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Gastroenteritis in Auckland: an aetiological and clinical study

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Summary

Faecal specimens from 60 patients (under six years old), most of whom were Maoris and Pacific Islanders admitted to Auckland Hospital with gastroenteritis during the months of June and July 1977, were examined for the presence of faecal viruses, bacterial pathogens and parasites. Faecal specimens from 18 non-diarrhoeal control patients were also examined, of which three contained rotavirus. Forty-three (72 per cent) gastroenteritis patients had rotavirus detectable in stools by electron microscopy or immune electron microscopy. Of the remainder, 17 patients were regarded as having non-rotavirus diarrhoea. Enterotoxigenic Esch. coli. was isolated from seven patients of whom six yielded stable toxin producers (ST+), four labile toxin producers (LT+) and two dual toxigenic strains (ST+/LT+). All ST+ isolates appeared to be of low enterotoxigenicity as indicated by low gut weight/carcass weight ratios in the infant mouse assay.

Rotavirus was the commonest aetiologic agent (72 per cent), bacterial pathogens (alone) accounted for only five per cent and no enteric pathogens were found in 15 per cent of cases. Non-agglutinable rotavirus, presumably a different serotype, was seen in both gastroenteritis and control patients. Rotavirus 'satellite' particles previously undescribed were demonstrated in a number of stool samples.

Introduction

There have been no studies of the relative incidence of agents of gastroenteritis in New Zealand. Prior to this survey, in the majority of cases admitted to Auckland Hospital, no pathogen has been found using traditional bacteriological and virological techniques. This study was designed to give some indication of the relative importance of aetiologic agents and clinical features in cases of gastroenteritis requiring hospital admission.

Rotavirus has been shown to be a major cause of gastroenteritis in other parts of the world (Bishop, Davidson, Holmes and Ruck, 1974; Flewett, Bryden, Davies, Woodc, Bridger and Derrick, 1974; Middleton, Szymanski, Abbott, Bortolussi and Hamilton, 1974; Kapikian, Kim, Wyatt, Rodriguez, Ross, Cline, Parrott and Chanock, 1974; Echeverria, Blacklow and Smith, 1975; Ryder, Wachsmuth, Buxton, Evans, Du Pont, Mason and Barrett, 1976; Lancet 1975; WHO 1975/76; British Medical Journal, 1977)—but with the advance of knowledge in this area and the finding of the virus in large

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numbers of non-diarrhoeal neonates (Totterdell, Chrystie and Banatvala, 1976; Murphy, Albrey and Crewe, 1977), uncertainty of its relationship with its host is evolving. Like rotavirus, enterotoxigenic Esch. coli has been implicated as a cause of gastroenteritis in young children in various parts of the world (Gorbach and Khurana, 1972; Nalin, McLaughlin, Rahaman, Yunus and Curlin, 1975; Sack, Hirschhorn, Brownlee, Cash, Woodward and Sack, 1975; Ryder, Wachsmuth, Buxton, Evans, Du Pont, Mason and Barrett, 1976; Evans, Olarte, Du Pont, Evans, Galindo, Portnoy and Conklin, 1977; Echeverria, Ho, Blacklow, Quinnan, Portnoy, Olson, Conklin, Du Pont and Cross, 1977). The significance of rotavirus and enterotoxigenic Esch. coli as causal agents of gastroenteritis was studied.

In June and July, 1977, patients admitted to Auckland Hospital with gastroenteritis were studied to determine the relative isolation rates of (1) rotavirus, (2) other viruses identifiable by electronmicroscopy of stools, (3) enterotoxigenic Esch. coli producing heat stable (ST) and /or heat labile (LT) enterotoxins, (4) salmonella species, (5) shigella species and (6) parasites as aetiological agents. In addition, the presence of red blood cells, leucocytes and mucus strands in stools was recorded.

**Patients and methods**

**Patients**

Sixty patients (30 males and 30 females) admitted to the infectious disease unit of Auckland Hospital with gastroenteritis were studied. Gastroenteritis was defined as acute development of unusually frequent and loose stools with or without vomiting. The first stool passed after admission was collected in a sterile plastic container and processed on the day of collection or the day following overnight storage at 4°C. Eighteen control patients (11 males and seven females) with non-diarrhoeal diseases were studied. Clinical features were obtained on review of the patients’ notes.

