# THE INFLUENCE OF RAIN EVENTS ON THE TRANSMISSION OF *CAMPYLOBACTER* THROUGH WATER SUPPLIES

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by

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# THE INFLUENCE OF RAIN EVENTS ON THE TRANSMISSION OF *CAMPYLOBACTER* THROUGH WATER SUPPLIES

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## SUMMARY

This study is an extension of the survey of *Campylobacter* in treated drinking waters undertaken in 2003–2004 (Nokes *et al.*, 2004). Its primary objectives were to quantify the concentrations of *Campylobacter* in source waters during rain events and to evaluate the extent of removal of the pathogen by full conventional treatment (coagulation/flocculation, clarification, filtration and disinfection) during these events.

Operators of four well-operated treatment plants drawing from source waters considered likely to contain *Campylobacter* were approached to be involved in the study. Staff at the treatment plants collected raw, partially treated and finished water samples eight times during each rain event. The timing of the sampling was at the discretion of the treatment plant staff, with the proviso that they should aim to collect samples before, during, and after the time of peak river turbidity at the plant intake.

Nine rain events were monitored. *Campylobacter* sampling was undertaken during six of the events.

The key findings of the study are:

- 1. Rain events can elevate *Campylobacter* concentrations in water supply source waters. The highest concentration of *Campylobacter* found in source water during this study was 93 MPN/100ml.
- 2. Monitoring of the turbidity, or the river or stream flow, cannot be used as a reliable indicator of when the threat from *Campylobacter* is at its greatest. The arrival of the peaks in *Campylobacter* concentrations relative to the arrival of the turbidity peak is variable and depends on the event.
- 3. A conservative estimate of the overall bacterial removal achievable by full conventional treatment is 7 log. Of this, 3.5 log is due to particle removal processes, showing their importance in the reduction of bacterial concentrations.
- 4. During a rain event full conventional treatment of the water will reduce the probability of illness from *Campylobacter* in a community to very low levels. In the absence of treatment, the estimated level of illness is much greater (ca.  $6 \times 10^5$  time greater if the event lasted three days). Illness levels from drinking untreated water are expected to be overestimates, although the extent of the overestimate is unknown.
- 5. *Under some conditions*, achieving an *E. coli* concentration of less than 1 *E. coli*/100ml *may* not reduce the disease burden in a community to a level below the WHO's health outcome target.

## IMPLICATIONS FOR WATER SUPPLIERS

There are several implications of this study for water suppliers. Some are already known but are reiterated here because of the evidence this study provides to support them.

1. Optimum performance of treatment processes is important during rain events, not only because of the need to removal particulates and ensure that levels of indicator bacteria, i.e., *E. coli*, meet the requirements of the *Drinking-water Standards for New Zealand* (DWSNZ), but because concentrations of *Campylobacter* also

increase during these events. The maximum *Campylobacter* concentration that may arise during rain events is variable, but can be sufficient to cause illness if the concentrations are not substantially attenuated by treatment.

- 2. Peaks in river flow and turbidity are not reliable indicators of the arrival of *Campylobacter* peaks, therefore the period of greatest threat should be regarded as starting from the time when the turbidity begins to rise, and ending as the turbidity approaches its base level.
- 3. The effectiveness of the particle removal processes needs to be maintained during rain events. As well as controlling water turbidity and protozoa, they achieve up to approximately 50% of the total log removal of bacteria accomplished by the treatment plant. Further, their ability to reduce turbidity helps to maintain the efficacy of the disinfection process.
- 4. If treatment fails, rapid remedial actions are necessary to stop the community receiving untreated, or partially treated water. A high probability of illness is expected from consumption of water with the *Campylobacter* concentrations found in the untreated raw water in this study. Other pathogens in the water will add to the risk of illness from inadequately treated water.

# **KEY POINTS FOR CONSIDERATION**

This work leads to the following points for consideration:

1. As part of their public health risk management plans, water supplies with run-ofthe-river abstraction should consider minimising the peaks in turbidity and microbial loadings in their raw water during rain events, by, for example: use of an infiltration gallery; impoundment of their source or installation of off-river storage; or shutting down their intake in the event of turbidity reaching a predetermine level.

These steps, while desirable in all supplies, are most important in supplies that are poorly equipped to cope with changes in raw water quality during rain events.

- 2. A sampling study, in which large sample volumes are collected, to allow:
  - reliable determination of log removal values for unit treatment processes used in New Zealand;
  - collection of improved information about pathogen levels in New Zealand source waters;

would assist in estimating levels of waterborne disease in New Zealand communities by risk assessment. and determining the degree of protection against pathogens afforded by the "<1 *E. coli*/100ml" water quality criterion.

3. Steps to understand the possible limitations of *E. coli* as an indicator of the microbial quality of treated drinking waters would prove valuable in determining when the "<1 *E. coli*/100ml" water quality criterion does not afford satisfactory protection against pathogens. The evaluation of the *E. coli* concentration distribution in the concentration range less than 1 *E. coli*/100ml in the study noted in 2) would assist with this.

## **1 INTRODUCTION**

From February 2003–February 2004 a survey of *Campylobacter* in selected water supplies in New Zealand was undertaken to assess the extent to which treated drinking waters were a possible transmission route for the pathogen (Nokes *et al.*, 2004). Source water in 11 of the 31 supplies included in the survey yielded at least one sample that was positive for *Campylobacter*. *Campylobacter* was not found in any of the samples taken post-treatment (either directly after the treatment plant or in the distribution network), except in one supply. This supply was a small privately-owned camping ground employing disinfection by UV irradiation. Further investigation showed the UV system to have been either poorly maintained, or switched off at the times samples were taken.

The survey concluded that untreated or inadequately treated waters are potential transmission routes for *Campylobacter*, because of the presence of *Campylobacter* in source waters. It also concluded that the risk of infection by waterborne *Campylobacter* is very low provided there are adequate treatment processes in place, and that these are properly operated.

The 2003–2004 survey did not focus on sample collection during rain events. This raised the concern that increased *Campylobacter* concentrations during heavy rains might exceed a treatment plant's ability to remove the pathogen, greatly increasing the risk of campylobacteriosis in the community. The role played by heavy rain in outbreaks of waterborne disease is evident in such incidents as the Milwaukee and Walkerton outbreaks, and others documented in a recent review of outbreaks in developed nations (Hrudey and Hrudey, 2004). An analysis of the outbreaks has led the Hrudeys to conclude that rapid, or dramatic, changes in conditions (including changes in water quality) are important in the occurrence of waterborne disease outbreaks. Water treatment processes do not cope well when operating conditions change, so wherever possible designers attempt to provide some buffering of the source water quality to minimise the threat to treated water quality and people's health.

This project was undertaken to examine the effect of rain events on the performance of treatment plants in New Zealand. The project's objectives were:

- a) to quantify the levels of *Campylobacter* challenging treatment plants during rain events;
- b) to assess the ability of the treatment plants to provide safe drinking water during these times.

## 2 METHODS

# 2.1 Approach overview

A group of suitable source/treatment plant combinations was identified, and, with the assistance of treatment plant staff, a series of samples collected during each rain event from the raw water, partially treated water, and the finished water, as the event progressed. Sampling was timed to try to provide information about the maximum *Campylobacter* concentrations during each rain event, and aimed to collect data before and during peak turbidity. Partially treated samples were intended to provide some indication of the importance of the particle removal processes in reducing bacterial numbers.

# 2.2 Selection of water treatment plants

The study was restricted to a small number of treatment plants. The logistics associated with sampling events that might occur anytime during the day or night precluded the involvement of larger numbers.

Initially, two treatment plants were included in the study (P1 and P2). These were selected on the basis that:

- a) they are well-operated treatment plants (the performance of poorly operated plants would provide no information about the reliability of conventional treatment processes in providing effective barriers to *Campylobacter*);
- b) the 2003-2004 survey had detected *Campylobacter* in 50% of raw water samples in their source waters, thereby providing a guide to the likelihood of finding high levels of *Campylobacter* during this study;
- c) they are sufficiently well-staffed that personnel would be available to collect samples when necessary.

During the latter months of the project, managers at a further two treatment plants, P3 and P4, were approached for assistance to augment the number of events being sampled. These treatment plants were also well-run and well-staffed, but a smaller percentage of their raw water samples collected during the original survey showed the presence of *Campylobacter*.

# 2.3 Sample collection

Treatment plant personnel undertook all sampling. They were requested to collect samples of raw water, partially treated water (preferably after the filters but before chlorination), and the finished water. The collection of partially treated and fully treated samples was staggered after the raw water sample was taken to allow for hydraulic delays through the treatment plant. By doing this all three samples should relate to approximately the same volume of water passing through the system.

Treatment plant personnel were asked to sample the water eight times during each event. It was intended that approximately half the samples should be collected during the increasing river flow, approximately two around or on the peak and two just after the peak flow to confirm that the peak had not been missed. In practice, turbidity levels were used to judge when samples should be taken.

