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Effects of Dietary Vitamin D Deficiency on Serum Ghrelin and Leptin Levels in New Zealand White Rabbits

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HIGHLIGHTS

- Energy balance may be linked to vitamin D status.
- Vitamin D plays a regulatory role in metabolic health.

Abstract

Ghrelin, an orexigenic hormone, promotes energy intake by stimulating appetite, whereas leptin, an anorexigenic hormone, plays a pivotal role in maintaining energy homeostasis by suppressing food consumption. The present study aimed to evaluate whether a 24-week vitamin D-deficient diet induces vitamin D deficiency in New Zealand White rabbits and to assess the associated changes in serum ghrelin and leptin levels. A total of sixteen two-month-old New Zealand White rabbits were included in the study. Following a 15-day acclimatization period, the animals were randomly divided into two groups, each consisting of eight rabbits. Group II received a standard rabbit diet supplemented with vitamin D, whereas Group I was fed a vitamin D–deficient diet for a period of 24 weeks. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured as 22.15 ± 1.70 ng/mL in Group I and 44.20 ± 6.09 ng/mL in Group II, confirming the successful induction of vitamin D deficiency. Serum ghrelin levels were found to be 0.13 ± 0.02 ng/mL in Group I and 0.24 ± 0.04 ng/mL in Group II. Similarly, serum leptin levels were measured at 2.39 ± 0.07 ng/mL in Group I and 2.69 ± 0.09 ng/mL in Group II. In Group 2, 25(OH)D, ghrelin and leptin levels were found to be statistically significantly higher than in Group 1 ($p < 0.05$).These findings suggest that vitamin D deficiency is associated with alterations in both ghrelin and leptin concentrations.

Keywords: Vitamin D; Ghrelin; Leptin; Energy Homeostasis; Appetite Control

1. Introduction

Vitamin D is one of the oldest hormone-like molecules in the world and plays a role in fundamental biological processes in virtually all living organisms, from phytoplankton to humans. Although it has long been considered solely in the context of skeletal health due to its critical role in regulating calcium and phosphorus homeostasis and in bone mineralization (Janoušek et al. 2022; Hao et al. 2023, Wimalawansa 2023), in recent years it has been demonstrated that it has multifaceted effects on immune system modulation, cell

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proliferation and differentiation, inflammation control, and metabolic health (Charoenngam and Holick 2020; Wimalawansa 2024). The expression of the vitamin D receptor (VDR) in tissues associated with energy metabolism, such as adipose tissue, the pancreas, the liver and the hypothalamus, has demonstrated that this molecule acts as a critical regulator not only of mineral metabolism but also of energy homeostasis. In particular, its ability to stimulate energy expenditure by increasing the expression of uncoupling proteins (UCPs) in adipocytes supports the role of vitamin D in body weight control (Rahme et al. 2024; Voiculescu et al. 2025). These findings suggest that vitamin D may exert regulatory effects on central and peripheral mechanisms controlling appetite and energy balance. The adverse health effects of vitamin D deficiency span a very broad spectrum. In addition to bone disorders such as rickets in children and osteomalacia and osteoporosis in adults, it has been reported to be associated with sarcopenia in older adults, immune system dysfunction (Kupisz-Urbańska et al. 2021) autoimmune diseases, diabetes, insulin resistance, obesity, hypertension, cardiovascular diseases and various types of cancer (Amrein et al. 2020; Christakos et al. 2019; Remelli et al. 2019; Pál et al. 2023). The role of vitamin D in energy metabolism is particularly noteworthy due to its relationship with the hormone's ghrelin and leptin. While ghrelin increases energy intake through its appetite-stimulating properties, leptin plays a balancing role in energy homeostasis through its appetite-suppressing effect. Vitamin D may influence this axis through VDR-mediated signaling pathways in the hypothalamus and peripheral metabolic tissues, thereby modulating appetite and energy expenditure (Sisley et al. 2016; *Su et al. 2021)* In the literature, the relationship between vitamin D and these hormones has yielded variable results across different populations. Some studies have reported that vitamin D supplementation increases ghrelin levels (Hajimohammadi et al. 2017; AlGhamdi et al. 2024), while its effects on leptin levels vary depending on individual factors (Mousa et al. 2020). Studies in animals have also shown that this relationship is species-specific and dependent on experimental conditions (Menendez et al. 2001; Kaneko et al. 2015). However, no study has systematically evaluated the temporal development of vitamin D deficiency and its direct effects on the ghrelin–leptin axis in New Zealand White rabbits. Research into vitamin D metabolism in rabbits has shown that severe deficiencies are common, particularly in animals housed indoors. while a drop in serum 25(OH)D levels below 17 ng/mL in domestic rabbits leads to significant changes in parathyroid hormone secretion and bone density (Mäkitaipale et al. 2020), a study conducted on wild rabbits found that average serum levels were only 3.3 ng/mL (Mäkitaipale et al. 2024). This finding demonstrates that vitamin D deficiency leads to metabolic and hormonal disorders in rabbits. Rabbits are widely used as experimental models due to their well-characterized metabolism, ease of handling, and relevance to human physiology, as well as their sensitivity to dietary vitamin D changes (Esteves et al. 2018).

