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Stage one pilot study of wastewater monitoring for mycotoxin exposure in Aotearoa New Zealand

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DISCLAIMER

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EXECUTIVE SUMMARY

Many homes in New Zealand are known to suffer from dampness or water damage, with numbers increasing due to flooding associated with extreme weather events such as Cyclone Gabrielle. One important, but often overlooked, consequence of dampness or water damage is mould development, which can lead to the presence of toxic fungal metabolites, or mycotoxins, on mouldy surfaces and in the air of water-damaged buildings. Classic approaches for assessing mycotoxin exposure generally focus on dietary exposure, with little consideration of inhalation or dermal exposure. Although urinary biomonitoring can provide information on overall exposure, there is currently no biomonitoring for mycotoxin exposure in New Zealand.

Recently, two European research groups pioneered the use of wastewater-based epidemiology for assessing mycotoxin exposure, however, the focus was predominantly on dietary exposure. In 2022, the Ministry of Health commissioned a feasibility study to determine whether wastewater-based epidemiology could be used to specifically assess mycotoxin exposure in mouldy indoor environments. This study identified 11 potential targets based on their presence in water-damaged and/or mouldy dwellings, potential effects on human health, and excretion in urine and/or faeces. In 2023, methods for detection of these target compounds in wastewater were developed and validated. This report details the results of the third phase of this project – a stage 1 pilot study in which wastewater collected from six sites across New Zealand was analysed for the presence of the 11 target mycotoxins, combined with assessment of the stability of these mycotoxins using analytical standards.

Of the 11 target mycotoxins, nine were detected in samples collected from the six sampling sites, which were located in Northland, Auckland, Hawke's Bay (two sites), Wellington and Canterbury. At least one mycotoxin was detected in each of the samples, with a maximum of five different mycotoxins detected in a single sample. One target, enniatin B, was detected in all of the samples analysed. Results from assessment of mycotoxin recovery using spiked controls indicated that for most samples, lack of detection was unlikely to be due to low recovery of the target from wastewater. Whether the lack of detection truly indicates lack of exposure in the community remains to be seen and further work is required to distinguish this from other possible explanations such as extensive metabolism of the target compound prior to excretion, or degradation of the target in-sewer prior to sample collection. It is also important to note that detection of a target mycotoxin in a given wastewater sample may not necessarily be indicative of indoor mould exposure, as there may be other potential exposure routes (eg, dietary) or environmental sources contributing to the presence of the target mycotoxin in wastewater. The aim of this stage 1 pilot study was not to distinguish between these different sources but rather to determine whether these compounds can be detected in municipal wastewater.

Overall, results of this stage 1 pilot study provide important information which can be used to guide future work needed for development of this method into an approach which can be used to assess indoor mycotoxin exposure in at-risk communities.

1. INTRODUCTION

Wastewater-based epidemiology (WBE) is a powerful public health tool which can be used to assess population-level exposure to a range of biological and non-biological contaminants (Eaton et al., 2022). Although this tool has only recently achieved public fame due to the indispensable role it played in global responses to the SARS-CoV-2 pandemic, it was first proposed back in 2001 as a non-intrusive method for monitoring illicit drug consumption within a community (Daughton, 2001). Now, more than 20 years later, the potential applications for WBE are almost limitless. These 'limitless' options have necessitated development of a means by which to evaluate and prioritize potential applications for their suitability for WBE. This was the focus of a previous report prepared for the Ministry of Health, in which a decision framework was developed to guide Public Health Authorities in determining whether a particular health determinant (eg, smoking, alcohol consumption, exposure to pesticides) is suitable for WBE (Eaton et al., 2022; Eaton et al., 2021).

In assessing potential applications of WBE that could be used to inform public health interventions or policy in New Zealand, the usage of WBE to assess exposure to mycotoxins in communities with water-damaged and mould affected housing (eg, due to flooding or poor housing stock) was identified as an ideal candidate for evaluation.

Mycotoxins are toxic secondary metabolites produced by some fungal species, many of which have been linked to adverse health effects in humans (Bennett & Klich, 2003). Humans are predominantly exposed to mycotoxins via consumption of contaminated foodstuffs but can also be exposed via inhalation of mycotoxins present in the air or dermal contact with contaminated surfaces. In contrast to fungal infections, or mycoses, exposure to mycotoxins leads to poisoning rather than infection (Bennett & Klich, 2003).

1.1. MYCOTOXINS IN WATER-DAMAGED DWELLINGS

The relationship between water ingress, be it from flooding, leakage or insufficient weathertightness, and development of mould in indoor environments is well-known. Over the last 20 years, several international studies have also demonstrated the presence of various mycotoxins in water-damaged buildings. For example, Bloom et al. (2009) detected mycotoxins in dust collected from homes in New Orleans flooded during Hurricane Katrina. Similarly, dust collected from flood-affected houses in the village of Gunja, Croatia (Jakšić et al., 2021) and floor dust, air and surface swabs collected from flood-affected dwellings in Arles, France (Charpin-Kadouch et al., 2006) were found to contain a range of mycotoxins.

In New Zealand, many homes suffer from dampness and mould, with Stats NZ reporting that in 2020, one in six homes had areas of visible mould totalling more than A4 in size (Stats NZ, 2020). Concerningly, Māori and Pasifika were noted to be more likely to live in homes affected by mould and dampness than other ethnicities (Stats NZ, 2020). Several factors likely contribute to the high proportion of damp and mould affected housing in New Zealand, including high outdoor humidity, poor housing stock having inadequate insulation and

heating (Howden-Chapman et al., 2009), the 'leaky homes' crisis (Clarke et al., 2021; Yates, 2003) and prevalence of extreme weather events (Mateparae et al., 2024).
Extreme weather events can cause water damage due to direct inundation or indirectly via prolonged exposure to extremely high relative humidity leading to water ingress into materials above the flood line (Neumeister-Kemp et al., 2023), which may potentially be overlooked during remediation. Flood waters often contain a variety of microorganisms, including fungi which can penetrate deep into building materials and proliferate, and unlike most bacteria and viruses which require moisture to survive, fungi are well suited to continue growing even once the materials have dried out. (Neumeister-Kemp et al., 2023). This mould contamination can then spread throughout the building via aerosolisation of spores (reproductive propagules) and hyphal fragments, and ingress of fungal spores from outdoors as fungal concentrations in outdoor spaces post-flooding are often elevated due to high humidity and increased nutrient levels on contaminated surfaces due to deposition of sediments and other flood debris (Neumeister-Kemp et al., 2023).
New Zealand ranks second in the Lloyds of London list of countries most exposed to natural hazards (Mateparae et al., 2024). Flooding is the most common natural disaster in New Zealand, occurring, on average, every eight months, with rates expected to increase due to climate change (Mason et al., 2021). Around two thirds of the New Zealand population live in flood-prone areas, including residents of several towns and cities that were built on floodplains (Mason et al., 2021). For example, in 2023 an estimated 47,443 people were living within 1 metre of sea level (Beggs et al., 2025). Certain population subgroups are also known to be more vulnerable to the impacts of flooding, including children, older adults (65 years +) and pregnant women (Mason et al., 2021). Concerningly, a nationwide assessment of 869 marae conducted by University of Auckland PhD student Haukapuanui Vercoe found that one third were in flood prone areas[1].
2023 was a particularly bad year for New Zealand, with three extreme weather events striking the North Island over a six-week period – Cyclone Hale (8 – 12 January), heavy rainfall over Auckland Anniversary weekend (26 January – 3 February) and Cyclone Gabrielle (12 – 16 February) (Mateparae et al., 2024). These three extreme weather events were collectively the most severe and destructive events in New Zealand's recent history.

1.2. METHODS FOR ASSESSING EXPOSURE TO MYCOTOXINS

Traditional approaches employed internationally for estimating human exposure to mycotoxins focus primarily on dietary exposure and include urinary biomonitoring studies, dietary surveys and analysis of foodstuffs (Eaton, 2022). More recently, two European studies used WBE to assess exposure to mycotoxins, but again the dominant focus was on mycotoxins present in food and dietary exposure (Berzina et al., 2022; Gracia-Lor et al., 2020). In New Zealand, there is currently no biomonitoring for mycotoxin exposure, but the Ministry for Primary Industries does assess the risk posed by dietary exposure via the Mycotoxin Surveillance Program.[2]

[1] https://www.rnz.co.nz/news/te-manu-korihi/512771/survey-finds-third-of-marae-in-flood-prone-areas-up-to-30-percent-prone-to-landslides Accessed 5 May 2025
[2] https://www.mpi.govt.nz/science/food-safety-and-suitability-research/food-science-research/chemical-hazard-and-mycotoxin-research/ Accessed 5 May 2025

1.3. BACKGROUND TO THIS PILOT STUDY

To determine the feasibility of assessing exposure to mycotoxins present in homes with a history of flooding or water damage using WBE, a review of grey and published literature was conducted to identify a) which mycotoxins have been found in indoor environments; b) the potential human health hazard posed by these toxins; c) whether these mycotoxins have previously been detected in urine/faeces or wastewater; and d) if anything is known about the metabolism of these toxins and stability in the environment (Eaton, 2022).

