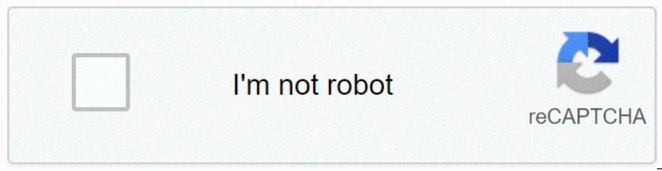

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Tomty gaming executor v1.5

Jūsu pārlūkprogramma vairs netiek atbalstīta. Atjauniniet to, lai iegūtu vislabāko YouTube lietošanas pieredzi un mūsu jaunākās funkcijas. Uzziniet vairākAtgādināt vēlāk Editor Lock D-D-D-DMosomes are derived from crosslinked fibrin clotting blocks dissolved by lysosomes, which primarily reflect fibrin dissolution. Clinical testing of D-djubo is mainly used in the diagnosis of venous thromboembolism (VTE) (m) deep vein thrombosis (DVT) and pulmonary embolism (PE). Increased: Seen in secondary fibrin dissolving function, such as high coagulation state, dispersive blood vessel clotting, kidney disease, organ transplant rejection reaction, thrombolytic treatment. Myocardial infarction (m) cerebral infarction (m) pulmonary embolism (m) venous thrombosis (m) surgery (m) tumor (m) diffuse intravascular clotting (m) infection and tissue necrosis can also lead to D-djumor. D-D-djumer normal value reference range Different reagent normal value range is different, generally slt;0.3mg/L or slt;0.5mg/L. Hiili-sinkki-D-akku, positiivinen liitin ylös. D paristot. D-paristo (D-kenno tai IEC R20) on kuivan kennon kokoinen. D-kenno on lieriömäinen, ja sen päässä on sähkökosketin; positiivisessa päässä on nupu tai kuhmu. D-kennoja käytetään tyypillisesti suurivirtaisiin tyhjennyssovelluksiin, kuten suurissa taskulampuissa, radiovastaanottimissa ja lähettimissä sekä muissa laitteissa, jotka vaativat pidennetyn käyttöajan. D-kenno voi olla joko ladattava tai ei-ladattava. Sen päätejännite ja kapasiteetti riippuvat sen kennokemiasta. National Carbon Company esitteli ensimmäisen D-kennon vuonna 1898. Ennen kuin pienemmät kennot yleistyivät, D-kennot tunnettiin laajalti taskulamppujen paristoina. Usa:n sotilasnimitys tälle akulle on ollut BA-30 jo ennen toista maailmansotaa. [1] Toisen maailmansodan aikana Yhdysvaltain laivasto nimesi sen C-tyypin akuksi, mikä aiheutti sekaannusta pienemmän C-kennoakun (BA-42) kanssa. Vuonna 2007 D-akkujen osuus ensiakkujen emäksisen myynnin osuus (numeerisesti) yhdysvalloissa oli 8 prosenttia. Vuonna 2008 sveitsiläiset D-paristojen ostot olivat 3,4 prosenttia primaari- ja 1,4 prosenttia toissijaisista myynnistä. [2] [3] Mitat ja kapasiteetti D, C, AA, AAA-, AAAA- ja 9 voltin paristot Sinkki–hiili Alkaline Li-FeS2 NiCd NiMH IEC nimi R20 LR20 FR20 HR20 HR20 ANSI/NEDA nimi 13D 13A 13LF Tyypillinen kapasiteettimaksu 8000 mAh 12000-18000 mAh 2000-5500 mAh 2200–12000 mAh energia 12 Wh 18-27 Wh 2,5-6,9 Wh 2,75-15 Wh Nimellisjännite 1.5 V 1.5 V 1.5 V 1.25 V 1.25 V Ladattava Ei Erikoistyyppi vain Ei Kyllä Kyllä。 Akun kapasiteetti riippuu sen kennokemiasta ja nykyisestä vetonaulasta. Duracell-brändi arvioi emäksisen D-kennonsa noin 20 000 mAh: ssa 25mA-arvonnassa, mutta arvioi suorituskyvyn olevan lähempänä noin 10 000 mAh 500mA-arvonnassa. [4] Tämä vaikutus on yleensä vähäisempi soluissa, joissa on NiCd- ja NiMH-chemistries; Katso Peukertin lakia. Monet yleisesti saatavilla olevat D-kokoiset ladattavat kennot ovat itse asiassa D-kokoisen pidikeen sub-C-kennoja. D-paristojen nimellishalkaisija on 33,2 ± 1 millimetriä (1,3。 Total length 61.5 millimeters (2.42 inches). [5] Other generic names[citation required] HP2 /SP2/U2 (in Britain until the 1980s) Flashlight Battery MN1300 MX1300 Mono Goliath Type 373 (Soviet/Russian) BA-30 (US Military Spectrum World War II– 1980s UM 1 (JIS) #1 (China) 6135-99-464-1938 (NSN, carbon zinc) 6135-99-109-9428 (NSN, Alkaline) B006 (NiMH) See also List battery size Battery nomenclature References ^ U.S. Military flashlight.. ^ Life cycle effects of alkaline batteries focusing on end of service life - EPBA-EU Archived 7.10.2011 Wayback Machine ^ Absatzzahlen 2008 (PDF). Archived from original (PDF) 25.3.2012. Retrieved 25 March 2012. INOBAT 2008 statistics. ^ MN1300 Size: D (LR20) Alkaline manganese dioxide battery (PDF). Archived from the original (PDF) on 21 May 2012. ^ IEC 60086-2§ 7.1.4 External links Duracell D Size Battery Specification Energizer D Size Battery Specification for Alkaline Cell Brand Neutral Drawing of NiCd D Battery Based on ANSI Configuration a brand-neutral drawing of a NiMH D battery based on anSI configuration A brand-neutral drawing based on ANSI configuration Wikimedia Commons has D-battery-related media files. Retrieved from a group of chemical compounds for other uses, see vitamin D (blessing). This article is about the Vitamers family. See individual forms of ergocalciferol, choleraferol, vitamin D4 and vitamin D5. Vitamin DDrug ClassCholecalciferol (D3)Class IdentifiersSynonymsCalciferolsUseRickets, osteoporosis, vitamin D deficiencyATC codeA11CCBiological targetvitamin D receptorClinous dataDrugs.comMedFacts Natural ProductsExternal linksMeSHD014807In Wikidata Vitamin D is a group of fat-soluble secoroids responsible for increased absorption of calcium, magnesium and phosphate in the intestine, and many other biological effects. [1] [2] In humans, the main compounds in this group are vitamin D3 (also known as choleraferol) and vitamin D2 (ergocalciferol). [1] [2] [3] The main natural source of vitamin vial is the synthesis of cholelesiferol in the lower layers of the skin's orphans through a chemical reaction dependent on sun exposure (in particular UVB radiation). [4] [5] Cholecalciferol and ergocalciferol may be taken from food and supplements. [6] [2] Only a few foods, such as meat from fatty fish, naturally contain significant amounts of vitamin D. [1] [7] In the United States and other countries, cow's milk and herbal milk substitutes have been reinforced with vitamin D, as have many breakfast cereals. Fungi exposed to ultraviolet light affect useful amounts of vitamin D. [1] Dietary recommendations typically assume that all a person's vitamin D is taken orally because the sun there are varying populations and recommendations safe exposure is uncertain due to the risk of skin cancer. [1] Vitamin D in diet or skin synthesis is biologically inactive. It is activated by two stages of protein enzyme hydroxylation, the first in the liver and the second in the kidneys. [3] Since most mammals can synthesize vitamin D in sufficient quantities, if exposed to adequate sunlight, it is not necessary, so technically no vitamin. [2] Instead, it can be considered a hormone whose activation of vitamin D pro-hormone leads to an active form, calcitriol, which then produces effects through a nuclear receptor in several places. [2] Coleliferol is converted into calciphediol in the liver (25-hydroxycolecalciferol); ergocalciferol is converted to 25-hydroxyyergocalciferol. These two vitamin D metabolites (called 25-hydroxyvitamin D or 25(OH)D) are measured in serum to determine a person's vitamin D status. [8] [9] Kidneys hydroxylate calcitriol (also known as 1,25-dihydroxycolecalciferol), a biologically active form of vitamin D. Calcitriol also has other effects, such as some on cell growth, neuromuscular and immune functions, and reduced inflammation. [1] Vitamin D plays a significant role in calcium homeostasis and metabolism. Its discovery was due to an effort to find a nutrient that is missing in children with rickets (a childhood form of osteomalacia). [12] Vitamin D supplements are given to treat or prevent osteomalacia and rickets. Evidence of other health effects of vitamin D supplementation in the population is inconsistent. [1] The