These findings indicate that vitamin D plays a significant regulatory role not only in bone metabolism but also in appetite control and metabolic health. Therefore, this study aimed to determine the time required for the development of vitamin D deficiency in New Zealand White rabbits fed a vitamin D-deficient diet and to investigate its effects on energy balance via the ghrelin–leptin axis. Understanding the progression of vitamin D deficiency over time is of vital importance for interpreting its metabolic and hormonal consequences; this study puts forward the hypothesis that vitamin D deficiency reduces ghrelin and leptin levels, thereby contributing to an imbalance in energy homeostasis.

2. Materials and Methods

2.1. Experimental animals and housing conditions.

This study was conducted following approval from the Kafkas University Animal Experimentation Ethics Committee (KAÜ-HADYEK/2016-114). The study utilized 16 two-month-old New Zealand White rabbits. The animals were obtained from the Ondokuz Mayıs University Centre for Laboratory Animal Application and Research and housed at the Kafkas University Centre for Laboratory Animal Application and Research.

After a 15-day adaptation period, the animals were randomly divided into two groups (n=8):

Group I: Fed a rabbit pellet diet without vitamin D for 24 weeks

Group II: Fed a standard rabbit pellet diet supplemented with vitamin D for 24 weeks.

Housing conditions were kept constant: a 12-hour light/12-hour dark cycle, a temperature of 21–23 °C and humidity of 55–65%. Feed and drinking water were provided *ad libitum*. Cages were cleaned daily, and feeders and waterers were regularly disinfected.

The nutritional compositions of the diets are summarised in Table 1. Vitamin D supplementation was added to the rabbit feed at a rate of 3000 IU/kg.

Table 1. Rabbit Feed Composition

Component	Standard Feed	Vitamin D–Deficient Feed
Dry Matter	90%	90%
Crude Protein	18%	18%
Raw Cellulose	11.20%	11.20%
Raw Ash	6.80%	6.80%
Crude Oil	4.50%	4.50%
Calcium	1%	1%
Phosphorus	0.62%	0.62%
Lysine	0.98%	0.98%
Methionine	0.58%	0.58%
Sodium	0.24%	0.24%
Energy (kkal/ kg)	2650	2650
Vitamin D3 (IU /kg)	3000	—

