Click to verify

Você já ouviu falar sobre melatonina e triptofano? Esses dois aminoácidos são fundamentais para a regulação do sono e, consequentemente, para o bem-estar do seu corpo e mente. Neste artigo, vamos explorar a relação entre essas substâncias e como você pode aproveitar ao máximo seus benefícios para ter uma noite de sono mais tranquila. O triptofano é um aminoácido essencial que nosso organismo não produz naturalmente. Ele deve ser obtido através da alimentação ou suplementação. Esse composto é importante porque é convertido em serotonina, um neurotransmissor responsável por regular diversas funções corporais, incluindo o humor e o ciclo de sono-vigília. Já a melatonina é um hormônio produzido pela glândula pineal durante a noite que ajuda a regular nosso ritmo circadiano – o relógio interno do corpo que controla os ciclos de alerta/sono. Qual é a triptofano? Aí, já ouviu falar do triptofano? Ele é um aminoácido essencial encontrado em alimentos como carnes, ovos e nozes. O triptofano é importante para a produção de neurotransmissores como a serotonina, relacionada ao bem-estar e regulação do humor. Além disso, ele é precursor da melatonina, hormônio responsável pelo sono. Os benefícios do triptofano incluem melhorias na qualidade do sono, redução da ansiedade e estresse e até mesmo aumento da sensação de felicidade. No entanto, é preciso tomar cuidado com os possíveis efeitos colaterais: quando consumido em excesso ou sem orientação médica adequada, o triptofano pode causar náuseas, tonturas e até convulsões. Por isso, é importante se informar bem antes de introduzir suplementos à base desse aminoácido na sua dieta. O papel da serotonina na regulação do sono A presença da serotonina no nosso organismo é fundamental para regular o sono e proporcionar uma noite tranquila. Esse neurotransmissor é responsável por manter nossos níveis de humor estáveis, além de controlar nossa ingestão alimentar. Quando os níveis de serotonina estão baixos, podemos sentir sintomas como ansiedade, irritabilidade e insônia. Os especialistas apontam que a serotonina tem um papel importante na regulação dos ciclos circadianos do sono. Ela ajuda a equilibrar nossos ritmos naturais e melhora a qualidade do nosso descanso. Por isso, é essencial manter uma dieta balanceada em triptofano para garantir que nosso corpo produza quantidades adequadas desse neurotransmissor tão importante. Melatonina e ciclos sono-vigília Você já deve ter ouvido falar sobre a importância do hormônio melatonina na regulação dos ciclos de sono-vigília, certo? Mas você sabia que a produção desse hormônio é influenciada pelo triptofano? Isso mesmo! Esse aminoácido é o precursor da serotonina, neurotransmissor responsável pela sensação de bem-estar e relaxamento, e também da melatonina, que ajuda a regular nosso ritmo circadiano. Para entender melhor essa relação, confira os seguintes pontos: A produção de melatonina acontece naturalmente durante a noite, em resposta à diminuição da luz solar; O triptofano é um aminoácido essencial que não pode ser produzido pelo corpo humano e precisa ser obtido por meio da dieta; Alimentos ricos em triptofano incluem frango, peru, peixe, ovos e laticínios; Quando consumimos alimentos com triptofano, ele é convertido em serotonina no cérebro; Como mencionado anteriormente, a serotonina é o precursor da melatonina. Ou seja: quanto mais serotonina temos disponível no cérebro (por exemplo, após comer uma refeição rica em triptofano), mais facilmente nosso organismo consegue produzir melatonina para ajudar na regulação do sono. Portanto, se você está buscando maneiras naturais de melhorar seu sono e regular seu ritmo circadiano (principalmente se estiver enfrentando problemas como insônia ou jet lag), vale a pena prestar atenção na sua ingestão de triptofano ao longo do dia! A relação entre melatonina e triptofano Você pode potencializar a regulação do seu sono e ritmo circadiano ao consumir alimentos como frango, peru, peixe, ovos e laticínios. Esses alimentos contêm um aminoácido essencial chamado triptofano que é convertido em serotonina, um neurotransmissor responsável pela sensação de bem-estar e relaxamento. A serotonina também é transformada em melatonina durante a noite, hormônio responsável por regular o ciclo sono-vigília. É importante lembrar que a absorção de triptofano é influenciada por outros nutrientes presentes na alimentação, assim como o consumo excessivo de proteínas pode prejudicar sua conversão em serotonina. Em alguns casos quando a produção natural de melatonina não é suficiente para regular o sono, suplementos podem ser utilizados com acompanhamento médico adequado para evitar possíveis efeitos colaterais. Alimentos ricos em triptofano Você pode encontrar muitos alimentos ricos em triptofano, como frango, peru, peixe, ovos e laticínios que podem ajudar na regulação do sono e ritmo circadiano. Além desses alimentos de origem animal, existem também fontes vegetais de triptofano, como a soja, feijões e lentilhas. É importante lembrar que o triptofano é um aminoácido essencial que não é produzido pelo corpo humano e precisa ser obtido através da alimentação. A suplementação com triptofano tem sido estudada para tratar problemas relacionados ao sono e humor. No entanto, sempre consulte um profissional de saúde antes de iniciar qualquer tipo de suplementação. Os benefícios da suplementação com triptofano são mais significativos em pessoas com deficiência desse aminoácido ou em casos específicos indicados por um médico. Lembre-se também de incluir uma variedade de alimentos ricos em triptofano na sua dieta diária para obter os benefícios deste aminoácido naturalmente presente nos alimentos. Dicas para melhorar a qualidade do sono Você já sabe que uma alimentação rica em triptofano pode ajudar a melhorar a qualidade do sono. Mas você sabia que existem outras técnicas para melhorar o seu descanso? Além de se alimentar bem, é preciso cuidar da higiene do sono e adotar algumas técnicas de relaxamento. A higiene do sono consiste em criar um ambiente propício para dormir, como manter o quarto escuro e silencioso, evitar eletrônicos antes de dormir e estabelecer horários regulares para ir para a cama e acordar. Já as técnicas de relaxamento podem incluir meditação, respiração profunda ou mesmo um banho quente antes de dormir. Experimente essas dicas e sinta a diferença na qualidade do seu sono! Conclusão Agora que você entende a relação entre melatonina e triptofano, pode usar esse conhecimento para melhorar a qualidade do seu sono. Certifique-se de incluir alimentos ricos em triptofano em sua dieta, como peru, frango, peixe e nozes. Além disso, tente regular seus ciclos de sono acordando e dormindo ao mesmo tempo todos os dias e evitando dispositivos eletrônicos antes de dormir. Com um pouco de planejamento e disciplina, é possível melhorar sua saúde geral através da promoção de um sono adequado. Boa sorte! InChI=1S/C11H12N2O2/c12-9(11(14)15)5-7-6-13-10-4-2-1-3-8(7)10/h1-4,6,9,13H,5,12H2,(H,14,15)/t9-/m0/s1InChIKeyInChIKey=QIVBCDIJIAJPQS-VIFPVBQESA-NSMILESC([C@@H](C(O)=O)N)C=1C=2C(NC1)=CC=CC2Canonical SMILESO=C(O)C(N)CC1=CNC=2C=CC=CC21Other Names for this SubstanceL-TryptophanTryptophan, L-l-β-3-IndolylalanineTryptophaneTryptophanDeleted or Replaced CAS Registry Numbers6912-86-3, 80206-30-0, 154635-35-5, 2416148-24-6, 2831375-12-1 O triptofano é um aminoácido essencial importante para a produção de serotonina e melatonina, regulando o humor, o sono e o apetite. A suplementação, quando indicada, pode melhorar a qualidade do sono, controlar os sintomas de depressão e ansiedade e apoiar a função cognitiva. Vale lembrar que, apesar dos potenciais benefícios em muitas situações, faltam estudos acerca da sua real eficácia em muitas delas. Continue a leitura para saber mais sobre as indicações de triptofano. Agendar consulta O que é triptofano? O triptofano é um aminoácido essencial que o corpo humano não pode sintetizar, ou seja, ele deve ser obtido por meio da dieta. Ele é um precursor na síntese de serotonina e melatonina. Esses hormônios atuam no sistema nervoso, ajudando a regular o humor, o sono, a memória e o apetite. Além disso, o triptofano também é necessário para a síntese de proteínas, manutenção dos músculos e outras funções metabólicas importantes do organismo. De forma geral, o triptofano é utilizado no tratamento e na prevenção de depressão, ansiedade, insônia e no processo de emagrecimento. Apesar do amplo uso, em muitas situações não existem evidências claras acerca da sua eficácia. Exame de triptofano: o que pode indicar? O exame de triptofano pode diagnosticar e monitorar diversas condições de saúde. Entre elas podemos citar: Deficiências nutricionais. Problemas no metabolismo. Investigar possíveis deficiências de triptofano que causam sintomas como fadiga, depressão, insônia e alterações no apetite. Avaliar o tratamento de doenças relacionadas à serotonina, como depressão e ansiedade. Monitorar o estado nutricional de pacientes com doenças crônicas, como renais, hepáticas ou intestinais, que podem afetar a absorção de triptofano. Auxiliar no diagnóstico de tumores raros, como carcinoides (que são ligados a secreção de hormônios). Vale ressaltar que não existe indicação de sua dosagem de rotina em situações de check-up. Para que serve o triptofano? Quando é indicado? O triptofano é um aminoácido essencial que desempenha várias funções importantes no organismo. Portanto, potencialmente pode oferecer benefícios nas seguintes situações, quando devidamente indicado: Auxílio no tratamento a depressão juntamente com outros medicamentos. Fortalecimento os músculos. Controle a ansiedade. Promoção do bem-estar. Melhoria a memória. Diminuição do estresse. Regulação do sono. Modulação do apetite. Auxílio no tratamento do déficit de atenção, hiperatividade, fadiga crônica e sintomas da síndrome pré-menstrual. Por outro lado, não há evidências para seu uso rotineiro em adultos saudáveis. Qual é a relação entre triptofano e melatonina? O triptofano é um aminoácido essencial que serve como precursor para a produção de melatonina. Ele é convertido em serotonina, que é posteriormente transformada em melatonina. Este hormônio é responsável por regular o ciclo sono-vigília, ajudando a promover um sono de qualidade. Portanto, uma ingestão adequada de triptofano pode ajudar a melhorar a produção de melatonina e, consequentemente, a qualidade do sono. Quais são os efeitos colaterais do triptofano? O uso de triptofano, principalmente na forma de suplementos, pode causar alguns efeitos colaterais. Entre os mais comuns estão: Náuseas Sonolência Dor de cabeça Tonturas Em casos mais raros, provoca: Boca seca Visão turva Dores musculares Importante observar o rótulo do suplemento. Muitos deles contêm outras substâncias não declaradas na sua composição. Portanto, procurar a informação acerca da certificação por alguma agência regulatória independente. Triptofano engorda? Não. O triptofano potencialmente ajuda a modular o apetite e melhorar o sono, fatores que contribuem para o controle de peso. Alimentos ricos em triptofano Alguns alimentos ricos em triptofano incluem: Carnes Peixes e frutos do mar Laticínios Ovos Queijos Iogurte Leguminosas, como feijão, grão-de-bico e lentilha Oleaginosas e sementes Cereais integrais, como aveia e arroz Frutas, como banana e abacate chocolate amargo Qual é o melhor horário para tomar triptofano? Para os indivíduos com indicação desse suplemento, a recomendação é ingerir o suplemento à noite, pouco antes de dormir. Isso ajuda a aumentar a produção de melatonina, hormônio que regula o sono e o ciclo sono-vigília. Portanto, promove um sono de qualidade. Quando o triptofano é indicado para regular o humor