

EXPERIMENTAL WORKS

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doi: <https://doi.org/10.15407/ubj97.04.034>EXPERIMENTAL PREECLAMPSIA DEVELOPMENT
DEPENDS ON VITAMIN D₃ STATUS
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Deficiency of vitamin D₃ during pregnancy is a widespread challenge associated with increased risk of complications, particularly preeclampsia (PE), a serious condition characterized by hypertension with proteinuria. This research aimed to study the experimental preeclampsia rates in pregnant rats depending on the vitamin D₃ supply. Eight-week-old female Wistar rats were divided into three experimental groups: control; vitamin D₃-deficient for 60 days before mating; vitamin D₃-deficient with oral vitamin D₃ supplement (1000 IU/kg b.w.t) two weeks before mating. Subgroups with and without PE induction were analyzed. PE was induced by administration of Nω-nitro-L-arginine methyl ester (L-NAME). The blood level of vitamin D₃ was measured using a 25-Hydroxyvitamin D₃ ELISA kit. Proteinuria was assessed using semi-quantitative urine test strips "Prototest". The highest blood pressure and proteinuria levels were recorded in animals with combined vitamin D₃ deficiency and induced preeclampsia. Administration of vitamin D₃ contributed to normalization of hemodynamic parameters and kidney function, indicating the importance of an adequate vitamin D₃ status for pregnancy health and PE prevention.

Key words: vitamin D₃ deficiency, L-NAME, preeclampsia, blood pressure, proteinuria level.

Vitamin D₃ (cholecalciferol) is currently recognized not only as a critical regulator of calcium-phosphate homeostasis and bone remodeling but also as a pleiotropic bioactive compound. It exerts significant effects through the vitamin D₃ auto-/paracrine system on cellular proliferation and differentiation, and participates in neurogenesis, angiogenesis, immune responses, and cell survival or apoptosis [1]. The hormonally active metabolite of vitamin D₃ – calcitriol (1α,25(OH)₂D₃) – acts in cells via both non-genomic and genomic mechanisms, similar to other steroid hormones. These mechanisms are primarily mediated by the vitamin D₃ receptor (VDR) [2]. In target cells, the interaction of 1α,25(OH)₂D₃ with VDR initiates a complex cascade of molecular events culminating in the modulation of transcription factors

activity that influences the expression of target genes [2-4].

Vitamin D₃ deficiency is a significant global health concern affecting various populations, including pregnant women [5-7]. Studies have shown that the prevalence of vitamin D₃ insufficiency among expectant mother's ranges from 28 to 96%, depending on geographic location, climate, dietary habits, and lifestyle [8]. On average, more than 65% of pregnant women have suboptimal vitamin D₃ levels, predisposing them to pregnancy-related complications [9].

Despite geographic differences in the prevalence of hypovitaminosis D₃ among pregnant women, its adverse effects on pregnancy outcomes are well documented, although the underlying mechanisms remain incompletely understood. Vitamin D₃ defi-

ciency has been associated with an increased risk of obstetric complications, including gestational hypertension, preeclampsia, preterm birth, fetal growth restriction, and higher rates of perinatal loss [10–12].

Among the most serious obstetric complications linked to vitamin D₃ deficiency is preeclampsia (PE) – a severe gestational disorder characterized by hypertension and proteinuria or signs of multiorgan dysfunction arising in the second half of pregnancy [13]. PE is a multifactorial pathological condition resulting from a combination of vascular dysfunction, impaired placental vascularization, chronic inflammation, and immune dysregulation.

To investigate the mechanisms of PE development and evaluate the effectiveness of therapeutic interventions, various experimental models using laboratory animals have been developed. These models aim to replicate the core pathophysiological features of PE, such as impaired placental blood flow, oxidative stress, low-grade chronic inflammation, and endothelial dysfunction. Among the commonly used experimental models of preeclampsia in rats are: surgical models, which involve uterine artery ligation or partial placental ischemia to simulate impaired uteroplacental blood flow characteristic of PE; genetic models, utilizing gene mutations or knockout animals to study the role of specific genes in PE pathogenesis; hormonal and immune-mediated models, based on administration of angiotensin II, TNF- α (tumor necrosis factor-alpha), or anti-VEGF (vascular endothelial growth factor) antibodies to induce pathology; pharmacological models, which rely on agents that provoke hypertension, vasoconstriction, and endothelial dysfunction [14].

