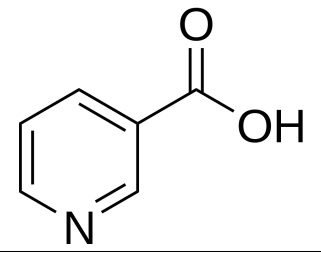
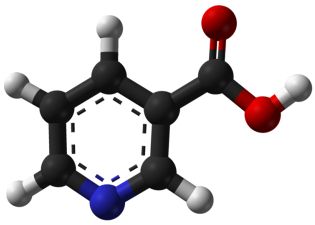
Niacin

Niacin	
	
	
Identifiers	
CAS number	59-67-6 [1] ✓
PubChem	938 [2] ✓
ChemSpider	913 [3] ✓
MeSH	Niacin [4]
IUPHAR ligand	1588 [5] ✓
SMILES	
Properties	
Molecular formula	$C_6H_5NO_2$
Molar mass	123.11 g/mol
Melting point	236.6 °C, 510 K, 458 °F
Boiling point	decomposes
✓ (what is this?) (verify) [6] Except where noted otherwise, data are given for materials in their standard state (at 25 °C, 100 kPa)	
Infobox references	

Niacin (also known as **vitamin B_3**, **nicotinic acid** and **vitamin PP**) is an organic compound with the formula $C_5H_4NCO_2H$ and, depending on the definition used, one of the between forty to eighty essential human nutrients. This colourless, water-soluble solid is a derivative of pyridine, with a carboxyl group (COOH) at the 3-position. Other forms of vitamin B_3 include the corresponding amide, nicotinamide ("niacinamide"), where the carboxyl group has been replaced by a carboxamide group ($CONH_2$), as well as more complex amides and a variety of esters. The terms niacin, nicotinamide, and vitamin B_3 are often used interchangeably to refer to any member of this family of compounds, since they have the same biochemical activity.

Niacin is converted to nicotinamide and then to NAD and NADP *in vivo*. Although the two are identical in their vitamin activity, nicotinamide does not have the same pharmacological effects as niacin, which occur as side effects

of niacin's conversion. Nicotinamide does not reduce cholesterol or cause flushing.[7] Nicotinamide may be toxic to the liver at doses exceeding 3 g/day for adults.[8] Niacin is a precursor to NAD+/NADH and NADP+/NADPH, which play essential metabolic roles in living cells.[9] Niacin is involved in both DNA repair, and the production of steroid hormones in the adrenal gland.

Niacin is one of five vitamins associated with a pandemic deficiency disease:

- niacin deficiency (pellagra)
- vitamin C deficiency (scurvy)
- thiamin deficiency (beriberi)
- vitamin D deficiency (rickets)
- vitamin A deficiency.

In larger doses, niacin can reverse atherosclerosis by lowering low-density lipoprotein (LDL) and favorably affecting other compounds.[10]

History

Niacin was first described by Hugo Weidel in 1873 in his studies of nicotine.[11] The original preparation remains useful: The oxidation of nicotine using nitric acid.[12] Niacin was extracted from livers by Conrad Elvehjem who later identified the active ingredient, then referred to as the "pellagra-preventing factor" and the "anti-blacktongue factor."[13] When the biological significance of nicotinic acid was realized, it was thought appropriate to choose a name to dissociate it from nicotine, to avoid the perception that vitamins or niacin-rich food contains nicotine, or that cigarettes contain vitamins. The resulting name 'niacin' was derived from **ni**cotinic **ac**id + vitam**in.**

Carpenter found in 1951 that niacin in corn is biologically unavailable, and can be released only in very alkaline lime water of pH 11.[14] This process is known as nixtamalization.[15]

Niacin is referred to as vitamin B_3 because it was the third of the B vitamins to be discovered. It has historically been referred to as "vitamin PP" or "vitamin P-P".

Dietary needs

The recommended daily allowance of niacin is 2–12 mg/day for children, 14 mg/day for women, 16 mg/day for men, and 18 mg/day for pregnant or breast-feeding women.[16] The upper limit for adult men and women is 35 mg/day, which is based on flushing as the critical adverse effect.

In general, niacin status is tested through urinary biomarkers,[17] which are believed to be more reliable than plasma levels.[18]

Deficiency

A man with pellagra, which is caused by a chronic lack of vitamin B_3 in the diet

At the present time, niacin deficiency is rarely seen in developed countries but it is usually apparent in conditions of poverty, malnutrition, and chronic alcoholism[19] . It also tends to occur in areas where people eat maize (corn, the only grain low in niacin) as a staple food. A special cooking technique called nixtamalization is needed to increase the bioavailability of niacin during maize meal/flour production.

Mild niacin deficiency has been shown to slow metabolism, causing decreased tolerance to the cold.

