**BEFORE THE INDEPENDENT COMMISSIONERS**

**IN THE MATTER** of the Resource Management Act 1991

**AND**

**IN THE MATTER** of the Proposed Waikato Regional Plan Change 1 - Waikato and Waipa River Catchments, and Variation 1 to proposed Plan Change 1

**AND**

**IN THE MATTER** of submissions under clause 6 First Schedule

**ON BEHALF OF** **BEEF + LAMB NEW ZEALAND**
**Submitter**

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# EXECUTIVE SUMMARY OF DR CHRISTOPHER A. DADA
## 26 MARCH 2019

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## INTRODUCTION

1. My full name is Christopher Ayokunle Dada.

2. I am I am an environmental health microbiologist, specializing in the fate, transport, detection, and control of pathogens in environmental media. I hold an MSc in Water Science, Policy and Management at Oxford University's Centre for the Environment. I completed a PhD in 2014 with a focus on the molecular characterization of faecal indicator bacteria and antibiotic resistant pathogens in aquatic environments. I have published extensively on public health aspects of faecal pollution in water. I have also been involved in several environmental effects assessment projects in New Zealand. This involved using a variety of catchment, hydrodynamic and empirical models to assess/predict the effect of past/future management decisions on water quality. I currently work as a Water Quality Specialist at Streamlined Environmental Limited.

3. I have been engaged by Beef + Lamb New Zealand to provide evidence on management responses in relation to land use and stock access to waterbodies with a particular focus on the effect of proposed fencing on *E.coli* freshwater outcomes and targets, for the hearing on Proposed Plan Change 1 for the Waikato and Waipa Rivers, and Variation 1 to this plan change (PC1).

4. I provided a Statement of Evidence in Chief on behalf of Beef + Lamb New Zealand dated 15 February 2019

5. I confirm the qualifications and experience set out in my Statement of Evidence in Chief.

6. As set out in my Evidence in Chief, I have read the Code of Conduct for Expert Witnesses in the Environment Court’s 2014 Practice Note and I have complied and continue to comply with it. I confirm that the opinions I have expressed represent my true and complete professional opinions. The matters addressed by my evidence are within my field of professional expertise. I have not omitted to consider material facts known to me that might alter or detract from the opinions expressed.

## EXECUTIVE SUMMARY

7. This executive summary provides comments on:

    a) assumptions used in the *E.coli* models underpinning the WRPC1;

    b) issues with monitoring waterborne pathogens in the WRPC1;

    c) sources, fate and transmission pathways of microbial contamination from primary productive land into receiving water; and,

    d) the effectiveness of fencing small waterbodies to reduce catchment microbial loads, based on a review of literature specific to the Waikato Region.

8. PC1 includes *E.coli* freshwater outcomes in Table 3.11-1, which are derived from an attempt to define a parameter and set of numerical outcomes which meet objective (k) of the Vision and Strategy.

9. The NPSFWM also requires that regional councils set instream *E.coli* attribute states to provide for contact recreation. These attribute states vary from region to region depending on level of protection required i.e. primary contact recreation versus secondary contact recreation.

10. In relation to providing for human health for primary contact recreation the focus is on managing the risk of zoonotic pathogens such as campylobacteria. As these parameters are difficult to measure in freshwater on a routine basis, a proxy for risk is provided in the NPSFWM in the form of an *E.coli* attribute. However, it is widely recognised that *E.coli* provides a very poor indicator of pathogenic risk and so should be used with caution.

11. *E.coli* is not correlated with pathogenic risk for these key reasons:

    a) Zoonotic pathogens from primary productive land are not reliably detected using the *E.coli* proxy. This is because there is often no correlation between concentrations of *E.coli* and zoonotic pathogens that they are meant to 'protect against'[1].

[1] National Research Council (US) Committee on Indicators for Waterborne Pathogens. Indicators for Waterborne Pathogens. Washington (DC): National Academies Press (US); 2004. 4, Attributes and Application of Indicators.

b) Also, not all *E.coli* are from faecal sources[2]. Non-fecal environmental sources of *E.coli* (e.g. decaying plants, algae and biofilms, indigenous strains in sands and soils) tends to confound our ability to predict the fate of pathogens in animal waste management systems both on and off farms. Besides, the potential for these non-environmental strains to predict human health effects has not been demonstrated in literature [3].

c) FIB can naturally survive and proliferate outside of animal intestines, in tropical and temperate habitats. That is, the quantity of *E.coli* is not necessarily correlated with increasing risk of infection[4]. The processes that control the survival and removal of microbes in water, such as competition, ultraviolet radiation, temperature, predation, and transport differ among pathogenic species. Thus, monitoring FIB alone is not sufficient to assess human health risk.

