**BEFORE THE CANTERBURY REGIONAL COUNCIL
AT CHRISTCHURCH**

IN THE MATTER   of the Resource Management Act 1991

SUBMITTER   **THE COMMUNITY AND PUBLIC HEALTH DIVISION OF THE CANTERBURY DISTRICT HEALTH BOARD**

SUBJECT   **HEARING GROUP 1 FOR THE PROPOSED LAND AND WATER REGIONAL PLAN**

SUBMITTER NO. 093

STATEMENT OF EVIDENCE OF WENDY WILLIAMSON

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Subject Area: Environmental Microbiology

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1   INTRODUCTION

1.1   My name is Wendy Williamson. I am an Environmental Microbiologist with over 18 years of experience. Since 2006 I have been employed by the Institute of Environmental Science and Research (ESR) at the Christchurch Science Centre.

1.2   I hold a BSc (Hons) and a PhD in Microbiology from the University of Canterbury. I currently provide scientific advice on microbiological issues relating to environmental microbiology and public health particularly on the "three waters" - drinking water, wastewater and storm water. The clients include the Ministry of Health, Public Health Services, regional, district and city councils, and consulting engineers.

1.3   I am a co-author of the 2009 *New Zealand Guidelines for Managing Cyanobacteria in Recreational Waters*, produced jointly by the Ministry for the Environment and the Ministry of Health.

1.4   My work has been presented at national and international conferences. Recent presentations include: greywater diversion from septic tank systems; the survival of indicator organisms (*E. coli*) and pathogens (bacterial and viral) in liquefaction silt; and, the inactivation of viruses in wastewater oxidation ponds.

2   SCOPE OF EVIDENCE

Scope of evidence

2.1.1   This evidence relates to the submission of the Community and Public Health Division of the Canterbury District Health Board (CPH) on the Proposed Canterbury Land & Water Regional Plan (LWRP). The submission is number 093 and various comments and recommendations are made as part of the submission.

2.1.2   My evidence is structured as follows.

   a.   *Cyanobacteria and cyanotoxins: potential impact on animal and human health.* The purpose of this section is to introduce cyanobacteria and the toxins they can produce, and show why human health should be explicitly included when considering the consequences of blooms of cyanobacteria.

   b.   *Public health issues of domestic on-site wastewater: management and failure of domestic on-site wastewater systems.* The purpose of this section of my evidence is to introduce how septic systems treat domestic wastewater including sewage, and to show why clusters of septic systems may pose a risk to human health. I include criteria that could be used to trigger community

discussion about upgrading wastewater treatment, with the workings of how these criteria were arrived at.

3 CYANOBACTERIA AND CYANOTOXINS: POTENTIAL IMPACT ON ANIMAL AND HUMAN HEALTH

3.1 Introduction to cyanobacteria (blue-green algae)

3.1.1 Cyanobacteria (blue-green algae) are ubiquitous environmental photosynthetic prokaryotic organisms with habitats ranging from urban soils and streams to Antarctic soils and volcanic hot springs. They are considered the organisms responsible for the early accumulation of oxygen in Earth's atmosphere. Cyanobacteria often exist where no other vegetation can and are an integral part of most ecosystems, tolerating a range of salinity and temperatures. In addition, they form symbiotic relationships with a number of other organisms (e.g., lichen and *Gunnera*).

3.1.2 Aquatic cyanobacteria are usually either planktonic (in the water column) or benthic (attached to surfaces at the bottom and margins of rivers and lakes). A high biomass of a single or a limited group of cyanobacteria species is usually referred to as a bloom (an algal bloom). These blooms typically form relatively quickly and usually last days and weeks rather than months, depending on weather conditions and water flows. Planktonic blooms in a lake can be moved around the water body depending on the wind and can accumulate at different portions of the lake. A bloom of benthic cyanobacteria usually progresses from covering a few rocks to covering large portions of a river bed in about a week, and as the bloom develops portions of the mats may lift off and accumulate on the river edge where they are readily accessible to animals and people. Canterbury's braided rocky rivers are ideal habitats for benthic cyanobacteria, while several Canterbury lakes have typical planktonic cyanobacteria seasonal blooms (Smith et al. 2012).

3.2 Cyanotoxins

3.2.1 Algal blooms can cause problems for humans and animals. The blooms can cause visual and odour problems and clog filters. When a bloom dies its decomposition can deplete the oxygen in water, which in turn kills fish. However, from a health perspective (human or animal) the greatest problem associated with algal blooms is the toxins that some cyanobacteria genera produce (cyanotoxins).

3.2.2 Cyanotoxins are natural metabolic products. Usually the toxins are retained within the cells of the organisms until the cells rupture or die, at which point the toxin is released into the water. While not all cyanobacterial blooms are toxic/harmful algal blooms, without analytical characterisation it is not possible to distinguish a toxic bloom from a non-toxic bloom.

