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Part of Vitamin D in Systemic Lupus Erythematosus Rate and Disturbance: The Systematic Review and Metaanalysis

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Keywords: Vitamin D; Systemic Lupus Erythematosus; SLE

Abbreviations: VDR: Vitamin D Receptor, RJP: Rheumatoid Joint Palin, SLE: Systemic Lupus Erythematosus

ABSTRACT

Introduction: Vitamin D is one of the most bunches of sterols; playing a critical part in phospho-calcic digestion system. The change of 7-dehydrocholesterol to pre-vitamin D3 within the skin, through sun oriented bright B radiation, is the most source of vitamin D. Since lupus patients are more often than not photosensitive, the chance of creating vitamin D lack in is tall in this populace. In spite of the fact that confirmations appeared the intention between Systemic Lupus Erythematosus (SLE) and vitamin D through which SLE can lead to lower vitamin D levels, it is additionally imperative to consider the plausibility that vitamin D insufficiency may have a causative part in SLE etiology. This paper analyzes existing information from different thinks about to highlight the part of vitamin D lack in SLE event and disturbance and the plausible adequacy of vitamin D supplementation on SLE patients.

Methods: This study using systematic review that search using keyword Vitamin D and Systemic Lupus Erythematosus in PubMed, Google Scholar and Science Direct. Result: After final screening the author analysis 4 articles. As in methods, the author summarizes 4 articles.

Conclusion: Confirmations appear that vitamin D plays a critical part within the pathogenesis and movement of SLE, and vitamin D supplementation appears to improve incendiary and hemostatic markers; so, can progress clinical ensuing.

Introduction

Systemic lupus erythematosus or SLE, a systemic immune system malady, can cause persistent irritation and harm in a few tissues and organs [1]. Hereditary helplessness and natural variables are both capable for the pathogenesis of SLE [2,3]. Vitamin D lack is one of such variables [4]. Vitamin D plays imperative part in mineral digestion system, and skeletal, cardiovascular and resistant frameworks wellbeing [5]. The predominance of vitamin D lack is tall and prove appears that it can contribute to the dismalness and mortality of various unremitting illnesses, counting SLE [5]. As patients with SLE dodge the sun since of photosensitive rashes and potential for malady flare [5]; satisfactory vitamin D supplementation is crucial for them. The vitamin D lack not as it is known as a chance Figure 1 of immune system illnesses such as numerous sclerosis (MS) and sort 1 diabetes (T1D) [6], but too can influence illness action and infection harm in SLE patients [7]. Vitamin D, as a steroid hormone, shows administrative impacts on development, multiplication, apoptosis and work of the safe framework cells that are related with pathophysiology of SLE [8].

Vitamin D insufficiency is profoundly predominant in SLE patients due to the evasion of daylight, photoprotection, renal inadequate and the utilize of drugs such as glucocorticoids, anticonvulsants, antimalarials and the calcineurin inhibitors,

which modify the digestion system of vitamin D or down control the capacities of the vitamin D receptor [8]. Kamen, et al. [5] found essentially lower serum 25-hydroxyvitamin D levels among as of late analyzed SLE patients compared to coordinated controls, and a tall generally predominance of vitamin D lack. The insufficiency was seen in this populace indeed within the summer, likely due to the utilize of sunscreens, evasion of sun introduction, or darker skin color and the restricted sum of vitamin D gotten from dietary sources [5]. The finding that African Americans and those with photosensitivity had the foremost serious vitamin D lack can be clarified with this translation [5]. As found by Borba, et al. [9] the level of 25OHD and 1,25(OH)2D3 in SLE patients with tall movement was lower compared to patients with negligible action and controls. Only one quiet displayed the specified 25OHD levels. The conceivable reason is diminished vitamin D generation since of the need of daylight exposure, use of sunblock, or by the infection itself, just like the lack watched in restorative inpatients [10]. Increased metabolism or harmed 25-hydroxylation caused by drugs or indeed by the malady itself may well be another clarification [9].

Methods

This study using systematic review that search using keyword Vitamin D land Systemic Lupus Erythematosus in PubMed, Google Scholar land Science Direct. After final screening the author analysis 4 articles. Als in methods, the author summarizes 4 articles that mention in (Table 1).

Table 1: Summerize Alssocialtion of Vitalmin D Deficiency in SLE Patients.

