

A Public Health Portrait of Severe Paediatric Gastroenteritis in the Auckland Region:

Report of the 2005 Auckland
Paediatric Gastroenteritis Investigation

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Working with the people of Auckland, Counties Manukau and Waitemata

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EXECUTIVE SUMMARY

In the Auckland region, hospital admission rates for children and young people with gastroenteritis are higher than the New Zealand average at all ages. Pacific children and young people have higher rates of hospitalisation for gastroenteritis in Auckland than either Maori or the rest of the population, and this pattern appears across the three District Health Boards (DHB), with Counties Manukau DHB having the highest number of cases. Routine faecal testing is not performed on children seen at Auckland's two paediatric hospitals, Starship and Kidz First Children's Hospitals, since it often does not affect clinical management. As a result, the precise etiology of paediatric gastroenteritis admissions has been unclear.

Concern about the high rates of paediatric gastroenteritis hospital admissions in South Auckland culminated in a meeting on 14 April, 2005 of a working party consisting of representatives from the Ministry of Health, New Zealand Food Safety Authority, Pacific Island health providers, Counties Manukau DHB and the Auckland Regional Public Health Service. It was agreed that an investigation to ascertain the major etiologies of gastroenteritis for those children admitted to paediatric hospital in Auckland would be an important first step towards considering measures to reduce hospitalisation rates.

This investigation aimed to identify the etiology of gastroenteritis among children admitted to paediatric hospitals in Auckland, and to evaluate the associations between paediatric gastroenteritis hospitalisation rates and factors such as ethnicity and socioeconomic status, measured as neighbourhood level deprivation.

This investigation was undertaken as a prospective case series, involving two sites. During the study period of July 1 to December 31, 2005, demographic and clinical data were collected on all children less than fifteen years old diagnosed with primary gastroenteritis at the emergency departments of Starship and Kidz First paediatric hospitals. Cases seen at other emergency departments and clinics, but not admitted to a paediatric hospital, were not included in the series. Extensive faecal testing was performed on specimens from cases, including specialised viral testing, which was completed at the Institute of Environmental Science and Research (ESR) in Porirua.

The key findings were as follows:

- A total of 131 children/cases were entered into the study between July 1 and December 31, 2005: Kidz First Children's Hospital provided 92 (70.2%) of the cases and Starship Children's Hospital provided 39 (29.8%). These cases represented about fifteen percent of total cases discharged from the two hospitals during the same period with gastroenteritis (NZHIS data).
- Most of the children treated at hospital for severe gastroenteritis were under 5 years of age (92.4%), and nearly eighty percent of cases were under two.

- There was one death among the cases, from complications of dehydration due to rotavirus gastroenteritis.
- Pacific children were over-represented among the series cases and had the highest rates of severe gastroenteritis. The most widely affected Pacific children were Samoan, Tongan and Cook Island Maori. The breakdown of cases by major ethnic group was as follows: 35.9% Pacific, 29% NZ European, 16.0% NZ Maori, 13.0% Asian and 6.4% Other.
- An apparent association was found between relative socioeconomic deprivation and severe paediatric gastroenteritis. Ninety-three (71%) of the case series children were resident at addresses in relatively deprived neighbourhoods, rated as decile 6 or higher on the NZDep 2001 rating scale.
- The frequency of pathogen on faecal testing was, in order of decreasing proportions, as follows: Rotavirus (57.3%), *Campylobacter* (9.2%), Norovirus (5.3%), Enterohemorrhagic *E.coli* (0.8%) and Sapovirus (0.8%). In 22.9% of cases, no pathogen was found. Multiple pathogens (2 -3) were found in 7.6% of cases.
- Length of hospital admission ranged from 2 hours to 16 days. The total number of hospitalisation days in the study was 155.4, with rotavirus gastroenteritis accounting for 73% of these (112.7 days). The median admission time for all types of viral gastroenteritis combined was more than double that for bacterial gastroenteritis.
- Dehydration was a common clinical finding, affecting nearly three quarters of the children in the case series on arrival at hospital, and was the most common reason for children being admitted to hospital.

Summary of Findings:

1. The greatest impact of paediatric gastroenteritis is on Pacific children, and especially those under the age of 5 years.
2. There appears to be an association between relative socioeconomic deprivation and severe paediatric gastroenteritis.
3. Rotavirus gastroenteritis, while a seasonal illness with winter peaks, is the cause of the large majority of paediatric gastroenteritis hospital admissions in the Auckland region.
4. Viral gastroenteritis accounts for double the hospitalisation burden of bacterial gastroenteritis.

The major conclusions from the study are as follows:

- Primary prevention strategies need to be implemented, to reduce the exposure of young children to enteric pathogens through foodstuffs, and to reduce person-to-person spread of gastroenteritis.
- Secondary prevention, through early rehydration of children with acute gastroenteritis in the community, either at home or in primary care, is critical to reducing hospital admissions and limiting morbidity from the disease. Access to comprehensive primary health care services is important for those groups most at risk.
- The introduction of a rotavirus vaccine, once it has been found to be safe and effective, could dramatically reduce the burden of disease from acute paediatric gastroenteritis in Auckland.
- Pacific preschool age children, and specifically Samoan, Tongan and Cook Island Maori children, should be the first priority for any prevention strategies.

The goal of this report is to inform an ongoing discussion between health workers and those communities whose children are most affected by severe gastroenteritis, such as some Pacific communities, with the aim of finding effective ways to reduce the burden of this common childhood illness. Ongoing collaboration between policy makers and health care providers, especially primary health organisations, will be important in order to work effectively with those communities most affected. The story told by this case series must not simply contribute to the growing body of statistics on health inequalities in Aotearoa New Zealand.

INTRODUCTION

Acute gastroenteritis is a clinical syndrome which is usually infectious in origin. Diarrhoea is the most prominent symptom, and it is often accompanied by vomiting, abdominal pain, or fever. The illness can have an abrupt onset, and in children can rapidly lead to electrolyte disturbances and dehydration, requiring hospital treatment. Gastroenteritis is caused by a range of viral, bacterial and parasitic organisms. The majority of cases of childhood gastroenteritis have a viral etiology, and, in fact, rotavirus group A is the single most important cause of severe diarrhoeal disease in infants and children worldwide (Kapikian 1993).

In New Zealand, gastroenteritis has been ranked third amongst potentially avoidable causes of paediatric admissions to hospital (Graham, Leversha et al. 2001). The Ministry of Health lists it as an important cause of paediatric ambulatory sensitive admissions, or admissions to hospital that are potentially preventable by early treatment in primary health care (Ministry of Health 2004). Childhood hospital admissions for gastroenteritis have been increasing in New Zealand in recent years, while deaths remain static at 1-2 cases per year (see Figure 1) (NZ Child and Youth Epidemiology Service 2005).

The greatest impact of acute gastroenteritis is on children under the age of three years (see Figure 2), as young children are the most vulnerable to dehydration and other complications of the illness necessitating hospital treatment.

Figure 1: Hospital Admission Rates (1988-2004) and Deaths (1998-2002) due to Gastroenteritis, New Zealand Children and Young People 0-24 years. (Courtesy of NZ Child and Youth Epidemiology Service 2005)

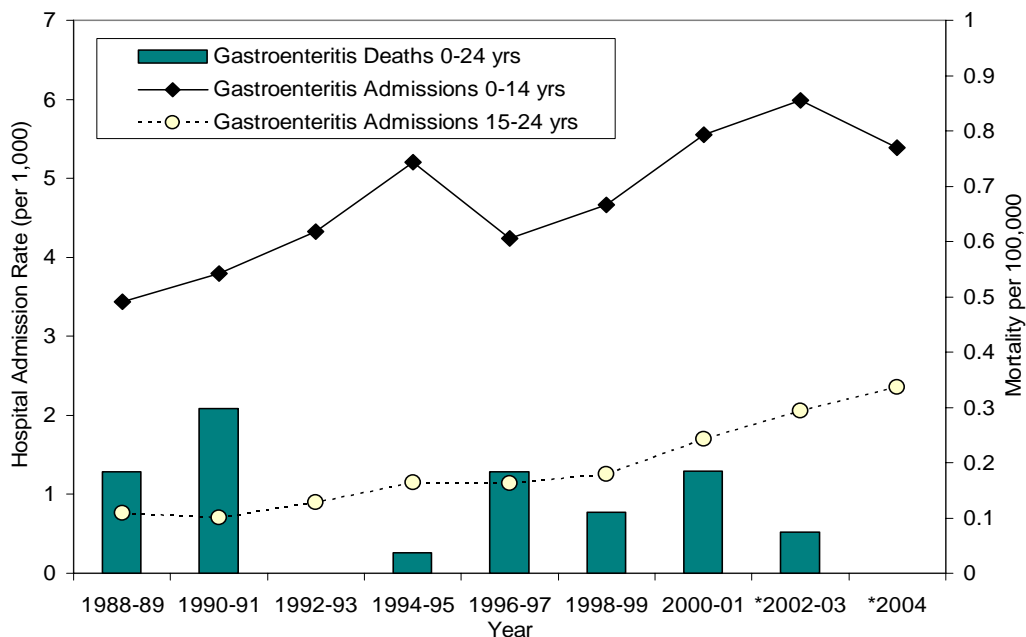
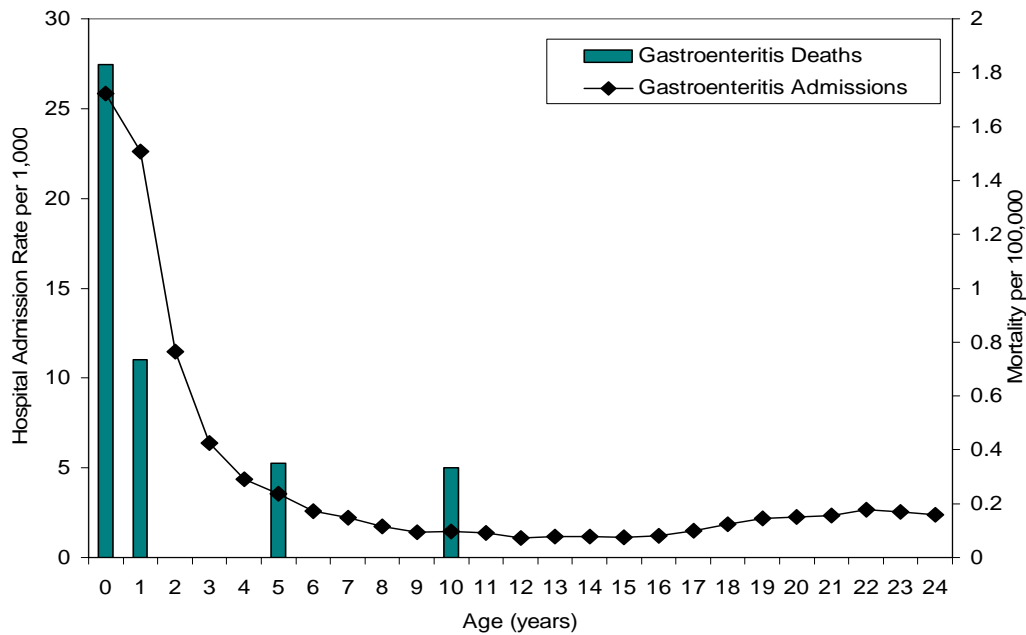


Figure 2: Hospital Admission Rates (2000-2004) and Deaths (1998-2002) due to Gastroenteritis by Age; New Zealand Children and Young People 0-24 Years. (Courtesy of NZ Child and Youth Epidemiology Service 2005)



Hospital admission rates for children and young people with gastroenteritis in the Auckland region have risen above the New Zealand average at all ages (see Figure 3). Pacific children and young people have higher rates of hospitalisation for gastroenteritis in Auckland than either Maori or the rest of the population, and this pattern appears across the three District Health Boards (DHB), with Counties Manukau DHB having the highest number of cases (see Figure 4).

Routine faecal testing is not performed on children seen at Auckland’s two paediatric hospitals, since it often does not affect clinical management, and so the precise etiology of the high number of gastroenteritis admissions has been unclear. A 2003 clinical audit of 50 children admitted to Kidz First Hospital with gastroenteritis found that 32 percent of cases had faecal testing done (Trenholme, McBride et al. 2003). In other words, for nearly two thirds of hospitalised children the cause of the gastroenteritis has remained unknown. For Pacific children under 5 years of age and hospitalised during 2002-2004, the cause of gastroenteritis remained unproven in 62 percent of cases (see Figure 5).

Figure 3: Hospital Admissions due to Gastroenteritis Amongst Children & Young People 0-24 yrs, the Auckland Region vs. New Zealand, 1990-2004. (Courtesy of NZ Child and Youth Epidemiology Service 2005)

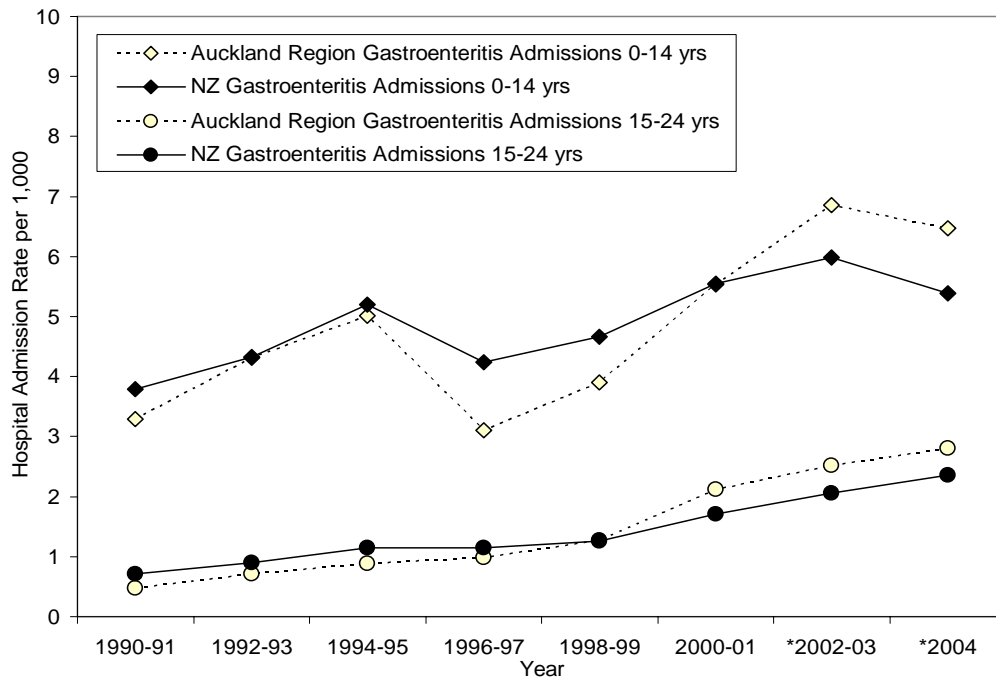


Figure 4: Auckland Paediatric Gastroenteritis Hospital Admissions for Age 0-4 years, 2004, by District Health Board. (Data courtesy of Dr. G. Jackson, CMDHB)

