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 Waipā River Catchments and Variation 1 to proposed Plan Change 1

AND

IN THE MATTER of submissions under clause 6 First Schedule

BY **BEEF + LAMB NEW ZEALAND LIMITED**
Submitter

BRIEF OF EVIDENCE OF DR CHRISTOPHER AYOKUNLE DADA

15 February 2019

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BACKGROUND

QUALIFICATIONS AND EXPERIENCE

1. My full name is Christopher Ayokunle Dada.

2. I am an environmental health microbiologist, specializing in the fate, transport, detection, and control of pathogens in environmental media.

3. I hold a BSc honours degree (First Class) in Microbiology from the University of Ado-Ekiti. I also completed an MSc in Water Science, Policy and Management at Oxford University’s Centre for the Environment which adequately equipped me to provide high-level advisorial support to decision makers, managers and policy makers in water policy and management. My PhD research focused on the molecular characterization of faecal indicator bacteria and antibiotic resistant pathogens in aquatic environments.

4. I have published extensively on public health aspects of faecal pollution in water (co-authored 38 peer-reviewed scientific publications, 10 as lead author and 26 in international journals). I am still actively engaged in research, especially around the environmental fate and effects of microbial contaminants in New Zealand.

5. I have also been involved in 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.

SCOPE OF EVIDENCE

6. I have been requested by Beef + Lamb New Zealand to provide expert evidence on the fate and transport of faecal indicator bacteria (FIB) and pathogens from pastures to receiving waters relevant to the proposed Waikato Regional Council proposed Plan Change 1 and variation 1 (henceforth PC1) for the Waikato and Waipa River Catchments. This analysis is undertaken as numerical *E.coli* freshwater outcomes and targets are provided for in WRPC1 through table 3.11-1, along with associated management responses in relation to land use and stock access to waterbodies. My evidence is structured under the following headings:

(a) An overview on the sources, fate and transmission pathways of microbial contamination from primary productive land into receiving water,

(b) Zoonotic diseases of concern from primary productive land uses conveyed through freshwater,

(c) Issues with monitoring waterborne pathogens in New Zealand,

(d) A summary of regionally relevant studies and comments on E coli reduction approaches/targets (including Table 3.11-1) and concerns specifically related to the assumptions used in the adopted *E.coli* models. This section also includes an analysis of *E.coli* data for streams in the PC1 catchment to identify the occurrences of peaks in FIB concentrations, during actual baseflow and stormflow conditions, and,

(e) An assessment on the proposed rules that require cattle, deer and pigs to be excluded from all permanently flowing waterbodies up to a land slope of 25 degrees. This section also includes an assessment on the effectiveness of fencing small waterbodies to reduce catchment microbial loads, which is supported by an analysis of relationships between *E.coli* and stream order using monitoring data and review of other regionally relevant studies.

EXPERT WITNESS CODE OF CONDUCT

7. I have read the Code of Conduct for Expert Witnesses in the Environment Court Practice Note 2014. This evidence has been prepared in accordance with it and I agree 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.

REPORTS USED IN PREPARING THIS EVIDENCE

8. In preparing this evidence I have reviewed the reports and statements of evidence of other experts including:

(a) Officers section 32 report;

(b) Officers section 42A report;

(c) Expert evidence of Mr Andrew Burtt;

(d) Expert evidence of Dr Jane Chrystal;

(e) Expert evidence of Mr Richard Parkes;

(f) Expert evidence of Dr Cox.

EXECUTIVE SUMMARY

9. 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. 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 best make the data fit and the failure to disclose this information means that the model on which the PC1 decision making was based cannot be independently verified to be trustworthy. 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. These uncertainties coupled with other reasons previously stated seem to render the model unfit to inform or underpin PC1.

10. 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. The 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. This evidence notes that 95th percentile *E. coli* concentrations are rare events that are associated with storm flows and will only reflect in 5% of the observed data used to make this judgement. In simple terms, only 5% of the monitoring data will be higher than the 95th percentile concentration, regardless of the number of "previous years" of data considered. A conservative threshold set at 540 colony forming units (CFU)/100mL 95th percentile concentration, regardless of the season may mean that health risks associated with exposure to pathogens are over-estimated, particularly during non-swimming periods when the FIB population are largely driven by periods of

high flow. Considerations for flow conditions may warrant the establishment of a stringent maximum limit for faecal coliform bacteria per 100mL sample during the "swimming season" (typically during base and low flows) and a less stringent limit for all other times (storm flows). Based on these conclusions , I recommend that:

(a) The *E.coli* targets need to be revised and the policy wording should be amended to read 'the *E.coli* concentration of the water must not exceed (table 3.11-1 revised numerical parameter given in CFU/100mL) when the river is at or below medium flow (the 50th percentile flow).

(b) If it is impossible to designate revised Table 3.11-1 *E.coli* targets in line with recommendation (a) above, then the *E.coli* targets should be amended to comply with the National Policy Statement for Freshwater Management (NPS-FM) *E.coli* Attribute State thresholds. Using this approach, an indicator of improvement in bacteriological water quality could be tied to at least two of the four numeric attribute statistics in the NPS-FM guidance document. For instance, this could be a combination of median and 95th percentile *E.coli* concentrations to infer improvement in NPS-FM Attribute States rather than a reliance on the single 95th percentile as it is currently in the PC1 Table 3.11-1. A table of suggested targets is also presented in this evidence. This approach will help authorities work with more realistic short-term targets hinged on improvements in the NPS-FM attribute state of the PC1 sites.

11. A key issue for the PC1 with respect to *E.coli* is the source of faecal pollution at the PC1 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 to certain levels (up to 2000% anticipated reduction for some streams). 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. 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. 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. 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.

12. Apart from critical source areas, 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, from a technical (microbiological) perspective, 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? While for some sites, it may be unreasonable to commit financial resources to erecting wired fences when the cause of elevated *E.coli* levels is mainly as a result of wildlife faecal deposits during low flows and overland flow during wet events, for some other sites, erecting barriers to prevent direct access to animals during low flows may actually be needed. At this stage, without the MST studies, it is difficut to apply a generic management option to tackle *E.coli* loads at the PC1 sites.

13. 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. Based on these arguments, I therefore 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-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 and suggesting pollution that is present.

14. While further work is undertaken to improve our understanding of the sources of in-stream *E.coli* concentrations in the PC1 sites, authorities can adopt tentative yet cautious approach that includes consideration for flow conditions since surface runoff is the major source of faecal pollution from agriculture in the Waikato Region (as in recommendations (a) and (b) above).

SOURCES, FATE AND TRANSMISSION PATHWAYS OF MICROBIAL CONTAMINATION FROM LAND USED FOR PRIMARY PRODUCTION PURPOSE

15. In the context of this evidence, and in line with international literature, land used for primary production purposes refer to land used for one or more of the following activities:

(a) Cultivating crops for the purposes of selling the produce, including in a processed or converted state.

(b) Cultivating or propagating plants, seedlings, vegetables, mushrooms or orchids for sale.

(c) Maintaining animals or poultry for the purposes of selling them, their offspring or bodily produce (e.g. beef and sheep farming and dairying)

16. I agree with previously published literature[1] that one of the most important issues related to primary productive land use is the impact and interdependence of this form of land use on water resources. Agricultural production requires a stable supply of fresh, clean water for stock watering, irrigation of crops and pasture, as well as for other aspects of the farming operation. Farm 'runoff' includes contaminants from farm operations, and intensive operations usually also produce a stream of spent water and solid waste that can potentially affect the quality of receiving waters.

17. The impact of primary productive land use on water quality can be observed at differing temporal and spatial scales. For instance, these could range from the presence of individual stock at a stock crossing or an unprotected stretch of a waterway, to impact of improper manure management at the farm scale, to whole catchment effects of land management practices (for example massive catchment-wide changes in production systems).

18. Sources of faecal contamination from primary productive land include humans, livestock and wild animals, with pathogens being excreted in the faeces and occasionally urine. Although human faecal wastes present the highest risk of waterborne disease, given that the probability of human pathogens (disease-causing microorganisms) being present is highest, waste from this source is almost always adequately disposed of through some form of on-site or reticulated treatment system. Notwithstanding this treatment of human wastes, direct discharge of post-treatment effluent into

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[1] Davies-Colley, R. (2003). Effects of rural land use on water quality. Ministry for the Environment.

FAO(1993) Water Resource Issues and Agriculture. In The State of Food and Agriculture , FAO Agriculture Series No. 26, ISSN 0081-4539

MfE (2004) Water Programme of Action: The Effects of Rural Land Use on Water Quality . Ministry for the Environment Technical Working Paper, July 2004. ME number: 563

PCE (2013) Water quality in New Zealand: Land use and nutrient pollution. Parliamentary Commissioner for the Environment report, November 2013.

receiving waters, or poorly-maintained septic tank systems can also be major sources of faecal loadings into receiving waters.

19. Per capita, faecal production by agricultural animals such as cattle and pigs exceeds that of humans. Hence, other than human waste, pastoral agriculture is the other major source of FIB in aquatic systems. Also, land use plays an important role in the inoculation, persistence, and dissemination of FIB. Faecal bacteria from primary productive lands can enter the stream network via direct deposition of faecal matter into the stream or via indirect pathways such as discharges of dairy effluent into streams, drainage via artificial drains, surface wash-off in areas of steep terrain, as well as from overland flow from excess irrigation water and water-logged conditions.

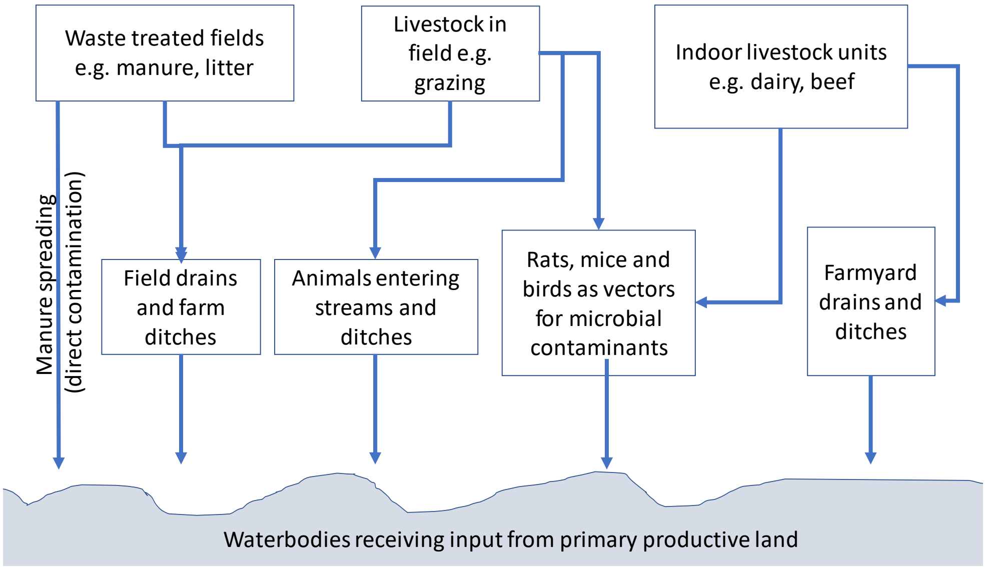

Figure 1: Sources and pathways of microbial water contamination from primary productive lands.

20. Of particular interest to this evidence are microorganisms associated with livestock (i.e. animal waste and animal manure) that are deposited on land, and on or near water bodies. The animal faecal wastes and other wastes (such as respiratory secretions and urine) of livestock and feral animals often contain high concentrations of pathogens. This include a variety of viruses such as hepatitis E virus, bacteria such as Salmonella species, and parasites such as Cryptosporidium parvum. A number of these pathogens

are endemic in commercial livestock and difficult to eradicate, and pose potential risks to human when transmitted to the wider environment

21. I agree with previous studies (e.g Doole, 2016[2], Romera and Doole, 2015[3]) that the amount of faeces deposited by livestock over the last 20 years has very likely increased given that stocking rates have increased. For example, the average national stocking rate for dairy cows increased by 18% from 2.44 cows ha-1 to 2.87 cows ha-1 over 1994–2014. It should be noted however that while this stocking rate versus faecal loading may logically appear positively correlated, the relationship between these is often cofounded by many other variables and uncertainties, as will be discussed later in this evidence.

22. Concentrations of some pathogens occur at levels of millions to billions per gram of wet weight faeces or millions per ml of urine. For instance, cattle manure contains up to 109 colony forming units (CFU) of indigenous bacteria g-1. Among this population of heterogenous bacteria, faecal indicator bacteria (Escherichia coli and enterococci) constitute up to 10^5 to 10^7 CFU g^{-1} of cattle manure. Proportions of other specific pathogens have been documented. For instance, faecal material from a cattle herd that has been colonized by Salmonella could contains up to 102 to 107 CFU g^{-1} of this pathogen[4]. In other studies[5], up to 2.6 × 107 oocysts g^{-1} of protozoans such as Cryptosporidium and Giardia have been documented in cattle excreta.