**Electronmicroscopy of stool specimens**

Viral pellets of stool specimens from patients with diarrhoea and from control patients were prepared for electronmicroscopy on a Philips EM 300 by the method of Totterdell, Chrystie and Banatvala (1976). The pellet was resuspended in a few drops of distilled water and stored at −20°C until examined. Specimens were negatively stained with three per cent potassium phosphotungstate (pH6). At least five suitable grid squares were scanned at approximately 41,000 magnification. Electronmicrographs were prepared for measurements of virus size and identification of morphologically distinct viruses.

**Immune electronmicroscopy**

All faecal viral pellets were examined after reacting with specific anti-rotavirus guinea pig serum kindly supplied by Dr M. D. Holdaway, Dunedin Hospital. Using Kayline 96 U-welled microtitre plates, 25 µl anti-rotavirus
serum (complement fixation titre 1:512) was diluted in phosphate buffered salines pH 7.01 to 1:64 and incubated with 25 μl faecal pellet at 37°C for three hours. After negatively staining and coding, grids were stored in LKB 4828B specimen grid boxes at room temperature until examined 'blind'.

**Stool light microscopy**
A wet preparation of stool stained with one per cent Loeffler's methylene blue (Harris and Coleman, 1963; Harris, Du Pont and Hornick, 1972) was examined for the presence of red blood cells, leucocytes, mucus strands and parasites.

**Bacteriology**
Stool specimens were examined for the presence of *Salmonella* sp. and *Shigella* sp. using MacConkey and XLD agar plates and selenite broth. These species were identified by standard methods (Edwards and Ewing, 1972).

**Enterotoxigenic isolates**
Ten lactose-fermenting colonies with the typical appearance of *Esch. coli* were discriminately selected from the last two streaks on MacConkey plates and were stored on nutrient (Columbia) agar slopes and inoculated into 3 ml Syncase medium (glucose substituted for sucrose) to make a pool suspensions of the 10 colonies for enterotoxin screening. Pure and predominant non-lactose fermenting growths were treated in a similar manner. Each pool was incubated stationary at 37°C for 48 hours, a 1 ml aliquot of broth suspension was removed and stored at -20°C for LT assay screening. The remainder of the suspension was centrifuged at 6000 rev/min for 30 minutes and the supernatant withdrawn for ST assay screening. All specimens were coded and tested 'blind'.

**Suckling mouse assay**
The assay for ST was carried out as described by Dean, Ching, Williams and Harden (1972). Pools of 10 isolates from each patient were tested for ST production. Each individual isolate from ST positive pools was then tested. Four mice were used per assay. A mean ratio of > 0.0700 was regarded as weakly positive, > 0.0800 was regarded as positive. Positive and negative control cultures were included in each assay. ST+/LT+ strains B7a (serotype 0148 K? H28), H10407 (serotype 078 H11) and ST-/LT- U5/41 (serotype 01K1H7) were kindly supplied by Dr B. Rowe, Salmonella and Shigella Reference Laboratory, Central Public Health Laboratory, Colindale Avenue, London NW9 5HT and ST+/LT+ strain 408-3 and ST-/LT- strain 408-4 were kindly supplied by Dr R. B. Sack, Baltimore City Hospital, 4940 Eastern Avenue, Baltimore, MD 21224.

**Y1-mouse adrenal cell assay**
A miniculture assay was carried out using whole bacterial pool culture as
described by Sack and Sack (1975). Individual strains of LT+ pools were tested later.

All enterotoxigenic isolates were identified by standard biochemical tests.

**Results**

**Clinical features**

The male:female ratio in diarrhoeic patients with stools positive for rotavirus was 0.75 and for non-rotavirus gastroenteritis was 2.4. For the purpose of comparing clinical features, gastroenteritis patients were placed into two diagnostic categories according to the presence or absence of rotavirus in their stools: (1) rotavirus gastroenteritis group and (2) non-rotavirus gastroenteritis group. Further subdivisions into age groups provided comparisons.

