Histomorphometric Study of Bone Healing Around Laminar Implants in Experimental Diabetes

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The aim of this study was to evaluate the effects of experimental diabetes on the healing period leading to osseointegration. Wistar rats were injected with a single dose of streptozotocin (STZ); body weight and food intake were assessed every 48 hours. On days 2, 12, 26, and 42 post-STZ, glucemia, plasma hemoglobin, and urea were determined. Twelve days post-STZ, a titanium laminar implant was placed in the right tibia of each rat. Two groups of 20 rats each were killed on days 14 and 30 postimplantation, respectively. Results (ANOVA test) showed STZ-treated rats to have 1) a significant decrease in body weight; 2) an increase in food intake; 3) normal hemoglobin and plasma urea values; 4) a significant increase in glucemia; and 5) a decrease in tibiae length. Microscopic evaluation 14 days postimplantation revealed the presence of woven bone, and, at 30 days, laminar bone was in contact with the implant. Our findings show that, in this model of periimplant bone repair and under the experimental conditions stated herein, STZ-induced diabetes retards periimplant bone healing.
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Key Words: diabetes, osseointegration, dental implant, streptozotocin

The first studies on titanium implants by Brånemark et al[1] have provided the basis for modern implantology. During the healing phase, several local and/or systemic factors (eg, radiotherapy, disorders of bone metabolism, intraosseous infections, traumatic surgical technique, and chemotherapy) may influence the degree of bone response to the implant environment.[2] Proper selection of patients is one of the most decisive issues regarding prognosis of implant treatments[3] because administration of pharmacological agents or disease may complicate or even exclude such therapeutic alternatives.[2,3]

It has been well documented that abnormalities of bone and mineral metabolism occur in several experimental models of diabetes.[4–11] These alterations have been reported in short-term or drug-induced diabetes, such as streptozotocin (STZ)-induced diabetes.[7,9,12,13]

In a previous experimental study, Cabrini et al[14] developed an experimental model (Laminar Implant Test) to quantitatively validate the periimplant reparative process during the unloaded healing stage. The aim of the present work was to carry out a histomorphometric study of the effects of STZ-induced diabetes on the healing period leading to periimplant osseointegration.

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Materials and Methods

Animals

Forty male Wistar rats weighing 47 ± 5 g were used. Animals were housed in plastic cages and maintained on a 12:12 hour light:dark cycle. They were fed rat chow and water *ad libitum*. National Institutes of Health guidelines for the care and use of laboratory animals (NIH Publication No. 85–23, Rev. 1985) were observed.

Diabetes Induction

Rats in the experimental group (n = 20) were given a single 65 mg/kg body weight intraperitoneal injection of STZ (Sigma, St. Louis, MO) dissolved in citrate buffer, pH 4.5. Using conventional methods, blood glucose was monitored in tail-nicked blood samples obtained 48 hours after induction of diabetes. A minimum blood glucose level of 180 mg/dL was adopted as the criterion level for having established moderate diabetes. Animals not responding (blood glucose <180 mg/dL) were excluded from the study. Body weight and food intake were assessed every 48 hours. On days 2, 12, 26, and 42 post-STZ, plasma glucose, hemoglobin, and urea were determined using conventional techniques.

Surgical Procedure

Twelve days post-STZ, under ether anesthesia, a CP titanium lami-

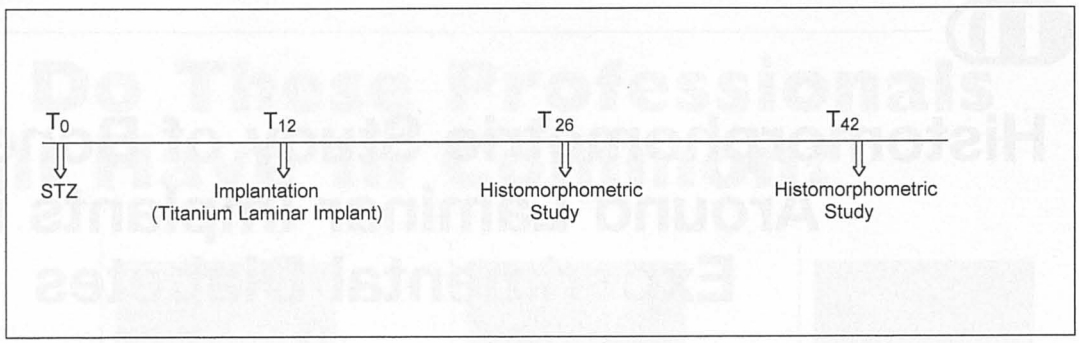

Fig. 1. Schedule of the experimental procedure.

