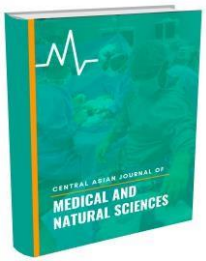

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Article

Activity of Superoxide Dismutase in Patients with Vitamin D3 Deficiency

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Abstract: Vitamin D, also known as vit D, is an important fat-soluble vitamin and steroid hormone that plays a crucial role in maintaining bone health and regulating calcium levels. In addition to its effects on the skeletal system, vit D also serves various functions in different target tissues, including the immune system, endocrine cells, heart, blood vessels, and pancreas. It exerts control over a significant portion of our genetic makeup, specifically about three percent of the human genome, which is involved in managing cell differentiation and the cell cycle. One member of the antioxidant enzyme family, known as superoxide dismutase (SOD), plays a vital role in regulating the production of reactive oxygen species (ROS) by facilitating the conversion of the enzyme into molecular oxygen and hydrogen peroxide. SOD is classified as a metalloenzyme, meaning it requires a metal cofactor to carry out its functions. The specific type of metal ion that SOD relies on determines its particular form. The primary isoforms of SOD that bind to metal ions and scavenge superoxide are iron (Fe), zinc (Zn), copper (Cu), and manganese (Mn). These metals present within the isoforms help differentiate and identify them. The three isoforms that catalyze the same interaction are named extracellular SOD3 (EC-SOD), cytosolic Cu-Zn-SOD (SOD1), and mitochondrial Mn-SOD (SOD2). The specific isoform of the proteins is determined by their subcellular locations and genes. Between July 21, 2022, and February 14, 2023, a collection of sixty samples was obtained, encompassing individuals aged 20 to 40. Using the device technique, the Mini Vidas were examined, showcasing its remarkable speed and accuracy in delivering results and its efficient utilization of elfa for early cancer detection. The study outcomes revealed that the D3 ratio ranged from 9.18 to 31.4 for men and 22.95 to 38.74 for women. Females exhibited a SOD ratio of 11.2 to 189.6, while males had a range of 3.44 to 190. Consequently, it can be inferred that women possess higher levels of vitamin D compared to males, despite males having higher levels of superoxide dismutase. The elevated proportion of vitamin D in females can be attributed to weight gain.

Keywords: vitamin D, superoxide dismutase, fat-soluble vitamin

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1. Introduction

Vitamin D

In addition to its functions in various non-skeletal target tissues, such as the immune system, heart, muscles, and adipose tissues, vitamin D plays a dual role as a steroid hormone and fat-soluble vitamin: it is essential for the regulation of calcium homeostasis and bone health. Moreover, it exerts control over a large number of genes responsible for cell differentiation and cycle regulation— accounting for about 3% of the human genome [1].

Sun exposure remains the primary source of vitamin D. Two available forms include cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2); ergocalciferol binds to the vitamin D-binding protein in human plasma, transports it to the liver where hydroxylation takes place to form 25-hydroxyvitamin D [2]. On the other hand, cholecalciferol is produced by human skin from 7-dihydrocholesterol (or 7DHC). UV light — an ability to swiftly transform 7-DHC into provitamin D3 [3]. The two main sources of ergocalciferol (vitamin D) are fish fat (cod) and green vegetables alongside mushrooms physiologically inactive; thus, vitamin D is obtained from these food sources or through exposure of the skin to sunlight. Transformation takes place at the liver and kidney hydroxylase enzyme level (13), where it converts into its active form known as 1,25 dihydroxy vitamin D (calcitriol). Adolf Windaus received the Nobel Prize in Chemistry for his work on sterols' structure and their interactions with vitamins— he presented vitamin D [4] as the first vitamin for which he was awarded a prize [5].

Vitamin D acts like a hormone since it can be acquired through diet or synthesized in the skin after exposure to sunlight via 7-dehydrocholesterol's natural production, acting through nuclear receptors (VDR) found not only in specific tissues but widely throughout the body [6], making it categorized as hormone-like. A link between vitamin deficiencies and obesity has been established by a few studies [7]. In terms of vitamin D levels, less than 20 ng/ml is considered inadequate, 21–29 ng/ml is considered insufficient, and anything above 30 ng/ml is deemed optimal [8].

Source of Vitamin D

The main source of vitamin D is exposure to sunlight, mainly UV radiation (290-315 nm) which is produced naturally by the body when it comes into contact with sunlight [9]. Vitamin D3 can be found in large quantities in fish and fish products, especially salmon. In addition to natural food sources, vitamin D is added to various foods including morning cereals, plant-based milk alternatives and cow's milk in many countries [10]. However, there are only a few natural food sources for vitamin D such as meat, eggs, fatty fish and whole dairy products [11]. It is recommended that individuals spend 10 to 20 minutes under the sun depending on the season, [12] latitude and skin tone as this helps to ensure adequate vitamin D production within the body; one reason people may not get enough sun exposure is spending long hours indoors due to work [13]. There are various factors that can lead to vitamin D insufficiency [14]: low intake of vitamin D-rich foods like milk and fortified foods, [15] reluctance towards high-fat diets that result in reduced vitamin D consumption, use of sunscreen, and lack of sun exposure [16].