3.2.3 There are five groups of cyanotoxins considered as priority hazards to human and animal health: anatoxins, cylindrospermopsin, microcystins, nodularins, and saxitoxins (Merel et al. 2010). The toxicity of these is: neurotoxins, which adversely affect function in both developing and mature nervous tissue (anatoxin-a, anatoxin-a(S), and saxitoxins); and hepatotoxins, which cause liver injury (microcystins, nodularin and cylindrospermopsin). Assignment of health effects to specific cyanobacterial species or toxins is often difficult because cyanobacteria species may co-exist in a water body and each species may produce a range of toxins.

3.2.4 A range of cyanobacteria in New Zealand freshwaters have been found to produce toxins. In addition, some cyanobacterial species that are internationally known to produce the toxins but which have not yet been show to produce toxins in New Zealand waters, have also been identified. (Wood et al. 2009).

3.2.5 Cyanobacteria and cyanotoxins are present in surface waters irregularly or seasonally and are usually present at health-relevant concentrations for only short periods. However, the concentration of the cells can increase rapidly, thus toxin concentration can also increase rapidly, and hence monitoring frequencies need to be higher for cyanotoxins than for other chemical contaminants at times when the cells or toxins are more likely to be present.

3.3 Drinking-water

3.3.1 The ideal protection of drinking-water supplies from cyanobacteria and their toxins is to prevent bloom formation. This can be done by catchment management to reduce the input of nutrient, such as phosphates into the water source, by mixing the water in a reservoir, and/or by maintaining a rapid river flow. Substances such as copper sulphate have been used in the past to control bloom formation in source waters, but as their mode of action causes cell lysis (rupture of the cells, which then releases intracellular toxins), these control measures are now generally avoided.

3.3.2 Managing the point of raw water abstraction can greatly reduce the number of cyanobacteria in the raw water. For lakes and reservoirs, changing the depth or location of the abstraction point can allow water to be removed from below the mass

of cyanobacterial cells. Using water booms (as used for oil spills) can keep cyanobacteria from entering the treatment plant intake. However, as cyanobacteria regulate their position in the water column depending on sunlight and nutrient availability, the depth at which fewest cells are likely to be abstracted can vary with time during the day. Furthermore, some species of cyanobacteria are found more commonly deeper in the lakes than other species.

3.3.3 Together, this means that for lakes and reservoirs used as sources for drinking-water, understanding bloom characteristics is very important and this requires multiple depth sampling and monitoring to determine the vertical profiles of cyanobacterial cell density. Cyanobacteria typically sink much lower in the water column during the night to access the cooler nutrient-rich water.

3.3.4 Most Canterbury river water supply intakes are fixed infiltration galleries. Infiltration galleries will achieve some (unknown) reduction in the number of cyanobacteria from entering the treatment plant, and the longer the travel time through the fine gravels from the river at the treatment plant the better the removal of cells (and other particular matter) will be. However, if the gavels are open (low proportion of small gravel and sand) then the natural filtration process will be limited. The effectiveness of infiltration galleries to remove toxins from the water has not been researched and it would be prudent to assume that toxins present in the river water will still be present by the time the water arrives at the treatment plant.

3.3.5 If despite the above interventions, cyanobacteria are present in the abstracted water these cells can be removed by treatment plants in several ways. Conventional water treatment facilities can remove the cells through the flocculation process, which clumps dirt and cells together and removes them from the water by allowing them to settle out under gravity. It is important that this sediment is regularly (daily) removed so that the cells do not die and release their toxins back into the water. The sludge needs to be disposed of so that it is not accessible to people or animals and the toxins cannot leach into water.

3.3.6 In conventional drinking-water treatment, cells that escape the flocculation process are removed by the filters, again with regular cleaning of the filters to ensure the cells do not accumulate and release their toxins. Filter backwash water should be discarded and not returned to the head of works.

3.3.7 Conventional water treatment processes may remove only a small amount or none of the toxins that were present in the source water, or that were released by physical damage to the cells as they passed through the early stages of treatment. However,

most released toxins will remain in the water and will require additional treatment steps to remove.

3.3.8 Canterbury has very few conventional water treatment plants. Most Canterbury drinking-water treatment plants do not have filtration. Therefore, vast majority of Canterbury drinking-water treatment plants do not currently have the ability to remove cyanobacteria cells or treat cyanotoxins.

3.3.9 Toxins remaining in the water can be removed with activated carbon treatment, and in some instances they may be destroyed by oxidising chemicals. It is necessary to have characterised the water to know exactly which toxins are present and in what concentrations, and what other impurities are in the water to ensure that the treatment process has the capacity to treat the toxins against the background of other natural organic (non-toxic) contaminants.

3.3.10 Most Canterbury water treatment plants do not have activated carbon treatment units as permanent water treatment options, nor do they have ozone or superchlorination-dechlorination facilities to treat cyanotoxins. For small systems, operation is too unreliable to risk having high chlorine concentrations entering the reticulation system if using superchlorination to remove cyanotoxins.

3.3.11 For these small plants, when cyanotoxins are confirmed in the river water, then powdered activated carbon could be used, which needs to be sourced and dosed at the correct concentration to manage the hazard, often at short notice.