In pharmacological approaches, the use of N ω -nitro-L-arginine methyl ester (L-NAME) – a nitric oxide synthase (NOS) inhibitor – has received particular attention [15]. L-NAME is widely used to induce PE in rodents due to its ability to inhibit nitric oxide (NO) production, a key modulator of vascular tone. When administered to pregnant rats, L-NAME induces hypertension, placental dysfunction, oxidative stress, and renal pathology, thus mimicking the clinical manifestations of PE in humans.

The main advantages of the L-NAME pharmacological model include its simplicity – administration can be oral, subcutaneous, or intraperitoneal – and it does not require complex or expensive equipment. The reproducibility of results makes this method a reliable and predictable model of PE, with characteristic biochemical and physiological alterations. Moreover, L-NAME is relatively inexpensive and widely available, making it ideal for experimental research. Importantly, its systemic action induces not only elevated blood pressure but also pathophysiological changes in the placenta, kidneys, and vasculature, thereby providing a comprehensive platform for investigating the cellular and molecular mechanisms underlying PE.

A relevant scientific challenge is to explore the mechanisms by which the pleiotropic effects of vitamin D₃ are realized during normal and complicated pregnancies. In particular, the relationship between alterations in vitamin D₃ status and vascular-endothelial dysfunction in PE remains insufficiently understood. In this context, the objective of our study was to assess the pathophysiological features of L-NAME-induced preeclampsia in rats with different levels of vitamin D₃ availability.

Materials and Methods

Animals and general experimental design. All experiments were conducted between November 2024 and February 2025 at the Palladin Institute of Biochemistry, National Academy of Sciences of Ukraine, in the Department of Vitamins and Coenzymes Biochemistry. A total of 37 eight-week-old female Wistar rats with an average body weight of 174 ± 12 g were used. Animals were divided into three groups (Table 1): group I ($n = 10$) – control females fed a standard vivarium diet without vitamin D₃ restrictions. Group II ($n = 13$) – females maintained on a rachitogenic diet devoid of vitamin D₃ for 60 days prior to mating to induce a vitamin D₃-deficient state. Group III ($n = 14$) – females also received a rachitogenic diet for 60 days, then switched to a standard laboratory diet with the ad-

Table 1. Experimental timeline for rat groups

Groups	Days 0–60	Days 61–74	Days 75–78	Days 79–100	GD5–GD15
I	Standard diet	Vehicle treatment	Mating	Pregnancy	L-NAME ($\pm$)
II	Rachitogenic diet	Vehicle treatment	Mating	Pregnancy	L-NAME ($\pm$)
III	Rachitogenic diet	Vit. D ₃ treatment	Mating	Pregnancy	L-NAME ($\pm$)

dition of oral vitamin D₃ dissolved in sunflower oil in a volume of 0.2 ml administered via gavage (oil solution, 1000 IU/kg body weight, cholecalciferol; Sigma, USA) two weeks before mating. Animals were housed under standard vivarium conditions ($22 \pm 2^\circ\text{C}$, relative humidity 55–60%, 12-hour light/dark cycle), with ad libitum access to food and water. Group II animals were protected from direct sunlight exposure to prevent endogenous synthesis of vitamin D₃.

Ethical statement. All procedures were carried out in accordance with bioethical principles and international standards for the use of laboratory animals (EU Directive 2010/63/EU), including the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, France; 1986) and the Guidelines for Bioethical Evaluation of Preclinical and Other Scientific Research Conducted on Animals (Kyiv, Ukraine; 2006).

Induction of vitamin D₃ deficiency. To induce vitamin D₃ deficiency, animals in groups II and III were fed a rachitogenic diet devoid of vitamin D₃ for 60 days before mating [16]. To confirm the development of vitamin D₃ deficiency and to monitor its status before mating and after the experimental endpoint, blood samples were collected from the tail vein and serum levels of 25-hydroxyvitamin D₃ (25(OH)D₃) were measured using a General 25-Hydroxyvitamin D₃ (HVD₃) ELISA kit (UNDL00047, Assay Genie). Sample concentrations were calculated using the Gain Data® (arigo's ELISA Calculator).

Mating and pregnancy monitoring. After confirming vitamin D₃ deficiency, the females were mated with fertile males at a male-to-female ratio of 2:1 (male : female). The presence of spermatozoa in vaginal smears the next morning was used to define gestational day 1 (GD1).

