Metabolites of Tryptamine: Endogenous psychedelic neurotransmitters, and N,N dimethyltryptamine (DMT) in explaining a new pathway to produce Serotonin, Melatonin and hallucinations.

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Abstract

Why naturally occurring psychoactive psychedelics, N,N-dimethyltryptamine (DMT), 5-hydroxy-DMT (Bufotenine, HDMT) and 5-Methoxy-DMT (MDMT) occur naturally in the human body has very little understanding at the cellular level, and even less how they give rise to hallucinations. This class of serotonergic hallucinogens will be discussed with new concepts to explain the chemical pathways and their associated enzymes in the brain. Inhibition of the tryptophan hydroxylase-2 (TPH2) enzyme, in forming serotonin (5HT), gives rise to the psychedelic metabolites of Tryptamine by activating stored information, as visual, auditory, tactile, gustatory or olfactory memories located to their correlating areas of the brain (Chaudhury, 2010) via Sigma-1, Serotonin (5HT) and Trace amine-associated receptors (TAARS). A direct synaptic pathway from the retina to the Dorsal Raphe Nucleus (DRN) (Fite, Janusonis, Foote, & Bengston, 1999) has been found. In addition, projections from DRN to the Pineal gland (Møler & Hay-Schmidt, 1998) and Olfactory Bulb (Steinfeld, Herb, Sprengel, Schaefer, & Fukunaga, 2015) results, which also correlates with findings of DMT presence in these areas (Barker, S., Borjigin, J., Lomnicka, I., Strassman, R., 2013). DMT accumulates in the cerebral cortex, putamen, caudate and amygdaloid nuclei (Yanai et al., 1986) and deactivates the default mode network (DMN) (Palhano-Fontes et al., 2015), hence the mind is unable to distinguish what is real and what is not (Li, 2014). A summary of these events will highlight the journey DMT takes from the intracellular makings to the sensory faking's renown as hallucinations.

Keywords: N,N-dimethyltryptamine (DMT), 5-hydroxy-DMT (Bufotenine, HDMT), 5-Methoxy-DMT (MDMT), Serotonin (5HT), Trace amine-associated receptors (TAARS), Indolethylamine-N-methyltransferase (INMT), default mode network (DMN), Tryptophan hydroxylase-2 (TPH2), Tryptamine hydroxylase enzyme (T5H), hydroxyindole-O-methyltrasferase (HIOMT), Tryptophan to 5-hydroxytrypotphan (5HTP), Traumatic Brain Injury (TBI), Tryptamine (TA).

Metabolites of Tryptamine: Endogenous psychedelic neurotransmitters, and N,N dimethyltryptamine (DMT) in explaining a new pathway to produce Serotonin, Melatonin and hallucinations.

Psychedelic chemicals, N,N-dimethyltryptamine (DMT), 5-hydroxy-DMT (Bufotenine, HDMT) and 5-Methoxy-DMT (MDMT), have been found in human cerebrospinal fluids – CSF (DMT and MDMT), blood (DMT and HDMT), and urine (DMT and HDMT) (Barker, S., Borjigin, J., Lomnicka, I., Strassman, R., 2013). HDMT has been found in hospital patients from surgical, medical and psychiatric wards, ten times more abundantly in stools than urine readings (Karkkainen, 2005) which could mean that excessive HDMT maybe a by-product, excreted from serotonergic cells in the gastrointestinal system. More recently psychedelics have been found in hair samples (Martin, Schurenkamp, Gasse, Pfeiffer, & Kohler, 2015) and combined with current data assessments possibly could find longitudinal data in future studies.

(Vitale et al., 2011) found labelled DMT is actively transported through the blood brain barrier (BBB) ten seconds after injection, and 0.1% was detected in the olfactory bulb 7 days later. This lead to the conclusion that DMT possibly is a plasma transporter, an extracellular substrate for Serotonin transporter (SERT) and an intracellular vesicle substrate for VMAT2 (vesicle monoamine transporter2), (Cozzi, N., Gopalakrishnan, A., Anderson, L., Feih, J., Shulgin, A., Daley, P., Ruoho, A., 2009) as shown in figure 1c.

DMT and Tryptamine bind with 5HT2A, 5HT2C, trace amine-associated (TAAR), sigma-1(σ-1) putative receptors. The methylation enzyme, Indolethylamine N-methyltransferase (INMT), has been found in the central nervous system, retina, pineal gland (Cozzi, N., Mavlyutov, T.A., Thompson, M.A., Ruoho, A.E. , 2011), (Barker, S., Borjigin, J., Lomnicka, I., Strassman, R., 2013) as well as motor neurons in the spinal cord (Mavlyutov et al., 2012) and is associated with DMT docking in INMT (Chu et al., 2014).

Figure 1(c) shows the schematic representation of the Serotonin (5HT) neuron and how neurotransmission is propagated. What will be discussed is a completed circuit of mechanisms intracellularly from the essential amino acid, Tryptophan (**3** in figure 1c), and how an alternative Tryptophan to Tryptamine pathway can explain numerous studies with the formation of Serotonin (5HT) and Melatonin.

