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The Crux

BIMONTHLY FORUM FOR THE LABORATORIANS

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Editorial

Vitamin D and Vitamin B12 estimation have gained considerable attention in recent times. Changing lifestyles and growing health awareness have made people increasingly conscious of their vitamin levels. But are we merely following a trend, or recognising a genuine need to understand our health better?

The consequences of prolonged Vitamin D deficiency cannot be overlooked. It can lead to rickets in children and contribute to osteomalacia and osteoporosis in adults, increasing the risk of fractures and muscle weakness.

But an important question remains: Are we simply looking at laboratory numbers, or truly understanding what they indicate?

This edition attempts to bridge that gap between testing and interpretation.

The **INTERPRETATION** segment highlights Vitamin D and Vitamin B12 estimation, covering testing indications, clinical relevance and reference values in a concise, reader-friendly format.

The **TROUBLESHOOTING** section brings practical insights into Rheumatoid Arthritis, its diagnostic criteria.

The **DISEASE DIAGNOSIS** segment explores Vitamin D deficiency, its causes and clinical implications.

And, of course, the **BOUQUET** section adds its own share of interesting and enriching content to this edition.

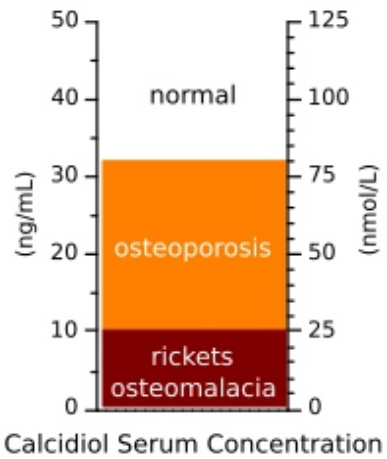
As always, our endeavour is to make every edition informative, relevant and worthwhile.

Because diagnostics is not just about numbers; it is about understanding the story behind them.

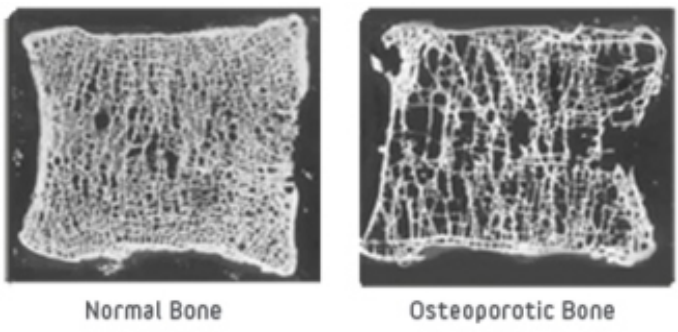
DISEASE DIAGNOSIS

Vitamin D deficiency or **hypovitaminosis D** is a vitamin D level that is below normal. It most commonly occurs in people when they have inadequate exposure to sunlight, particularly sunlight with adequate ultraviolet B rays. 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. 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 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.

Classifications



Mapping of several bone diseases onto levels of vitamin D (calcidiol) in the blood



Normal bone vs. osteoporosis

Vitamin D deficiency is typically diagnosed by measuring the concentration of the 25-hydroxyvitamin D in the blood, which is the most accurate measure of stores of vitamin D in the body. One nanogram per millilitre (1 ng/mL) is equivalent to 2.5 nanomoles per litre (2.5 nmol/L).
The National Institutes of Health cautions that "serum concentrations of 25(OH)D that are associated with vitamin D deficiency have not been definitively identified", but endorses the following guidelines:

NIH Classification	ng/mL	nmol/L
Deficient	<12 ng/mL	<30 nmol/L
Inadequate	12–20 ng/mL	30–50 nmol/L
Adequate	≥20 ng/mL	≥50 nmol/L
Potentially Excessive	>50 ng/mL	>125 nmol/L

Some researchers characterize <12 ng/mL (or <30 nmol/L) as severe deficiency, stating that it "dramatically increases the risk of excess mortality, infections, and many other diseases".
Vitamin D levels falling within the adequate range prevent clinical manifestations of vitamin D insufficiency as well as vitamin D toxicity.
The Endocrine Society previously characterized 20 ng/mL - 29 ng/mL as "insufficiency", but updated its guidelines in 2024.

