Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company’s public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Antigenic Sites

Antibodies are the first line of defense by the immune system against a viral infection. The epitopes combined with the neutralizing antibodies are mapped on a few isolated locations on the surface of viral proteins. The structure of human rhinovirus complexed with Fab fragments showed that the antibody makes contact with an area about 6 nm² and that the epitope spans different discontinuous polypeptides. Therefore, an effective vaccine usually needs to include a complete viral protein or a large fragment. The binding of the antibodies does not significantly change the structure of the antigen. The exact mechanism by which antibodies neutralize antigens is dependent upon the binding site and processes of the virus replication.

Antiviral Agents

Viral infectious diseases can be cured if an agent can be administered to stop viral infection. Such agents have been synthesized and shown to bind to the capsid of rhinovirus in the crystal structure. The compounds were inserted into the hydrophobic pocket within the β-barrel of the major capsid protein VP1. Binding of the compounds stops uncoating of the virion and the receptor binding, which resulted in the failure of releasing the viral RNA into the cytoplasm. These compounds inhibit infections of several other RNA viruses and may be effective against other viruses after modification since the β-barrel structure exists in many viruses.

The most successful antiviral drugs are the HIV protease inhibitors which are developed based on the atomic structure of the protease. Through iterative cycles of computer modeling, chemical synthesis and structural studies of the protein-inhibitor complexes, a panel of clinical effective drugs has been brought to the market and has shown great benefits to patients. Inhibitors of influenza virus neuraminidase have also been developed by the same method and marketed as antiviral drugs.

See also: Assembly of Viruses: Enveloped Particles; Theiler’s Virus.

Further Reading

Knipe DM, Howley PM, Griffin DE, et al. (eds.) (2002) Fields Virology, 4th edn. Philadelphia, PA: Lippincott Williams and Wilkins.
Rossmann MG and Johnson JE (1989) Icosahedral RNA virus structure. Annual Review of Biochemistry 58: 533–573.
Schneemann A (2006) The structural and functional role of RNA in icosahedral virus assembly. Annual Review of Microbiology 60: 51–67.

Astroviruses

L Moser and S Schultz-Cherry, University of Wisconsin – Madison, Madison, WI, USA
© 2008 Elsevier Ltd. All rights reserved.

Glossary

Enterocytes Epithelial cells lining the intestines.
Intussusception Obstruction of the intestine.
Interstitial nephritis Inflammation of the kidney.
Poult Young turkey.
Villi Finger-like intestinal projections lined with enterocytes.

Introduction

Astroviruses are enteric viruses first identified in the feces of children with diarrhea. Detection was originally based on a five- to six-pointed star morphology of virions by electron microscopy (EM). However, only about 10% of viral particles display these structures; the remaining 90% of particles have a smooth surface and a size similar to other small, round-structured viruses like picornaviruses and caliciviruses. Thus, accurate diagnostics were difficult to obtain and the true prevalence of astrovirus within a population was difficult to assess. Development of much more sensitive detection techniques like real time reverse transcription-polymerase chain reaction (RT-PCR), cell culture RT-PCR, and astrovirus-specific enzyme-linked immunosorbent assays (ELISAs) have made detection more accurate and specific, even allowing diagnosis of specific serotypes. Utilizing these techniques, astroviruses have been found in approximately 3–8% of children with diarrhea. Astroviruses can also be isolated in a subset of asymptomatic individuals, suggesting that a proportion of infected individuals shed the virus asymptptomatically or for some time after the
resolution of other symptoms of infection. Asymptomatic carriers may be a major reservoir for astroviruses in the environment and could contribute to dissemination of the virus.

The release of astroviruses into the environment is a concern due to the extreme stability of the virus. Astroviruses are resistant to inactivation by alcohols (propanol, butane, and ethanol), bleach, a variety of detergents, heat treatment including 50°C for an hour or 60°C for 5 min, and UV treatment up to 100 mJ cm⁻². Human astroviruses are known to survive up to 90 days in both marine and tap water, with survival potential increasing in colder temperatures. Studies have described the isolation of infectious virus from water treatment facilities. Furthermore, astroviruses can be concentrated by filter-feeding shellfish like oysters and mussels in marine environments. Astroviruses are transmitted fecal–orally, and contaminated food and water have been linked to astrovirus outbreaks.

