Research Advances on Animal Umami Receptors and Their Gene Expression Regulation

Authors: Wang Jing1 Zhang Yong1,2* Huang Tiejun2

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Abstract

Umami receptors in animals include metabotropic glutamate receptors (mGluR) and taste receptor heterodimers (T1R1/T1R3), which are type C G protein-coupled receptors. The N-terminal venus flytrap (VFT) domain can bind to umami ligands to recognize umami taste. This paper mainly discusses research progress on umami receptors, mechanisms of umami recognition and transduction, and expression regulation of umami receptor genes, aiming to provide references for related research.

Full Text

Research Advances on Animal Umami Receptors and Their Gene Expression Regulation

Wang Jing1 Zhang Yong1,2* Huang Tiejun2

(1. College of Animal Husbandry and Veterinary Medicine, Shenyang Agricultural University, Shenyang 110866;

2. Lucta (Guangzhou) Flavours Co., Ltd., Guangzhou 510530)

Abstract: Animal umami receptors include metabotropic glutamate receptors (mGluR) and taste receptor type 1 heterodimers (T1R1/T1R3). They are class C G-protein-coupled receptors, and the Venus flytrap (VFT) domain at the N terminus can bind umami ligands and recognize umami taste. This paper mainly discusses research progress on umami receptors, mechanisms of umami recognition and signal transduction, and regulation of umami receptor gene expression, with the aim of providing a reference for related studies.

Keywords: umami receptor; metabotropic glutamate receptor; taste receptor type 1 heterodimer; transduction mechanism; regulation

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Taste is the process by which animals identify the constituent components of ingested substances. It is generally divided into five types: sweet, sour, umami, bitter, and salty. Among them, umami is mainly elicited by monosodium glutamate (MSG), certain amino acids (mainly aspartic acid), large amounts of short peptides, and some organic acids (such as lactic acid and propionic acid)[1]. Taste discrimination is completed through the specific interactions between taste receptors (TRs) and different substances, which transmit information to the brain; taste receptors are specific. Existing studies indicate that umami receptors mainly include metabotropic glutamate receptors (metabotropic glutamate receptor, mGluR) and taste receptor type 1 heterodimers (T1R1/

functional expression in cells. The mGluRs that exist as taste receptors mainly include mGluR1[^3] and mGluR4[^4], whose structures are shown in Fig. 1-A. mGluR4 was the first substance discovered to exist as an umami receptor; it can be expressed in taste cells, and simulations of the taste aversion to *L*-glutamate by its ligands have confirmed it as an umami receptor. Related studies have shown that the mGluR1 inhibitor 1-aminoindan-1,5-dicarboxylic acid (AIDA) and the mGluR4 inhibitor α-cyclopropyl-4-phosphonophenylglycine (CPPG) can reduce the behavioral responses of taste cells and gustatory nerve fibers to umami substances in mice, also indirectly indicating the perception of umami by these two receptors. mGluR1 and mGluR4 mainly receive the umami taste of glutamate and certain analogs[^5-6]. Both are expressed not only in taste receptor cells of the circumvallate papillae and foliate papillae of the tongue, but also in brain tissue; mGluR1 also exists in the intestine of rats. Related studies have shown that the expression of both in the brain is directly related to the transduction of umami in the brain[^7]. They are both G protein-coupled receptors. After activation, mGluR1 mediates the phospholipase C signaling pathway, whereas mGluR4, after activation, is mainly coupled to the adenylate cyclase system. mGluR displays a certain degree of species-specificity among different species; the homology between mice and humans is only 80%, and this difference can also explain interspecies differences in umami perception.