Problems with judging when to take samples and the frequencies at which they should be taken were anticipated during the project's planning. "Test runs" (denoted by a "T") were therefore used, during which only samples for the cheaper indicator organism tests were taken. These were carried out to assist treatment plant personnel in estimating when to start sampling for *Campylobacter* in the "full runs" (denoted by an "F"). The test runs were of limited value, especially when the catchment was large and contained major subcatchments. Rainfall in different parts of such catchments could result in different timings of the arrival of higher river levels at the treatment plant. Consequently, test runs were undertaken at P1 and P2, but not at P3 and P4 to conserve resources for the potentially more informative full runs.

Physicochemical parameters were recorded for each sampling: raw water turbidity, and free available chlorine (FAC) in the fully treated water (as a check that chlorination was satisfactory).

All samples were chilled, and kept in the dark, and whenever possible, they were couriered to laboratories for analysis to start within 24 hours of the sample being taken. This target was met for all indicator samples and most *Campylobacter* samples. Exceedences of the 24-hour target usually arose from difficulties with courier timings. Delays, when they exceeded 24 hours, are recorded in the results given in the Appendix, and can be expected to have resulted in some reported concentrations being less than at the time of sampling.

# 2.4 Sample analysis

Microbiological laboratories routinely used by the water suppliers undertook assays for the indicator organisms (coliforms and *E. coli*). P1 and P3 used their own laboratories and P2 and P4 sent their samples to external laboratories. All laboratories were Ministry of Health recognised, and the methods employed were either Colilert<sup>®</sup> (APHA, 20<sup>th</sup> edition 1998) or membrane filtration (APHA, 20<sup>th</sup> edition 1998).

*Campylobacter* were quantified using a 3 x 3 MPN (most probable number) method at ESR's Public Health Laboratory in Christchurch. Primary enrichment in Exeter broth was undertaken at 37°C for a minimum of four hours, and then at 42°C to give a total incubation time of 48 hrs. A 10% CO<sub>2</sub> incubator provided a microaerophilic atmosphere. Three dilutions of this broth were then incubated, in triplicate, in tubes of Exeter broth at 42°C for 48 hrs. Subsamples from each tube were then looped onto Exeter agar plates and incubated. Presence/absence readings from the plates and reference to 3 x 3 MPN tables allowed enumeration of the *Campylobacter* concentrations.

For the raw water samples, the MPN dilutions were increased by a factor of 10 to reduce the likelihood of *Campylobacter* concentrations exceeding the method's upper quantification limit.

In the first full run (F1) from P3 and all second full runs (F2) from all treatment plants, determinations of the *Campylobacter* species present were made. Exeter plates from samples that were found positive during the MPN measurements were heat blasted to release the DNA, which was then analysed by multiplex PCR.

# 2.5 Treatment processes

All treatment plants that collaborated in this study used full conventional treatment, i.e. coagulation/flocculation, clarification, filtration and final disinfection by chlorine. At P4, chlorine is introduced ahead of the filters to assist in controlling biological growth, but this system was turned off prior to sampling.

# 2.6 Catchment information

2.6.1 <u>P1</u>

P1 draws its water from a medium-sized river (mean annual flow 43.5  $m^3/s$ ), which drains a catchment of 3,510 km<sup>2</sup> most of which is upstream of the plant. It rises in mountains flows firstly through high country tussock land, then through heavily stocked land (sheep, cattle and deer) in its middle and lower reaches. Much of this land was originally wetlands, but extensive drainage, flood control and channel clearance have been undertaken to convert it to productive farmland.

Several large tributaries flow into the river in its middle and lower reaches. Three of these receive considerable discharges of industrial and municipal effluent.

The regional council operates several hydrological monitoring stations along the river. The farthest of these from the treatment plant can provide 20–24 hours warning of an impending increase in river level at the treatment plant.

Data from WINZ indicates that this source water is subject to considerable pollution by animals, and that direct animal access to the river is possible. Heavy rains bring large changes in turbidity.

# 2.6.2 <u>P2</u>

The catchment area of the source water of P2, up stream of the plant intake, is 58 km<sup>2</sup>, and the mean flow (measured immediately up stream of the plant since 1980) is 7.8 m<sup>3</sup>/s. The tributaries that eventually create this source water form above the tree line. The river flows through agricultural and industrial land, and near the treatment plant flows into a small lake. The treatment plant can abstract water either from the lake or directly from the river. A large population of waterfowl on the lake is a potential source of faecal pollution.

The runoff rate in this catchment  $(0.13 \text{ m}^3/\text{s/km})$  is much higher than those of the other catchments in the study. Snowmelt and steeper topography contribute to the high runoff.

Information from WINZ indicates that the source is subject to considerable animal pollution and that stock access to the water is possible.

# 2.6.3 <u>P3</u>

Eighty-four percent of the 149km<sup>2</sup> catchment (125 km<sup>2</sup>) of the source river for P3 lies above the treatment plant. Six main tributaries drain the catchment. Land use in the upper catchment is native forest and regenerating bush. Indigenous forest covers approximately 36% of the catchment, while approximately 8% of the catchment contains exotic forest. Pastoral farming, predominantly sheep and beef farming with a small area occupied by deer farming, occupies 34% of the catchment.

The mean flow (data from 1975–2003) (Waugh *et al*, 2004) at P3 is 4.64 m<sup>3</sup>/s.

Information from WINZ indicates that the source is subject to some animal pollution and that stock access to the water is possible.

# 2.6.4 <u>P4</u>

The river system that provides the water abstracted by P4 consists of two major rivers, one with a catchment of 440 km<sup>2</sup> and the upper catchment of the other covering 1,100 km<sup>2</sup>. Both catchments are long and narrow with their tributaries being short and steep. Both are extensively bush-covered.

The mean flow of the river (data from 1957–1995) is 57 m<sup>3</sup>/s.

Information from WINZ indicates that the source is subject to considerable animal pollution and that stock access to the water is possible. Very large changes in turbidity occur during rain.

## 3 **RESULTS**

The results, for indicators and *Campylobacter* are discussed separately for each treatment plant.

## 3.1 P1

Data from two test runs (T1 and T2) and two full runs (F1 and F2) are available from P1. The full data sets from these runs are in the Appendix. Plots of the turbidity and bacterial concentrations in the raw water as a function of time are given in Figures 1-4 for T1, T2, F1, and F2, respectively.



Figure 1 Indicator bacteria concentrations and turbidity levels in the raw water during T1 at P1.



Figure 2 Indicator bacteria concentrations and turbidity levels in the raw water during T2 at P1



Figure 3 Indicator bacteria and *Campylobacter* concentrations and turbidity levels in the raw water during Full Run 1 at P1



Figure 4 Indicator bacteria and *Campylobacter* concentrations and turbidity levels in the raw water during Full Run 2 at P1

Initial attempts during T1 and T2 to judge the arrival time of peak turbidities based on the timing of peak river flows past one of the up-stream monitoring stations were unsuccessful. Figs.1 and 2 show that the turbidity peak was not captured in either event, although the coliform and *E. coli* levels in the raw water follow the changes in turbidity.

The river flow during F1 was high (maximum of  $210 \text{ m}^3/\text{s}$  - approximately 4 times that during F2) and although there had been a slight rise in flow during the previous week, it had been almost a month since the previous peak in river flow. Bacterial reservoirs in the catchment and in the river channel, therefore, had not been flushed by heavy rain for a month. The flow during F1 appears to have been sufficient to wash *Campylobacter* from these reservoirs. The river flow showed two peaks during F1. Samples were collected during the second peak in flow. The combined effects of the two peaks may have contributed to the poor correlation between the turbidity level and indicator concentrations (contrary to what was seen in T1 and T2), and the apparent fall in *Campylobacter* concentration despite the increasing turbidity shown in Fig.3.

There was a single peak in flow during F2. The bacterial concentrations (both indicators and *Campylobacter*) during F2 were lower than during F1. This can be explained in part by substantial depletion of bacterial reservoirs as a consequence of a large peak in flow 12-13 days prior (approximately three times the flow of the peak a month prior to F1). In addition, the maximum river flow during F2 was ca.  $62 \text{ m}^3/\text{s}$ . The rain event associated with F2 was therefore smaller than that giving rise to F1, which will have reduced the extent of mobilisation of bacteria remaining in the reservoirs.

Campylobacter concentrations were highest (43 MPN/100ml) in two samples collected during F1. The concentrations during F2 were only just detectable (maximum

concentrations ca. 4 MPN/100ml). None of the finished water samples in either full event contained detectable concentrations of the indicator organisms or *Campylobacter*.

*Campylobacter* speciation in the three positive samples in F2, showed that two samples contained thermotolerant *Campylobacter* that were neither *Campylobacter jejuni* nor *Campylobacter coli*. One sample contained *C. jejuni* only.

