Journal of Kerbala University , Vol. 14 No.1 Scientific . 2016

Effect of smoking on hard palate bone density
تأثير التدخين على كثافة عظم الحنك الصلب

Zahraa M. Al- Fadily, B.D.S., M.Sc.
University of Kufa college of denstray

Abstract

Background: The bone density of hard palate is key factor for success of palatal mini-implant. Smoking is an important determinant of osteoporosis.

Materials and method: Sixty males were selected with age range 20-39 years and divided into two groups according to smoking; smokers group (30), and nonsmokers (control) group (30). The measurements of hard palate bone density by HU (unit used to measure radiodensity of bone on CT scan) were made at 20 sites at the intersection of five anterioposterior and four mediolateral reference lines using Philips, Brilliance^{tm}, 64-multislice computed tomography scanner software.

Results: The results that were obtained showed that there was a statistically significant difference between the two groups in bone density at most areas of hard palate. The comparison between the two groups in the overall cortical bone density showed a highly statistically significant difference. The comparison between the two groups in the overall cancellous bone density showed a statistically significant difference.

Conclusion: Hard palate bone density decreased by smoking. In spite that, orthodontic mini-implants can be safely used for smoker persons in hard palate.

Keywords: Hard Palate, bone density, smoking, orthodontic mini-implant, computerized tomography.

الخلاصة

الخلفية: كثافة عظم الحنك الصلب عامل مهم لنجاح الزرعات التقويمية الحنكية. التدخين مسبب رئيسي لهشاشة العظام.

المواد والادوات: شملت العينة 60 ذكر تتراوح أعمارهم بين٢٠- ٣٩ سنة. قسمت العينة الى مجموعتين, المدخنين (30) وغير المدخنين (30). كثافة عظم الحنك الصلب قيست ل٢٠ موقع عند تقاطع خمس خطوط إشارة أمامية خلفية مع أربع خطوط إشارة قريبة بأستخدام برامجيات المفراس الحلزوني ٦٤- متعدد المقاطع لشركة فلبس المتألقة.

النتائج: النتائج أظهرت أن هنالك فروق ذات دلالة إحصائية في كثافة عظم الحنك الصلب بين المدخنين وغير المدخنين في معظم المناطق. مقارنة كثافة العظم القشري الكلية بين المجموعتين بينت فروق ذات دلالة إحصائية عالية . مقارنة كثافة العظم الإسفنجي الكلية بين المجموعتين بينت فروق ذات دلالة إحصائية.

الاستنتاج: كثافة عظم الحنك الصلب تتناقص بالتدخين. بالرغم من ذلك, الزرعات التقويمية من الممكن وضعها للمدخنين في عظم الحنك الصلب.

Introduction

In order to overcome problems associated with anchorage loss in orthodontic treatment, mini-implants have been developed and now used increasingly[1]. Palatal mini-implants are widely used as orthodontic anchorage device[2-4], which is optimal for supporting various treatment mechanics, including distalization, rapid maxillary expansion, protraction of buccal teeth, space closure, intrusion mechanics[5] and to reinforce anchorage in Angle Class II malocclusion patients in whom retraction of anterior teeth was achieved after the maxillary first premolars were extracted[6]. Despite that, the stability of a mini-implant is essential before it can be used as orthodontic anchorage and the clinical application of a mini-implant does not guarantee treatment success[7-9].The key determinant for stationary mini-implants anchorage is bone density[10,11]. As Previous studies showed a close association between the bone density factor and the failure rate of dental implants[12-14]. Many classification of bone density and its relation to dental implant treatments have been introduced[12,16-18]. Cigarette smoking was recognized as a key lifestyle factor for both bone loss (reduced bone

mineral density) and increase fractures risk. There are a wide variety of mechanisms by which smoking predispose bone loss[19]. Such as, a decrease in absorption of intestinal calcium[20,21], low body weight[21] and affects bone mass by some alterations in sex hormone metabolism[22].

Therefore, the purpose of this study was to know how much the cigarette smoking effect on the bone density of the hard palate and subsequently to know the possibility of placing palatal mini-implant for smoker persons.