nar implant (6 × 1 × 0.1 mm; Implant-Vel, Buenos Aires, Argentina) was placed in the right tibia of each rat (diabetic group n = 20 and control group n = 20), following the non-traumatic surgical technique described by Cabrini et al.[14] Half of the animals in each group (experimental diabetes n = 10 and control n = 10) were killed by ether overdose on day 14 postimplantation, and the remaining half underwent the same procedure 30 days postimplantation (26 and 42 days post-STZ, respectively) (Fig. 1). The tibiae were resected, fixed in 20% formalin solution, and radiographed.

Histological Processing

The tibiae were processed for embedding in methylmethacrylate resin. The samples were then sectioned using a saw. Three slices were cut perpendicular to the major axis of the tibiae; that is, at the middle of the implant and at two points equidistant from the middle. The cross sections were first ground using a grinding machine then were finished manually with sandpaper to obtain sections about 30 to 50 μm thick. The sections were then stained with 1% toluidine blue.

Histomorphometric Analysis

A Kontron MOP-AM 03 image analyzing system was used at ×80 magnification to evaluate the percentage of bone in direct contact with the implant surface (osseointegration) and the volume of periimplant bone tissue. Results are given as mean ± SD. Statistical significance was determined by ANOVA test.

Results

Body weight was significantly lower in diabetic animals than in controls, both at 14 and 30 days postimplantation (33% and 26%, respectively, $P < .001$) (Fig. 2). Diabetic animals also had a significant increase in plasma glucose (125% and 151% at 14 and 30 days postimplantation, respectively, $P < .001$) (Fig. 2) and in food intake (30%, $P < .01$) compared with controls. The difference between diabetic and control animals regarding plasma hemoglobin and plasma urea was not statistically significant at 14 or 30 days postimplantation. Histological observations at 14 days postimplantation revealed bone formation surrounding the implants, both in control and diabetic animals. Control animals showed lamellar bone close to the implant (Fig. 3*A*, *B*).

It must be pointed out that even though the extension of osteogenic repair process in STZ-treated rats was far greater than in control animals, this tissue was woven bone (Fig. 4*A*, *B*).

Thirty days postimplantation, additional lamellar bone growth was observed in control animals (Fig. 5). Diabetic animals showed a thin layer

	14 DAYS POST-IMPLANTATION		30 DAYS POST-IMPLANTATION	
	Control	Diabetics	Control	Diabetics
	$\bar{x}$ ± S.D.	$\bar{x}$ ± S.D.	$\bar{x}$ ± S.D.	$\bar{x}$ ± S.D.
	n : 10	n : 10	n : 10	n : 10
Body weight (g)	119.75 ± 2.47	80 ± 1.68	236.75 ± 3.30	175.50 ± 2.80
Plasma glucose (mg/dl)	105 ± 4.10	235.25 ± 14.64	92.75 ± 4.3	233.25 ± 42.6

± : S.D.

Fig. 2. Body weight and plasma glucose determinations at 14 and 30 days postimplantation.

