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Restorative Effects Of Vitamin D On Insulin, Luteinizing, Testosterone And Anti-Mullerian Hormone Levels As Markers Of Insulin Resistance In Polycystic Ovary Syndrome Patients Attending Moi Teaching And Referral Hospital In Western Part Of Kenya.

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Abstract
Introduction
The paucity of data and the controversies in the studies on the restorative effects of Vitamin D on improving insulin resistance and hyperglycemia, pose a major risk in the fight against metabolic complication associated with polycystic ovary syndrome especially in developing countries like Kenya.
Objectives
The objective of the study was to determine the restorative effects of Vitamin d on insulin, luteinizing, testosterone and anti-mullerian hormone levels as markers of insulin resistance in polycystic ovary syndrome patients attending Moi Teaching and Referral Hospital in Western part of Kenya.
Materials And Methods
The study adopted two phases; phase 1 was an observational study and phase 2 an interventional phase. A total of 100 participants were divided into 60 for observational comprising of PCOS (20) and controls (infertile-20 and fertile 20), while 40 PCOS served in the interventional study that were randomized into 60,000 IU/week of Vitamin D plus 500mg/day of Metformin (n=20) and Metformin 500mg/day only (n=20) for a period of 12 weeks. Quantification of HbA1c and Fasting blood sugars were done at AMPATH reference laboratories using fluorescence immunoassay and while Vitamin D and Insulin hormone levels were done at MTRH laboratories using Cobas automated Chemiluminescence Roche. Data was entered into a computer using excel sheets then analyzed using STATA version 15, Mann Whitney and Kruskal Wallis test and continuous variables are presented as median (interquartile range).
Results
The study established a statistical significant association between PCOS and predictors of insulin resistance including Anti Mullerian hormone, luteinizing hormone, Testosterone, insulin hormone, with one being a PCOS or non PCOS (P<0.05)). The study further established that Vitamin D administered at 60,000 IU per week plus Metformin 500mg per day for 12 weeks led to statistical significant difference from pre intervention to post intervention on AMH, LH, Testosterone, insulin, Vitamin D hormone (p<0.05). The study concludes that Vitamin D may augment Metformin in treatment of these hormones as markers of insulin resistance in women living with Polycystic ovary syndrome.
Key Words: *Polycystic Ovary Syndrome, Vitamin D, Insulin Resistance*

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I. Introduction

Polycystic ovary syndrome (PCOS) is a multifaceted condition considered by production of high androgen hormones, an ovulatory cycles and polycystic ovary on ultrasound (Magpool *et al.,* 2019) that affects women in reproductive age of between 15 years to 50 years. According to Choudhury *et al.,* (2022), PCOS is linked to a number of complications, including decreased fertility, pregnancy loss, IR, type 2 diabetes (T2DM) and psychological disorders (depression and anxiety). The root causes of PCOS have been widely researched but only a few of the contributing theories have been put up in the extensive studies on the origins of PCOS that includes genetics, in utero developmental programming, and environmental influences (Parker *et al.,* 2022).

PCOS based on the current diagnostic criteria is a reproductive disorder; however, it also has a significant metabolic component, with the majority of PCOS patients experiencing compensatory hyperinsulinemia and insulin resistance. According to Stepto *et al.,* (2019), insulin resistance affects between 38 and 80 percent of

women with PCOS and develops without regard to BMI, albeit aggravated. Early research suggests that PCOS patients have significant reduction in insulin sensitivity and that development of insulin resistance in PCOS differs from other metabolic disorders (Wikiera *et al.,* 2017).

The detrimental effects of IR on PCOS reproductive capability are brought on by hyperinsulinemia, which enhances androgen bioavailability through a number of pathophysiological mechanisms (Sanchez *et al.,* 2020). The mechanisms of peripheral IR are still unclear despite over two decades of research. This is most likely caused by PCOS' complex structure and varied etiology. It is still unclear which pathways are at play because several studies have produced conflicting results (Stepto *et al*., 2019).

