Viral gastroenteritis is the single most important cause of diarrhea in infancy world-wide and accounts for enormous morbidity and mortality in children in both the developing and developed worlds. Our laboratory is interested in the pathogenesis of viral gastroenteritis, specifically in intestinal factors which are critical to the outcome of rotavirus infection which range from asymptomatic viral shedding to severe clinical disease. Previous observations have shown that rotavirus has both a narrowly defined tissue tropism, essentially mature enterocytes of the intestinal villus tip, and a fairly narrow host range restriction, causing disease mainly in suckling animals and usually only with viral strains derived from the same host species. We believe that local gastrointestinal factors determine the outcome of rotavirus infection. Specific studies include:
1) Cell receptors for rotavirus are being identified and characterized by a variety of biochemical and immunological approaches.

2) Rotavirus penetration of target cell membranes. Work from our lab has shown that this is a critical step in determining host cell susceptibility to the virus.

3) Role of enteric secretions on rotavirus pathogenesis. Rotavirus requires exogenous trypsin for replication. We are studying the effects of gastrointestinal proteases and acid secretion on rotavirus pathogenesis in vitro and in vivo.

4) Role of intestinal mucins as a defense mechanism against rotavirus.

5) Mucosal immunity and protection from enteric viral pathogens.

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