1.2 T1R1/T1R3

The first family of taste receptors (taste receptor family 1 member, T1R) are G protein-coupled receptors. After their discovery in 2001, related studies in 2002 detected changes in the concentration of calcium ions (Ca^{2+}) after activation of G protein-coupled receptors in human embryonic kidney cells, and inferred that T1R1/T1R3 among the T1Rs is the principal receptor for umami. T1R1/T1R3 can sense glutamate and 20 kinds of *L*-amino acids, and inosine monophosphate (IMP) and guanosine monophosphate (GMP) have enhancing effects on it. T1R1/T1R3 is expressed not only in the fungiform papillae of the tongue and the taste buds of the palate, but also in non-gustatory systems such as the digestive tract, brain, liver, muscle tissue, and germ cells, and it can also be expressed in mouse neutrophils[^8]. The schematic structure of T1R1/T1R3

A and B schematic diagrams of umami receptor structure

Figure 1: A and B schematic diagrams of umami receptor structure

Flowchart of umami molecule transduction

Figure 2: Flowchart of umami molecule transduction

is shown in Fig. 1-B. Margolskee et al.[^9] found that in the intestines of mice, rats, and humans, T1R1 and T1R3 can recognize *L*-amino acids in the form of heterodimers. Related studies have shown that activated T1R1/T1R3 in the intestine can stimulate intestinal peristalsis[^{10}]; all of the above illustrates the expression of T1R1/T1R3 in the digestive tract. As a G protein-coupled receptor, T1R transmits taste signals through the phospholipase C signaling pathway after activation. In addition to umami recognition, T1R1/T1R3 can also regulate the secretion of cholecystokinin, insulin, and duodenal bicarbonate (HCO_3^-), and activate the inhibition of spontaneous secretion by mammalian trefoil factor complexes[^{11}]. T1R1/T1R3 shows a certain degree of specificity among different species; the T1R genes of humans and rodents have only 70% identity. The human T1R1 and T1R3 genes are both located on the short arm of human chromosome 1 and are relatively close to one another, whereas in mice both genes are located at the end of chromosome 4. In addition, the mammalian T1R1 gene contains six exons. This difference can also explain interspecies differences in umami ···

differences in perception.

A B

Extracellular region: extracellular region; Transmembrane region: transmembrane region; Cytoplasmic region: cytoplasmic region; Membrane: membrane.

Fig. 1 Structure of umami receptors

Fig. 1 The structure of umami receptors[1]

1.3 Recognition and transduction of umami by umami receptors

PLC: phospholipase C; TRPM5: transient receptor potential ion channel 5, melastatin 5; $P2X_2/P2X_3$: purinergic receptors.

Fig. 2 Transduction of umami molecules

Fig. 2 The transduction of umami molecules[2]

Although different species show certain differences in their umami receptors, the processes by which they recognize and transduce umami molecules such as glutamate salts are basically the same. The recognition process of umami-molecule

[1] 12-13
[2] 14

signals by T1R1/T1R3 has already been clarified. The *L*-glutamate molecule and IMP act together on the VFT domain of T1R1: *L*-glutamate binds to the VFT near the hinge region, inducing closure of the VFT structural domain; IMP binds to the site far from the VFT opening, which, after *L*-glutamate acts on the VFT, promotes closure of the VFT domain, thereby achieving umami recognition[3].

After umami molecules such as MSG are recognized, the process is as shown in Figure 2. Activation of the T1R1/T1R3 receptor initiates the phospholipase C signaling pathway, activating phospholipase C-β (PLC-β), which in turn hydrolyzes phosphatidylinositol 4,5-bisphosphate (PIP_2) to generate inositol trisphosphate (IP_3) and diacylglycerol (DAG). IP_3 is enzymatically degraded to produce degradation products, which can induce Ca^{2+} release from intracellular calcium stores, thereby activating TRPM5 channels. TRPM5 channels are monovalent cation channels that open instantaneously, allowing sodium ions (Na^+) to enter the cell. Studies by Akiyuki et al.[17] showed that calcium homeostasis modulator 1 (Calhm1) can form a link between Na^+ and the neurotransmitter ATP, ultimately leading to membrane depolarization and release of the neurotransmitter ATP.

In addition, if umami-molecule signals such as MSG are recognized by mGluR4, α-gustducin can be activated, thereby activating phosphodiesterase (PDE), reducing the content of cytoplasmic cyclic adenosine monophosphate (cAMP), and consequently eliminating the inhibitory effect of cyclic nucleotide (cNMP) on ion channels. Intracellular Ca^{2+} is released, so that membrane depolarization and neurotransmitter release can proceed[18].

