Vitamin D Deficiency Rickets

Major health organizations list thirteen vitamins: f427b2363d == Vitamin D vitamers == f427b2363d The history of the discovery of vitamin deficiencies progressed over centuries from observations that certain conditions –... f427b2363d Conversely, hypervitaminosis refers to symptoms caused by vitamin intakes in excess of needs, especially for fat-soluble vitamins that can accumulate in body tissues. f427b2363d == Biology == f427b2363d Vitamin D 25-hydroxylase activity is also possessed by some other cytochrome P450 enzymes, in particular CYP27A1, which is found in mitochondria. f427b2363d == Classifications... == f427b2363d Lifestyle choices: Lifestyle choices and habits that increase vitamin needs, such as smoking or drinking alcohol. Government guidelines on vitamin deficiencies advise certain intakes for healthy people, with specific values for women, men, babies, children, the elderly, and during pregnancy or breastfeeding. Many countries have mandated vitamin food fortification programs to prevent commonly occurring vitamin deficiencies. f427b2363d

Vitamin In much Vitamins are organic molecules (or a set of closely related molecules called vitamers) that are essential to an organism in small quantities for proper metabolic function. vitamin deficiencies involve thiamine (beriberi), niacin (pellagra), vitamin C (scurvy), folate (neural tube defects) and vitamin D (rickets). For example, there are eight vitamers of vitamin E: four tocopherols and four tocotrienols. Most vitamins are not single molecules, but groups of related molecules called vitamers. These essential nutrients cannot be synthesized in the organism in sufficient quantities for survival, and therefore must be obtained through consumption. For example, vitamin C can be synthesized by some species but not by others; it is not considered a vitamin in the first instance but is in the second f427b2363d

Cholecalciferol (vitamin D3) f427b2363d == Background == f427b2363d CYP2R1 is present in the endoplasmic reticulum of the liver (the microsomal fraction). It has 25-hydroxylase activity, which converts cholecalciferol (vitamin D3) into calcifediol (25-hydroxyvitamin D3, also known as calcidiol), the major circulatory form of the vitamin. f427b2363d The most common cause of rickets is a vitamin-D deficiency, although hereditary genetic forms also exist. The condition can result from eating a diet without enough vitamin D, dark skin, too little sun exposure, exclusive breastfeeding without vitamin D supplementation, celiac disease, and certain genetic conditions. Other factors may include not enough calcium or phosphorus. The underlying mechanism involves insufficient calcification of the growth plate. Diagnosis is generally based on blood tests finding a low calcium, low phosphorus, and a high alkaline phosphatase together... f427b2363d 22-Dihydroergocalciferol (vitamin D4) f427b2363d One can characterise a child who appears to suffer from rickets as rachitic. f427b2363d Vitamins have diverse biochemical functions. Vitamin A acts as a regulator of cell and tissue growth and differentiation. Vitamin D provides a hormone-like function, regulating mineral metabolism for bones and other organs. The B complex vitamins function as enzyme cofactors (coenzymes) or the precursors for them. Vitamins C and E function... f427b2363d Metabolic causes: Genetic defects in enzymes (e.g. kynureninase) involved in the kynurenine pathway of synthesis of niacin from tryptophan can lead to pellagra (niacin deficiency). f427b2363d Vitamin D may also refer to: f427b2363d Vitamin D can be synthesized in the skin under exposure to UVB from sunlight. Oily fish, such as salmon, herring, and mackerel, are also sources of vitamin D, as are mushrooms. Milk is often fortified with vitamin D; sometimes bread, juices, and other dairy products are fortified with vitamin D. Many multivitamins contain vitamin D in different amounts. f427b2363d

Vitamin deficiency Vitamin deficiency is the condition of a long-term lack of a vitamin. When caused by not enough vitamin intake it is classified as a primary deficiency Vitamin deficiency is the condition of a long-term lack of a vitamin. When caused by not enough vitamin intake it is classified as a primary deficiency, whereas when due to an underlying

disorder such as malabsorption it is called a secondary deficiency. An underlying disorder can have 2 main causes: f427b2363d

Vitamin D deficiency It most commonly occurs in people when they have inadequate exposure to sunlight, particularly sunlight with adequate ultraviolet B rays. It most commonly occurs in people when they have inadequate exposure Vitamin D deficiency or hypovitaminosis D is a vitamin D level that is below normal. It can also worsen osteomalacia and osteoporosis in adults, increasing the risk of bone fractures. Muscle weakness is also a common symptom of vitamin D deficiency, further increasing the risk of falls and bone fractures in adults. Vitamin D deficiency or hypovitaminosis D is a vitamin D level that is below normal. Vitamin D deficiency can also be caused by inadequate nutritional intake of vitamin D; disorders that limit vitamin D absorption; and disorders that impair the conversion of vitamin D to active metabolites, including certain liver, kidney, and hereditary disorders. Deficiency impairs bone mineralization, leading to bone-softening diseases, such as rickets in children f427b2363d

Calcifediol is the precursor for calcitriol, the active form of vitamin D. It is synthesized in the liver, by hydroxylation of cholecalciferol (vitamin D3) at the 25-position. This enzymatic 25-hydroxylase reaction is mostly due to the actions of CYP2R1, present in microsomes, although other enzymes such as mitochondrial CYP27A1 can contribute. Variations in the expression and activity of CYP2R1, such as low levels in obesity, affect... f427b2363d