Table I shows the main clinical features associated with rotavirus diarrhoea

| Table I Percentage positive clinical and other features by age |
| --- |
| Age group | Clinical group | <6M R | NR | 6M – <2Y R | NR | 2Y – 6Y R | NR |
| Clinical features |
| **Respiratory** |
| Pharyngitis | 31 | 14 | 35 | 71 | 70 | 0 |
| Coryza | 69 | 29 | 40 | 43 | 20 | 100 |
| Cough | 38 | 57 | 35 | 86 | 20 | 33 |
| Tachypnoea | 23 | 14 | 10 | 43 | 10 | 0 |
| Otitis media | 15 | 0 | 20 | 0 | 10 | 0 |
| Any of above respiratory symptoms or signs | 77 | 71 | 80 | 100 | 80 | 100 |
| **Other** |
| Vomiting | 69 | 86 | 80 | 100 | 100 | 67 |
| Abdominal pain | 8 | 14 | 0 | 0 | 0 | 67 |
| Shock | 15 | 0 | 15 | 0 | 10 | 0 |
| Convulsion | 0 | 0 | 5 | 14 | 10 | 33 |
| Fever | 100 | 71 | 95 | 100 | 100 | 100 |
| **Antibiotics** |
| Pre-admission | 31 | 43 | 20 | 57 | 20 | 0 |
| Post-admission | 46 | 57 | 20 | 57 | 10 | 33 |
| **Dehydration** |
| Clinical | 54 | 43 | 70 | 71 | 90 | 33 |
| > five percent by weight | 23 | 14 | 15 | 29 | 50 | 0 |
| Requiring IV therapy | 85 | 71 | 65 | 100 | 100 | 33 |
| **Ethnic groups** |
| Caucasians | 15 | 57 | 45 | 14 | 80 | 67 |
| Maoris | 46 | 29 | 30 | 14 | 10 | 0 |
| Pacific Islanders | 44 | 14 | 25 | 57 | 10 | 33 |
| Asians | 8 | 0 | 0 | 14 | 0 | 0 |
| Maoris and Pacific Islanders | 78 | 43 | 55 | 71 | 20 | 33 |
| No. in group | 13 | 7 | 20 | 7 | 10 | 3 |

R = Rotavirus gastroenteritis.
NR = Non-rotavirus gastroenteritis.
Gastroenteritis in Auckland

Fig. 1  Distribution of enteric pathogens according to age (Auckland June–July 1977). SHF6 = shigella flexneri 6; SHF2 = shigella flexneri 2; STM = salmonella enteritidis ser typhimurium; NARV = non-agglutinable rotavirus (on IEM); ST, LT, ST/LT = stable, labile and dually enterotoxigenic E. coli respectively. Merged ovals indicate toxigenic strains are from one patient.

and gastroenteritis due to other causes. A high incidence of upper respiratory symptoms and/or signs was seen in both gastroenteritis groups (71–100 per cent). Otitis media was exclusive to rotavirus gastroenteritis.

Diarrhoeic patients (in all age groups) with rotavirus in their stools had a propensity to present with marked dehydration and circulatory shock which was not encountered in other forms of diarrhoea.

On the whole, rotavirus gastroenteritis patients required intravenous fluids more often than the non-rotavirus group, but the numbers of patients in each group are small. Control patients had mainly respiratory infections and other non-diarrhoeal conditions. The relative distribution of enteric pathogens detected according to patient age is illustrated in Fig. 1.

Rotavirus was seen in non-diarrhoeal control stools in all age groups. Three of 18 (17 per cent) control stools contained rotavirus (one of which had non-agglutinable virus detectable on IEM). There was no evidence of gastroenteritis in these three patients, however, a three month old female infant had had transient loose bowel motions one week prior to admission to hospital with whooping cough syndrome.

