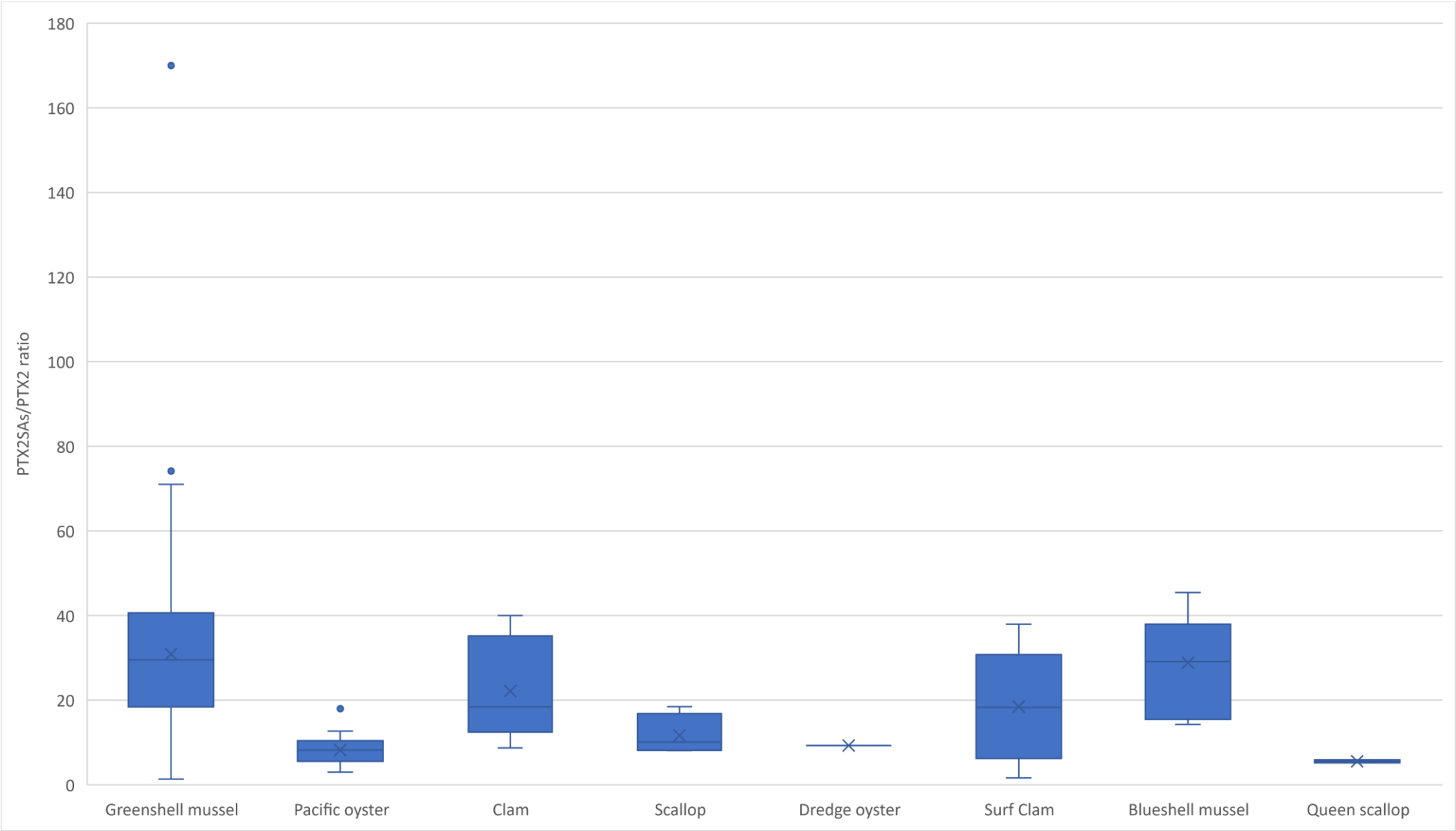


Figure D-5. Box and whisker plot of ratio between PTX2SAs and PTX in shellfish species in New Zealand over the 2009-2019 period

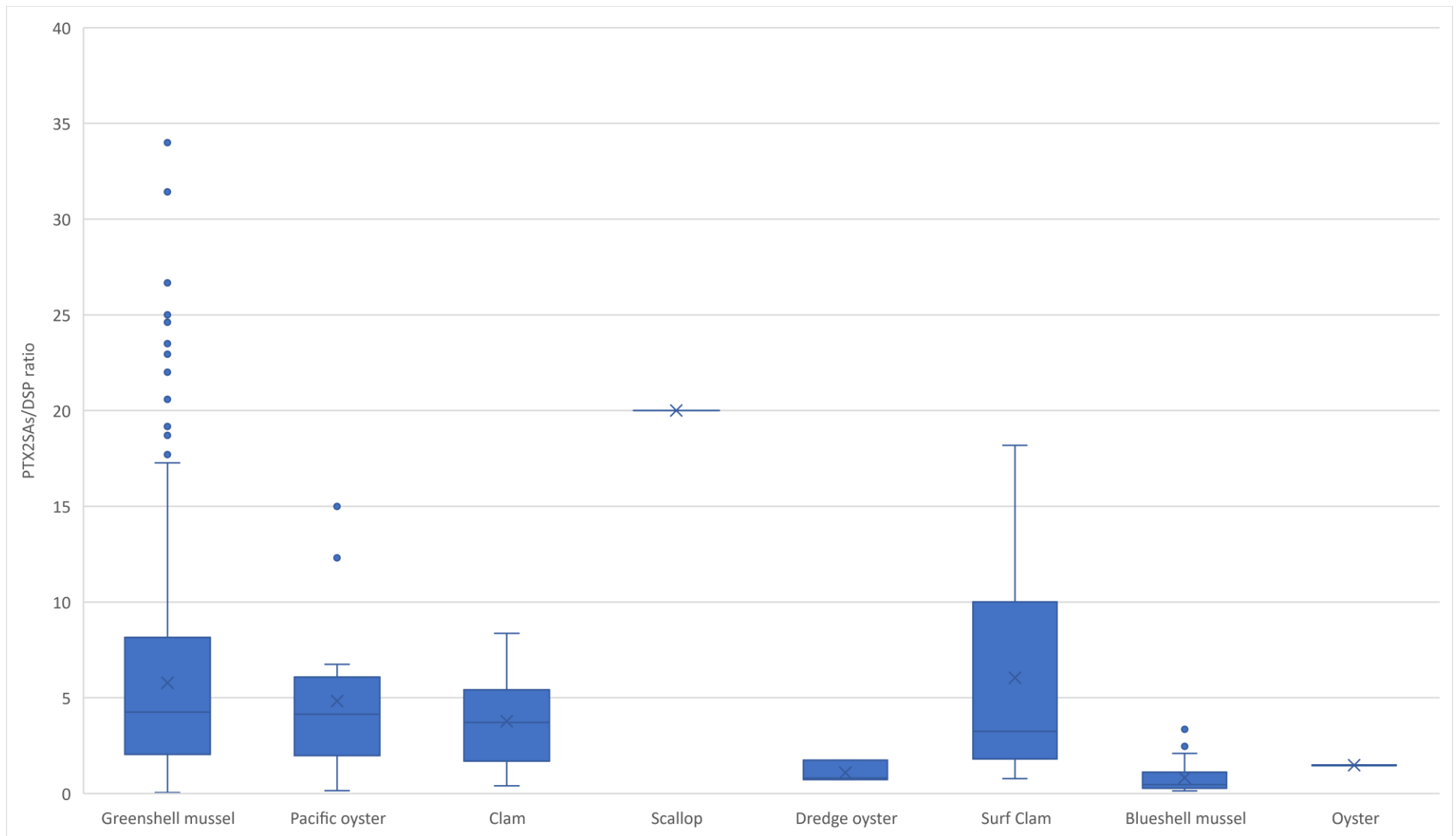


Figure D-6. Box and whisker plot of ratio between PTX2SAs and DSP in shellfish species in New Zealand over the 2009-2019 period



Figure D-7. Comparison of PTX2 concentrations against DSP in New Zealand over the 2009-2019 period by shellfish species

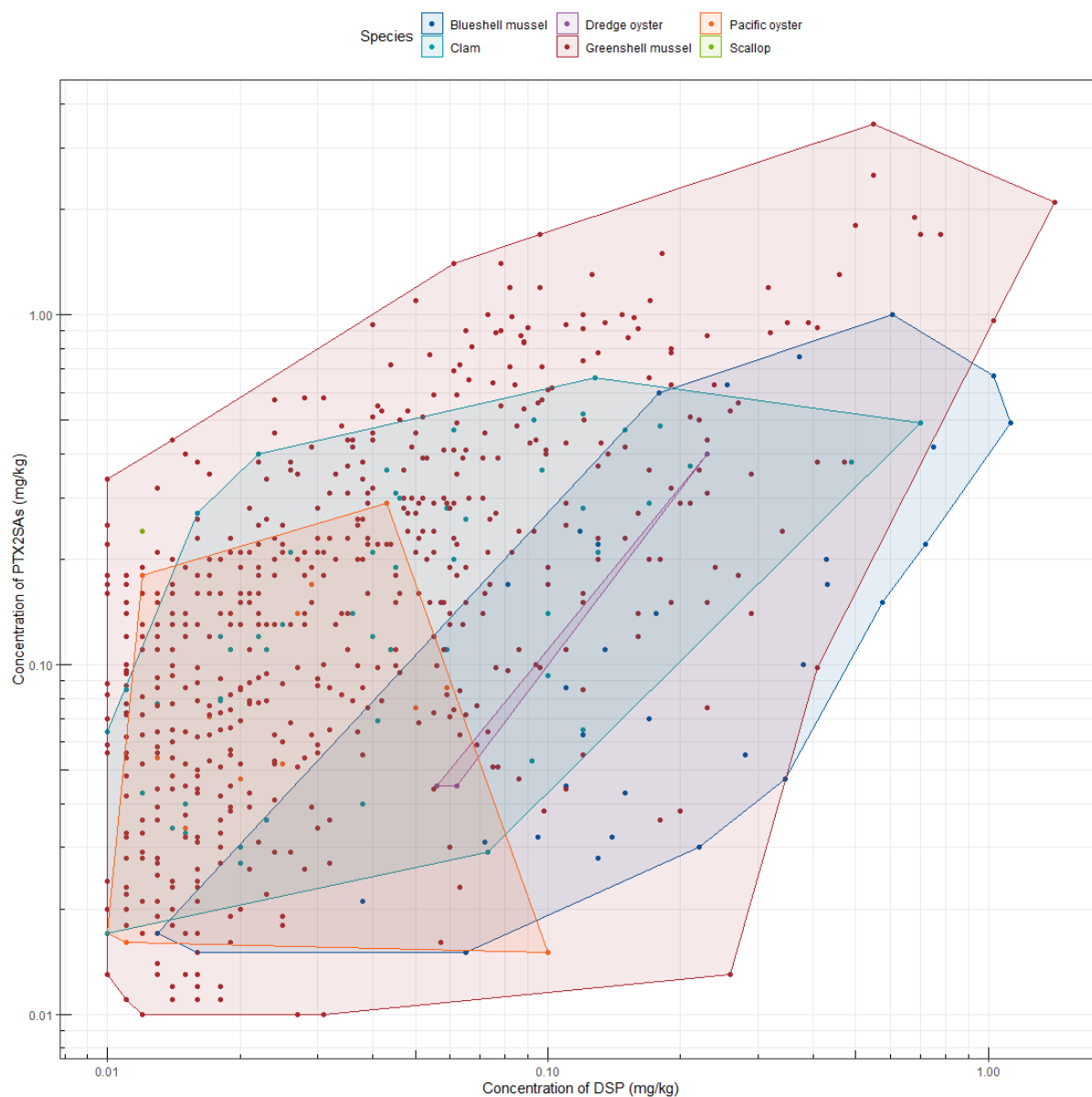


Figure D-8. Comparison of PTX2SAs concentrations against DSP in New Zealand over the 2009-2019 period by shellfish species

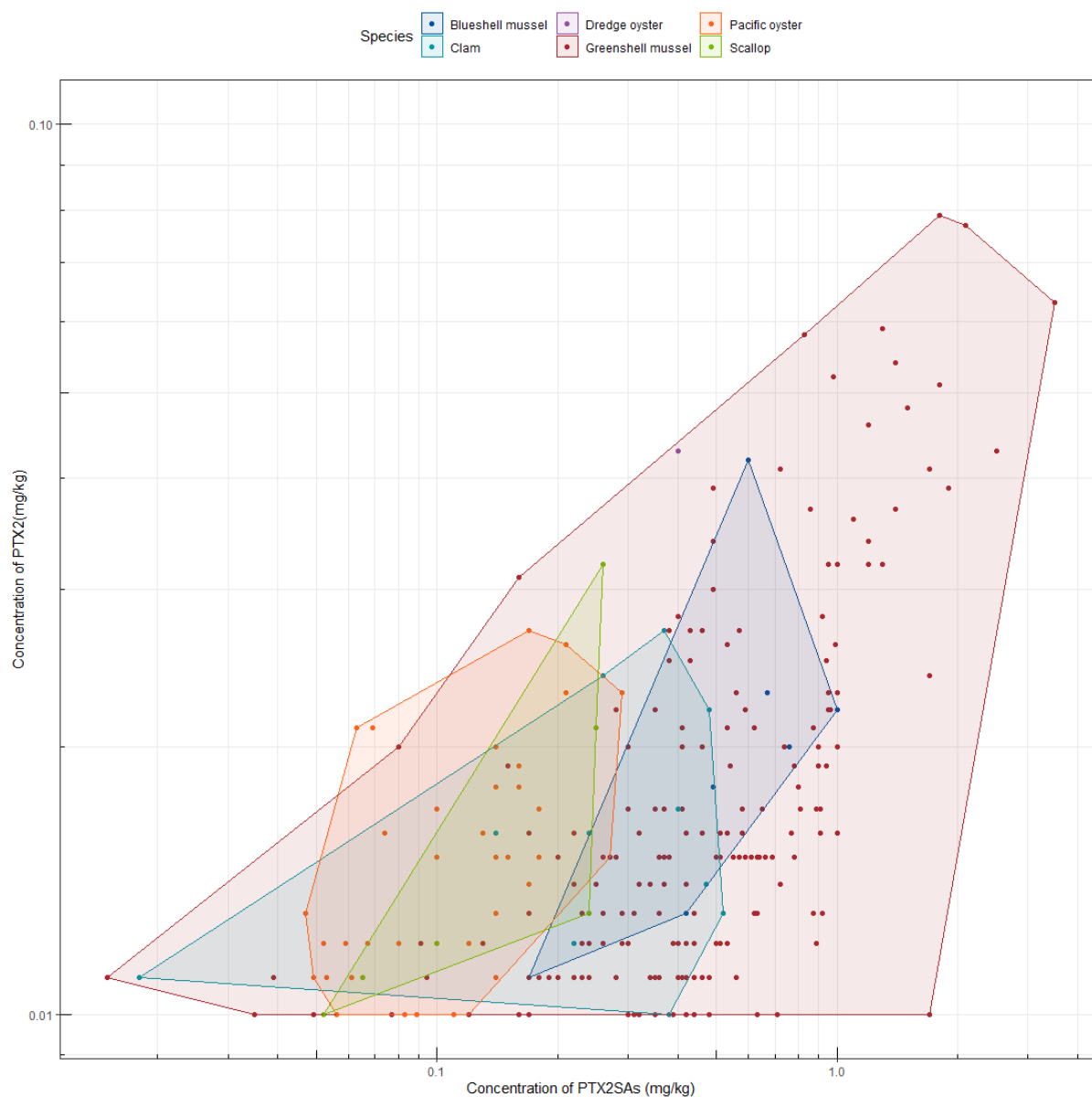


Figure D-9. Comparison of PTX2 concentrations against PTX2SAs in New Zealand over the 2009-2019 period by shellfish species

APPENDIX E. ANNUAL AND SEASONAL SUMMARIES

Table E-1. Summary of PTX2, PTX2SA, and DSP (mg/kg) in by year within New Zealand 2009-2019.

Year	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
2009	1688	56	3.3%	0.010	0.063	0.019	0.015	0.048	528	31.3%	0.010	3.500	0.102	0.025	0.778	35	2.1%	0.047	0.700	0.230	0.130	0.700
2010	1618	14	0.9%	0.010	0.041	0.014	0.011	0.035	245	15.1%	0.010	1.700	0.066	0.022	0.435	31	1.9%	0.050	0.780	0.132	0.071	0.488
2011	1684	21	1.2%	0.010	0.043	0.016	0.014	0.038	353	21.0%	0.010	0.950	0.052	0.021	0.282	54	3.2%	0.050	0.350	0.103	0.076	0.237
2012	1647	13	0.8%	0.011	0.025	0.015	0.013	0.024	330	20.0%	0.010	0.500	0.044	0.019	0.270	18	1.1%	0.050	0.260	0.096	0.066	0.243
2013	1723	5	0.3%	0.010	0.021	0.017	0.019	0.021	270	15.7%	0.010	1.700	0.058	0.024	0.228	5	0.3%	0.052	0.120	0.088	0.096	0.119
2014	1776	10	0.6%	0.010	0.016	0.013	0.014	0.016	390	22.0%	0.010	1.000	0.054	0.021	0.328	8	0.5%	0.051	0.160	0.079	0.067	0.150
2015	1871	66	3.5%	0.010	0.059	0.021	0.017	0.053	417	22.3%	0.010	1.500	0.144	0.031	0.986	162	8.7%	0.010	0.238	0.047	0.036	0.152
2016	1836	21	1.1%	0.010	0.079	0.026	0.021	0.078	363	19.8%	0.010	2.100	0.095	0.033	0.529	127	6.9%	0.010	1.415	0.084	0.021	0.486
2017	1924	14	0.7%	0.010	0.027	0.017	0.017	0.026	352	18.3%	0.010	0.670	0.063	0.030	0.290	174	9.0%	0.010	1.126	0.062	0.019	0.541
2018	1857	12	0.6%	0.011	0.058	0.023	0.017	0.054	285	15.3%	0.010	0.830	0.076	0.028	0.469	97	5.2%	0.010	0.346	0.038	0.023	0.157
2019	1323	19	1.4%	0.010	0.024	0.014	0.012	0.023	190	14.4%	0.010	0.580	0.085	0.036	0.380	80	6.0%	0.010	0.340	0.059	0.022	0.290
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