e controlar a ansiedade, a indicação é tomar de manhã ou à tarde. A estratégia é ajudar a manter a serotonina mais estável ao longo do dia. Qual médico procurar? O triptofano é um aminoácido essencial para o corpo e pode ser prescrito por profissional nutricionista, não apenas médicos. Abaixo a lista de especialistas úteis nessa avaliação:Psiquiatras Neurologistas Geriatras Nutricionistas Médicos de família Agendar consulta Fonte: Dra. Lívia Beraldo, psiquiatra O triptofano é um aminoácido essencial para a formação e manutenção dos músculos e para a produção de serotonina e melatonina, compostos que atuam no sistema nervoso, ajudando a regular o humor, o sono, a memória e o apetite, sendo usado no tratamento e na prevenção de depressão, ansiedade, insônia e no processo de emagrecimento. Encontre um Nutricionista perto de você! Parceria com Buscar Médico Por ser um aminoácido essencial, ou seja, que o organismo não consegue produzir, o triptofano deve ser obtido a partir da ingestão de alimentos como chocolate amargo, nozes, ovos e amêndoas, por exemplo. Veja outros alimentos ricos em triptofano. Além disso, o triptofano também pode ser obtido através da suplementação, como 5HTP, ou L-triptofano, em cápsulas. No entanto, esses suplementos devem ser consumidos apenas sob a orientação de um nutricionista, ou médico. Para que serve O triptofano é um aminoácido que participa de diversas funções no organismo, sendo indicado para: Combater a depressão; Formar e manter os músculos; Controlar a ansiedade; Promover o bem estar; Melhorar a memória; Diminuir o estresse; Regular o sono; Ajudar no emagrecimento. Além disso, o triptofano também é utilizado para ajudar no tratamento da dor, do déficit de atenção, da hiperatividade, da fadiga crônica e da TPM. O neurotransmissor serotonina ajuda na formação do hormônio melatonina que regula o ritmo do relógio biológico do organismo, melhorando a qualidade do sono, já que a melatonina é produzida durante a noite. Lista de alimentos ricos em triptofano Alguns alimentos que são ricos em triptofano são: Queijos; Ovos; Tofu; Salmão; Nozes; Amêndoas; Amendoim; Castanha-do-Pará; Banana. Além disso, o triptofano também pode ser encontrado na forma de suplementos, como 5-HTP, ou L-triptofano, em cápsulas, sendo indicada a ingestão de 100mg a 3 g por dia, de acordo com a situação a ser tratada. Entenda como usar o suplemento de triptofano. Triptofano ajuda a emagrecer? O triptofano participa da produção de serotonina, um neurotransmissor que promove o controle da ansiedade e depressão, diminuindo a compulsão alimentar e ajudando, assim, a emagrecer. Veja como usar o triptofano para ajudar no emagrecimento. A alimentação muitas vezes está relacionada com as emoções e, por isso, momentos de ansiedade e depressão podem estimular a ingestão de alimentos que promovem o prazer, como os alimentos ricos em carboidratos e gorduras, incluindo chocolates, sorvetes, bolos e frituras, por exemplo, aumentando as calorias da dieta e favorecendo, assim o ganho de peso. Ingestão diária recomendada A ingestão diária de triptofano, varia de acordo com a idade, o sexo e a fase da vida, como indicado na tabela a seguir: Além disso, mulheres grávidas precisam ingerir 7 mg / Kg de peso corporal, por dia, de triptofano. Já as mulheres que amamentam, devem consumir 9 mg / Kg de peso corporal, de triptofano por dia. "Tryptan" redirects here. For the type of anti-migraine drug, see Triptan. For the hydrocarbon, see Triptane. l-Tryptophan Skeletal formula of L-tryptophan ball-and-stick model[1] space-filling model[1] Names IUPAC name Tryptophan Systematic IUPAC name 2-Amino-3-(1H-indol-3-yl)propanoic acid Other names 2-Amino-3-(1H-indol-3-yl)propionic acid Identifiers CAS Number 73-22-3 Y 3D model (JSmol) Interactive imageZwitterion: Interactive image ChEBI CHEBI:16828 ChEMBL ChEMBL54976 Y ChemSpider 6066 Y DrugBank DB00150 Y ECHA InfoCard 100.000.723 IUPHAR/BPS 717 KEGG D00020 Y PubChem CID 6305 UNII 8DUH1N11BX Y CompTox Dashboard (EPA) DTXSID5021419 InChI InChI=1S/C11H12N2O2/c12-9(11(14)15)5-7-6-13-10-4-2-1-3-8(7)10/h1-4,6,9,13H,5,12H2,(H,14,15)/t9-/m0/s1 YKey: QIVBCDIJIAJPQS-VIFPVBQESA-N Y SMILES c1[nH]c2ccccc2c1C[C@H](N)C(=O)OZwitterion: c1[nH]c2ccccc2c1C[C@H]([NH3+])C(=O)[O-] Properties Chemical formula C11H12N2O2 Molar mass 204.229 g·mol−1 Solubility in water Soluble: 0.23 g/L at 0 °C, 11.4 g/L at 25 °C, 17.1 g/L at 50 °C, 27.95 g/L at 75 °C Solubility Soluble in hot alcohol, alkali hydroxides; insoluble in chloroform. Acidity (pKa) 2.38 (carboxyl), 9.39 (amino)[2] Magnetic susceptibility (χ) −132.0·10−6 cm3/mol Pharmacology ATC code N06AX02 (WHO) Supplementary data page Tryptophan (data page) Except where otherwise noted, data are given for materials in their standard state (at 25 °C [77 °F], 100 kPa). Infobox references Chemical compound Tryptophan ball and stick model spinning Tryptophan (symbol Trp or W)[3] is an α-amino acid that is used in the biosynthesis of proteins. Tryptophan contains an α-amino group, an α-carboxylic acid group, and a side chain indole, making it a polar molecule with a non-polar aromatic beta carbon substituent. Tryptophan is also a precursor to the neurotransmitter serotonin, the hormone melatonin, and vitamin B3 (niacin).[4] It is encoded by the codon UGG. Like other amino acids, tryptophan is a zwitterion at physiological pH where the amino group is protonated (–NH+3; pKa = 9.39) and the carboxylic acid is deprotonated (–COO−; pKa = 2.38).[5] Humans and many animals cannot synthesize tryptophan: they need to obtain it through their diet, making it an essential amino acid. Tryptophan is named after the digestive enzymes trypsin, which were used in its first isolation from casein proteins.[6] It was assigned the one-letter symbol W based on the double ring being visually suggestive to the bulky letter.[7] Metabolism of l-tryptophan into serotonin and melatonin (left) and niacin (right). Transformed functional groups after each chemical reaction are highlighted in red. Amino acids, including tryptophan, are used as building blocks in protein biosynthesis, and proteins are required to sustain life. Tryptophan is among the less common amino acids found in proteins, but it plays important structural or functional roles whenever it occurs. For instance, tryptophan and tyrosine residues play special roles in "anchoring" membrane proteins within the cell membrane. Tryptophan, along with other aromatic amino acids, is also important in glycan-protein interactions. In addition, tryptophan functions as a biochemical precursor for the following compounds: Serotonin (a neurotransmitter), synthesized by tryptophan hydroxylase.[8][9] Melatonin (a neurohormone) is in turn synthesized from serotonin, via N-acetyltransferase and 5-hydroxyindole-O-methyltransferase enzymes.[10] Kynurenine, to which tryptophan is mainly (more than 95%) metabolized. Two enzymes, namely indoleamine 2,3-dioxygenase (IDO) in the immune system and the brain, and tryptophan 2,3-dioxygenase (TDO) in the liver, are responsible for the synthesis of kynurenine from tryptophan. The kynurenine pathway of tryptophan catabolism is altered in several diseases, including psychiatric disorders such as schizophrenia,[11] major depressive disorder,[11] and bipolar disorder.[11][12] Niacin, also known as vitamin B3, is synthesized from tryptophan via kynurenine and quinolinic acids.[13] Auxins (a class of phytohormones) are synthesized from tryptophan.[14] The disorder fructose malabsorption causes improper absorption of tryptophan in the intestine, reduced levels of tryptophan in the blood,[15] and depression.[16] In bacteria that synthesize tryptophan, high cellular levels of this amino acid activate a repressor protein, which binds to the trp operon.[17] Binding of this repressor to the tryptophan operon prevents transcription of downstream DNA that codes for the enzymes involved in the biosynthesis of tryptophan. So high levels of tryptophan prevent tryptophan synthesis through a negative feedback loop, and when the cell's tryptophan levels go down again, transcription from the trp operon resumes. This permits tightly regulated and rapid responses to changes in the cell's internal and external tryptophan levels. Tryptophan metabolism by human gut microbiota (vte) Tryptophan Clostridiumsporogenes Lacto-bacilli Tryptophanase-expressingbacteria IPA I3A Indole Liver Brain IPA I3A Indole Indoxylsulfate AST-120 AhR Intestinalimmunecells Intestinalepithelium PXR Mucosal homeostasis: ↓TNF-α ↑Junction protein-coding mRNAs L cell GLP-1 T J Neuroprotectant: ↓Activation of glial cells and astrocytes ↓4-Hydroxy-2-nonenal levels ↓DNA damage–Antioxidant–Inhibits β-amyloid fibril formation Maintains mucosal reactivity: ↑IL-22 production Associated with vascular disease: ↑Oxidative stress ↑Smooth muscle cell proliferation ↑Aortic wall thickness and calcification Associated with chronic kidney disease: ↑Renal dysfunction–Uremic toxin Kidneys This diagram shows the biosynthesis of bioactive compounds (indole and certain other derivatives) from tryptophan by bacteria in the gut.[18] Indole is produced from tryptophan by bacteria that express tryptophanase.[18] Clostridium sporogenes metabolizes tryptophan into indole and subsequently 3-indolepropionic acid (IPA),[19] a highly potent neuroprotective antioxidant that scavenges hydroxyl radicals.[18][20][21] IPA binds to the pregnane X receptor (PXR) in intestinal cells, thereby facilitating mucosal homeostasis and barrier function.[18] Following absorption from the intestine and distribution to the brain, IPA confers a neuroprotective effect against cerebral ischemia and Alzheimer's disease.[18] Lactobacillaceae (Lactobacillus s.l.) species metabolize tryptophan into indole-3-aldehyde (I3A) which acts on the aryl hydrocarbon receptor (AhR) in intestinal immune cells, in turn increasing interleukin-22 (IL-22) production.[18] Indole itself triggers the secretion of glucagon-like peptide-1 (GLP-1) in intestinal L cells and acts as a ligand for AhR.[18] Indole can also be metabolized by the liver into indoxyl sulfate, a compound that is toxic in high concentrations and associated with vascular disease and renal dysfunction.[18] AST-120 (activated charcoal), an intestinal sorbent that is taken by mouth, adsorbs indole, in turn decreasing the concentration of indoxyl sulfate in blood plasma.[18] In 2002, the U.S. Institute of Medicine set a Recommended Dietary Allowance (RDA) of 5 mg/kg body weight/day of tryptophan for adults 19 years and over.[22] Tryptophan is present in most protein-based foods or dietary proteins. It is particularly plentiful in chocolate, oats, dried dates, milk, yogurt, cottage cheese, red meat, eggs, fish, poultry, sesame, chickpeas, almonds, sunflower seeds, pumpkin seeds, hemp seeds, buckwheat, spirulina, and peanuts. Contrary to the popular belief[23][24] that cooked turkey contains an abundance of tryptophan, the tryptophan content in turkey is typical of poultry.