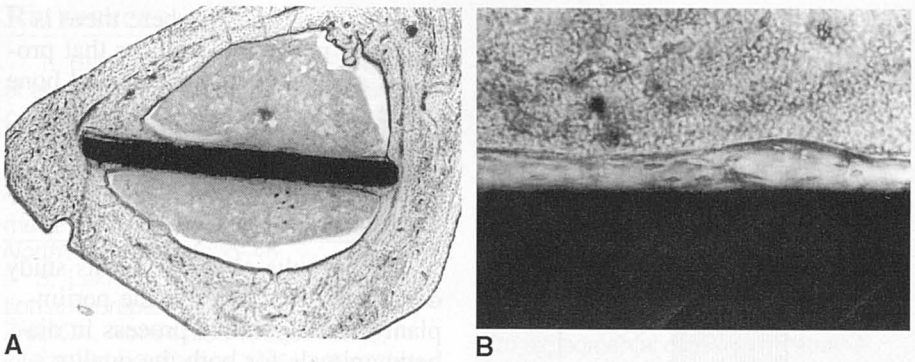

Fig. 3*A*, CONTROL 14 days postimplantation. Grinding section showing bone apposition to titanium laminar implant. Original magnification ×50; ***B***, CONTROL 14 days postimplantation. At higher magnification, close contact and full congruency at the level of the bone-implant interface is evident. Original magnification×400.

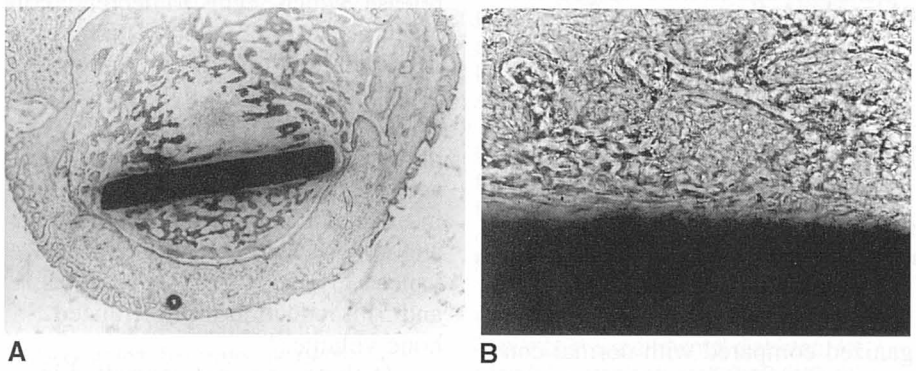

Fig. 4*A*, DIABETIC 14 days postimplantation. Grinding section showing woven bone formation in the periimplant microenvironment. Original magnification ×50; ***B***, DIABETIC 14 days postimplantation. Notice the woven bone-implant interface at higher magnification. Original magnification ×400.

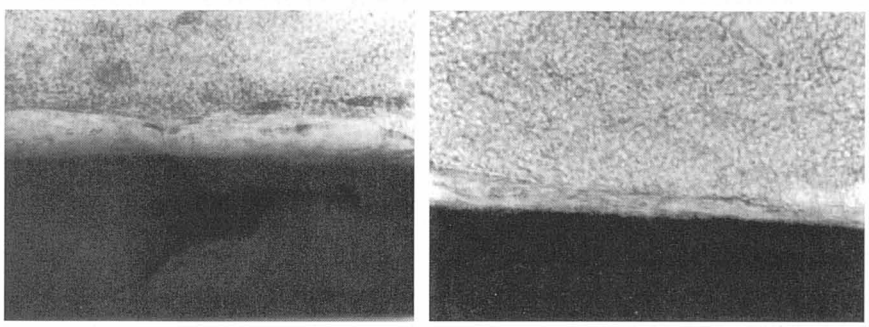

Fig. 5. CONTROL 30 days postimplantation. Grinding section showing osseointegration. Original magnification ×400.

Fig. 6. DIABETIC 30 days postimplantation. Grinding section showing close bone apposition to the implant. Original magnification ×100.

of osseointegrated lamellar bone (Fig. 6).

Histomorphometric studies revealed a significantly lower percentage of osseointegration in diabetic animals compared with controls, both at 14 and 30 days postimplantation (40% and 42%, respectively, $P < .001$) (Fig. 7).

The volume of periimplant bone tissue was greater in diabetic animals than in controls ($P < .001$) 14 days postimplantation and lower ($P < .001$) 30 days postimplantation (Fig. 8).

Discussion

According to previous studies reported in the literature,[15–17] induction of diabetes with STZ in rats is associated with a decrease in body weight and an increase in food intake and blood glucose concentration. Blood glucose concentration was more than two-fold greater than the control value. Mean serum creatinine concentration was <1 mg/dL in both groups (data not shown). Plasma urea concentration was also normal.