Serotonergic transmission in (a) the human brain (b) rat brain (c) serotonin neurons

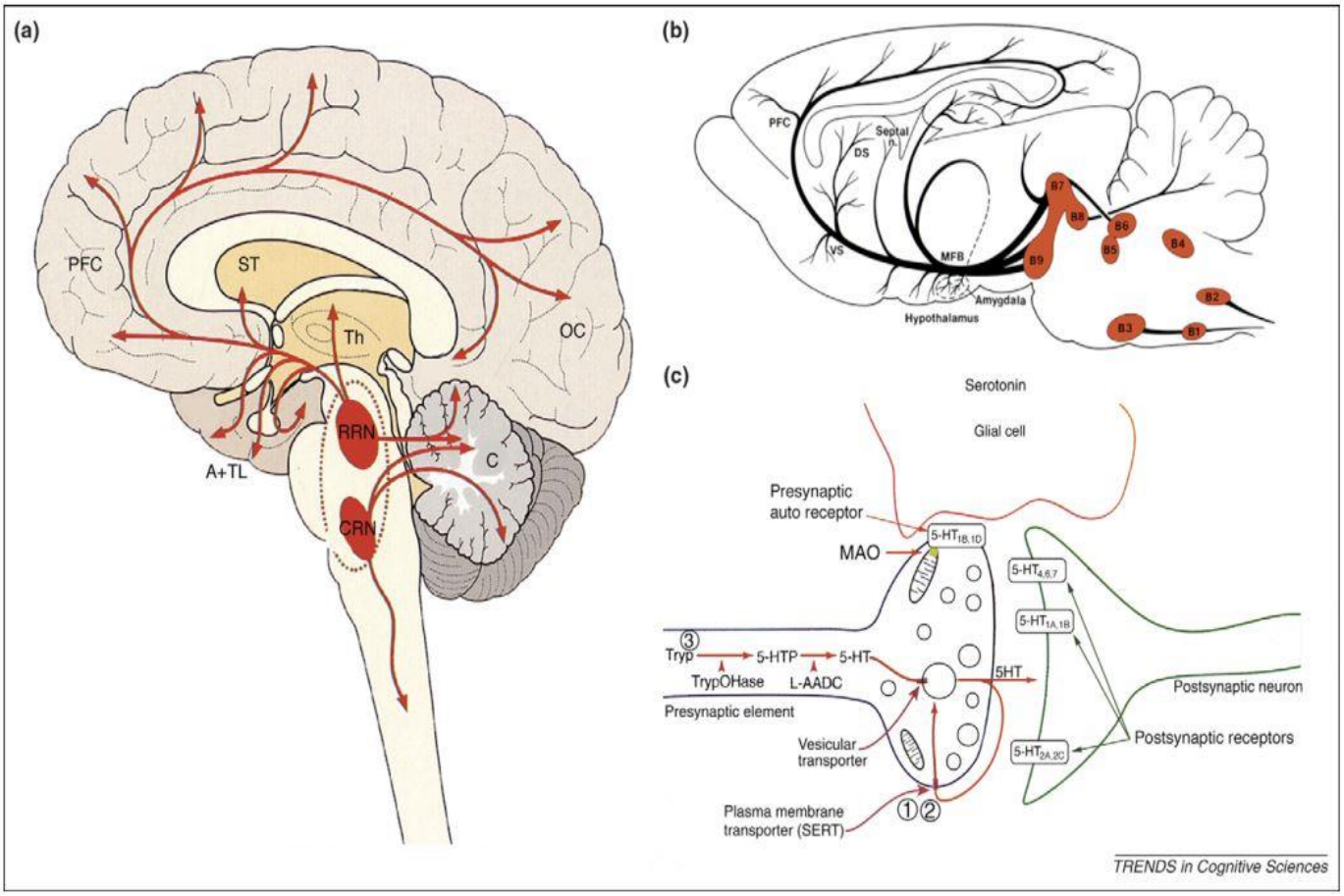


Figure 1. **(a)** Neuroanatomical diagram of serotonergic projections of the Dorsal Raphe Nucleus (DRN). Caudal Raphe Nucleus (CRN) projects down the brainstem to the spinal cord and to the Cerebellum (C). Rostral Raphe Nucleus (RRN) projects to the C, Thalamus (Th), Amygdala (A), Temporal lobe (TL), Striatum (ST), prefrontal cortex (PFC), occipital cortex (OC)

(b) Neuroanatomical projections in a rat brain. **(c)** Serotonergic neuron Tryptophan (Tryp), tryptophan hydroxylase (TrpOHase), 5-hydroxytryptophan (5-HTP), L-amino acid decarboxylase (L-AADC), serotonin transporter (SERT), monoamine oxidase (MAO), Serotonin (5HT) receptors; 5-HT1A, 5-HT1B, 5HT2A,C, etc. at the pre- and postsynaptic sites. (Cools, Roberts, & Robbins, 2008)

Tryptophan to Tryptamine, an alternative pathway

A study, (Christofides *et. al*, 2006), carried out with Tryptophan depletion/loading, in Traumatic Brain Injury (TBI), Huntington's disease (HD) patients and healthy controls, proposed there exists an alternative Tryptophan/Tryptamine pathway to Serotonin (5HT) and Melatonin production, from their findings with Tryptophan depletion, (fasting in TBI). Serotonin (5HT), the 5HT by-product 5-Hydroxyindoleacetic acid (5HIAA) and Melatonin *increased significantly* compared to the control group. However, no conclusive explanation why this resulted was given.

