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Rotavirus and Viral Gastroenteritis

THE AWARENESS THAT VIRUSES play a major role in the causation of diarrheal disease has been one of the outstanding medical advances of recent time. Previously, surveys of diarrheal disease had failed to determine the causative agent in up to two-thirds of all cases—no known pathogen could be isolated from the stools of these patients. Since 1972, however, several different viruses have been identified in stools by electronmicroscopy and have been shown to be associated with diarrheal disease throughout the world. Rotaviruses have received the most attention and have been accepted as a major cause of childhood gastroenteritis.

Description of Pathogens and Diseases

A growing number of different viruses are now associated with diarrheal disease (see table 9-1). Rotaviruses appear to be the most important of these and are therefore given greatest attention in this chapter. Several reviews of the rotaviruses have been published (Flewett and Woode 1978; Holmes 1979; McNulty 1978; Steinhoff 1980; Yolken and Kapikan 1979).

Identification

A number of different viruses may cause gastroenteritis, and the disease may vary accordingly. In rotavirus gastroenteritis, the onset is generally quite sudden, and vomiting may be the presenting symptom or may accompany diarrhea at the start. Vomiting is often the dominant feature rather than diarrhea. Fever is present in many cases. Dehydration frequently occurs, but may be more severe in combined infections with pathogenic *Escherichia. coli.* There is no consistent pattern of association between rotavirus and pathogenic *E. coli.* In hospitalized children the fever and vomiting usually resolve in the first 5 days, and recovery occurs within about a week. Mortality rates are low in hospitalized children in developed countries but may be considerable among untreated children in developing countries. Dehydration and shock are the most likely terminal processes, and oral rehydration is as effective in treating viral diarrhea as it is in treating bacterial diarrhea (Nalin and others 1979). Rotavirus gastroenteritis may occur as a single case, or one episode of an epidemic outbreak. There is also a form of continuing infection in some newborn nurseries in which a high proportion of the infants are asymptomatic.

Occurrence

Rotavirus appears to be almost universally distributed in human populations around the world. It has been found in the stools of children with diarrhea from Japan and New Zealand in the East, to Canada and Argentina in the West. It has been identified in tropical as well as temperate climates, although it may not be quite such an important etiological agent for diarrhea in tropical countries as in temperate regions (pathogenic *E. coli* may be more dominant in the tropics; see chapter 13).

Infectious agents

Acute nonbacterial gastroenteritis has long been recognized as a clinical entity. There was epidemiological evidence of outbreaks of infectious diarrheas in which neither bacterial nor parasitic organisms could be found. Volunteer experiments demonstrated that diarrhea could be transmitted by oral administration of bacteria-free fecal filtrates. A particle of sub-bacterial size, presumably a virus, seemed likely. The development of techniques to culture enteroviruses and adenoviruses from stool samples failed to identify any organisms that occurred predominantly in patients with diarrhea. The application of electronmicroscopy

to diarrheal stool samples was the decisive advance. The technique was enhanced by ultracentrifugation, antiserum clumping of particles (immuno-electron-microscopy, IEM), and negative staining. These methods have their limitations because, unless particles occur in concentration of greater than 10^6 per gram of feces, they may not be detectable. More recent techniques for the identification of rotavirus particles from stools include infected cell immunofluorescence, counter-immunoelectrophoresis, radioimmune assay, and enzyme-linked immunosorbent assay (ELISA), and the methodology is still rapidly improving.

Any form of examination of the feces may give a distorted indication of the pathophysiology of gastroenteritis, a condition in which the primary lesion is in the small bowel. The large bowel is distal to the site of infection and, even in a child, contains some 10^{13} bacteria and also many fungi, mycoplasmas, and protozoa. All of these frequently have their own viral infections and may shed particles into the feces. Bacteriophages, unless they have tails, may be very difficult to distinguish from small spherical human viruses. The colon and its flora alter the effluent from the small bowel in a number of ways before it presents as fecal material.