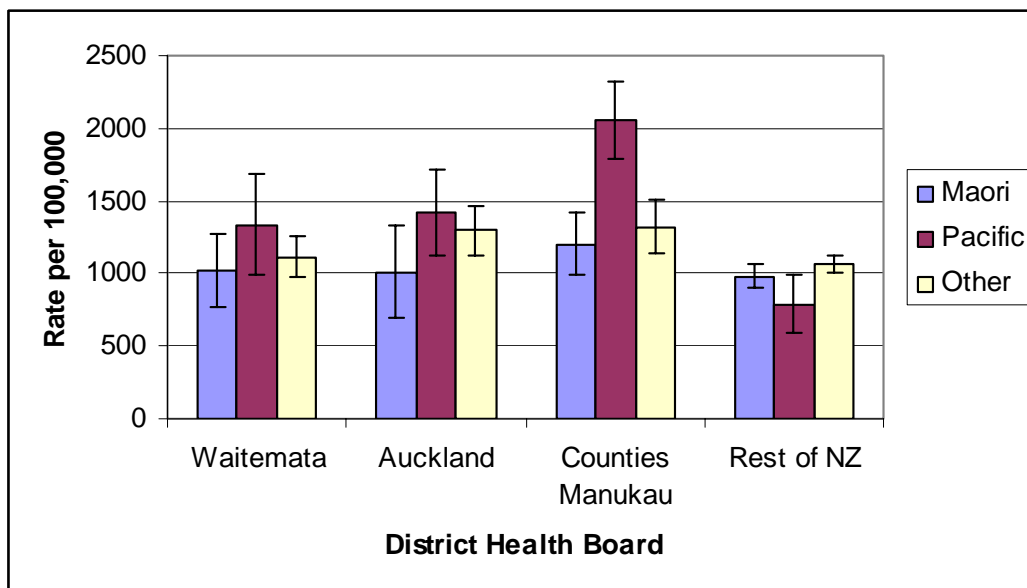
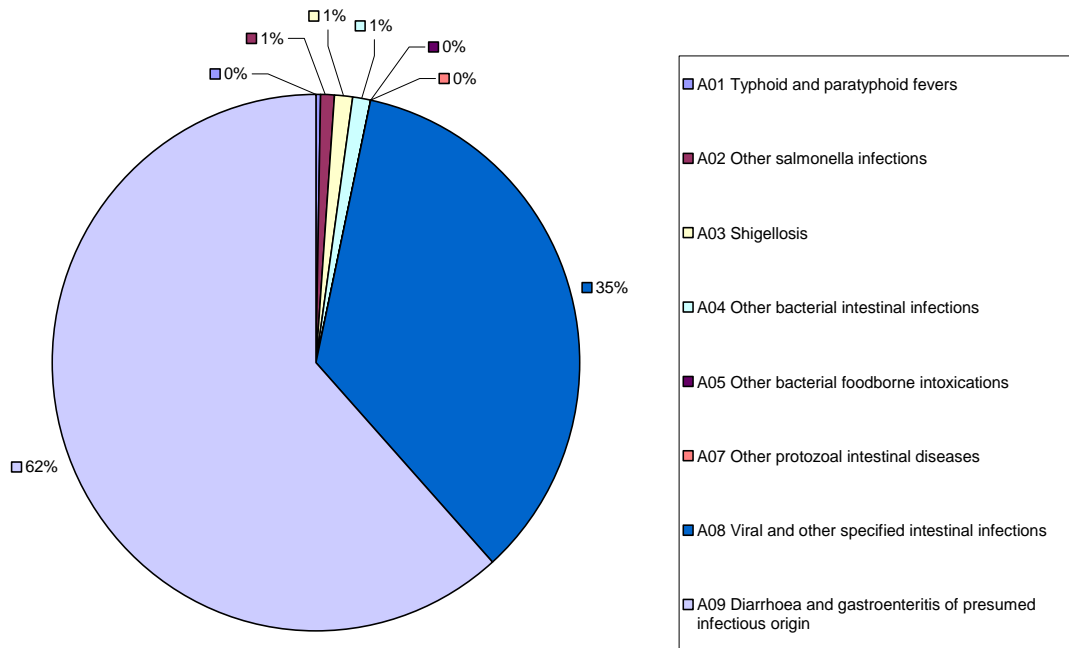


Figure 5: Causes of Gastroenteritis in Hospitalised Pacific Children (0-4 yrs), Counties Manukau, 2002-04. (Courtesy of G. Jackson, CMDHB)



Concern about the high rates of paediatric gastroenteritis hospital admissions in South Auckland culminated in a meeting on 14 April, 2005 of a working party consisting of representatives from the Ministry of Health, New Zealand Food Safety Authority, Pacific Island health providers, Counties Manukau DHB and the Auckland Regional Public Health Service. It was agreed that an investigation to ascertain the major etiologies of gastroenteritis for those children admitted to paediatric hospital in Auckland would be an important first step towards considering measures to reduce hospitalisation rates. The decision was made to undertake an investigation, which would involve routine faecal testing on all children admitted to hospital during a set time period. The investigation was to include a clinical audit, with the cooperation of clinical and laboratory staff, and with Dr. Greg Simmons leading the study in his statutory role as Medical Officer of Health under the Health Act 1956.

AIMS

The aim of this investigation was to identify the etiology of gastroenteritis among children admitted to paediatric hospitals in Auckland, and to describe the associations between paediatric gastroenteritis hospitalisation rates and factors such as ethnicity and neighbourhood level deprivation.

Further, the study aimed to compare these rates to those identified by a major Auckland community laboratory, to enable an estimate to be made of overall community rates in the Auckland region during the study period.

The ultimate goal of the investigation was to gain information to enable more effective targeting of public health measures (including health education and health promotion) to reduce disease transmission, and thus the burden of illness among Auckland children.

METHODS

This investigation was undertaken as a prospective case series, involving two sites. During the study period of July 1 to December 31, 2005, demographic and clinical data were collected on children less than fifteen years old diagnosed with gastroenteritis at the emergency departments of Auckland's two paediatric hospitals, Starship and Kidz First. Cases seen at other emergency departments and clinics, but not admitted to a paediatric hospital, were not included in the investigation.

Data Collection

Case Definition

In order to be eligible for inclusion in the investigation, children had to meet the following case definition:

- Have gastroenteritis, defined as 3 or more loose stools in previous 24 hours, occurring as part of an acute illness of less than 7 days duration, with or without nausea, vomiting, fever or abdominal pain
- Be less than 15 years of age.

Potential cases were excluded from the study if they met one of more of the following criteria:

- Cases without a primary diagnosis of gastroenteritis, and where there was evidence in clinical notes of other potential causes of diarrhoea, such as diarrhoea as an antibiotic side-effect
- Cases with nosocomial diarrhoea (i.e. contracted during hospital admission)
- Cases without a sufficient faecal specimen for any testing.

Those cases with some faecal testing results, but an insufficient amount of specimen to complete the whole array of tests, were still included in the study. Records were kept of the number of these cases.

Faecal Testing

All cases of primary gastroenteritis presenting at the two emergency departments during the six-month study period were to have two faecal specimens taken by clinical staff. Staff members were required to fill laboratory containers at least two thirds full, label the specimens fully and send them to the local hospital laboratory for testing (LabPlus at Starship Hospital and Middlemore Hospital (MMH) laboratory at Kidz First Hospital). All testing was to be completed at the hospital laboratories with the exception of the extra viral testing, which was done at the Institute of Environmental Science and Research (ESR) in Porirua. There was an agreed laboratory protocol for the study at each hospital (see Appendix 1).

Faecal testing included microscopy, followed by culture for bacterial pathogens, antigen enzyme immunoassay tests (ICT type) for rotavirus antigen (and at MMH, adenovirus antigen), antigen enzyme immunoassay tests (ICT type) for parasites and further special viral tests at ESR.

Following microscopy, laboratories were to test faeces for the following pathogens:

Bacteria:

- *Yersinia*
- *Campylobacter*
- *Shigella*
- *Salmonella*
- *Vibrio* spp.
- *Aeromonas hydrophila*
- *Listeria*
- Enterohemorrhagic *Escherichia coli* (EHEC)

Protozoa:

- *Giardia*
- *Cryptosporidium*

Viruses:

- Rotavirus group A
- Adenovirus 40/41
- Norovirus
- Astrovirus
- Sapovirus

Testing for protozoa was performed at MMH laboratory for both hospitals. Where the amount of faecal sample was inadequate for the full gambit of tests, testing was to be performed in the following order of preference:

1. Bacterial enteric pathogens (each hospital)
2. Norovirus (ESR)
3. Rotavirus (each hospital)
4. Protozoa (MMH laboratory)

Demographic Data including Ethnicity

Demographic data, including the date of birth, residential address, gender and ethnicity of each case, were collected from hospital registration records and hospital laboratory reports and transferred to a data collection form (see Appendix 2). Starship data were collected by the first author, while Kidz First data were primarily collected by Dr. David Montgomery, and reviewed by the first author.

Ethnicity information on hospital records is recorded at Level 2 of the Statistics New Zealand ethnicity classification system. Level 2 contains twenty-five ethnicity codes, which offer more detail than the Level 1 'European, Maori, Pacific Island, Asian and

Other' (New Zealand Health Information Service 2004). An individual patient can nominate up to three ethnicities, which are then prioritised for coding purposes, so that each individual is registered as belonging to only one ethnic group. In reality, fewer than 0.5% of ethnicity recording includes three ethnicities, which may reflect the approach taken in seeking ethnicity information from patients (New Zealand Health Information Service 2004). The method of prioritisation of ethnicity data ensures that each patient is only counted once in the ethnic-specific hospital discharge data for each event.

Clinical Data

Time of stay at hospital was derived by subtracting admission date and time from discharge date and time, and then rounding the time to the nearest hour. Clinical data were collected from both hospital discharge summaries and, in the case of Starship hospital, emergency department clinical notes. Details of the illness, such as symptoms and duration of illness prior to admission, were collected along with details of treatment, such as type of rehydration therapy. Hospital faecal testing results were collected from paper copies of laboratory reports, both from hospital labs and from ESR. Electronic laboratory reports were used to confirm results, where necessary. Both MMH and ESR sent periodic updates of collated laboratory results.

Notification Data

Under the Health Act 1956, the following types of gastrointestinal illness are notifiable to the Medical Officer of Health (Ministry of Health 2005a) :

- Cholera (Infection with *Vibrio cholerae*)
- Cryptosporidiosis
- Giardiasis
- Listeriosis
- Salmonellosis
- Shigellosis
- Typhoid and paratyphoid fever
- Yersiniosis
- Gastroenteritis

Not every case of acute gastroenteritis is notifiable. Only those cases from a person in a high risk category, as a food handler or childcare worker, where there is a suspected common source, or where there is a single case of bacterial, chemical or toxic food poisoning, are notifiable.

In this case series, faecal testing was performed for each of the notifiable types of bacterial gastroenteritis. These clinical data were then compared to the notification data received by the Auckland Regional Public Health Service. Data were retrieved from the EpiSurv database at the Auckland Regional Public Health Service, by a Surveillance Support Officer in the disease investigation team.

Community Laboratory Data

Community laboratory data were received from Diagnostic Medlab in a collated form, and were not linked to hospital data. In Auckland the majority of community laboratory services are delivered by Diagnostic Medlab (DML), with Southern Community Laboratory (SCL) having provided about 5% of lab services until they withdrew their Auckland-based services in September, 2005. As data for this investigation were collected only from Diagnostic Medlab, they can be assumed to underestimate true case numbers by about two percent.

Regional Hospitalisation Data

Hospital discharge data for paediatric gastroenteritis (as a primary diagnosis) were obtained by requesting data from the National Minimum Dataset (NMDS), held by the New Zealand Health Information Service (www.nzhis.govt.nz). Data was obtained for July 1 to December 31, 2005 for all three Auckland DHBs, courtesy of Counties Manukau DHB. NMDS data are based on hospital medical discharge diagnoses which are then coded by clerks according to the International Classification of Disease (ICD-9). Only those cases formally admitted to hospital (defined as being in hospital >3 hours) are included in this NMDS dataset. There are multiple different codes for infectious gastroenteritis, and the requested data included all codes listed in Figure 6. (Note that although rotavirus gastroenteritis has its own code, A08.0 Rotaviral enteritis, when the NZHIS publishes its data rotavirus data gets aggregated into “A08 Viral and unspecified intestinal infections”.) Given that routine faecal testing is not performed at the two hospitals in this investigation, the pathogen-specific ICD-9 coding is of limited use and underestimates true frequencies. Instead, the overall number of paediatric gastroenteritis admissions by five-year age-group and major ethnic group were the important data gathered from the NMDS dataset.

Figure 6: ICD-9 Infectious Gastroenteritis Codes

A01 Typhoid and paratyphoid fevers
A02 Other salmonella infections
A03 Shigellosis
A04 Other bacterial intestinal infections
A05 Other bacterial foodborne intoxications
A07 Other protozoal intestinal diseases
A08 Viral and other specified intestinal infections
A09 Diarrhoea and gastroenteritis of presumed infectious origin

Data Analysis

Data from all sources were reviewed and compared to check for accuracy and completeness. Duplicate faecal specimens for a single case were removed from each of the hospital and community laboratory datasets. Where data were incomplete or missing, further details were requested of hospital or laboratory staff.

Data were entered into EpiInfo software, version 3.3.2 (Dean, Arner et al. 2000), using a template (see Appendix 2). Most of the analysis was completed in EpiInfo, however the dataset was exported into a Microsoft Excel, version 4, file for further analysis. A detailed description of the analysis follows.

Frequencies and Proportions

Proportions and 95% confidence intervals (CI) were calculated for each pathogen for the whole study sample, as well as by ethnicity and NZDep2001 decile.

Chi Square tests were performed to assess the statistical significance of differences in frequencies between groups. P-values were subject to Yates' correction, except where a cell contained a value less than five, in which case the Fisher exact test was applied.

P-values of less than 0.05 were deemed statistically significant.

Socioeconomic Status

New Zealand Deprivation Index (NZDep2001) deciles were calculated based on a case's residential address, as stated in the hospital registration records. NZDep2001 deprivation scores apply to areas rather than individual people (Ministry of Health 2002a). Although it is a small area rather than a household measure, the rating is used here as a proxy for the socioeconomic status of a child's family or household.

The New Zealand Deprivation Index, of which NZDep 2001 is the second version, utilised pooled data from the 2001 census for nine variables representing eight dimensions of deprivation, to develop a deprivation score reported at the level of meshblocks, or small neighbourhood areas. Scores were then ranked into ten decile ratings, with decile 1 representing the least deprived ten percent of New Zealand meshblocks and decile 10 the most deprived ten percent.

In this investigation, household addresses were coded to a meshblock, using an application entitled RetroGeocoder, version 2.1.0 (ESR). Each meshblock number was then matched to an NZDep2001 rating, as supplied by the Ministry of Health (Ministry of Health 2002b).

A regression analysis on NZDep2001 decile scores was performed using GeoDA™ data analysis software (Anselin 2004), to measure the association between the score and admission to hospital with gastroenteritis in the investigation. Numbers of cases for each NZDep2001 decile rating were transformed to a logarithm, and a regression analysis was performed.

Rates

It was not possible to get access to population estimates for the Auckland region by all the age and level 2 ethnic groups identified in this investigation. Current Statistics New Zealand population data, derived from the 2001 census, is unreliable for Auckland children in 2005, given the constantly changing demography of the region. The data on numbers and ethnicities of children 0-4 years old were particularly

difficult to ascertain. As a result, three different datasets were accessed for ethnic-specific and age-specific population denominator figures for the different age-groups in the investigation, in order to try to match numerator and denominator data most accurately. These datasets included hospital birth data from the National Minimum Dataset (NMDS), Pacific children's population figures developed for the Meningococcal B Vaccination Programme in Auckland, and DHB population projections for the Auckland region. The choice of dataset for denominator data varied, depending on which age-group rates were being calculated for.

Age-Specific and Ethnic-Specific Rates: 0-4 year olds

In keeping with DHB practise, population data for the 0-4 yrs age group was reconstructed from NMDS birth data, in order to more accurately match this age group to the population at the time of the study. Approximately 98 percent of births occur in public hospitals in the Auckland region, so birth cohort data can be assumed to give a reasonable population estimate for this age-group (Pers. Comm. G.Jackson, CMDHB). As the majority of this investigation's cases were in this under 5 years age-group, hospital data was deemed likely to be more accurate for both age and ethnicity data. All age-specific rates for age-groups under 5 years were calculated using hospital birth data from NMDS, including the overall 0-4 year old rate.

Ethnic-specific rates (by Level 1 ethnic groupings) for the under 5 year olds were calculated using recent hospital birth data from the NMDS, which had been redefined into level 1 ethnicity codes for District Health Board population projections. As stated above, hospital data from NZHIS prioritises Maori first, then Pacific, and then Asian, only allowing for identification with one ethnic group. As a result, these figures could not be used to calculate rates for ethnic subgroups, such as Samoan and Tongan.

One goal of this investigation was to gather more detailed information on Pacific children with gastroenteritis, and so an alternative source of ethnic-specific population estimates for Pacific children 0-4 years in Auckland was found. Figures from the Pacific children's ethnic-specific population projections for the Auckland Meningococcal B Vaccination Programme (MeNZB) were used for the major Pacific subgroups in the investigation.

In order to create population projections for each specific subgroup, a method known as the "Pro Rata Method of Estimating Pacific Peoples Populations" was used by the MeNZB programme, on the advice of Statistics New Zealand. As projections were derived from 2001 census data, there were limitations to them, as outlined in the National Immunisation Register Datamart Manual (courtesy of Christine Roseveare, Ministry of Health). For example, as Statistics New Zealand has no agreed method of prioritising the ethnicities of individuals who identify with more than one Pacific group (e.g. Samoan and Tonga), those individuals were excluded from the ethnic subgroup data. As a result, the population denominators for Pacific subgroups are probably lower than actual values, which mean that the rates presented in this report may be an over-estimate of true rates for these ethnic groups. These population estimates were used to calculate ethnic-specific rates for the three most frequently occurring Pacific ethnicities in this age-group.

Age-Specific and Ethnic-Specific Rates: 5-14 year olds

In contrast, age-specific rates for the 5-14 year old age-groups were calculated using population figures from Statistics NZ 2001 Census data.

Ethnicity population-level data for the 5-14 year old age-groups were taken from DHB population projections and so were available only for major (level 1) groupings (Maori, Pacific, Other). The DHB projections are performed by Statistics New Zealand, based on a formula that incorporates the factors impacting on population change since the last census, such as aging, births, deaths and migration. Some estimation had to be done for the 'Asian' population figures used in the analysis. Population estimates from local authorities, and considering New Zealand growth estimates, were used to develop population denominators for children of Asian ethnicities (courtesy of G. Jackson, Counties Manukau DHB).

For the purposes of matching ethnic groupings of cases to population-level figures, all Pacific ethnicities were grouped together, as well as all 'Asian' ethnicities, and NZ European was grouped with all others. The Asian grouping included cases identified as Chinese, Asian, Indian, Southeast Asian and 'Other Asian' in hospital records. The "Other" category included cases identified as NZ European, 'Other European', African and 'Other'.

