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# Coffee restores expression of lncRNAs Involved in steatosis and fibrosis in a mouse model of NAFLD

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**Background and Aim:** Coffee intake exerts protective effects against non-alcoholic fatty liver disease (NAFLD), although without fully cleared mechanisms. In this study we aimed to assess whether coffee consumption may influence the expression of long non-coding RNAs (lncRNAs) in the liver. Methods: C57BL/6J mice were fed a 12-week standard diet (SD), High-fat diet (HFD) or HFD plus decaffeinated coffee solution (HFD +coffee). Expression of specific lncRNAs involved in NAFLD was analysed real-time PCR. For the most differentially expressed lncRNAs, the analysis was also extended to their mRNA targets.

**Results:** Decaffeinated coffee intake reduced body weight gain, prevented NAFLD, lowered hyperglycaemia and hypercholesterolemia. NAFLD was associated with lower hepatic expression of Gm16551, a lncRNA inhibiting de novo lip genesis, and higher expression of H19, a lncRNA promoting fibrogenesis. Coffee intake restored Gm16551 to levels observed in lean mice and down regulated gene expression of its targets acetyl coenzyme A carboxylase 1 and stearoyl coenzyme A desaturase1. Furthermore, coffee consumption markedly decreased hepatic expression of H19 and of its target gene collagen alpha-1(I) chain; consistently, in mice fed HFD + coffee liver expression of αSMA protein returned to levels of mice fed SD. Expression of lncRNA involved in circadian clock such as fatty liver-related lncRNA 1 (FLRL1) and fatty liver-related lncRNA 2 (FLRL2) were up regulated by HFD and were also modulated by coffee intake Conclusion. Hepatoprotective effects of Coffee may be depending on the modulation of lncRNAs involved in key pathways of NAFLD onset and progression.

## Biography

Dr Stefania Di Mauro has been involved in several projects concerning metabolic diseases. He conducted both bench top and translational research. One of her main interests has been to identify circulating noncoding RNAs in several kinds of body fluids as diagnostic biomarkers of metabolic diseases, she also focused on intracellular/tissue deregulated noncoding RNAs involved in pivotal metabolic, inflammatory and cellular stress pathways. In the context of NAFLD she developed two differential in vitro models of NAFLD where she identified intracellular and extracellular deregulated microRNAs involved in fundamental pathways of NAFLD progression; she also analysed through high throughput approach the whole transcriptase expressed in NAFLD NASH and control subjects. This study led to the identification of RNA panels that may be useful for NAFLD and fibrosis staging. She has also been involved in the study of hormone secretion deregulation of pancreatic cells and intestinal cells under lipotropic or glucotoxicity conditions.