The deleterious effects of diabetes on linear growth and body composition are well documented.[15,16] In the present study, diabetic rats showed a reduction in tibiae length, and microscopically, the metaphyseal area was similar to control animals (data not shown).

The laminar implant test used in this study was used to evaluate the effect of experimental diabetes on the healing period leading to periimplant osseointegration. In this experimental model, the area of osteogenic periimplant response 14 days postimplantation was greater in diabetic rats than in controls. However, it must be pointed out that this bone was woven bone. In a previous study,[18] we evaluated the different stages in the healing process leading to osseointegration and found that periimplant woven bone volume reaches its highest peak 6 days after implantation but then decreases and finally disappears around day 14. At this time lamellar bone can only be found on the implant surface (osseointegration). The presence of woven bone 14 days postimplantation and of a thin layer of lamellar bone 30 days postimplantation reveals that bone healing around a laminar implant (commercially pure titanium, smooth surface) is slower in STZ-induced diabetic animals than in controls.

Takeshita et al[19] reported that "the healing response accompanied by bone formation chiefly depends on the condition of the implanted site." These authors based their conclusion on the finding that the "implants in the cortical bone area were almost completely embedded in the compact bone tissue in diabetic

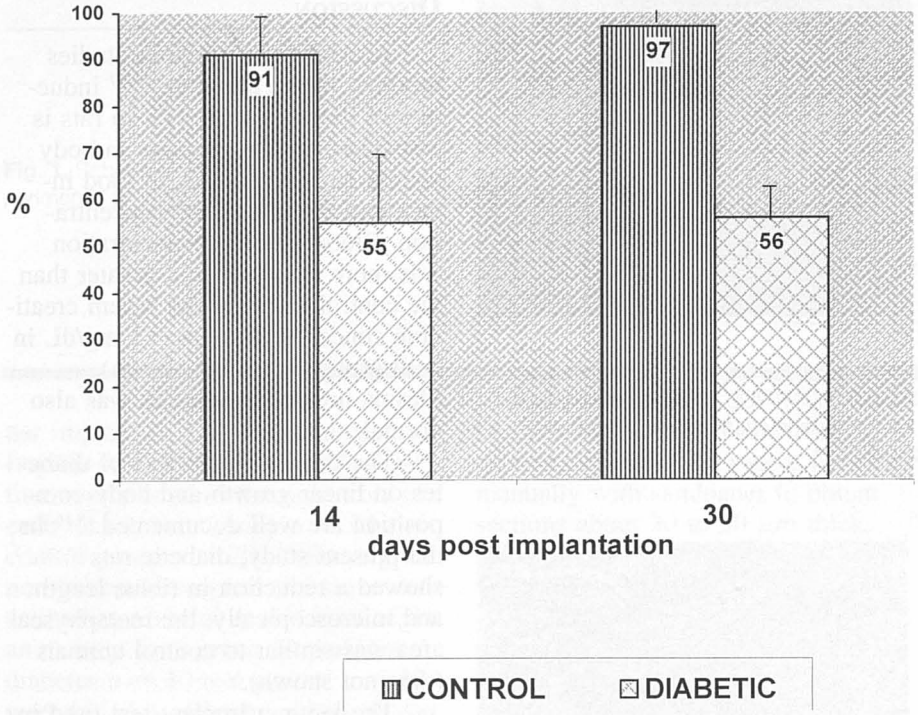

Fig. 7. Histomorphometric study. Percentage of osseointegration.

group as well as in the control group, in spite of the great differences in the bone marrow area."

Nevins et al[20] stated that "in the diabetic animals, osseointegration was observed, although with an altered pattern of bone quality. The morphology of the newly formed bone appeared immature and disorganized compared with normal controls." These data also correspond with bone marrow response.

The osteogenic capacity of bone marrow is a known fact. In our experimental model, we demonstrated that the osteogenic tissue in the medullary compartment (where there is no bone) reacts in a manner that produces a layer of osseointegrated bone tissue that covers a great proportion of the implant surface.

