http://dx.doi.org/10.14500/aro.11980

Impact of Vitamin D Deficiency on Iron Deficiency Anemia: A Comparative Analysis in Iraqi Population

Sleman Y. Omar[1], Awat H. Awla[2], Musher I. Salih[3†] and Badinan J. Hamadamin[1]

[1]Department of Biology, College of science, University of Raparin, Sulaymaniyah, Kurdistan Region – F.R. Iraq

[2]Department of Chemistry, College of science, University of Raparin, Sulaymaniyah, Kurdistan Region – F.R. Iraq

[3]Department of Chemistry, Faculty of Science and Health, Koya University, Koya KOY45, Kurdistan Region – F.R. Iraq

***Abstract*—Two important health problems are iron deficiency anemia and vitamin D deficiency, as well as the ability to cope with acute and chronic diseases, because iron and vitamin D are the main elements of physiological functions in the human body. Recent studies have indicated that generalized IDA in both healthy and diseased populations may be associated with inadequate levels of VD. The research comprised 132 participants, 65 with IDA and 67 controls, the study was conducted in a competent cross-sectional design, between the two groups matched by age and gender. The laboratory findings included the study of blood indices. Ferritin, iron, hemoglobin, mean corpuscular volume, red cell distribution width, and VD levels were measured for each participant. Iron metabolism markers showed highly significant variations between the groups. Patients with IDA exhibited considerably lower levels of iron indicators than the healthy control group, except for total iron binding capacity (TIBC), which was increased among the patients. The normal control group showed substantially higher serum VD levels than patients with iron deficient anemia ($p < 0.035$). This distinction suggests a high positive link between VD levels and iron metabolism markers, except for TIBC, which exhibited a negative correlation. The results showed a significant correlation between VD levels and several iron metabolism markers in the research participants. This suggests that VD may affect how iron is metabolized and help treat IDA. To fully understand the underlying mechanisms and any therapeutic benefit, further research is needed.**

***Index Terms* – Anemia, Hemoglobin Ferritin, Iron deficiency, Vitamin D deficiency.**

I. Introduction

Vitamin D deficiency (VDD) and iron deficiency anemia (IDA) are two of the most common nutritional disorders globally, particularly in developing countries such as Iraq. IDA, marked by impaired hemoglobin (Hb) production and oxygen transport, is often linked to poor dietary iron intake, impaired absorption, or chronic blood loss (Mohsen and Aljoofy, 2020). VDD, besides causing skeletal disorders, is also associated with extraskeletal complications such as inflammation, immune dysfunction, and altered iron metabolism. Recent studies suggest a complex interplay between Vitamin D (VD) and iron homeostasis, indicating that VDD may influence gene expression related to iron absorption and regulation (Caprio, et al., 2017). In addition, VD may modulate inflammatory responses, which further affects iron metabolism. This bidirectional relationship highlights the importance of simultaneously assessing both deficiencies, particularly in Iraq, where undernutrition and adverse environmental, nutritional, economic, and social factors contribute to widespread micronutrient deficiencies (Musaiger, Hassan and Obeid, 2011). Developing integrated nutritional interventions targeting these deficiencies is essential, especially for vulnerable populations such as children and elderly women. It plays an indispensable role in maintaining physiological homeostasis, and its deficiency can disrupt these vital functions, leading to IDA (Crichton, et al., 2002). IDA remains the most prevalent nutritional deficiency worldwide, contributing significantly to global morbidity and mortality, especially in vulnerable populations such as children, women of reproductive age, and individuals with chronic illnesses (Killip, Bennett and Chambers, 2007). The consequences of IDA extend beyond hematologic abnormalities, encompassing impaired cognitive function, delayed growth and development, and compromised immune response (Al-Zuhairy, et al., 2022). Iron homeostasis is tightly regulated by the digestive system, with iron absorption modulated according to the body's physiological needs. In addition, iron plays a critical role in neural development, myelin synthesis, and neurotransmission (Jáuregui-Lobera and treatment, 2014). VD, a fat-soluble secosteroid, is primarily synthesized in the skin on exposure to ultraviolet radiation, with smaller amounts derived from dietary sources. To become biologically active, VD undergoes

ARO-The Scientific Journal of Koya University
Vol. XIII, No.2 (2025), Article ID: ARO.11980. 6 pages
DOI: 10.14500/aro.11980
Received: 02 January 2025; Accepted: 14 July 2025
Regular research paper; Published: 09 August 2025
†Corresponding author's e-mail: musher.ismael@koyauniversity.org

two hydroxylation processes: First in the liver to form 25-hydroxyVD (Abdellateif, et al., 2020), and subsequently in the kidney to produce the active form, 1,25-dihydroxyVD (Boettger, et al., 2017) (Göring, 2018, Shobana and Usharani, 2022). Beyond its well-documented role in calcium and bone metabolism, VD has important immunomodulatory and anti-inflammatory properties, implicating its importance in overall health maintenance (El-Sharkawy and Malki, 2020).