**History and Classification**

Astroviruses were originally observed by Appleton and Higgins in 1975 as a small round virus in stools. Later that year, Madeley and Cosgrove identified the virus in association with diarrhea in children and bestowed the name astrovirus (from the Greek *astron*, meaning star) for the star-like morphology of a proportion of viral particles seen by EM (Figure 1(a)). Because of genomic similarities, astroviruses were originally thought to belong to either the families Picornaviridae or Caliciviridae. However, the lack of a helicase and use of a frameshifting event during replication (discussed below) distinguish astroviruses so completely that, in 1993, the International Committee on Taxonomy of Viruses (ICTV) classified astroviruses as a unique family, Astioviridae, composed of a single genus *Astrovirus*. Continued investigation into newly discovered astroviruses led to the division of the family into two genera, *Mamastrovirus* and *Avastrovirus*, by the ICTV in 2005. ICTV nomenclature abbreviates astrovirus AstV, with a single-letter abbreviation for the species type (i.e., human astrovirus; HAstV; turkey astrovirus; TAstV, etc.). Successive, serologically distinct isolates of astroviruses are named sequentially within that species (i.e., HAstV-1 through HAstV-8).

**Epidemiology**

**Humans**

Astroviruses have been detected throughout the world. While the exact incidences of infection vary from study to study, community-acquired astroviruses are found in 3–6% of children with infectious gastroenteritis. In some developing countries, infection rates as high as 20% have been observed. In many cases, astroviruses are the second most commonly detected viral pathogen in young children after rotavirus. Astrovirus infections are identified in up to 2% of asymptomatic individuals. These data may underrepresent actual astrovirus infections, as studies generally survey individuals visiting medical care centers. Because astrovirus disease is generally mild in humans (see the section discussing pathogenesis), hospital cases may represent only a slight proportion of actual infections in the community. In support of this, serological studies have demonstrated that up to 90% of children have been exposed to at least one strain of astrovirus by age 9.
Eight serotypes of human astrovirus have been identified to date, with all eight circulating within the global population to various levels. HAstV-1 is by far the most prevalent serotype, comprising 25–100% of astroviruses in a region, and the most prevalent reactivity of antibodies detected, although serological surveys of all serotypes have not been undertaken. HAstV-6, -7, and -8 are the least frequently detected, although three to four serotypes of HAstV are often detected in a region at any given time. The differing prevalence of serotypes could be a reflection of severity; perhaps HAstV-1 infection results in a higher frequency of hospital visits than other serotypes and is therefore overrepresented in hospital-based epidemiological studies. Alternatively, serotypes may be restricted by region. For example, one Mexican study identified HAstV-1 as the predominant serotype throughout the country, but HAstV-3 and -8 were prominent in select regions.

Viral infection occurs with equal frequency in boys and girls and predominantly in children under the age of 2. Infection is not restricted to young children, however, and has been noted in individuals of all ages, including immunocompetent adults and the elderly. Immunodeficient individuals, particularly those that are HIV-positive, appear to be at an increased risk of astrovirus infection.

Astrovirus infection occurs year-round, but with the highest frequency during the autumn and early winter months. In tropical climates, infection correlates with the rainy season. These seasonal correlations likely reflect the indoor confinement of the population as well as the increased stability of astroviruses in cold, damp conditions. Astrovirus outbreaks have also been associated with high-density environments, including childcare centers, primary and junior high schools, military recruiting centers, elderly care centers, and swimming pools. Astrovirus as a cause of hospital-acquired viral diarrhea in young children is second only to rotavirus, occurring at rates of 4.5–6%, and, in some studies, surpasses rotavirus in rates of nosocomial infections.

Interestingly, astrovirus infection occurs quite frequently (up to 50%) as a co-infection with other enteric pathogens. The most frequent co-pathogens are noroviruses and rotaviruses, but infections with adenoviruses, parasites, and enteric bacteria are often detected as well. The importance of this in humans is not entirely clear. In a study specifically examining co-infections, astrovirus co-infection with rotavirus increased the duration of diarrhea and vomiting over either virus alone, although whether this difference was statistically significant is unknown.