Insulin resistance, according to (Ruegsegger, 2018), is characterized by insulin's diminished capacity to reduce blood glucose. Despite the fact that IR decreases the absorption of glucose, it may also increase lipogenesis in type 2 diabetes by activating the MAP kinase pathway in the liver while at the same time enhancing lipolysis in adipose tissues (Cao *et al.,* 2023). The immune system is frequently linked to a reduction in intracellular glucose absorption mediated by insulin. Insulin resistance is one of the most common factors that contribute to type 2 diabetes, which is seen mostly in women living with PCOS (Ding *et al.,* 2021). Cellular insulin receptors and IR in eumenorrhoeic women are inversely correlated with the degree of hyperinsulinemia (Koysombat *et al.,* 2023) and in the study of Herman R *et al.,* (2023), it was established that insulin receptor's serine autophosphorylation is increased in the fibroblasts of PCOS women. Research on the pathophysiology of insulin resistance is still on going with many gaps still existing as presented by (Stepto *et al.,* 2019)

II. Materials And Methods

This was both a cross-sectional study and a randomized control trial that was conducted at Mediheal Fertility Center's and Moi Teaching and Referral Hospital in Western part of Kenya in the Reproductive, Endocrine and infertility clinic between July 2023 to May 2025. PCOS was diagnosed based on the Rotterdam criteria 2003, classifying participants into four phenotypes; A (full-blown syndrome PCOS: hyperandrogenism; HA + ovulatory dysfunction {OD} + PCO), Phenotype B (HA+OD), Phenotype C (ovulatory PCOS: HA+PCO), and Phenotype D (non-hyperandrogenic PCOS: OD+PCO). Diagnosis was confirmed if two out of three criteria were met. Sample size was determined using G* Power program 3.1.9.2 software, employing an exact statistical test correlation under a bivariate normal model. To achieve statistical significance, a sample size of 20 participants per group was calculated, later adjusted to 22 per group to account for a 10% drop out rate due to potential pre-analytic errors. In addition to evaluating the history and examination, demographic data were also collected.

Transvaginal ultrasound was performed on PCOS patients to assess ovarian morphology, with criteria including >20 follicles per ovary or ovarian volume >10ml. Serum analysis for vitamin D, AMH, LH, Testosterone and fasting insulin were performed using an automated Electro chemilumescent immune assay method (Roche Diagnostics; Basel, Switzerland e Cobas 311 series) at Moi Teaching and Referral Hospital laboratories. HOMA-IR was calculated using the formula ;(fasting insulin fasting blood glucose/405) with a cut-off score >3.5 indicating insulin resistance. HOMA-B (20Xfasting insulin/fasting blood sugar-3.5), QUICKI (1/log FBS plus log of fasting insulin) (Pispraseert V et al, 2013).

The study was approved by joint **MTRH/Moi university IREC (0004610)** and permission granted by the National Commission of Science and Technology (**NACOSTI/23/26592).** The trial was registered by Pan African trial registry in South Africa (**PACTR202401577278397**.) All participants provided written informed consent and the study adhered to the principles of Helsinki Declaration. Exclusion criteria included diabetes, hyperprolactinemia, hypothyroidism, androgen-secreting tumors, as well as Cushing syndrome. Data was imported on excel sheets and analysed using STATA version 15 for data analysis. Normality test were performed using Shapiro Wilks test and data was skewed and therefore non parametric test was adopted. ManWitney U test and Kruskal Wallis test was applied. Continuous variables are presented as median and interquartile ranges. Spearman's rank correlation on Vitamin D versus AMH, Testosterone Insulin, Vitamin D and LH was performed.

III. Results

(a) The pre-intervention findings on the levels of vitamin D-deficiency and levels of hormones in PCOS and Non PCOS.