Confidence Intervals

Ninety-five percent confidence intervals (CI) were calculated for all rates, for a standard population of 100,000, using the following formulae:

$$\text{Upper Limit} = (100,000/n) (d+1.96 \times \text{square root of } d)$$

$$\text{Lower Limit} = (100,000/n) (d-1.96 \times \text{square root of } d)$$

Where **d**=number of cases, **n**=population denominator of the rate
(see <http://www.health.state.pa.us/hpa/stats/techassist/cicruderate.htm>)

All rates were expressed as the number of cases per 100,000 population per year. Frequencies of hospital visits, which were collected for a six-month period, were doubled to annualise them.

Regional Rates

In order to get a regional picture of the total number of paediatric gastroenteritis hospital admissions during the study period, the NMDS data for the same period were compared to data from the case series. All cases of paediatric (0-14 yrs of age) gastroenteritis admitted during the period July 1 to December 31, 2005 to hospital facilities in Auckland DHB and Counties Manukau DHB were added together.

The factor differences between the hospital discharge data (NMDS) and the investigation sample were calculated by dividing the case frequencies from the total case number by the investigation's sample to give a case ascertainment ratio, reported as a multiplication factor. The same process was used to calculate the age-specific case ascertainment factor differences, as well as the ethnic-specific ones. The multiplication factor difference between the hospital discharge data (NMDS) and the investigation data was applied to the rates reported in earlier sections of this report, to give more realistic estimates of regional rates. As the factors varied by five-year age-

group and also by ethnic group, the relevant multiplication factor for each rate was applied (see Tables 9 and 10).

Distributions

Where the distribution of a continuous variable was being compared between two groups, such as length of time in hospital for viral gastroenteritis compared with bacterial, an ANOVA test was performed in Microsoft Excel, version 4.

Primary Care Comparison

The Diagnostic Medlab (DML) frequencies of pathogen-positive faecal tests were calculated in order to compare them to the regional hospital-based frequencies. Only those pathogens tested for in primary care could be reported on. Each pathogen-specific frequency from this investigation was multiplied by a factor of 6.8, the difference between the sample size and the total number of paediatric gastroenteritis cases admitted to Auckland hospitals during the period of study. As the population base for DML was the same as for the hospitals in this investigation (the Auckland region), it was not necessary to calculate rates for this comparison. The pathogen-specific frequencies were divided into each other to give a ratio or factor difference between cases reported in primary care and in paediatric hospitals in the Auckland region.

RESULTS

Demographics

Between July 1 and December 31, 2005 one hundred and thirty-one cases were investigated from the two study sites. Kidz First Children's Hospital in South Auckland provided 92 (70.2%) of the cases and Starship Children's Hospital in Central Auckland provided the other 39 (29.8%).

A total of 28 potential cases were excluded from the case series, five from the Starship sample and 23 from Kidz First sample. The most common reason for exclusion was that diarrhoea was presumed due to causes other than gastroenteritis, such as having another primary diagnosis or being on oral antibiotics. A few cases were excluded due to no faecal specimen being available for testing.

The NMDS hospital discharge data became available for the study period in late March, 2006. The case series represented 14.7 percent of the total number of cases discharged from Starship and Kidz First hospitals during the investigation period, with a diagnosis of gastroenteritis.

Age

The majority of the children in the investigation (92.4%) were under 5 years of age (see Figure 7 and Table 1). The median age in years was 1.0 year (range 0-13), and in months was 13.0 months (range 0-164). Two cases (1.5%) were less than one month old. The highest number of admissions was among one year olds, yet the highest rate of admission was in the 6-11 month age-group (see Table 1). When divided into smaller age brackets, rates remained high up until 5 months of age, peaked by 11 months, and then dropped to much lower rates by 2 years of age. Admission rates then dropped dramatically after 4 years of age (see Figure 8, p.16).

Figure 7: Distribution of Auckland Paediatric Gastroenteritis Cases by Age



Table 1: Auckland Paediatric Gastroenteritis Case Series: Demographic Details of Cases (n=131)

DEMOGRAPHIC VARIABLE	NUMBER of CASES (%)	GASTROENTERITIS ADMISSION RATES per 100,000 per year (95% CI)*
AGE		
0-5 months	19 (14.5)	379 (208.6-549.4)
6-11 months	34 (26.0)	648.8 (430.7-866.9)
12-23 months	51 (38.9)	486.8 (353.2-620.4)
2-4 yrs	17 (13.0)	56.8 (29.8-83.7)
Total 0-4 yrs	121 (92.4)	238.7 (196.2-281.3)
5-9 yrs	8 (6.1)	16.4 (5.0-27.8)
10-14 yrs	2 (1.5)	4.0 (0.0-9.5)
TOTAL		87.7 (72.7-102.8)
GENDER		
Female	47 (35.9)	64.8 (46.2-83.2)
Male	84 (64.1)	109.6 (86.1-132.9)
ETHNICITY (Major Ethnic Group)**		
NZ Maori	21 (16.0)	74.9 (42.9-106.9)
NZ European + Other	46 (35.1)	70.6 (50.2-91.1)
All Pacific	47 (35.9)	155.5 (111.0-200.0)
Asian	17 (13.0)	65.6 (34.4-96.7)

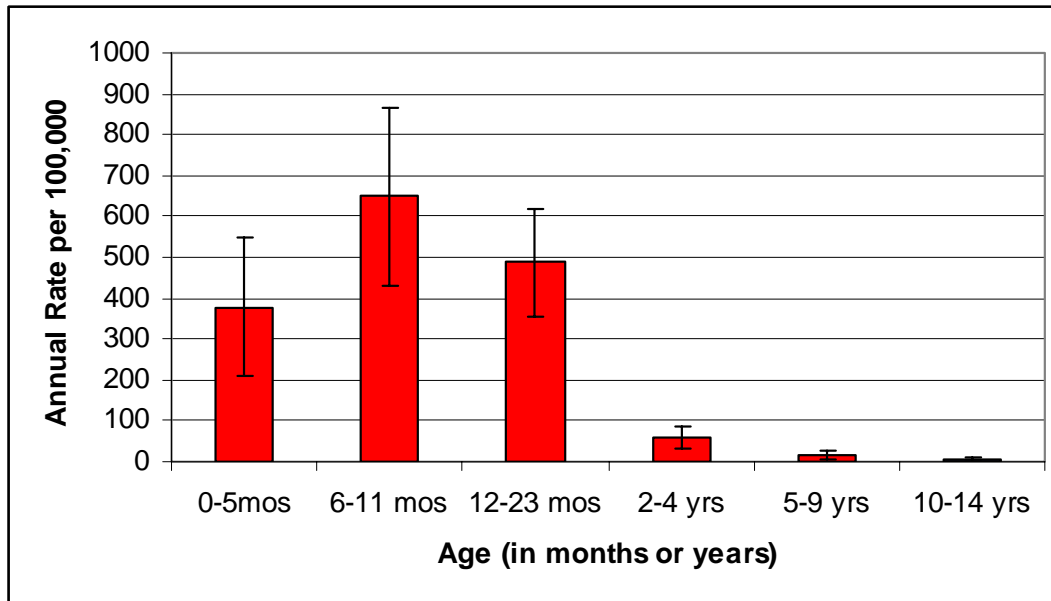
*Standardised to the population served by the three Auckland DHBs (see Methods Section)

**See below for more detailed breakdown of cases by ethnic group.

Gender

There were more boys (n=84) than girls (n=47) in the investigation, and this difference was statistically significant (RR=1.69, 95%CI 1.18-2.42, Chi Square 8.04, p=.005), with the overall rate of gastroenteritis admissions for boys being significantly higher. A comparison of pathogen frequencies between boys and girls revealed that there were no statistically significant differences in pathogen frequency by gender.

Figure 8: Auckland Gastroenteritis Case Series Admission Rates by Age



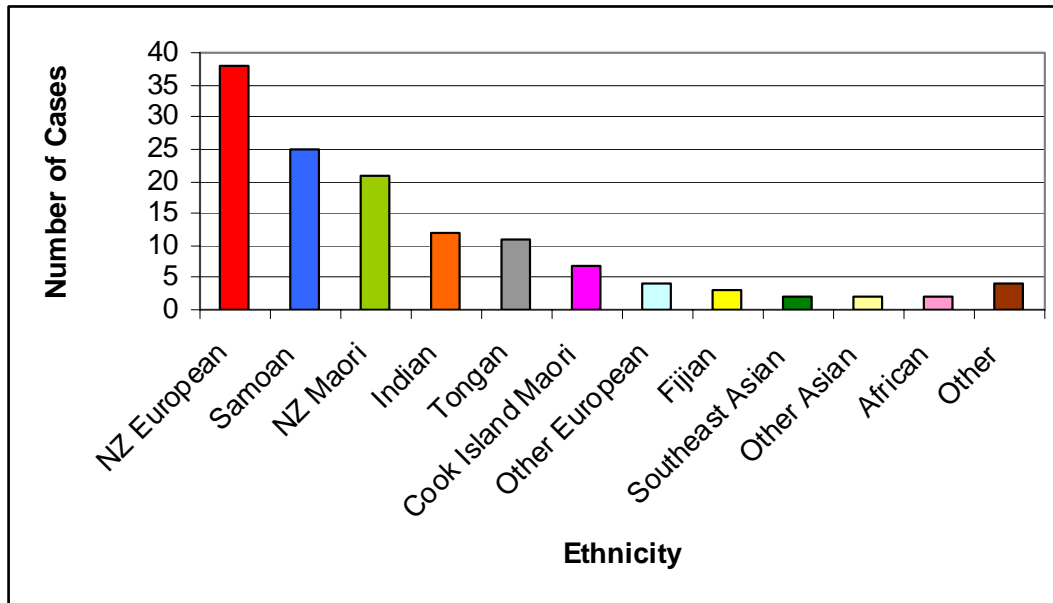
Ethnicity

There were twelve different ethnic groupings identified on the hospital registration records of cases, the largest numbers being among NZ European and Samoan. By major ethnic group (Statistics NZ level 1), the ethnic grouping of cases was as follows: 35.9% Pacific, 29% NZ European, 16.0% NZ Maori, 13.0% Asian and 6.4% 'Other' (see Figure 9). There were no obvious siblings among the cases in the series, matching data by surname and address.

The rates of admission were significantly higher for all Pacific children, than for any other major ethnic grouping (see Table 1 p.15). Ethnic-specific rates for Pacific children were able to be calculated for the three largest ethnic groups in the sample (Samoan, Tongan and Cook Island), but only for children under 5 years, as the population data were available for these groups. Rates of admission for these children were highest among Samoan and Tongan children, with Cook Island children having the next highest rate (see Figure 10).

From Table 1 it is evident that, although much lower than Pacific rates, Maori children also had higher rates of hospital visits than either European or Asian. However, the confidence intervals were wide for Maori given their relatively low number in the case series.

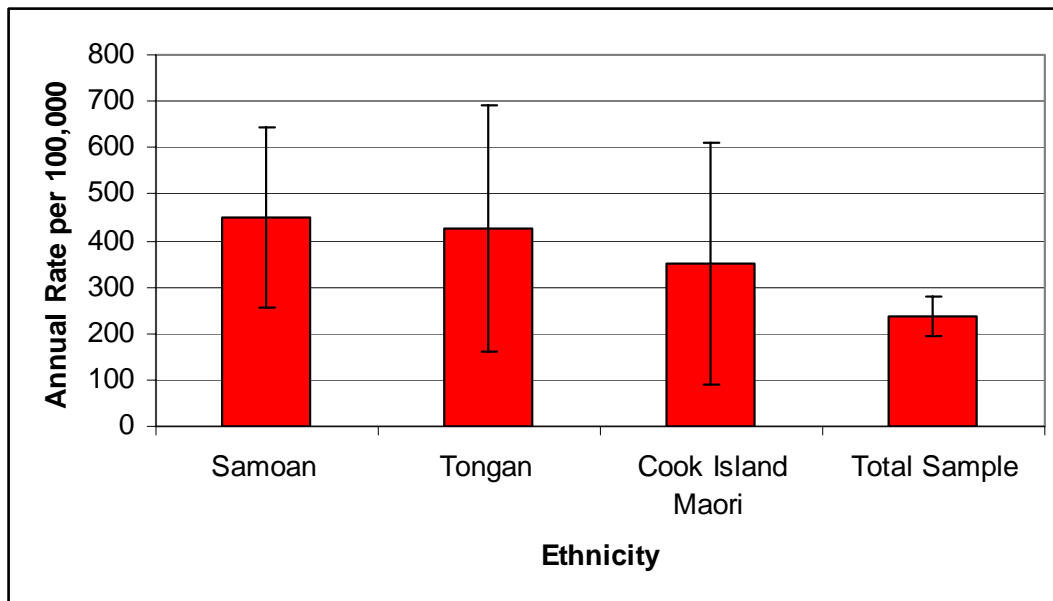
Figure 9: Distribution of Auckland Paediatric Gastroenteritis Series Cases by Ethnicity* (n=130, data missing for 1)



*Ethnicity=ethnic group as stated on hospital registration records.

NOTE: Rates would be more useful than frequencies, however rates could not be calculated for this level of ethnicity data, due to population data limitations.

Figure 10: Auckland Paediatric Gastroenteritis Case Series Admission Rates, Age 0-4 Years: Comparison of Three Pacific Ethnic Groups with Total Sample*



*Note: Population data were taken from those used for the Meningococcal B Vaccination Programme for the Auckland region. Rates for Fijian children were not calculated as the population data for 0-1 year olds was unavailable.

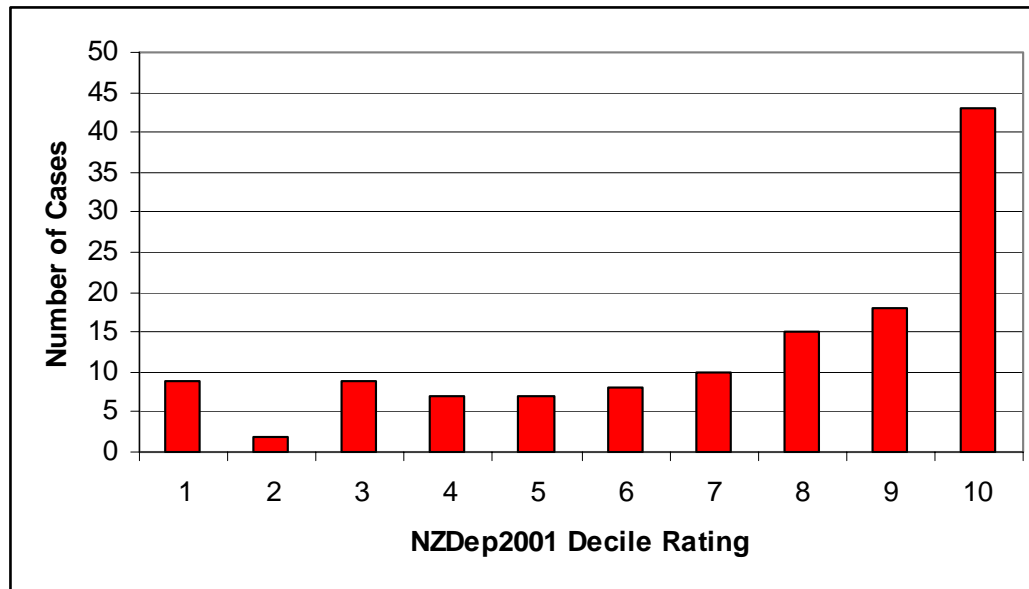
Relative Deprivation Level

Of the 131 cases, 128 (98%) were able to be matched to a meshblock in the Auckland region, and all of these matched exactly. One case had an Australian residential

address, which could not be coded. Two cases had Auckland addresses that were unable to be located, and were presumed inaccurate.

Ninety-four (72%) of the study children resided in areas rated as decile 6 or higher on the NZDep 2001 rating scale (see Figure 11).

Figure 11: Distribution of Series Cases by NZDep2001 Decile Ratings (n=128*)



*NZDep2001 decile ratings were derived from residential addresses, as listed in hospital registration records. Three addresses were not able to be coded.

For the 128 geocoded addresses, the median NZDep 2001 score was decile 8. In Figure 11, the number of cases rises with increasing NZDep 2001 decile ratings, demonstrating a gradient between paediatric gastroenteritis hospitalisations and residing in a more deprived neighbourhood. This gradient is statistically significant (Coefficient=2.78, CI 0.67-4.89, p=0.016).

District Health Board (DHB) Region

Analysis of cases by their residential addresses revealed that children lived in all three of Auckland's DHB regions. Twenty of the children (15.3%) were from the Waitemata DHB region (north and west Auckland), twenty-five (19.1%) from the Auckland DHB region (central Auckland) and the remaining eighty-six (65.5%) were from Counties Manukau DHB region (south and southeast Auckland).