ROTAVIRUSES. These viruses from the stools of children are morphologically identical to those found in the stools of some calves, piglets, foals, lambs, mice, and young monkeys with acute diarrhea. The human virus has been transmitted to a number of these animals. The virus particles are spherical, 70 nanometers in diameter, and made up of double-stranded RNA in two distinct capsid layers that on electron-microscopy give the appearance of a wheel, hence the name rotavirus (figure 11-1*a*). Rotaviruses are now classified as a genus of the family Reoviridae (see table 9-1). There are at least two, and possibly four, serotypes of human rotavirus.

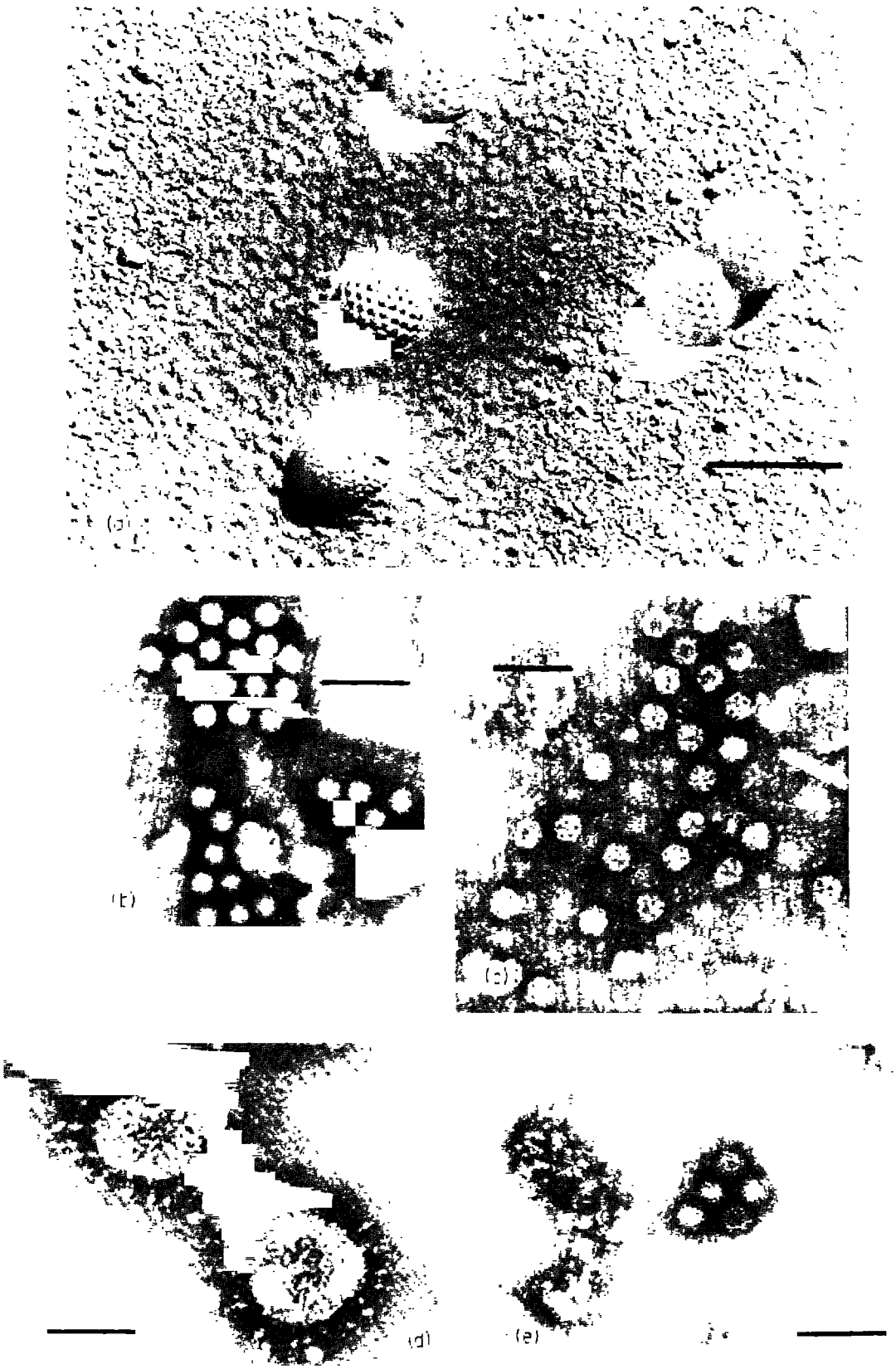
ADENOVIRUSES. These viruses are found in small numbers in feces of some patients with diarrhea, but occasionally in great numbers (see, for instance, Richmond and others 1979). The evidence that adenoviruses can cause diarrhea is circumstantial, and there is no equivalent animal model. However, their occasional presence in large numbers indicates that they must have replicated in and destroyed cells.