Metabolism of Vitamin D

Vitamin D, a steroid hormone, acts through the vitamin D receptor (VDR) that controls the musculoskeletal system— playing a role in various biological processes [17]. The two primary forms are: D2 (ergocalciferol), synthesized when ultraviolet radiation is absorbed by ergosterol in plants and fungi; and D3 (cholecalciferol), produced upon UV radiation absorption by 7-dehydrocholesterol in the skin [18]. Vitamin D binding protein (DBP), a liver-secreted serum glycoprotein, is mainly responsible for ferrying vitamin D into circulation [19]. Vitamin D itself is physiologically inert and hence hydroxylated with 25 hydroxy vitamin D (25(OH)D3) in the liver— as this particular isoform stands as the major metabolite of vitamin D, providing a more accurate reflection of the vitamin's quantity from all sources [19]; its half-life lingers at just twenty hours.

The ligand 1, 25 [OH]2D binds to Vitamin D Receptors (VDRs) in high affinity and thus helps increase calcium and phosphorus absorption in intestines. Vitamin D takes part in bone formation, bone resorption, bone mineralization, and neuro-muscular functions through actions on VDRs located in osteoblasts. Vitamin releases bio-chemical mediators

via the VDR which further lead to regulation of bone metabolism [21]. The presence of VDR has been noted in nearly all cells and tissues among humans [22]. This includes more than 3% control under genes for glucose metabolism as it falls within the scope of those genes that are necessary [23].

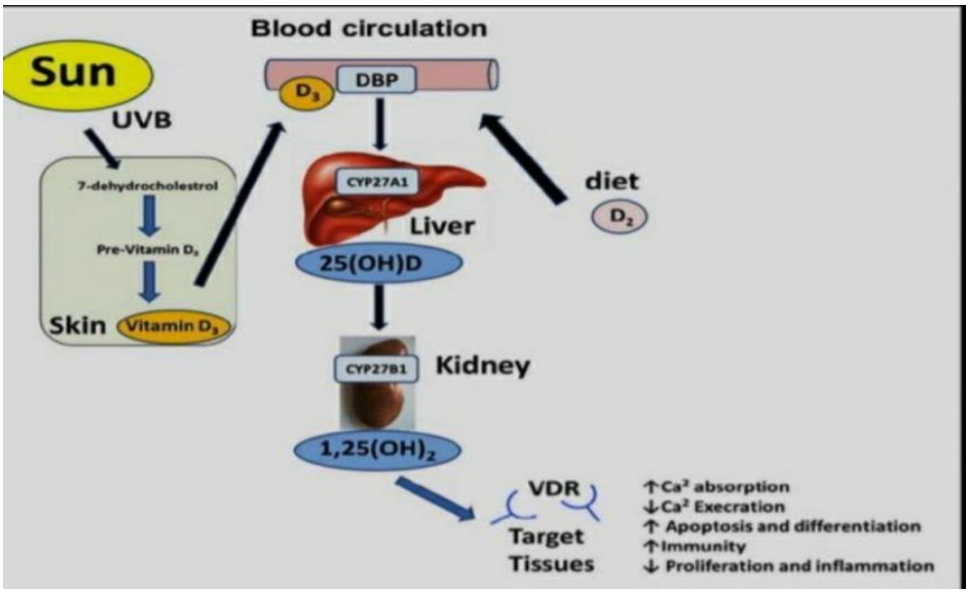


Figure 1. The metabolism of vitamin D [24]

The VDR gene in humans has been associated with a range of health conditions, including multiple sclerosis, osteoporosis, cancer, cardiovascular disease, diabetes, Parkinson's disease, and various autoimmune disorders. Furthermore, these genetic variations have a direct effect on the biological function of vitamin D [25].

Functions of Vitamin D in the body

Cell division, proliferation, and death are all processes in which vitamin D, an important steroid hormone, plays a crucial role [26]. Studies have demonstrated that vitamin D can increase clinical pregnancy rates and rates of patient implantation [27]. Recent research has also shown a connection between vitamin D3 and various diseases. In addition to conditions like rickets and osteomalacia, vitamin D deficiency can also impact non-structural illnesses such as cancer, cardiovascular disease, immunological diseases, and pregnancy complications. It is worth noting that every cell in the body contains the vitamin D receptor (VDR) [28].

A deficiency in vitamin D has been associated with a range of health issues including heart disease, infections, autoimmune diseases, cancer, obesity, osteoporosis, rheumatoid arthritis, inflammatory bowel disease, and type 1 diabetes [29]. Furthermore, lower levels of vitamin D have been linked to autoimmune thyroid disease [30]. Recent studies have shown that vitamin D plays a role in both innate and adaptive immunity, as well as in the development of cancer and autoimmune diseases. Moreover, research has found a connection between vitamin D deficiency, thyroid cancer, and autoimmune thyroid disorders [31]. Numerous studies conducted on humans and animals have linked vitamin D insufficiency to musculoskeletal diseases, male hypogonadism, type 1 and type 2 diabetes, cancer, autism, dementia, and cardiovascular disease. Research conducted in laboratory settings has also established a connection between insufficient levels of vitamin D and the development of hirsutism, obesity, irregular menstruation, and hyperandrogenism [32].