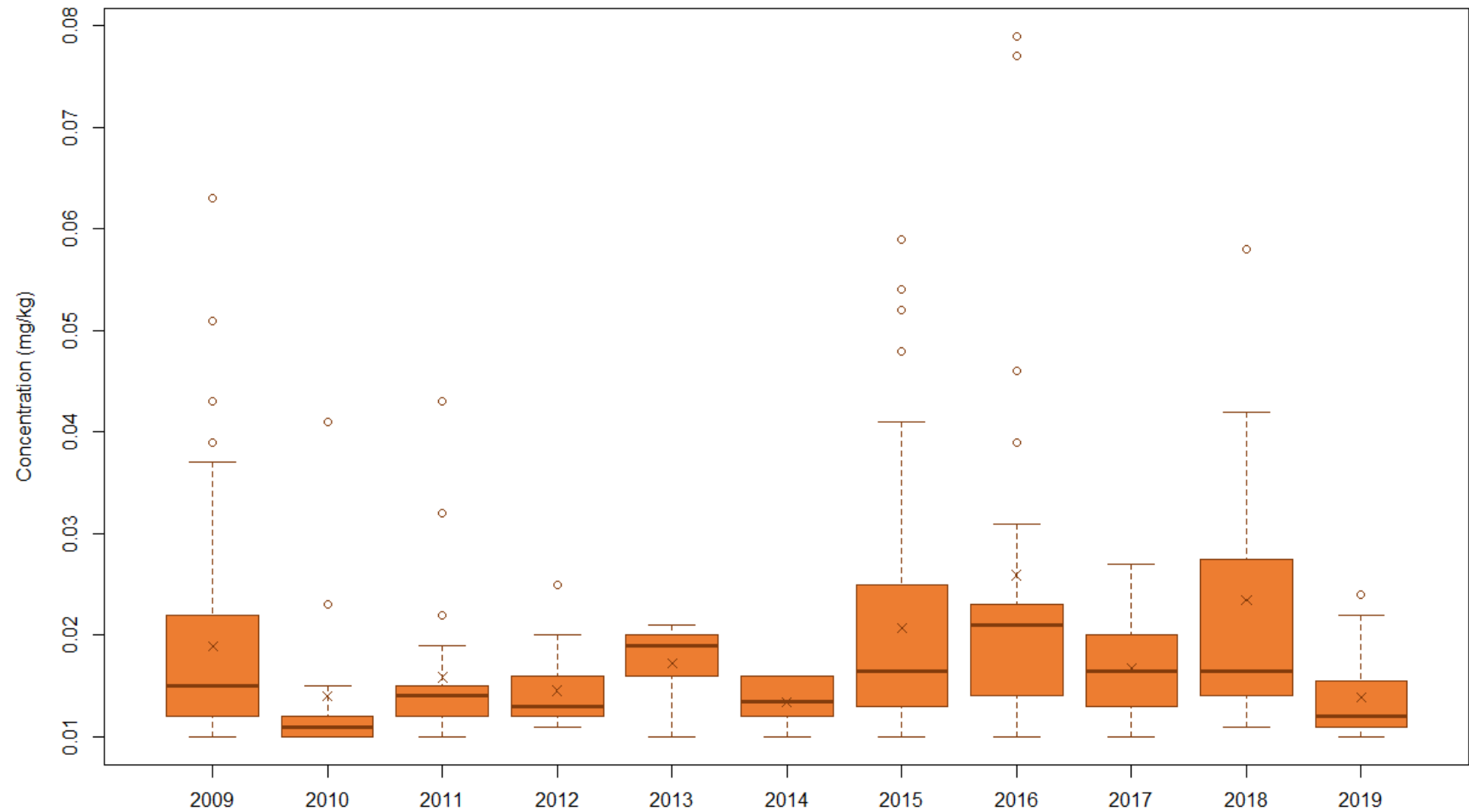


Figure E-1. Box and whisker plot of PTX2 concentrations for each year in New Zealand over the 2009-2019 period

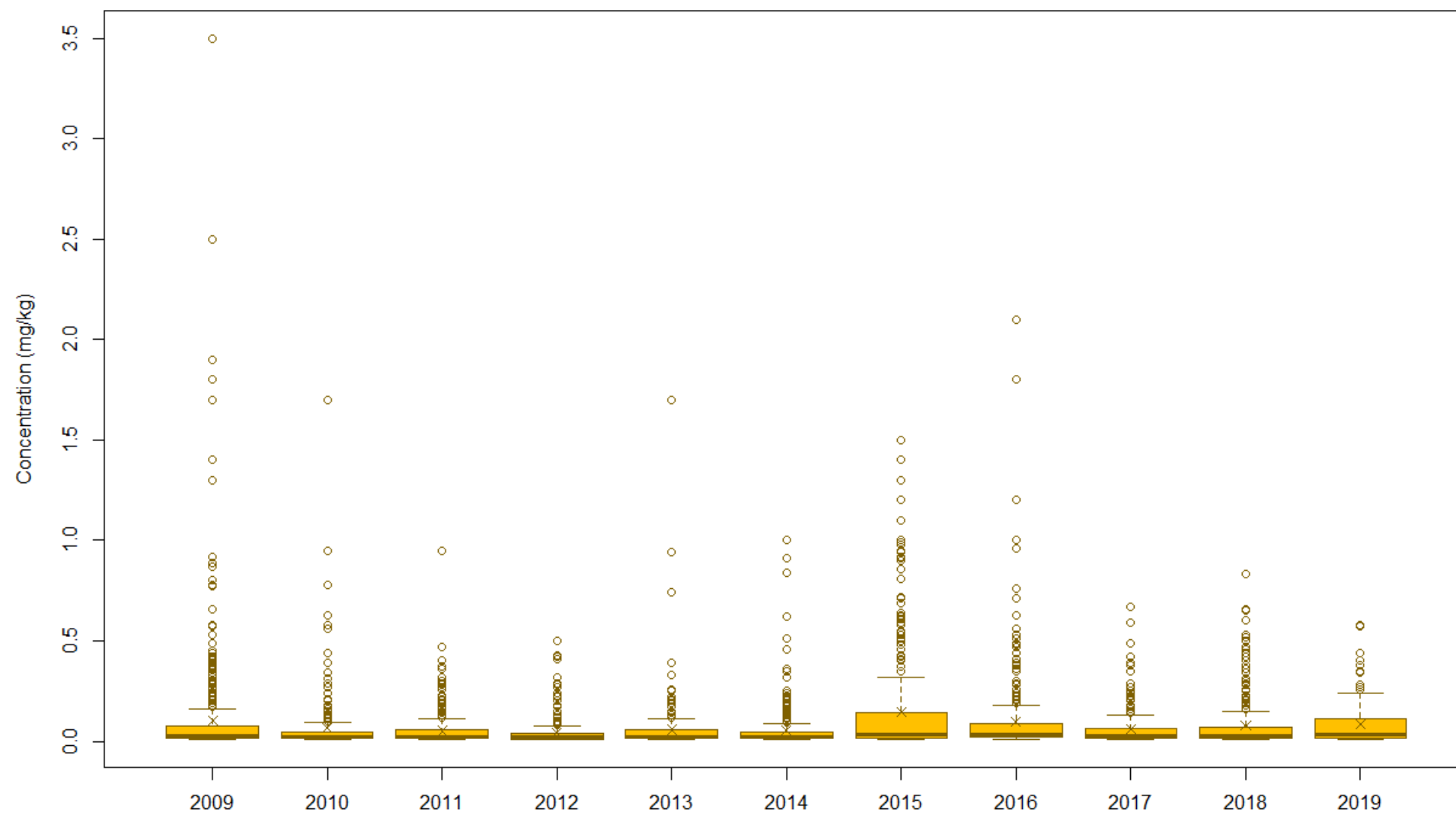


Figure E-2. Box and whisker plot of PTX2SAs concentrations for each year in New Zealand over the 2009-2019 period

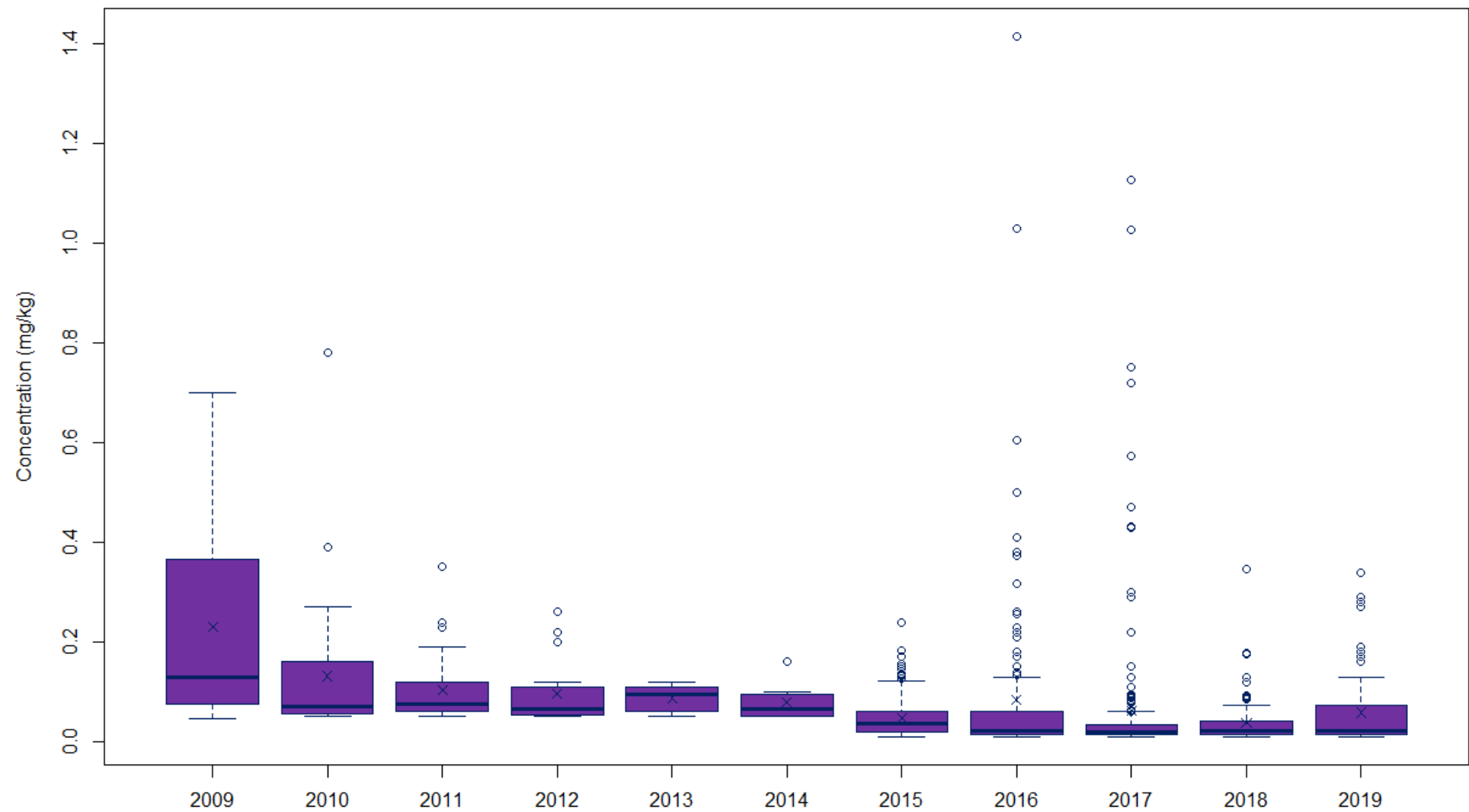


Figure E-3. Box and whisker plot of DSP concentrations for each year in New Zealand over the 2009-2019 period

Table E-2. Summary of PTX2, PTX2SA, and DSP (mg/kg) in by month within New Zealand 2009-2019.

Month	No Samples	PTX2							PTX2SA							DSP						
		Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL	Detections	%detected	Min	Max	Mean	Median	97.5 PCTL
January	1615	10	0.6%	0.011	0.043	0.020	0.016	0.041	176	10.9%	0.010	0.950	0.062	0.024	0.356	55	3.4%	0.010	0.350	0.070	0.053	0.297
February	1617	30	1.9%	0.010	0.027	0.014	0.012	0.026	155	9.6%	0.010	0.580	0.067	0.049	0.232	34	2.1%	0.010	0.290	0.052	0.029	0.208
March	1679	10	0.6%	0.010	0.023	0.013	0.011	0.021	155	9.2%	0.010	1.700	0.078	0.023	0.392	50	3.0%	0.010	0.390	0.070	0.031	0.329
April	1594	11	0.7%	0.010	0.039	0.016	0.014	0.035	168	10.5%	0.010	0.940	0.083	0.033	0.440	54	3.4%	0.010	0.280	0.056	0.030	0.187
May	1634	10	0.6%	0.011	0.022	0.015	0.015	0.022	247	15.1%	0.010	0.570	0.058	0.025	0.297	51	3.1%	0.010	0.135	0.032	0.020	0.120
June	1574	15	1.0%	0.010	0.058	0.020	0.016	0.052	309	19.6%	0.010	0.830	0.060	0.022	0.380	52	3.3%	0.010	0.179	0.027	0.018	0.082
July	1594	9	0.6%	0.011	0.027	0.016	0.015	0.026	326	20.5%	0.010	0.670	0.060	0.028	0.333	64	4.0%	0.010	1.026	0.077	0.019	0.733
August	1563	21	1.3%	0.010	0.052	0.022	0.018	0.047	273	17.5%	0.010	1.100	0.078	0.024	0.498	58	3.7%	0.010	1.126	0.074	0.025	0.513
September	1514	47	3.1%	0.010	0.059	0.021	0.018	0.054	409	27.0%	0.010	1.900	0.121	0.025	0.982	100	6.6%	0.010	0.680	0.089	0.063	0.421
October	1593	50	3.1%	0.010	0.046	0.017	0.015	0.034	696	43.7%	0.010	1.700	0.087	0.026	0.603	144	9.0%	0.010	1.030	0.089	0.045	0.431
November	1542	28	1.8%	0.010	0.079	0.024	0.017	0.078	544	35.3%	0.010	2.500	0.079	0.025	0.494	87	5.6%	0.010	1.415	0.089	0.029	0.543
December	1428	10	0.7%	0.010	0.063	0.021	0.013	0.058	265	18.6%	0.010	3.500	0.075	0.024	0.330	42	2.9%	0.011	0.780	0.098	0.051	0.547
Total	18947	251	1.3%	0.010	0.079	0.019	0.015	0.052	3723	19.6%	0.010	3.500	0.079	0.025	0.500	791	4.2%	0.010	1.415	0.073	0.033	0.430

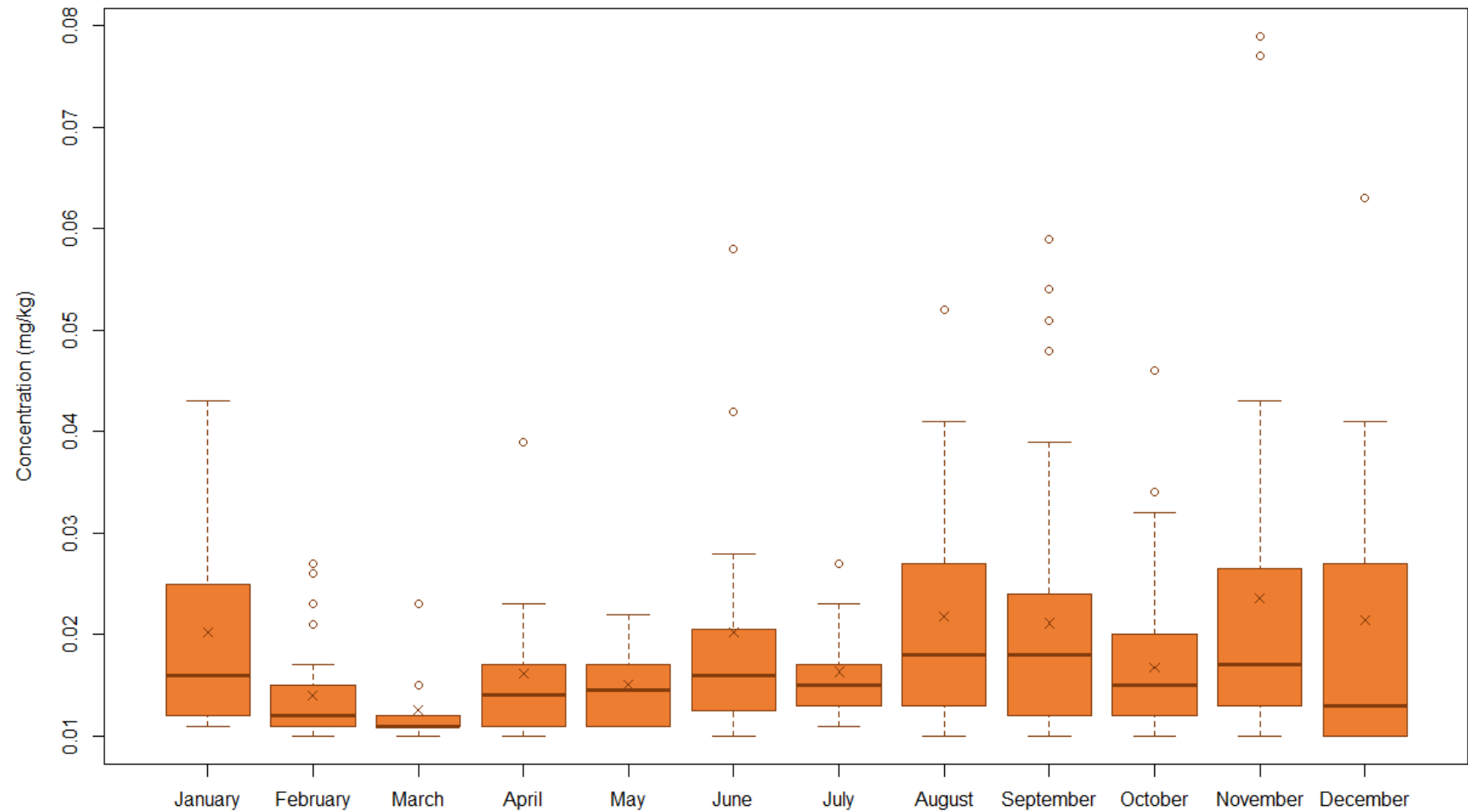


Figure E-4. Box and whisker plot of PTX2 concentrations for each month in New Zealand over the 2009-2019 period

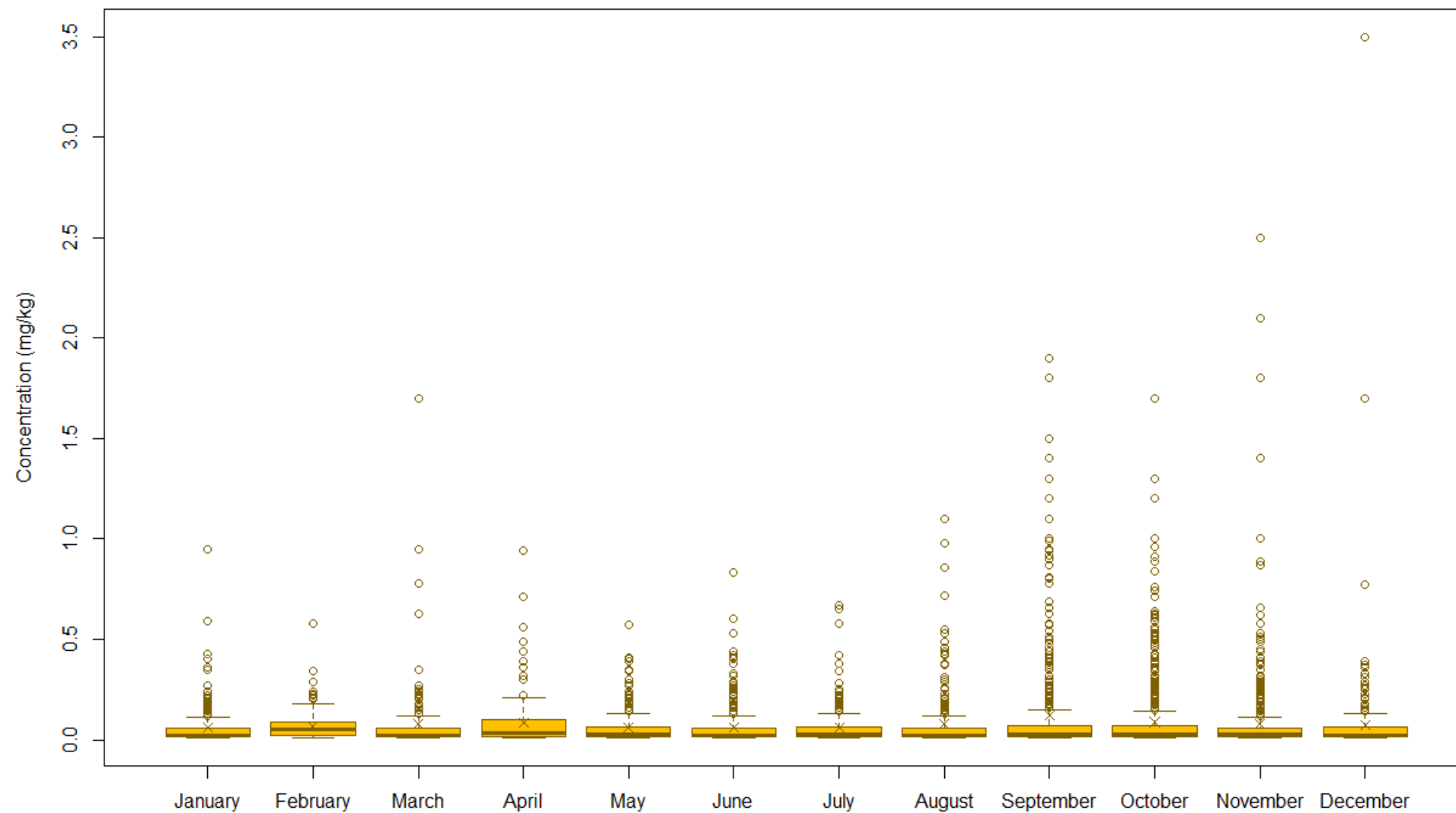


Figure E-5. Box and whisker plot of PTX2SAs concentrations for each month in New Zealand over the 2009-2019 period