With regards to the levels of vitamin D deficiency, the study established that PCOS category had statistically significant lower levels of Vitamin D as compared with both fertile and the infertile controls ($P<0.005$)**.** On the levels of hormones, the study established that the PCOS group had significantly high levels of hormones as compared to the infertile and fertile groups $p< 0.05$. It was therefore clear that hormonal derangements were a major challenge among the PCOS study group

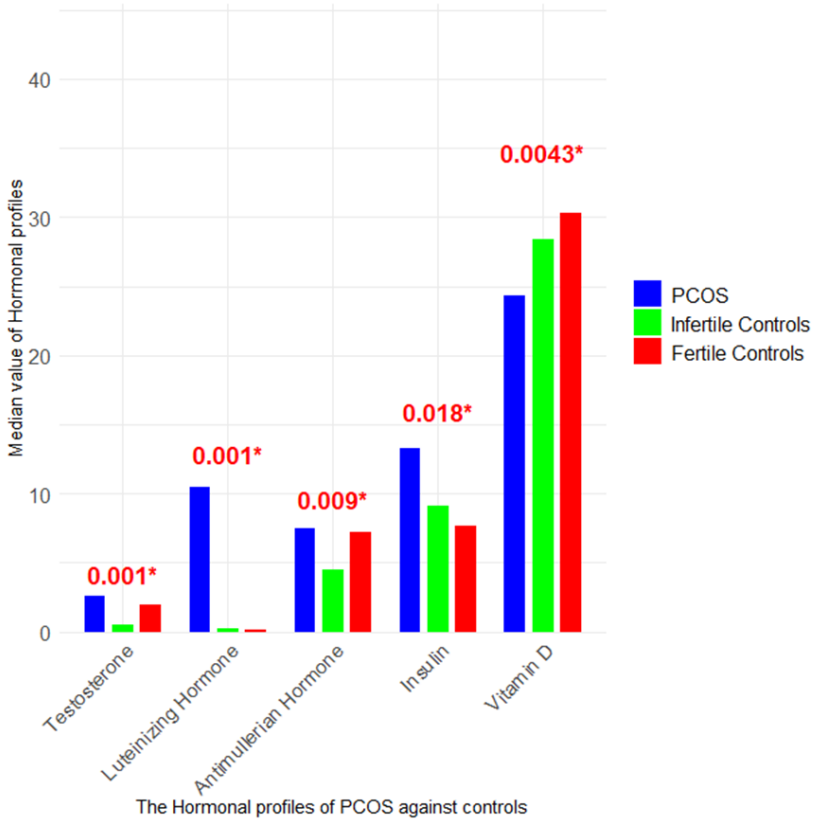

Figure showing the baseline results of median value levels of hormonal profiles of PCOS against the Infertile controls and fertile controls

On correlation analysis between Vitamin D against AMH, LH, Testosterone and Insulin hormone, it was established that Vitamin D correlated negatively with LH (rho = -0.3576642, p = 0.005022), Testosterone (rho = -0.3845513, p = 0.9378) and Insulin hormone (-0.1158432 p= 0.3781while AMH (rho =0.04648901, p= 0.7243)

correlated positively with Vitamin D suggesting that low levels of Vitamin D are associated with high levels of LH, Testosterone and Insulin and vice versa while high levels of Vitamin D are associated with high levels of AMH. However, the correlations were statistically significant only on LH ($p<0.05$)

The Correlation coefficient table between Vitamin D and the hormonal profiles assessed

Hormonal Profile	**R-OUTPUT**
Anti mullerian hormone	data: VITAMIN D ng/ml and AMH ng/ml S = 34317, p-value = 0.7243 alternative hypothesis: true rho is not equal to 0 sample estimates: rho 0.04648901
Leutinizing hormone	data: VITAMIN D ng/ml and LH ng/ml S = 48862, p-value = 0.005022 alternative hypothesis: true rho is not equal to 0 sample estimates: rho -0.3576642
Testosterone	data: VITAMIN D ng/ml and Testosterone ng/ml S = 22150, p-value = 0.002416 alternative hypothesis: true rho is not equal to 0 sample estimates: rho -0.3845513
Insulin	data: VITAMIN D ng/ml and FASTING INSULIN IU/ml S = 40159, p-value = 0.3781 alternative hypothesis: true rho is not equal to 0 sample estimates: rho -0.1158432