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[2] Doole (2016) Evaluation of scenarios for water-quality improvement in the Waikato and Waipa River catchments. Business-as-usual assessment 20 October 2016.

[3] Romera, A. J., & Doole, G. J. (2015). Optimising the interrelationships between intake per cow and intake per hectare. Animal Production Science, 55, 384-396.

[4] Himathongkham, S., Bahari, S., Riemann, H., and Cliver, D. (1999). Survival of Escherichia coli O157: H7 and Salmonella typhimurium in cow manure and cow manure slurry. FEMS Microbiol. Lett. 178, 251–257

[5] Medema, GJ; Shaw, S; Waite, M; Snozzi, M; Morreau, A; Grabow, W. Catchment characteristics and source water quality. In Assessing Microbial Safety of Drinking Water Improving Approaches and Method; WHO & OECD, IWA Publishing: London, UK, 2003; pp. 111–158.

Bradford, S. A., and J. Schijven. 2002. Release of Cryptosporidium and Giardia from dairy calf manure: impact of solution salinity. Environ. Sci. Technol. 36:3916-3923.

23. An understanding of the diverse fate and transport behaviour of faecal borne microorganism is critical for public health risk assessment and management. Following the discharge of faecal waste from animal sources, a number of factors and processes determine the fate (survival, growth, transmissibility, etc) of the pathogens in the excreta in its new environment. These pathways are broadly divided into two: (a) In-land processes and (b) In-stream processes.

(a) In-land processes:

  i. Processes that influence the faecal transmission pathway within the terrestrial environment of primary productive land and determine faecal content loadings that reach receiving waters. A general conceptual representation of pathways of transmission of faecally-associated microorganisms generated in primary productive land use is presented in Figure 2.

  ii. On agricultural productive land, risks potentially associated with the livestock faecal waste will depend on a number of factors, such as: (1) Composition (manure bulk density, aggregation, porosity, and water contents), (2) age and treatment of the manure, (3) characteristics of the faecal microbes[6], as well as (4) the degree of specific microbial association within the manure/soil matrix.

  iii. Risks potential associated with the livestock faecal waste also depends on climatic factors such as intensity and frequency of precipitation and ultraviolet radiation. For instance, rainfall energy and duration affect the release of microbes from manure and soil, higher intensities of rainfall increase levels of microbial release from manure on a farm. The increased water content in manure between rainfall events one and two may also promote bacterial survival and

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[6] e.g. differing specific physical and chemical properties (size, hydrophobicity, secrete extracellular polymeric materials, and possession of surface structures) of the faecal microorganisms that affect their propensity to dislodge from their microhabitats or surrounding soil layer, preferential attachment of different strains to soil particles of different size fractions (i.e., sand, silt, and clay)

growth and thus increased levels in run off. Conversely, faecal bacteria released from animal waste into surface runoff can also decrease during consecutive rainfall events, particularly when manure-borne bacteria is bound to soil particles below the manure deposition zone, or in gaps between manure so they are less susceptible to runoff removal in a subsequent rainfall event.

iv. Another important factor is land use, cover, and soil type. Differences in soil type and vegetative covers affects the transport of E. coli during rainfall events. Vegetation may also reduce microbial release by providing a canopy to reduce raindrop impact, thus protecting manure microhabitats from dispersion through overland flow during rainfall events. The export of matter from soils to an adjacent aquatic ecosystem is also partly controlled by the slope angle and the concentration of organic matter in the soils. For instance, in sloping lands, the export of organic matter and bacteria can be particularly high, presenting important implications for downstream aquatic ecosystems. Also, in intensive farming areas where manure production is high, there is a greater possibility of faecal contamination, particularly if these production zones are near to streams and rivers or if the animals have direct access to the stream. Land-use associated factors such as the stocking density of grazing animals, presence/absence of infected animals that carry zoonotic pathogens, the stage and severity of infection, the species and numbers of pathogens carried by the animals and shedding rates, the extent of direct access to the stream and/or its tributaries and the potential for live bacteria in cowpats and soil to be transported from ‘contributing areas’ into the stream.

(b) In-stream processes

i. In-stream processes are those processes that drive variabilities in levels of faecal bacteria in water bodies receiving input from primary productive lands. These relate to bacterial survival and transport in the water column, settling into sediments, survival in streambed sediments, release and resuspension into the water column, advection, and dispersion. Factors specifically related to the survival and persistence of faecal microbes include temperature and extent of sunlight inactivation. The presence of other bacteria viruses and predators, metabolic capacity, and associations with particles and other non-host organisms, all influence the decay (or loss) rates of microbes within water bodies.

ii. Most enteric pathogens have no means of transport (such as motility) in the aquatic environment other than being transported with the water flow. Hence, a critical factor which drives the occurrence and persistence of bacteria in the aquatic environment is frequency and intensity of storm events and inter-storm flow periods. The relative amount of groundwater[7] also exerts an influence on the magnitude of dilution effect of bacteria loads during floods, contrary to the situation in overland flows which strongly contributes to soil erosion and hence, bacteria erosion processes. Generally, inflows dominated by overland flow will contain elevated loads of suspended particles and bacteria. Once delivered to the river, sediment and bacteria can then accumulate on riverbeds before being re-suspended after an increase in river discharge. Highly erosive rain results in the resuspension of particles as a function of flow, thus leading to resuspension of several orders of magnitude of bacteria into the water column.

iii. Our knowledge of factors affecting fate and transport of pathogens in receiving waters stems largely from studies using FIB as the target organism(s). It is clear from these studies that stream sediments play an important role as reservoirs of microbes. My view is that better understanding of variabilities in FIB levels observable in water monitoring

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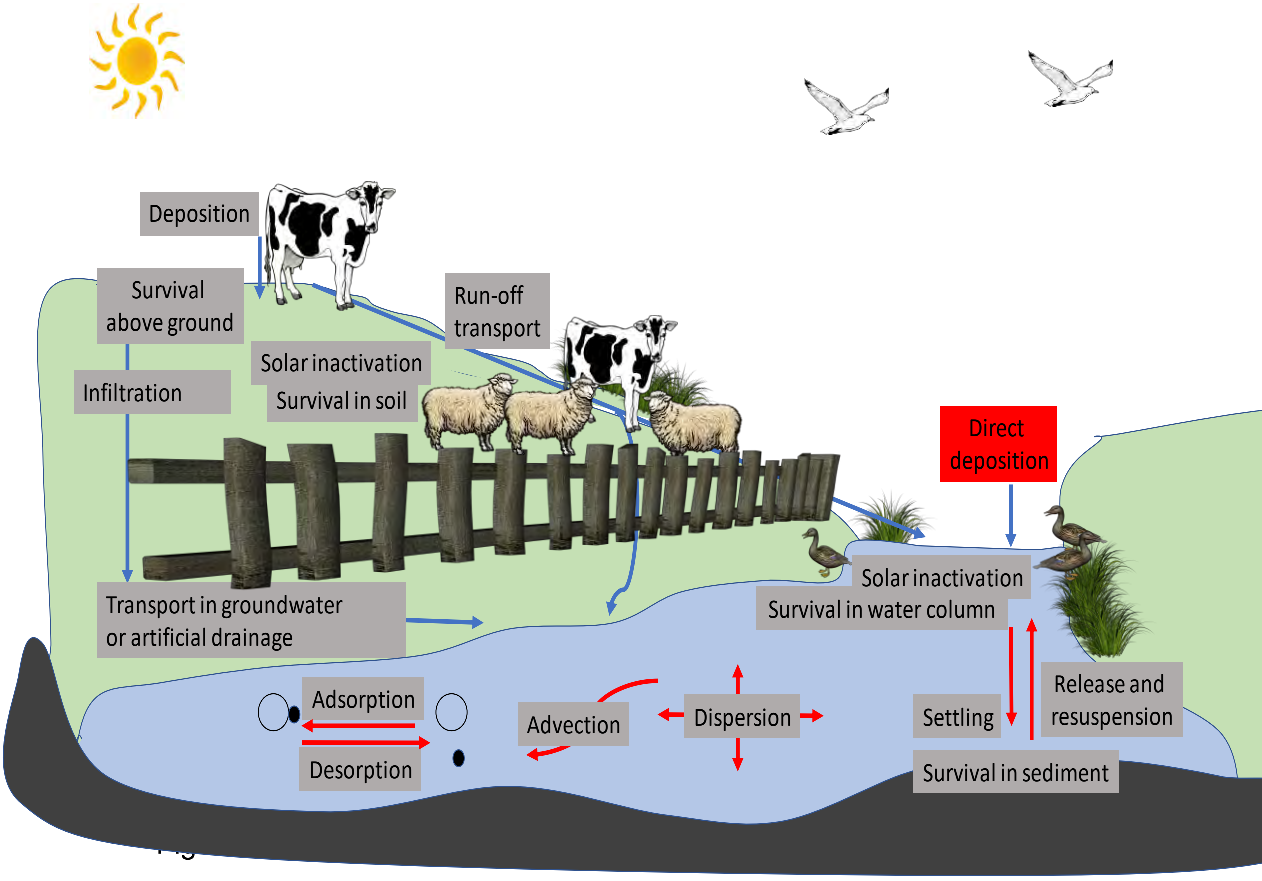
[7] Tend to have low microbial loads

programs hinges on our ability to understand the population dynamics of these sediment reservoirs under both base- and storm-flow conditions.

iv. I also agree with Stott et al (2011)[8] who suggest that a greater understanding of stream channel dynamics with respect to faecal microbes and considerations for microorganism specific factors is required before the ramifications of mitigations applied at a farm-scale can be determined at a catchment scale, in a similar manner to that done for nutrients.



concentrations in pastoral catchments.

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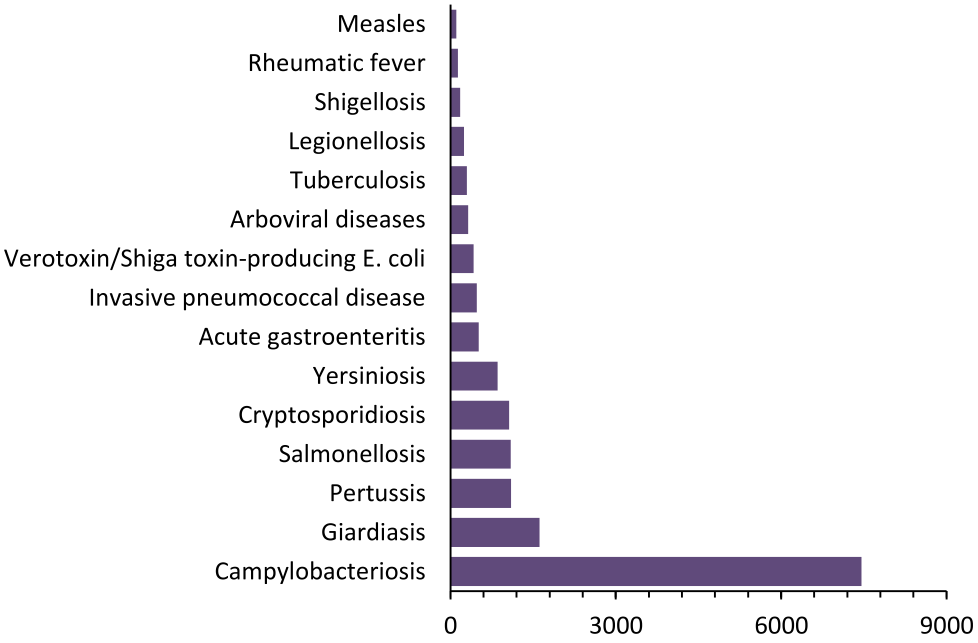
[8] Stott, R., Davies-Colley, R., Nagels, J., Donnison, A., Ross, C., Muirhead, R., 2011. Differential behavior of Escherichia coli and Campylobacter spp. In a stream draining dairy pasture. J. Water Health 09 (1), 59e69. http://dx.doi.org/10.2166/wh.2010.061.

ZOONOTIC DISEASES ASSOCIABLE WITH PRIMARY PRODUCTIVE LAND USE

24. Zoonotic pathogens—organisms that originate from animals and cause disease in humans—account for nearly two-thirds of emerging infectious diseases in humans (Voro et al 2007)[9]. In New Zealand and other developed countries, enteric zoonotic diseases are major contributors to water and food-borne disease, including gastroenteritis. Historically, New Zealand has a high incidence of enteric zoonotic diseases as reported for developed countries and the number of cases has increased annually. Enteric zoonotic diseases constitute about 80% of the total notified illnesses in New Zealand[10]. In New Zealand, the most significant micro-organisms causing zoonotic diseases are the bacteria Campylobacter spp., some strains of Escherichia coli, Salmonella spp., and the protozoa Giardia and Cryptosporidium[11]. For instance, in 2016, Campylobacteriosis, Giardiasis, and Cryptosporidiosis accounted for over 60% of notified diseases in New Zealand.