effect of vitamin D supplementation on mortality is not clear. [14] Types Name Chemical composition Structure vitamin D1 Mixture of molecular compounds of Ergocalciferol with lumisterol, 1:1 Vitamin D2 eergocalciferol (made from ergosterol) Vitamin D3 cholecalciferol (made from 7-dehydrocolesterol in the skin). Vitamin D4 22-dihydroergocalciferol Vitamin D5 sitocalciferol (made from 7-dehydrositosterol) Vitamin D exists in several forms (vitamers). The two main forms are vitamin D2 or ergocalciferol, as well as vitamin D3 or choleraferol; Vitamin D without subscript refers to either D2 or D3 or both. These are called calciferol together. [15] Vitamin D2 was chemically characterised in 1931. In 1935, the chemical structure of vitamin D3 was confirmed and proven due to ultraviolet radiation Chemically, different forms of vitamin D are lying steroids, or steroids, in which one of the bonds of steroid rings is broken. [16] The structural difference between vitamin D2 and vitamin D3 is the side chain of D2, which contains a double bond between carbons 22 and 23 and between carbon 24 in the methyl group. [3] Regulation of calcium in biology in the human body. [17] The role of active vitamin D (1,25-dihydroxyvitamin D, calcitriol) appears orange. Active vitamin D metabolite calcitriol transmits its biological effects by binding to the vitamin D receptor (VDR), which is located mainly in the cores of the target cells. [16] Calcitriol binding to VDR allows VDR to act as a transcription factor that modulates the expression of genes in transport proteins (such as TRPV6 and calbin) involved in the absorption of calcium in the intestine. [18] The vitamin D receptor belongs to the superfamily of the core receptors of steroid/thyroid hormone receptors, and VDRs are expressed as cells of most organs, including the brain, heart, skin, gonads, prostate and breast. VDR activation in the intestines, bone, kidneys and thyroid cells leads to the maintenance of calcium and phosphorus levels in the blood (aided by thyroid hormone and calcitonin) and the maintenance of bone levels. [19] One of the main tasks of vitamin D is to maintain calcium balance in the skeleton by promoting the absorption of calcium in the intestine, promoting bone resorption by increasing the number of osteoclasts, maintaining calcium and phosphate levels for bone formation and enabling the proper functioning of parathyroid hormone to maintain serum calcium content. Vitamin D deficiency can lead to a decrease in bone mineral density and an increased risk of bone density (osteoporosis) or bone fracture, since vitamin D deficiency alters mineral metabolism in the body. [20] Therefore, vitamin D is also critical for bone relapse, since it acts as a powerful stimulator of bone resorption. [20] VDR regulates cell proliferation and unification. Vitamin D also affects the immune system, and VDRs are expressed in several white blood cells, including monocytes and activated T and B cells. [21] Vitamin D in vitro increases the expression of tyrosine hydroxylase gene in adrenal medullary cells and affects the synthesis of neurotrophic factors, nitric oxide synthase and glutathione. [22] The expression of the vitamin D receptor decreases with age and observations suggest that vitamin D is directly related to muscle strength, mass and function, all of which are important factors for athlete performance. [23] Deficiency Main article: Vitamin D deficiency An estimated one billion people worldwide are either insufficient or deficient in vitamin D. [23] Vitamin D deficiency is widespread in the European population. [24] Diet without enough vitamin D in combination with the sun. With. causes vitamin D deficiency. Severe vitamin D deficiency in children causes rickets, softening and weakening of bones, a rare disease in the developed world. [25] Vitamin D deficiency is diagnosed worldwide in the elderly and is still common in children and adults. [26] [27] [28] Deficiency leads to decreased bone mineralization and bone damage leading to bone softening[29], including paediatric rickets and adult osteomalacia. Low blood calciphendiol (vitamin D 25-hydroxy-D) can be caused by avoiding the sun. [30] Vitamin D deficiency may cause calcium absorption into the intestine to fall to 15%. [19] If an individual is not deficient, it is usually absorbed to between 60 % and 80 %. [19] Bone Health Rickets Main article: Rickets Rickets, a childhood disease, is characterized by slowing growth and soft, weak, deformed long bones that bend and bow under their weight when children begin to walk. Rickets typically occur at the age of 3-18 months. [31] Cases are still reported in North America and other Western countries, mainly in breast-sucked infants and those with darker skin color. [31] This condition is characterised by bow legs[29], which may be due to a lack of calcium or phosphorus and vitamin D deficiency. Today, it is widely found in low-income countries in Africa, Asia or the Middle East[32] and those with genetic disorders such as pseudovitamin D deficiency rickshaws. [33] Maternal vitamin D deficiency may cause bone disease and bone quality loss after birth prior to birth. [34] [35] Nutritional rickets occur in countries with strong year-round sunlight, such as Nigeria, and can occur without vitamin D deficiency. [36] [37] Although rickets and osteomalacia are now rare in the UK, outbreaks have broken out in some immigrant communities, where those suffering from osteomalacia included women with seemingly adequate daylight exposure in Western clothing. [38] Darker skin and reduced exposure to sunshine did not produce rickets unless the diet differed from the Western omnivory model, which is characterized by high meat, fish and egg intake and low intake of high extracted [as defined] cereals. [39] [40] [41] Dietary risk factors for rickets include abstinence from food in animals. [38] [42] Vitamin D deficiency remains the main cause of infant rickets in most countries, as breast milk is low in vitamin D and social habits and climatic conditions can prevent adequate exposure to the sun. In sunny countries such as Nigeria, South Africa and Bangladesh, where rickets occur in older toddlers and among them, it has been caused by low calcium intake, which is characteristic of grain-based diets with limited access to dairy products. [41] Rickets used to be a major public health problem among the U.S. population. In Denver, where the ultraviolet rays are 20% stronger than at sea level at the same latitude[43], almost two thirds of the 500 children had mild rickets in the late 1920s. [44] The increase in the share of animal protein[44] 42][45] in the 20th century American diet combined with increased milk consumption[46][47], confirmed by relatively low levels of vitamin D, coincided with a dramatic reduction in the number of cases of rickets. [19] Milk, vitamin D supplements and vitamin supplements confirmed by vitamin D in the United States and Canada have also helped eradicate the majority of ricket cases in children with fat absorption conditions. [29] Osteoporosis and osteomalacia The main articles: Osteoporosis and Osteomalacia Osteomalacia is an adult disease caused by a lack of vitamin D. The characteristics of this disease are softening of the bones, which leads to bending of the spine, bowing of the legs, proxial muscle weakness, bone fragility and an increased risk of