In summary, after MSG umami-molecule signals are transduced through receptors, the cell membrane of taste cells is depolarized, and the neurotransmitter ATP is released to activate purinergic receptors ($P2X_2/P2X_3$) on adjacent gustatory nerve fibers[14]. In this way, umami receptor cells convert the received chemical signal into an electrical signal. Thereafter, the signal is transmitted along the glossopharyngeal nerve, the chorda tympani branch of the facial nerve, and the vagus nerve to the nucleus of the solitary tract in the medulla oblongata. In the transmission of umami signals in primates and humans, neurons in the nucleus of the solitary tract are second-order neurons and extend to the ventral posteromedial nucleus of the thalamus. In rodents, during umami signal transmission, neurons extend from the nucleus of the solitary tract to the parabrachial nucleus of the pons, and after relaying in the parabrachial nucleus, they split into two projections to the ventral posteromedial nucleus of the thalamus and the lateral hypothalamic area; after relaying there, they project to the cortex and then return from the central posterior region to the lowermost gustatory center for umami perception.

[3] 15–16

2 Factors affecting regulation of umami receptor gene expression

Umami receptors exist in the taste cells of the tongues of humans, primates, mammals, and many other animals. However, because of limitations imposed by many factors, experimental animals used to study regulation of umami receptor gene expression are mainly dogs and mice. Expression of umami receptor genes is affected by many factors. Related studies have shown that, within a certain range, the activity of TRM5 ion channels changes with temperature, thereby altering Na^+ concentration, reducing the action potential and the degree of taste-cell membrane depolarization, and consequently leading to decreased expression of umami receptor gene 1/umami receptor gene 3 (*Tas1r1/Tas1r3*), weakening the expression of umami receptors.

Okamoto et al.[19], through reverse transcription (RT)-PCR, showed that chronic restraint stress can markedly inhibit the expression of *T1R3* mRNA, thereby weakening the expression of umami receptors. Toyono et al.[20], using cultured human bile duct carcinoma cell lines, detected by luciferase assay and electrophoretic mobility shift assay that CCAAT enhancer-binding protein β (CCAAT enhancer binding protein β, C/EBPβ)

can activate the *Tas1r3* promoter, enhance the expression of *T1R3* mRNA, and thereby increase the expression of umami receptors. Studies by Kokabu et al.[9] showed that muscle regulatory factors can increase the expression level of *T1R3* mRNA by activating the promoter activity of *Tas1r3*, thereby increasing the expression of umami receptors. Many factors similarly regulate umami receptors. Below, several nutritional factors commonly used in production are selected to illustrate their regulation of umami receptor gene expression, and at the same time to explain their effects on the regulation of umami receptor expression in production practice.

2.1 Glucocorticoids (glucocorticoid, GCs)

GCs, also known as adrenal cortical hormones, are a class of steroid hormones synthesized by the adrenal cortex, mainly cortisol. GCs regulate the biosynthesis and metabolism of sugars, fats, and proteins, and are relatively common in both mammals and rodents. They also have anti-inflammatory effects and can be used for diseases not adequately treated by ordinary antibiotics or anti-inflammatory drugs, such as sepsis. They are commonly used drugs in daily production; for example, they are the principal components of drugs such as dexamethasone. Studies by Liu Lei et al.[21] showed that GCs can stimulate the appetite of broiler chickens through regulation of central AMP-activated protein kinase (AMPK), inducing chicks to prefer high-energy feed. GCs affect animal growth performance and have practical applicability in production.

GCs can regulate the gene expression of umami receptors. Oqawa et al.[22] used RT-PCR to detect the expression level of *T1R3* mRNA in the fungiform papillae of adrenalectomized rats, and found that *T1R3* mRNA expression in

the fungiform papillae was significantly reduced. Then, under fasting conditions, rats were fed 0.1, 10, and 1 000 ng/kg dexamethasone, respectively. In the fungiform papillae of rats fed 0.1 ng/kg dexamethasone, the expression level of *T1R3* mRNA recovered to the normal level. In the other two groups of rats, however, the expression level of *T1R3* mRNA in the fungiform papillae did not reach the normal level. This indicates that GCs can regulate the expression of the umami receptor *T1R1/T1R3*: low concentrations of GCs can induce *T1R3* mRNA expression, thereby enhancing the expression of the *T1R1/T1R3* receptor; high concentrations of GCs, however, suppress the expression of umami receptor *T1R3* mRNA, thereby suppressing the expression of the *T1R1/T1R3* receptor. Thus, they affect the regulation of umami receptor gene expression, and also indicate that in production practice they may affect umami receptor expression. In addition, this also indirectly illustrates the harm of abusing GCs.