The main differences in results from healthy controls and TBI could be explained due to inhibition of Tryptophan hydroxylase-2 (TPH2), figure 2. TPH2 gene is produced in the brainstem and seems likely that damage to this area significantly reduces TPH2 production in the traditional hydroxylation step of Tryptophan to 5-hydroxytrypotphan (5HTP). This inhibition of TPH2 forces an alternative step of Tryptophan decarboxylating to Tryptamine (TA). Instead of a lack of 5HT production, from the absence of TPH2, an alternative pathway takes place for more 5HT to be produced and cognition to continue. This process could even possibly enhance cognition by activating divergent neurons from other areas, giving rise to creative problem solving through deactivating the default mode network (DMN). Studies have shown DMT's effects on consciousness can be likened to the results found with sleeping and meditation (Palhano-Fontes et al., 2015) with connections in the posterior and anterior cingulate cortex in the DMN, and ability to make distinctions (Li, 2014). This could be one reason, people are not able to distinguish what is real and what is not. As each individual neuron is activated a highly elaborate collection of information could be likened to a series of snapshots, or as a series of holograms, found like in an old flicking movie. With each screen (neuron) added, and slight differences of information developing the overall picture. Thus

appearing very real to the individual seeing it. In the case for smell and auditory, the same mechanisms and concepts could be involved, just different states experienced due to the location in different areas of activation.

Biosynthesis of Serotonin, Melatonin and the Tryptamine Pathway

Biosynthesis of Tryptophan (Tryp) has several pathways. The major pathway, (1) Kynurenine (Jones, Guillemin, & Brew, 2013),

(2) Current understanding, the enzyme Tryptophan hydroxylase-2 (TPH2) for the brain and TRH1 for the body (Walther et al., 2003), produces 5HT by hydroxylation to 5-hydroxytryptophan (5HTP). (Nakamura & Hasegawa, 2009)

(3) Will be explained (figure2), the metabolites from Tryptamine (TA) production.

Decarboxylation, using tryptophan decarboxylase (TDC) (K. Kang, Kang, Lee, Park, & Back, 2008), producing Tryptamine (TA) has a very short half-life of around 18-42 seconds (Juorio, 1990). Yuwen, 2013, also proved by bioinformatics, using the aromatic L-amino acid (AADC) in vitro gene cloning, that Tryptophan forms Tryptamine.

Kang, 2007, showed that the enzyme Tryptamine-5-hydroxylase (T5H), converts Tryptamine (TA) into Serotonin, by hydroxylation (OH-), in rice seedlings. (Kang, S., Kang, Lee, & Back, 2007), figure 2. When this occurs another possible alternative pathway, from TA, methylate's with the enzyme, Indolethylamine-N-methyltransferase INMT (twice)→N,N-dimethyltryptamine (DMT) → hydroxylated DMT produces 5-OH-DMT (Bufotenine) and possibly other naturally occurring psychedelics like, 4-OH-DMT, 5-MeO-DMT, displayed in figure 2. (Rosengarten & Friedhoff, 1976), (Gomes et al., 2014), (Szára, 2007).