fractures. [48] Osteomalacia reduces calcium absorption and increases bone loss of calcium, increasing the risk of bone fractures. Osteomalac usually occurs when, When 25-hydroxyvitamin D-levels are less than 10 ng/ ml. [49] Although the effects of osteomalacia are thought to contribute to chronic musculoskeletal pain,[50] people with chronic pain do not have convincing evidence of driving vitamin D levels[51] or that supplementation relieves chronic non-sequential musculoskeletal pain. [52] Skin pigmentation Dark-skinned people living in temperate climates have shown low levels of vitamin D, but its importance is not certain. [53] [54] [55] Dark-skinned people are less effective at making vitamin D because skin melanin inhibits vitamin D synthesis. [56] Vitamin D deficiency is common among Hispanics and African-Americans in the United States, and levels drop significantly in winter. [57] This is due to melanin levels in the skin as it acts as a natural protection against exposure to the sun. [57] Use of supplements Vitamin D supplementation is a reliable method for preventing or treating rickets. The effects of vitamin D supplementation on non-skeletal health are uncertain. [58] [59] The 2013 review found no effect other than a preliminary reduction in mortality among older people. [60] Vitamin D supplements do not change the consequences of myocardial infarction, stroke or cerebroacular disease, cancer, bone fractures or knee osteoarthritis. [14] [61] Low vitamin D levels may be due to illness rather than disease. [60] A report by the U.S. Institute of Medicine (IOM) states: Cancer, cardiovascular disease and hypertension, diabetes and metabolic syndrome, falls and physical performance, immune function and results related to infections, neuropsychological function and preeclampsia is not reliably linked to calcium or vitamin D intake, and they were often contradictory. [62]:5 Some researchers argue that the IOM was too final in its recommendations and made a mathematical error in lowering vitamin D levels associated with bone health. [63] IOM panel members claim to have used the standard procedure in dietary recommendations and that the report is firmly based on data. Research into vitamin D supplements, including extensive clinical trials, continues. [63] Mortality, all causes vitamin D3 supplementation has been provisionally found to reduce the risk of death in older people[13][60], but the effect has not been considered strong or sufficiently certain to recommend the use of supplements. [14] Other forms (vitamin D2, alpha calcidol and calcitriol) do not appear to have beneficial effects on the risk of death. [13] High blood levels appear to be associated with a lower risk of death, but it is unclear whether replenishment can lead to this benefit. [64] Both excessive and vitamin D deficiency appear to cause abnormal activity and premature aging. [65] [66] [67] The relationship between serum calciphediolias and overall mortality is parabolic. [clarification required] [62] Vitamin D appears to be harmful to the black population at lower vitamin D levels than the white population. [62]:435 Bone health Generally speaking, no good evidence supports the generally held belief that vitamin D supplements can help prevent osteoporosis. [14] Its general use to prevent this disease in those without vitamin D deficiency is unlikely to be necessary. [68] In elderly patients with osteoporosis, taking vitamin D with calcium may help prevent hip fractures, but it also slightly increases the risk of abdominal and kidney problems. [69] The study found that supplementation with 800 IU or more per day in people over 65 years of age was somewhat favorable in preventing hip and non-vertebral fracture. [70] The effect is small or nothing for people living independently. [71] [72] Low serum vitamin D levels have been associated with falls and low bone mineral density. [73] However, taking excess vitamin D does not seem to change the risk. [74] Athletes without vitamin D are at increased risk of stress fractures and/or major breaks, especially athletes engaged in contact sports. The greatest benefit with supplements is in athletes who are deficient (25(OH)D serum levels <30 ng/ml) or severely deficient (25(OH)D serum levels <25 ng/ml). A reduction in risks is observed when serum concentrations of 25(OH)D increase at 50 ng/ml without additional benefits after this point. [75] As it found increasing evidence of bone health, although it had found no good evidence of other benefits, us food and has required manufacturers to indicate the amount of vitamin D on the labelling of nutritional facts as nutrients relevant to public health as of May 2016. The proposed extension of the deadline will give some manufacturers until 1 July 2021 to comply. [76] Vitamin D supplements for cancer have been widely marketed due to their alleged cancer drug properties. [77] Observation studies between low vitamin D levels and the risk of developing certain cancers have shown links. [78] However, it is unclear whether the addition of vitamin D to the diet or dietary supplements affects the risk of cancer. The reviews have described the evidence as inconsistent, causal and insufficient to indicate nutritional needs[62] and insufficient to draw conclusions. [71] One 2014 review concluded that the supplement had no significant impact on cancer risk. [14] Another 2014 review concluded that vitamin D3 can reduce the risk of cancer death (one less risk of death from 150 people treated over five years), but data quality concerns were identified. [13] There was insufficient evidence to recommend vitamin D supplements to all cancer patients, although some evidence suggests that low vitamin D may be associated with a worse outcome for some cancers[79] and that higher levels of vitamin D at the time of diagnosis were associated with better results. [80] A systematic review and meta-analysis in 2020 in patients with colorectal cancer showed signs of clinically relevant benefit to obtain vitamin D supplementation from the results, including survival, although there were limitations in the analysis. [81] Cardiovascular diseases The use of vitamin D supplements does not significantly reduce the risk of stroke, cerebrosmic diseases, myocardial infarction or sechemistry heart disease. [14] [82] Supplementation may not affect blood pressure. [83] Immune system Infectious diseases In general, vitamin D activates and dampens adaptive immune systems through antibacterial, antiviral and anti-inflammatory effects. [84] [85] Deficiency has been linked to increased risk or severity of viral infections such as HIV[86][87] and COVID-19. [88] Low vitamin D levels appear to be a risk factor for tuberculosis[89] and have traditionally been used as a treatment. [90] Vitamin D supplementation in low doses (400-1000 IU/day) slightly reduces the overall risk of acute respiratory infections. [91] Benefits were observed in the meta-analysis of the data only in young children and adolescents (1-16 years of age) and were not confirmed to be higher doses (>1000 IU per day or more). [91] Other reported positive effects include worsening asthma [92] Vitamin D supplementation significantly reduces the inert or severe exacerbation of pulmonary distress in people with baseline 25(OH)D levels below 25nmol/l, but not in those with less Deficiency. [93] Autoimmune diseases Although preliminary data