Catnip

Nepeta cataria
family *Lamiaceae*

Catnip in Door County

There are around 300 species of *Nepeta*, or "catmints", and some new species are still being found in the Old World, where they originated. Many wild and cultivated forms are found in Door County. Like other mint family plants, it grows exuberantly. Now this Mediterranean import is a common roadside weed throughout the United States. I find it in abandoned fields, everywhere I go. Fortunately it does not seem inclined to form a monoculture, but grows in isolated patches, respecting other flora. If you aren't certain you have catnip, try my method: pinch off a piece, and present this to your nearest cat for identification. They will quickly let you know if you are correct.

Cultivated versions have lovely lavender flowers and blue-green foliage, and are an easy garden perennial to grow in Door County. According to local naturalist Roy Lukes, their tubular flowers attract hummingbirds, too.

History and Folklore

Catnep, catmint, and catswort are common names for catnip. Despite these names' obvious allusion to cats, the plant's power to amuse cats was strangely regarded with skepticism by the 1834 drug authority, *The United States Dispensatory*. It took over 50 years for the reference to admit, “Cats are very fond of it.” Humans are also traditionally fond of it.

In the ancient Roman city of Nepete, which gives the herb its Latin name, catnip was grown for food and seasoning. European gardeners later popularized the herb, which was used in salads, stews, and on meat. Many species are still used widely in the Middle East and Mediterranean as a food and herb. (Much of the research that I found on this plant came out of labs in Iran, Italy, and Turkey.) Catnip has a minty, sweet, lemony smell, and resembles mint, with its square stem, opposite leaves, and white or purple flowers.

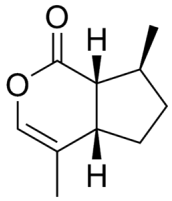
Catnip’s ability to launch cats into a euphoric frenzy is due to a molecule called nepetalactone. Cats have a receptor for nepetalactone in their noses. The existence of any particular cognitive effect in humans is not obvious, or at least it is a lot more subtle. According to the current scientific literature, we probably do not have receptors for nepetalactone in our noses or anywhere else. Although catnip excites cats, it has been used in traditional cultures around the world to calm both humans and livestock. Macerated poultices are used as folk remedies for contusions and inflammation. According to worldwide folk wisdom, catnip promotes sleep, calms nerves, and soothes upset stomachs.

An aggravating aspect of science is that, once an authority pronounces an error, it becomes practically impossible to erase that misconception from popular culture. A 1969 *Journal of the American Medical Association* article mistakenly labeled catnip as marijuana, confusing the effects of the two plants. Despite 1,612 letters to the beleaguered editor pointing out the error, the retraction failed to sway members of the 70’s drug counterculture, who enthusiastically commenced smoking catnip. This was, no doubt, anticlimactic, because it does not have any

effect like marijuana. There is, however, some preliminary evidence from non-cat animals that its historic use as a calming agent may be justified.

What Molecules From Catnip Do

To be vulnerable to catnip's cat-intoxicating effects, you need two things. But humans don't have either of these, as far as we know. The first is a functional *vomeronasal organ*, or VNO. The VNO is in many vertebrate animals' noses, with the exception of birds and fish. It picks up chemical *pheromones*. These chemicals signal behavioral clues, like alarm, food, or mating readiness and arousal. Catnip contains a pheromone that cats respond to, called nepetalactone.



Nepetalactone (the 4aα,7α,7aα version)

Although humans have a rudimentary VNO, it has apparently degraded through lack of use, thanks to our ability to communicate using words and quite a variety of other means. Nerves from our VNO appear to dead-end, failing to reach the brain. There is some controversy about how well we respond to pheromones. Some scientists say that, if we *do* respond to them, receptors in your everyday sniffer, the olfactory bulb, may have taken over the job of the VNO, to a slight extent.