# 3.2 P2

Data from only one test run (T1) are available from P2; no full runs were undertaken at this treatment plant. The results are tabulated in the Appendix, and raw water data are shown in Fig.5. At the time of this event, water was being drawn from both the river and lake intakes.



# Figure 5 Indicator bacteria concentrations and turbidity levels in the raw water during T1 at P2

Sampling for this event did encompass peaks in turbidity and indicator bacteria. The increase in turbidity at P2 of 3-4 NTU was much less than the changes seen at the other treatment plants, although the indicator organism concentrations were as high as, and in some instances higher than, those at the other plants.

The coliform concentration increased between the raw water and the point in the treatment plant where the partially treated samples were taken (see Appendix) – a phenomenon not evident at the other treatment plants. This observation had been made previously by the treatment plant staff, and was reproduced in checking the results for this study. The same phenomenon is not seen in the *E. coli* concentrations. At this treatment plant, the partially treated samples were obtained after the clarifiers, rather than after the filters. The treatment plant supervisor believes that organisms attached to small flocs contained in water after the clarifiers probably contribute to the high coliform concentrations measured in these

samples. The increase in coliform numbers, but decrease in *E. coli* numbers, could result from the growth of coliforms of environmental (rather than faecal) origin in the clarifiers.

Neither indicator organism was detected in any finished water sample.

# 3.3 P3

Data from two full events (F1 and F2) are available from P3. The results are given in the Appendix and the raw water data are plotted in Figs. 6-9. For clarity, the indicator and *Campylobacter* data are plotted separately, each with the turbidity for reference.



Figure 6 Turbidity and indicator bacteria levels in the raw water during F1 at P3



Figure 7 Turbidity and *Campylobacter* concentrations in the raw water during F1 at P3



Figure 8 Turbidity and indicator bacteria levels in the raw water at P3 during F2



Figure 9 Turbidity levels and *Campylobacter* concentrations in the raw water at P3 during F2

There was a three-fold increase in turbidity during F1 and a 60-fold increase in turbidity at P3 during F2. The concentrations of indicator bacteria were correspondingly greater during F2 than F1, but the highest *Campylobacter* concentration (93 MPN/100ml) was measured during F1. No *Campylobacter* were detected in any partially treated or finished waters from P3.

The maximum flow during F1 was ca. 7.5  $\text{m}^3$ /s. Eight days prior to F1 there had been a rain event resulting in a peak flow of ca. 14  $\text{m}^3$ /s. This earlier event probably did little to flush the bacterial reservoirs in the catchment and river channel, but the low flow during F1 may have been unable to release high bacterial numbers from the reservoirs.

In contrast the maximum flow during F2 was ca. 70-80  $\text{m}^3$ /s. This flow will have more likely mobilised bacteria and sediment than the much lower flow during F1. Six days prior to F2 there had been a small rain event, which, because of the low flow it created, was unlikely to have greatly depleted bacterial reservoirs. However, 13 days prior to F2 an event leading to a maximum river flow of ca. 160  $\text{m}^3$ /s had occurred. Despite the likely depletion of the bacterial reservoirs during an event of this magnitude, the high levels of organisms in the raw water during F2 show that some replenishment of the reservoirs in the sediment must also have occurred during the 160m<sup>3</sup>/s event, perhaps during its latter stages.

Sampling during F1 and F2 captured the peaks in turbidity and bacteria concentrations. The indicator bacteria peaked slightly in advance of the turbidity in both events. During F1 *Campylobacter* peaked before the turbidity, but during F2, the *Campylobacter* and turbidity peaks virtually coincide.

*C. jejuni* was present in all samples in which *Campylobacter* was detected in both full events. *C. coli* was only present in two samples in each event. These were samples in which the *E. coli* concentration was the highest and the *Campylobacter* concentration was at, or near, peak concentration. No other thermotolerant *Campylobacter* species were detected.

# 3.4 P4

Data were collected from two full events (F1 and F2) from P4. The results are tabulated in the Appendix, and the raw water data from both events are plotted in Figs. 10-12. Data for the coliform measurements are not included in the figures as the concentrations exceeded the highest quantifiable values in both events.

Both F1 and F2 encompassed peaks in turbidity and *E. coli* concentrations (see Figs.10 and 12). The *Campylobacter* concentration also appears to have reached a peak during F2, but the data from F1 are so variable that it is difficult to determine whether a peak in concentration occurred. The peak in *E. coli* during F1 arrived slightly ahead of the turbidity peak, but the *E. coli* and *Campylobacter* peaks during F2 occurred as the turbidity levels were dropping.

Turbidity levels were higher during F1 than F2, but the opposite was true of the indicator bacteria, and highest *Campylobacter* concentrations were found during F2. High concentrations of coliforms and *E. coli* in the raw water resulted in their detection after the filters, but neither indicators nor *Campylobacter* were detected in the treated water from this treatment plant during either event.

The maximum flow during F1 (157 m<sup>3</sup>/s) was very much greater that the flow during F2 (58 m<sup>3</sup>/s). F1 had been preceded by three rain events in the previous 3 weeks, with flows of 295, 99 and 128 m<sup>3</sup>/s respectively. The hydrograph for the month prior to F2, on the other hand, showed a gradually falling river flow with no significant peaks. Bacterial reservoirs prior to F1 were therefore likely to have been depleted by the earlier events, but the high flow during F1 ensured mobilisation of particulates to contribute to the high turbidities. The flow during F2 was too low to lead to the turbidity levels observed during F1, but as there had been no events to deplete the bacterial reservoirs the bacterial concentrations were very much higher.

Prechlorination was turned off during the event to avoid interference with the data obtained prior to filtration. However, the treatment plant supervisor did note that there was still a trace of chlorine present in the system when the first "partially treated" sample was taken during F1. This may have affected bacterial numbers in the first sample taken.

Speciation showed *C. jejuni* present in all samples and *C. coli* was present in three. *C. coli* was detected when the *Campylobacter* concentration was at its highest, or when *E. coli* was at, or close to, its highest concentration. Thermotolerant *Campylobacter* that were neither *C. jejuni* nor *C. coli* were present in four of the eight raw water samples.



Figure 10 Turbidity levels and *E. coli* concentrations in the raw water during F1 at P4



Figure 11 Turbidity levels and *Campylobacter* concentrations in the raw water during F1 at P4



Figure 12 Turbidity levels and *E. coli* and *Campylobacter* concentrations in the raw water during F2 at P4.

#### 4 **DISCUSSION**

#### 4.1 *Campylobacter* in the raw waters

#### 4.1.1 *Campylobacter* concentrations and species

*Campylobacter* concentrations were not quantified during the original 2003-04 survey of *Campylobacter* in drinking water supplies. Presence/absence measurements only were made. One of this study's objectives was to quantify the *Campylobacter* concentrations to assist in understanding the level of risk associated with *Campylobacter* during rain events. A complete record of the *Campylobacter* concentrations is provided in the Appendix. A summary of the maximum *Campylobacter* concentrations measured during each event is given in Table 1.

Treatment Plant	Event	Maximum <i>Campylobacter</i> concentration (MPN/100ml)					
D1	F1	43					
PI	F2	4					
D2	F1	93					
P3	F2	43					
D4	F1	43					
P4	F2	93					

Table 1	Maximum	Camnvlohacter	concentrations	measured in	each full	event
		Campyiovacici	concenti ations	measureu m	cach lun	c v c m

With the exception of the F2 event at P1, the maximum concentrations measured during all the events are remarkably similar given the differences in catchment size and river flows. (F2 at P1 occurred relatively soon after an earlier major rain fall event which probably depleted the *Campylobacter* reservoirs). Too few events were monitored to determine whether this similarity in maximum concentrations might be a more general phenomenon. The Freshwater Microbiology Research Programme (FMRP) (McBride *et al.*, 2002) reported *Campylobacter* concentrations in excess of 110 MPN/100ml in some of the waters studied, but these concentrations were not quantified and may have been of the same order as the concentrations found in this study.

The maximum concentration of 93MPN/100ml found in this study can only be regarded as a lower limit for the maximum concentration that might arise in source waters.

The *Campylobacter* concentrations in source waters will be determined by the balance between:

- i) the total numbers of *Campylobacter* in reservoirs both on the land surrounding the source and in the river or stream channel;
- ii) the intrinsic *Campylobacter* die-off rate and inactivation by environmental factors such as sunlight;
- iii) the efficiency of processes transporting *Campylobacter* into the source water and re-suspending *Campylobacter* in river or stream sediment;
- iv) the extent of dilution by the water flow.

Factor i) depends on the nature of activities in the catchment, the number of faecal sources in the catchment (e.g., live stock), and on the effects of past rain events. The die-off rate is intrinsic to the organism and unaffected by the conditions during the rain event. Factors iii) and iv) are controlled by the rainfall during the event under consideration. It could be argued that there is a limiting maximum *Campylobacter* concentration determined by:

- a maximum number of animals that can graze near a water source and have access to it;
- the depletion of *Campylobacter* numbers in reservoirs by the die-off rate and environmental factors
- the balance maintained between iii) and iv) as both increase with rainfall.