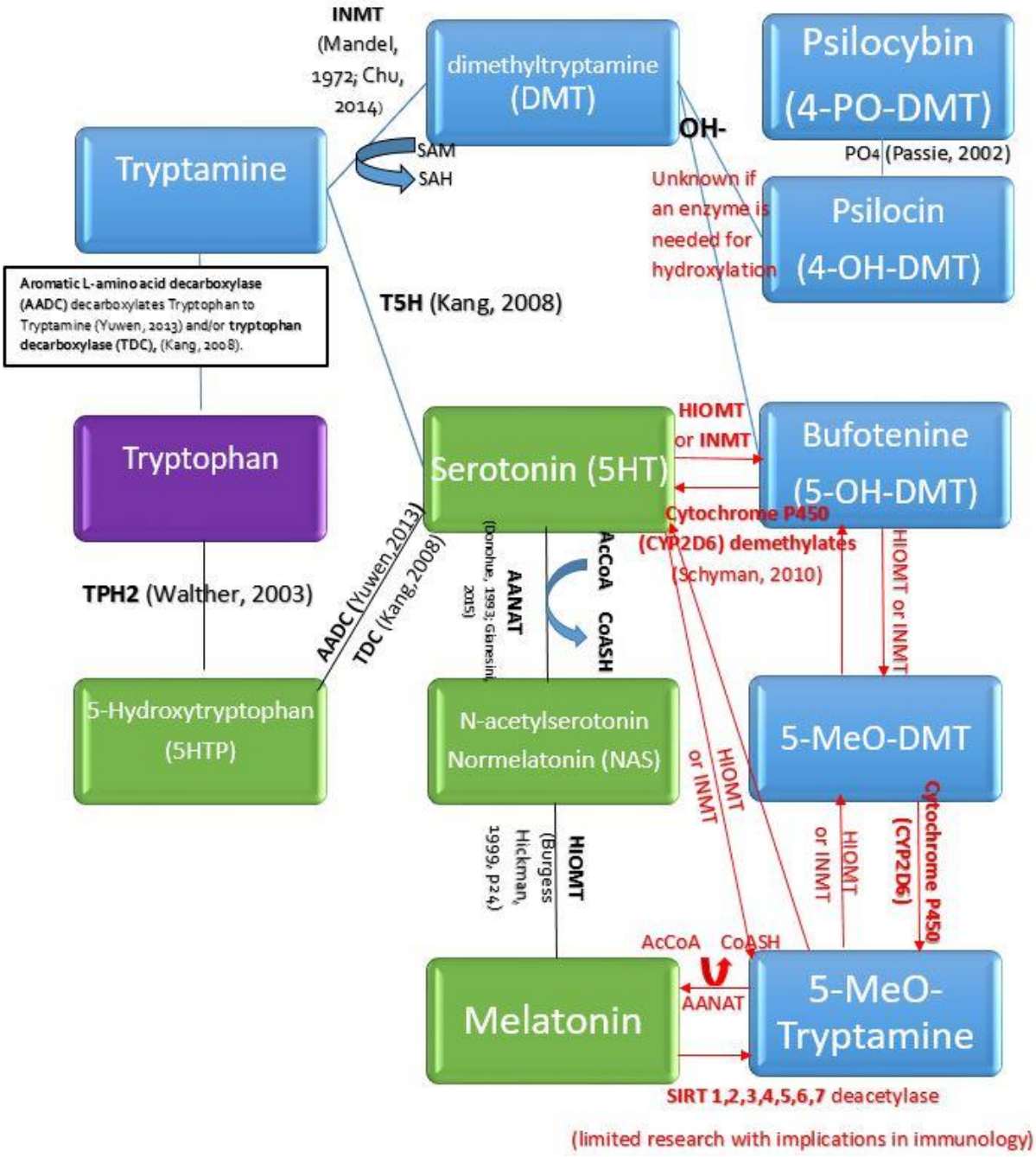


Figure 2. The Tryptophan to Tryptamine neurotransmitter pathway. Biosynthesis model collating studies, enzymes and metabolites of Tryptamine.
Blue represents psychedelic pathway, red highlighting hypothesised pathways and enzymes areas for further research, green shows established and accepted Tryptophan →5HTP→ Serotonin → Melatonin pathway.
HIOMT-hydroxyindole-O-methyltrasferase, **INMT**-Indolethylamine-N-methyltransferase, **TPH2-**Tryptophan hydroxylase-2, **T5H**-Tryptamine hydroxylase enzyme, **OH-** hydroxyl ion, **TDC**- tryptophan decarboxylase, **AANAT**- aralkylamine N-acetyltransferase, **AcCoA**- Acetyl coenzyme A, **CoASH**-coenzyme A, **SIRT NAD+-dependent deacetylase** - sirtuin (silent mating

type information regulation 2 homolog). (Axelrod, 1961; Barker, Borjiginb,Lomnicka, Strassman, 2013; Baxter, Canavier, Clark, & Byrne, 1999; Beaton & Morris, 1984; Burgess Hickman, 1999; Chauhan, 2009; Chilton, Bigwood, & Jensen, 1979; Chu et al., 2014; Cozzi, Gopalakrishnan, Anderson, Feih, Shulgin, Daley, Ruoho, 2009; Cozzi, Mavlyutov, Thompson, Ruoho, 2011; Donohue, 1993; Fontanilla et al., 2009; Funakoshi, 2011; Gianesini, Clesse, Tosini, Hicks, & Laurent, 2015; Gomes et al., 2014; Juorio, 1990; Kang et al., 2008; Kang et al., 2007; Karkkainen, 2005; Lipinski, 2011; Mack, Mulvena, & Slaytor, 1988; Mackay et al., 2006; Mandel, Ahn, & VandenHeuvel, 1972; Matuszak, Reszka, & Chignell, 1997; Meyer, Gehlhaus, Knoth, & Volk, 2007; Nakamura & Hasegawa, 2009; Paredes, 2009; Park et al., 2011; Passie, 2002; Riba et al., 2003; Rosengarten & Friedhoff, 1976; Schyman, 2010; Sekiduka-Kumano, 2013; Shen, Jiang, Winter, & Yu, 2010; Shi et al., 2015; Sullivan et al., 1986; Szabo, 2014; Takahashi, 1985; Takano, 1977; Takeda, 1994; Tan, 2010; Thomas, Adams, Nessler, Brown, & Bohnert, 1995; Thompson et al., 1999; Torrente, Gelenberg, & Vrana, 2012; van der Stelt, Broersen, Olivier, & Westenberg, 2004; Vitale et al., 2011; Walther et al., 2003; Yanai, et. al., 1986; Yang, Fu, Pestell, & Sauve, 2006; Young & Gauthier, 1981; Yuwen et al., 2013)