Induction of preeclampsia. To model L-NAME-induced preeclampsia (PE), each experimental group was subdivided into subgroups A and B. Subgroup A animals continued their respective dietary regimens. Rats in subgroup B received subcutaneous injections of L-NAME (N ω -nitro-L-arginine methyl ester, 100 mg/kg body weight) on GD5 and GD7. From GD5 to GD15, the drug was administered via drinking water at a dose of 40 mg/kg/day.

Blood pressure measurement. Systolic blood pressure was assessed using the CODA® Monitor (Kent Scientific, USA), a non-invasive tail-cuff system based on volume pressure recording (VPR). Measurements were taken twice: on GD1 (baseline)

and GD15 (after 10 days of L-NAME treatment). During measurement, rats were placed in restrainers on a thermostatic platform ($37\text{--}38^\circ\text{C}$) to ensure stable blood flow. The system recorded 3–5 readings within 5–7 min to minimize variability.

Assessment of proteinuria. Proteinuria was assessed using semi-quantitative urine test strips “Pro-totesT” (Norma, Ukraine). On GD15 of preeclampsia modeling, proteinuria was assessed by placing the rats in metabolic cages with free access to water to minimize stress. The indicator zone was moistened with a urine sample and assessed after 3 minutes by comparing color changes against a standard scale. The measurement range was 0–2.0 g/l.

Statistical analysis. Data were analyzed using conventional methods of variation statistics, with the calculation of the $M \pm m$. Statistical significance of differences between groups was assessed using one-way ANOVA followed by Bonferroni post hoc correction. Differences were considered significant at $P < 0.05$. Statistical processing was performed using Origin Lab 8.5 software.

Results and Discussion

The results of serum 25(OH)D₃ levels – recognized as the primary marker of vitamin D₃ status and a precursor of its hormonally active form – demonstrated significant intergroup differences in pregnant Wistar rats (Table 2). In the control group, which received adequate vitamin D₃ intake, the highest serum levels of 25(OH)D₃ were recorded: 49.8 ± 2.6 ng/ml in subgroup I A and 40.8 ± 3.7 ng/ml in subgroup IB, corresponding to optimal vitamin D₃ status by widely accepted standards [5, 6].

In pregnant rats from subgroup IIA, which were subjected to dietary vitamin D₃ restriction, 25(OH)D₃ concentrations dropped to 17.0 ± 1.1 ng/ml, indicating a pronounced deficiency. Such a deficiency is typically associated with disturbances in calcium-phosphate metabolism and may increase the risk of gestational complications [7, 8]. In contrast, subgroup IIIA, which received cholecalciferol in an oil-based formulation to correct deficiency, showed a more than twofold increase in serum 25(OH)D₃ levels (35.3 ± 2.2 ng/ml), demonstrating a high degree of therapeutic efficacy. The choice of lipid-based over water-soluble formulations was based on evidence from the literature, particularly studies by Durá-Travé T. et al. [17] and Wagner C.L. et al. [18], which emphasize the superior bioavailability of oil-based vitamin D₃.

Table 2. Serum 25(OH)D₃ levels in the studied rats ($M \pm m$)

Indicator	Control (Group I), $n = 10$		D ₃ Hypovitaminosis (Group II), $n = 13$		D ₃ Hypovitaminosis + Vitamin D ₃ (Group III), $n = 14$	
	IA (without PE), $n = 5$	IB (PE), $n = 5$	IIA (without PE), $n = 7$	IIB (PE), $n = 6$	IIIA (without PE), $n = 7$	IIIB (PE), $n = 7$
25(OH)D ₃ , ng/ml	49.8 ± 2.6	40.8 ± 3.7*	17.0 ± 1.1*^	–	35.3 ± 2.2*#	30.5 ± 1.2*#&^

Note. *Statistically significant difference compared to Group IA ($P < 0.05$); #statistically significant difference compared to Group IIA ($P < 0.05$); &statistically significant difference compared to Group IIIA ($P < 0.05$); ^statistically significant difference compared to Group IB ($P < 0.05$)