Material and method

The total sample consisted of 60 Iraqi subjects (males), with age range of 20-39 years old, collected from the MRI and CT department of AL-Sader Medical City in AL-Najaf. The sample divided into two groups according to smoking; smoker group (30), and control group (30). Sample selection criteria included:

a. Skeletal class I, with normal occlusion.
b. They have full set of permanent teeth in both jaws left and/or right side "excluding the 3rdmolar"
c. No impacted teeth and/or any erupted supernumerary within the area of measurement.
d. No history of a systemic disease and/or chronic use of any medication that could affect the bone density.
e. No regular alcohol drinking.
f. No history of dentofacial deformities, pathologic lesions in the jaws or facial trauma.
g. No history of previous orthodontic and/or orthopedic treatment.
h. For smokers were daily consumption of at least 15 cigarettes (for at least two years)[22].

The study confined to males depending on previous study that refer there were no statistical significant gender difference in the hard palate bone density[23,24,25.] Each individual was informed about the study and asked if he agrees to participate in it. To measure the bone density by Hounsfield unit (HU) for the hard palate, CT images were obtained by 64-slice multi-detector CT scanner (Philips, Holland, Brilliancetm CT, V4.0). The measurements of hard palate bone density by (HU) were made at 20 sites at the intersection of five anterioposterior reference lines with 6 mm interval from incisive foramen (anterior area at 0,6, middle area at 12 and posterior area at 18,24 mm) and four mediolateral reference lines with 3 mm interval from mid-palatal suture (mid-palatal area at 0, medial area at 3, middle area at 6 and lateral area at 9 mm) (Figure 1). The measurements made either in the left or right side depending on the previous studies[23,24,26] that refer there were no statistical significant differences between the left and right side measurements. In the sagittal view for all points, the cortical bone density was measured at the midpoint of the cortical bone thickness. Also, the cancellous bone density was measured at the trabeculae, located halfway incisoapically between the two cortical plates.

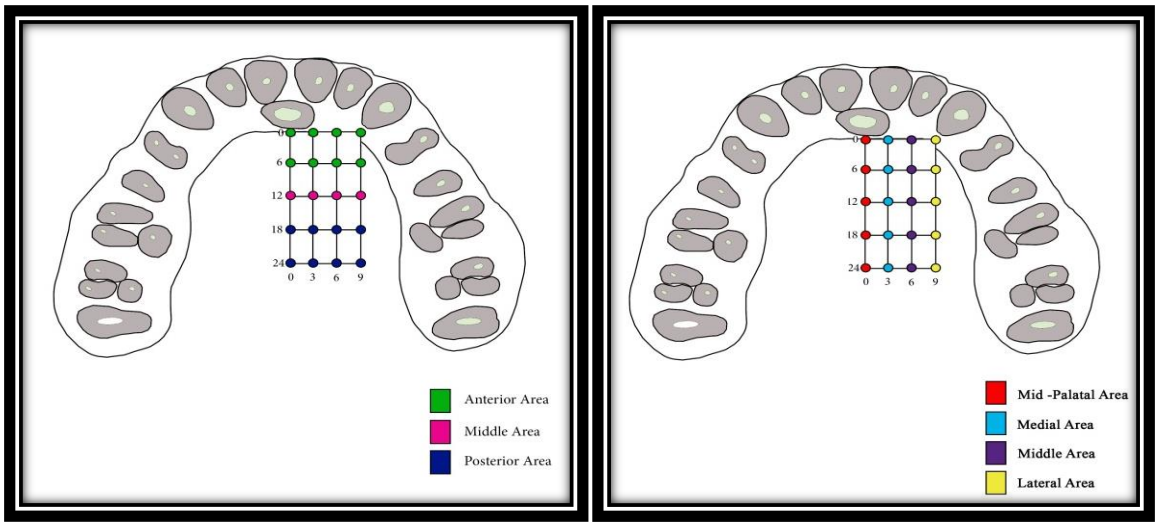

Figure (1): The designated areas (A) anteroposterior areas; (B) mediolateral areas.