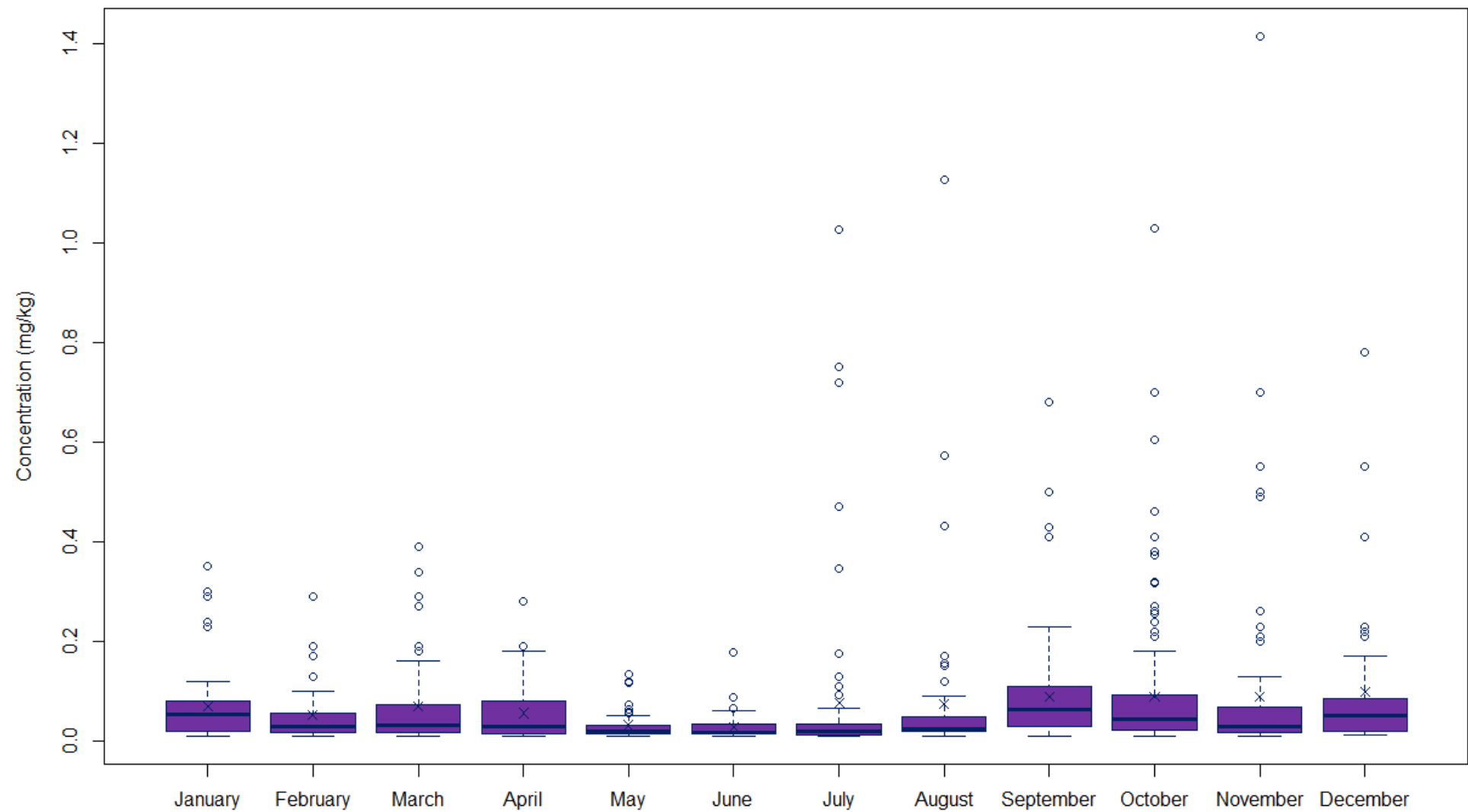


Figure E-6. Box and whisker plot of DSP concentrations for each month in New Zealand over the 2009-2019 period

APPENDIX F. PECTENOTOXIN PROFILES

Samples over the C|201507-12 and I|pbk|201607-201703 bloom events were reprocessed to quantify pectenotoxins 1, 6 and 11 which are acquired in the LC-MS method of analysis although not processed as part of the routine monitoring programme.

No detections of pectenotoxins 1, 6 or 11 were observed above the 0.01 mg/kg reporting limit. Trace detections were observed for pectenotoxin 1 and 11 in some samples. Pectenotoxin profiles were assessed including all trace detections below the reporting limit.

The number of samples, number of detections, percent detections, mean, 97.5th percentile (PCTL) and max concentrations each for pectenotoxin 1, 2, 6 and 11 for each site in bloom are summarised in Table F-1 for bloom event C|201507-12, Table F-3 for bloom event I|pbk|201607-201703, Table F-5 for bloom event I|bpk|200904-201005, Table F-7 for bloom event Bloom Event B|201509-12, and Table F-9 for bloom event A|boi|201506-12.

The number of samples, number of detections, percent detections, mean, 97.5th percentile (PCTL) and max concentrations each for pectenotoxin 1, 2, 6 and 11 for each species are summarised in Table F-2 for bloom event C|201507-12, Table F-4 for bloom event I|pbk|201607-201703, Table F-6 for bloom event I|bpk|200904-201005, Table F-8 for bloom event Bloom Event B|201509-12, and Table F-10 for bloom event A|boi|201506-12.

Concentrations of pectenotoxins 1, 2, 6 and 11 over the duration blooms are shown in Figure F-1 for bloom event C|201507-12, Figure F-4 for bloom event I|pbk|201607-201703, Figure F-7 for bloom event I|bpk|200904-201005, Figure F-10 for bloom event Bloom Event B|201509-12, and Figure F-13 for bloom event A|boi|201506-12.

The concentrations of pectenotoxins 1, 2, 6, 11 and pectenotoxins 2 seco acids are shown in **Error! Reference source not found.** for bloom event C|201507-12, **Error! Reference source not found.** for bloom event I|pbk|201607-201703, Figure F-8 for bloom event I|bpk|200904-201005, Figure F-11 for bloom event Bloom Event B|201509-12, and Figure F-14 for bloom event A|boi|201506-12.

The concentrations of pectenotoxins 1, 2, 6, and 11 (excluding pectenotoxin 2 seco acids) are shown in Figure F-3 for bloom event C|201507-12, Figure F-6 for bloom event I|pbk|201607-201703, Figure F-9 for bloom event I|bpk|200904-201005, Figure F-12 for bloom event Bloom Event B|201509-12, and Figure F-15 for bloom event A|boi|201506-12.

F.1. Bloom Event C|201507-12

Table F-1. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from sites within bloom C|201507-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
C002	12	12	100%	0.00911	0.03078	0.03600	2	17%	0.00025	0.00030	0.00030	0					0				
C003	3	3	100%	0.00367	0.00546	0.00560	0					1	33%	0.00040	0.00040	0.00040	0				
C004	3	3	100%	0.00353	0.00500	0.00510	0					2	67%	0.00040	0.00040	0.00040	0				
C009	31	28	90%	0.00702	0.02065	0.02200	2	6%	0.00010	0.00010	0.00010	0					0				
C029	33	31	94%	0.00971	0.03800	0.04100	8	24%	0.00015	0.00020	0.00020	0					0				
C038	29	25	86%	0.00393	0.01340	0.01400	2	7%	0.00010	0.00010	0.00010	0					0				
C041	4	4	100%	0.00270	0.00400	0.00410	0					0					0				
C056	3	2	67%	0.00060	0.00108	0.00110	0					0					0				
C059	11	11	100%	0.01046	0.03275	0.03600	1	9%	0.00020	0.00020	0.00020	0					0				
C060	15	15	100%	0.01174	0.04780	0.05900	2	13%	0.00010	0.00010	0.00010	0					0				
C061	12	12	100%	0.01086	0.03035	0.03200	4	33%	0.00028	0.00039	0.00040	0					0				
C063	25	23	92%	0.00693	0.03970	0.05400	3	12%	0.00027	0.00030	0.00030	0					0				
C065	1	1	100%	0.00600	0.00600	0.00600	0					0					0				
C323	13	13	100%	0.01597	0.05080	0.05200	4	31%	0.00025	0.00039	0.00040	0					0				
Total	195	183	94%	0.00838	0.03880	0.05900	28	14%	0.00019	0.00040	0.00040	3	2%	0.00040	0.00040	0.00040	0				

Table F-2. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from different shellfish species within bloom C|201507-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
Greenshell Mussel	182	171	94%	0.00877	0.04000	0.05900	28	15%	0.00019	0.00040	0.00040	0					0				
Pacific Oyster	10	10	100%	0.00324	0.00549	0.00560	0					3	30%	0.00040	0.00040	0.00040	0				
Scallop	3	2	67%	0.00060	0.00108	0.00110	0					0					0				
Total	195	183	94%	0.00838	0.03880	0.05900	28	14%	0.00019	0.00040	0.00040	3	2%	0.00040	0.00040	0.00040	0				

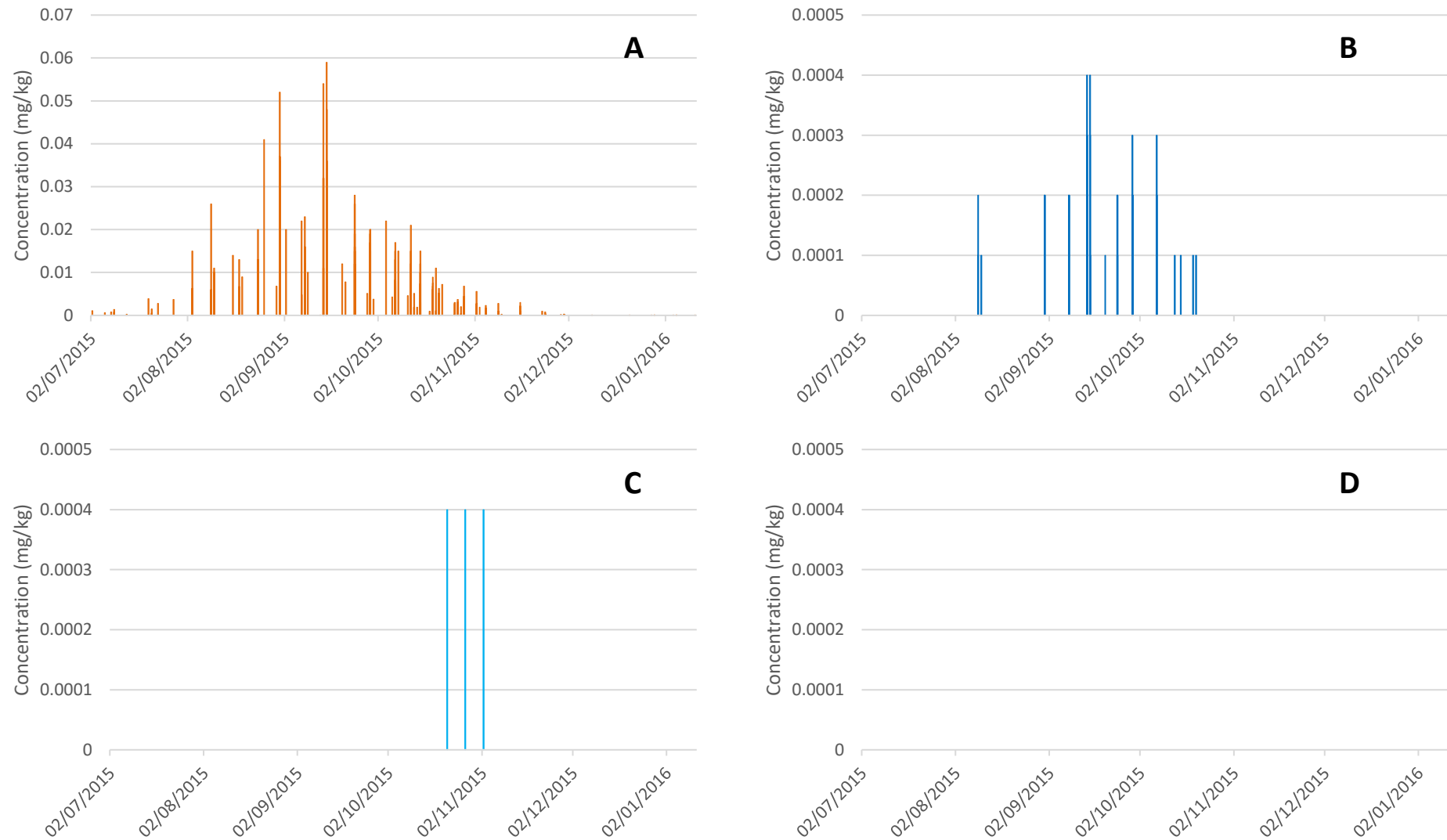


Figure F-1. Plot of pectenotoxins over time for bloom event C|201507-12. A) PTX2 B) PTX1 B) PTX11 B) PTX6

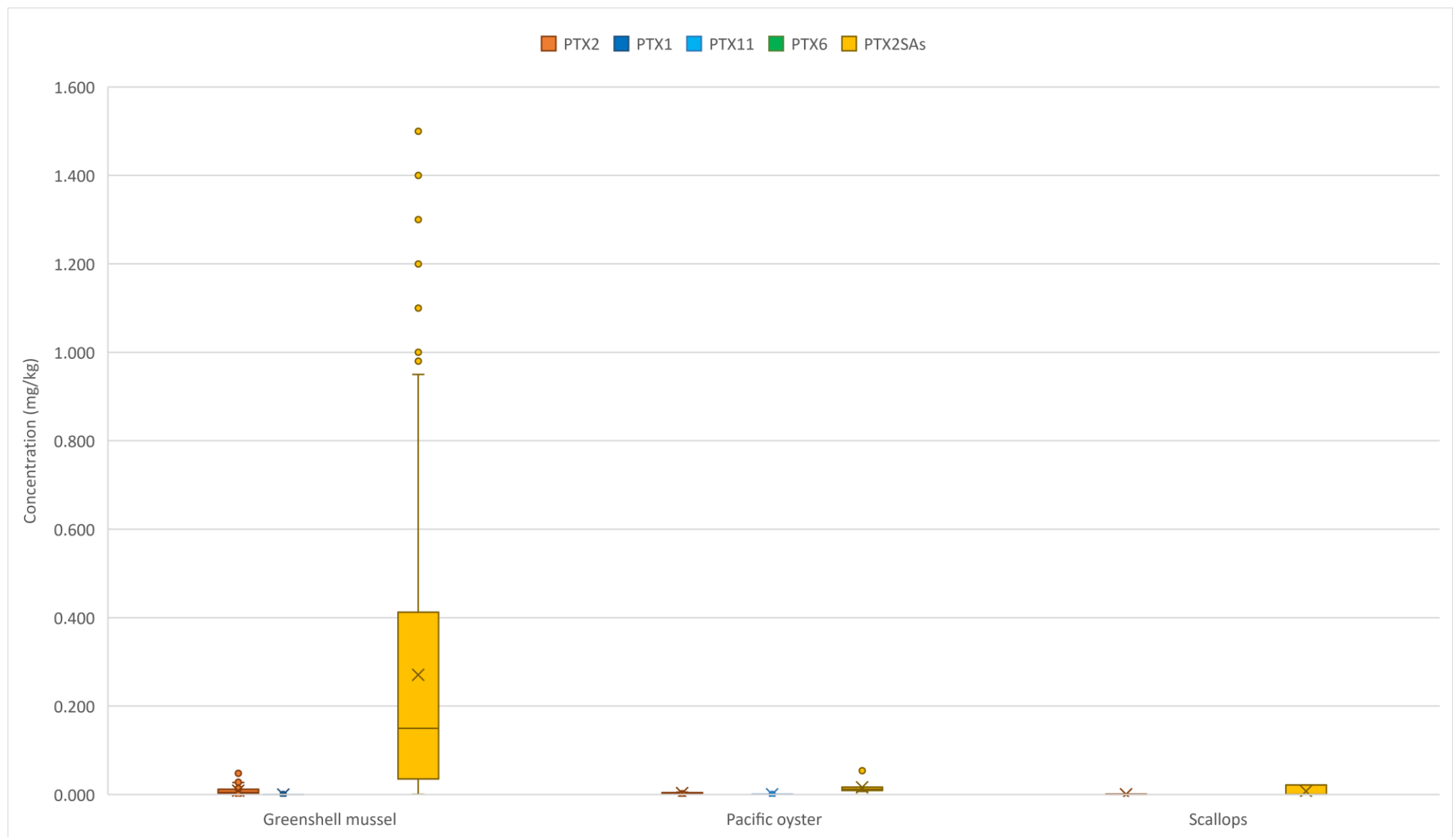


Figure F-2. Box and whisker plot of PTXs in each matrix for bloom event C|201507-12

