Scd1 genetic deficiency protects mice against ethanol induced liver injury.

Presented by: Karl- Frédérik Bergeron PhD

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Meakins Theatre rm. 521
McIntyre Medical Building
10:30-11:30

Abstract: Hepatic steatosis is the accumulation of fat in the form of lipid droplets within liver hepatocytes. Obese patients often suffer from non-alcoholic fatty liver disease and may develop liver inflammation and fibrosis that can progress further to liver cirrhosis, and to hepatocellular carcinoma. Non-alcoholic fatty liver disease and alcoholic liver disease share a similar pathological spectrum, including hepatic steatosis. Chronic alcohol consumption induces hepatic lipid synthesis, contributing to fat accumulation. Stearoyl-CoA desaturase 1 (SCD1) is an enzyme that catalyzes the synthesis of monounsaturated fatty acids. These are typically incorporated into triglycerides and stored within lipid droplets. SCD1 is a critical regulator of hepatic lipogenesis, able to tip the balance between lipid degradation and synthesis. Scd1 genetic knockout mice exhibit cutaneous abnormalities and narrow eye fissure. Moreover, Scd1 knockout mice are protected against obesity and non-alcoholic fatty liver disease. We therefore set off to determine if these knockout mice are also resistant to the development of alcoholic liver disease. The alcohol feeding protocol used to cause liver injury was chosen to reflect alcoholic behaviour and induce typical responses (steatosis, inflammation, impaired liver function) in a short time frame. This “chronic plus binge” protocol (Bertola et al., Nat Protoc. 2013) is based on an ethanol-supplemented liquid diet, followed by a single ethanol oral gavage. This approach is relatively simple to implement and limits the number of interventions having to be made with the animals.

Bio: Karl-F. Bergeron, PhD
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Karl obtained a BSc and an MSc in biochemistry at University of Montreal then switched gears a little and completed a PhD in molecular biology in 2010 at Simon Fraser University, Vancouver. He then came back to Montreal to work as a post-doctoral fellow with Dr Pilon (UQAM; molecular genetics of development laboratory) on the migration and differentiation of neural cells during mouse intestine embryonic development. He is now working as a research associate in Dr Mounier’s lab, using genetic and diet-induced mouse models of obesity and steatosis (fat accumulation in the liver) to explore the role of specific fatty acids in various diseases like diabetes, neuro-degeneration and cancer.

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