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[9] Vorou, R. M., Papavassiliou, V. G., & Tsiodras, S. (2007). Emerging zoonoses and vector-borne infections affecting humans in Europe. *Epidemiology & Infection*, *135*(8), 1231-1247.

[10] https://thewaternetwork.com/_/climate-change-and-the-environment/blog-Jl6/zoonoses-in-new-zealand-lNbgfO1psWDbvKpOvPwOWw

[11] https://www.health.govt.nz/system/files/documents/pages/zoonosespmlt_0.pdf

Figure 3: Number of notifications by disease, New Zealand (2016)[12]

25. Campylobacteriosis[13], caused by Campylobacter species, is the most common human bacteria-related diarrhoeal illness in New Zealand, as well as in developed and developing countries of the world. Although seldom disease-causing in animals, Campylobacter infects most warm-blooded wild and domestic animals. Humans become infected through ingestion of contaminated unpasteurized milk, drinking water, or undercooked meat (US Centers for Disease Control and Prevention 2009). Infection rates in New Zealand have steadily increased since 1980, peaking in 2006 at over 15,000 notifications (Baker et al. 2012[14]). While the incidence rate for Campylobacteriosis in New Zealand has reduced since 2016, the current incidence is still 1.5 to 3 times higher than reported incidence rates in Australia, England and Wales, and several other developed countries[15]. Although previous surveillance efforts identified poultry as the primary source of human disease, it also found that other animal sources such as sheep and cows account for disease transmission, probably due to environmental and occupational exposures.

26. Cryptosporidiosis is an important cause of gastroenteritis worldwide, and New Zealand has one of the highest reported rates in the world with between 26·1 and 32·3 new cases per 100,000 population per year[16]. Cryptosporidiosis is caused by infection with protozoan parasites of the genus Cryptosporidium. *S*ymptoms of gastroenteritis typically last from several days to several weeks. Routes of transmission are largely from poorly treated drinking water, swimming in swimming pools, contact with

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[12] https://surv.esr.cri.nz/surveillance/annual_diseasetables.php

[13] http://scientists.org.nz/files/journal/2011-68/NZSR_68_2.pdf

[14] Baker, M. G., Kvalsvig, A., Zhang, J., Lake, R., Sears, A., & Wilson, N. (2012). Declining Guillain-Barré Syndrome after Campylobacteriosis Control, New Zealand, 1988–2010. Emerging Infectious Diseases, *18*(2), 226-233.

[15] Lane, R., Briggs, S. (2014) Campylobacteriosis in New Zealand: room for further improvement. The New Zealand Medical Journal. 127(1391), 6-9

[16] Learmonth JJ et al (2004). Genetic characterization and transmission cycles of Cryptosporidium species isolated from humans in New Zealand. Applied and Environmental Microbiology. 70:3973–3978.

farm animals and person-to-person transmission. In New Zealand, Lake et al (2008)[17] argued that human cryptosporidiosis demonstrates spring and autumn peaks of incidence. The authors argued that in the spring livestock are most infectious due to the birth of large numbers of new, and hence highly infectious, livestock while the autumn cryptosporidiosis peak is related to increased recreational water use, swimming, outdoor activities and increased person-to-person spread.

27. Giardiasis is an important cause of gastroenteritis worldwide. It is one of the most commonly notified waterborne disease in New Zealand, which has high incidence rates compared with other developed countries[18]. Giardiasis is caused by Giardia, a protozoan parasite that can cause water-borne diarrhoeal infections to both man and animals. Transmission occurs from ingestion of faecally-contaminated food or drinking-water, swallowing recreational water (for example, swimming and wading pools, streams and lakes), exposure to faecally contaminated environmental surfaces, and person to person by the faecal-oral route. Like C. parvum, Giardia cysts are very resistant to conventional water disinfection treatments. Prevention of their spread is, therefore, essential to prevent contamination of fresh waters.

ISSUES WITH MONITORING ZOONOTIC DISEASE IN NEW ZEALAND

28. Surface waters are prone to contamination by zoonotic pathogens (from various point and nonpoint sources) as a result of faecal wastes from intensive agriculture-related practices on primary productive lands. Detection of these infectious pathogens requires the use of recovery and isolation methods employing multiple steps of cultivation for bacteria, cell cultures or experimental animals. Going beyond presence/absence enumeration analysis, detecting pathogens by their infectivity or cultivability is more important for decision making about pathogen risks to human and animal health, because only live or infectious pathogens pose health risks. Unfortunately, even some advanced technologies (e.g. nucleic acid

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[17] Lake IR, Pearce J, Savill M (2008) The seasonality of human cryptosporidiosis in New Zealand. Epidemiology and Infection 136 (10): 1383–1387

[18] Hoque E, Hope V, Scragg R, Baker M, Shrestha R. A descriptive epidemiology of giardiasis in New Zealand and gaps in surveillance data. N Z Med J. 2004;117:U1149.

amplification by PCR, immunoassays, etc.) will still capture dead or inactivated pathogens during analysis of agricultural waste samples. As dead cells no longer pose health risks, detection of these dead or inactivated pathogens are “false-positives” which tend to confound our ability to accurately determine risks of infectivity.

29. In New Zealand, current risk assessment is based on a monitoring system that assesses the levels of Escherichia coli. *E.coli* is typically used an indicator of the presence of potential enteric pathogens given that it is commonly present at high concentrations in the intestinal tracts and faeces of animals, including humans. Despite the widespread use as of *E.coli* as an indicator organism, it is quite debatable as to whether the levels of FIB adequately predict the presence of all types of pathogens, including viruses and parasites. Zoonotic pathogens from primary productive land are not reliably detected using the *E.coli* proxy. This is because there is often no correlation between *E.coli* and zoonotic pathogens that they are meant to ‘protect against’. Hence, merely measuring *E.coli* as an indicator of risk on streams receiving input from primary productive lands may fail to protect the public from exposure to zoonotic pathogens. These concerns are well documented[19]

30. Another consideration is that not all FIB are from faecal sources (Ferguson 2006; Ksol et al 2007; Yan et al 2011)[20]. Non-fecal environmental sources of FIB (e.g. decaying plants, algae and biofilms, indigenous *E.coli* 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 relationship of the FIB from non-fecal sources to the occurrence and

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[19] Sobsey, M.D.; Khatib, L.A.; Hill, V.R.; Alocilja, E.; Pillai, S. Pathogens in Animal Wastes and the Impacts of Waste Management Practices on Their Survival, Transport and Fate. In White Paper, Midwest Plan Service; Iowa State University: Ames, IA, USA, 2001

[20] 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.

distribution of enteric pathogens[21] in these examples has not been demonstrated (EPA 2014[22]).

31. Also, current risk assessment is based on a monitoring system that does not distinguish between animal versus human faecal contamination, or even between animal strains such as ruminate or avian. It is intuitive to believe that non-human faeces probably carry fewer pathogens that might be hazardous to humans. For example, viruses that are specific to humans do not normally occur in animals; therefore, the risk from animal faeces may not be equivalent to that associated with human faeces. The dilemma, however, is that the presence of such faecal indicators may or may not be an indication of actual risk from pathogens at that time and are of little use in determining if their faecal source is human or animal. A detailed knowledge of the sources of faecal material in the catchment impacting on a waterway, be they human or animal in origin, and data related to the spatial and temporal loadings of expected pathogens from such sources will, in profound ways, assist the assessment of a public health risk.

32. Another limitation to the current risk assessment system, which relies on FIB as indicator bacteria, is that FIB can naturally survive and proliferate outside of animal intestines, in tropical and temperate habitats. This calls into question their reliability as indicators in these habitats. That is, the quantity of *E.coli* is not necessarily correlated with increasing risk of infection. Also, viral and protozoan pathogens are not well correlated with standard bacterial indicators such as FIB[23]. 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.

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[21] and the potential for those microbes to predict human health effects

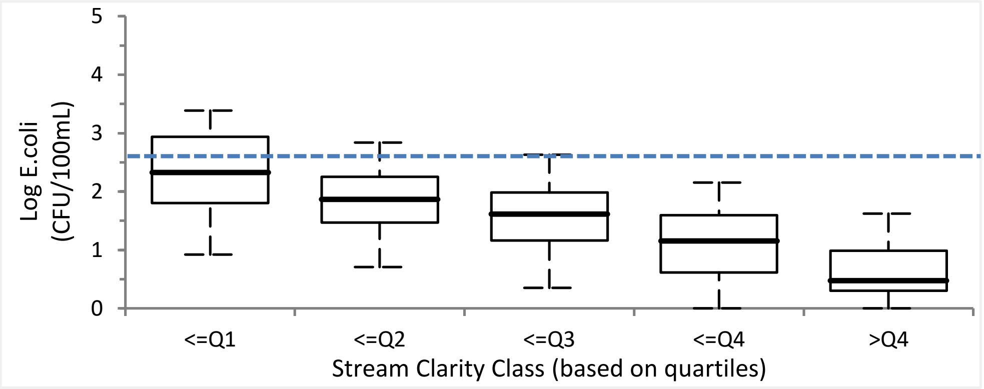
[22] EPA (2014) Overview of Technical Support Materials: A Guide to the Site-Specific Alternative Recreational Criteria TSM Documents. EPA-820-R-14-010 U.S. Environmental Protection Agency Office of Water Office of Science and Technology Health and Ecological Criteria Division

[23] 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.

33. In New Zealand, levels of FIB in water is used to determine whether the water intended for drinking or recreational purposes are free of zoonotic pathogens. For contact recreation, less than 540 CFUs/100 mL of E. coli are recommended by the NPS-FM 2014 and warnings (advisories) are usually issued to the public when contaminant levels exceed these concentrations[24].

34. *E.coli* concentrations in New Zealand Rivers are strongly correlated with water clarity (e.g. Dada and Hamilton 2017; Davies-Colley et al 2018; Dada 2019)[25]. The same observation holds for rivers and tributaries in the Waikato region (Figure 4a,b). Correlations between water clarity (reflective of turbidity) and *E.coli* concentrations is understandable as the primary pathway for pathogens to enter surface water from agricultural land uses is via overland flow pathways (Paragraph 59). The strong coupling of water clarity and *E.coli* concentrations suggest that efforts geared towards monitoring and improving water clarity may also quite reasonably allow for concomitant reductions in *E.coli* levels in New Zealand waterways.

(a) New Zealand



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[24] National Policy Statement on Freshwater Management

[25] Davies-Colley, R., Valois, A., & Milne, J. (2018). Faecal pollution and visual clarity in New Zealand rivers: Correlation of key variables affecting swimming suitability. Journal of Water and Health, wh2018214.

Dada, A. C., & Hamilton, D. P. (2016). Predictive models for determination of E. coli concentrations at inland recreational beaches. Water, Air, & Soil Pollution, 227(9), 347.

Dada (2019) Seeing is Predicting: Water Clarity-based Nowcast Models for E.coli Prediction in Surface. Accepted for publication, Water Global Journal of Health Science

(b) Waikato

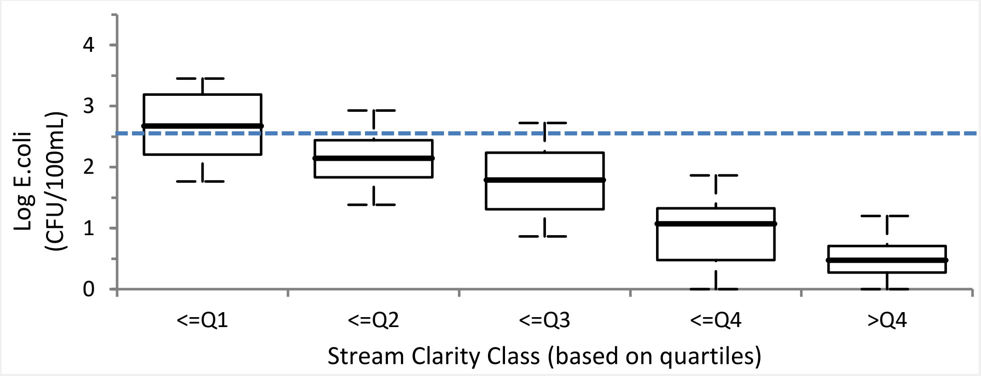

Figure 4: Box plots of Region *E.coli* concentrations versus water clarity grouped by quartiles, 2005-2013 for (a) New Zealand and (b) Waikato Region (Dotted blue line is the log-transformed bathing water standard of 540 CFU/100mL (i.e. 2.54 LogCFU/100mL, Q1-Q4 represent first, second, third and fourth quartile of the water clarity values, based on black disc measurements)

35. I note that the approach taken in PC1to monitoring *E.coli* levels as a proxy for the presence of zoonotic pathogens does not seem to distinguish between concentrations during different flow conditions (e.g. see Figure 8). A conservative threshold set at 540CFU/100mL 95th percentile concentration regardless of the season may actually mean that health risks associated with exposure to pathogens are over-estimated, particularly during non-swimming periods when the FIB population are largely driven by periods of high flow. Considerations for flow conditions may warrant the establishment of a stringent maximum limit for faecal coliform bacteria per 100mL sample during the “swimming season” (typically during base and low flows) and a less stringent limit for all other times (storm flows).

