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# Therapeutic Efficacy of Homeopathic Medicine of the Plant *Conium Maclatum*

***Abesh Das[1]* Debolina Kundu[2]****

[1,2]*Department of Pharmaceutics,
Guru Nanak Institute of Pharmaceutical Sciences

**ABSTRACT**

*Conium maculatum* is a highly toxic herbaceous biennial plant that is native to North Africa, western Asia, and northern Europe. Its natural habitat has been extensively invaded, and it is now found in many regions of America, southern Africa, China, New Zealand, and Australia. In addition to invading native vegetation and competing with pasture and crops, *C. maculatum* poses a serious health risk to almost all humans and livestock. C. maculatum is growing and becoming more common in crops, even in its native range. An age-old cure, made classical by Plato's vivid account of its application in Socrates' demise. The provings produce many symptoms, including difficult gait, trembling, sudden loss of walking strength, painful stiffness in the legs, etc. Conium is an excellent remedy for these symptoms. Its ascending paralysis, which ends in death by failure of respiration, shows the ultimate tendency of these symptoms. Elderly age is a time of weakness, languor, local congestion, and sluggishness, and it is common to find such a condition there. Conium has chosen this particular setting in which to express itself. This condition correlates with weakness, hypochondriasis, urination issues, memory loss, and sexual debility. Problems with aging and single status in life. Tumor growth also invites it. the overall sensation of being struck and bruised. Very weak in bed first thing in the morning. palpitations, shaking, and weakness of body and mind. Diathesis is cancerous. Arteriosclerosis. sternum caries. larger glands. acts on the glandular system, causing conditions such as scrofulous and cancer to engorge and indurate it, changing its structure. after grippe, a tonic. Multiple neuritis causing insomnia.

**Keywords:** Conium maclatum, biennial, languor, tumor, diathesis, insomnia.

## Introduction

Native to Europe and North Africa, Conium maculatum is a highly poisonous biennial herbaceous flowering plant in the Apiaceae family, which includes carrots. It is also referred to by the common names hemlock, poison hemlock, and wild hemlock. Hemlock is a hardy plant that can withstand a wide range of conditions. It has been introduced to areas outside of its natural range, including parts of Australia, West Asia, North and South America, and other regions. It has the potential to spread, turning into an invasive weed. The entire plant is poisonous, but the seeds and roots are particularly dangerous when consumed. With a long, penetrating root, the plant can grow up to 2.4 meters in height during the growing season under the correct circumstances. It grows fairly quickly. The plant emits a unique, often disagreeable smell that moves with the wind. Before the plant dies and finishes its biennial lifecycle, the hollow stems are typically speckled with a dark maroon color. The plant then turns brown and becomes dry. This poisonous plant's hollow stems can remain toxic for up to three years after the plant dies.

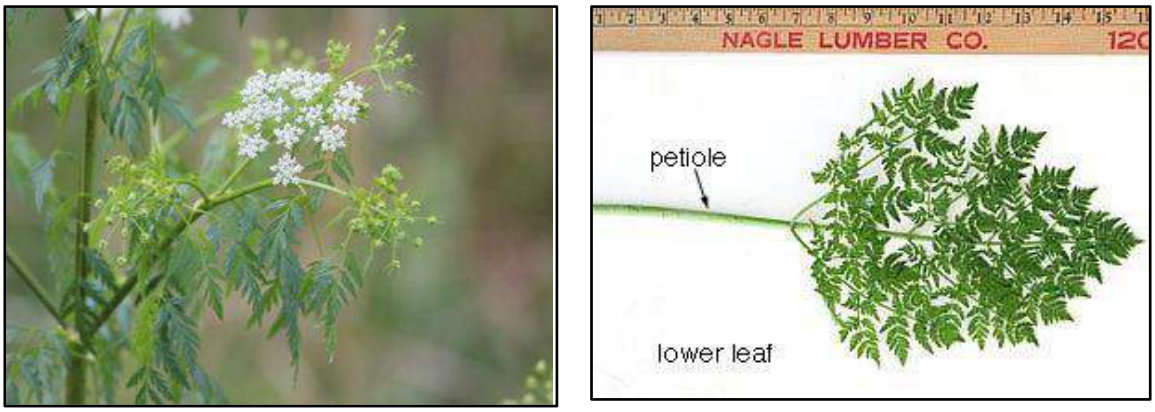


Figure 1: Picture of the plant *Conium maclatum*

## Morphological Character

Herbaceous and biennial, Conium maculatum is a flowering plant that reaches a height of 1.5–2.5 meters (5–8 feet), with an exceptional 3.6 meters (12 feet). Its smooth, hollow, green stem is often speckled or streaked with purple or red on both the upper and lower portions. The entire plant is hairless, or glabrous; its leaves are triangular in shape and can grow up to 50 centimeters (20 inches) in length and 40 centimeters (16 inches) in width. The leaves are two to four pinnate, finely divided, and lacy. The tiny, white flowers of hemlock are loosely clustered and have five petals apiece. Hemlock is a biennial plant that yields no flowers its first year but leaves at the base. It bears white flowers in umbrella-shaped clusters in its second year of growth.

