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Vitamin D and Reproductive Dysfunction in Polycystic Ovary Syndrome: Metabolic, Hormonal and Fertility Implications

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ABSTRACT

Background. Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder affecting 15–20% of reproductive-age women and characterized by hyperandrogenism, anovulation, insulin resistance and chronic low-grade inflammation. Vitamin D deficiency is markedly prevalent in PCOS, affecting approximately 67–85% of patients, and has been increasingly recognized as a potential contributor to the metabolic, endocrine, and reproductive disturbances associated with the syndrome. Given its widespread receptor expression in reproductive and metabolic tissues, vitamin D may influence ovarian steroidogenesis, follicular maturation, endometrial receptivity and glucose–insulin homeostasis. Recent evidence indicates that vitamin D status may influence metabolic health, menstrual regularity, and the success of assisted reproductive technologies, including in vitro fertilization (IVF).

Aim. To evaluate the relationship between vitamin D, metabolic and hormonal functions, and fertility outcomes in women with PCOS, as well as to summarize current evidence on the role of vitamin D in natural conception and IVF success.

Material and methods. A literature review was conducted using the PubMed database with the keywords: “Polycystic Ovary Syndrome (PCOS)”, “vitamin D”, “fertility”, “IVF”, “insulin resistance”, “folliculogenesis”, and “reproductive outcomes”. Studies involving clinical trials, observational research and experimental models were included. This study was conducted as a narrative literature review and did not involve original patient data.

Results. Across multiple clinical trials, vitamin D supplementation has been associated with significant improvements in insulin resistance (HOMA-IR), fasting insulin, lipid parameters, and inflammatory markers such as hs-CRP. Several studies demonstrated reductions in total testosterone and free androgen index alongside increases in SHBG, indicating beneficial modulation of hyperandrogenism. In women undergoing ART, higher serum and follicular fluid vitamin D levels were linked to improved ovarian response, oocyte quality, and IVF outcomes. Mechanistic studies support these findings, showing that vitamin D enhances follicular viability, regulates aromatase expression, and modulates cytokine profiles—suppressing pro-inflammatory mediators (TNF-α, IL-6, IFN-γ) while enhancing anti-inflammatory pathways and supporting trophoblast function. However, results regarding ovulation and pregnancy rates remain heterogeneous and appear dependent on baseline vitamin D status, dosage, and treatment duration.

Conclusions. Vitamin D deficiency is associated with worsened insulin resistance, elevated androgen levels, and impaired reproductive function in PCOS. Supplementation has been shown to improve metabolic markers, reduce inflammation, support menstrual regularity, and favorably influence ovarian function. Higher serum and follicular fluid vitamin D levels correlate with improved oocyte quality and IVF outcomes. Optimizing vitamin D status may therefore serve as a valuable adjunctive strategy, rather than a standalone therapy, in managing both metabolic and reproductive aspects of PCOS

Keywords: Polycystic Ovary Syndrome (PCOS), Vitamin D, Fertility, IVF, Insulin resistance, Reproductive function, Folliculogenesis

1. Introduction

Polycystic ovary syndrome (PCOS) is the leading endocrine disorder causing anovulation and infertility in reproductive-age women, affecting up to 20 % of this population and imposing a substantial metabolic burden [1, 2]. PCOS is often associated with hyperinsulinemia, impaired glucose tolerance, and sometimes even type 2 diabetes mellitus (T2DM)[3]. Other contributing factors include insulin resistance (IR), dyslipidemia, endothelial dysfunction, and systemic inflammation, all of which together increase the risk of cardiovascular diseases in women with PCOS compared to healthy individuals[4, 5]. It is likely that reduced insulin sensitivity leads to compensatory hyperinsulinemia, which in turn contributes to the development of hyperandrogenism through chronic stimulation of ovarian theca cells[6]. Among the many modifiable factors explored in PCOS-related subfertility, vitamin D status has emerged as a potential contributor. Polycystic Ovary Syndrome (PCOS) is frequently accompanied by vitamin D deficiency, which has been reported in approximately 67–85% of cases[7, 8]. Women with PCOS frequently exhibit low circulating 25-hydroxyvitamin D levels, and deficiency has been associated with adverse metabolic profiles that may impair oocyte quality and endometrial receptivity. Consequently, assessing and correcting vitamin D insufficiency is often recommended as part of pre-IVF optimisation, alongside lifestyle modification and other adjuvants, to enhance fertility outcomes in women with PCOS[1].