36. The WRPC1 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. This is based on the assumption that ‘the 95th percentile of sample results from the previous 5 years accommodates infrequent or rare high flow events’. It is important to note that the 95th percentile *E. coli* concentrations are rare events that are associated with storm flows and will only reflect in 5% of the observed data used to make this judgement. In simple terms, only 5% of the monitoring data will be higher than the 95th percentile

concentration, regardless of the number of “previous years” of data considered.

37. The WRPC1 also argues that the proposed 95th percentile target and monitoring regime already provides for the exclusion of extreme events, and hence no need is required for an amendment of the Table 3.11-1 such that the targets for E. coli do not apply during high flow events. Details are however not available about the ‘proposed monitoring regime’ and the exclusion criteria that will be used to adjudge when sampling should be conducted to implement targets in the Tables 3.11-1. Are the monitoring officers to use their discretion to determine when sampling is to be done while monitoring compliance with the targets specified in the WRPC1 Table 3.11-1? These issues require clarification, as footnote 1 of the NPS-FM *E.coli* Attribute State Table differs from this position of ‘subjective determination of monitoring regime that excludes high flow events’. The NPS-FM 2017 states categorically that ‘…samples should be <u>collected on a regular basis regardless of weather and flow conditions</u>’. Hence, the proposed attribute monitoring programme (to determine achievement of the targets) is <u>NOT</u> consistent with the guidance contained in the NPS-FM as it presents ambiguities associated with when monitoring officers are to sample and not to sample.

38. Based on the arguments in paragraphs 33-36, I recommend that:

    (a) the proposed WRPC1 monitoring needs to be consistent with the NPS-FM guidance document with samples <u>collected on a regular basis regardless of weather and flow conditions</u>;

    (b) The *E.coli* targets however need to be revised and the policy wording should be amended to read ‘the *E.coli* concentration of the water must not exceed [table 3.11-1 revised numerical parameter given in CFU/100mL] when the river is at or below medium flow (the 50th percentile flow)’.

    (c) If it is impossible to designate revised Table 3.11-1 *E.coli* targets in line with recommendation (ii) above, then the *E.coli* targets should be amended to comply with the NPS-FM *E.coli* Attribute State thresholds. Using this approach, an indicator of improvement in bacteriological water quality could be tied to at least two of the four numeric attribute statistics in the NPS-FM guidance document. For instance this could be a combination of median and 95th percentile *E.coli* concentrations rather than a reliance on the single 95th percentile as it is currently in the PC1 Table 3.11-1. A table of suggested targets based on this criterion are presented in Appendix 1 of this document. In this way, authorities can work towards progressive improvement of the NPS-FM Attribute State of the particular site being considered.For instance, the Attribute State of Mangauika Stm Te Awamutu Borough W/S Intake, is currently C (Yellow) which is equivalent to median *E.coli* concentrations <130 CFU/100mL and 95th percentile concentration <1200. A short term target should be set at improving the Maramaruaa NPS FM attributre state to B (Green) which is equivalent to median *E.coli* concentrations < 130 CFU/100mL and 95th percentile concentration <1000 CFU/100mL. This approach does not only comply with the NPS-FM requirements, it also makes monitoring and reporting of progress seamless.

REVIEW OF REGIONALLY RELEVANT STUDIES AND COMMENTS ON *E.COLI* REDUCTION APPROACH/TARGETS

39. I have read a number of reports that have been published to support WRPC1. These reports, and a synopsis of their objectives are presented in Paragraph 39 to 40 of this evidence.

40. Doole et al. (2015[26]) described outputs from a predictive-modeling approach that aimed to identify the economic implications of altering land and point-source management to achieve the water-quality limits proposed for each of four scenarios:

(a) Substantial improvement in water quality for swimming, taking food, and healthy biodiversity,

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[26] Doole et al (2015) Economic evaluation of scenarios for water quality improvement in the Waikato and Waipa River catchments. Assessment of first set of scenarios 24 August 2015. Report No. HR/TLG/2015-2016/4.1, Draft for discussion purposes, 10 November 2015

(b) No further degradation and improving sites to at least minimum acceptable standard for all attributes

(c) Some general improvement in water quality for swimming, taking food, and healthy biodiversity, and,

(d) No further degradation in spite of lags.

41. The modelling approach used also predicted the economic implications of these scenarios at the farm, catchment, regional, and national scales.

42. Other relevant reports arising from the WRPC1 are highlighted below:

(a) Doole et al. (2015[27]) further described using the predictive-modeling approach the implications of altering land and point-source management to achieve the water-quality limits proposed for steps towards Scenario 1, across a number of alternatives.

(b) Doole et al. (2016[28]) employed the HRWO economic model to simulate the policy mix associated with WRPC1 under several different situations, to assess its impact on economic and water-quality outcomes within the Waikato River and Waipa River catchments.

(c) Doole et al. (2016[29]) estimated the state of water quality in the Waikato and Waipa River catchments in 1863 using predictive modelling and highlighted the effect that future policy actions—derived from the HRWO process—are likely to have on surface water quality. The science model behind the predictions was the *E. coli* model previously reported by Semadeni-Davies et al. (2015)[30].

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[27] Doole et al (2015) Evaluation of scenarios for water-quality improvement in the Waikato and Waipa River catchments. Assessment of second set of scenarios 24 September 2015. Report No. HR/TLG/2015-2016/4.2, Draft for discussion purposes, 10 November 2015

[28] Doole et al (2016) Simulation of the proposed policy mix for the Healthy Rivers. Report No. HR/TLG/2016-2017/4.5, Draft for discussion purposes, 13 July 2016

[29] Doole et al (2016) Prediction of water quality within the Waikato and Waipa River catchments in 1863. Report No. HR/TLG/2016-2017/4.3, Draft for Discussion Purposes, 2 August 2016

[30] Semadeni-Davies et al. (2015) Modelling E. coli in the Waikato and Waipa River Catchments: Development of a catchment-scale microbial model Prepared for the Technical Leaders Group of the Healthy. Rivers/Wai Ora Project. Report No. HR/TLG/2015-2016/2.6

(d) Doole 2015[31] outlined the cost and levels of mitigation achieved for each of the four contaminants for a range of management practices across a broad array of land uses. A feature of this report is an extensive sensitivity analysis that is performed to test how profit changes within the catchment-level model utilized within the HRWO process

(e) In Doole (2016)[32], an economic model — considering the farm-, catchment-, regional-, and national-level economic implications of water-quality limits — was utilised to investigate and predict the changes that may be associated with partial movements from the current state towards the most aspirational of the initial water-quality scenarios previously developed (Scenario 1). (Scenario 1, key output of the HRWO process, involves an improvement in water quality everywhere in the Waikato and Waipa catchments, even if it is already meeting minimum acceptable state).

(f) Doole et al (2016[33]) outlined the reasons why certain key decisions have been made during the design and development of this HRWO economic model

(g) Doole (2016)[34] outlined the potential implications of what would happen in the absence of the proposed policy mix—the prediction of outcomes associated with moving forward according to a "business-as-usual" scenario.

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[31] Doole (2015) Description of mitigation options defined within the economic model for Healthy Rivers Wai Ora Project. Description of options and sensitivity analysis 28 September 2015. Report No. HR/TLG/2015-2016/4.6, Draft for discussion purposes, 10 November 2015

[32] Doole (2016) Model structure for the economic model utilised within the Healthy Rivers Wai Ora process. Report No. HR/TLG/2015-2016/4.8, Draft for Discussion Purposes, 23rd February 2016

[33] Doole et al (2016) General principles underlying the development of the Healthy Rivers Wai Ora (HRWO) economic model. Report No. HR/TLG/2015-2016/4.7, Draft for Discussion Purposes, 23rd February 2016

[34] Doole (2016) Evaluation of scenarios for water-quality improvement in the Waikato and Waipa River catchments -Business-as-usual assessment. Report No. HR/TLG/2016-2017/4.4, Draft for discussion purposes, 21 October 2016

(h) Semandeni-Davies and Elliot (2016)[35] reported on the calibration of a national catchment-scale model that predicts the effect of stock exclusion (i.e., fencing to restrict stock access to waterways and their riparian margins) on water quality.

43. I note that the Doole et al. (2015) report cited a successful integration of diverse hydrological/water quality models that relate contaminant losses within and across subcatchments to pollutant concentrations at the various monitoring sites represented within the catchment. These models concern *E. coli* (Semadeni-Davies et al., 2015)[36], sediment, nitrogen and phosphorus. Given that hydrological/water quality models are a core driver of the HWRO model, I have decided to focus on a review of the *E.coli* model (Semadeni-Davies et al., 2015) that informed the HRWO model and plan change decision-making. The Semadeni-Davies et al (2015) study reported the calibration of three steady-state catchment models to estimate *E. coli* loads and concentrations in the Waikato and Waipa River catchments from Lake Taupo to Port Waikato. In this evidence, my comments, thus, specifically relate to concerns on the assumptions used in the adopted *E.coli* models, in relation to the estimated fate-transport matrices and processes that drive variabilities in the flow and attenuation of *E.coli*. These are presented in subsequent sections.

44. First, in the Semadeni-Davies et al (2015) study, the authors note that *E.coli* concentrations and loadings were generated for sites that do not have flow data. I note that out of the 63 sites that formed the basis for the study, only 20 of these sites had flow data (see Figure 2-1 in Semadeni-Davies et al 2015). This represents less than 30% of the entire dataset. Using a model with 'generated dataset' comprising of more than 70% of the observed dataset is technically flawed, potentially vulnerable to bias and could be distorted in the directions of certain vested policy interests.

45. While rainfall and flow were considered as variables in the Semadeni-Davies et al (2015) *E.coli* model, my experience analyzing *E.coli* data in

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[35] Semadeni-Davies, A. Elliott, s (2016) Modelling the effect of stock exclusion on E. coli in rivers and streams: National Application. MPI Technical Paper No: 2017/10. Prepared for Ministry for Primary Industries by NIWA, 229 pages.

[36] As previously cited.

New Zealand reveals that antecedent rainfall and antecedent flow (which could be incorporated as a lagged component) explains a higher proportion of variability in *E.coli* dataset than actual rainfall or flow does (Dada and Hamilton, 2017).

46. The load models reported in the Semadeni-Davies et al (2015) study are simply steady-state models that predict mean annual loads. The implication of this is that the more important seasonal changes in *E. coli* generation and transport are not captured by the models[37]. This reduces the importance of the model as there is a huge variability of *E.coli* loads, travel time and in-land/in-stream dynamics that is missed out during varying seasons and flow conditions.

47. The Semadeni-Davies et al (2015) study states that '…under the NPSFWM (2014) National Objectives Framework (NOF), it is assumed that if *E. coli* are present in fresh water bodies, then other more pathogenic faecal micro-organisms are also likely to be present.' This is a technically inappropriate statement. It is not the fact that *E.coli* may be present that is material. Rather the correct approach is to note that *E.coli* may be indicative of a heightened probability of potentially infective pathogens if the *E.coli* is present at levels above certain thresholds that have been previously demonstrated to be so.

48. I note that the Semadeni-Davies et al (2015) study used a rating curve method to estimate measured mean annual *E. coli* loads at sites where there were sufficient concurrent flow data at or near the site. Following an intense search into published literature, the lack of refereed publications that use this approach for bacteria indicates that it is rarely used for *E.coli*. Besides, the number of the formula and coefficients stated in this report were not explicitly stated, which prevents independent verification of inputs and outputs of the model. For instance, a, b and s in the 'bacteria rating curve' equation are not stated. In another instance, the authors mention that '…in the ratio method, the median concentration is multiplied by a factor to convert to flow-weighted concentration' (page 22) but fail to mention what

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[37] Based on an analysis of available data, I note specifically that storm flow conditions is responsible for at least 80% of total *E.coli* loads in the Waikato region. Steady state models used in the Semadeni-Davies et al (2015) *E.coli* model will not capture these storm flow loads.

the factor is and how it was generated. This is important because modellers 'optimise' these coefficients/functions to best make the data fit and the failure to disclose this information means that the model on which the WRPC1 decision making was based cannot be independently proved to be trustworthy.

49. I also note that the Semadeni-Davies et al (2015) modelling study only incorporated 4 out of at least 21 factors that influence variabilities in *E.coli* levels in primary productive land. Variables incorporated into the model were surface decay, drainage type, rainfall class and land use class incorporating a conservative per hectare animal population[38]. Important but missing variables in the calibrated model are detailed in paragraph 22(a) of this evidence. Concentration of organic matter in different soil types which affect microbial survival, presence/absence of infected grazing animals that carry zoonotic pathogens, the stage and severity of infection, the species and numbers of pathogens carried by the animals and shedding rates, the extent of direct access to the stream, manure composition, degree of specific microbial association within the manure/soil matrix.