2.2. *Blood Samples and Biochemical Analyses*

Blood samples were collected only once at the end of the 24-week experimental period. The samples were centrifuged at 3000 rpm for 10 minutes to separate the serum, which was then stored at –20 °C until analysis. Serum 25-hydroxyvitamin D [25(OH)D], ghrelin, and leptin levels were measured using commercial ELISA kits (USCN Life Science Inc., Wuhan, China; Catalog No: [UCEA920Ge] for 25(OH)D; detection range; 4.94-400ng/mL, Sensitivity: the minimum detectable dose of this kit is typically less than 1.71ng/mL, EIAab Science Co., Wuhan, China; Catalog No: [UMEA991Ra] for ghrelin detection range: 123.5-10,000pg/mL, Sensitivity; The minimum detectable dose of this kit is typically less than 51.5pg/mL, and leptin Catalog No: [USEA084Hu]), detection range; 0.156-10ng/mL, Sensitivity; The minimum detectable dose of this kit is typically less than 0.058ng/mL, according to the manufacturers' instructions.

2.3. *Statistical Analysis*

The data were analysed using SPSS 20.0 software (SPSS Inc., Chicago, IL, USA). Differences between the two groups were analyzed after assessing the normality of the data using the Shapiro–Wilk test; an independent samples t-test was applied for normally distributed data, whereas the Mann–Whitney U test was used for data that did not follow a normal distribution. Results are expressed as mean ± standard error ($\bar{x}$ ± SE). A p-value of <0.05 was considered statistically significant.

3. Results and Discussion

The parameters measured in the two groups (n = 8 each) are shown in Table 2. Serum 25(OH)D level: 22.15 ± 1.70 ng/mL in Group 1, 44.20 ± 6.09 ng/mL in Group 2 ($p < 0.05$). Serum ghrelin level: 0.13 ± 0.02 ng/mL in Group 1, 0.24 ± 0.04 ng/mL in Group 2 ($p < 0.05$). Serum leptin level: 2.39 ± 0.07 ng/mL in Group 1, 2.69 ± 0.09 ng/mL in Group 2 ($p < 0.05$). In Group 2, 25(OH)D, ghrelin and leptin levels were found to be statistically significantly higher than in Group 1 ($p < 0.05$). The findings suggest that vitamin D deficiency exerts an effect on endocrine hormones that regulate the appetite mechanism.

Table 2. Serum 25(OH)D and hormone levels in Group I and Group II

Traits	n	Group 1 $\bar{X} \pm S_{\bar{X}}$	Group 2 $\bar{X} \pm S_{\bar{X}}$	p-value
25(OH)D (ng/mL)	8	22.15 ± 1.70	44.20 ± 6.09	<0.05
Ghrelin (ng/mL)	8	0.13 ± 0.02	0.24 ± 0.04	<0.05
Leptin (ng/mL)	8	2.39 ± 0.07	2.69 ± 0.09	<0.05

n: Number of animals, $\bar{X}$: Mean, $S_{\bar{X}}$: Standart error

This study has demonstrated the effects of vitamin D deficiency on serum 25(OH)D, ghrelin and leptin levels in New Zealand White rabbits and has supported the critical role of vitamin D in energy homeostasis and endocrine regulation. According to our findings, 25(OH)D levels in rabbits fed a vitamin D-deficient diet showed a significant decrease compared to Group II. (22.15 ± 1.70 ng/mL vs. 44.20 ± 6.09 ng/mL; $p<0.05$). However, the relatively small sample size (n=8 per group) may limit statistical power and generalizability. In parallel with this decline, there was a decrease in ghrelin levels (0.13 ± 0.02 vs. 0.24 ± 0.04 ng/mL; $p<0.05$) and a more modest but significant decrease in leptin levels (11%, 2.39 ± 0.069 vs. 2.69 ± 0.09 ng/mL; $p<0.05$) were observed. Additionally, food intake, body weight, and overall energy balance were not assessed, which could influence the observed hormonal changes. The marked decrease in ghrelin levels in Group I supports the regulatory role of vitamin D in appetite and energy metabolism. Our findings are consistent with the study by Biercewicz et al. (2023), which demonstrated an association between low vitamin D levels (<20 ng/mL) and low ghrelin levels in geriatric women. Similarly, Hajimohammadi et al. (2017) reported that vitamin D supplementation significantly increased ghrelin levels in patients with major depressive disorder, and AlGhamdi et al. (2024) reported the same in individuals with type 2 diabetes. These data suggest that ghrelin production in the gastric mucosa may be regulated via the vitamin D receptor (VDR) and that vitamin D deficiency may suppress appetite mechanisms.

