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Guest editorial:

HIGHLIGHT REPORT: GENERAL DETERMINANTS OF STEATOSIS

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Recently Christian Hudert and colleagues from the Charité in Berlin published a study about genetic determinants in pediatric non-alcoholic liver disease (Hudert et al., 2018). Non-alcoholic fatty liver disease (NAFLD), the most frequent chronic liver disease in children, is known to be strongly influenced by genetic factors (Nobili et al., 2016; Schwimmer et al., 2006; Makkonen et al., 2009; Anstee et al., 2016). However, genetic determinants of a portal/zone-1 pattern of steatosis in children are not yet known. This would be important, because a portal/zone-1 pattern of steatosis leads to an increased risk of disease progression to fibrosis (Africa et al., 2018; Mann et al., 2016). To address this question, the authors established the Berlin adolescence NAFLD cohort (BaNA) and studied a set of single nucleotide polymorphisms. Interestingly, a variant of the retinyl-palmitate lipase PNPLA3 (rs738409) was associated with a periportal pattern of steatosis and also with an increased risk of progression to fibrosis (Hudert et al., 2018). Therefore, obese children with the PNPLA3 variant may be candidates for a more intensive clinical follow-up and intervention.

Due to the current increase in the incidence of liver diseases a better understanding of their pathophysiology is of major importance (Jansen et al., 2017; Vartak et al., 2016; Hammad et al., 2014; Hassan, 2016; Stöber, 2016; Bolt, 2017; Ekhlasi et al., 2017). For this purpose systems modeling as well as the analysis of expression patterns in relation to a phenotype represent frequently applied tools (Godoy et al., 2016; Crespo Yanguas et al., 2016; Jain et al., 2016; Saleem et al., 2016; Schenk et al., 2017; Thiel et al., 2015). The newly established BaNA cohort of adolescent NAFLD with its careful phenotyping and availability of proteome data is an important milestone for a better understanding of disease progression in steatosis.

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