(b) The post intervention findings with Vitamin D plus Metformin on the hormonal profiles on PCOS patients

Following the vitamin D supplementation and Metformin treatments, it was noted that the levels of vitamin D deficiency among PCOS patient significantly improved from median value of 27.7 and interquartile ranges of (22.8-30.1) to median value of 68 and interquartile ranges of(57.5-72.8) pre to post intervention period ($P<0.05$), at the same time the findings on the median values and interquartile ranges on all the hormonal profiles of PCOS were noted to have significantly reduced after the intervention with Vitamin D following the 12-week administration $p< 0.05$. as shown below.

The comparative results of hormonal profiles for PCOS group before intervention and post intervention with Vitamin D and Metformin

	Results of hormonal profiles for PCOS group pre and post intervention with Vitamin D and Metformin				**p-value of median values tested between pretest and post test**
	median values and interquartile ranges before intervention ((PRE TEST)		**median values and interquartile ranges after intervention ((POST TEST))**		
	Median value	Interquartile range	Median value	Interquartile range	
Testosterone	0.49	0.30-0.71	0.34	0.23-038	0.040*
Luteinizing hormone	16.23	10.88-18.58	10.70	10.07-14.55	0.048*
Antimullerian hormone	9.2	7.5-11.3	8.4	7.4-9.0	0.030*
Insulin	12.35	10.06-14.06	9.11	8.11- 9.60	0.007*
Vitamin D	27.7	22.8-30.1	68	57.5-72.8	<0.001*

***denotes statistically significant difference*

C The post intervention findings with metformin on the hormonal profiles on PCOS patients (positive controls)

Following the Metformin treatments, it was noted that the levels of vitamin D deficiency among PCOS patient significantly improved from median value of 27.3 and interquartile ranges of (25.3-31.0) to median value of 33.2 and interquartile ranges of(30.3-46.5) pre to post intervention period ($P<0.001$), at the same time the findings on the median values and interquartile ranges on all the hormonal profiles of PCOS were noted to have not significantly reduced after the intervention with Vitamin D following the 12-week administration $p>0.05$ as shown in the table below.

The comparative findings on the median values and interquartile ranges of hormonal profiles for PCOS group before and after intervention with Metformin

Hormonal profiles	Findings of hormonal profiles for PCOS group pre and post intervention with Metformin				**p-value of median values tested between pretest and post test**
	median values and interquartile ranges before intervention (PRE TEST)		**median values and interquartile ranges after intervention (POST TEST)**		
	Median value	Interquartile range	Median value	Interquartile range	
Testosterone	0.71	0.37-0.86	0.57	0.25-0.84	0.208
Luteinizing hormone	11.8	6.2-13.7	7.0	1.4-13.1	0.194
Antimullerian hormone	9.2	7.5-11.3	8.2	6.6-9.7	0.083
Insulin	12.7	8.4-17.4	10.1	9.3-14.7	0.261
Vitamin D	27.3	25.9-31.0	33.2	30.3- 46.5	<0.001*

***denotes statistically significant difference*

IV. Discussion

Researchers are interested in the relationship between AMH and HOMA-IR because it may have substantial therapeutic ramifications on management of insulin resistance and diabetes mellitus (Jun *et al.,* 2020).