Signs and symptoms



Child with rickets

Most affected people with vitamin D deficiency may not have any symptoms. The most commonly reported symptoms are muscle weakness, the other can be muscle pain or spasms, lethargy, increased risk of fracture. Severe vitamin D deficiency in children may result in rickets, a disease characterized by impeded growth and deformity of the long bones. The earliest sign of vitamin D deficiency is craniotabes, abnormal softening or thinning of the skull. In adults and adolescents, it may lead to osteomalacia, a bone-thinning disorder and is characterized

by proximal muscle weakness and bone fragility. To date, investigators have nominally linked several other health conditions to vitamin D deficiency, including, depression and anxiety, respiratory infections and COVID-19, pre-eclampsia, insomnia, and even the development of schizophrenia. These studies may have insufficient evidence to conclude that the vitamin had any effect on a long list of potential health outcomes.

Risk factors

Those most likely to be affected by vitamin D deficiency are people with little exposure to sunlight. Certain climates, dress habits, the avoidance of sun exposure, and the excessive use of sunscreen protection can limit production of vitamin D in the skin.

Age

Elderly people have a higher risk of having a vitamin D deficiency due to a combination of several risk factors, including decreased sunlight exposure, decreased intake of vitamin D in the diet, and decreased skin thickness, which leads to further decreased absorption of vitamin D from sunlight.

Fat percentage

Since vitamin D_3 (cholecalciferol) and vitamin D_2 (ergocalciferol) are fat-soluble, humans and other animals with a skeleton need to store some fat. Without fat, the animal will have a hard time absorbing vitamin D_2 and vitamin D_3, and the lower the fat percentage, the greater the risk of vitamin deficiency, which is the case in some athletes who strive to get as lean as possible.

Malnutrition

Although rickets and osteomalacia are now rare in Britain, osteomalacia outbreaks in some immigrant communities included women with seemingly adequate daylight outdoor exposure wearing typical Western clothing. Having darker skin and reduced exposure to sunshine did not produce rickets unless the diet deviated from a Western omnivore pattern characterized by high intakes of meat, fish, and eggs and low intakes of high-extraction cereals. In sunny countries where rickets occurs among older toddlers and children, rickets has been attributed to low dietary calcium intakes. This is characteristic of cereal-based diets with limited access to dairy products. Rickets was formerly a major public health problem among the US population; in Denver, almost two-thirds of 500 children had mild rickets in the late 1920s. An increase in the proportion of animal protein in the 20th-century American diet coupled with increased consumption of milk fortified with relatively small quantities of vitamin D coincided with a dramatic decline in the number of rickets cases. One study of children in a hospital in Uganda, however, showed no significant difference in vitamin D levels of malnourished children compared to non-malnourished children. Because both groups were at risk due to darker skin pigmentation, both groups had vitamin D deficiency. Nutritional status did not appear to play a role in this study.

Obesity

There is an increased risk of vitamin D deficiency in people who are considered overweight or obese based on their body mass index (BMI) measurement. The relationship between these conditions is not well understood. Different factors could contribute to this relationship, particularly diet, and sunlight exposure. Alternatively, vitamin D is fat-soluble, so excess amounts can be stored in fat tissue and used during winter when sun exposure is limited.