The second thing you need is a nepetalactone *receptor* in your VNO. We don't have this either, but about 80% of cats do (though I question this; see my editorial text box), which explains why they need only smell the plant to become engrossed. When nepetalactone binds to its receptor, sensual pleasure areas in the cat brain are stimulated. If you are a cat lover like myself, you can testify that cats try to saturate their entire bodies with the plant, rubbing and licking it ecstatically. Without a nepetalactone receptor, we can only enjoy this pleasure vicariously. Catnip might, however, affect different receptors in our own brains: there is evidence it interacts with opiate and GABA receptors in rodent brains, at least. It remains to be seen whether the human version of these receptors are also effected, since ours are not the same.

[begin text box]

It's nepetalactones, plural, actually, and they are not just in catnip

Actually, if you read the scientific literature, you will quickly come across the term *nepetalactones,* plural. Any molecule that has the same atoms bonded in the exact same way to each other in the picture above is called a nepetalactone. However, the *directions* that their bonds point relative to each other may assume different three dimensional orientations in space, so there are different versions or *isomers* of this molecule. Most molecules are not flat, despite our depictions of them on flat surfaces like paper and computer screens. It is a molecule's average

three dimensional shape, if it is relatively constant, that dictates much of the activity of the molecule. Thus, if one nepetalactone is not active, another one might well be more active.

One of the most common nepetalactones in catnip is depicted in the drawing above. Notice the dark wedge shaped lines on the structure. In chemistry shorthand, the wedges indicate the fatter end of the wedge is closer to you, in space, than the thinner end, and a wedged bond is used to indicate the atom at the end of it is coming out of the page *toward you*. (Hatched lines are used to indicate bonds going away from you, into the paper.)

This is only one possible nepetalactone, but others exist, where any one or all of these three wedged lines are going in the opposite direction, going into the page. This creates a variety of molecules with the same connection pattern between the same atoms, but different shape in space. This is very common in molecules, analogous to having a right glove and a left glove. While the gloves share the same basic structure, you know they are different if you try to put the left one on the right hand--it doesn't work! These different shapes of the same molecule are called *stereoisomers*.

A little math formula chemists learn to predict the number of stereoisomers possible for a particular molecule first counts the number of *chiral centers*. These are locations in the molecule where a mirror image of that region would not be the same as the original--like left-handed and right-handed gloves. If you have n chiral centers, you have 2^n different stereoisomers possible. Since nepetalactone has three chiral centers, it has 2^3 versions of itself. That's 2 x 2 x 2 = 8 nepetalactones. The same thing would happen if you had three hands, you could be right right right, or left left left, or right left left, and so on...up to eight possible versions of you.

Just because we can calculate the theoretical number possible does not mean all eight exist in nature. Much of the biological machinery used to make these molecules, like enzymes inside plant cells, are *also* three dimensional, and as such can be expected to churn out one version of a chiral center, but not another. Indeed, one of these theoretical eight molecules (the 4aβ,7β,7aβ version and mirror image of the one above) has not been found in nature yet, but has been synthesized in a lab[1]. The form of the molecule matters, because of these eight versions of nepetalactone, a some appear to be more active than others, at least in rodents, at relieving pain and anxiety, and promoting sleep.

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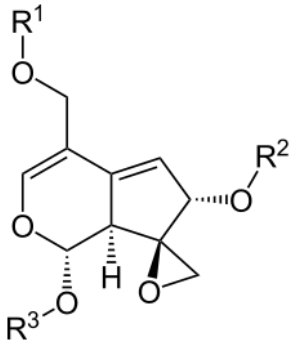
Not quite nepetalactone but close to it

Furthermore, there are other molecules in catnip that are not nepetalactones but are structurally related, with a few extra or missing bits, with names like dihydronepetalactone and nepetalic acid. These could have similar effects, or affect nepetalactone's activity when in a mixture. If you still can't get enough nepetalactone-like molecules, consider that other plants besides catnip make them as well. Other mint family plants like the toxic pennyroyal (*Mentha pulegium*) also contain the molecule. Even some unrelated plants like tartarian honeysuckle (*Lonicaera tatarica*) make nepetalactone; its bark shavings are often used as a catnip substitute in cat toys. In addition, other plants make molecules that resemble nepetalactone. Valerian, unrelated to catnip, contains molecules called valepotriates that resemble nepetalactone closely enough such that some cats like valerian, but most do not. Some cats even prefer valerian to catnip, to their owner’s surprise.