12. Because of these limitations with the *E.coli* as an indicator, in the context of PC1 the application of stringent *E.coli* outcomes are:

a) impossible to achieve; and

b) unnecessary in relation to providing for human safety in relation to swimming;

13. The modelling that underpins the PC1 decision making failed to include key factors that influence variabilities in *E.coli* levels in primary productive land and receiving streams (e.g. in-stream attenuation). Furthermore, formula and coefficients applied in the model were not explicitly stated, thus preventing independent verification of inputs and outputs of the model. This is important because modellers 'optimise' these coefficients/functions to

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[2] Ferguson, D. (2006). Growth of E. coli and Enterococcus in Storm Drain Biofilm. Presentation at 2006 U.S. EPA National Beaches Conference.

Ksoll, W.B., Ishii, S., Sadowsky, M.J., Hicks, R.E. 2007. Presence and Sources of Fecal Coliform Bacteria in Epilithic Periphyton Communities of Lake Superior. Applied and Environmental Microbiology 73: 3771-3778.

Yan, T., Goto, D.K., Feng, F. 2011. Concentration dynamics of fecal indicators in Hawaii's coastal and inland sand, soil, and water during rainfall events. PATH6R09. Water Environment Research Foundation, Alexandria, VA.

[3] EPA (2014) Overview of Technical Support Materials: A Guide to the Site-Specific Alternative Recreational Criteria TSM Documents. EPA-820-R-14-010 U.S. Environmental Protection Agency Office of Water Office of Science and Technology Health and Ecological Criteria Division

[4] National Research Council (US) Committee on Indicators for Waterborne Pathogens. Indicators for Waterborne Pathogens. Washington (DC): National Academies Press (US); 2004. 4, Attributes and Application of Indicators.

best make the data fit and the failure to disclose this information means that the model cannot be independently verified. Also, the *E.coli* models that informed the decision making process in the PC1 were not tested with new measured data not originally included during the model development, a standard process in model validation.

14. The approach taken in PC1 to monitoring *E.coli* levels as a proxy for the presence of zoonotic pathogens does not distinguish between concentrations during different flow conditions (Figure 1). PC1 uses the 95th percentile sample results from the previous 5 years as an indicator of an overall achievement of the *E.coli* target in Table 3.11-1. A conservative threshold set at 540 colony forming units (CFU)/100mL 95$^{th}$ percentile concentration, regardless of the season, is overly constraining in relation to providing for human health for primary contact recreation such as swimming and may be unachievable. It over estimates health risks associated with exposure to pathogens, particularly during non-swimming periods when the FIB population are largely driven by periods of high flow.