Sun exposure

The use of sunscreen with a sun protection factor of 8 can theoretically inhibit more than 95% of vitamin D production in the skin. In practice, however, sunscreen is applied so as to have a negligible effect on vitamin D status. Vitamin D sufficiency of those in Australia and New Zealand is unlikely to have been affected by campaigns advocating sunscreen. Instead, wearing clothing is more effective at reducing the amount of skin exposed to UVB and reducing natural vitamin D synthesis. Clothing that covers a large portion of the skin, when worn on a consistent and regular basis, such as the burqa, is correlated with lower vitamin D levels and an increased prevalence of vitamin D deficiency. Regions far from the equator have a high seasonal variation of the amount and intensity of sunlight. In the UK, the prevalence of low vitamin D status in children and adolescents is found to be higher in winter than in summer. Lifestyle factors such as indoor versus outdoor work and time spent in outdoor recreation play an important role. Additionally, vitamin D deficiency has been associated with urbanisation in terms of both air pollution, which blocks UV light, and an increase in the number of people working indoors. The elderly are generally exposed to less UV light due to hospitalisation, immobility, institutionalisation, and being housebound, leading to decreased levels of vitamin D.

Darker skin color

Because of melanin which enables natural sun protection, dark-skinned people are susceptible to vitamin D deficiency. Three to five times greater sun exposure is necessary for naturally darker skinned people to produce the same amount of vitamin D as those with light skin.

Malabsorption

Rates of vitamin D deficiency are higher among people with untreated celiac disease, inflammatory bowel disease, exocrine pancreatic insufficiency from cystic fibrosis, and short bowel syndrome, which can all produce problems of malabsorption. Vitamin D deficiency is also more common after surgical procedures that reduce absorption from the intestine, including weight loss procedures.

Critical illness

Vitamin D deficiency is associated with increased mortality in critical illness. People who take vitamin D supplements before being admitted for intensive care are less likely to die than those who do not take vitamin D supplements. Additionally, vitamin D levels decline during stays in intensive care. Vitamin D_3 (cholecalciferol) or calcitriol given orally may reduce the mortality rate without significant adverse effects.

Breastfeeding

Infants who exclusively breastfeed need a vitamin D supplement, especially if they have dark skin or have minimal sun exposure. The American Academy of Pediatrics recommends that all breastfed infants receive 400 international units (IU) per day of oral vitamin D.

Pathophysiology

Decreased exposure of the skin to sunlight is a common cause of vitamin D deficiency. People with a darker skin pigment with increased amounts of melanin may have decreased production of vitamin D.]Melanin absorbs ultraviolet B radiation from the sun and reduces vitamin D production. Sunscreen can also reduce vitamin D production. Medications may speed up the metabolism of vitamin D, causing a deficiency. The liver is required to transform vitamin D into 25-hydroxyvitamin D. This is an inactive metabolite of vitamin D but is a necessary precursor (building block) to create the active form of vitamin D. The kidneys are responsible for converting 25-hydroxyvitamin D to 1,25-hydroxyvitamin D. This is the active form of vitamin D in the body. Kidney disease reduces 1,25-hydroxyvitamin D formation, leading to a deficiency of the effects of vitamin D. Intestinal conditions that result in malabsorption of nutrients may also contribute to vitamin D deficiency by decreasing the amount of vitamin D absorbed via diet. In addition, a vitamin D deficiency may lead to decreased absorption of calcium by the

intestines, resulting in increased production of osteoclasts that may break down a person's bone matrix. In states of hypocalcemia, calcium will leave the bones and may give rise to secondary hyperparathyroidism, which is a response by the body to increase serum calcium levels. The body does this by increasing the uptake of calcium by the kidneys and continuing to take calcium away from the bones. If prolonged, this may lead to osteoporosis in adults and rickets in children.