Speciation of the *Campylobacter* showed that *C. jejuni* was the predominant themotolerant *Campylobacter* species present. *C. coli* was detectable in a few samples showing high levels of microbial contamination, i.e., high concentrations of *Campylobacter* or *E. coli*. The original survey also only found *C. coli* in samples with high levels of turbidity or *E. coli*. These observations are consistent with *C. coli* numbers in faecal sources being much lower than *C. jejuni* numbers, and consequently much higher levels of faecal contamination of the water being required to render *C. coli* detectable.

Thermotolerant *Campylobacter* that were neither *C. jejuni* nor *C. coli* were found in a few samples, and sometimes in the absence of *C. jejuni* and *C. coli*. Such species had also been found in the original survey in the absence of *C. jejuni* and *C. coli* and when contamination levels were low. The significance of this is unclear.

# 4.1.2 Relationships between *Campylobacter*, turbidity and *E. coli*

# 4.1.2.1 Concentration correlations

Direct pathogen monitoring is not required by the *Drinking-water Standards for New Zealand* (DWSNZ), and consequently *Campylobacter* assays are not undertaken by water suppliers. *E. coli* and turbidity measurements, on the other hand, are routinely made at water treatment plants, and in the case of turbidity can be rapidly made by in-line probes. If relationships between *Campylobacter* concentrations and the *E. coli* concentrations or turbidity levels can be identified, readily available surrogate measurements (*E. coli* and turbidity) could be used to better understand the risk associated with *Campylobacter* in water supplies.

The question of interest is: "Can an estimate of the *Campylobacter* concentration in the raw water at any time be made from a turbidity or *E. coli* measurement at that time?". If the answer to this question is "Yes", real time estimates of *Campylobacter* concentrations could be made based on the turbidity. Real time estimates could not be made from *E. coli* data because of analytical delays, but these data could assist in retrospective assessments of risk.

To this end, correlations between *Campylobacter* concentration and turbidity, and *Campylobacter* concentration and *E. coli* concentration, were sought from the data sets for the six full events that were monitored. Student's t-Test was used to identify the existence of correlations at the 95% confidence level (Zar, 1996 s.18.2). No linear correlations were found between *Campylobacter* and *E. coli*, and the only linear correlation (r = 0.89) found between *Campylobacter* and turbidity was for the F2 event at P3.

A simple linear relationship between *Campylobacter* and turbidity or *E. coli* that holds for all source waters is not apparent from this study. Moreover, the existence of such a relationship for a specific catchment depends on the rain event. Numerous factors interact before and during a rain event to determine the bacterial and particulate loadings in the water. For this reason, any relationship between *Campylobacter* and the surrogates can be expected to be catchment- and rain-event- dependent, and therefore cannot be identified in advance.

## 4.1.2.2 Relative timing of the turbidity and *Campylobacter* peaks

Examination of Figs.1-12 shows that the peaks in *Campylobacter* (and indicator bacteria) concentrations and turbidity in the raw water reach the treatment plant at different times. There are examples of bacterial peaks preceding the turbidity peak (Figs.3, 6, 7, 8, 10), being approximately coincident with (Figs.4, 5, and 9) and following (Fig.12) the turbidity peak.

Two factors that influence the temporal relationship between the turbidity and *Campylobacter* (and indicator) concentration may explain bacterial peaks preceding the

turbidity peak. The first is the numbers of organisms available for release from reservoirs, both in the surrounding catchment and in the river sediment.

River sediments and faecal matter on land act as reservoirs for bacteria. Rainfall depletes these stores by washing microbes from faecal material on land into waterways, or by increasing river flow and thereby re-suspending microbes contained in channel sediment. Concentrations of micro-organisms in the water column will increase for a while as a result of these processes, but during an extended rain event concentrations will eventually fall as the numbers of organisms available for release from the reservoirs diminish. The reservoirs of clays, silts and sand that contribute to turbidity in the water are more extensive than the bacterial reservoirs. As a result, the timing of turbidity peaks is determined by decreasing rainfall or river flow rather than the particle sources becoming depleted. Increasing levels of turbidity may therefore be accompanied by declining bacterial concentrations.

The arrival of a peak *Campylobacter* concentration before the *E. coli* peak can be explained by the *E. coli* bacteria being more numerous in the environment than *Campylobacter*. This would result in an earlier depletion of the *Campylobacter* reservoirs. The reverse order of peak arrivals cannot be explained in this manner. Uncertainty in the timing of *Campylobacter* peaks, because of substantial uncertainties in the measured concentration of the organism, could lead to the *E. coli* peak appearing to arrive prior to *Campylobacter*.

The second factor leading to bacterial peaks preceding the turbidity peak is dilution. During the early stages of a rain event increasing volumes of water will wash increasing numbers of bacteria into the river with a resulting increase in their concentration. At some point, the increasing volume of water entering the river will outstrip the bacterial input and dilution will start. The effect of dilution on turbidity will not become evident as rapidly because of the more plentiful supply of particulates being carried by the run-off water.

The relative importance of dilution and reservoir depletion in influencing the bacterial concentrations found in this study could not be determined.

The variability in the timing of the arrival of peak bacterial concentrations relative to the arrival of peak turbidities means that monitoring of the turbidity cannot be used as a reliable indicator of when the threat from bacterial pathogens is at its greatest.

# 4.1.2.3 The influence of river flow on *Campylobacter* concentration

River flows, and the accompanying rainfall, are expected influence bacterial concentrations and turbidity in the source waters. As the river flow increases, more material will be resuspended from the river channel, and more contaminants carried in run-off will be washed into the river. Opposing the processes acting to increase contaminant concentrations will be increased dilution, and exhaustion of bacterial reservoirs.

Table 2 gives, for each full event monitored, the river flow and the mean and maximum *Campylobacter* concentrations measured in samples collected during the event. No consistent relationship between river flow and *Campylobacter* concentration is evident from Table 2. At P3 and P4 the higher *Campylobacter* concentrations were found during the events with the lower river flows. The opposite is true at P1.

What has occurred in the catchment prior to the event of interest is likely to play a major role in determining the *Campylobacter* concentrations during the event. Factors such as the intensity and duration of previous events, and the period between these events and the event of interest, will influence the *Campylobacter* concentration. The likely level of *Campylobacter* in the source water during a rain event cannot therefore be estimated using the river flow as the only guide; more sophisticated modelling is required.

Treatment Plant	Event	River Flow <sup>1</sup> (m <sup>3</sup> /s)	Mean <i>Campylobacter</i> Concentration (MPN/100ml)	Maximum Campylobacter Concentration (MPN/100ml)
D1	F1	210	14.6	43
PI	F2	62	2.4	4
<b>D</b> 2	F1	7.5	34.3	93
P3	F2	75	26.0	43
D4	F1	157	28.0	43
P4	F2	58	55.9	93

Table 2River flows, and mean and maximum Campylobacter concentrations for the full<br/>events monitored during the study

Approximate maximum flow during the event

## 4.2 Treated waters

1

## 4.2.1 Treated water quality

Data from the original *Campylobacter* survey showed no detectable *Campylobacter* in finished drinking waters that were properly treated. It was concluded that adequately treated drinking water was not a transmission route for the pathogen. Indicator organisms (coliforms and *E. coli*) and *Campylobacter* were also undetectable in all fully-treated water samples taken during this study, i.e. *E. coli* concentrations were less than 1 *E. coli* MPN/100ml, and *Campylobacter* concentrations were less than 0.3 *Campylobacter* MPN/100ml. This supports the conclusion of the survey.

*Campylobacter* were not detected in any partially-treated sample, but indicator organisms were detected in some partially treated samples, i.e., those collected before chlorination, although their concentrations were much lower than those entering the treatment plant. The detection of some indicator bacteria after the particle removal processes is not unexpected given the high concentrations of indicator bacteria in some raw waters, and the fact that particle removal processes are not intended as the primary treatment barrier to bacteria.

The treatment plants monitored in this study complied with the DWSNZ with respect to *E. coli* on all occasions, despite the degraded quality of the raw water during the rain events, i.e., *E. coli* was undetected in all 100ml samples of fully treated water. The 2005 edition of the DWSNZ does not specify a maximum acceptable value for pathogenic bacteria, and therefore there is no benchmark against which to assess plant performance with respect to *Campylobacter*. It is possible, however, to estimate the likelihood of illness due to *Campylobacter* in the treated water.

## 4.2.2 Treatment plant performance –removal of bacteria

Two pieces of information required for estimating the likelihood of illness due to *Campylobacter* in a water supply are the concentration of the organism in the raw water and the efficacy of the treatment processes. This section discusses the information this study provides about the efficacy of treatment.

