[BROKEN](/wiki/BROKEN" \o "BROKEN) [Template:Drug](/wiki/Template:Drug) [Template:Psychedelic sidebar](/wiki/Template:Psychedelic_sidebar) ***N*,*N*-Dimethyltryptamine** (**DMT** or ***N*,*N*-DMT**) is a [psychedelic compound](/wiki/Psychedelic_drug) of the [tryptamine](/wiki/Tryptamine) family. It is a [structural analog](/wiki/Structural_analog) of [serotonin](/wiki/Serotonin) and [melatonin](/wiki/Melatonin) and a [functional analog](/wiki/Functional_analog_(chemistry)) of other psychedelic tryptamines such as [4-AcO-DMT](/wiki/4-AcO-DMT), [5-MeO-DMT](/wiki/5-MeO-DMT), [5-HO-DMT](/wiki/5-HO-DMT), [psilocybin](/wiki/Psilocybin) (4-PO-DMT), and [psilocin](/wiki/Psilocin) (4-HO-DMT).

Historically, it has been consumed by indigenous [Amazonian Indian](/wiki/Amazonian_Indians#Amazon) cultures in the form of [ayahuasca](/wiki/Ayahuasca) for divinatory and healing purposes.[[1]](#cite_note-1)

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## History[[edit](/index.php?title=(none)&action=edit&section=1)]

DMT was first synthesized in 1931 by Canadian chemist Richard Helmuth Fredrick Manske (1901–1977).[[2]](#cite_note-2)[[3]](#cite_note-3) In general, its discovery as a natural product is credited to Brazilian chemist and [microbiologist](/wiki/Microbiology) Oswaldo Gonçalves de Lima (1908–1989) who, in 1946, isolated an alkaloid he named *nigerina* (nigerine) from the root bark of *jurema preta*, that is, [*Mimosa tenuiflora*](/wiki/Mimosa_tenuiflora).[[3]](#cite_note-3)[[4]](#cite_note-4)[[5]](#cite_note-5) However, in a careful review of the case [Jonathan Ott](/wiki/Jonathan_Ott) shows that the [empirical formula](/wiki/Empirical_formula) for nigerine determined by Gonçalves de Lima, which notably contains an atom of oxygen, can match only a partial, "impure" or "contaminated" form of DMT.[[6]](#cite_note-6) It was only in 1959, when Gonçalves de Lima provided American chemists a sample of *Mimosa tenuiflora* roots, that DMT was unequivocally identified in this plant material.[[6]](#cite_note-6)[[7]](#cite_note-7) Less ambiguous is the case of isolation and formal identification of DMT in 1955 in seeds and pods of [*Anadenanthera peregrina*](/wiki/Anadenanthera_peregrina) by a team of American chemists led by Evan Horning (1916–1993).[[6]](#cite_note-6)[[8]](#cite_note-8) Since 1955, DMT has been [found in a host of organisms](/wiki/#Endogenous_DMT): in at least fifty plant species belonging to ten [families](/wiki/Family_(biology)),[[9]](#cite_note-9) and in at least four animal species, including one [gorgonian](/wiki/Gorgonian)[[10]](#cite_note-10) and three mammalian species.

Another historical milestone is the discovery of DMT in plants frequently used by Amazonian natives as additive to the vine [*Banisteriopsis caapi*](/wiki/Banisteriopsis_caapi) to make [ayahuasca](/wiki/Ayahuasca) decoctions. In 1957, American chemists Francis Hochstein and Anita Paradies identified DMT in an "aqueous extract" of leaves of a plant they named *Prestonia amazonicum* (*sic*) and described as "commonly mixed" with *B. caapi*.[[11]](#cite_note-11) The lack of a proper botanical identification of [*Prestonia amazonica*](/wiki/Prestonia_amazonica) in this study led American [ethnobotanist](/wiki/Ethnobotany) [Richard Evans Schultes](/wiki/Richard_Evans_Schultes) (1915–2001) and other scientists to raise serious doubts about the claimed plant identity.[[12]](#cite_note-12)[[13]](#cite_note-13) Better evidence was produced in 1965 by French pharmacologist Jacques Poisson, who isolated DMT as a sole alkaloid from leaves, provided and used by [Aguaruna](/wiki/Aguaruna) Indians, identified as having come from the vine [*Diplopterys cabrerana*](/wiki/Diplopterys_cabrerana) (then known as *Banisteriopsis rusbyana*).[[13]](#cite_note-13) Published in 1970, the first identification of DMT in the plant [*Psychotria viridis*](/wiki/Psychotria_viridis),[[4]](#cite_note-4) another common additive of ayahuasca, was made by a team of American researchers led by pharmacologist Ara der Marderosian.[[14]](#cite_note-14) Not only did they detect DMT in leaves of *P. viridis* obtained from Cashinahua Indians, but they also were the first to identify it in a sample of an ayahuasca decoction, prepared by the same Indians.[[4]](#cite_note-4)