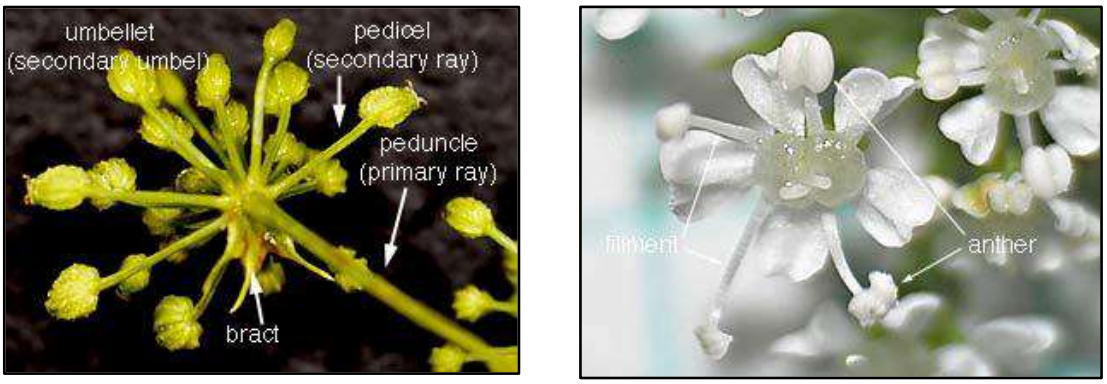


Figure 2: Parts of plant *Conium maclatum.*

## Geographical Distribution

The hemlock tree is indigenous to the Mediterranean and European continents. In most British Isles counties, it can be found in some woodland areas. In Ulster, these counties include County Down, County Antrim, and County Londonderry. In Australia, New Zealand, Asia, and North America, it has attained naturalization. In Tasmania and southeast Australia, it can occasionally be found near rivers. Because of its extreme toxicity, infestations, and human contact with the plant occasionally make news in the United States. The plant is frequently found in soil that has poor drainage, especially in the vicinity of ditches, streams, and other wet areas. Along roadside edges, in cultivated fields, and in waste areas, it can also be seen. Conium maculatum grows on roadsides, disturbed ground, and drier, rough grasslands in addition to extremely damp soil. The larvae of certain Lepidoptera, notably the poison hemlock moth (Agonopterix alstroemeriana) and silver-ground carpet moths, use it as a food plant. The latter has been applied extensively to the plant as a biological control agent. In the spring, when a lot of undergrowth is dormant and possibly not even in leaf, hemlock grows. The plant is toxic in every part.