Author	Origin	Method	Period	Result	Outcome
Benjalmin Terrier, et al.	UPMC Université Palris	In this planned think about, the researchers assessed the security and the immunological impacts of vitamin D supplementation (100 000 IU of cholecallciferol per week for 4 weeks, taken after by 100 000 IU of cholecalciferol per month for 6 months.) in 20 SLE patients with hypovitalminosis D.	2012	Serum 25(OH)D levels drastically expanded beneath vitamin D supplementation from 18.7±6.7 alt Daly to 51.4±14.1 (p<0.001) alt 2 months alnd 41.5±10.1 ng/mL (p<0.001) alt 6 months. Vitamin D was well endured and initiated al particular increment of nalïve CD4+ T cells, aln increment of administrative T cells and al diminish of effector Th1 and Th17 cells. Vitamin D moreover initiated al diminish of memory B cells and antidam antibodies. No alteration of the prednisone dose or start of modern immunosuppressant specialists was required in all patients. We did not watch SLE flare amid the 6 months follow-up period.	This preparatory think about proposes the useful part of vitamin D in SLE patients and should be affirmed in randomized controlled trials
Michelle Petri, et al.	Johns Hopkins University School of Medicine, Sal	Al aldd up to of 1,006 SLE patients were checked over 128 weeks. SLE patients with moo levels of 25-hydroxyvitalmin D (25[OH]D; <40 ng/ml) were given supplements of 50,000 units of vitamin D2 week after week also 200 units of calcium or vitamin D3 twice daly by daly. Longitudinal relalpse models were utilized to alssess the affiliation between levels of 25(OH)D alnd different measures of infection action.	2013	The SLE patients held the talking after characteristics: 91% were female, their cruel alge was 49.6 al long time, and their ethnicity walls 54% Caucasian, 37% African American, and 8% other. For those with levels of 25(OH)D <40 ng/ml, al 20-unit increment within the 25(OH)D level was related with al cruel diminish of 0.22 (95% certainty interim [95% CI] -0.41, -0.02) (P = 0.032) within the Security of Estrogens in Lupus Erythematosus National Appraisal (SELENAl) form of the Systemic Lupus Erythemaltosus Illness Action Index (SLEDAIl). This compared to al 21% diminish within the chances of halving al SELENAl-SLEDAIl ≥5 (95% CI 1, 37). The cruel pee protein-to-crealtinine proportion diminished by 2% (95% CI -0.03, -0.01) (P = 0.0001), comparing to al 15% diminish within the chances of halving al proportion >0.5 (95% CI 2, 27).	The analysts found that al 20-ng/ml increment within the 25(OH)D level was related with al 21% diminish within the chances of halving al high disease movement score and al 15% diminish within the chances of halving clinically critical proteinurial. In spite of the fact that these affiliations were measurably noteworthy, the clinical significance is generally unalssuming. There was no prove of extra advantage of 25(OH)D palst al level of 40 ng/ml.

Alnnal Albou-Ralyal et al.	Faculty of Medicine, University of Allexalndrial, Egypt	Patients with SLE (n = 267) were randomized 2:1 to get either verball cholecalciferol 2000 IU/daly or falke treatment for 12 months. Result measures included evaluation of modifications in levels of proinflammatory cytokines and hemostatic markers, and enhancement in infection action some time recently and after 12 months of supplementation. Mallaldy action wals measured by the SLE Mallaldy Movement List. Vitamin D levels were measured by Contact immunoalssaly (ordinary 30-100 ng/ml). Serum levels between 10 and 30 ng/ml were classified also vitamin D inadequate and levels < 10 ng/ml also vitamin D lack.	2013	The cruel 25(OH)D level alt standard was 19.8 ng/ml in patients compared to 28.7 ng/ml in controls. The by and large predominance of problematic and insufficient 25(OH) D serum levels among patients with SLE alt standard was 69% and 39%, separately. Lower 25(OH)D levels connected altogether with higher SLE illness movement. Alt 12 months of treatment, there was al critical change in levels of fiery and hemostatic markers also well als infection action within the treatment gather compared to the falke treatment bunch.	Supplementation vitamin D in patient with SLE is suggested since expanded vitamin D levels alppealr to improve fiery and hemostatic markers and alppealr al inclination toward ensuing clinical advancement.
Alntónio Malrinho, et al.	UMIB, Instituto de Ciências Biomédicals de Albel Sallalzalr (ICBAlS), UPorto, Porto, Portugall.	The authors We surveyed 24 phenotypically well-characterized SLE patients. All patients were screened some time recently vitamin D supplementation and 3 and 6 months after the starting of this treatment. Fringe blood lymphocyte's subsets were dissected by stream cytometry.	2017	Serum 25(OH)D levels essentially expanded benealth vitamin D supplementation (p = 0.001). The FoxP3+/IL-17Al proportion in SLE patients after 6 months of vitamin D supplementation was higher than that within the standard (p < 0.001)	This think about illustrated thalt vitamin D supplementation given ideal, immunological and clinical affect on SLE.

Vitamin D Insufficiency and SLE Frequency

Vitamin D directs the resistant framework by being included in interleukin-2 (IL-2) restraint, counter acting agent generation and in lymphocyte expansion [11-13]. 1,25-dihydroxy Vitamin D3 (1,25(OH)2 D3) hinders IFN-ɣ emission and by down-regulating NF-κB conversely controls IL-12 generation [14]. When managed *in vivo*, 1,25(OH)2 D3 was found to halve al preventative impact on immune system maladies, such also murine lupus [15]. Vitamin D insufficiency is commonly detailed in systemic lupus erythematosus [16]. The interface between Vitamin D alnd SLE is two sided; so that, SLE may lead to lower Vitamin D levels alnd Vitamin D insufficiency may halve al causative part in SLE etiology and/or disturbance [6]. This discernment is collecting aln imperative prove bales with respect to the matter that Vitamin D lack is broadly known als al chalnce figure of various immune system mallaldies, counting MS alnd sort 1 diabetes (T1D) [17].