2. Polycystic Ovary Syndrome (PCOS)

2.1. Pathogenesis of Polycystic Ovary Syndrome (PCOS)

Polycystic Ovary Syndrome (PCOS) is a complex, multifactorial endocrine disorder affecting up to 15–20% of women of reproductive age worldwide, depending on diagnostic criteria and population studied[9]. It represents a multisystem condition characterized by reproductive, metabolic, and endocrine abnormalities that often coexist and reinforce one another. The pathogenesis of PCOS involves interactions among neuroendocrine dysregulation, metabolic disturbances—particularly insulin resistance—and excessive androgen production[10].

2.2. Neuroendocrine Mechanisms

One of the core disturbances in PCOS lies in the hypothalamic–pituitary–ovarian (HPO) axis. Women with PCOS exhibit increased frequency and amplitude of gonadotropin-releasing hormone (GnRH) pulses, leading to excessive secretion of luteinizing hormone (LH) and a relative deficiency of follicle-stimulating hormone (FSH). The elevated LH/FSH ratio drives excessive androgen production by the ovarian theca cells, while the insufficient FSH secretion impairs follicular maturation[5].

Furthermore, elevated androgen levels disrupt the normal negative feedback loop between the ovaries and the hypothalamus, perpetuating the abnormal gonadotropin rhythm and contributing to chronic anovulation and menstrual irregularities[11]. Morphologically, the ovaries often present a polycystic appearance (polycystic ovarian morphology, PCOM), characterized by numerous small, immature follicles.

2.3. Metabolic Mechanisms

Insulin resistance (IR) is a hallmark feature of PCOS and occurs independently of obesity in many cases. Peripheral tissues—especially muscle and adipose tissue—demonstrate reduced

insulin sensitivity, leading to compensatory hyperinsulinemia[12]. Elevated insulin levels act synergistically with LH to further stimulate androgen production in theca cells and simultaneously suppress hepatic synthesis of sex hormone-binding globulin (SHBG), resulting in increased circulating free androgens [13, 14].
In addition, PCOS is associated with low-grade chronic inflammation and oxidative stress, which exacerbate insulin resistance and contribute to endothelial dysfunction and dyslipidemia[15]. These metabolic abnormalities explain the higher prevalence of metabolic syndrome, impaired glucose tolerance, and even type 2 diabetes mellitus (T2DM) in women with PCOS[8].

2.4. Androgenic, Genetic, and Environmental Mechanisms

Hyperandrogenism—whether ovarian or adrenal in origin—is both a defining feature and a driving force in PCOS pathophysiology. Excessive androgen production inhibits normal follicular development and oocyte maturation, leading to arrested folliculogenesis and the formation of multiple small cystic follicles characteristic of PCOS[16, 17].
Genetic studies have identified several susceptibility loci associated with PCOS, including variants in CYP11A1, FSHR, and the androgen receptor (AR) genes, which influence steroidogenesis and gonadotropin signaling[18].
Moreover, epigenetic modifications, such as DNA methylation and altered microRNA expression, have been implicated in the long-term dysregulation of ovarian and metabolic pathways [19]. Environmental factors—including obesity, vitamin D deficiency, oxidative stress, and even prenatal androgen exposure—can further modulate gene expression and exacerbate PCOS symptoms [20].
This convergence of genetic predisposition, hormonal imbalance, and environmental triggers creates a self-perpetuating cycle in which androgen excess, insulin resistance, and inflammation continuously reinforce one another.

VITAMIN D

3.1. Biological Functions of Vitamin D and Its Relevance to PCOS

Vitamin D is a fat-soluble vitamin that also functions as a hormone in the human body[21, 22]. One of its primary roles is to support calcium and phosphorus absorption in the intestine, which is essential for proper bone mineralization[23]. Vitamin D deficiency is highly prevalent worldwide, affecting both young and older populations[24]. Beyond bone health, vitamin D influences the immune system, modulating inflammatory responses and autoimmunity. Epidemiological studies suggest that low vitamin D levels are associated with an increased risk of cardiovascular disease, certain cancers, and metabolic disorders[25]. Supplementation is commonly recommended for individuals with vitamin D deficiency or at high risk of deficiency[26]. Recommendations regarding dose and frequency vary due to heterogeneity in study populations and designs[27]. Benefits of supplementation appear greater in individuals with a deficiency rather than those with adequate baseline levels[28], however, excessive intake can cause hypercalcemia and other adverse effects[29]. In summary, vitamin D plays a crucial role not only in skeletal health but also in metabolism, immunity, and overall physiological homeostasis[30].