## Biosynthesis[[edit](/index.php?title=(none)&action=edit&section=2)]

[thumb|left|Biosynthetic pathway for *N*,*N*-dimethyltryptamine](/wiki/Image:DMT_biosynthetic_pathway.png) Dimethyltryptamine is an [indole alkaloid](/wiki/Indole_alkaloid) derived from the [shikimate](/wiki/Shikimate) pathway. Its [biosynthesis](/wiki/Biosynthesis) is relatively simple and summarized in the picture to the left. In plants, the parent amino acid [L-tryptophan](/wiki/L-tryptophan) is produced endogenously where in animals [L-tryptophan](/wiki/L-tryptophan) is an [essential amino acid](/wiki/Essential_amino_acid) coming from diet. No matter the source of [L-tryptophan](/wiki/L-tryptophan), the biosynthesis begins with its [decarboxylation](/wiki/Decarboxylation) by an [aromatic amino acid decarboxylase](/wiki/Aromatic_amino_acid_decarboxylase) (AADC) [enzyme](/wiki/Enzymes) (step 1). The resulting decarboxylated tryptophan [analog](/wiki/Analog_(chemistry)) is [tryptamine](/wiki/Tryptamine). Tryptamine then undergoes a [transmethylation](/wiki/Transmethylation) (step 2): the enzyme [indolethanolamine-N-methyltransferase](/wiki/Tryptamine-N-methyltransferase) (INMT) [catalyzes](/wiki/Catalysis) the transfer of a [methyl group](/wiki/Methyl_group) from [cofactor](/wiki/Cofactor_(biochemistry)) [S-adenosyl-methionine](/wiki/S-adenosyl-methionine) (SAM), via [nucleophilic](/wiki/Nucleophilic) attack, to tryptamine. This reaction transforms SAM into [S-adenosylhomocysteine](/wiki/S-adenosylhomocysteine) (SAH), and gives the intermediate product [*N*-methyltryptamine](/wiki/N-methyltryptamine) (NMT).[[15]](#cite_note-15)[[16]](#cite_note-16) NMT is in turn transmethylated by the same process (step 3) to form the end product *N*,*N*-dimethyltryptamine. Tryptamine transmethylation is regulated by two products of the reaction: SAH,[[17]](#cite_note-17)[[18]](#cite_note-18)[[19]](#cite_note-19) and DMT[[17]](#cite_note-17)[[19]](#cite_note-19) were shown *ex vivo* to be among the most potent inhibitors of rabbit INMT activity.

This transmethylation mechanism has been repeatedly and consistently proven by [radiolabeling](/wiki/Isotope_labeling) of SAM methyl group with [carbon-14](/wiki/Carbon-14) (14C-CH3)SAM).[[15]](#cite_note-15)[[17]](#cite_note-17)[[19]](#cite_note-19)[[20]](#cite_note-20)[[21]](#cite_note-21)

### Evidence in mammals[[edit](/index.php?title=(none)&action=edit&section=3)]

Published in [*Science*](/wiki/Science_(journal)) in 1961, [Julius Axelrod](/wiki/Julius_Axelrod) found an *N*-[methyltransferase](/wiki/Methyltransferase) enzyme capable of mediating biotransformation of tryptamine into DMT in a rabbit's lung.[[15]](#cite_note-15) This finding initiated a still ongoing scientific interest in endogenous DMT production in humans and other mammals.[[16]](#cite_note-16)[[22]](#cite_note-22) From then on, two major complementary lines of evidence have been investigated: localization and further characterization of the *N*-methyltransferase enzyme, and [analytical studies](/wiki/Analytical_chemistry) looking for endogenously produced DMT in body fluids and tissues.[[16]](#cite_note-16) In 2013 researchers first reported DMT in the [pineal gland](/wiki/Pineal_gland) [microdialysate](/wiki/Microdialysis) of rodents.[[23]](#cite_note-23) A study published in 2014 reported the biosynthesis of N,N-dimethyltryptamine (DMT) in the human melanoma cell line SK-Mel-147 including details on its metabolism by peroxidases.[[24]](#cite_note-24) In a 2014 paper a group first demonstrated the immunomodulatory potential of DMT and [5-MeO-DMT](/wiki/5-MeO-DMT) through the [Sigma-1 receptor](/wiki/Sigma-1_receptor) of human immune cells. This immunomodulatory activity may contribute to significant anti-inflammatory effects and tissue regeneration.[[25]](#cite_note-25)

