Pesticides: debunking the myths

Pyrethroids and Health Effects

Pyrethroids have irritant and/or sensitizing properties. They are not easily absorbed through the skin, but are absorbed through the gut and pulmonary membrane. Tests of some pyrethroids on laboratory animals reveal striking neurotoxicity when administered by injection or orally. Systemic toxicity by inhalation and dermal absorption is low. The acute toxicity, calculated by LD50's, ranges from low to high, depending on the specific formulation. Low toxicity is attributed to two factors: limited absorption of some pyrethroids, and rapid biodegradation by mammalian liver enzymes (ester hydrolysis and oxidation). Insects, without this liver function, exhibit greater susceptibility to the chemicals (Reigart et al., 1999).

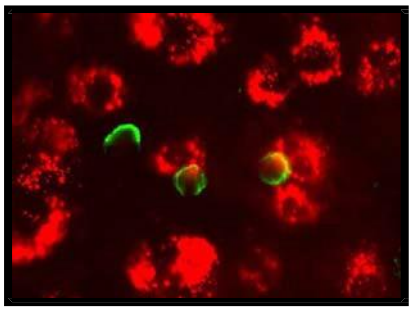
Pyrethroids interfere with the ionic conductance of nerve membranes by prolonging the sodium current. This stimulates nerves to discharge repeatedly causing hyper-excitability in poisoned animals. The World Health Organization explains that synthetic pyrethroids are neuropoisons acting on the axons in the peripheral and central nervous systems by interacting with sodium channels in mammals and/or insects. The main systems for metabolism include breakage of the ester bond by esterase action and oxidation at various parts of the molecule. Induction of liver microsomal enzymes has also been observed (WHO, 1999).

Copper Naphthenate

The myth...

Widely used as a wood preservative and also in many polymeric applications for protection against insects and rodents. Copper naphthenate or more specifically copper naphthenate solution which consists of 20% copper naphthenate mixed with white spirits or mineral turps type solvents have been popularly reintroduced in the market as a formulation which is effective in its primary purpose of being a wood preservative and yet provide positive benefits with respect to safety of human health and environment.

But dig deeper and you would find yourself busting a myth and getting closer to the truth which is that it is not only not harmless b u t in fact much more toxic than it claims in its so called 'non toxic' status.



While copper naphthenate is said to be comprising of copper naphthenate in combination with white contains spirits/ mineral turps type solvents, the truth is that it about 20% copper salts of naphthenic acids which comprise of an unknown mixture of certain petroleum by products and contaminants and about 80% secret unknown ingredients. These contaminants and by products haven't been researched enough to conclude that they have no side effects: harmful or otherwise in the long or short run. **Most petroleum-based constituents are known acutely toxic, chronically toxic and carcinogenic compounds!** Moreover they are volatile which means that at any point of time the air surrounding you could be laden with lethal carcinogens: not a very comforting thought especially since toxic!!!

Numerical Measures of Toxicity:

Product Toxicity Data:
Oral rat LD50: >2000 mg/kg
Dermal rabbit: LD50 >2000 mg/kg

Copper Naphthenate:
Oral rat LD50: 2000 mg/Kg

Finland, Indonesia, Korea, Netherlands, New Zealand, Saint Lucia, Sweden, Australia, Austria, Cyprus, Norway and Sri Lanka have banned the use of the Copper Naphthenate.

Pyrethroids- the different types:

Bifenthrin

Bifenthrin is an off-white to pale tan waxy solid, characterized by its slightly sweet smell. **As a Restricted Use Pesticide**, Bifenthrin may only be purchased or applied by certified applicators or persons under the direct supervision of a certified applicator. **EPA has classified products containing Bifenthrin as toxicity class II (I = most toxic, IV = least toxic), and the word WARNING must appear on all product labels.** Bifenthrin is toxic to mammals when ingested (oral rat LD50 = 54 to 70 mg/kg), and like all pyrethroids affects the central nervous system.

A study on laboratory mice shows that Bifenthrin causes gene mutation in white blood cells (ETN, Bifenthrin, 1995). **EPA classifies Bifenthrin as a Class C (possible human) carcinogen (EPA, 1997).**

Symptoms of poisoning include in-coordination, tremor, salivation, vomiting, diarrhea, and irritability to sound and touch (ETN Bifenthrin, 1995). Of concern in the environment, Bifenthrin is very highly toxic to fish, crustaceans, other aquatic animals and bees, and is moderately toxic to birds. Scientists are particularly concerned about possible bioaccumulation in birds.