A study with Traumatic Brain Injury to explain WHY an alternative pathway could exist

Figure 2, inspired by the study, Christofides, 2006, did with trying to find a Tryptophan pathway using patients with Huntington's disease (HD) and Traumatic Brain Injury (TBI). HD is a progressive autosomal disorder with neuronal dysfunction and striatal loss (Roze et al., 2011). Whereas TBI is a rapid neuronal loss, with possible internal bleeding, resulting in inflammation and oxidative stress often damaging the brainstem and thus possibly inhibiting TPH-2 enzyme formation, however, studies would be needed to confirm this. Christofides, comments that the blood results shown in Tryptophan depletion (through fasting over 24 hours and using an amino acid depleting solution) found that Serotonin (5HT) levels did not change in control and HD subjects but **increased** with TBI patients. This seems to contradict the concept that Serotonin is produced from Tryptophan (Young & Gauthier, 1981). To explain these results, Christofides, considered the Kynurenine pathway from previous studies (Stoy et al, 2005; Mackay et al, 2006) with Tryptophan but didn't find any conclusive explanation. "Possibly 'the amino acid concoction' that was used in the depleting of Tryptophan may have

led to the results". This still did not explain the significantly different results HD and controls had to TBI patients. What was not proposed to explain these findings is the hypothesis, *stored endogenous Tryptophan, along with the inhibition of the TPH2 enzyme produces Tryptamine, Serotonin and thus ultimately increases Melatonin.*

Further proposals that require validation include, *in an abundance of Tryptamine with, Tryptamine hydroxylase enzyme (T5H) inhibition, DMT, and by-products are produced*. Or if INMT is not available, hydroxyindole-O-methyltransferase (HIOMT) could be used from 5HT to Bufotenine to 5-Me-O-DMT to Melatonin-based on (Burgess Hickman, 1999). Figure 2.

The following results in figure 3, found from Christofides, 2006, will be referred to with an explanation using the Tryptamine pathway from figure 2.

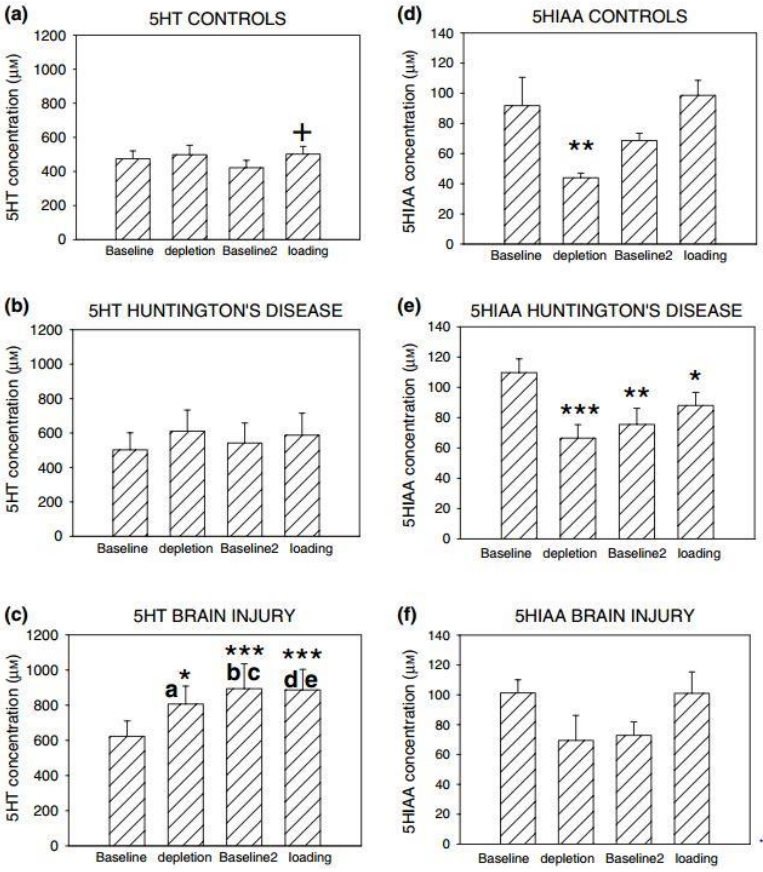


Figure 3. Results displayed as a histogram of *5HT concentration* in (a), (b), (c) versus baseline, tryptophan depletion, baseline2 taken 24 hours after tryptophan depletion and Tryptophan loading (5 hours after baseline2).

5HIAA concentration (d), (e), (f) versus tryptophan depletion, baseline 24hours after tryptophan depletion and Tryptophan loading, (5 hours later), in control, Huntington's disease and traumatic brain injury subjects respectively. (Christofides 2006, page 1081).