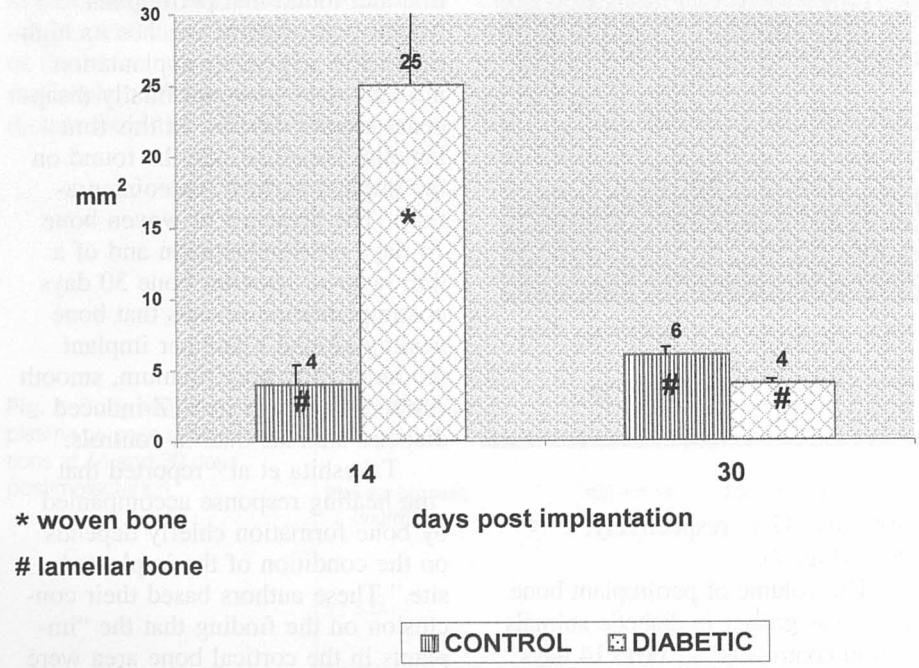

Fig. 8. Histomorphometric study. Volume of periimplant bone tissue.

Conclusions

The results obtained in this study demonstrated a delay in the periimplant bone reparative process in diabetic animals for both the quality and quantity of bone tissue.

To date, diabetes-related bone tissue disorders remain unclear. Some of the possible explanations may be 1) the reduction of insulin release, which seems to decrease collagen production; 2) bone turnover is depressed due to the inactivation of osteoblasts followed by impaired osteoclast activity;[21] 3) decreased protein synthesis and phosphatase activities in osteoblasts;[22] 4) changes in extracellular matrix components and bone metabolism caused by advanced glycosylation end products;[23] and 5) a reduction in mineralized bone volume.[24]

Diabetes was not controlled in the experimental model used in this study. Additional research should aim at studying the role of insulin control. It is likely that patients with well-controlled diabetes are not a risk group for surgical procedures, whereas those with poorly controlled diabetes may experience a failure in wound healing.[25]

Balshi and Wolfinger[26] reported that the results of their retrospective study indicate that a high success rate is achievable when dental implants are placed in diabetic patients whose disease is under control. These authors stated that " it has become increasingly common for controlled diabetic patients to be considered as candidates for dental implants."