Hazards of handling bifenthrin

- Causes dizziness, headache, tingling and numbness sensation, muscle spasms and tremors.
- Develops rash, hives, blisters, sores and itchiness.
- Can result in redness, pain and swelling of eyes, itchy watery eyes and blurred vision.
- Suspected of causing cancer.

Cypermethrin

Cypermethrin is registered to control cockroaches, fleas and other indoor pests in homes, restaurants, hospitals, schools and food processing plants, and also in agriculture to control pests on cotton, fruits and vegetables. **Depending on the specific product formulation, EPA classifies pesticides containing Cypermethrin as toxicity class II (I = most toxic, IV = least toxic) and must display the word WARNING or CAUTION on the labels.** Cypermethrin is considered to be toxic (oral male rat LD50 =187 to 326 mg/kg, dermal rat LD50 = 1600 mg/kg) and like all pyrethroids, affects the central nervous system (ETN, Cypermethrin, 1996). Symptoms of Cypermethrin poisoning in humans include numbness, burning, loss of bladder control, vomiting, in- coordination, seizures, coma and death. In California, Cypermethrin is the fourth most common cause of pesticide related illness in pest control operators. EPA classifies Cypermethrin as a class C (possible human) carcinogen (EPA, 1997). Studies in laboratory animals have shown exposure to cypermethrin **to cause reproductive effects, including abnormal sperm and disruption of sex hormones (Cox, 1996). Cypermethrin should not be applied near water, because it is very toxic to fish and other aquatic organisms.**

Hazards of handling Cypermethrin

- This material can cause eye irritation and damage in some persons
- The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage
- Causes clinical signs of neurotoxicity (body tremors, decreased motor activity and impaired gait) following acute, subchronic or chronic exposure.

Deltamethrin

Deltamethrin is pyrethroid insecticide that kills insects on contact and through digestion. It works by paralyzing the insects' nervous system and therefore giving a quick knock-down effect. **Deltamethrin pesticides may range in toxicity from EPA toxicity class I to class III (I = most toxic, IV = least toxic), bearing the words DANGER-POISON, WARNING or CAUTION on the label (PANNA, 2000). Deltamethrin products may be general or Restricted Use Pesticides.** Deltamethrin produces different signs of poisoning than other pyrethroids. When exposed to

Deltamethrin, mammals exhibit typical type II motor symptoms, which include a writhing syndrome in rodents, as well as copious salivation. **The acute oral LD50 in male rats has been reported as low as 128 mg/kg to greater than 5,000 mg/kg depending on the carrier and conditions of the study (ETN, Deltamethrin, 1995).** Some studies have shown Deltamethrin to cause skin irritation. Especially characteristic of Deltamethrin poisoning is rolling convulsions. The sequence of the signs of poisoning is clearly defined, progressing from chewing, salivation, and pawing to rolling convulsions, tonic seizures, and death (ETN, Deltamethrin, 1995). In humans, symptoms of poisoning include ataxia, convulsions leading to muscle fibrillation and paralysis, dermatitis, edema, diarrhea, dyspnea, headache, hepatic microsomal enzyme induction, irritability, peripheral vascular collapse, rhinorrhea, serum alkaline phosphatase elevation, tremors, vomiting and death due to respiratory failure. Deltamethrin is a suspected endocrine disruptor. Deltamethrin is also toxic to fish, aquatic organisms, amphibians and bees.

Permethrin

Permethrin resembles pyrethrins chemically but is chlorinated to increase its stability. There are four isomeric forms, two cis - and two trans- of technical permethrin. Although the acute toxicity of the mixture (oral rat LD50 > 5000 mg/kg, oral mouse LD50 = 500) is less than that of natural pyrethrins, the cis-isomer is considerably more toxic (oral LD50 = 100), and in rats, mouse metabolites of the cis-isomer are more persistent biologically.

Permethrin receives an EPA toxicity class of II or III (I = most toxic, IV = least toxic), and carries either the word CAUTION on its label, depending on the formulation.