Additionally, in the realm of male reproduction, there exists a correlation between vitamin D and testosterone levels, as well as sperm motility and quality [33]. Notably,

males with severe vitamin D deficiency (10 ng/mL) exhibited a decrease in the occurrence of motile sperm [34].

Vitamin D plays a vital role in neurological development and brain function, and a deficiency has been associated with schizophrenia [35]. Furthermore, vitamin D has been found to contribute to the prevention of coronavirus disease (COVID-19) infection, as it has demonstrated protective effects against severe respiratory infections. Studies have shown a correlation between vitamin D levels and COVID-19 cases, particularly in terms of mortality rates. Older individuals, who often have low levels of vitamin D, are particularly susceptible to COVID-19 infection [36]. It also plays a crucial role in strengthening the immune system [37]. Additionally, vitamin D levels have been linked to psoriasis and other skin conditions [38]. In terms of bone health, vitamin D is implicated in skeletal hypertrophy, primary and secondary osteoporosis, rickets, and myofascial pain [39].

Superoxide Dismutase and Its Isozymes

Superoxide dismutase (SOD) is a crucial intracellular antioxidant enzyme (EC 1.15.1.1) [34] that plays a significant role in protecting cells against superoxide and peroxynitrite. SOD belongs to a family of oxidoreductases that facilitate the conversion of superoxide to hydrogen peroxide through oxygen dismutation. This enzyme is classified as a metalloenzyme, requiring a metal cofactor for its proper functioning [39]. The specific metal ion supplied by SOD determines the different forms of the enzyme. Typically, SOD binds to iron (Fe), zinc (Zn), copper (Cu), or manganese (Mn) ions (Figure 3). In mammals, there are three primary isoforms of SOD, each characterized by the metal they contain and their ability to degrade superoxide [41]. These isoforms are known as extracellular SOD3 (EC-SOD), cytosolic Cu-Zn-SOD (SOD1), and mitochondrial Mn-SOD (SOD2). Although originating from separate genes and subcellular locations, all three isoforms catalyze the same action [42]. The main intracellular SOD, SOD1, exists as a dimer with a molecular mass of 32 kDa [43].

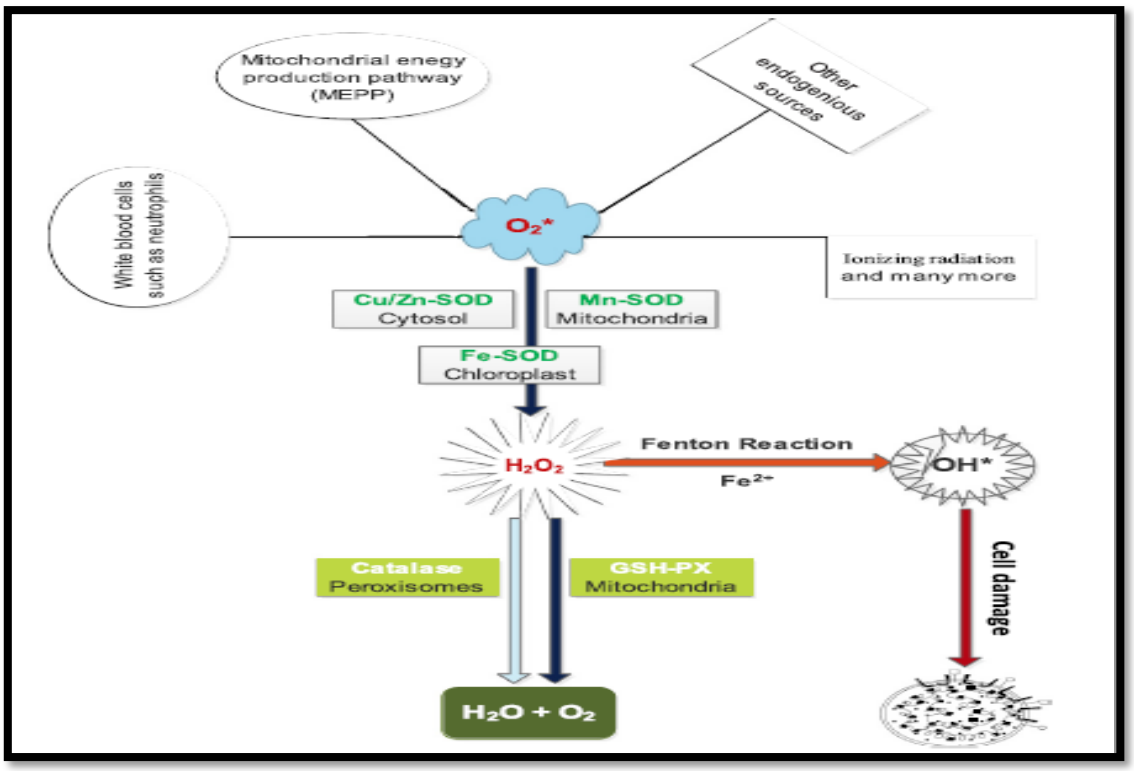


Figure 2. Sources of superoxide anion and types of SOD [44]

The primary antioxidant enzyme, which can be found in every part of a cell, accounts for 80% of the overall SOD activity [45]. It is abundantly present in the cytoplasm of all mammalian cells, and to a lesser extent in the gaps of the inner cell membrane in the nucleus, lysosomes, mitochondria, and peroxisomes [46].