Acknowledgments

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References

1. Brånemark P-I, Breine BE, Adell R, et al. Intraosseous anchorage of dental prostheses: I. Experimental studies. *Scand J Plast Reconstr Surg*. 1969;3:81–100.
2. Blanchaert RH. Implants in the medically challenged patient. *Dent Clin North Am*. 1998;42:35–45.
3. Lekholm U, Zarb GA. Patient selection and preparation. In: Brånemark P-I, Zarb GA, Albrektsson T, eds. *Tissue-Integrated Prostheses. Osseointegration in Clinical Dentistry.* Chicago: Quintessence; 1985:199–209.
4. Hough S, Avioli LV, Bergfeld MA, et al. Correction of abnormal bone and mineral metabolism in chronic streptozotocin-induced diabetes mellitus in the rat by insulin therapy. *Endocrinology*. 1981;108:2228–2234.
5. Dixit PK, Ekstrom RA. Decreased breaking strength of diabetic rat bone and its improvement by insulin treatment. *Calcif Tissue Int*. 1980;32:195–199.
6. Igarashi CH, Maruyama T, Ezawa I, et al. WBN/Kob rat: A new model of spontaneous diabetes, osteopenia and systemic hemosiderin deposition. *Bone Mineral*. 1994;27:133–144.
7. Spenser EM, Khalil M, Tobiassen O. Experimental diabetes in the rat causes an insulin-reversible decrease in renal 25-hydroxyvitamin D3–1 alpha-hydroxylase activity. *Endocrinology*. 1980;107:300–305.
8. Hough S, Fausto A, Sonn Y, et al. Vitamin D metabolism in the chronic streptozotocin-induced diabetic rat. *Endocrinology*. 1983;113:790–796.
9. Schneider LE, Schedl HP, McCain T, et al. Experimental diabetes reduces circulating 1,25-dihydroxyvitamin D in the rat. *Science*. 1977;196:1452–1454.
10. Verhaegne J, Van Herck E, Visser WJ, et al. Bone and mineral metabolism in BB rats with long-term diabetes. *Diabetes*. 1990;39:477–482.
11. Nyomba B, Bouillon R, Lissens W, et al. 1,25-Dihydroxyvitamin D and vitamin D-binding protein are both decreased in streptozotocin-diabetic rats. *Endocrinology*. 1985;116:2483–2488.
12. Tein MS, Breen SA, Loveday BE, et al. Bone mineral density and composition in rat pregnancy: Effects of streptozotocin-induced diabetes mellitus and insulin replacement. *Exp Physiol*. 1998;83:165–174.
13. Yoshino T, Yamaguchi I. Bone mineral density at metaphysis is specifically reduced in STZ-treated diabetic rats. *Life Sci*. 1996;59:161–168.
14. Cabrini RL, Guglielmotti MB, Almagro JC. Histomorphometry of initial bone healing around zirconium implants in rats. *Implant Dent*. 1993;2:264–267.
15. Cortright RN, Collins HL, Chandler MP, et al. Diabetes reduces growth and body composition more in male than in female rats. *Physiol Behav*. 1996;60:1233–1238.
16. Friedman MI. Hyperphagia in rats with experimental diabetes mellitus: A response to a decreased supply of utilizable fuels. *J Comp Physiol Psych*. 1978; 92:109–117.
17. Tepper BJ, Kanarek RB. Dietary self-selection patterns of rats with mild diabetes. *J Nutr*. 1985;115:699–709.
18. Guglielmotti MB, Guerrero C and Cabrini RL. Chronodynamic evaluation of stages of osseointegration in zirconium laminar implants. *Acta Odontol Latinoamer*. 1997;10:11–23.
19. Takeshita F, Murai K, Iyama S, et al. Uncontrolled diabetes hinders bone formation around titanium implants in rat tibiae: A light and fluorescence microscopy, and image processing study. *J Periodontol*. 1998;69:314–320.
20. Nevins ML, Karimbux NY, Weber HP, et al. Wound healing endosseous implants in experimental diabetes. *Int J Maxillofac Implants*. 1998;13:620–629.
21. Kaneko H, Sasaki T, Ramamurthy NS, et al. Tetracycline administration normalizes the structure and acid phosphatase activity of osteoclasts in streptozotocin-induced diabetic rats. *Anat Rec*. 1990;227:427–436.
22. Sasaki T, Ramamurthy NS, Golub LM. Insulin-deficient diabetes impairs osteoblast and periodontal ligament fibroblast metabolism but does not affect ameloblast and odontoblasts: Response to tetracycline administration. *J Biol Buccale*. 1990;18:215–226.
23. Browlee M. Glycation products and the pathogenesis of diabetic complications. *Diabetes Care*. 1992;15:1835–1843.
24. Goodman WG, Hori MT. Diminished bone formation in experimental diabetes. *Diabetes*. 1984;33:825–831.
25. Smith RA, Berger R, Dodson TB. Risk factors associated with dental implants in healthy and medically compromised patients. *Int J Oral Maxillofac Implants*. 1992;7:367–372.
26. Balshi T, Wolfinger GJ. Dental implants in the diabetic patient: A retrospective study. *Implant Dent*. 1999; 8:355–359.