ASTROVIRUSES. These are particles of 28 nanometers in diameter that are roughly spherical with surface indentations that result in the appearance of a 5- or 6-pointed star (figure 11-1*b*). They have been found in the feces of normal children and of those with gastro-enteritis (see, for instance, Kurtz, Lee and Pickering 1977). There is still no firm evidence that they are pathogenic.

CALICIVIRUSES. These are picornaviruses of approximately 35 nanometers in diameter (figure 11-1*c*) and were previously known as fecal viruses in such diverse species as sealions, pigs, and cats. Calicivirus has now been associated with several outbreaks of gastro-enteritis and has also been found in the stools of asymptomatic individuals (Schaffer 1979; Studdert 1978).

CORONAVIRUSES. These are well-known agents of acute gastroenteritis in piglets and calves. They vary in shape and size but have a distinctive appearance in electron micrographs (figure 11-1*d*). Recently they have been described in feces from young adults in Britain and from children in Canada and Australia. They have been isolated in tissue culture. They are not proven human enteric pathogens, although they are a major cause of the common cold (Clarke, Caul and Egglestone 1979; McIntosh 1979; Robb and Bond 1979).

Figure 11-1. *Agents of viral gastroenteritis.* (*a*) *Rotaviruses* under scanning electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: J. Cohen, Station de Recherches de Virologie et d'Immunologie, Institute National de la Recherche Agronomique, Thiverval, France.) (*b*) *Astroviruses* under transmission electronmicroscopy, showing the characteristic 5- or 6-pointed star appearance. Scale bar = 0.1 micrometers. (Photo: C. R. Madeley, Royal Victoria Infirmary, Newcastle-upon-Tyne, UK.) (*c*) *Caliciviruses* under transmission electronmicroscopy, showing the characteristic surface hollows. Scale bar = 0.1 micrometers. (Photo: A. J. Zuckerman, London School of Hygiene and Tropical Medicine, London, UK.) (*d*) *Coronaviruses* under transmission electronmicroscopy. The virus particles vary in shape and size and possess a distinctive array of widely spaced surface projections, approximately 20 nanometers long, which give the characteristic "corona" appearance. Scale bar = 0.1 micrometers. (Photo: E. O. Caul, Public Health Laboratory Service, Bristol, UK.) (*e*) *Norwalk agent particles* under transmission electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: E. O. Caul, Public Health Laboratory Service, Bristol, UK)


(a)
(b)
(c)
(d)
(e)

ENTEROVIRUSES. Most studies have indicated that the occurrence of these viruses is no more common in the stools of children with diarrhea than in those from control groups. However, in one outbreak of gastroenteritis, believed to be due to a failure of water supply purification, coxsackie- and echoviruses were found as frequently as *Shigella sonnei* in the stools of those with diarrhea (Green and others 1968).

MEASLES VIRUS. In temperate climates measles is now an exanthem which affects children primarily with respiratory symptoms and a systemic upset. In the past, however, diarrhea was associated with measles and 28 percent of children with measles in a London clinic in 1904 had diarrhea (Balme 1904). In tropical countries where measles is frequently more severe, not only is the rash very prominent, but there is evidence of invasion of the bowel epithelium. The giant cells associated with measles have been seen in the mucosa of biopsy specimens, and the excretion of giant cells in the feces may be prolonged. Diarrhea is frequently associated with measles in the tropics. A study in Guatemala showed that half of the children under 5 years old with measles had acute diarrhea (Scrimshaw and others 1966).