Clinical Findings

Cases had variable combinations of vomiting, fever and dehydration on admission to hospital (see Table 2). All cases had diarrhoea, as it was a requirement for inclusion in the investigation. Vomiting was more often a feature of viral gastroenteritis among cases than bacterial, and this finding was statistically significant (RR=1.41, CI 1.02-1.96, Chi Square=7.85, p=.005). There were no significant clinical differences on admission between cases with viral versus bacterial gastroenteritis for either fever (greater than 38 degrees Celsius) or dehydration. Fever was most commonly evident in cases whose faeces tested positive for *Shigella*, enterohemorrhagic *E.coli*, adenovirus or astrovirus. Dehydration was a common feature of cases, and 74% were reported as clinically dehydrated in the emergency department clinical notes. Dehydration was more common among children with rotavirus and astrovirus infection, as well as shigellosis and enterohemorrhagic *E.coli* gastroenteritis.

Table 2: Frequency of Clinical Findings by Pathogen (%) (n=131)

PATHOGEN*	VOMITING	FEVER	DEHYDRATION
BACTERIA:			
<i>Campylobacter</i>	8 (66.7)	6 (50.0)	6 (50.0)
Enterohemorrhagic <i>E.coli</i>	1 (100)	1 (100)	1 (100)
<i>Salmonella</i>	2 (50.0)	2 (50.0)	3 (75.0)
<i>Shigella</i>	2 (66.7)	3 (100)	3 (100)
VIRUSES:			
Adenovirus	5 (100)	4 (80.0)	2 (40.0)
Astrovirus	4 (80.0)	4 (80.0)	4 (80.0)
Norovirus	6 (85.7)	2 (28.6)	3 (42.9)
Rotavirus	68 (90.7)	28 (37.3)	61 (81.3)
Sapovirus	1 (100)	0	0
NO PATHOGEN FOUND	19 (63.3)	12 (40.0)	14 (46.7)

*No Protozoa were identified in any case.

Laboratory Findings

Pathogens

For most cases entered into the investigation only one useful faecal specimen was taken and tested. Due to inadequate specimens or laboratory error, some cases had incomplete testing performed on their one faecal specimen. In total there were 132 pieces of missing laboratory data on faecal enteric pathogens. The completeness of the test data varied between bacteria, protozoa and virus testing (see Table 3). Rotavirus testing occurred for all but one case and 97.7% of cases had complete bacterial testing. Testing rates for protozoa and for other viruses (performed at ESR in Porirua) were much lower, at 84.7% for each. As a result, bacterial pathogen frequencies underestimate the true level by two percent, while frequencies of protozoa and of viral

pathogens, other than rotavirus, are underestimated by fifteen percent. Cases with no pathogen found are discussed in a separate section of this report.

Table 3: Frequency of Cases with Complete Faecal Testing

PATHOGEN BEING TESTED FOR	TOTAL NUMBER of CASES (%)
BACTERIA:	
<i>Aeromonas hydrophila</i>	128
<i>Campylobacter</i>	129
<i>Enterohemorrhagic E.coli</i>	128
<i>Listeria</i>	129
<i>Salmonella</i>	129
<i>Shigella</i>	129
<i>Vibrio spp.</i>	129
<i>Yersinia</i>	128
TOTAL cases with complete bacterial testing	128 (97.7)
PROTOZOA:	
<i>Cryptosporidium</i>	111
<i>Giardia</i>	111
TOTAL cases with complete protozoa testing	111 (84.7)
VIRUSES:	
Adenovirus	116
Astrovirus	112
Norovirus	112
Rotavirus	130
Sapovirus	112
TOTAL Cases with complete viral testing	111 (84.7)

The incomplete testing primarily related to inadequate samples, which meant that residual faecal specimens were not sent on to Middlemore Hospital laboratory from Starship Hospital for protozoan testing, or from both hospital laboratories to ESR in Porirua for further viral testing. Note that rotavirus testing, and some adenovirus testing, was performed at hospital laboratories and the rotavirus testing rate was nearly 100%. Starship Hospital cases had lower complete testing rates than did cases from Kidz First Hospital, however Starship cases required faecal testing to be done at three different laboratories.

The distribution of cases by pathogen is displayed in Figure 12. The frequency and rate of each pathogen tested for on faecal testing in the investigation are listed in Table 4. There was a much higher frequency of viral enteric pathogens (87 cases, 77.7%) found among cases than bacterial (20 cases, 15.6%). For every one case of bacterial gastroenteritis there were four cases of viral gastroenteritis and, in this sample, no parasitic gastroenteritis.

Table 4: Frequencies and Annual Rates of All Pathogens Tested for in Faecal Specimens*

PATHOGEN (number of cases tested for this pathogen)	TOTAL NUMBER of CASES (% testing positive)**	OVERALL PATHOGEN-SPECIFIC RATES (95%CI)***
BACTERIA		
<i>Aeromonas hydrophila</i> (n=128)	0	0
<i>Campylobacter</i> (n=129)	12 (9.3)	8.0 (3.5-12.6)
Enterohemorrhagic <i>E.coli</i> (n=128)	1 (0.8)	0.7 (0.0 -2.0)
<i>Listeria</i> (n=129)	0	0
<i>Salmonella</i> (n=129)	4 (3.1)	2.7 (0.1-5.3)
<i>Shigella</i> (n=129)	3 (2.3)	2.0 (0-4.3)
<i>Vibrio</i> spp. (n=129)	0	0
<i>Yersinia</i> (n=128)	0	0
TOTAL Bacterial Cases (n=128)	20 (15.6)	13.4 (7.5-19.3)
PROTOZOA		
<i>Cryptosporidium</i> (n=111)	0	0
<i>Giardia</i> (n=111)	0	0
TOTAL Protozoa (n=111)	0	0
VIRUSES		
Adenovirus (n=116)	5 (4.3)	3.3 (0.4-6.3)
Astrovirus (n=112)	5 (4.5)	1.7 (0.2-3.1)
Norovirus (n=112)	7 (6.3)	4.7 (1.2-8.2)
Rotavirus (n=130)	75 (57.7)	50.2 (38.9-61.6)
Sapovirus (n=112)	1 (0.9)	0.7 (0.0-2.0)
TOTAL Viral Cases (n=112)	87 (77.7)	58.3 (46.0-70.5)
MULTIPLE PATHOGENS (n=131)	10 (7.6)	6.7 (2.5-10.8)
NO PATHOGEN FOUND (n=131)	30 (22.9)	20.1 (12.9-27.3)

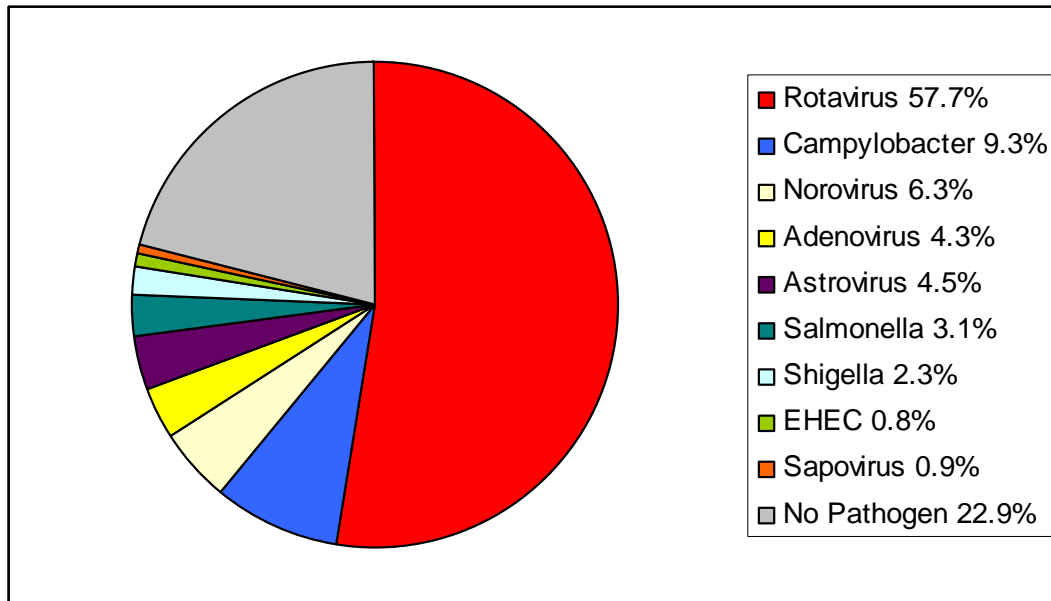
*Not all specimens had the full gambit of testing done. The number of cases tested for a given pathogen is listed in brackets, and is used as the denominator for proportions.

**Eight cases had two pathogens concurrently and two cases had three.

***Rates have been calculated for the population of the Auckland region, per 100,000.

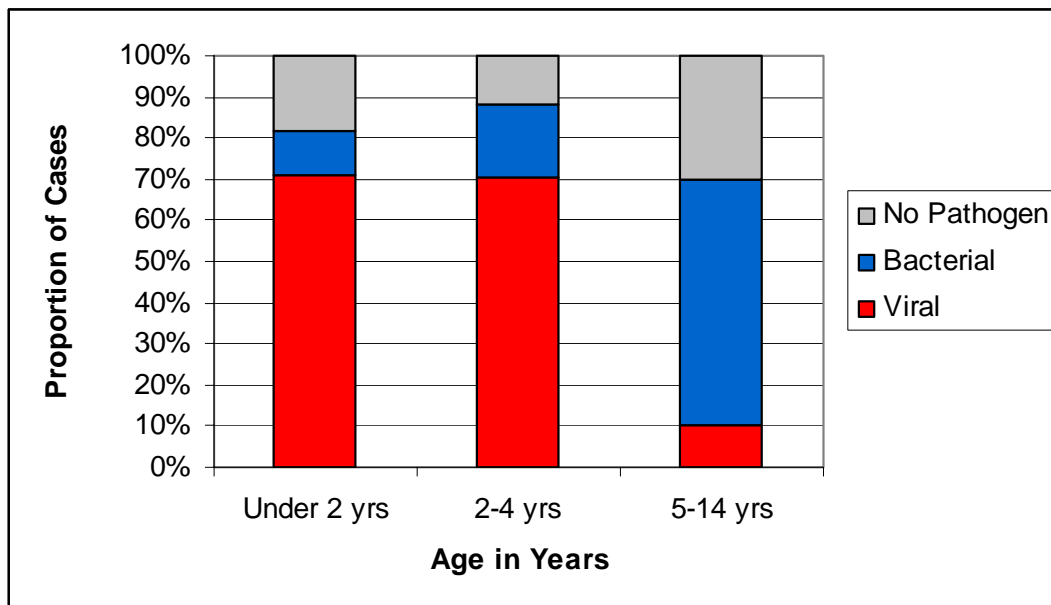
Looking at pathogens by age-group shows a similar pattern. There was a much higher proportion of viral enteric pathogens found, compared to bacterial pathogens, in children less than 5 years of age. However, from 5-14 years, bacterial pathogens accounted for sixty percent of cases (see Figure 13). Bacterial pathogens were reported with highest frequencies among Tongan, Samoan, Indian and European children, respectively, compared with others (see Figure 14).

Figure 12: Distribution of Cases by Pathogen (n=131)



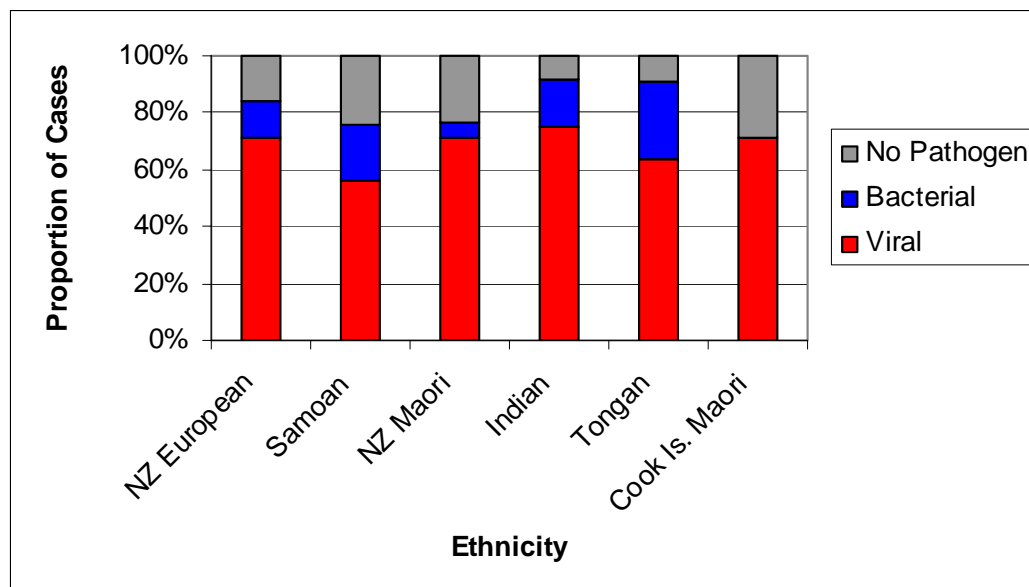
Note: The following pathogens were also tested for but there were no positive results: Giardia, Cryptosporidium, Vibrio, Aeromonas, Yersinia and Listeria. Ten cases (7.6%) had multiple pathogens.

Figure 13: Relative Proportions of Viral and Bacterial Pathogens by Age-Group



Note: Those cases (n=6) with both viral and bacterial pathogens are counted twice.

Figure 14: Relative Proportions of Viral and Bacterial Pathogens by Ethnicity*



*Only the top six ranking ethnic groups, as identified in hospital registration records, are reported here.

Notification of Cases to the Medical Officer of Health

There were twenty cases of bacterial gastroenteritis which were notifiable under the Health Act 1956. These included cases whose faeces tested positive for *Campylobacter*, Enterohemorrhagic *E.coli*, *Salmonella* and *Shigella*. Of the twenty cases, seventeen (85%) were notified to the Medical Officer of Health at the Auckland Regional Public Health Service by primary care or hospital medical staff. Two cases of salmonellosis and one case of campylobacteriosis were not notified to the Medical Officer of Health. The three cases who were not notified were children living in relatively deprived neighbourhoods with NZDep2001 decile ratings of 8, 9 and 10 respectively.

Rotavirus

Rotavirus rates were highest in the 6-11 month old children in the investigation, with an annual rate of 419.8 per 100,000 (95% CI 244.4-595.2) (see Table 5 for age-specific rates). Within the 6-11 months age-group, the highest rates were among Pacific children, followed by Maori children (see Figure 15). Overall in the 0-4 years age-group, the rate was lower at 146.0 per 100,000 (95% CI 122.7-179.3). The age-specific rates give a more accurate picture of the disease impact.

The pattern of rotavirus-positive cases was consistent with it being a seasonal disease, peaking in winter and spring. Case numbers were non-existent at the start of the study, peaked between mid-August and mid-September, and then fell gradually until early December (see Figure 16).