To be able to make these calculations, the concentrations of organisms before and after a particular treatment process must be known. This information is available for the indicator bacteria for many of the datasets collected in this study, but because *Campylobacter* was undetectable in all partially and fully-treated samples, no removal efficacies specifically for *Campylobacter* can be calculated. *Campylobacter* is more readily inactivated by chlorine than *E. coli* (Blaser *et al.*, 1986), therefore estimations of the extent of the removal of indicator bacteria should provide a conservative guide to the extent of *Campylobacter* removal.

All treatment plants that participated in this study used full conventional treatment, i.e., coagulation/flocculation, clarification, filtration and disinfection. Samples of partially treated water obtained after the filters<sup>1</sup>, but before chlorination, provide a measure of the efficacy of the particle removal processes in reducing bacterial concentrations. In principle, the difference in bacterial concentrations between the partially- and fully- treated samples allows the efficacy of the chlorination process to be determined. In practice, only a lower limit for bacterial removal by disinfection could be estimated, because the indicator bacteria were always undetectable in the finished water.

Treatment efficacy can be expressed as a percentage removal, but it is more often expressed as

$$\log \text{removal} = \log_{10} \left[ \frac{C_B}{C_A} \right]$$

where  $C_B$  is the bacterial concentration before the treatment process and  $C_A$  is the concentration following treatment. Expressing the removal in this way allows the overall removal achieved by a series of treatment processes to be calculated by adding the log removal values of the individual processes.

The estimated log removals for the indicator bacteria are given in Table 3. A log removal value is tabulated if  $C_B$ , at least, was quantified. When  $C_A$  was less than the limit of detection (LoD), it was arbitrarily assigned a value of 50% of the LoD. Log removal values are not tabulated when both  $C_A$  and  $C_B$  were "less than" values.

Minimum, maximum and mean log removals are calculated for each treatment component (particle removal and disinfection) and each event. The spread of results shows that a particular treatment plant's performance is quite variable even when the system is well run and under close manual or automated control. Uncertainty in the bacterial concentrations and the difficulty in maintaining optimum treatment conditions during relatively rapid changes in raw water quality probably contribute to the variability in the log removal values.

<sup>&</sup>lt;sup>1</sup> With the exception of P2 where partially treated samples had to be taken after the clarifiers and before filtration.

The maximum removal of bacteria achieved by the particle removal processes is ca. 5 log based on the coliform data, or ca. 4.4 log if the *E. coli* data are considered. A more conservative estimate of the level of removal being achieved could be derived from the maximum of the minimum values: ca. 3.5 log from coliform data and 3.1 log from *E coli*. The WHO (2004, Table 7.6) states that coagulation might achieve 1 log removal and filtration could achieve 2 log removal under optimum coagulation conditions, i.e., a total of 3 log. Hijnen *et al.* (2004) report approximately 1.5 log removal for the coagulation and clarification step at one full scale treatment plant, which if combined with 2 log removal by filtration would give a total for the combined processes of 3.5 log.

The log removal values for chlorination (Table 3 "Removal between filtered and finished water") range from 0.8-2.7 log for coliforms and 0.3-2.5 log for *E. coli* (if the data from T1 at P2 are excluded). These are underestimations of the reduction achieved by chlorination, because all  $C_A$  values had to be assumed to be 50% of the LoD and may have been lower concentrations. Hijnen *et al.* (2000) report a removal of 3.6 log for the chlorination process in a full scale treatment plant they studied, but they also note that the removal is low for the Ct value (40 mg.min/L) at the treatment plant compared with removals reported in a review by Sobsey (1989). Sobsey reported a removal of ca. 4.5 log for a Ct value of 3 mg.min/L, but this was in demand-free water under laboratory conditions, not a full scale treatment plant.

Event T1 at P2 did result in much greater coliform concentrations in the partially-treated water ( $C_B$ ) than was observed in other events. As a result, it was possible to quantify at least a 5 log reduction in the coliform concentration. This value cannot be used directly as an estimate of the log removal achieved by chlorination, however, because at this treatment plant the partially treated sample had to be taken before the filters. A contribution to the 5 log removal therefore comes from removal by the filters. If the WHO estimate of 2 log removal by filters is assumed for the filters at P2, the data from P2 would imply at least 3 log removal by chlorination.

From the above considerations, a conservative estimate of the overall removal of bacteria by full conventional treatment is ca. 7.0 log, taking a 3.5 log contribution from the particle removal processes, and 3.5 log contribution from disinfection. A value of 3.5 log is taken as the contribution from disinfection because it is closer to the value found in the Dutch study (Hijnen *et al.*, 2000), and the value of ca. 3 log from this study noted in the previous paragraph is probably an underestimate.

The disinfection process is generally regarded as key to providing protection against bacterial pathogens. The figures in Table 3, however, show that during rain events particle removal processes make a similar contribution to the overall removal of bacteria: an example of the benefits of having multiple barriers capable of removing a contaminant. Particle removal reduces the concentration of bacteria challenging the disinfection process, and also improves disinfection efficacy by reducing the turbidity of the water.

To get a more accurate measure of the efficacy of treatment processes in New Zealand, large volume sampling, like that undertaken by Hijnen *et al.* (2004), is needed. This would allow the limit of detection to be lowered and the bacterial concentrations that would otherwise be reported as "less than" to be quantified. Overall removal of coliforms in the Dutch treatment plants ranged from 3.2 to 6.3 logs, with the highest value being obtained at a treatment plant operating without a disinfection step. The study showed the robustness of

the multiple barrier strategy in water treatment through the observation that the variation in the overall removal was less than the sum of the variations in the unit processes that together made up the treatment trains. The study also found bacterial breakthroughs at levels that would have been undetectable using routine small volume monitoring. One of these breakthroughs occurred when suboptimal performance of the coagulation/flocculation process compromised the efficacy of disinfection by ozone.

	Coliforms								E. coli						
		Removal between raw and filtered water			Removal between filtered and finished water			Removal between raw and filtered water			Removal between filtered and finished water				
Treatment plant	Event	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max		
P1	T1	1.3	1.9	2.3	0.8	1.5	2.7	1.0	1.8	2.2	0.3	0.9	2.5		
	T2	3.2	3.5	3.8	0.8	1.0	1.3	2.5	3.2	3.7	0.3	0.5	0.9		
	F1	2.2	2.4	2.7	2.0	2.1	2.3	1.9	2.2	2.4	1.7	1.9	2.1		
	F2	1.4	1.9	2.6	0.9	1.4	2.0	1.9	2.2	3.0	0.3	0.6	1.6		
P2 <sup>1</sup>	T1				(4.8)	(4.9)	(5.1)	(1.5)	(2.1)	(2.5)	(1.2)	(1.7)	(2.2)		
P3	F1	3.2	3.4	3.6				2.7	3.0	3.3					
	F2	3.5	4.2	5.0				3.1	3.7	4.4					
P4	F1							2.8	3.1	3.3					
	F2							3.0	3.2	3.5					
Maximum values		3.5	4.2	5.0	2.0	2.1	2.7	3.1	3.7	4.4	1.7	1.9	2.5		
Average values		2.5	2.9	3.3	1.1	1.5	2.1	2.4	2.8	3.2	0.7	1.0	1.8		
Minimum values		1.3	1.9	2.3	0.8	1.0	1.3	1.0	1.8	2.2	0.3	0.5	0.9		

#### Table 3 Estimated logs of removal of indicator bacteria

1 Partially treated water taken after clarifiers but before filters in this treatment plant. As a result, the log removal values do not measure the efficacy of the same treatment processes as for the other treatment plants. Log removal values are given but are not included in the summary statistics.

## 4.3 Heavy rain events and public health

In this section estimates are made of the probability of illness caused by *Campylobacter* during a rain event, if untreated and treated waters raw waters are consumed. Consideration is also given to the level of illness that might be associated with *Campylobacter* for a water supply producing finished water that meets the requirement that the *E. coli* concentration is less than 1/100ml.

## 4.3.1 Estimates of illness due to Campylobacter during heavy rain events

This study and the earlier survey (Nokes *et al.*, 2004) have shown no evidence of *Campylobacter* being detected in suitably treated waters, and the public health risk from waterborne campylobacteriosis is therefore expected to be very low when treatment processes are working well. It has to be acknowledged, however, that because of the increase in *Campylobacter* concentrations in source waters during rain events the risk of infection from *Campylobacter*, even in well-operated supplies, increases.

Two factors have to be taken into account when evaluating the risk to public health from waterborne disease during a rain event: the "likelihood" of treatment failure and the "consequences" of exposure to the pathogen concentrations that would result from the failure. During a rain event, the likelihood of treatment failure increases because the changing water quality challenging the treatment plant makes maintaining optimum treatment conditions more difficult. In well-operated treatment plants with suitable treatment processes in place the likelihood of a breakthrough of pathogens into the finished water remains very small. At the same time that the possibility of treatment failure increases, *Campylobacter* concentrations and the consequences of failure for public health also increase, i.e., risk to public health increases, and is probably at its greatest, during rain events. This study has not tried to evaluate the likelihood of treatment failure, but the consequences of failure, in terms of the probability of illness, can be estimated.