50. In addition the *E.coli* model in the Semadeni-Davies et al (2015) study incorporated only 3 out of at least ten factors/variables that influence variabilities in *E.coli* levels in streams. Important but missing variables in the calibrated model are detailed in stated in paragraph 22(b) of this evidence. This include factors related to bacterial survival and transport in the water column, settling into sediments, survival in streambed sediments, release and resuspension into the water column, advection, and dispersion.

51. I note that the in-stream attenuation factor was calibrated to zero in the Semadeni-Davies et al (2015) model. Despite the importance of microbial die-offs and growth potentials in streams, Semadeni-Davies et al (2015) argued that 'adding microbial die-off into the model significantly reduced the performance of the model and hence it was avoided in the model'. Whilst it could be argued this makes the model conservative, I would argue that by ignoring a process known to be important by all environmental microbiologists, it fails to properly demonstrate accurate and realistic *E.coli*

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[38] (see Table 2-4 in Semadeni-Davies et al 2015)

loadings. In my view, it is not surprising that there were 'anomalies'[39] in the results of the model.

52. It is important also that the *E.coli* models reported in the Semadeni-Davies et al. (2015) study were not validated. This means that the models are not fit to inform or underpin Plan Change 1, that is the models are not fit for purpose. Model validation assesses if a model possesses a satisfactory range of accuracy consistent with the intended application of the model. Validation checks the accuracy of the model's representation of the catchment as the modeller compares the model input-output transformations to corresponding input-output transformations for the catchment. In layman terms, this means the Semadeni-Davies et al. (2015) *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. This is worrying. When the authors decide to test one of the developed models in another published report (Doole et al. 2016[40]), they chose a year for which there was no observational data (1863), thus allowing heavy reliance on 'generated data'. The authors applied the 'developed model' to predict water quality outcomes 'thought to have existed in 1863' with the current state and with the established long-term goal for water quality established within the HRWO process—known broadly as "Scenario 1". I am of the opinion that to robustly assess the *E.coli* loads prediction the application of the empirical models to estimate water-quality outcomes in past natural conditions across the Waikato and Waipa River catchments should have been done for other years for which there are observed data, for the sake of comparison. This would greatly reduce potential uncertainties and errors associated with the *E.coli* loads prediction and the HWRO decision making. These uncertainties coupled with other reasons previously stated seem to render the model unfit to inform or underpin PC1.

53. While the targets for microbial reduction as stated in Scenario 1 are a step in the right direction, it is important to note that the estimates that formed

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[39] As the authors quoted in Semadeni-Davies et al (2015)

[40] Graeme Doole1, Neale Hudson2, and Sandy Elliott (2016) Prediction of water quality within the Waikato and Waipa River catchments in 1863. Report No. HR/TLG/2016-2017/4.3

the targets are associated with very significant uncertainties Doole (2016[41]) stated categorically that ‘changes in microbial loadings to water that will occur over the next decade—as indicated by *E. coli* yields—are problematic to assess’. Unlike the case for nutrients where a lot of research work has been undertaken to help our understanding of in-land and in-stream processes, there is a general lack of knowledge regarding key elements of their generation, survival, preponderance, and transport from farming systems in receiving waters[42]. These uncertainties coupled with other reasons stated in Section 23 make it impractical to realistically estimate loads or in-stream *E.coli* concentrations.

54. I have reviewed the Semandeni-Davies and Elliot (2016) report on ‘Modelling the effect of stock exclusion on E. coli in rivers and streams: National Application’. The report used a national model to analyse changes in *E. coli* concentrations in freshwater around the country as a result of fencing. Eight fencing scenarios were modelled and the predicted *E.coli* concentrations during these scenarios were used to classify rivers into bands (attribute states). These scenarios are:

    (a) Scenario 1 – current level of fencing;

    (b) Scenario 2 (status quo) – current level of fencing, with further fencing in regions which either have fencing policy in place or are planning new fencing policies to be in place by 2017;

    (c) Scenarios 3a to 3e (Land and Water Forum progressive) – status quo with fencing along Water Accord streams on land with an average slope of less than 16° (a) dairy platform; (b) dairy runoff on land owned or leased by dairy farmers; (c) dairy grazing on land owned by a third party; (d) sheep and beef; and (e) deer;

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[41] Doole 2016 Evaluation of scenarios for water-quality improvement in the Waikato and Waipa River catchments: Business-as-usual assessment 20 October 2016. Report No. HR/TLG/2016-2017/4.4

[42] Muirhead, R. (2015), ‘A farm-scale index for reducing faecal contamination of surface waters’, Journal of Environmental Quality 44: 248–255.

(d) Scenario 4 (Steep Hill Country) – fencing along all streams, including non-Accord streams, accessible to all stock on land with an average slope of less than 28°.

55. I note a number of critical issues with the Semandeni-Davies and Elliot (2016) report and highlight these below:

(a) Band classification: the classification used to delineate rivers into bands (attribute states) is predicated on outdated numeric attributes (NPS-FM 2014). For example, assuming reported annual median *E.coli* concentrations are consistent[43], rivers adjudged to be in the best attribute state (Band A) in the Semandeni-Davies and Elliot (2016) report have been re-classified by MfE as A, B, C and D in the revised NPS-FM document (2017). Hence, a river adjudged to be of excellent quality (Band A) in the Semandeni-Davies and Elliot (2016) report may actually be poor, based on the updated policy document. It is thus not surprising that, during the 'do nothing' scenarios, Semandeni-Davies and Elliot (2016) reported that 'around 80% of non-Accord streams and 90% of Accord streams nationally have median E. coli concentrations in NOF Band A'. This outdated classification scheme used in the Semandeni-Davies and Elliot (2016) report thus makes it unreliable for the current policy decision making related to stock exclusion.

(b) Meanwhile a careful analysis of the results of the Semandeni-Davies and Elliot (2016) study in Table 1 shows that that only very marginal increases (1.0 – 8.7%) in the proportion of stream length in Band A/B/C/D was associable with 'upgrades' in fencing approach. For example:

i. Only 1.06% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 2 instead of Scenario 1;

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[43] i.e. over the space of 5 years, as stipulated in the updated NPS-FM (2017)

ii. Only 1.19% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 3a instead of Scenario 1;

iii. Only 1.20% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 3b instead of Scenario 1;

iv. Only 1.33% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 3c instead of Scenario 1;

v. Only 2.61% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 3d instead of Scenario 1;

vi. Only 2.64% increase in the stream length categorised as Band A was predicted when policy decision makers apply fencing conditions in Scenario 3e instead of Scenario 1;

vii. Even during conditions of Scenario 4 (Steep Hill Country), less than 10% increase in the stream length categorised as Band A was predicted.

56. Analysis of results reported in the Semandeni-Davies and Elliot (2016) study indicates that additional fencing investment does not produce significant additional improvement in *E.coli* conditions or Band classifications nationwide. The potential for live bacteria soil to be transported from 'contributing areas' into the stream, as depicted in Figure 2, also aligns with this conclusion. That is, fencing may be beneficial in some intensively farmed areas where livestock can disturb stream beds and transport soil into waterways if not excluded. However, in other areas, fences can only stop direct deposition from animals but not overland flow of pathogens into the stream. For example, 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 (Collins et al 2007[44]) is largely associated with overland transport into receiving streams (McDowell and Wilcock 2008[45]).

Table 1: Length and proportions of nation-wide streams with estimated median E. coli concentrations in the NOF bands for each scenario. Included in this table also are increases and decreases in the % of stream length in each band.

		NOF Bands			
		A	**B**	**C**	**D**
Parameters	**Fencing Scenarios**	***E.coli* median : ≤260**	***E.coli* median : > 260 and ≤ 540**	***E.coli* median : > 540 and ≤ 1000**	***E.coli* median : >1000**
Total Stream Length (km) in Band	S1	353295	45810	968	16
	S2	357551	41613	911	14
	S3a	358050	41143	884	13
	S3b	358083	41113	879	13
	S3c	358603	40594	879	13
	S3d	363727	35514	835	13
	S3e	363855	35389	832	13
	S4	388209	11460	417	3
% of Stream Length in Band	S1	88.30	11.45	0.24	0
	S2	89.37	10.40	0.23	0
	S3a	89.49	10.28	0.22	0
	S3b	89.50	10.28	0.22	0
	S3c	89.63	10.15	0.22	0
	S3d	90.91	8.88	0.21	0
	S3e	90.94	8.85	0.21	0
	S4	97.03	2.86	0.10	0
Change in % Stream Length in Band after fencing upgrade	S1	N/A	N/A	N/A	N/A
	S2	1.06	-1.05	-0.01	0
	S3a	1.19	-1.17	-0.02	0
	S3b	1.20	-1.17	-0.02	0
	S3c	1.33	-1.30	-0.02	0
	S3d	2.61	-2.57	-0.03	0
	S3e	2.64	-2.60	-0.03	0
	S4	8.73	-8.59	-0.14	0

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[44] Collins, R., Mcleod, M., Hedley, M., Donnison, A., Close, M., Hanly, J., ... & Matthews, L. (2007). Best management practices to mitigate faecal contamination by livestock of New Zealand waters. New Zealand Journal of Agricultural Research, 50(2), 267-278.

[45] 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

57. I recommend therefore, 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.

RESTRICTION OF ANIMAL ACCESS: IMPLICATIONS FOR TARGET REDUCTIONS IN *E.COLI* LEVELS IN RECEIVING STREAMS

58. I have reviewed the Ritchie and Donnison (2010[46]) report on ‘Faecal Contamination of Rural Waikato Waterways: Sources, Survival, Transport and Mitigation Opportunities’. The report generally supports the focus in the draft Regional Policy Statement on possible mitigation efforts i.e. stock effects in and near water bodies, including access to the beds and banks of waterways and intensive grazing near water, particularly when soils are saturated or poorly-drained. Ritchie and Donnison (2010) also reached some important conclusions: (a) that transportation pathways by which microbes reach water are important; (b) that direct deposition is a minor percentage of total annual catchment *E.coli* loads to stream, and (c) that direct deposition into a typical stream would not produce a measurable change in the concentration of Campylobacter when considered on an annual contribution basis.

59. It is logical to raise questions related to stocking class and effects on *E.coli* loadings in streams flowing through agricultural catchments, as these are important considerations for risk assessments. Inputs like volume and composition of manure, proximity to stream/watering radius and watering requirements tend to vary between stocking class e.g. sheep versus cattle, etc. For instance, in pastoral lands, a study has shown that sheep normally graze within a radius of about 2.5km of a watering point while cattle within a radius of about 5km, cattle need between 40-100 litres per day of water while sheep require 2-6L per day (Table 2). Cattle have longer legs and sturdier bodies and can wade through streams that sheep would panic to enter. Contaminants from their legs and hoof disturbance of streambed

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[46] Ritchie, H. and Donnison, A. (2010) Faecal Contamination of Rural Waikato Waterways: Sources, Survival, Transport and Mitigation Opportunities. A review for Environment Waikato. Document #: 1789463

sediments and banks are more vigorous with cattle than is the case with sheep. Although Moriarty (2013)[47] reported a slightly higher proportion of *E.coli* concentration in sheep faeces than cattle faeces[48], the higher requirement for water and longer water radius distance in cattle invariably has implications on the probability of direct deposition of *E.coli*-laden faecal material in or close to water bodies. Another NZ study[49], reported that 246 cows deposited 37kg of faeces on just two crossing events. The study concluded that cows are much more (up to 50 times) likely to defecate in stream water than on adjacent raceways. In general, however, associated data based on robust microbiological science to affirm the relative importance and or contribution of different livestock are largely unavailable. On this basis, I disagree with previous studies [50], that have, merely on the basis of *E.coli* counts in culture media, argued that given the same stocking rate, losses of *E. coli* in overland flow are similar among stock classes.

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[47] Moriarty (2013) Sheep as a Potential Source of Faecal Pollution in Southland Waterways. Report Prepared for Environment Southland

[48] It is important to note that the observation reported in Moriarty (2013)[48] of a slightly higher proportion of *E.coli* concentration in sheep faeces than cattle faeces, does not necessarily mean that cattle faeces present relatively lower risks than sheep faeces. Additional FIB-pathogen correlational analysis for the different animal sources will be required to confirm this.

[49] Davies-Colley, R., Nagels, J., Smith, R., Young, R., Phillips, C. (2002) Water quality impact of cows crossing the Sherry River, Tasman District. Cows and Creeks, LandCare Knowledge Base.

