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Research Article

Impact of Antidiabetic Medications on Bone Remodeling Markers and Bone Structure in Type 2 Diabetic Wistar Rats

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ABSTRACT

Background: The presence of impaired bone quality in the presence of normal or even elevated bone mineral density (BMD) has been increasingly recognized as a feature of Type 2 diabetes mellitus (T2DM) and is thought to be due to changes in bone remodeling.

Objective: To assess the effect of the commonly used antidiabetic drugs on serum bone remodeling markers and bone histomorphometric structure in Wistar rats with type 2 diabetes mellitus.

Materials and Methods: The experimental animal study was conducted at Faisalabad Medical University Faisalabad, Punjab, from August 2023 to January 2024. The animals were divided into 6 equal groups of 10 animals each: Group I (Normal Control); Group II (Diabetic Control); Group III (Diabetic + Metformin); Group IV (Diabetic + Pioglitazone); Group V (Diabetic + Sitagliptin); Group VI (Diabetic + Insulin). SPSS version 25 was used for data analysis, and p-values <0.05 were considered statistically significant.

Results: Diabetic control rats had significantly lower levels of osteocalcin and higher levels of CTX than normal controls ($p<0.001$), suggesting reduced bone formation and enhanced bone resorption. The bone marker profile was least favorable in the pioglitazone group and most favorable in the Insulin and Metformin groups, indicating improvement toward normal control values.

Conclusion: Bone remodeling markers and bone microarchitecture were significantly altered in Wistar rats with untreated type 2 diabetes.

Keywords: Type 2 Diabetes Mellitus, Bone Remodeling, Osteocalcin, Ctx, Antidiabetic Drugs, Wistar Rats.

INTRODUCTION

There is growing recognition that type 2 diabetes mellitus is a metabolic disease that can have significant, and sometimes overlooked, implications for skeletal health. Al-Shmmari and Al-Hadrawy investigated the effect of antidiabetic drugs on bone remodeling biomarkers and bone histological structure in diabetic Wistar rats and revealed that the diabetic state and the type of antidiabetic treatment had a significant effect on bone turnover markers and bone histological structure (1). In type 2 diabetes mellitus rats, Wu et al. evaluated the effects of verapamil on bone mass, microstructure, and mechanical properties. They observed that verapamil had measurable effects on several parameters of bone quality in the diabetic condition (2). Martiniakova et al. investigated the effects of chronic administration of the aldose reductase inhibitor cemtirestat on bone quality parameters in non-diabetic and streptozotocin-induced diabetic rats and observed that cemtirestat had a beneficial effect on several bone quality parameters in diabetic rats (3).

The effects of natural and pharmacological interventions on the skeleton have been studied in diabetic rodent models. Maksimović et al. investigated the effects of a polyherbal mixture on the microarchitecture of the femur and tibia in diabetic OP rats and reported that the polyherbal preparation had positive effects on several microarchitectural parameters compared with diabetic control rats (4). Kotb et al. investigated the possible effects of vitamin D and MSCs on reducing bone-remodeling changes in streptozotocin-induced diabetic rats, using functional, histological, and immunohistochemical studies, and reported that both treatment groups normalized bone-remodeling markers and histological indices (5). In a systematic review, Viggers et al. investigated the impact of incretin therapy on skeletal health in T2D and generally neutral to positive effects of incretin-based therapy on bone outcomes in the included studies (6).

Biochemical and structural aspects have also been studied, in particular those related to incretin-based therapies. Alhashem et al. investigated the impact of liraglutide on biochemical markers of bone turnover in rats treated with dexamethasone and concluded that liraglutide partially reduced the negative effects of dexamethasone on bone (7). Abdi et al. investigated the role of incretin pathway components in bone properties and observed that these agents had a measurable effect on bone biomechanical parameters, which were mostly beneficial (8). In an extensive review, Zhao et al. investigated the impact of hypoglycemic agents on the musculoskeletal system and found that different antidiabetic drug classes have varying effects, sometimes opposing, on bone and muscle health (9).