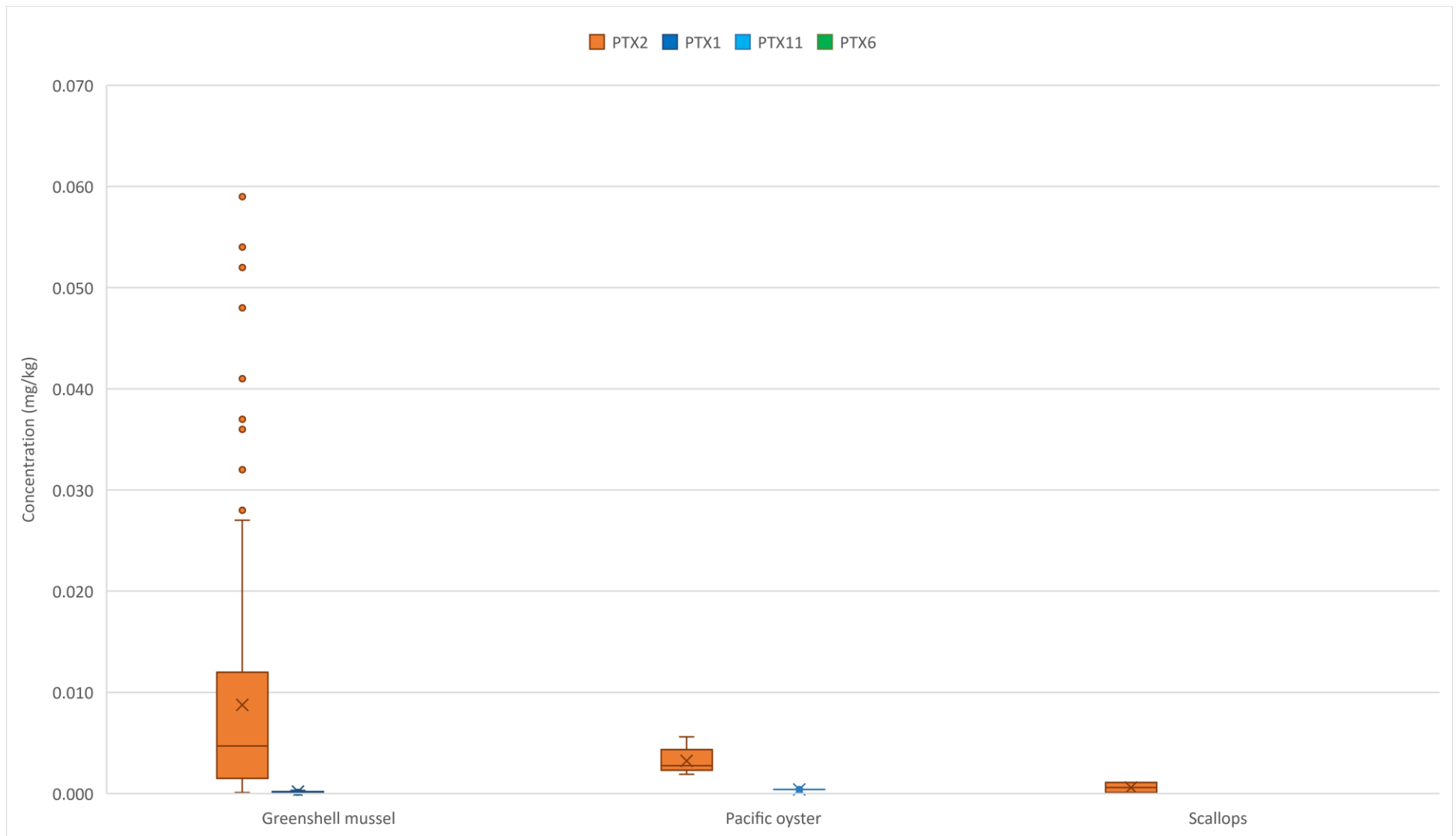


Figure F-3. Box and whisker plot of PTXs (excluding PTX2SAs) in each matrix for bloom event C|201507-12

F.2. Bloom Event I|pbk|201607-201703

Table F-3. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from sites within bloom I|pbk|201607-201703.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
I008A	24	22	92%	0.00310	0.01780	0.02200	1	4%	0.00010	0.00010	0.00010	0					0				
I007	15	15	100%	0.01157	0.05775	0.07700	2	13%	0.00030	0.00040	0.00040	0					0				
I035	14	14	100%	0.00674	0.05690	0.07900	0					0					0				
I036	8	8	100%	0.00714	0.03965	0.04600	0					0					0				
I032	6	5	83%	0.00030	0.00048	0.00050	0					0					0				
I002	3	3	100%	0.01270	0.02170	0.02200	1	33%	0.00020	0.00020	0.00020	0					0				
I021	2	2	100%	0.00425	0.00449	0.00450	0					0					0				
I038	1	1	100%	0.00180	0.00180	0.00180	0					0					0				
I026	1	1	100%	0.00060	0.00060	0.00060	0					0					0				
I031	1	1	100%	0.00020	0.00020	0.00020	0					0					0				
I004	1	1	100%	0.00500	0.00500	0.00500	0					0					0				
Total	76	73	96%	0.00615	0.05220	0.07900	4	5%	0.00023	0.00039	0.00040	0					0				

Table F-4. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from shellfish species within bloom I|pbk|201607-201703.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
Greenshell Mussel	41	40	98%	0.00798	0.07705	0.07900	2	5%	0.00020	0.00020	0.00020	0					0				
Clams	24	22	92%	0.00310	0.01780	0.02200	1	4%	0.00010	0.00010	0.00010	0					0				
Blueshell Mussel	11	11	100%	0.00560	0.02150	0.02200	1	9%	0.00040	0.00040	0.00040	0					0				
Total	76	73	96%	0.00615	0.05220	0.07900	4	5%	0.00023	0.00039	0.00040	0					0				

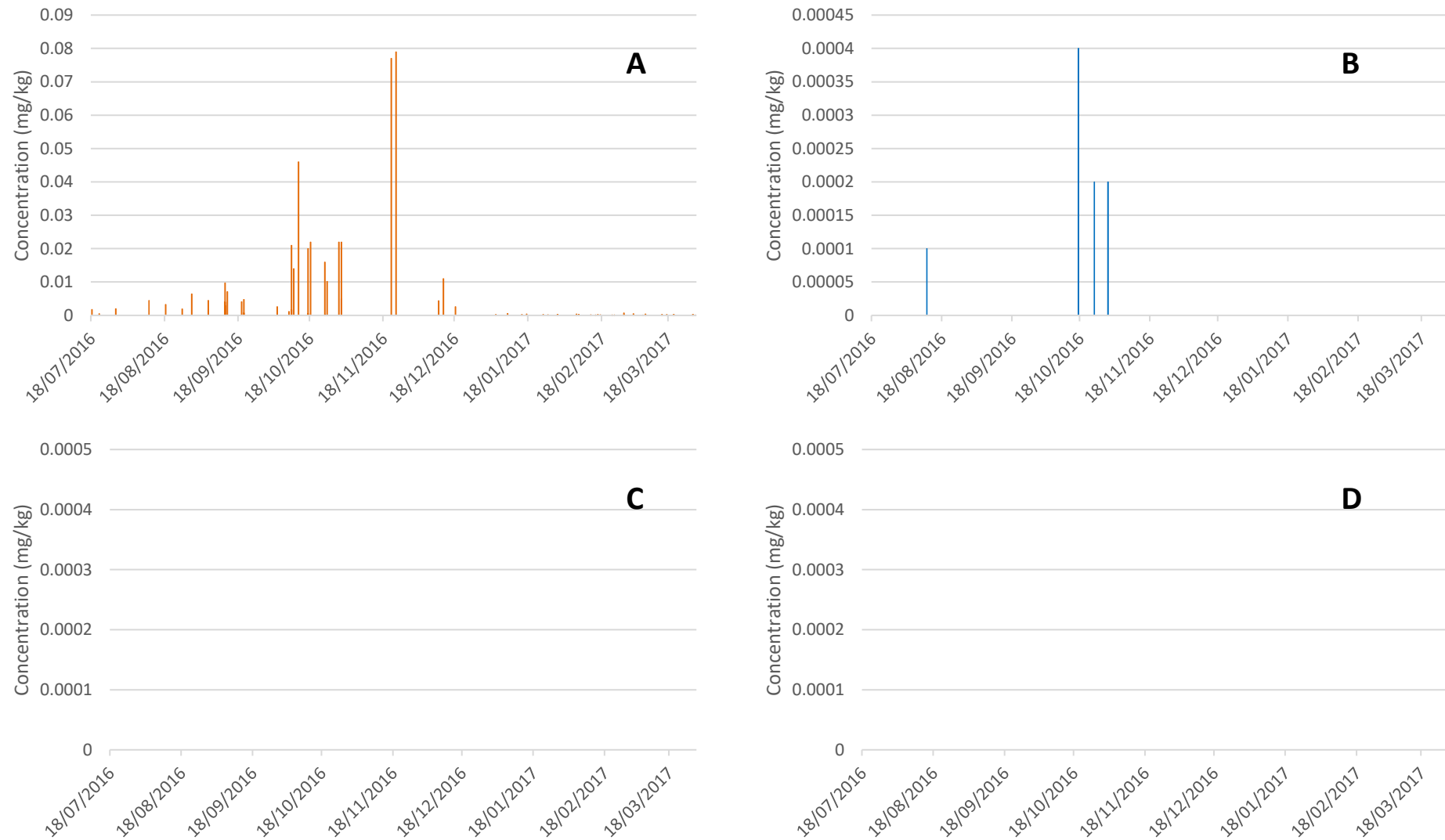


Figure F-4. Plot of pectenotoxins over time for bloom event I|pbk|201607-201703. A) PTX2 B) PTX1 B) PTX11 B) PTX6

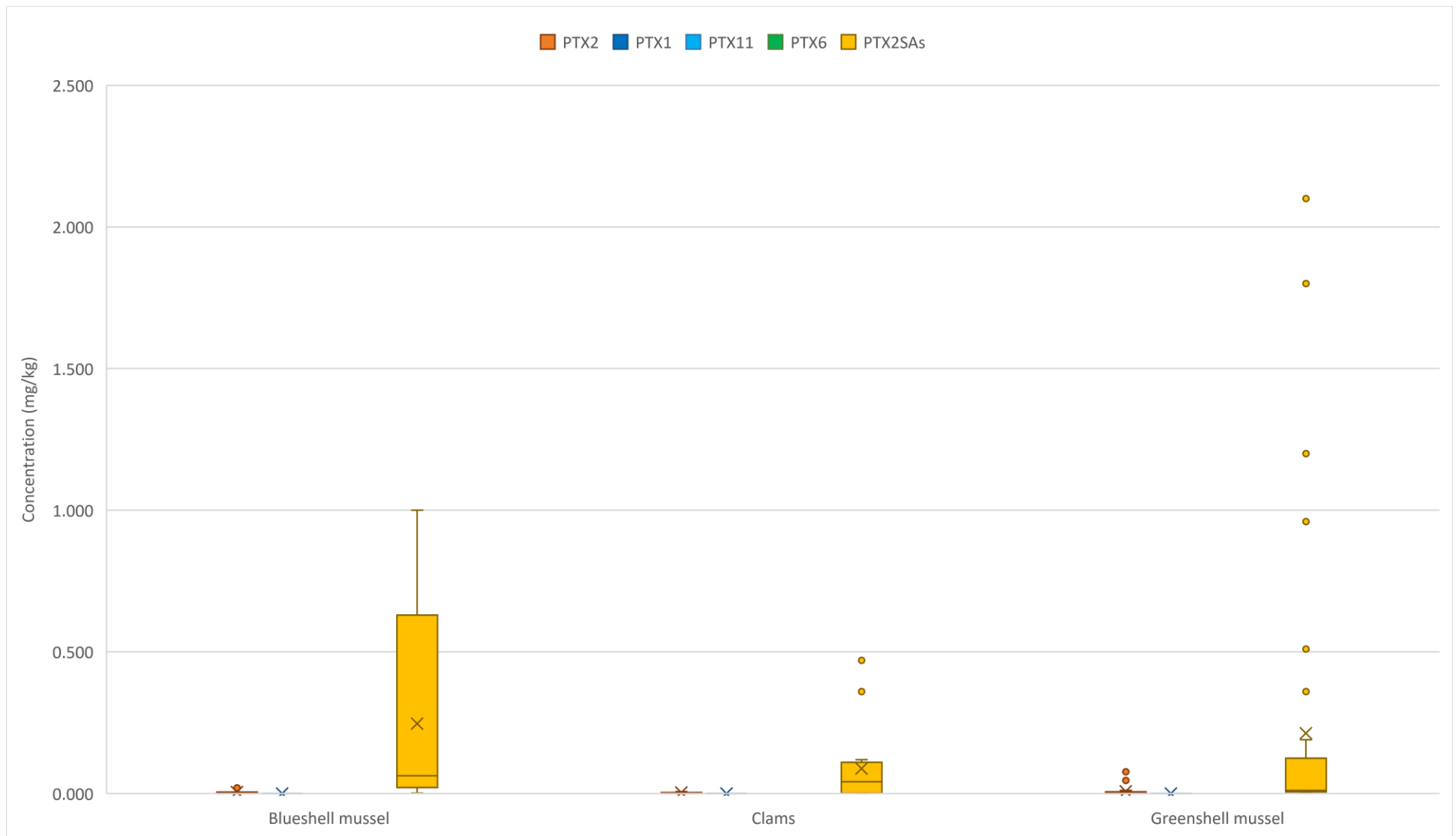


Figure F-5. Box and whisker plot of PTXs in each matrix for bloom event I|pbk|201607-201703

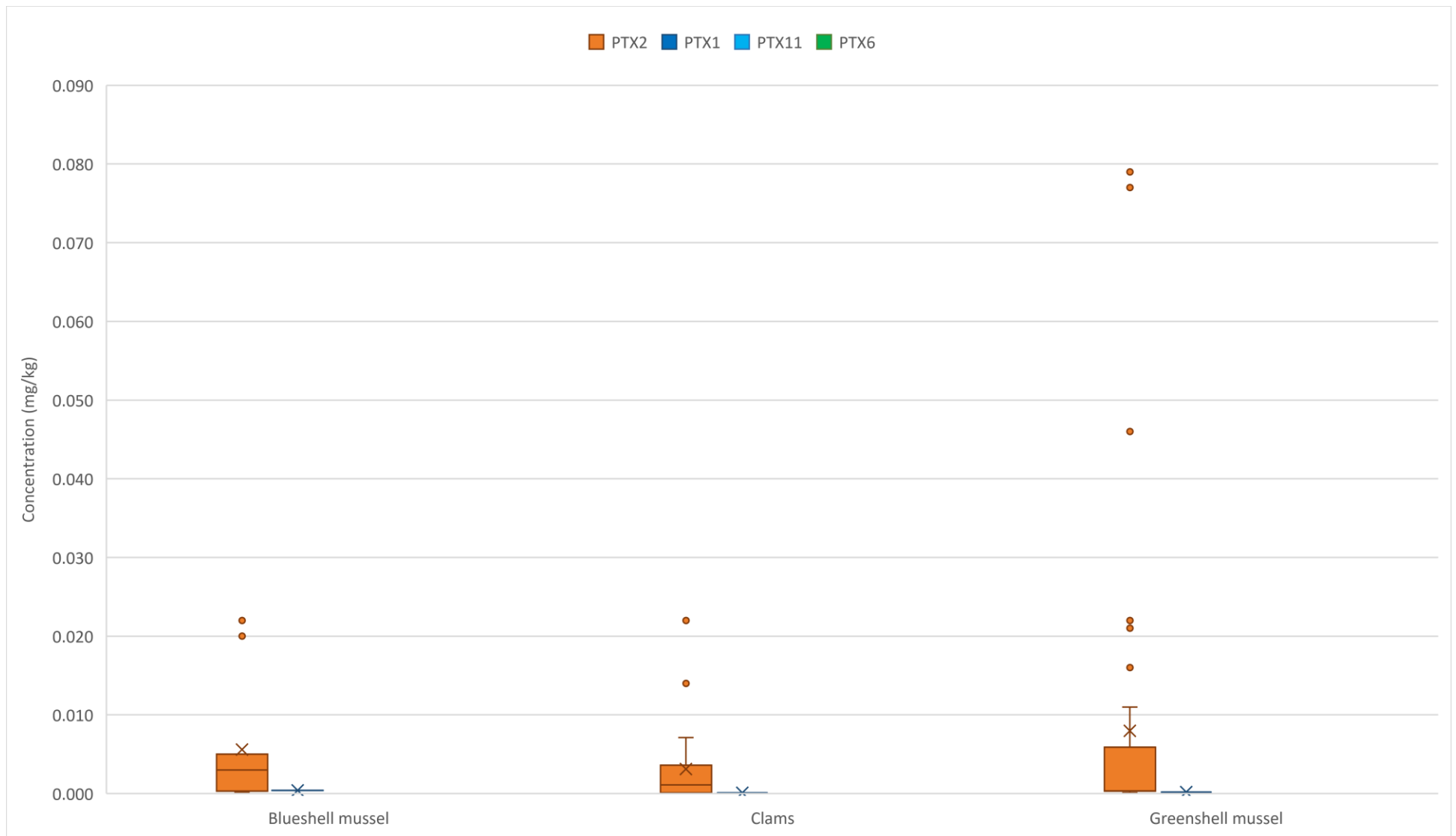


Figure F-6. Box and whisker plot of PTXs (excluding PTX2SAs) in each matrix for bloom event I|pbk|201607-201703

F.3. Bloom Event I|bpk|200904-201005

Table F-5. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from sites within bloom I|bpk|200904-201005.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
I008A	8	8	100%	0.00423	0.01314	0.01440	0					0					0				
I032	23	18	78%	0.00839	0.05973	0.07520	0					0					0				
I036	37	27	73%	0.01126	0.05235	0.06060	0					0					0				
I108	1	1	100%	0.00550	0.00550	0.00550	0					0					0				
Total	69	54	78%	0.00916	0.05647	0.07520	0					0					0				

Table F-6. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from shellfish species within bloom I|bpk|200904-201005.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
Clams	8	8	100%	0.00311	0.00708	0.00720	0					0					0				
Greenshell mussel	61	46	75%	0.01021	0.05901	0.07520	0					0					0				
Total	69	54	78%	0.00916	0.05647	0.07520	0					0					0				

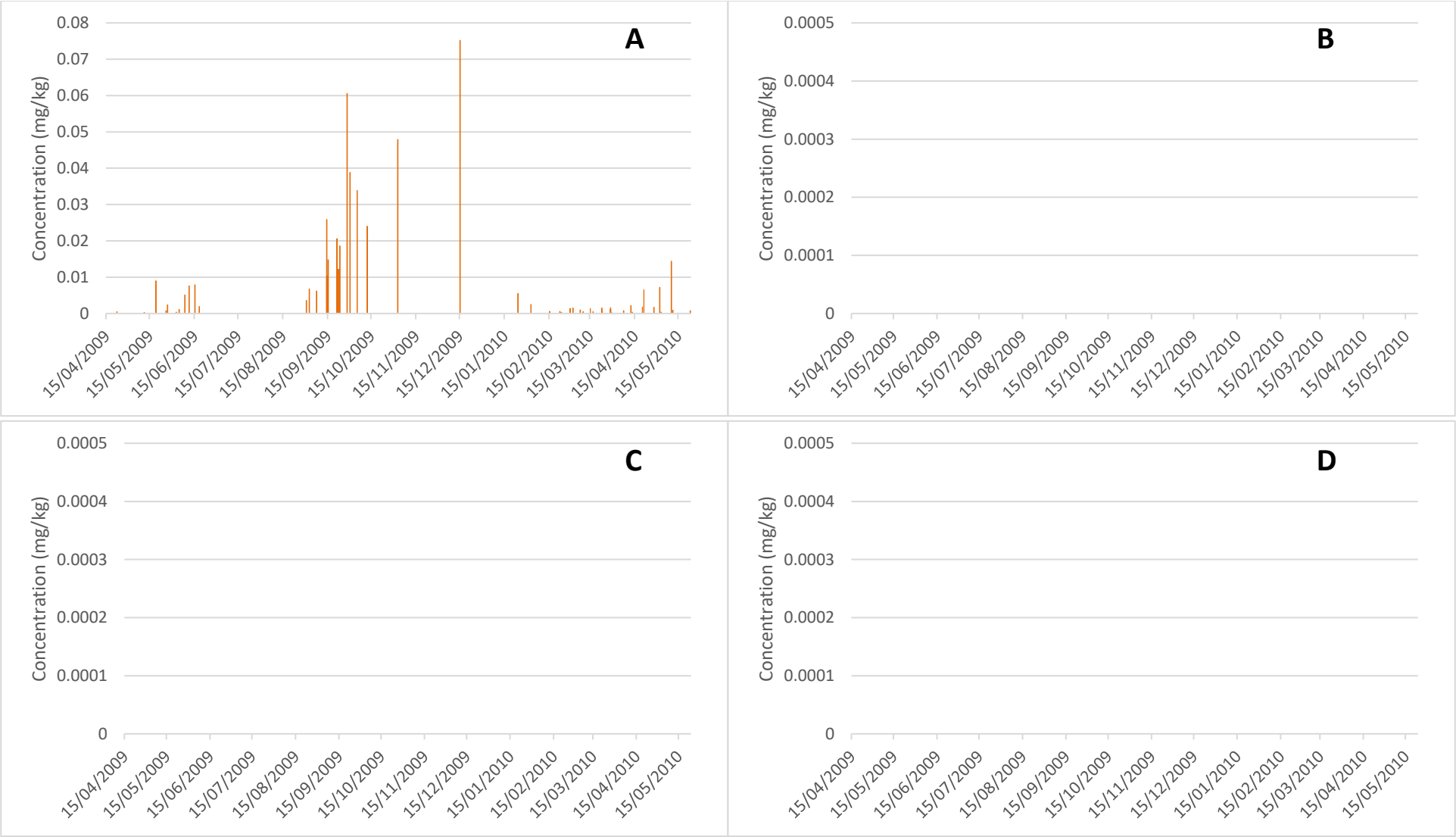


Figure F-7. Plot of pectenotoxins over time for bloom event l|b|pk|200904-201005. A) PTX2 B) PTX1 B) PTX11 B) PTX6