Global data from the World Health Organization (WHO) indicate that anemia affects approximately 42.6% of children under the age of five, with even higher prevalence rates reported in the Eastern Mediterranean region (48.6%) and Iraq (36%) (De Benoist, et al., 2008). Iron deficiency contributes substantially to this burden, with nearly half of Iraqi children under five suffering from iron deficiency, particularly in northern regions (Al-Zuhairy, et al., 2022). Simultaneously, VDD (VDD) is also a pervasive public health problem, with up to 40% of children having insufficient VD levels, and 12.1% classified as VD deficient (Akhtar, et al., 2013). Notably, emerging research has highlighted the frequent coexistence of IDA and VDD, particularly in children, raising important questions about potential interactions between these two deficiencies (Azizi-Soleiman, et al., 2016).

Although iron and VD metabolism involve distinct regulatory pathways, recent evidence suggests potential physiological cross-talk between the two. VD has been shown to modulate the expression of hepcidin, a key regulator of iron homeostasis. Specifically, 1,25-dihydroxyVD suppresses the expression of the hepcidin gene (HAMP), promoting increased iron absorption and mobilization (Rochette, et al., 2015). This emerging evidence highlights a potential bidirectional relationship between VD and iron metabolism, particularly in populations with high rates of nutritional deficiencies (Azizi-Soleiman, et al., 2016).

This study provides novel insights into the potential interplay between VDD and IDA, aiming to enhance the understanding of micronutrient deficiencies in this population. Existing evidence on this topic is fragmented, with most research focusing on pediatric populations, while limited attention has been given to young adults and middle-aged groups. In addition, data from resource-constrained settings, such as Iraq, remain scarce, despite the high prevalence of both deficiencies in this region. Previous studies have primarily relied on basic hematological parameters, overlooking detailed iron biomarkers such as ferritin, total iron-binding capacity (TIBC), and red cell indices, alongside VD status. To address these gaps, this study investigates the relationship between VD levels and a comprehensive panel of iron metabolism markers in a cross-sectional cohort of Iraqi patients with IDA, compared to age- and sex-matched healthy controls. The research employs advanced diagnostic tools and applies standardized WHO criteria to define both VD and iron deficiency, ensuring a rigorous and clinically relevant analysis.

II. Materials and Methods

This investigation was conducted from August 2023 to April 2024 in Ranya, Sulaimani, where 132 patients of both genders, aged 1–70 years, were randomly admitted to the clinic of Smart Hospital. Informed consent forms were distributed to all participants, and each participant signed the form to indicate their willingness to participate in the study. A specially designed questionnaire was used during the patients' interviews with the doctor to collect information related to their health status.

Several laboratory tests were performed as part of this investigation, including Complete Blood Count, iron levels, TIBC, ferritin, and VD. Based on their iron status, participants were categorized into two groups: IDA and normal iron status. IDA was defined as Hb ≤11 g/dL, while normal iron status was defined as Hb > 11 g/dL (El-Adawy, et al., 2019, Auerbach and Adamson, 2016b). The IDA group included subjects with impaired iron-binding capacity (≤15%) due to infection-related disturbances in iron metabolism.

Ferritin levels were also measured, as ferritin is a key marker for diagnosing IDA. The normal ferritin range is 30–400 ng/mL for adult males and 13–175 ng/mL for adult females. For infants, the normal range is 25–200 ng/mL, and it can reach up to 600 ng/mL after the 1st month (Mathuthu and Mason, 2021).

In addition, VD status was assessed. VDD was defined as a level ≤20 ng/mL, insufficiency as 20–30 ng/mL, and normal VD as ≥30 ng/mL (Azizi-Soleiman, et al., 2016, Grygorieva, et al., 2023).

It is important to note that in some patients, inflammation can lead to iron metabolism disturbances, resulting in decreased iron-binding capacity (Chouraqui, 2022, Bacchetta, et al., 2014b). As a result, individuals with inflammation-related iron abnormalities were classified appropriately based on their iron status.

A. Study Design

A detailed study of the complex interactions of low serum iron status with VDD was carried out. The purpose of this study provides a detailed comparative analysis of patients with IDA and VDD. Individuals in attendance: Those with iron deficiency and VDD were used to sample that 132 people were selected. Every participant carefully provided their informed consent, which guaranteed a strong ethical basis.

