The liver of obese patients with hepatic steatosis exhibits a severe dysregulation of key splicing machinery components as compared to obese patients without hepatic steatosis.

Obesity, a disease that is reaching epidemic proportions worldwide, is caused by a combination of genetic and lifestyle factors. One of the most common pathologies associated with obesity is hepatic steatosis, an accumulation of fat within the liver that can progress to liver fibrosis, cirrhosis, and ultimately lead to hepatocellular carcinoma. There is emerging evidence suggesting that alternative mRNA splicing, the key mechanism providing transcript and protein diversity, is dysregulated in many tissues under pathological conditions, such as obesity and cancer. Moreover, the splicing variants generated by the alteration of the normal splicing process could contribute to the aggressiveness and comorbidities of these diseases.

We hypothesized that an alteration in the splicing machinery could occur in the liver of obese patients with hepatic steatosis, which could contribute to the dysregulation of the splicing process and might ultimately be associated with the progression to hepatic fibrosis/cirrhosis/carcinoma. Therefore, the objective of this work was to determine the dysregulation of the splicingosome components and splicing factors in the liver of obese women with steatosis compared to control women.

In conclusion, the expression of specific splicing machinery components is significantly altered in the liver of obese patients with hepatic steatosis, wherein correlates with relevant clinical parameters. Ongoing studies would clarify the potential pathological implications of these findings, which could help predict a worsening in steatosis, and may provide novel diagnostic biomarkers and therapeutic tools for this disease.