**Animals**

Most animals are not routinely screened for astrovirus infection, so our knowledge of the prevalence of infection is limited to surveillance studies. Astroviruses have been found in association with most animals examined, although the effect of infection varies with species (see below). While astroviruses were originally identified in humans, they have since been identified in both mammalian and avian species, including rabbits, mice, calves, sheep, piglets, dogs, red-tailed deer, kittens, mink, turkeys, ducks, chicken, and guinea fowl. At least three serotypes of bovine astroviruses are postulated to exist based on distinct neutralizing antibodies (one in the United States and two in the United Kingdom). In addition, astroviruses have been isolated from mink across Scandinavia, and serological studies have demonstrated that astroviruses were prevalent in chicken flocks in the 1980s as well as in 2001. Interestingly, two very different manifestations of chicken astrovirus infection have been described (see the section on pathogenesis), suggesting that distinct chicken astroviruses may circulate; however, this is yet to be proven. The best epidemiologically characterized animal astroviruses are the turkey astroviruses. Surveillance of turkey flocks in the 1980s isolated astrovirus from 78% of diseased flocks, but only 29% of normal flocks. Astroviruses were the first pathogen detected in many flocks and were most commonly detected in birds less than 4 weeks of age. Similar to human infections, turkey astrovirus was frequently isolated with other pathogens, most commonly rotavirus-like viruses. The early age of infection and the prevalence of co-infections led one group to postulate that astrovirus infection may predispose birds to infection by other viruses.

**Virus Propagation**

Attempts at *in vitro* propagation of astroviruses have been met with varying degrees of success. The most successful techniques utilize cultured cells from the host species and provide exogenous trypsin in the culture. Successful propagation of human astroviruses was originally achieved by repeated passage through primary human embryonic kidney cells; it was later discovered that direct passage through the human intestinal cell line Caco-2 would also yield infectious virus. Propagation of porcine, bovine, and chicken astroviruses has been successful in their respective host cells *in vitro*. However, many astroviruses still have not been adapted to propagation *in vitro* for unknown reasons, while others lose infectivity with subsequent passages and therefore cannot be maintained continuously. This problem has been circumvented in some systems by passing the virus through an animal system, as is the case for the turkey astrovirus, in which highly concentrated virus can be obtained from infected turkey embryos *in ovo*. 
Molecular Virology and Protein Expression

Astroviruses contain one copy of positive-sense, single-stranded RNA. The genome is approximately 6.8 kb long and contains three open reading frames (ORFs), ORF1a, -1b, and -2, as well as 5' and 3' untranslated regions (UTRs) (Figure 2). The RNA is polyadenylated, but lacks a 5' cap structure. The 5' and 3' UTRs are highly conserved and are believed to contain signals important for genome replication.

Astroviruses initiate infection by binding to an unknown receptor and entering the cell via receptor-mediated endocytosis. The plus-strand genome is released into the cytoplasm by unknown mechanisms and ORF1a and -1b are immediately translated by the host machinery. ORF1a is 2.8 kb and encodes a polypeptide of approximately 110 kDa. This polypeptide contains a variety of conserved motifs, including several putative transmembrane domains, a bipartite nuclear localization sequence (NLS), and a serine protease motif. The translated polypeptide is cleaved by both cellular protease(s) and the viral protease into at least five peptides. The actual function of each protein remains largely unknown. The transmembrane domains may localize to the endoplasmic reticulum (ER) membrane to facilitate replication, as all plus-strand RNA viruses have been shown to replicate in association with a membrane. One peptide, NSP1a/4, colocalizes with the viral RNA at the ER membrane; mutations in NSP1a correlate with increased viral titers in vitro and in vivo, suggesting a role for this protein in viral replication. The role for the NLS remains unclear; some reports suggest viral antigen is observed in the nucleus, while others find that it is excluded.

The second reading frame, ORF1b, overlaps ORF1a by 70 nucleotides and has no detectable start codon. Intensive research has determined that ORF1b is translated by a frameshift into the −1 frame. This frameshifting event is unique among plus-strand animal RNA viruses and requires a highly conserved shift heptamer sequence (A_A_C) as well as a downstream hairpin structure. This event, which occurs with frequencies up to 25% in cells, results in an ORF1a/1b fusion peptide. Cleavage near the 1a/1b border releases the ORF1b gene product: the viral RNA-dependent RNA polymerase (RdRp). Astrovirus polymerase is a supergroup I RdRp, a group which generally utilizes a VPg to initiate transcription. Although a VPg is postulated to exist and a putative VPg genomic linkage site has been identified, its existence is yet to be empirically proven.