In this study it has been established that AMH was 7.5 (6.2, 11.1) in PCOS, while fertile controls had AMH of 7.2 (4.0, 13.0) and the infertile control group had AMH of 4.5 (2.2, 7.2). This was statistically significant with a (P< 0.009)**.** On correlation analysis Vitamin D correlated positively with AMH (rho=0.0756, p=0.56) suggesting that low levels of Vitamin D are associated with low levels of AMH though it was not statistically significant, this study agrees with the study of (Merh *et al.,* 2014) which established a positive association between vitamin D and AMH but differs with the study of (Perce *et al.,* 2015) that found a negative correlation between AMH and Vitamin D. The intervention study established that Vitamin D plus metformin administered for 12 weeks led to decline of AMH from a median and interquartile range of 9.2 (7.5, 11.3) to 8.4 (7.4, 9.0) and the difference was statistically significant with a p<0.030 as compared to comparison group that received metformin only for the same duration that led AMH to decline from 9.2 (7.5,11.3) to 8.2 (6.6, 9.7) but the difference was not statistically significant p<0.083.This study is in agreement with the study of **(**Wang *et al*., 2019), (Bakeer et al., 2018) and (Dastorani *et al.,* 2018), who established that Vitamin led to decline of AMH. However, the studies of (SeyyedAbootorabi *et al.,* 2018), Soya *et al.,* 2019, Cappy *et al.,* 2016 and Gupta *et al.,* 2015 failed to establish the effects of Vitamin D on anti mullerian hormone (AMH).

Hyperandrogenemia is common in PCOS and drives the pathophysiology of insulin resistance and hyperinsulinemia (Armanini *et al.,* 2022).In this study, the median levels and interquartile ranges of LH in PCOS was 10.46 (5.71, 12.98) that varied with the controls who had LH of 0.16 (0.05, 0.29) and 0.21 (0.08, 0.64) for fertile and infertile controls respectively and the difference was statistically significant p=0.001, Testosterone in PCOS had a median and interquartile range of 2.56 (1.21, 4.39) that varied with controls who had testosterone levels of 2.0 (1.67, 2.25) and 0.49 (0.31, 0.77) for fertile and infertile controls respectively and the difference was significant p=0.001. On the correlation analysis Vitamin D correlated negatively with both LH and testosterone (rho=-0.185, p=0.156) and (rho=-0.01, p=0.93) respectively though not significant difference but suggests that lower levels of Vitamin D are associated with high levels of LH and testosterone hormones and vice versa. On the interventional study it was established that Vitamin D in combination with metformin for a period of three months led to a decline of LH and testosterone with median and interquartile ranges of 16.23 (10.88, 18.58) and 0.49 (0.30, 0.71) to 10.70 (10.07, 14.55) and 0.34 (0.23, 0.38) respectively and the variation was statistically significant p<0.05 as compared to comparison group which received metformin for the same duration and had a decline of LH and testosterone with median and interquartile ranges of 11.8 (6.2, 13.7) and 0.71 (0.37, 0.86) to 7.0 (1.4, 13.1) and 0.57 (0.25, 0.84) respectively but the difference was not statistically significant p>0.05. This study is in agreement with the study of (Gandhari *et al.,* 2023) who established that Vitamin D deficiency leads to increase in androgens levels in rats induced PCOS, and its administration led to decline of LH and FSH ratio thus lowering plasma testosterone. This was also agreed by (Behmanesh *et al.,* 2019), that rats exhibiting PCOS like features improved their testosterone levels when Vitamin D was administered. Our study disagrees with the study of (Werh *et al.,* 2011) which found that Vitamin D failed to reduce testosterone in women living with PCOS. Others have argued that Vitamin D can improve LH, FSH and even testosterone but not hyperandrogenism (Karadag *et al.,* 2018). Insulin resistance leads to hyperinsulinemia, which controls the production of androgens, and it is believed that IR is the primary cause of PCOS symptoms.