Within the mitochondrial matrix, an enzyme called SOD2 (Mn-SOD) operates with the assistance of manganese (Mn) [47]. Transported from the cytosol to the mitochondria by a signal peptide, this 96 kDa tetramer dismutates superoxide generated by the respiratory chain. The active regions of SOD2, through the process of Mn transformation, convert superoxide into oxygen and water (46). In the extracellular region of the vasculature, the homotetramer SOD3, also known as extracellular SOD, weighs 135 kDa and is widely recognized [48]. Its primary location is the extracellular matrix (ECM) and the surfaces of cells containing extracellular fluids and plasma fraction (142). Iron-SOD-containing enzymes are abundant in plants, predominantly in chloroplasts and increasingly in peroxisomes and mitochondria. These SODs are believed to be the oldest in their class [49] (Figure 3). However, a lack of regulation can result in various forms of cellular damage [50].

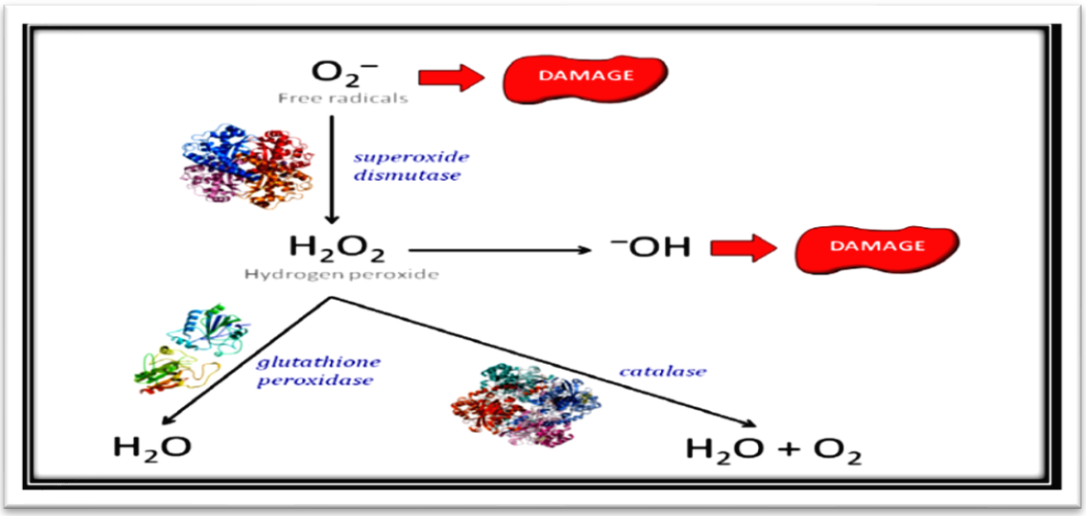


Figure 3. Mechanism action of superoxide dismutase and the fate of its end product [15]

2. Materials and Methods

Equipment

This experiment employed standard laboratory materials and equipment, all of which are of analytical quality and they get them from the companies indicated in Table (1, 2):

Table 1. List of Laboratory Equipment

Equipment	Company	Origin
Electronic Sensitive balance	Sartorius	Germany
Water bath	Memmert	Germany
Centrifuge	Hettich	Germany
Incubator	Memmert	Germany
Micropipettes	Hettich	Germany

Spectrophotometer		Germany
pH meter	Hanna	Romania
Timer with alarm	Hettich	Germany
Refrigerator (deep freeze)	National	Japan

Chemicals

Table 2. Chemical used in the study

Chemicals	Company	Origin
Dipotassium Hydrogen Orthophosphate. Trihydrate ($K_2HPO_4.3H_2O$)	BDH	England
Ethylenediaminetetraacetic acid disodium (EDTA-2Na)	BDH	England
Potassium dihydrogen Orthophosphate .Trihydrate ($K_2HPO_4.3H2O$)	BDH	England
Nitro blue tetrazolium (NBT-2HCl)	Sigma	USA
Triton x-100	Fluka	Switzerland
Riboflavin	BDH	England
Potassium Cyanide(KCN)	BDH	England
L-Methionine	Fluka	Switzerland

Sampling

Samples were collected from individuals, both those who were ill and those who were healthy, during the early morning hours between 8.30 and 11:00 A.M. A blood sample, ranging from five to ten milliliters, was obtained from each participant, including both control and patient groups. Once the blood had clotted for approximately ten minutes at a temperature of 25°C, it was separated using a gel tube and subjected to centrifugation at 3000 rpm for a duration of ten minutes. Subsequently, the resulting serum was divided into three Eppendorff tubes and stored in a deep freezer at a temperature of -20°C.

Measurement of Body Mass Index (BMI)

Equation (1) [51] demonstrates the method by which the body mass index (BMI) is determined, taking into account an individual's height and weight. This straightforward calculation involves dividing one's weight in kilograms by their height in meters squared. The resulting value is then used to classify adults as underweight, normal, overweight, or obese.

$$BMI = \frac{\text{Weight (Kg)}}{\text{Height (m)}^2} \quad \ldots(1)$$

The ranking of weight by BMI, was shown in the following Table (2-4) [52].