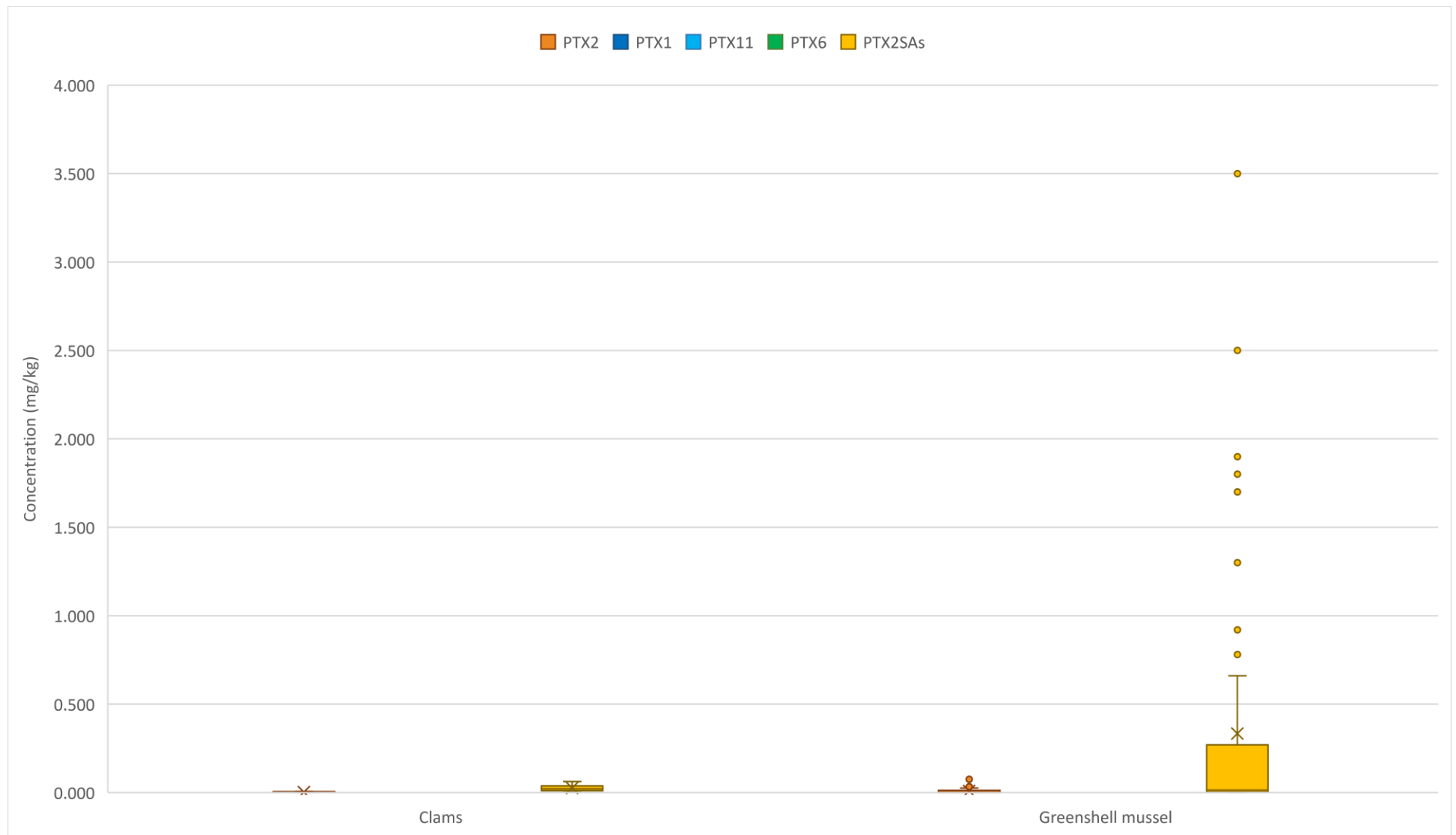


Figure F-8. Box and whisker plot of PTXs in each matrix for bloom event I|bpb|200904-201005

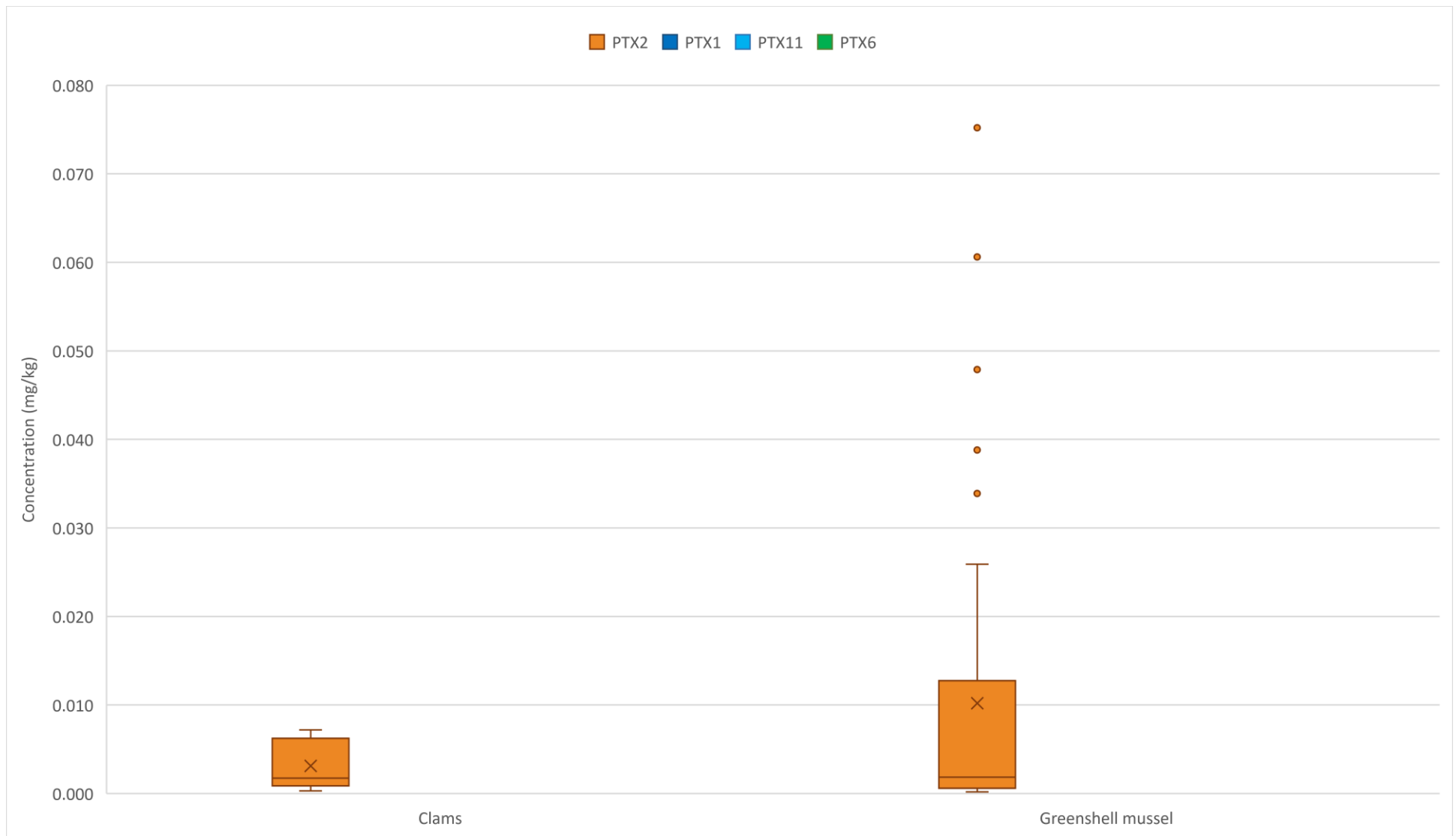


Figure F-9. Box and whisker plot of PTXs (excluding PTX2SAs) in each matrix for bloom event I|bpk|200904-201005

F.4. Bloom Event B|201509-12

Table F-7. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from sites within bloom B|201509-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
B007	4	4	100%	0.00363	0.00824	0.00860	0					0					0				
B015B	1	1	100%	0.01480	0.01480	0.01480	0					0					0				
B024B	11	11	100%	0.00875	0.02688	0.02940	0					0					0				
Total	16	16	100%	0.00784	0.02561	0.02940	0					0					0				

Table F-8. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from shellfish species within bloom B|201509-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
Pacific Oyster	5	5	100%	0.00586	0.01418	0.01480	0					0					0				
Scallop	11	11	100%	0.00875	0.02688	0.02940	0					0					0				
Total	16	16	100%	0.00784	0.02561	0.02940	0					0					0				

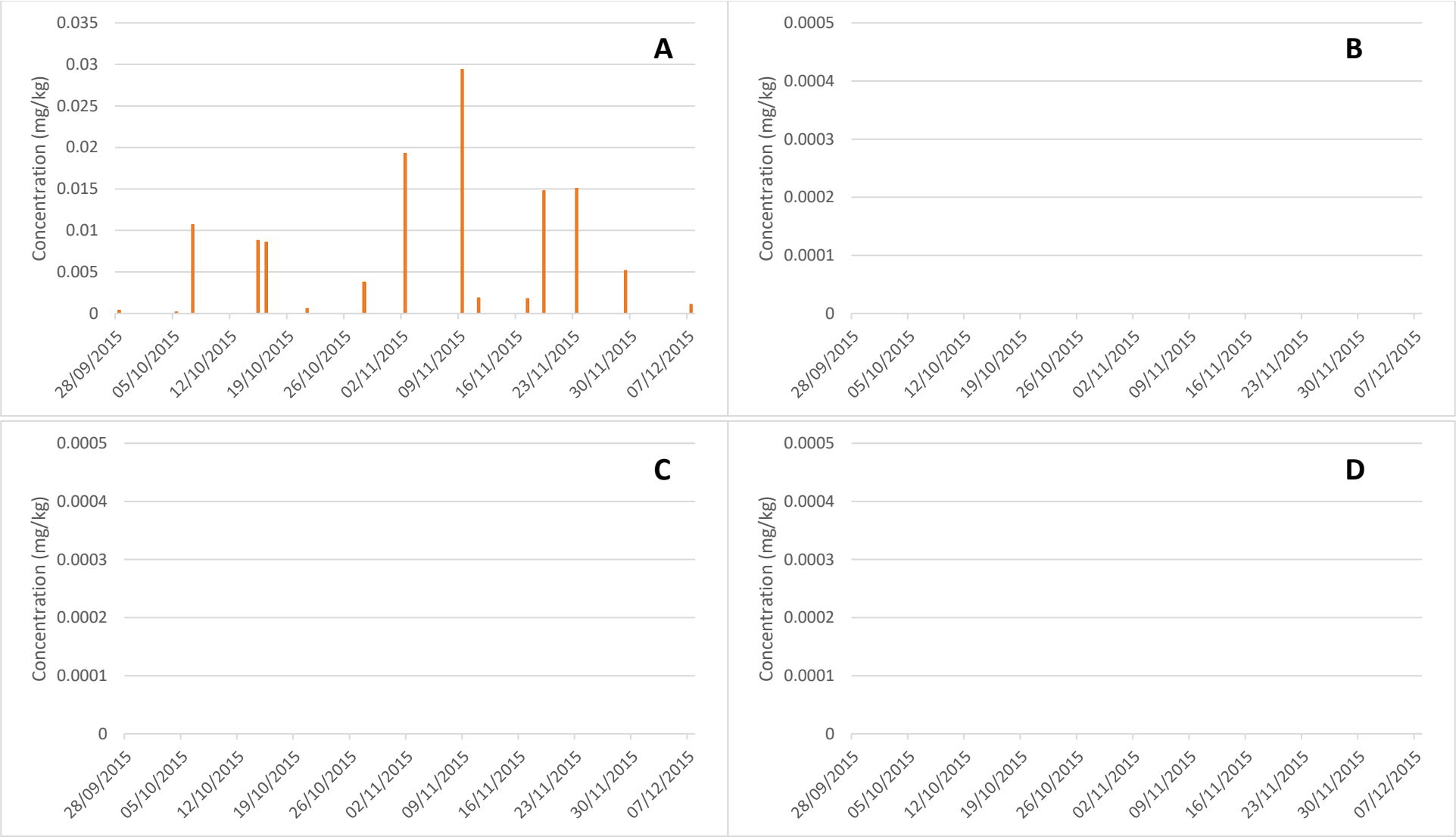


Figure F-10. Plot of pectenotoxins over time for bloom event B|201509-12. A) PTX2 B) PTX1 B) PTX11 B) PTX6

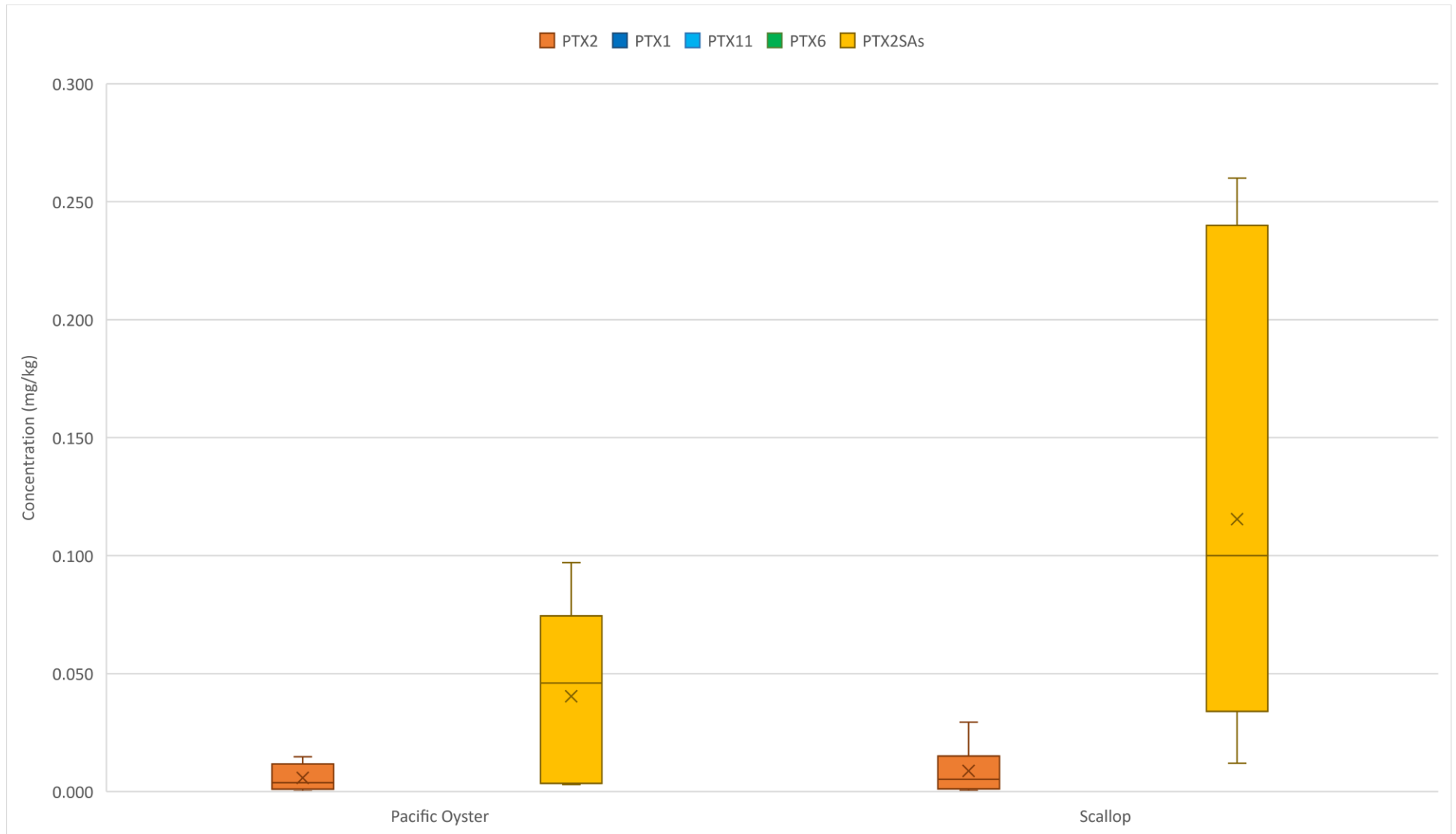


Figure F-11. Box and whisker plot of PTXs in each matrix for bloom event B|201509-12

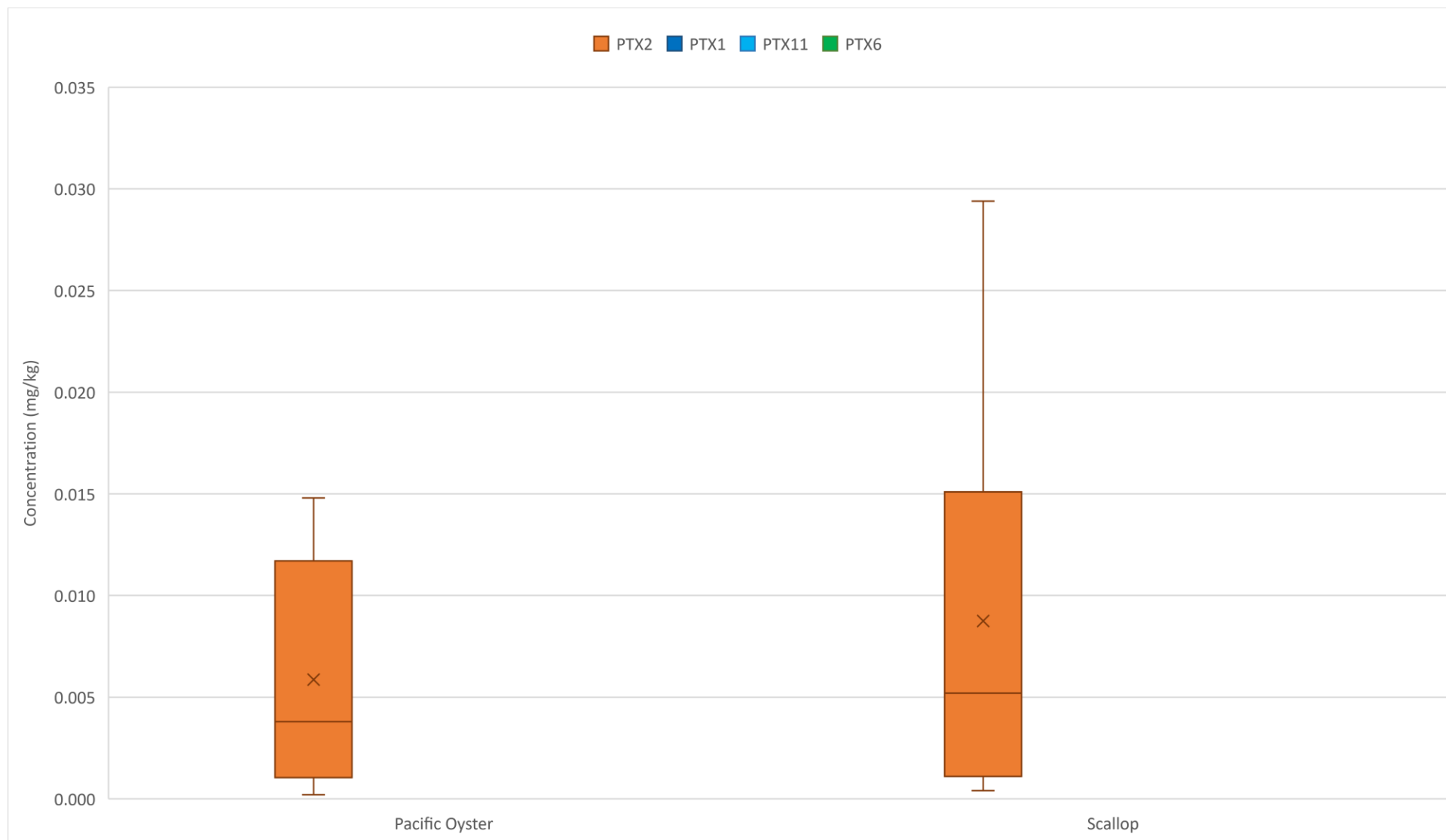


Figure F-12. Box and whisker plot of PTXs (excluding PTX2SAs) in each matrix for bloom event B|201509-12

F.5. Bloom Event A|boi|201506-12

Table F-9. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from sites within bloom A|boi|201506-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
A014	14	14	100%	0.00290	0.00890	0.01000	3	21%	0.00010	0.00010	0.00010	13	93%	0.00028	0.00077	0.00080	0				
A015	16	16	100%	0.00523	0.01849	0.02010	7	44%	0.00051	0.00181	0.00200	14	88%	0.00056	0.00100	0.00100	0				
A030	3	3	100%	0.02140	0.03295	0.03330	2	67%	0.00485	0.00727	0.00740	0					0				
Total	33	33	100%	0.00571	0.02762	0.03330	12	36%	0.00113	0.00600	0.00740	27	82%	0.00043	0.00100	0.00100	0				

Table F-10. Summary of PTX 1, 2, 6 and 11 (mg/kg) samples from shellfish species within bloom A|boi|201506-12.

