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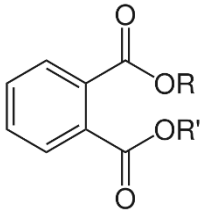
Early career investigator

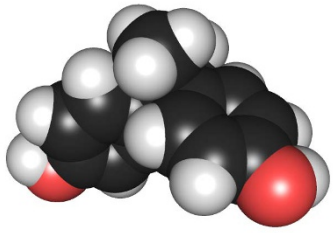
Congratulations to Adam Frymoyer, named this issue's Early Career Investigator. Check out why he was drawn to pediatric research, who his mentors are, and his lessons learned. **See page 850**

Prenatal exposure to BPA increases the risk of allergic disease



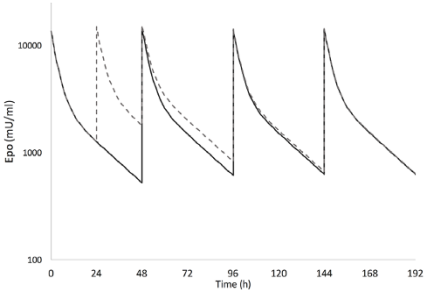
Prenatal exposure to bisphenol A (BPA) has been associated with several health outcomes. In this study from China, Zhou *et al.* observed an association between maternal urinary concentrations of BPA and infants' allergic diseases at 6 months of age. They found an increased risk in female infants of exposed pregnancies. **See page 851**

BPA and phthalates associated with endothelial dysfunction



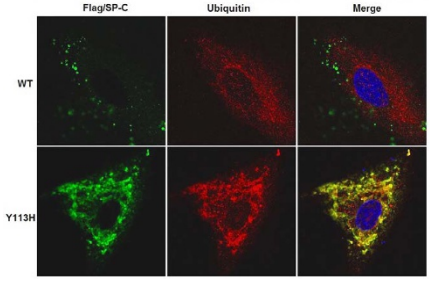
In a predominantly Mexican American and Hispanic population, Kataria *et al.* found that increasing urinary concentrations of bisphenol A (BPA), phthalates, and the BPA-substitute bisphenol S were associated with cardiometabolic risk factors even after correcting for body mass index. **See page 857**

Pharmacokinetics of high-dose erythropoietin in HIE (new ECI article!)



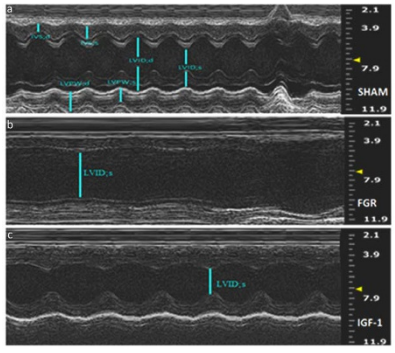
Erythropoietin is a promising drug in combination with cooling in the treatment of hypoxic–ischemic encephalopathy (HIE). Frymoyer *et al*. studied the pharmacokinetics of administered erythropoietin to develop dosing regimens that achieve target neuroprotective exposure levels. **See page 865**

A novel SPC mutation affects protein processing and location



Protein C mutations cause interstitial lung disease. Hong *et al.* studied a novel mutation that resulted in arrest of protein processing and/or excessive degradation of the proprotein. They found that the mutated protein colocalizes with ubiquitin, implying a degradation pathway other than that for wild type. **See page 891**

Prevention of fetal growth restriction induced cardiac dysfunction by IGF-1



Physiological and structural changes are a frequent outcome in growth-restricted infants. Alsaied *et al*. found that intraplacental transfer of insulin-like growth factor-1 (IGF-1) prevented cardiac dysfunction in a mouse model of fetal growth restriction. **See page 919**