Importantly, 25(OH)D₃ levels were consistently higher in all subgroup A animals compared to their subgroup B counterparts, which were exposed to the nitric oxide synthase inhibitor L-NAME to model preeclampsia (e.g., 49.8 ± 2.6 ng/ml in IA vs. 40.8 ± 3.7 ng/ml in IB; 35.3 ± 2.2 ng/ml in IIIA vs. 30.5 ± 1.2 ng/ml in IIIB; $P < 0.05$). This may reflect both a direct impairment of cholecalciferol hydroxylation and an indirect effect of L-NAME-induced hypertension and impaired microcirculation on vitamin D metabolism, ultimately resulting in reduced synthesis of the vitamin D₃ prohormone. Rats in subgroup IIB, which experienced both vitamin D₃ deficiency and L-NAME-induced preeclampsia, did not survive. The high mortality rate in this subgroup may be attributable to inadequate physiological adaptation to L-NAME in the context of vitamin D₃ deficiency, late initiation and insufficient duration of vitamin D therapy, or the need for higher doses – issues that warrant further investigation.

Moreover, the lowest serum 25(OH)D₃ levels were observed in subgroup IIA compared to all other groups ($P < 0.05$), confirming the effectiveness of the model in inducing vitamin D₃ deficiency and validating the correction potential of cholecalciferol.

In summary, the obtained data indicate a significant influence of L-NAME on vitamin D₃ bioavailability as assessed by serum 25(OH)D₃. High-dose cholecalciferol supplementation in the setting of preeclampsia modeling may have practical implications for enhancing pharmacological strategies against this pregnancy complication, particularly when vitamin D₃ deficiency is present as a contributing pathophysiological factor.

In the next phase of the experiment, we assessed blood pressure dynamics in pregnant rats with different vitamin D₃ statuses and in the presence or absence of preeclampsia modeling. Blood pressure was measured on gestational day 1 (GD1) to deter-

mine baseline hemodynamic status, and repeated on GD15 – a critical period for fetoplacental development and for evaluating the preeclampsia model.

At the onset of pregnancy, both systolic (SBP) and diastolic (DBP) blood pressure values were within physiological norms across all groups (Table 3), with SBP ranging from 115.20 ± 4.48 to 122.60 ± 1.34 mmHg and DBP from 70.80 ± 3.26 to 78.70 ± 1.25 mmHg. This homogeneity in baseline values indicates the absence of systemic hypertension prior to experimental interventions, allowing for a valid assessment of each factor's contribution (vitamin D₃ deficiency, preeclampsia, vitamin D₃ supplementation) to hemodynamic changes.

By GD15, blood pressure varied significantly among groups, depending on vitamin D₃ status and preeclampsia modeling. In control rats (subgroup IA), blood pressure remained stable throughout pregnancy, confirming the protective role of adequate vitamin D₃ in maintaining normotension during gestation. In contrast, subgroup IIA animals, with vitamin D₃ deficiency, showed a moderate but statistically significant increase in SBP (132.00 ± 2.31 mmHg) and DBP (88.90 ± 2.04 mmHg) by GD15, even in the absence of additional stressors. This suggests that vitamin D₃ deficiency may act as an independent risk factor for gestational hypertension, potentially mediated by decreased bioavailability of endothelial NO, activation of the renin-angiotensin-aldosterone system (RAAS), and heightened sensitivity to vasoconstrictors [19, 20].

In subgroup III A, cholecalciferol-treated rats with prior D₃ deficiency maintained normotensive levels (SBP: 118.50 ± 4.27 mmHg; DBP: 77.00 ± 1.69 mmHg), further supporting the hypothesis that low vitamin D₃ status contributes directly to pregnancy-associated hypertension. The antihypertensive effect of vitamin D₃ may be mediated through vitamin D₃ receptor (VDR)-dependent

Table 3. Dynamics of blood pressure changes in pregnant rats at different gestational stages ($M \pm m$)

Blood pressure, mmHg	Control (Group I), $n = 10$		D ₃ hypovitaminosis (Group II), $n = 13$		D ₃ hypovitaminosis + vitamin D ₃ (Group III), $n = 14$	
	IA (without PE), $n = 5$	IB (PE), $n = 5$	IIA (without PE), $n = 7$	IIB (PE), $n = 6$	IIIA (without PE), $n = 7$	IIIB (PE), $n = 7$
<i>Blood pressure on GD1</i>						
SBP	120.60±0.87	112.60±3.57*	121.30±1.55	122.60±1.34	120.50±2.55	115.20±4.48
DBP	77.60±1.94	70.80±3.26*	78.40±1.34	78.70±1.25^	78.00±2.01^	74.80±4.23
<i>Blood pressure on GD15</i>						
SBP	110.20±4.56	163.80±2.76*	132.00±2.31*^	—	118.50±4.27#^	174.70±5.11*#&^
DBP	69.60±4.01	105.20±3.57*	88.90±2.04*^	—	77.00±1.69*#^	118.00±5.67*#&^