NORWALK AGENT AND OTHER SMALL ROUND VIRUSES. Norwalk agent is a small round virus particle of 27 nanometers in diameter (figure 11-1*e*) that caused an outbreak of nonbacterial gastroenteritis in 50 percent of students and staff of an elementary school in Norwalk, Ohio (USA) in 1968. There was a 32 percent attack rate among family contacts. Rectal swab filtrates produced disease in volunteers, and subsequently the particle was visualized by IEM. Other morphologically similar viruses (Montgomery County agent and Hawaii agent) have been isolated from different epidemics of diarrhea (Dolin 1979). Some of these show a cross-immunity, but several distinct serotypes have been demonstrated. These agents produce a mild, self-limited gastroenteritis that lasts 24–48 hours and affects older children and adults more often than the rotavirus.

Particles of similar size have been associated with small epidemics of winter vomiting disease in Britain: the W and Ditchling agents. These particles differ antigenically from the Norwalk agent and a high proportion of adults appear to have antibody against them. Some patients continued to excrete the particles for over 2 months after the illness. Smaller spherical particles of 25–26 nanometers were recently found in the feces of a high proportion of patients suffering from food poisoning in Britain after eating seafood cocktails containing cockles (Appleton and Pereira 1977). IEM suggests this agent (cockle virus) is antigenically similar to W agent, but different from Norwalk agent.

There is still considerable confusion in identifying viruses in feces, especially when variations in methods, possible artifacts, and the presence of bacteriophages are considered. It is far from proven that all the viruses discussed above cause gastroenteritis, and all have been isolated from healthy persons as well as from those with gastroenteritis. On some occasions, more than one virus has been present. However, it appears likely that, just as the upper respiratory tract reacts to a range of viruses by producing the symptoms of the common cold, the alimentary tract will react to a range of viruses by developing gastroenteritis. It is only the rotaviruses that are unquestionably a major cause of gastroenteritis worldwide, and it is these which are discussed in the rest of this chapter.

Reservoirs

Man is probably the only important reservoir for human rotavirus infection.

Transmission

It is probable that transmission modes are similar to those of the enteroviruses and hepatitis A virus; that is, fecal-oral and usually person-to-person but sometimes via contaminated water, food, or shellfish. Airborne respiratory transmission remains an additional possibility.

Incubation period

Studies that include information about more than one case of rotavirus diarrhea within a family or closed community indicate that the incubation period is between 24 and 72 hours.

Period of communicability

This is very uncertain because many details about the route and mode of spread are unknown. Moreover, rotavirus diarrhea apparently disappears from a community for months at a time in hot weather; also, the organism cannot be found in stools unless it is present in high concentrations ($>10^6$ per gram). If transmission is dependent on the ingestion of a large number of virus particles, communicability from a patient will be at its maximum on about the third to fourth day of the disease, coinciding with the period of maximum virus shedding (10^{11} per gram or more) and

would be unlikely after the eighth day—although excretion of rotavirus can continue for more than 20 days. Asymptomatic infection and excretion certainly occur, but persistent carriage has not been demonstrated.

Resistance

Facts are limited by ignorance about the epidemiology and pathophysiology of rotavirus infection. Newborn babies are apparently susceptible to the infection, particularly in the nursery situation, but only a proportion of them develop clinical symptoms. The low pathogenicity at this age may possibly be due to passively acquired maternal immunoglobulins. Although the majority of older children and adults have antibody to rotavirus, adults can be infected—as is shown by rising antibody titers and sometimes by clinical infections. The existence of at least two rotavirus serotypes that are not cross-protective may partly explain repeat attacks.

