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REVIEW

VIRAL DIARRHEA OF YOUNG ANIMALS: A REVIEW*

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Abstract—A brief presentation is given of the enteric viral infections of young animals. The general characteristics of rotaviruses, coronaviruses and parvoviruses are reported, and the different aspects of the diseases associated with these viruses are discussed. Certain suggestions are made regarding the prevention of these diseases.

Key words: Viral diarrhea, rotaviruses, coronaviruses, parvoviruses

LA DIARRHEE NEONATALE DES ANIMAUX: REVUE

Résumé—L'objet de cette publication est de faire le point, de manière synthétique, de connaissances acquises au cours de ces dernières années sur les infections entériques d'origine virale chez les nouveaunés dans diverses espèces de mammifères. En particulier on fait mention des principales caractéristiques des rotavirus, coronavirus et parvovirus, de certains aspects des infections associées à ces agents viraux et de la prévention des correspondantes maladies.

Mots-clefs: Diarrhée virale, rotavirus, coronavirus, parvovirus

INTRODUCTION

It is well known that the diarrheas of young animals are caused by protozoa, bacteria and viruses. Rotaviruses, coronaviruses, parvoviruses and parvovirus-like agents, enteropathogenic strains of *E. coli* and *Salmonella* species, seem to be the most commonly involved in the etiology of the condition.

Viruses only will be considered in this review.

ROTAVIRAL INFECTIONS

The virus

Mebus *et al.* [1] were the first to report on having reproduced diarrhea in colostrum-deprived calves by filtrates of fecal material from infected calves given by the oral route. It was shown also that feces from calves which were exposed to either natural or experimental infection, contained viral particles resembling members of the *Reoviridae* family [2–4]. Later, similar viral particles were observed in the duodenal mucosa and in the feces of children with diarrhea [5–7]. Since the viral isolates from calves and children resembled

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each other closely, a new genus, "*Rotavirus*", was created under the family *Reoviridae*, and these viruses were assigned to it. Rotaviruses or agents resembling rotaviruses, were also found to be the cause of epizootic diarrhea of infant mice (EDIM) [8]. Similar viruses were detected also in the monkey (S.A. 11), in sheep and cattle (O agent) [9].

Recently the genus *Rotavirus* was officially recognized by the International Committee for the Nomenclature of Viruses [10] and the following properties were stated for the genus: double-stranded RNA viruses, resistant to pancreatin RNA-ase, divided into 11 or 12 segments with the molecular weight (MW) ranging from 0.2 to 2.3×10^6D. On the basis of the size of the RNA fragments of the strains isolated from man and from cattle, four classes of rotavirus RNA have been detected [11]. The most significant differences in the MW of the RNA between the human and the bovine isolates are in fragments 1, 4 and 5. This property serves to differentiate the genoma of human rotaviruses from that of bovine rotaviruses [12].

The virion contains 8–10 polypeptides having a MW ranging from 15 to 130×10^3D. Its buoyant density is 1.33–1.35 g/ml in CsCl [13]; it is stable at pH 3.0, resistant to ether, and relatively so to heat. Virus particles are 65–75 nm in diameter and possess an hexagonal core surrounded by an electron-lucent area from which the capsomeres radiate. Thirty-two capsomeres, which form an inner capsid layer, are described. Surmounting the inner layer is an outer layer which gives the viral particle the appearance of a smooth rim on the ends of 20 short spikes radiating from a wide hub [14].

Viral replication occurs in the cytoplasm of the cell where complete particles are seen, together with viral particles lacking the outer capsid [15, 16]. The morphogenesis of the virus takes place in association with the cisternae of the endoplasmic reticulum [17]. Granular or fibrillar masses,which are considered as precursor material of the virus or viroplasm, are found exterior to the cisterna. The virus is released from the cell following the rupture of the cell itself [17].

The Nebraska strain of rotavirus was found to agglutinate human group O cells [18]. The same red blood cells and also cells from guinea pigs and rabbits are agglutinated in the presence of fecal suspension obtained from a calf experimentally infected with the Nebraska strain of rotavirus [19]. The hemagglutination (HA) is inhibited by specific immune serum (IHA).