Results

The descriptive statistics of the cortical and cancellous bone density at different points for two group are presented in Table1 and 2 respectively. Regarding cortical bone density, there were a highly statistically significant difference ($p < 0.001$) between the two groups at anterior area, middle area (anteroposterior areas), mid-palatal area, medial area and lateral area (mediolateral areas). While at posterior area (anteroposterior areas) and middle area (mediolateral areas) showed a statistically significant difference. The results of the cancellous bone density were a highly statistically significant difference between two group at anterior area (anteroposterior areas) and mid-palatal area (mediolateral areas). At posterior area showed a statistically significant difference. In the remaining areas the difference were a non- statistically significant (Table 3 and 4). The comparison between the two groups in the overall cortical bone density was a highly statistically significant difference. The comparison between the two groups in the overall cancellous bone density was a statistically significant difference (Table 5).

Discussion

Orthodontics treatment in last years depend mainly on mini-implant to provide anchorage clinically. The selection of hard palate for placement of temporary skeletal anchorage devices resulting from sufficient bone quality, less possibility of root damage to the adjacent teeth (nontooth-bearing area) and this anchorage is highly successful during treatment without hindering tooth movement[27]. The success of mini-implant is determined by many factors including bone mineral density (quality of bone)[28,29].

Growth and development of the bone mass are products from complex interactions of genetic and environmental factors[30]. These factors can be broadly classified into factors that cannot be modified, such as age, gender, body size, genetics and ethnicity, and modified factors, such as lifestyle factors including physical activity levels, smoking and alcohol consumption patterns, hormonal status, and diet [31].

Previous studies done to evaluate bone density of the hard palate in normal persons to provide guidelines in order to select the most suitable sites for placement of mini-implants[23,25,26]. This study aimed to evaluate hard palate bone density in smoker persons because the effect of smoking on bone health and prevalence of smoking in our community. The study made comparison between the two groups at different areas and in the overall cortical and cancellous bone density. This study found highly statistically significant difference between them in cortical bone density at most areas. The comparison of the overall cortical bone density between the two groups was a highly statistically significant difference. on the other hand, The results of the cancellous bone density were a highly statistically significant difference between two groups at anterior area (anteroposterior areas) and mid-palatal area (mediolateral areas). At posterior area showed a statistically significant difference. In the remaining areas the difference were a non- statistically significant. The comparison between the two groups of the overall cancellous bone density was a statistically significant difference. There is no previous study examining these relationships. This results can be explained as a direct relationship between tobacco use and decreased bone density found by many previous studies at various areas of human body[22,32-35].

Low bone density caused by smoking is thought to be through various pathways including: changes in hormone household, leading to a decrease in estrogen levels and parathyroid hormone (thus reducing calcium absorption) in addition to an increase in the level of cortisol and adrenal androgens, changes that have been linked to an increased risk of osteoporosis[36]; body mass reduced by smoking, which is postulated to provide an osteogenic stimulus and is linked to higher bone mineral density[37]; increasing free radicals and oxidative stress which affects bone resorption associated with smoking[38]; reduces the level of Vitamin D in the body, which is essential for good bone health[39]; also the smokers are more likely to suffer from peripheral vascular disease leading to reducion blood supply to the bones[40]; Finally, direct toxic effects of many of the constituents in tobacco smoke on bone cells may also exist[41].

The study found that the greatest effect of smoking was on the cortical bone density than cancellous bone density, this may belong to the fact that the cancellous bone is a trabecular network, surrounds

marrow spaces that may contain either hematopoietic or fatty tissue, located subjacent to the cortical bone[42] and the water ratio is higher in trabecular than in cortical bones[43].
The mean of cortical and cancellous bone density at all areas not reach to D4 (150-350 HU) category of Misch's classification **(1990)**[11]. In D4 bone, the placement of implant is not recommended due to the high failure rate associated with it (35-50%)[44].
In conclusion, smoking seems to be associated with a decrease in hard palate bone density. However, this effects according to **Misch's** classification **(1990)**[11] will not be so sever to prevent placement of palatal mini-implant.