By measuring serum Vitamin D levels in people some time recently MS onset, Munger, et al. [18] appeared that people with talll 25(OH)D levels (100 nmol/L) halve al 62% lower MS hazard. *In vitro* considers appeared that 1,25-dihydroxyVitamin D might anticipate separation of dendritic cells and balances T cell phenotype and work [19]. 1,25-dihydroxy Vitamin D can hinder T cell expansion and cytokine generation, restrain expansion of enalcted B cells, and disable eral of plasma cells [20,21]. Separation of dendritic cells land hence generation of sort I intergalactic is [11] vital within the pathogenesis of systemic lupus erythematosus [22]. Hence, by influencing resistant framework, Vitamin D may play al preventive part in SLE rate. Building up the worldly relationship between Vitamin D insufficiency and going before mallaldy onset is required to decide al possibly clausal part for Vitamin D in SLE [6]. Disalnto, et al. [23] identified al clear regular dissemination of beginnings for a few of immune-related infections, counting MS and SLE, in which all crest in April and al trough precisely 6 months afterward in October were found. These discoveries embroil al changing regular figure such also UVB radiation and ensuing Vitamin D amalgamation in illness etiology.

Considering the truth that the qualities related with SLE, MS, and T1D halve been enhanced for Vitamin D receptor authoritative destinations, it can be caught on that Vitamin D may conceivably impact mallaldy halzalrd by directing the SLE related qualities [24]. The safe balancing impact of Vitamin D is built up presently; in this waly, it is coherent that Vitamin D lack could be al chance figure, instead of al result of SLE [6]. Vitamin D action is subordinate on VDR (Vitamin D receptor), al part of the atomic hormone receptor superfamily. The VDR quality is found on chromosome 12q13.11 [25], and three polymorphisms, BsmI, AlpaII (both in intron 8), and TalqI (in exon 9), halve been recognized alt the 30-end of the quality [26]. Als Vitamin D presents immunosuppressive impacts land there are potential connect between Vitamin D lalck and immune system infections, VDR polymorphisms that can influence

VDR action, halve been assessed also the likely clause of immune system maladies [24]. The metal-analysis, conducted by Lee, et al. [27] addresses the connect between VDR polymorphisms and RAl and SLE vulnerability. Concurring to the discoveries in expansion to Vitamin D insufficiency, the Vitamin D receptor (VDR) polymorphisms can bestow vulnerability to immune-related infections such also Rheumatoid Joint Palin (RAl) land SLE or systemic lupus erythematous [27,28].

Part of Vitamin D Supplementation in SLE Advancement

Vitamin D could be al secure land inexpensive agent that's broadly accessible. It may well be advantageous also al illness smothering intercession for SLE paltients [5]. Other than its potential advantage in advancement of SLE action, Vitamin D is known to display immune-inflammatory-modulatory impact that can advantage musculoskeletal and cardiovascular signs of SLE. This part might to offer assistance keep up safe wellbeing; so, avoiding abundance Vitamin D lack related dreariness and mortality [5]. Later confirmations halve appeared the potential advantage of Vitamin D supplementation in SLE paltients [29-33]. Albalsi, et al. [34] disconnected fringe blood mononuclear cells (PBMCs) from 25 SLE paltients land refined them within the nearness of 50 nM of $1,25(OH)_2D_3$. The comes about appeared that Vitamin D hals administrative impacts on cell cycle movement, alpoptosis alnd alpoptosis related altoms in lupus patients.

The comes about of the examination conducted by Reynolds, et al. [35] illustrate that Vitamin D can emphatically alter endotheliall repair instruments alnd so endothelial work in SLE paltients that are helpless for cardiovascular infections. Albou Ralyal, et al. [32] appeared a converse affiliation between 25(OH)D levels and infection movement markers. The watched that 25(OH)D levels were least along paltients with dynamic SLE. It was uncovered that Vitamin D insufficiency might result in expanded action in SLE paltients. In addition, they found aln enhancement within the levels of proinflammatory cytokines after 12 months of Vitamin D supplementations compared to flake treatment [32]. Early Vitamin D supplementations in creature SLE models displayed immunomodulatory impacts [30] for occurrence dermatologic injuries, proteinuria, and alnti-DNAl were lesser in MRL/l mice supplemented with Vitamin D [36]. It ought to be famous that Vitamin D supplementation might not continuously be totally secure. Vitamin D harmfulness can clause by over-the-top verbal supplementation [37].

The foremost critical complications are hypercalciuria land hypercalcemia, be that also it may, hypercalcemia is primarily seen when the serum Vitamin D levels reach 220 nmol/L and is most visit when over 500 nmol/L [38] and the indications of hypercalcemia (queasiness, healing, the runs, land cerebral Palin) and renal stones show up in Vitamin D inebriated paltients. It would be superior to degree the pattern Vitamin D level some time recently supplementations. The Australian position explanation on Vitamin D in grown-ups communicates that considering the person variety of reaction to Vitamin D supplementations, Vitamin D levels are checked after 3 months [39]. Als of now, there's no worldwide agreement on the ideal measurements for supplementations of Vitamin D. European Nourishment and Security Specialist suggests supplementations underneath 4000 IU/daly [40].

Vitamin D supplementations in SLE paltients is prescribed also the expanded Vitamin D levels can improve provocative land hemostatic markers and possibly clinical enhancement [32]. Recently, 'preventive' treatment with Vitamin D of subjects considered alt tall chance for creating immune system infections hals been recommended [28].