B. Participants

After enrolling in the study, the 132 individuals were divided into two groups: Those with low and normal iron levels. These 65 patients had iron deficiency, while 67 participants had normal iron levels. There were 132 participants, 28 (46.7%) men and 37 (51.4%) women with iron deficiency, and 32 (53.3%) men and 35 (48.6%) women were healthy control. Participants ranged in age from 1 to 70 years old.

The study was conducted in compliance with ethical norms and guidelines. At the time of the investigation, the patient had given consent. VD and iron deficiency were diagnosed based on tests and clinical observations. It makes the diagnosis of iron and vitamin deficiencies dependent on

http://dx.doi.org/10.14500/aro.11980

laboratory studies. A standardized questionnaire that covered demographic data, medical history, and test findings was used to gather data.

C. Data Collection

Patient data were collected using a variety of techniques, including medical history questionnaires. Additionally, blood samples were obtained in order to quantify 25-hydroxyvitamin D (Abdellateif et al.). After that, sterile procedures were used to remove blood samples from the arm via capillaries, and vacationer tubes were used to hold them. After the participant ID number was written on the tubes, they were delivered to a laboratory for testing.

Data were collected over eight months. Participants were recruited and their eligibility was checked throughout this period. After identifying eligible participants, they followed the necessary data collection procedures and were included in the study. To maintain participant privacy, all patient data were uniformly collected and kept private and safe.

D. Ethical Approval

The Raparin University Faculty of Science's ethics committee formally approved the study idea. Before any samples were collected, all participants provided informed written consent. In addition, all procedures adhered to Iraqi regulations and protocols for biomedical research.

E. Statistical Analysis

Version 23 of the SPSS statistical program (IBM Corporation, Armonk, NY, USA) was used to analyze the data. The continuous variables between normal controls and patients with iron-deficiency anemia were compared using the independent t-test. Mean ± standard deviation was used to report the results. The categorical variables in the two groups were compared using the Chi-square test. Potential correlations between the analyzed parameters were evaluated using Pearson correlation evaluation, with a significance level of $p < 0.05$.

TABLE I
General Characteristic Data for all Studied Groups

Characteristics	Study groups		p-value
	Iron deficiency anemia (n=65)	Healthy control group (n=67)	
Age (years) Mean±SE	35.44±26.919	28.850±19.88	0.111
Age distribution No. (%)			
<15	17 (26.2)	22 (32.8)	0.588
15–29	17 (26.2)	15 (22.4)	
30–45	12 (18.5)	15 (22.4)	
>46	19 (29.1)	15 (22.4)	
Gender no. (%) Mean±SE			
Male	28 (46.7)	32 (53.3)	0.589
Female	37 (51.4)	35 (48.6)	
Iron (mcg/dL)	28.65±16.79	100.1±40.68	<0.001
Ferritin (ng/ml)	41.32±54.29	84.22±76.52	<0.001
TIBC (mcg/dL)	444.5±43.80	330.2±43.05	<0.001
Hb (g/dl)	10.07±1.806	15.35±13.581	0.002
MCV	79.44±5.260	88.62±6.53	<0.001
RDW	12.04±1.294	14.268±1.84	<0.001
Vitamin D (ng/mL)	24.31±13.67	29.36±14.16	0.039

TIBC: Total iron binding capacity, Hb: Hemoglobin, MCV: Mean corpuscular volume, RDW: Red cell distribution width. *Data are presented as median. **Data are given as mean±standard deviation (SD). P<0.05 indicates significance, and P>0.05 indicates non-significant

TABLE II
Comparison of Biochemical Parameters among Iron Deficiency Anemia Patients with Different Vitamin D Levels.

General characteristics	Studied groups			p-value
	Optimal (≥30 ng/mL) (n=17)	Intermediate (20–<30 ng/mL) (n=13)	Deficient (<20 ng/mL) (n=35)	
Iron (mcg/dL)	32.72±18.44	31.20±17.08	25.73±15.75	0.312
Ferritin (ng/mL)	68.67±61.01	46.83±84.25	25.98±25.77	0.024
TIBC (mcg/dL)	434.3±50.17	443.9±43.07	446.2±44.85	0.673
Hb (g/dL)	10.51±1.983	10.27±2.188	9.789±1.552	0.375
MCV	81.49±5.670	80.00±4.158	78.23±5.208	0.1
RDW	11.75±0.963	12.03±1.331	12.18±1.423	0.53