References

1. Kanomi R. Mini-implant for orthodontic anchorage. J Clin Orthod 1997;31:763-7.
2. Bernhart T, Freudenthaler J, Dortbudak O, Bantleon HP, Watzek G. Short epithetic implants for orthodontic anchorage in the paramedian region of the palate. A clinical study. Clin Oral Implants Res 2001; 12: 624-31.
3. Jung BA, Kunkel M, Gollner P, Liechti T, Wehrbein H. Success rate of second-generation palatal implants. Angle Orthod 2009; 79: 85-90.
4. Flieger S, Ziebura T, Kleinheinz J, Wiechmann D. A simplified approach to true molar intrusion. Head and Face Medicine 2012; Open Access: 1-7.
5. Ludwig B, Glasel B, Bowman SJ, Wilmes B, Kinzinger GSM. Overview Anatomical Guidelines for Miniscrew Insertion Palatal Sites. J Clin Orthod 2011 b; 45(8): 433-441.
6. Wehrbein H, Feifel H, Diedrich P. Palatal implant anchorage reinforcement of posterior teeth: prospective study. Am J OrthodDentofacial Orthop 1999;116:678-86.
7. Lee SY, Cha JY, Yoon TM, Park YC. The effect of loading time on the stability of mini-implant. Korean J Orthod 2008;38:149-58.
8. Park YC, Chu JH, Choi YJ, Choi NC. Extraction space closure with vacuum-formed splints and miniscrew anchorage. J Clin Orthod 2005;39:76-9.
9. Im DH, Kim YS, Cho MA, Kim KS, Yang SE. Interdisciplinary treatment of Class III malocclusion using mini-implant: problemoriented orthodontic treatment. Korean J Orthod 2007;37:305-14.
10. Lee JS, Kim DH, Park YC, Kyung SH, Kim TK. The efficient use of midpalatal miniscrew implants. Angle Orthod 2004;74:711-4.
11. Misch CE. Density of bone: effect on treatment plans, surgical approach, healing, and progressive bone loading. Int J Oral Implantol 1990;6:23-31.
12. Lekholm U, Zarb GA. Patient selection and preparation. In: Brånemark PI, Zarb G, Albrektsson T. (Ed.). Tissue-integrated prostheses. Osseointegration in clinical dentistry. Chicago: Quintessence, 1985, Page 199-209.
13. Jaffin RA, Berman CL. The excessive loss of Branemark fixtures in type IV bone: a 5-year analysis. J Periodontol 1991;62:2–4.
14. Truhlar RS, Morris HF, Ochi S, Winkler S. Second-stage failures related to bone quality in patients receiving endosseous dental implants: DICRG Interim Report No. 7. Dental Implant Clinical Research Group. Implant Dent 1994;3:252–5.
15. Linkow LI, Chercheve R. Theories and techniques of oral implantology, vol. I, St Louis Mosby, 1970.
16. Roberts WE, Turley PK, Brezniak N, Fiedler PJ. Bone physiology and physiology and metabolism. J Calif Dent Assoc 1987; 15: 54-61.
17. Misch CE. Bone character: second vital implant criterion. DentToday. 1988;7(5):39.
18. Misch CE, Kircos LT. Diagnostic imaging and techniques. In: Misch CE, ed. Contemporary Implant Dentistry. 2nd ed. St. Louis: Mosby; 1999:73e87.
19. Yoon V, Maalouf NM, Sakhaee K. The effect of smoking on bone metabolism. International Osteoporosis Foundation and National Osteoporosis Foundation 2012; 23(8): 2081-92.