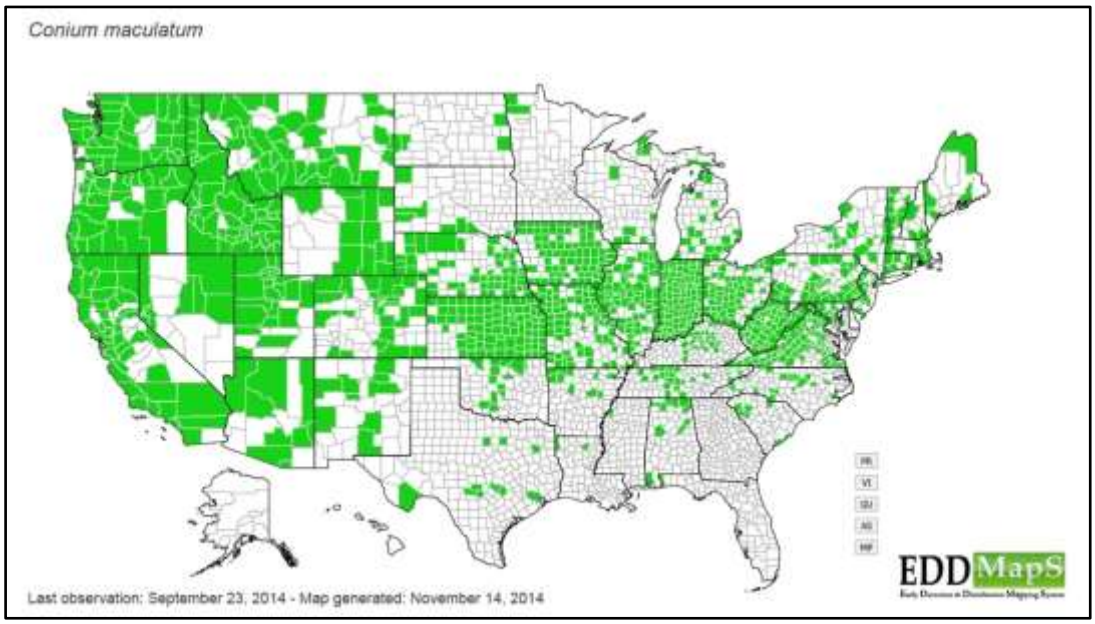


Figure 3: Geographical distribution of plant *Conium maclatum.*

## Chemical Constituents

It is well known that C. maculatum is incredibly poisonous. Its tissues are home to several distinct alkaloids. γ-coniceine is the main alkaloid present in flower buds. Coniine is later produced from this molecule during the fruit's development. Because alkaloids are volatile, scientists believe that they are crucial in drawing pollinators like bees and butterflies. Coniine, N-methylconiine, conhydrine, pseudoconhydrine, and gamma-coniceine (or g-coniceïne), the precursor of the other hemlock alkaloids, are among the piperidine alkaloids found in conium.

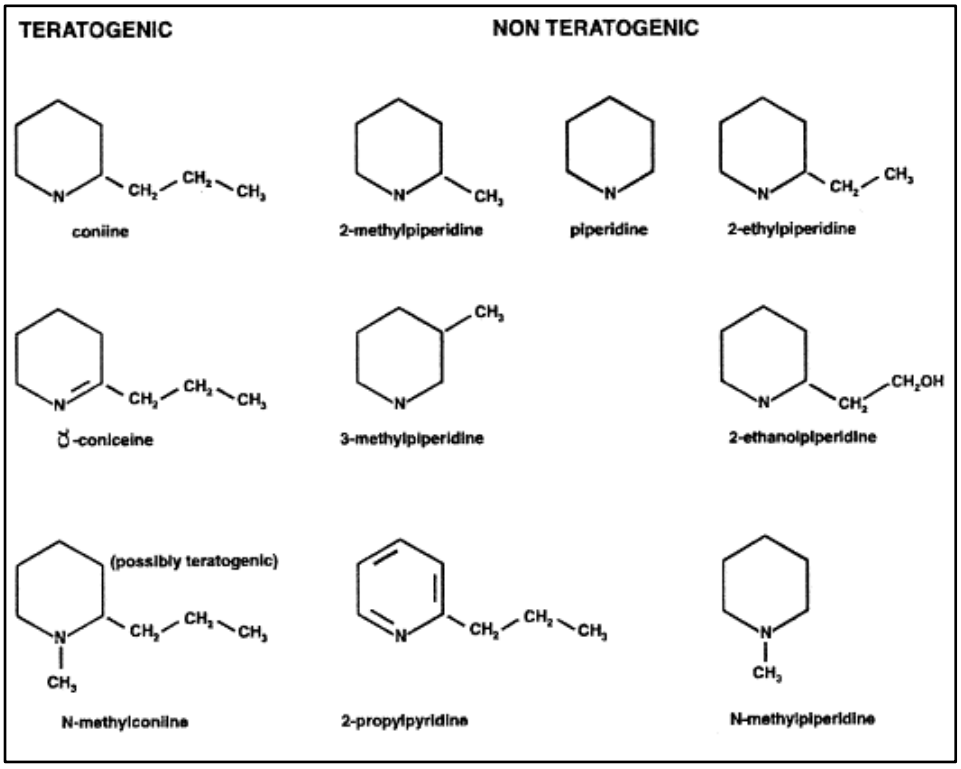


Figure 4: Chemical constituents of the plant *Conium maclatum.*

## Mechanism of Action

Moore and Row conducted one of the earliest investigations into the biological effects of coniine in the body in 1898. A frog weighing approximately 25 g was subcutaneously injected with 10–20 mg of coniine. The animal suffered from total muscular paralysis as a result. The paralysis of the intramuscular portion of the muscle nerves prevented the frog from responding when its nerves were stimulated with electricity. The peripheral nervous system was initially impacted by the coniine poisoning. Applying 50–60 mg of coniine to the superior cervical ganglion of a rabbit caused partial paralysis, which quickly subsided. Coniine also slowed the hearts of mammals and amphibians, according to the authors' observations. Arterioles induced by coniine dilated three times their normal diameter. Coniine caused a noticeable deepening and slight quickening of respiration in mammals. Subsequently, the rate of breathing decreased and the depth of each breath increased. A cat or dog given an injection of coniine (20–70 mg) experienced a decline in breathing that eventually stopped as a result of peripheral paralysis of the respiratory muscles. The outcome was unclear and did not show the lethal dosage. According to Moore and Row, coniine plays physiological roles that are comparable to those of piperidine and nicotine, albeit to varying degrees.