A valepotriate from valerian. Notice how, if you flipped this picture upside down, the six-sided and five-sided rings would look like those of nepetalactone, above.

In humans, valerian is said to have a mild sedative or anti-anxiety effect. Hard evidence that valerian sedates humans is currently disappointing, however. Nonetheless scientists seeking a mechanism for this debatable effect postulate that valerian's valepotriates enhance the action of GABA, an inhibitory, sedating brain neurotransmitter. Is it just a coincidence that cultures around the world have used both valerian and catnip as sedatives?

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Cat naps for rodents and chicks

Cats might like catnip even better if they knew that there are a number of studies showing it puts rodents[2,3,4,5], and in one study, chicks[6], to sleep faster. However, this generally requireds injecting rodents intraperitoneally with a diluted extract of some species of *Nepeta* in doses ranging from 50 to 100 mg/kg, something cats are unlikely to manage.

Stimulating in higher doses

Furthermore, a few of these studies note that the higher doses appear stimulant, preventing sleep in both rodents and chicks. In mice a 100 mg/kg dose of *Nepeta persica* extract "increased the locomotor activity" which, given what I've seen of an average lab mouse's activity level, must have been impressive. One paper describes an "inverted U shaped curve" for *Nepeta* extracts' ability to put rodents to sleep. It will be interesting to learn how that mechanism works on a molecular level.

Pain relief for rodents, too

Several of these studies note that these extracts decrease both "peripheral and central" pain in these animals. This means that the extract appeared to work both in the brain and at the source of the discomfort, like a limb or a tail. Mild sources of discomfort, like heat, irritating chemicals, or mechanical stimuli were used. Then the ability of injected *Nepeta* extracts to keep the animal from flicking its tail or moving away from a heat source was noted. The extracts

seemed to work for everything but thermal discomfort. This distinction might provide a clue to which receptor in the brain was working to block the pain.

Calmer mice

A standard anxiety test for mice, using an elevated maze, which they don't like much, was used to determine whether *Nepeta persica* extracts made it any easier for them[3]. At a Goldilocks zone dose somewhere between too low (which was sedative) and too high (which was stimulating) the intermediate 50 mg/kg dose succeeded in helping the mice face their fears.

What is the active ingredient? Lots of things.

Something from *Nepeta* appears to be affecting these animals' brains, but what, and how? The active agent in from is not pinned down in these studies. There is most likely more than one, in any case, which probably complicates finding it. Instead, some studies correlate the abundance of some ingredient with the most effective preparation in a study, and the active agent would be guessed from that. These are usually various nepetalactones, but also other terpenes, like epinepetalactone, ursolic acid, linalool, and eucalyptol. These other non-nepetalactone molecules are quite common in other plants, as well.

Opiate receptor binding, specifically, the non mu kind

In a few of these studies, the sedative and pain-relieving activity was blocked by naloxone. Naloxone binds to opioid receptors in the brain, preventing them from being activated. Thus the researchers theorized that something in the catnip oil was interacting specifically with opiate receptors in the brain. There is more than one type of opiate receptor in the brain, with names like mu, delta, gamma, and nociceptin, each possessing different pain-relieving and calming powers. Because the extracts failed to provide pain relief for thermal discomfort, the researchers further surmised that non-mu opiate receptors are affected by the plants' extracts.

An effect on GABA receptors in mice

GABA, gamma-amino butyric acid, is a brain signaling molecule that mostly inhibits brain activity, and can promote sleep and calm. Something in *Nepeta* extracts appears to have some influence on this system, at least in mice. What does this mean? Well, other drugs that act on GABA are benzodiazepines; the best recognized one is valium. I think if catnip acted like valium in people we would all know that by now, but there could, in theory, be a more subtle process in effect.