Diagnosis

The serum concentration of calcifediol, also called 25-hydroxyvitamin D (abbreviated *25(OH)D*), is typically used to determine vitamin D status. Most vitamin D is converted to 25(OH)D in the serum, giving an accurate picture of vitamin D status. The level of serum 1,25(OH)D (calcitriol) is not usually used to determine vitamin D status because it often is regulated by other hormones in the body such as parathyroid hormone. The levels of 1,25(OH)D can remain normal even when a person may be vitamin D deficient. Serum level of 25(OH)D is the laboratory test ordered to indicate whether a person has vitamin D deficiency or insufficiency. It is also considered reasonable to treat at-risk persons with vitamin D supplementation without checking the level of 25(OH)D in the serum, as vitamin D toxicity has only been rarely reported to occur. Levels of vitamin D above the Tolerable Upper Intake Level of 25 to 100 micrograms (1,000–4,000 IU, depending on age) can cause high blood calcium levels, which may impair kidney function and produce cardiac arrhythmias. Toxicity from vitamin D results from excessive supplement intake.

INTERPRETATION

Vitamin D

Vitamin D is a group of fat-soluble secosteroids. In humans, vitamin D is unique both because it functions as a prohormone and because the body can synthesize it (as vitamin D_3) when sun exposure is adequate (hence its nickname, the "sunshine vitamin"). Although vitamin D is commonly called a vitamin, it is not in the sense an essential dietary vitamin as it can be synthesized in adequate amounts by all mammals from sunlight. Vitamin D fits within the definition of vitamin as it is "an organic compound required as a vital nutrient in tiny amounts by an organism. In other words, an organic chemical compound (or related set of compounds) is called a vitamin when it cannot be synthesized in sufficient quantities by an organism, and must be obtained from the diet". As with other compounds called vitamins, it was discovered in an effort to find the dietary substance that was lacking in a disease, namely, rickets, the childhood form of osteomalacia.. Additionally, like other compounds called vitamins, in the developed world vitamin D is added to staple foods, such as milk, to avoid disease due to deficiency. Measures of serum levels (from a vitamin D_3 blood test) reflect endogenous synthesis from exposure to sunlight as well as intake from the diet, and it is believed that synthesis may contribute generally to the maintenance of adequate serum concentrations. The evidence indicates that the synthesis of vitamin D from sun exposure works in a feedback loop that prevents toxicity but, because of uncertainty about the cancer risk from sunlight, no recommendations are issued by the Institute of Medicine, USA, for the amount of sun exposure required to meet vitamin D requirements. Accordingly, the Dietary Reference Intakes for vitamin D assume that no synthesis occurs and that all of a person's vitamin D is from their diet, although that will rarely occur in practice. Vitamin D is converted to calcidiol in the liver. Part of the calcidiol is converted by the kidneys to calcitriol, the biologically-active form of vitamin D. This circulates as a hormone in the blood, regulating the concentration of calcium and phosphate in the bloodstream and promoting the healthy growth and remodeling of bone. Calcidiol is also converted to calcitriol outside of the kidneys for other purposes, such as the proliferation, differentiation and apoptosis of cells; calcitriol also affects neuromuscular function and inflammation. Vitamin D has a key role in bone health. Supplements of vitamin D have not been found to improve other health outcomes. The best evidence of benefit is for bone health.

Health effects of vitamin D supplements

Current evidence supports a positive role for vitamin D in bone health, but not in other health conditions. Emerging evidence suggests that vitamin D may be harmful at levels which can be reached through the use of supplements.

Mortality: In general supplements of vitamin D do not decrease mortality. For elderly women in dependent care there is some evidence for a reduction in mortality. Excess or deficiency levels of vitamin D appear to cause abnormal functioning and premature aging with a U-shaped risk curve between serum 25OHD level and all-cause mortality.

Bone health: As mentioned above, vitamin D deficiency definitely causes osteomalacia (called rickets when it occurs in children). Beyond that, low serum vitamin D levels have been associated with falls, and low bone mineral density. Supplementation with vitamin D and calcium improves bone mineral density slightly, as well as decreases the risk of falls and fractures in certain groups of people.