[25] Tryptophan (Trp) content of various foods[25][26] Food Tryptophan [g/100 g of food] Protein [g/100 g of food] Tryptophan/protein [%] Egg white, dried 1.00 81.10 1.23 Spirulina, dried 0.92 57.47 1.62 Cod, Atlantic, dried 0.70 62.82 1.11 Soybeans, raw 0.59 36.49 1.62 Cheese, Parmesan 0.56 37.90 1.47 Chia seeds, dried 0.44 16.50 2.64 Sesame seed 0.37 17.00 2.17 Hemp seed, hulled 0.37 31.56 1.17 Cheese, Cheddar 0.32 24.90 1.29 Sunflower seed 0.30 17.20 1.74 Pork, chop 0.25 19.27 1.27 Turkey 0.24 21.89 1.11 Chicken 0.24 20.85 1.14 Beef 0.23 20.13 1.12 Oats 0.23 16.89 1.39 Salmon 0.22 19.84 1.12 Lamb, chop 0.21 18.33 1.17 Perch, Atlantic 0.21 18.62 1.12 Chickpeas, raw 0.19 19.30 0.96 Egg 0.17 12.58 1.33 Wheat flour, white 0.13 10.33 1.23 Baking chocolate, unsweetened 0.13 12.90 1.23 Milk 0.08 3.22 2.34 Rice, white, medium-grain, cooked 0.03 2.38 1.18 Quinoa, uncooked 0.17 14.12 1.20 Quinoa, cooked 0.05 4.40 1.10 Potatoes, russet 0.02 2.14 0.84 Tamarind 0.02 2.80 0.64 Banana 0.01 1.03 0.87 Because tryptophan is converted into 5-hydroxytryptophan (5-HTP) which is then converted into the neurotransmitter serotonin, it has been proposed that consumption of tryptophan or 5-HTP may improve depression symptoms by increasing the level of serotonin in the brain. Tryptophan is sold over the counter in the United States (after being banned to varying extents between 1989 and 2005) and the United Kingdom as a dietary supplement for use as an antidepressant, anxiolytic, and sleep aid. It is also marketed as a prescription drug in some European countries for the treatment of major depression. There is evidence that blood tryptophan levels are unlikely to be altered by changing the diet,[27][28] but consuming purified tryptophan increases the serotonin level in the brain, whereas eating foods containing tryptophan does not.[29] In 2001 a Cochrane review of the effect of 5-HTP and tryptophan on depression was published. The authors included only studies of a high rigor and included both 5-HTP and tryptophan in their review because of the limited data on either. Of 108 studies of 5-HTP and tryptophan on depression published between 1966 and 2000, only two met the authors' quality standards for inclusion, totaling 64 study participants. The substances were more effective than placebo in the two studies included but the authors state that "the evidence was of insufficient quality to be conclusive" and note that "because alternative antidepressants exist which have been proven to be effective and safe, the clinical usefulness of 5-HTP and tryptophan is limited at present".[30] The use of tryptophan as an adjunctive therapy in addition to standard treatment for mood and anxiety disorders is not supported by the scientific evidence.[30][31] The American Academy of Sleep Medicine's 2017 clinical practice guidelines recommended against the use of tryptophan in the treatment of insomnia due to poor effectiveness.[32] Potential side effects of tryptophan supplementation include nausea, diarrhea, drowsiness, lightheadedness, headache, dry mouth, blurred vision, sedation, euphoria, and nystagmus (involuntary eye movements).[33][34] Tryptophan taken as a dietary supplement (such as in tablet form) has the potential to cause serotonin syndrome when combined with antidepressants of the MAOI or SSRI class or other strongly serotonergic drugs.[34] Because tryptophan supplementation has not been thoroughly studied in a clinical setting, its interactions with other drugs are not well known.[30] The isolation of tryptophan was first reported by Frederick Hopkins in 1901.[35] Hopkins recovered tryptophan from hydrolysed casein, recovering 4–8 g of tryptophan from 600 g of crude casein.[36] As an essential amino acid, tryptophan is not synthesized from simpler substances in humans and other animals, so it needs to be present in the diet in the form of tryptophan-containing proteins. Plants and microorganisms commonly synthesize tryptophan from shikimic acid or anthranilate:[37] anthranilate condenses with phosphoribosylpyrophosphate (PRPP), generating pyrophosphate as a by-product. The ring of the ribose moiety is opened and subjected to reductive decarboxylation, producing indole-3-glycerol phosphate; this, in turn, is transformed into indole. In the last step, tryptophan synthase catalyzes the formation of tryptophan from indole and the amino acid serine. The industrial production of tryptophan is also biosynthetic and is based on the fermentation of serine and indole using either wild-type or genetically modified bacteria such as B. amyloliquefaciens, B. subtilis, C. glutamicum or E. coli. These strains carry mutations that prevent the reuptake of aromatic amino acids or multiple/overexpressed trp operons. The conversion is catalyzed by the enzyme tryptophan synthase.[38][39][40] There was a large outbreak of eosinophilia-myalgia syndrome (EMS) in the U.S. in 1989, with more than 1,500 cases reported to the CDC and at least 37 deaths.[41] After preliminary investigation revealed that the outbreak was linked to intake of tryptophan, the U.S. Food and Drug Administration (FDA) recalled tryptophan supplements in 1989 and banned most public sales in 1990,[42][43][44] with other countries following suit.[45][46] Subsequent studies suggested that EMS was linked to specific batches of L-tryptophan supplied by a single large Japanese manufacturer, Showa Denko.[42][47][48][49] It eventually became clear that recent batches of Showa Denko's L-tryptophan were contaminated by trace impurities, which were subsequently thought to be responsible for the 1989 EMS outbreak.[42][50][51] However, other evidence suggests that tryptophan itself may be a potentially major contributory factor in EMS.[52] There are also claims that a precursor reached sufficient concentrations to form a toxic dimer.[53] The FDA loosened its restrictions on sales and marketing of tryptophan in February 2001,[42] but continued to limit the importation of tryptophan not intended for an exempted use until 2005.[54] The fact that the Showa Denko facility used genetically engineered bacteria to produce the contaminated batches of L-tryptophan later found to have caused the outbreak of eosinophilia-myalgia syndrome has been cited as evidence of a need for "close monitoring of the chemical purity of biotechnology-derived products".[55] Those calling for purity monitoring have, in turn, been criticized as anti-GMO activists who overlook possible non-GMO causes of contamination and threaten the development of biotech.[56] See also: Postprandial somnolence § Turkey and tryptophan A common assertion in the US and the UK[57] is that heavy consumption of turkey meat—as seen during Thanksgiving and Christmas—results in drowsiness, due to high levels of tryptophan contained in turkey.[24] However, the amount of tryptophan in turkey is comparable with that of other meats.[23][25] Drowsiness after eating may be caused by other foods eaten with the turkey, particularly carbohydrates.[58] Ingestion of a meal rich in carbohydrates triggers the release of insulin.[59][60][61][62] Insulin in turn stimulates the uptake of large neutral branched-chain amino acids (BCAA), but not tryptophan, into muscle, increasing the ratio of tryptophan to BCAA in the blood stream. The resulting increased tryptophan ratio reduces competition at the large neutral amino acid transporter (which transports both BCAA and aromatic amino acids), resulting in more uptake of tryptophan across the blood–brain barrier into the cerebrospinal fluid (CSF).[62][63][64] Once in the CSF, tryptophan is converted into serotonin in the raphe nuclei by the normal enzymatic pathway.[60][65] The resultant serotonin is further metabolised into the hormone melatonin—which is an important mediator of the circadian rhythm[66]—by the pineal gland.[10] Hence, these data suggest that "feast-induced drowsiness"—or postprandial somnolence—may be the result of a heavy meal rich in carbohydrates, which indirectly increases the production of melatonin in the brain, and thereby promotes sleep.[59][60][61][65] In 1912 Felix Ehrlich demonstrated that yeast metabolizes the natural amino acids essentially by splitting off carbon dioxide and replacing the amino group with a hydroxyl group. By this reaction, tryptophan gives rise to tryptophol.[67] Tryptophan affects brain serotonin synthesis when given orally in a purified form and is used to modify serotonin levels for research.[29] Low brain serotonin level is induced by administration of tryptophan-poor protein in a technique called acute tryptophan depletion.[68] Studies using this method have evaluated the effect of serotonin on mood and social behavior, finding that serotonin reduces aggression and increases agreeableness.[69] See also: 5-Hydroxytryptophan § Psychedelic effects Tryptophan produces the head-twitch response (HTR) in rodents when administered at sufficiently high doses.[70] The HTR is induced by serotonergic psychedelics like lysergic acid diethylamide (LSD) and psilocybin and is a behavioral proxy of psychedelic effects.[71][72] Tryptophan is converted into the trace amine tryptamine and tryptamine is N-methylated by indolethylamine N-methyltransferase (INMT) into N-methyltryptamine (NMT) and N,N-dimethyltryptamine (N,N-DMT), which are known serotonergic psychedelics.[70][73][74][75][76][77] Main article: Fluorescence spectroscopy § Tryptophan fluorescence Tryptophan is an important intrinsic fluorescent probe (amino acid), which can be used to estimate the nature of the microenvironment around the tryptophan residue. Most of the intrinsic fluorescence emissions of a folded protein are due to excitation of tryptophan residues. 5-Hydroxytryptophan (5-HTP) α-Methyltryptophan Acree–Rosenheim reaction Adamkiewicz reaction Attenuator (genetics) N,N-Dimethyltryptamine Hopkins–Cole reaction Serotonin Tryptamine ^ a b Görbitz CH, Törnroos KW, Day GM (2012). "Single-crystal investigation of L-tryptophan with Z′ = 16". Acta Crystallographica Section B. 68 (Pt 5): 549–557. doi:10.1107/S0108768112033484. PMID 22992800. ^ Dawson RM, et al. (1969). Data for Biochemical Research. Oxford: Clarendon Press. ISBN 0-19-855338-2. ^ "Nomenclature and Symbolism for Amino Acids and Peptides". IUPAC-IUB Joint Commission on Biochemical Nomenclature. 1983. Archived from the original on 2 December 2021. Retrieved 22 October 2022. ^ Slominski A, Semak I, Pisarchik A, Sweatman T, Szczesniewski A, Wortsman J (2002). "Conversion of L-tryptophan to serotonin and melatonin in human melanoma cells". FEBS Letters. 511 (1–3): 102–6. Bibcode:2002FEBSL.511..102S. doi:10.1016/s0014-5793(01)03319-1. PMID 11821057. S2CID 7820568. ^ "L-tryptophan | C11H12N2O2 - PubChem". pubchem.ncbi.nlm.nih.gov. Retrieved 22 December 2016. ^ Curzon G (31 December 1987), Bender DA, Joseph MH, Kochen W, Steinhart H (eds.), "Hopkins and the Discovery of Tryptophan", Progress in Tryptophan and Serotonin Research 1986, Berlin, Boston: De Gruyter, pp. XXIX–XL, doi:10.1515/9783110854657-004, ISBN 978-3-11-085465-7, retrieved 19 February 2024 ^ "IUPAC-IUB Commission on Biochemical Nomenclature A One-Letter Notation for Amino Acid Sequences". Journal of Biological Chemistry. 243 (13): 3557–3559. 10 July 1968. doi:10.1016/S0021-9258(19)34176-6. ^ Fernstrom JD (1983). "Role of precursor availability in control of monoamine biosynthesis in brain". Physiological Reviews. 63 (2): 484–546. doi:10.1152/physrev.1983.63.2.484. PMID 6132421. ^ Schaechter JD, Wurtman RJ (1990). "Serotonin release varies with brain tryptophan levels" (PDF). Brain Research. 532 (1–2): 203–10. doi:10.1016/0006-8993(90)91761-5. PMID 1704290. S2CID 8451316. Archived from the original (PDF) on 9 August 2020. Retrieved 30 May 2014. ^ a b Wurtman RJ, Anton-Tay F (1969). "The mammalian pineal as a neuroendocrine transducer" (PDF). Recent Progress in Hormone Research. 25: 493–522. doi:10.1016/b978-0-12-571125-8.50014-4. ISBN 978-0-12-571125-8. PMID 4391290. Archived from the original (PDF) on 31 May 2014. ^ a b c Marx W, McGuinness AJ, Rocks T, Ruusunen A, Cleminson J, Walker AJ, Gomes-da-Costa S, Lane M, Sanches M, Diaz AP, Tseng PT (23 November 2020). "The kynurenine pathway in major depressive disorder, bipolar disorder, and schizophrenia: a meta-analysis of 101 studies". Molecular Psychiatry. 