This assessment revealed that over 140 different fungal secondary metabolites have been detected in indoor environments. After evaluation of the suitability of these compounds for WBE, 10 candidate targets were identified (Eaton, 2022). These include:

- Enniatins A, A1, B, B1 and beauvericin: structurally related cyclic hexadepsipeptide toxins mainly produced by *Fusarium* species.
- Sterigmatocystin: a polyketide-derived mycotoxin structurally similar to the highly toxic and carcinogenic aflatoxins and produced by more than a dozen *Aspergilli* and several other species.
- Alternariol and alternariol monomethyl ether: dibenzo-α-pyrone mycotoxins produced by *Alternaria* species.
- Roquefortine C: an indole alkaloid mycotoxin produced by more than 30 different *Penicillium* species.
- Griseofulvin: an antifungal compound first isolated from *Penicillium griseofulvum* and previously used as a pharmaceutical.

The structures of the target mycotoxins are shown in Figure 1. The griseofulvin metabolite 6-desmethyl griseofulvin (6-DMG) was also identified as a good target for assessing indoor mycotoxin exposure as approximately 84% of absorbed griseofulvin is excreted in urine as this metabolite and a commercial standard for 6-DMG is available to support analyses.

Following this feasibility study, methods for detection of these 11 mycotoxin targets in wastewater were developed, informed by the methodology of Gracia-Lor et al. (2020) and Berzina et al. (2022) and validated via inclusion of commercially available standards (Chappell & Li, 2024).

1.4. APPROACH AND SCOPE

This report summarises results of a stage one pilot study in which samples were collected from six sites across New Zealand four times between July 2024 and June 2025 and analysed for the presence of the 11 mycotoxin targets using the methodology developed by Chappell and Li (2024). The stability of these 11 targets in wastewater was also assessed. The objective of this initial stage one pilot study was not to compare mycotoxin concentrations between locations or indeed even across time-points, but rather to determine if the developed methodology was suitable for detecting these chemicals, which will likely be present in low levels, in the complex wastewater matrix.

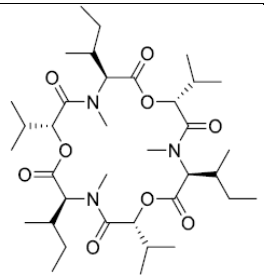
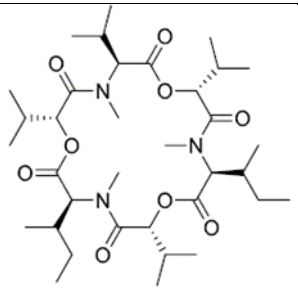
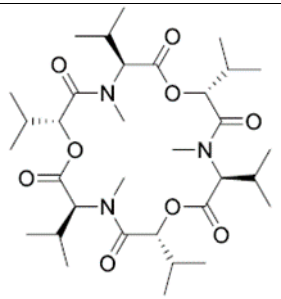
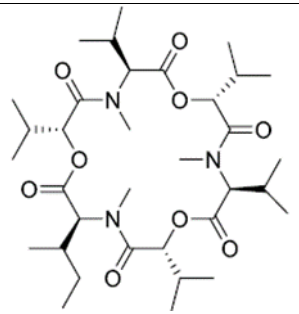
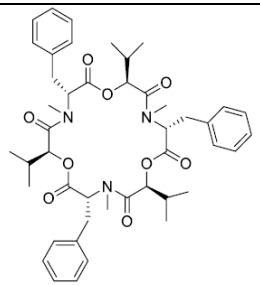
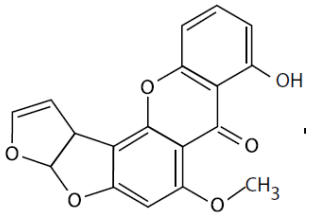
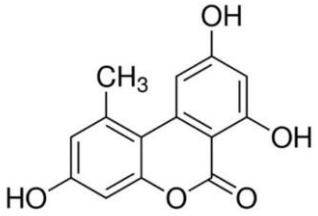
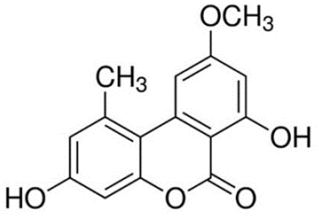
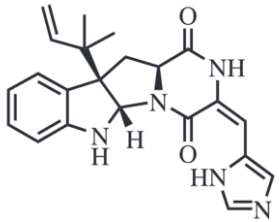
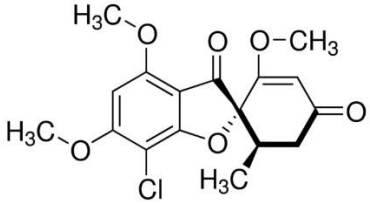
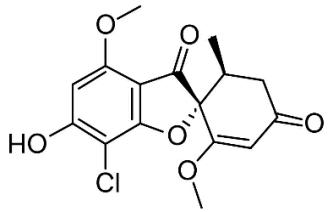
			
Enniatin A	**Enniatin A1**	**Enniatin B**	**Enniatin B1**
			
Beauvericin	**Sterigmatocystin**	**Alternariol**	**Alternariol monomethyl ether**
			
Roquefortine C	**Griseofulvin**	**6-desmethylgriseofulvin**	

Figure 1 Structures of target mycotoxins

2. METHODOLOGY

2.1. SAMPLE COLLECTION AND PROCESSING

Wastewater samples analysed during this pilot study were subsampled from samples collected for the National Drugs in Wastewater (DIWW) monitoring programme. The DIWW samples are collected using autosamplers set up to continuously sample influent wastewater arriving at the wastewater treatment plant over the course of seven days, with 24-hour composite samples (~ 1 litre volume) collected daily by Council staff. After collection, the composites were acidified by addition of hydrochloric acid and stored at 4°C until being couriered chilled to ESR at the end of the sampling week. On arrival at ESR, ~ 150 ml was taken from each of the seven daily composite samples and combined to give a sample representative of the entire week of sampling to be used for mycotoxin analysis. These combined samples were then stored at -20°C until processing.

Immediately prior to analysis, samples were defrosted and then filtered through a Whatman™ GF/D glass microfibre filter followed by a Whatman™ GF/F glass microfibre filter to remove particulates. Samples were then pH adjusted to 4.5 with acetic acid or sodium hydroxide.

Samples were collected from six sites across New Zealand four times between July 2024 and June 2025 (October, January, March and May). These six sites were located in the Northland, Auckland, Hawkes Bay (two sites), Wellington and Canterbury regions. Exact sampling locations will remain anonymous in this report. Due to maintenance at the Auckland site during May, which made it unsafe for samples to be collected, only three samples were analysed for this site.

2.2. STABILITY ASSESSMENT

To assess the stability of the target mycotoxins in wastewater, 3,960 ml of a representative wastewater sample (not selected from any specific sampling location) was filtered and spiked with analytical standards for the 11 targets at varying concentrations (as listed in Table 1), then subdivided into eighteen 220 ml samples and stored at room temperature, 4°C and -20°C. Nine 220 ml non-spiked samples were also stored with the spiked samples. Analysis of stored samples occurred at varying time intervals of zero, three, seven and 28 days. At each timepoint three samples were analysed from each storage temperature – two spiked and one non-spiked. The non-spiked sample was spiked to the same initial concentration as the spiked samples on the day of analysis for recovery assessment. By comparing concentrations of the stored samples to this time zero spiked sample, any loss of the target mycotoxins over time can be assessed and optimal storage conditions can be determined.

Table 1 Analytical and isotope-labelled internal standards for the target mycotoxins

Standard	Product ID	Manufacturer	Concentration (ng/l)
Enniatin A	FIA000245	FIANOVIS, Vindry-sur-Turdine, France	12.5
Enniatin A1	FIA000248	FIANOVIS, Vindry-sur-Turdine, France	12.5
Enniatin B	E5411	Merck, Darmstadt, Germany	25
Enniatin B1	FIA000254	FIANOVIS, Vindry-sur-Turdine, France	12.5
Beauvericin	FIA000233	FIANOVIS, Vindry-sur-Turdine, France	12.5
Sterigmatocystin	DRE-V16974700AL-50	LGC, Middlesex, United Kingdom	25
U-[$^{13}C_{18}$]-Sterigmatocystin	10002828	Romer Labs®, Tulln, Austria	NA
Alternariol	10003681	Romer Labs®, Tulln, Austria	125
U-[$^{13}C_{14}$]-alternariol	10006457	Romer Labs®, Tulln, Austria	NA
Alternariol monomethyl ether	10003682	Romer Labs®, Tulln, Austria	25
U-[$^{13}C_{15}$]-alternariol monomethyl ether	10006458	Romer Labs®, Tulln, Austria	NA
Roquefortine C	TRC-R700875	LGC, Middlesex, United Kingdom	25
Griseofulvin	PHR1534	Merck, Darmstadt, Germany	25
U-[$^{13}C_{17}$]-griseofulvin	TRC-G787502	LGC, Middlesex, United Kingdom	NA
6-desmethyl griseofulvin	TRC-D231080	LGC, Middlesex, United Kingdom	25

NA, not applicable.