In women with Polycystic Ovary Syndrome (PCOS), vitamin D deficiency is particularly prevalent, affecting approximately 67–85% of patients[4]. Deficiency is associated with worse metabolic profiles, including increased insulin resistance, dyslipidemia, and impaired glucose

metabolism[31].Insulin resistance promotes hyperinsulinemia, which stimulates excessive androgen production in ovarian theca cells, further impairing ovulation[32]. Numerous studies have demonstrated that vitamin D supplementation in women with PCOS improves menstrual cycle regularity, increases the number of developing follicles, and reduces circulating testosterone levels[26]. In assisted reproductive technology (ART) contexts, higher vitamin D levels correlate with better ovarian response and a higher number of retrieved oocytes[33]. Mechanistically, vitamin D acts through its receptor in ovarian and endometrial cells, modulating sex hormone levels, reducing inflammation, and mitigating oxidative stress—all factors critical in PCOS[34]. Meta-analyses have reported improvements in hormonal parameters such as total testosterone (TT), free androgen index (FAI), and inflammatory markers like hs-CRP after vitamin D supplementation[35]. The beneficial effects of vitamin D may, at least in part, be attributed to its immunomodulatory properties, which play a crucial role in reproductive physiology. In conditions of vitamin D deficiency, the immune balance shifts toward Th2 cytokine dominance and a reduction in overall inflammatory cytokine activity, disrupting normal immune regulation[36]. Vitamin D has been shown to inhibit the secretion of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interferon-gamma (IFN-γ), while simultaneously enhancing the production of human chorionic gonadotropin (hCG) in trophoblast cell models[37]. Furthermore, studies on trophoblast cultures exposed to Escherichia coli demonstrated that vitamin D exhibits anti-inflammatory and antibacterial properties[38], highlighting its broader protective effects within the reproductive system[39]. However, not all studies show significant improvements in ovulation rates or pregnancy outcomes, suggesting that effectiveness depends on dose, duration, baseline vitamin D levels, and comorbid conditions such as obesity and insulin resistance[30, 40].Optimizing vitamin D levels is thus recommended as part of a broader strategy to improve reproductive outcomes in women with PCOS, alongside lifestyle modifications and conventional endocrine therapies[4].

Table 1. Polycystic ovary syndrome and vitamin D deficiency.

Author/Year	Article Type	Study Group	Findings / Summary
Iran M. et al., 2015 [41]	Randomized, placebo-Controlled Trial (RCT)	68 vitamin D-deficient women with PCOS (45 treated, 23 placebo)	Supplementation of 50,000 IU/week for 8 weeks reduced TGF-β1 bioavailability and improved PCOS clinical parameters.
Javed Z. et al., 2019 [42]	Randomized, controlled trial	40 women with PCOS randomized to vitamin D (3,200 IU/day) or placebo over 3 months	Vitamin D group showed significant increase in 25(OH)D levels and modest improvement in HOMA-IR; liver