26 (8): 4158–4178. doi:10.1038/s41380-020-00951-9. ISSN 1476-5578. PMID 33230205. S2CID 227132820. ^ Bartoli F, Misiak B, Callovini T, Cavaleri D, Cioni RM, Crocamo C, Savitz JB, Carrà G (19 October 2020). "The kynurenine pathway in bipolar disorder: a meta-analysis on the peripheral blood levels of tryptophan and related metabolites". Molecular Psychiatry. 26 (7): 3419–3429. doi:10.1038/s41380-020-00913-1. PMID 33077852. S2CID 224314102. ^ Ikeda M, Tsuji H, Nakamura S, Ichiyama A, Nishizuka Y, Hayaishi O (1965). "Studies on the biosynthesis of nicotinamide adenine dinucleotide. II. A role of picolinic carboxylase in the biosynthesis of nicotinamide adenine dinucleotide from tryptophan in mammals". The Journal of Biological Chemistry. 240 (3): 1395–401. doi:10.1016/S0021-9258(18)97589-7. PMID 14284754. ^ Palme K, Nagy F (2008). "A new gene for auxin synthesis". Cell. 133 (1): 31–2. doi:10.1016/j.cell.2008.03.014. PMID 18394986. S2CID 9949830. ^ Ledochowski M, Widner B, Murr C, Sperner-Unterweger B, Fuchs D (2001). "Fructose malabsorption is associated with decreased plasma tryptophan" (PDF). Scandinavian Journal of Gastroenterology. 36 (4): 367–71. doi:10.1080/003655201300051135. PMID 11336160. Archived from the original (PDF) on 19 April 2016. ^ Ledochowski M, Sperner-Unterweger B, Widner B, Fuchs D (June 1998). "Fructose malabsorption is associated with early signs of mental depression". European Journal of Medical Research. 3 (6): 295–8. PMID 9620891. ^ Gollnick P, Babitzke P, Antson A, Yanofsky C (2005). "Complexity in regulation of tryptophan biosynthesis in Bacillus subtilis". Annual Review of Genetics. 39: 47–68. doi:10.1146/annurev.genet.39.073003.093745. PMID 16285852. ^ a b c d e f g h i Zhang LS, Davies SS (April 2016). "Microbial metabolism of dietary components to bioactive metabolites: opportunities for new therapeutic interventions". Genome Med. 8 (1): 46. doi:10.1186/s13073-016-0296-x. PMC 4840492. PMID 27102537. Lactobacillus spp. convert tryptophan to indole-3-aldehyde (I3A) through unidentified enzymes [125]. Clostridium sporogenes convert tryptophan to IPA [6], likely via a tryptophan deaminase. ... IPA also potently scavenges hydroxyl radicalsTable 2: Microbial metabolites: their synthesis, mechanisms of action, and effects on health and diseaseFigure 1: Molecular mechanisms of action of indole and its metabolites on host physiology and disease ^ Wikoff WR, Anfora AT, Liu J, Schultz PG, Lesley SA, Peters EC, Siuzdak G (March 2009). "Metabolomics analysis reveals large effects of gut microflora on mammalian blood metabolites". Proc. Natl. Acad. Sci. U.S.A. 106 (10): 3698–3703. Bibcode:2009PNAS..106.3698W. doi:10.1073/pnas.0812874106. PMC 2656143. PMID 19234110. Production of IPA was shown to be completely dependent on the presence of gut microflora and could be established by colonization with the bacterium Clostridium sporogenes.IPA metabolism diagram ^ "3-Indolepropionic acid". Human Metabolome Database. University of Alberta. Retrieved 12 June 2018. ^ Chyan YJ, Poeggeler B, Omar RA, Chain DG, Frangione B, Ghiso J, Pappolla MA (July 1999). "Potent neuroprotective properties against the Alzheimer beta-amyloid by an endogenous melatonin-related indole structure, indole-3-propionic acid". J. Biol. Chem. 274 (31): 21937–21942. doi:10.1074/jbc.274.31.21937. PMID 10419516. S2CID 6630247. [Indole-3-propionic acid (IPA)] has previously been identified in the plasma and cerebrospinal fluid of humans, but its functions are not known. ... In kinetic competition experiments using free radical-trapping agents, the capacity of IPA to scavenge hydroxyl radicals exceeded that of melatonin, an indoleamine considered to be the most potent naturally occurring scavenger of free radicals. In contrast with other antioxidants, IPA was not converted to reactive intermediates with pro-oxidant activity. ^ Institute of Medicine (2002). "Protein and Amino Acids". Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Washington, DC: The National Academies Press. pp. 589–768. doi:10.17226/10490. ISBN 978-0-309-08525-0. ^ a b Ballantyne C (21 November 2007). "Does Turkey Make You Sleepy?". Scientific American. Retrieved 6 June 2013. ^ a b McCue K. "Chemistry.org: Thanksgiving, Turkey, and Tryptophan". Archived from the original on 4 April 2007. Retrieved 17 August 2007. ^ a b c Holden J. "USDA National Nutrient Database for Standard Reference, Release 22". Nutrient Data Laboratory, Agricultural Research Service, United States Department of Agriculture. Retrieved 29 November 2009. ^ Rambali B, Van Andel I, Schenk E, Wolterink G, van de Werken G, Stevenson H, Vleeming W (2002). "[The contribution of cocoa additive to cigarette smoking addiction]" (PDF). RIVM (report 650270002/2002). The National Institute for Public Health and the Environment (Netherlands). Archived from the original (PDF) on 8 November 2005. ^ Soh NL, Walter GT (2011). "Tryptophan and depression: can diet alone be the answer?". Acta Neuropsychiatrica. 23 (1): 1601–5215. doi:10.1111/j.1601-5215.2010.00508.x. S2CID 145779393. ^ Fernstrom JD (2012). "Effects and side effects associated with the non-nutritional use of tryptophan by humans". The Journal of Nutrition. 142 (12): 2236S – 2244S. doi:10.3945/jn.111.157065. PMID 23077193. ^ a b Wurtman RJ, Hefti F, Melamed E (1980). "Precursor control of neurotransmitter synthesis". Pharmacological Reviews. 32 (4): 315–35. doi:10.1016/S0031-6997(25)06841-3. PMID 6115400. ^ a b c Shaw K, Turner J, Del Mar C (2002). Shaw KA (ed.). "Tryptophan and 5-hydroxytryptophan for depression" (PDF). The Cochrane Database of Systematic Reviews. 2010 (1): CD003198. doi:10.1002/14651858.CD003198. PMID 11869656. ^ Ravindran AV, da Silva TL (September 2013). "Complementary and alternative therapies as add-on to pharmacotherapy for mood and anxiety disorders: a systematic review". Journal of Affective Disorders. 150 (3): 707–19. doi:10.1016/j.jad.2013.05.042. PMID 23769610. ^ Sateia MJ, Buysse DJ, Krystal AD, Neubauer DN, Heald JL (February 2017). "Clinical Practice Guideline for the Pharmacologic Treatment of Chronic Insomnia in Adults: An American Academy of Sleep Medicine Clinical Practice Guideline". J Clin Sleep Med. 13 (2): 307–349. doi:10.5664/jcsm.6470. PMC 5263087. PMID 27998379. ^ Kimura T, Bier DM, Taylor CL (December 2012). "Summary of workshop discussions on establishing upper limits for amino acids with specific attention to available data for the essential amino acids leucine and tryptophan". The Journal of Nutrition. 142 (12): 2245S – 2248S. doi:10.3945/jn.112.160846. PMID 23077196. ^ a b Howland RH (June 2012). "Dietary supplement drug therapies for depression". Journal of Psychosocial Nursing and Mental Health Services. 50 (6): 13–6. doi:10.3928/02793695-20120508-06. PMID 22589230. ^ Hopkins FG, Cole SW (December 1901). "A contribution to the chemistry of proteids: Part I. A preliminary study of a hitherto undescribed product of tryptic digestion". The Journal of Physiology. 27 (4–5): 418–428. doi:10.1113/jphysiol.1901.sp000880. PMC 1540554. PMID 16992614. ^ Cox G, King H (1930). "L-Tryptophane". Org. Synth. 10: 100. doi:10.15227/orgsyn.010.0100. ^ Radwanski ER, Last RL (1995). "Tryptophan biosynthesis and metabolism: biochemical and molecular genetics". The Plant Cell. 7 (7): 921–34. doi:10.1105/tpc.7.7.921. PMC 160888. PMID 7640526. ^ Ikeda M (2002). "Amino acid production processes". Microbial Production of l-Amino Acids. Advances in Biochemical Engineering/Biotechnology. Vol. 79. pp. 1–35. doi:10.1007/3-540-45989-8_1. ISBN 978-3-540-43383-5. PMID 12523387. ^ Becker J, Wittmann C (2012). "Bio-based production of chemicals, materials and fuels -Corynebacterium glutamicum as versatile cell factory". Current Opinion in Biotechnology. 23 (4): 631–40. doi:10.1016/j.copbio.2011.11.012. PMID 22138494. ^ Conrado RJ, Varner JD, DeLisa MP (2008). "Engineering the spatial organization of metabolic enzymes: mimicking nature's synergy". Current Opinion in Biotechnology. 19 (5): 492–9. doi:10.1016/j.copbio.2008.07.006. PMID 18725290. ^ Allen J, Varga J (2014). "Eosinophilia–Myalgia Syndrome". In Wexler P (ed.). Encyclopedia of Toxicology (3rd ed.). Burlington: Elsevier Science. ISBN 978-0-12-386455-0. ^ a b c d "Information Paper on L-tryptophan and 5-hydroxy-L-tryptophan". FU. S. Food and Drug Administration, Center for Food Safety and Applied Nutrition, Office of Nutritional Products, Labeling, and Dietary Supplements. 1 February 2001. Archived from the original on 25 February 2005. Retrieved 8 February 2012. ^ "L-tryptophan: Uses and Risks". WebMD. 12 May 2017. Retrieved 5 June 2017. ^ Altman LK (27 April 1990). "Studies Tie Disorder to Maker of Food Supplement". The New York Times. ^ Castot A, Bidault I, Bournerias I, Carlier P, Efthymiou ML (1991). "["Eosinophilia-myalgia" syndrome due to L-tryptophan containing products. Cooperative evaluation of French Regional Centers of Pharmacovigilance. Analysis of 24 cases]". Thérapie. 46 (5): 355–65. PMID 1754978. ^ "COT Statement on Tryptophan and the Eosinophilia-Myalgia Syndrome" (PDF). UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. June 2004. ^ Slutsker L, Hoesly FC, Miller L, Williams LP, Watson JC, Fleming DW (July 1990). "Eosinophilia-myalgia syndrome associated with exposure to tryptophan from a single manufacturer". JAMA. 264 (2): 213–7. doi:10.1001/jama.264.2.213. PMID 2355442. ^ Back EE, Henning KJ, Kallenbach LR, Brix KA, Gunn RA, Melius JM (April 1993). "Risk factors for developing eosinophilia myalgia syndrome among L-tryptophan users in New York". The Journal of Rheumatology. 20 (4): 666–72. PMID 8496862. ^ Kilbourne EM, Philen RM, Kamb ML, Falk H (October 1996). "Tryptophan produced by Showa Denko and epidemic eosinophilia-myalgia syndrome". The Journal of Rheumatology. Supplement. 46: 81–8, discussion 89–91. PMID 8895184. ^ Mayeno AN, Lin F, Foote CS, Loegering DA, Ames MM, Hedberg CW, Gleich GJ (December 1990). "Characterization of "peak E," a novel amino acid associated with eosinophilia-myalgia syndrome". Science. 250 (4988): 1707–8. Bibcode:1990Sci...250.1707M. doi:10.1126/science.2270484. PMID 2270484. ^ Ito J, Hosaki Y, Torigoe Y, Sakimoto K (January 1992). "Identification of substances formed by decomposition of peak E substance in tryptophan". Food and Chemical Toxicology. 30 (1): 71–81. doi:10.1016/0278-6915(92)90139-C. PMID 1544609. ^ Smith MJ, Garrett RH (November 2005). "A heretofore undisclosed crux of eosinophilia-myalgia syndrome: compromised histamine degradation". Inflammation Research. 54 (11): 435–50. doi:10.1007/s00011-005-1380-7. PMID 16307217. S2CID 7785345. ^ Michael Predator Carlton. "Molecular Biology and Genetic Engineering explained by someone who's done it". Archived from the original on 24 June 2007. ^ Allen JA, Peterson A, Sufit R, Hinchcliff ME, Mahoney JM, Wood TA, Miller FW, Whitfield ML, Varga J (November 2011). "Post-epidemic eosinophilia-myalgia syndrome associated with L-tryptophan". Arthritis and Rheumatism. 63 (11): 3633–9. doi:10.1002/art.30514. PMC 3848710. PMID 21702023. ^ Mayeno AN, Gleich GJ (September 1994). "Eosinophilia-myalgia syndrome and tryptophan production: a cautionary tale". Trends in Biotechnology. 12 (9): 346–52. doi:10.1016/0167-7799(94)90035-3. PMID 7765187. ^ Raphals P (November 1990). "Does medical mystery threaten biotech?". Science. 250 (4981): 619. Bibcode:1990Sci...250..619R. doi:10.1126/science.2237411. PMID 2237411. ^ Harding N (21 December 2023). "How to stop Christmas food from ruining your sleep". The Telegraph. ISSN 0307-1235. Retrieved 25 December 2023. ^ "Food & mood. (neuroscience professor Richard Wurtman) (Interview)". Nutrition Action Healthletter. September 1992.[dead link] ^ a b Lyons PM, Truswell AS (March 1988). "Serotonin precursor influenced by type of carbohydrate meal in healthy adults". The American Journal of Clinical Nutrition. 