Site	No Samples	PTX2					PTX1					PTX11					PTX6				
		Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max	Detections	%detected	Mean	97.5 PCTL	Max
Greenshell Mussel	3	3	100%	0.02140	0.03295	0.03330	2	67%	0.00485	0.00727	0.00740	0					0				
Pacific Oyster	30	30	100%	0.00414	0.01698	0.02010	10	33%	0.00039	0.00171	0.00200	27	90%	0.00043	0.00100	0.00100	0				
Total	33	33	100%	0.00571	0.02762	0.03330	12	36%	0.00113	0.00600	0.00740	27	82%	0.00043	0.00100	0.00100	0				

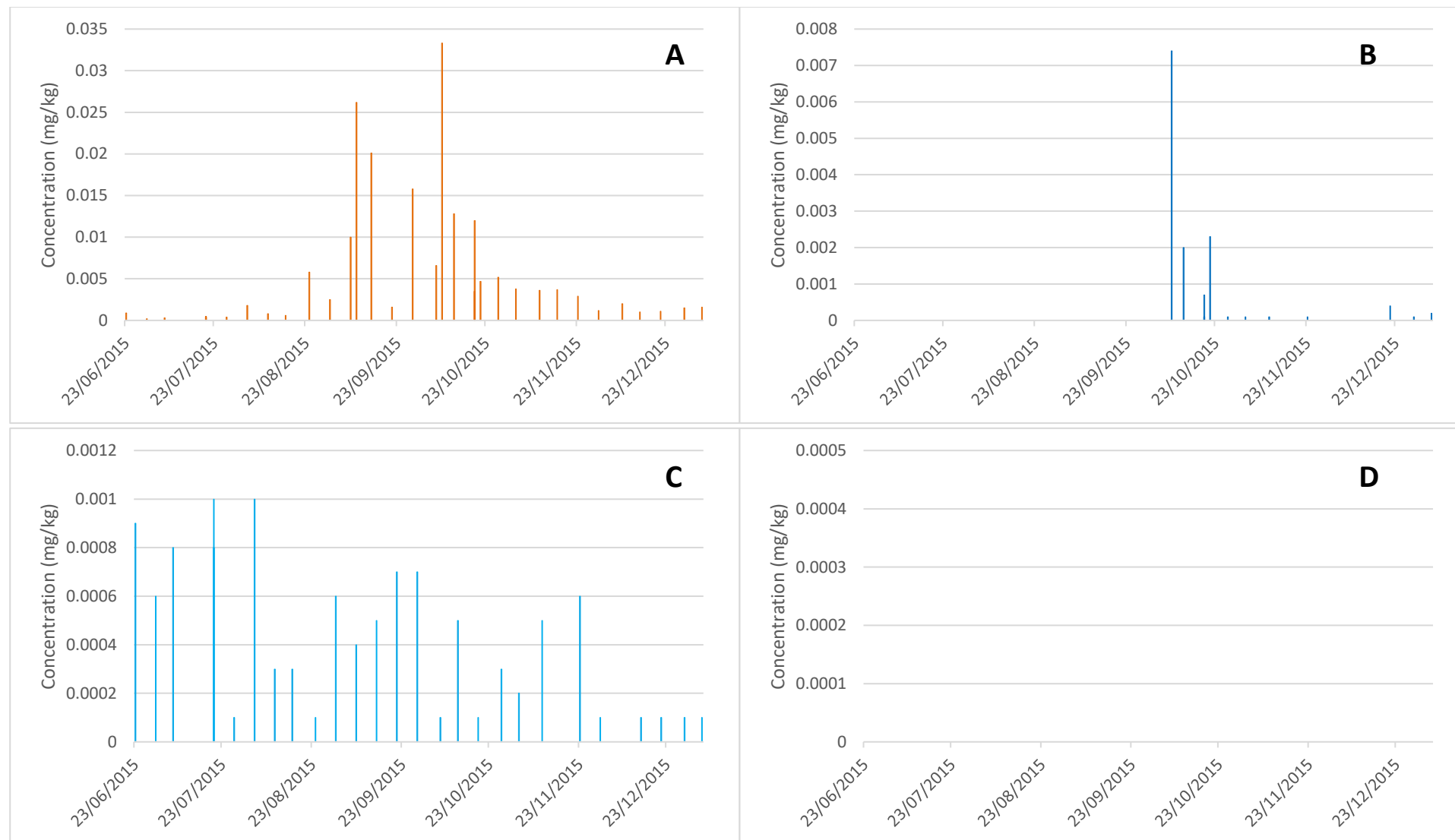


Figure F-13. Plot of pectenotoxins over time for bloom event A|boi|201506-12. A) PTX2 B) PTX1 B) PTX11 B) PTX6

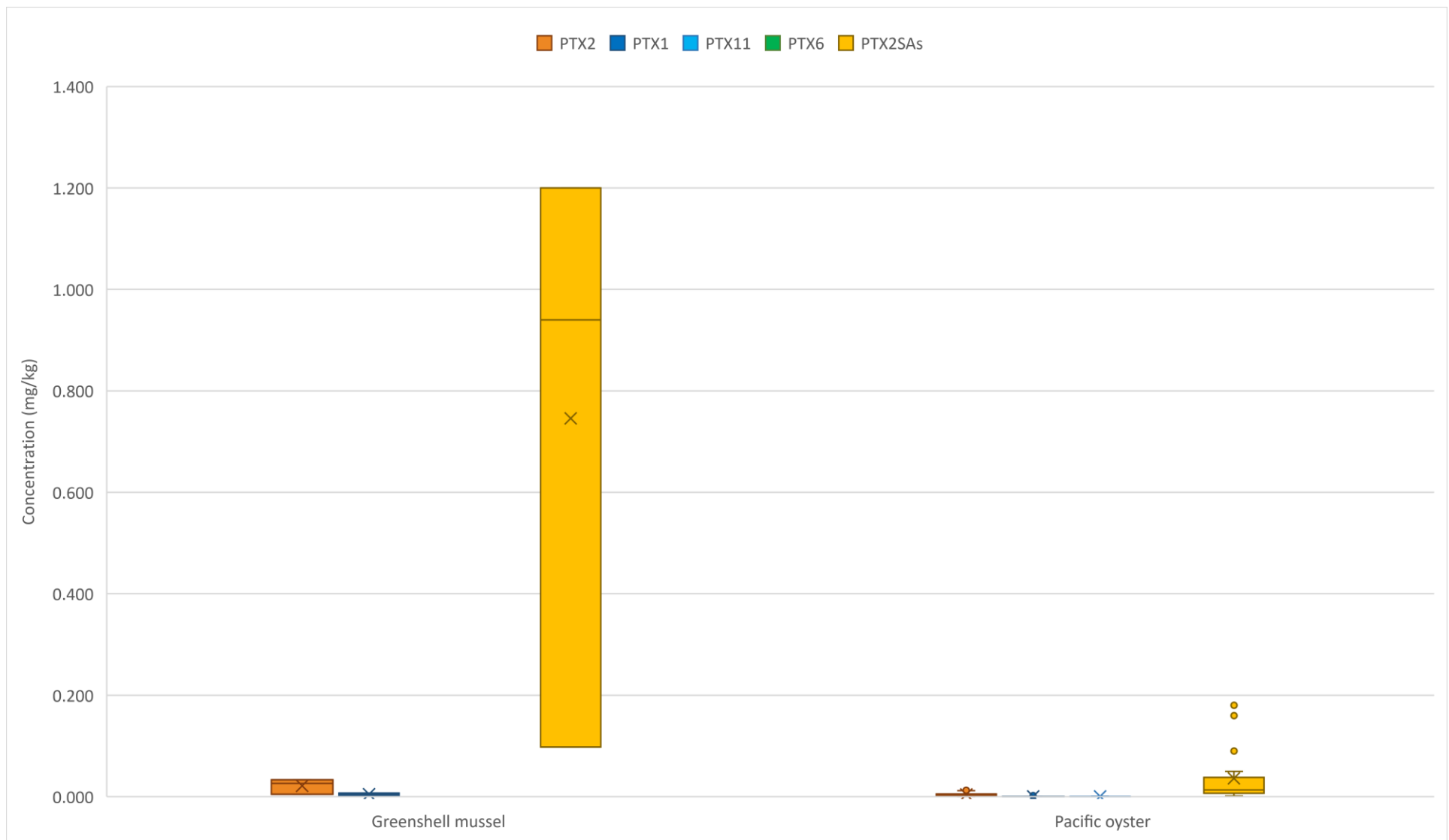


Figure F-14. Box and whisker plot of PTXs in each matrix for bloom event A|boi|201506-12

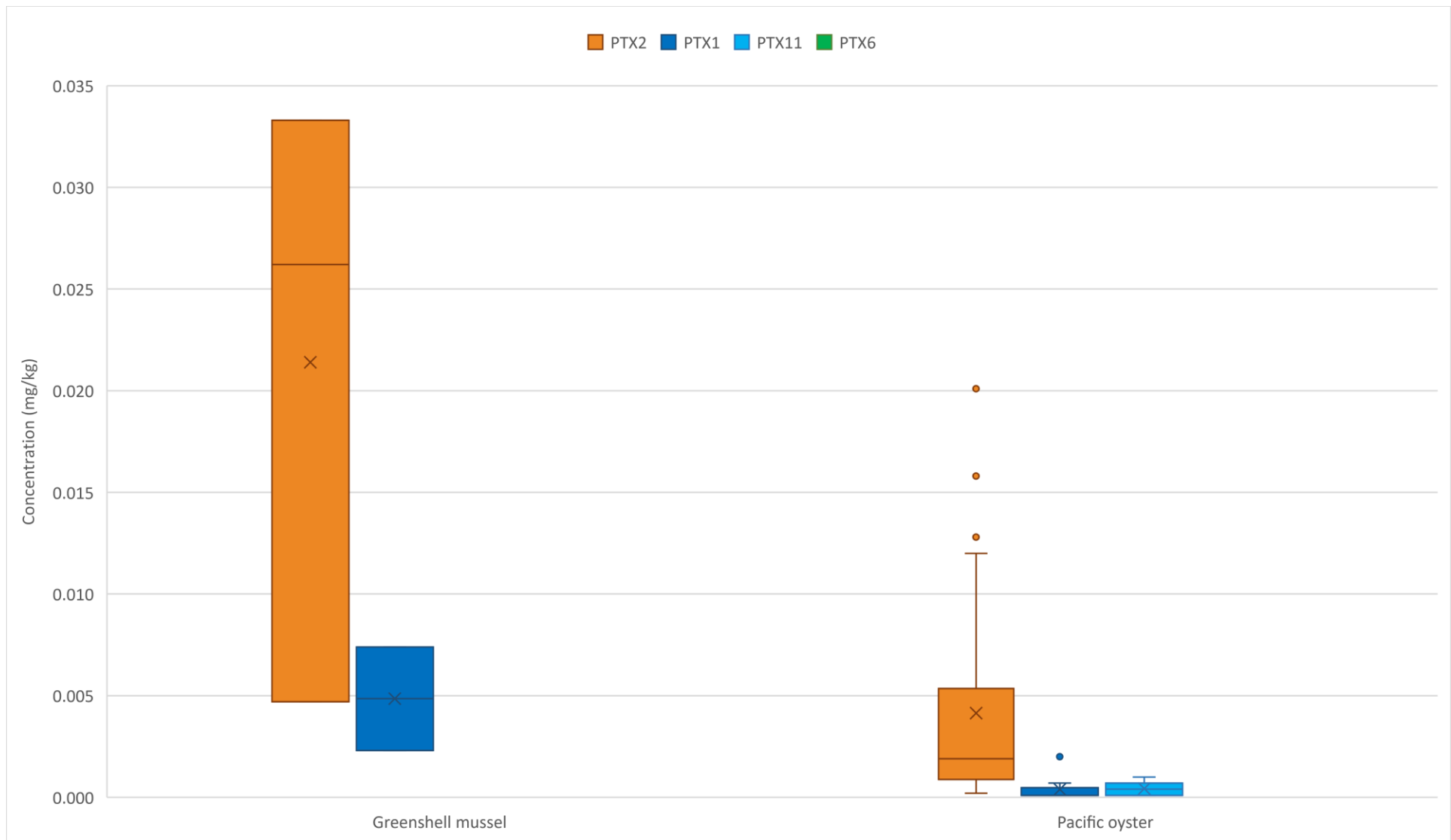


Figure F-15. Box and whisker plot of PTXs (excluding PTX2SAs) in each matrix for bloom event A|boi|201506-12

APPENDIX G. EXPOSURE ASSESSMENT AND RISK CHARACTERISATION

G.1. Pectenotoxins

G.1.1. Consumption amount of shellfish

The amount of bivalve shellfish meat consumption was assumed to follow a triangular distribution with a minimum of 0g, most likely value (mode) of 100g and a maximum value of 400g as follows:

```
## For consumption use a Triangular distribution with  
## Min=0, Mode=0.1kg, Max=0.4kg - capture both variability and uncertainty  
mc.cons <- mcstoc(rtriang, min=0, mode=0.1, max=0.4, type="V")
```

G.1.2. Distribution of PTX2 Concentration

There are two approaches that could be used for the simulation of exposure in relation to the distribution of PTX2 from which realisations are drawn. The first approach is to fit a suitable parametric distribution to the observed PTX2 values, and hence this approach can result in realisations of PTX2 that were not directly observed. The second is to draw realisations from the empirical distribution of PTX2 concentration, and hence only concentrations that were actually observed can be sampled.

To investigate the first approach, a Cullen and Frey graph was produced to compare the skewness and kurtosis of the detectable PTX2 results with various candidate distributions (Figure G-1). From this graph the sample distribution, and vast majority of bootstrapped samples, fall in the grey region, which represents a range of beta distributions (based on shape and scale parameters). Interestingly, EFSA used a beta distribution for the PTX2 detections to evaluate the probabilistic dietary exposure to PTX2 in the EU.

Cullen and Frey graph

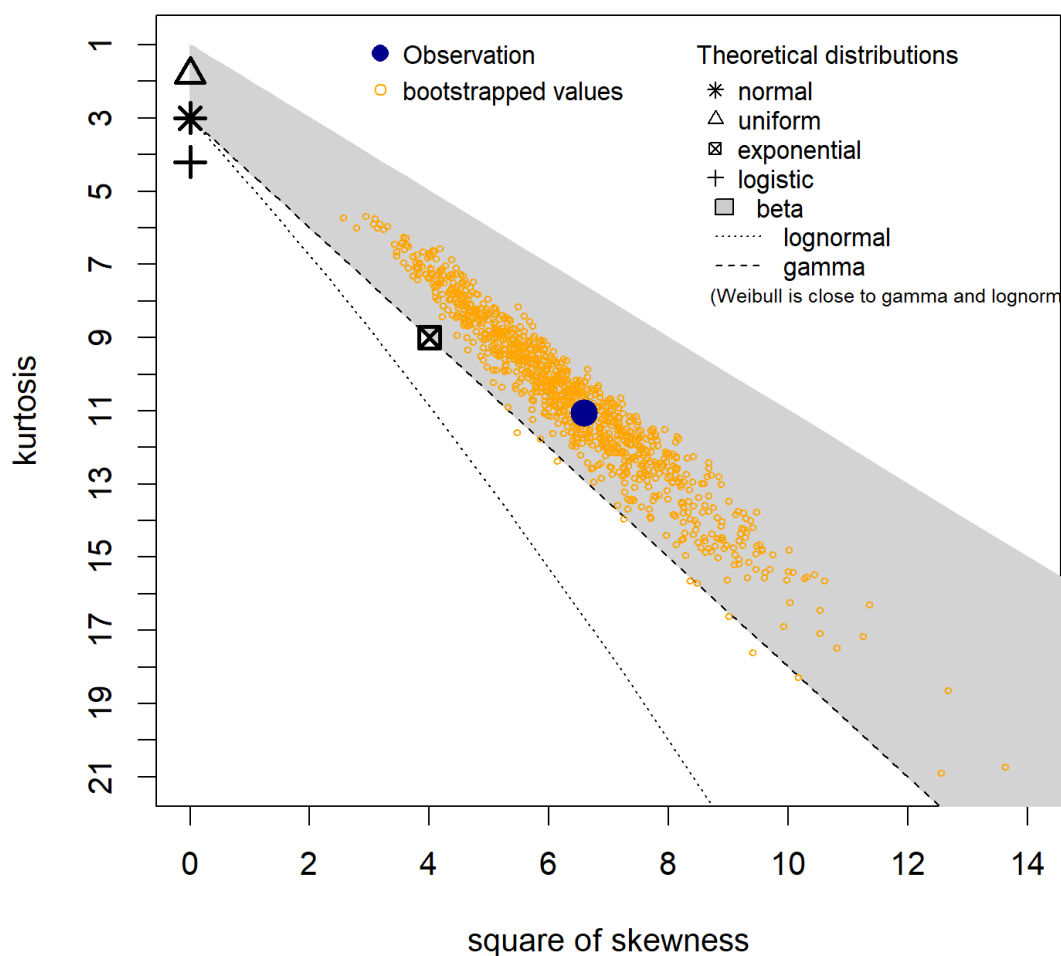


Figure G-1. Cullen and Frey graph showing the data various potential theoretical distributions for PTX2

In addition to fitting a beta distribution to the PTX2 detections, the log-normal and Weibull distributions were also fitted. The summary output, comparing the distributions in terms of goodness-of-fit, shown below indicates that the log-normal distribution fits the data better than the beta or Weibull distribution (as indicated by smaller GoF statistics and criteria). The cumulative distribution functions (CDFs) of the candidate distributions are compared against the empirical distribution function (*i.e.* data) in Figure G-2, which also indicates that the log-normal distribution fits slightly better than the beta distribution.