Human and bovine rotaviruses, as well as strains isolated from rabbits, pigs, lambs and foals, have been found to be antigenically related by complement fixation (CF), immunofluorescence (IF) and serum neutralization (SN) [20–25]. The group-specific antigen is located on the inner capsid [25–27] whereas the type-specific antigen is situated on the outer capsid of the virus [28]. The strains of rotavirus isolated from calves in either the United States or the United Kingdom, appear to be very closely related [29]. However, rotaviruses seem to be antigenically unrelated to reoviruses.

Although the virus is present in high concentration in the feces of infected animals, it is difficult to cultivate in cell culture. Only a small number of cells in the culture are susceptible to the virus, so that serial passage of the virus *in vitro* frequently fails. Successful attempts to cultivate the virus have been made recently by infecting suspensions of trypsinized cells, or by carrying out subpassages at long intervals of time. Once adapted to cell culture, the virus multiplies readily. It was suggested that the lactase which is present at the edges of the brush-bordered epithelial cells of the intestine acts as an uncoating enzyme for the virus [30]. This hypothesis has not been proved.

In the cytoplasm of PK-15 cells infected with bovine, swine or ovine rotaviruses are mature virions, as well as numerous empty particles which most probably are defective viruses, unable to multiply in permissive cells [28]. More recently it has been demonstrated that pig rotaviruses can be serially passaged in pig kidney cell cultures when the virus was treated with pancreatin or trypsin prior to inoculation of the cultures [23]. Moreover, the capacity of bovine rotaviruses to produce plaques appears to be greatly promoted when trypsin or DEAE-dextran are used [31]. Therefore, in order to facilitate the replication of wild strains of the virus in cell culture, it is recommended that 1:10 suspension of infected feces be treated with a solution containing 500 μg/ml of trypsin for 20 min [32]. The bovine rotaviruses replicate in bovine fetal intestinal cell cultures [33] and also in MDBK cells, in which the cytopathic effects (CPE) are characterized by the production of intracytoplasmatic inclusion bodies and vacuolization of the cytoplasm [2, 34]. The human and murine rotavirus multiply in organ-explants of the intestinal mucosa of the respective susceptible host [35, 36]. If the culture is performed on roller tubes the CPE are significantly enhanced [34, 37].

The infection

Rotaviral infection usually spreads horizontally but it does not appear that invertebrate are involved in the transmission. Rotaviral infections occur generally in young animals during the first few weeks of life. The infection has been observed in calves [38–43], pigs [44–46], foals [47], lambs [22, 48, 49], mice [50], rabbits [51, 52], deer, mink and buffaloes [53]. In Scotland the infection appears to be widespread in sheep, in both the adults (38%) and in the lambs (56%), with excretion of the virus in the feces of lambs possessing specific serum antibody [48].

Rotaviral infections in humans generally occur in children. While they may be inapparent in the newborn, they are always associated with gastroenteritis in children from 6 months to 1 year of age [28]. Serologic studies in Germany on 263 samples from children up to 10 years of age indicated that the highest positive rate of antibody to the Nebraska calf strain virus was among children 2-years of age or older [54].

Infection of humans and animals occurs through contact with infected individuals or through ingestion of contaminated materials. Because of the enormous number of virus particles usually present in the feces (about 10^{10}/g), and owing to the stability of the virus in the feces and its resistance to the commonly used disinfectants, it is extremely difficult to prevent the dissemination of the virus once the infection has been introduced to the premises [28]. Analogous difficulties are encountered in hospitals but in these cases the situation is readily brought under control [28]. It seems unlikely that adult animals carry the virus; on the other hand it appears that the virus is readily transmitted to the bovine fetus through the placenta [55]. This finding is supported by demonstrating antibody to rotavirus in the serums of 46% of 69 bovine fetuses that were examined.

Rotaviruses from one species can infect members of other species. This is the case of human strains with which the infection has been experimentally transmitted to pigs [56], calves [57], lambs and monkeys (*Macaca mulatta*) [58] and of bovine strains which have been used to infect pigs [25].