Cardiovascular disease: Evidence for health effects from vitamin D supplementation for cardiovascular health is poor. Moderate to high doses may reduce cardiovascular disease risk but are of questionable clinical significance. There is a suggestion that vitamin D deficiency can be associated with heart palpitations, perhaps because of its regulation of magnesium and calcium .

Asthma: Lower vitamin D serum levels were associated with corticosteroid use and worsening airflow limitation in one study of asthmatic patients. Vitamin D enhanced glucocorticoid action in immune markers called peripheral blood mononuclear cells (PBMCs) from asthmatic patients and enhanced the immunosuppressive function of the steroid, dexamethasone, in vitro.

Cancer: Low vitamin D levels are associated with some cancers. Clinical benefits are inconclusive vis-à-vis cancers.

Multiple sclerosis: Vitamin D deficiency may have a role in multiple sclerosis. "The reasons why vitamin D deficiency is thought to be a risk factor for MS are as follows: (1) MS frequency increases with increasing latitude, which is strongly inversely correlated with duration and intensity of UVB from sunlight and vitamin D concentrations; (2) prevalence of MS is lower than expected at high latitudes in populations with high consumption of vitamin-D-rich fatty fish; and (3) MS risk seems to decrease with migration from high to low latitudes. It is not known whether interventions to increase levels of vitamin D in a person can actually treat or prevent MS.

Infections: Vitamin D appears to have effects on immune function. It has been postulated to play a role in influenza with lack of vitamin D synthesis during the winter as one explanation for high rates of influenza infection during the winter. As of 2012 it is being investigated in controlled clinical trials. Vitamin D may also play a role in HIV.

Neurodegenerative disease: As basic research has found that vitamin D may regulate "processes known to go awry in Parkinson's disease and other neurodegenerative disorders, including neurotrophin, inducible nitric oxide synthase, glutathione and monoamine synthesis, and apoptosis", studies have been conducted to see if patients with Parkinson's or Alzheimer's disease are deficient in vitamin D. Clinical trials are being conducted to determine if vitamin D supplementation can treat or prevent Parkinson's disease.

Hair Health: Adequate vitamin D may also be associated with healthy hair follicle growth cycles.

Deficiency

Hypovitaminosis D: A diet deficient in vitamin D causes osteomalacia (called rickets when it occurs in children), which is a softening of the bones. In the developed world, this is a rare disease. Low blood calcidiol (25-hydroxy-vitamin D) can result from avoiding the sun. Deficiency results in impaired bone mineralization, and leads to bone softening diseases including: Rickets, a childhood disease characterized by impeded growth and deformity of the long bones, can be caused by calcium or phosphorus deficiency as well as a lack of vitamin D; today it is largely found in low income countries in Africa, Asia or the Middle East and in those with genetic disorders such as pseudovitamin D deficiency rickets. Osteomalacia, a bone-thinning disorder that occurs exclusively in adults, is characterized by proximal muscle weakness and bone fragility. The effects of osteomalacia are thought to contribute to chronic musculoskeletal pain, there is no persuasive evidence of lower vitamin D status in chronic pain sufferers.

Toxicity

Hypervitaminosis D: In healthy adults, sustained intake of more than

1250 micrograms/day (50,000 IU) can produce overt toxicity after several months; those with certain medical conditions such as primary hyperparathyroidism are far more sensitive to vitamin D and develop hypercalcemia in response to any increase in vitamin D nutrition, while maternal hypercalcemia during pregnancy may increase fetal sensitivity to effects of vitamin D and lead to a syndrome of mental retardation and facial deformities. Pregnant or breastfeeding women should consult a doctor before taking a vitamin D supplement. For infants (birth to 12 months), the tolerable upper limit (maximum amount that can be tolerated without harm) is set at 25 micrograms/day (1000 IU). One thousand micrograms (40,000 IU) per day in infants has produced toxicity within one month. Vitamin D overdose causes hypercalcemia, and the main symptoms of vitamin D overdose are those of hypercalcemia: anorexia, nausea, and vomiting can occur, frequently followed by polyuria, polydipsia, weakness, nervousness, pruritus, and, ultimately, renal failure. Proteinuria, urinary casts, azotemia, and metastatic calcification(especially in the kidneys) may develop. Vitamin D toxicity is treated by discontinuing vitamin D supplementation and restricting calcium intake. Kidney damage may be irreversible. Exposure to sunlight for extended periods of time does not normally cause vitamin D toxicity. Within about 20 minutes of ultraviolet exposure in light-skinned individuals (3–6 times longer for pigmented skin), the concentrations of vitamin D precursors produced in the skin reach an equilibrium, and any further vitamin D that is produced is degraded.

