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Genomic and Genetic Evidence for the Loss of Umami Taste in Bats

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Abstract

Umami taste is responsible for sensing monosodium glutamate, nucleotide enhancers, and other amino acids that are appetitive to vertebrates and is one of the five basic tastes that also include sour, salty, sweet, and bitter. To study how ecological factors, especially diets, impact the evolution of the umami taste, we examined the umami taste receptor gene *Tas1r1* in a phylogenetically diverse group of bats including fruit eaters, insect eaters, and blood feeders. We found that *Tas1r1* is absent, unamplifiable, or pseudogenized in each of the 31 species examined, including the genome sequences of two species, suggesting the loss of the umami taste in most, if not all, bats regardless of their food preferences. Most strikingly, vampire bats have also lost the sweet taste receptor gene *Tas1r2* and the gene required for both umami and sweet tastes (*Tas1r3*), being the first known mammalian group to lack two of the five tastes. The puzzling absence of the umami taste in bats calls for a better understanding of the roles that this taste plays in the daily life of vertebrates.

Key words: bats, umami taste, pseudogenization, *Tas1r1*, diet.

Evolution of the Umami Taste and Its Receptor

Umami taste is one of the five primary tastes that also include salty, sour, bitter, and sweet (Kinnamon and Margolskee 1996; Lindemann 1996). It detects monosodium glutamate (MSG), nucleotide enhancers, and other amino acids that are appetitive to vertebrates (Yarmolinsky et al. 2009) and is believed to be important for identifying nutritious food (Herness and Gilbertson 1999). Humans have developed MSG as an additive to make food savory or meaty (Ikeda 2002). Umami taste signal transduction starts from the binding of umami tastants by a heterodimeric receptor composed of Tas1r1 and Tas1r3. Although Tas1r1 is used exclusively in the Tas1r1–Tas1r3 heterodimer, Tas1r3 can also couple with Tas1r2 to form the Tas1r2–Tas1r3 heterodimer that binds to the sweet tastants and transmits the sweet signal (Mombaerts 2004; Yarmolinsky et al. 2009). Tas1r1, Tas1r2, and Tas1r3 are homologous with one another. Genomic and phylogenetic analyses suggest that the Tas1r family originated in the common ancestor of jawed vertebrates (Grus and Zhang 2009) and that the three distinct Tas1rs were already present in the common ancestor of bony vertebrates (Shi and Zhang 2006). In all mammals studied, Tas1r1, Tas1r2, and Tas1r3 are each encoded by one gene (Shi and Zhang 2006).

There have been few behavioral studies of the umami taste in vertebrates. Nonetheless, revelation of the molecular genetic basis of umami perception allows the use of *Tas1r1* as a genetic marker to probe the umami taste across diverse species. Thus far, *Tas1r1* is known to be intact in all mammals except the giant panda (Li et al. 2010; Zhao, Yang, et al. 2010), a bear that nevertheless feeds almost exclusively on bamboos. Because amino acids are more abundant in meat than in plants, it has been hypothesized that the pseudogenization of *Tas1r1* in the giant panda was related to its dietary switch from a carnivore to a vegetarian (Li et al. 2010; Zhao, Yang, et al. 2010). This hypothesis was supported by the approximate match in inferred time between the *Tas1r1* pseudogenization and the dietary switch (Zhao, Yang, et al. 2010). However, other vegetarians, such as horse and cow, still possess an intact *Tas1r1*, suggesting that additional factors shape mammalian *Tas1r1* evolution (Zhao, Yang, et al. 2010).

Examining the relatively closely related species that exhibit a high dietary diversity can help discern the dietary impact on the evolution of *Tas1r1* and umami taste. Bats are potentially useful for this purpose. Two-thirds of bat species feed primarily on insects, although some of them supplement with small mammals, birds, reptiles, amphibians, fish, and other arthropods. For simplicity, these bats are referred to as insect eaters. Around one-third of the bats are primarily plant eaters, consuming fruits, flowers, nectar, pollen, and foliage. Plant eaters are divided into two groups: Old World (OW) and New World fruit bats; the latter occasionally eat insects. In addition to the insect eaters and plant eaters, three bat species, known as vampire bats, feed exclusively on blood (Altringham 1996).