20. Rapuri PB, Gallagher JC, Balhorn KE, Ryschon KKL. Smoking and bone metabolism in elderly women. Bone 2000; 27:429-436.
21. Krall EA, Dawson-Hughes B. Smoking and bone loss among postmenopausal women. J Bone Miner Res 1991; 6:331-337.
22. Supervía A, Nogués X, Enjuanes A, Vila J, Mellibovsky L, Serrano S, Aubía J, Díez-Pérez A. Effect of smoking and smoking cessation on bone mass, bone remodeling, vitamin D, PTH and sex hormones. J Musculoskelet Neuronal Interact 2006; 6(3):234-241.
23. Han S, Bayome M, Lee J, Lee YJ ,Song HH, Kook YA. Evaluation of palatal bone density in adults and adolescents for application of skeletal anchorage devices. Angle Orthod 2012; 82: 625-631.
24. Tewfiq SM. Bone density determination for the maxilla and the mandible in different age groups by using computerized tomography, a Master Thesis, Department of Orthodontics, College of Dentistry, University of Baghdad, 2012.
25. Al- Fadily ZM. Hard palate bone density and thickness determination using CT scan and their relationships with body compositions measured by bioelectrical impedance analysis for Iraqi adult sample, a Master Thesis, Department of Orthodontics, College of Dentistry, University of Baghdad, 2014.
26. Moon SH, Park SH, Lim WH, Chun YS. Palatal Bone Density in Adult Subjects: Implications for Mini-Implant Placement. Angle Orthodontist 2010; 8(1): 137-144.
27. Jung BA, Kunkel M, Gollner P, Liechti T, Wehrbein H. Success rate of second-generation palatal implants. Angle Orthod 2009;79: 85-90.
28. Motoyoshi M, Matsuoka M, Shimizu N. Application of orthodontic mini-implants in adolescents. Int J Oral Maxillofac Surg 2007; 36:695-9.
29. Cha JY, Kil JK, Yoon TM, Hwang CJ. Miniscrew stability evaluated with computerized tomography scanning. Am J Orthod Dentofacial Orthop 2010; 137(1): 73-79.
30. Gkiatas I, Lykissas M, Kostas-Agnantis I, Korompilias A, Batistatou A, Beris A. Factors affecting bone growth. Am J Orthop (Belle Mead NJ) 2015;44(2):61-7.
31. **Cashman KD.** Diet and control of osteoporosis. In: Remacle C, Reusens B, editors. Functional foods, ageing and degenerative disease. Cambridge, UK: Wood head Publishing Limited 2004; 83-114.
32. Valimaki MJ, Karkkainen M, Lamberg-Allardt C, Laitinen K, Alhava E, Heikkinen J, Impivaara O, Makela P, Palmgren J, Seppanen R. Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass: Cardiovascular Risk in Young Finns Study Group.
Br Med J 1994; 309:230-235.
33. Vogel JM, Davis JW, Nomura A, Wasnich RD, Ross PD. The Effects of smoking on bone mass and the rates of bone loss among elderly Japanese-American men. Journal of bone and mineral research 1997; 12 (9): 1495-1501.
34. Law MR, Hackshaw AK. A meta-analysis of cigarette smoking, bone mineral density and risk of hip fracture: recognition of a major effect. Br Med J 1997; 315:841-846.
35. Ward KD, Klesges RC. A meta-analysis of the effects of cigarette smoking on bone mineral density. Calcif Tissue Int 2001; 68:259-270.
36. Kapoor D, Jones TH. Smoking and hormones in health and endocrine disorders. Eur J Endocrinol 2005; 152(4):491– 499.
37. Daniel M, Martin AD, Drinkwater DT. Cigarette smoking, steroid hormones, and bone mineral density in young women. Calcif Tissue Int 1992; 50(4):300–305.
38. Duthie GG, Arthur JR, James WP. Effects of smoking and vitamin E on blood antioxidant status. Am J Clin Nutr 1991; 53(4 Suppl):1061S–1063S.
39. Brot C, Jorgensen NR, Sorensen OH. The influence of smoking on vitamin D status and calcium metabolism. Eur J Clin Nutr 1999; 53(12):920–926.
40. Vestergaard P, Mosekilde L. Fracture risk associated with smoking: a meta-analysis. J Intern Med 2003; 254(6):572–583.