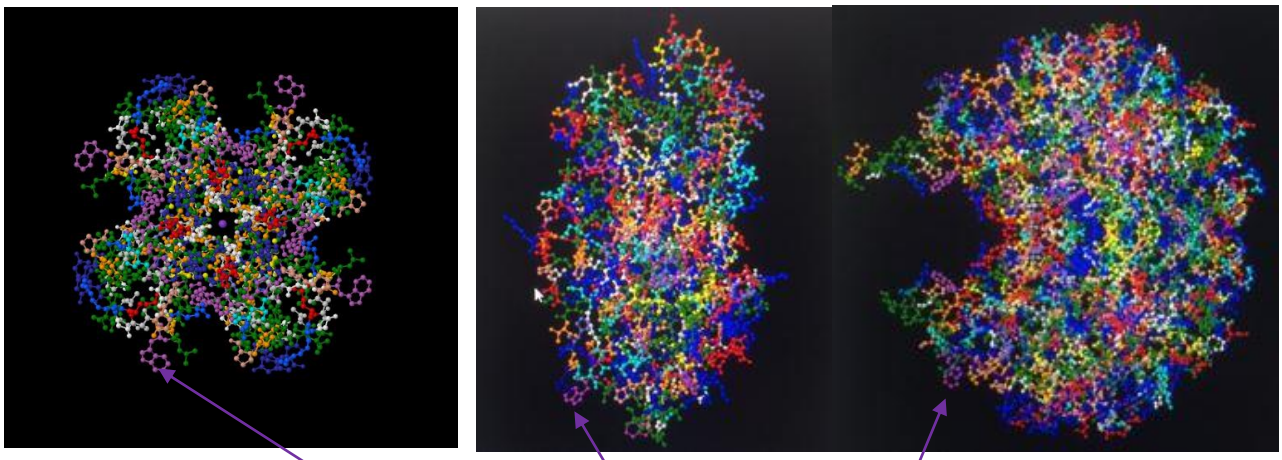
Results of 5HT concentrations observed

The baseline starts around twenty percent higher in TBI patients to start with, which suggests TBI patients are generally producing more 5HT. With Tryptophan depletion in

controls, a minimal increase was observed. With HD patients around twenty percent increase and in TBI around a twenty-five percent increase can be calculated. Thus, TBI has more increase with Tryptophan depletion and somewhat to a lesser degree for HD and nearly no change for controls. These results are interesting as it would have been thought with Tryptophan depletion less 5HT production would result and with Tryptophan loading, more production of 5HT would be observed. (Young & Gauthier, 1981). However, the results showed the reverse in TBI and HD patients. Hence, an alternative pathway was posed to explain the increase in 5HT for Tryptophan depletion in all subjects and most significantly observed in TBI patients.

Tryptophan hydroxylase-2 (TPH-2), produced in the brain stem, could possibly have been inhibited, 'switching' an alternative route to producing Tryptamine using either Aromatic L-amino acid decarboxylase (AADC) or tryptophan decarboxylase (TDC). Since the body was depleted of Tryptophan, stored (endogenous) Tryptophan could have been used for this process, (pictures 1a,b,c below). For example, Potassium channels, monoamine oxidase A (MAOA) and monoamine oxidase B (MAOB), pictures1,2,3, are observed to be composed of Tryptophan which could possibly 'break' off in the decarboxylation process and pick up a Hydrogen ion (H+) to form Tryptamine. HD patients could have fewer neurons, MAOA, MAOB and Potassium channels in comparison to TBI patients. Also, TBI may have a greater number of free radicals from the rapid onset, possible bleeding, inflammation and oxidative stress thus have more hydroxyl ions for reactions with Tryptamine, forming 5HT or methylate's twice to form DMT. It is very difficult to test for Tryptamine with such a short half-life and could be a reason why results are hard to record.

A longitudinal study greater than 2 weeks would be recommended in confirming these explanations, in addition to testing with naturally occurring psychedelics.



Picture1a. Potassium channel **Picture1b** Monoamine Oxidase A (MAOA) **Picture1c** (MAOB)

Molecular structure from, RCSB Protein Data Bank, for (a) 1BL8, Potassium channel (KCSA) from Streptomyces Lividans (b) 2BXS - Human Monoamine Oxidase A, in complex with Clorgyline (c) 4A7A Crystal structure of human monoamine oxidase B (MAO B) in complex with rosiglitazone. Stored endogenous Tryptophan is purple.(Doyle et al., 1998), (De Colibus et al., 2005).

The second baseline for 5HT was taken 24 hours after the first base line. It can be observed that in TBI patients, 5HT continued to rise with Tryptophan depletion quite significantly whereas both control and HD, 5HT had reduced. This is consistent with the explanation that an alternative TA route could exist.

With Tryptophan **loading**, 5HT in both control and HD patients increased as expected but TBI had no significant increase. This can be explained as 5HT was already being produced at significantly higher amounts and 5HT quite likely was also being converted to Melatonin with the results seen in figure 4. Also, psychedelic concentrations were not tested which could also allow for differences.