3.4 Animal poisoning

3.4.1 The first recorded observation of animal poisoning directly attributable to cyanobacteria was in 1878 in Lake Alexandrina, South Australia where toxin(s) produced by *Nodularia spumigena* killed cattle, sheep (death in 1-6 h), dogs (death in 4-5 h), horses (death in 8-24 h) and pigs (death in 3-4 h). (Francis 1878)

3.4.2 Numerous case studies of animal poisoning directly linked through analytical chemistry to cyanotoxins are documented including: waterbirds in Denmark and Japan (in 1993, 1995, respectively); dogs in South Africa and France (in 1994, 2003, respectively); cattle and sheep in Australia (in 2001, 1994, respectively). (Stewart et al. 2008)

3.4.3 In mouse bioassays, which have been used traditionally to screen the toxicity of field and laboratory water samples, cyanobacterial hepatotoxins caused death by liver haemorrhage within a few hours of the acute doses being administered, whereas

neurotoxins caused rapid death by respiratory arrest (within 2-30 minutes). (Chorus and Bartram 1999)

3.4.4 During New Zealand summers there are usually cases of dogs (and sometimes livestock) dying after consuming cyanobacterial mats or drinking water where cyanobacteria are or have recently bloomed. Post-mortem results from dogs have found cyanobacteria in the stomach. It is thought that some dogs may actively seek out and consume benthic cyanobacteria (Stewart et al. 2008), which may be why dogs are over-represented in New Zealand animal deaths attributed to cyanotoxins.

3.5 Human health

3.5.1 The international literature indicates that many people have become ill or some died from exposure to freshwater cyanobacterial toxins. However, there are few instances that can be definitively attributed to drinking water. This is mainly because most people will not drink water that has a scum or smells "off", and generally drinking-water resources are managed to control taste, odour and other algae-related problems.

3.5.2 In a 1979 example, illness was attributed to cyanotoxins in drinking-water: "In November 1979 an outbreak of hepatoenteritis at Palm Island, in northern Queensland, involved 148 people, mainly children, the majority of whom required hospitalization. The outbreak occurred a few days after treatment of Solomon Dam, a drinking-water supply for the island, with copper sulfate to control a dense algal bloom. ... An epidemiological investigation of the incident revealed that only people in households connected to the reticulated water supply from Solomon Dam were affected … From all available evidence it was, retrospectively, postulated that the sickness was related to algal toxicity …" (Hawkins et al. 1985)

3.5.3 In a 1988 example, death was attributed to cyanotoxins in drinking-water: "… a severe gastroenteritis epidemic in the Paulo Afonso region of Brazil ... Some 2,000 gastroenteritis cases, 88 of which resulted in death, were reported over a 42-day period … results revealed that the source of the outbreak was water impounded by the [Itaparica] dam and pointed to toxin produced by cyanobacteria as the responsible agent …" (Teixeira et al. 1993)

3.5.4 It is possible that extended exposure to low levels of cyanobacterial hepatotoxins could have long-term or chronic effects in humans. (Chorus and Bartram 1999)

3.5.5 The toxins themselves do not impart a smell or taste. Therefore, if there has been a bloom in the water body or upstream of a drinking-water abstraction site, it is not

possible to know whether the abstracted water has toxins present without analytical testing. Thus, it is necessary to assume the toxins are present until testing shows otherwise.

3.5.6   Boiling water does not remove or eliminate the toxicity of cyanotoxins.

3.5.7   Symptoms attributed to exposure to cyanobacterial toxins in humans include liver failure, gastroenteritis, dermatitis, headaches, aching, and malaise. These symptoms are consistent with many other causes, and exposure to cyanotoxins is not usually what people think of as the cause of such symptoms in the first instance. Consequently, linking cause and effect is difficult, usually equivocal, more by process of elimination and often well after-the-fact so that toxicity assays may be inconclusive or negative. To the best of my knowledge, cyanotoxin metabolites are not usually assayed in clinical samples for which there are no other causative explanations, indicating under-reporting likely of cyanotoxin exposure.

3.5.8   Data on the effects of cyanobacterial blooms on animal and human health indicates that much more is known about the effects on animals than on humans. The reasons for this include:

- there is no single disease or symptom complex in humans associated with exposure to cyanobacteria
- a specific diagnostic test for cyanobacterial exposure is not available
- diagnosis in humans is frequently a diagnosis made by exclusion of other possible causes
- mild cases are frequently missed, as those affected may not seek medical attention or the attending doctors may not associate the presenting symptoms with previous exposure to cyanobacteria
- toxin identification has been frequently omitted in the human studies.

3.5.9   To the best of my knowledge there have been no human deaths in New Zealand directly attributed to cyanotoxin exposure and I am not aware of any illness in New Zealand unequivocally attributed to cyanotoxin exposure.

3.6   Conclusion to cyanobacteria

3.6.1   Humans should be included in the *Canterbury Land and Water Regional Plan* when planning for the management of cyanobacteria.

3.6.2   Cyanobacteria are natural and important components of terrestrial and aquatic ecosystems. They generally only become problematic to us when their cell density is (or has been) high in water we wish to use for recreation or for human or animal

drinking-water. The problem is because some species of cyanobacteria sporadically and seasonally produce toxins that contaminate water. These toxins can be difficult to remove by most conventional treatments and if consumed can cause severe adverse health effects.