#### INMT[[edit](/index.php?title=(none)&action=edit&section=4)]

Before techniques of [molecular biology](/wiki/Molecular_biology) were used to localize [indolethylamine N-methyltransferase](/wiki/Indolethylamine_N-methyltransferase) (INMT),[[19]](#cite_note-19)[[21]](#cite_note-21) characterization and localization went on a par: samples of the biological material where INMT is hypothesized to be active are subject to [enzyme assay](/wiki/Enzyme_assay). Those enzyme assays are performed either with a radiolabeled methyl donor like (14C-CH3)SAM to which known amounts of unlabeled substrates like tryptamine are added[[16]](#cite_note-16) or with addition of a radiolabeled substrate like (14C)NMT to demonstrate [in vivo](/wiki/In_vivo) formation.[[17]](#cite_note-17)[[20]](#cite_note-20) As qualitative determination of the radioactively tagged product of the enzymatic reaction is sufficient to characterize INMT existence and activity (or lack of), analytical methods used in INMT assays are not required to be as sensitive as those needed to directly detect and quantify the minute amounts of endogenously formed DMT (see DMT subsection below). The essentially qualitative method [thin layer chromatography](/wiki/Thin_layer_chromatography) (TLC) was thus used in a vast majority of studies.[[16]](#cite_note-16) Also, robust evidence that INMT can catalyze transmethylation of tryptamine into NMT and DMT could be provided with [reverse isotope dilution analysis](/wiki/Isotopic_dilution) coupled to [mass spectrometry](/wiki/Mass_spectrometry) for rabbit[[26]](#cite_note-26)[[27]](#cite_note-27) and human[[28]](#cite_note-28) lung during the early 1970s.

Selectivity rather than sensitivity proved to be an Achilles’ heel for some TLC methods with the discovery in 1974–1975 that incubating rat blood cells or brain tissue with (14C-CH3)SAM and NMT as substrate mostly yields tetrahydro-β-carboline derivatives,[[16]](#cite_note-16)[[17]](#cite_note-17)[[29]](#cite_note-29) and negligible amounts of DMT in brain tissue.[[16]](#cite_note-16) It is indeed simultaneously realized that the TLC methods used thus far in almost all published studies on INMT and DMT biosynthesis are incapable to resolve DMT from those tetrahydro-β-carbolines.[[16]](#cite_note-16) These findings are a blow for all previous claims of evidence of INMT activity and DMT biosynthesis in avian[[30]](#cite_note-30) and mammalian brain,[[31]](#cite_note-31)[[32]](#cite_note-32) including [in vivo](/wiki/In_vivo),[[33]](#cite_note-33)[[34]](#cite_note-34) as they all relied upon use of the problematic TLC methods:[[16]](#cite_note-16) their validity is doubted in replication studies that make use of improved TLC methods, and fail to evidence DMT-producing INMT activity in rat and human brain tissues.[[35]](#cite_note-35)[[36]](#cite_note-36) Published in 1978, the last study attempting to evidence [in vivo](/wiki/In_vivo) INMT activity and DMT production in brain (rat) with TLC methods finds biotransformation of radiolabeled tryptamine into DMT to be real but "insignificant".[[37]](#cite_note-37) Capability of the method used in this latter study to resolve DMT from tetrahydro-β-carbolines is questioned later.[[17]](#cite_note-17)  
To localize INMT, a qualitative leap is accomplished with use of modern techniques of [molecular biology](/wiki/Molecular_biology), and of [immunohistochemistry](/wiki/Immunohistochemistry). In humans, a gene encoding INMT is determined to be located on [chromosome 7](/wiki/Chromosome_7_(human)).[[21]](#cite_note-21) [Northern blot analyses](/wiki/Northern_blot) reveal INMT [messenger RNA](/wiki/Messenger_RNA) (mRNA) to be highly expressed in rabbit lung,[[19]](#cite_note-19) and in human [thyroid](/wiki/Thyroid), [adrenal gland](/wiki/Adrenal_gland), and lung.[[21]](#cite_note-21)[[38]](#cite_note-38) Intermediate levels of expression are found in human heart, skeletal muscle, trachea, stomach, small intestine, pancreas, testis, prostate, placenta, [lymph node](/wiki/Lymph_node), and spinal cord.[[21]](#cite_note-21)[[38]](#cite_note-38) Low to very low levels of expression are noted in rabbit brain,[[21]](#cite_note-21) and human [thymus](/wiki/Thymus), liver, [spleen](/wiki/Spleen), kidney, colon, ovary, and [bone marrow](/wiki/Bone_marrow).[[21]](#cite_note-21)[[38]](#cite_note-38) INMT mRNA expression is absent in human peripheral blood [leukocytes](/wiki/White_blood_cell), whole brain, and in tissue from 7 specific brain regions (thalamus, subthalamic nucleus, caudate nucleus, hippocampus, amygdala, substantia nigra, and corpus callosum).[[21]](#cite_note-21)[[38]](#cite_note-38) [Immunohistochemistry](/wiki/Immunohistochemistry) showed INMT to be present in large amounts in [glandular epithelial cells](/wiki/Goblet_cell) of small and large intestines. In 2011, immunohistochemistry revealed the presence of INMT in primate nervous tissue including retina, spinal cord motor neurons, and pineal gland.[[39]](#cite_note-39)