TIBC: Total iron binding capacity, Hb: Hemoglobin, MCV: Mean corpuscular volume, RDW: Red cell distribution width. *Data are presented as median. **Data are given as mean±standard deviation (SD). P<0.05 indicates significance, and P>0.05 indicates non-significant

III. Results and Discussion

This investigation included two groups: Iron deficiency anima (n = 65) and healthy control (n = 67). The groups under study shared in the Table I the following general characteristics:

Our result according to VD included three groups: Optimal (n = 17), intermediate (n = 13), and deficiency (n =35). The groups under study shared in the Table II the following general characteristics:

The mean iron content in the adequate VD group was 32.72 ± 18.44 mcg/dL, and in the intermediate VD group was 31.20 ± 17.08, also mean iron for VDD is 25.73 ± 15.751. Were the p-value for iron comparison between groups was not significant at p value 0.312). The mean ferritin content in the adequate VD group, intermediate VD group, and VDD was 68.67 ± 61.01 ng/mL, 46.83 ± 84.25 ng/mL, and 25.98 ± 25.77 ng/mL, respectively, p-value for ferritin comparison between groups was significant at 0.024.

The mean TIBC content in the adequate VD group, intermediate VD group, and VDD was 434.3 ± 50.17 mcg/dL, 443.9 ± 43.07 mcg/dL, and 446.2 ± 44.85 mcg/dL, respectively. p-value for TIBC comparison between groups was not significant at 0.673.

The mean HB in the adequate VD group, intermediate VD group, and VDD was 10.51 ± 1.983 g/dL, 10.271 ± 2.188 g/dL, and 9.789 ±1.552 g/dL, respectively. p-value for Hb comparison between groups was not significant at 0.375.

The mean corpuscular volume (MCV) in the adequate VD group, intermediate VD group, and VDD was 81.49 ± 5.670, 80.0 ± 4.158, and 78.234 ± 5.208, respectively. p-value for MCV comparison between groups was not significant at 0.1.

The mean red cell distribution width (RDW) in the adequate VD group, intermediate VD group, and VDD was 11.75 ± 0.963, 12.031 ± 1.331, and 12.18 ± 1.423,

respectively. p-value for RDW comparison between groups was not significant at 0.53.

Correlation analysis between reduced VD level and iron metabolism marker in the patient's group is displayed in Table III. VD and Iron metabolism were correlated significantly and negatively with iron (r = 0.242, p = 0.005), ferritin (r = 0.432, p = 0.001), TIBC (r = −0.231, p = 0.008), MCV (r = 0.279, p = 0.001), and RDW (r = 0.208, p = 0.017). At the same time, VD and Iron metabolism recorded a non-significant correlation with Hb. Inversely, a significant negative correlation was noted between TIBC, these findings are visually supported by the scatter plots in Figure 1 and Table III, which illustrate the positive correlation between vitamin D and serum iron, and the absence of correlation between vitamin D and hemoglobin.

IV. Discussion

This research has greatly enriched the knowledge regarding the specific association between VDD and IDA in the Iraqi population. The inclusion of people from different age groups increases the generality of the results and gives information about the combination of nutritional deficiencies through the life stages. It stresses the importance of managing several nutritional deficiencies at the same time to enhance the health status of at-risk groups. Knowledge of the combinations and effects of various nutrients may lead to better and more precise strategies for IDA management and other comorbidities. In the present study, 132 patients were observed and divided into two groups: Healthy control (n = 67) and iron-deficient anemia (n = 65) groups, according to age, sex, ferritin, VD level, MCV, TIBC, RDW, and iron deficiency. We used SPSS programming and scatter plots to evaluate the results and determine the relationship between these factors (Auerbach and Adamson, 2016a). This study's findings expose a strong correlation between VD insufficiency and an increased risk of anemia – especially iron deficient anemia. Consistent with current epidemiological data, this connection was found to be more pronounced in women than in men (Lee, et al., 2015). Menstrual iron loss, lower socioeconomic level, and nutritional deficiencies are probably the many elements influencing the increased risk among women. Due to their large iron losses during heavy menstrual cycles – up to 42 mg each cycle – menstruating women especially are more likely to get IDA. These results highlight the need of gender-specific treatments to solve micronutrient deficits, especially in areas where anemia and VD shortage are major public health issues (Wintrobe, 2009).

This work has great strength in its thorough investigation of hematological and biochemical indicators, which provide strong proof of the link between VD and iron deficits, as in recent study (De Martinis, et al., 2021, Vaquero, et al., 2024). VD may affect iron metabolism and erythropoiesis, whereas iron is crucial for VD production. We investigated the association between VD deficits (VDD) and diminished iron status, as well as whether advancing iron insufficiency (ID) correlates with suboptimal VD status (Malczewska-Lenczowska, et al., 2018).

