In human, capsaicin, the pungent compound in hot pepper, is detected through transient receptor potential (TRP) ion channel vanilloid 1 (TRPV1). TRPV1 is also activated by heat (43˚C) and protons. Since the discovery of TRPV1 (1), several thermosensitive TRP channels have been found. Food components activating TRPV1 inhibit abdominal fat deposition through sympathetic nerve stimulation. We have searched for agonists for TRPV1 and TRPA1 in vitro from Asian spices by the use of TRPV1- and TRPA1-expressing cells. Further, we performed food component addition tests to high-fat and high-sucrose diets in mice. We found capsiate, capsicionate, capsainol from hot and sweet peppers, several piperine analogs from black pepper, gingeriols and shogaols from ginger, and sanshools and hydroxysanshools from sansho (Japanese pepper) to be TRPV1 agonists. We also identified several sulfur compounds from garlic and durian, hydroxy fatty acids from royal jelly, and capsiate from Capsicum frutescens (2). We found that all of them were weak agonists but had similar potency as that in human sensory analyses (8). Further, we found that 1-monoyacylglycerol having certain acyl moieties from wheat, mioga (Zingiber mioga) and onion are agonists for TRPV1 (9).

In Vitro Experiments Using TRPV1- or TRPA1-Expressing Cells

Human embryonic kidney (HEK) 293 cells were used.
Food Compounds Activating Thermosensitive TRP Channels in Asian Spice

Animal Experiments

To show the possibility that TRPA1 agonist could enhance energy metabolism, adrenaline responses to AITC and cinnamaldehyde (CNA) were measured in blood from the adrenal vein of anesthetized rats. Intravenous administration of AITC or CNA induced adrenalineline secretion (13). These responses were blocked by cholinergic blockers showing the participation of adrenergic sympathetic nerve activity. Further, capsaicin-treatment, which impairs sensory nerve function, abolished the response due to AITC or CNA. These results suggest that TRPA1 agonists induce adrenergeline secretion via sensory nerve activation like TRPV1 agonist capsaicin.

Effect of food components on visceral fat accumulation in mice fed high-fat and high-sucrose diet was also evaluated as an in vivo test. Feeding a high-fat and high-sucrose (HFS) diet for 1 mo induced obesity in C57BL mice. Addition of 1-oleoylglycerol to this diet resulted in an increase of UCP1 in interscapular brown adipose tissue (BAT) (14). With the addition of 0.03 or 0.05% of piperine or black pepper extract containing same amount of piperine, the deposition of visceral fats was abolished and UCP1 contents in interscapular BAT increased (15). In addition, CNA feeding with the HFS diet inhibited the accumulation of visceral fats at least due to BAT activation (16).

Conclusion

We found that several agonists of TRPV1 and TRPA1 and some agonists of TRPV1 and TRPA1 inhibit visceral fat deposition in mice. The effects of such compounds on humans remain to be clarified, but we expect that they will be helpful in the prevention of obesity. In fact, supplementation of capsaicin-like compound (CH-19 Sweet) reduces visceral fat in humans (17).

REFERENCES