#### Endogenous DMT[[edit](/index.php?title=(none)&action=edit&section=5)]

The first claimed detection of mammalian [endogenous](/wiki/Endogenous) DMT was published in June 1965: German researchers F. Franzen and H. Gross report to have evidenced and quantified DMT, along with its [structural analog](/wiki/Structural_analog) bufotenin (5-HO-DMT), in human blood and urine.[[40]](#cite_note-40) In an article published four months later, the method used in their study was strongly criticized, and the credibility of their results challenged.[[41]](#cite_note-41) Few of the analytical methods used prior to 2001 to measure levels of endogenously formed DMT had enough sensitivity and selectivity to produce reliable results.[[42]](#cite_note-42)[[43]](#cite_note-43) [Gas chromatography](/wiki/Gas_chromatography), preferably coupled to [mass spectrometry](/wiki/Mass_spectrometry) ([GC-MS](/wiki/GC-MS)), is considered a minimum requirement.[[43]](#cite_note-43) A study published in 2005[[22]](#cite_note-22) implements the most sensitive and selective method ever used to measure endogenous DMT:[[44]](#cite_note-44) [liquid chromatography](/wiki/High-performance_liquid_chromatography)-[tandem mass spectrometry](/wiki/Tandem_mass_spectrometry) with [electrospray ionization](/wiki/Electrospray_ionization) (LC-ESI-MS/MS) allows for reaching limits of detection (LODs) 12 to 200 fold lower than those attained by the best methods employed in the 1970s. The data summarized in the table below are from studies conforming to the abovementioned requirements (abbreviations used: CSF = [cerebrospinal fluid](/wiki/Cerebrospinal_fluid); LOD = [limit of detection](/wiki/Limit_of_detection); n = number of samples; ng/L and ng/kg = nanograms (10−9 g) per litre, and nanograms per kilogram, respectively):

|  |  |  |
| --- | --- | --- |
| **Species** | **Sample** | **Results** |
| **Human** | [Blood serum](/wiki/Blood_serum) | < LOD (n = 66)[[22]](#cite_note-22) |
| [Blood plasma](/wiki/Blood_plasma) | < LOD (n = 71)[[22]](#cite_note-22)  ♦  < LOD (n = 38); 1,000 & 10,600 ng/L (n = 2)[[45]](#cite_note-45) |
| Whole blood | < LOD (n = 20); 50–790 ng/L (n = 20)[[46]](#cite_note-46) |
| Urine | < 100 ng/L (n = 9)[[22]](#cite_note-22)  ♦  < LOD (n = 60); 160–540 ng/L (n = 5)[[43]](#cite_note-43)  ♦  Detected in n = 10 by GC-MS[[47]](#cite_note-47) |
| Feces | < 50 ng/kg (n = 12); 130 ng/kg (n = 1)[[22]](#cite_note-22) |
| Kidney | 15 ng/kg (n = 1)[[22]](#cite_note-22) |
| Lung | 14 ng/kg (n = 1)[[22]](#cite_note-22) |
| [Lumbar](/wiki/Lumbar_puncture) CSF | 100,370 ng/L (n = 1); 2,330–7,210 ng/L (n = 3); 350 & 850 ng/L (n = 2)[[48]](#cite_note-48) |
| **Rat** | Kidney | 12 &16 ng/kg (n = 2)[[22]](#cite_note-22) |
| Lung | 22 & 12 ng/kg (n = 2)[[22]](#cite_note-22) |
| Liver | 6 & 10 ng/kg (n = 2)[[22]](#cite_note-22) |
| Brain | 10 &15 ng/kg (n = 2)[[22]](#cite_note-22)  ♦  Measured in [synaptic vesicular](/wiki/Synaptic_vesicle) [fraction](/wiki/Fractionation)[[49]](#cite_note-49) |
| **Rabbit** | Liver | < 10 ng/kg (n = 1)[[22]](#cite_note-22) |
| **DMT** in body fluids and tissues *(NB: units have been harmonized)* | | |

