

## Why are chillies pungent?

Early voyagers to the Americas, including Central America, Mexico, Peru, and Chile, found many forms of peppers, among them the 'hot' (pungent) ones. In Spain these hot peppers are called chili, meaning from Chile, and in India, chillies. When asked to guess the source of chillies, one might think of Mexico. However, despite the plant's popularity in that country, it is believed that chillies – or, to use the misleading but widely used name by which they are called in the United States, chili peppers – originated in South America, after which they spread to Central America. Most of the varieties of pepper referred to as chili peppers belong to *Capsicum annuum* L; some varieties with "chili" in their name are actually *C. frutescens* L. Precise categorization can be difficult because of the large number of varieties and the constant creation of new ones by hybridization. Their weedy nature, combined with the easy transportability of their seeds, made chili peppers among the first plants to be domesticated (Andrews 1984). Remains found in Tehuacan, Mexico, have been dated to approximately 7000 BC. Columbus mistook the chili pepper for a relative of black pepper, *Piper nigrum*, whence the term 'pepper' (Robbins 1992).

Dr Diego Alvarez Chanca, who accompanied Columbus on his second voyage, described chili peppers as the principal food of native Americans and compared them to the turnip (Andrews 1984). The seeds of the chili pepper were brought back to Spain and the plant was grown in monastery gardens. Portuguese traders spread it to many countries including Persia, India and Indonesia (Andrews 1984). The earliest reference to chillies in India is in a poem in Kannada by Purandara Dasa (1480–1564) that extols its praises; the *Bhojana Kutuhala*, a Marathi text on the enjoyment of food by Raghunatha (1650), also refers to chillies. But "... not a single recipe of over fifty given in the *Ain-i-Akbari* of 1590 used anything except black pepper to impart pungency" (Achaya 1994). While most people know the chili pepper as a food, it had other uses in ancient times. The pre-Columbian Indians used them as a medicine, as a punishment for children (inhalation of the smoke of burning chili peppers), and as a kind of tear gas during warfare (chili peppers were burned and the smoke blown by the wind over to enemy lines) (Andrews 1984). The basic sensation produced by chili peppers is extreme pungency or 'heat'.

The substance that produces all of the heat sensation is known as capsaicin (N-vanillyl-8-methyl-6-(E)-nonamide). Capsaicin is made by specialized gland cells found in the cross-walls or ribs of the pepper and is composed of several different alkaloids which vary in amounts depending on the species (Rowland *et al* 1983). Wilbur Scoville developed a scale in 1912 to measure the "heat levels" of chilli peppers. In the original Scoville test, a panel of volunteers would be asked to determine what dilution of the chilli pepper solution no longer cause burning discomfort in the mouth. Approximately one part per million of "heat" is equivalent to 1.5 Scoville units. Until recently, the hottest chilli pepper ever recorded was a Habanero which had 577,000 Scoville pungency units while in contrast the fiery Jalapeno has between 2,500 and 10,000 units, and in complete contrast the Sweet Italian Bell Pepper has a pungency of 0 units (Bellringer 2001). Indian scientists have recently claimed that a type of chilli grown in the country's northeast has the highest Scoville units of pure capsaicin. Called the Tezpur chilli, after the area where it is grown, scientists say the pepper has beaten Mexico's Red Savina Habanero. "The Tezpur chilli was rated having 855,000 Scoville units . . . the Mexican chilli contained 557,000 Scoville units of pure capsaicin," one of the scientists, who asked not to be identified, told Reuters (<http://www.cnn.com/2000/FOOD/news/09/04/india.chilli.reut/>). Pure capsaicin has a pungency of 16 million Scoville units. Thankfully for the tasters, the original Scoville taste test has given way to HPLC measurements.

The sensations of heat and pain in the mouth are the result of the stimulation of local heat receptors in the skin and mucous membranes by capsaicin, providing one answer to the question posed in the title. The capsaicin (vanilloid) receptor VR1 is a sensory neuron-specific ion channel that serves as a polymodal detector of pain-producing chemical and physical stimuli. Capsaicin is a trigeminal stimulant that is important in gustatory physiology (Liu and Simon 2000). Interestingly, capsaicin can also help in the mediation of pain: prolonged application of capsaicin is thought to cause the desensitization of sensory neurons responsible for pain. This might occur via the depletion of substance P, a peptide neurotransmitter in sensory "pain" fibres (Goettl *et al* 1997), the final outcome being the release of

B-endorphins which are endogenous opioids. Capsaicin can induce sweating, which is why chillies are popular in hot dry climates. Further, it stimulates the actions of the muscles of the stomach and intestine; this improves digestion and makes chili peppers an attractive condiment for a food that might upset the stomach (Andrews 1984). Most importantly, it appears that capsaicin was developed by plants as a way of preventing animals with digestive systems that can destroy chili pepper seeds from eating them, while allowing animals who will pass the seeds to eat them with no ill effects (Robbins 1992; Tewksbury and Nabhan, 2001). That may be the evolutionary explanation of why chillies are so pungent.