41. Broulik PD, Jarab J. The effect of chronic nicotine administration on bone mineral content in mice. Horm Metab Res 1993; 25(4):219–221.
42. Clarke B. Normal Bone Anatomy and Physiology. Clin J Am Soc Nephrol 2008; 33(4): S131-9.
43. Gong JK, Arnold JS, Cohn SH. Composition of trabecular and cortical bone. The Anatomical Record 1964; 149(3): 325-331.
44. Kravitz ND, Kusnoto B, Tsay TP, Hohit WF. The use of temporary anchorage devices for molar intrusion. J Am Dent Assoc 2007; 138(1): 56-64.

Table 1: Descriptive statistics of the cortical bone density at different points

Variables	Descriptive statistics					
	Smoker Group (N=30)			Control Group (N=30)		
	Mean	SD	SE	Mean	SD	SE
$AP^{0}ML^{0}$	1065.600	143.847	26.263	1180.367	152.271	27.801
$AP^{0}ML^{3}$	1090.367	192.037	35.061	1230.700	168.000	30.672
$AP^{0}ML^{6}$	1211.100	164.523	30.038	1341.133	131.641	24.034
$AP^{0}ML^{9}$	1255.700	149.346	27.267	1394.533	146.345	26.719
$AP^{6}ML^{0}$	1007.700	141.790	25.887	1108.133	136.910	24.996
$AP^{6}ML^{3}$	1062.667	186.442	34.040	1190.733	193.966	35.413
$AP^{6}ML^{6}$	1025.133	248.363	45.345	1150.767	235.819	43.054
$AP^{6}ML^{9}$	1114.667	182.880	33.389	1246.700	181.431	33.125
$AP^{12}ML^{0}$	918.100	170.441	31.118	1038.967	148.325	27.080
$AP^{12}ML^{3}$	1044.833	196.613	35.896	1153.067	182.660	33.349
$AP^{12}ML^{6}$	950.267	233.962	42.715	1072.867	218.313	39.858
$AP^{12}ML^{9}$	995.467	211.410	38.598	1095.267	212.845	38.860
$AP^{18}ML^{0}$	908.200	166.490	30.397	1026.567	144.527	26.387
$AP^{18}ML^{3}$	981.733	252.885	46.170	1089.333	247.187	45.130
$AP^{18}ML^{6}$	877.867	297.501	54.316	983.133	301.100	54.973
$AP^{18}ML^{9}$	937.600	280.142	51.147	1048.167	292.192	53.347
$AP^{24}ML^{0}$	968.733	164.986	30.122	1079.133	155.365	28.366
$AP^{24}ML^{3}$	944.100	213.761	39.027	1056.167	211.565	38.626
$AP^{24}ML^{6}$	828.233	267.058	48.758	922.167	269.600	49.222
$AP^{24}ML^{9}$	748.267	241.006	44.001	844.133	231.932	42.345

Table 2: Descriptive statistics of the cancellous bone density at different points

Variables	Descriptive statistics					
	Smoker Group (N=30)			Control Group (N=30)		
	Mean	SD	SE	Mean	SD	SE
AP^0ML^0	765.433	143.543	26.207	889.433	117.519	21.456
AP^0ML^3	488.733	245.516	44.825	596.967	222.215	40.571
AP^0ML^6	436.000	211.074	38.537	505.700	173.867	31.744
AP^0ML^9	344.233	186.164	33.989	428.700	147.794	26.983
AP^6ML^0	776.267	170.345	31.101	884.167	145.202	26.510
AP^6ML^3	453.233	268.433	49.009	533.367	252.189	46.043
AP^6ML^6	578.600	261.690	47.778	675.000	254.435	46.453
AP^6ML^9	586.267	281.502	51.395	679.133	271.270	49.527
$AP^{12}ML^0$	778.700	148.197	27.057	889.767	135.902	24.812
$AP^{12}ML^3$	469.600	271.049	49.487	540.767	266.844	48.719
$AP^{12}ML^6$	734.433	292.104	53.331	824.800	313.909	57.312
$AP^{12}ML^9$	651.700	347.488	63.442	715.067	363.854	66.430
$AP^{18}ML^0$	760.733	184.728	33.727	864.133	155.977	28.477
$AP^{18}ML^3$	528.933	322.399	58.862	602.967	315.249	57.556
$AP^{18}ML^6$	676.400	382.215	69.783	783.900	383.782	70.069
$AP^{18}ML^9$	743.967	357.418	65.255	852.033	384.194	70.144
$AP^{24}ML^0$	697.433	213.086	38.904	807.600	183.907	33.577
$AP^{24}ML^3$	665.633	260.129	47.493	764.467	275.685	50.333
$AP^{24}ML^6$	778.167	333.169	60.828	855.867	342.645	62.558
$AP^{24}ML^9$	737.867	304.795	55.648	824.533	307.205	56.088