Pseudogenization of *Tas1r1* in Bats

We began by examining the two bat draft genome sequences at Ensembl (http://www.ensembl.org/). Mammalian Tas1r1 is encoded by six exons, of which the first five encode a long extracellular domain of this G-protein coupled receptor, whereas exon 6 encodes the remaining segment composed of the seven transmembrane domains, three extracellular loops, three intracellular loops, and the intracellular C-terminus. From the genome sequence of *Pteropus vampyrus* (fig. 1), commonly known as the large flying fox and one of the largest bats, we identified the complete exon 1 (182 bp), a partial exon 3 (137 bp), and a partial exon 6 (743 bp) of a single-copy *Tas1r1* (supplementary fig. S1, Supplementary Material online). Although the open reading frame (ORF) is retained in exon 1 (despite a frame shifting deletion) and exon 3, it is disrupted in exon 6 by five insertions/deletions (indels) that result in four premature stop codons (supplementary fig. S1, Supplementary Material online), suggesting that *Tas1r1* is a pseudogene in *P. vampyrus*.

We could not find *Tas1r1* from the genome sequence of the little brown bat *Myotis lucifugus* (fig. 1). In the dog genome, *Tas1r1* is flanked by *NOL9* (ENSCAFG00000019604) on one side and *ZBTB48* (ENSCAFG00000019615) on the other. This linkage is conserved across human, mouse, cow, and cat. We found *NOL9* and *ZBTB48* adjacent to each other in GeneScaffold_4671 of the *M. lucifugus* genome, strongly indicative of a true loss of *Tas1r1* rather than the incomplete genomic sequencing in this species.

Based on the well-established bat phylogeny, the bat order Chiroptera is divided into two suborders: Yinpterochiroptera and Yangochiroptera (Teeling et al. 2005). The two species analyzed above, *P. vampyrus* and *M. lucifugus*, belong to these two suborders, respectively (fig. 1), suggesting the possibility that the absence of a functional *Tas1r1* may be widespread in bats. To test this hypothesis, we attempted to amplify exon 6 of *Tas1r1* from *P. vampyrus* and 29 additional species representing all major lineages of bats (fig. 1). We focused on exon 6, because exon 1 and exon 3 identified from the *P. vampyrus* genome are short. We were able to amplify and sequence longer fragments (559–709 bp) from five species and shorter fragments (190–342 bp) from another five species (fig. 1). The longer fragments span from the second transmembrane domain to the C-terminus, whereas the shorter fragments contain the region from the end of the third transmembrane domain to the fifth or sixth transmembrane domain depending on the species (fig. 2). For the remaining 20 species, amplification was unsuccessful (fig. 1). The amplification success rate is higher for yinpterochiropterans (7/14 = 50%) than for yangochiropterans (3/16 = 19%) ($P = 0.077$, one-tail Fisher's exact test), probably because the primers were designed according to *P. vampyrus*, a yinpterochiropteran.

After aligning our newly obtained sequences with dog *Tas1r1* (GenBank accession no. XM_546753), we identified premature stop codons in each sequence (fig. 2). In nine of the ten sequences, the 5′ most premature stop codon is located before the final transmembrane domain (fig. 2), leading to the loss of at least one transmembrane domain and the C-terminus of the receptor. In the remaining sequence (*Rhinopoma hardwickii*), the first premature stop codon is located within the seventh transmembrane domain, but a 26-bp deletion is found in the sixth transmembrane domain, resulting in a shift of the reading frame. Thus, none of the 10 amplified *Tas1r1* genes are functional.