[50] 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

McDowell, R.W. (2006). Contaminant losses in overland flow from cattle, deer and sheep dung. *Water, Air, and Soil Pollution* 174, 211–22

Wilcock, R.J. Assessing the Relative Importance of Faecal Pollution Sources in Rural Catchments. Technical Report TR 2006/41, Environment Waikato, Hamilton, NZ, 2006

Table 2: Average water requirements of stock

Stock type	**Consumption Per head per day (L)**
Sheep (weaners)	2-4
Sheep (adult dry sheep)	2-6
Sheep (ewes with lambs)	4-10
Cattle (lactating cows)	40-100
Cattle (young stock)	25-50
Cattle (dry stock, 400kg)	35-80
Horses	40-50

60. Meanwhile, studies[51] which have analysed *E.coli* loadings in waterways in the Waikato region affirm that surface runoff is the major source of faecal pollution from agriculture, despite inputs from dairy herds crossing streams and from drains (Figure 2). For instance, based on datasets for Toenepi, Davies-Colley et al. (2008[52]) estimated that direct deposition accounted for only about 0.23% of the total annual *E. coli* 'production' from the catchment streams and that 95% of the annual yield was exported during the thirty storm flood events that occurred over a twelve-month period. In a particular instance, stream *E. coli* concentrations were significantly reduced following the installation of bridge crossings for dairy herds over the Sherry River near Motueka, but this reduction was not sufficient to meet contact recreation standards (Ritchie and Donnison 2010) [53]. In a previous study by McDowell

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[51] 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

McDowell, R.W. (2006). Contaminant losses in overland flow from cattle, deer and sheep dung. Water, Air, and Soil Pollution 174, 211–22

Wilcock, R.J. Assessing the Relative Importance of Faecal Pollution Sources in Rural Catchments. Technical Report TR 2006/41, Environment Waikato, Hamilton, NZ, 2006

[52] Davies-Colley R, Lydiard E, Nagels J 2008. Stormflow-dominated loads of faecal pollution from an intensively dairy-farmed catchment. Water, Science and Technology 57:1519-1523.

[53] Ritchie, H. and Donnison, A. (2010) Faecal Contamination of Rural Waikato Waterways: Sources, Survival, Transport and Mitigation Opportunities. A review for Environment Waikato. Document #: 1789463

(2008)[54], water quality was monitored on a tributary of the Dow Stream with the goal of assessing if fencing-off an area of the stream channel with a known contaminant source (a wallow) and riparian planting improved water quality as measured by the two-weekly concentrations and annual loads. Results revealed that mean concentrations of *E. coli* showed no significant difference with fencing-off and planting.

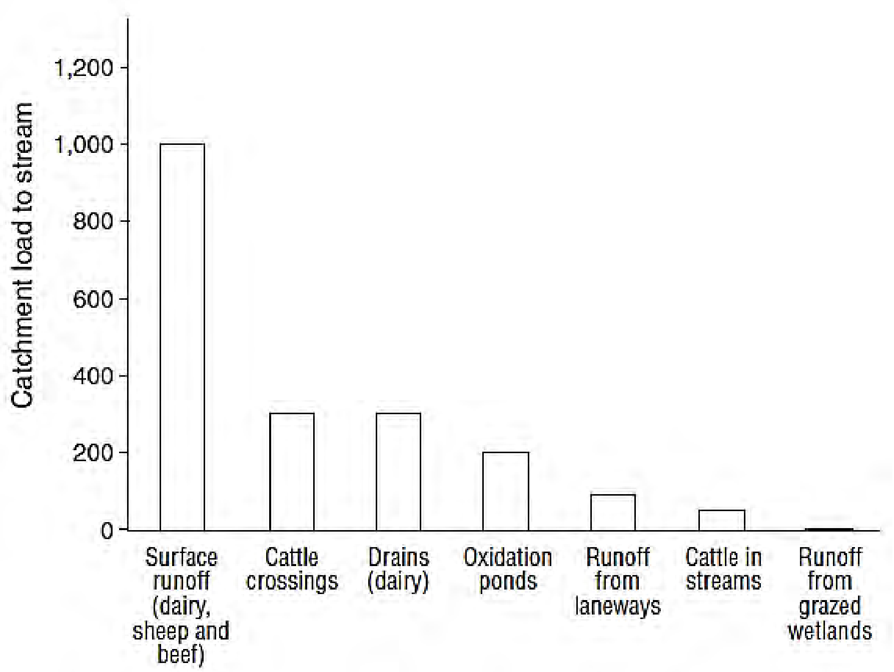

Figure 5: Waterway loadings of Escherichia coli (CFU x 108/ha./pasture/year for major sources of faecal matter in the Waikato Region, New Zealand. Source: McDowell and Wilcock 2008)

61. These published information suggest that if the streambank fencing is erected for reducing the delivery of *E. coli* to water ways, there could still be elevated *E.coli* levels in these streams (listed in Table 3.11.1 in the WRPC1) that run through agricultural catchments. At this juncture, it is important to mention that the Doole (2015)[55] report (which describes the mitigation

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[54] McDowell (2008) Water quality of a stream recently fenced-off from deer. New Zealand Journal of Agricultural Research 51(3):291-298

[55] Doole (2015) Description of mitigation options defined within the economic model for Healthy Rivers Wai Ora Project. Description of options and sensitivity analysis. Report No. HR/TLG/2015-2016/4.6

options defined within the economic model for Healthy Rivers Wai Ora Project), applied certain estimates 58% and 65% for median and 95th percentile dairy and drystock loads when estimating the efficacy of streambank fencing for reducing the delivery of *E. coli* to water ways. These values were, according to the report, based on personal communication and published studies (see Table 3). It should be noted that a review of the published studies cited as a basis for these estimates indicate that a more conservative estimate of 36% should have been applied, going by the average of these variously published figures which range from 20-65%. Applying a near maximum stream bank efficacy estimate as was done in the Doole 2015 report tends to allow for a gross overestimate of the stream bank fencing efficacy for reducing the delivery of *E. coli* to water ways. This suggests that the estimated stream bank fencing efficacy which formed the basis for the decision making may actually be unrealistic or over-optimistic.

Table 3: Reported efficacy levels for streambank fencing for reducing *E. coli* loadings, extracted with modifications* from Doole 2015 report

Reduction in *E.coli* delivery (%)		Land use	Reference**
	27.5*	Cattle	McKergow et al. (2007)
	40	Cattle	Monaghan and Quinn (2010)
	60	Dairy and drystock	Monaghan and Quinn (2010)
	25	Dairy and drystock	Muirhead et al. (2011)
	20	Dairy and drystock	Longhurst (2012)
	24	Drystock	Longhurst (2012)
	47.5*	Dairy and drystock	Quinn
	20	Dairy and drystock	Semadeni-Davies and Elliot (2012)
	24	Dairy and drystock	Semadeni-Davies and Elliot (2012)
	20	Dairy and drystock	Semadeni-Davies and Elliot (2013)
	50	Dairy and drystock	Semadeni-Davies and Elliot (2013)
	20	Dairy and drystock	Elliot et al. (2013)
	50	Dairy and drystock	Elliot et al. (2013)
	55	Drystock	McDowell et al. (2013)
	20	Dairy	Ross Monaghan (pers. Comm., 2015)
	30	Median reductions in dairy and drystock 95th percentile	Ross Monaghan (pers. Comm., 2015)
	58		Richard Muirhead (pers. Comm., 2015)
	65	Reductions in dairy and drystock	Richard Muirhead (pers. Comm., 2015)
Average	36.44		
	*	an average of min and max estimates reported in the study	

62. A recent paper by McDowell et al. (2017) based on GIS modelling, concluded that fencing small waterbodies in head water hill catchments will be required to significantly reduce catchment contaminant loads. It is important that decision makers are confident that endorsing the proposed fencing rules for all stock classes, will result in the *E. coli* reductions in streams predicted by the PC1 modelling. The McDowell et al (2017), which is based on analysis of stream orders appears to reinforce the WRPC1 approach, albeit at a national level and based on modelling. I therefore re-examined historical water quality monitoring data[56], by comparing *E.coli* concentrations in rivers and streams with varying stream order classification with a view to evaluating if the proposed fencing requirements will be effective in mitigating pathogens. A total of 8108 nation-wide *E.coli* datasets which had associated discharge and water clarity data were used. Based on this statistical analysis, I found that trends in *E.coli* concentrations and loads in New Zealand rivers are not related to stream order (Figure 6 and Figure 7). In contrast to the results of McDowell report, this indicates that stream order is not relevant to the faecal indicator bacteria levels observable during monitoring programs. On the basis of this statistical analysis on actual monitoring data (as against modelled input in the McDowell et al. 2017 study), I posit that if potential regulation in New Zealand is requiring livestock to be fenced off from certain rivers based on their stream order classification, there might be no notable effect on *E.coli* loadings in the receiving waters. This position is also strengthened by those of other studies (see paragraph 59) which affirm that surface runoff is the major source of faecal pollution from agriculture in the Waikato Region, as opposed to direct defaecation in streams. Fencing, without additional measures such as riparian buffer strips, is therefore unlikely to have a meaningful effect on

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[56] A total of 145,040 water quality dataset that have been routinely collected by regional authorities from as early as the late 1980s for New Zealand rivers and tributaries (https://data.mfe.govt.nz/) was used in the analysis. This dataset contained measured values for several notable parameters such as ammoniacal nitrogen, total nitrogen, nitrate-nitrogen, dissolved reactive phosphorus, total phosphorus and *E.coli*. All *E.coli* datasets were extracted (n=8170). Among these, a total of 8103 *E. coli* datasets which had corresponding discharge data were thus used for the analysis. *E.coli* data used thus spanned from 2005 to 2013.

stream *E coli* concentrations, particularly with hill country sheep and beef properties.

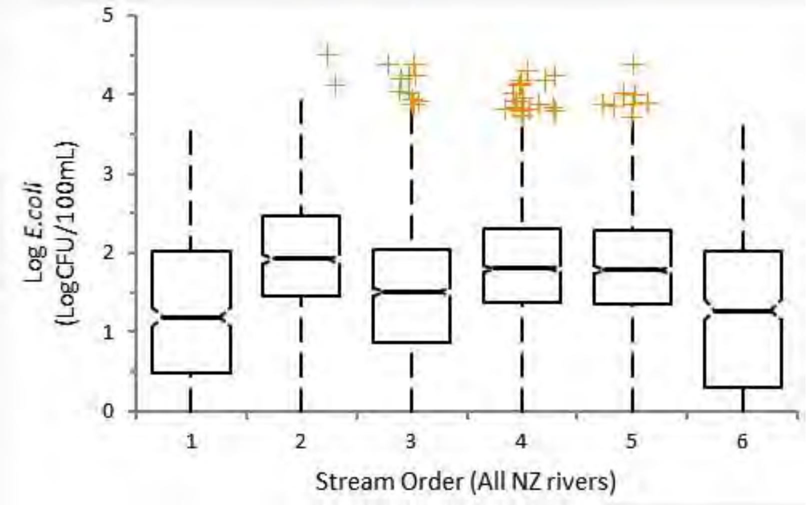

Figure 6: *E.coli* concentrations in New Zealand rivers in relation to stream order designation

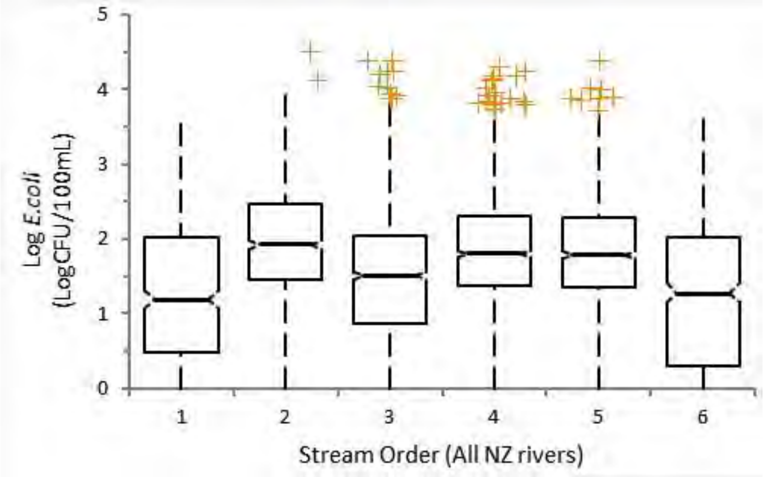

Figure 7: *E.coli* loads in New Zealand rivers in relation to stream order designation

63. I, however, agree with the arguments of Ritchie and Donnison (2010)[57], Moriarty (2015)[58] and Monaghan et al. (2010[59]) that the short-term and immediate effects of direct deposition in smaller lowland streams cannot be discounted. This is particularly so because, from the health risk point of view, direct faecal deposition could still be important given that it occurs at base flows when there is less dilution, and when downstream use is more likely. Also, in-stream faecal deposition delivers viable pathogens directly to water, with no land-based die-off effects thus leading to an erratic elevation in *E.coli* levels.

64. Although exceedances are also associable with low flow river discharge conditions, elevated *E.coli* levels are more pronounced during storm flow discharge conditions for rivers and tributaries in the Waikato region (Figure 8). In Figure 8, it is however, difficult to decipher from an analysis of discharge conditions versus FIB concentrations, what factor (direct stream deposition, over land flow, etc.) is responsible for elevated *E.coli* concentrations in the receiving water (i.e. sites identified in - Table 3.11.1). While it may be convenient to statistically analyse ‘box plots’ of *E.coli* concentrations under varying land use and river discharge scenarios, and posit that ‘higher’ *E.coli* concentrations observed in New Zealand streams are due to a particular factor/source, the *E.coli* concentrations may actually be confounded by *E.coli* from other hitherto unidentified sources (such as non-faecal environmental sources highlighted paragraph 29).