Table 3: Descriptive statistics and comparison between the groups in the cortical and cancellous bone density at the anterioposterior areas

Bone	Areas	Groups	Descriptive statistics			Groups' difference (df=58)	
			Mean	SD	SE	t-test	p-value
Cortical	Anterior area	Smoker	8832.93	937.85	171.23	4.180	0.000**
		Control	9843.07	933.81	170.49		
	Middle area	Smoker	3908.67	657.60	120.06	2.737	0.008**
		Control	4360.17	619.65	113.13		
	Posterior area	Smoker	7194.73	1278.52	233.43	2.598	0.012*
		Control	8048.80	1268.04	231.51		
Cancellous	Anterior area	Smoker	4428.77	1136.49	207.49	2.941	0.005**
		Control	5192.47	854.97	156.09		
	Middle area	Smoker	2634.43	807.21	147.38	1.620	0.111
		Control	2970.40	799.11	145.90		
	Posterior area	Smoker	5589.13	1484.79	271.08	2.021	0.048*
		Control	6355.50	1452.68	265.22		

***= Significant $0.05 \geq P > 0.01$; **=Highly significant $0.05 \geq P > 0.001$**

Table 4: Descriptive statistics and comparison between the groups in the cortical and cancellous bone density at the mediolateral areas

Bone	Areas	Groups	Descriptive statistics			Groups' difference (df=58)	
			Mean	SD	SE	t-test	p-value
Cortical	Mid-palatal area	Smoker	4868.33	611.71	111.68	3.664	0.001**
		Control	5433.17	581.90	106.24		
	Medial area	Smoker	5123.70	769.92	140.57	3.008	0.004**
		Control	5720.00	765.63	139.78		
	Middle area	Smoker	4892.60	931.96	170.15	2.411	0.019*
		Control	5470.07	923.15	168.54		
	Lateral area	Smoker	5051.70	717.60	131.02	3.059	0.003**
		Control	5628.80	743.62	135.77		
Cancellous	Mid-palatal area	Smoker	3778.57	650.46	118.76	3.670	0.001**
		Control	4335.10	516.66	94.33		
	Medial area	Smoker	2606.13	924.38	168.77	1.937	0.058
		Control	3038.53	800.64	146.18		
	Middle area	Smoker	3203.60	1056.04	192.81	1.638	0.107
		Control	3645.27	1032.95	188.59		
	Lateral area	Smoker	3064.03	1019.43	186.12	1.674	0.099
		Control	3499.47	994.94	181.65		

***= Significant 0.05 ≥ P >0.01; **=Highly significant 0.05 ≥ P > 0.001**

Table 5: Descriptive statistics and comparison between the groups in the overall cortical and cancellous bone density

Overall Bone density	Groups	Descriptive statistics			Groups' difference (d.f.=58)	
		Mean	SD	SE	t-test	p-value
Cortical	Smoker	19936.33	2673.98	488.20	3.369	0.001**
	Control	22252.03	2649.93	483.81		
Cancellous	Smoker	12652.33	2975.81	543.31	2.586	0.012*
	Control	14518.37	2600.47	474.78		

***= Significant 0.05 ≥ P >0.01; **=Highly significant 0.05 ≥ P > 0.001**