

## Dinitrophenol

The Maryland Poison Center was contacted regarding an adult male who ingested dinitrophenol powder as a weight loss supplement once daily for an unspecified length of time. On that day he increased his dose, and 4 hours later was in the emergency department complaining of palpitations and nausea. He was diaphoretic, tachycardic (120 bpm) and tachypneic (22 breaths/min). By 7 hours after ingestion mental status was altered, temperature was elevated, and pulse increased to 150 bpm. At 10 hours, his temperature was 41.1°C. Two hours later, after receiving succinylcholine for a difficult intubation, he became rigid, arrested, and received CPR unsuccessfully for 30 minutes. He died approximately 10 hours after ingesting dinitrophenol.

2,4-Dinitrophenol (also known as DNP, DNOC, Dinosam, Solfo Black and others) has been used as a dye, wood preservative, pesticide and dietary supplement for weight loss and body building. It was widely used in the 1930s for 'rapid, safe weight loss' but was later deemed dangerous and not fit for human consumption (Federal Food, Drug and Cosmetic Act of 1938). The ban on human consumption was reissued by the Food and Drug Administration in 2003. Although banned in the U.S. as a weight loss supplement, it is available over the internet. DNP is a thermogenic agent which uncouples oxidative phosphorylation, stimulates cellular metabolism (glycolysis) and inhibits lipogenesis from pyruvate to lactate.

The suggested dose for weight loss is 2 mg/kg/day up to 200-400 mg daily. Dinitrophenol has a very narrow therapeutic index such that the toxic dose is very close to its therapeutic dose. With acute overdoses, symptoms may begin in as early as 3-4 hours. Death can occur rapidly with an average time of death of 14 hours after the overdose. (*J Med Toxicol.* 2011;7:205-12). A review of the literature in 2011 found 62 published deaths from DNP (*J Med Toxicol.* 2011;7:205-12).

Toxicity includes headache, weakness, malaise, lethargy, tachypnea, hyperthermia, diaphoresis, thirst, seizures, coma, methemoglobinemia, renal and hepatic failure and cardiac arrhythmias. Hyperthermia occurs as a result of uncoupling of oxidative phosphorylation which disrupts ATP synthesis and dissipation of energy produced by metabolism as heat. Death is associated with cardiovascular collapse and/or severe hyperthermia.

Diagnosis is based on history and signs and symptoms as there is no easily available blood test for DNP. Depending on the circumstances, DNP should be in the differential diagnosis for patients with tachypnea, diaphoresis, hyperthermia and metabolic acidosis.

Treatment is largely supportive care; there is no specific antidote. Activated charcoal can be considered for a large acute overdose. Patients may require large volume intravenous fluids, rapid and aggressive external methods of cooling (ice, cooling blankets), benzodiazepines for agitation and seizures, and methylene blue for methemoglobinemia. Difficulty with mechanical ventilation due to muscle rigidity as experienced by the patient described above has been reported.



### Did you know?

**Medications to avoid when treating patients with DNP toxicity include salicylates and anticholinergic drugs.**

Aspirin and other salicylates uncouple oxidative phosphorylation which can worsen hyperthermia and metabolic acidosis. Hyperthermia can also worsen with anticholinergic drugs which interfere with sweating and heat dissipation.

(*Olson. Poisoning & Drug Overdose, 7<sup>th</sup> ed, pp364-5.*)

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