Implant Prosthodontics in Medically Challenged Patients: The University of Toronto Experience

(Les prothèses sur implant chez les patients ayant des problèmes de santé : l'expérience de l'Université de Toronto)

• Emad S. Elsubeihi, BDS, MS, Dip Prostho •
• George A. Zarb, BChD, DDS, MS, MS, FRCD(C) •

Sommaire

Une série d'études prospectives, commencées au milieu des années 80 à l'Université de Toronto, ont fourni des preuves de l'efficacité et de la rentabilité des implants dans le traitement de patients entièrement et partiellement édentés. Ces études se sont concentrées essentiellement sur les résultats du traitement aux niveaux de la chirurgie et de la dentisterie prothétique, avec un taux d'échec global de 7,7 % sur une période de 20 ans. Étant donné qu'une importante proportion de ces échecs (4,2 %) ont eu lieu avant l'insertion de la prothèse et que l'ostéo-intégration consiste essentiellement en un processus de cicatrisation, les facteurs qui interfèrent avec la cicatrisation, y compris les états systémiques, peuvent contribuer à l'échec de l'implant. Cet article examine les études sur l'impact de conditions systémiques sélectionnées, notamment l'ostéoporose, les maladies cardiovasculaires, le diabète sucré et l'hypothyroïdie, ainsi que l'usage du tabac, sur le succès ou la «survie» des implants dentaires chez les patients traités à l'Unité de dentisterie prothétique de l'Université de Toronto.

Mots clés MeSH : *dental implants; osseointegration/physiology; risk factors*

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A series of prospective studies undertaken since the mid-1980s in the Implant Prosthodontic Unit (IPU) at the University of Toronto have provided evidence of the efficacy and effectiveness of the osseointegration technique in the treatment of completely and partially edentulous patients. Data from this patient population have revealed impressive prosthodontic treatment outcomes: of the 1852 implants placed in 464 patients between 1979 and 1999, 143 (7.7%) failed, 78 (4.2%) of them failing to osseointegrate before insertion of the prosthesis.[1] Furthermore, studies have shown that failures tended to be concentrated in a few individuals, an occurrence described in the literature as the "cluster phenomenon".[2] Because osseointegration is essentially a wound-healing process, these observations suggest that factors that interfere with healing may contribute to implant failure. Hence, we contend that conditions shown to adversely affect wound healing may decrease the potential for successful osseointegration.

In this article we review IPU studies on the possible impact of certain systemic conditions, including osteoporosis, cardiovascular diseases, diabetes mellitus, and hypothyroidism, as well as smoking behaviour, on the success or "survival" of dental implants in 464 consecutively treated patients. The protocols for patient selection and treatment in these studies have been previously published.[3-7]

Osteoporosis

The term osteoporosis has been used loosely in the dental literature, often to imply postmenopausal osteoporosis. Both human and animal studies indicate that bone loss associated with postmenopausal osteoporosis results from an increase in bone turnover, in which the rates of both bone resorption and bone formation are increased. However, because the former

Table 1 **Effect of age (a marker of osteoporosis) on implant failure rate[a]**

	Age group; total no. of patients in group (and implant failure rate, %[b])	
	< 50 years	**≥ 50 years**
Women	48 (18.8%)	45 (22.2%)
Men	18 (11.1%)	18 (22.2%)

[a]Table courtesy of Dao and others.[19]
[b]Failure rate is based on the number of patients in each age group with implants, in contrast to Tables 3 to 5, where the failure rate is based on the total number of implants; consequently, the failure rates here appear higher than the failure rates in Tables 3 to 5. The failure rates were not significantly different between men and women 50 years of age or older. Likewise, no significant difference was detected between women less than 50 years of age and those 50 or older.

exceeds the latter, the net result is bone loss. Evidence at the gene expression,[8] cellular[9] and tissue[10,11] levels indicate that the rate of bone formation increases in postmenopausal osteoporosis. Furthermore, experimental evidence has shown that estrogen depletion leads to a significant loss of bone mass in the edentulous mandible but not in the dentate mandible.[12] Other experimental studies showed that reduced masticatory function resulted in a reduction of mandibular bone mass associated with reduced cortical thickness[13] and reduction in mineral apposition rates[14] consistent with findings from osteoporosis associated with immobilization (unloading).[15] It is not known, however, if mandibular bone loss observed in the edentulous mandible in association with estrogen deficiency[12] results from increased bone resorption alone or the combined effect of increased bone resorption and reduction in bone formation rate.

Interestingly, biochemical analysis of bone derived from the human osteoporotic femoral head showed evidence of overhydroxylation of lysine and a consequent reduction in the stabilizing cross-links of the collagenous framework, which has been suggested to contribute to increased fragility of bone.[16] Indeed, mechanical testing of healing femoral fractures in rats indicated that ovariectomy impairs fracture healing for up to 4 weeks after fracture. Healing returned to normal 6 weeks after fracture.[17] Furthermore, when 17-β-estradiol was administered during the period of fracture repair, there was a dose-dependent increase in the peak force required to re-break the fracture.[18] The impact of such findings on the bone–implant interface is not known.

In investigating the effect of osteoporosis on the success of dental implants, Dao and others[19] examined data from 93 women and 36 men treated in the IPU. The authors used the Smith and Zarb[20] criteria of success and compared implant failure between women and men 50 years of age or older and between women less than 50 years of age and those 50 years of age or older. The study design was based on the assumption that because osteoporosis is more prevalent in women at least 50 years of age (i.e., subjects at risk for osteoporosis), the frequency of failure would be greater in this patient group than in other groups. Patients were followed for 2 to 11 years, and failure rates were analyzed according to the number of patients with implants (not the total number of implants) (Table 1). Dao and others[19] concluded that patients at risk for osteoporosis did not appear to be at greater risk for implant failure. This observation led us to persist in our clinical judgement that a clinical diagnosis of osteoporosis should not preclude a prescription for implant prosthodontics. Subsequent studies[21,22] have supported this premise. The specific role of osteoporotic bone in the interfacial osteogenesis upon which successful osseointegration depends is incompletely understood.