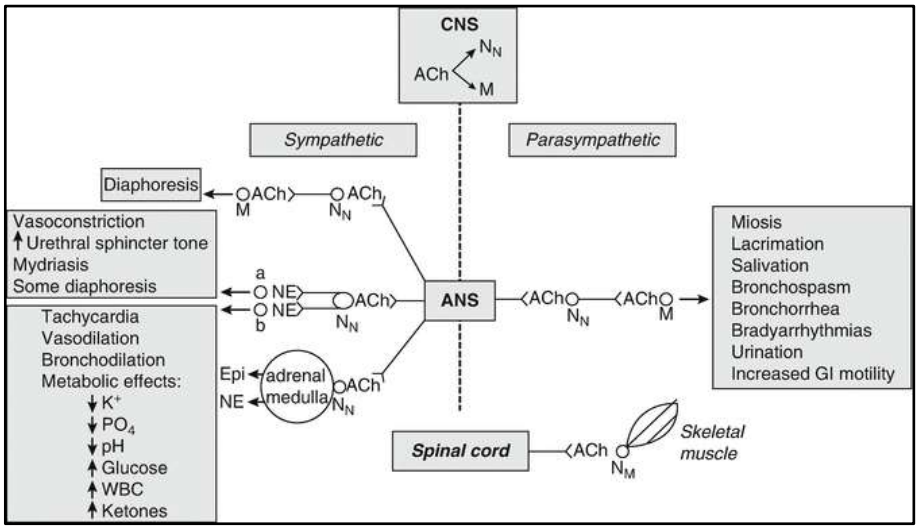


Figure 5: Mechanism of Action of *Conium maclatum*

## Mechanism of Toxicity

Nicotine-like alkaloids interact agonistically with nicotinic-type acetylcholine receptors (AChRs). These sodium-gated receptors can be found in the neuromuscular junction, autonomic and central nervous systems, and pre-or postsynaptic sites. Among their autonomic sites are the adrenal medulla and the autonomic ganglia; in the latter case, postsynaptic receptors are located at the start of the postganglionic autonomic neurons, whose junction with the end organs involves either cholinergic (muscarinic type) or norepinephrine receptors. Because they are agonists of nAChRs, nicotinic alkaloids lengthen membrane depolarization and improve action potential propagation by increasing sodium ion influx through the channel. Increased adrenal gland catecholamine release and ganglionic transmission to the postganglionic sympathetic fibers may cause tachycardia and hypertension. Tremor and muscular fasciculations can also be brought on by direct spinal cord discharges. Furthermore, increased activity of the postganglionic parasympathetic fibers may result in bronchospasm, lacrimation, salivation, and miosis. However, at neuroeffector junctions, muscarinic cholinergic (and noradrenergic) receptors are more prevalent, and nAChRs are not directly involved. Indeed, because there are so many different types of neuronal receptors, the dose-response relationships of nicotine and related alkaloids are complicated.

## Side Effects

Humans who have unintentionally consumed the leaves of parsley plants (Petroselinum crispum), parsnip roots (Pastinaca sativa), or anise seeds (Pimpinella anisum) have been reported to have contracted hemlock poisoning. Hemlock primarily affects the central nervous system. The plant has an effect akin to nicotine poisoning. While the symptoms of animal poisoning are generally similar to those of human poisoning, acute renal failure appears to be specific to human poisoning. They examined myoglobinuria, serum muscle enzymes, and renal function in the non-rapidly fatal cases. Microscopic kidney examinations and actin and myoglobin tubule identification using immunohistochemistry were conducted on patients suffering from acute renal failure. Coniine, an alkaloid, was found in tissues, serum, and urine. In each case, coniine exhibited neurological characteristics, such as a curare-like effect on the neuromuscular junction. Alkaloids containing conium have a dual mode of action. The most detrimental effect is at the neuromuscular junction, where they function similarly to curare as non-depolarizing blockers. In most cases, respiratory failure results in death. The effects of Conium alkaloids are biphasic, similar to those of nicotine. Abdominal pain, diarrhea, excessive salivation, nausea, and vomiting are some of the first stimulatory effects. Increased adrenergic tone is reflected in hypertension and tachycardia, which may be partially related to coronary artery constriction. Pallor resulting from peripheral blood vessel constriction is also frequently observed. Deep tendon reflexes may also be compromised. Early neurological effects include ataxia, tremor, restlessness, headache, visual and auditory disturbances, confusion, dizziness, muscle fasciculations, and seizures. Tachypnea and diaphoresis are other early symptoms.