Following the guidelines outlined in CLSI protocol EP9-A2, we conducted a thorough evaluation of the precision of the VIDAS® 25-OH Vitamin D Total Assay. This evaluation encompassed the use of assay controls and blood samples across the dynamic range. To ensure accuracy, each sample was assessed twice daily for five consecutive days using three different reagent lots on two separate VIDAS® systems. In order to assess the accuracy of the assay, we utilized samples with 25(OH)D levels ranging from approximately 10 to 130 ng/mL. Additionally, we determined the blank limit, functional sensitivity, and detection limit of the assay in accordance with CLSI protocol EP17-A2. The Limit of Quantification (LoQ), also known as functional sensitivity, represents the lowest concentration of 25(OH)D that can be accurately measured with a specific coefficient of variation (CV).

Assay Methodology

The VIDAS® 25-OH Vitamin D Total Assay utilizes a two-step competitive immunoassay approach. Initially, the protein carrier (DBP) is separated from the serum or plasma 25(OH)D and combined with an alkalinephosphatase (ALP)-conjugated antibody specific to vitamin D. In the second step, the unbound ALP antibody is exposed to a solid phase receptor coated with a vitamin D mimic. Following the purification of the solid phase, the fluorescence process is initiated by the addition of the substrate reagent. The system detects relative fluorescence units, which are inversely proportional to the amount of 25(OH)D present in the sample.

Precision

The reproducibility and repeatability of the VIDAS® 25-OH Vitamin D Total Assay were evaluated using standard deviation and CV%. These measures were used to assess the precision between runs, between days, between calibrations, between lots, and between instruments, as well as the precision within lots, within runs, and within instruments. The accuracy profile of the assay revealed that the total accuracy CV% ranges from 16.0% at 10.5 ng/mL to 2.4% at 119.8 ng/mL.

Linearity

The anticipated outcome for the low sample pool was 7.1 ng/mL, while the high sample pool was expected to yield a value of 132.1 ng/mL. Analysis of the test results revealed a linearity of less than 12% within the specified range of [8.1-126] ng/mL, as determined by a weighted linear regression analysis. In terms of detection, the VIDAS® 25-OH Vitamin D Total Assay has a limit of blank (LoB) of 6.2 ng/mL and a limit of detection (LoD) of 8.1 ng/mL. The quantification limit (LoQ) is determined by the coefficient of variation (CV).

25(OH)D2 cross reactivity determination

To calculate the 25(OH)D2 cross reactivity, the concentrations of 25(OH)D2 and 25(OH)D3 were assessed through LC-MS/MS analysis. This involved the use of unaltered blood samples that naturally contained these compounds, without any artificial additions. The formula was then applied to determine the cross reactivity of 25(OH)D2 for each individual sample.

$$\text{D2 cross reactivity (\%)} = \frac{[25(OH)D\,\text{Total}]_{VIDAS} - [25(OH)D3]_{LCMS}}{[25(OH)D2]_{LCMS}} \times 100$$

The VIDAS® 25-OH Vitamin D Total Assay exhibits a cross reactivity of 91% with 25(OH)D2, yielding this outcome.

Determination of Total Serum Superoxide Dismutase (SOD) Activity

In the last decade, different methods were used to estimate the most important antioxidant enzyme SOD activity. Some methods used expensive commercial kit or enzymatic reaction or such as using cytochrome C (266) and xanthine oxidase (267). These methods are expensive and very protein sensitive which susceptible to denaturation. Nitro blue tetrazolium was also used where the sample was radiated using a straight neon lamp 20 watt (268).

The Principle

Serum SOD activity was measured using Beyer's indirect method, which built upon the riboflavin/NBT technique (268). The fundamental principle of this method is that superoxide radicals degrade nitro blue tetrazolium (NBT) to violet formazan, which can be measured at 470–560 nm using spectrophotometry [53] as seen in Figure 3.

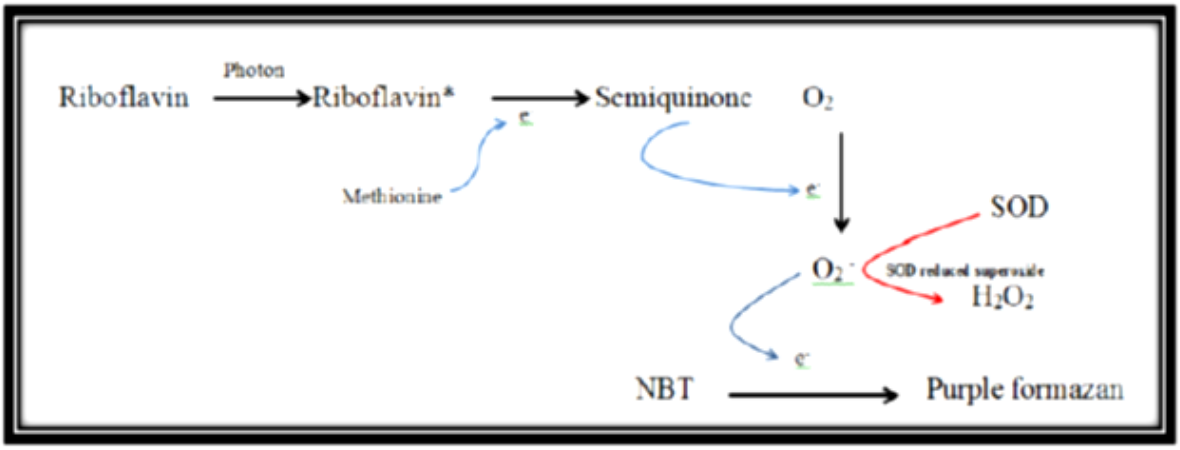


Figure 3. The riboflavin/NBT method is used to indirectly evaluate the activity of superoxide dismutase. When riboflavin is exposed to light in the presence of oxygen and an electron donor, such methionine, superoxide anions are created. NBT can be changed into formazan by these anions, however SOD prevents this from happening