With significant p = 0.001, the research revealed a substantial adverse association between iron levels and TIBC. This suggests that, a characteristic of iron insufficiency, TIBC levels grow while iron levels fall. Moreover, substantiated by p = 0.039, a statistically significant correlation between iron deficit and VDD was noted. This result points to a possible bidirectional link between these two micronutrients wherein shortages of one can aggravate or cause shortages of the other. Given the functions of iron and VD in important physiological processes such as erythropoiesis, immunological function, and cellular metabolism, such a link seems scientifically reasonable (Hwalla, et al., 2017). A deficiency of VD increases the risk of many hematological

TABLE III
Correlation Analysis Between Serum Vitamin D Levels, and Iron Metabolism Markers

Parameters	Correlation coefficient (r)	p-value
Iron (mcg/dL)	0.242**	0.005
Ferritin (ng/ml)	0.432**	<0.001
TIBC (mcg/dL)	−0.231**	0.008
Hb (g/dl)	0.006	0.941
MCV	0.279**	0.001
RDW	0.208*	0.017

TIBC: Total iron binding capacity, Hb: Hemoglobin, MCV: Mean corpuscular volume, RDW: Red cell distribution width * Correlation is significant at the 0.05 level (2-tailed), ** Correlation is significant at the 0.01 level (2-tailed).

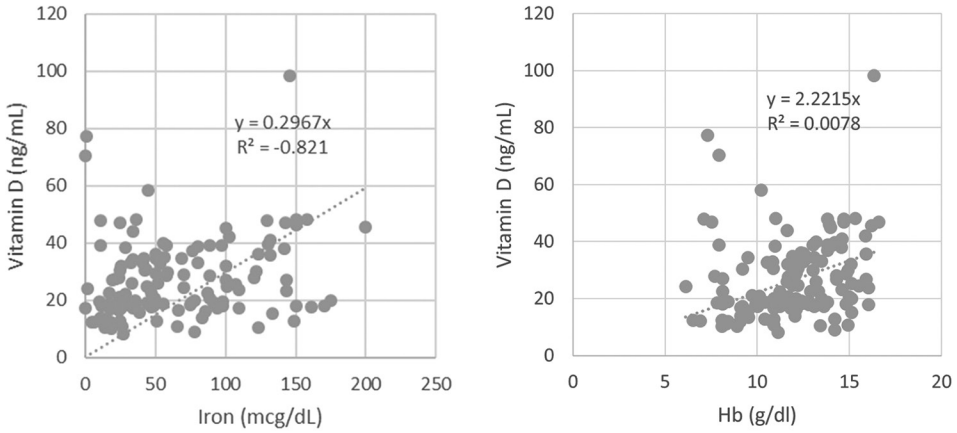

Fig. 1. Scatter plot showing correlation between iron, hemoglobin, with Vitamin D levels.

diseases and disruptions in iron metabolism (Kulling, et al., 2017). Research has shown that a deficiency in VD increases the risk of several hematological diseases and problems in iron metabolism, particularly evident in individuals with diverse health conditions. A VDD induces proinflammatory effects, resulting in increased hepcidin synthesis through the stimulation of pro-inflammatory cytokines and the activation of the JAK-STAT3 pathway. Sun et al. (Sun, et al., 2012) indicated that VD may downregulate hepcidin transcription, while the underlying mechanism remains unidentified. Elevated hepcidin levels may facilitate the sequestration of iron in macrophages and hepatocytes, hence contributing to the onset of inflammatory anemia (Bacchetta, et al., 2014a). The anti-inflammatory action of VD is substantiated by studies indicating a decrease in hepcidin levels and an increase in 25(OH)D concentration in VD deficient individuals after treatment with this vitamin (Smith, et al., 2017).