Surprisingly, most of the work on pain-induced by capsaicin had concentrated on mammals, with very little work on gustatory responses in birds. Recently Bryant *et al* (2000) cultured trigeminal nociceptors (pain receptors) from the Norway rat (*Rattus norvegicus*, laboratory strain), white leghorn chicken (*Gallus gallus*), coyote (*Canis latrans*), white-tailed deer (*Odocoileus virginianus*) and Canada goose (*Branta canadensis*) and then applied digital fluorescence microscopy to measure changes in intracellular calcium (an index of cellular activation) in response to applications of known and effective repellents. They found that capsaicin was a more effective stimulus for rat, coyote, and deer neurons than cells from chicken. Does this mean that birds do not have capsaicin-stimulated or vanilloid-receptors in their oral linings, or do they have potent antagonists?

The limited effect of capsaicin on birds appears to be why capsaicin is now believed to cause directed toxicity or directed deterrence of potential mammalian seed predators in the chiltepine chilli plant (*Capsicum annuum* var. *glabriusculum*) in southern Arizona, while having no effect on seed-dispersing birds, the curve-billed thrashers (*Toxostoma curvisrostre*) (Tewksbury and Nabhan 2001). To find out whether small fruit-consuming mammals such as cactus mice (*Peromyscus eremicus*) and packrats (*Neotoma lepida*) avoid chillies because of their capsaicin content, the authors presented these mammals as well as desert thrasher birds with the pungent chilli fruit (*C. annuum*), fruit of a non-pungent mutant variety of *C. chacoense* which is similar in all other aspects to the fruit of *C. annuum* except in lacking pungency, as well as desert hackberry fruit (*Celtis pallida*) as a control. They found that while the birds consumed all three types of fruits equally, the mammals consumed no pungent chilli fruit, an intermediate amount of the non-pungent chilli fruit and all the hackberry fruit. It appears that the capsaicin in the chiltepine chilli fruit deters consumption by mammals. Furthermore, germination trials of *C. chacoense* fruit (non-pungent chilli) showed that there was zero germination following gut passage through the mammals, while germination levels following gut passage of chiltepine seeds and non-pungent chilli seeds through the birds were excellent and comparable with that of control seeds taken directly from the fruit and planted. If seeds can germinate just as well with and without passage through bird guts, what is the advantage to being consumed by the birds? Tewksbury and Nabhan (2001) found that birds that consumed chilli fruit are more likely to deposit these seeds in shaded sites suitable for germination. This fact coupled with the zero germination on passage through mammalian guts can explain why the chiltepine chilli plant “wants to encourage consumption” by the thrasher birds and to deter consumption by mammals.

Some years ago, Cipollini and Levey (1997a, b) proposed two sets of hypotheses to address patterns of secondary metabolites in ripe fruit pulp. The first set of hypotheses dealt with toxicity of the metabolites. The compounds could either have directed deterrence i.e. be targeted towards specific harmful consumers, or have general toxicity, wherein retention of toxins in ripe fruit is a manifestation of a trade-off between seed defense and frugivore attraction. The second set of hypotheses addressed whether fruit removal rates and fruit pulp nutrient content could explain the secondary metabolite patterns. Several workers have since then attempted to test these sets of hypotheses. Tewksbury and Nabhan (2001) believe they have for the first time shown directed toxic deterrence in a fruit, and how this may influence the interaction between plants and their fruit-eating visitors.

Have we finally understood why chillies are so pungent? Have mammalian seed predators and their VR1 nociceptive oral receptors been the driving force behind capsaicin evolution? Interestingly, an ultrapotent analogue of capsaicin, resiniferatoxin, is found in the latex of the succulent spiny shrub *Euphorbia resinifera*, and its use in pain mediation has been known since Roman times (Appendino and Szallasi 1997). Resiniferatoxin has been shown to have acute emetic effects in the house musk shrew (Andrews *et al* 2000). It would be worth investigating whether a similar selective pressure may explain the origin and maintenance of this potent vanilloid too.

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