The majority of individuals in the world are vitamin D deficient or insufficient (Garg *et al*., 2017), he further opines that the prevalence may be normal (> 30ng/ml), insufficient (below 30 ng/ml) or deficient (below 20 ng/ml), depending on the area, time, and country of origin. In this study, it was established that PCOS group had significantly lower Vitamin D levels with a median and interquartile range of 24.3(21.1, 27.6) while the controls had 30.3 (26.2, 32.7) and 28.4 (23.2, 30.7) in fertile and infertile controls respectively (p<0.0043)**.** In the intervention study it was established that Vitamin D levels improved in both arms of treatment with p<0.001. The study also established significant variations on insulin hormone where the median and interquartile range for PCOS was 13.3(8.4, 15.3) while the controls had a median and interquartile ranges of 7.7 (5.81, 12.20) and 9.1 (6.6, 13.0) in fertile and infertile controls respectively p<0.18**.**On correlation analysis between Vitamin D and insulin hormone levels, the study established a positive relation though not significant (rho=0.087, p=0.5049) indicating that low levels of Vitamin D are associated with low insulin and vice versa. The intervention phase established that combination of Vitamin D and metformin for a period of 12 weeks led to a decline of insulin median and interquartile ranges of 12.35 (10.06, 14.06) to 9.11 (8.11, 9.60) and it was statistically significant p=0.007 as compared to metformin group for the same duration which led to a decline of insulin median and interquartile ranges of 12.7 (8.4, 17.4) to 10.1 (9.3,14.7) though the difference was not statistically significant p=0.261**.** This results are in agreement with the study of Jamilian et al., (2017) who investigated the effect of 4,000 IU of cholecalciferol daily versus 1,000 IU of calciferol versus placebo over 12 weeks in 90 women who were living with PCOS and established significant decrease on serum insulin, HOMA-IR and improvement in Vitamin D. However, this study disagrees with the study of (Rajan *et al.,* 2014) who established no significant effect of 12,000 IU of cholecalciferol daily versus placebo over a period of 12 weeks on insulin hormone in 28 PCOS women. The current studies on the effects of Vitamin D and hormones as markers of IR is therefore quite inconsistent and the main reason could be attributed to genetic variations and geographical differences.

V. Conclusion And Recommendation

The study concludes that Anti Mullerian hormone (AMH), Leutinizing hormone (LH), Testosterone and insulin hormone were significantly higher in PCOS as compared to controls while the levels of Vitamin D were significantly lower in PCOS as compared to controls and Vitamin D in combination with Metformin significantly reduces AMH, LH, testosterone, insulin and significantly increases Vitamin D as compared to Metformin alone which only increases Vitamin D. The study also concludes that Vitamin D correlates negatively with LH, testosterone and Insulin but positively with AMH showing that higher levels of LH, testosterone and Insulin are associated with lower levels of Vitamin D but higher levels of AMH are associated with higher levels of Vitamin D.

Further research should explore additional hormonal factors influencing insulin resistance in PCOS, such as leptin, pancreatic polypeptide and assess the potential synergistic effects of Vitamin D supplementation and hypoglycemic agents on other hormonal dysregulations. Ensure adequate Vitamin D levels in women living with PCOS to alleviate hormonal derangements that would lead to insulin resistance.

References

[1]. Argano, C., Mirarchi, L., Amodeo, S., Orlando, V., Torres, A., & Corrao, S. (2023). The Role Of Vitamin D And Its Molecular Bases In Insulin Resistance, Diabetes, Metabolic Syndrome, And Cardiovascular Disease: State Of The Art. International Journal Of Molecular Sciences, 24(20), 15485.

[2]. Bakeer, E.; Radwan, R.; El Mandoury, A.; El Rahman, A.A.; Gad, M.; El Maksoud, S.A. (2018) Anti-Müllerian Hormone As A Diagnostic Marker In Egyptian Infertile Polycystic Ovary Syndrome Females: Correlations With Vitamin D, Total Testosterone, Dyslipidemia And Anthropometric Parameters. Journal Of Medical Biochemistry., 37,448–455.