Estimating the probability of illness starts by estimating the probability of infection in a community exposed to *Campylobacter*. The worst case would arise if the community were exposed to the untreated raw water. This might occur as the result of disinfection failure in a treatment plant in which disinfection is the only treatment barrier. In addition to the *Campylobacter* concentration, calculation of the infection probability requires:

a) An estimate of daily water consumption

The third edition of the *Guidelines for Drinking-water Quality* (WHO, 2004) assumes that the average daily intake of unboiled water is 1 litre per person per day.

b) An equation relating the probability of infection to the dose (number of pathogenic organisms ingested)

A beta-Poisson model is often used for calculating infection probabilities. The general form of this model is (Teunis *et al.*, 2005):

$$P_{inf} = 1 - {}_{1}F_{1}(\alpha, \alpha + \beta, -N)$$
 Eqn.1

where  $P_{inf}$  is the probability of infection,  $_1F_1$  is the Kummer confluent hypergeometric function, N is the mean dose, and  $\alpha$  and  $\beta$  are two parameters defining the beta distribution.

A simplified form, which gives results similar to Eqn.1 provided the conditions required for the approximations made in its derivation are fulfilled, is (Teunis and Havelaar, 2000):

$$P_{inf} = 1 - [1 + N/\beta]^{-\alpha} \qquad \text{Eqn.2}$$

Teunis and Havelaar (2000) give values for  $\alpha$  and  $\beta$  for Eqn. 1 of 0.145 and 8.007, respectively for *C. jejuni*. Speciation of the *Campylobacter* in this study has shown the predominance of *C. jejuni* in the waters studied. It is assumed for these calculations that *C. jejuni* is the primary organism that will lead to infection through ingestion of waters from all three treatment plants.

Multiplication of the mean Campylobacter concentration measured during each rain event by the volume of unboiled water consumed daily, allows a mean daily dose of Campylobacter, N, to be determined. From these values of N the probabilities of infection can be calculated for each event using Eqn.1. Calculations using the hypergeometric carried on-line function were out the Wolfram Research website at (http://functions.wolfram.com/webMathematica/FunctionEvaluation.jsp?name=Hypergeo metric1F1).

Table 4 lists the calculated infection probabilities for events monitored at P1, P3 and P4 for the consumption of untreated water.

Р	Event	Mean raw water <i>Campylobacter</i> concentration (MPN/100ml)	Probability of infection (/day)
D1	F1	15	0.35
PI	F2	2.4	0.19
D2	F1	34	0.43
P3	F2	26	0.40
D4	F1	28	0.41
Ľ4	F2	56	0.47

Table 4Probability of infection by Campylobacter calculated for full events monitored<br/>at P1, P3 and P4, for mean concentrations in raw waters.

"Less than values" were assigned a concentration of 50% of the Limit of Detection for calculation of the mean.

The probability of infection over a period of t days ( $P_{inf,t}$ ), can be calculated from:

$$P_{inf,t} = 1 - (1 - P_{inf,d})^t$$
 Eqn.3

where  $P_{inf,d}$  is the daily probability of infection. The annual probability of infection, i.e., the probability of an individual becoming infected as the result of consuming water with this concentration of *Campylobacter* for a year is obtained by giving t a value of 365.

Estimates of infection and illness probabilities over periods of 3 and 365 days for consumption of treated and untreated water are given in Table 5. The assumed concentration of *Campylobacter* in the raw water for these calculations was the mean taken over all full events monitored during the study (ca. 26 MPN/100ml). Eqn.3. was used to calculate the probability of infection for the two periods. The probabilities are only the probability of becoming infected; illness does not necessarily result from infection. An estimate can be made of the probability of becoming infected and ill by multiplying the probability of infection by the probability of illness given infection (the conditional probability of illness). A value for the conditional probability of illness of 0.3 is given in Table 7.3 of the WHO *Guidelines for Drinking-water Quality* (WHO, 2004).

			,	
Water	Period	Daily probability of infection	Probability of infection during the period	Probability of illness during the period
Untreated water	3 days	0.41	0.79	0.24 (24%)
	1 year	0.41	1	0.30 (30%)
Treated water (7 log removal)	3 days	4.8 x 10 <sup>-7</sup>	1.4 x 10 <sup>-6</sup>	4.1 x 10 <sup>-7</sup> (4.1 x 10 <sup>-5</sup> %)
	1 year	4.8 x 10 <sup>-7</sup>	1.7 x 10 <sup>-4</sup>	5.0 x 10 <sup>-5</sup> (0.0050%)

Table 5Estimates of Infection and illness probabilities for periods of 3 days and 1 year<br/>assuming a raw water concentration equal to the mean concentration from all<br/>full events (ca. 26 Campylobacter/100ml)

Plant failure during a rain event should ideally be detected immediately and actions taken to protect the community from receiving contaminated water. In small, poorly equipped and monitored systems, this may not happen and it may be some days before the problem is identified and corrected. To evaluate what this might mean for public health in a community a three day period for the calculations was arbitrarily chosen. The estimate of an annual probability of illness would be more appropriate if untreated water were typically provided to consumers because the water supply had no treatment plant, and *Campylobacter* were frequently present in the water at the levels found in this study. This is an extreme situation, and unlikely to be encountered in reality in a community water supply. However, the calculation does allow comparison with probabilities expressed on an annual basis reported elsewhere, e.g., WHO (2004).

Both figures for the probability of illness based on consumption of untreated water are likely overestimates. Models are presently unable to take account of the development of immunity in the exposed community. This is not likely to lead to major inaccuracies when infection probabilities are low, but for the high infection probabilities resulting from exposure to raw water overestimations will result.

The probability of illness resulting from consumption of untreated water is very much reduced if the water is adequately treated. From Section 4.2.2 a conservative estimate of the removal of *Campylobacter* achievable by full conventional treatment is 7.0 log. This is the removal assumed for the calculations for Table 5, and would result in the treated water concentration being ca.  $2.6 \times 10^{-6}$  *Campylobacter*/100ml (mean concentration found in thus

study x  $10^{-7}$ ). Exposure to this concentration would lead to illness in one in 20,000 people over the course of a year. Over the three day period, treatment reduces the probability of infection by a factor of ca. 6 x  $10^5$ .

The levels of faecal contamination found during rain events in the source waters included in this study are probably typical of many drinking-water sources in New Zealand during rain events. The treatment plants that participated in the study are well operated and the calculated likelihoods of campylobacteriosis arising from drinking waters in the communities they serve are correspondingly low. However, where water supplies are untreated, or poorly treated, the above calculations show rain events can be expected to lead to an increased likelihood of waterborne campylobacteriosis in the community. Other pathogens that increase in concentration in source waters as the result of rain are also likely to contribute to increased levels of water-borne disease.

Where treatment plants have difficulty in producing water of satisfactory quality during rain events, water suppliers need to consider steps to minimise the deterioration in raw water quality. Possible preventive measures include: closing the intake once raw water turbidity reaches a predetermined level; abstraction through an infiltration gallery; or use of a raw water reservoir that buffers the change in water quality.

## 4.3.2 Protection afforded by meeting the MAV of less than 1 E. coli/100ml

Monitoring of the indicator bacterium *E. coli* is required at water treatment plants to show compliance of the plant with the DWSNZ, unless there is continuous chlorine monitoring. The bacteriological quality of the water is considered acceptable provided the *E. coli* concentration is less than 1 per 100ml. This "test" of the microbiological safety of water has been used internationally for many years, but retrospective investigation of disease outbreaks and advances in the understanding of pathogens in water are showing there are shortcomings in its use in establishing the microbiological safety of water (p.142, WHO, 2004). Opportunities for improving the way indicator organisms, such as *E. coli*, are used arise as more information on the concentrations of pathogens in water supplies becomes available. This section uses data from the present study to consider the protection against *Campylobacter* afforded by compliance with the *E. coli* MAV.

The requirement that the *E. coli* concentration is less than 1 per 100ml can be met by *E. coli* concentrations a little less than 1 per 100ml. Disinfection processes that reduce the *E. coli* to less than 1 per 100ml may achieve *E. coli* concentrations well below this, but routine monitoring using 100ml sample volumes does not allow this to be determined.

To evaluate a worst case situation for public health, the following calculations assume that the actual *E. coli* concentration in a sample recorded as "<1 *E. coli*/100ml" is 0.5 *E. coli*/100ml (i.e. 50% of the MAV). An estimate of the *Campylobacter* concentration in the finished water can be made from ratios of *E. coli*:*Campylobacter* determined in the raw water by this study. This ratio ranged from 10 to over ca. 4,000. The ratio in a finished water will be less than the ratio in the corresponding raw water because of the greater susceptibility of *Campylobacter* to disinfectants, but like the raw water ratio it will be time-dependent because of the different rates of inactivation of the two organisms<sup>2</sup>.