Epidemiology

Rotavirus gastroenteritis is primarily a disease of children, especially those between 6 months and 3 years old. Rotavirus infection can spread very rapidly among neonates in nurseries, but many of these infections are asymptomatic (Jesudoss and others 1979). Rotavirus infection has been recorded in adults, often in association with infection of their children (for instance. Wenman and others 1979; Zissis and others 1976). In all age groups asymptomatic infection is fairly common, but persistent carriage is not demonstrated.

Seroepidemiological surveys show that neonates have a high prevalence of rotavirus antibodies (presumably of maternal origin) that falls over the first 6 months of life. Antibody prevalence then rises again until, by about 3 years, 80–90 percent have rotavirus antibodies, and this high prevalence is maintained thoughout adult life. For instance, a survey of 266 children in Vellore (India) showed that the antibody prevalence was 75 percent among neonates, 30 percent among 5–6 month old infants, and 87 percent among 3 year olds (Jesudoss and others 1978). Similar results are found in affluent communities, indicating that rotavirus transmits successfully even in conditions of good hygiene, pure water, and full sewerage. This suggests direct person-to-person, fecal-oral, or respiratory routes of transmission, particularly within family groups. Parent-to-child, child-to-parent, and sibling-to-sibling spread are all likely (see, for instance, Wyn-Jones, Lillington and Alzaka 1978).

By 18 months of age 85 percent of children in the area of Washington, DC (USA) have acquired antibodies to both Type 1 and Type 2 rotaviruses, and the high antibody prevalence is maintained throughout life (Yolken and others 1978*a*). In contrast, in the same area, only about 10 percent of 3 year old children have Norwalk agent antibodies, and this prevalence rises to only around 50 percent later in life (Kapikian and others 1978). The same picture of rapid acquisition of rotavirus antibodies by nearly all children, contrasted with gradual acquistion of Norwalk agent antibodies (to a maximum prevalence of only 33 percent), was found in Bangladesh (Kapikian and others 1978; Sack and others 1980).

In temperate countries there is a striking seasonal variation, with most cases occurring in the coldest months of the year, whereas in tropical climates (and poorer communities) there appears to be much less seasonal variation. At a children's hospital in Washington, DC (USA) during 1974–78, rotavirus accounted for 39 percent of inpatient diarrhea and 22 percent of outpatient diarrhea. The equivalent figures in January were 71 percent and 62 percent, whereas during June and July they were 4.4 percent and 4.8 percent for inpatients and outpatients, respectively (Brandt and others 1979). A comparative study in Dallas (Texas, USA) and San Jose (Costa Rica) showed that in both settings rotavirus accounted for 50–60 percent of acute nonbacterial pediatric gastroenteritis episodes occurring from December through February. This is the cool period in Dallas and the dry season in San Jose. During the rest of the year the virus was not recovered from any Dallas patients but was found in 30–40 percent of Costa Rican patients in every month except May (Hieber and others 1978).

In developed and temperate countries (such as Australia, Britain, Japan, and the USA), about half of all diarrhea in children that requires hospitalization is caused by rotavirus infection. During the summer, 0–20 percent of cases are rotavirus associated, and in winter this figure rises to 70–80 percent. Studies in developing and tropical countries have indicated that rotavirus accounts for a somewhat lower proportion of hospitalized childhood diarrhea cases—maybe 25–50 percent.

Schnagl, Holmes and Mackay-Scollay (1978) studied 537 episodes of diarrhea among 473 hospitalized children under 6 years old in Western Australia. Among aboriginal children the percentages of 387 diarrheal stools from which known pathogens could be isolated were: parasites 17 percent, rotavirus 16 percent, *Salmonella* or *Shigella* 13 percent, pathogenic *E. coli* 9 percent, adenovirus 3 percent, and astrovirus 2

percent. Among nonaboriginal children the percentages of 150 diarrheal stools from which known pathogens could be isolated were: rotavirus 25 percent, parasites 11 percent, *Salmonella* or *Shigella* 10 percent, pathogenic *E. coli* 10 percent, adenovirus 4 percent, and astrovirus 3 percent. The data suggest the possibility of a winter peak of rotavirus gastroenteritis among nonaboriginals and a summer peak among aboriginals. Rotavirus was detected in the stools of only 2 out of 170 children without diarrhea.