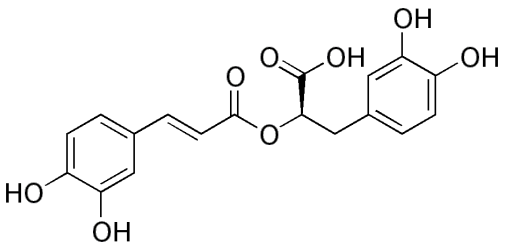
Benzodiazepines enhance GABA receptors in the brain such that they are more strongly influenced by the brain-calming effects of GABA. However, benzodiazepines have limited, short-term effectiveness, with serious side effects and a problem of increasing tolerance, in treating insomnia, anxiety, and seizures. So, other molecules with fewer side effects with action on GABA would be nice to find.

Italian researchers found that extracts of *Nepeta sibthorpii*, ("Greek catmint"), prevented mouse seizures[7]. This effect stopped when they co-administered a benzodiazepine blocker, suggesting that the catmint extract enhanced the action of GABA at its receptor. They correlated this seizure-stopping activity with the extract's epinepetalactone content.

Keeping GABA from breaking down

GABA transaminase is an enzyme that keeps this molecule from building up; it breaks GABA down. So, one way to induce calm and sleep is to inhibit GABA transaminase, which allows GABA levels to increase. There are molecules common to mint plants like catnip that

inhibit GABA transaminase. Rosmarinic acid, found in catmints, but also dead common in many mint plants like--as you may expect--rosemary, but also in lemon balm, bee balm, basil, sage, thyme, and marjoram, peppermint, spearmint, heal all, lavender, and quite a variety of other plants, appears to have GABA transaminase inhibiting activity. Catmints may not have a lot of rosmarinic acid compared to other plants (Heal all and oregano appear to be one of the best sources[8]) but what it does have could, in theory, contribute to its calming, sedative effect.



Rosmarinic acid, a GABA transaminase inhibitor

Anti-inflammatory action linked to calcineurin inhibition

A number of papers found that in animals, *Nepeta* extracts prevent inflammation. One study pinned down minor constituents of *Nepeta cataria*, which are not even nepetalactones, that inhibit a pro-inflammatory molecule called calcineurin[9]. Better known anti-inflammatory calcineurin inhibitors are cyclosporin, used to prevent organ transplant rejection, and some topical eczema, dermatitis, and ulcerative colitis medications.

Calcineurin does its dirty pro-inflammatory work by activating T-cells to start producing inflammatory molecules such as TNF-α and IL-2. This is fine in a crisis but does damage when the process goes overboard. Lamiuside A and verbascoside from catnip inhibited calcineurin in test tube studies. Before you get all excited about this, realize that these molecules, and others of their class (caffeoyl phenylethanoid glycosides, and no, they have no relationship with caffeine) are common in plants, and other unrelated plants might have even more of these agents. So, although many constituents of plant oils can be irritating to the skin, (particularly small fragrance molecules like monoterpenes,) it is interesting to note that at least *some* components might act like topical anti-dermatitis medications, and relieve inflammation.

Why would plants bother to make something that inhibits an inflammatory molecule in people? Well, calcineurin is actually found in most cells (eukaryotes) including fungi, and fungi depend on calcineurin to prevent membrane stress. Plants have likely evolved calcineurin-inhibitors to kill pathogenic fungi. So, these molecules may be expected to have some fungicidal activity, as well.

Relaxing muscles you can't control on your own might help you breathe and digest better. Like other mint plant oils, the presence of small terpenes probably helps the involuntary muscles surrounding a spasmodic gut or throat to chill. A normal influx of calcium ions into these muscle cells makes them contract. For the gut, this contraction is required to push items along their route through the gut, but when these muscles act up and get out of synch, pain and gas result.

In the respiratory system, spasmodically contracting involuntary muscles make breathing a strenuous act, too. There's good reason for traditional use of these plants with these fragrant oils for easing digestion and respiratory issues. Specifically, an extract of *Nepeta cataria* (rich in eucalyptol, alpha-humulene, alpha-pinene, and geranyl acetate) relaxed isolated, involuntary muscles from the gut and trachea[10]. The extract acted like a calcium channel blocker in these muscles, a mechanism discovered for other mint oils as well, like peppermint oil's menthol.