The 5HIAA concentrations initially found in control, HD and TBI were similar. HD displaying the most 5HIAA and control displayed least. TBI may have slightly less than HD due to higher Melatonin production, and more free radicals to continue reactions utilizing 5HT for Melatonin production. 5HIAA being a by-product of 5HT the results are consistent.

Results for 5HIAA concentrations in Tryptophan depletion

The controls showed around fifty percent decrease, HD around 40% decrease and then least decrease with TBI patients. This supports less Tryptophan means less 5HT is produced and thus less by-product 5HIAA. However, at the same time TBI 5HT went up significantly and 5HIAA decreased. This could possibly be due to Melatonin production. Interestingly, fatigue and tiredness often expressed by TBI patients can be explained by these results. Higher Melatonin, affecting sleep/wake circadian rhythms. (Brown, 1994).

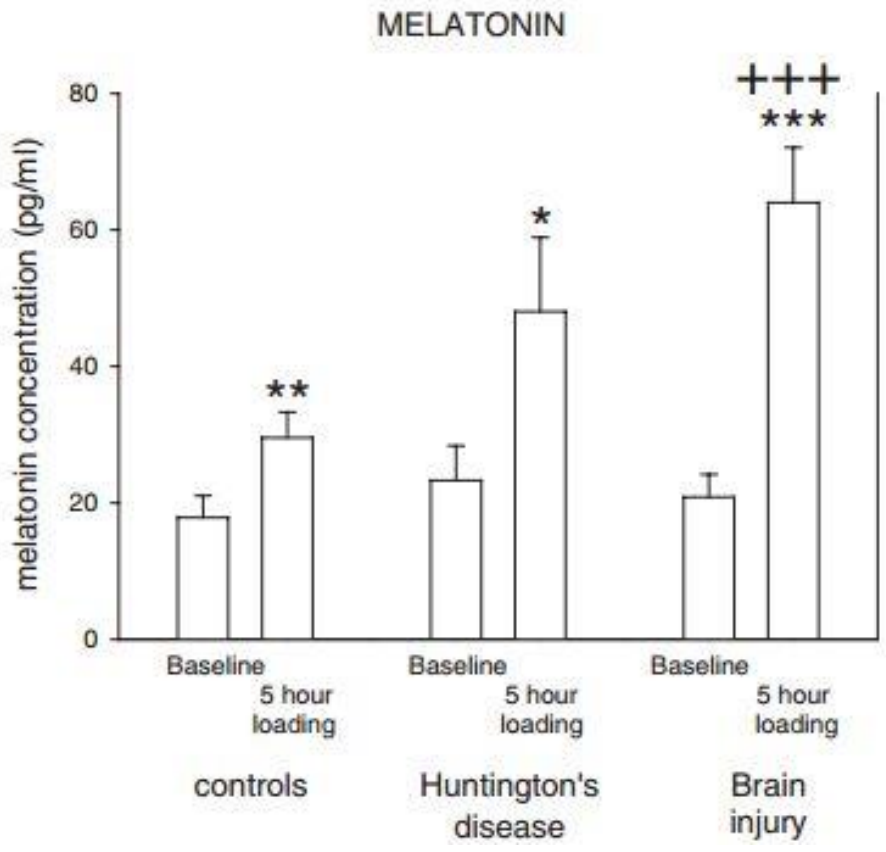


Figure 4. The concentration of Melatonin in control, HD and TBI patients taken initially at baseline and then taken five hours after Tryptophan loading. (Christofides et al., 2006).

Serotonin, Bufotenine, and Psilocin were found in Amanita species, mushrooms, with DMT at low doses. Chilton, 1979 stated, from research done by Nettleship and Slaytor in 1974, “oxidation of 5-hydroxyl indole alkaloids (like Bufotenine) was from tryptamine or a Tryptamine derivative.” The derivative could possibly have been DMT because in the same study, page 66, Udenfriend 1959, confirmed by Gomes, 2011, showed hydroxylation of DMT in cultures of mushrooms resulted with Psilocin. The reaction of Bufotenine produced from DMT was not proposed at the time and the enzymatic methylator-INMT has since been discovered demonstrating DMT can be formed from Tryptamine. (Thompson et al., 1999),(Chu et al., 2014).

Survival mechanism

In young individuals (15-24 years), with the onset of sleep, visual hallucinations have been reported in more than a third of a randomly selected non-institutionalized UK population (Ohayon, Priest, Caulet, & Guilleminault, 1996) and showed consistent and significant decreases with age. This implies that hallucinations or dreams are a normal healthy phenomenon.

People who experience negative hallucinations, as seen in Schizophrenia, could have had various physiological factors that led to significant findings of hydroxylated DMT, Bufotenine (Karkkainen, 2005). Injury, genetic differences, heavy metals (creating more free radical ions) or with further studies possible mutations on of TPH2 gene, ID: 121278 (found on the National Centre for Biotechnology Information-NCBI), chromosome 11p15-p14 and

12q21, updated on 25-Oct-2015. Since internal injuries can't be seen this hallucinogenic 'alert system' is the body's natural result for self-discovery, emotional learning and cognitive mechanisms to continue production of Serotonin and Melatonin hence, survival. With an altered view, patients thinking could be assisted to not be fearful but enlightened from knowledge they gain thus allowing the experiences to be dignifying and less traumatic for the individual. This ultimately benefits them in dealing with and resolving fear responses. No thought (deactivating DMN), no fear.