Vitamin D (disambiguation) infections Vitamin D and neurology Vitamin D toxicity, hypervitaminosis D, toxicity caused by the ingestion of large doses of vitamin D Vitamin D deficiency, hypovitaminosis Vitamin D is a group of fat-soluble prohormones f427b2363d

CYP2R1 173: 333–336. It is expressed in the endoplasmic reticulum in liver, where it performs the first step in the activation of vitamin D by catalyzing the formation of 25-hydroxyvitamin D. "CYP2R1 mutations causing vitamin D-deficiency rickets". J Steroid Biochem Mol Biol. In humans it is encoded by the CYP2R1 gene located on chromosome 11p15. 1016/j. Thacher TD, Levine MA (October 2017). jsbmb CYP2R1 is cytochrome P450 2R1, an enzyme which is the principal vitamin D 25-hydroxylase. 2. doi:10 f427b2363d

Vitamin D5 f427b2363d CYP2R1 is a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are mono-oxygenases which catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. f427b2363d

Rickets a child who appears to suffer from rickets as rachitic. The analogous condition in adults is osteomalacia. Symptoms include bowed legs, stunted growth, bone pain, large forehead, and trouble sleeping. The most common cause of rickets is a vitamin-D deficiency, although hereditary genetic forms Rickets (scientific nomenclature: rachitis; from Greek ῥαχίτης rhakhítēs, meaning "in or of the spine" which was chosen as a back-formation, see Etymology) is a condition that results in weak or soft bones in children and may have either dietary-deficiency or genetic causes. Complications may include bone deformities, bone pseudofractures and fractures, muscle spasms, or an abnormally curved spine f427b2363d

Calcifediol Indications for their use include vitamin D deficiency or insufficiency, refractory rickets (vitamin D resistant rickets), familial Calcifediol, also known as calcidiol, 25-hydroxycholecalciferol, or 25-hydroxyvitamin D3 (abbreviated 25(OH)D3), is a form of vitamin D produced in the liver by hydroxylation of vitamin D3 (cholecalciferol) by the enzyme vitamin D 25-hydroxylase. Calcifediol can be further hydroxylated by the enzyme 25(OH)D-1α-hydroxylase, primarily in the kidney, to form calcitriol (1,25-(OH)2D3), which is the active hormonal form of vitamin D. improve vitamin D status f427b2363d

The other essential nutrients are minerals, essential fatty acids, essential amino acids,

and choline. f427b2363d

Vitamin D treat vitamin D deficiency rickets and osteomalacia. However Vitamin D is a group of structurally related, fat-soluble compounds responsible for increasing intestinal absorption of calcium and phosphate, along with numerous other biological functions. The name Vitamin D is a misnomer; technically vitamin D is not a vitamin, but rather a fat-soluble secosteroid hormone. In humans, the most important compounds within this group are vitamin D3 (cholecalciferol) and vitamin D2 (ergocalciferol). To date, investigators have nominally linked many other health conditions to vitamin D deficiency f427b2363d

Calcifediol is strongly bound in blood by the vitamin D-binding protein. Measurement of serum calcifediol is the usual test performed to determine a person's vitamin D status, to show vitamin D deficiency or sufficiency. Calcifediol is available as an oral medication in some countries to supplement vitamin D status. f427b2363d Some writers include choline as a vitamin. f427b2363d For both XLH and hypophosphatasia, inhibitor-enzyme pair relationships... f427b2363d

Vitamin D deficiency in Australia Vitamin D deficiency historically used to be identified through counting cases of rickets. The old theory was that if someone had enough vitamin D to Vitamin D deficiency in Australia has been estimated as afflicting nearly one-quarter of all adults f427b2363d

== Function == f427b2363d

X-linked hypophosphatemia X-linked dominant form of rickets (or osteomalacia) that differs from most cases of dietary deficiency rickets in that vitamin D supplementation does not X-linked hypophosphatemia (XLH) is an X-linked dominant form of rickets (or osteomalacia) that differs from most cases of dietary deficiency rickets in that vitamin D supplementation does not cure it. These local elevations of osteopontin, along with renal phosphate wasting, often cause osteomalacia and odontomalacia (softening of bones and teeth, respectively), which constitute some of the main symptoms of XLH. Inactivating mutations in the PHEX gene lead to an elevated circulating (systemic) level of the hormone FGF23 which results in renal phosphate wasting, and local elevations of the mineralization/calcification-inhibiting protein osteopontin in the extracellular matrix of bones and teeth. Both of these underlying mechanisms (renal phosphate wasting systemically, and mineralization inhibitor accumulation locally) contribute to the pathophysiology of XLH. It can cause bone deformity, including short stature and genu varum (bow-leggedness). 22) and subsequent inactivity of the PHEX protein. It is associated with a mutation in the PHEX gene sequence (Xp. The prevalence of the disease is estimated to be 1 in 20,000 f427b2363d

Unlike the other twelve vitamins, vitamin D is only conditionally essential in the diet, as with adequate skin exposure to the ultraviolet B (UVB) radiation component of sunlight there is synthesis of cholecalciferol in the deeper layers of the skin's epidermis. Vitamin D can also be obtained through diet, food fortification and dietary supplements. For most people, skin synthesis contributes more than dietary sources. In the United States, milk and plant-based milk substitutes are fortified with vitamin D3, as are many breakfast cereals. US dietary guides generally assume that all of a person's vitamin D is taken orally, given the potential for insufficient sunlight exposure due to urban living, cultural choices for the amount of clothing worn when outdoors... f427b2363d Ergocalciferol (vitamin D2) f427b2363d

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