2.3. MYCOTOXIN QUANTIFICATION

For quantification of mycotoxins in the wastewater samples, samples were extracted by solid phase extraction (SPE) using an Oasis PRiME HLB cartridge (Waters Corporation) followed by detection with liquid chromatography coupled with tandem mass spectrometry (LC-MS/MS). For each sample, a total of 200 ml of wastewater was analysed as this was shown during method development to be the best volume for optimising sample detection whilst minimising matrix suppression (Chappell & Li, 2024).

Isotope-labelled standards were available for four of the mycotoxins (sterigmatocystin, alternariol, alternariol monomethyl ether and griseofulvin) (see Table 1), allowing for automatic recovery correction. A 50 µl mixture of these isotope-labelled standards was added to all samples prior to SPE. The eluant was then removed by drying under a stream of nitrogen gas at 40°C. Samples were then reconstituted in 1 ml methanol and analysed by LC-MS/MS (Chappell & Ashmore, 2018). All samples were analysed in duplicate (A and B samples) together with a spiked control sample (S) to which a 50 µl mixture of the non-labelled mycotoxin standards listed in Table 1 were added to allow for recovery correction. As the samples collected in January and March 2025 were processed together, a single spiked control was prepared for each location to cover both sampling times by adding the unlabelled mycotoxin standards to a mixed wastewater sample consisting of an equal volume of wastewater from the January and March samples.

During mass spectrometry analysis, enniatin A, enniatin A1, beauvericin, alternariol and alternariol monomethyl ether were analysed in negative ionisation mode whilst the remainder of the targets were analysed in the positive ionisation mode.

2.4. DATA ANALYSIS

As noted above, for each sampling time and location three results are recorded – A, B and S. The A and B results correspond to the concentration of mycotoxin measured in the two duplicate samples, whilst the S result corresponds to the concentration in the spiked control, from which mycotoxin recovery can be determined. Concentrations of the A and B samples were averaged and then recovery corrected by dividing by the recovery rate of the spiked control for the given sampling iteration. The resulting concentrations were then divided by the volume of wastewater passed through the SPE cartridge to give the mycotoxin concentration in the wastewater sample.

3. RESULTS AND DISCUSSION

3.1. STABILITY OF THE TARGET MYCOTOXINS IN WASTEWATER

To determine the optimal temperature for storing wastewater samples prior to mycotoxin analysis, and to determine whether there is likely to be any substantial reduction in mycotoxin concentration whilst the wastewater samples are stored at 4°C for up to a week before being sent to ESR, analytical standards for the 11 target mycotoxins were used. The stability of these standards was examined in wastewater stored at room temperature for up to seven days, and 4°C and -20°C for up to 28 days. Summarised results from this analysis are presented in Table 2 and are graphically represented in Figures 2 – 4. The full data from this analysis is included in the Appendix.

As was expected, the concentrations of almost all the target mycotoxins decreased after seven days at room temperature, apart from alternariol, alternariol monomethyl ether and griseofulvin whose concentrations remained relatively stable (Figure 2). The concentrations of most of the mycotoxin targets were stable after seven days at 4°C, except for beauvericin, sterigmatocystin and roquefortine C whose concentrations fell by approximately 30, 20 and 50% respectively (Figure 3). In contrast, concentrations of enniatin A, A1, B, B1, alternariol, alternariol monomethyl ether and griseofulvin remained relatively even after 28 days at 4°C. These findings suggest that there may be some degradation of beauvericin, sterigmatocystin and roquefortine C prior to the wastewater samples being sent to ESR, and as such the concentrations of these mycotoxins may be underestimated.

With regards to stability at -20°C, although concentrations of alternariol and alternariol monomethyl ether remained relatively stable after 28 days, concentrations of the other target mycotoxins decreased, with this decrease being particularly evident between the 7- and 28-day time points. This may have been caused by an additional filtration step conducted before the 28-day samples were analysed as it was noticed that there was some additional particulate matter in these samples which needed to be removed. The exact cause for this remains unclear at this stage. Given this uncertainty, it is deemed that the best approach moving forward is to store the wastewater samples at 4°C upon receipt at ESR and aim to complete solid phase extraction within one month of the sampling date.

Table 2 Stability of the 10 target mycotoxins and 6-desmethyl griseofulvin in wastewater

Storage temp.	Change in concentration	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Room temp (~25°C)	% change after 3 days	+42	+49	+7	+2	-8	-16	+5	+6	-14	-1	-35
	% change after 7 days	-34	-40	-40	-29	-47	-49	+16	-2	-8	0	-15
4°C	% change after 3 days	-5	+2	+2	-12	-16	-4	+22	-7	-32	+6	+14
	% change after 7 days	-3	-2	-27	-13	-30	-21	-7	-5	-53	+12	0
	% change after 28 days	+6	+9	+3	+1	-30	-29	0	0	-42	-4	-12
-20°C	% change after 3 days	+9	+19	-18	-3	-23	-23	-2	-5	-48	-28	-33
	% change after 7 days	+11	+20	-23	-27	-38	-46	+19	+1	-58	-17	-26
	% change after 28 days	-30	-25	-24	-25	-57	-71	+28	-3	-87	-54	-58

ENA, enniatin A; ENA1, enniatin A1; ENB, enniatin B; ENB1, enniatin B1; BEA, beauvericin; AOH, alternariol; AME, alternariol monomethyl ether; ROQC, roquefortine C; STE, sterigmatocystin; GRI, griseofulvin; 6-DMG, 6-desmethyl griseofulvin.

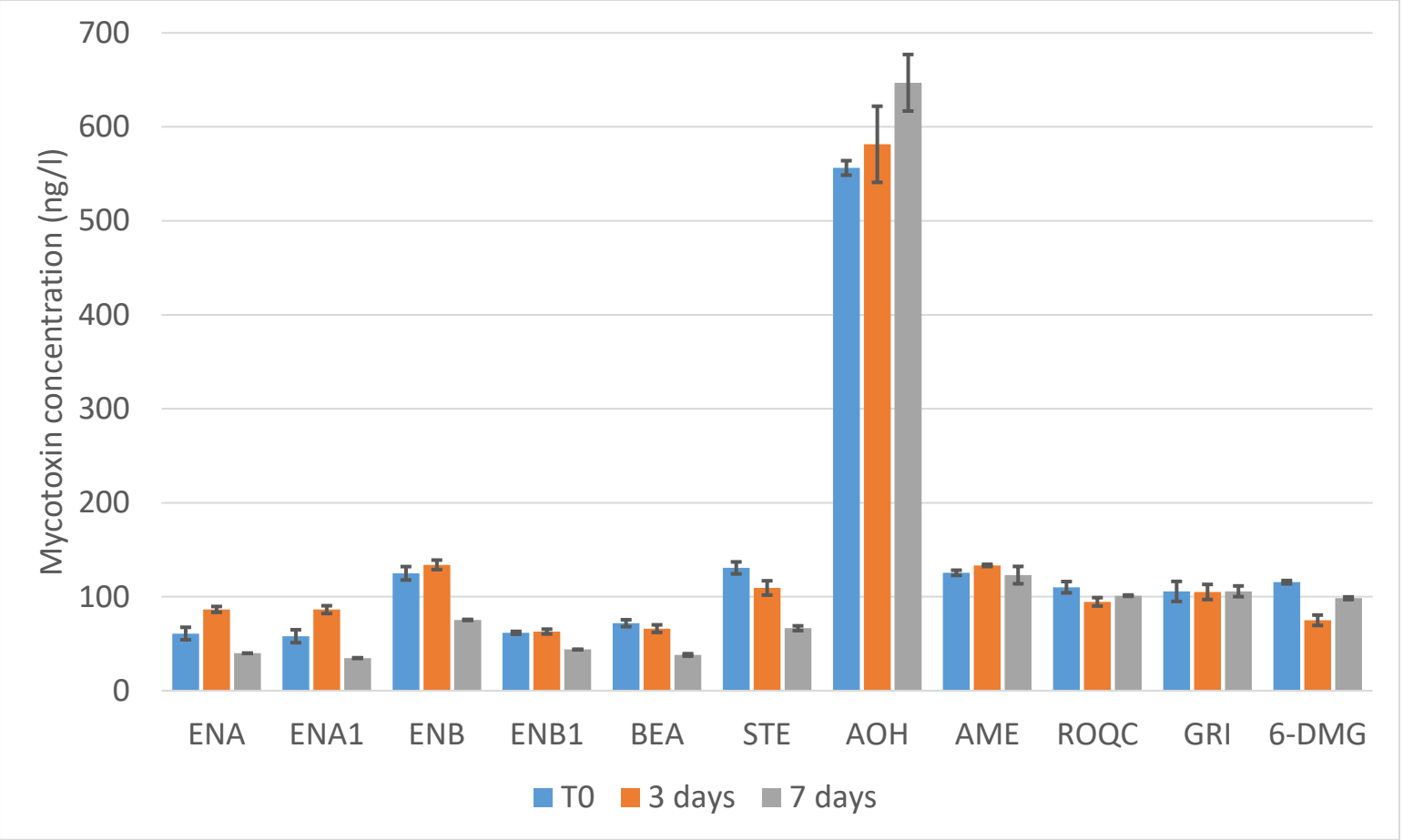


Figure 2 Graph showing changes in mycotoxin concentration in wastewater samples stored for up to 7 days at room temperature

ENA, enniatin A; ENA1, enniatin A1; ENB, enniatin B; ENB1, enniatin B1; BEA, beauvericin; AOH, alternariol; AME, alternariol monomethyl ether; ROQC, roquefortine C; STE, sterigmatocystin; GRI, griseofulvin; 6-DMG, 6-desmethyl griseofulvin. Error bars indicate standard deviation between duplicate samples.