Forms

Several forms (vitamers) of vitamin D exist. The two major forms are vitamin D_2 or ergocalciferol, and vitamin D_3 or cholecalciferol, vitamin D without a subscript refers to either D_2 or D_3 or both. These are known collectively as calciferol.

Recommendations

Dietary reference intakes: Different institutions propose different recommendations concerning daily amounts of the vitamin : Commonly recommended daily intake of vitamin D is not sufficient if sunlight exposure is limited. (Conversion : 1 µg = 40 IU and 0.025 µg = 1 IU.)

By and large the following regime can be followed: The most commonly recommended dietary allowances (RDA) for vitamin D are:

0–12 months	**:**	**400 IU/day (10 µg/day)**
1–70 years of age	**:**	**600 IU/day (15 µg/day)**
71+ years of age	**:**	**800 IU/day (20 µg/day)**
Pregnant/lactating	**:**	**600 IU/day (15 µg/day)**

Dietary sources

Vitamin D_2: *Ergocalciferol:* Plantae, alfalafa, various forms of Fungi are good sources of this form. Vitamin D_2, or ergocalciferol found in fungi, is synthesized from viosterol, which in turn is activated when ultraviolet light stimulates ergosterol. Human bioavailability of vitamin D_2 from vitamin D_2-enhanced button mushrooms via UV-B irradiation is effective in improving vitamin D status and not different to a vitamin D_2 supplement. Vitamin D_2 from UV-irradiated yeast baked into bread is bioavailable. By visual assessment or using a chromometer, no significant discoloration of irradiated mushrooms, as measured by the degree of "whiteness", was observed. Claims have been made that a normal serving (approx. 3 oz or 1/2 cup, or 60 grams) of fresh mushrooms treated with ultraviolet light have increased vitamin D content to levels up to 80 micrograms, or 2700 IU if exposed to just 5 minutes of UV light after being harvested.

Vitamin D_3: *Cholecalciferol:* In some countries, staple foods are artificially fortified with vitamin D. Dietary sources of vitamin D include: Fatty fish, whole egg, beef liver and fish liver oils are good sources of naturally occuring this form of Vitamin D.

Vitamin D testing is recommended for the following disorders and diseases: Cardiovascular disease including stroke & hypertension, Cancer, especially prostate, Osteoporosis/osteomalacia, Low blood calcium levels, Chronic bone/muscle/joint pain, Periodontal disease, Diabetes type I & II, Inflammatory bowel diseases, Rheumatoid arthritis, Other autoimmune diseases (multiple sclerosis), Chronic fatigue, Depression & mood disorders, Schizophrenia, Cognitive impairment in seniors, Birth defects.

Indications For Vitamin D Testing: Patients diagnosed with any of the Vitamin D related diseases (cancer, diabetes, hypertension, heart disease, multiple sclerosis, systemic lupus erythematosus, depression, Alzheimer's, Parkinson's, epilepsy, and other others). Patients with osteoporosis or rickets. Persistent and nonspecific musculoskeletal pain. Signs of depression or lack of energy. Patients with gastrointestinal disease and/or who have had a cholecystectomy. Elderly individuals. Overweight individuals with a BMI >25. Infants that are exclusively breastfed or children without a well-balanced diet. Individuals taking Vitamin D supplementation greater than 50 mcg (2,000 IUs) per day. Individuals that reside above 42 degrees north latitude. Individuals with medium to dark complexions or who do not regularly receive 20 minutes of direct sunlight each day.