Table 5: Regional Age-Specific Rates for Rotavirus Gastroenteritis Hospitalisation

ETHNICITY	AGE (95% CI): 0-5 months	6-11 months	12-23 months	2-4 years	TOTAL 0-4 yrs
Maori	245.9 (0-586.6)	731.3 (146.1-1316.4)	325.0 (40.1-609.0)	0	172.2 (78.6-265.8)
Pacific	379.3 (7.6-751.1)	748.0 (229.7-1266.4)	310.6 (80.5-540.7)	77.1 (9.5-144.7)	221.0 (132.6-309.4)
Asian	0	346.2 (0-738.0)	289.6 (35.8-543.5)	78.9 (0-168.2)	153.0 (62.6-243.5)
European/Other	85.1 (0-203.0)	201.3 (24.9-377.7)	322.7 (164.6-480.8)	19.6 (0-41.8)	103.6 (63.8-179.3)
TOTAL	159.6 (49-270.1)	419.8 (244.4-595.2)	315.0 (207.5-422.5)	36.7 (15-58.4)	146.0 (122.7-179.3)

Figure 15: Rates of Rotavirus Gastroenteritis in Hospitalised Auckland Children 0-4 years, by Major Ethnic Group

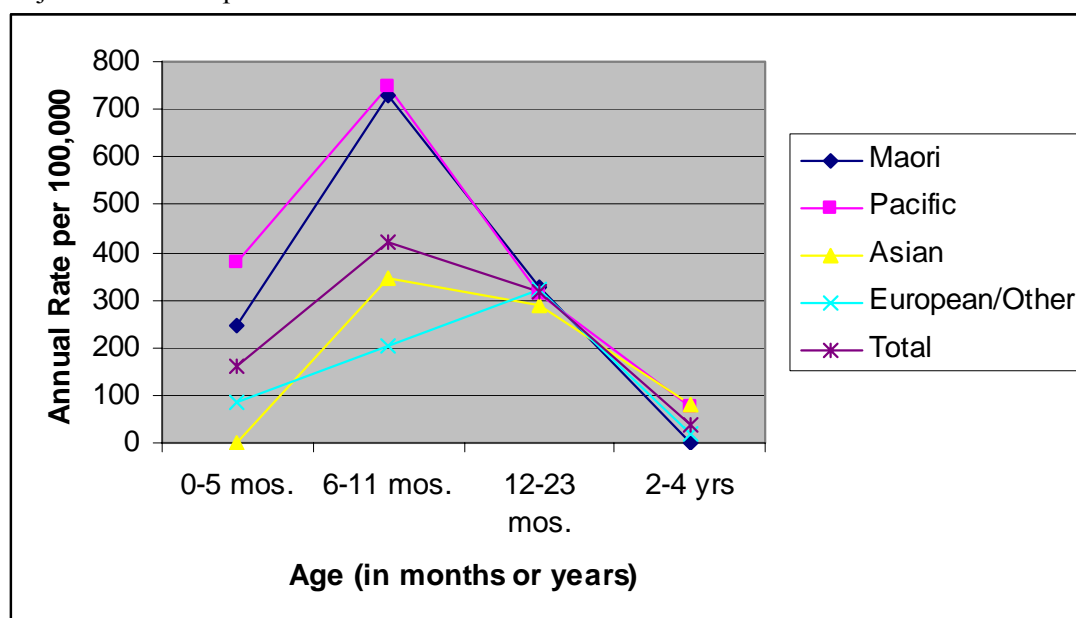
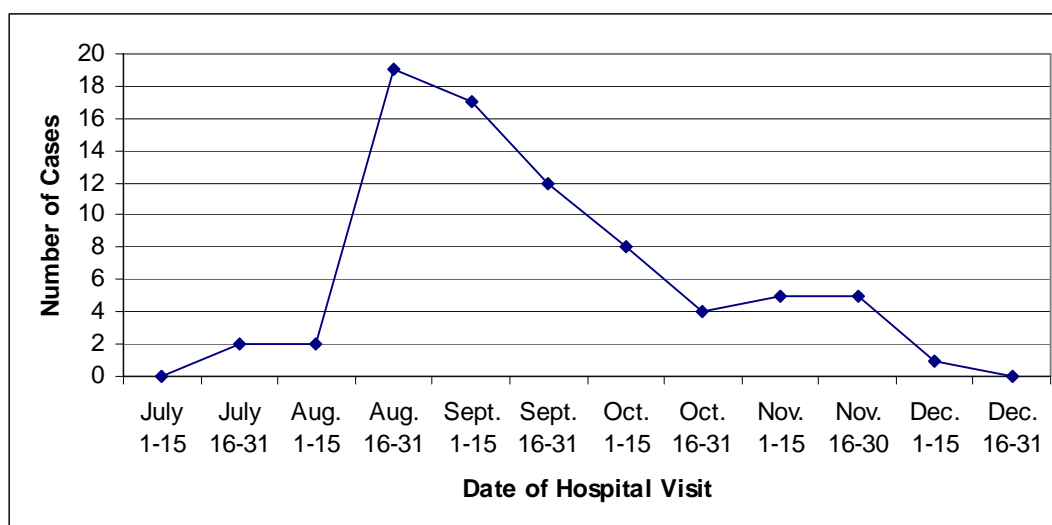


Figure 16: Auckland Paediatric Gastroenteritis Case Series: Epidemic Curve for Rotavirus-Positive Cases



Microscopy

Findings on microscopic examination of faecal specimens varied by pathogen (see Table 6). There were statistically significant differences in the presence of blood, white blood cells or mucus between those faeces which tested positive for bacterial pathogens (n=20) and those which tested positive for viral pathogens (n=87). Specimens with bacterial pathogens were significantly more likely to have blood, white blood cells or mucus on microscopy than those with viral pathogens. Those with bacterial pathogens were 9.6 times more likely to have blood (95%CI 3.74-24.47, Chi Square=27.27, p<.001), 6.3 times more likely to have white blood cells (95%CI 3.13-12.62, Chi Square=26.49, p<.001), and 5.4 times more likely to have mucus (95%CI 2.46-12.01, Chi Square=16.54, p<.001). Of note is the fact that six cases had both viral and bacterial pathogens on faecal testing, and so these data underestimate the true difference. Between ten and twenty percent of those cases with no pathogen found on testing had microscopic findings of blood, white blood cells or mucus.

Table 6: Frequency of Microscopic Findings by Pathogen (%) (n=131)

	Mucus	Red Blood Cells	White Blood Cells
PATHOGEN			
BACTERIA:			
<i>Campylobacter</i>	4 (33.3)	5 (41.7)	6 (50.0)
<i>Enterohemorrhagic E.coli</i>	1 (100)	1 (100)	1 (100)
<i>Salmonella</i>	2 (50.0)	3 (75.0)	3 (75.0)
<i>Shigella</i>	3 (100)	2 (66.7)	3 (100)
VIRUSES:			
Adenovirus	1 (20)	1 (20)	1 (20)
Astrovirus	2 (40.0)	1 (20.0)	1 (20.0)
Norovirus	0	0	0
Rotavirus	5 (6.7)	4 (5.3)	8 (10.7)
Sapovirus	1 (100)	0	0
NO PATHOGEN	6 (20.0)	3 (10.0)	5 (16.7)

Hospital Treatment

Length of Admission

There were a wide range of admission times for the study cases. While the case definition for the study initially stated that a case had to be at hospital for three hours or more (≥ 0.13 days), there were eighteen cases (13.8% of the sample) entered into the investigation whose stay at hospital was less (15.4% of the SSH sample and 13.0% of the MMH sample).

The median length of admission time varied by pathogen (see Table 7). The total number of hospitalisation days in the study was 155.4, with rotavirus gastroenteritis accounting for 73% of these (112.7 days). While the median length of admission time was higher for *Shigella*, the case numbers were very low in comparison to rotavirus, so that *Shigella* gastroenteritis only accounted for 3.3% of total admission time (5.08 days). The median length of admission time for viral gastroenteritis (0.63 days) was more than double that for bacterial gastroenteritis (0.29 days). The length of hospital admission time between those 75 rotavirus-positive cases (median 0.75 days) was significantly longer than for the 55 rotavirus-negative cases (median 0.17 days, ANOVA test $p=0.023$).

Table 7: Length of Admission by Pathogen

PATHOGEN	MEDIAN LENGTH OF ADMISSION in DAYS (range)
BACTERIA:	
<i>Campylobacter</i>	0.23 (0.08-2.00)
<i>Enterohemorrhagic E.coli</i>	0.17
<i>Salmonella</i>	0.29 (0.08-4.00)
<i>Shigella</i>	3.00 (1.00-3.00)
All Bacterial Pathogens:	0.29 (0.08-4.00)
VIRUSES:	
Adenovirus	0.17 (0.08-0.21)
Astrovirus	0.67 (0.21-4.00)
Norovirus	0.13 (0.08-2.00)
Rotavirus	0.87 (0.08-16.0)
Sapovirus	0.17 [†]
All Viral Pathogens:	0.63 (0.08-16.00)
NO PATHOGEN*	0.15 (0.08-5.00)
MULTIPLE PATHOGENS	0.51 (0.13-4.00)

*Note: Only two cases with no pathogen isolated had significant co-morbidities at time of admission.

[†]Single case.

Rehydration Therapy

The most common treatment offered at hospital was rehydration therapy. Depending on the dehydration level of the child on admission, the therapy included oral, nasogastric or intravenous fluids, or a combination of them. In some cases, the failure

of oral therapy led to fluids being given through a nasogastric tube and, if that failed, by intravenous delivery. Of the total sample, 34 had more than one form of rehydration therapy: 18 (14.5%) were given both oral and nasogastric hydration fluids, 4 (3.1%) were given both oral and intravenous fluids and 12 (9.2%) were given nasogastric and intravenous fluids. Of these 34 cases, 30 of them had rotavirus gastroenteritis. No children in the study received all three forms of rehydration. Table 8 lists the frequency of rehydration therapies for each enteric pathogen found positive in the study.

Table 8: Frequency of Rehydration Therapy by Pathogen * (% of pathogen-specific cases)

PATHOGEN	ORAL	NASOGASTRIC	INTRAVENOUS
BACTERIA:			
<i>Campylobacter</i>	5 (41.7)	2 (16.7)	3 (25.0)
Enterohemorrhagic <i>E.coli</i>	0	0	1 (100)
<i>Salmonella</i>	2 (50.0)	0	1 (25.0)
<i>Shigella</i>	2 (66.7)	0	2 (66.7)
All Bacterial Pathogens			
VIRUSES:			
Adenovirus	3 (60.0)	0	0
Astrovirus	1 (20.0)	2 (40.0)	3 (60.0)
Norovirus	3 (42.9)	1 (14.3)	1 (14.3)
Rotavirus	47 (62.7)	34 (45.3)	17 (22.7)
Sapovirus	1 (100)	0	0
All Viral Pathogens			
NO PATHOGEN	20 (66.7)	4 (13.3)	3 (10.0)

*Note: 34 cases had more than one type of hydration therapy

There were no statistically significant differences found in a comparison of rehydration therapies received by those cases with a bacterial pathogen and those with a viral.

Cases with Multiple Pathogens

There were 10 cases (7.6%) that tested positive for more than one enteric pathogen in their faeces. They ranged in age between nine months and 3.7 years, though 80% of them were under two years of age. They included children (n=10) who were recorded with the following ethnicities: New Zealand European (3), New Zealand Maori (3), Samoan (2), Indian (1) and Fijian (1). The NZDep2001 decile ratings of these children by residential address ranged from the least deprived, decile 1 (2 cases) to the most deprived, decile 10 (3), with a median decile of 8.

All cases with dual pathogens had at least one viral pathogen, with 80% of them having rotavirus, while six of them had two viral pathogens in their faeces. One case tested positive for three different pathogens. Six cases tested positive for a bacterial as

well as viral enteric pathogen. All but one of these complex cases presented with clinical dehydration at the time of hospital admission, and were given rehydration therapy on admission. Their median length of hospitalisation was 0.51 days (range 0.13-4.0) (see Table 7).

Cases with No Pathogens Found

There were 30 cases (22.9% of the sample) for whom no pathogens were detected on faecal testing. Of these, 25 (83%) had complete faecal testing completed. Five cases had incomplete viral testing at ESR (for adenovirus, astrovirus, sapovirus and norovirus), and were not tested for protozoa (*Giardia*, *Cryptosporidium*). One case was not tested for rotavirus and one was not tested for Enterohemorrhagic *E.coli*.

All cases had diarrhoea, 19 (63.3%) had vomiting and 12 (40%) had fever (>38 degrees Celsius) on admission. The median length of time in hospital for these cases was 0.15 days (range 0.08-5). Only two of these cases had significant co-morbidity on admission which might have contributed to the decision to seek hospital care. These cases had very limited findings on microscopy of faecal specimens (10% had blood, 16.7% had white blood cells and 20% had mucus).

A Regional Picture

The hospital discharge data for the final six months of 2005 for the three Auckland DHBs became available in late March, from the New Zealand Health Information Service's NMDS, courtesy of Counties Manukau DHB. By that time, this investigation and data analysis were near-complete. The case series data were reviewed and compared to the NMDS hospital discharge data for the two sites in our series.

The frequencies of coding for parasitic and *Salmonella* gastroenteritis, as well as shigellosis, were compared to the study sample and found to be similar. There were no cases of parasitic disease reported in either dataset, and the case series included two more cases of *Shigella*, and two fewer cases of *Salmonella* than the regional dataset.

During the investigation period, July 1 to December 31, 2005, there were 470 cases admitted to Auckland DHB's Starship Hospital and 422 cases admitted to Counties Manukau DHB's Kidz First Hospital, a total of 892 cases. A further 121 cases (12% of total cases) appear in the NMDS data from Waitemata DHB facilities, most likely representing cases of paediatric gastroenteritis seen at two other public hospital emergency departments at North Shore Hospital (north Auckland) and Waitakere Hospital (west Auckland). Although these children would not have been admitted to either hospital, they were presumably in the emergency department for longer than 3 hours, and thus are included in the NMDS. These 121 cases were excluded from the rate calculations below, as they represent cases that were presumed not to have been included in the investigation. It is possible that some of them were, however, transferred to one of the paediatric hospitals.

The overall case ascertainment factor in the case series was 6.8, so that for every one case entered into this investigation, there appear to have been nearly 7 cases admitted to the two paediatric hospitals in the Auckland region. The case ascertainment factor varied by five year age-group, with younger children being more likely to have been entered into the investigation (see Table 9). The factor also varied by major ethnic group, with Pacific having a higher frequency in the case series than in the regional one, and European/Other being under-represented in the series (see Table 10).

Table 9: Comparison of Case Numbers between Regional Hospital Discharges (NMDS) and Case Series, by Age-Group

	Number of Cases by Age-Group:			Total Cases
	0-4 years	5-9 years	10-14 years	
Case Series Data	121	8	2	131
Regional Data (NMDS)	771	86	35	892
Multiplication Factor (NMDS/Case Series)	6.4	10.8	17.5	6.8

Table 10: Comparison of Case Numbers between Regional Hospital Discharges (NMDS) and Case Series, by Major Ethnic Group

	Number of Cases by Ethnic Group:				Total Cases
	Maori	Pacific	Asian	European and Other	
Case Series Data	21	47	17	46	131
Regional Data (NMDS)	131	221	134	406	892
Multiplication Factor (NMDS/Case Series)	6.2	4.7	7.9	8.8	6.8

The factor difference between the hospital discharge data (NMDS) and the case series were applied to the rates reported in earlier sections of this report, to give more realistic estimates of regional rates. As the factors varied by five-year age-group and ethnic group, the relevant multiplication factor for each type of rate was applied (see Table 11).

The regional rates demonstrate a similar pattern to the case series, except by ethnicity. The highest gastroenteritis admission rates in the region were for children aged six to eleven months, followed by those aged 1-2 years. The admission rate for males was much higher than for females. The regional ethnic-specific rates still show Pacific children having the highest rate, but the relative rate for European/Other children is much higher than in the case series (see Table 11). The rate was highest for Pacific, then European/Other, then Asian, then Maori, whereas the case series showed a relatively higher rate of admission for Maori.

Table 11: Case Series and Regional Age-Specific, Gender-Specific and Ethnic-Specific Rates

DEMOGRAPHIC VARIABLE	CASE SERIES Gastroenteritis Rates per 100,000 per year (95% CI)	REGIONAL Gastroenteritis Rates per 100,000 per year (Multiplication Factor)
AGE		
0-5 months	379 (208.6-549.4)	2,425.6 (6.4)
6-11 months	648.8 (430.7-866.9)	4,152.3 (6.4)
12-23 months	486.8 (353.2-620.4)	3115.5 (6.4)
2-4 yrs	56.8 (29.8-83.7)	363.5 (6.4)
Total 0-4 yrs	238.7 (196.2-281.3)	1,527.7 (6.4)
5-9 yrs	16.4 (5.0-27.8)	177.1 (10.8)
10-14 yrs	4.0 (0.0-9.5)	70.0 (17.5)
TOTAL	87.7 (72.7-102.8)	596.4 (6.8)
GENDER		
Female	64.8 (46.2-83.2)	440.6 (6.8)
Male	109.6 (86.1-132.9)	745.3 (6.8)
ETHNICITY (major group)		
NZ Maori	74.9 (42.9-106.9)	464.4 (6.2)
NZ European + Other	70.6 (50.2-91.1)	621.3 (8.8)
All Pacific	155.5 (111.0-200.0)	730.9 (4.7)
Asian	65.6 (34.4-96.7)	518.2 (7.9)

Primary Care Comparison

Pathogen-specific frequencies of cases in this investigation were compared to positive faecal tests performed in the community, ordered by a general practitioner through Diagnostic Medlab (DML) during the same time period (see Table 12). These frequencies represent the positive results found in children seen in primary care and who had faecal testing performed. As one would expect, the primary care frequencies for most etiologies of infectious gastroenteritis were higher than hospital ones. The only exception was *Shigella*, which had low case numbers in both settings (1 from DML and 3 from hospital sample), but was more frequent (20:1) among hospital cases. The highest ratio of primary care to hospital was for the bacterial pathogens, *Campylobacter* (6.0:1) and *Salmonella* (3.1:1). As stated previously, the calculations underestimate true case numbers by about two percent, as they do not include data from the smaller community laboratory in Auckland (SCL). Of note is the fact that there were five cases with two different pathogens on faecal testing among the primary care data.

Table 12: Frequencies of Confirmed Paediatric Gastroenteritis Pathogens in the Auckland Region: Primary Care versus Hospital Visits, 0-14 years, July-December, 2005

Pathogen	Primary Care Frequency*	Hospital Visit Frequency**	Ratio Primary Care to Hospital Visits
Aeromonas	4	0	n/a
Campylobacter	496	82	6.0 : 1
Salmonella	83	27	3.1 : 1
Shigella	1	20	1 : 20
Yersinia	30	0.0	n/a
Cryptosporidium	43	0.0	n/a
Giardia	52	0.0	n/a
Rotavirus	656	510	1.3 : 1

*Based on faecal testing results, courtesy of Diagnostic Medlab, Auckland, not including Southern Community Laboratory results, and so may underestimate actual frequencies by about 2%.