Catnip makes irritating iridoids for bugs. A satisfying number of nuisance insects--cockroaches, aphids, mosquitos, termites, and house flies--do not like catnip. In particular, studies report nepetalactone (the 4aα,7α,7aα and 4aα,7α,7aβ versions) repels them, in some cases, better than DEET[1].

One possible reason for this is that the nepetalactone structure is not just a pheromone for cats, but some insects, as well. This structure, known as an iridoid, is one that certain ants use to say "I'm not dead yet", chemically. This prevents other congenitally blind worker ants from robotically burying them, a valuable service to the colony when the buried ants are actually dead. So, it repels the ants from burying ants that are alive. I'm conjecturing here that this repulsion, mutated into a variety of signals in different insects, might be the mechanism behind nepetalactone's bug-repelling power, but as far as I know, the mechanism has not been pinned down. (One reference says that antennectomized insects are immune to catnip's repulsive force.) Catnip essence also contains a bit of citronellal, the active ingredient in citronella oil, renowned for repelling mosquitos.

Use and Misuse

Catnip is safe to ingest, just don't overdo it. Unlike many herbs, catnip has an almost completely unblemished safety record. There is only one documented report—that of a one-year-old boy who was taken to an emergency room after eating some questionable, old, fermenting food he had discovered, in addition to what was his mother claimed was catnip tea. (Whether or not it was really catnip was questioned.) He “looked drugged,” but recovered quickly[2]. Based on catnips' apparent effect on rodents' brains, however, you might not want go overboard in youngsters; moderation is always wise.

Take as tea or as a flavoring herb. Catnip’s relatively untarnished safety record allows most herbalists to unreservedly recommend a tea made from the leaves. This is either sipped to ease jangled nerves, or drunk before bedtime to ensure sleep. The tea is even given to infants to ease colic. If you are an adventurous cook, experiment with adding this traditional Middle Eastern herb to your cooking by sprinkling dried catnip on cooked food or adding it fresh to salads. Some varieties have a strong flavor so a little bit goes a long way.

Watch out if you have GERD. If you have gastroesophageal reflux, mint family plants like catnip can be your undoing. The relaxation of involuntary muscles caused by these plants makes stomach acid more likely to climb up a relaxed esophagus.

Be cautious with the essential oil. Plant essential oils are dangerous because they are concentrated, and should never be eaten in any way. Essential oils can be diluted with water and then used topically or in baths, but even then they are likely to irritate the skin.

Experiment with your own bug repellent. You can try strategically placing catnip oil in pest infested locations in your home or garden if you are OK with these spots becoming cat magnets. Some people place cooled catnip tea in a spray bottle as a mosquito repellent. (As someone allergic to mosquito bites and suffering greatly every Wisconsin summer, I am eager to give this DEET alternative a try as soon as the snow melts). Nepetalactone is reportedly a skin sensitizing agent, which makes its use as a topical bug repellent problematic. It might be best to put catnip-based bug repellents on your clothing, rather than your skin. You can buy commercial catnip-based repellents, too.

Don't smoke catnip. If smoke catnip to get a high, you will be disappointed. Lots of people tried it out of desperation in the 60's, but all it reportedly did was cause a lot of sore throats. Of course, inhaling smoke from *anything* causes a sore throat, and neither doctors nor firefighters advise it. Smoking is one of the worst things you can do for your cardiovascular system and lungs and will shorten your life. Why not just have a nice cup of tea?

[begin sidebar] Safe Storage

Enterprising cats can go to great lengths to obtain catnip. I once had a determined neighbor's cat break into my apartment, and open an elevated kitchen cabinet to steal catnip-containing tea bags. Seeing the cabinet open and things awry, I first thought I was the victim of a burglary, yet realized the intruder left muddy paw prints around a broken window screen, and half-chewed tea bags were scattered on the kitchen floor. "A cat has done this!" I surmised. I found my neighbor's cat intoxicated outside. If you have similar opportunist cats around you, you may want to store the leaves in a more impregnable location, like a refrigerator or freezer. This helps keep the herb's volatile oil from evaporating away, in any case.