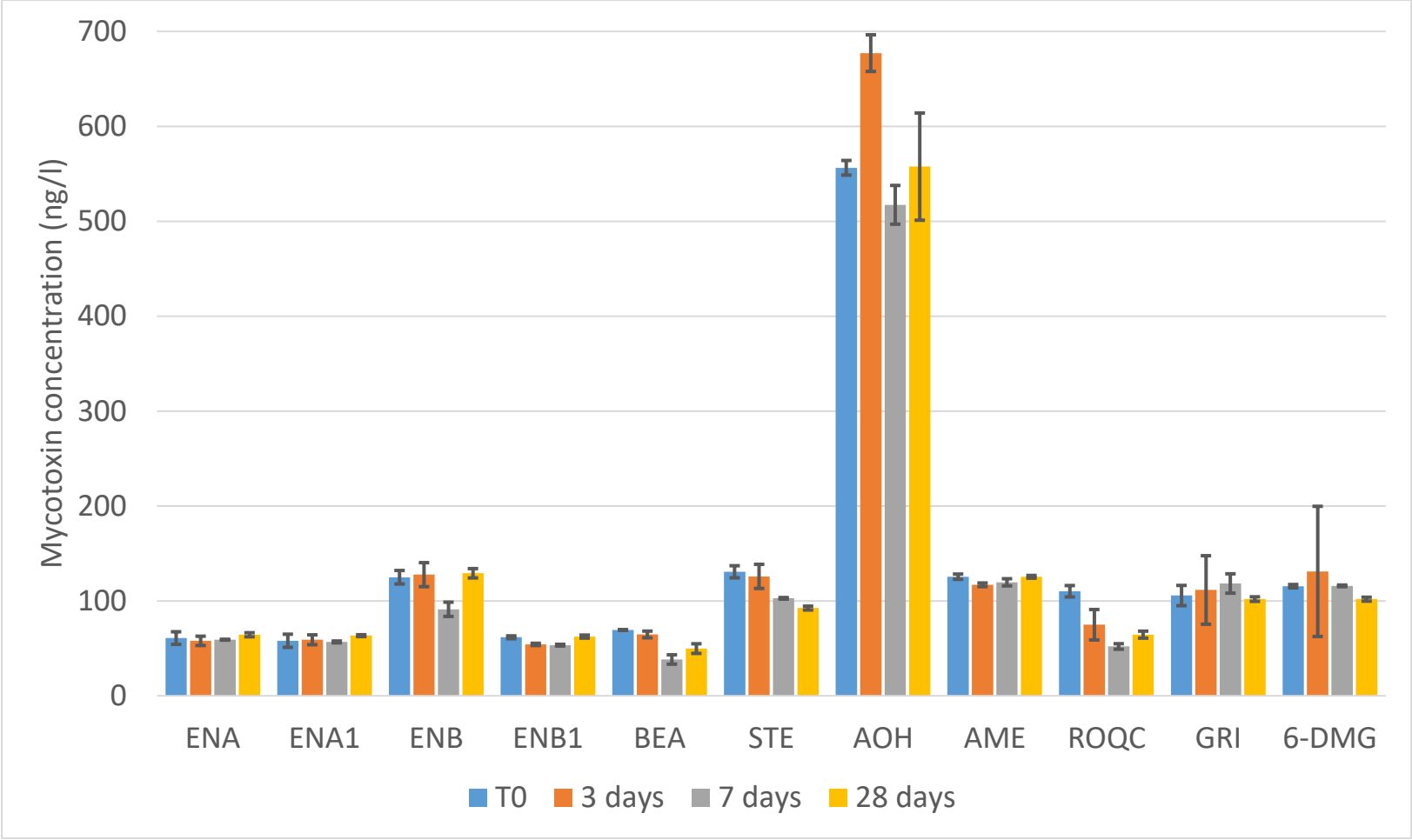


Figure 3 Graph showing changes in mycotoxin concentration in wastewater samples stored for up to 28 days at 4°C

ENA, enniatin A; ENA1, enniatin A1; ENB, enniatin B; ENB1, enniatin B1; BEA, beauvericin; AOH, alternariol; AME, alternariol monomethyl ether; ROQC, roquefortine C; STE, sterigmatocystin; GRI, griseofulvin; 6-DMG, 6-desmethyl griseofulvin. Error bars indicate standard deviation between duplicate samples.

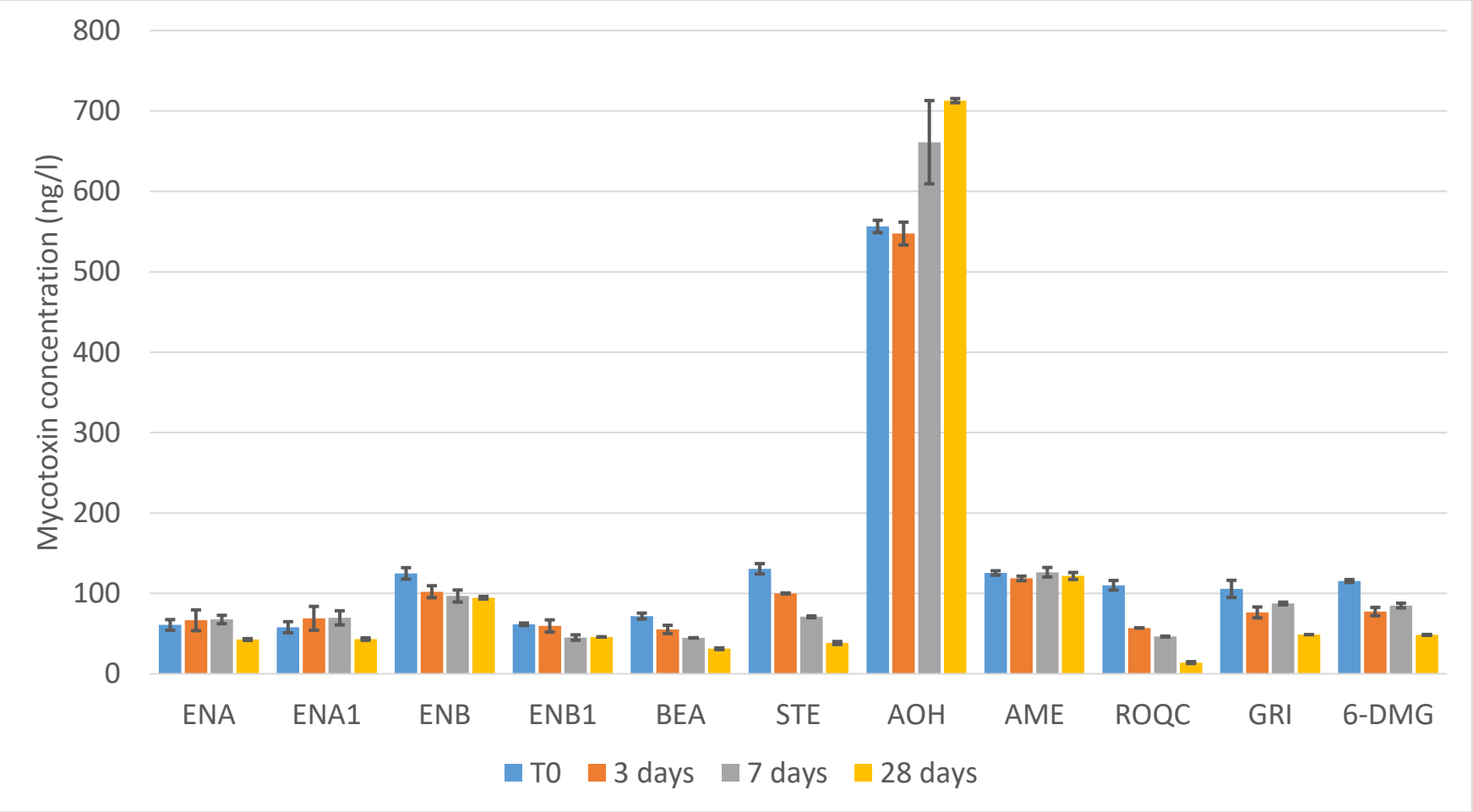


Figure 4 Graph showing changes in mycotoxin concentration in wastewater stored for up to 28 days at -20°C

ENA, enniatin A; ENA1, enniatin A1; ENB, enniatin B; ENB1, enniatin B1; BEA, beauvericin; AOH, alternariol; AME, alternariol monomethyl ether; ROQC, roquefortine C; STE, sterigmatocystin; GRI, griseofulvin; 6-DMG, 6-desmethyl griseofulvin. Error bars indicate standard deviation between duplicate samples.