Among yinpterochiropterans, two OW fruit bats (*Rousettus leschenaultii* and *Cynopterus sphinx*) share two frame shifting insertions and two premature stop codons, which are unshared with another OW fruit bat *P. vampyrus* (fig. 2), suggesting that these ORF-disrupting mutations occurred in the common ancestor of *R. leschenaultii* and *C. sphinx* after its separation from *P. vampyrus* (fig. 1). Although we sequenced several individuals of *P. vampyrus* sampled from different locations, none of our sequences contain the first 1 bp insertion that was discovered from the draft genome sequence (supplementary fig. S1, Supplementary Material online). Thus, the first 1 bp insertion is probably a sequencing error in the low-coverage (2.6×) *P. vampyrus* genome sequence, which makes the locations of premature stop codons different between the newly obtained sequence and the genome sequence (fig. 2 and supplementary fig. S1, Supplementary Material online). In *Megaderma spasma*, we identified a very long deletion (126 bp) unshared with any other species. We found no ORF disruption that is shared by all seven amplified yinpterochiropteran *Tas1r1* genes, suggesting the possibility that multiple independent pseudogenizations occurred in Yinpterochiroptera. Alternatively, there might be a single pseudogenization event in the common ancestor of all yinpterochiropterans, but no common ORF-disrupting substitution occurred in exon 6 because of the limited length of the exon and/or the potentially short time between the pseudogenization event and yinpterochiropteran divergence.

