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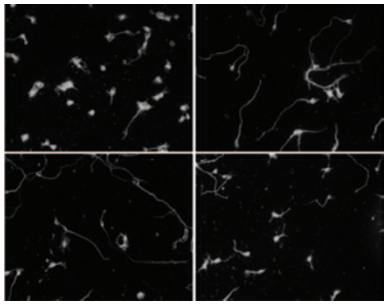
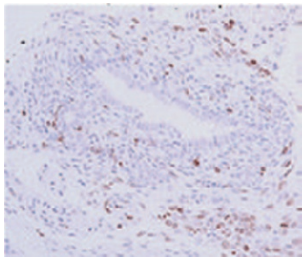
Chlorhexidine inhibits neurite outgrowth



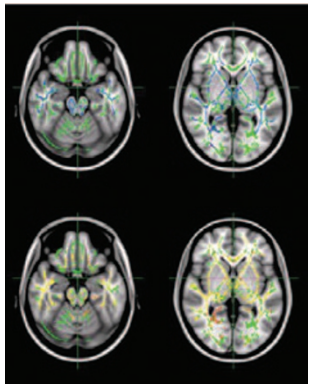
Milstone and colleagues assessed the potential neurotoxicity of chlorhexidine on the developing central nervous system using a well-established *in vitro* model of neurite outgrowth. It was found that chlorhexidine at concentrations detected in the blood following topical application in preterm infants specifically inhibited L1-mediated neurite outgrowth of cerebellar granule neurons. **See page 8**

Cholangitis in Reo-2-infected mice



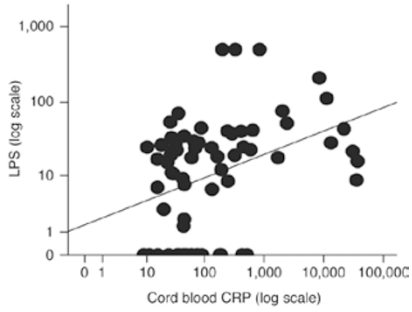
Reovirus is a proposed cause of infantile biliary atresia, but mechanistic insight regarding Reo-2 as a potential cholangiotropic virus is lacking. Nakashima and colleagues analyzed lesions of bile ducts in newborn DBA/1J mice infected with Reo-2 and found that Reo-2 infection induced T-helper cell type 1–dependent injury to bile ducts in weaning mice. **See page 29**

Pediatric diabetic ketoacidosis



The pathophysiology that results in cerebral edema in pediatric diabetic ketoacidosis (DKA) is unknown. To investigate changes in white matter microstructure in this disease, Dervan *et al*. measured diffusion tensor imaging (DTI) parameters in children with DKA at two time points during treatment. Their findings support an association between clinical illness and DTI markers of microstructural change in white matter. **See page 62**

Lipopolysaccharide and inflammation



Lipopolysaccharide (LPS), an endotoxin of Gram-negative bacteria, has been implicated as a factor triggering preterm labor and systemic complications. Martinez-Lopez *et al*. explored LPS levels in the cord blood of term and preterm infants. The findings suggest that LPS is indeed associated with both preterm labor and inflammation. **See page 67**

Body composition and asthma

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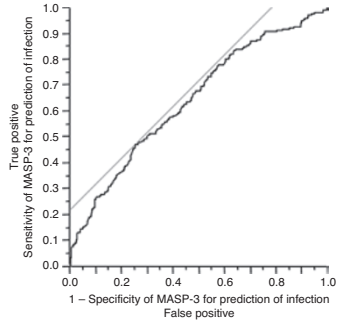
Jensen and coauthors investigated whether BMI *z*-score and body composition were associated with lung function in asthmatic children. Clinical assessment of 48 asthmatic children indicated that lean mass, but not fat mass, is associated with respiratory function in children with asthma. **See page 93**

Lectin pathway



Deficiencies within the innate immunity lectin pathway of complement activation have been implicated in a child's vulnerability to infections, but their role during critical illness remains unclear. Ingels and coinvestigators studied protein levels in 130 healthy children and 700 critically ill children upon admission to an intensive-care unit (ICU). Low levels of MASP-3 (mannose-binding lectin–associated serine protease 3) were independently associated with both subsequent acquisition of infection and prolonged ICU stay in critically ill children. **See page 99**