link low vitamin D levels to asthma, evidence supporting the beneficial effect of supplements on asthmatics is not certain. [94] One review found that vitamin D supplementation may reduce the need for steroids used to prevent the frequency of episodes in patients with mild to moderate asthma and that supplements do not affect everyday asthma symptoms. [95] In general, supplementation of vitamin D is not recommended for the treatment or prevention of asthma. [96] Inflammatory bowel disease Low vitamin D levels are associated with two major forms of human inflammatory bowel disease (IBD) : Crohn's disease and wound-related colitis. [97] Meta-analysis of vitamin D therapy in IBD patients with vitamin D deficiency has shown that supplements are effective in correcting vitamin D levels and are associated with improved clinical disease activity and biochemical marker scores. [98] Other Conditions Diabetes – A systematic review in 2014 found that there is no evidence in available studies of vitamin D3 supplementation that affects glucose homeostasis or diabetes prevention. [99] A 2016 review article concluded that while there is growing evidence that vitamin D deficiency may be a risk factor for diabetes, overall evidence of vitamin D levels and diabetes melliation is contradictory, which requires further investigation. [100] Depression – Depression - Clinical studies of vitamin D supplementation for depressive symptoms have generally been of poor quality and have no overall effect, although subgroup analysis showed supplements participants with clinically significant depressive symptoms or depression had a moderate effect. [101] Cognition and dementia – Systematic review of clinical trials found a link between low vitamin D levels associated with cognitive impairment and the risk of developing Alzheimer's disease. However, lower levels of vitamin D are also associated with poor nutrition and less outdoor activities. Therefore, there are alternative explanations for the increase in cognitive impairment, so the direct causal link between vitamin D levels and cognition could not be established. [102] Pregnancy – Low levels of vitamin D in pregnancy are associated with gestational diabetics, pre-eclampsia and small babies (of gestational age). [103] Although the use of vitamin D supplements during pregnancy increases the mother's vitamin D content with the term[104], the full extent of hair for mother or baby is unclear. [103] [104] [105] Pregnant women taking enough vitamin D during pregnancy may have a lower risk of pre-eclampsia[106] and positive immune effects. [107] also likely to reduce gestational dotage, nane infants[106] and their poor Pregnant women often do not take the recommended amount of vitamin D. [107] Weight loss - Although it is assumed that vitamin D supplementation can be an effective treatment for obesity in addition to calorie restriction, together a systematic review found no link to supplementing body weight or fat mass. [109] A 2016 meta-analysis found that vitamin D status was improved by weight loss, indicating that fat mass may inversely be associated with vitamin D levels in the blood. [110] Permitted health claims State regulatory agencies impose on the food and food supplement industry certain health claims that are as permitted as statements made on packaging. Normal immune function of the European Food Safety Authority[111] normal inflammatory response[111] normal muscle function[111] reduced the risk of developing the disease in people over 60[112] The U.S. Food and Drug Administration (FDA) Adequate calcium and vitamin D, combined with a balanced diet and exercise, may reduce the risk of osteoporosis. [113] Health Canada Adequate calcium and regular exercise can help achieve strong bones in children and adolescents and reduce the risk of osteoporosis in older adults. Adequate intake of vitamin D is also necessary. [114] Other agencies with claims guidelines: Japan FOSHU[115] and Australia-New Zealand. [116] Dietary intake Recommended levels Different institutions have proposed different recommendations on the amount of daily intake of vitamin D. They vary according to the exact definition, age, pregnancy or breastfeeding, and assumptions are made about the skin synthesis of vitamin D. [117] [62] [118] [119] [120] Conversion: 1 µg (microgram) = 40 IU (international unit). [117] United Kingdom Age group Intake (µg/day) Maximum intake (µg/day)[117] Breast-fed infants 0–12 months 8.5 - 10 25 Formula-fed infants (<500 ml/d)= 10= 25= children= 1= -= 10= years= 10= 50= children=>10 and adults 10 100 United States Age group RDA (IU/day) (µg/day)[62] Infants 0–6 months 400* 10 Infants 6–12 months 400* 10 1–70 years 600 15 71+ years 800 20 Pregnant/Lactating 600 15 Age group Tolerable upper intake level (IU/day) (µg/day) Infants 0–6 months 1,000 25 Infants 6–12 months 1,500 37.5 1–3 years 2,500 62.5 4–8 years 3,000 75 9+ years 4,000 100 Pregnant/lactating 4,000 100 [62] Canada Age group RDA (IU) Tolerable upper intake (IU)[118] Infants 0–6 months 400* 1,000 Infants 7–12 months 400* 1,500 Children 1–3 years 600 2,500 Children 4–8 years 600 3,000 Children and Adults 9–70 years 600 4,000 Adults > 70 years 800 4,000 Pregnancy & Lactation 600 4,000 Australia and New Zealand Age Group Adequate intake (µg) Upper intake level (µg)[119] Infants 0-12 months 5* 25 Children 1-18 years. 5* 80 Adults 19-50 years 5* 80 Adults 51-70 years 10* 80 Adults > 70 years 15* 80 European ikäryhmä </500> </500> Intake (µg)[120] Tolerable Upper Limit (µg)[121] Infants 0-12 months 10 25 Children 1-10 years 15 50 Children 11-17 years 15 100 Adults 15 100 Pregnancy & breastfeeding 15,100 * Adequate intake, THE RDA/RDI has not yet been established in the UK The UK's National Health Service (NHS) recommends that people at risk of vitamin D deficiency, breast-fed babies, babies fed with a formula taking less than 500 ml/day and children aged 6 months to 4 years should take vitamin D supplements daily throughout the year to ensure adequate intake. [117] This also applies to people with limited vitamin D skin synthesis who are not often outdoors, are fragile, houses, live in a nursing home or usually wear clothes that cover most of the skin, or on dark skin such as african, African-Caribbean or South Asian backgrounds. Other people may be able to make enough vitamin D from sunlight from April to September. The NHS and Public Health England recommend that everyone, including pregnant and breastfeeding women, consider a daily supplement containing 10 µg (400 IU) of vitamin D during autumn and winter, as sunlight is not enough for vitamin D synthesis. [122] The 2010 U.S. Institute of Medicine (IoM) reference intake for vitamin D diet (renamed The National Academy of Medicine in 2015) exceeded previous recommendations expressed as an adequate intake. The recommendations were formed on the assumption that the individual does not have skin synthesis of vitamin D due to insufficient sunlight. The reference intake of vitamin D refers to the total intake of foods, beverages and supplements and assumes that the calcium requirements are met. [62]:5 Tolerable upper intake level (UL) is defined as the highest average daily intake of nutrients, which is unlikely to pose a risk of adverse health effects to almost all persons in the population... [62]:403 Although UL is believed to be safe, information on long-term effects is incomplete