While it is not extremely toxic to humans, there are numerous reports on transient skin, eye and respiratory irritation. Like all pyrethroids, permethrin is a central nervous system poison.

Other studies have shown effects on the immune system, enlarged livers and at high doses, decreased female fertility and endocrine disruption. Permethrin is extremely toxic to aquatic life, bees and other wildlife. It should not be applied in crops or weeds where foraging may occur (ETN, Permethrin, 1996).

Hazards overview - overview of concerns

Pyrethroids have irritant and/or sensitizing properties. They are not easily absorbed through the skin but are absorbed through the gut and pulmonary membrane. Tests of some pyrethroids on laboratory animals reveal striking neurotoxicity when administered by injection or orally. The acute toxicity, calculated by LD50's, ranges from low to high, depending on the specific formulation Pyrethroids interfere with the ionic conductance of nerve membranes by prolonging the sodium current. This stimulates nerves to discharge repeatedly causing hyper-excitability in poisoned animals. **The World Health Organization explains that synthetic pyrethroids are neuropoisons acting on the axons in the peripheral and central nervous systems by interacting with sodium channels in mammals and/or insects.** The main systems for metabolism include breakage of the ester bond by esterase action and oxidation at various parts of the molecule. Induction of liver microsomal enzymes has also been observed (WHO, 1999).

Signs and symptoms of poisoning by pyrethroids may take several forms. Because of the similarities to crude pyrethrum, pyrethroids may act as dermal and respiratory allergens. Exposure to pyrethroids has resulted in contact dermatitis and asthma-like reactions. Persons, especially children, with a history of allergies or asthma are particularly sensitive, and a strong cross-reactivity with ragweed pollen has been recognized. Severe anaphylactic (allergic) reactions with peripheral vascular collapse and respiratory difficulty are rare. Other symptoms of acute toxicity due to inhalation include sneezing, nasal stuffiness, headache, nausea, in-coordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensations. The most severe poisonings have been reported in infants, who are not able to efficiently break down pyrethroids (ETN, Pyrethroids, 1994). With orally ingested doses, nervous symptoms may occur, which include excitation and convulsions leading to paralysis, accompanied by muscular fibrillation and diarrhea (ETN, Pyrethroids, 1994). Death in these cases is due to respiratory failure. Symptoms of acute exposure last about 2 days.

Endocrine Disruption and Breast Cancer

Many pyrethroids have also been linked to disruption of the endocrine system, which can adversely affect reproduction and sexual development, interfere with the immune system and increase chances of breast cancer. Pyrethroids contain human-made, or xenoestrogens, which can increase the amount of estrogen in the body (Garey et al., 1998). When tested, certain pyrethroids demonstrate significant estrogenicity and increase the levels of estrogen in breast cancer cells (Go et al., 1999). Because increased cell division enhances the chances for the formation of a malignant tumor in the breast, artificial hormones, like those found in pyrethroids, may increase breast cancer risk (PCBR, 1996). Some pyrethroids are classified by EPA as class C (possible human) carcinogens.

Pyrethroids and the Environment

While the development of the synthetic pyrethroids was heralded with claims of selective toxicity to insects, both pyrethroids and pyrethrins are extremely toxic to aquatic organisms, including fish such as the bluegill and lake trout, with LC50 values less than 1.0 parts per billion. Lobster, shrimp, mayfly nymphs and zooplankton are the most susceptible non-target aquatic organisms (Mueller-Beilschmidt, 1990). The nonlethal effects of pyrethroids on fish include damage to the gills and behavioral

changes. Pyrethroids are toxic to birds, with most LD50 values greater than 1000 mg/kg. Birds can also be indirectly affected by pyrethroids, because of the threat to their food supply. Waterfowl and small insectivorous birds are the most susceptible (Mueller-Beilschmidt, 1990). Because pyrethroids are toxic to all insects, both beneficial insects and pests are affected by pyrethroid applications. In some cases, predator insects may be susceptible to a lower dose than the pest, disrupting the predator-prey relationship.

Pyrethroids Residues /Persistence

As mentioned before, pyrethroids are designed to breakdown more slowly than the naturally occurring pyrethrins. While pyrethrins, extremely sensitive to light, heat and moisture, break down in a few hours, the synthetic pyrethroids are stable and persist in the environment much longer. With a few exceptions, pyrethroids break down most quickly in direct sunlight, usually just a few days after application, with a few exceptions. However, in areas with limited sunlight, such as grain silos and subway tunnels, pyrethroids can persist for months. For more specific breakdown times see the sections below on Bifenthrin, Cypermethrin, Deltamethrin, Permethrin and so on.

