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A natural treatment for chronic pain by capsaicin

I Yuri*

Department of Biochemistry, Goodman Cancer Centre, McGill University, Montréal, Canada

*Corresponding author. E-mail: Ivanyuri@gmail.com

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DESCRIPTION

To prevent obesity caused by high-fat diets, capsaicin activates transient receptor potential vanilloid subfamily numerous studies indicate that Capsaicin (CAP) causes white adipocytes *in vitro* to turn brown or inguinal white adipose tissue to turn brown *in vivo*. Data on the dose response for CAP to prevent HFD-induced obesity are still lacking, though. Therefore, in order to prevent HFD-induced weight gain in wild-type mice, we first conducted experiments to correlate the effect of different CAP doses. Then, we conducted a sub chronic safety study using WT mice that were given either an eight-month HFD CAP diet or a regular chow diet.

We performed histological analyses of the vital organs, measured the inflammatory cytokines in plasma and iWAT, and assessed the functions of the liver and kidneys, in addition to analyzing the expression of adipogenic and thermo genic genes and proteins in the iWAT from these mice. The dose-response study demonstrated that CAP prevented HFD-induced obesity in mice at doses above 0.001% in HFD. However, at doses above 0.003% in HFD, there was no difference in the anti-obesity effect of CAP. Additionally, sirtuin-1 and thermogenic uncoupling protein 1 expression in the iWAT was improved by CAP at levels higher than 0.001%. According to safety analyses, CAP did not result in inflammation.

However, CAP reversed the effects of iWAT hypertrophy and hepatic steady states caused by HFD, elevated plasma alanine aminotransferase, and these conditions. Our research shows that CAP is well tolerated by mice and inhibits the metabolic stress and inflammation that the HFD-induced diet causes. The main component of natural chilli peppers is capsaicin. In recent years, a great deal of research has been done on the advantages of CAP for the treatment of cancer, metabolic syndrome, and neuropathic pain. CAP was apparently safe and well tolerated in prior experimental studies using local CAP patches on rats and rabbits. Additionally, research on the use of an external CAP patch to treat neuropathic pain indicates that CAP is safe and well-tolerated by people.

The effectiveness and safety of feeding CAP orally over the long term, however, are not well understood. In both rodents and people, CAP reduces obesity and increases energy expenditure. To counter high-fat diet-induced obesity in mice without reducing energy intake, CAP activates the browning of brown fat and enhances the thermogenic programme in inguinal white fat. Additionally, CAP reduces hyperlipidemia, improves glucose intolerance, and raises plasma levels of glucagon-like peptide 1. In contrast, CAP had no effect on obesity in transient receptor potential vanilloid subfamily 1-deficient mice.

Earlier studies using different forms of capsaicin, such as chilli pepper powder, its less pungent analogue, capsiate, or other forms of capsaicinoids and their safety reporting varied. The kind and purity of capsaicinoids or capsinoids used in these studies are the cause of these variations. Additionally, recent research indicates that pungent CAP is more effective than non-pungent derivatives at binding to TRPV1 and promoting thermogenesis. There is also no correlation between the dose of CAP and the inhibition of HFD-induced weight gain, despite the fact that the majority of these rodent data were obtained using 0.01% of CAP in HFD. Due to a lack of information on the pharmacological effect of oral feeding of various doses of pure CAP and its long-term safety in mice, we conducted a dose response study and 8-month subchronic safety analyses by feeding mice with pure CAP either in regular chow diet or HFD.