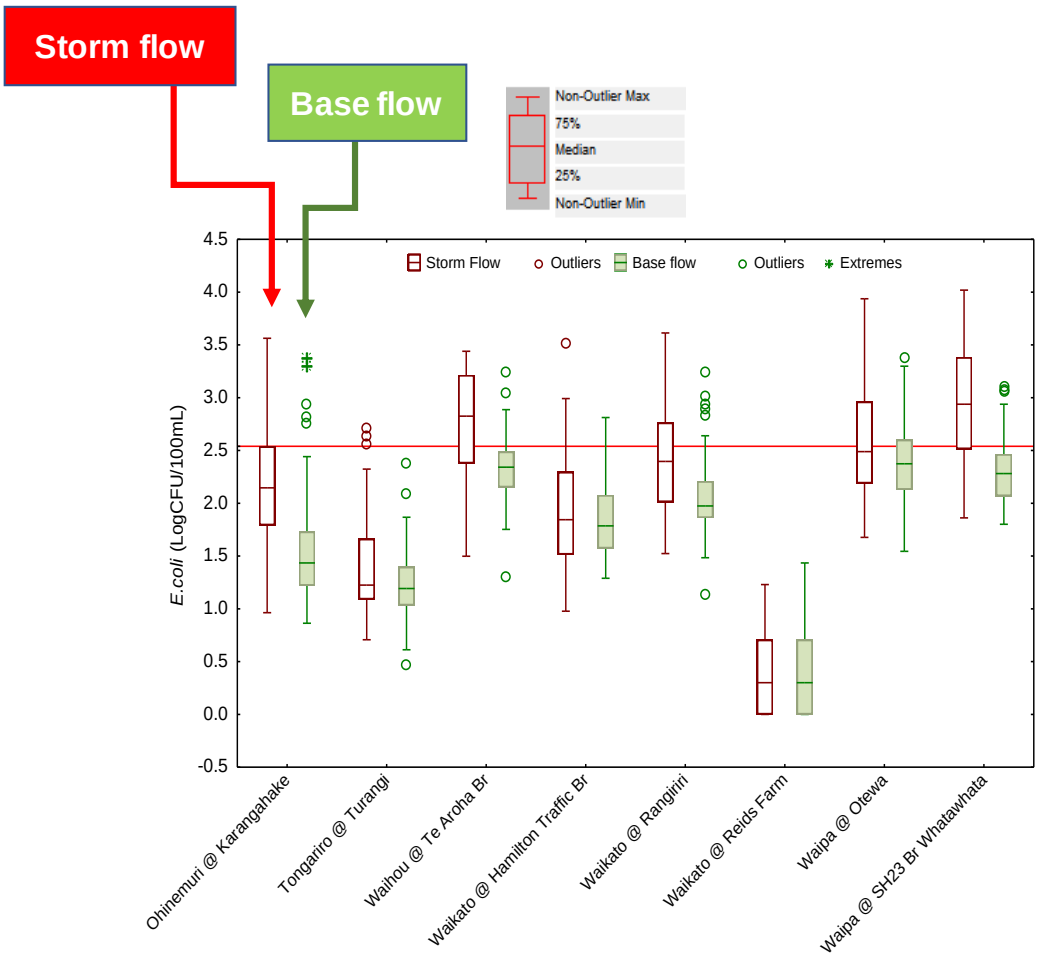

Figure 1. Box plots of *E.coli* concentrations during baseflow and storm flow conditions, Waikato Region waterways, 2007-2013. Red horizontal line is the 540 CFU/100mL *E.coli* threshold

15. My expert position is that instream *E.coli* concentrations, where they are set, should take into account different flow conditions in relation to providing for primary contact recreation. Considerations for flow conditions warrant the establishment of a stringent maximum limit for *E.coli* during the "swimming season" (typically during base and low flows (i.e. flows below medium flow) and a less stringent limit for all other times (storm flows). Based on these conclusions, I recommend that:

    a) The *E.coli freshwater outcomes* be revised, and amended as follows[5]:

        (i) When flow is < 50th percentile (when the river is below or at medium flow), *E.coli* concentration must not exceed 260/100ml

        (ii) When flow is < 75th percentile (when the river is less than the top 25% of flow), *E.coli* concentration must not exceed 540/100ml all year round.

        (iii) When flow is > 75th percentile (when the river is greater than the top 25% of flow), *E.coli* concentration must not exceed 1000/100ml all year round.

    b) While option (a) is my preferred approach, an alternative approach could be to amend the Table 3.11-1 *E.coli* targets in line with the National Policy Statement for Freshwater Management (NPS-FM) *E.coli* Attribute State thresholds. This approach complies with the NPS-FM requirements, and it will help authorities work with more realistic short-term

[5] This is similar to the approach adopted by Horizons Regional Council in their One Plan

outcomes. It also makes monitoring and reporting of progress seamless.

c) Using the 2017 NPS approach, all four or at least two of the four numeric attribute statistics for *E.coli* in the NPS-FM 2017 guidance document should be applied. For instance, the short-term targets could be a combination of median and 95$^{th}$ percentile *E.coli* concentrations rather than a reliance on the single 95$^{th}$ percentile as it is currently in the PC1 Table 3.11-1. Improvement in water quality can then be tied to a movement up the 2017 attribute state to the next higher state (e.g. Yellow to Green, as stated in my evidence in Chief, Appendix 1).

d) Using the 2014 NPS approach, the current river state could be judged as Best (A), Good (B), Poor (C) and Very Poor (D) based on 5-year median concentrations of <260, <540, <1000 and >1000 CFU/100mL,respectively. Improvement in bacteriological water quality could then be tied to a movement up the attribute state to the next higher state. For example, if current 5-year median concentration of River X is currently less than 980 CFU/100mL, the river is deemed to have a current state of Poor (i.e. C). A short-term future target of 540 CFU/100mL should be applied (i.e. the river is expected to have moved from State C to State B, in the short-term).

e) Given the lack of relationship between *E.coli* and pathogenic risk I propose that the long-term *E.coli* outcomes be deleted. Meanwhile, I am aware that the NPSFWM *E.coli* attributes are currently under review, and amendments to the NPSFWM in relation to an appropriate indicator for pathogenic risk is due in 2020, or 2021.