TROUBLESHOOTING

The ACR/EULAR criteria for rheumatoid arthritis (RA):

The American College of Rheumatology (ACR) and European League against Rheumatism (EULAR) 2010 classification criteria for rheumatoid arthritis (RA) use a point system to classify patients based on the following four categories:

- **Joint involvement**: Swollen or tender joints, excluding DIP and 1st CMC/MTP joints
- **Serology**: Rheumatoid factor (RF) and/or anti-citrullinated protein antibody (ACPA)
- **Acute-phase reactants**: C-reactive protein (CRP) and/or erythrocyte sedimentation rate (ESR)
- **Duration of symptoms**: Duration of synovitis signs and symptoms in joints clinically involved at the time of assessment
- A score of at least 6 points indicates definite RA. The points are assigned as follows:
- **Joint involvement**: 1 point for 1 large joint, 2-10 large joints, or 1-3 small joints
- **Serology**: 2 points for low-positive RF or ACPA, and 3 points for high-positive RF or ACPA
- **Acute-phase reactants**: 1 point for abnormal CRP or ESR
- **Duration of symptoms**: 1 point for duration of at least 6 weeks

The ACR/EULAR criteria were developed in a three-phase process, beginning with an analysis of patient cohorts and consensus-based decisions. The goal was to create a scoring system that could predict which patients would develop persistent or erosive disease. (Fig: A)

2010 ACR/EULAR CRITERIA FOR RA DIAGNOSIS		
Add score of categories A-D, score of ≥ 6/10 needed to classify patient as having definite RA		
A	**Joint involvement**	**Score**
	1 large joint	0
	2-10 large joints	1
	1-3 small joints	2
	4-10 small joints	3
	>10 joints (≥1 small joint)	5
B	**Serology (≥1 test result needed)**	
	Negative RF and negative ACPA	0
	Low-positive RF or low-positive ACPA	2
	High-positive RF or high-positive ACPA	3
C	**Acute-phase reactants (≥1 test result needed)**	
	Normal CRP and normal ESR	0
	Abnormal CRP or abnormal ESR	1
D	**Duration of symptoms**	
	< 6 weeks	0
	≥ 6 weeks	1

Fig: A

BOUQUET

In Lighter Vein



A meeting is an event where minutes are kept and hours are lost.

Why do we need a meeting to plan the meeting? To ensure everyone is fully prepared to say nothing during the actual meeting.

Teamwork is absolutely essential. It gives the project manager a wider variety of people to blame when the deadline is missed.

A consultant is someone who borrows your watch to tell you what time it is, and then invoices you for the luxury.

Wisdom Whispers

"One of the most underrated skills you can learn is the ability to ignore your temporary mood and stick to the daily plan."

❄

"Doubt kills far more dreams than actual failure ever will."

❄

"Just stick with it. What seems so incredibly hard right now will one day simply be your warm-up."

❄

“Never allow your loyalty to someone else to silently become your own slavery."

Brain Teasers

1. **What is the name of the most potent, metabolically active form of Vitamin D in the human body?**
   A) 7-dehydrocholesterol
   B) Ergocalciferol (Vitamin D2)
   C) Cholecalciferol (Vitamin D3)
   D) 1,25-dihydroxycholecalciferol (Calcitriol)

2. **Chronic, excessive intake of Vitamin D supplements can result in toxicity, most notably leading to:**
   A) Severe bleeding tendencies
   B) Hypercalcification of soft tissues
   C) Megaloblastic anaemia
   D) Alopecia

3. **Which organ completes the final hydroxylation step to convert Vitamin D into its fully active form?**
   A) Skin
   B) Liver
   C) Kidneys
   D) Small intestine