<sup>&</sup>lt;sup>2</sup> Lund (1996) reported the time for 99.9% inactivation of *E. coli* by chlorine to be ca. 2.5 times that of *C. jejuni* at  $10^{\circ}$ C

Calculations based on the range of *E. coli/Campylobacter* ratios in the raw water, without taking account of chlorine susceptibility, give an estimated *Campylobacter* concentration range in the finished water of 0.00013-0.05 *Campylobacter*/100ml.

The probability of infection by *Campylobacter* per day for this concentration range is ca.  $2.2 \times 10^{-5}$ -8.6 x  $10^{-3}$  (calculated using Eqn.1). The range of annual probabilities of infection calculated from these daily probabilities is 0.008-0.96 per year, which gives annual probabilities of illness of 0.0024-0.29 (0.008-0.96 x 0.3), i.e., ca. 0.2-29%. WHO (Table 7.3, (2004)) gives an estimated disease burden in DALYs (disability adjusted life years) per case of illness of 4.6 x  $10^{-3}$ . Multiplying this figure by the annual probability of illness yields an annual estimated disease burden of ca. 1–100 x  $10^{-5}$  DALYs per person per year.

This estimated disease burden is 10–1,000 times the WHO health outcome target of 1 x  $10^{-6}$  DALYs per person per year (Section 3.3.2, WHO (2004)), but caution is needed in drawing conclusions from this comparison. *E. coli* is a conservative indicator for *Campylobacter* because of its greater resistance to chlorine, and this calculation does not change this. The calculation shows that because of the likely *E. coli:Campylobacter* ratio, and the infectivity of *Campylobacter*, *under the worst-case condition* in which an *E. coli* concentration reported as "<1/100ml" is actually 0.5 *E. coli*/100ml, undesirable levels of illness *might* result. The extent to which this constitutes a public health concern depends, *inter alia*, on the frequency at which the "worst-case condition" is encountered. No data are available to allow an estimate of this frequency. Provided a water supply is adequately disinfected, the *E. coli* concentration in a water reported as containing "<1 *E. coli*/100ml" is likely to be several orders of magnitude lower. It is supplies that are not, or inadequately, disinfected, where *E. coli* concentrations could be much closer to the 1 *E. coli*/100ml limit.

While this unsophisticated estimate of the disease burden cannot provide firm guidance on the protection against water-borne illness afforded by the "<1 *E. coli*/100ml" water quality criterion, it does show that a better understanding of its limitations would be valuable. This could start by using large volume sampling to quantify *E. coli* concentrations that are below the detection limit for routine test protocols (i.e., <1 *E. coli*/100ml). This will provide information about the frequency at which the "worst case condition" is likely to occur. Quantification of *Campylobacter* and other pathogens during such as study would also provide direct measurement of pathogen exposure levels.

# 5 CONCLUSIONS

This study of *Campylobacter* in water supply source waters during rain events, and the ability of treatment plants to produce safe water under these conditions, has collected data from nine events, six of which were sampled for *Campylobacter*. The key findings of the study are:

- 1. Rain events can lead to elevated *Campylobacter* concentrations in water supply source waters. Indicator organism concentrations are also elevated during rain. The highest concentration of *Campylobacter* found in source water during this study was 93 MPN/100ml with the mean concentrations from the three treatment plants from which *Campylobacter* samples were obtained, ranging from ca. 2.4–56MPN/100ml.
- 2. Water turbidity levels increase during rain, but the highest turbidity level does not necessarily arrive at the treatment plant intake at the same time as the maximum concentrations of *Campylobacter* or indicator bacteria. The relative timings of peaks in turbidity and bacteria are expected to be influenced by, *inter alia*: the levels of microbes in reservoirs, such as river sediments, the rate at which the reservoirs are recharged and depleted, and the degree of dilution caused by the rainfall.
- 3. The arrival of the peaks in bacterial concentrations relative to the arrival of the turbidity peak is variable and depends on the event. Monitoring of the turbidity, or the river or stream flow, cannot be used as a reliable indicator of when the threat from bacterial pathogens is at its greatest.
- 4. A simple linear relationship between *Campylobacter* concentration and turbidity or *Campylobacter* and *E. coli* concentration in the raw water that would allow turbidity or indicator measurements to be used to estimate *Campylobacter* concentrations in the water could not be found.
- 5. Neither *Campylobacter* nor indicator bacteria were detected in any fully-treated water sample. This is consistent with the findings of the original survey, and supports the survey's conclusions that the risk from *Campylobacter* infection from well-treated drinking water is low.
- 6. Comparison of indicator bacteria concentrations in the raw and partially treated waters shows that the particle removal processes in full conventional treatment (coagulation/flocculation, clarification and filtration) can conservatively achieve 3.5 log removal of bacteria and at times up to 5 log.
- 7. Only minimum log removal values achieved by chlorination could be estimated because the indicator bacteria concentration in the fully-treated water was always below the limit of detection. The maximum log removal value determined was 2.7 log. Consideration of estimates from other sources showed that a log removal value of 3.5 log is probably a conservative estimate of bacterial removal by chlorination.
- 8. A conservative estimate of the overall bacterial removal achievable by full conventional treatment is 7 log. During rain events particle removal processes can make a major contribution (up to 50% of the total log removal of bacteria achieved by the treatment plant) to the removal of bacteria from waters. Thereby providing a further barrier, in addition to disinfection, to the barriers to bacteria.

- 9. The estimated probability of illness resulting from the consumption of untreated raw water (with the mean *Campylobacter* concentration found in this study) for a period of three days is calculated to be ca. 24%, i.e., one in every four people would be ill. However, this figure is expected to be an overestimate because the development of immunity by exposed individuals cannot be taken into account.
- 10. Probabilities of illness in communities exposed to untreated raw water are calculated to be much greater than in communities receiving adequately treated water. The concentration of *Campylobacter* in water that has been adequately treated is estimated to result in only 1 case of campylobacteriosis in every 20,000 people per year, and this assumes a raw water concentration over this period equal to the mean concentration found during these rain events.
- 11. Calculations based on the *E. coli:Campylobacter* ratios found in raw water from this study indicate that waters with an *E. coli* concentration of 0.5 *E. coli*/100ml *may* contain sufficient *Campylobacter* to produce a disease burden greater than the WHO health target outcome of  $1 \times 10^{-6}$  DALYs per person per year. This does *not* indicate a universal failure of the criterion of "<1 E. coli/100ml" to constitute a safe water, but it does signal a need to better understand the limitations of the criterion.

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## **APPENDIX - BACTERIOLOGICAL DATA FROM TREATMENT PLANTS**

Throughout this Appendix

\* denotes samples analysed over 24 hours and up to and including 36 hours after collection

\*\* denotes samples analysed over 36 hours and up to and including 48 hours after collection

#### P1 data

TEST RUN 1		Turbidity				Total Coliform			E. coli			Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	
2-Sep-04	8:00 a.m.	6.1	0.963	0.232	236	11	<1	88	1	<1				
2-Sep-04	11:30 a.m.	18.5	0.148	0.637	1,300	6.3	<1	308	4	<1				
2-Sep-04	3:00 p.m.	18.5	0.101	0.212	649	3.1	<1	122	2	<1				
2-Sep-04	6:30 p.m.	15.5	0.136	0.303	866	9.8	<1	96	1	<1				
2-Sep-04	10:00 p.m.	13.5	0.103	0.166	816	11	<1	105	2	<1				
3-Sep-04	1:30 a.m.	17.2		0.668	2,419		<1	281		<1				
3-Sep-04	5:00 a.m.	22.7	0.419	1.41	>2,419	30.9	<1	921	6	<1				
3-Sep-04	8:30 a.m.	35.5	0.606	0.815	5,475	261.3	<1	1,515	150	<1				

Units: all bacterial concentrations MPN/100ml, turbidity NTU

TEST RUN 2		Turbidity				Total Coliform			E. coli			Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	
3-May-05	11:00 a.m.	25	0.034	0.093	>24,192	4	<1	3,968	4	<1				
3-May-05	2:30 p.m.	19	0.035	0.098	>24,192	5	<1	4,569	1	<1				
3-May-05	6:00 p.m.	15	0.034	0.091	24,192	5	<1	3,873	2	<1				
3-May-05	9:30 p.m.	13	0.036	0.093	17,329	3	<1	1,785	1	<1				
4-May-05	1:00 a.m.	9	0.058	0.106	12,033	4	<1	1,723	1	<1				
4-May-05	4:30 a.m.	7	0.035	0.117	14,136	9	<1	1,553	1	<1				
4-May-05	8:00 a.m.	6	0.032	0.088	11,199	<1	<1	1,119	<1	<1				
4-May-05	11:30 a.m.	5	0.031	0.085	7,270	4	<1	1,043	3	<1				