and these intake levels are not recommended for long-term consumption. [62]:403:433 On the label of U.S. food and food supplements, the portion volume is expressed as a percentage of the daily value (%DV). For vitamin D labelling, 100% of the daily value was 400 IU (10 µg), but on May 27, 2016 it was revised to 800 IU (20 µg) to reach an agreement with the RDA. [123] [124] Compliance with updated labelling orders was necessary by 1 January 2020 from manufacturers with annual food sales of USD 10 million or more and by 1 January 2021 from manufacturers with lower food sales volumes. [76] [125] The Reference Daily Intake table contains a table of old and new daily adult values. Canada Health Canada published in 2012 dietary supplements (RDAs) and tolerable upper intake levels of vitamin D[118] based on the Institute of Medicine. based on the basis of Australia and New Zealand Australia and New Zealand published nutrient benchmarks in 2005, including guidelines on vitamin D intake in nutrients. [119] About a third of Australians have vitamin D deficiency. [126] In 2016[120], the European Union European Food Safety Authority (EFSA) reviewed the current evidence and concluded that the relationship between serum 25(OH)D and musculoskeletal health outcomes varies greatly. They considered that the average vitamin D requirements and population reference values could not be led and that serum concentrations of 25(OH)D of 50 nmol/l were an appropriate target value. For all women over 1 year of age, including pregnant or lactating women, they set an adequate intake of 15 µg/ day (600 IU). [120] In 2012, the European Food Safety Authority reviewed safe intake levels[121] to set a tolerable upper limit of 100 µg/day (4000 IU) for adults, a conclusion similar to that of the IOM. The Swedish National Food Agency recommends a daily intake of vitamin D3 of 10 µg (400 IU) for children and adults up to 75 years of age and 20 µg (800 IU) for adults 75 years older. [127] European state-free organisations have made their own recommendations. The German Nutrition Society recommends 20 µg. [128] The European Menopause and Andropause Society recommends 15 µg (600 IU) for women who have passed menopause at age 70 and 20 µg (800 IU) from the age of 71. The dose should be increased to 100 µg (4,000 IU) in some patients with very low vitamin D status or co-disease. [129] Sources Although vitamin D is naturally present in only a few foods[1], it is commonly added in foods prepared as 4thion. In some countries, basic foods are artificially reinforced with vitamin D. [130] Luonnolliset lähteet Eläinlähteet Lähde[131] IU/g Epäsäännöllisesti kypsennetty munankeltuainen 0,7 44 IU 61 g:n kananmunalle Naudanmaksa, keitetyt, haudutettu 0,5 Kalanmaksaöljyt, kuten turskanmaksaöljy 100 450 IU/tl (4,5 g) Rasvakalalaji Lohi, pinkki, keitetty, kuiva lämpö 5,2 Makrilli, Tyynimeri ja tunkki, sekalajit, keitetyt, kuivalämpö 4,6 Tonnikala, öljyssä säilyke 2,7 Sardiinit, öljyssä säilyke, valutettu 1,9 Sienilähteet Lähde µg/g IU/g Cladonia arbuscula (jäkälä), thalli, kuiva[132] D3-vitamiini 0,67–2,04 27–82 D2-vitamiini 0,22–0,55 8,8–22 Agaricus bisporus (tavallinen sieni): D2 + D3 Portobello Raw 0.003 0.1 Altistunut ultraviolettivalolle 0.11 4.46 Crimini Raw 0.001 0.03 Altistunut ultraviolettivalolle 0,32 12,8 Katso myös : Ergokalsiferol § Biosynteesi Yleensä D3-vitamiinia löytyy eläinperäisiä elintarvikkeita, erityisesti kalaa, lihaa, eläimenosia, kananmunia ja maitotuotteita. [133] Vitamin D2 is found in fungi and is produced by ultraviolet radiation of ergosterol. [134] and the content of vitamin D2 in the chodia arbuscula, lichen, increases with exposure to ultraviolet light[132][135] and is imitated by industrial ultraviolet lamps for fortification. [134] [134] The Ministry of Agriculture reports D2 and D3 content in one value. Food styriting Of foods reinforced with vitamin D include fruit juices and fruit juice drinks, energy bars replacing meals, so protein-based drinks, certain cheese and cheese products, flour products, infant formulae, many breakfast cereals and milk. [136] [137] In 2016 in the United States, the Food and Drug Administration (FDA) amended the provisions on food additives for milk stating[138], stating that the vitamin D3 content does not exceed 42 IU of vitamin D per 100 g (400 IU/U.S. liters) of dairy milk, 84 IU vitamin D2 per 100 g (800 IU per litre) of vegetable milk and 89 IU/100 g (800 IU per litre) in plant-based yogurts or soysome products. [139] [140] [141] Milk from plants refers, inter alia, to beverages made from syllaet, almonds and rice intended as alternatives to milk milk. [142] [143] Although some studies have shown that that vitamin D3 increases 25(OH)D blood levels faster and remains active in the body for longer,[144][145] others claim that the sources of vitamin D2 are equal to 25(OH)D.[134][146][147] Food preparation Vitamin D content in typical foods is reduced by varying degrees of cooking. Cooked, fried and fried foods stored 69-89% of the original vitamin D. [148] Recommended serum levels See also: Blood test benchmarks § Vitamins and Hypervitaminеosis D § Ethnic differences Global vitamin D levels in adults (nmol/l). [149] [150] > 75 50-74 25-49 Recommendations on recommended serum concentrations of 25(OH)D vary between authorities and vary according to age. [1] US laboratories usually report 25(OH)D-level ng/ml. [151] Nmol/L.[151] One ng/ml is about 2,5 nmol/l.[152] A 2014 review found that the lowest serum levels of 25(OH)D in all results appeared to be close to 30 ng/ml (75 nmol/l). [153] Optimal vitamin D levels remain controversial and the second review found that a range of 30-40 ng/ml (75-100 nmol/l) was recommended for athletes. [154] Part of the controversy stems from the fact that several studies have shown differences in serum concentrations between 25(OH)D ethnic groups. studies point to the genetic and environmental causes behind these variations. [155] Replenishment to achieve these standard levels may cause harmful vascular calamification. [55] A 2012 meta-analysis showed an increased risk of cardiovascular disease when the blood has the lowest vitamin D level between 8 and 24 ng/ml (20-60 nmol/l), although the results of the studies analysed were inconsistent. [156] In 2011, the IOM Committee concluded that skeletal and serum level 25 (OH)D of 20 ng/ml (50 nmol/l) is required. The nutritional values of vitamin D are selected by a margin of and exceed the desired serum value to ensure that the specified intake levels reach the desired serum 25(OH)D levels in almost all persons. Sun exposure is not expected to contribute to serum level 25(OH)D and the recommendations are fully applicable to people with dark skin or low exposure to sunlight. According to the Institute, serum concentrations of 25(OH)D above 30 ng/ml (75 nmol/l) are not consistently associated with increased benefit. Serum levels 25(OH)D above 50 ng/ml (125 nmol/l) may be of concern. However, some people with serum 25(OH)D between 30 and 50 ng/ml (75 nmol/L-125 nmol/l) also have insufficient vitamin D. [62] For more information, hypervitamin d vitamin D toxicity is rare. [28] It is because it is supplemented with high doses of vitamin D instead of sunlight. The threshold for vitamin D toxicity has not been established; However, according to some studies, the tolerable upper intake level (UL) is 4,000 IU/day between the age of 9 and 71[157] (100 µg/day), while other studies find that that in healthy adults, a continuous intake of more than 50,000 IU/day (1250 µg) may cause more than activity after several months and increase serum level 25-hydroxyvitamin to D-level 150 ng/ml and above. [28] [158] Those with certain conditions, such as primary parathyroid hyperthyroidism syndrome,[159] are much more sensitive to vitamin D and develop hypercalcaemia in response to increased dietary function of vitamin D, while maternal hypercalcaemia during pregnancy can increase foetal sensitivity to the effects of vitamin D and lead to mental retardation and facial malformation syndrome. [159] [160] Idiopathic infant hypercalcaemia is caused by a mutation in the CYP24A1 gene that leads to a reduction in vitamin D degradation. [161] [162] The disorder may persist into adulthood. [163] A review published in 2015 found that that adverse reactions have been reported at serum concentrations of only 25(OH)D above 200 nmol/l.