			markers improved.
Jamilian et al. 2017 [43]	Randomized, double-blind, placebo-controlled clinical trial	The study included 90 insulin-resistant women with polycystic ovary syndrome (HOMA-IR > 2.5) who were randomly assigned into three equal groups (n = 30): one group received 4000 IU/day of vitamin D_3 plus metformin, the second group received 1000 IU/day of vitamin D_3 plus metformin, and the third group received placebo plus metformin for 12 weeks.	Twelve weeks of high-dose (4000 IU/day) vitamin D (with metformin) produced significant improvements in androgenic profile (lower total testosterone and FAI, higher SHBG), decreased hirsutism and hs-CRP, and increased antioxidant capacity compared with low-dose vitamin D (1000 IU/day) and placebo in insulin-resistant women with PCOS.
Lerchbaum et al., 2021 [44]	Randomized Controlled Trial (RCT)	180 women aged 18–40 years with diagnosed PCOS according to the Rotterdam criteria. Participants were randomized to receive either 20,000 IU of vitamin D_3 weekly or placebo for 24 weeks. Serum 25(OH)D levels, reproductive hormones, and metabolic parameters were measured at baseline and after intervention.	In women with PCOS, 24-week Vitamin D supplementation significantly increased FSH levels and reduced the LH/FSH ratio, while showing no significant effect on AMH. In non-PCOS women, no meaningful hormonal changes were observed. Overall, Vitamin D appeared to influence gonadotropin balance only in women with PCOS.
Study 2024 [45]	Randomized Controlled Trial	60 women with PCOS and vitamin D deficiency/insufficiency	Vitamin D supplementation (2,000 IU/day for 12 weeks) resulted in lower BMI, WHR, insulin, HOMA-IR, TG, TC and LDL-C compared to control.
Meta-analysis 2023 [46]	Systematic review & meta-analysis	Multiple RCTs in PCOS women (total subjects >1,000)	Supplementation reduced total testosterone (TT) and hs-CRP, improved TAC and reduced oxidative stress markers in PCOS patients.

3.2. Integrating Vitamin D Into the Framework of Fertility and Assisted Reproduction

Vitamin D has emerged as a significant regulator of human fertility, owing to the widespread expression of vitamin D receptors (VDRs) throughout the reproductive system. VDRs are present in the hypothalamus, pituitary, ovaries, granulosa cells, endometrium, placenta, decidua, and testes, indicating that vitamin D signaling is integral to both female and male reproductive physiology. Experimental evidence demonstrates that vitamin D influences key reproductive processes, including ovarian steroidogenesis, follicular maturation, endometrial receptivity, androgen synthesis, and spermatogenesis. Furthermore, several studies report that vitamin D modulates the expression of critical genes involved in reproductive function, such as aromatase, FSH receptor, and growth hormone–related pathways. Owing to these broad physiological actions, vitamin D has been increasingly examined as a potential modulator of fertility and as an adjunctive factor influencing outcomes of assisted reproductive technologies, including in vitro fertilization (IVF). Table 2 summarizes key experimental, observational, and clinical findings exploring the impact of vitamin D status and VDR signaling on reproductive performance and IVF success rates [54-58].

Table 2. Influence of Vitamin D on Fertility and IVF [7].

Author / Year	Article Type	Study Group	Findings / Summary
Xu et al., 2018 [54]	Experimental animal/cell study	Preantral follicles isolated from rhesus monkey ovaries	Vitamin D enhanced preantral follicle survival, maintained AMH production, and promoted progression to the antral stage, suggesting a direct role in early folliculogenesis.
Kinute et al., 2010 [55]	Animal study (VDR-null mouse model)	Male and female VDR-null mice	Female VDR-null mice showed uterine hypoplasia and reduced aromatase expression; males exhibited reduced sperm count, decreased motility, and testicular abnormalities, demonstrating that absence of VDR causes reproductive dysfunction in both sexes.
Akhavizadegan et al., 2017 [56]	Case–control clinical study	116 fertile men vs. 114 infertile men	Men with vitamin D < 20 ng/mL had significantly lower sperm counts; fertile men exhibited higher mean vitamin D levels, indicating a relationship between vitamin D deficiency and impaired semen quality.
Dennis et al., 2012 [57]	Observational + interventional study	Premenopausal women, adult men, adolescent boys	Women demonstrated seasonal AMH fluctuations (∼18% decrease in winter); vitamin D supplementation prevented this decline, suggesting vitamin D

			may stabilize ovarian reserve markers.
Ozkan et al., 2010 [58]	Prospective observational cohort (IVF)	84 women undergoing IVF	Follicular fluid vitamin D strongly correlated with serum 25(OH)D (r = 0.94); higher follicular vitamin D was associated with improved IVF outcomes, independently of age, BMI, ethnicity, and embryo transfer factors.