Diabetic bone models have also been used to investigate the effect of pharmacological and phytochemical interventions. Abd-Elrhman et al. reported that the effect of the rose hips (Rosa canina L.) extract on the development of osteoarthritis secondary to type 2 diabetes mellitus in rats treated with pioglitazone or vanadyl sulfate was that the pioglitazone treatment had a comparatively less favorable joint and bone profile than vanadyl sulfate, which was improved by rose hips supplementation (10). Ambomo et al. investigated the aqueous extract of Alstonia boonei in a diabetic rat model using both in vivo and in silico studies and showed that the extract improved several indices of bone health (11). In a systematic review, García-Rios et al. analyzed the effects of diabetes on orthodontic treatment and emphasized the clinical significance of the changes in bone remodeling in diabetic patients when undergoing orthodontic tooth movement (12).

The skeletal effects of GLP-1-based and other emerging therapies have been discussed and investigated in other settings. In a systematic review, Daniilopoulou et al. investigated the effects of GLP-1 agonists on bone metabolism in the included studies, which showed mostly favorable or neutral effects on bone turnover markers (13). Antimicrobial photodynamic therapy and systemic resveratrol were found to enhance peri-implant bone healing parameters in type 2 diabetic rats (14). Zofou et al. evaluated the effect of vitamin D co-administration with antidiabetic drugs in a streptozotocin-induced albino Wistar rat model and concluded that vitamin D improved metabolic effects and, consequently, skeletal effects of antidiabetic drugs (15).

Lastly, a few recent studies have focused on the wider molecular and systemic implications of diabetes management for the current project. Yousaf et al. examined the recent progress of plant-based nanotechnology and molecular understanding for the diagnosis and therapy of type 2 diabetes, providing new therapeutic insights that could eventually cross over to the skeletal arena (16). Basha et al. investigated the anti-inflammatory effect of semaglutide on diabetic rats with experimental periodontitis, and they observed that the treatment with semaglutide decreased the inflammatory mediators in the periodontal-bone interface in diabetic rats (17). The systemic and tissue-protective effects of some antidiabetic agents extending beyond glycemic control were illustrated by Kashmoola et al. in their study on adipokine dysregulation and oxidative stress in type 2 diabetes and its implications for neurodegeneration and the neuroprotective effect of antidiabetic therapies (18), while Odetayo et al. observed the effect of orange peel ethanolic extract and physical exercise on preventing testicular toxicity in streptozotocin and high-fat diet-induced type 2 diabetic rats through Nrf2/NF-κB signaling, further illustrating the broad tissue-protective potential of adjunctive antioxidant interventions in diabetic rodent models (19).

Although this growing body of evidence exists, there is still a lack of direct comparative data on the differential effects of commonly used antidiabetic drug classes, metformin, pioglitazone, sitagliptin, and insulin, on bone remodeling markers and histomorphometric

bone structure in the same experimental model. The present study was designed to assess the effect of the commonly used antidiabetic drugs on the serum bone remodeling markers and bone histomorphometric structure in a type 2 diabetes mellitus Wistar rat model to support the clinical relevance of selecting antidiabetic therapy with a favourable skeletal profile, especially in patients who are at risk of osteoporosis and fracture.

Objective: To assess the effect of frequently used antidiabetic agents on serum bone remodeling markers and bone histomorphometric structure in type 2 diabetes mellitus Wistar rat model.

MATERIALS AND METHODS

Study Design: Experimental animal study.

Setting: Faisalabad Medical University Faisalabad, Punjab.

Study Duration: From August 2023 to January 2024.

Sample Size: The total number of rats used was 60 (10 rats/group, 6 groups), which was adequate to detect a significant difference between groups with 80% power, based on the resource equation and previous similar experimental studies (ALShmmari and AL-Hadrawy).

Inclusion Criteria: Wistar rats were used and were healthy adult males (180-220g), and no visible signs of illness or skeletal deformity were observed at baseline.