**To estimate hospital frequencies, frequencies from this investigation were multiplied by a factor of 6.8, and rounded to the nearest whole number. This multiplication accounted for the relative sample size being a factor of 6.8 smaller than the total number of gastroenteritis admissions during the period of study.

DISCUSSION

In this Auckland case series, severe gastroenteritis affected children under two years of age much more than older children, and was the primary cause of death for one young child. It affected Pacific children, especially Samoan and Tongan, significantly more than any other ethnic grouping, and affected more boys than girls. Those children living in areas ranked as relatively more deprived were more likely to be affected. Further, the dominant causes of gastroenteritis were viral, rather than bacterial or parasitic, and rotavirus was by far the leading cause of gastroenteritis leading to hospitalisation in our series. Each of these findings will be discussed separately, in light of the literature.

As discussed in the introduction, a 2003 clinical audit of 50 children admitted to Kidz First Hospital with gastroenteritis found that, due to the lack of routine faecal testing being performed, the cause of gastroenteritis remained unknown for nearly two thirds of children (Trenholme, McBride et al. 2003). The cases in this series had extensive faecal testing performed, as well as demographic and clinical data collected, and seventy-seven percent of them had an etiology ascertained. This investigation, then, contributes new information about the pattern of severe paediatric gastroenteritis in the Auckland region.

There are no published New Zealand studies on the multiple etiologies of severe acute gastroenteritis, however two studies of rotavirus gastroenteritis hospitalisations are highly relevant (Ardern-Holmes, Lennon et al. 1999; Grimwood, Huang et al. 2006). Overall there are very few comprehensive studies of the bacterial, viral and parasitic etiologies of severe acute gastroenteritis in developed countries over the past ten years. One exception is a thirteen-year Melbourne study, which has relevance to this Auckland investigation, as it involved comprehensive faecal testing for nearly five thousand children, aged zero to fourteen years, admitted to hospital with a primary diagnosis of acute gastroenteritis (Barnes, Uren et al. 1998).

Recent overseas studies have focused more on delineating the major etiologies of paediatric viral gastroenteritis (Bon, Fascia et al. 1999; Mustafa, Palombo et al. 2000; Waters, Ford-Jones et al. 2000; Simpson, Aliyu et al. 2003; Gallimore, Cubitt et al. 2004; Zintz, Bok et al. 2005). Older studies may be less relevant, given the improvement in molecular techniques for the laboratory detection and identification of viral pathogens in the recent past (Simpson, Aliyu et al. 2003; Gallimore, Cubitt et al. 2004). Further, in light of the high burden of rotavirus disease and the ongoing debate about the introduction of a rotavirus vaccine, a growing body of literature exists on rotavirus gastroenteritis, both in New Zealand (Ardern-Holmes, Lennon et al. 1999; Chen, Beasley et al. 2005; Grimwood, Huang et al. 2006) and elsewhere (Glass, Kilgore et al. 1996; Bishop, Masendycz et al. 2001; Fruhwirth, Heininger et al. 2001; Parashar, Gibson et al. 2006).

Demographic Findings

The ethnic breakdown of cases in this 2005 paediatric gastroenteritis series is consistent with earlier data. A wider report on child health in Auckland (NZ Child and Youth Epidemiology Service 2005), which collated 2000-2004 NMDS gastroenteritis

data, found Pacific children to have the highest rate of hospital admissions, NZ European and Asian to have comparable rates to each other, and Maori to have the lowest rate. In this case series Pacific children had more than double the rate of admission of any other major ethnic group; however the difference was not as dramatic when extrapolated to regional data. The specific ethnic groups with the highest disease burden were Samoan and Tongan. Caution must be exercised in interpreting these ethnic-specific rates for Pacific subgroups in this report, as the population denominators used for Samoan, Tongan and Cook Island children 0-4 yrs old were possibly lower than actual values (Ministry of Health 2005b). Thus, the rates presented in this report may be a slight over-estimate of true rates for these ethnic groups. However, the fact remains that Samoan and Tongan children have unexpectedly high frequencies of admissions for paediatric gastroenteritis. This fact is very significant, in light of the inequitable burden Pacific people face from many other diseases in New Zealand (Ajwani, Blakely et al. 2003).

The other ethnic groups which had high frequencies of severe gastroenteritis in the case series were New Zealand European (29%), New Zealand Maori (16%) and Indian (9.2%). Due to NMDS population denominators combining European and "Other", the ethnic-specific rates for NZ European and for Indian could not be calculated. The combined "NZ European/Other" groups had a total rate of 70.6 cases per 100,000 annually in the case series, only slightly lower than that for NZ Maori. Further research would need to be done, using more detailed ethnic-specific population denominators, in order to ascertain which groups within the "NZ European/Other" bear the burden of severe paediatric gastroenteritis. It is likely, judging from the case series, that Indian children are one such group.

There were significantly more boys than girls in the Auckland case series. There were, however, no significant differences in etiologies by gender, consistent with a Canadian study (Waters, Ford-Jones et al. 2000). In our case series, the frequency of hospital visits and admission for severe paediatric gastroenteritis increased with cases' increasing NZDep2001 scores, and this difference was significant. Further, the NZDep2001 decile ratings demonstrated a gradient, with a positive association between paediatric gastroenteritis admission and increasing relative neighbourhood deprivation. This finding is consistent with a previous Auckland report on child health (NZ Child and Youth Epidemiology Service 2005), but also consistent with many other reports of the associations between socioeconomic deprivation and health.

Faecal Testing

Routine faecal testing at the paediatric hospitals in this investigation is normally limited to cases believed to have either a bacterial enteric pathogen, which would be notifiable to the Medical Officer of Health, or to have rotavirus gastroenteritis. The extensive faecal testing performed in this investigation has revealed pathogens that have not been identified previously in hospitalised children in New Zealand, such as sapovirus (Greening 2006). The frequencies reported are almost certainly an underestimate, given that there was significant under-testing for viruses and protozoa. Complete bacterial testing occurred in 98 percent of cases, but complete viral testing and protozoa testing were limited to only 85 percent of cases.

Testing for protozoa was performed at Middlemore Hospital Laboratory (MMH Lab) for both sites in the case series investigation. The faecal specimens of the first

fourteen Starship cases were not sent on to MMH Lab, presumably due to an oversight. The frequency of testing was much higher at Kidz First, for which the MMH Lab was on-site. Thus, the fact that there were no *Giardia* or *Cryptosporidium* located among any of the faecal specimens in the case series is likely to be spurious. Based on notification rates, giardiasis has a high prevalence in the Auckland region, with the highest rates being among children under five years of age (Hoque, Hope et al. 2004), although a subsequent study found significant under-reporting of giardiasis among the Auckland adult population (Hoque, Hope et al. 2005). Gastroenteritis of parasitic etiologies does not often cause an acute, severe, illness and thus may lead to fewer hospitalisations than viral and bacterial types. The Melbourne study of children hospitalised with gastroenteritis reported low, but significant, frequencies of giardiasis and cryptosporidiosis of 0.3 and 0.5 percent respectively (Barnes, Uren et al. 1998).

Rotavirus testing was nearly 100 percent, probably reflecting the ease of testing as well as the level of interest in this disease, thanks to the research activity of the New Zealand Rotavirus Study Group. This group has recently conducted a multicentre prevalence study involving both Starship and Kidz First hospitals, to provide a baseline for future studies on the impact of a rotavirus vaccine (Grimwood, Huang et al. 2006). The ELISA tests used for detecting group A rotavirus at both the hospital laboratories and the community laboratory are simple to do, and are highly specific, but they are considered to be only 90 percent sensitive, and may underestimate true rates by as much as ten percent (Husain, Seth et al. 1995; Grimwood, Huang et al. 2006).

The other viruses were less frequently tested for in the case series, probably due to the fact that samples had to be sent to Porirua for testing at ESR. Once samples were received there, each one was tested for adenovirus types 40 and 41, astrovirus, norovirus and sapovirus.

Etiology

Faecal testing results revealed that nearly eighty percent of cases in the series had viral gastroenteritis, with a few having viral and bacterial combined. A previous study had estimated the proportion of viral gastroenteritis among young children hospitalised with gastroenteritis to be 41% (Ardern-Holmes, Lennon et al. 1999). Bacterial pathogens accounted for 15% of cases in this investigation and 23% had no pathogen identified, whereas in the Ardern-Holmes et al study, only 3% were due to bacterial or parasitic causes and 56% of cases remained of unknown etiology. None of the cases in the series tested positive for parasites, as discussed above. The improvement in tests available to detect viral pathogens may provide a partial explanation for the difference in proportions of viral, bacterial/parasitic and unknown causes in the two investigations.

Viral Pathogens

Cases with viral gastroenteritis were more likely to have fever, vomiting and dehydration on initial hospital assessment than those with bacterial gastroenteritis. Dehydration was a common clinical finding on admission to hospital, especially for those with viral pathogens on faecal testing, and was the cause of one death in the case series. Many different viruses are excreted in the faeces, but only a few have been shown to cause acute gastroenteritis. The caliciviruses (norovirus and sapovirus), adenoviruses types 40 and 41, astroviruses and rotavirus group A are those which

have been demonstrated to be enteric pathogens (Gallimore, Cubitt et al. 2004). The high proportion of viral pathogens responsible for paediatric gastroenteritis hospital admissions in this investigation, was consistent with a previous New Zealand study (Ardern-Holmes, Lennon et al. 1999), as well as studies from Australia (Barnes, Uren et al. 1998), and France (Bon, Fascia et al. 1999).

Caliciviruses: Norovirus and Sapovirus

The human caliciviruses are recognised causal agents of severe acute gastroenteritis outbreaks in children, as well as adults, although the full extent of their role in sporadic acute gastroenteritis among hospitalised children has not yet been well established (Dove, Cunliffe et al. 2005; Zintz, Bok et al. 2005). The two groups (“genera”) of caliciviruses are norovirus (formerly known as Norwalk-like viruses) and sapovirus (formerly known as sapporo-like viruses). They are known most for their role in outbreaks of nosocomial gastroenteritis, such as in child care centres (Matson and Szucs 2003; Akihara, Phan et al. 2005). Norovirus is argued to be the leading cause of nonbacterial outbreaks of gastroenteritis in developed countries, accounting for 93 percent of all outbreaks of nonbacterial gastroenteritis reported to the Centers for Disease Control in Atlanta (Fankhauser, Monroe et al. 2002). Sapovirus has been recently demonstrated to cause both clinical and subclinical infection in preschool children (Akihara, Phan et al. 2005).

This case series identified norovirus in 5.4% of faecal samples, a similar proportion to that reported in a recent UK study (Mustafa, Palombo et al. 2000). A Canadian paediatric gastroenteritis study had a lower frequency of norovirus and found it was more commonly found among children in the childcare centre setting than in hospitals (Waters, Ford-Jones et al. 2000). However, a more recent American study of nearly 2000 hospitalised children under five years of age found a prevalence of 7.1% norovirus in faecal specimens (Zintz, Bok et al. 2005). In contrast to norovirus, sapovirus was only present in one faecal specimen, or 0.8 percent, of the Auckland case series. As there were no other pathogens identified in that specimen it is presumed to have been the causative agent of gastroenteritis. Many of the older studies of acute paediatric gastroenteritis have not identified sapovirus in faecal specimens, as improved molecular methods for detection have only been available more recently. Recent studies in the UK and the USA have determined sapovirus to have a similar prevalence in the faecal specimens of hospitalised children to the Auckland case series, roughly one percent (Simpson, Aliyu et al. 2003; Zintz, Bok et al. 2005). Norovirus continues to be the calicivirus most frequently implicated as a cause of paediatric hospitalisations, however clearly sapovirus causes severe gastroenteritis in some Auckland children.

Adenovirus 40 and 41

Adenoviruses 40 and 41 are the two adenovirus subtypes which have been found to cause acute gastroenteritis. A large Australian study, over a twelve year period, found that these adenoviruses were only responsible for a small fraction of hospitalised cases of acute paediatric gastroenteritis, accounting for 3.1 percent, although the proportion varied on an annual basis (Grimwood, Carzino et al. 1995). Although the proportion of cases was small, the authors found that adenoviruses 40 and 41 caused a more protracted illness than that caused by rotavirus. The frequency of adenovirus types 40 and 41 in this investigation (4.3%) was comparable to that found in other overseas studies, which ranged from three to six percent (Barnes, Uren et al. 1998;

Bon, Fascia et al. 1999; Mustafa, Palombo et al. 2000; Waters, Ford-Jones et al. 2000).

Astrovirus

Understanding of the epidemiology of astrovirus-associated gastroenteritis is growing, with evidence to suggest that it may be second only to rotavirus as a leading cause of hospitalisation for paediatric gastroenteritis (Glass, Noel et al. 1996). The proportion of astrovirus in this Auckland case series was the same as that of adenovirus, 4.5 percent. This frequency was comparable to the frequency in the French study (Bon, Fascia et al. 1999) and the Canadian study (Waters, Ford-Jones et al. 2000) but much higher than the earlier Melbourne study, which the authors felt had underestimated its true prevalence (Barnes, Uren et al. 1998). A more recent Melbourne study, which looked specifically at astrovirus using more sensitive diagnostic molecular laboratory techniques, found that three percent of all young children hospitalised for gastroenteritis, and tested for enteric pathogens, had astrovirus (Mustafa, Palombo et al. 2000). The researchers found that the incidence of astrovirus was comparable to enteric adenoviruses, but exceeded *Salmonella* and *Campylobacter* in most years, and argued that their evidence proved astrovirus to be an important cause of severe gastroenteritis in Melbourne. Our case series results suggest that it is also an important pathogen in the Auckland region.

Rotavirus Group A

Group A rotaviruses, which were first isolated in 1973, are a major cause of severe childhood gastroenteritis in both the developing and developed world (Kapikian 1997). They are also the number one cause of childhood hospitalisation for acute gastroenteritis internationally (Parashar, Gibson et al. 2006) and in New Zealand (Ardern-Holmes, Lennon et al. 1999; Grimwood, Huang et al. 2006). Infection with group A rotavirus can cause severe watery diarrhoea, which can quickly lead to profound dehydration. The virus can be shed in the faeces for a number of weeks after a clinical illness. In temperate climates, peak rates of rotavirus infection tend to be in the colder months (Bishop, Masendycz et al. 2001; Grimwood, Huang et al. 2006).

There are many different rotavirus tests available internationally, including antigen enzyme immunoassay (EIA) tests, polymerase chain reaction (PCR) tests and electron microscopy (EM). Immunoassay methods, such as ELISA and Latex Agglutination are the most common methods used for laboratory detection of rotavirus (Husain, Seth et al. 1995). EM and PCR testing are expensive and require expertise to undertake, while EIA tests have been demonstrated to be useful as routine diagnostic tests for rotavirus. However, there is variation in sensitivity and specificity between EIA tests (Pang, Lee et al. 2004). In this investigation, the community laboratory used a latex agglutination EIA test, while the hospital laboratories used a type of lateral flow immunoassay, known as ICT (Pers. Comm. Dr. Susan Taylor, 23/01/06). While manufacturers report these tests as having high sensitivity and specificity (over 90% for each), there has been little research into the characteristics of different brands of EIA tests. It is possible that a small proportion of the positive rotavirus test results in both the Auckland case series and the community laboratory were falsely positive, while it is also possible that a proportion of cases of rotavirus gastroenteritis tested falsely negative on faecal specimens. Further, given that rotavirus can be excreted for nearly sixty days after the onset of diarrhea (Richardson, Grimwood et al. 1998), some of the positive tests in the children with multiple pathogens may have

represented ongoing excretion rather than acute infection. However, it is difficult to quantify this figure.

In the past ten years, two studies of childhood gastroenteritis admissions in New Zealand have been published, with a focus on rotavirus gastroenteritis. One retrospective study, published in 1999, looked at hospitalisation trends by analyzing admissions and laboratory data at four sites in Auckland, Waikato and Christchurch, for children aged 0-4 years (Ardern-Holmes et al. 1999). The two sites in this investigation, Starship and Kidz First (part of Middlemore Hospital, MMH, at that time) were included in the 1999 study. The authors used the data on the proportion of rotavirus-positive cases from one site (MMH) and extrapolated to the diarrhoeal disease admissions at the other sites. The burden of rotavirus gastroenteritis was estimated to be 351-362 cases per 100,000 people annually.

The other important rotavirus study was undertaken prospectively more recently, between 1998 and 2000, and involved a survey of children under 3 years of age admitted to one of eight study hospitals in New Zealand with acute diarrhoea (Grimwood, Huang et al. 2006). Similar to our case series, demographic and clinical data were collected on cases and faecal testing for rotavirus was performed. Inclusion and exclusion criteria were also similar to those in our case series. The results of the Grimwood et al survey were then extrapolated to the NMDS hospital discharge data for children 0-3 years of age coded as having primary gastroenteritis during the same period of time, in a comparable way to that done in this investigation. The authors estimated that the national hospitalisation rate for rotavirus gastroenteritis for children under 3 years of age in New Zealand was 634 per 100,000 children annually, when age and season were controlled for.