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[57] As previously cited

[58] As previously cited

[59] Monaghan R, Semadeni-Davies A, Muirhead R, Elliott S, Shankar U 2010. Land use and land management risks to water quality in Southland. Report prepared for Environment Southland. Invermay, AgResearch

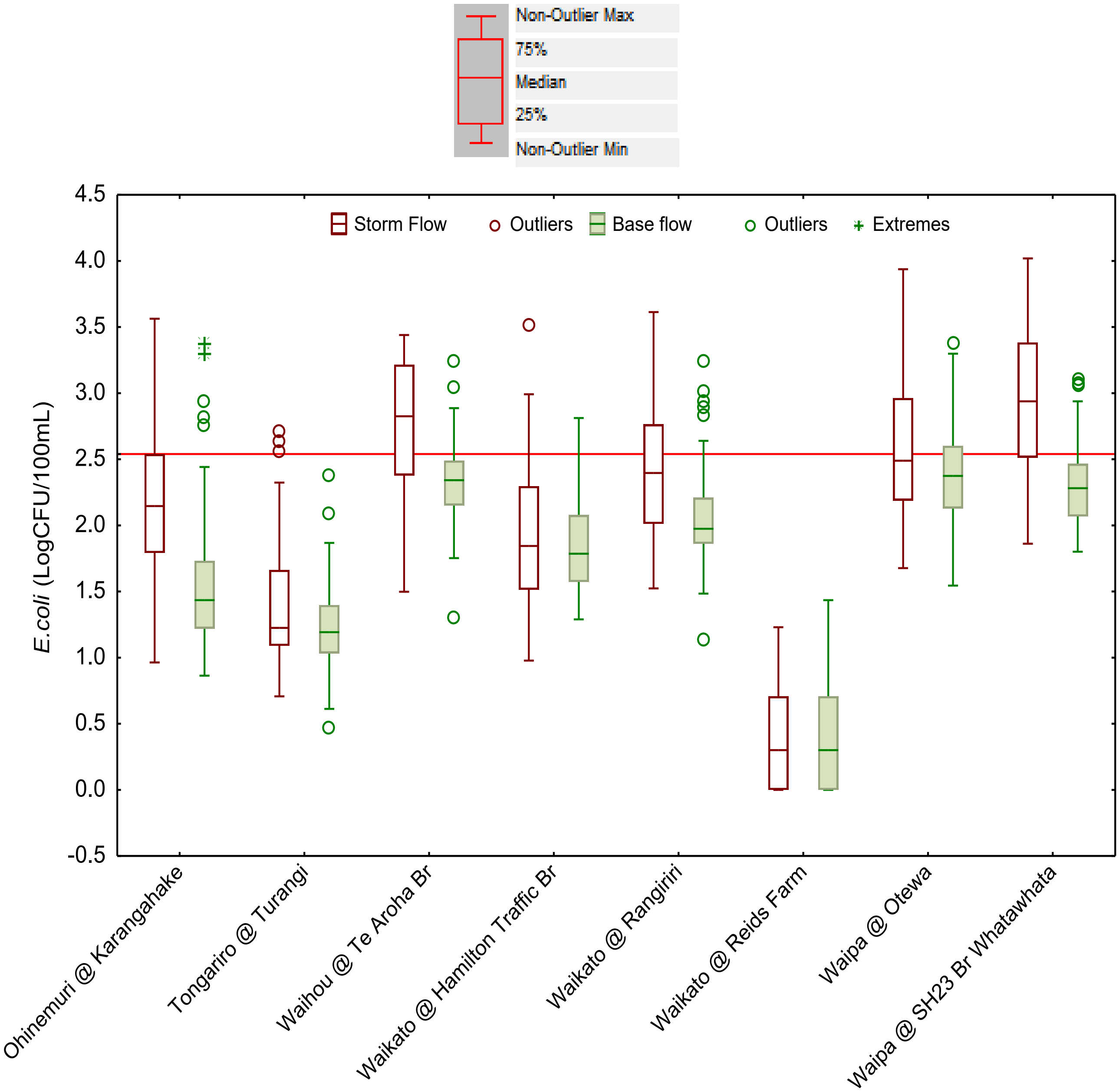

Figure 8: Box plots of *E.coli* concentrations during baseflow and storm flow conditions, Waikato Region waterways, 2007-2013. Red horizontal line is the 540 CFU/100mL *E.coli* threshold

65. Summarily, 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, 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 streams (listed in Table 3.11.1 in the WRPC1) that run through agricultural catchments. I therefore 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-specific management intervention rather than a blanket approach to effect fences for stock exclusion which only stops direct deposition.

SHORT & LONG-TERM *E.COLI* TARGETS STATED IN PC1 TABLE 3.11-1

66. Table 3.11-1 in the PC1 sets out the *E.coli* concentrations to be achieved by actions taken in the short-term and long term (at 80 years) for rivers and tributaries. I note that these projected reductions are generally less than[60] 10% reductions for the short term but could be as high as 2000% reduction for the 80-year reduction target. For instance, Mangakotukutuku Stream which currently has a base 95th percentile of >12,000CFU/100mL has a long-term target of 540 CFU/100mL.

67. From a technical (microbiological) perspective, I am of the opinion that these targets related to *E.coli* reductions at the freshwater sites listed in WRPC1 are ambitious, unrealistic, and unecessary, and they present a cart 'before the horse' approach. Management options applied for the mitigation of *E.coli* in the PC1 need to be site-specific and this would be dependent on the successful execution of a reliable microbial source tracking (MST) study at each site to determine the contributory source of faecal pollution.

68. Currently, it is not known for certain what the sources of faecal pollution are for these streams and rivers, yet ambitious declarations are made to drastically reduce *E.coli* levels to certain levels (up to 2000% anticipated reduction for some streams). Only when we cross over the first milestone of reliably answering the teething question related to sources responsible for elevated bacteria levels at each site, can we begin to identify an appropriate

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[60] or equal to

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.

69. 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? While for some sites, it may be unreasonable to commit financial resources to erecting wired fences when the cause of elevated *E.coli* levels is mainly as a result of wildlife faecal deposits during low flows and overland flow during wet events, for some other sites, erecting barriers to prevent direct access to animals during low flows may actually be needed. To answer these questions, there is the need to commission a carefully designed MST study targeted at these sites. Such study has to be longitudinal, capturing samples collected from different seasons and flow conditions for each identified site in the WRPC1 Table 3.11.1.

70. Also, from a technical perspective, I suggest the need to commission a study that distinguishes if these elevated bacteria levels identifiable for sites listed in PC1 Table 3.11.11 are due to naturalized *E.coli* from the stream bed and channel sediments, which become resuspended following sheer disturbances that allow releases of additional microbial contamination to the water column during low flow conditions. These "naturalized" *E. coli* populations may survive and proliferate[61] in terrestrial (soil) and aquatic environments independent of pollution events (as have been documented in literature[62]). The genetic structure of these naturalized *E.coli* tends to be different from those isolated from animals and often suggesting that they were not recently deposited by animals. "Naturalized" *E. coli* populations could also falsely inflate measurement levels, leading to exceedances of

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[61] i.e. grow

[62] Ishii, Satoshi et al. “Presence and Growth of Naturalized Escherichia Coli in Temperate Soils from Lake Superior Watersheds.” Applied and Environmental Microbiology 72.1 (2006): 612–621. PMC. Web. 3 Jan. 2018

Perchec Merien, A. M. (2014). Naturalization of Escherichia coli in New Zealand freshwater streams (Doctoral dissertation, ResearchSpace@ Auckland).

Ishii, S., and M.J. Sadowsky. 2008. Escherichia coli in the environment: implications for water quality and human health. Microbes Environ. 23:101–108.

available thresholds and suggesting pollution that is present (Devane, 2015)[63]. On the one hand, management options may be targeted towards restricting access to agents (animals or humans) that disrupt streambed sediments[64] during low flow conditions. On the other hand, while access restriction may be possible for animals, humans can also stir up and remobilize bed sediment with its faecal reservoir during contact recreation or food harvesting at base flows. Invariably, elevated concentrations of *E.coli* may continue to be recorded during restricted animal access and low flow conditions at these freshwater sites. A crucial piece of the puzzle thus lies with our ability to decipher by way of phylogenetic studies, if these elevated *E.coli* are due to naturalized *E.coli* and also to assess risks of exposure to pathogens during conditions of elevated levels of naturalized *E.coli.*

71. To shed more light on the arguments above on identifying sources of faecal contamination in waterways before a management solution or target is set, I reviewed the MST results of a recent study (Moriarty 2015)[65] that was completed on five sites with typically elevated concentrations of *E. coli* in the routine Environment Waikato testing (Karapiro, Komakorau, Mangaone, Mangaonua and Mangawhero Streams). These sites are also five out of the 62 sites identified in the proposed plan change (Table 3.11.1). Sampling occurred both during dry weather for 'base-flow' sources and following heavy rainfall. In Mangawhero, during base flow conditions, mean concentrations of *E.coli* was 9933 CU/100mL (higher than the 540CFU/100mL primary contact benchmark). However, further MST investigation under these base flow conditions revealed that wildfowl pollution was the dominant faecal source detected while pollution from ovine and bovine sources was not or rarely detected at Mangawhero Stream (Table 7, Moriarty 2015). Only after heavy downpour (>10mm of rain) was ovine, bovine and wildlife pollution detected, indicating additional pressure from the catchment during rainfall impacted conditions. A similar

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[63] Devane M (2015) The sources of "natural" microorganisms in streams. Client Report CSC15004, Prepared for Environment Southland and West Coast Regional Council

[64] Stock access can also serve to re-charge bed sediment stores of microbes, thereby increasing peak concentrations during rainfall events.

[65] 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

observation was made for samples collected from Mangaone River during baseflow and rainfall impacted conditions, although sheep faecal pollution was not detected under these conditions. Similarly, wildfowl markers were found present in one of three Komakorau Stream samples with extremely elevated *E.coli* concentrations during baseflow conditions. During rainfall-impacted conditions, wildfowl pollution was detected in all samples collected, as well as faecal pollution from humans and ruminants in some of the samples, indicating additional pressure from the catchment during rainfall impacted conditions.

72. Based on the Moriarty (2015) MST results, the high prevalence of wildfowl markers during conditions of low flow (the most critical times for public exposure to health risk) coupled with the comparatively low prevalence of cattle markers during conditions of low flow (Table 5) suggest that pressure due to cattle droppings in these streams during low flow conditions may, in reality, be insignificant compared to wildlife droppings on streams marked in the WRPC1 as having elevated *E.coli* concentrations. Sunohara et al. (2012)[66] found that the cattle exclusion fencing promoted greater numbers and types of plant species and notably greater degrees of wildlife. In another study[67], protecting habitat through cattle exclusion fencing increased inputs of wildlife (C. goose) faecal material significantly, yet where cattle have open access to a stream (where they eat plants, trample soil and plants, etc.), the wildlife faecal markers were significantly reduced in relation to protected upstream sites.

73. The Moriarty (2015) study also reported total coliform and *E.coli* concentrations for the water samples collcted durign the MST study. While the total coliform analysis is not specific to bacteria of faecal origin[68] and

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[66] Sunohara MD, Topp E, Wilkes G, Gottschall N, Neumann N, Ruecker N, Jones TH, Edge TA, Marti R, Lapen DR. 2012. Impact of riparian zone protection from cattle on nutrient, bacteria, F-coliphage, and loading of an intermittent stream. J. Environ. Qual. 41:1301–1314

[67] Wilkes, G., Brassard, J., Edge, T. A., Gannon, V., Jokinen, C. C., Jones, T. H., … Lapen, D. R. (2013). Coherence among Different Microbial Source Tracking Markers in a Small Agricultural Stream with or without Livestock Exclusion Practices. Applied and Environmental Microbiology, 79(20), 6207–6219.

[68] In extreme cases, a high count for the total coliform group may be associated with a low, or even zero, count for faecal coliforms, this would not necessarily indicate the presence of faecal contamination (WHO 1996).

may be related to decaying organic matter surrounding the streams or in the stream bed, the test for *E. coli* is a more specific indicator of faecal contamination due to human sewage or animal droppings which could contain other bacteria, viruses, or disease-causing organisms. Generally lower *E.coli* to total coliform ratios were recorded during baseflow compared to rainfall impacted flow (Table 5) at the five Waikato Streams reported in the Moriarty (2015) study. Without further sampling and analysis to prove otherwise, this results tends to suggest that non-faecal contamination was higher compared to faecal contamination during low flow conditions.