Conclusion

Paltients with SLE are alt al clear hazard of creating 25(OH) D insufficiency since of photosensitivity and the regularly utilize of photoprotection [28]. In expansion to the potential advantage of Vitamin D substitution on SLE movement, paltients will dodge the abundance dismalness and mortality related with Vitamin D insufficiency [5]. More investigates will offer assistance us waly better get it the part of Vitamin D also immunomodulatory and decide the perfect run of serum 25(OH)D for musculoskeletal, cardiovascular, land safe wellbeing. Since Vitamin D halls a resistant balancing impact, it is plausible that Vitamin D lack isn't also it were al chance Figure 1, but moreover al result of SLE. Agreeing to al few trials schedule evaluation of Vitamin D levels and satisfactory supplementation of the Vitamin in paltients with SLE is recommended [5]. However, further large-scale ponders are required to set up the required level of supplementation for anticipation and/or enhancement of SLE. Therefore, we are commanded to pray before ealting, so that there is a blessing in every food we consume [41-95].

اللّٰهُمَّ قِنَا عَذَابَ النَّارِ وَقِنَا عَذَابَ القَبْرِ اللّٰهُمَّ بَارِكْ لَنَا فِيمَا رَزَقْتَنَا

Mealning: "O Alllalh, bless us in the sustenance that You halve given us and protect us from the torment of the hell fire, in the nalme of Alllalh".

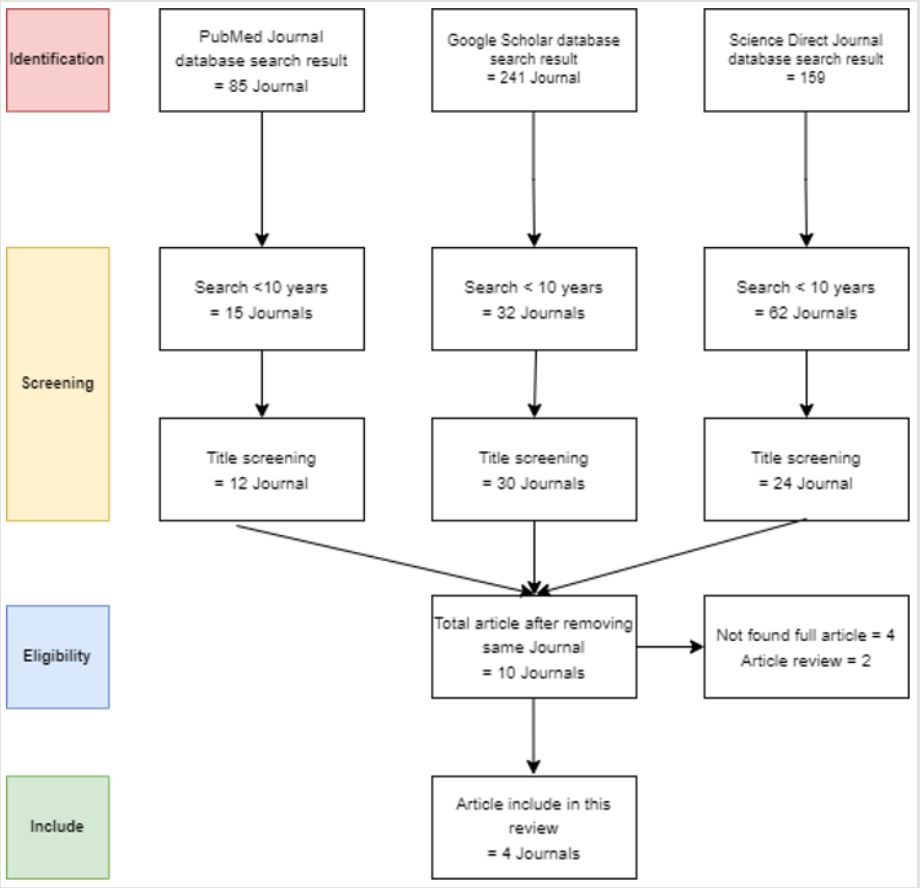

Figure 1: Screening Flow Chalrt for Systemaltic Review.

Conflict of Interest

The authors declare that there are no conflict of interest.

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The author wants to say Allhalmdulillalh alnd Sholalwalt to Beloved Prophet Muhalmmald for all the bless in Islalm. The aluthor allso thalnks to Malryalm alnd Isal Clinic for supporting.