16. An important issue for PC1 is the source of faecal pollution at the sites for which *E.coli* reduction targets are set. Currently, it is not known for certain what the sources of faecal pollution are for these streams and rivers, yet declarations have been made to drastically reduce *E.coli* levels (nearly

100% anticipated *E.coli* reduction for some streams with as high as 12,000 CFU/100mL). Only when we cross over the first milestone of reliably identifying sources responsible for elevated bacteria levels at each site, can we begin to identify an appropriate solution that will drive down observed elevations in *E.coli* levels, rather than a mere declaration of anticipated reduction targets without the means of achieving it.

17. In hilly or steep lands in New Zealand and in flat, poorly drained land in the greater Waikato region, high runoff potential under high rainfall is largely associated with overland transport into receiving streams (Figure 2). A review of published studies indicate that direct deposition is a minor percentage of total annual catchment *E.coli* loads to waterways in the Waikato Region, and that surface runoff is the major source of faecal pollution from agriculture in the Waikato Region (as seen in Figure 3). It is logical that if the streambank fencing is erected for reducing animal access and delivery of *E. coli* to water ways, there could still be elevated *E.coli* levels in PC1 streams that run through agricultural catchments. Rather than a 'blanket fencing approach' currently proposed in the WRPC1, a more effective response to reduce the risk of pathogens from agricultural land uses entering waterbodies is the identification and management of critical source areas.

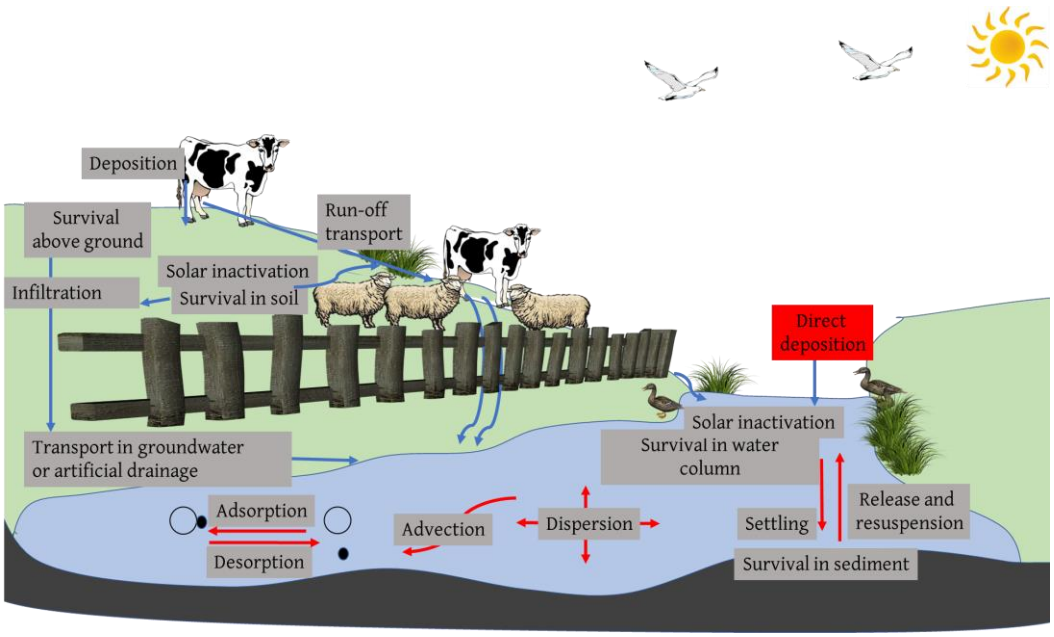

Figure 2: Fate of microbial pathogens (on-land and in-stream) showing that overland flow of pathogens evade stream fencing in pastoral catchments. Position of animals in the image are imaginary.