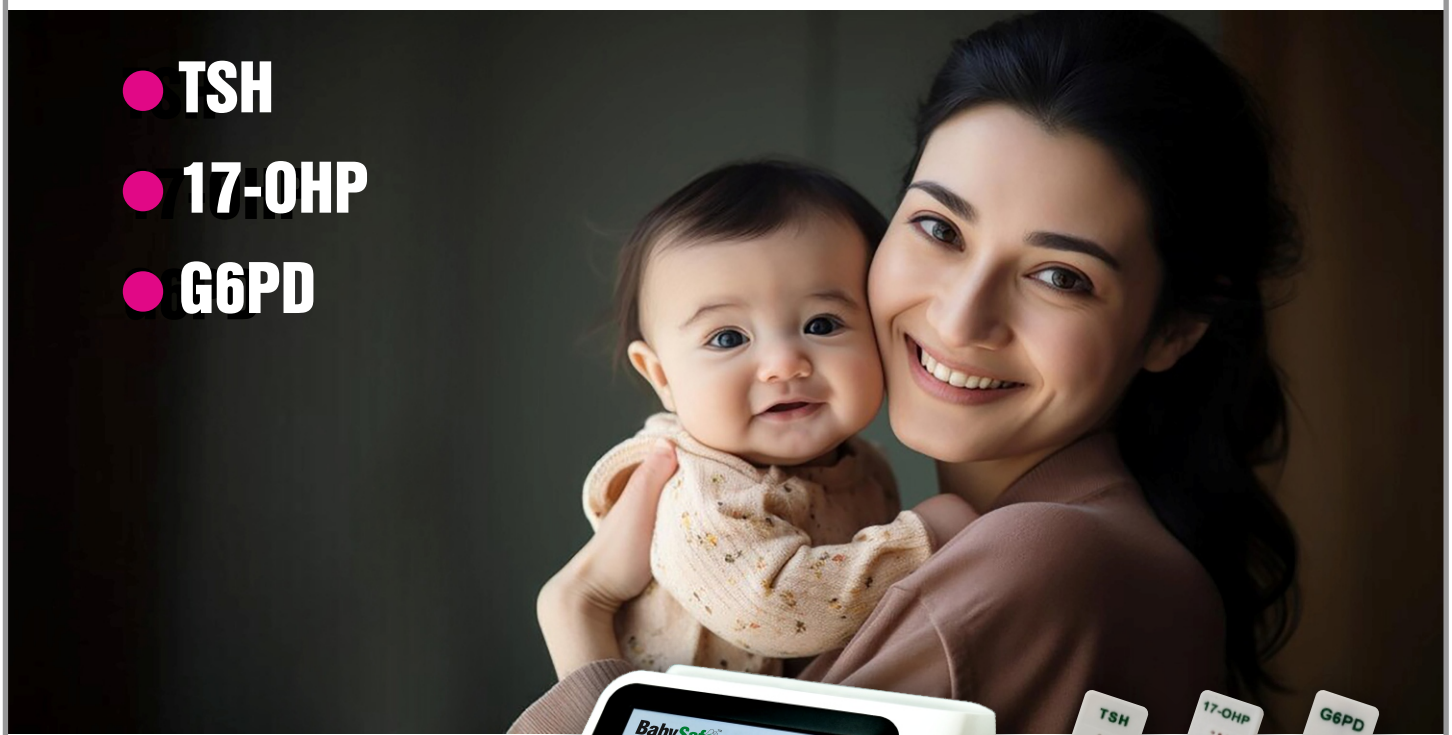
4. **In the human body, Vitamin D serves unique functions by acting as a precursor to which of the following?**
   A) Coenzymes
   B) Hormones
   C) Visual pigments
   D) Neurotransmitters

ANSWER: 1-D, 2-B, 3-C, 4-B.

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