3.6.3   In general, Canterbury does not have conventional drinking-water treatment in place, so there is almost no ability to remove toxins from drinking-water if these toxins are present in the water off-take. This means that catchment protection is the tool available for protecting humans and animal health from cyanotoxins.

4   PUBLIC HEALTH ISSUES OF DOMESTIC ON-SITE WASTEWATER: MANAGEMENT AND FAILURE OF DOMESTIC ON-SITE WASTEWATER SYSTEMS

4.1   Introduction to on-site sewage management

4.1.1   One of the greatest advances in public health and for the improvement in the health status of New Zealanders has been the provision of safe drinking-water and the separation of people from their wastes through wastewater treatment. Treatment of wastewater is essential to reduce the chance of people being exposed to pathogens and to ensure that soils, surface water and groundwater do not become receiving bodies for pathogens, excess nutrients and other contaminants typically associated with wastewater.

4.1.2   Many New Zealanders live beyond reticulated and centralised sewerage treatment systems, and therefore, on-site treatment is needed. Septic systems are the typical way in which on-site treatment takes place. A basic individual domestic septic system is composed of two parts: (1) a septic tank, and (2) a disposal field. The septic tank's primary purpose is separation of solids from liquid and the degradation of the solid matter. The disposal's field is where the wastewater is slowly distributed over a large area to allow contact with the soil.

4.1.3   Most of the wastewater treatment occurs during passage of wastewater through the soil and subsoil. During this passage the effluent is treated by soil microorganisms and it is the bio-physical properties of the subsoil that removes or inactivates pathogens and nutrients before they enter a receiving environment.

4.1.4   Overtime, the sludge in the septic tank increases and needs to be removed. If this is not done regularly (3-5 years), then the storage capacity of the septic tanks is reduced (displaced by sludge) and the retention time of the wastewater is too short

for organic matter to settle or be decomposed. When this happens, the wastewater passes through the septic tank quickly and enters the disposal field still containing particular matter and fats. Particular matter and fats block outlet nozzles and is one cause of failure of the disposal field. Also, if the flow rate through the septic tank is too fast, the water holding capacity of the receiving soil may be exceeded and surface flooding by untreated wastewater may occur.

4.1.5   Therefore, all three components (tank, disposal field and soil passage) function together to effect wastewater treatment. The septic tank and the disposal field remove only a small proportion of microorganisms that may be present in the wastewater, most removal will occur during the passage through the subsoil.

4.1.6   If the wastewater moves too quickly through the soil (as may happen in Canterbury's alluvial soils), then there may be insufficient time for either pathogens or nutrients to be removed before the plume reaches groundwater.

4.1.7   The standard that relates to the design and installation of domestic on-site wastewater management is AS/NZS 1547:2012. There is currently no National Environmental Standard for wastewater systems, nor any national "warrant of fitness" that monitors or regulated whether on-site wastewater systems are effective.

4.2   Failure of on-site systems

4.2.1   Septic system failure typically occurs one of two ways catastrophic or cumulative.

- o   Catastrophic is a more immediate event and can include odour and breakthrough of sewage to the soil surface.
- o   Cumulative failure is the progressive contamination of ground and surface waters by microorganisms of sewage origin.

4.2.2   Cumulative failures are often difficult to detect until the consequences are severe, by which time remediating the receiving environment is difficult.

4.2.3   A range of factors may contribute to the failure of on-site wastewater systems and based on ESR research there is no one consistent cause of failure. Reasons it can occur include:

- o   Lithology (of underlying landforms) that limits system placement
- o   Poor subdivision planning
- o   Sections too small
- o   Poor drainage leading to inconsistent sorption of effluent and effluent running off

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- o Too rapid drainage, where the wastewater receives too little treatment during its passage through the subsurface
- o Thin soils that provide too little interaction with soil microorganisms and have too little water holding capacity
- o Under sizing of drainage trenches (especially for aerated wastewater treatment systems, which may be because of under estimating the need to still remove the same volume of water from a home with a secondary treatment system as with a primary system)
- o Poor storm water control
- o Unsuitable systems installed for the conditions
- o Poor installation.

4.2.4   User contribution to failures included

- o Aging tanks cracking
- o Drainage fields blocking either through aging or through too much organic matter/grease entering the disposal field
- o Poor operation and maintenance (such as not cleaning filters and not getting the tank pumped out every 3-5 years)
- o Lack of education of home occupiers about operating on-site wastewater systems
- o Poor water-use behaviours
- o Addition of devices such as hot tubs and other high water use devices can cause periodic overloading
- o Use of incompatible cleaning or personal hygiene products
- o Hydraulic overloading of the disposal area.

4.3   Separation distances and system design

4.3.1   A wide range of disease-causing microorganisms (pathogens) may be present in sewage, with the main pathogens being bacteria, viruses and protozoa. For individual households and small communities, pathogen presence in wastewater will be sporadic and is dependent on the level of infection in the people contributing to the sewage. In the soil environment, wastewater bacteria will die off more quickly than wastewater viruses. This is because bacteria are metabolically active organisms and require to food and moisture to retain their viability. Viruses are intracellular parasites and are biologically inert in the environment until they encounter a new host and initiate a new infection. Virus inactivation is mainly by physical means such as abrasion that damages them, with a minor inactivation mechanism being consumed

as food by other microorganisms in the soil, vadose zone (the vadose zone is the area under the soil layer and before the groundwater) and aquifer.