The studies summarized in Table 2 collectively highlight the multifaceted role of vitamin D in reproductive biology, integrating evidence from cellular models, animal experiments and human clinical investigations. Experimental studies demonstrate that vitamin D may exert direct effects on ovarian folliculogenesis: Xu et al. showed that vitamin D supplementation enhances preantral follicle survival, supports anti-Müllerian hormone (AMH) production, and promotes progression to the antral stage in primate ovarian tissue, suggesting a beneficial role in early follicular development [54]. Complementary evidence from VDR-knockout mouse models illustrates the necessity of intact vitamin D signaling for reproductive competence. In VDR-null females, uterine hypoplasia and impaired aromatase activity have been observed, while males exhibit reduced sperm count, diminished motility, and testicular histological abnormalities, collectively indicating that absence of VDR leads to profound infertility in both sexes [55].

Human studies further reinforce these biological observations. In a case–control study of men, Akhavizadegan et al. reported that individuals with serum 25(OH)D levels below 20 ng/mL displayed significantly reduced sperm concentration compared with fertile controls, underscoring the potential contribution of vitamin D deficiency to male-factor infertility [56]. Additional evidence from Dennis et al. demonstrated a seasonal variation in AMH levels among premenopausal women, with reductions of approximately 18% during winter months; importantly, vitamin D supplementation appeared to attenuate this decline, suggesting that vitamin D may help stabilize ovarian reserve markers across seasons [57].

In the context of assisted reproduction, Ozkan et al. found that follicular fluid vitamin D concentrations strongly correlated with serum 25(OH)D levels and were independently associated with improved IVF outcomes, even after adjusting for confounders such as age, BMI, ethnicity and embryo transfer characteristics [58]. These findings imply that vitamin D may influence oocyte competence or local follicular microenvironment in ways that enhance implantation or early embryonic development.

4. Discussion

Polycystic ovary syndrome is a heterogeneous endocrine disorder in which reproductive dysfunction is closely intertwined with metabolic abnormalities, chronic low-grade inflammation, and hormonal dysregulation. Increasing evidence suggests that vitamin D deficiency—highly prevalent among women with PCOS—may represent an important, potentially modifiable factor contributing to both metabolic and reproductive disturbances characteristic of the syndrome. The findings summarized in Tables 1 and 2 collectively highlight the multifaceted role of vitamin D in PCOS pathophysiology and fertility outcomes.

The studies presented in Table 1 consistently demonstrate adverse metabolic and hormonal profiles in vitamin D–deficient women with PCOS. Randomized controlled trials and meta-analyses indicate that vitamin D supplementation is associated with improvements in insulin sensitivity, as reflected by reductions in fasting insulin concentrations and HOMA-IR values. Given that insulin resistance is a central pathophysiological driver of PCOS—independent of obesity in many patients—these findings are of particular clinical relevance. Hyperinsulinemia amplifies ovarian androgen production and suppresses hepatic synthesis of sex hormone-binding globulin, thereby exacerbating hyperandrogenism and anovulation. By improving insulin sensitivity, vitamin D may indirectly attenuate these maladaptive endocrine pathways.

In addition to metabolic effects, several studies summarized in Table 1 report favorable changes in hormonal parameters following vitamin D supplementation. Reductions in total testosterone and free androgen index, accompanied by increases in SHBG levels, have been observed, particularly with higher-dose supplementation and longer treatment duration. Furthermore, normalization of gonadotropin balance—manifested as increased FSH levels and a reduced LH/FSH ratio—suggests that vitamin D may influence hypothalamic–pituitary–ovarian axis regulation in women with PCOS. These changes are clinically meaningful, as abnormal gonadotropin secretion plays a key role in impaired follicular development and chronic anovulation.

Inflammation and oxidative stress represent additional mechanisms through which vitamin D may modulate PCOS severity. Chronic low-grade inflammation is a recognized feature of PCOS and contributes to both insulin resistance and ovarian dysfunction. Trials included in Table 1 demonstrate reductions in inflammatory markers such as hs-CRP and improvements in antioxidant capacity following vitamin D supplementation. Of particular interest is the observed reduction in TGF-β1 bioavailability, a cytokine implicated in ovarian fibrosis, follicular arrest, and altered extracellular matrix remodeling within the ovary. These anti-inflammatory and antifibrotic effects provide mechanistic support for the observed metabolic and endocrine improvements.