Severe deficiency of niacin in the diet causes the disease pellagra which is characterized by diarrhea, dermatitis, and dementia as well as "necklace" lesions on the lower neck, hyperpigmentation, thickening of the skin, inflammation of the mouth and tongue, digestive disturbances, amnesia, delirium, and eventually death, if left untreated[20] . Common psychiatric symptoms of niacin deficiency include irritability, poor concentration, anxiety, fatigue, restlessness, apathy, and depression[20] . Studies have indicated that, in patients with alcoholic pellagra, niacin deficiency may be an important factor influencing both the onset and severity of this condition. Alcoholic patients typically experience increased intestinal permeability leading to negative health outcomes .

Hartnup's disease is a hereditary nutritional disorder resulting in niacin deficiency[20] . This condition was first identified in the 1950s by the Hartnup family in London. It is due to a deficit in the intestines and kidneys, making it difficult for the body to break down and absorb dietary tryptophan. The resulting condition is similar to pellagra, including symptoms of red, scaly rash, and sensitivity to sunlight. Oral niacin is given as a treatment for this condition in doses ranging from 40–200 mg, with a good prognosis if identified and treated early[20] . Niacin synthesis is also deficient in carcinoid syndrome, because of metabolic diversion of its precursor tryptophan to form serotonin.

Lipid-modifying effects

In pharmacological doses, niacin has been proven to reverse atherosclerosis by reducing total cholesterol, triglyceride, very-low-density lipoprotein (VLDL), and low-density lipoprotein (LDL); and increasing high-density lipoprotein (HDL). It has been proposed that niacin has the ability to lower lipoprotein(a), which is beneficial at reducing thrombotic tendency.[21]

Niacin, prescribed in doses between 1000 and 2000 mg two to three times daily,[22] blocks the breakdown of fats in adipose tissue, more specifically the very-low-density lipoprotein (VLDL), precursor of low-density lipoprotein (LDL) or "bad" cholesterol. Because niacin blocks breakdown of fats, it causes a decrease in free fatty acids in the blood and, as a consequence, decreased secretion of VLDL and cholesterol by the liver.[23]

By lowering VLDL levels, niacin also *increases* the level of high-density lipoprotein (HDL) or "good" cholesterol in blood, and therefore it is sometimes prescribed for patients with low HDL, who are also at high risk of a heart attack.[24] [25]

The ARBITER 6-HALTS study, reported at the 2009 annual meeting of the American Heart Association and in the New England Journal of Medicine[26] concluded that, when added to statins, 2000 mg/day slow-release niacin was more effective than ezetimibe (Zetia) in reducing carotid intima-media thickness, a marker of atherosclerosis.[27]

As of August 2008, a combination of niacin with laropiprant is tested in a clinical trial. Laropiprant reduces facial flushes induced by niacin. [28]

Toxicity

Pharmacological doses of niacin (1.5 - 6 g per day) often lead to side effects that can include dermatological conditions such as skin flushing and itching, dry skin, skin rashes including acanthosis nigricans. Gastrointestinal complaints, such as dyspepsia (indigestion) and liver toxicity [fulminant hepatic failure], have also been reported. Side effects of hyperglycemia, cardiac arrhythmias and "birth defects in experimental animals" have also been reported.[29] The flush lasts for about 15 to 30 minutes, and is sometimes accompanied by a prickly or itching sensation, in particular, in areas covered by clothing. This effect is mediated by prostaglandin and can be blocked by taking 300 mg of aspirin half an hour before taking niacin, or by taking one tablet of ibuprofen per day. Taking the niacin with meals also helps reduce this side effect. After several weeks of a consistent dose, most patients no longer flush.[30] Slow- or "sustained"-release forms of niacin have been developed to lessen these side effects.[23] [31] One study showed the incidence of flushing was significantly lower with a sustained release formulation[32] though doses above 2 g per day have been associated with liver damage, in particular, with slow-release formulations.[29] Flushing is often thought to involve histamine, but histamine has been shown not to be involved in the reaction.[33] Prostaglandin (PGD_2) is the primary cause of the flushing reaction, with serotonin appearing to have a secondary role in this reaction.[33]

High-dose niacin may also elevate blood sugar, thereby worsening diabetes mellitus.[29]

Hyperuricemia is another side effect of taking high-dose niacin, and may exacerbate gout.[34]

Niacin at doses used in lowering cholesterol has been associated with birth defects in laboratory animals, with possible consequences for infant development in pregnant women.[29]

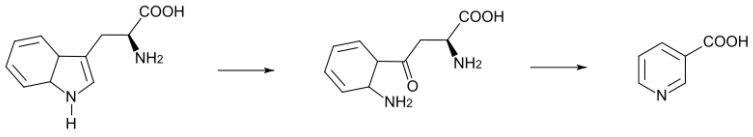
Niacin at extremely high doses can have life-threatening acute toxic reactions.[35] Extremely high doses of niacin can also cause niacin maculopathy, a thickening of the macula and retina, which leads to blurred vision and blindness. This maculopathy is reversible after stopping niacin intake.[36]