4.3.2   Viruses in sewage represent a high risk to public health because they cause infection and illness at very low concentrations of the viruses. Pathogenic viruses are shed (primarily in faeces) from infected people in exceptionally high concentrations and continue to be shed for some time following an illness (i.e., when symptom are no longer present). In addition, people may or may not present as being ill – a person could be asymptomatic or have low-level illness but still shed infective viruses in faeces (that is, they have "just a bit of an upset tummy"). Children can continue to shed infective viruses for weeks or months post-symptoms.

4.3.3   Viruses can be present when bacterial indicators such as *E. coli* are no longer detectable, because they are environmentally more robust.

4.3.4   For managing wastewater a precautionary and risk-based approach can be based on the potential for exposure to viruses.

4.3.5   The removal of viruses from the septic tank part of a septic system is usually less than 1-log_{10}. Where 1-log_{10} is equivalent to 90% removal, 2-log_{10} removal is 99% removal, 3-log_{10} removal is 99.9%, 4-log_{10} removal 99.99%, etc.

4.3.6   Log-reduction of viruses in the immediate disposal area (gravel or sand around disposal pipes) is less than 0.5-log_{10}. Therefore, most virus removal needs to occur during the wastewater passage through the subsurface, and this passage needs to be sufficiently slow (long) to allow the inactivation of pathogens to occur within the boundaries of a property (remembering that home owners cannot actually see where this is happening, which is why the standards and other guidance documents have been produced).

4.3.7   Viruses are typically present at about 10^9 or more per gram faeces (i.e., a thousand million per g faeces) from an infected person. People produce about 100 to 250 g faeces per day, which means septic tank effluent can contain very high concentrations of viruses when people are shedding pathogens. For several viruses, including norovirus and rotavirus, fewer than 10 viruses can cause infection.

4.3.8   Moore et al. (2010) have produced guidelines for estimating separation distances from septic tanks to drinking-water wells, aiming for virus reduction at drinking-water to achieve no viruses present. (Note: the *Draft Guidelines for Drinking-water Quality Management for New Zealand 2005* state: "… *it should be understood that if they [viruses] are specifically sought, they should not be detected* …").

4.3.9 Moore et al. (2010) use target $\log_{10}$ reductions for rotavirus or hepatitis A virus because both are waterborne viruses and pathogenic to humans, with rotavirus being one of the more infectious waterborne viruses, while the long-term consequences from contracting hepatitis A are greater than from rotavirus (in New Zealand).

4.3.10 Separation distances calculated based on rotavirus will provide satisfactory protection against most waterborne viruses. The reductions required from a septic tank to drinking-water well were calculated to be 16.2 $\log_{10}$ and 11.1 $\log_{10}$ for rotavirus and hepatitis A virus, respectively, based on virus concentration in faecal matter, shedding and the annual probability of infection of 1×10^{-4} (Table A10 in Moore et al. (2010)). The septic system (tank and disposal field) is assumed to achieve about 2.7 $\log_{10}$-removal, indicating that for rotavirus a further 13.5 $\log_{10}$-removal is required in the soil, sub-soil, vadose zone and aquifer before the abstraction point.

4.3.11 To achieve this reduction for a gravel vadose zone and a gravel aquifer (such as in Canterbury), it is estimated that there needs to be more than 300 m between a septic tank and a drinking-water bore of 60 m deep to achieve satisfactory removal of viruses for drinking-water consumption (log reduction Table 1, Moore et al. (2010)).

4.3.12 The calculations in Moore et al. (2010) assume a single effluent plume. In most small settlements multiple plumes are produced, and it is likely that these combine. If effluent plumes intersect, then the time and distance required to inactive viruses progressively increases; as more plumes combine, their collective load of pathogens increases and so will the treatment distance and time. Therefore, lot size is critical to ensure the pathogens in the wastewater are completely treated (inactivated or removed) within a property's boundaries so that cumulative pathogen loadings do not enter surface or ground water.

4.3.13 Good design and best practice in planning for long-term on-site wastewater management includes a reserve disposal area that can become operational when the primary disposal field becomes blocked or is otherwise no longer treating pathogens within the property's boundaries. The reserve disposal area needs to be managed for the lifetime of the property in exactly the same manner as the primary field. New Zealand Ministry for the Environment surveys show that 15 to 50% of New Zealand's on-site wastewater systems are failing at any one time (Ministry for the Environment 2008).

4.3.14 There is currently no information from manufacturers on the performance of aerated/secondary treatment on-site systems for removal of viruses. In the absence

of that knowledge, a precautionary approach means I assume there is minimal reduction in viruses from these septic tanks. This assumption is supported scientifically, as national and international literatures shows that viruses persist in the environment longer after bacteria.