While Table 1 focuses primarily on metabolic and hormonal outcomes, Table 2 expands the discussion toward fertility and assisted reproductive technologies. Experimental and animal studies indicate that vitamin D exerts direct effects on ovarian tissue, including enhanced follicle survival, maintenance of anti-Müllerian hormone production, and promotion of follicular progression to more advanced developmental stages. Findings from VDR-deficient animal models further underscore the necessity of intact vitamin D signaling for normal reproductive function, as absence of the vitamin D receptor results in profound reproductive impairment in both females and males.

Human observational and clinical studies support these experimental observations. Higher serum and follicular fluid vitamin D concentrations have been associated with improved oocyte quality, fertilization rates, and IVF outcomes, independent of age, body mass index, and other confounding factors. These data suggest that vitamin D may influence the follicular microenvironment and endometrial receptivity through both endocrine and paracrine mechanisms, thereby supporting implantation and early embryonic development.

Nevertheless, despite growing evidence of biological plausibility and surrogate marker improvement, the relationship between vitamin D supplementation and definitive reproductive endpoints—such as ovulation, clinical pregnancy, and live birth rates—remains inconsistent. Heterogeneity across studies with respect to baseline vitamin D status, supplementation dose,

treatment duration, study design, and population characteristics limits direct comparability. Importantly, the most pronounced benefits appear to occur in women with documented vitamin D deficiency rather than in those with sufficient baseline levels, emphasizing the importance of individualized assessment and targeted intervention.

Taken together, the findings discussed herein support the concept that vitamin D deficiency may exacerbate the metabolic, hormonal, and inflammatory milieu underlying PCOS and, in turn, impair reproductive potential. Vitamin D supplementation should not be viewed as a standalone therapy for PCOS but rather as an adjunctive strategy that may enhance the effectiveness of established treatments, including lifestyle modification, insulin sensitizers, and fertility-oriented interventions. Future large-scale, well-designed randomized controlled trials with standardized supplementation protocols and long-term follow-up are needed to clarify the optimal dosing strategy and to determine whether correction of vitamin D deficiency translates into meaningful improvements in live birth outcomes among women with PCOS.

5. Conclusion

Polycystic ovary syndrome (PCOS) is a prevalent endocrine and metabolic disorder affecting 15–20% of women of reproductive age, depending on diagnostic criteria and population differences [47, 48]. Its pathogenesis is multifactorial, involving genetic susceptibility, environmental influences, and hormonal dysregulation, with insulin resistance and hyperandrogenism being central features[49] . Vitamin D deficiency, reported in approximately 67–85% of PCOS patients, has emerged as a significant modifier of both metabolic and reproductive dysfunction[50, 51].

Growing evidence indicates that vitamin D influences ovarian steroidogenesis, follicular maturation, and endometrial receptivity through its action on vitamin D receptors (VDR) expressed in granulosa and endometrial cells [29]. Adequate vitamin D levels have been correlated with improved menstrual regularity, ovulation, and pregnancy rates, both spontaneous and assisted, while deficiency has been linked to adverse outcomes, including higher insulin resistance and poorer embryo quality [28, 30]. Moreover, vitamin D supplementation appears to enhance insulin sensitivity, reduce androgen excess, and improve metabolic markers such as fasting glucose and HOMA-IR index.

From a reproductive perspective, studies suggest that higher vitamin D levels, particularly in the follicular fluid, are associated with improved oocyte quality and higher fertilization rates during IVF procedures [52] . Mechanistically, vitamin D has immunomodulatory effects, including the potential to reduce pro-inflammatory cytokines such as TNF-α and IL-6, while supporting anti-inflammatory pathways that may favor implantation and early embryonic development [53].

The evidence suggests that vitamin D deficiency not only exacerbates the metabolic and hormonal imbalance characteristic of PCOS but may also impair fertility potential through its effects on ovarian and endometrial physiology. Correcting hypovitaminosis D should therefore be considered an integral component of PCOS management, complementing established therapies such as lifestyle intervention and insulin sensitizers. Further randomized controlled trials with standardized dosages and long-term follow-up are needed to clarify the optimal supplementation strategy and to determine whether sustained vitamin D sufficiency translates into improved live birth outcomes in women with PCOS.

Disclossure

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Declaration on the use of AI

In preparing this manuscript, the authors used Artificial intelligence for language improvement and enhancing readability. Following the use of this tool, all content was reviewed and edited by the authors, who take full responsibility for the accuracy and integrity of the final version.

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