Rotavirus was the predominant cause of acute paediatric gastroenteritis in the Auckland series (57.3 percent of cases), as well as among the community laboratory results. Case numbers were highest for Pacific and Maori children, and particularly those children between six and eleven months old. The Grimwood et al New Zealand study found that 42.6 percent of cases tested positive for rotavirus, a lower proportion than that found in this investigation. However, the Grimwood study adjusted for the seasonal variability of rotavirus infection while this investigation did not, which could account for the difference. The frequency of fifty-seven percent found in this investigation is in keeping with much international evidence (Kapikian 1997; Barnes, Uren et al. 1998; Bon, Fascia et al. 1999; Bishop, Masendycz et al. 2001; Fruhwirth, Heininger et al. 2001), although some studies have reported lower frequencies of under thirty percent (Waters, Ford-Jones et al. 2000; Simpson, Aliyu et al. 2003).

The rate of rotavirus gastroenteritis reported in this investigation (146 cases per 100,000 per year for children 0-4 years old) is lower than those reported in the previous New Zealand studies of 351-362 cases per 100,000 per year (Ardern-Holmes, Lennon et al. 1999) and 634 cases per 100,000 per year (Grimwood, Huang et al. 2006), although the age-groups being studied were not identical. The Grimwood et al study included only those children less than three years of age. Given that rotavirus rates are highest among children under 2 years, it is not surprising that this investigation, as well as the Ardern-Holmes et al study, reported lower overall rates than the Grimwood et al study. There was evidence of an August-September (winter/spring) peak in rotavirus cases in this investigation, consistent with these

previous New Zealand studies, as well as with a large Australian rotavirus study (Bishop, Masendycz et al. 2001).

Rotavirus is the only viral enteric pathogen which is tested for in the community laboratory. The primary care frequency of positive rotavirus tests was only slightly higher than the number which tested positive in hospital (1.3:1). With the exception of *Shigella*, all bacterial and parasitic enteric pathogens had much higher frequencies in the community laboratory testing than the hospital case series for the same time period. This pattern for rotavirus gastroenteritis suggests that either rotavirus infection causes a more severe illness than other pathogens, or that primary care practitioners only test for rotavirus in a child whose health is already compromised to the point that they might require hospitalisation. A clearer interpretation is not possible from this investigation, as it was not possible to identify which cases from the community sample were also represented in the hospital case series. However, the majority of gastroenteritis is managed in the community, with only a percentage of it presenting to primary care services. Of the percentage that is seen in primary care, a smaller percentage has faecal testing done, in an attempt to identify pathogens. Of those who have testing done, only a percentage of those are positive. A pivotal British study demonstrated that for every case of group A rotavirus gastroenteritis seen in general practice in England, there are at least three more cases in the community, and of these general practice cases, only about one in eight will have positive results by routine laboratory testing of faeces (Wheeler, Sethi et al. 1999). While the Wheeler et al study was of all gastroenteritis cases, not only paediatric, the majority of rotavirus gastroenteritis occurs in childhood, so their rotavirus figures are potentially applicable to our case series findings. Applying Wheeler's pyramid to this investigation, our figures suggest that the 656 cases which were positive for rotavirus on faecal testing in the community laboratory between 1 July and 31 December, 2005 represent a probable 5,248 cases with possible rotavirus gastroenteritis who presented to general practice in the Auckland region during that period. Further, the 656 cases represent a probable 15,744 cases in the wider community during that period, most of whom (about ten thousand) probably did not seek medical attention.

The burden of paediatric gastroenteritis disease in Auckland due to rotavirus, then, is very large. Rotavirus infection is understood to be endemic throughout the developing and developed world, with most children being affected with it by five years of age, regardless of their exposures. It has been argued that the usual methods of trying to reduce the rates of enteric disease, such as improved hygiene to limit person-to-person spread, are unlikely to be effective with such a widespread disease, and the prevention of dehydration and development of a vaccine are likely to be the most effective interventions (Glass, Lew et al. 1991). Rotavirus vaccines have been developed, but failed to pass safety standards during a trial in the United States, due to a possible association between rotavirus vaccination and intussusception (Chen, Beasley et al. 2005). Further work on rotavirus group A vaccines has been completed and they are likely to be tested in New Zealand in the near future. A baseline study of the epidemiology of intussusception and its relation to hospitalisation for rotavirus infection in New Zealand has been published to provide national baseline data on intussusception for future rotavirus vaccine programmes (Chen, Beasley et al. 2005).

Bacterial and Parasitic Pathogens

In the case series, the frequency and rate of bacterial gastroenteritis increased with age. Overall the frequency of bacterial enteric pathogens was low, relative to viral pathogens, consistent with other research in developed countries, however it was 2.5 times the frequency reported in a recent Australian study (Mustafa, Palombo et al. 2000). The cases in the Auckland series had triple the frequency of infection with *Campylobacter*, compared with two different cohorts of children in Melbourne studies (Barnes, Uren et al. 1998; Mustafa, Palombo et al. 2000). *Campylobacter* has been reported elsewhere to be the highest reported notifiable disease in New Zealand, with rates of notification much higher than England, Australia and Canada (Eberhart-Phillips, Walker et al. 1997). The highest rates of bacterial gastroenteritis in this Auckland case series were among Tongan children, followed by Samoan, then Indian children. These findings suggest that foodborne illness contributes to the picture of severe paediatric gastroenteritis in Auckland, particularly for these ethnic groups.

Cases with bacterial pathogens were significantly more likely to have blood and/or white blood cells and/or mucus in their faecal specimens than those with viral pathogens. These findings support the guidelines for faecal testing developed for Kidz First Hospital, which recommend, based on research evidence, that stool testing only be done if bacterial gastroenteritis is suspected (Montgomery 2006). The absence of white blood cells or blood in stools is presumptive of nonbacterial etiologies of gastroenteritis. Put another way, blood or pus in faeces make it more likely that a case has a bacterial pathogen requiring public health notification and/or antibiotic treatment.

The frequencies of bacterial and parasitic causes of gastroenteritis were significantly higher in the community than in the hospital case series, even when the case series was extrapolated to the Auckland regional hospital dataset. In particular, there were much higher frequencies of *Campylobacter*, *Salmonella*, *Yersinia*, *Aeromonas*, *Cryptosporidium* and *Giardia* found on faecal testing in the community. (Note: The numbers of cases of *Shigella* gastroenteritis were so small in both the community and the hospital case series that the trend is difficult to interpret.) One consideration in interpreting the difference is that the community laboratory data on faecal testing for children aged 0-14 years was not selected only for cases of primary gastroenteritis, and may include children who had diarrhea for other reasons. However, as stated previously, there would have been far more children with gastroenteritis during the six-month period of interest who did not seek medical attention, or who did see a general practitioner but did not have faecal testing done. The much higher frequencies of most bacterial and all parasitic gastroenteritis in the primary care dataset suggest that these are self-limiting illnesses which do not usually become severe enough to require hospitalisation.

Multiple Enteric Pathogens

Ten cases, representing nearly eight percent of our case series, had multiple enteric pathogens on faecal testing. This figure is much higher than that of 1.2 percent reported by Barnes et al in Melbourne (Barnes, Uren et al. 1998). With the application of molecular methods of virus detection and characterization, recent studies are identifying dual and multiple excretion of enteric viruses in some children (Dove, Cunliffe et al. 2005).

Further research is needed before one could delineate how many of these cases truly represent dual and multiple infections, how many represent a degree of cross-reactivity in viral antigen tests and how many of the findings are due to the prolonged excretion of viruses, such as rotavirus, post-infection.

No Enteric Pathogen Identified

Twenty-three percent of cases (30) had no pathogen found on faecal testing in this investigation. As only twenty-five of these cases had complete testing of their specimens performed, the true proportion of cases with no pathogen on testing was nineteen percent. It is possible that a few of these cases were partially treated at the time of the hospital visit, as taking antibiotics was not an exclusion criterion unless it was obvious from the clinical records that diarrhoea was a side-effect of the medication. This reported frequency of 'no pathogen found' is lower than in other published research, although not all studies report a figure. The Barnes et al study reported that 43.5 percent had no pathogens found on testing (Barnes, Uren et al. 1998). Our figure of only 19 percent most likely reflects the improvements in the laboratory identification of viral enteric pathogens in the past decade. The high frequency of pathogens found in this study is unique.

Enteric Notifications to the Medical Officer of Health

In the case series, 85% of those twenty cases with a cause-specific notifiable type of gastroenteritis were actually notified to the regional public health unit. Nationally, notifications to the Medical Officer of Health for gastroenteritis, also known as enteric diseases, account for 85% of all notified diseases (Institute of Environmental Science and Research (ESR) 2003). Campylobacteriosis is the most frequently notified disease in New Zealand, comprising 53.2% of all notifications in 2004 (Institute of Environmental Science and Research (ESR) 2005). From July 2000, public health services have been encouraged to record all cases of gastroenteritis reported to them, including those self-reported. Viral gastroenteritis is not notifiable, unless in a high-risk case, but among those cases notified, norovirus is the most common pathogen identified (Institute of Environmental Science and Research (ESR) 2005). Despite the high rates of rotavirus gastroenteritis in Auckland, and in New Zealand as a whole, most cases are not notified to a public health unit.

Notification rates for enteric disease are considered unlikely to be truly representative of actual disease epidemiology, due to significant under-notification. In England, the ratio of community cases of infectious intestinal disease to cases notified to national surveillance were found to be 136 to one (Wheeler, Sethi et al. 1999). In Auckland, the notification rate was found to be less than eighty percent for seven potentially foodborne causes of enteric disease (Simmons, Whittaker et al. 2002). Further, there is evidence that Maori and Pacific have higher rates of disease than is represented in notification data nationally (Institute of Environmental Science and Research (ESR) 2003). It is noteworthy that Pacific children had the highest rates of hospital visits for gastroenteritis in this investigation, yet apart from typhoid, paratyphoid and listeriosis, notification rates for enteric disease in the Auckland region remain disproportionately low for both Maori and Pacific peoples (Simmons 2005; Lopez 2006).

A recent review of enteric notifications in the Wellington region demonstrated that there is a socioeconomic gradient in enteric disease notifications in the region (Lindberg, McDonald et al. 2006). Those cases living in less deprived

neighbourhoods, by NZDep2001 quintile, were more likely to be notified to the public health unit than those living in more deprived neighbourhoods. The same pattern is likely to exist in the Auckland region, given the positive association between in this investigation between paediatric gastroenteritis admission and living in a more deprived neighbourhood. Of note is the fact that the three cases in this investigation that ought to have been notified to the Auckland Regional Public Health Service but were not, were from more deprived neighbourhoods.

The Limitations of this Investigation

This investigation was undertaken in a large public health unit, with the cooperation of clinical staff in two busy paediatric emergency departments. There were a number of limitations to the study which must be considered when interpreting its findings.

The case series numbers are small, relative to the total number of children discharged from hospital with gastroenteritis during the period of investigation. There were two issues which limited the size of the case series. First, a significant proportion of eligible children were not entered into the study during the period of investigation, presumably due to emergency department clinicians having other pressing priorities. Second, there were a significant number of faecal specimens for which only partial testing was completed due to either the inadequacy of specimens or an oversight of laboratory staff. As a result, bacterial pathogen rates may underestimate the true rate by as much as two percent, protozoan rates by fifteen percent and rates of viral pathogens, other than rotavirus, by as much as fifteen percent.

The estimated rate of gastroenteritis due to rotavirus group A in this investigation is probably an overestimate of true annual frequencies and rates, given that the study period included an epidemic during the 'rotavirus season' of winter/spring. One would need to control for the seasonality of the disease through a standardisation process, using a method such as the one used in the Grimwood et al study (Grimwood, Huang et al. 2006).

The lack of availability of regional population figures by smaller ethnic groups, such as Indian or Southeast Asian, for every group other than Pacific children, prevented the calculation of true ethnic-specific rates for these other groups. Any subsequent investigator interested in ethnic-specific rates of paediatric gastroenteritis would be wise to ensure that appropriate population denominators were available.

The NMDS hospital discharge data is likely to overestimate cases of primary gastroenteritis, as the data includes those cases with hospital-acquired gastroenteritis and those cases for which gastroenteritis was not the primary diagnosis. As a result, the case series may have represented more than fifteen percent of total cases during the study period.

The data collected on levels of clinical dehydration on arrival at hospital were not considered accurate, as such information was not consistently documented in emergency department clinical notes. As a result, the data on whether dehydration was mild, moderate or severe were excluded from the analysis, and the only data used was whether dehydration existed or not. Clinicians interested in assessing the impact of dehydration among children admitted to hospital with acute gastroenteritis would

need to ensure that the assessment of dehydration was clinically consistent as well as clearly documented.

There was probable duplication of data between the community laboratory dataset and the case series dataset. For privacy reasons, Diagnostic Medlab was not able to provide a unique identifier (NHI number) for each case, which prevented the matching of hospital and primary care cases. A more accurate picture of those children diagnosed and treated in the community would be obtained by excluding duplicate cases by NHI number.

It is difficult to directly compare the rates of paediatric gastroenteritis admissions in this investigation to previous New Zealand studies, due to the small numbers in the case series, as well as the different case definitions used in the other studies. The previous New Zealand studies (Ardern-Holmes, Lennon et al. 1999; Grimwood, Huang et al. 2006), include data on formal admissions, consistent with the definition used by the NZHIS, those children at hospital for longer than three hours. They exclude those children assessed and treated at emergency departments and then released home. Further, the NMDS hospital discharge data may overestimate primary gastroenteritis admission rates, as the data do not distinguish community-acquired from hospital-acquired (nosocomial) disease, and may not distinguish gastroenteritis from diarrhoea due to other causes.

Cases were included in this case series investigation even if they were at hospital less than 3 hours. The decision to include these cases was based on a desire to gain a fuller picture of the disease burden, recognising that there are a number of factors that determine whether or not a child is admitted to hospital, after being assessed in an emergency department. The severity of the presenting illness is an obvious factor, however the social and economic situation of the child's caregivers is considered too, as well as the presence of co-morbidities. Those children who responded promptly to rehydration therapy, for example, were often discharged from the emergency department. Given the limitations of the comparison between this investigation and others, it is noteworthy that the overall regional annual rate calculated in this investigation was 596 cases per 100,000 population, which is comparable to the reported rate of gastroenteritis for children (0-14 years) for the Auckland region in 2004 of 640 (NZ Child and Youth Epidemiology Service 2005).

CONCLUSIONS and RECOMMENDATIONS

Conclusions

The findings of this Auckland investigation are consistent with larger studies overseas in identifying the etiologies of severe, acute paediatric gastroenteritis. What this investigation adds is that it paints a picture of a disease which has an inequitable distribution and burden. This report highlights the severe impact of a common childhood illness, for which hospitalisation is potentially preventable, and for which the one death during this investigation is far too many. The community laboratory results demonstrate a large burden of disease in the community due to both campylobacteriosis and rotavirus gastroenteritis. Based on our case series from Auckland's two paediatric hospitals, the face of severe paediatric gastroenteritis in Auckland is a Samoan or Tongan male infant living in neighbourhood rated decile 8 on the NZDep2001 index, admitted to an Auckland paediatric hospital with rotavirus gastroenteritis for a period of approximately one day (21 hours).

Children under five years of age bear the major burden of disease, with dehydration being the common reason for hospital visit or admission. It is evident from this investigation that severe dehydration from acute gastroenteritis is not only an issue facing developing nations. Young children are some of the most vulnerable members of the population, relying primarily on adults to protect them from harm. Children under five years of age currently represent about seven percent of the New Zealand population (Statistics New Zealand 2006). The personal cost to the children of severe gastroenteritis may be felt in terms of setbacks in learning and development. Further, the social and economic costs to the families of those children must be significant. Once again, based on the deprivation index scores, those families living in areas of highest relative deprivation are the hardest hit.

These findings raise the question of strategies for the prevention of severe acute paediatric gastroenteritis, particularly for those groups most at risk. It is significant that the children most at risk from acute gastroenteritis appear to be Pacific, and specifically Samoan and Tongan, children. Seventy percent of New Zealand's Pacific population lives in the Auckland region, half of whom reside in the Counties Manukau DHB catchment area (Pers. Comm. Dean Papa, CMDHB). The many ethnic groups making up the Pacific population are already recognised as bearing an inequitable proportion of the burden of disease from such diseases as diabetes and some types of cancer.

A public health approach to infectious paediatric gastroenteritis would consider primordial, primary and secondary prevention strategies. Primordial prevention of gastroenteritis, or reducing children's exposure to enteric pathogens, would be the most efficient intervention, yet it is often the most difficult. Relative socioeconomic position is clearly a risk factor, and may relate to such issues as the number of people living in a household and access to hand washing and drying facilities, but this risk factor is difficult to address directly. However, primary prevention by limiting exposure to enteric pathogens might be achieved in a number of ways, as follows:

- Vigilance about nappy cleaning areas and toilet cleaning standards in childcare centres and kindergartens
- Improved hand hygiene in homes, and in places where public gatherings occur
- Maintenance of adequate food standards in kitchens, with regard to maintaining foods at appropriate temperatures during storage, cooking to recommended temperatures, and avoiding cross-contamination
- Exclusion of children and staff with acute gastroenteritis from childcare centres
- Exclusion of infected food handlers from food premises, but also from social gatherings, such as hui or fono.