47 (3): 433–9. doi:10.1093/ajcn/47.3.433. PMID 3279747. ^ a b c Wurtman RJ, Wurtman JJ, Regan MM, McDermott JM, Tsay RH, Breu JJ (January 2003). "Effects of normal meals rich in carbohydrates or proteins on plasma tryptophan and tyrosine ratios". The American Journal of Clinical Nutrition. 77 (1): 128–32. doi:10.1093/ajcn/77.1.128. PMID 12499331. ^ a b Afaghi A, O'Connor H, Chow CM (February 2007). "High-glycemic-index carbohydrate meals shorten sleep onset". The American Journal of Clinical Nutrition. 85 (2): 426–30. doi:10.1093/ajcn/85.2.426. PMID 17284739. ^ a b Banks WA, Owen JB, Erickson MA (2012). "Insulin in the Brain: There and Back Again". Pharmacology & Therapeutics. 136 (1): 82–93. doi:10.1016/j.pharmthera.2012.07.006. ISSN 0163-7258. PMC 4134675. PMID 22820012. ^ Pardridge WM, Oldendorf WH (August 1975). "Kinetic analysis of blood-brain barrier transport of amino acids". Biochimica et Biophysica Acta (BBA) - Biomembranes. 401 (1): 128–36. doi:10.1016/0005-2736(75)90347-8. PMID 1148286. ^ Maher TJ, Glaeser BS, Wurtman RJ (May 1984). "Diurnal variations in plasma concentrations of basic and neutral amino acids and in red cell concentrations of aspartate and glutamate: effects of dietary protein intake". The American Journal of Clinical Nutrition. 39 (5): 722–9. doi:10.1093/ajcn/39.5.722. PMID 6538743. ^ a b Fernstrom JD, Wurtman RJ (1971). "Brain serotonin content: increase following ingestion of carbohydrate diet". Science. 174 (4013): 1023–5. Bibcode:1971Sci...174.1023F. doi:10.1126/science.174.4013.1023. PMID 5120086. S2CID 14345137. ^ Atul Khullar MD (10 July 2012). "The Role of Melatonin in the Circadian Rhythm Sleep-Wake Cycle". Psychiatric Times. 29 (7). ^ Jackson RW (1930). "A synthesis of tryptophol" (PDF). Journal of Biological Chemistry. 88 (3): 659–662. doi:10.1016/S0021-9258(18)76755-0. ^ Young SN (September 2013). "Acute tryptophan depletion in humans: a review of theoretical, practical and ethical aspects". Journal of Psychiatry & Neuroscience. 38 (5): 294–305. doi:10.1503/jpn.120209. PMC 3756112. PMID 23428157. ^ Young SN (2013). "The effect of raising and lowering tryptophan levels on human mood and social behaviour". Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences. 368 (1615): 20110375. doi:10.1098/rstb.2011.0375. PMC 3638380. PMID 23440461. ^ a b Halberstadt AL, Geyer MA (2018). "Effect of Hallucinogens on Unconditioned Behavior". Behavioral Neurobiology of Psychedelic Drugs. Current Topics in Behavioral Neurosciences. Vol. 36. pp. 159–199. doi:10.1007/7854_2016_466. ISBN 978-3-662-55878-2. PMC 5787039. PMID 28224459. ^ Canal CE, Morgan D (2012). "Head-twitch response in rodents induced by the hallucinogen 2,5-dimethoxy-4-iodoamphetamine: a comprehensive history, a re-evaluation of mechanisms, and its utility as a model". Drug Test Anal. 4 (7–8): 556–576. doi:10.1002/dta.1333. PMC 3722587. PMID 22517680. ^ Kozlenkov A, González-Maeso J (2013). "Animal Models and Hallucinogenic Drugs". The Neuroscience of Hallucinations. New York, NY: Springer New York. pp. 253–277. doi:10.1007/978-1-4614-4121-2_14. ISBN 978-1-4614-4120-5. ^ Carbonaro TM, Gatch MB (September 2016). "Neuropharmacology of N,N-dimethyltryptamine". Brain Res Bull. 126 (Pt 1): 74–88. doi:10.1016/j.brainresbull.2016.04.016. PMC 5048497. PMID 27126737. Endogenous DMT is synthesized from the essential amino acid tryptophan, which is decarboxylated to tryptamine. Tryptamine is then transmethylated by the enzyme indolethylamine-N-methyltransferase (INMT) (using S-adenosyl methionine as a substrate), which catalyzes the addition of methyl groups resulting in the production of N-methyltryptamine (NMT) and DMT. NMT can also act as a substrate for INMT-dependent DMT biosynthesis (Barker et al., 1981). ^ Barker SA (2018). "N, N-Dimethyltryptamine (DMT), an Endogenous Hallucinogen: Past, Present, and Future Research to Determine Its Role and Function". Front Neurosci. 12: 536. doi:10.3389/fnins.2018.00536. PMC 6088236. PMID 30127713. After the discovery of an indole-N-methyl transferase (INMT; Axelrod, 1961) in rat brain, researchers were soon examining whether the conversion of tryptophan (2, Figure 2) to tryptamine (TA; 3, Figure 2) could be converted to DMT in the brain and other tissues from several mammalian species. Numerous studies subsequently demonstrated the biosynthesis of DMT in mammalian tissue preparations in vitro and in vivo (Saavedra and Axelrod, 1972; Saavedra et al., 1973). In 1972, Juan Saavedra and Julius Axelrod reported that intracisternally administered TA was converted to N-methyltryptamine (NMT; 4, Figure 2) and DMT in the rat, the first demonstration of DMT's formation by brain tissue in vivo. ^ Cameron LP, Olson DE (October 2018). "Dark Classics in Chemical Neuroscience: N, N-Dimethyltryptamine (DMT)" (PDF). ACS Chem Neurosci. 9 (10): 2344–2357. doi:10.1021/acschemneuro.8b00101. PMID 30036036. Like serotonin and melatonin, DMT is a product of tryptophan metabolism.25 Following tryptophan decarboxylation, tryptamine is methylated by an N-methyltransferase (i.e., INMT) with S-adenosylmethionine serving as the methyl donor. A second enzymatic methylation produces DMT (Figure 3A).26 [...] The enzyme indolethylamine N-methyltransferase (INMT) catalyzes the methylation of a variety of biogenic amines, and is responsible for converting tryptamine into DMT in mammals.140 ^ Colosimo FA, Borsellino P, Krider RI, Marquez RE, Vida TA (26 February 2024). "The Clinical Potential of Dimethyltryptamine: Breakthroughs into the Other Side of Mental Illness, Neurodegeneration, and Consciousness". Psychoactives. 3 (1). MDPI AG: 93–122. doi:10.3390/psychoactives3010007. ISSN 2813-1851. The metabolism of DMT within the body begins with its synthesis. Endogenous DMT is made from tryptophan after decarboxylation transforms it into tryptamine [22,25]. Tryptamine then undergoes transmethylation mediated by indolethylamine-N-methyltransferase (INMT) with S-adenosyl methionine (SAM) as a substrate, morphing into N-methyltryptamine (NMT) and eventually producing N,N-DMT [26]. Intriguingly, INMT is distributed widely across the body, predominantly in the lungs, thyroid, and adrenal glands, with a dense presence in the anterior horn of the spinal cord. Within the cerebral domain, regions such as the uncus, medulla, amygdala, frontal cortex, fronto-parietal lobe, and temporal lobe exhibit INMT activity, primarily localized in the soma [26]. INMT transcripts are found in specific brain regions, including the cerebral cortex, pineal gland, and choroid plexus, in both rats and humans. Although the rat brain is capable of synthesizing and releasing DMT at concentrations similar to established monoamine neurotransmitters like serotonin [27], the possibility that DMT is an authentic neurotransmitter is still speculative. This issue has been controversial for decades [28] and requires the demonstration of an activity-dependent release (i.e., Ca2+-stimulated) of DMT at a synaptic cleft to be fully established in the human brain. ^ Araújo AM, Carvalho F, Bastos Mde L, Guedes de Pinho P, Carvalho M (August 2015). "The hallucinogenic world of tryptamines: an updated review". Arch Toxicol. 89 (8): 1151–1173. Bibcode:2015ArTox..89.1151A. doi:10.1007/s00204-015-1513-x. PMID 25877327. Wood RM, Rilling JK, Sanfey AG, Bhagwagar Z, Rogers RD (May 2006). "Effects of tryptophan depletion on the performance of an iterated Prisoner's Dilemma game in healthy adults". Neuropsychopharmacology. 31 (5): 1075–84. doi:10.1038/sj.npp.1300932. PMID 16407905. Huang Y, Zhao M, Chen X, Zhang R, Le A, Hong M, Zhang Y, Jia L, Zang W, Jiang C, Wang J, Fan X, Wang J (June 2023). "Tryptophan Metabolism in Central Nervous System Diseases: Pathophysiology and Potential Therapeutic Strategies". Aging Dis. 14 (3): 858–878. doi:10.14336/AD.2022.0916. PMC 10187711. PMID 37191427. "KEGG PATHWAY: Tryptophan metabolism - Homo sapiens". KEGG: Kyoto Encyclopedia of Genes and Genomes. 23 August 2006. Retrieved 20 April 2008. G. P. Moss. "Tryptophan Catabolism (early stages)". Nomenclature Committee of the International Union of Biochemistry and Molecular Biology (NC-IUBMB). Archived from the original on 13 September 2003. Retrieved 20 April 2008. G. P. Moss. "Tryptophan Catabolism (later stages)". Nomenclature Committee of the International Union of Biochemistry and Molecular Biology (NC-IUBMB). Archived from the original on 13 September 2003. Retrieved 20 April 2008. B. Mikkelson, D. P. Mikkelson (22 November 2007). "Turkey Causes Sleepiness". Urban Legends Reference Pages. Snopes.com. Retrieved 20 April 2008. Retrieved from " 2Not to be confused with 5-hydroxytryptamine. This article is about 5-hydroxytryptophan as a biological compound. For its role as a medication and supplement, see Oxitriptan. 5-Hydroxytryptophan Names IUPAC name 2-amino-3-(5-hydroxy-1H-indol-3-yl)propanoic acid Other names 5-HTP; Oxitriptan; α-Carboxy-5-hydroxytryptamine; α-Carboxy-5-HT Identifiers CAS Number 56-69-9 N 3D model (JSmol) Interactive image ChEBI CHEBI:17780 Y ChEMBL ChEMBL350221 Y ChemSpider 388413 Y ECHA InfoCard 100.022.193 IUPHAR/BPS 4671 KEGG D07339 Y MeSH 5-Hydroxytryptophan PubChem CID 144 UNII C1LJO185Q9 Y CompTox Dashboard (EPA) DTXSID1025437 InChI InChI=1S/C11H12N2O3/c12-9(11(15)16)3-6-5-13-10-2-1-7(14)4-8(6)10/h1-2,4-5,9,13-14H,3,12H2,(H,15,16)/t9-/m0/s1 YKey: LDCYZAJDBXYCGN-VIFPVBQESA-N YInChI=1/C11H12N2O3/c12-9(11(15)16)3-6-5-13-10-2-1-7(14)4-8(6)10/h1-2,4-5,9,13-14H,3,12H2,(H,15,16)/t9-/m0/s1Key: LDCYZAJDBXYCGN-VIFPVBQEBZ SMILES O=C(O)C[C@@H](N)Cc2c1cc(O)ccc1[nH]c2 Properties Chemical formula C11H12N2O3 Molar mass 220.228 g·mol−1 Density 1.484 g/mL Melting point 298 to 300 °C (568 to 572 °F; 571 to 573 K) Boiling point 520.6 °C (969.1 °F; 793.8 K) Except where otherwise noted, data are given for materials in their standard state (at 25 °C [77 °F], 100 kPa). N verify (what is YN ?) Infobox references Chemical compound 5-Hydroxytryptophan (5-HTP), used medically as oxitriptan, is a naturally occurring amino acid and chemical precursor as well as a metabolic intermediate in the biosynthesis of the neurotransmitter serotonin. 5-HTP can be manufactured and used as a drug and supplement with the INNTooltip International Nonproprietary Name oxitriptan. Brand names include Cincofarm, Levothym, Levotonine, Oxyfan, Telesol, Tript-OH, and Triptum. As a drug, it is used in the