Exclusion Criteria: Rats that died during the induction or treatment period, did not reach the diabetic threshold fasting glucose following streptozotocin injection, or developed severe illness or weight loss of more than 20% of their body weight were excluded from the final analysis.

Methods: After 2 weeks of acclimatization, five groups (Groups II-VI) were made diabetic by feeding a high-fat diet for 4 weeks and then a single injection of low-dose streptozotocin (35 mg/kg) into the peritoneum. Five groups (Groups II-VI) were made diabetic after 2 weeks of acclimatization to a high-fat diet for 4 weeks, followed by a single low-dose streptozotocin injection (35 mg/kg) into the peritoneum. Fasting blood glucose ≥200 mg/dL 72 hours after injection was confirmation of diabetes. Group I (normal control) was fed with a normal diet and vehicle injection only. After confirmation of diabetes, Group II was not subjected to any pharmacological treatment (diabetic control), Group III was given oral metformin (100 mg/kg/day), Group IV was given oral pioglitazone (10 mg/kg/day), Group V was given oral sitagliptin (10 mg/kg/day), and Group VI was given subcutaneous insulin (4 IU/kg/day) for 8 weeks. Rats were fasted overnight and at the end of the treatment period were put to sleep under general anesthesia, and blood was taken by cardiac puncture for the measurement of serum osteocalcin, CTX, and ALP using an ELISA kit. The two femurs were harvested, one of which was subjected to hematoxylin and eosin staining and then analyzed for trabecular bone volume (%) and cortical thickness (mm) under light microscopy. The data were analyzed with SPSS version 25. Mean ± standard deviation was used to report data on continuous variables. Means were compared between the 6 groups using one-way ANOVA with post-hoc Tukey test. A p-value of ≤0.05 was deemed significant.

RESULTS

The study included 60 rats, equally distributed among six groups, all of which finished the study. Baseline fasting blood glucose was similar between diabetic groups after streptozotocin induction.

Table 1: Baseline Body Weight and Fasting Blood Glucose across Study Groups (N = 60)

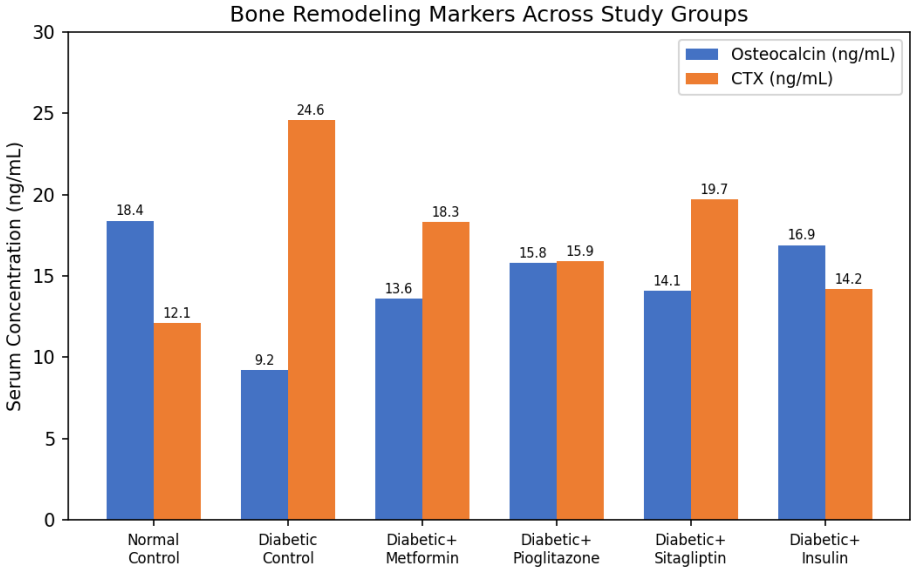
Group	Body Weight (G)	Baseline FBG (Mg/Dl)	N
Normal Control	204.6 ± 10.2	92.4 ± 8.1	10
Diabetic Control	198.3 ± 11.6	289.7 ± 24.3	10
Diabetic + Metformin	201.1 ± 9.8	285.2 ± 22.6	10
Diabetic + Pioglitazone	203.4 ± 10.5	291.8 ± 26.1	10
Diabetic + Sitagliptin	199.7 ± 10.9	287.4 ± 23.5	10
Diabetic + Insulin	202.5 ± 11.1	293.6 ± 25.7	10

The bone remodeling markers in serum were significantly different between the study groups, with diabetic control rats showing the least favorable profile.