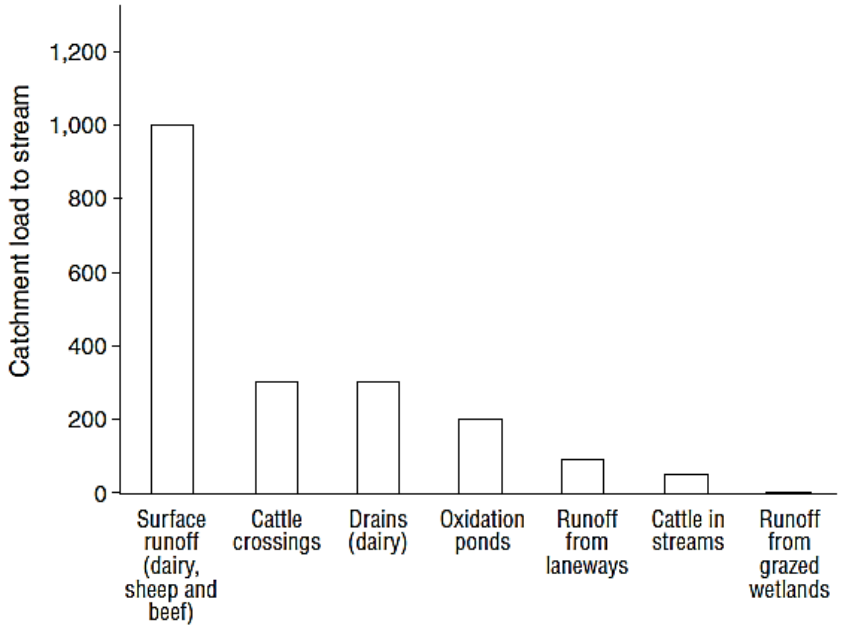


Figure 3: Waterway loadings of *E.coli* (CFU x 108/ha./pasture/year for major sources of faecal matter in the Waikato Region, New Zealand. Source: McDowell and Wilcock 2008[6])

18. Site-specific management options informed by microbial source tracking (MST) studies at each PC1 site can help determine the contributory source of faecal pollution, and hence support mitigation efforts for the PC1 streams. Without these MST studies, I am of the opinion that the targets related to *E.coli* reductions at the freshwater sites listed in PC1 are ambitious, unrealistic, and unecessary, and they present a cart 'before the horse' approach. We need to begin to ask the hard questions. Are elevated bacteria due to direct deposition of farm animals? If yes, which animals are largely responsible for these faecal droppings? At this stage, without the

[6] McDowell, R.W and Wilcock, R.J. (2008) Water quality and the effects of different pastoral animals. New Zealand Veterinary Journal 56(6): 289-296

MST studies, it is difficut to apply a generic management option to tackle *E.coli* loads at the PC1 sites.

19. Currently, the MST approach has only been applied to 5 out of the 62 WRPC1 sites. Even then, preliminary MST results show that wildfowl is the predominant source of faecal indicator bacteria in the WRPC1 streams and that cattle markers only become prevalent following heavy rainfall impacted (i.e. surface run-off and overland) conditions (see Table 1).

Table 1: ESR *E. coli* and faecal source tracking results for Karapiro, Komakorau, Mangaone, Mangaonua and Mangawhero Streams (adapted from Moriarty, 2015[7])

| Discharge condition | Faecal Pollution Source | No. of samples positive for marker | Total No. of observations | Prevalence (%) |
|---|---|---|---|---|
| Low flow | Wildfowl | 11 | 14 | 78.6 |
| Low flow | Cattle | 6 | 14 | 42.9 |
| Rainfall-impacted | Wildfowl | 15 | 15 | 100 |
| Rainfall-impacted | Cattle | 11 | 15 | 73.3 |

20. While further work is undertaken to improve our understanding of the sources of in-stream *E.coli* concentrations in the PC1 sites, I recommend that authorities:

a) Delete requirements to fence hill country streams, considering that it is a counter-intuitive approach to stopping overland flow.

b) Increase requirements to identify and manage critical source areas and overland flow pathways. This will then lead to catchment-

[7] Moriarty, E (2015) Sources of Faecal pollution in Selected Waikato Rivers - July 2015. Report commissioned by Dairy NZ. Report No. HR/TLG/2015-2016/7.3

specific management intervention rather than a blanket approach to effect fences for stock exclusion which only stops direct deposition.

c) Commission longitudinal site-specific MST studies targeted for each identified site in the WRPC1 Table 3.11.1. The study should also incorporate phylogenetic dimensions that are able to distinguish if these elevated bacteria levels in each WRPC1 site are due to naturalized *E.coli* from the stream bed and channel sediments. "Naturalized" *E. coli* populations falsely inflate measured *E.coli* levels, leading to exceedances of available thresholds, this incorrectly suggesting that pollution is present.

d) Amend Table 3.11-1 E.coli freshwater outcomes aligned with my para 14.

**DATED** this 26 day of March 2019

Christopher A. Dada