treatment of depression and for certain other indications. 5-HTP is produced from the amino acid tryptophan through the action of the enzyme tryptophan hydroxylase. Tryptophan hydroxylase is one of the biopterin-dependent aromatic amino acid hydroxylases. Production of 5-HTP is the rate-limiting step in 5-HT (serotonin) synthesis. 5-HTP is normally rapidly converted to 5-HT by amino acid decarboxylase.[1] 5-HTP is decarboxylated to serotonin (5-hydroxytryptamine or 5-HT) by the enzyme aromatic-L-amino-acid decarboxylase with the help of vitamin B6.[2] This reaction occurs both in nervous tissue and in the liver.[3] 5-HTP crosses the blood–brain barrier,[4] while 5-HT does not. Excess 5-HTP, especially when administered with vitamin B6, is thought to be metabolized and excreted.[5][6] Metabolic pathway from tryptophan to serotonin. 5-HTP AAAD Serotonin PLP See also: Tryptophan § Dietary sources Though 5-HTP is found in food only in insignificant quantities, it is a chemical involved intermediately in the metabolism of tryptophan, an amino acid found in all unfractionated foods, with lower total amino acid content correlating with increased tryptophan absorption.[7] Main article: Oxitriptan 5-HTP is used medically and as a supplement under the name oxitriptan in the treatment of depression and for certain other indications. It can be potentiated in combination with a peripherally selective aromatic L-amino acid decarboxylase (AAAD) inhibitor such as carbidopa or benserazide. These agents increase the strength and duration of oxitriptan. An investigational combination formulation is oxitriptan/carbidopa. See also: Tryptophan § Psychedelic effects 5-HTP robustly produces the head-twitch response (HTR) in rodents when administered at relatively high doses.[8][9][10][11][12] It dose-dependently induces the HTR in mice across a dose range of 50 to 250 mg/kg via intraperitoneal administration, with an inverted U-shaped dose–response curve and maximal induction of the HTR at a dose of 200 mg/kg.[12][1] Similarly to the case of 5-HTP, intracerebroventricular injection of serotonin, but not peripheral administration of serotonin, produces the HTR.[9][8][11] The HTR is induced by serotonergic psychedelics like lysergic acid diethylamide (LSD) and psilocybin and is a behavioral proxy of psychedelic effects.[13][8] The HTR of 5-HTP is blocked by serotonin 5-HT2A receptor antagonists, which block the hallucinogenic effects of serotonergic psychedelics in humans, is prevented by aromatic L-amino acid decarboxylase (AAAD) inhibitors, which block conversion of 5-HTP into serotonin, and is potentiated by monoamine oxidase A (MAO-A) inhibitors, which prevent the degradation of serotonin and other endogenous tryptamines.[9][8][10][11][12] It is also suppressed by the serotonin 5-HT1A receptor full agonist 8-OH-DPAT, is greatly augmented by the serotonin 5-HT2C receptor antagonist RS-102221, and is reduced by the trace amine-associated receptor 1 (TAAR1) antagonist EPPTB.[12] In addition, the HTR of 5-HTP is abolished by indolethylamine N-methyltransferase (INMT) inhibitors, which block conversion of serotonin and other endogenous tryptamines into N-methylated tryptamines, such as N-methylserotonin (NMS; norbufotenin), bufotenin (5-hydroxy-N,N-dimethyltryptamine; 5-HO-DMT), and N,N-dimethyltryptamine (DMT).[8][14][11] These N-methylated tryptamines are well-known for their psychedelic effects, whereas serotonin itself, without biotransformation, does not seem to produce psychedelic effects.[8][11] 5-HTP has not been found to produce psychedelic effects in humans, which has been attributed to the high doses required to produce such effects.[8][10] The 5-HTP doses that produce the HTR in rodents are orders of magnitude higher than the doses of 5-HTP that have been used safely and therapeutically in humans.[10][12] It remains unknown whether 5-HTP can produce psychedelic effects in humans.[15][12] The highest dosage of 5-HTP that is known to have been evaluated in humans is about 3,000 mg per day.[12][1] Serotonin syndrome and associated hallucinations have been reported with overdose of serotonin-elevating drugs, but psychedelic-like effects have not been reported.[12] The lack of the HTR and psychedelic effects with serotonin itself has been attributed to the fact that these effects appear to be dependent on activation of a population of intracellular 5-HT2A receptors expressed in cortical neurons in the medial prefrontal cortex (mPFC) that lack the serotonin transporter (SERT) and are inaccessible to serotonin.[16][17] Serotonin itself is too hydrophilic to enter serotonergic neurons without the SERT, whereas serotonergic psychedelics and serotonin's N-methylated metabolites and analogues are lipophilic and readily enter these neurons.[16][17] These findings may also explain why selective serotonin reuptake inhibitors (SSRIs) and related serotonergic agents do not produce psychedelic effects.[16] The properties of 5-HTP in animal drug discrimination tests have been studied.[18][19][20][21][22][23] 5-HTP generalizes with the serotonin releasing agent fenfluramine and its cue is markedly potentiated by the selective serotonin reuptake inhibitor (SSRI) fluoxetine.[18][19] However, numerous serotonin receptor antagonists, including methysergide, cyproheptadine, metergoline, methiothepin (metitepine), ketanserin, pirenperone, pizotifen, and mianserin, all failed to block the discriminative stimulus properties of 5-HTP.[18][19][20][21] Conflictingly however, in a subsequent study, pizotifen was able to fully block the discriminative stimulus properties of 5-HTP.[18][21] The inability of serotonin 5-HT2A receptor antagonists to block the discriminative stimulus properties of 5-HTP is in notable contrast to their ability to block the 5-HTP-induced HTR.[24] 5-HTP only partially substitutes for LSD in drug discrimination tests, whereas LSD and quipazine fully substitute for 5-HTP.[20] The full substitution of LSD and quipazine for 5-HTP can be blocked by the serotonin 5-HT2A receptor antagonist ketanserin.[20] The findings of drug discrimination tests suggest that 5-HTP has a more complex or compound discriminative stimulus compared to other agents like LSD and that its stimulus properties may not be readily explained by either the serotonin 5-HT1 or 5-HT2 receptors alone.[18][20][23] Instead, a combination of actions at these and/or other receptors may be involved in its stimulus effects.[18][20][23] α-Methyl-5-hydroxytryptophan ^ a b c Turner EH, Loftis JM, Blackwell AD (March 2006). "Serotonin a la carte: supplementation with the serotonin precursor 5-hydroxytryptophan". Pharmacology & Therapeutics. 109 (3): 325–338. doi:10.1016/j.pharmthera.2005.06.004. PMID 16023217. S2CID 2563606. 5-HTP is commonly given to rats or mice to test the SSRI potency of putative antidepressants (O'Neil & Moore, 2003). This simple in vivo test measures the potency of a compound in potentiating the serotonin syndrome induced by the administration of 5-HTP (Grahame-Smith, 1971). The behavioral and physiological features of this syndrome include hypolocomotion, head twitch, forepaw treading, tremors, hindlimb abduction, flat body posture or hunched back, cyanosis, and hyperthermia. In rodents, 5-HTP induces a serotonin syndrome at dosages of 100– 200 mg/ kg (Casal et al., 2000; Nisijima et al., 2000, 2001; see Section 4.4.3 for more on serotonin syndrome). ^ Rahman MK, Nagatsu T, Sakurai T, Hori S, Abe M, Matsuda M (October 1982). "Effect of pyridoxal phosphate deficiency on aromatic L-amino acid decarboxylase activity with L-DOPA and L-5-hydroxytryptophan as substrates in rats". Japanese Journal of Pharmacology. 32 (5): 803–11. doi:10.1254/jjp.32.803. PMID 6983619. ^ Bouchard S, Bousquet C, Roberge AG (September 1981). "Characteristics of dihydroxyphenylalanine/5-hydroxytryptophan decarboxylase activity in brain and liver of cat". Journal of Neurochemistry. 37 (3): 781–7. doi:10.1111/j.1471-4159.1982.tb12555.x. PMID 6974228. S2CID 43853143. ^ Nakatani Y, Sato-Suzuki I, Tsujino N, Nakasato A, Seki Y, Fumoto M, Arita H (May 2008). "Augmented brain 5-HT crosses the blood-brain barrier through the 5-HT transporter in rat". The European Journal of Neuroscience. 27 (9): 2466–72. doi:10.1111/j.1460-9568.2008.06201.x. PMID 18445233. S2CID 18940166. ^ Bouchard S, Roberge AG (July 1979). "Biochemical properties and kinetic parameters of dihydroxyphenylalanine--5-hydroxytryptophan decarboxylase in brain, liver, and adrenals of cat". Canadian Journal of Biochemistry. 57 (7): 1014–8. doi:10.1139/o79-126. PMID 39668. ^ Amamoto T, Sarai K (September 1976). "On the tryptophan-serotonin metabolism in manic-depressive disorders. Changes in plasma 5-HT and 5-HIAA levels and urinary 5-HIAA excretion following oral loading of L-5HTP in patients with depression". Hiroshima Journal of Medical Sciences. 25 (2–3): 135–40. PMID 1088369. ^ "5-Hydroxytryptophan". University of Maryland Medical Center. Archived from the original on 6 January 2010. Retrieved 21 January 2010. ^ a b c d e f g Kozlenkov, Alexey; González-Maeso, Javier (2013). "Animal Models and Hallucinogenic Drugs". The Neuroscience of Hallucinations. New York, NY: Springer New York. pp. 253–277. doi:10.1007/978-1-4614-4121-2_14. ISBN 978-1-4614-4120-5. ^ a b c Schmid, Cullen L.; Bohn, Laura M. (2018). "βArrestins: Ligand-Directed Regulators of 5-HT2A Receptor Trafficking and Signaling Events". 5-HT2A Receptors in the Central Nervous System. Cham: Springer International Publishing. pp. 31–55. doi:10.1007/978-3-319-70474-6_2. ISBN 978-3-319-70472-2. ^ a b c d Jaster AM, de la Fuente Revenga M, González-Maeso J (July 2022). "Molecular targets of psychedelic-induced plasticity". J Neurochem. 162 (1): 80–88. doi:10.1111/jnc.15536. PMC 9068831. PMID 34741320. ^ a b c d e Schmid CL, Bohn LM (October 2010). "Serotonin, but not N-methyltryptamines, activates the serotonin 2A receptor via a β-arrestin2/Src/Akt signaling complex in vivo". J Neurosci. 30 (40): 13513–24. doi:10.1523/JNEUROSCI.1665-10.2010. PMC 3001293. PMID 20926677. ^ a b c d e f g h Shahar O, Botvinnik A, Esh-Zuntz N, Brownstien M, Wolf R, Lotan A, Wolf G, Lerer B, Lifschytz T (November 2022). "Role of 5-HT2A, 5-HT2C, 5-HT1A and TAAR1 Receptors in the Head Twitch Response Induced by 5-Hydroxytryptophan and Psilocybin: Translational Implications". Int J Mol Sci. 23 (22): 14148. doi:10.3390/ijms232214148. PMC 9698447. PMID 36430623. HTR was first described in mice after administration of the serotonin precursor, 5-hydroxytryptophan (5-HTP) [22], and has been further characterized by subsequent investigators [23–29]. Although extensive research has documented the effect of 5-HTP to induce HTR in rodents [30–33], psychedelic effects have not been reported at doses administered to humans [34]. [...] 5-HTP-induced HTR has previously described by multiple authors [30–33,48]. However, 5-HTP has not been reported to have psychedelic effects in humans [49]. Although, overdoses of compounds that increase serotonin release can result in serotonin syndrome, which may include hallucinations [50,51], classic psychedelic effects resembling those induced by tryptaminergic and other psychedelic drugs have not been reported. In our study, administration of 5-HTP at 150–250 mg/kg induced significant HTR. The implications of administering equivalent high doses of 5-HTP to humans are unknown. There are two instances of administering up to 3000 mg 5-HTP per os per day but not as a single dose. Such prolonged exposure that can result in tolerance effects [49]. ^ Canal CE, Morgan D (2012). "Head-twitch response in rodents induced by the hallucinogen 2,5-dimethoxy-4-iodoamphetamine: a comprehensive history, a re-evaluation of mechanisms, and its utility as a model". Drug Test Anal. 4 (7–8): 556–576. doi:10.1002/dta.1333. PMC 3722587. PMID 22517680. ^ Halberstadt AL, Geyer MA (2018). "Effect of Hallucinogens on Unconditioned Behavior". Behavioral Neurobiology of Psychedelic Drugs. Current Topics in Behavioral Neurosciences. Vol. 36. pp. 159–199. doi:10.1007/7854_2016_466. ISBN 978-3-662-55878-2. PMC 5787039. PMID 28224459. ^ Hanks JB, González-Maeso J (January 2013). "Animal models of serotonergic psychedelics". ACS Chem Neurosci. 4 (1): 33–42. doi:10.1021/cn300138m. PMC 3547517. PMID 23336043. Following these initial studies, it was shown that a large dose of the serotonin precursor 5-hydroxytryptophan (5-HTP) induces head-twitch behavior in mice.32 However, to our knowledge, equivalent doses of 5-HTP have not been tested in healthy volunteers, and therefore, it remains unknown whether 5-HTP is psychedelic in humans. Subsequently, numerous psychedelic compounds were shown to induce head-twitch behavior.27,33−36 ^ a b c Sapienza, Jacopo (13 October 2023). "The Key Role of Intracellular 5-HT2A Receptors: A Turning Point in Psychedelic Research?". Psychoactives. 