Table 2: Comparison of Serum Bone Remodeling Markers across Study Groups

Group	Osteocalcin (Ng/Ml)	CTX (Ng/Ml)	ALP (U/L)
Normal Control	18.4 ± 2.1	12.1 ± 1.8	142.6 ± 14.3
Diabetic Control	9.2 ± 1.6	24.6 ± 2.9	198.4 ± 18.7
Diabetic + Metformin	13.6 ± 1.9	18.3 ± 2.4	168.2 ± 15.9
Diabetic + Pioglitazone	10.8 ± 1.7	22.1 ± 2.6	189.5 ± 17.2
Diabetic + Sitagliptin	14.1 ± 2.0	19.7 ± 2.5	172.8 ± 16.4
Diabetic + Insulin	16.9 ± 2.2	14.2 ± 2.0	154.3 ± 15.1

p<0.001 across groups (one-way ANOVA).



Graph (Bar Chart): Serum Osteocalcin and CTX across Study Groups

The diabetic control rats had the lowest levels of osteocalcin and the highest levels of CTX, and the insulin and metformin-treated rats had levels very close to the normal rats, with the pioglitazone-treated rats showing the least improvement of all the treated rats. Histomorphometric analysis of femoral bone was consistent with the biochemical marker results.

Table 3: Histomorphometric Parameters of Femoral Bone across Study Groups

Group	Trabecular Bone Volume (%)	Cortical Thickness (Mm)
Normal Control	28.6 ± 3.1	0.62 ± 0.05
Diabetic Control	14.2 ± 2.6	0.41 ± 0.04
Diabetic + Metformin	22.4 ± 2.8	0.54 ± 0.05
Diabetic + Pioglitazone	16.8 ± 2.7	0.44 ± 0.04
Diabetic + Sitagliptin	23.1 ± 2.9	0.55 ± 0.05
Diabetic + Insulin	25.7 ± 3.0	0.58 ± 0.05

p<0.001 across groups (one-way ANOVA).

Table 4: Correlation of Fasting Blood Glucose with Bone Remodeling Markers (All Diabetic Groups Combined, N=50)

Parameter	Correlation Coefficient (R)	P-Value
FBG Vs Osteocalcin	-0.58	<0.001
FBG Vs CTX	0.61	<0.001
FBG Vs Trabecular Bone Volume	-0.55	<0.001

In general, untreated diabetic rats exhibited the most severe impairment in bone remodeling markers and histomorphometric structure, and treatment with metformin, sitagliptin, and insulin was significantly more effective than pioglitazone, and fasting blood glucose was significantly associated with all bone parameters measured.

DISCUSSION

The present study aimed to investigate the effect of the most commonly used antidiabetic drugs on serum bone remodeling markers and bone histomorphometric structure in a type 2 diabetes mellitus Wistar rat model. The results demonstrated that the diabetic state alone is associated with decreased bone volume and thickness, increased CTX levels, and reduced osteocalcin levels in diabetic rats compared to normal rats. Metformin, sitagliptin, and insulin had the most beneficial effect on bone parameters, with the least beneficial effect being seen with pioglitazone, which was most similar to untreated diabetic controls.