Note. *Statistically significant difference vs. Group IA ($P < 0.05$); #statistically significant difference vs. Group IIA ($P < 0.05$); &statistically significant difference vs. Group IIIA ($P < 0.05$); ^statistically significant difference vs. Group IB ($P < 0.05$)

anti-inflammatory pathways and improved vascular homeostasis. These effects may include downregulation of angiotensin II type 1 receptors (AT1R), improved NO bioavailability, and upregulation of pro-angiogenic factors such as VEGF and placental growth factor (PlGF) [21, 22]. This is consistent with other studies showing that vitamin D₃ reduces the expression of the antiangiogenic protein sFlt-1 (soluble fms-like tyrosine kinase-1), improves trophoblast invasion, and modulates RAAS function [23, 24].

In subgroup IIIB, which received L-NAME in addition to cholecalciferol therapy, blood pressure rose markedly (SBP: 174.70 ± 5.11 mmHg; DBP: 118.00 ± 5.67 mmHg). Notably, 100% mortality was observed in subgroup IIB (vitamin D₃ deficiency + L-NAME), likely due to acute hemodynamic instability. These findings suggest a synergistically detrimental effect of vitamin D₃ deficiency and preeclampsia-associated pathophysiological changes – particularly immune activation and endothelial dysfunction. Literature indicates that vitamin D₃ deficiency exacerbates immune dysregulation, inflammation, and oxidative stress, all of which contribute to angiogenic imbalance, a hallmark of preeclampsia pathogenesis [25, 26]. Additionally, vitamin D₃ deficiency impairs the synthesis of nitric oxide, a key vasodilator. This effect may amplify vascular tone when combined with the NO synthase inhibition induced by L-NAME [27].

It is noteworthy that preeclampsia modeling in control animals (subgroup IB) also led to a rise in blood pressure (SBP: 163.80 ± 2.76 mmHg; DBP:

105.20 ± 3.57 mmHg); however, these values were significantly lower than those observed in subgroup IIIB ($P < 0.05$). This suggests a partial attenuation of the adverse effects of preeclampsia under conditions of optimal vitamin D₃ status.

Our findings indicate that hypovitaminosis/deficiency of vitamin D₃ can be considered an independent risk factor for the development of gestational hypertension. Its insufficiency markedly exacerbates the hypertensive response in animals with L-NAME-induced preeclampsia. Vitamin D₃ supplementation resulted in a statistically significant reduction in blood pressure parameters, supporting its potential role as both a preventive and therapeutic agent in the management of preeclampsia.

Arterial hypertension may be closely linked to renal dysfunction and activation of the RAAS, while at the same time contributing to kidney injury and the development of renal failure. Proteinuria is a key marker of renal impairment and is widely used to assess the progression of nephropathies, including those associated with preeclampsia, which is often accompanied by glomerular filtration disturbances. The results of proteinuria assessment revealed marked intergroup differences, reflecting the functional status of the kidneys in pregnant rats depending on their vitamin D₃ status and hemodynamic alterations.

As shown in Figure, no proteinuria was detected in the urine of pregnant control females (subgroup IA), indicating the absence of structural kidney pathology in animals with sufficient serum

25(OH)D₃ levels. This underscores the importance of maintaining optimal vitamin D₃ status during pregnancy to preserve renal health [9]. In contrast, in control subgroup IB, 66.7% of the animals exhibited proteinuria at a level of 1.0 g/l, and 33.3% at 2.0 g/l – an indication of progressive nephropathy caused by L-NAME-induced preeclampsia, even in the presence of relatively adequate vitamin D₃ supply. This clearly highlights the multifactorial nature of preeclampsia, in which suboptimal vitamin D₃ status represents only one of many contributing pathophysiological mechanisms [10].