3.2. PRESENCE OF THE TARGET MYCOTOXINS IN THE WASTEWATER SAMPLES

As noted earlier, the emphasis of this pilot study was to determine whether the different target mycotoxins could be detected in municipal wastewater collected from different sites in New Zealand, rather than to provide a quantitative comparison, as given the small number of samples it is not possible to make any statistically reliable conclusions. A summary of the detections of these target mycotoxins in the analysed wastewater samples is given in Table 3, and for completeness the quantitative data can be found in the Appendix.

In interpreting these data, other potential routes for human exposure to these toxins and potential non-human sources contributing to the presence of these compounds in municipal wastewater should be taken into consideration. All the target mycotoxins have been detected in a range of foodstuffs (as detailed in Eaton (2022)) and as such, exposure to these toxins may arise via a combination of inhalation and dietary exposure, as well as potential dermal exposure via contact with contaminated surfaces. All these routes will likely contribute to the amount of a given mycotoxin excreted into the wastewater network. The amount of a particular mycotoxin present in wastewater may also be affected by disposal of mould-contaminated substances to the municipal network eg, mouldy foodstuffs via kitchen waste disposal units, residues from cleaning of mould-contaminated surfaces. The aim of this pilot study was not to distinguish these different potential sources for mycotoxins entering the wastewater network, but rather to determine if the target compounds can be detected in wastewater in New Zealand, as discussed above.

3.2.1. ENNIATIN A

Enniatin A was not detected in any of the 23 wastewater samples analysed in this project. This was surprising as a previous study conducted in Riga, Latvia detected this mycotoxin in 90% of wastewater samples analysed (n = 29) (Berzina et al., 2022). Recovery of enniatin A in the spiked controls was highly variable, ranging from 31.4 – 108.4% across the six sites, with significant variation also observed within samples from a single site (eg, 41.0 – 89.6% for samples collected from the site in Northland). As such, the absence of detection of enniatin A is not solely due to low recovery. There are several possible explanations for the lack of detection of enniatin A, including low exposure in the assessed population, significant metabolism of this toxin prior to excretion, and/or degradation of this toxin in-sewer.

3.2.2. ENNIATIN A1

Enniatin A1 was detected in two of the 23 samples analysed in this study – once in a sample from the Canterbury site and once in a sample from Hawke's Bay site 2. Recovery of enniatin A1 in the spiked controls ranged from 49.7 – 106%, with the two positive sites having recovery of 61.0 and 113.6% (for Canterbury and Hawke's Bay site 2 respectively). The absence of detection of enniatin A1 in several samples with high recovery (> 70%) indicates that this is not solely due to low recovery. As for enniatin A, there are several possible reasons for the low detection of this mycotoxin as discussed above.

Table 3 Presence of selected mycotoxins in wastewater samples

Location	Sampling month	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Northland	Oct 24											
	Jan 25											
	Mar 25											
	May 25											
Auckland	Oct 24											
	Jan 25											
	Mar 25											
Hawke's Bay site 1	Oct 24											
	Jan 25											
	Mar 25											
	May 25											
Hawke's Bay site 2	Oct 24											
	Jan 25											
	Mar 25											
	May 25											
Wellington	Oct 24											
	Jan 25											
	Mar 25											
	May 25											
Canterbury	Oct 24											
	Jan 25											
	Mar 25											
	May 25											

ENA, enniatin A; ENA1, enniatin A1; ENB, enniatin B; ENB1, enniatin B1; BEA, beauvericin; AOH, alternariol; AME, alternariol monomethyl ether; ROQC, roquefortine C; STE, sterigmatocystin; GRI, griseofulvin; 6-DMG, 6-desmethyl griseofulvin.

3.2.3. ENNIATIN B

Enniatin B was detected in all 23 samples analysed during this study. Recovery in the spiked controls ranged from 12.7 – 81.1%. The detection of this mycotoxin in all analysed wastewater samples suggests that this mycotoxin is prevalent in all six locations included in this study. As such, it may be possible to use this mycotoxin to measure baseline exposure across the country. However, further work is needed to confirm if this is possible.

3.2.4. ENNIATIN B1

Enniatin B1 was detected in 11 of the 23 samples analysed in this study, with at least one sample from each of the six sampling sites testing positive. Recovery of this mycotoxin in the spiked controls ranged from 21.5 – 74.7%. Several of the samples in which enniatin B1 was detected had relatively low recovery of the spiked control (< 50%). Further work is needed to determine why this mycotoxin was detected in some samples and not others with similar rates of recovery in the spiked control sample.

3.2.5. BEAUVERICIN

Beauvericin was detected in only one of the 23 samples analysed in this study – a sample collected from Hawke's Bay site 2. Recovery of beauvericin in the spiked controls ranged from 20.0 – 108.6%. There are several possible explanations for the low rate of detection of beauvericin including low exposure, significant metabolism and/or degradation in-sewer as discussed for enniatin A above.

3.2.6. STERIGMATOCYSTIN

Sterigmatocystin was detected in three of the 23 samples analysed in this study – one from the Northland site, one from Hawke's Bay site 2 and one from the Canterbury site. Recovery of sterigmatocystin from the spiked controls ranged from 64.8 – 114.2%. As a considerable number of the samples had high spike recovery rates, the absence of detection of sterigmatocystin is unlikely to be due to low recovery. Similar to enniatin A and beauvericin, there are several possible explanations for the low detection of this toxin, including low exposure, in-sewer degradation and/or high metabolism of this mycotoxin by exposed individuals.

3.3. ALTERNARIOL

Alternariol was not detected in any of the samples analysed during this study. As recovery in the spiked controls ranged from 71.5 – 111.3%, this is unlikely to be due to low recovery of this toxin from the wastewater samples. During the method development phase of this project, significant loss of alternariol was observed when the analytical standard was spiked

into fresh tap water (approximate pH of 7.7) (Chappell & Li, 2024). It was hypothesised that this loss was due to the presence of chlorine in tap water, as chlorine is a strong oxidising agent which may react with some mycotoxins, degrading them. In support of this hypothesis, loss of alternariol was ameliorated by replacing tap water with chlorine-free deionised (DI) water. As such, the absence of detection of this mycotoxin in the analysed wastewater samples may be due to significant chlorine-induced degradation in-sewer. However, further work would be needed to distinguish this from the other possible explanations for absence of detection discussed for the other target mycotoxins above.

3.4. ALTERNARIOL MONOMETHYL ETHER

Alternariol monomethyl ether was only detected in two of the 23 wastewater samples, both of which came from Hawke's Bay site 2. Recovery in the spiked controls ranged from 74.9 – 102.8%, indicating that the lack of detection was unlikely due to low recovery of this toxin from the wastewater samples. Similar to alternariol, significant loss of alternariol monomethyl ether was observed during the method development phase when fresh tap water was used Chappell & Li, 2024). As such, the low detection of this mycotoxin may be due to high degradation in-sewer. However, other possible explanations such as those detailed above cannot be excluded.

3.5. ROQUEFORTINE C

Roquefortine C was detected in five of the wastewater samples analysed during this study – a single sample each from the Auckland, Canterbury and Hawke's Bay 2 sites, and two samples from Hawke's Bay site 1. Recovery of roquefortine C in the spiked controls was low, ranging from 7.4 – 36%. However, this low recovery is insufficient to fully explain the low detection of roquefortine C as recovery rates for the spiked controls corresponding to those samples where this toxin was detected ranged from 7.4 – 32.2%. Similar to alternariol and alternariol monomethyl ether, significant loss of roquefortine C was observed during method development when the analytical standard was spiked into tap water but not deionised water (Chappell & Li, 2024). Thus, it is possible that the low detection is due to significant degradation in-sewer, but as for alternariol and alternariol monomethyl ether, further work would be needed to distinguish this from other possible explanations.