The knowledge of the immunity to rotavirus infection is incomplete. In the bovine, local immunity arising from exposure of the epithelium of the small intestine is more important

than the circulating antibody in protecting animals against re-infection. Colostral antibody plays an important role with regard to the behavior of the infection in the calf in that the appearance or absence of clinical signs of the disease is dependent on viral concentration in the intestinal lumen. The administration of approximately 3 liters of colostrum which brings the Ig level in the blood serum to a concentration exceeding 30 mg/ml is sufficient to prevent diarrhea [55].

The clinical signs of rotavirus infections appear after an incubation period ranging from about 15 hr to 3–4 days. The principal signs are diarrhea, depression, anorexia and dehydration [28]. The fever is erratic and in some cases may be absent. The disease has a duration of 4–8 days and may be fatal. In calves a mortality ranging from 1 to 50% was observed [59]. It is not clear whether death is caused by the virus infection or whether it is due to secondary bacterial invasion.

The changes associated with the infection are seen in the epithelial cells of the intestinal villi where viral multiplication occurs, sometimes characterized by the formation of sincytia [60]. Shortening or stunting of the villi is observed [61]; the columnar villous epithelial cells undergo degeneration and are replaced by cuboidal or squamous cells, while there is an increase in the number of reticular-like cells in the lamina propria. Goblet cells do not become infected. As the infection progresses the mucosa becomes flat because of the loss of the villous formation [44, 61, 62].

As regards the pathogenesis of rotaviral infections, the virus appears in the epithelial cells situated at the top of the villi, early during the incubation period of the disease [63]. The infected cells are destroyed and replaced by immature cells from the glandular crypts [64]. This process starts in the small intestine and proceeds to the other sections of the intestinal tract. It was suggested that the presence of the new cuboidal-shaped cells might represent an attempt by the organism to protect the lamina propria. On the other hand, this attempt may result in the acquisition of resistance to a re-infection. In fact, these new cells are apparently refractory to infection, being devoid of the specific receptors for the virus [64]. However, the cuboidal-shaped cells contain a very low level of disaccharidases, hence have little capacity to absorb glucose and galactose [65, 66]. Because of this there is a decreased capacity to utilize the lactose which accumulates in the intestine. Thus, the fermentation of lactose by bacteria results in the production of short chain fatty acids. The inflammatory reaction of the intestine might cause an increase in the peristaltic activity, predisposing the animal to diarrhea since the more rapid passage of the food through the intestine does not allow enough time for the completion of digestion and water adsorption.

The diagnosis or rotaviral infections is generally based on demonstration of the virus or the viral antigen in the feces. This is determined by electron microscopy (EM) or by fluorescent antibody (FA) [1, 67].

The virus can be detected by FA also in cell cultures infected with fecal preparations. For this purpose LLC-MK2 and PK-15 cells are used because of their proven susceptibility to human as well as to a variety of animal rotaviruses [22, 24]. Generally, there is a good correlation of the results obtained by EM and FA. Other procedures for detecting virus in the feces are the CF test [20], immuno-electrophoresis, immuno-electron microscopy [68] and the solid-phase radioimmunoassay. However these methods, even if they might give comparable results to those of the EM, are not of practical value.

Recently the ELISA (enzyme-linked-immunosorbent-assay) test was proposed for the diagnosis of rotaviral infections. In this method, stable and non-radioactive reagents are

used; moreover, sophisticated equipment is not required and the test can be read with the naked eye or by a colorimeter [69–71]. The method is so sensitive that as little as 20–30 μg of virus/ml can be detected in either cell cultures or in fecal samples [70].

Serological diagnosis of the infection can be accomplished by CF [21], FA [25], SN [24] and IHA [19]. For the IHA test a filtered fecal suspension may be used as antigen [19].

Sanitary measures alone are insufficient to control rotaviral infections because of the large number of virus particles contained in the infected feces. Thus, it appears that immunization of susceptible animals provides the best method for prevention of the disease. Good results can be obtained by the use of colostral antibody. However, the resistance induced by these antibodies does not last more than 5–6 days [72]. Therefore, in order to provide adequate protection in calves it is necessary to stimulate a local immunity at the level of the intestinal mucosa [72].