Observations and Remarks

A review of *Histomorphometric Study of Bone Healing Around Laminar Implants in Experimental Diabetes* by Thomas J. Balshi, DDS

This was a well-conducted study that supports findings of previous investigations that uncontrolled diabetes can play a role in the decreased bone apposition to a titanium implant during the initial healing phase. It is clinically important to be aware of this potential complication that could arise in treating an uncontrolled diabetic patient. The question yet to be answered is, "What degree of decreased bone formation resulting from uncontrolled diabetes will ultimately affect the clinical use of the implant, ie, inhibit osseointegration to render the implant incapable of sustaining stability under loading forces?"

The clinician must stress to the diabetic patient the need for careful monitoring of blood glucose levels and control of the disease process. There are many undiagnosed diabetic patients. Many practitioners have treated patients who have demonstrated poor healing to dental procedures. Recognizing poor healing around the dental implant could be an indication of a systemic abnormality such as diabetes in some patients.

In our center we have found that controlled diabetics can experience the same positive results with osseointegration as nondiabetic patients if they are closely monitored and controlled during all treatment.

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Abstract Translations [German, Spanish, Portuguese, Japanese]

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ABSTRACT: Ziel dieser Studie war die Bewertung der Auswirkungen experimentellen Diabetes auf die Heilungsdauer bis zur vollen Integration in das Knochengewebe. Wistarratten wurde eine einzelne Dosis Streptozotocin (STZ) injiziert; Körpergewicht und Nahrungsaufnahme wurden alle 48 Stunden bestimmt. Am 2., 12., 26., und 42. Tag nach STZ-Verabreichung wurden der Blutzucker-, der Plasma-Hämoglobin- sowie der Harnstoffgehalt gemessen. Zwölf Tage nach STZ-Verabreichung wurde jeder Ratte ein laminares Titanimplantat in das rechte Schienbein eingesetzt. Zwei Gruppen von jeweils zwanzig Ratten wurden 14 bzw. 30 Tage nach Implantateinbringung getötet. Der (ANOVA-) Test ergab an mit STZ behandelten Ratten folgende Ergebnisse: A) eine signifikante Abnahme des Körpergewichts, B) eine erhöhte Nahrungsaufnahme, C) normale Hämoglobin- und Plasma-Harnstoffwerte, D) einen signifikanten Anstieg der Blutzuckerwerte, E) eine Verkürzung des Schienbeins. Bei der mikroskopischen Untersuchung 14 Tage nach Implantateinbringung zeigte sich Geflechtknochen; nach 30 Tagen bestand Kontakt zwischen laminarem Knochen und Implantat. Unsere Ergebnisse zeigen, daß sich durch STZ-induzierten Diabetes bei diesem Modell der Augmentationsplastik im umliegenden Bereich von Implantaten und unter den hier beschriebenen Versuchsbedingungen eine verzögerte Heilung des Knochengewebes im Bereich des Implantats ergibt.

