Dear Sir;

We read with interest the article by Jyotaki et al. [1], entitled “Modulation of sweet taste sensitivity by orexigenic and anorexigenic factors.”, which summarized recent findings regarding the roles of leptin and endocannabinoids as modulators of the peripheral components of sweet taste. Polymorphism of the cannabinoid receptor (CNR1) gene was implicated in the modulation of the endocannabinoid system metabolic and center effects [2]. The gene that codes for CNR1 is located in chromosome 6q14-q15. At the 3’ extreme of CNR1, there is a polymorphic (AAT)n triplet (number of 12-20) which facilitates classification of the population according to the length of the repeats. The presence of long alleles, thus, a higher number of AAT triplets, yields the conformation of a Z shape in the DNA, which may lead to altered gene transcription. Ballon et al. reported that the (AAT)12 repeat allele near the CNR1 gene was associated with a predisposition to cocaine addiction in an African-Caribbean population [3].

Therefore, we investigated the association between sweet taste and this repeat in 32 obese females (mean age: 57 ± 6 years, body mass index (BMI): 26.0 ± 1.9 kg/m²). The study was approved by the ethics committee of Kyoto Medical Center. All subjects gave informed consent prior to participation. The sweet taste threshold was determined according to the whole-mouth gustatory method [4]. Leptin was measured by a radioimmunoassay. Zinc was measured with an atomic absorption spectrophotometer. Copper was measured by a colorimetric method. Genetic analyses were performed using the allele-specific DNA assay. The sweet taste threshold was significantly higher in subjects without (n = 19) than in those with the (AAT)12 repeat (n = 13) at the baseline (0.48 ± 0.42 % vs. 0.22 ± 0.33 % in a solution of sucrose; p = 0.018). There was no difference in age, BMI, zinc, copper or serum leptin levels between groups. Stepwise multiple linear regression using age, BMI, zinc, copper, serum leptin levels and (AAT)12 repeats as potential factors of log-transformed sweet taste threshold yielded only the (AAT)12 repeat as a significant determinant (β = -0.496; 95 % confidence interval = -0.119 to -0.874; p = 0.012). The taste organ is a peripheral target of endocannabinoids. The positive effect of endocannabinoids on sweet sensitivity opposes the action of leptin, which suppresses sweet sensitivity. Leptin and endocannabinoids, therefore, not only regulate food intake through the central nervous systems, but also may modulate the palatability of foods by altering peripheral sweet taste responses through their cognate receptors [5]. Orexigenic and anorexigenic factors such as endocannabinoids and leptin may affect energy homeostasis by regulating taste sensitivity. These studies provide data to suggest that the ATT repeat in the CNR1 gene influences sweet taste in obese females. A major limitation of this trial is the small sample size. More studies with a larger sample size are necessary to confirm these findings.

References