However, the research also showed that iron insufficiency does not always follow from VDD. For certain studies, non-significant p-values for indicators such as Hb, MCV, RDW, and TIBC clearly showed this. With p-value of 0.005, a direct and important link between ferritin – a critical indication of iron stores – and VD levels was noted, however. This result is consistent with other studies stressing the co-existence of IDA and VD insufficiency, especially in areas with limited access to foods high in nutrients or enough sunshine exposure (Lee, et al., 2015). Studies have shown, for example, that subclinical rickets – often linked to VDD – often coexists with IDA in young infants and need combination supplementation approaches to successfully treat both deficits (Misra, et al., 2008). The research demonstrated a statistically significant negative connection between VD levels and TIBC ($p = 0.008$), therefore affecting erythrocyte indices. This implies that, as shown by increased TIBC levels, iron dysregulation can be aggravated by VD deprivation. Highly significant ($p < 0.001$) the relationship between VD insufficiency and MCV suggests that it may affect red blood cell size and volume, therefore perhaps contributing to the pathogenesis of anemia (Smith and Tangpricha, 2015). The detection of a noteworthy connection between RDW and VDD ($p < 0.017$) supported the theory that VD is engaged in erythropoietic regulation. These results are in line with other studies showing that a lack of VD might change the structure and operation of red blood cells, hence producing anemia (Sim, et al., 2010). Despite these significant findings, the study also highlights areas of unknown interaction between VD and iron metabolism. For instance, a finding of the non-significant link between Hb and VD levels ($p = 0.941$) defines certain past research (Malczewska-Lenczowska, et al., 2018). Variations in study populations, methods, or the multifactorial nature of anemia which might be influenced by a wide range of genetic, environmental, and nutritional elements could assist to explain this difference (Zouine, et al., 2024). Furthermore, the studies do not establish causality even if they provide strong evidence of a link between deficiencies of iron and VD. The basic mechanisms linking these deficiencies are most likely complex and involve hormonal regulation, inflammatory pathways, and genetic predispositions (Bacchetta, et al., 2014a). One idea maintains that VD's anti-inflammatory properties may gently affect iron metabolism (Smith and Tangpricha, 2015). VD is well recognized to maybe restrict the synthesis of pro-inflammatory cytokines, therefore influencing iron intake and use (Andrukhov, et al., 2014). Higher hepcidin levels, a required control of iron homeostasis that reduces iron absorption and mobilization, are linked to chronic inflammation. Changing inflammatory pathways might help to lower hepcidin levels and boost iron availability. Furthermore, included among other organs related with iron metabolism are the intestines and bone marrow, which have VD receptors suggesting a presumably direct function of VD in erythropoiesis and iron absorption (Bacchetta, et al., 2014a, Nairz, et al., 2010). The research emphasizes the more general consequences of nutritional shortages, especially in countries like Iraq with low resources. Deficiencies in iron and VD are linked not just with anemia but also with other serious health implications including worse immune function, more sensitivity to infections, and delayed cognitive and physical development in children. These issues especially worry in poorer countries when food shortages, inadequate healthcare access, and environmental circumstances like inadequate sunshine raise their primary frequency. Dealing with these problems calls for a complete approach including dietary changes, supplement programs, and public health campaigns meant to increase food and drug availability.

V. Conclusion

Underlying the complex interaction between iron deficiency anemia (IDA) and VD insufficiency, this study reveals a significant association between these two micronutrients in the Iraqi population. The noted relationships between VD levels and key iron markers including ferritin, TIBC, MCV, and RDW lead to most probable impact of VD on iron metabolism and erythropoiesis. Although a direct link between VD and hemoglobin was not identified, the results line studies suggesting effects of VD iron homeostasis, most likely by means of inflammatory pathways and hepcidin regulation. Given the great frequency of both VD and iron insufficiencies in resource-limited countries like Iraq, our findings emphasize the need of coordinated public health campaigns treating both deficiencies concurrently.

VI. Recommendations

Based on our findings, we recommend: Healthcare providers should consider sunlight exposure and family history as important factors in assessing iron deficiency and developing early intervention strategies. Further research is needed to fully understand the bidirectional relationship between VDD and iron deficiency health. These recommendations aim to improve early diagnosis, treatment, and management of iron deficiency, ultimately reducing the risk factors.

References

Abdellateif, M.S., Shaarawy, S., Elesawy, Y.F., Mansour, M., Tharwat, E., Ibrahim, N.H., and Eissa, M.S., 2020. The role of vitamin D, platelet-derived growth factor and insulin-like growth factor 1 in the progression of thyroid diseases. *Asian Pacific Journal of Cancer Prevention*, 21, pp.2083-2089.

Akhtar, S., Ismail, T., Atukorala, S., and Arlappa, N., 2013. Micronutrient deficiencies in South Asi-current status and strategies. *Trends Food Science and Technology*, 31, pp.55-62.

Al-Zuhairy, S.H., Darweesh, M.A., Othman, M.A.M., and Al-Zuhairy, N.A.S., 2022. Vitamin D deficiency in young children with iron deficiency in Misan province, Iraq. *Journal of Medicine and Life*, 15, pp. 387-391.