3.6. GRISEOFULVIN AND 6-DESMETHYL GRISEOFULVIN

Griseofulvin was detected in six of the wastewater samples analysed during this study – all three samples from the Auckland site, two samples from Hawke's Bay site 1 and a single sample from the Wellington site. The griseofulvin metabolite 6-desmethyl griseofulvin was detected in all three of the samples from the Auckland site but no other samples. This was somewhat surprising as it was hypothesised that 6-desmethyl griseofulvin would be more prevalent than griseofulvin as approximately 84% of absorbed griseofulvin is excreted as this metabolite. However, similar to alternariol, alternariol monomethyl ether and roquefortine C,

significant loss of 6-desmethyl griseofulvin was observed during the method development phase of this project when tap water was used (Chappell & Li, 2024). As such, the apparently low presence of this metabolite in the wastewater samples analysed may be due degradation in-sewer prior to sampling. Recovery of griseofulvin from the spiked controls ranged from 65.7 – 99.5% versus 14.4 – 73.5% for 6-desmethyl griseofulvin, providing another possible explanation for the more frequent detection of griseofulvin compared to 6-desmethyl griseofulvin.

4. CONCLUSIONS

The results of this stage one pilot study have confirmed that it is possible to detect several different mycotoxins in municipal wastewater in New Zealand. Of the 11 target compounds, nine were detected in at least one wastewater sample, and in some samples up to five different mycotoxins were detected. At least one mycotoxin was detected in each of the analysed samples. Enniatin B was detected in all samples analysed and the closely related enniatin B1 was detected in samples from five of the six sampling locations. Analysis of recovery using spiked control samples indicated that most non-detections could not simply be explained by low recovery of the target mycotoxin from wastewater. It remains to be seen whether the lack of detection of a given mycotoxin at a specific site is due to non-exposure, or other factors such as extensive metabolism prior to excretion by exposed individuals and/or degradation in-sewer prior to sampling. Analysis of the stability of the target mycotoxins in wastewater using analytical standards confirmed that the majority are relatively stable at 4°C for up to seven days, which corresponds to the maximum length of time samples may be stored at the wastewater treatment plant prior to being shipped to ESR. Unfortunately, an apparent anomaly with samples stored at -20°C for 28 days which necessitated additional filtration meant the reliability of results for these samples was uncertain. As such, during the next phase of this project samples will be stored at 4°C with the aim to analyse these samples as quickly as possible, ideally within one month of collection.

APPENDIX

Table 4 Stability of the 11 target mycotoxins in wastewater at room temperature

Time point	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
	Initial spike concentration (ng/l)	**12.5**	**12.5**	**25**	**12.5**	**12.5**	**25**	**125**	**25**	**25**	**25**	**25**
T_0	Duplicate A	8.1	13.7	8.6	4.3	6.5	19.2	99.6	20.5	2.4	19.6	3.6
	Duplicate B	7.0	11.6	7.9	4.1	7.0	20.6	101.6	21.2	2.2	17.0	3.6
	Spike control (S)	7.8	13.6	8.2	4.3	5.8	19.0	113.0	20.8	2.6	21.6	3.9
	Recovery %	62	109	33	34	12	76	90	83	10	87	16
	Corrected conc. A (ng/l)	13.1	12.6	26.0	12.5	13.8	25.2	110.2	24.7	22.9	22.6	23.3
	Corrected conc. B (ng/l)	11.2	10.6	24.0	12.1	14.9	27.0	112.4	25.5	21.2	19.6	22.8
	Final conc. A (ng/l)	65.5	62.9	130.0	62.7	69.2	126.2	550.9	123.5	114.4	113.2	116.7
	Final conc. B (ng/l)	56.1	53.1	119.9	60.6	74.4	135.2	561.8	127.5	105.9	98.1	114.2
	Average final conc. (ng/l)	60.8	58.0	125.0	61.6	71.8	130.7	556.4	125.5	110.2	105.7	115.5
3 days	Duplicate A	7.7	14.0	8.3	3.9	6.1	18.3	94.6	20.4	1.9	15.7	1.8
	Duplicate B	7.3	13.0	8.7	4.2	5.6	16.6	104.5	20.6	1.8	14.1	2.0
	Spike control (S)	5.4	9.8	7.9	4.0	5.5	20.0	107.0	19.2	2.4	17.7	3.1
	Recovery %	43	78	32	32	44	80	86	77	10	71	12
	Corrected conc. A (ng/l)	17.7	17.8	26.1	12.2	13.8	23.0	110.6	26.5	19.6	22.2	14.2
	Corrected conc. B (ng/l)	16.9	16.7	27.5	13.0	12.6	20.8	122.0	26.8	18.3	19.9	15.8
	Final conc. A (ng/l)	88.7	89.2	130.4	61.1	68.9	114.8	552.8	132.6	97.8	110.8	71.1
	Final conc. B (ng/l)	84.3	83.4	137.5	64.8	63.2	104.0	610.2	134.2	91.5	99.4	78.9
	Average final conc. (ng/l)	86.5	86.3	133.9	62.9	66.1	109.4	581.5	133.4	94.6	105.1	75.0
7 days	Duplicate A	3.4	5.5	10.3	4.1	2.1	11.5	103.2	18.1	3.3	14.2	4.2
	Duplicate B	3.4	5.4	10.4	4.1	2.2	10.9	96.6	16.3	3.3	13.2	4.2
	Spike control (S)	5.3	9.9	17.2	5.9	3.6	21.0	96.5	17.5	4.1	16.2	5.3
	Recovery %	42	79	69	47	29	84	77	70	16	65	21
	Corrected conc. A (ng/l)	8.0	7.0	15.0	8.8	7.4	13.7	133.6	25.9	20.1	22.0	19.9
	Corrected conc. B (ng/l)	8.0	6.9	15.1	8.8	7.8	12.9	125.1	23.3	20.3	20.4	19.5
	Final conc. A (ng/l)	39.8	35.0	74.9	43.9	37.2	68.3	668.2	129.6	100.7	109.8	99.4
	Final conc. B (ng/l)	40.0	34.4	75.7	43.9	39.0	64.7	625.7	116.5	101.7	101.8	97.4
	Average final conc. (ng/l)	39.9	34.7	75.3	43.9	38.1	66.5	647.0	123.0	101.2	105.8	98.4

Table 5 Stability of the 11 target mycotoxins in wastewater after 3, 7 and 28 days at 4°C

Time point	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
	Initial spike concentration (ng/l)	**12.5**	**12.5**	**25**	**12.5**	**12.5**	**25**	**125**	**25**	**25**	**25**	**25**
3 days	Duplicate A	5.4	9.0	7.9	3.8	5.6	20.6	112.5	19.5	1.5	14.9	2.8
	Duplicate B	4.8	7.9	9.1	3.7	5.1	17.8	108.1	20.0	2.0	23.7	6.1
	Spike control (S)	5.5	9.0	8.3	4.3	5.5	19.1	101.8	21.1	2.9	21.7	4.3
	Recovery %	44	72	33	35	44	76	81	84	12	87	17
	Corrected conc. A (ng/l)	12.3	12.5	23.7	11.0	12.7	27.0	138.2	23.1	12.7	17.2	16.5
	Corrected conc. B (ng/l)	10.9	11.1	27.3	10.7	11.5	23.4	132.7	23.7	17.2	27.4	35.9
	Final conc. A (ng/l)	61.4	62.7	118.7	55.0	63.6	134.8	690.8	115.6	63.5	86.0	82.6
	Final conc. B (ng/l)	54.4	55.4	136.6	53.3	57.4	116.8	663.5	118.3	86.2	137.0	179.6
	Average final conc. (ng/l)	57.9	59.0	127.6	54.1	60.5	125.8	677.2	116.9	74.9	111.5	131.1
7 days	Duplicate A	5.9	11.0	11.3	4.4	4.8	18.3	102.8	18.5	1.6	17.1	5.0
	Duplicate B	5.9	11.2	10.0	4.3	4.8	18.1	97.2	19.3	1.7	19.3	5.1
	Spike control (S)	6.2	12.2	14.6	5.1	6.0	22.2	120.8	19.7	4.0	19.2	5.4
	Recovery %	50	98	58	41	48	89	97	79	16	77	22
	Corrected conc. A (ng/l)	11.8	11.2	19.3	10.8	10.0	20.7	106.4	23.4	10.0	22.2	23.0
	Corrected conc. B (ng/l)	11.9	11.5	17.2	10.6	10.0	20.4	100.6	24.5	10.8	25.1	23.3
	Final conc. A (ng/l)	58.8	56.1	96.5	54.0	50.1	103.3	531.8	116.9	49.9	111.1	115.2
	Final conc. B (ng/l)	59.4	57.5	85.9	52.8	50.2	102.1	502.9	122.3	54.1	125.6	116.5
	Average final conc. (ng/l)	59.1	56.8	91.2	53.4	50.1	102.7	517.3	119.6	52.0	118.3	115.9
28 days	Duplicate A	4.3	8.4	12.1	4.9	2.7	15.2	118.2	18.9	1.1	19.5	3.3
	Duplicate B	4.5	8.6	12.8	5.0	3.1	15.7	102.4	19.2	1.2	18.8	3.4
	Spike control (S)	4.3	8.3	12.1	5.0	3.6	20.8	123.7	19.0	2.2	23.5	4.1
	Recovery %	34	67	48	40	29	83	99	76	9	94	17
	Corrected conc. A (ng/l)	12.6	12.5	25.1	12.3	9.5	18.2	119.5	24.9	12.4	20.7	20.1
	Corrected conc. B (ng/l)	13.2	12.8	26.5	12.7	10.6	18.8	103.5	25.3	13.4	20.0	20.7
	Final conc. A (ng/l)	62.8	62.7	125.6	61.3	47.3	91.0	597.5	124.3	61.8	103.6	100.4
	Final conc. B (ng/l)	65.8	64.1	132.6	63.5	52.9	93.9	517.7	126.4	67.0	100.2	103.3
	Average final conc. (ng/l)	64.3	63.4	129.1	62.4	50.1	92.4	557.6	125.4	64.4	101.9	101.8

T_0 values are shown in Table 4.