Inositol hexanicotinate

One popular form of dietary supplement is inositol hexanicotinate (IHN), which is inositol that has been esterified with niacin on all six of inositol's alcohol groups. IHN is usually sold as "flush-free" or "no-flush" niacin in units of 250, 500, or 1000 mg/tablet or capsule. It is sold as an over-the-counter formulation and often marketed and labeled as niacin, thus misleading consumers into thinking they are getting the active form of the medication. While this form of niacin does not cause the flushing associated with the immediate-release products, the evidence that it has lipid-modifying functions is contradictory, at best. As the clinical trials date from the early 1960s (Dorner, Welsh) or the late 1970s (Ziliotto, Kruse, Agusti), it is difficult to assess them by today's standards.[37] One of the last of those studies affirmed the superiority of inositol and xantinol esters of nicotinic acid for reducing serum free fatty acid, [38] but other studies conducted during the same period found no benefit.[39] A more recent placebo-controlled trial was small (n=11/group), but results after three months at 1500 mg/day showed no trend for improvements in total cholesterol, LDL-C, HDL-C or triglycerides. [40] Thus, so far there is not enough evidence to recommend IHN to treat dyslipidemia. Furthermore, the American Heart Association and the National Cholesterol Education Program both take the position that only prescription niacin should be used to treat dyslipidemias, and only under the management of a physician. The reason given is that niacin at effective intakes of 1500–3000 mg/day can also potentially have severe adverse effects. Monitoring of liver enzymes is necessary.

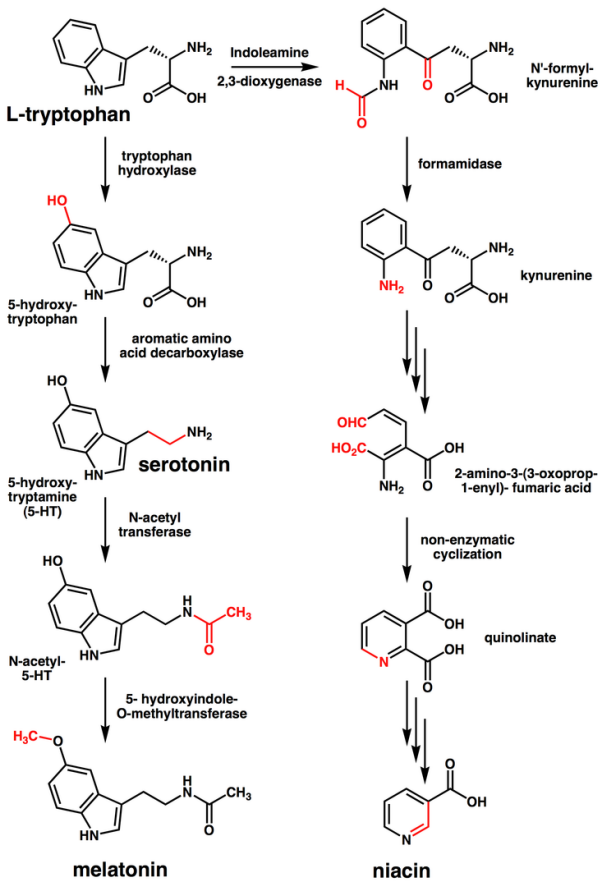
Biosynthesis and chemical synthesis

The liver can synthesize niacin from the essential amino acid tryptophan, requiring 60 mg of tryptophan to make one mg of niacin.[41] The 5-membered aromatic heterocycle of tryptophan is cleaved and rearranged with the alpha amino group of tryptophan into the 6-membered aromatic heterocycle of niacin.



Biosynthesis: Tryptophan → kynurenine → niacin

Several million kilograms of niacin are manufactured each year, starting from 3-methylpyridine.



Biosynthesis

Receptor

The receptor for niacin is a G protein-coupled receptor called HM74A.[42] It couples to Gi alpha subunit.[43]

Food sources

Niacin is found in variety of foods including liver, chicken, beef, fish, cereal, peanuts and legumes and is also synthesized from tryptophan, which is found in meat, dairy and eggs.

Animal products:

- liver, heart and kidney
- chicken
- beef
- fish: tuna, salmon
- milk
- eggs

Fruits and vegetables:

- avocados
- dates
- tomatoes
- leaf vegetables
- broccoli
- carrots
- sweet potatoes
- asparagus

Seeds:

- nuts
- whole grain products
- legumes
- saltbush seeds

Fungi:

- mushrooms
- brewer's yeast

Other:

- Vegemite (from spent brewer's yeast)

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