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Murine Astroviruses

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Astroviruses are small, non-enveloped RNA viruses that have been identified in humans and a variety of animal species (including cattle, pig, sheep, dog, cat, mouse, bat, chicken, duck, turkey). Astroviruses isolated from mammals belong to the genus *Mamastrovirus*, those isolated from birds belong to the genus *Avastrovirus*. These viruses are an important cause of acute gastroenteritis. Extra-intestinal manifestations include severe to fatal hepatitis in ducklings, nephritis in chickens and encephalitis in minks (shaking mink syndrome), cattle and immunocompromised humans. Human astroviruses primarily cause enteric disease in young children and immunocompromised patients. Transmission occurs via the faecal-oral route or via contaminated food and drinking water.

Murine astroviruses (MuAstV) were first observed in 1985 in the gut contents of laboratory mice by electron microscopy (1). They were then detected by using molecular biological techniques in the faeces of a wild house mouse (*Mus musculus*) in 2011 (2) and subsequently in laboratory mice, too (3-6). In the study by Yokoyama et al. (4), astroviruses were detected in the intestinal tract and faeces of immunodeficient and immunocompetent mice as well as in the liver, kidney and spleen of immunodeficient *Rag1-/-* mice by RT-PCR. Clinical symptoms were not observed. The duration of infection varies depending on the virus strain (7) and on the mouse strain; infection may persist particularly in immunodeficient mice (e.g. *interferon (Ifn)-α receptor-/-* mice) (6, 7).

Infections with MuAstV can be diagnosed directly by RT-PCR on faecal or environmental samples (e.g. cage dust, IVC exhaust air dust) and indirectly by serology (8-10). Transmission of MuAstV to sentinel animals by used bedding is effective (8). Surveys in the US, Japan and Germany indicate a wide distribution of MuAstV infections in mice from research institutes and commercial breeders (5, 9). Schmidt et al. (9) found MuAstV-specific antibodies in 38% of 661 serum samples of mice from 15 different countries.

The impact of MuAstV on animal experiments remains to be evaluated. While Marvin et al. (6) show that MuAstV may increase intestinal barrier permeability, no impact of MuAstV on the disease phenotype could be observed in a mouse model of inflammatory bowel disease (11). Of note, a recent study (12) demonstrates that MuAstV can complement adaptive immunodeficiency in *Rag2-/-Il2rg-/-* mice to protect against murine norovirus and rotavirus infections. This protection corresponded with the presence of a specific strain of MuAstV in the gut and required IFN-λ signalling in gut epithelial cells. Hence, other immunodeficient models could also be affected by virus-mediated altered IFN-λ responses, which might alter experimental outcomes (e.g. in infection studies or transplantation studies).

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