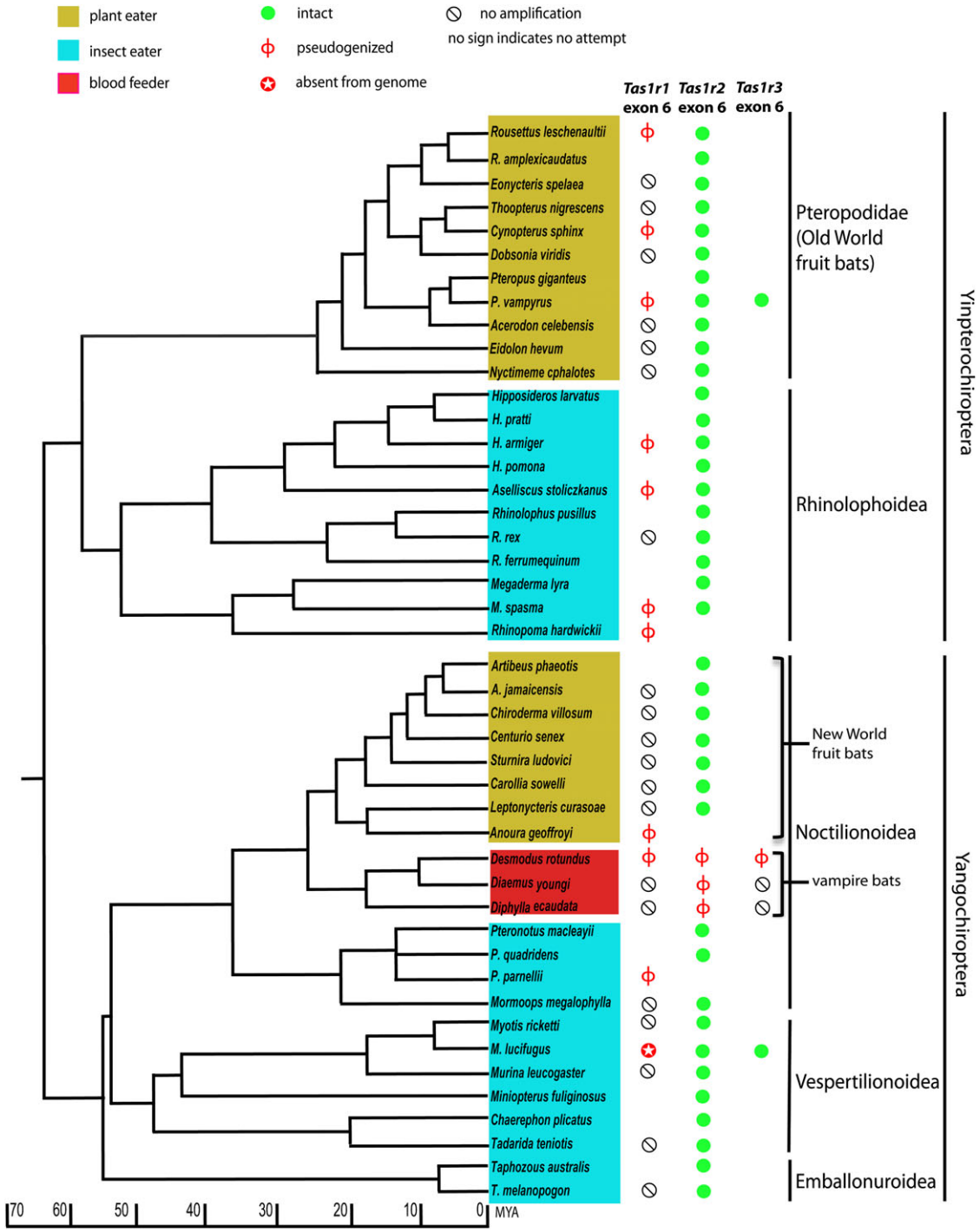

Fig. 1.—The species tree of the bats examined for *Tas1r* taste receptor genes. The phylogeny and divergence times follow Teeling et al. (2005). Dietary preferences are indicated by various colors, and the functional status of the *Tas1r* genes is also indicated. *Tas1r1* is umami specific, *Tas1r2* is sweet specific, and *Tas1r3* is used for both tastes. The *Tas1r2* data are from Zhao, Zhou, et al. (2010), whereas those of *Tas1r1* and *Tas1r3* are from the present study.

There are three superfamilies in Yangochiroptera, but all three amplified yangochiropteran *Tas1r1* sequences are from the superfamily Noctilionoidea. One common large deletion (92 bp) and a shared premature stop codon were observed among the three sequences, suggesting that the *Tas1r1* pseudogenization predated the radiation of Noctilionoidea. In the superfamily Vespertilionoidea, the draft genome sequence of *M. lucifugus* suggests the loss of