Antibiotic treatment

The proportion of patients receiving antibiotics prior to and during admission to hospital is shown in Table I.
Table II Complications

| Complication                    | Rotavirus gastroenteritis | Non-rotavirus gastroenteritis |
|---------------------------------|---------------------------|------------------------------|
| 2° disaccharidase deficiency    | 2                         | 1                            |
| Malabsorption requiring         |                           |                              |
| hyperalimentation               |                           | 1                            |
| Hypernatraemia                  | 3                         | 1                            |
| Hypoglycaemia                   |                           | 1                            |
| Pneumonia                       | 2                         | 2                            |
| Convulsion                      | 3                         | 2                            |

Complications

Table II shows the complications arising in the two gastroenteritis groups of patients.

Racial groups

Table I shows the distribution of various ethnic groups. Maoris and Pacific Islanders predominated in the younger age groups affected by both rotavirus and non-rotavirus gastroenteritis. Controls showed a similar ethnic pattern.

Enterotoxigenic strains

Of the 78 pools tested, eight were weakly ST+ (ratios > 0.0700 but < 0.0800) and six were LT+. Assay of the individual ten strains of each pool identified enterotoxigenic strains. Table III summarises the enterotoxigenic isolates.

Table III Enterotoxigenic Esch. coli isolates

| Age | Patient group                        | Pool number | ST+ pool | LT+ pool | Number of individual strains from pool |
|-----|--------------------------------------|-------------|----------|----------|----------------------------------------|
|     |                                      |             | ST+      | LT+      | ST+/LT+                               |
| 3M  | Rotavirus gastroenteritis            | 26          | +        | +        | 3/10 4/10 1/10                        |
|     | Non-rotavirus gastroenteritis        |             |          |          |                                        |
| 16M | Rotavirus gastroenteritis            | 28          | +        | -        | 1/10 0/10 0/10                        |
|     | Non-rotavirus gastroenteritis        |             |          |          |                                        |
| 7M  | Rotavirus gastroenteritis            | 51          | +        | +        | 4/10 3/10 0/10                        |
|     | Non-rotavirus gastroenteritis        | 54*         | +        | +        | 10/10 10/10 10/10                      |
| 16M | Rotavirus gastroenteritis            | 65          | +        | -        | 2/10 0/10 0/10                        |
|     | Control                              | 78          | -        | +        | 0/10 5/10 0/10                        |
| 23M | Rotavirus gastroenteritis            | 86          | +        | +        | 4/9 8/9 3/9                           |
|     | Control                              | 88          | +        | -        | 1/10 0/10 0/10                        |

*Salmonella typhimurium.*

isolates. Dual enterotoxigenicity of a strain (ST+/LT+) was encountered only in two patients' stools. Mixtures of ‘toxitypes’ within one pool were encountered.

Interpretation of gut weight/remaining body weight ratios

Strain U5/41 (serotype 01K1H7) was used as the non-toxigenic control and
gave a mean ratio of 0.0612 (range 0.0487–0.0700, standard deviation ± 0.0062). Strains B7a (serotype 0148 K?H28), H10407 (serotype 078H11) and 408-3 (?serotype) were used as positive enterotoxigenic (ST+/LT+) strains and gave mean ratios of 0.1077, 0.0843 and 0.1203 respectively with ranges of 0.0769–0.1466, 0.0700–0.1066 and 0.0707–0.2877 respectively, with standard deviations of ± 0.022, ± 0.011 and ± 0.0516 respectively.

Assuming that greater ratios are associated with a higher degree of toxigenicity, a comparison of the mean ratios for each control strain was carried out. The mean ratios of strains H10407 and B7a were significantly different (P < 0.001). This suggests that these differences are true and that the idea of degrees of toxigenicity thus can be entertained (Klipstein, Engert and Short, 1977). The mean ratio of test strains regarded as being ST+ was 0.0736 (range 0.0701 to 0.0793, standard deviation ± 0.0025) being significantly greater than that of the control ST negative strain U5/41 (P < 0.001). From Table III it is seen that enterotoxigenic isolates were found in both groups of gastroenteritis patients and also in non-diarrhoeal controls.