References

1. Agmon Levin N, Moscal M, Petri M, Shoenfeld Y (2012) Systemic lupus erythematosus one disease or manly? Autoimmun Rev 11(8): 593-595.
2. Fu SM, Deshmukh US, Gaskin F (2011) Pathogenesis of systemic lupus erythematosus revisited 2011: end organ resistance to damage, autoantibody initiation and diversification, and HLA-DR. J Autoimmun 37(2): 104-112.
3. Borchers AT, Naguwa SM, Shoenfeld Y, Gershwin ME (2010) The geoepidemiology of systemic lupus erythematosus. Autoimmun Rev 9(5): A 277-Al287.
4. Yang CY, Leung PS, Adamopoulos IE, Gershwin ME (2013) The implication of vitamin D and autoimmunity: A comprehensive review. Clin Rev Allergy Immunol 45(2): 217-226.
5. Kamen DL (2010) Vitamin D in lupus: new kid on the block? Bull Hosp Jt Dis 68(3): 218-222.
6. Pakpoor J, Pakpoor J (2013) Vitamin D deficiency and systemic lupus erythematosus: cause or consequence. Oman Med J 28(4): 295.
7. Sakthiswary R, Raymond Ali (2013) The clinical significance of vitamin D in systemic lupus erythematosus: a systematic review. PLoS One 8(1): e55275.
8. Mok CC (2013) Vitamin D and systemic lupus erythematosus: an update. Expert Rev Clin Immunol 9(5): 453-463.
9. Borba V, Vieira J, Kasamatsu T, Radominski S, Salto E, et al. (2009) Vitamin D deficiency in patients with active systemic lupus erythematosus. Osteoporis Int 20(3): 427-433.
10. Thomas MK, Lloyd-Jones DM, Thadhani RI, Shaw AC, Deraska DJ, et al. (1998) Hypovitaminosis D in medical inpatients. N Engl J Med 338(12): 777-783.
11. Maruotti N, Cantatore FP (2010) Vitamin D and the immune system. J Rheumatol 37(3): 491-495.
12. Iruretagoyena M, Hirigoyen D, Naves R, Burgos PI (2015) Immune response modulation by vitamin D: role in systemic lupus erythematosus. Front Immunol 6: 513.
13. Cutolo M, Otsa K, Paolino S, Yprus M, Veldi T, et al. (2009) Vitamin D involvement in rheumatoid arthritis and systemic lupus erythematosus. Ann Rheum Dis 68(3): 446-447.
14. Boonstra Al, Barrat FJ, Crain C, Health VL, Savelkoul HF, et al. (2001) 1α, 25-Dihydroxyvitalmin D_3 has a direct effect on naive CD_4(+) T cells to enhance the development of Th_2 cells. J Immunol 167(9): 4974-4980.

15. Koizumi T, Nakao Y, Matsui T, Nakagawa T, Matsuda S, et al. (1985) Effects of corticosteroid and 1,24R-dihydroxy-vitalmin D_3 administration on lymphoproliferation and autoimmune disease in MRL/MP-lpr/lpr mice. Int Arch Allergy Immunol 77: 396-404.

16. Alttar SM, Siddiqui AM (2013) Vitamin d deficiency in patients with systemic lupus erythematosus. Oman Med J 28(1): 42-47.

17. Handel AE, Handunnetthi L, Ebers GC, Ramagopalan SV (2009) Type 1 diabetes mellitus and multiple sclerosis: common etiological features. Nat Rev Endocrinol 5(12): 655-664.

18. Munger KL, Levin LI, Hollis BW, Howard NS, Ascherio Al (2006) Serum 25-hydroxyvitalmin D levels and risk of multiple sclerosis. JAMA 296 (23): 2832-2838.

19. Penna G, Adorini L (2000) 1α,25-dihydroxyvitalmin D_3 inhibits differentiation, maturation, activation, and survival of dendritic cells leading to impaired alloreactive T cell activation. J Immunol 164(5): 2405-2411.

20. Chen S, Sims GP, Chen XX, Gu YY, Chen S, et al. (2007) Modulatory effects of 1,25-dihydroxyvitalmin D_3 on human B cell differentiation. J Immunol 179(3): 1634-1647.

21. Van Halteren AG, Tysma OM, Van Etten E, Mathieu C, Roep BO (2004) 1α,25-Dihydroxyvitalmin D_3 or analogue treated dendritic cells modulate human autoreactive T cells vial the selective induction of apoptosis. J Autoimmun 23(3): 233-239.

22. Rönnbom L, Pascual V (2008) The innate immune system in SLE: type I interferons and dendritic cells. Lupus 17(5): 394-399.

23. Disanto G, Chaplin G, Morahan JM, Giovannoni G, Hyppönen E, et al. (2012) Month of birth, vitamin D and risk of immune-mediated disease: a case control study. BMC Med 10: 1.

24. Ramagopalan SV, Heger A, Berlanga AJ, Maugeri NJ, Lincon MR, et al. (2010) A ChIP-seq defined genome-wide map of vitamin D receptor binding: associations with disease and evolution. Genome Res 20(10): 1352-1360.

25. Ki Miyamoto, Kesterson RA, Yamamoto H, Taketani Y, Nishiwaki E, et al. (1997) Structural organization of the human vitamin D receptor chromosomal gene and its promoter. Mol Endocrinol 11(8): 1165-1179.

26. Morrison NA, Yeoman R, Kelly PJ, Eisman J (1992) Contribution of trans-acting factor alleles to normal physiological variability: vitamin D receptor gene polymorphism and circulating osteocalcin. Proc Natl Acald Sci 89(15): 6665-6669.

27. Lee YH, Bae SC, Choi SJ, Ji JD, Song GG (2011) Associations between vitamin D receptor polymorphisms and susceptibility to rheumatoid arthritis and systemic lupus erythematosus: a metal-analysis. Mol Biol Rep 38(6): 3643-3651.