For this purpose, a live attenuated calf rotavirus vaccine is available. Its administration soon after birth has significantly reduced the incidence and the severity of the disease [73]. On the other hand, cows can be vaccinated with an inactivated vaccine. This method also seems to be successful, probably because of the increased level of colostral and milk antibodies in the intestinal lumen of the newborn [73, 74].

CORONAVIRAL INFECTIONS

The virus

Coronaviruses have been isolated from several animal species and from humans [75]. The viruses are included in the Family of *Coronaviridae,* Genus *Coronavirus* [10, 75].

The genome of the virus consists of single-stranded RNA with a MW $5.5–6.1 \times 10^6$D; 4–6 polypeptides are present, one of which, has a MW $50–60 \times 10^3$D, is associated with the RNA. The virus contains lipids and carbohydrates, the latter being associated with high MW proteine (glycosylated proteins). The virus is sensitive to lipid solvents and to heat but resists heating at 50°C for 60 min in the presence of 1 M $MgCl_2$; it is stable at pH of 3.0. The buoyant density in sucrose is between 1.15 and 1.23 g/ml.

The particles are pleomorphic, they range in diameter from 75 to 160 nm, and are surrounded by an envelope which contains projections known as peplomeres. The peplomeres may be tear-shaped, or petal-shaped, spherical or filamentous. In bovine strains the peplomeres are usually petal-shaped.

The morphogenesis of the virus occurs in the cytoplasmic vesicles of the infected cell.

The virus has two principal antigenic determinants. All members of the family seem to be antigenically related.

Coronaviruses agglutinate red blood cells of the rat, mouse, hamster and guinea pig [76–78].

The bovine strains can be propagated in MDBK, VERO and PK-15 cells. Viral multiplication in these cultures results in CPE characterized by cytoplasmic vacuolization and rounding of the cells. The virus can be cultured also in primary bovine embryo kidney and in intestinal organ explants [79], as well as in bovine embryo kidney (BEK-1) cell line [80], in which it induces formation of syncytia, and reaches a titer of $10^{6.0}$–$10^{7.0}$, median-tissue-culture-infective-dose (TCID). When cultured in the presence of actinomycin D

(0.05 μg/ml), trypsin (20 μg/ml) and DEAE-dextran (25 μg/ml) the yield of the virus increases and CPE is enhanced [79–81].

In cells infected with coronaviruses, tubular formation of 9–10 nm in diameter are seen by EM. These formations most likely correspond to the nucleocapsids of the virus [82].

Bovine coronaviral isolates from the United States and Canada are not pathogenic for newborn mice, hamsters or guinea pigs [83], nor do they replicate in chicken embryo [83]. These strains, however, are readily transmitted to newborn gnotobiotic or colustrum-deprived calves, with diarrhea ensuing [78, 84, 85].

The infection

Spread of the infection occurs via the respiratory tract (avian infectious bronchitis) or the oral route (transmissible gastroenteritis (TGE) of swine and diarrhea of young animals and children [76, 82, 83, 86].

The host range of the virus includes several species of mammals and birds [75].

Recently it was demonstrated that coronavirus infections are quite common in cattle [84, 87] and it seems that (as TGE in swine) the bovine infection is the most important disease caused by coronaviruses in terms of economic losses. In a survey carried out in Nebraska, coronavirus was found in the feces of calves with diarrhea from 19 herds [84]. Virus was detected in similar studies conducted in Belgium [88], the United Kingdom [89], Italy [90–92] and Canada [93]. It is interesting that in Canada, where 134 fecal samples from calves affected with diarrhea were examined, 107 were found to contain rotavirus and/or coronavirus. While 54% of the samples were positive for both viruses, 53 and 14% of them contained coronavirus or rotavirus, respectively. Of the 107 samples, 4 contained, in addition, bovine viral diarrhea (BVD) virus, 2 the bovine infectious rhinotracheitis (IBR) virus and 2 contained enteroviruses.