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Who doesn't like catnip?

Scientific articles tell me that around 80% of cats are attracted to catnip. The gene for it is autosomal, that is, not located on a sex chromosome, and dominant. This means that only one copy, either from the tom or the queen, is required for the kitten to express the trait. It takes time to develop functional nepetalactone receptors. Kittens take a few weeks for this to occur, and may even avoid catnip before then. Other catnip lovers include lions, pumas, and leopards, but not tigers. In cats, it is nonaddicting, nontoxic, and causes no hangover. Cats generally sleep off the effects.

Because I am "gifted" with cat-taming powers, I have more experience with them than most people. The total number I've had under my feet throughout my almost fifty years of life probably adds up to over thirty. (I now have five, and though I love them dearly, I don't recommend more than two.) I know the scientific literature says that 20% don't respond to catnip, but I have yet to meet a single one of these theoretical non-responders. One article in Science and Engineering News even said that 50% of cats are non-responders to nepetalactone, and I find that difficult to believe. It isn't that I don't believe the existence of non-responders, but I can't imagine they are that common. It's true that young kittens start off ambivalent, yet quickly learn to relish it. Perhaps it is an acquired taste.

I once met a cat that I *thought* was, at long last, an example of the catnip ambivalent. First I should explain that I have never met a feral cat that I could not tame; but I don't know

whether or not to call this a gift. My husband calls me *The Cat Whisperer* after watching me in action for ten years. Along the bluff where we live along Green Bay, wild felines claw out a miserable living in the bitter winters, having litter after litter of kittens. These usually die from exposure or are eaten by coyotes. Meanwhile the starving cats ravenously eat up our beloved wildlife. (You can put a good dent in this predation by putting out piles of alternative cat food during the day and bringing it in at night to prevent raccoons and other nocturnal animals from gobbling it all up). These wild cats are terrified of people. I catch them with a humane trap, get them neutered and vaccinated, and bring them into my office where I write, also known as "the taming room".

So I end up with a lot of domesticated cats on my hands that I cherish but don't know what to do with. I am often lucky to find loving owners who promise to keep them indoors where the cats are not able to snack on the local wildlife. It's a win win win. It feels like the right thing to do.

One of the most frightened wild cats that I ever tamed perfected an unusual survival strategy that fooled me into thinking that she was immune to catnip. She assumed a striking state of complete paralysis after I caught her and had her spayed and vaccinated. Convinced she was blind or mortally ill, Carrie the vet examined her twice, assuring me that the cat was faking it. She was frozen in what I call meatloaf position, did not track anything with her eyes, and the strongest catnip right in front of her nose could not budge her rigid pose. I dashed to my computer to type into the search window "cats that don't like catnip", thinking at last I had found a 20 percenter. Months later, after she finally convinced herself that I was not going to eat her, she revealed she had legs and could walk around! Furthermore, she proved a typical, playful catnip fiend. I marvel that she resisted the chemical pull of that molecule when she was rigid with fear. Perhaps she started out with some resistance, and learned to love it. Or, fear for her life may overcame the grip of the drug.

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Evidence of Action in Humans

There are regrettably no well-designed published, scientific, clinical trials assessing the effects of catnip on humans.

What's in it

(For an impressively detailed reference to the constituents of hundreds of *Nepeta*, see Formisano et. al.[1]) The lion's share of the volatile oil is stereoisomers of nepetalactone, which may be 80-90%, but this varies, not just for the species of *Nepeta*, but also can widely vary for one species grown in different locations. *Nepeta* also contain a variety of iridoid derivatives of nepetalactone like epinepetalactone, and dihydronepetalactone, and their glycosides. Studies also note the presence of eucalyptol, linalool, caryophyllene, citronellal, camphor, thymol, carvacrol, and pulegone, as well as Lamiuside A and verbascoside.

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