Black and others (1979) investigated 4,498 diarrhea cases reporting to Matlab hospital (Bangladesh) and were able to identify a pathogen in the stools of 85 percent (see table 13-1). Rotavirus was associated with 23 percent of all reported diarrhea cases and with 40 percent of diarrhea cases under 5 years old.

During January–June 1976, Echeverria and others (1978) studied 82 hospitalized infants and children with diarrhea in Manila (Philippines). A viral etiology was indicated in 17 percent of cases, enterotoxigenic *E. coli* in 11 percent, *Salmonella* or *Shigella* in 7 percent, *Vibrio cholerae* in 4 percent, *Giardia lamblia* in 5 percent, and *Entamoeba histolytica* in 2 percent. Ten percent of children had evidence of infection with more than one enteric pathogen. Only 1 out of 49 healthy children had rotavirus particles in their stools. Echeverria and others (1977) found evidence of rotavirus infection in 56 percent (42/75) of children (3 days to 4 years old) with diarrhea seen at hospitals in Taipei (Taiwan) during the summer.

Studies of 293 hospitalized children under 5 years old with diarrhea in Caracas (Venezuela) showed a rotavirus etiology in 41 percent of cases (Viera de Torres, Mazzali de Ilja and Esparza 1978). Only 3 out of 66 healthy children were excreting rotavirus. Espejo and others (1978) studied 242 children under 5 years old with acute diarrhea in two hospitals in Mexico City (Mexico) and found rotavirus excretion in 25 percent. Although the peak of all diarrhea cases in Mexico occurred in June–September, the peak of rotavirus diarrhea occurred in October. The highest age-specific proportions of diarrhea cases with rotavirus excretion were in the 4–10 month age group. Of the 60 children who excreted rotavirus, 22 also excreted *Salmonella*, *Shigella*, or potentially pathogenic serotypes of *E. coli*. Rotavirus infection was less common among breastfed infants with diarrhea (10 percent) than among nonbreastfed infants with diarrhea (27 percent).

Little information is yet available on rotavirus gastroenteritis in Africa. Brookfield and others (1979) detected rotavirus in the stools of 31 percent of 123 hospitalized children under 4 years with diarrhea in Dar es Salaam (Tanzania). Mutanda (1980*a*) studied infants and children with diarrhea at 3 hospitals in Kenya. Rotavirus accounted for 41 percent of inpatients in Nairobi, for 14 percent of inpatients and 17 percent of outpatients in Mombasa, and for 29 percent of inpatients and 11 percent of outpatients in Kisumu (see also Mutanda 1980*b*; Mutanda, Cruickshank and Itotia 1979). Hansen and others (1978) found that 6 percent of adult inpatients with diarrhea in Nairobi had serological evidence of rotavirus infection, whereas 26 percent had *Shigella* and 18 percent had enterotoxigenic *E. coli*.

Some studies have found that rotavirus infection is associated with diarrhea of more than average severity; if this is the case, the proportion of hospitalized diarrhea cases due to rotavirus may be greater than the proportion of all diarrhea cases. Two community studies support this hypothesis. Spencer and others (1980) isolated rotavirus from only 7 percent (5 of 74) of nonhospitalized children under 4 years old with diarrhea in a coastal area of El Salvador. Similarly, rotavirus accounted for only 14 percent (26 of 183) of diarrheal episodes, from which no bacterial or protozoal pathogen could be isolated, among 0–3 year old nonhospitalized children in a highland village in Guatemala (Wyatt and others 1979). If all diarrhea among these children is considered, the proportion due to rotavirus was approximately 7 percent. The incidence of rotavirus diarrhea was estimated at only 1.1 episode per child during the first 3 years of life.