Full Run 1		Turbidity			Total Coliform			E. coli			Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
21-Dec-05	10:00 a.m.	46	0.215	1.630	24,810		<1	6,200		<1	43	<0.3	<0.3
21-Dec-05	12:00 p.m.	69	0.126	0.756	19,350	98.5	<1	4,570	60.9	<1	9	<0.3	<0.3
21-Dec-05	2:00 p.m.	65	0.081	0.325	17,250	50.4	<1	5,280	25.9	<1	4	<0.3 **	<0.3 **
21-Dec-05	4:00 p.m.	80	0.060	0.155	12,330	71.7	<1	4,500	42.8	<1	43 **	<0.3 **	<0.3 **
21-Dec-05	6:00 p.m.	100	0.062	0.121	15,650	61.3	<1	7,270	31.8	<1	<3 **	<0.3 **	<0.3 *
21-Dec-05	8:00 p.m.	127	0.060	0.096	18,420	95.9	<1	7,590	45.5	<1	11 **	<0.3 *	<0.3 *
21-Dec-05	10:00 p.m.	127	0.056	0.095	16,160	57.1	<1	4,800	25.3	<1	<3 *	<0.3 *	<0.3 *
22-Dec-05	12:00 a.m.	105	0.060	0.102	29,090	65	<1	8,620	41.6	<1	4 *	<0.3 *	<0.3 *

## P1 data (continued)

Full	Run 2		Turbidity			Total Coliform			E. coli		(	r	
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
9-Oct-06	10:00 a.m.	2.2	0.031	0.063	645	23.1	<1	272	3.1	<1	4 *	<0.3 *	<0.3
9-Oct-06	1:30 p.m.	2.23	0.031	0.060	496	6.3	<1	74	1	<1	<3	<0.3	<0.3
9-Oct-06	5:00 p.m.	2.4	0.031	0.061	393	6.3	<1	85	1	<1	<3	<0.3	<0.3
9-Oct-06	8:30 p.m.	2.25	0.030	0.059	368	4.1	<1	86	1	<1	<3	<0.3	<0.3
10-Oct-06	12:00 a.m.	6.4	0.036	0.058	2,560	11.9	<1	970	1	<1	<3	<0.3	<0.3
10-Oct-06	3:30 a.m.	17	0.109	0.100	2,620	46.4	<1	1,460	5.2	<1	<3	<0.3	<0.3
10-Oct-06	7:00 a.m.	17.3	0.121	0.142	16,100	41.3	<1	3,100	18.3	<1	4	<0.3	<0.3
10-Oct-06	10:30 a.m.	13	0.044	0.109	960	14.8	<1	410	2	<1	4	<0.3	<0.3

TEST	RUN 1		Turbidity			Total Colifor	m		E. coli		(	Campylobacte	r
Date	Time of raw water sample collection	Raw water	Post clarifier	Finished water	Raw water	Post clarifier	Finished water	Raw water	Post clarifier	Finished water	Raw water	Post Filter	Finished water
8-Feb-05	12:30	2.2	0.089	0.058	3700	39000	<1	1500	20	<1			
8-Feb-05	13:00	2.78	0.085	0.058	3600	37000	<1	1800	<4	<1			
8-Feb-05	13:30	2.89	0.088	0.059	4100	35000	<1	1800	8	<1			
8-Feb-05	14:00	3.19	0.096	0.063	5200	40000	<1	2200	20	<1			
8-Feb-05	14:30	3.69	0.103	0.064	6300	32000	<1	2800	80	<1			
8-Feb-05	15:00	4.43	0.109	0.065	7600	44000	<1	4000	12	<1			
8-Feb-05	15:30	4.8	0.111	0.066	8700	60000	<1	2600	40	<1			
8-Feb-05	16:00	4.57	0.111	0.068	8700	44000	<1	4200	36	<1			
8-Feb-05	16:30	6.04	0.12	0.068	13000	50000	<1	5500	52	<1			
8-Feb-05	17:00	5.91	0.112	0.068	11000	>10000	<1	6000	24	<1			
8-Feb-05	17:30	5.5	not collected	not collected	7900	not collected	not collected	4800	not collected	not collected			

#### P2 data Units: Total coliform and *E. coli* concentrations cfu/100ml, *Campylobacter* MPN/100ml, turbidity NTU

Yellow highlighting indicates results outside the normal quantification range which are approximate values only

#### P3 data

#### Units: Total coliform and E. coli concentrations cfu/100ml, Campylobacter MPN/100ml, turbidity NTU

FULL	FULL RUN 1		Turbidity			Total Coliform			E. coli		Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
23-May-06	9:20 p.m.	5.0	-	-	800	<10	<1	260	<1	<1	15	<0.3	<0.3
23-May-06	10:20 p.m.	7.7	-	-	1,230	<10	<1	330	<1	<1	23	<0.3	NA
23-May-06	11:20 p.m.	7.6	-	-	1,420	<10	<1	440	<1	<1	43	<0.3	<0.3
24-May-06	12:20 a.m.	9.9	-	-	1,520	10 <sup>‡</sup>	<1	670	<1	<1	39	<0.3	<0.3
24-May-06	1:20 a.m.	13.6	-	-	2,080	<10	<1	990	<1	<1	93	<0.3	<0.3
24-May-06	2:20 a.m.	17.4	-	-	1,960	<10	<1	830	<1	<1	23	<0.3	<0.3
24-May-06	3:20 a.m.	18.6	-	-	1,260	<10	<1	680	<1	<1	15	<0.3	<0.3
24-May-06	4:20 a.m.	17.5	-	-	1,160	<10	<1	540	<1	<1	23	<0.3	<0.3

‡ One colony found in a 1:10 dilution

## P3 data (continued)

FULL RUN 2		Turbidity			Total Coliform				E. coli		Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
30-Nov-06	7:40 a.m.	8	-	-	1,920	<1	<1	650	<1	<1	<3 *	<0.3	<0.3
30-Nov-06	8:40 a.m.	16	-	-	3,650	<1	<1	1,600	<1	<1	9	<0.3	<0.3
30-Nov-06	9:40 a.m.	220	-	-	33,000	<1	<1	8,000	<1	<1	9	<0.3	<0.3
30-Nov-06	10:40 a.m.	440	-	-	65,000	1	<1	14,000	1	<1	43	<0.3	<0.3
30-Nov-06	11:40 a.m.	500	-	-	48,000	<1	<1	13,000	<1	<1	43	<0.3	<0.3
30-Nov-06	12:40 p.m.	311	-	-	>5000	1	<1	4,200	<1	<1	43	<0.3	<0.3
30-Nov-06	1:40 p.m.	68	-	-	2,900	1	<1	1,600	1	<1	9	<0.3	<0.3
30-Nov-06	2:40 p.m.	25	-	-	1,820	<1	<1	1,500	1	<1	<3	<0.3	<0.3

#### P4 data

Units: *Campylobacter* MPN/100ml, turbidity NTU

FULL	. RUN 1		Turbidity			Total Coliform			E. coli		(	Campylobacte	÷r
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
20-Jun-06	5:45 a.m.	382			>2005	<1	<1	591	<1	<1	23 *	<0.3	<0.3
20-Jun-06	6:30 a.m.	437			>2005	<1	<1	782	<1	<1	23 *	<0.3	<0.3
20-Jun-06	7:15 a.m.	530			2005	1.0	<1	1013	<1	<1	43 *	<0.3	<0.3
20-Jun-06	8:00 a.m.	542			>2005	3.1	<1	1091	<1	<1	23 *	<0.3	<0.3
20-Jun-06	8:45 a.m.	580			>2005	2.0	<1	2005	1.0	<1	23	<0.3	<0.3
20-Jun-06	9:30 a.m.	630			>2005	2.0	<1	1184	2.0	<1	39	<0.3	<0.3
20-Jun-06	10:15 a.m.	575			>2005	3.1	<1	885	1.0	<1	7	<0.3	<0.3
20-Jun-06	11:00 a.m.	535			>2005	2.0	<1	591	<1	<1	43	<0.3	<0.3

## P4 data (continued)

FULL	RUN 2	Turbidity			Total Coliform				E. coli		Campylobacter		
Date	Time of raw water sample collection	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water	Raw water	Post Filter	Finished water
14-Mar-07	7:15 a.m.	150			>20050	>200.5	<1	16,520	5	<1	4 *	<0.3	<0.3
14-Mar-07	7:45 a.m.	173			>20050	>200.5	<1	16,520	10	<1	43 *	<0.3	<0.3
14-Mar-07	8:15 a.m.	178			>20050	>200.5	<1	20,050	19	<1	75 *	<0.3	<0.3
14-Mar-07	8:45 a.m.	170			>20050	>200.5	<1	20,050	14	<1	21	<0.3	<0.3
14-Mar-07	9:15 a.m.	158			>20050	144	<1	11,450	11	<1	75	<0.3	<0.3
14-Mar-07	9:45 a.m.	127			>20050	144	<1	16,520	6	<1	93	<0.3	<0.3
14-Mar-07	10:15 a.m.	106			>20050	144	<1	12,980	8	<1	93	<0.3	<0.3
14-Mar-07	10:45 a.m.	89			>20050	165	<1	10,130	9	<1	43		

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