Expression of the RdRp results in production of a minus-strand viral template. This generates multiple copies of the plus-strand genome as well as a polyadenylated subgenomic RNA (sgRNA) containing short 5' and 3' UTRs and ORF2. ORF2 is in the 0 frame and overlaps ORF1b slightly (four nucleotides) in human astroviruses. Production of the capsid protein from a sgRNA not only temporally restricts capsid production to later in the viral replication cycle, but also allows for massive capsid protein expression; it is estimated that sgRNA is produced in tenfold excess of the viral genome by 12 h post infection (hpi). The sgRNA is about 2.4 kb and encodes the single structural protein of approximately 87 kDa. This peptide is cleaved by an intracellular protease to approximately 79 kDa; mutational analyses suggest that this 8 kDa stretch is required for efficient expression of the capsid protein. Individual capsid proteins multimerize spontaneously to form icosahedral structures of about 32 nm (Figure 1(b)).
Positive-sense genomes are packaged into these viral-like particles (VLPs), possibly through interactions with the first 70 amino acids of the capsid protein. The virions are released by an unknown mechanism, which may involve cellular caspases, after which the capsid undergoes an extracellular trypsin-mediated maturational cleavage. This increases infectivity up to $10^5$ fold, condenses the virion to approximately 28 nm, and transforms the 79 kDa capsid protein into at least three smaller peptides of approximately 34, 29, and 26 kDa. Computational predictions suggest that VP34 may comprise the core of the virion while VP29 and VP26 form spike-like projections that may be important for viral tropism and receptor binding. This is corroborated by studies suggesting that VP26 is only loosely associated with the virion. These spikes are also thought to be responsible for the star morphology visible by EM (Figure 1(a)).

**Evolution**

Examination of nucleotide changes and nonsynonymous amino acid changes from the whole genome and across species suggests that an ancient divergence between avian and mammalian astroviruses occurred approximately 310 million years ago. Mammalian astroviruses split more recently into two distinct clades: human astroviruses and feline/mink-associated astroviruses. Phylogenetic clustering of the human astroviruses together argues against continual human–animal interspecies transmission. It is hypothesized that at least two interspecies transmission events (avian to porcine, porcine to feline) led to the current division of viruses. Further comparison of synonymous mutations by codon usage generates an astrovirus evolutionary pattern which mirrors the evolution of respective hosts, suggesting that recent evolution of the virus may have been in adaptation to the host. As RNA viruses, astroviruses are expected to undergo frequent genetic changes. However, nucleotide changes occur at rates of approximately 5% in human viruses over time, despite the co-circulation of multiple serotypes within a region. Nucleotide and amino acid comparisons of ORF1a of human astroviruses demonstrate two distinct lineages, known as genogroup I (HAstV-1 to -5) and genogroup II (HAstV-6 and -7). Comparisons of ORF1b or ORF2 lack these distinct groups, leading investigators to postulate that a recombination event at the ORF1a/1b junction occurred before HAstV-6 or -7 diverged.

**Clinical Features, Pathology, and Pathogenesis**

**Mammalian Astroviruses**

Astrovirus infection in mammals presents clinically as gastroenteritis. Disease has been most closely studied in humans and, in volunteer studies, astrovirus-infected individuals develop diarrhea, the most prominent symptom, as well as vomiting, nausea, anxiety, headache, malaise, abdominal discomfort, and fever. Onset of symptoms at 2–3 days post infection (dpi) correlates with shedding of the virus in feces, although shedding can continue after resolution of other symptoms. Astrovirus infection has also been associated with intussusception, although a causative role has not been established.

The earliest studies of astrovirus pathogenesis utilized gnotobiotic sheep and calves as models. In calves, astrovirus infection was localized to the dome epithelial cells overlying Peyer’s patches. These cells appeared flat or rounded and released cells were identified in the intestinal lumen. Astrovirus infection in calves was shown to be specifically targeted to M cells and led to the sloughing of necrotic M cells into the intestinal lumen. Enterocytes were never observed to be infected. Specific tropism of the virus for immune cells suggests that astrovirus may have an immunomodulatory role in calves. While the virus replicated in these animals and could be detected in their feces, the calves displayed no clinical signs. In most bovine studies, viral infection is asymptomatic, although changes in the feces from solid and brown to soft and yellow were noted in one study. Mild villus atrophy and slight changes in villus-to-crypt ratios have been noted but no changes in xylose absorption were observed. Despite the lack of symptoms, viral shedding continued until the termination of the experiment.