Control Measures

The spread of infection may be reduced by improved personal and domestic hygiene and by the sanitary disposal of excreta, but this is uncertain. The very high prevalence of antibodies to rotavirus in children over 2 years old in affluent communities indicates that rotavirus transmits successfully even in conditions of near optimum hygiene, water supply, and sanitary facilities.

Infections by rotavirus in breast-fed infants are less likely and less severe than in bottle-fed infants. Breast milk has been shown to contain specific antibodies to rotavirus (Yolken and others 1978*a*), but it now appears that other unidentified properties of breast milk are responsible for its protective effect (Totterdell, Chrystie and Banatvala 1980).

The development of rotavirus vaccines is a distinct possibility within the next few years, but delivering them to the most vulnerable individuals (children aged 5–24 months) will be a difficult task in most developing countries.

Occurrence and Survival in the Environment

There is no direct evidence on the behavior or occurrence of human rotavirus in the environment because the virus cannot be routinely isolated from environmental samples. Tissue culture methods have been developed (Wyatt and others 1980), but rotavirus does not grow readily in cell culture, and demonstration of cytopathic effects is difficult. Investigations into rotaviruses in the environment must await the development of improved tissue culture techniques or sensitive immunological antigen-detecting techniques.

The available evidence on human rotavirus in the environment is indirect. The knowledge that rotavirus may be excreted in large numbers (10^{11} per gram) by infected individuals, and that incidence of infection appears to be very high in some communities, leads to the assumption that human rotavirus may be present where fecal pollution is present, especially where high concentrations of enteroviruses are found (see chapter 9). However, far too little is known about the prevalence of rotavirus excretion, or about the numbers of viruses excreted by asymptomatic excreters, to predict at what concentration human rotaviruses might be found in, for instance, sewage. Some evidence is provided by outbreaks of gastroenteritis believed to be of viral etiology that have been traced by epidemiological analysis to a particular source, such as contaminated water or shellfish.

Many outbreaks of gastroenteritis have been linked to polluted water, and many of these have had an undetermined etiology and could be of viral origin. Craun (1978) reported that during 1975 and 1976 an etiological agent could not be identified in 75 percent of waterborne gastroenteritis outbreaks in the USA. From observations of the symptoms, it is more than probable that some of these outbreaks were due to rotaviruses or other diarrhea-causing viruses. However, there is no evidence that water conforming to conventional bacteriological criteria has ever caused rotavirus infection, and waterborne gastroenteritis occurs in circumstances similar to those which may lead to other outbreaks of waterborne, fecal-oral gastroenteritis (see, for instance, Morens and others 1979).

Other sources of circumstantial evidence of rotavirus behavior in the environment are the documented accounts of gastroenteritis associated with the ingestion of contaminated shellfish. The so-called cockle virus was detected during outbreaks of gastroenteritis in the UK affecting 797 people who had a common history of eating seafood cocktails containing cockles grown in waters polluted by sewage (Appleton and Pereira 1977).

Dismukes and others (1969) reported 33 cases of gastroenteritis of unknown etiology, and 4 cases of hepatitis, occurring among 128 persons attending a picnic at which raw clams were eaten. Ironically, at an annual convention of a shellfish sanitation association held at New Haven (Connecticut, USA) in November 1968, 19 persons ate raw clams and 17 of them developed acute gastroenteritis of unknown etiology (Ratzan and others 1969). There was subsequently a 37 percent secondary attack rate among family contacts of the 17.

The largest outbreak of viral gastroenteritis so far reported occurred in Australia during June and July 1978 (Murphy and others 1979). At least 2,000 cases were reported throughout the country; cases had a common history of eating rock oysters harvested from polluted estuaries near Sydney. The causative organism was shown to be Norwalk agent. As a result of this outbreak, the New South Wales state government has required that all oysters harvested from the incriminated areas be depurated for at least 2 days in disinfected water, and a panel of volunteers has been set up to test-consume samples of oysters prior to marketing. The data reviewed in chapter 9 on the elimination of enteroviruses from oysters in sterilized water suggest that a 2-day depuration time is inadequate to remove the risk of viral contamination with reliability.