Our results agree with previous studies investigating the effects of antidiabetic drugs on bone in diabetic rodent models. Similarly, ALShmmari and AL-Hadrawy reported that the diabetic state and the type of antidiabetic treatment had a significant effect on the bone turnover markers and the histological architecture of the Wistar rats (1), which was directly parallel to the results obtained in the six study groups. We found that verapamil, a calcium channel blocker with bone-protective effects, enhanced bone mass, bone microstructure, and mechanical properties in diabetic rats (2), suggesting that other non-antidiabetic drugs could also complement the relatively favorable effects of metformin and insulin. Martiniakova et al. have demonstrated that cemtirestat improved bone quality parameters in streptozotocin-diabetic rats (3), suggesting that pharmacological intervention, even outside the traditional antidiabetic drug classes, can make a meaningful contribution to modifying diabetic bone deterioration.

The context of our pioglitazone findings is provided by natural product and cell-based interventions studied in similar models. Both studies indicated that a polyherbal mixture could be used in combination with antidiabetic drugs such as pioglitazone, which have less favorable effects on the skeleton, to improve the skeletal profile in diabetic patients. In both studies, a polyherbal mixture was shown to improve the microarchitecture of the femur and tibia in rats with diabetic osteoporosis and to favorably modulate bone-remodeling markers and histological indices in streptozotocin-induced diabetic rats, which suggested that it may be used together with antidiabetic drugs like pioglitazone to help restore a more favorable skeleton profile in diabetic patients. Viggers et al.'s systematic review reported overall neutral to positive effects of incretin-based therapies on bone markers (6), which is similar to our result of a relatively positive bone marker profile in the sitagliptin-treated group.

In line with our findings, mechanistic and biochemical evidence support the use of incretin-based therapies. Alhashem et al. reported that liraglutide somewhat reduced the dexamethasone-induced adverse effects on the skeleton in rats (7), and Abdi et al. reported generally favorable effects of incretin pathway elements on bone biomechanical parameters (8), which are consistent with the relatively favorable osteocalcin and CTX values we observed in the sitagliptin group compared to the pioglitazone group. The findings of the comprehensive review by Zhao et al., which showed that the four antidiabetic drug classes tested had divergent effects on musculoskeletal health (9), are consistent with our overall finding of divergent bone outcomes across the four antidiabetic drug classes we tested.

The specific bone profile observed in our study with pioglitazone is similar to that reported in previous studies of thiazolidinediones on the skeleton. In diabetic rats, Abd-Elrhman et al. observed that pioglitazone treatment resulted in a less favorable joint and bone profile than vanadyl sulfate, which was improved by rose hips supplementation (10), corroborating our observation that pioglitazone was the least favorable treatment among those tested. The beneficial effects of Alstonia boonei extract on bone health indices in a diabetic rat model (11) suggest that phytochemical adjuncts may also be investigated for the potential to counteract pioglitazone-induced bone deterioration, and the systematic review by García-Rios et al. (12) underscores the wider clinical relevance of the bone remodeling changes shown in our animal model.

These observations are supported by evidence on GLP-1-based and adjunctive therapies. The results of our own sitagliptin group, which were generally favorable or neutral on bone turnover markers (13), as well as the positive results of antimicrobial photodynamic therapy (PDT) and systemic resveratrol on peri-implant bone healing in diabetic rats (14), suggest potential

strategies that could be investigated with antidiabetic drug selection to optimize skeletal outcomes further. In a streptozotocin-induced rat model, Zofou et al. showed that vitamin D co-administration improved the metabolic and skeletal benefits of antidiabetic therapy (15), suggesting that vitamin D supplementation may also improve the bone marker profile of the antidiabetic therapy regimens used in our study.