4.4   Mitigation of public health issues

4.4.1   The link between exposure to pathogens encountered in our environment and actually getting sick is difficult to establish unambiguously, and why a risk-based approach to protecting public health is considered necessary. It can be very difficult for lay people to make the link between exposure to microbial pathogens and the consequences of that exposure. The onset of symptoms may be 24 to 72 hours after exposure, in some cases several weeks later.

4.4.2   Epidemiological studies rely on people remembering what they were doing before they presented with symptoms. An incidental, and probably unrecognised, exposure to poorly treated wastewater is unlikely to be remembered and therefore not documented. Without the pathogen exposure being accurately recorded, the link between wastewater exposure and illness is extremely difficult to determine or to distinguish from person-to-person exposure.

4.4.3   Failure of septic systems is a serious health and environmental hazard and can lead to one or more of the following:

  o Spread of infectious diseases
  o Breeding of mosquitoes and attraction of flies and rodents
  o Nuisance and unpleasantness (e.g., objectionable smells)
  o Pollution and contamination of waterways, beaches, streams and shellfish beds
  o Contamination of bores, wells, and groundwater
  o Alteration of the local ecology.

4.4.4   Section T5.2 of AS/NZS 1547:2012 (page 200) provides some practical ways in which home occupiers can ensure that their on-site system is able to operate optimally, however none of those suggestions will be sufficient if the system people have is inadequate for the conditions.

4.4.5   The rules proposed by CPH for deciding whether a community should be considering reticulation of their wastewater are aimed at estimating the risk to human health arising from a cluster of septic systems and are practical and informative.

4.4.6   Reticulation of wastewater to a central treatment plant or to a decentralised system with disinfection is the best protection for public health from wastewater-borne pathogens. For small communities situated too far from a centralised wastewater treatment plant, decentralised wastewater treatment plants are also a viable option.

4.5   Suggested criteria for considering reticulating a small community

4.5.1   The criteria suggested below are derived by considering Darfield, Canterbury, as an example of a community currently using on-site systems to manage its wastewater.

4.5.2   I am suggesting some initial criteria for considering reticulation of a community's wastewater that is triggered when the cumulative effects from clusters of septic systems may represent risks to human health. Establishing these criteria was carried out by Dr Lee Burbery and me using a combination of estimations and consideration of vulnerable groups within the community. My evidence includes our combined discussion and Dr Burbery's calculations.

4.5.3   The method used is a simple mass balance approach (Hantzche and Finnemore 1992). The concept is that the concentration of nitrate-nitrogen leaving the septic system is treated only by dilution, and all of the dilution is assumed to be derived from rainfall (i.e., land-surface recharge). We have assumed that the wastewater-groundwater system is well mixed (which is a simplified assumption).

4.5.4   We have further assumed that:

   i)   nitrogen is mass conservative in the wastewater treatment system (i.e., any N in the wastewater is converted to and is output as nitrate, with no denitrification occurring)

   ii)   N-production = 11.5 g N/person/day

   iii) water use (i.e., water flowing in to septic tank) = 200 L/person/day

   iv)   household occupancy = 2.6 people/unit (as per Darfield 2001 census; which compares to the national average of 2.7)

   v)   30% of rainfall infiltrates as land surface recharge (typical estimates for Canterbury Plains are in the range 20 – 36%, dependent upon climate, plant and soil conditions).
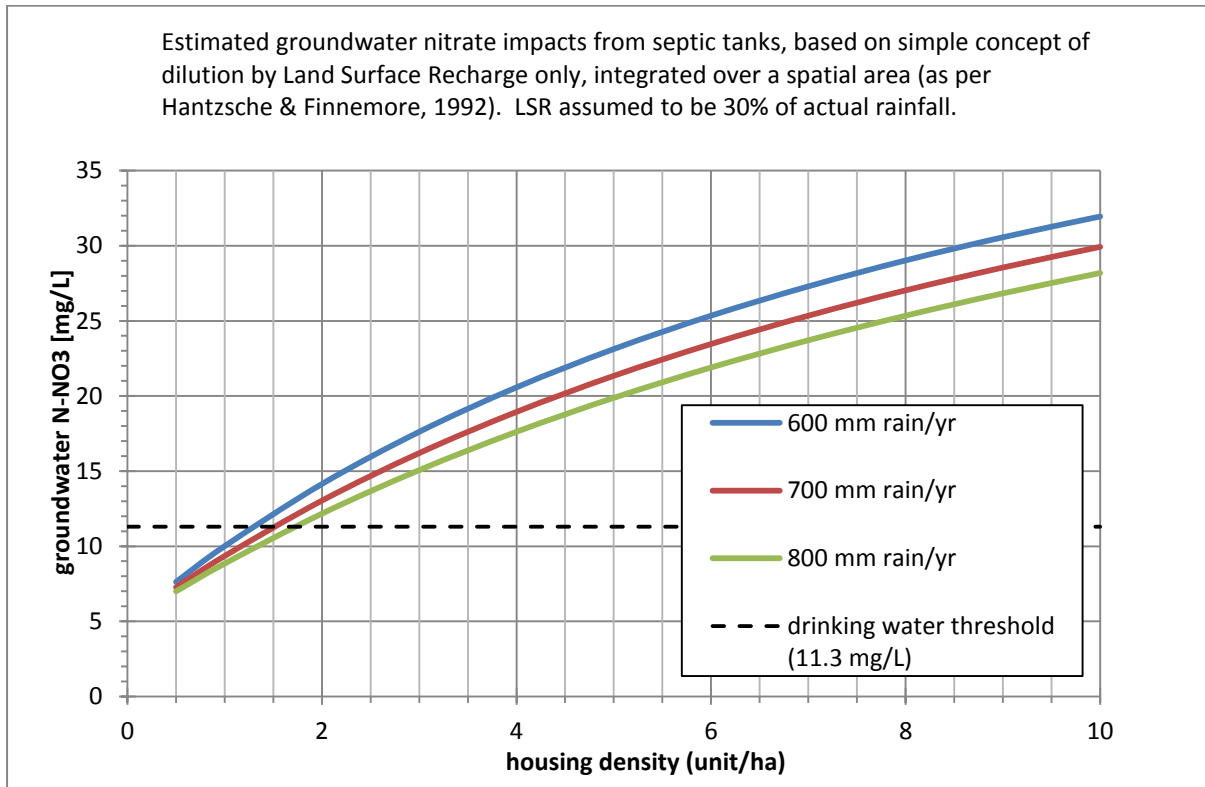
4.5.5   Various additional assumptions:

   vi)   a range of rainfalls have been assessed (600, 700 and 800 mm/year), which are consistent with the Canterbury Plains range of approximately 600 at coast to 1000 mm/year at foothills

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vii)   the concentration of nitrate in the land surface recharge water (i.e., the diluent of any nitrate released from the septic systems is the background N level) has been assessed at two levels: 0 ppm = pure water; and 5 ppm = typical concentration of N in water leached from moderate landuse (similar to the median and average nitrate levels measured in ECAN's 2010 Annual Groundwater Quality Survey)

viii)   that a sector of the community vulnerable to elevated nitrate concentrations are children, especially those under 1 year.
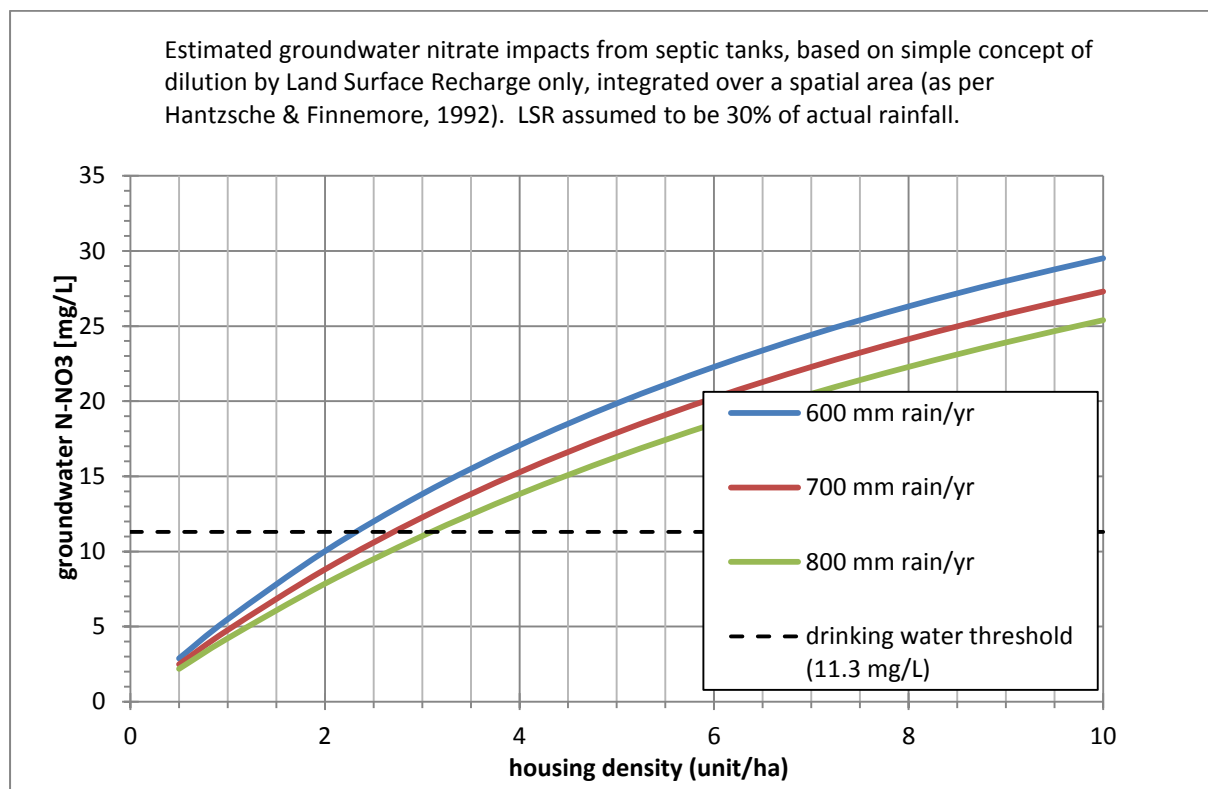
4.5.6   Two graphs were prepared, based on these assumptions. The graphs are used to estimate how many houses per hectare is a maximum when septic systems are contributing nitrate to the environment.