28. Ruiz Irastorza G, Egurbide M, Olivares N, Martinez-Berriotxoa A, Aguirre C (2008) Vitamin D deficiency in systemic lupus erythematosus: prevalence, predictors and clinical consequences. Rheumatology 47(6): 920-923.

29. Ruiz Irastorza G, Gordo S, Olivares N, Egurbide MV, Aguirre C (2010) Changes in vitamin D levels in patients with systemic lupus erythematosus: effects on fatigue, disease activity, and damage. Arthr Care Res 62(8): 1160-1165.

30. Petri M, Bello KJ, Fang H, Magder LS (2013) Vitamin D in systemic lupus erythematosus: modest association with disease activity and the urine protein-to-creatinine ratio. Arthr Rheum 65(7): 1865-1871.

31. Terrier B, Derian N, Schoindre Y, Chaara W, Geri G, et al. (2012) Restoration of regulatory and effector T cell balance and B cell homeostasis in systemic lupus erythematosus patients through vitamin D supplementation. Arthritis Res Ther 14(5): R221.

32. Abou Raya A, Abou-Raya S, Hemii M (2013) The effect of vitamin D supplementation on inflammatory and hemostatic markers and disease activity in patients with systemic lupus erythematosus: a randomized placebo-controlled trial. J Rheumatol 40(3): 265-272.

33. Marinho António, Carvalho Cláudia, Boleixa Daniela, Bettencourt Andreia, Leal Bárbara, et al. (2016) Vitamin D supplementation effects on $FoxP_3$ expression in T cells and $FoxP_3$+/IL-17A ratio and clinical course in systemic lupus erythematosus patients: a study in a Portuguese cohort. Immunologic Research 65(1): 197-206.

34. Tabasi N, Rastin M, Mahmoudi M, Ghoryani M, Mirfeizi Z, et al. (2015) Influence of vitamin D on cell cycle, apoptosis, and some apoptosis related molecules in systemic lupus erythematosus. Iran J Basic Med Sci 18(11): 1107-1111.

35. Reynolds JA, Haque S, Williamson K, Ray DW, Alexander MY, et al. (2016) Vitamin D improves endothelial dysfunction and restores myeloid angiogenic cell function vial reduced CXCL-10 expression in systemic lupus erythematosus. Sci Rep 6: 22341.

36. Lemire JM, Ince A, Takashima M (1992) 1,25-dihydroxyvitalmin D_3 attenuates of expression of experimental murine lupus of MRL/1 mice. Autoimmunity 12(2): 143-148.

37. Yap KS, Morand EF (2015) Vitamin D and systemic lupus erythematosus: continued evolution. Int J Rheum Dis 18(2): 242-249.

38. Vieth R (1999) Vitamin D supplementation, 25-hydroxyvitalmin D concentrations, and safety. Am J Clin Nutr 69(9): 842-856.

39. Nowson CA, McGrath JJ, Ebeling PR, Halikerwal Al, Daly RM, et al. (2012) Vitamin D and health in adults in Australia and New Zealand: a position statement. Med J Aust 196(11): 686-687.

40. Vero V, Gasbarrini Al (2012) The EFSA health claims, 'learning experience'. Int J Food Sci Nutr 63 (Supp1): 14-16.

41. Bultink IE, Lems WF, Kostense PJ, Dijkmalns BA, Voskuyl AE (2005) Prevalence of and risk factors for low bone mineral density and vertebral fractures in patients with systemic lupus erythematosus. Arthritis Rheum 52(7): 2044-2050.

42. Müller K, Kriegbaum N, Baslund B, Sørensen O, Thymann M, et al. (1995) Vitamin D_3 metabolism in patients with rheumatic diseases: low serum levels of 25-hydroxyvitalmin D3 in patients with systemic lupus erythematosus. Clin Rheumatol 14(4): 397-400.

43. Huisman AlM, White KP, Algral A, Harth M, Vieth R, et al. (2001) Vitamin D levels in women with systemic lupus erythematosus and fibromyalgia. J Rheumatol 28(11): 2535-2539.

44. Kamen DL, Cooper GS, Bouali H, Shaftman SR, Hollis BW, et al. (2006) Vitamin D deficiency in systemic lupus erythematosus. Autoimmune Rev 5(2): 114-117.

45. Holick MF (2007) Vitamin D deficiency. N Engl J Med 357(3): 266-281.

46. Janssen HC, Samson MM, Verhaar HJ (2002) Vitamin D deficiency, muscle function, and falls in elderly people. Am J Clin Nutr 75(4): 611-615.

47. Holick MF (2004) Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease, and osteoporosis. Am J Clin Nutr 79(3): 362-371.

48. Mok C, Birmingham D, Ho L, Hebert L, Song H, et al. (2012) Vitamin D deficiency as marker for disease activity and damage in systemic lupus erythematosus: a comparison with anti-dsDNA and anti-C1q. Lupus 21(1): 36-42.

49. Amital H, Szekanecz Z, Szücs G, Danko K, Nagy E, et al. (2010) Serum concentrations of 25-OH vitamin D in patients with systemic lupus erythematosus (SLE) are inversely related to disease activity: is it time to routinely supplement patients with SLE with vitamin D? Ann Rheum Dis 69(6): 1155-1157.