Andrukhov, O., Andrukhova, O., Hulan, U., Tang, Y., Bantleon, H.P., and Rausch-Fan, X., 2014. Both 25-hydroxyvitamin-D3 and 1,25-dihydroxyvitamin-D3 reduces inflammatory response in human periodontal ligament cells. *PLoS One,* 9, p.e90301.

Auerbach, M., and Adamson, J.W., 2016a. How we diagnose and treat iron deficiency anemia. *American Journal of Hematology*, 91, pp.31-38.

Auerbach, M., and Adamson, J.W., 2016b. How we diagnose and treat iron deficiency anemia. *American Journal of Hematology*, 91, pp.31-38.

Azizi-Soleiman, F., Vafa, M., Abiri, B., and Safavi, M., 2016. Effects of iron on vitamin D metabolism: A systematic review. *International Journal of Preventive Medicine*, 7, p.126.

Bacchetta, J., Zaritsky, J.J., Sea, J.L., Chun, R.F., Lisse, T.S., Zavala, K., Nayak, A., Wesseling-Perry, K., Westerman, M., Hollis, B.W., Salusky, I.B., and Hewison, M., 2014a. Suppression of iron-regulatory hepcidin by vitamin D. *Journal of the American Society of Nephrology*, 25, pp.564-572.

Bacchetta, J., Zaritsky, J.J., Sea, J.L., Chun, R.F., Lisse, T.S., Zavala, K., Nayak, A., Wesseling-Perry, K., Westerman, M., Hollis, B.W., Salusky, I.B., and Hewison, M., 2014b. Suppression of iron-regulatory hepcidin by vitamin D. *Journal of the American Society of Nephrology*, 25, pp.564-572.

Boettger, P.C., Knupp, C.L., Liles, D.K., and Walker, K., 2017. Vitamin D deficiency in adult sickle cell patients. *Journal of the National Medical Association*, 109, pp.36-43.

Camaschella, C., 2019. Iron deficiency. Blood, *The Journal of the American Society of Hematology*, 133, pp.30-39.

Caprio, M., Infante, M., Calanchini, M., Mammi, C., and Fabbri, A., 2017. Vitamin D: Not just the bone. Evidence for beneficial pleiotropic extraskeletal effects. *Eating and Weight Disorders*, 22, pp.27-41.

Chouraqui, J.P., 2022. Dietary approaches to iron deficiency prevention in childhood-a critical public health issue. *Nutrients*, 14, p.1604.

Crichton, R.R., Wilmet, S., Legssyer, R., and Ward, R.J., 2002. Molecular and cellular mechanisms of iron homeostasis and toxicity in mammalian cells. *Journal of Inorganic Biochemistry*, 91, pp.9-18.

De Benoist, B., Cogswell, M., Egli, I., Wojdyla, D., and Mclean, E., 2008. Worldwide prevalence of anaemia, WHO vitamin and mineral nutrition information system, 1993-2005. *Public Health Nutrition*, 12, pp.444-454.

De Martinis, M., Allegra, A., Sirufo, M.M., Tonacci, A., Pioggia, G., Raggiunti, M., Ginaldi, L., and Gangemi, S., 2021. Vitamin D deficiency, osteoporosis and effect on autoimmune diseases and hematopoiesis: A review. *International Journal of Molecular Sciences*, 22, p.8855.

El-Adawy, E.H., Zahran, F.E., Shaker, G.A., and Seleem, A., 2019. Vitamin D status in Egyptian adolescent females with iron deficiency anemia and its correlation with serum iron indices. *Endocrine Metabolic and Immune Disorders Drug Targets*, 19, pp.519-525.

El-Sharkawy, A., and Malki, A., 2020. Vitamin D signaling in inflammation and cancer: Molecular mechanisms and therapeutic implications. *Molecules*, 25, p.3219.

Göring, H., 2018. Vitamin D in nature: A product of synthesis and/or degradation of cell membrane components. *Biochemistry (Mosc)*, 83, pp.1350-1357.

Grygorieva, N.V., Tronko, M.D., Kovalenko, V.M., Komisarenko, S.V., Tatarchuk, T.F., Dedukh, N.V., Veliky, M.M., Strafun, S.S., Komisarenko, Y.I., Kalashnikov, A.V., Orlenko, V.L., Pankiv, V.I., Pankiv, O.V., Gogunska, I.V., and Regeda, S.I., 2023. Diagnosis, prevention and treatment of vitamin D deficiency in adults. *Pain Joints Spine*, 13, pp.60-76.