2 (4): 287–293. doi:10.3390/psychoactives2040018. ISSN 2813-1851. ^ a b Vargas MV, Dunlap LE, Dong C, Carter SJ, Tombari RJ, Jami SA, Cameron LP, Patel SD, Hennessey JJ, Saeger HN, McCorvy JD, Gray JA, Tian L, Olson DE (February 2023). "Psychedelics promote neuroplasticity through the activation of intracellular 5-HT2A receptors". Science. 379 (6633): 700–706. Bibcode:2023Sci...379..700V. doi:10.1126/science.adf0435. PMC 10108900. PMID 36795823. ^ a b c d e f Glennon RA (1988). "Site-selective serotonin agonists as discriminative stimuli". Psychopharmacol Ser. 4: 15–31. doi:10.1007/978-3-642-73223-2_2. PMID 3293039. ^ a b c Barrett RJ, Blackshear MA, Sanders-Bush E (1982). "Discriminative stimulus properties of L-5-hydroxytryptophan: behavioral evidence for multiple serotonin receptors". Psychopharmacology (Berl). 76 (1): 29–35. doi:10.1007/BF00430750. PMID 6805004. ^ a b c d e f Cunningham KA, Callahan PM, Appel JB (January 1985). "Differentiation between the stimulus effects of l-5-hydroxytryptophan and LSD". Eur J Pharmacol. 108 (2): 179–186. doi:10.1016/0014-2999(85)90723-x. PMID 3156756. ^ a b c Friedman R, Barrett RJ, Sanders-Bush E (1983). "Additional evidence that L-5-hydroxytryptophan discrimination models a unique serotonin receptor". Psychopharmacology (Berl). 80 (3): 209–213. doi:10.1007/BF00436154. PMID 6137018. ^ Witkin JM, Brady LS, Barrett JE (1988). "Antagonism by ketanserin of the behavioral effects of quipazine but not l-5-hydroxytryptophan in squirrel monkeys". Psychopharmacology (Berl). 94 (3): 302–305. doi:10.1007/BF00174679. PMID 3128804. ^ a b c Winter JC, Rabin RA (July 1988). "A comparison of the discriminative stimulus properties of l-5-hydroxytryptophan in the presence of either citalopram or Ro 4-4602". Pharmacol Biochem Behav. 30 (3): 613–616. doi:10.1016/0091-3057(88)90073-1. PMID 3264918. ^ Glennon, Richard A. (23 October 1992). "Animal Models for Assessing Hallucinogenic Agents". Animal Models of Drug Addiction. Vol. 24. New Jersey: Humana Press. p. 345–382. doi:10.1385/0-89603-217-5:345. ISBN 978-0-89603-217-0. Retrieved 20 May 2025. Retrieved from " 3Not to be confused with 5-hydroxytryptamine. This article is about 5-hydroxytryptophan as a biological compound. For its role as a medication and supplement, see Oxitriptan. 5-Hydroxytryptophan Names IUPAC name 2-amino-3-(5-hydroxy-1H-indol-3-yl)propanoic acid Other names 5-HTP; Oxitriptan; α-Carboxy-5-hydroxytryptamine; α-Carboxy-5-HT Identifiers CAS Number 56-69-9 N 3D model (JSmol) Interactive image ChEBI CHEBI:17780 Y ChEMBL ChEMBL350221 Y ChemSpider 388413 Y ECHA InfoCard 100.022.193 IUPHAR/BPS 4671 KEGG D07339 Y MeSH 5-Hydroxytryptophan PubChem CID 144 UNII C1LJO185Q9 Y CompTox Dashboard (EPA) DTXSID1025437 InChI InChI=1S/C11H12N2O3/c12-9(11(15)16)3-6-5-13-10-2-1-7(14)4-8(6)10/h1-2,4-5,9,13-14H,3,12H2,(H,15,16)/t9-/m0/s1 YKey: LDCYZAJDBXYCGN-VIFPVBQESA-N YInChI=1/C11H12N2O3/c12-9(11(15)16)3-6-5-13-10-2-1-7(14)4-8(6)10/h1-2,4-5,9,13-14H,3,12H2,(H,15,16)/t9-/m0/s1Key: LDCYZAJDBXYCGN-VIFPVBQEBZ SMILES O=C(O)[C@@H](N)Cc2c1cc(O)ccc1[nH]c2 Properties Chemical formula C11H12N2O3 Molar mass 220.228 g·mol−1 Density 1.484 g/mL Melting point 298 to 300 °C (568 to 572 °F; 571 to 573 K) Boiling point 520.6 °C (969.1 °F; 793.8 K) Except where otherwise noted, data are given for materials in their standard state (at 25 °C [77 °F], 100 kPa). N verify (what is YN ?) Infobox references Chemical compound 5-Hydroxytryptophan (5-HTP), used medically as oxitriptan, is a naturally occurring amino acid and chemical precursor as well as a metabolic intermediate in the biosynthesis of the neurotransmitter serotonin. 5-HTP can be manufactured and used as a drug and supplement with the INNTooltip International Nonproprietary Name oxitriptan. Brand names include Cincofarm, Levothym, Levotonine, Oxyfan, Telesol, Tript-OH, and Triptum. As a drug, it is used in the treatment of depression and for certain other indications. 5-HTP is produced from the amino acid tryptophan through the action of the enzyme tryptophan hydroxylase. Tryptophan hydroxylase is one of the biopterin-dependent aromatic amino acid hydroxylases. Production of 5-HTP is the rate-limiting step in 5-HT (serotonin) synthesis. 5-HTP is normally rapidly converted to 5-HT by amino acid decarboxylase.[1] 5-HTP is decarboxylated to serotonin (5-hydroxytryptamine or 5-HT) by the enzyme aromatic-L-amino-acid decarboxylase with the help of vitamin B6.[2] This reaction occurs both in nervous tissue and in the liver.[3] 5-HTP crosses the blood–brain barrier,[4] while 5-HT does not. Excess 5-HTP, especially when administered with vitamin B6, is thought to be metabolized and excreted.[5][6] Metabolic pathway from tryptophan to serotonin. 5-HTP AAAD Serotonin PLP See also: Tryptophan § Dietary sources Though 5-HTP is found in food only in insignificant quantities, it is a chemical involved intermediately in the metabolism of tryptophan, an amino acid found in all unfractionated foods, with lower total amino acid content correlating with increased tryptophan absorption.[7] Main article: Oxitriptan 5-HTP is used medically and as a supplement under the name oxitriptan in the treatment of depression and for certain other indications. It can be potentiated in combination with a peripherally selective aromatic L-amino acid decarboxylase (AAAD) inhibitor such as carbidopa or benserazide. These agents increase the strength and duration of oxitriptan. An investigational combination formulation is oxitriptan/carbidopa. See also: Tryptophan § Psychedelic effects 5-HTP robustly produces the head-twitch response (HTR) in rodents when administered at relatively high doses.[8][9][10][11][12] It dose-dependently induces the HTR in mice across a dose range of 50 to 250 mg/kg via intraperitoneal administration, with an inverted U-shaped dose–response curve and maximal induction of the HTR at a dose of 200 mg/kg.[12][1] Similarly to the case of 5-HTP, intracerebroventricular injection of serotonin, but not peripheral administration of serotonin, produces the HTR.[9][8][11] The HTR is induced by serotonergic psychedelics like lysergic acid diethylamide (LSD) and psilocybin and is a behavioral proxy of psychedelic effects.[13][8] The HTR of 5-HTP is blocked by serotonin 5-HT2A receptor antagonists, which block the hallucinogenic effects of serotonergic psychedelics in humans, is prevented by aromatic L-amino acid decarboxylase (AAAD) inhibitors, which block conversion of 5-HTP into serotonin, and is potentiated by monoamine oxidase A (MAO-A) inhibitors, which prevent the degradation of serotonin and other endogenous tryptamines.[9][8][10][11][12] It is also suppressed by the serotonin 5-HT1A receptor full agonist 8-OH-DPAT, is greatly augmented by the serotonin 5-HT2C receptor antagonist RS-102221, and is reduced by the trace amine-associated receptor 1 (TAAR1) antagonist EPPTB.[12] In addition, the HTR of 5-HTP is abolished by indolethylamine N-methyltransferase (INMT) inhibitors, which block conversion of serotonin and other endogenous tryptamines into N-methylated tryptamines, such as N-methylserotonin (NMS; norbufotenin), bufotenin (5-hydroxy-N,N-dimethyltryptamine; 5-HO-DMT), and N,N-dimethyltryptamine (DMT).[8][14][11] These N-methylated tryptamines are well-known for their psychedelic effects, whereas serotonin itself, without biotransformation, does not seem to produce psychedelic effects.[8][11] 5-HTP has not been found to produce psychedelic effects in humans, which has been attributed to the high doses required to produce such effects.[8][10] The 5-HTP doses that produce the HTR in rodents are orders of magnitude higher than the doses of 5-HTP that have been used safely and therapeutically in humans.[10][12] It remains unknown whether 5-HTP can produce psychedelic effects in humans.[15][12] The highest dosage of 5-HTP that is known to have been evaluated in humans is about 3,000 mg per day.[12][1] Serotonin syndrome and associated hallucinations have been reported with overdose of serotonin-elevating drugs, but psychedelic-like effects have not been reported.[12] The lack of the HTR and psychedelic effects with serotonin itself has been attributed to the fact that these effects appear to be dependent on activation of a population of intracellular 5-HT2A receptors expressed in cortical neurons in the medial prefrontal cortex (mPFC) that lack the serotonin transporter (SERT) and are inaccessible to serotonin.[16][17] Serotonin itself is too hydrophilic to enter serotonergic neurons without the SERT, whereas serotonergic psychedelics and serotonin's N-methylated metabolites and analogues are lipophilic and readily enter these neurons.[16][17] These findings may also explain why selective serotonin reuptake inhibitors (SSRIs) and related serotonergic agents do not produce psychedelic effects.[16] The properties of 5-HTP in animal drug discrimination tests have been studied.[18][19][20][21][22][23] 5-HTP generalizes with the serotonin releasing agent fenfluramine and its cue is markedly potentiated by the selective serotonin reuptake inhibitor (SSRI) fluoxetine.[18][19] However, numerous serotonin receptor antagonists, including methysergide, cyproheptadine, metergoline, methiothepin (metitepine), ketanserin, pirenperone, pizotifen, and mianserin, all failed to block the discriminative stimulus properties of 5-HTP.[18][19][20][21] Conflictingly however, in a subsequent study, pizotifen was able to fully block the discriminative stimulus properties of 5-HTP.[18][21] The inability of serotonin 5-HT2A receptor antagonists to block the discriminative stimulus properties of 5-HTP is in notable contrast to their ability to block the 5-HTP-induced HTR.[24] 5-HTP only partially substitutes for LSD in drug discrimination tests, whereas LSD and quipazine fully substitute for 5-HTP.[20] The full substitution of LSD and quipazine for 5-HTP can be blocked by the serotonin 5-HT2A receptor antagonist ketanserin.[20] The findings of drug discrimination tests suggest that 5-HTP has a more complex or compound discriminative stimulus compared to other agents like LSD and that its stimulus properties may not be readily explained by either the serotonin 5-HT1 or 5-HT2 receptors alone.[18][20][23] Instead, a combination of actions at these and/or other receptors may be involved in its stimulus effects.[18][20][23] α-Methyl-5-hydroxytryptophan ^ a b c Turner EH, Loftis JM, Blackwell AD (March 2006). "Serotonin a la carte: supplementation with the serotonin precursor 5-hydroxytryptophan". Pharmacology & Therapeutics. 109 (3): 325–338. doi:10.1016/j.pharmthera.2005.06.004. PMID 16023217. S2CID 2563606. 5-HTP is commonly given to rats or mice to test the SSRI potency of putative antidepressants (O'Neil & Moore, 2003). This simple in vivo test measures the potency of a compound in potentiating the serotonin syndrome induced by the administration of 5-HTP (Grahame-Smith, 1971). The behavioral and physiological features of this syndrome include hypolocomotion, head twitch, forepaw treading, tremors, hindlimb abduction, flat body posture or hunched back, cyanosis, and hyperthermia. In rodents, 5-HTP induces a serotonin syndrome at dosages of 100– 200 mg/ kg (Casal et al., 2000; Nisijima et al., 2000, 2001; see Section 4.4.3 for more on serotonin syndrome). ^ Rahman MK, Nagatsu T, Sakurai T, Hori S, Abe M, Matsuda M (October 1982). "Effect of pyridoxal phosphate deficiency on aromatic L-amino acid decarboxylase activity with L-DOPA and L-5-hydroxytryptophan as substrates in rats". Japanese Journal of Pharmacology. 32 (5): 803–11. doi:10.1254/jjp.32.803. PMID 6983619. ^ Bouchard S, Bousquet C, Roberge AG (September 1981). "Characteristics of dihydroxyphenylalanine/5-hydroxytryptophan decarboxylase activity in brain and liver of cat". Journal of Neurochemistry. 