Reagents

Reagents Preparation

Reagents	Preparation
R1	Solution (A): Using deionized water, two distinct weights (2.9942 g of K2HPO4.3H2O and 0.0098 g of EDTA-2Na) were mixed and diluted up to 100 milliliters. Option (B): 1.7854 g of KH2PO4 and 0.0098 g of EDTA-2Na were dissolved in a little amount of deionized water to bring the volume up to 100 mL. The working phosphate buffer solution was prepared as follows: The pH was raised to 7.8 by mixing a volume of solution (B) with 50 milliliters of solution (A).

R2 Weights of 0.0044g of riboflavin were dissolved in a small amount of deionized water, and the volume was then raised to 100 mL using the same deionized water.

The working solution was prepared as following:

Component	Concentration W/V[a]	Volume (mL)	Final Concentration
Phosphate buffer (pH 7.8)		11.875	103 mM
L-Methionine	300 mg/10mL	1.5	19.8 mM
NBT-2HCl[b]	14.1 mg/10 mL	1	0.114 mM
Triton x-100	100 mg/10 mL	0.75	$4.96x10^{-2}$ (W/V)
Total		15.125	

a: Solutions were prepared in deionized water.

b: The brown container containing this solution was refrigerated.

Procedure

1. The working mixture solutions were diluted with deionized water by adding 0.4 mL to aliquots measuring 0.5 mL.
2. The serum, consisting of 10 µL, was thoroughly mixed in a covered tube, while a separate tube containing 10 µL of buffer solution served as the control.
3. Immediately after the addition and mixing of R2 (10µL) to each tube, the absorbance at 560 nm was promptly measured.
4. In order to assess the absorbance, the tubes were subjected to illumination within an aluminum foil box containing two 20-Watt fluorescent lamps at a temperature of 25°C for a duration of 7 minutes. After the 7-minute period, the tubes were extracted and the absorbance was promptly measured at a wavelength of 560 nm.

Calculations

From the following formula the inhibition percentage of SOD activity was determined:

$$\text{SOD activity (inhibition \%)} = \frac{(AB2-AB1)-(AS2-AS1)}{(AB2-AB1)} \times 100 \quad \ldots(2)$$

Where:

AS1 = The sample's absorbance prior to illumination.

AS2 = The sample's absorbance upon lighting.

AB1 = The blank's absorption prior to illumination.

AB2 = The blank's absorption upon lighting.

New unit of SOD is 1 the enzyme activity that inhibits 50% of NBT formazan formation (270, 271). Therefore, the SOD activity in the assay of riboflavin/NBT in unit (U/mL) is expressed according to the following equations:

50% inhibition =1 Unit of SOD = Y

Y unit= X% * 1/50

Y unit is from =10µL of serum contain enzyme

Total volume of serum contain enzyme = 10µL/1000 (1 ml =1000 µL)

(Y/10) *1000 = SOD activity (unit /ml)

Statistical Analysis

Statistical analysis was conducted using the Prism Graph Pad 8.0.1 software. To determine the significance between the mean values of the sick group and the healthy control group, a t-test was employed. All the compared values demonstrated significance, with a P value of less than 0.05 indicating significance.

3. Results and Discussion

Table 3 showcases the demographic information of (30) individuals who were in good health and (50) patients with a deficiency in vitamin D, along with the mean and t-value for the research parameters compared to the control group. Similarly, Table 1 presents the demographic data and study features for (30) healthy participants and (50) patients with vitamin D deficiency. In order to further analyze the data, both the vitamin D deficient group and the healthy group were divided based on gender. This division resulted in 25 male and 25 female patients with vitamin D deficiency, as well as 15 healthy males and females serving as controls.

In addition to age and BMI, the table probably contains information regarding blood vitamin D levels and other pertinent laboratory measurements. It is highly probable that the mean and t-values of the study parameters, which indicate the extent to which each parameter differs between the vitamin D deficient group and the healthy group, are also presented.

One must bear in mind that while t-values may signify statistical significance, they do not necessarily imply clinical or practical importance. Therefore, when analyzing the findings, it is imperative to consider the research question and pertinent clinical benchmarks.

Playing a crucial role in the body's defense against oxidative stress is the enzyme known as superoxide dismutase (SOD) 52. This enzyme facilitates the dismutation of the superoxide radical, a highly reactive compound that poses a threat to proteins, lipids, and DNA within cells. Through its catalytic action, SOD transforms the superoxide radical into both hydrogen peroxide and molecular oxygen [54].