Table IV  Combinations of potential enteric pathogens isolated

| Enterotoxigenic E. coli | Number of patients | Controls |
|-------------------------|--------------------|---------|
| LT+                     | 3                  | 0       | 1       |
| ST+                     | 3                  | 2       | 1       |
| ST+/LT+                 | 2                  | 0       | 0       |
| Shigella spp.           | 1                  | 1       | 0       |
| Salmonella enteritidis  | 1                  | 0       | 0       |
| (see typhimurium)       |                    |         |         |
| Salmonella typhi        | 0                  | 0       | 0       |
| Rotavirus               | 41                 | 0       | 2       |
| Non-agglutinable rotavirus | 2              | 0       | 1       |
| on IEM                  |                    |         |         |
| Adenovirus              | 5                  | 7       | 4       |
| Reovirus                | 0                  | 0       | 1       |
| Unknown                 | 0                  | 9       | —       |
| Number in group         | 43                 | 17      | 18      |

Table IV shows combinations of enteric pathogens isolated from gastroenteritis patients and control patients.

Electronmicroscopy (EM) and immune electronmicroscopy (IEM)
Mean rotavirus diameters for smooth and rough particles (Flewett, 1977), were 64.5 nm and 53.8 nm respectively (standard deviation ± 2.4 nm and ± 4.3 nm respectively). Rotavirus-like particles that failed to agglutinate on IEM were seen in two gastroenteritis patients' stools and in one control patient's stool. Solitary particles indicated non-agglutination.

Small round particles (which I shall call 'satellite particles') were seen in
close proximity to rotaviruses. These satellite particles were between 13 and 26 nm in diameter (mean 16.9 nm, standard deviation ± 3.8 nm) and appeared to have a surface substructure and were seen with increasing frequency with patient age. (Plates 1 and 2).

On four occasions direct EM of faecal viral pellets failed to show rotavirus that was later easily seen in large clumps on IEM. Table V summarises the per

Table V  Percent incidence of viruses seen by EM and IEM according to patient age and group

| Age group | <6/12 | 6/12 – <2Y | 2Y – 6Y |
|-----------|-------|-----------|--------|
| Rotavirus | R     | NR        | C       |
| Non-agglutinable rotavirus (on IEM) | 15 | 0 | 0 | 0 | 9 | 0 | 0 | 0 |
| Adenovirus | 23 | 43 | 0 | 5 | 29 | 18 | 0 | 33 | 25 |
| Corona-like virus | 54 | 29 | 0 | 45 | 100 | 91 | 60 | 100 | 75 |
| Reovirus | 0 | 0 | — | 0 | 0 | 9 | 0 | 0 | 0 |
| Rotavirus 'Satellite' particle | 31 | — | 0 | 55 | — | 0 | 70 | — | 0 |
| Capsomeres | 31 | 57 | 33 | 65 | 43 | 36 | 60 | 33 | 75 |
| Phage | 62 | 57 | 33 | 45 | 43 | 55 | 20 | 33 | 25 |
| Small round viruses | 85 | 86 | 100 | 100 | 100 | 100 | 90 | 100 | 100 |

R = Rotavirus gastroenteritis; NR = Non-rotavirus gastroenteritis; C = Control.

Tubular capsid protein structures as described Flewett (1977) were common to all groups of stool whether or not rotavirus was present and failed to agglutinate on IEM. Adenovirus (54–78 nm) were seen in stools from all patient groups. Their link with diarrhoea could not be established. Coronavirus-like particles (mean size 93 × 161 nm) were commonly seen in all three patient groups. Reovirus (80 nm) was seen in one control patient’s stool. It was distinguished from rotavirus by its size, capsid structure and failure to react with immune rotavirus serum on IEM. Small round viruses (SRV) 13–29 nm not identifiable as astroviruses or calciviruses were an almost invariable inhabitant of all stool types. Bacteriophages of various shapes and sizes were common inhabitants of stools regardless of clinical condition.

Stool light microscopy

Table VI shows the per cent incidence of stool red blood cells, leucocytes and mucus strands according to age and clinical group. Generally, all three features were common to rotavirus gastroenteritis where comparisons with non-rotavirus gastroenteritis and controls were possible. No parasites were recorded.
Plate 1  Electronmicrograph showing 'satellite particles' approximately 25 and 26 nm diameter associated with 'smooth' rotaviruses. Magnification approximately $\times 340,000$. 
Plate 2 Immune electronmicrograph showing rotavirus satellite particles in association with 'rough' rotaviruses. Magnification approximately $\times 136,000$. 