2.2 MSG

MSG, chemically named monosodium α-aminoglutarate, is the sodium salt of glutamic acid, is the main component of monosodium glutamate, and is often added to feed as an umami agent. Glutamic acid participates in many metabolic activities in the organism. Glutamic acid mediates rapid excitatory transmission at the vast majority of synapses in the central nervous system and participates in most brain-function regulatory processes. In a certain sense, glutamic acid is part of umami. Excessive glutamic acid affects animal growth performance and affects the absorption of trace elements such as calcium and zinc. In Chen Li' s[23] experiment, suckling piglets were used as the research subjects, and 0, 0.06, 0.50, and 1.00 g/(kg · d) MSG were added, respectively. The experimental results showed that low

the average daily gain in the low-dose and medium-dose groups increased by 3.25% and 7.54%, respectively, compared with the control group; compared with the control group, the average daily gain in the high-dose group decreased by 9.41%. MSG affects animal growth performance and has practical value in production.

MSG affects the regulation of gene expression of *mGluR* and *T1R1/T1R3*, and the three influence one another. The study by Zhang Ce et al.[24] showed that excessively high glutamate concentrations activate *mGluR4* and increase the expression of *mGluR* mRNA. *mGluR4* mediates the inhibitory effect of parallel fibers induced by *L*-2-amino-4-phosphonobutyric acid (*L*-AP4) (originating from granule cells) on the presynaptic terminals of Purkinje cells, suppressing neurotransmitter release and thereby inhibiting the entire pathway of umami molecular-signal transduction. Recognition of umami by T1R1/T1R3 is inhibited; the expression of *T1R1/T1R3* mRNA decreases; expression of *mGluR* and *Tas1r1/Tas1r3* mRNA is suppressed; and mRNA expression of *mGluR* and *T1R1/T1R3* is inhibited. This also indirectly indicates the hazards of excessive addition of monosodium glutamate. In addition, Zhang et al.[25], using RT-PCR and Western blot analysis, showed that addition of a normal amount

of MSG increases the expression of *T1R1/T1R3* mRNA in the gastrointestinal tract of piglets, increases *T1R1/T1R3* mRNA expression, strengthens regulation of umami genes, and thus demonstrates regulation by MSG of umami receptor gene expression; at the same time, it also shows its effect on umami receptor expression in production practice.

2.3 Inosine Nucleotides

The effects of inosine nucleotides on umami receptors are mainly reflected in IMP and GMP. Their salt forms, disodium inosinate and disodium guanylate, are the principal components of umami agents and have synergistic effects. In addition to being used to enhance flavor, inosinate can also be used to increase the breast-muscle percentage of chickens, reduce the content of triglycerides in serum, increase the contents of glucose, high-density lipoprotein, and cholesterol in serum, and improve carcass quality[26]. The study by Yang Yufen et al.[27] showed that adding IMP and GMP separately to feed had no significant negative effects on the production performance of piglets, indicating their practical value in production. IMP is mainly produced by the degradation of ATP in muscle, whereas GMP is mainly derived from RNA treated with snake-venom phosphodiesterase. As umami substances, the two mainly enhance the umami taste of sodium glutamate and glutamic acid, and this flavor-enhancing effect is chiefly manifested in their regulation of umami receptor gene expression.

The regulation of umami receptor gene expression by IMP and GMP mainly has two aspects. On the one hand, both can interact with the VFT of T1R1.

The research methods reported show that IMP can act on taste cells, increasing the concentration of Ca^{2+} in taste cells, thereby activating TRPM5 channels and causing Na^{+} influx, which leads to ATP release, enhanced umami signal transduction, increased expression of *T1R1/T1R3* mRNA, and increased expression of the umami receptor T1R1/T1R3. That is, both can regulate the expression of the *Tar1r1/Tar1r3* genes by modulating intracellular Ca^{2+} concentration, thereby achieving an umami-enhancing effect. These two aspects both indicate that IMP and GMP affect the regulation of umami receptor gene expression, and also demonstrate their influence on umami receptor expression in production practice.

