Jacob E Friedman, J Gastroint Dig Syst 2014, 4:3
http://dx.doi.org/10.4172/2161-069X.S1.021

OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference on
Gastroenterology & Urology
July 28-30, 2014 DoubleTree by Hilton Hotel San Francisco Airport, USA

# Developmental origins of pediatric nonalcoholic fatty liver disease: redefining the"first hit"

**Jacob E Friedman**
University of Colorado School of Medicine, USA

The incidence of pediatric nonalcoholic fatty liver disease has increased dramatically, and growing evidence indicates that the pathophysiology may be unique from the adult form, suggesting a role for early-life events. Recent radiologic techniques have now demonstrated that maternal obesity contributes to hepatic fat storage in newborn infants. Emerging evidence now suggests that NAFLD may involve a complex interaction between diet, modifications in gut microbial composition, and a dysregulated immune response leading to progressive liver injury. The developing human fetus may be vulnerable to steatosis because immature fetal adipose depots are not available to buffer the excess trans-placental lipid delivery in maternal obesity. In non-human primate models, *in utero* exposure to high fat diet results in an increase in the accumulation of liver triglycerides prior to birth and increased hepatic oxidative stress and apoptosis, perhaps priming the liver for later development of NAFLD. Innate immune dysfunction and necro-inflammatory changes have been observed in postnatal offspring liver of animals born to high-fat fed dams. Post-weaning, livers of offspring exposed to maternal high-fat feeding share pathophysiologic features with human NAFLD, including increased de-novo lipogenesis and decreased FFA oxidation. Human studies using MRI have shown that maternal body mass index predicts infant intrahepatocellular lipid storage, as seen in animal models. The generational transfer of NAFLD from mother to infant may occur via epigenetic changes in offspring liver. Transmission of microbiota from mother to infant may also impact energy retention and immune function that contribute to a predisposition to NAFLD.

## Biography

Jacob E Friedman is the Founding Director of the Colorado Program for Nutrition and Healthy Development, sponsored by the Colorado Children's Hospital Research Institute. He has trained more than 27 Post-Docs, MD fellows, and graduate students the majority of whom are Assistant Professor or above in academic institutions. He is a PI, Co-PI, or Co-I on multiple NIH funded basic, clinical, as well as large-scale epidemiological studies of pregnancy and obesity and maternal-fetal outcomes. More recently, with funding from the Bill and Melinda Gates Foundation he has begun investigating the role of the microbiome and DNA methylation in infants with NAFLD.

Jed.Friedman@ucdenver.edu