Table 6 Stability of the 11 target mycotoxins in wastewater after 3, 7 and 28 days at -20°C

Time point	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
	Initial spike concentration (ng/l)	**12.5**	**12.5**	**25**	**12.5**	**12.5**	**25**	**125**	**25**	**25**	**25**	**25**
3 days	Duplicate A	6.2	10.1	7.5	3.4	4.6	17.7	117.5	21.8	1.8	14.9	2.4
	Duplicate B	4.7	7.5	8.4	4.1	4.0	17.8	113.3	22.5	1.8	16.9	2.7
	Spike control (S)	5.1	8.0	9.7	3.9	4.9	22.2	131.7	23.3	3.9	26.0	4.1
	Recovery %	41	64	39	31	39	89	105	93	15	104	16
	Corrected conc. A (ng/l)	15.2	15.9	19.4	10.8	11.8	19.9	111.5	23.3	11.4	14.3	14.8
	Corrected conc. B (ng/l)	11.5	11.7	21.5	13.0	10.3	20.1	107.5	24.1	11.4	16.2	16.2
	Final conc. A (ng/l)	75.8	79.6	96.9	54.2	58.9	99.6	557.6	116.7	57.2	71.6	73.8
	Final conc. B (ng/l)	57.4	58.6	107.4	64.9	51.6	100.4	537.5	120.7	57.0	81.2	81.2
	Average final conc. (ng/l)	66.6	69.1	102.2	59.6	55.2	100.0	547.6	118.7	57.1	76.4	77.5
7 days	Duplicate A	4.7	6.9	12.8	4.5	2.6	11.3	93.0	16.7	1.9	12.6	3.9
	Duplicate B	4.2	5.8	14.3	5.0	2.6	11.1	103.9	15.7	1.8	12.2	4.1
	Spike control (S)	4.1	5.7	17.5	6.5	3.6	19.7	93.1	16.0	5.0	17.7	5.9
	Recovery %	33	46	70	52	29	79	74	64	20	71	23
	Corrected conc. A (ng/l)	14.3	15.2	18.3	8.6	8.9	14.3	124.9	26.1	9.4	17.7	16.6
	Corrected conc. B (ng/l)	12.8	12.7	20.4	9.5	9.0	14.0	139.6	24.4	9.2	17.3	17.4
	Final conc. A (ng/l)	71.3	75.9	91.5	42.8	44.7	71.7	624.6	130.6	46.8	88.7	82.9
	Final conc. B (ng/l)	64.0	63.4	102.1	47.5	44.9	70.1	697.8	122.2	46.0	86.3	87.1
	Average final conc. (ng/l)	67.6	69.7	96.8	45.2	44.8	70.9	661.2	126.4	46.4	87.5	85.0
28 days	Duplicate A	2.3	3.7	8.8	3.6	1.3	5.1	97.2	16.6	0.4	7.3	1.3
	Duplicate B	2.4	3.9	8.6	3.6	1.2	4.7	96.7	15.8	0.3	7.3	1.3
	Spike control (S)	3.4	5.5	11.5	4.9	2.5	16.0	85.0	16.6	3.1	18.8	3.4
	Recovery %	27	44	46	39	20	64	68	66	12	75	14
	Corrected conc. A (ng/l)	8.4	8.4	19.2	9.2	6.4	7.9	142.9	25.0	3.0	9.8	9.6
	Corrected conc. B (ng/l)	8.7	8.9	18.7	9.2	6.1	7.4	142.2	23.7	2.6	9.8	9.8
	Final conc. A (ng/l)	41.9	42.2	95.9	45.9	32.0	39.7	714.7	124.8	14.8	48.9	48.0
	Final conc. B (ng/l)	43.5	44.3	93.5	46.0	30.5	37.0	711.0	118.6	13.2	48.8	48.8
	Average final conc. (ng/l)	42.7	43.3	94.7	46.0	31.2	38.3	712.9	121.7	14.0	48.9	48.4

T_0 values are shown in Table 4.

For the January and March 2025 samples, a single mixed wastewater sample was used for the spike control, as discussed in the methods section. As such, to avoid duplication, information on spike control (S) concentration and percentage recovery is omitted for the March samples.

Table 7 Results of wastewater LC-MS/MS analysis for the 11 target mycotoxins

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Northland	Oct 24	Duplicate A	ND	ND	0.32	0.15	ND	ND	0.72	0.27	ND	ND	ND
		Duplicate B	ND	ND	0.32	0.15	ND	ND	ND	0.22	ND	ND	ND
		Spike control (S)	1.03	1.59	1.18	0.88	0.78	5.37	22.25	5.15	0.64	4.25	2.76
		Recovery (%)	41.0	63.5	17.2	28.9	31.2	107.4	89.0	98.1	12.7	85.1	55.1
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.87	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**9.36**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	Jan 25	Duplicate A	ND	ND	0.60	0.20	ND	ND	ND	ND	ND	ND	ND
		Duplicate B	ND	ND	0.59	0.21	ND	ND	ND	ND	ND	ND	ND
		Spike control (S)	2.24	2.36	1.38	0.96	2.40	3.48	23.4	4.12	1.16	3.43	1.24
		Recovery (%)	89.6	94.3	27.7	38.4	95.9	69.6	93.5	82.3	23.2	68.6	24.8
		Corrected conc. (mg/l)	<LOQ	<LOQ	2.15	0.54	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**5.38**	**1.34**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	Mar 25	Duplicate A	ND	ND	0.79	0.23	ND	ND	ND	ND	ND	ND	ND
		Duplicate B	ND	ND	0.63	0.21	ND	ND	ND	ND	ND	ND	ND
		Corrected conc. (mg/l)	< LOQ	< LOQ	2.56	0.57	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**6.39**	**1.42**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	May 25	Duplicate A	ND	ND	0.64	ND	ND	0.24	ND	ND	ND	ND	ND
		Duplicate B	ND	ND	0.75	ND	ND	0.28	ND	ND	ND	ND	ND
		Spike control (S)	1.03	1.50	1.80	0.94	1.54	4.18	20.1	4.66	1.08	3.61	0.77
		Recovery (%)	41.2	60.1	36.0	37.5	61.5	83.6	80.4	93.1	21.7	72.3	15.3
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.93	< LOQ	< LOQ	0.31	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**4.82**	< LOQ	< LOQ	**0.78**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Auckland	Oct 24	Duplicate A	< LOQ	< LOQ	0.74	0.17	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	2.16	1.77
		Duplicate B	< LOQ	< LOQ	0.70	0.16	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	2.90	1.78
		Spike control (S)	0.94	1.48	1.79	0.82	0.50	5.14	27.19	5.14	0.47	6.13	4.44
		Recovery (%)	37.6	59.3	21.5	26.0	20.0	102.8	108.8	102.8	9.3	72.0	53.2
		Corrected conc. (mg/l)	< LOQ	< LOQ	3.35	0.64	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	3.51	3.35
		Final conc. (ng/l)	< LOQ	< LOQ	**16.74**	**3.19**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**17.54**	**16.74**
	Jan 25	Duplicate A	< LOQ	< LOQ	0.89	0.21	< LOQ	< LOQ	< LOQ	< LOQ	0.47	1.76	0.94
		Duplicate B	< LOQ	< LOQ	0.83	0.21	< LOQ	< LOQ	< LOQ	< LOQ	ND	1.87	1.02
		Spike control (S)	1.54	1.83	1.49	0.84	1.23	3.59	20.90	4.12	1.00	3.73	0.72
		Recovery (%)	61.7	73.2	29.8	33.8	49.3	71.7	83.6	82.4	20.0	74.6	14.4
		Corrected conc. (mg/l)	< LOQ	< LOQ	2.90	0.62	< LOQ	< LOQ	< LOQ	< LOQ	1.18	2.43	6.84
		Final conc. (ng/l)	< LOQ	< LOQ	**7.25**	**1.55**	< LOQ	< LOQ	< LOQ	< LOQ	**2.95**	**6.07**	**17.09**
	Mar 25	Duplicate A	< LOQ	< LOQ	1.24	0.23	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	1.73	0.74
		Duplicate B	< LOQ	< LOQ	0.75	0.21	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	1.77	0.66
		Corrected conc. (mg/l)	< LOQ	< LOQ	3.35	0.66	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	2.35	4.86
		Final conc. (ng/l)	< LOQ	< LOQ	**8.38**	**1.65**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**5.87**	**12.15**