2.4 Inorganic salts

The effects of inorganic salts on umami are mainly reflected in Na^{+} and Ca^{2+}. Among them, NaCl is the main component of table salt. Salt has an umami-enhancing effect; in ancient China there was the saying that "without blandness there is no taste, and without salt there is no taste," which indicates the regulatory effect of salt on flavor.

The regulation of umami gene expression by Na^{+} is mainly due to the fact that changes in intracellular Na^{+} concentration cause membrane depolarization

and a large release of the neurotransmitter ATP; a large amount of umami molecules is transduced, thereby causing a sharp increase in *T1R1/T1R3* mRNA expression, increased expression of the T1R1/T1R3 receptor, and completion of the regulation of umami receptor gene *Tar1r1/Tar1r3* expression.

The regulation of umami gene expression by Ca^{2+} is mainly manifested in the regulation of TRPM5 channels by changes in Ca^{2+} concentration. That is, an increase in intracellular Ca^{2+} concentration activates intracellular TRPM5 channels; activation of TRPM5 channels leads to Na^{+} influx, followed by membrane depolarization and release of the neurotransmitter ATP; *T1R1/T1R3* mRNA expression increases sharply, T1R1/T1R3 receptor expression increases, and the effect on regulating umami receptor gene *Tar1r1/Tar1r3* expression is completed. At the same time, this can also indicate its influence on umami receptor expression in production practice. In addition, changes in Ca^{2+} concentration are associated with the N-methyl-*D*-aspartic acid receptor (N-methyl-*D*-aspartic acid receptor, NMDA). NMDA is a type of glutamate receptor and affects the concentration of glutamate, but the specific regulation of its expression remains to be further studied.

3 Summary

As one of the five basic tastes, umami can enhance animal appetite and improve animal production efficiency. In summary, umami receptors can complete the recognition and transduction of umami, and umami signals stimulate the brain to generate appetite. The gene expression of umami receptors is regulated by many factors, among which there are many components with practical value in production practice. Umami receptors are very important for the application of umami agents; however, research on umami receptors both in China and abroad remains clearly insufficient. At present, research on umami receptors, on the one hand, is mainly concentrated on T1R1/T1R3, with relatively little research on mGluR, and studies of the T1R1/T1R3 heterodimer are also mainly focused on T1R3. On the other hand, research on umami receptors is mainly concentrated in humans and rodents, while studies in animals such as pigs and cattle are relatively few. There are also few studies on the regulation of umami receptor gene expression by nutrients, and the mechanism by which nutrients regulate umami receptor gene expression is still not completely clear. This review, through the research progress on umami receptors, the mechanism of umami recognition and transduction, and the effects of nutrients on ···

The introduction above to the regulation of umami receptor gene expression is intended to provide a reference for subsequent related research. With the widespread use of umami additives in feed, research on umami receptors is receiving extensive attention. It is hoped that, in the near future, studies of umami receptors will achieve breakthroughs and yield favorable results in the fields of gene regulation and livestock production.

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Umami Receptors: Research Progress and Gene Expression Regulation

WANG Jing[1] ZHANG Yong[1,2*] HUANG Tiejun[2]

(1. College of Veterinary and Animal Science, Shenyang Agricultural University, Shenyang 110866, China; 2. Lucta (Guangzhou) Flavors Co., Ltd., Guangzhou 510530, China)

Abstract: Umami receptors include metabolic glutamate receptors and taste heteromeric receptor dimmers, which are members of G protein coupled receptors C family. They all have N-terminal

VFT region, which enables them to bind umami ligands and recognize umami. In this paper, the research progress on umami receptors, the transduction of umami recognition, and the regulation of umami receptor gene expression were summarized.

Key words: umami receptor; mGluR; T1R1/T1R3; transduction mechanism; regulation

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*Corresponding author, professor, E-mail: syndzhy@126.com (Responsible editor: Wu Hailong)

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