Given that 25(OH)D₃ concentrations were still significantly lower in preeclamptic control rats compared to healthy pregnant controls, it is plausible that higher-dose cholecalciferol supplementation could have provided more pronounced antihypertensive and nephroprotective effects in these animals.

In pregnant females of subgroup IIA, who did not receive correction for vitamin D₃ deficiency, proteinuria levels ranged from 0.15 to 0.5 g/l. Nearly half of the animals (42.9%) exhibited proteinuria levels of 0.3 g/l, indicative of moderate glomerular filtration impairment. This may represent the early stages of nephropathy, which could potentially progress to more severe forms in the context of vitamin D₃ deficiency [6]. As previously noted, the females in subgroup IIB, who were administered L-NAME, did not survive the experiment. This likely reflects significant pathophysiological disturbances

in the organism, including possible renal involvement, which may have contributed to mortality.

In rats with partially corrected vitamin D₃ deficiency through cholecalciferol supplementation (subgroup IIIA), a moderate reduction in proteinuria levels was observed compared to the deficiency subgroup (IIA), correlating with reduced hypertension levels. The vast majority of animals in subgroup IIIA (85.7%) exhibited proteinuria around 0.15 g/l, supporting the potential nephroprotective effect of vitamin D₃. However, in the subgroup with induced preeclampsia against the background of vitamin D₃ correction (IIIB), proteinuria levels remained elevated: nearly 57% of the animals had urinary protein concentrations of 2.0 g/l. The degree of proteinuria in this subgroup exceeded that of the respective preeclamptic control animals (40%), aligning with higher blood pressure values and incomplete restoration of 25(OH)D₃ levels compared to the control. Thus, the observed intergroup differences demonstrate a clear relationship between the severity of arterial hypertension and proteinuria and serum 25(OH)D₃ levels under conditions of L-NAME-induced preeclampsia.

Overall, our results confirm that vitamin D₃ deficiency is associated with more severe renal injury in preeclampsia. This aligns with numerous clinical and experimental studies indicating that vitamin D₃ deficiency can disrupt glomerular filtration barrier function, increasing urinary protein excretion [28].

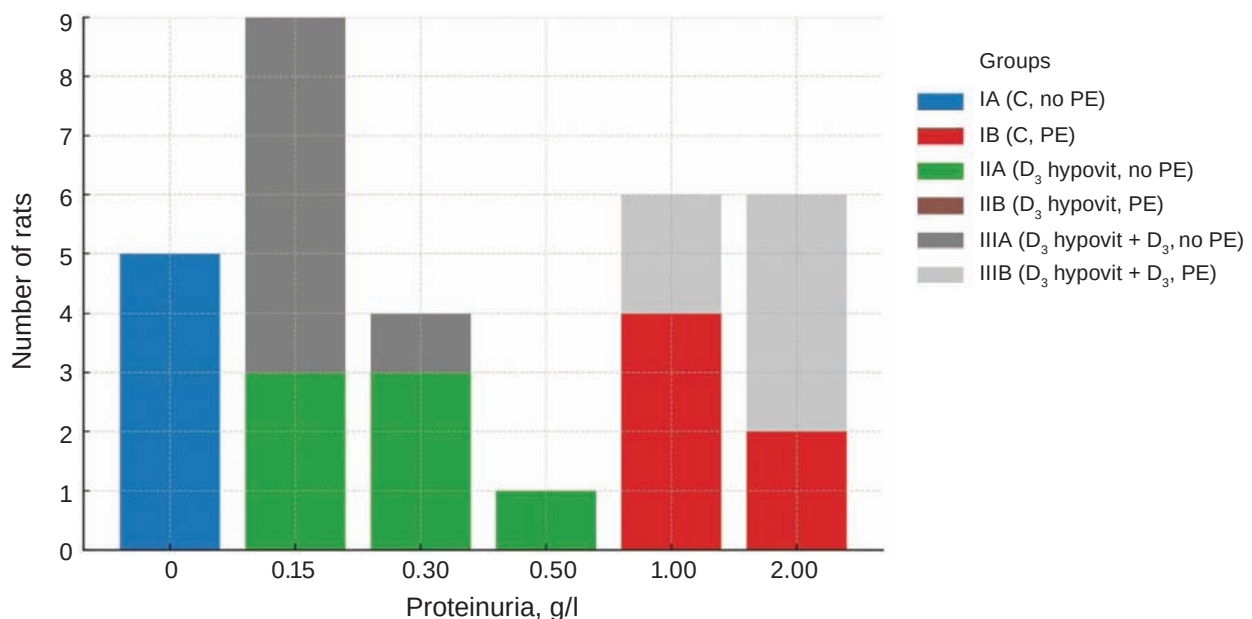


Figure. Distribution of female rats by level of proteinuria in the studied groups