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Hawke's Bay site 1	Oct 24	Duplicate A	< LOQ	< LOQ	0.59	0.18	< LOQ	0.01	< LOQ	0.09	0.22	1.06	< LOQ
		Duplicate B	< LOQ	< LOQ	0.53	0.18	< LOQ	< LOQ	< LOQ	0.11	0.21	0.79	< LOQ
		Spike control (S)	0.78	1.48	2.04	1.09	0.69	5.12	25.5	5.14	0.59	5.19	3.68
		Recovery (%)	31.4	59.1	29.4	36.3	27.7	102.4	102.2	102.8	7.4	85.3	73.5
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.83	0.49	< LOQ	< LOQ	< LOQ	< LOQ	2.94	1.08	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**9.16**	**2.47**	< LOQ	< LOQ	< LOQ	< LOQ	**14.72**	**5.42**	< LOQ
	Jan 25	Duplicate A	< LOQ	< LOQ	0.59	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.41	< LOQ
		Duplicate B	< LOQ	< LOQ	0.57	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.54	< LOQ
		Spike control (S)	2.26	2.65	2.13	1.25	2.72	4.18	19.20	4.19	1.49	3.98	1.50
		Recovery (%)	90.3	106.0	42.6	50.0	109.0	83.5	76.8	83.9	29.9	79.7	30.0
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.36	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.60	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**3.40**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**1.49**	< LOQ
	Mar 25	Duplicate A	< LOQ	< LOQ	0.85	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.78	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.92	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**4.80**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	May 25	Duplicate A	< LOQ	< LOQ	0.88	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.27	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.66	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.23	< LOQ	< LOQ
		Spike control (S)	0.83	1.32	1.65	0.57	1.16	4.01	17.88	4.49	0.57	3.28	0.74
		Recovery (%)	33.1	53.1	32.9	22.8	46.2	80.3	71.5	89.9	11.4	65.7	14.8
		Corrected conc. (mg/l)	< LOQ	< LOQ	2.34	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	2.19	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**5.85**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**5.46**	< LOQ	< LOQ

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Hawke's Bay site 2	Oct 24	Duplicate A	< LOQ	< LOQ	0.34	0.15	< LOQ	0.42	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.33	0.15	< LOQ	0.45	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Spike control (S)	0.90	1.43	1.67	0.69	0.76	5.64	19.90	5.18	0.43	4.62	2.69
		Recovery (%)	35.9	57.1	26.7	21.5	30.3	104.1	79.6	96.9	8.7	92.5	53.8
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.26	< LOQ	< LOQ	0.42	< LOQ	0.34	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**6.30**	< LOQ	< LOQ	**2.11**	< LOQ	**1.71**	< LOQ	< LOQ	< LOQ
	Jan 25	Duplicate A	< LOQ	< LOQ	0.65	< LOQ	< LOQ	< LOQ	< LOQ	0.62	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.66	< LOQ	< LOQ	< LOQ	< LOQ	0.65	< LOQ	< LOQ	< LOQ
		Spike control (S)	1.64	1.71	3.83	1.77	1.37	4.23	20.76	3.75	1.06	3.55	1.75
		Recovery (%)	65.8	68.4	76.6	70.6	54.7	84.5	83.1	74.9	21.2	71.1	35.0
		Corrected conc. (mg/l)	< LOQ	< LOQ	0.85	< LOQ	< LOQ	< LOQ	< LOQ	0.84	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**2.14**	< LOQ	< LOQ	< LOQ	< LOQ	**2.11**	< LOQ	< LOQ	< LOQ
	Mar 25	Duplicate A	< LOQ	< LOQ	0.57	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.56	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Corrected conc. (mg/l)	< LOQ	< LOQ	0.73	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**1.83**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	May 25	Duplicate A	< LOQ	0.41	0.54	0.20	0.51	< LOQ	< LOQ	< LOQ	0.51	< LOQ	< LOQ
		Duplicate B	< LOQ	0.42	0.57	0.20	0.53	< LOQ	< LOQ	< LOQ	0.53	< LOQ	< LOQ
		Spike control (S)	2.71	2.84	0.63	0.88	1.73	4.05	20.86	4.20	0.85	3.37	0.85
		Recovery (%)	108.4	113.6	12.7	35.0	69.2	80.9	83.5	84.0	17.0	67.4	17.0
		Corrected conc. (mg/l)	< LOQ	0.37	4.39	0.58	0.75	< LOQ	< LOQ	< LOQ	3.04	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	**0.91**	**10.9**	**1.45**	**1.88**	< LOQ	< LOQ	< LOQ	**7.61**	< LOQ	< LOQ

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Wellington	Oct 24	Duplicate A	< LOQ	< LOQ	0.51	0.17	< LOQ	< LOQ	< LOQ	0.09	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.49	0.17	< LOQ	< LOQ	< LOQ	0.15	< LOQ	< LOQ	< LOQ
		Spike control (S)	1.09	1.68	2.39	1.19	0.89	4.80	24.99	4.79	0.57	3.45	2.66
		Recovery (%)	43.6	49.7	37.8	40.7	35.6	96.0	100.0	93.3	11.4	69.1	53.2
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.32	0.41	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**6.60**	**2.07**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	Jan 25	Duplicate A	< LOQ	< LOQ	0.63	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	1.12	< LOQ
		Duplicate B	< LOQ	< LOQ	0.65	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	1.13	< LOQ
		Spike control (S)	1.55	1.73	2.90	1.23	1.31	4.16	22.17	4.06	1.45	3.69	1.67
		Recovery (%)	62.1	69.3	58.1	49.1	52.6	83.3	88.7	81.1	28.9	73.8	33.4
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.11	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	1.53	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**2.77**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**3.82**	< LOQ
	Mar 25	Duplicate A	< LOQ	< LOQ	1.19	0.23	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	1.10	0.24	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.97	0.48	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**4.93**	**1.19**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	May 25	Duplicate A	< LOQ	< LOQ	0.90	0.30	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.75	0.30	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Spike control (S)	1.04	1.80	2.95	1.87	2.22	4.20	18.86	4.89	1.80	4.97	1.79
		Recovery (%)	41.5	71.9	58.9	74.7	88.7	83.9	75.4	97.7	36.0	99.5	35.8
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.40	0.40	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**3.49**	**1.00**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ

Location	Date	Sample type	ENA	ENA1	ENB	ENB1	BEA	STE	AOH	AME	ROQC	GRI	6-DMG
Canterbury	Oct 24	Duplicate A	< LOQ	< LOQ	0.39	< LOQ	< LOQ	0.15	< LOQ	0.10	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	0.13	0.37	0.16	< LOQ	0.11	< LOQ	0.15	< LOQ	< LOQ	< LOQ
		Spike control (S)	1.02	1.53	1.74	1.07	0.66	5.84	27.81	5.18	0.55	4.84	3.32
		Recovery (%)	40.8	61.0	27.2	36.4	26.4	114.2	111.3	101.2	11.0	96.8	66.5
		Corrected conc. (mg/l)	< LOQ	0.27	1.40	0.44	< LOQ	0.11	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	**1.33**	**6.99**	**2.22**	< LOQ	**0.57**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	Jan 25	Duplicate A	< LOQ	< LOQ	0.54	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.50	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Spike control (S)	1.65	1.98	1.92	0.74	1.15	3.97	19.49	4.20	1.59	4.09	1.40
		Recovery (%)	66.1	79.3	38.3	29.4	46.0	79.5	78.0	84.0	31.8	81.9	28.0
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.35	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**3.38**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	Mar 25	Duplicate A	< LOQ	< LOQ	0.57	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.53	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Corrected conc. (mg/l)	< LOQ	< LOQ	1.44	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**3.60**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ
	May 25	Duplicate A	< LOQ	< LOQ	0.68	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.21	< LOQ	< LOQ
		Duplicate B	< LOQ	< LOQ	0.64	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.22	< LOQ	< LOQ
		Spike control (S)	1.72	2.01	4.05	1.84	2.71	3.24	18.74	5.09	1.61	4.77	1.44
		Recovery (%)	68.9	80.5	81.1	73.7	108.6	64.8	75.0	101.7	32.2	95.4	28.8
		Corrected conc. (mg/l)	< LOQ	< LOQ	0.81	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	0.66	< LOQ	< LOQ
		Final conc. (ng/l)	< LOQ	< LOQ	**2.03**	< LOQ	< LOQ	< LOQ	< LOQ	< LOQ	**1.66**	< LOQ	< LOQ

Red text indicates where the percentage recovery is < 50%.

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