Goodness-of-fit statistics

	Beta	Lognormal	Weibull
Kolmogorov-Smirnov statistic	0.1764861	0.1468933	0.2213077
Cramer-von Mises statistic	2.0386956	1.2553224	2.7137689
Anderson-Darling statistic	11.8291487	7.7508921	15.7943388

Goodness-of-fit criteria

	Beta	Lognormal	Weibull
Akaike's Information Criterion	-1692.859	-1747.744	-1632.918
Bayesian Information Criterion	-1685.808	-1740.693	-1625.867

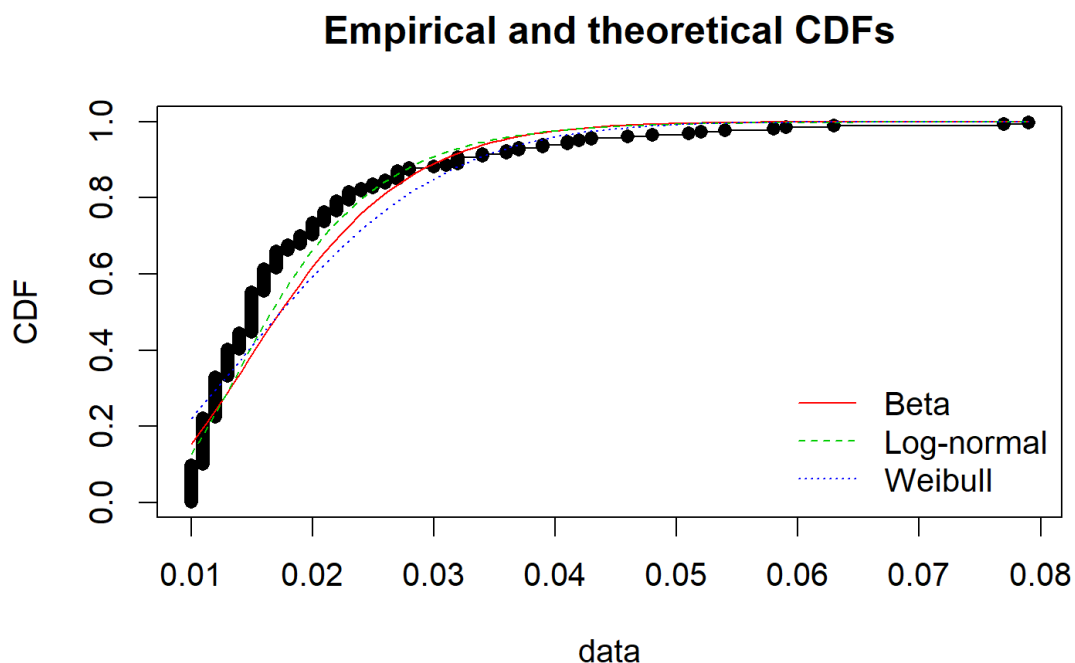


Figure G-2. Comparison of empirical CDF for PTX2 and potential theoretical distributions

The estimated maximum likelihood parameters are shown below and yield the summary plots in Figure G-3. From these it can be seen that the histogram has a very high mode at the very low detectable concentrations, higher than the best fitting distribution. This also affects the rest of the distribution, especially values in the range of about 0.013 to about 0.023 mg/kg, where the parametric distribution 'over-predicts' the probabilities while for larger values it 'under-predicts' the probabilities with which PTX2 concentrations have been observed.

Fitting of the distribution 'lnorm' by maximum likelihood

Parameters :

estimate Std. Error

meanlog -4.0979566 0.02806483

sdlog 0.4446305 0.01984438

Loglikelihood: 875.8719 AIC: -1747.744 BIC: -1740.693

Correlation matrix:

meanlog sdlog

meanlog 1 0

sdlog 0 1

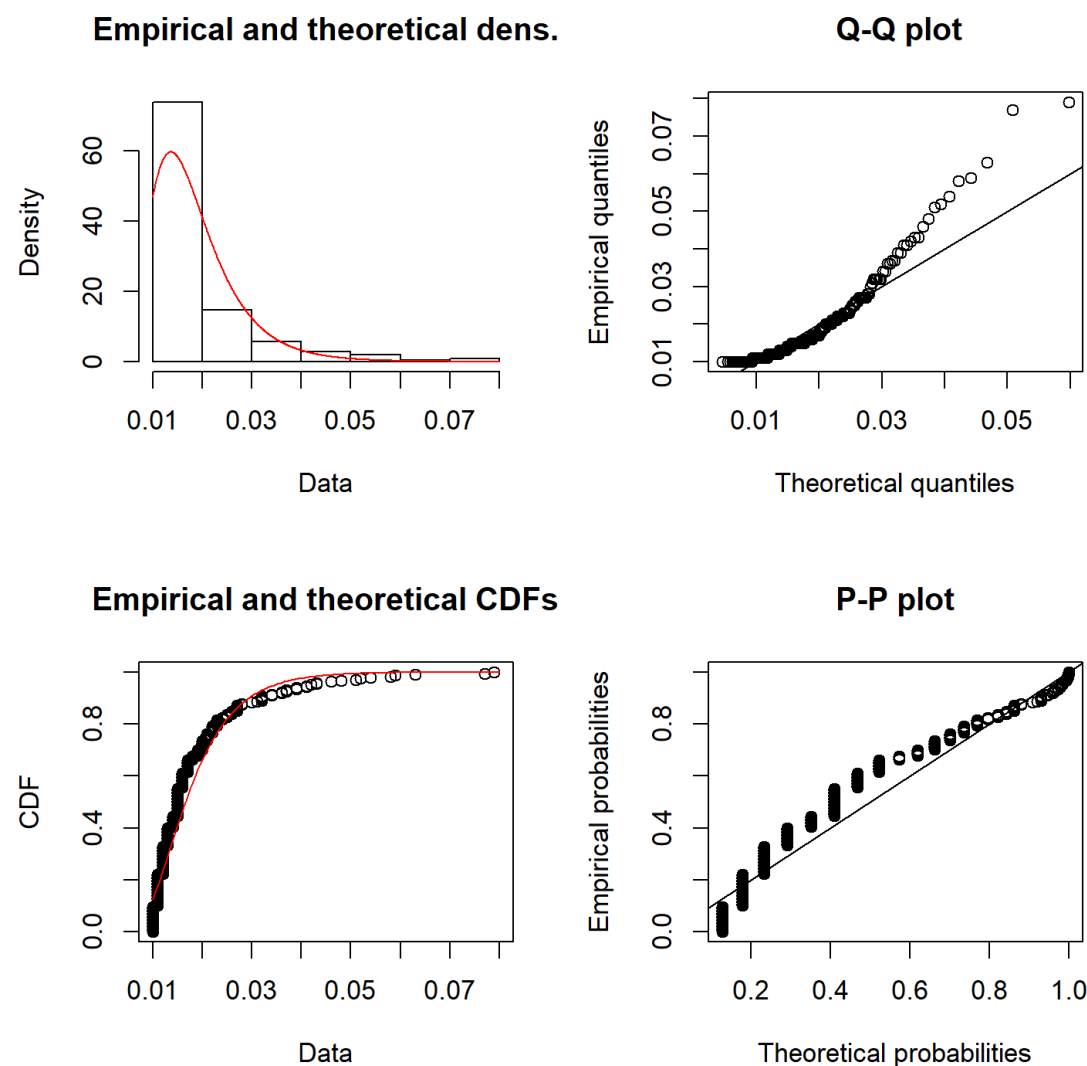


Figure G-3. Summary plots showing the fit of the log-normal distribution to the PTX2 detections

EFSA included a binomial distribution to model whether a particular serving contained PTX2, or not. This approach was further complicated by the assumption that not all non-detects were true zero PTX2 concentrations, but that 20% were assumed to contain PTX2, but below the lower limit of quantification (LoQ). It should be noted that how non-detects are dealt with in the probabilistic assessment is of limited value—that is, if the PTX2 concentration is less than or equal to the LoD/Q of 0.01 mg/kg, then

this implies that a 60kg consumer would have to consume 4.8kg, or more, of bivalve shellfish. This value clearly far exceeds the consumption amount distribution. For this reason, the simplifying assumption is made that the distribution of PTX2 concentrations is made up of two components:

- Detects and non-detects are randomly sampled from a binomial distribution with probabilities of a detection/non-detection equal to those in the bloom data set, *i.e.* 6.55% and 93.45%, respectively.
- Non-detects are assigned a PTX2 concentration of 0.01 mg/kg.
- Detects are a realisation from a parametric distribution with parameters estimated above, and left truncated at 0.01 mg/kg to ensure quantified detections below the LoQ.

In addition, a second scenario for the distribution of PTX2 concentrations is evaluated, using the empirical distribution of PTX2 concentrations, again assuming that non-detects are equal to the LoQ of 0.01 mg/kg.

```
## First approach:
## For the PTX2 concentrations we need two components:
## 1) A probability tree (i.e. binomial) where non-detects are
## assigned a PTX2 concentration of LoQ = 0.01
## 2) Detects, are sampled from a beta distribution (estimated
## previously), these are left truncated to the LoQ = 0.01.
mc.ptx2.detp <- c(sum(is.na(NZ.bloom$ptx2)),
sum(!is.na(NZ.bloom$ptx2)))/nrow(NZ.bloom)
mc.ptx2.conc1 <-
  mcprobtree(mc.ptx2.detp,
    list("1"=mcdata(0.01, type="0"),
          "2"=mcstoc(rlnorm, meanlog=coef(fit.ptx2.ln)[1],
                    sdlog=coef(fit.ptx2.ln)[2], type="V",
                    rtrunc=TRUE, linf=0.01) ), type="V" )
## Second approach: We simply sample from the actual data, again
## assuming that values below the LoQ are set to 0.01.
mc.ptx2.conc2 <- mcstoc(rempiricalD, NZ.bloom$ptx2.r, type="V")
```

The following output provides summary statistics for the two concentration scenarios.

```
summary(mc(mc.ptx2.conc1, mc.ptx2.conc2),
  probs=rpt.p)

mc.ptx2.conc1 :
      mean      sd  Min  50%   95%  97.5%   99%  Max    nsv Na's
NoUnc 0.0106 0.00319 0.01 0.01 0.0138 0.0201 0.0272 0.127 1000000 0

mc.ptx2.conc2 :
      mean      sd  Min  50%   95%  97.5%   99%  Max    nsv Na's
NoUnc 0.0106 0.00358 0.01 0.01 0.012 0.017 0.027 0.079 1000000 0
```

G.1.3. Estimating Exposure

The final step in this model is to combine the consumption and concentrations data to estimate the amount of PTX2 consumed in a single sitting, adjusting for adult weight—

three scenarios for adult weight were assessed, namely a 'standard' 60kg adult, and average male and female NZ adult weights, which were estimated as 86.7kg and 73.3kg (Pearson et al. 2018), respectively. Finally, the results are also converted to µg/kg bw to allow comparison with the Acute Reference Dose (ARfD).

```
## Define the body weights for standardisation
```

```
mc.bw <- mcddata(60, type="0")
mc.bw.m <- mcddata(86.7, type="0")
mc.bw.f <- mcddata(73.3, type="0")
```

The exposure estimates are calculated for each of the three body weight and two concentration distribution scenarios.

```
## Calculate exposure for different body weight scenarios
```

```
mc.ptx2.exp1 <- 1000*mc.cons*mc.ptx2.conc1/mc.bw
mc.ptx2.exp1.m <- 1000*mc.cons*mc.ptx2.conc1/mc.bw.m
mc.ptx2.exp1.f <- 1000*mc.cons*mc.ptx2.conc1/mc.bw.f
mc.ptx2.exp2 <- 1000*mc.cons*mc.ptx2.conc2/mc.bw
mc.ptx2.exp2.m <- 1000*mc.cons*mc.ptx2.conc2/mc.bw.m
mc.ptx2.exp2.f <- 1000*mc.cons*mc.ptx2.conc2/mc.bw.f
```

The output below summarises the exposure to PTX2 for each of the six scenarios. From these it can be seen that:

- the two scenarios for the concentration of PTX2 result in similar exposures hence which one approach is used has little effect on the results.
- using a standard body weight of 60kg results in greater dietary exposure to PTX2 compared with using the larger average NZ adult body weights for males and females.

```
summary(mc(mc.ptx2.exp1, mc.ptx2.exp1.m, mc.ptx2.exp1.f,
           mc.ptx2.exp2, mc.ptx2.exp2.m, mc.ptx2.exp2.f),
        probs=rpt.p)
```

```
mc.ptx2.exp1 :
  mean      sd      Min    50%    95%  97.5%    99%    Max      nsv Na's
NoUnc 0.0296 0.0181 0.000025 0.0266 0.0572 0.063 0.0892 0.533 1000000 0

mc.ptx2.exp1.m :
  mean      sd      Min    50%    95%  97.5%    99%    Max      nsv Na's
NoUnc 0.0205 0.0125 0.0000173 0.0184 0.0396 0.0436 0.0618 0.369 1000000 0

mc.ptx2.exp1.f :
  mean      sd      Min    50%    95%  97.5%    99%    Max      nsv Na's
NoUnc 0.0242 0.0148 0.0000205 0.0218 0.0468 0.0516 0.073 0.436 1000000 0

mc.ptx2.exp2 :
  mean      sd      Min    50%    95%  97.5%    99%    Max      nsv Na's
NoUnc 0.0294 0.0187 0.000025 0.0265 0.0564 0.0616 0.0814 0.505 1000000 0

mc.ptx2.exp2.m :
  mean      sd      Min    50%    95%  97.5%    99%    Max      nsv Na's
NoUnc 0.0203 0.0129 0.0000173 0.0183 0.0391 0.0426 0.0563 0.35 1000000 0
```

```
mc.ptx2.exp2.f :  
      mean      sd      Min      50%      95%  97.5%      99%      Max      nsv Na's  
NoUnc 0.024 0.0153 0.0000205 0.0217 0.0462 0.0504 0.0666 0.414 1000000 0
```

G.1.4. Risk Characterisation

The risk characterization in the current context is a simple matter of comparing the exposure distributions to the corresponding Health Based Guidance Value, which for PTX2 is the ARfD of 0.8 µg/kg bw.

From the dietary exposure to PTX2 from bivalve shellfish calculated above, it can be seen that none of the 1,000,000 iterations resulted in an exposure exceeding the ARfD (based on the maximum).

G.2. Okadaic acid (OA) group toxins

G.2.1. Consumption amount of shellfish

The amount of bivalve shellfish meat was assumed to follow a triangular distribution with a minimum of 0g, most likely value (mode) of 100g and a maximum value of 400g as follows:

```
## For consumption use a Triangular distribution with  
## Min=0, Mode=0.1kg, Max=0.4kg - capture both variability and uncertainty  
mc.cons <- mcstoc(rtriang, min=0, mode=0.1, max=0.4, type="V")
```

G.2.2. Distribution of DSP Concentration

For DSP concentration, a similar approach as for PTX2 is used. First, the distribution of DSP detections is visualised using the Cullen and Frey graph (Figure G-4). From this graph it can be seen that the sample distribution, and vast majority of bootstrapped samples, fall in the grey region, which represents a range of beta distributions (based on shape and scale parameters). However, the beta distribution is defined only on the interval of 0 to 1, and hence cannot be used in this circumstance, and the gamma distribution is fitted instead. Interestingly, EFSA used a log-normal distribution for the DSP detections to evaluate the probabilistic dietary exposure to DSP in the EU.

Cullen and Frey graph

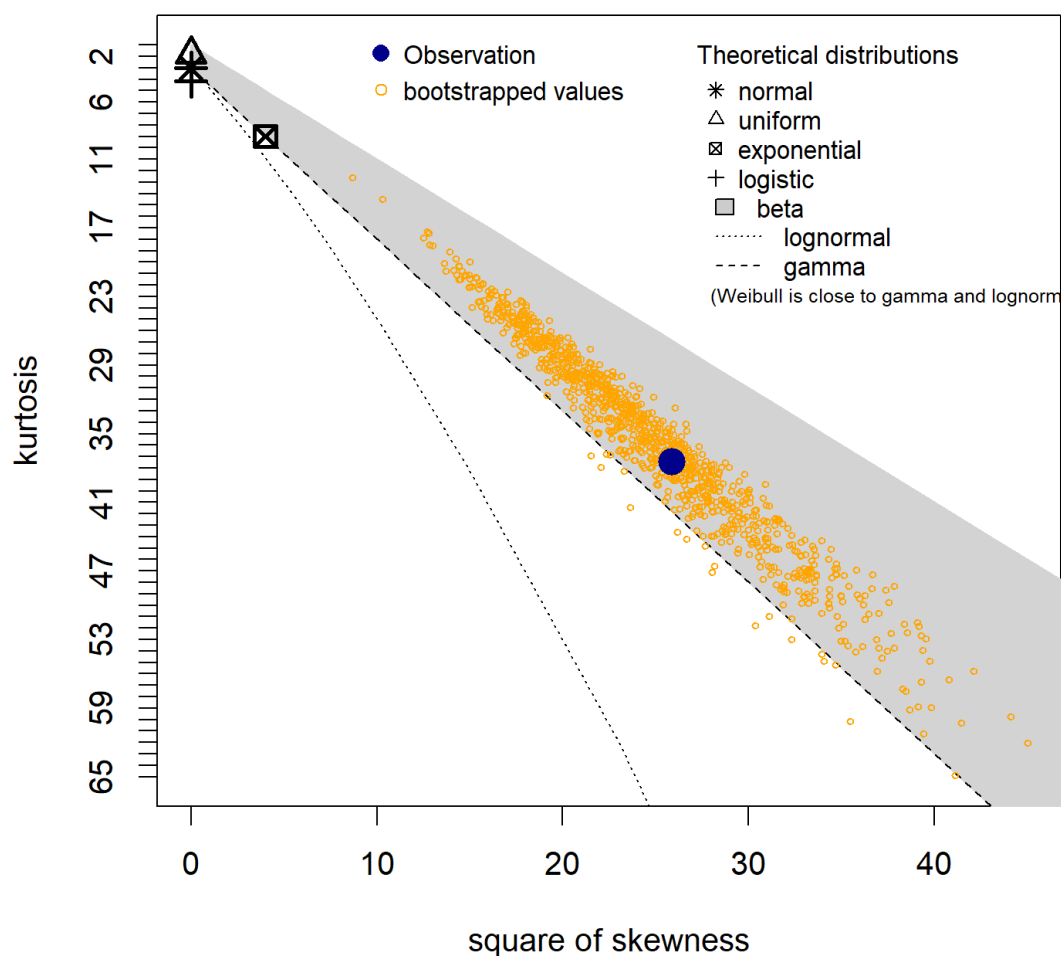


Figure G-4. Cullen and Frey graph showing the data various potential theoretical distributions for DSP

The GoF statistics and criteria for three candidate distributions—the gamma, log-normal and Weibull—are shown below. Similar to PTX2, the log-normal distribution fits best and this is also shown in the CDF comparison plot (Figure G-5).