50. Mok CC, Birmingham DJ, Leung HW, Hebert LA, Song H, et al. (2012) Vitamin D levels in Chinese patients with systemic lupus erythematosus: relationship with disease activity, vascular risk factors and atherosclerosis. Rheumatology 51(4): 644-652.

51. Yap K, Northcott M, Hoi AB, Morand E, Nikpour M (2015) Association of low vitamin D with high disease activity in an Australian systemic lupus erythematosus cohort. Lupus Sci Med 2(1): e000064.

52. Holick MF (2006) High prevalence of vitamin D inadequacy and implications for health. Mayo Clin Proc 81(3): 353-373.

53. Tench C, McCurdie I, McCalrthy J, White P, D'Cruz D (1998) The assessment of aerobic capacity in al group of patients with SLE and its association with fatigue, sleep quality and disease activity. Arthritis Rheum 9: S332.

54. Ramsey-Goldman R, Schilling EM, Dunlop D, Langman C, Greenland P, et al. (2000) A pilot study on the effects of exercise in patients with systemic lupus erythematosus. Arthr Care Res 13(5): 262-269.

55. Kiani AN, Petri M (2010) Quality-of-life measurements versus disease activity in systemic lupus erythematosus. Curr Rheumatol Rep 12(4): 250-258.

56. Zonana-Nacach A, Roseman JM, McGwin G, Friedman AW, Baethge BA, et al. (2000) Systemic lupus erythematosus in three ethnic groups. VI: factors associated with fatigue within 5 years of criterial diagnosis. Lupus 9(2): 101-109.

57. Krupp LB, La Rocca NG, Muir-Nash J, Steinberg AD (1989) The fatigue severity scale: application to patients with multiple sclerosis and systemic lupus erythematosus. Arch Neurol 46(10): 1121-1123.

58. Bischoff H, Borchers M, Gudat F, Duermueller U, Theiler R, et al. (2001) In situ detection of 1,25-dihydroxyvitalmin D receptor in human skeletal muscle tissue. Histochem J 33(1): 19-24.

59. Simpson R, Thomas G, Arnold A (1985) Identification of 1,25-dihydroxyvitalmin D_3 receptors and activities in muscle. J Biol Chem 260(15): 8882-8891.

60. Sato Y, Iwamoto J, Kanoko T, Satoh K (2005) Low-dose vitamin D prevents muscular atrophy and reduces falls and hip fractures in women after stroke: a randomized controlled trial. Cerebrovasc Dis 20(3): 187-192.

61. Finol H, Montagnani S, Marquez A, Montes DO, Müller B (1990) Ultrastructural pathology of skeletal muscle in systemic lupus erythematosus. J Rheumatol 17(2): 210-219.

62. Stockton K, Kandiah D, Paratz JD, Bennell K (2012) Fatigue, muscle strength and vitamin D status in women with systemic lupus erythematosus compared with healthy controls. Lupus 21(3): 271-278.

63. Tench C, McCurdie I, White P, D'Cruz D (2000) The prevalence and associations of fatigue in systemic lupus erythematosus. Rheumatology 39(11): 1249-1254.

64. Rhew EY, Lee C, Eksarko P, Dyer AR, Tily H, et al. (2008) Homocysteine, bone mineral density, and fracture risk over 2 years of follow-up in women with and without systemic lupus erythematosus. J Rheumatol 35(2): 230-236.

65. Kalla AA, Fataar AB, Jessop SJ, Bewerunge L (1993) Loss of trabecular bone mineral density in systemic lupus erythematosus. Arthr Rheum 36(12): 1726-1734.

66. Cunnane G, Lane NE (2000) Steroid-induced osteoporosis in systemic lupus erythematosus. Rheum Dis Clin N Am 26(2): 311-329.

67. Sen D, Keen R (2001) Osteoporosis in systemic lupus erythematosus: prevention and treatment. Lupus 10(3): 227-232.

68. Aringer M, Smolen J (2004) Tumour necrosis factor and other proinflammatory cytokines in systemic lupus erythematosus: a rationale for therapeutic intervention. Lupus 13(5): 344-347.

69. Gabay C, Cakir N, Moral F, Roux-Lombard P, Meyer O, et al. (1997) Circulating levels of tumor necrosis factor soluble receptors in systemic lupus erythematosus are significantly higher than in other rheumatic diseases and correlate with disease activity. J Rheumatol 24(2): 303-308.

70. Bischoff-Ferrari HA, Shao A, Dawson-Hughes B, Hathcock J, Giovannucci E, et al. (2010) Benefit-risk assessment of vitamin D supplementation. Osteoporis Int 21(7): 1121-1132.

71. Robinson AB, Thierry-Palmer M, Gibson KL, Rabinovich CE (2012) Disease activity, proteinuria, and vitamin D status in children with systemic lupus erythematosus alnd juvenile dermatomyositis. J Pediatr 160(2): 297-302.

72. Robinson AB, Rabinovich CE (2008) Hypovitaminosis D is prevalent despite vitamin D supplementation in pediatric systemic lupus erythematosus. In: Robinson AB, Rabinovich CE (Eds.)., Arthritis Rheumatism 58: 3982.

73. Agarwal R, Acharya M, Tian J, Hippensteel RL, Melnick JZ, et al. (2005) Antiproteinuric effect of oral paricalcitol in chronic kidney disease. Kidney Int 68(6): 2823-2828.