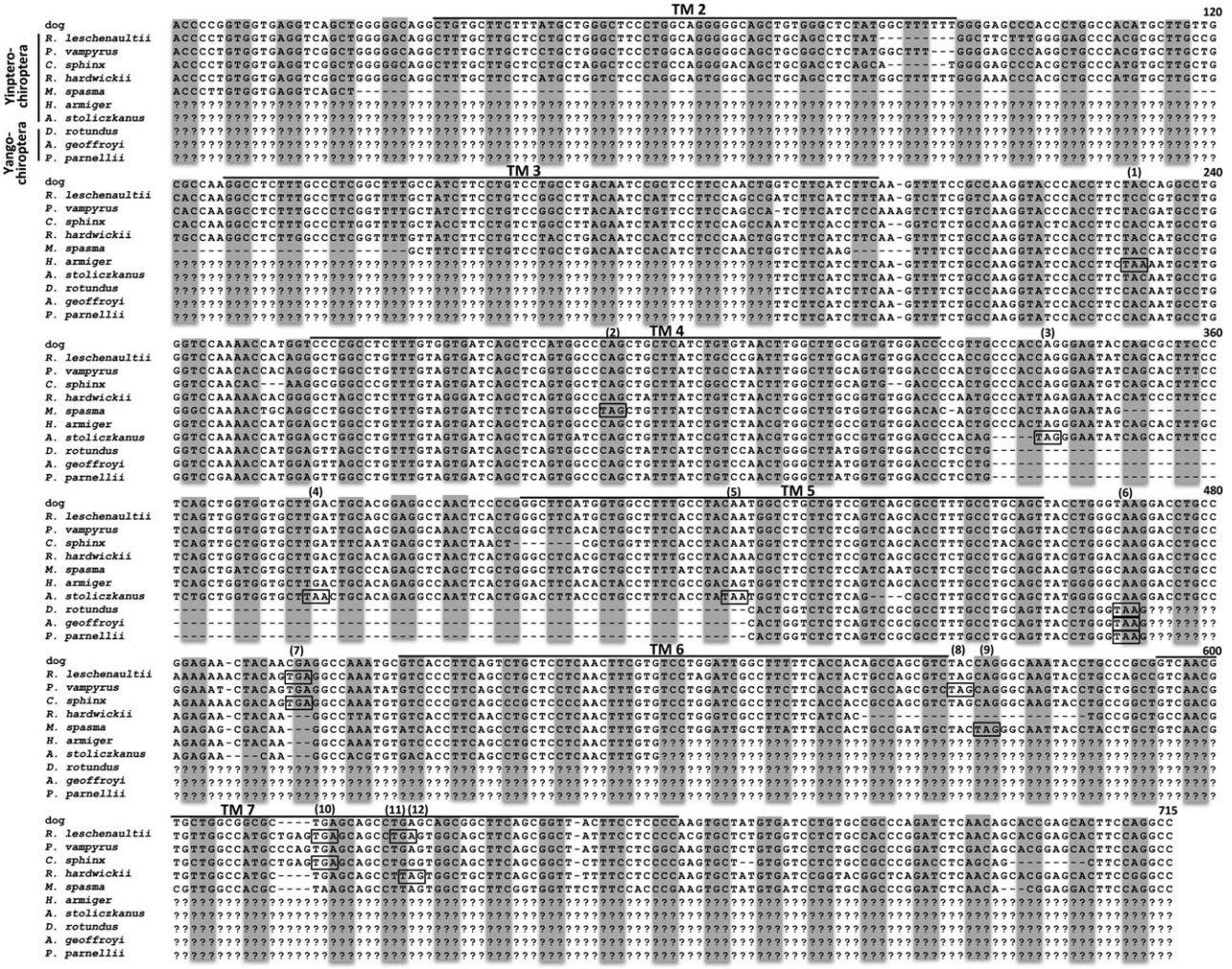

Fig. 2.—Alignment of a fragment of *Tas1r1* exon 6 from 10 bats, with dog *Tas1r1* used as a reference. Dashes (–) indicate alignment gaps and the question marks (?) represent the nucleotides that are not sequenced. Codons in the correct reading frame are indicated by shading and the premature stop codons are boxed. Premature stop codons created by nonsense mutations should appear in the correct reading frame, whereas those created by the indels should appear in an incorrect reading frame. The numbers in parentheses indicate the order of premature stop codons. Regions corresponding to the transmembrane domains (TM2–TM7) are indicated. See figure 1 for the full species names.

Tas1r1, as aforementioned. In Emballonuroidea, the third superfamily of Yangochiroptera, we failed to amplify *Tas1r1*.

As mentioned, we could not amplify *Tas1r1* from 20 bat species even after trying multiple primer pairs. Because an evolutionarily conserved sequence is easier to amplify than an unconserved one, the lack of amplification most likely indicates either a loss or a severe degeneration of the gene in these species. In other words, it is likely that none of the 31 bat species examined has an intact *Tas1r1*. Based on our wide taxon sampling, we conclude that *Tas1r1* is lost or pseudogenized in most, if not all, bat lineages. Because of the essential role of Tas1r1 in umami taste signal transduction, demonstrated by targeted gene deletion in mouse (Zhao et al. 2003), we infer that most, if not all, bats have lost the umami taste. However, whether the absence of an intact *Tas1r1* in bats was caused by one pseudogenization event or multiple events cannot be unambiguously determined, due to the lack of any common ORF-disrupting substitution in the 10 partial coding sequences obtained. In the past, we were able to date pseudogenization events in a number of other cases (Zhang and Webb 2003; Wang et al. 2004; Wang et al. 2006; Zhao, Yang, et al. 2010). But, we are unable to do so for bat *Tas1r1* because we only have the sequences of one of the six exons, whereas our dating requires inferring the timing of the first ORF-disrupting substitution in the entire gene.

Why Is the Umami Taste Dispensable in Bats?