Table 4: ESR *E. coli* and faecal source tracking results for Karapiro, Komakorau, Mangaone, Mangaonua and Mangawhero Streams (adapted from Moriarty, 2015)

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

Table 5: *E.coli*: Total Coliform Ratio of Samples collected during the Moriarty (2015) MST study (adapted from Moriarty, 2015)

Flow conditions	**Sample No-Date**	***E.coli*: Total Coliform Ratio**				
		Karapiro	**Komakorau**	**Mangaone**	**Mangaonua**	**Mangawhero**
Base flow	Sample 1 -4 May	0.03	0.11	0.12	0.20	0.29
	Sample 2 -20 May	0.06	0.10	0.04	0.06	0.01
	Sample 3 - 11 June	0.07	0.09	0.04	0.11	0.06
	Mean	**0.05**	**0.10**	**0.07**	**0.12**	**0.12**
Rainfall impacted flow	Sample 1 - 13 April	0.02	1.00	1.00	0.07	1.00
	Sample 2 - 20 April	0.09	0.30	0.08	0.13	0.12
	Sample 3 - 28 April	0.30	0.29	0.10	0.30	0.13
	Mean	**0.14**	**0.53**	**0.39**	**0.17**	**0.42**

74. Care, however, should be taken in interpreting results from the Moriarty (2015) for decision making with regards to sources of elevated *E.coli* levels in Waikato waterways. The adopted sampling regime was limited in scope and frequency e.g. no sampling was conducted during summer (the most critical times for public exposure to health risk). The study also did not adequately capture considerations for flow in the study design. Instead, it defined baseflow as the period which there is no antecedent 24-hour rainfall greater than 10mm. Depending on the peculiarities of the catchment being considered (e.g., size, predominant land use, etc), what constitutes baseflow to each would differ. For instance, in some catchments, antecedent rainfall of up to 72 hours can impact on the flow of the downstream water bodies, despite the absence of rain in the previous 24 hours before sampling for faecal bacteria. Without any stream flow measurements reported in the MST study, it is difficult to know what flow conditions were referred to in the report as 'during base flow'. Further MST studies are needed that adopt comparative approaches in a way that can reliably inform our understanding on the drivers of *E.coli* variability during

different flow and animal stream access conditions within the Waikato Region[69]. Only upon the successful execution of these source tracking studies shall we be able to inform appropriate management interventions that set realistic and achievable *E.coli* reduction targets for these streams.

75. Based on the above-mentioned, I recommend that:

(a) Site-specific management options, which is supported by flow-specific microbial source tracking (MST) studies at each site to determine the contributory source of faecal pollution, be applied for the mitigation of *E.coli* in the streams listed in the WRPC1. At the phylogenetic level, these studies will help to distinguish if these elevated bacteria levels identifiable for PC1 sites are due to faecal sources or non-faecal environmental *E.coli* from natural stream processes. Currently only 5 out of the 62 PC1 sites have adopted this approach. Even then, preliminary MST results show that wildfowl is the predominant source of faecal indicator bacteria in the streams and that cattle markers only become prevalent following heavy rainfall impacted (i.e. surface run-off and overland flow) conditions. Results from MST studies for the PC1 sites will then inform appropriate site-specific solutions that will drive down observed peaks in *E.coli* levels;

(b) While further work is undertaken to improve our understanding of the sources of in-stream *E.coli* concentrations in the PC1 sites, authorities can adopt tentative approaches already stated in paragraph 38c in order to meet the requirements of the NPS-FM.

CONCLUSIONS

76. I have within the ambit of available published literature (globally and regionally), as well as region-specic data analysis, presented evidence that supports the following arguments:

(a) The *E.coli* modelling science underpinning the economic modelling used to justify draft PC1 rules associated with very significant uncertainties and hence unreliable. It also does not effectively capture important variables related to sources, fate and transmission

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[69] Technologies to achieve this are available, tests could be easily executed at ESR

pathways of microbial contamination from primary productive land into receiving water

(b) Targets related to *E.coli* reductions at the freshwater sites listed in PC1 are not based on scientific evidence and somewhat ambitious as they present a cart 'before the horse' approach. Management options applied for the mitigation of *E.coli* in the PC1 need to be site-specific and this would be dependent on the successful execution of reliable microbial source tracking studies at each site to determine the contributory source of faecal pollution.

(c) *E.coli* does not reliably predict the presence of all types of zoonotic pathogens associated with primary productive land. Also, not all FIB are from faecal sources, hence non-fecal environmental sources of FIB confound *E.coli*-pathogen correlations in streams. These uncertainties suggest the need to be cautious when determining *E.coli* targets as stated in Table 3-11.1 and associated interventions on land use.

(d) Until such time as reliable microbial source tracking is undertaken I propose that long term targets should be deleted from Table 3.11-1 given the myraids of uncertainties associated with the PC1.I also propose that the *E.coli* freshwater objectives be included in Table 3.11-1 in a way that meets the requirements of the NPS-FM. For instance, short term targets could be amended to include a combination of median and 95th percentile *E.coli* concentrations rather than a reliance on the single 95th percentile as it is currently in the PC1 Table 3.11-1. In this way, authorities can work towards a more realistic short-term target that is hinged on improvements in the NPS-FM attribute state of the P1 sites.

(e) Considering that surface runoff is the major source of faecal pollution from agriculture in the Waikato Region, as opposed to direct defaecation in streams, the proposed fencing rules are unlikely to be cost-effective in reducing the delivery of E. coli to Waikato water ways.

Christopher Dada
15 February 2019

APPENDIX 1: SUGGESTED ALTERNATIVE *E.COLI* TARGETS FOR PC1 SITES

			Current PC1 approach				Suggested PC1 Approach		
			Short term	80 year target	Base level		short term		
Catchment number	Catchment description	Site	95th percentile (E. coli/100mL)	95th percentile (E. coli/100mL)	95th percentile (E. coli/100mL)	Current NPS FM Attribute State	median	95th percentile (E. coli/100mL)	Short term target NPS-FM attribute state
73	Upper	Waikato River Ohaaki Br	70	70	70	Green	≤130	<540	Blue
66	Upper	Waikato River Ohakuri Tailrace Br	15	15	15	Green	≤130	<540	Blue
67	Upper	Waikato River Whakamaru Tailrace	60	60	60	Green	≤130	<540	Blue
64	Upper	Waikato River Waipapa Tailrace	162	162	162	Green	≤130	<540	Blue
74	Upper	Pueto Stm Broadlands Rd Br	92	92	92	Green	≤130	<540	Blue
72	Upper	Torepatutahi Stm Vaile Rd Br	216	216	216	Green	≤130	<540	Blue
65	Upper	Waiotapu Stm Homestead Rd Br	281	281	281	Green	≤130	<540	Blue
69	Upper	Mangakara Stm (Reporoa) SH5	1584	540	1700	Red	≤130	<1200	Yellow
62	Upper	Kawaunui Stm SH5 Br	2335	540	2534	Red	≤130	<1200	Yellow
58	Upper	Waiotapu Stm Campbell Rd Br	18	18	18	Green	≤130	<540	Blue
59	Upper	Otamakokore Stm Hossack Rd	680	540	696	Green	≤130	<540	Blue
56	Upper	Whirinaki Stm Corbett Rd	98	98	98	Green	≤130	<540	Blue
54	Upper	Tahunaatara Stm Ohakuri Rd	783	540	810	Green	≤130	<540	Blue
57	Upper	Mangaharakeke Stm SH30 (Off Jct SH1)	684	540	700	Green	≤130	<540	Blue
70	Upper	Waipapa Stm (Mokai) Tirohanga Rd Br	1147	540	1214	Red	≤130	<1200	Yellow
71	Upper	Mangakino Stm Sandel Rd	251	251	251	Green	≤130	<540	Blue
49	Upper	Whakauru Stm SH1 Br	2106	540	2280	Red	≤130	<1200	Yellow
48	Upper	Mangamingi Stm Paraonui Rd Br	2151	540	2330	Red	≤130	<1200	Yellow
45	Upper	Pokaiwhenua Stm Arapuni - Putaruru Rd	1363	540	1454	Red	≤130	<1200	Yellow
44	Upper	Little Waipa Stm Arapuni - Putaruru Rd	1377	540	1470	Red	≤130	<1200	Yellow
33	Middle	Waikato River Narrows Boat Ramp	340	260	340	Green	≤130	<540	Blue
25	Middle	Waikato River Horotiu Br	774	540	800	Green	≤130	<540	Blue
32	Middle	Karapiro Stm (Hickey Rd Bridge)	4518	540	4960	Red	≤130	<1200	Yellow
35	Middle	Mangawhero Stm (Cambridge- Ohaupo Rd)	2920	540	3184	Red	≤130	<1200	Yellow
29	Middle	Mangaonua Stm Hoeka Rd	6372	540	7020	Red	≤130	<1200	Yellow
31	Middle	Mangaone Stm Annebrooke Rd	2052	540	2220	Red	≤130	<1200	Yellow
30	Middle	Mangakotukut uku Stm Peacockes Rd	11394	540	12600	Red	≤130	<1200	Yellow
28	Middle	Waitawhiriwhiri Stm Edgecumbe Street	5922	540	6520	Red	≤130	<1200	Yellow
23	Middle	Kirikiriroa Stm Tauhara Dr	2124	540	2300	Red	≤130	<1200	Yellow
20	Lower	Waikato River, Huntly-Tainui Br	1944	540	2100	Red	≤130	<1200	Yellow
9	Lower	Waikato River, Mercer Br	1494	540	1600	Red	≤130	<1200	Yellow
4	Lower	Waikato River, Tuakau Br	1584	540	1700	Red	≤130	<1200	Yellow
22	Lower	Komakorau Stm, Henry Rd	3474	540	3800	Red	≤130	<1200	Yellow
17	Lower	Mangawara Stm Rutherford Rd Br	4955	540	5446	Red	≤130	<1200	Yellow
19	Lower	Awaroa Stm (Rotowaro) Sansons Br @ Ro	1800	540	1940	Red	≤130	<1200	Yellow
14	Lower	Matahuru Stm Waiterimu Road Below, Con	6147	540	6770	Red	≤130	<1200	Yellow
16	Lower	Whangape Stm Rangiriri-Glen Murray Rd	584	540	589	Green	≤130	<540	Blue
12	Lower	Waerenga Stm SH2 Maramarua, Taniwha R	5098	540	5604	Red	≤130	<1200	Yellow
8	Lower	Whangamari no River Jefferies Rd Br	4712	540	5176	Red	≤130	<1200	Yellow
2	Lower	Mangatangi River SH2 Maramarua	5567	540	6126	Red	≤130	<1200	Yellow
1	Lower	Mangatawhir i River Lyons Rd	5108	540	5616	Red	≤130	<1200	Yellow
68	Lower	Waipa River Mangaokewa Rd	2417	540	2626	Red	≤130	<1200	Yellow
60	Lower	Waipa River Otewa	2036	540	2202	Red	≤130	<1200	Yellow
51	Lower	Waipa River SH3 Otorohanga	3289	540	3594	Red	≤130	<1200	Yellow
43	Lower	Waipa River, Pirongia-Ngutunui Rd Br	4441	540	4874	Red	≤130	<1200	Yellow
34	Lower	Waipa River Whatawhata Bridge	3657	540	4003	Red	≤130	<1200	Yellow
26	Lower	Ohote Stm Whatawhata/Horotiu Rd	2142	540	2320	Red	≤130	<1200	Yellow
36	Lower	Kaniwhaniwha Stm Wright Rd	1917	540	2070	Red	≤130	<1200	Yellow
38	Lower	Mangapiko Bowman Rd Stm	7074	540	7800	Red	≤130	<1200	Yellow
39	Lower	Mangaohoi Stm South Branch Maru Rd	943	540	988	Green	≤130	<540	Blue
37	Lower	Mangauika Stm Te Awamutu Borough W/S	1008	540	1060	Yellow	≤130	<1000	Green
40	Lower	Puniu River Bartons Corner Rd Br	2790	540	3040	Red	≤130	<1200	Yellow
47	Lower	Mangatutu Stm Walker Rd Br	738	540	760	Green	≤130	<540	Blue
46	Lower	Waitomo Stm SH31 Otorohanga	1453	540	1554	Red	≤130	<1200	Yellow
53	Lower	Mangapu River Otorohanga	4284	540	4700	Red	≤130	<1200	Yellow
52	Lower	Waitomo Stm Tumutumu Rd	2241	540	2430	Red	≤130	<1200	Yellow
63	Lower	Mangaokewa Stm Lawrence Street Br	6224	540	6856	Red	≤130	<1200	Yellow
10	Lower	Whangamari no River Island Block Rd	655	540	668	Green	≤130	<540	Blue
3	Lower	Whakapipi Stm	1773	540	1910	Red	≤130	<1200	Yellow
7	Lower	SH22 Br Ohaeroa Stm	4667	540	5126	Red	≤130	<1200	Yellow
11	Lower	SH22 Br Opuatia Stm	2898	540	3160	Red	≤130	<1200	Yellow
5	Lower	Ponganui Rd Awaroa River	1017	540	1070	Yellow	≤130	<1000	Green