The disease affects calves from 1–3 weeks of age. It seems to be highly contagious, and is characterized by depression, fever, salivation associated with ulcerative lesions in the oral mucosa, diarrhea, and loss of weight. The feces are fluid and contain mucus and blood. During the diarrhea there is loss of water, sodium and potassium chlorides. Moreover, a state of acidosis occurs as a result of the loss of bicarbonate. Affected calves are hypoglycemic [94]. The animals that survive recover very slowly over a period of several weeks [88].

In experimentally infected calves the gross lesions consist of large amounts of yellowish fluid in the intestinal lumen, and the thinning of the small intestine wall which becomes transparent as a result.

The microscopic changes include atrophy and fusion of the intestinal villi, vacuolization of the epithelial cells, a reduction in the number of columnar cells, enlargement and hypertrophy of the crypts. The ultrastructural changes include swelling of the endoplasmic reticulum, the presence of granular/fibrillar material in the endoplasmic cysternae and paranuclear foci of virus replication in the epithelial infected cells. Viral particles are found in the columnar epithelial cells of the villi and, occasionally, in the fibroblasts and the endothelial cells of the mucosa. These changes are similar to those observed in TGE of pigs and in the coronal infection of dog. However, none of these lesions is considered specific for coronavirus infection [78].

The pathogenesis of the diarrhea in calves caused by coronavirus is quite similar to that described above for rotavirus infection. In the gnotobiotic calves exposed to oral infection, the diarrhea appears after approximately 20 hr incubation. From 42–96 hr post-infection weakness, depression and loss in weight are observed. At this stage neither gross lesions nor histologic changes are detected in the intestine; however, virions and/or viral antigens are seen by FA tests or EM. The infection initially involves the epithelial cells of the anterior section of small intestine, then spreads to the posterior sections. When the diarrhea appears all cells have become infected. As the infection proceeds, the infected cells are lost and are replaced by immature, cuboidal or squamous cells. The persistence of diarrhea is due to the new villous cells which lack digestive enzyme, and also to the impaired absorbing activity of the intestine.

In colostrum-fed calves inoculated at 4–5 days of age the anterior portion of the intestinal tract remained uninfected and the diarrhea was slight. However, in the case of 12 day old colostrum-fed calves which were seropositive for coronavirus antibody, the entire intestinal tract became infected, accompanied by severe diarrhea. In the case of calves inoculated at 4–5 days of age the IgA and the IgM might have been protective. In calves infected when 12 days old, the IgA and the IgM have both disappeared but IgG persists. Therefore it appears that the latter antibody does not protect against coronavirus infection [85].

Once the virus has reached the intestine in orally infected calves, it becomes adsorbed to the microvilli of the susceptible cells whose plasma membrane fuses with the envelope of the virion, hence the nucleocapsids are released in the cysternae of the endoplasmic reticulum.

Replication of the virus results in the production of granulous material in which structures of the viral core are recognized. Because of the presence of this material (viroplasm) the cysternae, as well as the Golgi's bodies, increase in size. The mitochondria undergoes a degenerative process, depriving the cell of its capacity to produce energy [95]. On lysis of the cell the viral particles are released into the intestinal lumen [96].

For diagnosis, the same methods used for other enteric infections in general are applicable to coronaviruses. These methods include EM observation of fecal preparations, isolation of the virus in cell culture, and the FA test carried out directly on the intestinal mucosa. The presence of viral particles with a diameter of 110–180 nm, surrounded by petal-shaped projections of 17–24 nm in length, can be considered as specific for diagnosis of coronavirus infection.

Coronaviruses are extremely fastidious insofar as their isolation in cell culture is concerned, which renders this method of diagnosis of questionable value. In the United Kingdom one strain of coronavirus from the infected intestinal villi of a calf was isolated in bovine kidney cells, but the infectivity of the virus was so low that it could not be readily passed serially. Moreover, this virus isolate does not induce CPE so that its presence in the culture can be detected only by EM or the FA test [97]. On the other hand, a strain of coronavirus was isolated in Denmark in organ cultures of bovine embryonic trachea [97].