[3]. Behmanesh, N., Abedelahi, A., Charoudeh, H. N., & Alihemmati, A. (2019). Effects Of Vitamin D Supplementation On Follicular Development, Gonadotropins And Sex Hormone Concentrations, And Insulin Resistance In Induced Polycystic Ovary Syndrome. Turkish Journal Of Obstetrics And Gynecology, 16(3), 143

[4]. Cappy, H., Giacobini, P., Pigny, P., Bruyneel, A., Leroy-Billiard, M., Dewailly, D., & Catteau-Jonard, S. (2016). Low Vitamin D3 And High Anti-Müllerian Hormone Serum Levels In The Polycystic Ovary Syndrome (PCOS): Annales d'Endocrinologie (Vol. 77, No. 5, Pp. 593-599).

[5]. Cao, R., Tian, H., Zhang, Y., Liu, G., Xu, H., Rao, G., & Fu, X. (2023). Signaling Pathways And Intervention For Therapy Of Type 2 Diabetes Mellitus. Medcomm, 4(3), E283.

[6]. Chaudhary, I., Prasad, V., Ganavi, Y. P., Navikala, K., Manjunath, P. R., Selvan, C., & Kalra, P. (2024). A Comparative Analysis Of Serum Irisin Levels And Body Composition Indices In Women With And Without Polycystic Ovary Syndrome (PCOS). Cureus, 16(10), E71426.

[7]. Dastorani, M., Aghadavod, E., Mirhosseini, N., Foroozanfard, F., Zadeh Modarres, S., Amiri Siavashani, M., & Asemi, Z. (2018). The Effects Of Vitamin D Supplementation On Metabolic Profiles And Gene Expression Of Insulin And Lipid Metabolism In Infertile Polycystic Ovary Syndrome Candidates For In Vitro Fertilization. Reproductive Biology And Endocrinology, 16, 1-7.

[8]. Ding, H., Zhang, J., Zhang, F., Zhang, S., Chen, X., Liang, W., & Xie, Q. (2021). Resistance To The Insulin And Elevated Level Of Androgen: A Major Cause Of Polycystic Ovary Syndrome. Frontiers In Endocrinology, 12, 741764.

[9]. Garg G, Kachhawa G, Ramot R, Khadgawat R, Tandon N, Sreeni Vas V, Kriplani A, Gupta N (2015) Effect Of Vitamin D Supplementation On Insulin Kinetics And Cardiovascular Risk Factors In Polycystic Ovarian Syndrome: A Pilot Study. Endocr Connect 4:108–116.

[10]. Gandhari, A. A. S. A. A., Wiyasa, I. W. A., Hariyati, S. N., & Baihaqi, I. (2023). The Influence Of Vitamin D3 Supplementation On LH And FSH Levels In Female Rats (Rattus Norvegicus) With Polycystic Ovary Syndrome (PCOS) Model. Asian Journal Of Health Research, 2(3), 31-37.

[11]. Gupta T, Rawat M, Gupta N, Arora S (2017) Study Of Effect Of Vitamin D Supplementation On The Clinical, Hormonal And Metabolic Profile Of The PCOS Women. Journal Of Obstetetric Gynecology India 67:349–355

[12]. Herman, R., Sikonja, J., Jensterle, M., Janez, A., & Dolzan, V. (2023). Insulin Metabolism In Polycystic Ovary Syndrome: Secretion, Signaling, And Clearance. International Journal Of Molecular Sciences, 24(4), 3140

[13]. Jamilian M, Foroozanfard F, Rahmani E, Talebi M, Bahmani F, Asemi Z (2017) Effect Of Two Different Doses Of Vitamin D Sup Plementation On Metabolic Profiles Of Insulin-Resistant Patients With Polycystic Ovary Syndrome. Nutrients 9:1280.

[14]. Jun TJ, Jelani AM, Omar J, Abdul Rahim R, Yaacob NM. (2020). Serum Anti-Müllerian Hormone In Polycystic Ovary Syndrome And Its Relationship With IR, Lipid Profile And Adiponectin. Indian Journal Of Endocrine And Metabolism; 24:191-5.