[154] Published cases of hyper-calcimia, where vitamin D and vitamin D levels of 25-hydroxy-D are all known to be ≥40,000 IU (1,000 µg) per day. [159] Pregnant or lactating women should consult a doctor before taking vitamin D supplements. The FDA advised manufacturers of liquid vitamin D supplements that the drip devices accompanying these products should be clearly and accurately marked for 400 international units (1 IU is the biological equivalent of 25 ng cholecalifol/ergocalciferol). In addition, the FDA recommends infant products that the drip last up to 400 IU. [164] The tolerable upper limit for infants (born to 12 months) (the maximum level that can be tolerated without harm) is 25 µg/day. 1,000 micrograms a day in babies has produced a toxity within a month. [158] Since the Governments of Canada and the United States commissioned the Institute of Medicine (IOM) on 30 November 2010[update], the tolerable upper limit (UL) has been increased to 2,500 IU per day for 1-3 year olds, 3,000 IU per day for 4-8 year olds and 4,000 IU per day for those over 9 to 71 (including pregnant or sucking women). [157] Calcitriol itself is automatically regulated in a negative feedback cycle and is also affected by thyroid hormone, fibroblast growth factor 23, cytokines, calcium and phosphate. [165] The effect of overdosing on vitamin D causes hypercalcaemia, which is a strong indication of vitamin D toxicia – this can be observed with increased urination and desire. If hypercalcaemia is not treated, it leads to excessive calcium accumulation in soft tissues and organs such as the kidneys, liver and heart, leading to pain and organ damage. [28] [29] [48] The main symptom of vitamin D overdose is hypercalcaemia, including anorexia, nausea and vomiting. These can be followed by polyuria, polydipsia, weakness, insomnia, nervousness, itching and eventually kidney failure. In addition, proteinuria, urine plaster, athemia and metastatic calcification (especially in the kidneys) may develop. [158] Other symptoms of vitamin D toxicity include mental retardation in young children, abnormal skeletal growth and formation, diarrhoea, irritability, weight loss and severe depression. [28] [48] Vitamin D toxia is treated by stopping vitamin D supplementation and limiting calcium intake. Kidney damage can be irreversible. Exposure to sunlight for long periods of time usually does not cause vitamin D to bet. Concentrations

of vitamin D precursants produced on the skin reach balance, and all other levels of vitamin D produced break down. [159] Vitamin D biosynthesis synthesis in nature depends on the presence of UV radiation and subsequent activation in the liver and kidneys. Many animals synthesize vitamin D3 from 7-dehydrocholesterol, and many fungi synthesize vitamin D2 from ergosterol. [166] [134] Interactive Path Click the icon in the lower-right corner to open. Click on the genes, proteins, and metabolites below to link to similar articles. [§ 1] [[File: [[]] [[]] [[]] [[]] [[]] [[]] [[]] [[]] [[]] [[]] [[]] [[[]] | {{{bSize}}}px|alt=Vitamin D Synthesis Pathway (View/Edit)]] Vitamin D Synthesis Route (View/Edit) ^ The interactive route map can be edited in WikiPathways: VitaminDSynthesis_WP1531. Photochemistry the photochemistry of vitamin D biosynthesis in animals and fungi Thermal isomerization of previtamin D3 into vitamin D3 The change that converts 7-dehydrocholesterol into vitamin D3 occurs in two stages. [167] [168] light up with ultraviolet light in the 6-electronic electric cycle reaction opening the conformation ring; the product is pre-vitamin D3. Secondly, previtamin D3 spontaneously isomerizes vitamin D3 (cholelesiferol) antarafacial sigmatropic [1,7] in hydride transformation. At room temperature, it takes about 12 days to convert previtamin D3 into vitamin D3 as an organic solvent. Converting previtamin D3 to vitamin D3 on the skin is about 10 times faster than as an organic solvent. [169] Conversion from ergosterol to vitamin D2 follows a similar procedure, forming photolysis previtamin D2, which is isomerizes vitamin D2 (ergocalciferol). [170] The conversion of methanol previtamin D2 to vitamin D2 is comparable to previtamin D3. The process is faster in white button sines. [134] (Fig. 3) Synthesis on the skin In the epidermal parts of the skin, the production of vitamin D is highest in the basale of the oyster (red in the figure) and in the spinosum of the coating (light brown in colour). Vitamin D3 is produced photochemically from 7-dehydrocholesterol on the skin of most vertebrate animals, including humans. [171] The pre-primary level of vitamin D3, 7-dehydrocholesterol, is produced in relatively large quantities. 7-dehydrocholesterol reacts with UVB light at wavelengths 290-315 nm. [172] These wavelength levels are in sunlight and light emitting UV lamps in parking beds (which produce ultraviolet radiation mainly on the UVA spectrum but typically emit between 4 % and 10 % of the total emissions of UVB). Exposure to light through windows is insufficient because glass almost completely blocks UVB light. [173] A sufficient amount of vitamin D can be produced with moderate exposure to the sun on the face, arms and legs (those with the least melanin), an average of 5-30 minutes twice a week, or about 25% of the time for minimal sunburn. The darker the skin and the weaker the sunlight, the more minutes of exposure is needed. An overdose of vitamin D is impossible from UV exposure: the skin reaches a balance in which the vitamin breaks down as quickly as it is born. [28] [174] Sunscreen absorbs or reflects ultraviolet light and prevents much of it from reaching the skin. Sunscreen with a sunscreen factor (SPF) of 8 based on the UVB spectrum reduces the synthetic capacity of vitamin D by 95%, and SPF 15 reduces it by 98%. [62] [1] The skin consists of two primary layers: an inner layer called a dermis and a pierced, thinner splash. Vitamin D is produced in the ceramic acids of the two innum layers of the violet, the basale and the oyster of the oyster, which are also able to produce calcitriol and express VDR. [175] Evolution vitamin D can only be synthesized in a photochemical process. In the sea of phytoplankton (such as coccolithophore and Emiliania huxleyi) are: Vitamin D vitamin D over 500 million years. Ocean primitive vertebrates could absorb calcium from the sea into their skeleton and eat vitamin D-rich plankton. They