Vitamin D₃ is believed to exert a protective effect on glomerular endothelial cells by reducing inflammation and oxidative stress – key pathogenic mechanisms in the development of preeclampsia. Specifically, low levels of vitamin D₃ have been shown to correlate with increased concentrations of inflammatory markers such as interleukin-6 and C-reactive protein, which may contribute to endothelial dysfunction and vascular tone dysregulation [29]. Vitamin D₃ insufficiency/deficiency has also been associated with reduced activity of antioxidant enzymes, exacerbating pro-oxidant processes and oxidative renal damage [30].

The data obtained underscore the need for routine monitoring of vitamin D₃ status in pregnant women and for its timely correction as a potential preventive measure against preeclampsia and its complications. Recent studies suggest the preventive effectiveness of vitamin D₃ supplementation in pregnant women at high risk for preeclampsia, with benefits for both maternal and fetal outcomes [31, 32].

Conclusions. The results indicate that vitamin D₃ deficiency in pregnant rats is associated with a significant increase in blood pressure and the development of proteinuria, implicating its involvement in the pathogenesis of preeclampsia. Administration of cholecalciferol contributed to normalization of hemodynamic parameters and kidney damage. The highest blood pressure values and proteinuria levels were recorded in animals with combined vitamin D₃ deficiency and induced preeclampsia, suggesting a synergistic effect of these factors. Therefore, the findings support the pathogenic role of vitamin D₃ deficiency in the development of gestational complications and highlight the potential of vitamin D₃ as a preventive and pathogenetic therapeutic agent in preeclampsia.

Practical implications. Routine monitoring of vitamin D₃ levels in pregnant women at high risk for preeclampsia is important for the prevention of vascular and renal complications. Correction of vitamin D₃ status may be an effective strategy to prevent preeclampsia and its complications, including hypertension and proteinuria, as demonstrated by the findings of this study and recent clinical research.

Conflict of interest. Authors have completed the Unified Conflicts of Interest form at http://ukr-biochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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ВІТАМІН-D₃ СТАТУС ЯК ВИЗНАЧАЛЬНИЙ ЧИННИК РОЗВИТКУ ЕКСПЕРИМЕНТАЛЬНОЇ ПРЕЕКЛАМПСІЇ У САМИЦЬ ЩУРІВ ЛІНІЇ WISTAR

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Дефіцит вітаміну D₃ під час вагітності є поширеною проблемою, що асоціюється з підвищеним ризиком ускладнень, зокрема преєклампсії (ПЕ) – серйозного стану, який характеризується артеріальною гіпертензією з протеїнурією. Метою цього дослідження було вивчення частоти розвитку експериментальної преєклампсії у вагітних щурів у залежності від забезпеченості вітаміном D₃. Самки щурів лінії Wistar віком 8 тижнів були розподілені на три експериментальні групи: контроль; дефіцит вітаміну D₃ протягом 60 днів до спаровування; дефіцит вітаміну D₃ з пероральною корекцією вітаміном D₃ (1000 МО/кг маси тіла) за два тижні до спаровування. Аналізувалися підгрупи з індукованою та неіндукованою ПЕ. Преєклампсію моделювали шляхом введення N^ω-нітро-L-аргінін метилового естеру (L-NAME). Рівень вітаміну D₃ у крові визначали за допомогою набору ELISA для 25-гідроксिवітаміну D₃. Протеїнурію оцінювали напівкількісними тест-смужками для сечі «Прототест». Найвищі показники артеріального тиску та рівня протеїнурії були зафіксовані у тварин із поєднаним дефіцитом вітаміну D₃ та індукованою преєклампсією. Введення вітаміну D₃ сприяло нормалізації гемодинамічних показників і функції нирок, що вказує на важливість адекватного вітамін-D₃ статусу для нормального перебігу вагітності та профілактики ПЕ.

Ключові слова: дефіцит вітаміну D₃, L-NAME, преєклампсія, артеріальний тиск, рівень протеїнурії.

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