Studies in sheep have shed more light on histological changes associated with infection. Astrovirus-infected sheep developed a transient diarrhea as early as 2 dpi, but virus was detected at early as 14 hpi and initially confined to the luminal tips of the intestinal villi. By 23 hpi, virus was observed coating the microvilli and infection had spread to the apical two-thirds of the villi. This correlated with sloughing of degenerate cells from the apical portion of the villi, which continued through 38 hpi. At this time, villus blunting was apparent in the ileum and midgut. Furthermore, normal epithelial cells lining the villi were replaced with immature, cuboidal cells reminiscent of crypt cells. Neither these immature cells nor crypt cells were ever observed to be infected, suggesting that only mature enterocytes are susceptible to infection. By 5 dpi, viral infection had cleared and intestinal histology had returned to normal.

Volunteer studies in humans have not explored the underlying causes of astrovirus pathogenesis; our knowledge is therefore limited to intestinal biopsies taken for other reasons, but generally support the observations described above. In a biopsy from a child shedding large quantities of astrovirus, slight histological changes, including mild villous blunting and irregular epithelial cells, were observed. Infection increased distally through the small intestine. Similarly to animal models, astrovirus
infection was restricted to the apical two-thirds of intestinal villi and could be identified in infected cells.

**Avian Astroviruses**

In avian species, astrovirus infection has a much broader range of disease than in mammals. While astrovirus does cause gastroenteritis in turkeys and chickens, it can also cause nephritis in chickens and a severe, often fatal, hepatitis in young ducklings.

Turkey astrovirus was the first discovered avian astrovirus and remains the best characterized in terms of pathogenesis, due in part to the development of the turkey as a small animal model. In these animals, virus could be detected from 1 to 12 dpi in the intestines. Viral replication was limited to the enterocytes on the apical portion of the villi, but the virus could be detected throughout the body, including the blood. The development of viremia is rare among enteric viruses and its function remains unclear. Infected turkeys developed a yellow, frothy, gas-filled diarrhea from 1 to 12 dpi. Diarrhea occasionally contained undigested food, but never blood. The intestines of infected birds became thin walled, flaccid, and distended. Despite these changes, histological examination suggested that only mild changes occur during infection. A mild crypt hyperplasia and shortening of the villi were noted from 4 or 5 to 9 dpi, and single degrading enterocytes could be identified. However, TUNEL staining suggested that the amount of cell death in infected intestines is similar to control birds. D-xylene absorption, a measure of intestinal absorption, was significantly decreased from 2 to 5 dpi in one study and up to 13 dpi in another. This effect was exacerbated in the presence of another enteric pathogen, turkey coronavirus. Astrovirus infection also caused a significant growth depression in turkey poulets by 5 dpi; infected birds never recovered from this, leading to flock unevenness. Infected birds also demonstrated a transient (3–9 dpi) reduction of the thymus, which returned to normal by 12 dpi.

Avian infection by astroviruses can present with nonenteric symptoms as well. Infection of ducklings with duck astrovirus causes a severe hepatitis. Infected birds develop liver hemorrhage, swollen kidneys, and hepatocyte necrosis. On farms, infection leads to mortality rates of 10–25% in adult (4–6-week-old) ducks, but can reach 50% in ducklings under 14 days of age. In chickens, infection with the astrovirus avian nephritis virus (ANV) results in discoloration of the kidney, development of renal lesions, and interstitial nephritis by 3 dpi. Pathogenesis is age dependent, with 1-day-old chicks the most susceptible and adult birds the least. ANV infection can result in mortality rates of up to 33%, although rates appear to be strain specific.

**Immune Response**

The immunological response to astrovirus infection is poorly defined; however, observations in humans and animal models suggest that both the adaptive and innate responses play important roles in controlling and eliminating the virus.

The humoral immune response likely plays a major role in astrovirus immunity. The biphasic infection pattern of young children and the elderly suggests that antibodies are protective during the middle of life. Indeed, serological studies have indicated that approximately 50% of neonates have maternally acquired antibody to HAstV, which wane by 4–6 months of age. Children then acquire anti-HAstV antibodies rapidly due to astrovirus exposure. By the age of 9, up to 90% of the population has been exposed to HAstV-1. Furthermore, volunteer experiments demonstrate that astrovirus exposure generally leads to an increase in anti-astrovirus antibody titer. While astrovirus antibodies protected individuals from symptoms associated with infection, virus was identified in the feces, suggesting that such antibodies do not necessarily prevent viral replication. Additionally, immunoglobulin treatment has been attempted as a treatment for severe or chronic astrovirus infection. The results have been mixed and difficult to interpret, as the presence of astrovirus-specific antibodies in the immunoglobulin treatment was not always confirmed.