DMT is difficult to detect, along with Tryptamine, which appears as insignificant findings however, the possibility that these substances could act as free radical scavengers supports antioxidant studies (Frecska, Szabo, Winkelman, Luna, & McKenna, 2013), (Szabo, 2015), and possibly could be rapidly converted into significant Serotonin and Melatonin concentrations found by Christofides, 2006.

Increases of synaptic transmission activates neuronal systems from increases in calcium ions (Ca++). DMT bound to sigma-1 receptors has been found to modulate voltage-gated sodium ions (Na+) channels (Fontanilla et al., 2009). Concentration gradients could also be affected from free radical ions, like hydroxyl ions, Hydrogen ions (H+), Nitric Oxide (NO) being scavenged by DMT and Tryptamine. Hence, understanding how psychedelics interact physiologically is a vital step towards understanding consciousness and subconscious mechanisms of how the mind and body works, learns, sleeps, and heals.

So why the body produces these substances can now be explained as a necessary pathway possibly found in fasting, (Mackay et al., 2006) or enzyme inhibition. If this pathway was not present it is likely that cognitive processes would cease and the individual would have less chance of survival, hence termed the 'survival mechanism'.

Conclusion

The Greek word for psychedelic, psyche-ψυχή, (soul) and dēloun-δηλοῦν, (to make visible or reveal), aptly describes research with psychedelics. Increased understanding of the mind and body is being revealed through these studies despite the many questions that remain unresolved. A few of these questions include why do psychedelic substances like N,N dimethyltryptamine and Bufotenine exist in our bodies and throughout nature? What is the physiological role they have? A natural conclusion would seem that these psychedelic substances, naturally found, are the cause of hallucinations however, what has been discussed is that they are a result of necessary pathways required to produce Serotonin and Melatonin, vital for cognition, sleep/wake cycles and could be considered the 'survival mechanisms'.

Cozzi, 2009, suggests there may exist several mechanisms, at intracellular levels, that result from complex interactions giving rise to hallucinations. Naturally occurring psychoactive psychedelics, like Ayahuasca (DMT and MAOI beta-carbolines) in South America, Peyote (Mescaline) in North America, Magic Mushrooms (Psilocin and Psilocybin) in Siberia, were used for thousands of years leading to current studies investigating psychedelics as medicinal therapies (McKenna, 2007), (Frecska et al., 2013), (Szabo, 2015).

The lack of understanding of endogenous mechanisms that produce Serotonin, Melatonin and hallucinations have been a major hole in many studies. The model, figure 2, of an alternative pathway from Tryptophan to Tryptamine provides an explanation of results observed by Christofides, 2006, for significant production of Serotonin and Melatonin in fasting Traumatic Brain Injury patients who could have experienced inhibition of Tryptophan hydroxylase-2 (TPH2) enzyme. Hallucinations possibly result when neural activation occurs amplifying sensory systems and internally stored information to be retrieved either visually, auditory, tactile, smelt or even possibly tasted. This then could account for a greater chance for

survival, if cognitive systems are inhibited like observed in fasting (Christofides, 2006). Other studies with; Schizophrenia, Alzheimer's, Delirium, PTSD, ear and eye diseases, Parkinson's, Lewy body dementia, Traumatic Brain injuries (TBI), postpartum, drug induced, starvation, affective and sleep disorders, like depression and narcolepsy. (Chaudhury, 2010), (Dobry, Novakovic, Barkin, & Sundaram, 2014), (Mittal & Khan, 2010), (Strassman, 2000) also potentially can be explained.

One metabolite, DMT, has been found to deactivate the default mode network (Palhano-Fontes et al., 2015), related to being able to make distinctions (Li, 2014). Through deactivation of the DMN, it is possible people are not able to decipher senses in perceiving what is real and what is not, hence hallucinate.

Open for discussion is, *how does Tryptamine and DMT hydroxylate to Serotonin and Bufotenine?* As discussed, Tryptamine has a very short half-life and thus may be questionable if an enzyme is needed for hydroxylation to occur. If an enzyme is required further studies into Tryptamine hydroxylase (T5H) could be considered to verify.

Clearly there are significant opportunities for future research with; pharmacology (enzyme supplementation/inhibitors, biomarkers, and medicine), hallucinogenic education, physiology (DMN), and interdisciplinary molecular studies are examples of the boundless and emerging fields in areas of sleep, mental health and immunology.

Studies with psychedelic neurotransmitter pathways could ultimately assist with restructuring drug scheduling policies that would reverse the set-back it has had and advance hallucinogenic research for medicinal discoveries that could prove to have significant economic, and social benefits in health care and employment.

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