SCHLÜSSELWÖRTE: Diabetes, Knochenintegration, Zahnimplantat, Streptozotocin

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ABSTRACTO: Los factores locales o sistémicos que condicionan el fracaso de los implantes dentales es un tema de interés creciente. Es de destacar que un alto porcentaje de estas variaciones actúan en las etapas reparativas tempranas postimplante. El objetivo del presente fue evaluar histomorfométricamente los efectos de la diabetes experimental en la cascada de la cicatrización ósea peri-implante que conduce a la oseointegración. Ratas Wistar macho recién destetadas fueron inyectadas con una dosis de streptozotocina (STZ) (65mg/kg. de peso corporal), determinándose el peso corporal y la ingesta de alimento cada 48 horas. A los 2, 12, 26 y 42 días post-STZ, se determinaron glucemia, hemoglobina, y urea en plasma mediante técnicas convencionales. A los 12 días post-SZT se colocaron implantes laminares de titanio* en la tibia derecha de cada rata (Cabrini et al. Imp Dent 1993;2:264–267). Dos grupos de 20 ratas cada una fueron sacrificados por sobredosis de éter a los 14 y 30 días post-implantación, respectivamente, se resecaron las tibias, radiografiaron y procesaron para su inclusión en resina acrílica. Los resultados obtenidos mostraron (Test de ANOVA) en las ratas tratadas con STZ: a) disminución del peso corporal (26%) a los 30 días post-implantación, b) aumento de la ingesta de alimento (30%), c) valores normales de hemoglobina y urea en plasma y d) aumento significativo de la glucemia (151%) a los 30 días post-implantación. La evaluación microscópica en los animales control evidenció la presencia de tejido óseo laminar a los 14 y 30 días post-implantación. En los animales diabéticos a los 14 días post-implantación se reveló la presencia de hueso reticular mientras que a los 30 días se encontró hueso laminar en contacto con el implante. El estudio histomorfométrico evidenció en las ratas diabéticas: a) una disminución significativa del porcentaje de oseointegración a 14 y 30 días post-implantación (40% y 42% respectivamente) y b) un volumen de tejido óseo peri-implante mayor (25%) a los 14 días post-implante en comparación al encontrado a los 30 días post-implante (4%). Es importante destacar que si bien se encontró un mayor volumen óseo peri-implante a los 14 días post-implantación, el estudio histológico determinó la presencia de hueso reticular. Los hallazgos obtenidos en el modelo experimental utilizado sugieren que la diabetes inducida por estreptozotocina retardaría la cicatrización ósea peri-implante.

PALABRAS CLAVES: diabetes, oseointegración, implante dental, estreptozotocina

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SINOPSE: O objetivo desse estudo era avaliar os efeitos da diabetes experimental no período de cicatrização que leva à integração óssea. Uma dose única de streptozotocin (STS) foi injetada em Ratos Wistar; o peso corporal e a ingestão de alimentos foram avaliados a cada 48 horas. Nos dias 2, 12, 26 e 42 após a dose de STZ, foram determinados os níveis de glicemia, hemoglobina plásmica e uréia. Doze dias após a dose de STZ, foi colocado um implante laminar de titânio na tíbia direita de cada rato. Dois grupos de vinte ratos foram sacrificados nos dias 14 e 30 após o implante, respectivamente. Os resultados, (teste ANOVA) mostraram que os ratos tratados com STZ apresentaram: A) diminuição considerável no peso corpóreo; B) aumento na ingestão de elementos; C) valores normais de hemoglobina e uréia plasmática, D) aumento considerável na glicemia e E) diminuição no comprimento das tíbias. A avaliação microscópica 14 dias após o implante revelou a presença de osso entrelaçado, e em 30 dias, o osso laminar estava em contato com o implante. Nossas descobertas mostram que neste modelo de reparo do osso por peri-implante e sob as condições experimentais citadas acima, a diabetes induzida por STZ retardaria a cicatrização óssea peri-implante.

PALAVRAS-CHAVES: diabetes, integração óssea, implante dentário, streptozotocin

実験的糖尿病におけるlaminarインプラント周囲の骨治癒についての組織形態計測学的研究

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概要：

この研究の目的は、実験的糖尿病が骨統合に向かう治癒過程に与える影響を評価することであった。wistar ratにストレプトゾトシン（STZ）を1回量注射後、体重と食餌摂取量が48時間ごとに記録された。STZ注射後2日目、12日目、26日目、42日目に、glucemia、血漿ヘモグロビン、尿素が測定された。STZ注射後12日目に、チタンlaminarインプラントが各ラットの右脛骨に設置された。おのおの20匹で構成される2つのグループのラットが、インプラント設置後各14日目と30日目にそれぞれ安楽死させられた。ANOVAテストの結果は、STZを与えられたラットはA)体重が有意に増加し、B)食餌摂取量が増加し、C)ヘモグロビンと血漿中尿素の数値は正常であり、D)glucemiaが有意に増加し、E)脛骨が短縮化していた。インプラント後14日目のグループの顕微鏡的観察では、woven boneの存在が認められ、30日目のグループでは、laminar boneのインプラントへの接触が認められた。この研究結果から、今回のようなインプラント周囲の骨修復モデルと実験的環境のもとでは、STZによる人為的な糖尿病がインプラント周囲の骨の治癒を遅らせることがわかった。

キーワード：

糖尿病、骨統合、デンタルインプラント、ストレプトゾトシン

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