[15]. Karadağ, C., Yoldemir, T., & Yavuz, D. G. (2018). Effects Of Vitamin D Supplementation On Insulin Sensitivity And Androgen Levels In Vitamin-D-Deficient Polycystic Ovary Syndrome Patients. Journal Of Obstetrics And Gynaecology Research, 44(2), 270-277.

[16]. Koysombat, K., Dhillo, W. S., & Abbara, A. (2023). Assessing Hypothalamic Pituitary Gonadal Function In Reproductive Disorders. Clinical Science, 137(11), 863-879.

[17]. Maqbool, M., Gani, I., & Geer, M. I. (2019). Polycystic Ovarian Syndrome-A Multifaceted Disease: A Review. Int J Pharm Sci Res, 10(3), 1072-79.

[18]. Merhi, Z. (2019). Vitamin D Attenuates The Effect Of Advanced Glycation End Products On Anti-Mullerian Hormone Signaling. Molecular And Cellular Endocrinology, 479, 87-92.

[19]. Parker, J., O'brien, C., Hawrelak, J., & Gersh, F. L. (2022). Polycystic Ovary Syndrome: An Evolutionary Adaptation To Lifestyle And The Environment. International Journal Of Environmental Research And Public Health, 19(3), 1336.

[20]. Pearce, K.; Gleeson, K.; Tremellen, K. (2015). Serum Anti-Mullerian Hormone Production Is Not Correlated With Seasonal Fluctuations Of Vitamin D Status In Ovulatory Or PCOS Women. Human Reproduction., 30, 2171–2177

[21]. Raja-Khan N, Shah J, Stetter CM, Lott ME, Kunselman AR, Dod Son WC, Legro RS (2014) High-Dose Vitamin D Supplementation And Measures Of Insulin Sensitivity In Polycystic Ovary Syndrome: A Randomized, Controlled Pilot Trial. Fertility And Sterility 101:1740–1746.

[22]. Ruegsegger, G. N., Creo, A. L., Cortes, T. M., Dasari, S., & Nair, K. S. (2018). Altered Mitochondrial Function In Insulin-Deficient And Insulin-Resistant States. The Journal Of Clinical Investigation, 128(9), 3671-3681.

[23]. Sanchez-Garrido, M. A., & Tena-Sempere, M. (2020). Metabolic Dysfunction In Polycystic Ovary Syndrome: Pathogenic Role Of Androgen Excess And Potential Therapeutic Strategies. Molecular Metabolism, 35, 100937.
[24]. Seyyedabootorabi M, Ayremlou P, Behroozi-Lak T, Nourisaeidlou S. (2018); The Effect Of Vitamin D Supplementation On IR, Visceral Fat And Adiponectin In Vitamin D Deficient Women With Polycystic Ovary Syndrome: A Randomized Placebo-Controlled Trial. Gynecology And Endocrinology.; 34:489–94.
[25]. Stepto, N.K., Moreno-Asso, A., Mcilvenna, L.C., Walters, K.A., Rodgers, R.J., (2019). Molecular Mechanisms Of IR In Polycystic Ovary Syndrome. Unraveling The Conundrum In Skeletal Muscle? Journal Of Clinical. Endocrinology And Metabolism. 104, 5372–5381.
[26]. Wang, J., Wu, D., Guo, H., & Li, M. (2019). Hyperandrogenemia And Insulin Resistance: The Chief Culprit Of Polycystic Ovary Syndrome. Life Sciences, 236, 116940
[27]. Wehr, E., Pilz, S., Schweighofer, N., Giuliani, A., Kopera, D., Pieber, T. R., & Obermayer-Pietsch, B. (2009). Association Of Hypovitaminosis D With Metabolic Disturbances In Polycystic Ovary Syndrome. European Journal Of Endocrinology, 161(4), 575-582.
[28]. Wikiera, B., Zubkiewicz-Kucharska, A., Nocoń-Bohusz, J., & Noczyńska, A. (2017). Metabolic Disorders In Polycystic Ovary Syndrome. Pediatric Endocrinology Diabetes And Metabolism, 23(4).