37 (3): 781–7. doi:10.1111/j.1471-4159.1982.tb12555.x. PMID 6974228. S2CID 43853143. ^ Nakatani Y, Sato-Suzuki I, Tsujino N, Nakasato A, Seki Y, Fumoto M, Arita H (May 2008). "Augmented brain 5-HT crosses the blood-brain barrier through the 5-HT transporter in rat". The European Journal of Neuroscience. 27 (9): 2466–72. doi:10.1111/j.1460-9568.2008.06201.x. PMID 18445233. S2CID 18940166. ^ Bouchard S, Roberge AG (July 1979). "Biochemical properties and kinetic parameters of dihydroxyphenylalanine--5-hydroxytryptophan decarboxylase in brain, liver, and adrenals of cat". Canadian Journal of Biochemistry. 57 (7): 1014–8. doi:10.1139/o79-126. PMID 39668. ^ Amamoto T, Sarai K (September 1976). "On the tryptophan-serotonin metabolism in manic-depressive disorders. Changes in plasma 5-HT and 5-HIAA levels and urinary 5-HIAA excretion following oral loading of L-5HTP in patients with depression". Hiroshima Journal of Medical Sciences. 25 (2–3): 135–40. PMID 1088369. ^ "5-Hydroxytryptophan". University of Maryland Medical Center. Archived from the original on 6 January 2010. Retrieved 21 January 2010. ^ a b c d e f g Kozlenkov, Alexey; González-Maeso, Javier (2013). "Animal Models and Hallucinogenic Drugs". The Neuroscience of Hallucinations. New York, NY: Springer New York. pp. 253–277. doi:10.1007/978-1-4614-4121-2_14. ISBN 978-1-4614-4120-5. ^ a b c Schmid, Cullen L.; Bohn, Laura M. (2018). "βArrestins: Ligand-Directed Regulators of 5-HT2A Receptor Trafficking and Signaling Events". 5-HT2A Receptors in the Central Nervous System. Cham: Springer International Publishing. pp. 31–55. doi:10.1007/978-3-319-70474-6_2. ISBN 978-3-319-70472-2. ^ a b c d Jaster AM, de la Fuente Revenga M, González-Maeso J (July 2022). "Molecular targets of psychedelic-induced plasticity". J Neurochem. 162 (1): 80–88. doi:10.1111/jnc.15536. PMC 9068831. PMID 34741320. ^ a b c d e Schmid CL, Bohn LM (October 2010). "Serotonin, but not N-methyltryptamines, activates the serotonin 2A receptor via a β-arrestin2/Src/Akt signaling complex in vivo". J Neurosci. 30 (40): 13513–24. doi:10.1523/JNEUROSCI.1665-10.2010. PMC 3001293. PMID 20926677. ^ a b c d e f g h Shahar O, Botvinnik A, Esh-Zuntz N, Brownstien M, Wolf R, Lotan A, Wolf G, Lerer B, Lifschytz T (November 2022). "Role of 5-HT2A, 5-HT2C, 5-HT1A and TAAR1 Receptors in the Head Twitch Response Induced by 5-Hydroxytryptophan and Psilocybin: Translational Implications". Int J Mol Sci. 23 (22): 14148. doi:10.3390/ijms232214148. PMC 9698447. PMID 36430623. HTR was first described in mice after administration of the serotonin precursor, 5-hydroxytryptophan (5-HTP) [22], and has been further characterized by subsequent investigators [23–29]. Although extensive research has documented the effect of 5-HTP to induce HTR in rodents [30–33], psychedelic effects have not been reported at doses administered to humans [34]. [...] 5-HTP-induced HTR has previously described by multiple authors [30–33,48]. However, 5-HTP has not been reported to have psychedelic effects in humans [49]. Although, overdoses of compounds that increase serotonin release can result in serotonin syndrome, which may include hallucinations [50,51], classic psychedelic effects resembling those induced by tryptaminergic and other psychedelic drugs have not been reported. In our study, administration of 5-HTP at 150–250 mg/kg induced significant HTR. The implications of administering equivalent high doses of 5-HTP to humans are unknown. There are two instances of administering up to 3000 mg 5-HTP per os per day but not as a single dose. Such prolonged exposure that can result in tolerance effects [49]. ^ Canal CE, Morgan D (2012). "Head-twitch response in rodents induced by the hallucinogen 2,5-dimethoxy-4-iodoamphetamine: a comprehensive history, a re-evaluation of mechanisms, and its utility as a model". Drug Test Anal. 4 (7–8): 556–576. doi:10.1002/dta.1333. PMC 3722587. PMID 22517680. ^ Halberstadt AL, Geyer MA (2018). "Effect of Hallucinogens on Unconditioned Behavior". Behavioral Neurobiology of Psychedelic Drugs. Current Topics in Behavioral Neurosciences. Vol. 36. pp. 159–199. doi:10.1007/7854_2016_466. ISBN 978-3-662-55878-2. PMC 5787039. PMID 28224459. ^ Hanks JB, González-Maeso J (January 2013). "Animal models of serotonergic psychedelics". ACS Chem Neurosci. 4 (1): 33–42. doi:10.1021/cn300138m. PMC 3547517. PMID 23336043. Following these initial studies, it was shown that a large dose of the serotonin precursor 5-hydroxytryptophan (5-HTP) induces head-twitch behavior in mice.32 However, to our knowledge, equivalent doses of 5-HTP have not been tested in healthy volunteers, and therefore, it remains unknown whether 5-HTP is psychedelic in humans. Subsequently, numerous psychedelic compounds were shown to induce head-twitch behavior.27,33−36 ^ a b c Sapienza, Jacopo (13 October 2023). "The Key Role of Intracellular 5-HT2A Receptors: A Turning Point in Psychedelic Research?". Psychoactives. 2 (4): 287–293. doi:10.3390/psychoactives2040018. ISSN 2813-1851. ^ a b Vargas MV, Dunlap LE, Dong C, Carter SJ, Tombari RJ, Jami SA, Cameron LP, Patel SD, Hennessey JJ, Saeger HN, McCorvy JD, Gray JA, Tian L, Olson DE (February 2023). "Psychedelics promote neuroplasticity through the activation of intracellular 5-HT2A receptors". Science. 379 (6633): 700–706. Bibcode:2023Sci...379..700V. doi:10.1126/science.adf0435. PMC 10108900. PMID 36795823. ^ a b c d e f Glennon RA (1988). "Site-selective serotonin agonists as discriminative stimuli". Psychopharmacol Ser. 4: 15–31. doi:10.1007/978-3-642-73223-2_2. PMID 3293039. ^ a b c Barrett RJ, Blackshear MA, Sanders-Bush E (1982). "Discriminative stimulus properties of L-5-hydroxytryptophan: behavioral evidence for multiple serotonin receptors". Psychopharmacology (Berl). 76 (1): 29–35. doi:10.1007/BF00430750. PMID 6805004. ^ a b c d e f Cunningham KA, Callahan PM, Appel JB (January 1985). "Differentiation between the stimulus effects of l-5-hydroxytryptophan and LSD". Eur J Pharmacol. 108 (2): 179–186. doi:10.1016/0014-2999(85)90723-x. PMID 3156756. ^ a b c Friedman R, Barrett RJ, Sanders-Bush E (1983). "Additional evidence that L-5-hydroxytryptophan discrimination models a unique serotonin receptor". Psychopharmacology (Berl). 80 (3): 209–213. doi:10.1007/BF00436154. PMID 6137018. ^ Witkin JM, Brady LS, Barrett JE (1988). "Antagonism by ketanserin of the behavioral effects of quipazine but not l-5-hydroxytryptophan in squirrel monkeys". Psychopharmacology (Berl). 94 (3): 302–305. doi:10.1007/BF00174679. PMID 3128804. ^ a b c Winter JC, Rabin RA (July 1988). "A comparison of the discriminative stimulus properties of l-5-hydroxytryptophan in the presence of either citalopram or Ro 4-4602". Pharmacol Biochem Behav. 30 (3): 613–616. doi:10.1016/0091-3057(88)90073-1. PMID 3264918. ^ Glennon, Richard A. (23 October 1992). "Animal Models for Assessing Hallucinogenic Agents". Animal Models of Drug Addiction. Vol. 24. New Jersey: Humana Press. p. 345–382. doi:10.1385/0-89603-217-5:345. ISBN 978-0-89603-217-0. Retrieved 20 May 2025. Retrieved from " 4 The following pages link to 5-Hydroxytryptophan External tools (link count transclusion count sorted list) · See help page for transcluding these entries Showing 50 items. View (previous 50 | next 50) (20 | 50 | 100 | 250 | 500)Alkaloid (links | edit) Dimethyltryptamine (links | edit) Déjà vu (links | edit) Serotonin (links | edit) Tyrosine (links | edit) Psilocybin (links | edit) Putrescine (links | edit) Tryptophan (links | edit) Eosinophilia–myalgia syndrome (links | edit) A-Methyltryptamine (links | edit) Catecholamine (links | edit) Benzphetamine (links | edit) Melatonin (links | edit) Dipropyltryptamine (links | edit) Bupropion (links | edit) Orlistat (links | edit) Tryptamine (links | edit) 5-MeO-DMT (links | edit) TiHKAL (links | edit) Fenfluramine/phentermine (links | edit) Sumatriptan (links | edit) B-Carboline (links | edit) Cathine (links | edit) Anorectic (links | edit) Phenylpropanolamine (links | edit) Frovatriptan (links | edit) Dopamine reuptake inhibitor (links | edit) Norepinephrine reuptake inhibitor (links | edit) Transdermal patch (links | edit) Yohimbine (links | edit) 5-MeO-DiPT (links | edit) ATC code A (links | edit) ATC code A08 (links | edit) 5-MeO-AMT (links | edit) A-Ethyltryptamine (links | edit) N-Ethyltryptamine (links | edit) Triptan (links | edit) 4-HO-DiPT (links | edit) Psilocin (links | edit) L-DOPA (links | edit) Aromatic L-amino acid decarboxylase (links | edit) Pyridoxal phosphate (links | edit) Harmala alkaloid (links | edit) Rizatriptan (links | edit) 2,4-Dinitrophenol (links | edit) ZMA (supplement) (links | edit) Fenfluramine (links | edit) Methylethyltryptamine (links | edit) Anti-obesity medication (links | edit) DiPT (links | edit) View (previous 50 | next 50) (20 | 50 | 100 | 250 | 500) Retrieved from " WhatLinksHere/5-Hydroxytryptophan" O triptofano é um aminoácido essencial para a formação e manutenção dos músculos e para a produção de serotonina e melatonina, compostos que atuam no sistema nervoso, ajudando a regular o humor, o sono, a memória e o apetite, sendo usado no tratamento e na prevenção de depressão, ansiedade, insônia e no processo de emagrecimento. Encontre um Nutricionista perto de você! Parceria com Buscar Médico Por ser um aminoácido essencial, ou seja, que o organismo não consegue produzir, o triptofano deve ser obtido a partir da ingestão de alimentos como chocolate amargo, nozes, ovos e amêndoas, por exemplo. Veja outros alimentos ricos em triptofano. Além disso, o triptofano também pode ser obtido através da suplementação, como 5HTP, ou L-triptofano, em cápsulas. No entanto, esses suplementos devem ser consumidos apenas sob a orientação de um nutricionista, ou médico. Para que serve O triptofano é um aminoácido que participa de diversas funções no organismo, sendo indicado para: Combater a depressão; Formar e manter os músculos; Controlar a ansiedade; Promover o bem estar; Melhorar a memória; Diminuir o estresse; Regular o sono; Ajudar no emagrecimento. Além disso, o triptofano também é utilizado para ajudar no tratamento da dor, do déficit de atenção, da hiperatividade, da fadiga crônica e da TPM. O neurotransmissor serotonina ajuda na formação do hormônio melatonina que regula o ritmo do relógio biológico do organismo, melhorando a qualidade do sono, já que a melatonina é produzida durante a noite. Lista de alimentos ricos em triptofano Alguns alimentos que são ricos em triptofano são: Queijos; Ovos; Tofu; Salmão; Nozes; Amêndoas; Amendoim; Castanha-do-Pará; Banana. Além disso, o triptofano também pode ser encontrado na forma de suplementos, como 5-HTP, ou L-triptofano, em cápsulas, sendo indicada a ingestão de 100mg a 3 g por dia, de acordo com a situação a ser tratada. Entenda como usar o suplemento de triptofano. Triptofano ajuda a emagrecer? O triptofano participa da produção de serotonina, um neurotransmissor que promove o controle da ansiedade e depressão, diminuindo a compulsão alimentar e ajudando, assim, a emagrecer. Veja como usar o triptofano para ajudar no emagrecimento. A alimentação muitas vezes está relacionada com as emoções e, por isso, momentos de ansiedade e depressão podem estimular a ingestão de alimentos que promovem o prazer, como os alimentos ricos em carboidratos e gorduras, incluindo chocolates, sorvetes, bolos e frituras, por exemplo, aumentando as calorias da dieta e favorecendo, assim o ganho de peso. Ingestão diária recomendada A ingestão diária de triptofano, varia de acordo com a idade, o sexo e a fase da vida, como indicado na tabela a seguir: Além disso, mulheres grávidas precisam ingerir 7 mg / Kg de peso corporal, por dia, de triptofano. Já as mulheres que amamentam, devem consumir 9 mg / Kg de peso corporal, de triptofano por dia.