[facing page 347]
Table VI  Percent incidence of light microscopic features of stools according to age and clinical group

| Age group Clinical group   | <6M | 6M - 2Y | 2Y - 6Y |
|----------------------------|-----|---------|---------|
|                            | R   | NR      | C       |
|                            | R   | NR      | C       |
|                            | R   | NR      | C       |
| Stool red blood cells      | 31  | 0       | 0       | 25  | 0       | 0       | 30  | 33      | 0       |
| Stool leucocytes           | 31  | 14      | 33      | 35  | 29      | 0       | 70  | 33      | 0       |
| Stool mucus strands        | 38  | 0       | 0       | 25  | 29      | 18      | 80  | 33      | 0       |

R = Rotavirus gastroenteritis; NR = non-rotavirus gastroenteritis; C = control.

Discussion

This article points out the high incidence of upper respiratory symptoms and signs associated with rotavirus gastroenteritis and other causes of diarrhoea. This was alluded to (without reference) in a leading article (1977) but a respiratory mode of transmission of rotavirus was not suggested. From this study there is clinical evidence for this, but the hypothesis would also support a similar mode of transmission of non-rotavirus gastroenteritis and its largely unknown cause or causes. It should be noted that this study took place during winter when respiratory admissions are most common. However, respiratory symptoms and signs were also a common finding in Washington children with rotavirus gastroenteritis reported by Rodriguez, Kim, Arrobio, Brandt, Chanock Kapikian, Wyatt and Parrott (1977) and Lewis, Parry, Davies, Parry, Mott, Dourmashkin, Sanderson, Tyrrell and Valman (1979) showed a significant excess of rotavirus infected children with respiratory illness. A female predominance in the younger age groups affected by rotavirus was noted. The finding of rotavirus or non-agglutinable rotavirus in non-diarrhoeal stools in all age groups suggests asymptomatic infection. This is contrary to current evidence elsewhere except that newborn infants in neonatal units appear to be colonised by rotavirus with quite variable consequences (Chrystie Totterdell and Banatvala, 1975; Totterdell, Chrystie and Banatvala 1976; Murphy, Albrey and Crewe, 1977).

The predominance of Maoris and Pacific Islanders in the younger age groups with or without gastroenteritis may be a reflection of socio-economic, nutritional or other factors. Evidence of spread within families was seen with equal frequency in Maoris and Caucasians. Rotavirus more often spread to siblings than other agents of gastroenteritis.