## Medicinal Use

**Decontamination**

Nicotine is adsorbable by activated charcoal in vitro, and early decontamination may help to reduce the full effects of intoxication. In patients with an intact or protected airway, gastrointestinal decontamination with activated charcoal may be considered, despite the fact that the efficacy of the treatment has not been formally evaluated and symptoms may appear rapidly. General supportive measures such as hemodynamic stabilization, assisted ventilation, seizure control, and airway securing and stabilization should be prioritized over decontamination in symptomatic patients. It's also not advised to induce emesis.

**Supportive measures**

Supportive care is the mainstay of management with primary emphasis on cardiovascular and respiratory support.

**Cardiovascular Activity**

Although tachycardia and hypertension are frequent early signs of toxicity, the initial phase of adrenergic stimulation is usually short-lived and doesn't usually call for special treatment. Adrenergic antagonist therapy should therefore be avoided as it may exacerbate cardiovascular symptoms during the blockade phase, potentially leading to shock and cardiovascular collapse. Intravenous fluids should be used to treat hypotension; if this doesn't work, sympathomimetics like norepinephrine or dopamine, as well as other inotropic and/or vasopressor drugs, may be needed to restore blood pressure and circulation. Bradycardia is a common finding in the second phase and should be treated with atropine if symptomatic.

**Respiratory Effect**

Respiratory failure may arise from paralysis of the respiratory muscles, bronchoconstriction, and increased mucosal secretions. When hypersalivation, bronchorrhea, or wheezing are signs of excessive parasympathetic stimulation, atropine should be used. Respiratory support, including positive pressure ventilation and rapid sequence intubation, is essential in cases of declining respiratory function.

**Gastrointestinal Effect**

Effects on the digestive system can result from both central and peripheral mechanisms. Stimulation of the emetic chemoreceptor trigger zone is the primary mechanism responsible for the vomiting response. Diarrhea and vomiting are frequent and can cause hypovolemia hypotension as well as electrolyte and fluid imbalances. Antiemetic medication and appropriate intravenous fluid and electrolyte replacement are part of the management. When muscarinic parasympathetic stimulation is excessive, gastrointestinal hyperactivity can often be resolved with the administration of atropine.

**Seizure**

Although they are rare, seizures can occur when high doses of nicotine or related alkaloids are consumed. A benzodiazepine should be used to treat severe agitation or seizures; lorazepam or diazepam are recommended. Usually, unnecessary, longer-acting anticonvulsants might be needed as a second-line treatment if benzodiazepines are not working to control the seizures. Phenobarbital should be infused in this case. The use of barbiturates or benzodiazepines may cause respiratory depression.

**Anti-cancer Activity of *Conium maculatum***

Mondal et al. (2014) investigated the anti-cancer potential of Conium maculatum extract against cancer cells in vitro. Conium has the ability to interact with DNA, which makes it difficult for cells to divide and go through the cell cycle. Treatment with conium decreased HeLa cells' viability and capacity to form colonies; it also reduced cell proliferation and caused cell cycle arrest; it started the buildup of reactive oxygen species (ROS) in HeLa cells; it depolarized mitochondrial membrane potentials; it caused morphological changes in HeLa cells with nucleosomal fragmentation; and it altered the expression of several proteins linked to Hela cells' ability to proliferate and undergo apoptosis. Apoptosis's true mechanism is based on the up-and down-regulation of a few proteins.

**Economic Importance**

Animal poisoning from poison hemlock can result in significant losses. Both direct and indirect losses are possible. Because the majority of plant deaths are undetected and unreported, the economic losses can only be approximated. Conium maculatum adds to the estimated $100 million or more in livestock losses caused by poisonous plants in the US each year.

## Conclusion

Conium maculatum is a very common weed species of invasive character, i.e. the habitat of the plant is increasing. The plant grows first of all on soil with higher or nitrogen content, it is said to be a nitrophile species. Its chemical control (for example by auxin-like compounds) seems to be the most economical and relatively safe method. Conium maculatum has a very significant capacity to synthesize the alkaloids of the piperidine type. The main component is coniine the production of which is localized in different types of plant tissues.

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