Decrease in blood glucose as well as increase in fatty acids are essential information for hunger in the brain hypothalamus. During meal, the gustatory information is important to identify the food containing required nutrients or not. Food with familiar taste and flavor is to be allowed for swallowing. Subsequently, food debris with nutrient information stimulates the luminal layer of the stomach, as chemical senses and tactile information.

The vagal afferent fiber composed of two types; one is responsive to nutrients, and the other is stomach fullness. In recent, we defined the vagal afferent responding to glutamate alone among 20 kinds of amino acids. Additionally, particular 5'-ribonucleotides, inosine 5'-monophosphate (IMP) and guanylic acid (GMP) are umami materials and also positive stimulants for gastric vagal afferent. These data manifest that umami taste materials are essential either to recognize the food intake or to onset digestion. But celiac and hepato-portal vagal afferent respond to any nutrients including umami taste materials.

This glutamate signaling causes the digestive juice secretion in the gut directly and indirectly via brain function. Therefore, the cascade of digestion processes should need glutamate in digests and operate normally for absorption of yielded nutrients and consequent metabolic control due to these homeostasis in the body.

Finally glutamate signaling from the gut reaches the hypothalamus in the brain i.e. feeding and satiety centers, the basic metabolic rate regulator and body temperature controller. In facts, we defined previously that diet with glutamate enhanced thermogenesis in awake rats. So, diet-induced thermogenesis during and after meal seem to be triggered by glutamate signaling via the vagal afferent. Similar mechanism for other nutrients might exist.

This symposium will discuss interrelationship between the taste and visceral information and brain functional changes during meal from the experimental to clinical levels.