Following this, additional enzymes such as glutathione peroxidase and catalase play a role in breaking down hydrogen peroxide into water and molecular oxygen. By breaking down superoxide and hydrogen peroxide, SOD plays a crucial role in protecting tissues and cells from oxidative damage.

There are three primary types of SOD: SOD3, SOD2, and SOD1. SOD3 is the extracellular copper-zinc form, SOD2 is the mitochondrial manganese form, and SOD1 is the human cytosolic copper-zinc form. These enzymes are located in different compartments within cells and serve various functions in the body. SOD1 and SOD3 primarily work to counteract superoxide radicals in the extracellular environment, while SOD2 targets superoxide radicals in the mitochondria, which play a crucial role in providing energy for the body [55].

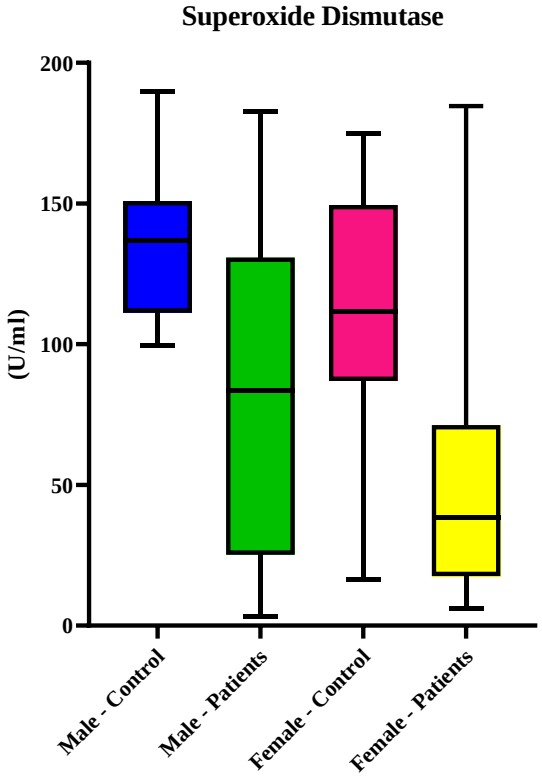
Table 3. The demographic data for (50) patients with Vit-D deficiency and (30) healthy

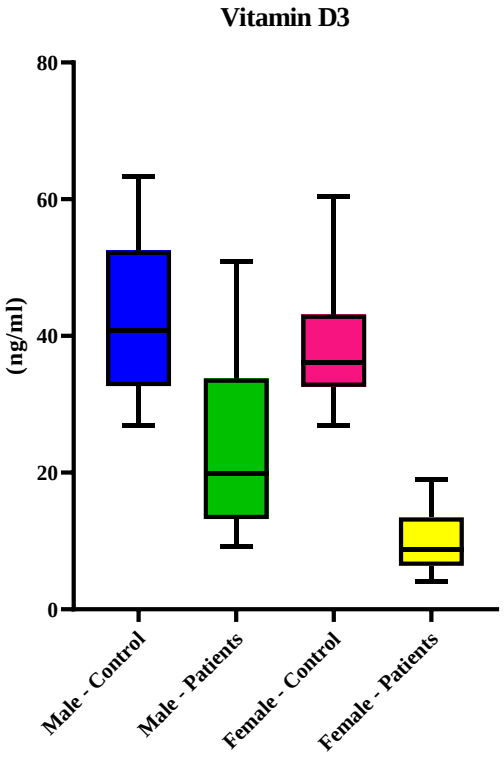
Parameters	Groups	Mean	t-value	95% Confidence Interval	P-value	P-value summary
BMI (kg/m²)	Male - Control	29.73	22.05	26.97 To 32.49	<0.0001	****
	Male - Patients	25.55	19.65	25.58 To 31.52	<0.0001	****
	Female - Control	30.06	27.91	27.85 To 32.26	<0.0001	****
	Female - Patients	28.01	36.18	26.43 To 29.43	<0.0001	****
Age (Years)	Male - Control	27.28	22.94	24.84 To 29.71	<0.0001	****
	Male - Patients	31.13	31.73	29.13 To 33.14	<0.0001	****
	Female - Control	31.07	23.31	28.34 To 33.79	<0.0001	****
	Female - Patients	28.97	28.25	26.87 To 31.06	<0.0001	****
Vitamin D3 (ng/ml)	Male - Control	41.90	20.34	37.68 To 46.12	<0.0001	****
	Male - Patients	24.00	10.06	19.12 To 28.88	<0.0001	****
	Female - Control	38.37	26.86	35.45 To 41.30	<0.0001	****
	Female - Patients	10.09	11.67	8.32 To 11.86	<0.0001	****
SOD (U/ml)	Male - Control	134.50	28.44	124.80 To 144.20	<0.0001	****