Cellular immunity may also play a role in controlling and/or preventing astrovirus infection. Studies have demonstrated that most individuals possess HLA-restricted, astrovirus-specific T cells. When stimulated with astrovirus in vitro, these cells produce tumor necrosis factor, interferon gamma, and occasionally interleukin (IL)-5 but not IL-2 or IL-4. These cytokines are typical of the T-helper-type response thought to be important in controlling viral infections. Individuals deficient in T and B-cell functions are unable to control infection, shedding virus to very high titers (≥10^14 particles ml^-1) and for extended periods of time (up to 18 months), further supporting the importance of cellular immunity.

Experiments in a turkey model demonstrate that the adaptive response is not the only important immunological response. In this model, no increase in T cells (CD4^+ or CD8^+) could be demonstrated after TAstV-2 infection. Moreover, while infected turkeys produced a slight increase in antibody production, these antibodies were not neutralizing and did not prevent against future infection. However, it was noted that macrophages from TAstV-2 infected turkeys produced significantly higher levels of nitric oxide (NO) both in vivo and upon stimulation ex vivo. Inhibition of NO in vivo led to a significant increase in viral production, while addition of exogenous
NO decreased viral production to below the detection limit, suggesting that NO is an important factor in controlling astrovirus infection. The importance of macrophages and their role in astrovirus infection has been corroborated by observations in astrovirus-infected lambs, where EM showed virions within macrophages. Furthermore, it is possible that astroviruses have a mechanism to combat this response, as macrophages in astrovirus-infected turkeys demonstrate a reduced ability to phagocytose.

Treatment, Prevention, and Control

Because astrovirus infection is generally mild and self-limiting in humans, treatment is generally restricted to fluid rehydration therapy. This can often be accomplished at home; thus, hospital admittance is rare. No vaccine is yet available for humans, and as noted above, immunoglobulin treatment for immunocompromised individuals has been met with varying degrees of success. Additionally, no treatment for astrovirus-infected animals exists. The best solution, therefore, is prevention of transmission, which is best done in humans by conscientious hand and food washing. The stability of astroviruses and their resistance to inactivation make them difficult to eliminate after introduction. This is a significant problem in hospitals, where individuals are generally immunocompromised and therefore more susceptible to infection. One outbreak in a bone marrow transplant ward prompted the hospital to scrub the entire ward with warm, soapy water. However, surveillance of the subsequent inhabitants demonstrated fecal shedding of astroviruses, underscoring the difficulty in removing the virus. This is also a significant problem in commercial farming, where astrovirus infection of animals significantly decreases productivity. Its introduction and maintenance in this environment can mean drastic financial losses. In each of these environments, early detection and thorough disinfection are keys to limiting transmission and controlling infection.

See also: Caliciviruses; Enteric Viruses; History of Virology; Vertebrate Viruses; Picornaviruses: Molecular Biology; Replication of Viruses; Picornaviruses: Molecular Biology; Replication of Viruses; Rotaviruses; Viral Pathogenesis; Virus Particle Structure: Nonenveloped Viruses.

Further Reading

Koci MD (2005) Immunity and resistance to astrovirus infection. Viral Immunology 18: 11–16.
Koci MD and Schultz-Cherry S (2002) Avian astroviruses. Avian Pathology 31: 213–227.
Matsui SM and Greenberg HB (1996) Astroviruses. In: Fields BN, Knipe DM, and Howley PM (eds.) Fields Virology, 3rd edn., pp. 875–893. Philadelphia: Lippincott-Raven.
Monroe SS, Carter MJ, Herrmann JE, Kurtz JB, and Matsui SM (1995) Astroviridae. In: Murphy FA, Fauquet CM, Bishop DHL, et al. (eds.) Virus Taxonomy: Sixth Report of the International Committee on Taxonomy of Viruses, pp. 364–367. Vienna: Springer.
Monroe SS, Jiang B, Stine SE, Koopmans M, and Glass RI (1993) Subgenomic RNA sequence of human astrovirus supports classification of Astroviridae as a new family of RNA viruses. Journal of Virology 67: 3611–3614.