Hwalla, N., Al Dhaheri, A.S., Radwan, H., Alfawaz, H.A., Fouda, M.A., Al-Daghri, N.M., Zaghloul, S., and Blumberg, J.B., 2017. The prevalence of micronutrient deficiencies and inadequacies in the middle east and approaches to interventions. *Nutrients*, 9, p.229.

Jáuregui-Lobera, I., 2014. Iron deficiency and cognitive functions. *Neuropsychiatric Disease and Treatment*, 10, pp.2087-2095.

Killip, S., Bennett, J.M., and Chambers, M.D., 2007. Iron deficiency anemia. *American Family Physician*, 75, pp.671-678.

Kulling, P.M., Olson, K.C., Olson, T.L., Feith, D.J., and Loughran, T.P. Jr., 2017. Vitamin D in hematological disorders and malignancies. *European Journal of Haematology*, 98, pp.187-197.

Lee, J.A., Hwang, J.S., Hwang, I.T., Kim, D.H., Seo, J.H., and Lim, J.S., 2015. Low vitamin D levels are associated with both iron deficiency and anemia in children and adolescents. *Pediatric Hematology and Oncology,* 32, pp.99-108.

Malczewska-Lenczowska, J., Sitkowski, D., Surała, O., Orysiak, J., Szczepańska, B., and Witek, K., 2018. The association between iron and vitamin D status in female elite athletes. *Nutrients,* 10, p.167.

Misra, M., Pacaud, D., Petryk, A., Collett-Solberg, P.F., Kappy, M., and Drug and Therapeutics Committee of the Lawson Wilkins Pediatric Endocrine Society., 2008. Vitamin D deficiency in children and its management: Review of current knowledge and recommendations. *Pediatrics,* 122, pp.398-417.

Mohsen, A.A., and Aljoofy, I.K., 2020. Correlation between vitamin D3 level and iron deficiency anemia in Iraqi high school students in Baghdad city, Iraq. *Biochemical and Cellular Archives*, 20, p.6457.

Musaiger, A.O., Hassan, A.S., and Obeid, O., 2011. The paradox of nutrition-related diseases in the Arab Countries: The need for action. *International Journal of Environmental Research and Public Health*, 8, pp.3637-3671.

Nairz, M., Schroll, A., Sonnweber, T., and Weiss, G., 2010. The struggle for iron-a metal at the host-pathogen interface. *Cellular Microbiology,* 12, pp.1691-1702.

Rochette, L., Gudjoncik, A., Guenancia, C., Zeller, M., Cottin, Y., and Vergely, C., 2015. The iron-regulatory hormone hepcidin: A possible therapeutic target? *Pharmacology and Therapeutics*, 146, pp.35-52.

Sim, J.J., Lac, P.T., Liu, I.L.A., Meguerditchian, S.O., Kumar, V.A., Kujubu, D.A., and Rasgon, S.A., 2010. Vitamin D deficiency and anemia: A cross-sectional study. *Annals of Hematology,* 89, pp.447-452.

Smith, E.M., Alvarez, J.A., Kearns, M.D., Hao, L., Sloan, J.H., Konrad, R.J., Ziegler, T.R., Zughaier, S.M., and Tangpricha, V., 2017. High-dose vitamin D3 reduces circulating hepcidin concentrations: A pilot, randomized, double-blind, placebo-controlled trial in healthy adults. *Clinical Nutrition,* 36, pp.980-985.

Smith, E.M., and Tangpricha, V., 2015. Vitamin D and anemia: Insights into an emerging association. *Current Opinion in Endocrinology Diabetes and Obesity,* 22, pp.432-438.

Sun, C.C., Vaja, V., Babitt, J.L., and Lin, H.Y., 2012. Targeting the hepcidin-ferroportin axis to develop new treatment strategies for anemia of chronic disease and anemia of inflammation. *American Journal of Hematology*, 87, pp.392-400.

Vaquero, M.P., García-maldonado, E., Gallego-narbón, A., Zapatera, B., Alcorta, A., and Martínez-Suárez, M., 2024. Iron deficiency is associated with elevated parathormone levels, low vitamin D status, and risk of bone loss in omnivores and plant-based diet consumers. *International Journal of Molecular Sciences,* 25, p.10290.

Wintrobe, M.M., 2009. *Wintrobe's Clinical Hematology*. Lippincott Williams and Wilkins, United States.

Zouine, N., Lhilali, I., Godderis, L., El Midaoui, A., El Jaafari, S., and Filali-Zegzouti, Y., 2024. The interplay between vitamin D deficiency, iron status, and anemia risk in moroccan women of reproductive age: A cross-sectional analysis. *Epidemiologia (Basel)*, 5, pp.805-827.