Although leptin levels were reduced in Group I in our study, this relationship has been reported inconsistently in the literature. Juliaty et al. (2022) and Madhu et al. (2022) reported that low vitamin D levels are associated with higher leptin levels in obese individuals. In contrast, animal models and cell culture studies have shown that vitamin D exerts varying effects on leptin production. Kong et al. (2013) reported that $1,25(OH)_2D_3$ supplementation increased leptin expression in mice, whereas Menendez et al. (2001) and Kaneko et al. (2015) reported that it suppressed it. Dinca et al. (2016) and AlGhamdi et al. (2024) reported that supplementation did not result in a significant change in leptin levels, while Mousa et al. (2020) and Hajimohammadi et al. (2017) reported an increase in certain specific clinical conditions. These conflicting findings suggest that leptin synthesis is regulated differently depending on species-specific mechanisms and experimental conditions.

The fact that vitamin D deficiency leads to a reduction in ghrelin and leptin levels supports its systemic effects beyond its role in appetite and energy metabolism. Rahme et al. (2024) demonstrated that vitamin D promotes energy expenditure and lipolysis by increasing the expression of the mitochondrial uncoupling protein (UCP). Mäkitaipale et al. (2020; 2024) noted that low 25(OH)D levels in rabbits are associated with increased parathyroid hormone (PTH) secretion and impaired bone mineralization.

The low vitamin D levels observed in our study were found to be consistent with these metabolic abnormalities. Furthermore, it has been demonstrated that vitamin D deficiency in humans is associated with an increased risk of diabetes, insulin resistance, obesity (Berridge 2017), hypertension and cardiovascular diseases (Pál et al. 2023), as well as certain types of cancer (De Martinis et al. 2021). Finally, species-specific differences should be considered when extrapolating these findings to humans, and the observed endocrine changes may also reflect broader metabolic and immunomodulatory consequences of vitamin D deficiency.

Our findings suggest that these systemic effects may also be explained via the ghrelin–leptin axis. Furthermore, the effects of vitamin D deficiency on the immune system should not be overlooked. Charoenngam and Holick (2020) noted that vitamin D plays a critical role in modulating the immune system, suppressing pro-inflammatory cytokines while enhancing the anti-inflammatory response. The changes in ghrelin and leptin levels observed in our study may indicate an indirect interaction with these

immunomodulatory effects. Particularly given leptin's close association with the immune response, it can be suggested that vitamin D deficiency may have both metabolic and immunological implications. Future studies with larger sample sizes, mechanistic molecular analyses, and controlled energy balance assessments are warranted to further elucidate these effects.

4. Conclusion

The homogeneous genetic makeup of New Zealand White rabbits and their endocrine response profiles, which are similar to those of humans, enhance the translational value of our study. This study demonstrated that feeding a diet deficient in vitamin D significantly reduced serum 25(OH)D levels in New Zealand White rabbits and that this reduction was associated with a significant suppression of ghrelin and leptin levels. Although the leptin decrease was at a low level, it may still reflect meaningful endocrine alterations. Furthermore, it was found that feeding rabbits a vitamin D-deficient pellet diet for 24 weeks was sufficient to induce vitamin D deficiency. The findings demonstrate that vitamin D plays a critical role not only in skeletal metabolism but also in energy homeostasis and endocrine regulation. Our findings suggest that the ghrelin–leptin axis may be modulated by vitamin D and that vitamin D deficiency may play a role in the pathogenesis of obesity, metabolic syndrome and endocrine disorders.

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