The absence of the umami taste in vampire bats (genus *Desmodus*) is not unexpected because the sense of taste in vampire bats is generally poorly developed; they do

not even learn taste aversion, which is crucial for avoiding the ingestion of harmful food (Ratcliffe et al. 2003). Because vampire bats are blood-feeding specialists, the extremely narrow diet may have rendered their tastes unimportant. Furthermore, vampire bats use olfactory cues to locate preys (Bahlman and Kelt 2006) and use infrared sensors to locate blood flows close to the skin (Kishida et al. 1984; Fenton 1992; Gracheva et al. 2011). The acquisition of the infrared sensory system by vampire bats may have further diminished the importance of taste perception. This said, we note that the loss of *Tas1r1* apparently predated the origin of vampire bats (fig. 1) and thus cannot be a consequence of their unique feeding behavior.

In addition to the loss of the umami taste, our previous study (Zhao, Zhou, et al. 2010) discovered that the sweet receptor gene *Tas1r2* has been pseudogenized in all vampire bats but not other bats (fig. 1), consistent with an earlier behavioral study that found common vampire bats (*D. rotundus*) indifferent to high concentrations of sugar (Thompson et al. 1982). Because both *Tas1r1* and *Tas1r2* are nonfunctional in vampire bats, *Tas1r3*, which is required for both the sweet and umami tastes, must be useless. We attempted to amplify exon 6 of *Tas1r3* from each of the three vampire bat species but were able to amplify it only from *D. rotundus* (fig. 1). Indeed, we found its ORF disrupted by multiple deletions and three premature stop codons (supplementary fig. S2, Supplementary Material online). Specifically, the first premature stop codon is located in the fourth transmembrane domain, which would lead to a truncated Tas1r3 without three transmembrane domains and the C-terminus (supplementary fig. S2, Supplementary Material online). This finding contrasts the identification of an intact *Tas1r3* from the genome sequences of both *P. vampyrus* and *M. lucifugus* (fig. 1). Our results about *Tas1r3* further confirm the losses of umami and sweet tastes in vampire bats. To our knowledge, vampire bats are the first reported mammalian group to have lost two basic tastes. Future work is needed to assess the other three basic tastes in vampire bats.

By contrast, the absence of the umami taste in non-vampire bats is surprising. Our previous study (Zhao, Zhou, et al. 2010) showed that the sweet taste receptor gene *Tas1r2* is conserved in non-vampire bats (fig. 1). A number of *Tas2r* bitter taste receptor genes were also reported in a non-vampire bat (Zhou et al. 2009). Behavioral studies showed that plant-feeding and insect-feeding bats have strong ability to learn taste aversion (Ratcliffe et al. 2003). Why do bats specifically not require the umami taste, a basic taste that is conserved in almost all other mammals according to *Tas1r1* analysis (Shi and Zhang 2006)?

As mentioned, only one mammal, the giant panda, was known to have lost *Tas1r1* and the loss approximately coincided with the panda's dietary shift from meat to bamboo (Li et al. 2010; Zhao, Yang. et al. 2010). But all bats, regardless of their diets, have lost *Tas1r1*. Furthermore, the earliest bat fossil, *Onychonycteris finneyi*, that lived ~52.5 Ma was insectivorous (Simmons et al. 2008). So, it is unlikely that primitive bats were vegetarians. Bats are distinct among mammals in their ability to fly. But the ability to fly seems unrelated to the umami taste because we found *Tas1r1* intact in a number of birds, including chicken, turkey, zebra finch, egret, loon, and tubenose seabirds (Zhao H, Zhang J, unpublished data). Many bats use echolocation to orient and forage, whereas OW fruit bats do not echolocate (Jones and Teeling 2006). However, OW fruit bats lack the umami taste as the other bats do. Hence, it remains unanswered why the umami taste is dispensable in non-vampire bats. In the future, a better understanding of the physiological functions of *Tas1r1* and the umami taste in nature may help answer this question.

