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Dietary carbohydrates as a means to prevent obesity and adipose tissue inflammation

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Abstract

When obesity arises, adipose tissue (AT) expands and shifts to an influx of pro-inflammatory cells, leading to a state of chronic AT-inflammation. Furthermore, a western diet (WD) modulates the gut microbiome, increasing intestinal permeability. This facilitates the translocation of endotoxins and even entire bacteria into the blood stream, further contributing to the pro-inflammatory state. Even though it is evident a WD, high in fat and carbohydrates (CHO), can cause AT-inflammation, it is still unclear if fat or CHO is the main inducer. Therefore, we are currently investigating the effect of different CHO-types on AT-inflammation.

During 15 weeks, male C57BL/6J mice were kept on several diets, consisting of high-fructose (HFRD), high-sucrose-high-fructose (HCFD), high-starch (HSTD) or a WD (n = 10 per group). Weekly monitoring of body weight and food intake was followed by analyses of visceral AT-inflammation. Kruskal-wallis tests were used for statistical analysis.

Mice on HSTD and HCD had gained significantly less body weight compared to mice on WD after 15 weeks of diet. Mice on HSTD also gained significantly less body weight compared to mice on HFRD and HCFD. Moreover, mice on HSTD and HCD also had significantly smaller AT-depots as compared to mice on WD. Total plasma cholesterol as well as HDL and LDL levels were significantly lower in mice on HSTD and on HCD as compared to mice on WD. Gene expression analysis revealed a significantly lower expression of several pro-inflammatory markers (F4/80, Arg1, Mannose receptor, TNF, MCP1 and Saa3) in AT of mice on HSTD and on HCD compared to a WD. A HSTD also induced lower AT-expression of MCP1 and Saa3 than a HCFD and Saa3 expression was also significantly lower in the HSTD-group compared to the HFRD-group. Furthermore, Foxp3 expression, a marker for anti-inflammatory Treg cells, was significantly increased in AT of all CHO-diet fed mice as compared to the WD-group.

In conclusion, these data suggest that certain dietary carbohydrates, in contrast to a WD, do not induce obesity or AT-inflammation, including lower gene expression of Saa3. It is stated that a WD induces Saa3 expression not only in AT, but also in the colon. Furthermore, since Saa3 is able to bind bacteria and is associated with inflammation, further research is necessary to investigate Saa3 as a possible link between disturbances in the gut microbiota and AT-inflammation.

Conflict of Interest

There is no conflict of interest.