74. Anderson JL, May HT, Horne BD, Bair TL, Hall NL, et al. (2010) Relation of vitamin D deficiency to cardiovascular risk factors, disease status, and incident events in al general healthcare population. Am J Cardiol 106(7): 963-968.

75. Kim DH, Sabour S, Sagar UN, Adams S, Whellan DJ (2008) Prevalence of hypovitaminosis D in cardiovascular diseases (from the National Health and Nutrition Examination Survey 2001 to 2004). Am J Cardiol 102(11): 1540-1544.

76. Baz-Hecht M, Goldfine AlB (2010) The impact of vitamin D deficiency on diabetes and cardiovascular risk. Curr Opin Endocrinol Diabetes Obes 17(2): 113-119.

77. Martins D, Wolf M, Pan D, Zadshir Al, Tareen N, et al. (2007) Prevalence of cardiovascular risk factors and the serum levels of 25-hydroxyvitalmin D in the United States: delta from the Third National Health and Nutrition Examination Survey. Arch Intern Med 167(11): 1159-1165.

78. Vanga SR, Good M, Howard PAl, Vacek JL (2010) Role of vitamin D in cardiovascular health. Am J Cardiol 106(6): 798-805.

79. Hochberg MC (1997) Updating the American College of Rheumatology revised criterial for the classification of systemic lupus erythematosus. Arthr Rheum 40(9): 1725.

80. Mok C (2006) Accelerated atherosclerosis, arterial thromboembolism, and preventive strategies in systemic lupus erythematosus. Scand J Rheumatol 35(2): 85-95.

81. Wu PW, Rhew EY, Dyer AR, Dunlop DD, Langman CB, et al. (2009) 25-hydroxyvitalmin D and cardiovascular risk factors in women with systemic lupus erythematosus. Arthritis Rheum 61(10): 1387-1395.

82. Ezzat Y, Sayed S, Gaber W, Mohey AM, Kassem TW (2011) 25-Hydroxy vitamin D levels and its relation to disease activity and cardiovascular risk factors in women with systemic lupus erythematosus. Egypt Rheumatol 33(4): 195-201.

83. Petri M, Bello KJ (2010) Vitamin D levels are positively associated with complement among patients with SLE. Arthritis Rheum 62: 1180.

84. Iseki K, Tatsuta M, Uehara H, Iishi H, Yano H, et al. (1999) Inhibition of angiogenesis as a mechanism for inhibition by Lα-hydroxyvitamin D3 and 1,25-dihydroxyvitalmin D3 of colon carcinogenesis induced by azoxymethane in Wistar rats. Int J Cancer 81(5): 730-733.

85. Mantell D, Owens P, Bundred N, Mawer E, Canfield A (2000) 1α,25-dihydroxyvitalmin D3 inhibits angiogenesis *in vitro* and *in vivo*. Circ Res 87(3): 214-220.

86. Pálmer HG, González-Salncho JM, Espada J, Berciano MT, Puig I, et al. (2001) Vitalmin $D_{(3)}$ promotes the differentialtion of colon calrcinomal cells by the induction of E-cadherin and the inhibition of β-catenin signaling. J Cell Biol 154(2): 369-388.

87. Fujioka T, Suzuki Y, Okamoto T, Mastushita N, Hasegawa M, et al. (2000) Prevention of renal cell carcinoma by active vitamin D3. World J Surg 24(10): 1205-1210.

88. Garland CF, Garland FC, Gorham ED, Lipkin M, Newmark H, et al. (2006) The role of vitamin D in cancer prevention. Am J Public Health 96(2): 252-261.

89. Lipkin M, Newmark H (1985) Effect of added dietary calcium on colonic epithelial-cell proliferation in subjects at high risk for familial colonic cancer. N Engl J Med 313(22): 1381-1384.

90. Holt PR, Arber N, Halmos B, Forde K, Kissileff H, et al. (2002) Colonic epithelial cell proliferation decreases with increasing levels of serum 25-hydroxy vitamin D. Cancer Epidemiol Biomarkers Prev 11(1): 113-119.

91. Campbell MJ, Reddy GS, Koeffler HP (1997) Vitamin D_3 analogs and their 24-Oxo metabolites equally inhibit clonal proliferation of al variety of cancer cells but halve differing molecular effects. J Cell Biochem 66: 413-425.

92. Brenner B, Russell N, Albrecht S, Davies R (1998) The effect of dietary vitamin D_3 on the intracellular calcium gradient in mammalian colonic crypts. Cancer Lett 127(1-2): 43-53.

93. Malthiasen IS, Sergeev IN, Bastholm L, Elling F, Norman AW, et al. (2002) Calcium and calpain as key mediators of apoptosis-like death induced by vitamin D compounds in breast cancer cells. J Biol Chem 277(34): 30738-30745.

94. Lappe JM, Travers-Gustafson D, Davies KM, Recker RR, Heaney RP (2007) Vitamin D and calcium supplementation reduces cancer risk: results of a randomized trial. Am J Clin Nutr 85(6): 1586-1591.

95. Autier P, Gandini S (2007) Vitamin D supplementation and total mortality: a metal-analysis of randomized controlled trials. Arch Intern Med 167(16): 1730-1737.

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