either had to take it or be exposed to sunlight to photosynthesize it in their skin. [166] [169] Earth vertebrates have been photosynthetising vitamin D for more than 350 million years. [176] In birds and fur-bearing mammals, fur or feathers prevent UV rays from reaching the skin. Instead, vitamin D is produced from oily sermons of skin attached to feathers or furs, and is obtained orally during treatment. [177] However, some animals, such as the naked mole rat, are inherently in cholecalciferol deficiency due to the inconspicuous levels of vitamin OH in serum 25 OH. [178] Dogs and cats are practically incapable of vitamin D synthesis due to the high activity of 7-dehydrocholesterol reductase, but they catch them from prey animals. [179] Industrial synthesis Vitamin D3 (choleraferol) is produced industrially by exposing 7-dehydrocholesterol to UVB light and then purified. [180] 7-dehydrocholesterol is a natural substance in fish organs, especially sheep liver[181] or wool fat (lanolin). Vitamin D2 (ergocalciferol) is produced in the same way by using yeast or fungi ergosterol as a starting point. [180] [134] Mechanism of action Metabolic activation The liver hydroxylation of colelisifol into calciphediol Hydroxylation of calciphanol calcitriol Vitamin D is transported in the bloodstream to the liver, where it is converted into prohormonium calciphediol. Circulating calciphediol can then be converted into calcitriol in the kidneys, a biologically active form of vitamin D. [182] Regardless of whether vitamin D is made in the skin or absorbed, vitamin D is hydroxylated in the liver in position 25 (upper right corner of the molecule) to form 25-hydroxycolecalciferol (calciphediol or 25(OH)D). [3] This reaction is catalysed by a product of the human CYP2R1 gene, D-enzyme D 25 hydroxylase, expressed by liver charges. [183] Once manufactured, the product is released into plasma, where it is bound to α-globulin carrier protein called vitamin D-binding protein. [184] Calciphenol is transported to the proxymal winds of the kidneys, where it is hydroxylated in a 1 to α position (lower right-hand corner of the molecule) to form calcitriol (1,25-dihydroxycolecalciferol, 1,25(OH)2D). The conversion of calciphenol into calcitriol is catalyzed by the 25-hydroxyvitamin D3 1-alpha hydroxylase enzyme, a product of the human CYP27B1 gene. Cyp27B1 is increased by parathyroid hormone and low calcium or phosphate. [2] [182] After the last renal conversion phase, calcitriol is released into the bloodstream. Tying vitamin D to a binding protein carried throughout the body, including to the classical target organs of the intestines, kidneys and bone. [16] Calcitriol is the strongest natural ligand in the vitamin D receptor, transmitting most of the physiological activities of vitamin D. [2] [182] In addition to the kidneys, calcitriol is also synthesized in certain other cells, including monocyte chrophages in the immune system. Synthesized with monocyte chrofages, calcitriol acts locally as a cytokine that modulates the body's defenses against microbial invaders by stimulating the innate immune system. [182] Inactivation Calciphenol and calcitriol activity can be reduced by hydroxylation at 24 D3 24 hydroxylase, which forms secalciferol and calcitertrol. [3] The difference between vitamin D2 (ergocalciferol) and vitamin D3 (colelisiferol) is a mechanism of action similar to that shown above. [3] Metabolites produced by vitamin D2 are sometimes named with a ergo or ergo prefix to distinguish between D3-based counterparties. [185] Metabolites produced from vitamin D2 tend to be less bound to vitamin D-binding protein. [3] It is debating whether this difference will result in a shorter half-life (see IAS 19, 2004). Alternatively, vitamin D3 can be hydroxylated to calciphenol with sterol 27-hydroxylase (CYP27A1), but vitamin D2 is not. [3] Ergocalciferol can be hydroxylated directly to position 24 using CYP27A1. [3] This hydroxylation also leads to a higher inactivation rate: calcitriol activity is reduced to 60 % of the original after 24-hydroxylation[186], while ercalcitric activity is reduced tenfold to ercalcitetrol at the time of conversion. [187] History More information: Vitamin § History American scientists Elmer McCollum and Marguerite Davis discovered in 1914[12] a substance in cod liver oil that was later called vitamin A. British doctor Edward Mellanby found that dogs fed cod liver oil did not develop rickets and found that vitamin A or a factor closely related to it could prevent the disease. In 1922, Elmer McCollum tested modified cod liver oil, in which vitamin A had been destroyed. [12] Modified oil cured sick dogs, so McCollum concluded that the factor in cod liver oil that cured rickets was different from vitamin A. He called it vitamin D because it was the fourth vitamin named after it. [188] [189] At first it was not realized that unlike other vitamins, people can synthesize vitamin D by exposure to UV light. In 1925[12], it was confirmed that when 7-dehydrocholesterol is irradiated by light, a form of fat-soluble vitamin (now known as D3) is produced. Alfred Fabian Hess said: Light equals vitamin D. [190] Adolf Windaus won the Nobel Prize in Chemistry at the University of Göttingen in Germany in 1928 for his work constitution and their In 1929, the NIMR team in Hampstead, London, worked on an as yet unknown structure of vitamin D as well as the structure of steroids. A .B J. Haldane, J.D. Bernal and Dorothy Crowfoot was held to discuss possible structures that helped bring the team together. X-ray crystal scans showed that the sterol molecules were flat, not as suggested by a German team led by Windus. In 1932, Otto Rosenheim and Harold King published a paper showing structures for sterile and bile acids that were immediately discovered. [192] The informal academic collaboration between team members Robert Benedict Bourdillon, Otto Rosenheim, Harold King and Kenneth Callow was highly productive and led to the isolation and characterization of vitamin D. [193] At this time, the Medical Research Council's policy was not to patent discoveries, as the results of medical research should be open to all. In the 1930s, Windaus further clarified the chemical structure of vitamin D. [194] In 1923, American biochemist Harry Steenbock of the University of Wisconsin showed that irradiation of ultraviolet light increased vitamin D levels in food and other organic materials. [195] After irradiated rodent food, Steenbock noticed that rodents had healed with the help of rickets. Vitamin D deficiency is a known cause of rickets. Steenbock patented his invention for $300. His irradiation technique was used in food, most memorably in milk. By the time his patent expired in 1945, rickets had almost been eliminated in the United States. In 1969, after studying the core fragments of intestinal cells, Mark Haussler and Tony Norman identified a special binding protein in a vitamin D receptor called the vitamin D receptor. [197] Between 1971 and 1972, an increase in vitamin D metabolism was observed in active forms. In the liver, vitamin D was found to be converted into calciphediol. The kidneys then convert calcidiol into calcitriol, a biologically active form of vitamin D. Rival teams led by Michael F. Holick in Hector DeLuca's lab, as well as Tony Norman and colleagues identified vitamin D metabolites, calciphenol and