The direct FA test can be carried out successfully on infected intestinal villi, since the virus antigen persists for several days after infection, and virus can be detected also at necroscopy.

However, owing to the difficulty of detecting viral particles in all fecal samples from calves with diarrhea, the diagnosis of coronavirus infection should be based on at least two

of the three methods mentioned above, and it should be carried out on a herd basis rather than on a single animal [97].

In order to prevent coronavirus infection the measures for protecting against rotaviruses are generally effective. Good results have been obtained in the control of diarrhea in newborn calves by the oral administration of 5.0 ml of a coronavirus strain culture after 13 serial passages in bovine embryo kidney cells [87]. When such animals were challenged with the virulent virus they did not develop any signs of the disease.

PARVOVIRAL INFECTIONS

The virus

The parvoviruses are included in the family *Parvoviridae*. They contain single-stranded DNA with a MW of $1.5–2.2 \times 10^6$ D and have a complementary base composition with G+C content of 41–53%. Virions possess three polypeptides whereas they lack enzymes, lipids and carbohydrates. The virus has a MW of $5.5–6.2 \times 10^6$ D, a sedimentation coefficient of 110–122 S, and a buoyant density of 1.42 g/ml. Virus infectivity is stable between pH 3.0 and 9.0, at 56°C for 60 min and is ether-resistant. It is inactivated by formalin, betapropiolactone, hydroxylamine, oxidative agents and ultraviolet rays. The viral particle is naked, being unenveloped, it possesses a capsid formed most probably by 32 capsomeres, and a core with dimensions of 14–17 nm. The polypeptides are antigenically distinct but immunological related. The virus induces the formation of FC, SN and hemagglutination inhibiting antibodies; the corresponding antigens are type-specific.

The virus multiplies in the nuclei of the infected cells, where mature virions and empty capsids are seen. Replication of the virus depends on the particular physiologic condition of the cell, and is enhanced during the cell division phase. The virus produces intranuclear inclusions.

The infection

The virus has a wide host range, including cattle and cat, dog, goose, mink, mouse, swine, rabbit and rat, which are susceptible either to natural or experimental infection.

The virus is transmitted to the adult host mechanically by vectors [10] and to the fetus by way of the placenta.

The first parvovirus infection to be described was feline panleukopenia [98]. In 1959 a parvovirus was isolated from a calf with an intestinal infection [99]. Subsequently, enteric infections caused by parvovirus were described in dogs [77, 100], rabbits [101] and in rats [102].

The first bovine parvovirus was isolated in Maryland, followed by the isolation of other strains of the virus from fecal samples of calves with enteric troubles in Colorado, South Dakota, Oregon, Algeria and Japan [98]. On several occasions parvoviruses were isolated from the same animal in association with different viruses, including enteroviruses, coronaviruses and adenoviruses.

Bovine isolates of parvovirus are not defective, they replicate in bovine embryo kidney

cell cultures and in bovine testicle, lung and spleen cells. They are antigenically different from human parvoviruses and from strains of the virus isolated from other animal species. They agglutinate guinea pig and human group O red blood cells.

Parvoviruses were isolated from the feces of calves affected by enteric syndromes and also from fecal samples of healthy calves. The presence of specific antibody in the blood of the calves did not interfere with isolation of the virus from their feces. Virus was isolated also from aborted fetuses and also from the conjunctiva and tonsils of calves with diarrhea [98].

When newborn, colostrum-deprived calves are exposed to parvoviruses by oral or intravenous infection, they develop enteritis associated with the passage of watery, blood stained feces 24–48 hr later. Calves inoculated intravenously with the virus also undergo a significant febrile response (41.0°C).

Antihemagglutinating antibody appears in the blood of calves 5–7 days following the infection.