It should be mentioned that, while the Tas1r1 + Tas1r3 heterodimer is undoubtedly the predominant receptor for the umami taste (Behrens and Meyerhof 2011), there were conflicting reports of whether mice lacking the heterodimer possess residual umami sensitivity (Damak et al. 2003; Zhao et al. 2003). If the residual sensitivity truly exists, it has been suggested to be mediated by metabotropic glutamate receptors (brain-mGluR1 and its truncated variant taste-mGluR1; brain-mGluR4 and its truncated variant taste-mGluR4) that also perform other brain functions (Yasumatsu et al. 2009). In humans, the Tas1r1 + Tas1r3 heterodimer appears to be the sole umami taste receptor (Yasumatsu et al. 2009). As expected, we found intact *mGluR1* and *mGluR4* in the genome sequences of both *P. vampyrus* and *M. lucifugus*, but the lack of their expression information prohibits us from inferring the possibility of a residual umami taste in bats. Regardless, the absence of a functional *Tas1r1* gene suggests that the umami taste is completely or almost completely lost in bats, which should be behaviorally verified in future.

Materials and Methods

Among mammals with high-coverage genome sequences, the dog is phylogenetically the closest to bats (Murphy et al. 2007). We thus first identified the dog *Tas1r1* (GenBank accession no. XM_546753) from its genome sequence (7.6× coverage) and then used it as a Blast query to identify the corresponding gene in the genome sequences of *P. vampyrus* (2.6×) and *M. lucifugus* (1.7×). Based on the *P. vampyrus* sequence, we designed a pair of primers (TR1LF: 5′- CTG TTT GCC TGG CAC TTA GA-3′ and TR1LR: 5′-GCA GAG GAC CAC AGA GCA C-3′) to amplify exon 6 of *Tas1r1* in various bats. When this primer pair did not work, we used the forward primer TR1SF3 (5′- TCT TCA TCT TCA AGT TTT CTG CCA A-3′) and either TR1SR3 (5′-CCT TCA GCC TGC TCC TCA ACT TYG TG-3′) or TR1SR4 (5′-CCT TTG CCT GCA GYT ACC TGG GYA AG-3′) as the backward primer. The two degenerate backward primers were designed based on an alignment of the

publicly available *Tas1r1* sequences from human, mouse, dog, cat, and fox. We also tried several additional degenerate primers when the above primers did not work, but none of the additional primers worked. To examine *Tas1r3* in the common vampire bat, primers TR3VF (5′-GTG TGA CGA GGA CAA GTG GT CC-3′) and TR3VR (5′-ACG CCC TCC CAG GAA GAA CTC-3′) were used. Bat tissues were collected over the years and the genomic DNAs were isolated using the DNeasy Blood & Tissue Kit (Qiagen). Polymerase chain reactions (PCRs) were performed with *Taq* DNA polymerase (Takara). Each PCR mixture (50 μl) contained 1 μl (50 ng/μl) genomic DNA, 25 μl 2× buffer, 7.5 μl (50 mM) $MgCl_2$, 5 μl (10 μM) of each primer, and 1 μl (5U/μl) Taq DNA polymerase (Takara). PCR reactions were conducted as follows: 5 min of initial denaturation, 30 cycles of denaturation at 94 °C for 30 s, annealing at a temperature gradient of 45 °C to 58°C for 30 s, extension at 72 °C for 60 s, and a final extension at 72 °C for 5 min. PCR products were gel purified and cloned into the pMD19-T vector (Takara). Clones were grown on agar plates containing 100 μg/ml ampicillin. Plasmid DNA was purified using Quick Plasmid Miniprep Kit (Invitrogen). Multiple plasmids (3–5) from a single PCR product were sequenced in both directions by the Sanger method, using an ABI DNA sequencer with the sequencing primer pair (M13-47:5′-CGC CAG GGT TTT CCC AGT CAC GAC-3′ and M13-48:5′-GAG CGG ATA ACA ATT TCA CAC AGG-3′). All newly acquired sequences have been deposited into the GenBank (supplementary table S1, Supplementary Material online). DNA sequences were aligned with CLUSTAL_X 1.81 (Thompson et al. 1997) after the removal of primer regions.

Supplementary Material

Supplementary figures S1 and S2 and table S1 are available at *Genome Biology and Evolution* online (http://www.gbe.oxfordjournals.org/).

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