Goodness-of-fit statistics

	Gamma	Lognormal	Weibull
Kolmogorov-Smirnov statistic	0.1467938	0.1000071	0.1782962
Cramer-von Mises statistic	6.3764028	1.8616815	4.5822944
Anderson-Darling statistic	36.8765137	12.8231252	30.4078344

Goodness-of-fit criteria

	Gamma	Lognormal	Weibull
Akaike's Information Criterion	-2555.329	-2877.800	-2592.398
Bayesian Information Criterion	-2545.983	-2868.454	-2583.052

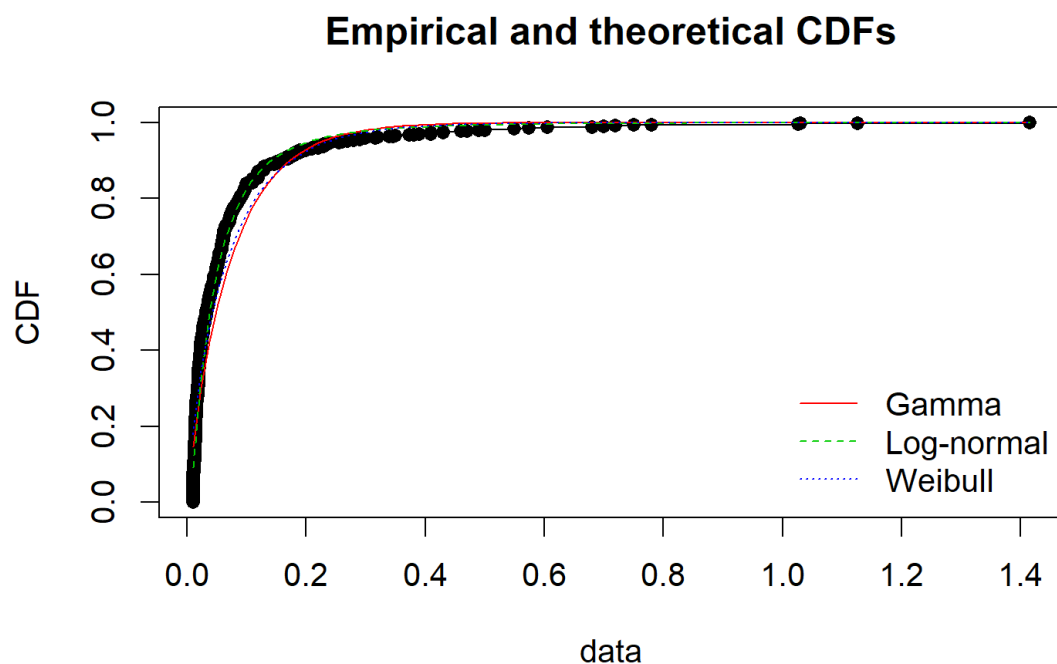


Figure G-5. Comparison of empirical CDF for DSP and potential theoretical distributions

The estimated maximum likelihood parameters are shown below and yield the summary plots in Figure G-6. While some lack of fit is again evident, the deviations do not appear as large as those for PTX2.

```
Fitting of the distribution 'lnorm' by maximum likelihood
Parameters :
      estimate Std. Error
meanlog -3.257020 0.03614612
sdlog    1.016599 0.02555905
Loglikelihood: 1440.9   AIC: -2877.8   BIC: -2868.454
Correlation matrix:
      meanlog sdlog
meanlog      1      0
sdlog        0      1
```

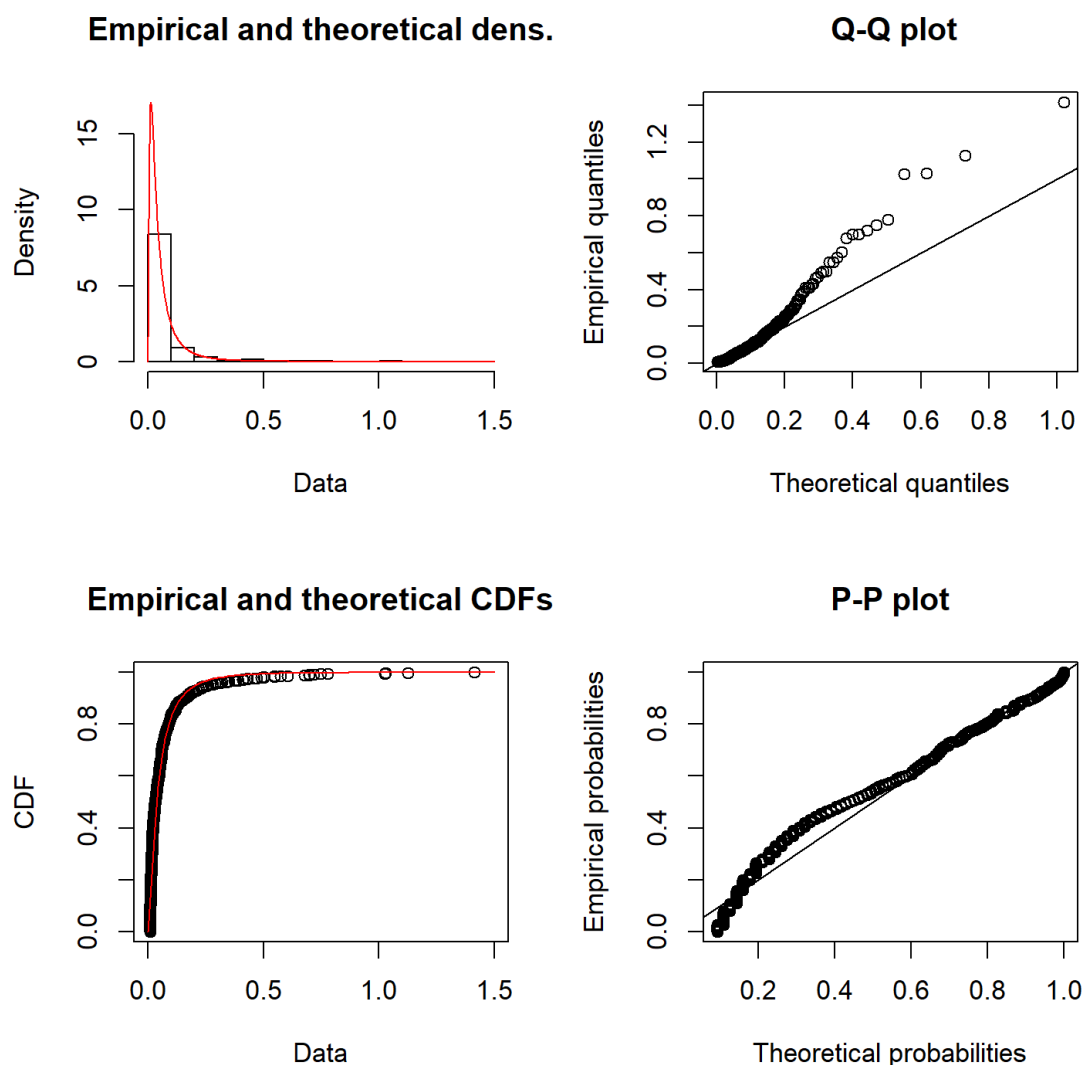



Figure G-6. Summary plots showing the fit of the log-normal distribution to the DSP detections.

As for PTX2, the distribution of DSP concentrations is model through two components:

- Detects and non-detects are randomly sampled from a binomial distribution with probabilities of a detection/non-detection equal to those in the bloom data set, *i.e.* 20.63% and 79.37%, respectively.
- Non-detects are assigned a DSP concentration of 0.03mg/kg (the sum of the three analogue reporting limits after 2015, which was assumed to apply to *all* non-detects).
- Detects are realisation from a parametric distribution with parameters estimated above, left truncated at the LoQ=0.01mg/kg, as 373/791 detections were between 0.01 and 0.03 (which relates to how the three analogues are analysed and combined for reporting).

In addition, a second scenario for the distribution of DSP concentrations is also evaluated, using the empirical distribution of DSP concentrations, again assuming that non-detects are equal to the LoQ of 0.03 mg/kg—as noted above, values between 0.01 and 0.03 were left unchanged. These two scenarios do not place any restrictions on the DSP concentrations. However, the current NZ regulatory limit for DSP is 0.16mg/kg and hence an additional two scenarios are included here, limiting to the DSP concentration to 0.16mg/kg or below.

```
## First approach:
## For the DSP concentrations we need two components:
## 1) A probability tree (i.e. binomial) where non-detects are
## assigned a DSP concentration of LoQ = 0.03
## 2) Detects, are sampled from a beta distribution (estimated
## previously), these are left truncated at 0.01 as many
## concentrations were between 0.01 and 0.03.
mc.dsp.detp <- c(sum(is.na(NZ.bloom$dsp)),
sum(!is.na(NZ.bloom$dsp)))/nrow(NZ.bloom)
mc.dsp.conc1 <-
  mcprobtree(mc.dsp.detp,
    list("1"=mcdata(0.03, type="0"),
         "2"=mcstoc(rlnorm, meanlog=coef(fit.dsp.ln)[1],
                    sdlog=coef(fit.dsp.ln)[2], type="V",
                    rtrunc=TRUE, linf=0.01) ), type="V" )
mc.dsp.conc11 <-
  mcprobtree(mc.dsp.detp,
    list("1"=mcdata(0.03, type="0"),
         "2"=mcstoc(rlnorm, meanlog=coef(fit.dsp.ln)[1],
                    sdlog=coef(fit.dsp.ln)[2], type="V",
                    rtrunc=TRUE, linf=0.01, lsup=0.16) ), type="V" )
## Second approach: We simply sample from the actual data, again
## assuming that values below the LoQ are set to 0.01.
mc.dsp.conc2 <- mcstoc(rempricalD, NZ.bloom$dsp.r, type="V")
mc.dsp.conc21 <- mcstoc(rempricalD, NZ.bloom$dsp.r, type="V",
rtrunc=TRUE, lsup=0.16)
```

The following output provides summary statistics for the two concentration scenarios without (mc.dsp.conc1 and mc.conc2) and with applying the regulatory limit (mc.dsp.conc11 and mc.conc21). From this it can be seen that the maximum concentration observed without application of the regulatory limits is about 1 order of magnitude greater than with the application of the limit. Nevertheless, the 99 percentiles are considerably lower and reasonably similar between the scenarios. In addition, using the log-normal distribution results in slightly larger summary statistics (mean and percentiles) than using the empirical distribution and this is due to the (small) chance of observing values in the extremes of the tail area of the distribution.

```
summary(mc(mc.dsp.conc1, mc.dsp.conc2,
           mc.dsp.conc11, mc.dsp.conc21),
        probs=rpt.p)

mc.dsp.conc1 :
      mean      sd  Min  50%   95% 97.5%   99%  Max      nsv Na's
NoUnc 0.0383 0.0436 0.01 0.03 0.0843 0.134 0.219 3.06 1000000 0
```

```
mc.dsp.conc2 :
      mean      sd  Min  50%   95% 97.5% 99%  Max      nsv Na's
NoUnc 0.0389 0.0608 0.01 0.03 0.074 0.13 0.27 1.41 1000000 0

mc.dsp.conc11 :
      mean      sd  Min  50%   95% 97.5% 99%  Max      nsv Na's
NoUnc 0.0341 0.0179 0.01 0.03 0.0695 0.0974 0.127 0.16 1000000 0

mc.dsp.conc21 :
      mean      sd  Min  50%   95% 97.5% 99%  Max      nsv Na's
NoUnc 0.0305 0.0156 0.01 0.028 0.057 0.085 0.1 0.16 1000000 0
```

G.2.3. Estimating Exposure

The final step in this model is to combine the consumption and concentrations data to estimate the amount of PTX2 or DSP consumed in a single sitting, adjusting for adult weight—three scenarios for adult weight were assessed, namely a 'standard' 60kg adult, and average male and female NZ adult weights, which were estimated as 86.7kg and 73.3kg (Pearson et al. 2018), respectively. Finally, the results are also converted to $\mu\text{g/kg bw}$ to allow comparison with the corresponding Acute Reference Dose (ARfD).

```
## Define the body weights for standardisation
mc.bw <- mcddata(60, type="0")
mc.bw.m <- mcddata(86.7, type="0")
mc.bw.f <- mcddata(73.3, type="0")
```

G.2.4. DSP Exposure

The exposure estimates are calculated for each of the three body weights and two concentration distribution scenarios, ignoring the regulatory limit. The scenarios are summarised below and it can be seen that:

- the two scenarios for the concentration of DSP results in similar exposures, though, as expected, using the log-normal distribution increases the proportion of exposures in the right tail and results in large maximum exposures. Nevertheless, the choice of approach does not appear to have a large effect on the results.
- using a standard body weight of 60kg results in greater dietary exposure to PTX2 compared with using the larger average NZ adult body weights for males and females.

```
## Calculate exposure for different body weight scenarios
mc.dsp.exp1 <- 1000*mc.cons*mc.dsp.conc1/mc.bw
mc.dsp.exp1.m <- 1000*mc.cons*mc.dsp.conc1/mc.bw.m
mc.dsp.exp1.f <- 1000*mc.cons*mc.dsp.conc1/mc.bw.f
mc.dsp.exp2 <- 1000*mc.cons*mc.dsp.conc2/mc.bw
mc.dsp.exp2.m <- 1000*mc.cons*mc.dsp.conc2/mc.bw.m
mc.dsp.exp2.f <- 1000*mc.cons*mc.dsp.conc2/mc.bw.f
summary(mc(mc.dsp.exp1, mc.dsp.exp1.m, mc.dsp.exp1.f,
           mc.dsp.exp2, mc.dsp.exp2.m, mc.dsp.exp2.f),
       probs=rpt.p)
```

```
mc.dsp.exp1 :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.106 0.146 0.0000751 0.0808 0.229 0.385 0.662 14.1 1000000 0

mc.dsp.exp1.m :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0737 0.101 0.0000519 0.0559 0.159 0.267 0.458 9.73 1000000 0

mc.dsp.exp1.f :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0872 0.119 0.0000614 0.0662 0.188 0.316 0.542 11.5 1000000 0

mc.dsp.exp2 :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.108 0.199 0.0000751 0.0779 0.203 0.373 0.767 9.23 1000000 0

mc.dsp.exp2.m :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0749 0.138 0.0000519 0.0539 0.141 0.258 0.531 6.39 1000000 0

mc.dsp.exp2.f :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0885 0.163 0.0000614 0.0637 0.167 0.305 0.628 7.56 1000000 0
```

Similarly, exposure estimates are calculated for the various concentration and body weight scenarios, this time applying the regulatory limit for DSP; the scenarios are summarised below. Clearly, applying this regulatory limit has a substantial effect on the larger percentiles and maximum, but has little effect on the average or median.

Calculate exposure for different body weight scenarios

```
mc.dsp.exp11 <- 1000*mc.cons*mc.dsp.conc11/mc.bw
mc.dsp.exp11.m <- 1000*mc.cons*mc.dsp.conc11/mc.bw.m
mc.dsp.exp11.f <- 1000*mc.cons*mc.dsp.conc11/mc.bw.f
mc.dsp.exp21 <- 1000*mc.cons*mc.dsp.conc21/mc.bw
mc.dsp.exp21.m <- 1000*mc.cons*mc.dsp.conc21/mc.bw.m
mc.dsp.exp21.f <- 1000*mc.cons*mc.dsp.conc21/mc.bw.f
summary(mc(mc.dsp.exp11, mc.dsp.exp11.m, mc.dsp.exp11.f,
           mc.dsp.exp21, mc.dsp.exp21.m, mc.dsp.exp21.f),
        probs=rpt.p)
```

```
mc.dsp.exp11 :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0948 0.0739 0.0000751 0.0798 0.192 0.283 0.418 1.03 1000000 0

mc.dsp.exp11.m :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0656 0.0512 0.0000519 0.0552 0.133 0.196 0.289 0.711 1000000 0

mc.dsp.exp11.f :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0776 0.0605 0.0000614 0.0653 0.157 0.232 0.342 0.841 1000000 0

mc.dsp.exp21 :
  mean    sd      Min    50%   95% 97.5%   99% Max      nsv Na's
NoUnc 0.0848 0.065 0.0000705 0.072 0.169 0.235 0.359 1.04 1000000 0
```

```
mc.dsp.exp21.m :
      mean      sd      Min      50%      95%      97.5%      99%      Max      nsv Na's
NoUnc 0.0587 0.045 0.0000488 0.0498 0.117 0.163 0.249 0.722 1000000 0

mc.dsp.exp21.f :
      mean      sd      Min      50%      95%      97.5%      99%      Max      nsv Na's
NoUnc 0.0695 0.0532 0.0000577 0.0589 0.139 0.193 0.294 0.853 1000000 0
```

G.2.5. Risk Characterisation

The risk characterization in the current context is a simple matter of comparing the exposure distributions to the corresponding Health Based Guidance Value, which for DSP a value of 0.33 µg/kg bw has been used by the World Health Organization and a value of 0.3 µg/kg bw by EFSA.

The output below shows the proportion of exposures which exceed the Health-Based Guidance Value (HBGV) of 0.3 µg/kg bw proposed/used by EFSA (shown under the heading "mean"; the "sd" is of no relevance here). From this it can be seen that when no regulatory limit is applied for DSP, 3.58% of exposures exceed the HBGV assuming a 60kg adult when using the log-normal distribution to model the DSP concentrations. A slightly lower percentage of 3.26% is obtained when the DSP concentrations are sampled from the empirical DSP distribution during blooms. Using the larger NZ adult weight for males and females, reduces the percentages of exposures exceeding the HBGV, though these remain at above 2% of exposures for both genders.

```
hbgv <- mcddata(0.3, type="0")
mc.dsp.1.hbgv <- (mc.dsp.exp1 > hbgv)
mc.dsp.1m.hbgv <- (mc.dsp.exp1.m > hbgv)
mc.dsp.1f.hbgv <- (mc.dsp.exp1.f > hbgv)
mc.dsp.2.hbgv <- (mc.dsp.exp2 > hbgv)
mc.dsp.2m.hbgv <- (mc.dsp.exp2.m > hbgv)
mc.dsp.2f.hbgv <- (mc.dsp.exp2.f > hbgv)
summary(mc(mc.dsp.1.hbgv, mc.dsp.1m.hbgv, mc.dsp.1f.hbgv,
           mc.dsp.2.hbgv, mc.dsp.2m.hbgv, mc.dsp.2f.hbgv),
        probs=NULL)

mc.dsp.1.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0358 0.186 1000000 0

mc.dsp.1m.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0209 0.143 1000000 0

mc.dsp.1f.hbgv :
      mean      sd      nsv Na's
NoUnc 0.027 0.162 1000000 0

mc.dsp.2.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0326 0.178 1000000 0

mc.dsp.2m.hbgv :
```

```

      mean      sd      nsv Na's
NoUnc 0.0208 0.143 1000000    0

mc.dsp.2f.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0256 0.158 1000000    0

```

Applying a regulatory limit of 0.16mg/kg for DSP results in a reduction in the percentage of exposures exceeding the HBGV, irrespective of which distribution is used for DSP. Using the log-normal distribution results in more values in the tail area, even though these are restricted to <0.16mg/kg. Consequently, even with the regulatory limit in place it is estimated that 0.90% and 1.42% of exposures exceed the HBGV for NZ adult males and females, respectively.

```

hbgv <- mcdata(0.3, type="0")
mc.dsp.1l.hbgv <- (mc.dsp.exp1l > hbgv)
mc.dsp.1lm.hbgv <- (mc.dsp.exp1l.m > hbgv)
mc.dsp.1lf.hbgv <- (mc.dsp.exp1l.f > hbgv)
mc.dsp.2l.hbgv <- (mc.dsp.exp2l > hbgv)
mc.dsp.2lm.hbgv <- (mc.dsp.exp2l.m > hbgv)
mc.dsp.2lf.hbgv <- (mc.dsp.exp2l.f > hbgv)
summary(mc(mc.dsp.1l.hbgv, mc.dsp.1lm.hbgv, mc.dsp.1lf.hbgv,
           mc.dsp.2l.hbgv, mc.dsp.2lm.hbgv, mc.dsp.2lf.hbgv),
        probs=NULL)

mc.dsp.1l.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0223 0.148 1000000    0

mc.dsp.1lm.hbgv :
      mean      sd      nsv Na's
NoUnc 0.00896 0.0942 1000000    0

mc.dsp.1lf.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0142 0.118 1000000    0

mc.dsp.2l.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0155 0.124 1000000    0

mc.dsp.2lm.hbgv :
      mean      sd      nsv Na's
NoUnc 0.0058 0.0759 1000000    0

mc.dsp.2lf.hbgv :
      mean      sd      nsv Na's
NoUnc 0.00948 0.0969 1000000    0

```