

**Thursday October 20, 2016 – 13h30**

Conference room AI 1153 (\*) - EPFL - Lausanne

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## **THE EFFECT OF AMBIENT TEMPERATURE AND THE GUT MICROBIOTA ON THE DEVELOPMENT OF DIET-INDUCED OBESITY**

**Host:** Prof. Kristina Schoonjans

### **Abstract:**

Variations in adiposity phenotype can be regulated by cold exposure or as recently described by the gut microbiota. The former intensifies thermogenesis in brown adipocytes via *Ucp1* induction, the latter may involve mechanisms enhancing the thermogenic capacity by microbial-derived bile acids (BAs), which act through the thyroid hormone axis. We proposed that increased energy expenditure at lower ambient temperature is linked to gut microbiota metabolism. In three independent experiments we showed that at 12°C mice fed a high-fat diet had reduced adiposity and increased total energy expenditure that together with induced *Ucp1* mRNA level in iBAT and increased hepatic fatty acid oxidation blocked diet-induced obesity. These changes were accompanied by alterations in gut microbiota composition that was significantly clustered in response to low ambient temperature, with the strongest effect on a high-fat diet. Mice with reduced adiposity were characterized by increased number of *Adlercreutzia*, *Mogibacteriaceae*, *Ruminococcaceae*, *Desulfovibrio* associated with leanness. At 12°C higher production of taurine-conjugated bile acids, particularly TαMCA, TβMCA led to increased AMPK activation and decreased FXR signaling that resulted in lean phenotype. Cold-shaped microbiota maintained lean phenotype and energy metabolism, when transferred to germ free host. Importantly, the changes in gut microbiota composition and microbial metabolism were observed already after 1 day of cold exposure and were independent of reduced adiposity. We present a novel pathway by which cold-activated thermogenesis modulates the structure and function of the gut microbiota to affect microbiota-liver-BAT signaling and energy expenditure. In our mouse model, lower ambient temperature phenocopies the germ-free state and protects against developing diet-induced obesity.

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