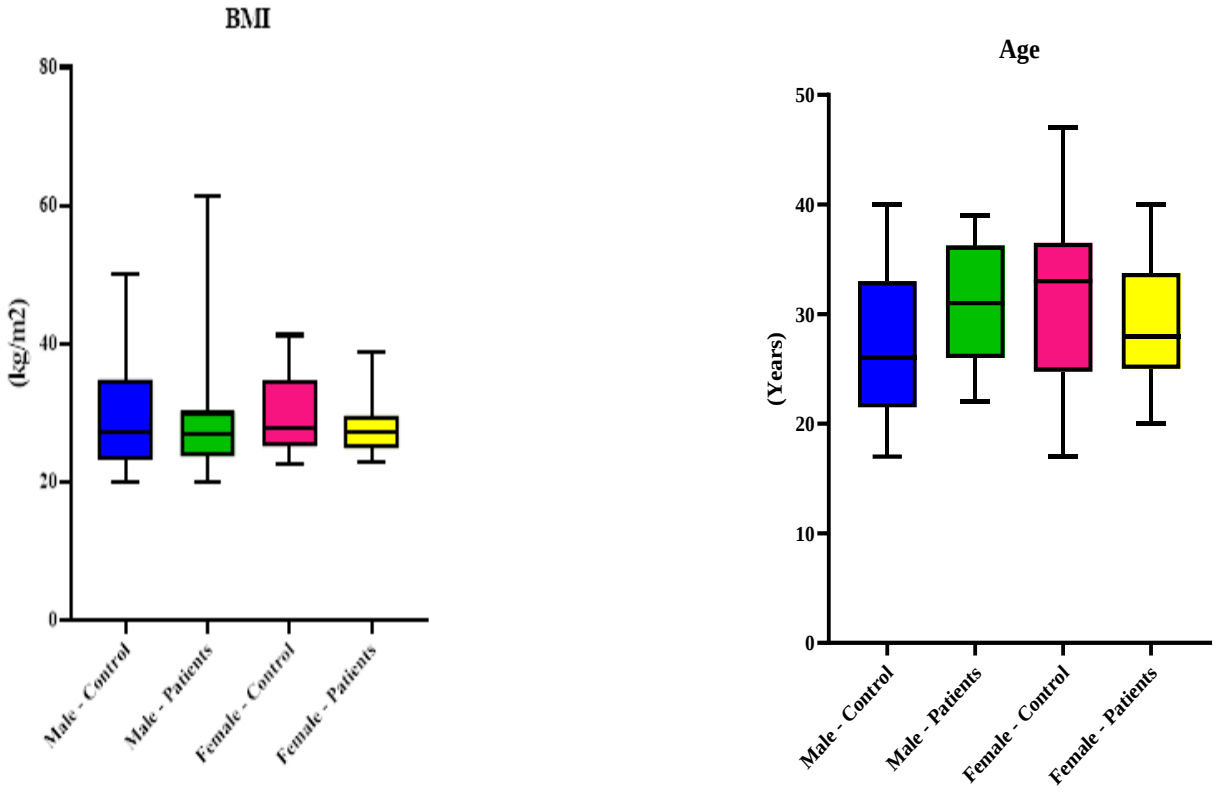
Male - Patients	80.32	7.24	57.63 To 103.00	<0.0001	****
Female - Control	114.10	15.32	98.86 To 129.30	<0.0001	****
Female - Patients	45.10	6.75	31.92 To 59.67	<0.0001	****

The body's antioxidant defense system relies on an essential enzyme called SOD to protect cells and tissues from oxidative damage. SOD plays a crucial role in safeguarding against harmful oxidation.

Based on the study's results, it seems that the levels of the SOD enzyme in the group with the illness were elevated compared to those in the control group. The body's antioxidant defense system relies on the presence of an enzyme called superoxide dismutase, or SOD, which breaks down superoxide radicals into oxygen and hydrogen peroxide [56].







Elevated levels of SOD can serve as a potential indicator of oxidative stress or inflammation within the body. Nevertheless, it is crucial to acknowledge that relying solely on one biomarker, such as SOD, may not provide a comprehensive understanding of the intricate biochemical mechanisms taking place internally. Therefore, it is imperative to take into account other pertinent biomarkers and clinical factors in order to accurately interpret the findings of the study [57].

Playing a pivotal role in the body's defense against oxidative stress, the enzyme SOD, also referred to as superoxide dismutase, is of utmost importance. This enzyme facilitates the disputation of the superoxide radical, an extremely reactive compound that has the potential to harm proteins, lipids, and DNA within cells. Through its catalytic action, SOD transforms the superoxide radical into hydrogen peroxide and molecular oxygen.

4. Conclusion

With a focus on individuals who suffer from vitamin D insufficiency, the study examined the function and importance of the enzyme superoxide dismutase (SOD) in the body's defense against oxidative stress. According to the findings, patients with vitamin D insufficiency had far higher levels of SOD than the group of healthy controls. Our data implies that elevated SOD levels in these patients could be a compensatory mechanism for elevated oxidative stress. This work demonstrated the critical function of SOD in oxidative damage mitigation by facilitating the transformation of reactive superoxide radicals into less reactive molecules such as oxygen and hydrogen peroxide.

There are important ramifications for the higher SOD levels seen in vitamin D deficient people. These point to an elevated oxidative stress load in these people, which may be linked to the onset and advancement of a number of medical disorders. Healthcare practitioners can better manage and treat illnesses associated with oxidative damage by knowing the relationship between vitamin D deficiency and oxidative stress. This research

also emphasizes how crucial it is to keep vitamin D levels sufficient in order to support the body's antioxidant defense mechanisms and general health.

5. Recommendations

Developing scientific research related to Superoxide dismutase and to reach good scientific results and in new, sophisticated ways Contribute to the development of the field of research Scientific.

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