Nonivamide (synthetic capsaicin)

Aptly defined as the **pain producing component,** Nonivamide is a synthetic capsaicin belonging to the genus Capsicum.

Nonivamide is also called **pelargonic acid vanillylamide** or **PAVA** and is a capsaicinoid. It is present in chili peppers but is commonly manufactured synthetically. The reddish brown, oily liquid obtained by extracting dried, ripe fruit of chili peppers is known as Oleoresin capsicum. More than 100 compounds have been identified in oleoresin capsicum, but capsaicin is the most pungent and particularly irritant component in many peppers comprising of 0.007% to 0.7% of dried mass. [2]

2 most unfortunate myths associated with this synthetic capsaicin are discussed in this article.

MYTH #1:

"Nonivamide can be used to deter pests"

Capsaicin compound is sold popularly as mammalian pests, bird and insect deterrents. A common example is the use of ground-up or crushed dried chili pods in birdseed to deter squirrels, since birds are unaffected by capsaicin. However, these solutions may be a realistic option for commercial applications.

Although hot chili pepper extract is commonly used as a component of household and garden insect repellent formulas, extensive studies show that it is not clear that the capsaicinoid elements of the extract are responsible for any repellency. Even if the effects are seen, they are only temporary.

Infact, the matter of fact is that- Unlike many fruits, which have evolved to seed dispersal with the zoochory, the seeds of Capsicum plants are predominantly dispersed by birds themselves, in which capsaicin has an analgesic rather than irritant property! **Chili pepper seeds consumed by birds pass through the digestive tract unharmed, whereas those**

consumed by mammals do not germinate at all.

Most of the pests possess evolutionary advantages: Birds & mammals do not have the same sensitivity to capsaicin anymore, because it targets a specific pain receptor which is now immune to the hotness. Chili peppers are eaten by birds living in the chili peppers' natural range. The seeds of the peppers are distributed by the birds that drop the seeds while eating the pods, and the seeds pass through the digestive tract unharmed. This relationship may have promoted the evolution of the protective capsaicin. In the picture above, you can see a grey squirrel inside a chili ring of fire, eating the bird food.

MYTH #2:

"Nonivamide is non- toxic and safe to humans"

Since capsaicin is universally used as self- defense and riot- control agent in form of pepper sprays, tear gas, etc, the notes and documents from various statutory bodies world- wide have been sadly misunderstood. In the summary report on Nonivamide by The European Agency for the Evaluation of Medicinal Products (Veterinary Medicines Evaluation Unit), a conclusion that Nonivamide has low oral toxicity and recommendation for use in topical treatment is given. Similarly, a Committee on Toxicology of Chemicals in Food, Consumer Products & the Environment has given a statement on the use of PAVA as an incapacitant spray. The report concludes that the available information, both from the toxicity data in experimental studies, and experience in use, indicates that the low exposures arising from the use of PAVA incapacitant spray would not be expected to be associated with any significant adverse health effects.

Capsaicin is almost non- toxic, as also highlighted by these reports. But the glitch here is that in food/ pharma applications the dosage of Nonivamide is not more than 1%. Most of the self-defense or pepper sprays contains not more than 0.32% of capsaicin. **In large quantities, capsaicin can cause death**. Symptoms of overdose include difficulty breathing, blue skin, and convulsions. Eye exposure produces intense tearing, pain, conjunctivitis and blepharospasm.

Acute toxicity values:

The lethal dose (LD50 in mice) of capsaicin is 47.2 mg/kg. According to WHO Recommended Classification of Pesticides by Hazard, this compound falls in Class 1b (5-50 mg/kg rat) meaning a **"highly hazardous substance"**.