Little is known about the survival of human rotavirus in the environment, and it is reasonable to assume, for the time being, that its environmental behavior is similar to that of the enteroviruses (chapter 9). Simian rotaviruses and reoviruses may provide a closer model for human rotavirus in the environment than the enteroviruses, and recently data on simian rotaviruses in water have been reported. Hurst and Gerba (1980) compared the survival of poliovirus 1, echovirus 7, coxsackievirus B3, and simian rotavirus in clean and polluted freshwaters and in estuarine waters of various salinities (1.2–2.8 percent) at 20°C. All viruses survived for a very similar time, undergoing a 3 log reduction in concentration in 6 to over 14 days in freshwaters and in 2 to 3 days in estuarine waters. This preliminary experiment suggests that simian rotavirus in fresh and saline water exhibits death rates well within the range reported for enteroviruses. Rotavirus is very stable under some conditions. The virus is stable in the pH range 2–9.8 and survives for at least 7 months, but not 4 years, at 18–20°C. Rotavirus in feces remained infectious and virulent for calves after

5 years storage at 4°C. Preliminary studies indicate that rotavirus may resist a temperature of 60°C, but not 63°C, for 30 minutes (G. N. Woode, personal communication).

Inactivation by Sewage Treatment Processes

The lack of adequate detection techniques has prevented any direct studies on the inactivation of human rotavirus by sewage treatment. However, there is some indication that rotaviruses may be less inactivated in treatment systems than polioviruses.

Farrah and others (1978) compared the ability of poliovirus, human rotavirus and simian rotavirus to adsorb to aluminum hydroxide flocs and activated sludge flocs. Aluminum hydroxide flocs reduced the concentration of poliovirus in tap water by 3 log units but only reduced the concentration of simian rotavirus by 1 log unit or less and did not noticeably reduce the number of rotavirus particles present in a dilute stool suspension. Activated sludge flocs reduced the concentration of added poliovirus by 0.7 to 1.8 log units but reduced simian rotavirus numbers by 0.5 log units or less. This suggests that the adsorptive characteristics of poliovirus and rotavirus are different and that lesser removals of rotavirus occur during water coagulation or activated sludge treatment than have been reported for polioviruses (see chapter 9).

Goyal and Gerba (1979) compared the proportion of 27 different excreted viruses that were adsorbed to a sandy loam soil when shaken in water for 30 minutes. Between 91 and 99.99 percent of all viruses adsorbed to the soil, except for echovirus 1 (55 percent), echovirus 12 (78 percent), echovirus 29 (14 percent), and simian rotavirus (52 percent). However, in another series of adsorption experiments using nine different soils, simian rotavirus tended to adsorb more than all other viruses studied except poliovirus 1, echovirus 7, and bacteriophage T4. A considerable amount of additional experimentation will be required before it is clear whether rotaviruses are less readily adsorbed than enteroviruses or merely less readily adsorbed than poliovirus 1.

The next few years will undoubtedly see many investigations into the removal of rotavirus from sewage and water. Pending the development of adequate concentration and detection techniques for human rotaviruses, studies may be done using seeded simian rotaviruses and reoviruses, which may provide suitable models for human rotavirus.

Inactivation by Night Soil and Sludge Treatment Processes

As with sewage treatment processes, no direct evidence is available on the inactivation of human rotavirus by night soil or sludge treatment. As noted above, reoviruses may provide a suitable model for environmental studies on human rotaviruses, and several differences in the environmental characteristics of enteroviruses and reovirus are known to exist. For instance, a series of studies by Ward and Ashley (1976, 1977*a*, 1977*b*, 1977*c*, 1978) have shown that reovirus is more heat resistant than poliovirus but that, unlike poliovirus, it is not protected against heat inactivation by being in sludge. These studies also showed that ammonia, although it is highly virucidal to enteroviruses at pH above 8, does not affect reoviruses, and that some detergents sensitize reovirus to heat inactivation while they protect poliovirus.

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