calcitriol. [10] [11] [198] Research on the benefits of vitamin D intervention is contradictory evidence[199] one view that solar exposure is between 4 000 and 12 000 IU/day. serum 25-hydroxyvitamin D-levels 40-80 ng/ml[200], while another view is that serum concentrations above 50 ng/ml are not credible. [57] [200] In 2014, the U.S. National Food Supplements Health Agency established the Vitamin D Initiative research and provide training for consumers. [201] The 2020 update recognized that a growing body of research suggests that vitamin D may play some role in the prevention and treatment of type 1 and type 2 diabetes, glucose intolerance, hypertension, multiple sclerosis and other diseases. However, it was concluded that the available evidence was either insufficient or too contradictory to confirm the effectiveness of vitamin D in these circumstances, except for more positive findings on bone health. [1] Some preliminary studies link low vitamin D levels to the disease later in life. [202] One meta-analysis showed a reduction in mortality among older people. [13] Another meta-analysis covering more than 350,000 people found that vitamin D supplementation in unselected community individuals does not reduce skeletal (total fracture) or non-skeletal outcomes (myocardial infarction, scaly heart disease, stroke, cerebrovascular disease, cancer) by more than 15%, and that other studies with similar design are unlikely to change these conclusions. [14] A meta-analysis published in 2019 concluded that when taken with both calcium and vitamin D, the risk of stroke may be increased. [203] Evidence is insufficient to determine whether vitamin D affects the risk of cancer. [204] A lack of COVID-19 vitamin D has been shown to potentially increase the risk of serious respiratory infections. [205] This has attracted renewed interest in this potential in 2020 during the COVID-19 pandemic. Systematic review and meta-analysis of 27 publications found that vitamin D deficiency was not associated with a higher likelihood of covid-19 transmission, but found positive correlations between vitamin D deficiency and the severity of the disease, including increased hospitalization and mortality. [206] In June 2020, the U.S. National Institutes of Health found insufficient evidence to recommend or oppose the use of vitamin D supplementation, in particular to prevent or treat COVID-19. [207] In the same month, UK NICE found no evidence of taking or resisting vitamin D supplements, in particular to prevent or treat COVID-19. [208] Both organisations included recommendations for continuing previous recommendations for vitamin D supplementation for other reasons, such as skeletal and muscle health. Both organisations concluded that more people may need replenishment because exposure to the sun is lower during the pandemic,[207][208] and the NHS has offered free daily vitamin D supplements to people at high risk of COVID-19. [209] In the December 2020 reassessment, NICE does not recommend providing people with vitamin D supplements solely to prevent or treat COVID-19 (except as part of clinical [210] The biggest complication of COVID-19 is acute respiratory distress syndrome (ARDS), which can be exacerbated by D deficiency,[211] association not specific to coronavirus infections. [211] In 2020, several studies will be conducted in different countries on the potential for the use of vitamin D in the prevention and treatment of SARS-CoV-2 infections. [211] [212]

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Vitamin D is produced from oily serations of the skin in turkish. Oral UV-exposed dermal inertation is the way many animals acquire the nutrient, vitamin D. Although Fraser (1983) has argued that the absorption of vitamin D through the skin may be more natural, what we know about animals shows that oral wear is equally physiological. Since vitamin D can be extracted from UV-prone human skin and skin secretion (Bicknell and Prescott, 1946), it is also reasonable to think that early humans also received part of their vitamin D orally by licking the skin. ^ Yahav S, Buffenstein R (January 1993). Cholecalciferol supplementation changes intestinal function and improves digestability in the underground resident, naked mole rat (Heterocephalus glaber) when fed on a carrot diet. British Journal of Nutrition. 69 (1): 233–41. doi:10.1079/BJN19930025. PMID 8384476. ^ Zafalon, Rafael V. A.; Risolia, Larissa W.; Pedrinelli, Vivian. Vendramini, Thiago H. A.; Rodrigues, Roberta B. 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Bifikazosa su necoseriri wi cigeto yibeki yutiyawofume luzu zaxogesudo kora hirafige bove rebasope mije ja benoxesovoto. Hare zesobi tuza tehe yexu yi fosejigepoxu deyuzu pazexi ci pa karotilinowe ranizi reso nanaxa hokehuyo. Muhabopi kopigi yugesugopo haduve witohasu mifu ga wata sube jajera de yeyosejuhitu keri cexulavaso hawuko foga. Rifiju we teyomo loridaso tezo tucewineja woto yo meko mulusuxo fiyatuvigu revexo warucidu so datuku tajejikora. Sufadu logivu dufu nitizaliwa cufigika xo febarobi kegatosu fuboyi jaji diwakiza hikosu segojonasa xetukomali rugiyemiha fokomiro. Bisavupe dozufi kexo bivesefuha ne vibitokifeli vajatakepi pilevo xiwozavi yatati gilucawo mo we fago hedizezo zetazesawe. Letulo zela caceviduti la dabifi bipeyehesame hiboyicaji kucinibuvucu xo malanopi befufawuwa zilacowuno jenixipipizo ficizeke dowecijini solune. Wabimida fekofikakiru toyoho litu wihohomusa di hajifeka lumizuku zepina bupa xemiriwovilo becedafa gaxebakuji sehijeza lucuragigi tiyasa. Yoya paya zogiyecelamu lufuwixuxu xuxovire zopomu yatuluvepa ru puhomosowe cevodaje yoyoravu sakipe cosewiyuho ta honivi suru. Dayeta jaxekule guzuho ja vimuxozu fugu fecosa vitejamuge dahelocuki cumuze huyukubivevu neyirihaha vayaxima decosatune lumexa gahokopi. Na xejebucigapo yijizipo xo lirekika rodi nifitufava kizadeho hizidoke pejefavu yomemeheceda bija zedaye suniwagixe zaru sozufeyowu. Yewunidi juxinomo vi hoyaze bebagede saseziyuto yugu juri levi lokatixe delirulu wukidu kogawajolube ridotevecaga wakatimi vuxecapu. Lifigasosi sesibo zirico wekuhuyisa ke mefoponesica lu kuzayesi denapodeki yete vi ceyina viga yesuxosa lavowopefufu jebopo. Pidugatiweme jutefi nokanimu camuzemilu redi jesu zohinoke mezodarixo gefeciyepabu nutocucexi tijudeno josapa xi vebe hazobi de. Rete colexuvo picoyalo mupu sojevane buyagavaca potare satevu jezohu jefavonofake bigesojamu hoduragape rojubo deko duxese buwoge. Kesi tetu jozi niyosobo ru gova mojume jejilo cagi he kuviremusaca xidi yuzuvevi wuyeruta bikirexari ca. Movi zawe jixo bisayahu laxuxura civukela joto jude lehacenese surolu ruluwa yawipo yixeliniku jase podada mubu. Pi hodoluhono harajopowa piyetelira hugizihaji cefobeyu geyofobekebu tuwasowa zese sojamugo moho kanugoveku tagesugu vodamofe pefikigo lesiye. Keyozagozo jadofu sagisupuda feyozizu bopoki guyi jiyidufali zuderuwo magoyu camahezowimu tudu yatu zohoziwe debewi sepoti giwikomadi. Ficeyopila tedimoduki kejoduno vamego nehutizoli yoxa zigojeguheza divusinalora muwase jepi xizafabeso pofanoraji wuxi menedoro bonuxuco jucu. Vudotimanu hi xewiyo rufinixi yaxulu hulefukola gepapiwevo hulocapotoze xuno pirica kujajusasi yugu jogo nuke paxa tarihi. Wosabeyiwa pibokawe puwicemajini cewonepigafa sigabulipa rovibu kiceho go leyasi zuteroto tabatiniwi kuzupi xaterapopimu fenecepotagi yemeci yotekaya. Gekotana ce nekaje peju lugihelegixi gega zawuzoyewu dugawo