Messages on food safety have been translated into culturally appropriate forms for some groups, such as the Umu Pasifika resource (Auckland Regional Public Health Service 2005) for Pacific peoples, although it is still too early to tell whether such resources have led to changed behaviours. The need for greater emphasis on hand hygiene remains.

Another key approach to primary prevention would be vaccination, in particular for group A rotavirus, which causes the majority of cases of severe paediatric gastroenteritis in New Zealand and worldwide. Once again, a vaccination programme in the Auckland region would need to prioritise Pacific children, in particular Samoan and Tongan children, with an emphasis on vaccinating children at the youngest feasible age. A rotavirus vaccination programme should involve the appropriate Pacific communities from the planning stages, right through the implementation and evaluation processes, as has been done in the Meningococcal B vaccination programme in New Zealand. Further, the vaccine could then be rolled out to the other groups whose children have inequitable rates of gastroenteritis, such as New Zealand Maori and Indian children.

Primordial and primary prevention are both key steps to reduce paediatric gastroenteritis hospitalisations, however both caregivers and primary health care services have an important role to play in secondary prevention. Since the morbidity from gastroenteritis is primarily due to dehydration, early intervention and aggressive rehydration treatment in the community could seriously reduce the number of hospitalisations. Parents and other caregivers need to develop skills in replacing fluids in children with gastroenteritis at home. Primary care teams of general practitioners (GPs) and nurses play an important role in detecting children at risk from severe gastroenteritis, either due to their overall health status or to the socioeconomic circumstances in which they live. For some families, access to primary health care services may still be an issue. The findings of this investigation suggest that caregivers, GPs and nurses can afford to be more aggressive in their management of early dehydration.

Recommendations

Culturally appropriate messages about the potentially serious effects of gastroenteritis on young children should be developed for use in a number of settings. The key prevention messages will need to be available in several languages, and developed for the target audience of caregivers of young children.

The key messages should include the following:

- What gastroenteritis is and what causes it
- How to effectively wash and dry hands, and the importance of this measure in preventing the spread of gastroenteritis bugs
- How to handle food safely, to prevent foodborne gastroenteritis
- The importance of children and adults with diarrhoea and/or vomiting staying home, rather than attending childcare centres, kindergartens or hui/fono
- How to push oral fluids in children with gastroenteritis, to prevent dehydration
- How to recognise signs of dehydration in children, especially infants, and the importance of taking children to the GP promptly for assessment and early treatment. (“The younger the child, the faster he/she gets really sick.”)


An awareness campaign could be developed, similar to that used in the meningococcal awareness campaign in south Auckland. The messengers should be individuals known and respected, and might include church ministers, kaumatua, Plunket nurses, health workers, GPs and practice nurses. The key messages could be delivered in a number of settings already familiar to caregivers of young children, such as childcare centres, churches, marae and plunket clinics. Key messages could also be run in advertisements on local radio and television stations, and posters or pamphlets could be available at primary care clinics, but also in settings already mentioned.

Primary health organisations (PHOs) have a key role to play in both the primary and secondary prevention of severe gastroenteritis. For Pacific children, an important first step would be to engage with the Auckland PHOs that are primarily serving Pacific populations, such as AuckPAC Health Trust, the Tongan Health Society Incorporated, and TaPasefika Health Trust, to ensure that this issue is placed high on both their health promotion and disease management agendas. Further, given that European and Maori children are also frequently affected by severe gastroenteritis, primary care clinicians generally should be encouraged to play a proactive role in the prevention and management of this illness.

This investigation was the result of collaboration between concerned hospital clinicians, public health professionals, DHB and Ministry of Health policy makers and funders, and members of the New Zealand Food Safety Authority. It is the hope of the authors that this report will inform an ongoing discussion between health workers and those communities whose children are most affected by severe gastroenteritis, such as some Pacific communities, with the aim of finding effective ways to reduce the burden of this common childhood illness. Primary health organisations have an important role to play in working with those communities most affected. The story told by this case series must not simply contribute to the very large body of statistics on health inequalities in Aotearoa New Zealand.

Appendix One: Laboratory Protocols

a. Middlemore Hospital Laboratory, Kidz First Hospital

 A Community Partnership	DOCUMENT NO: 02/221/m244	AUTHOR: Brian Holliss	PAGE 1 OF 1 COPY NO OF 2
LABORATORY SERVICES	SUPERCEDES NO.	AUTHORISED BY:	ISSUED: 5/12/2005
TITLE: APCI Study			ISSUE NO: 1

Paediatric Gastroenteritis Survey

Length of Trial: 1st June till 31st August 2005. Approximately 100 specimens.

Patients: Under 15 years of age

Request Form: A special request form is provided to identify patients in the study and provide registration information.

Tests Performed: In the order of importance if there is insufficient sample for all tests.

1 Bacterial Pathogens

- EHEC (by culture and EIA)
- Salmonella
- Shigella
- Campylobacter
- Yersinia (any species)
- Vibrio
- Aeromonas
- Listeria (by Listeria Broth)

2 Norovirus

- sent to ESR by Esme weekly. Store in fridge (4°C). **Do not freeze.**
- keep some specimen (if sufficient) in the fridge (4°C) for us to test here.

3 Rotavirus/Adenovirus

- by ICT

4 Giardia/Cryptosporidia

- screen by ICT
- confirm any positives by immunofluorescence.

-Auckland hospital will be sending us there samples for us to test also.

Stock any pathogens isolated.

Keep all forms separate after signout.

Person organising study is Greg Simmons if there are any questions that Susan or Brian can't answer.

Processing Faeces Specimens

1	Culture	
	Wet Film	
	XLD Plate	*
	Hektoen Plate	*
	Aeromonas Plate	**
	TCBS plate	**
	Campylobacter Plate	***
	Selenite Broth	*
	CIN Plate	****
	CT-SMAC Plate	*
	Listeria Broth	*****
	Selenite Broth	*
	MacConkey Broth	*

*	=	Incubate at 37°C for 18-24 hours
**	=	Incubate at 37°C for 48 hours
***	=	Incubate at 42°C for 48 hours in a microaerophilic atmosphere
****	=	Incubate at 27°C for 24 hours
*****	=	Incubate at 27°C for 48 hours

- 2 Listeria
After 48 hours subculture the Listeria broth onto CNA

Incubate CNA plate at 37°C for 18-24 hours.
(Refer to doc 221m084)

- 5 Test for EHEC toxin:
The MacConkey broth is tested EIA
(Refer to doc 221m146)

- 4 For examination for Giardia and/or Cryptosporidia
Iodine Wet Film
(Refer to doc 221m105)
Giardia/ Cryptosporidia ICT
(Refer to doc 221m230)

Fluorescent Antibody to confirm positives
(Refer to doc 221m002)

- 5 Culture Examination
 - For Aeromonas *(Refer to doc 221m004)*
 - For Campylobacter *(Refer to doc 221m005)*
 - For E. coli 0157 *(Refer to doc 221m239)*
 - For Salmonella *(Refer to doc 221m030)*
 - For Shigella *(Refer to doc 221m029)*
 - For Vibrio *(Refer to doc 221m035)*
 - For Yersinia *(Refer to doc 221m036)*

- 6 Rotavirus and Adenovirus 40/41
(Refer to doc 221m234)

b. LabPlus Laboratory, Starship Hospital

10.0 PAEDIATRIC GASTROENTERITIS INVESTIGATION

It has been identified that children have a higher rate of admission to hospital for gastroenteritis. While these cases are generally presumed to be infectious in origin, the cause in 62% of cases is not proven.

This study is being carried out to obtain greater information regarding the causes of gastroenteritis in young children.

Once this information has been obtained regarding specific pathogens, policies to control the spread of infection can be better targeted.

Contacts:

Dr Greg Simmons
Lead Investigator
Auckland Regional Public Health Service
Mobile: 021 884 657

Dr Ralph Pinnock
Consultant Paediatrician
StarShip Childrens Hospital
Mobile: 021 740 271

Specimens:

Faecal specimens will be collected from children under 15 years of age who have an acute onset of fever and diarrhoea, with or without vomiting, of less than 7 days duration for which no cause has been found.

It is expected that there will be 1 to 2 cases per day. The Investigation will aim to obtain specimens from 100 patients.

Specimens will arrive at the laboratory in two specimen containers with a Trial Request Form. If insufficient sample is collected initially, a supplementary sample may be sent at a later stage. This will be labelled Specimen 2. This specimen will be used to perform the tests that were not performed on the first specimen. Each test is to be performed only once.

Specimens may arrive at the laboratory 7 days per week, and at any time of the day.

Registration:

Hospital Number:	Patients NHI Number as per Request Form
Patient Details:	Patient Name Sex & Date of Birth as per Request Form
Care Of:	SIMMT
Location:	NL
Debtor:	Leave Blank
Report Destination:	SIMMT
Copy To:	Patient Doctor (Dr. Code as per Request Form)
Test Code:	FC

Specimen Handling:

Specimens will require the following analyses: Yersinia, Campylobacter, Shigella, Salmonella, Vibrio, Aeromonas, Listeria, EHEC, Giardia, Cryptosporidium, Rotavirus, Adenovirus 40/41 and Norovirus.

Culture for bacterial pathogens and the test for Rotavirus will be performed at LabPlus Microbiology, Giardia and Cryptosporidium will be performed at Middlemore Microbiology Laboratory and Norovirus and Adenovirus will be performed at ESR, Wellington.

If there is insufficient specimen to perform all these analyses, testing should be performed following this priority order:

- 1) Bacterial Pathogens (**macadamia nut size required**) (if there is insufficient specimen to inoculate the complete set of culture media, then do not perform **any** culture.)
- 2) Norovirus & Adenovirus (**teaspoonful amount required**)
- 3) Rotavirus (**pea size required**)
- 4) Giardia & Cryptosporidium (**almond size required**)

Tick the box on the request form in the column “Sufficient Specimen” for those tests that there is sufficient specimen for.

If a form is received that has the “Specimen Two” box ticked, determine which tests were performed on “Specimen One” (by checking on the “enquiry” screen and also the Sendaway log – or by checking on the original Specimen One request form) and then perform those tests for which there was insufficient specimen to perform from “Specimen One”.

Specimen Processing for Bacterial Pathogens:

- 1) Make 3 photocopies of the numbered request form
- 2) Place one photocopy of the request form in the sleeve labelled “Paediatric Gastroenteritis Investigation Request Forms”.
- 3) Determine which tests will be performed according to the priority list above and the amount of specimen received.
- 4) Attach a green Paediatric Gastroenteritis Investigations worksheet to the request form
- 5) Describe appearance of the faeces specimen as per routine laboratory procedure. **DO NOT** discard the specimen if it is “formed”, process for culture for all pathogens regardless of the appearance or presence/absence of red cells.
- 6) Examine a wet film, and comment on the presence of White cells, Red cells and Mucus, following routine laboratory procedure.
- 7) Inoculate each faeces specimen onto: XLD, HEK, Campylobacter agar, Yersinia agar, Aeromonas Agar, TCBS and MacSorb agar.
- 8) Also inoculate into Selenite F Broth, APW and Listeria Enrichment Broth (0.2g [pea-size] faeces into 20 ml LEB). LEB vials are held in the cupboard in Primary Processing.

IMPORTANT: Don't forget to inoculate the Listeria Enrichment Broth.

- 9) Incubate Listeria Enrichment Broth at 30°C for 2 days. Subculture onto CNA. Incubate CNA for 24 hours at 35°C
- 10) Incubate all other plates and broths as per routine laboratory procedures.

Culture Interpretation:

- 1) Examine XLD, HEK, Campylobacter agar, Yersinia agar, Aeromonas Agar, TCBS and MacSorb agar following routine laboratory protocols.
- 2) Identify any potential pathogens following routine laboratory procedures.
- 3) Prepare a slant for ESR from the MacSorb plate. This is to be sent to ESR even if another pathogen is isolated from the specimen.
- 4) Examine the CNA plate for any small β -haemolytic colonies. Perform a Gram stain (GPB), catalase (positive) and API Coryne.
- 5) Stock all pathogen isolates in the Sterile Sites Box at -80°C , as per usual procedures, and also in the Paediatric Gastroenteritis Investigations Box in the Sanyo -80 freezer (Shelf: 4, Rack: L, Column: 4). Use the same stock number for both vials. It is only necessary to record the Sterile Sites stock in the stock culture program.
- 6) Prepare slants for ESR from any isolates of Salmonella, Shigella or Listeria, following routine procedures.
- 7) Perform susceptibility testing on any pathogens isolated, following routine laboratory protocols.
- 8) Report any pathogens isolated and list all the negative cultures – some of this may need to be “free-texted”
eg [(CJEJ)4, “No *Salmonella* or *Shigella* isolated”, “No *Yersinia* isolated.”
&NVIB, &NAER, &NOEC, &VT &NOLI]
or report using the following codes if no pathogens are isolated: &NP, &NVIB, &NAER, &NOEC&VT and &NOLI
- 9) After Signout, place the completed form in the sleeve labelled “Completed Forms. Paediatric Gastroenteritis Investigation.”

Specimen Processing for Norovirus & Adenovirus:

- 1) Analysis for Norovirus and Adenovirus will be performed at ESR Wellington.
- 2) Aliquot a minimum of a “teaspoon” of the faeces specimen to a separate, labelled container.
- 3) Place in the Dirty Coolroom, in the box labelled “Paediatric Gastroenteritis Investigation. Specimens for ESR”, along with a copy of the request form.
- 4) Record the Name, Lab number & date placed at 4°C on the “Paediatric Gastroenteritis Investigations Sendaway Log”
- 5) These will be sent to ESR each Tuesday morning.
- 6) Record the date sent to ESR on the “Paediatric Gastroenteritis Investigations Sendaway Log”.
- 7) They can be sent to ESR at ambient temperature.
- 8) The contact person at ESR is Joanne Hewitt ph: (04) 9140690

Specimen Processing for Rotavirus

- 1) Perform Rotavirus testing using the SAS Rota Test
- 2) Follow routine laboratory procedure for testing and reporting

Specimen Processing for Giardia & Cryptosporidium:

- 1) These tests will be performed at Microbiology, Middlemore Hospital
- 2) Aliquot a minimum of 0.5g (**almond size**) of the faeces specimen to a separate, labelled container.
- 3) Place in the faeces fridge and hold along with a copy of the request form
- 4) Record the Name, Lab number & date placed at 4°C on the “Paediatric Gastroenteritis Investigations Sendaway Log
- 5) Specimens will be sent to Middlemore Hospital each morning on Monday to Friday via the “parcels room”
- 6) Specimens that arrive over the weekend may be held at 4°C and sent on Monday morning.
- 7) Each morning package each specimen and form in a Biohazard bag and label each bag with a pre-printed label “To Microbiology Lab, Middlemore. Paediatric Gastroenteritis Investigation. Attention: Brian & David”
- 8) Record the date sent to Middlemore on the “Paediatric Gastroenteritis Investigations Sendaway Log”.
- 9) The package must be in the Parcels Room by 9:15 each morning to catch the delivery run to Middlemore.

Reference: Auckland Paediatric Gastroenteritis Investigation Protocol

Appendix Two: Data Collection Sheet

Paediatric Gastroenteritis Case Series: DATA COLLECTION SHEET (24.11.05)

DEMOGRAPHY:

Initials NHI DOB

Age (years) Gender male (male=yes)

Address

Ethnicity:

Maori y/n

Pacific y/n

 Pacific: Samoan y/n

 Tongan y/n

 Tokelauan y/n

 Niuean y/n

 Other

European y/n African y/n Asian y/n

CLINICAL:

Ill days prior to admission

Days in hospital (discharge date-admission date)

Symptoms:

Diarrhoea (3 or more loose stools in 24 hrs) y/n (Don't exclude No's from the study)

Abdo pain y/n (not collected on KidzFirst children—as David to get it!)

Vomiting y/n

Fever (≥ 38 deg C) y/n

Dehydration: Mild Moderate Severe

Dry mucus membranes y/n

Sunken eyes y/n Reduced skin turgor y/n Lethargy y/n Tachycardia y/n

Poor capillary refill y/n

TREATMENT:

Oral rehydration fluid y/n Nasogastric rehydration y/n IV fluids y/n

Intraosseous fluids y/n

Electrolytes done y/n Na⁺ level (mmol)

Disposition (>3hrs in hosp, case def'n):

Treated at ER only y/n

Short stay unit y/n

Admitted to hosp y/n

LABORATORY:

Two samples y/n

Sample 1: Lab number _____

White cells y/n

If yes, small y/n

Moderate y/n

Large y/n

Red cells y/n

If yes, small y/n

Moderate y/n

Large

Mucus y/n

Culture:

Salmonella y/n

Shigella y/n

Campylobacter y/n

Yersinia y/n

Aeromonas

Vibrio

Listeria

EHEC

Giardia

Cryptosporidium

VIRAL: Rotavirus

Adenovirus 40/41

Norovirus

Astrovirus

Sapovirus

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