Hazards of handling capsaicin:

- Causes serious irritation, conjunctivitis and lacrimation via contact with eyes. It induces a burning sensation and pain in case of contact with eyes and skin.
- As it is also irritating to the respiratory system, it causes lung irritation and coughing as well as bronchoconstriction.
- Other respiratory effects include laryngospasm, swelling of the larynx and lungs, chemical pneumonitis, respiratory arrest and central nervous system effects such as convulsions and excitement.
- In case of ingestion, gastrointestinal tract irritation may be observed along with a sensation of warmth or painful burning

Denatonium Benzoate

It is world's bitterest known substances. The bitterness of the compound guides most applications of denatonium. Denatonium benzoate is used to denature ethanol so that it is not treated as an alcoholic beverage with respect to taxation and sales restrictions.

MYTH:

"Denatonium Benzoate can be used to deter pests"

Denatonium is used in denatured alcohol, antifreeze, nail biting preventions, animal repellents, liquid soaps, and shampoos.

It should be noted that animals are known to have different sensitivities to the effects of denatonium. It has been used to safeguard rat poisons from human consumption, **so presumably rats are not deterred by it, says Macfarlan Smith** of Edinburgh, Scotland. For denatonium benzoate, a concentration of 0.000008 moles per cubic meter is discernible to humans.

Few studies have been conducted to assess the effectiveness of denatonium benzoate in discouraging tasting, swallowing, or otherwise repelling wildlife. A recent review concluded that with respect to carnivores, 'products that contain denatonium derivates are ineffective repellents.

DNS is classified as a class III toxicity pesticide, on a scale of I to IV, I being the highest toxicity class (FCH 2000). According to toxicology studies performed by the manufacturer, 1g/kg of DNS has shown to produce weakness, gasping and imbalance in mice. The acute oral LD50 (lethal dose for 50% of the test population) for rabbits is 1.39g/kg. It can cause skin and eye irritation.

Buprofezin

Buprofezin is an insecticide used for control of insect pests such as mealybugs, leafhoppers and whitefly on vegetable crops. It is a growth regulator, acting as an inhibitor of chitin synthesis. It is banned in some countries due to its negative environmental impacts, being especially toxic to aquatic organisms as well as non-target insects.

Acute oral LD50 - 2198-2355 mg/kg (rats), >10000 mg/kg (mouse)

It is mildly irritating and may cause mild trasient discomfort. Stinging, reddening and watering of the eyes and lengthy exposure or delayed treatment may cause permanent damage.

It has been reported very toxic to aquatic life with long lasting effects.

Chlorpyrifos

Chlorpyrifos, a pesticide commonly used on food crops, has garnered international media attention in recent months due to its links to neurotoxic health issues.

For half a century, staple food crops in the United States — such as corn, wheat, apples and citrus — have been sprayed with chlorpyrifos, a dangerous pesticide that can damage the developing brains of children, causing reduced IQ, loss of working memory, and attention deficit disorders.

In November 2016, EPA released a revised human health risk assessment for chlorpyrifos that confirmed that there are no safe uses for the pesticide. EPA found that:

- All food exposures exceed safe levels, with children ages 1–2 exposed to levels of chlorpyrifos that are 140 times what EPA deems safe.
- There is no safe level of chlorpyrifos in drinking water.
- Pesticide drift reaches unsafe levels at 300 feet from the field's edge.
- Chlorpyrifos is found at unsafe levels in the air at schools, homes, and communities in agricultural areas.
- All workers who mix and apply chlorpyrifos are exposed to unsafe levels of the pesticide even with maximum personal protective equipment and engineering controls.
- Field workers are allowed to re-enter fields within 1–5 days after pesticide spraying, but unsafe exposures continue on average 18 days after applications.

HUMAN RIGHTS WATCH

Europe's Opportunity to Ban a Dangerous Pesticide

September 9, 2019

In several European countries, pregnant women risk consuming food contaminated with the pesticide chlorpyrifos, a neurotoxin that could be harmful to the fetus.

Last month, the European Food Safety Authority (EFSA) stated that the pesticide does not meet the safety criteria for renewed approval by the European Union. The agency joins eight EU member states and a growing number of children's rights and environmental advocacy groups around the world pushing back against the use of this dangerous chemical.

The EFSA's finding that there does not exist a safe exposure level for chlorpyrifos comes at a critical time. The approval period for chlorpyrifos use in the EU expires in January 2020 and the manufacturer's application to renew this approval is currently under review by the European Commission.