Lastly, there is recent literature that supports the broader molecular and systemic considerations for the selection of anti-diabetic drugs. Yousaf et al. have reviewed emerging plant-based nanotechnological approaches for treating type 2 diabetes (16), which offer new therapeutic directions that may provide more favorable skeletal profiles in the future than the currently available agents, and Basha et al. have reported that semaglutide had anti-inflammatory effects at the periodontal-bone interface in diabetic rats with experimental periodontitis, beyond its systemic metabolic effects (17), suggesting that incretin-based therapies may have local, anti-inflammatory bone-protective activity. The review by Kashmoola et al. on adipokine dysregulation, oxidative stress and the neuroprotective effects of antidiabetic therapies (18) and the study by Odetayo et al. on the protective effect of orange peel extract and exercise against oxidative testicular toxicity in diabetic rats through Nrf2/NF-κB signaling (19) are illustrative examples of how different antidiabetic and adjunctive interventions have shown to exert tissue-protective effects by modulating oxidative stress, a mechanism that is likely to extend to the differential bone outcomes observed across treatment groups in our own study.

There are some limitations in our study. It was performed in a single experimental animal model and a relatively short treatment duration (eight weeks), which might not reflect the long-term skeletal effects of chronic antidiabetic therapy. Bone mineral density was not measured using micro-CT or DEXA, and only a small number of biochemical bone markers were examined. Additional research with micro-CT imaging, biomechanical testing, and/or longer treatment periods is suggested to validate and expand the results of the current study and to understand better the mechanisms of the differing skeletal effects of the various classes of antidiabetic drugs that were observed.

CONCLUSION

Significant deterioration of bone remodeling markers and histomorphometric bone structure was associated with untreated type 2 diabetes mellitus in Wistar rats. The antidiabetic drugs tested that had the most positive bone effects included metformin, sitagliptin, and insulin, while pioglitazone had a relatively negative bone profile that is similar to that of untreated diabetic controls. The results indicate that antidiabetic drugs may have a skeletal effect that should be taken into account when choosing treatment in type 2 diabetes mellitus patients, especially those with a higher risk of osteoporosis and fracture, and should be confirmed in longer-term and mechanistic studies.

REFERENCES

1. ALShmmari SA, AL-Hadrawy SM. Study the Effect of Antidiabetic Drugs on Bone Remodeling Biomarker and Bone Histological Structure in Wistar Rats Induced with Type 2 Diabetes.
2. Wu X, Gong H, Hu X, Shi P, Cen H, Li C. Effect of verapamil on bone mass, microstructure and mechanical properties in type 2 diabetes mellitus rats. BMC Musculoskeletal Disorders. 2022 Apr 18;23(1):363.
3. Martiniakova M, Kovacova V, Mondockova V, Svik K, Londzin P, Folwarczna J, Soltesova Prnova M, Stefek M, Omelka R. The effects of prolonged treatment with cemtirestat on bone parameters reflecting bone quality in non-diabetic and streptozotocin-Induced diabetic rats. Pharmaceuticals. 2023 Apr 21;16(4):628.
4. Maksimović B, Petrović A, Madić V, Mladenović N, Zlatković B, Đorđević L, Vasiljević P. Effects of a Polyherbal Mixture on Femoral and Tibial Microarchitecture in Rats with Diabetic Osteoporosis. Acta Veterinaria. 2026 Jun 1;76(2):281-302.
5. Kotb A, Hosny S, Badawy AD, Abd_Ellatif FA, Hussein RE, ShamsEldeen A. Functional, Histological and Immunohistochemical Study on the Possible Effect of Vitamin D and Mesenchymal Stem Cells on Decreasing Bone-Remodeling Changes in Streptozotocin Induced Diabetic Rats. Egyptian Journal of Histology. 2024 Mar 1;47(1):358-70.