A 2013 study found DMT in [microdialysate](/wiki/Microdialysis) obtained from a rat's pineal gland, providing evidence of endogenous DMT in the mammalian brain.[[23]](#cite_note-23)

## Physical and chemical properties[[edit](/index.php?title=(none)&action=edit&section=6)]

[thumb|DMT crystals](/wiki/File:D-Tryp.jpg) [thumb|right|DMT crystal at 400× magnification](/wiki/Image:Dmtx400tt9.jpg) DMT is commonly handled and stored as a [fumarate](/wiki/Fumaric_acid),[[50]](#cite_note-50) [RIMAs](/wiki/Reversible_inhibitor_of_monoamine_oxidase_A) should be used with caution as they can have lethal interactions with some prescription drugs such as SSRI antidepressants, and some over-the-counter drugs.[[101]](#cite_note-101) Induced DMT experiences can include profound time-dilation, visual and auditory illusions, and other experiences that, by most firsthand accounts, defy verbal or visual description. Some users report intense erotic imagery and sensations and utilize the drug in a ritual sexual context.[[104]](#cite_note-104)[[105]](#cite_note-105)[[106]](#cite_note-106)

## Detection in body fluids[[edit](/index.php?title=(none)&action=edit&section=17)]

DMT may be measured in blood, plasma or urine using chromatographic techniques as a diagnostic tool in clinical poisoning situations or to aid in the medicolegal investigation of suspicious deaths. In general, blood or plasma DMT levels in recreational users of the drug are in the 10–30 μg/L range during the first several hours post-ingestion.[Template:Citation needed](/wiki/Template:Citation_needed) Less than 0.1% of an oral dose is eliminated unchanged in the 24-hour urine of humans.[[107]](#cite_note-107)[[108]](#cite_note-108)[Template:Clarify](/wiki/Template:Clarify)

## Effects[[edit](/index.php?title=(none)&action=edit&section=18)]

### Dependence liability[[edit](/index.php?title=(none)&action=edit&section=19)]

The dependence potential of DMT and the risk of sustained psychological disturbance are minimal.[[109]](#cite_note-109)

### Physical[[edit](/index.php?title=(none)&action=edit&section=20)]

According to a dose-response study, "dimethyltryptamine dose slightly elevated blood pressure, heart rate, pupil diameter, and rectal temperature, in addition to elevating blood concentrations of beta-[endorphin](/wiki/Endorphin), [corticotropin](/wiki/Corticotropin), [cortisol](/wiki/Cortisol), and [prolactin](/wiki/Prolactin). [Growth hormone](/wiki/Growth_hormone) blood levels rose equally in response to all doses of DMT, and [melatonin](/wiki/Melatonin) levels were unaffected."[[53]](#cite_note-53)