Enterotoxigenic strains were isolated from six patients with diarrhoea and three control patients. No strain producing ST had mouse mean gut weight/remaining body weight ratios exceeding 0.0800 indicating a low level of ST production. From the work of Klipstein, Engert and Short, 1977, relative degrees of enterotoxigenicity seem to occur amongst toxigenic enterobacteriaceae. Whether or not the weakly ST+ strains found in this study produced the symptoms in those patients with gastroenteritis remains to be seen. To date there is no information concerning the amount of toxigenic activity
required to classify a strain as toxigenic except arbitrary infant mouse mean
gut weight body ratios which vary from one published study to another
(Dean, Ching, Williams and Harden, 1972; Morris, Merson, Sack, Wells,
Martin, De Witt, Feeley, Sack, Bessudo, 1976; Donta, Wallace, Whipp and
Olarte, 1977). The finding of enterotoxigenic isolates in control patients
indicates that LT and ST plasmids are probably circulating freely in the
community, and as pointed out by Pickering, Du Pont, Evans, Evans and
Olarte (1977), asymptomatic subjects are probably important in transmis-
sion of infection. Combinations of enteric pathogens isolated from gastroen-
teritis patients and control patients were a common finding. The isolation of
multiple enteric pathogens supports the findings of Evans, Olarte, Du Pont,
Evans, Galindo, Portnoy and Conklin (1977); Schoub, Gref, Lecatsas,
Prozesky, Hay, Prinsloo and Ballard (1977); Echeverria, Ho, Blacklow,
Quinnan, Portnoy, Olson, Conklin, Du Pont and Cross (1977); and Madeley,
Cosgrove, Bell and Fallon (1977). Rotavirus was responsible for a large
proportion of non-bacterial gastroenteritis in most age groups accounting for
74 per cent of diarrhoeal illness in the six months to less than two year age
group, and 77 per cent in the two to six year group. In the youngest age group
(less than six months) its prevalence was 65 per cent. Rotavirus’ high overall
prevalence (72 per cent) may be explained by the winter season during which
the study took place. From overseas reports (Middleton, Szymanski, Abbott,
Bortolussi and Hamilton, 1974; Bryden, Davies, Hadley, Flewett, Morris
and Oliver, 1975; Davidson, Bishop, Townley, Holmes and Riuck, 1975;
Kapikian, Kim, Wyatt, Cline, Arrobio, Brandt, Rodriguez, Sack, Chanock
and Parrott, 1976), rotavirus diarrhoea appears to occur at the cooler times
of the year in temperate climates but neonates in Sydney showed no
seasonal variation of rotavirus excretion (Murphy, Albrey and Crewe, 1977).
It remains uncertain whether seasonal variation of rotavirus infection occurs
in New Zealand. However, this high detection rate may be reflected by the
use of IEM. The increased sensitivity of IEM over EM was shown by the
detection of rotavirus in four patients which would otherwise have been
missed by the latter method. Rotaviruses of two gastroenteritis patients and
one control patient failed to agglutinate on IEM. The particles were identical
in structure to agglutinable rotavirus and the severity of attributable disease
appeared to be similar. Zissis and Lambert (1978) have shown that IEM is a
valid means of serotyping rotavirus. At least two serotypes of rotavirus (one
predominant) were responsible for a large proportion of paediatric gastroen-
teritis admissions in Auckland at the time of the study.

Adenoviruses, small round viruses and coronavirus-like particles
appeared (on EM) to a similar extent in gastroenteritis and control patients’
stools. Without immune serum, identification of parvoviruses and their dis-
tinction from other small round viruses by IEM was not possible. Astrovir-
uses and caliciviruses (Madeley and Cosgrove, 1975 and 1976) were not
seen.

‘Satellite particles’ were a common accompaniment of rotavirus. The
proximity of these small isometric particles to rotavirus may only be an accidental encounter in the overcrowded tube journey to the outside world. But that this was seen with a frequency of 54 per cent suggests that some design was involved in their meeting. It is possible that these particles represent small aggregates of coat proteins. They have not been described previously. The author has not seen these particles in rotavirus positive specimens from patients at St. Thomas' Hospital.

The capsid protein described by Flewett (1977) as an association with rotaviruses of various animal species was seen in all types of stool whether or not rotavirus was detectable. This material failed to agglutinate on IEM suggesting that it is not of rotavirus origin.

In 15 per cent of gastroenteritis cases no pathogen could be found. It is possible that these infections were due to rotavirus excreted in numbers not great enough for detection even by IEM of faecal viral pellets. The features of most cases of rotavirus gastroenteritis and non-rotavirus gastroenteritis were so similar that a separate aetiology for each group could not be distinguished on clinical evidence. That only one stool specimen from each patient was examined may explain negative results, but the overall yield of potential pathogens is higher or similar to other published studies already cited where multiple specimens had been collected. The role of enteropathogenic serotypes of *Esch. coli* (EPEC) and their relationship with enterotoxin production may cast some light on the area of aetiologically unexplained gastroenteritis. Data on EPEC serotypes is being prepared for publication.

(My thanks to Dr R. B. Ellis-Pegler and Dr T. E. Miller for their helpful criticism. Additional thanks to Dr W. E. Lang, Dr C. Howden and Dr Keitha Farmer and other clinicians for giving me access to their patients and clinical notes. My gratitude to the nursing staff who collected the specimens and without whose help this work would not have been possible. Dr Sharon Hannan's help with ultracentrifugation is gratefully appreciated. My thanks to Merck, Sharp and Dohme who supported this work by means of a fellowship.)

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