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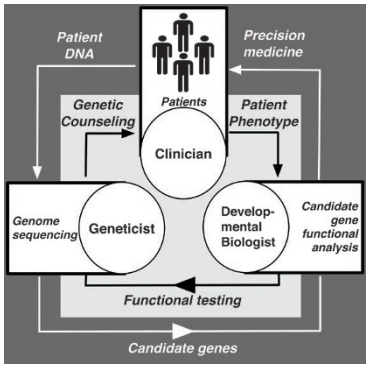
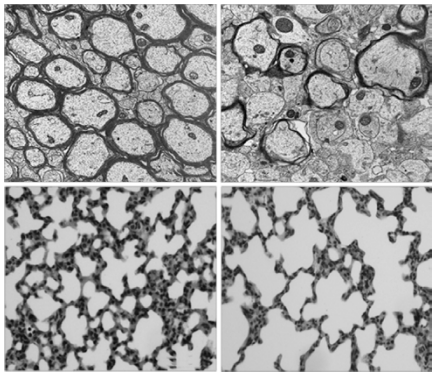
Genetic causes of birth defects



Emerging genomic sequencing technologies provide the capacity to identify genetic variants, including those that cause birth defects. In their review, Khokha and colleagues outline the need for a birth defect genomics initiative and conclude that it must be a trans–National Institutes of Health effort that spans disciplines. **See page 282**

Mitochondria and diseases of prematurity



Some organs in premature infants are not developed enough at birth to meet the challenges of extrauterine life. Ten's integrated mechanism review introduces the hypothesis that mitochondrial bioenergetic dysfunction is a fundamental mechanism of organ maturation failure in premature infants. He suggests that experimental prevention or attenuation of white matter injury and bronchopulmonary dysplasia manifestation via preservation of mitochondrial function will serve as therapeutic proof of the hypothesis and may emerge as a novel therapeutic strategy against diseases of postnatal developmental failure in premature neonates. **See page 286**

Infection and hearing screen failure

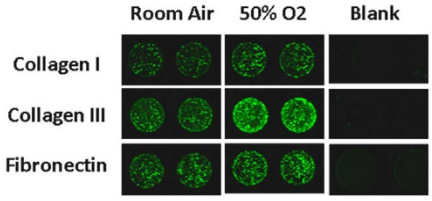
Jung and coauthors aimed to determine whether intra-amniotic infection and elevated proinflammatory cytokine levels in amniotic fluid (AF) are associated with newborn infants' failing the newborn hearing screen (NHS) test. In a retrospective cohort study of premature singletons born preterm at ≤32 weeks, AF obtained through amniocentesis was cultured and interleukin-6 (IL-6) and -8 levels determined. The results appear to demonstrate that the presence of intra-amniotic infection, but not elevated levels of AF IL-6 or -8, may contribute to the risk of failing the NHS test for preterm babies. **See page 349**

Pioglitazone for NEC in a rat model

Peroxisome proliferator-activated receptors (PPARs) may be crucial in the pathogenesis of necrotizing enterocolitis (NEC). Corsini *et al*. hypothesized that the PPARγ agonist pioglitazone (PIO) might be effective in preventing the development of NEC. Using the hypoxia–hypothermia model, they induced NEC in preterm rats. The treatment group received enteral PIO for 72 h,

and animals were killed 96 h after birth. NEC occurrence was found to be significantly higher in the control group than in the treatment group, demonstrating for the first time that PIO is effective in reducing the incidence and severity of NEC in a preterm rat model. **See page 364**

Hyperoxia induces extracellular matrix remodeling



Vogel and colleagues assessed the impact of 24–72 h of moderate hyperoxia (50%) on human fetal airway smooth muscle extracellular matrix (ECM) deposition using western blot, modified in-cell western, and zymography techniques. Among other results, hyperoxia exposure significantly increased collage I and III deposition and increased matrix metalloproteinase 9 (MMP9) activity. These results demonstrate that moderate hyperoxia enhances ECM deposition in developing airways by altering the balance between MMPs and their inhibitors and by increasing collagen deposition. **See page 376**