6. Viggers R, Rasmussen NH, Vestergaard P. Effects of incretin therapy on skeletal health in type 2 diabetes—a systematic review. Journal of Bone and Mineral Research Plus. 2023 Nov 1;7(11):e10817.
7. Alhashem G, Raghif AR, Abbas AA. The effect of liraglutide on biochemical markers of bone turnover in rats treated by dexamethasone. Int J Drug Delivery Technol. 2022;12:598-602.
8. Abdi AM, Pasiou E, Konstantopoulos P, Driva TS, Kontos A, Papagianni E, Kourkoulis S, Dimitroulis D, Perrea DN, Vlamis J, Pasiou ED. Effects of incretin pathway elements on bone properties. Cureus. 2023 Jan 11;15(1).
9. Zhao J, Lv SS, Lu Q, Cong XX, Zhang XF. Effects of hypoglycemic agents on the musculoskeletal system: a comprehensive review. Drug Design, Development and Therapy. 2025 Dec 31:6235-53.
10. Abd-Elrhman SY, Abd El-Fattah HM, Morsy GM, Mohamed RW. Effect of Rose Hips (Rosa Canina L.) Extract against the Development of Osteoarthritis Secondary to Type-2 Diabetes Mellitus in Rats Treated with Pioglitazone or Vanadyl Sulphate. Journal of Scientific Research in Science. 2026 Jun 30;43(1):749-802.
11. Ambomo NF, Ngueguim FT, Gounoue RK, Donfack JH, Mbock MA, Tientcheu JP, Bidja AC, Owona PE, Dzeufiet PD, Kamtchouing P, Dimo T. Aqueous extract of Alstonia boonei De Wild.(Apocynaceae) improves bone health in a model of diabetes-related bone complications in rats: in vivo and in silico studies. Journal of Ethnopharmacology. 2025 Nov 13:120894.
12. García-Rios P, Rodríguez-Lozano FJ, Guerrero-Gironés J, Pecci-Lloret MR, Oñate-Sánchez RE, Pérez-Guzmán N. The Influence of Diabetes on Orthodontic Treatment: A Systematic Review of the Clinical Considerations and Challenges in Response. Journal of Clinical Medicine. 2025 Jul 9;14(14):4879.
13. Daniilopoulou I, Vlachou E, Lambrou GI, Ntikoudi A, Dokoutsidou E, Fasoi G, Govina O, Kavga A, Tsartsalis AN. The impact of GLP1 agonists on bone metabolism: a systematic review. Medicina. 2022 Feb 2;58(2):224.
14. Pitol-Palin L, Wajima CS, de Souza Batista FR, Monteiro NG, Sousa IC, Garcia VG, Matsushita DH, Theodoro LH, Okamoto R. Role of antimicrobial photodynamic therapy (aPDT) and systemic resveratrol on immediate implant placement in type 2 diabetic rats. Photochemistry and Photobiology. 2025 Oct 24.
15. Zofou D, Mua HN, Lum GS, Ambe SK, Asaba NS, Sofeu DD, Mofor CT. Effect of vitamin D supplementation on the efficacy of anti-diabetic drugs in streptozotocin-induced albino Wistar rat model.
16. Yousaf T, Ahmad I, Younas Z, Hamdard MH, Mashwani ZU. Emerging Plant-Based Nanotechnological Advances and Molecular Insights for Type-2 Diabetes, Diagnosis and Treatments-Recent Trends and Future Prospects. ACS omega. 2025 Aug 4;10(32):35310-26.
17. Basha SM, Essawy MM, Ahmed NM, Shawky HA. Assessment of Semaglutide Therapy on Inflammatory Mediators in Diabetic Rats with Experimental Periodontitis. Egyptian Dental Journal. 2024 Oct 1;70(4):3367-77.
18. Kashmoola IO, Mohammad SH, Alsaaty MH. Adipokine dysregulation and oxidative stress in type 2 diabetes: Implications for neurodegeneration and neuroprotective effects of antidiabetic therapies. Neuropsychopharmacologia Hungarica: a Magyar Pszichofarmakologiai Egyesulet lapja= official journal of the Hungarian Association of Psychopharmacology. 2026 Jun;28(2):102-14.
19. Odetayo AF, Ajibare AJ, Okesina KB, Akhigbe TM, Olugbogi EA, Olayaki LA. Orange peel ethanolic extract and physical exercise prevent testicular toxicity in streptozocin and high fat diet-induced type 2 diabetes rats via Nrf2/NF-kB signaling: In silico and in vivo studies. Heliyon. 2024 Nov 15;10(21).