Hazards of handling chlorpyrifos

- People may sweat, and develop headache, nausea, and dizziness.
- Vomiting, abdominal muscle cramps, muscle twitching, tremors and weakness, and loss of coordination
- Diarrhea or blurred or darkened vision.
- In severe poisoning cases, exposure can lead to unconsciousness, loss of bladder and bowel control, convulsions, difficulty in breathing, and paralysis.

PESTS DEVELOPING RESISTANCE TOWARDS INSECTICIDES AND PESTICIDES

Cockroaches Are So Tough They Develop Resistance to Pesticides They Haven't Even Met

Mike Mcrae | 28th June 2019

A study on how quickly populations of German cockroach (*Blattella germanica*) bounce back after being doused with several classes of insect killer has revealed they can evolve a general resistance to pesticides they've never even encountered.

Researchers from Purdue University set up an experiment to evaluate how roaches evolve pesticide resistance over successive generations, hoping to determine which methods for eradication might be optimal.

Back in the lab, their two caged populations helped the researchers pull apart what they saw in the field. By testing pesticides in the lab, they could work out how long it took for generations to breed back into previous numbers.

Answer: not long enough.

If even a handful of resistant cockroaches survive that first treatment of one or even multiple pesticides, their offspring will reclaim the land for super-fun-happy times and a carefree existence dancing joyfully in their poisonous paradise.

The Telegraph

New 'super rats' evolve resistance to poison

Genetic mutations have produced a new breed of "super rat" with DNA that protects the vermin from standard toxins, according to Professor Robert Smith at the University of Huddersfield.

Ratcatchers in Berkshire and Hampshire were the first to report that their poisons were no longer effective, which experts put down to increased immunity among the pests.

But as the poison-resistant rats continue to spread, tests have revealed that they boast an entirely new strand of DNA that wards off attacks from pesticides.

Swindon in Wiltshire is the latest town to suffer an infestation, with exterminators reporting a 500 per cent increase in the rodents. Many are turning to traps, air rifles and even dogs in an effort to keep the populations under control.

Prof Smith of the university's applied sciences department warned that "super rats" may be thriving in communities across Britain. The Government no longer provides funding to track resistance, meaning the scale of the problem is unclear.

"Natural selection means that when you have a rat population in your town, **poison will kill the ones that aren't resistant, the ones that survive may have the gene, they then have babies who can receive the gene themselves," he said.**

BUSINESS INSIDER
INDIA

Cockroaches are becoming resistant to common insecticides, which could make them nearly impossible to exterminate

Morgan Mcfall-Johnsen | July 3, 2019

- In a recent study, researchers tried out common insecticide treatments in about 100 cockroach-infested apartments across two Midwestern cities.
- Almost none of the treatments were effective, and some cockroach populations seemed to increase despite them.
- This means cockroaches are developing increasing resistance to insecticides - traits they're passing on to their offspring.

The problems on the shop floor

Pesticides are not designed for polymeric applications and they are definitely not designed for use as a masterbatch as they pose problems during processing via extrusion and also on account of leachability, compatibility and toxicity.

They have an extremely low thermal stability thus volatilizes easily during the manufacturing processes.

These hazardous substances or their degradation products can be released during all phases of plastic life cycle. This is an unwanted situation for the manufacturer, since the leaching of additives shortens the polymer lifetime, but is certainly harmful also to the humans and the environment.

These harmful pesticide additives volatilize at polymer processing temperatures and release extremely toxic fumes. These toxic fumes are many a times more lethal than the original. This poses fatal hazards to workers handling such products at the shop floor. As (air) temperature increases, vapour hazards will increase. The vapours from many pesticides increase three to four times for each 10° C increase in temperature. This is one reason why pesticide should be kept away from sunlight and why it is typically recommended that pesticides not be applied when air temperatures are above 30° C. Extrusion temperatures are as high as 100 - 300° C.

Conclusion

Thus it can be seen that pesticides are extremely toxic to animals, human health and life and the environment. Moreover, they are not meant for use with polymeric applications.

The above reported results show how pesticides have been found to be toxic and therefore extensive study of the same is the need of the hour which would then probably reveal in alarming proportions whatever has been found so far to have affected humans and animals alike! What however we can do is stop or reduce the use of such compounds as ultimately it is us the end users and the workers handling these products who suffer the consequences and not the ones who supply it or market it and develop use of better nontoxic and effective alternatives.

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