In experimentally infected calves the virus antigen was detected in several organs and tissue by FA. The antigen is present in the jejunum, ileum, cecum and is concentrated particularly in the crypts of Lieberkuhn's glands. The histologic sections of infected cells show the presence of intranuclear inclusions. Virus can be recovered from the feces, the intestinal mucosa, the mesenteric lymph nodes, the suprarenal glands and the thymus. Virus isolation is readily accomplished in parasynchronized bovine spleen cell cultures, or by means of explant cultures of kidney or testicle cells obtained from infected calves [98].

Because of the possibility that either the calf serum or the fetal bovine serum, which are generally added to tissue culture mediums, may contain antibody to the virus, it is advisable to check the serum prior to their use in parvovirus isolation work. Lamb serum which has an inhibiting hemagglutination titer less than 1:8 is satisfactory [98].

Epidemiological studies revealed that antibody to parvoviruses are wide-spread in cattle. This indicates that a naturally occurring passive immunity in the young animals might reduce the incidence of the infection in the herds. At the present it is still unknown whether parvovirus infections cause a significant economic loss in cattle. It seems likely that the herds most heavily exposed to the disease are those in which the infection occurs for the first time, when the herds are devoid of specific parvovirus antibody.

OTHER DIARRHEAL VIRUSES

Parvovirus-like agents

Particles approximately 22–27 nm in diameter have frequently been observed in feces of clinically ill and clinically normal children [103], and of a variety of animals [104]. However, while these particles, which includes six different isolates (Norwalk, Hawaii, MC, W, Ditchling, Crockle), have been associated with enteric infections of children [105], the role, if any, that they play in the enteric diseases of animals [104] is still unknown.

Astroviruses

The virions of astroviruses are spherical, approximately 29 nm in diameter and have a star-like structure. They have frequently been found in the feces of children and were

reported also in cattle [103]. The bovine isolate was serially passaged in gnotobiotic calves and found to be antigenically distinct from the human strains.

Calicivirus

The caliciviruses have been detected in feces of children, and one strain causing diarrhea was isolated from calves [103].

"Fuzzy-Wuzzys"

These viruses which are often associated with adenoviruses, have a diameter of about 28 nm. The proposed term for these viruses (fuzzy-wuzzy) [103] derives from their shape which is villous and indistinct, whereas their characteristics have not yet been precisely described. Fuzzy-wuzzy agents have been found in human feces [103].

CONCLUSIONS

Elucidation of the epidemiology of the viral diarrheas which affect young animals is still incomplete so there is little information on the manner in which outbreaks of the disease occur. Thus, it is unknown how the infection can reappear in the same herd after a considerable length of time following the previous outbreak. Of course the possibility that the infection could spread with the introduction of new animals in the herd must be considered. However, aside from that possibility, it is conceivable that virus or viruses associated with diarrhea remain latent in the recovered animals and subsequent stress could reactivate the virus, causing the disease to reappear. Furthermore, the possibility for interspecies infection is an additional point worthy of consideration in attempts to understand the mode of spread of the disease. In this connection it has been reported that young pigs might represent a potential source of rotaviral infection for calves [43].

Very little is known about the aftermath in calves after recovery from a viral enteric infection except that they undergo a significant decrease in their normal growth rate. The serious changes which affect the mucosa of small intestine, principally the virus induced inhibition of the absorptive mechanism of the epithelial cells, could result in the loss by the calf of some essential nutritional factors which are required for a normal growth.

Owing to the differences in virulence which exist among strains of rotaviruses, studies on the pathogenesis of the infection should be pursued. Such studies might provide information which could be of value in correcting the altered physiology induced by the disease [106].

The selective nature of viral damage in the intestine has to be considered carefully. In the case of TGE infection of pigs the villous epithelium is destroyed, while crypt epithelium is spared and regenerates the villi. In contrast, the rotaviruses infect the most mature absorptive cells which are situated at the median and at the apical parts of the villi. On the other hand, the parvoviruses have a predilection for the immature proliferative cells of the crypts, so that as the disease progresses, the crypts become aplastic and fail to replace the villous absorptive cells, causing the atrophy of the villi to occur [107].

A better knowledge also is needed of the antigenicity of the enteropathogenic viruses in order to understand the epidemiology of the infections, and to develop effective vaccines against neonatal diarrheas.

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