## Conjecture[[edit](/index.php?title=(none)&action=edit&section=21)]

Several speculative and yet untested hypotheses suggest that [endogenous](/wiki/Endogenous) DMT is produced in the [human brain](/wiki/Human_brain) and is involved in certain [psychological](/wiki/Psychology) and [neurological](/wiki/Neurology) states.[[110]](#cite_note-110)[[111]](#cite_note-111) DMT is naturally occurring in small amounts in rat brain, human cerebrospinal fluid, and other tissues of humans and other mammals.[[22]](#cite_note-22)[[48]](#cite_note-48)[[49]](#cite_note-49)[[112]](#cite_note-112) A biochemical mechanism for this was proposed by the medical researcher J. C. Callaway, who suggested in 1988 that DMT might be connected with visual dream phenomena: brain DMT levels would be periodically elevated to induce visual dreaming and possibly other natural states of mind.[[113]](#cite_note-113) A role of endogenous hallucinogens including DMT in higher level sensory processing and awareness was proposed by J. V. Wallach based on a hypothetical role of DMT as a neurotransmitter.[[111]](#cite_note-111) Neurobiologist Andrew R. Gallimore suggests that while DMT might not have a modern neural function, it may have been an ancestral neuromodulator once secreted in psychedelic concentrations during [REM sleep](/wiki/Rapid_eye_movement_sleep) - a function now lost.[[114]](#cite_note-114) Dr. [Rick Strassman](/wiki/Rick_Strassman), while conducting DMT research in the 1990s at the [University of New Mexico](/wiki/University_of_New_Mexico), advanced the controversial hypothesis that a massive release of DMT from the [pineal gland](/wiki/Pineal_gland) prior to death or near death was the cause of the [near death experience](/wiki/Near_death_experience) (NDE) phenomenon. Several of his test subjects reported audio or visual hallucinations. His explanation for this was the possible lack of panic involved in the clinical setting and possible dosage differences between those administered and those encountered in actual NDE cases. Several subjects also reported contact with "other beings", alien like, insectoid or reptilian in nature, in highly advanced technological environments[[5]](#cite_note-5) where the subjects were "carried", "probed", "tested", "manipulated", "dismembered", "taught", "loved" and "raped" by these "beings". Basing his reasoning on his belief that all the enzymatic material needed to produce DMT is found in the pineal gland, and moreover in substantially greater concentrations than in any other part of the body, Strassman has speculated that DMT is made in the [pineal gland](/wiki/Pineal_gland)([[5]](#cite_note-5) p. 69).

In the 1950s, the endogenous production of psychoactive agents was considered to be a potential explanation for the hallucinatory symptoms of some psychiatric diseases; this is known as the transmethylation hypothesis.[[115]](#cite_note-115) In 2011, Nicholas V. Cozzi, of the [University of Wisconsin School of Medicine and Public Health](/wiki/University_of_Wisconsin_School_of_Medicine_and_Public_Health), concluded that [INMT](/wiki/INMT), an enzyme that may be associated with the biosynthesis of DMT and endogenous hallucinogens, is present in the primate (rhesus macaque) pineal gland, retinal ganglion neurons, and spinal cord.[[39]](#cite_note-39)

## Legal status[[edit](/index.php?title=(none)&action=edit&section=22)]

### International law[[edit](/index.php?title=(none)&action=edit&section=23)]

DMT is classified as a Schedule I drug under the [UN](/wiki/United_Nations) 1971 [Convention on Psychotropic Substances](/wiki/Convention_on_Psychotropic_Substances), meaning that use of DMT is supposed to be restricted to scientific research and medical use and international trade in DMT is supposed to be closely monitored. Natural materials containing DMT, including ayahuasca, are explicitly not regulated under the 1971 Psychotropic Convention.[[116]](#cite_note-116)

### By country[[edit](/index.php?title=(none)&action=edit&section=24)]

#### Australia[[edit](/index.php?title=(none)&action=edit&section=25)]

Between 2011 and 2012, the [Australian Federal Government](/wiki/Australian_Federal_Government) was considering changes to the [Australian Criminal Code](/wiki/Criminal_law_of_Australia) that would classify any plants containing any amount of DMT as "controlled plants".[[117]](#cite_note-117) DMT itself was already controlled under current laws. The proposed changes included other similar blanket bans for other substances, such as a ban on any and all plants containing Mescaline or Ephedrine. The proposal was not pursued after political embarrassment on realisation that this would make the official [Floral Emblem of Australia](/wiki/List_of_Australian_floral_emblems), [Acacia pycnantha](/wiki/Acacia_pycnantha) (Golden Wattle), illegal. The Therapeutic Goods Administration and federal authority had considered a motion to ban the same, but this was withdrawn in May 2012 (as DMT may still hold potential entheogenic value to native and/or religious people).[[118]](#cite_note-118) DMT is listed as a Schedule 9 prohibited substance in [Australia](/wiki/Australia) under the [Poisons Standard](/wiki/Standard_for_the_Uniform_Scheduling_of_Medicines_and_Poisons) (October 2015).[[119]](#cite_note-119)