- Graph 1. The graph is used to estimate house density in order to protect the health of the group most vulnerable to nitrate consumption – young children – if there **IS** background nitrogen (such as due to farming activity in the local area).

- Graph 2. The graph is used to estimate house density in order to protect the health of the group most vulnerable to nitrate consumption – young children, if there is **NO** background nitrogen.

Graph 1: Nitrate hazard WITH background nitrate present. The dashed line indicates the drinking-water limit of 11.3 mg N/L. The nitrogen from the dwellings needs to be diluted (by rainfall) to decrease the nitrogen load from households before the nitrogen reaches a drinking-water well to achieve safe drinking-water for people most vulnerable to nitrate (such as young children). The graph indicates that 1.5 houses per ha is approaching the drinking-water limit, while 2 houses per ha requires more rain that is likely to fall to sufficiently dilute the nitrogen contributed from a household. Graph assumed occupancy of 2.6 people per household.

Estimated groundwater nitrate impacts from septic tanks, based on simple concept of dilution by Land Surface Recharge only, integrated over a spatial area (as per Hantzsche & Finnemore, 1992). LSR assumed to be 30% of actual rainfall.

- 600 mm rain/yr
- 700 mm rain/yr
- 800 mm rain/yr
- drinking water threshold (11.3 mg/L)

Graph 2: Nitrate hazard WITHOUT background nitrate. The dashed line indicates the drinking-water limit of 11.3 mg N/L. The nitrogen from the dwellings needs to be diluted (by rainfall) to decrease the nitrogen load from households before the nitrogen reaches a drinking-water well to achieve safe drinking-water for people vulnerable to nitrate. The graph indicates that 2 houses per ha is approaching the drinking-water limit, while 3 houses per ha requires more rain that is likely to fall to sufficiently dilute the nitrogen contributed from a household (except in the highest rainfall area). This indicates that the background concentration of nitrate in groundwater is critical to determine how many septic systems can be located in an area and still protect public health from a nitrate hazard. Graph assumed occupancy of 2.6 people per household.

Estimated groundwater nitrate impacts from septic tanks, based on simple concept of dilution by Land Surface Recharge only, integrated over a spatial area (as per Hantzsche & Finnemore, 1992). LSR assumed to be 30% of actual rainfall.

4.5.7   Septic systems are likely to fail, with estimates of 15% to 50% failing at any one time. There is no reason to assume that Canterbury is different to the national average.

- A population of 500 people, living at a population density of 2.6 people per household would have 192 septic systems. If 20% of these systems are failing by effluent reaching the soil surface (catastrophic failure; 38 systems are failing), the community is exposed to the effluent from about 100 people, some of whom will probably be shedding infectious viruses.

- A population of 1000 people, living at a population density of 2.6 people per household would have 384 septic systems. If 20% of these systems are failing by effluent reaching the soil surface (77 systems are failing), the community is

exposed to the effluent from about 200 people, some of whom will probably be shedding infectious viruses.

4.5.8   In either case (community = 500 or 1000 people), if people are shedding pathogens, then other people in the household or community could be exposed to wastewater pathogens due to failing septic systems. From a pragmatic perspective (what is the rating base to support paying for infrastructure to build and operate a wastewater treatment plant), I considered that a community of 1000 people should be assessing what exposure to pathogens the community is prepared to accept. I have no data to support or refute this "line in the sand".

4.5.9   Therefore, based on Dr Burbery and my discussions and calculations, the criteria that we are proposing for initiating community discussion about upgrading wastewater management are:

   o   when there is an average 1.5 or more domestic dwellings per hectare, *and*

   o   1000 or more people are served by septic systems within the settlement's urban boundary.

4.5.10   Darfield 9as a case study) has a population of about 1,400 people, in about 525 houses (average occupancy 2.6 people per household), and has an urban area of approximately 350 ha (Darfield urban area was estimated to be ~3,500,000 m^2 = 350 ha, using a rough polygon in GIS). This gives Darfield a housing density of 1.5 houses/ha. Thus, Darfield meets the criteria for population and housing density to have discussions about whether the community wants and can afford a reticulated wastewater service.

4.5.11   Should the suggested criteria for considering reticulation be met by a community, discussion with the local Health Protection Officers would help understand options from a public health perspective.

4.6   Conclusion to septic systems

4.6.1   Where individual domestic on-site wastewater systems are (or will be) used, the size (and shape) of sections, the number of houses per hectare, and the soil type are the primary factors that need to be considered to ensure sufficient setback/separation distances from property boundaries and drinking-water sources are available to protect public health.

4.6.2   The criteria I am suggesting for triggering a community discussion about wastewater management are:

- o when there are on average 1.5 or more domestic dwellings per hectare, *and*

- o 1000 or more are people served by septic systems within the settlement's urban boundary.

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