Table 2 **Effect of cardiovascular diseases (CVD) on implant failure rate[a,b]**

	No. of patients	No. (and %) of patients with failed implants
Patients with CVDs of interest[c]	39	5 (13)
Control groups		
Healthy patients	98	12 (12)
Patients with systemic disease including CVDs not of interest[d]	109	15 (14)

[a]Table courtesy of Khadivi and others.[23]
[b]Failure rate is based on the number of patients in each age group with implants, in contrast to Tables 3 to 5, where the failure rate is based on the total number of implants; consequently, the failure rates here appear higher than the failure rates in Tables 3 to 5.
[c]CVDs of interest included hypertension, atherosclerosis, vascular stenosis, coronary artery disease and congestive heart failure.
[d]CVDs not of interest include dysrhythmia and heart murmur.

Cardiovascular Diseases

Certain cardiovascular diseases, including hypertension, artherosclerosis, vascular stenosis, coronary artery disease, and congestive heart failure, can compromise blood flow and reduce oxygen tension and nutrient supply to tissues. They might therefore be expected to compromise the outcome of osseointegration.

In a retrospective study of 246 consecutively treated patients (153 women and 93 men) Khadivi and others[23] investigated the impact of these diseases on the outcome of osseointegration at stage II surgery using the Smith and Zarb[20] criteria of success. The prevalence of cardiovascular diseases in the examined population was 23.9%. There was no significant difference in the rate of implant failure between patients with cardiovascular diseases (13%) and the control population (12%) (Table 2). The authors concluded that patients with controlled cardiovascular diseases are not at higher risk of osseointegration failure than patients without such conditions.

Diabetes Mellitus

Diabetes mellitus is a common metabolic disorder affecting 4% of the Canadian population.[24] Diabetic patients have a wide range of defects that delay the healing process and that increase their susceptibility to infection.[25,26] Furthermore, the prevalence of osteopenia among patients with diabetes tends to be greater

Table 3 **Effect of medically controlled diabetes mellitus on implant failure rate[a,b]**

	Total no. of implants	No. (and %) of failed implants
Diabetic group	59	4 (7)
Control group	111	7 (6)

[a]*Table courtesy of Accursi.*[27]
[b]*Implant failure rate is based on the total number of implants. Failure was assessed according to the criteria of Zarb and Albrektsson.*[28]

Table 4 **Effect of medically controlled hypothyroidism on implant failure rate[a,b]**

	Total no. of implants	No. (and %) of failed implants
Hypothyroid group	82	3 (4)
Control group	81	2 (2)

[a]*Table courtesy of Attard.*[33]
[b]*Implant failure rate is based on the total number of implants. Failure was assessed according to the criteria of Zarb and Albrektsson.*[28] *There was no statistically significant difference in implant failure rate between the hypothyroid and control groups.*

than among the general population; this difference may be related to hyperglycemia in the former group. It has been reported that long-term bone loss is more severe among patients with type 1 diabetes than among those with type 2 diabetes and that bone mineral density in patients with type 1 diabetes is at least 10% lower than among sex- and age-matched healthy people.[27] Studies performed with untreated type 1 diabetic animal models showed evidence of lower numbers of osteoblasts, less osteoid surface, and lower plasma osteocalcin levels, consistent with decreased rates of bone formation.[28,29]

Accursi[30] examined the impact of diabetes on the success of dental implants in patients who were followed for 1 to 17 years. In that study each of 15 diabetic patients (representing 3.9% of the patient population at the IPU) was matched to 2 control subjects by age, sex, location of implants, type of prosthetic restoration, opposing dentition, and duration of edentulism. A total of 59 implants in the diabetic group were compared with 111 implants in the control group according to the Zarb and Albrektsson[31] criteria of success. The diabetic patients were no more likely to experience implant failure than the nondiabetic patients (Table 3). In assessing changes in crestal bone levels around the implants, the researchers found that diabetic patients had greater loss of crestal bone during the first year of implant loading (mean ± standard deviation 0.25 ± 0.07 mm) than nondiabetic controls (0.06 ± 0.03 mm). However, this difference disappeared in subsequent years, when loss of crestal bone among diabetic patients matched that for nondiabetic patients. Accursi[30] also reported interesting soft-tissue and neurological complications in the diabetic group. He found that soft-tissue complications were similar in number in the diabetic and control groups; in both groups, these complications were mainly of a minor nature (including redness, bleeding and minor swelling) and resolved with improvement in oral hygiene. Conversely, the incidence of paresthesia among diabetic patients was higher than among nondiabetic controls, and the diabetic patients reported less postoperative pain. These findings suggested that controlled diabetic patients are not at higher risk of implant failure.

Hypothyroidism

Hypothyroidism decreases recruitment, maturation and activity of bone cells, leading to decreased bone resorption and formation.[32] Thyroid hormone exerts a direct effect on bone to increase production of both insulin-like growth factor-I (IGF-I) and IGF binding protein II.[33] IGF-I increases the number of osteoblasts, enhances osteoblast differentiation and increases bone remodelling, but the levels of circulating IGF-I are decreased in hypothyroidism.[34] Experimental evidence[35] has suggested that hypothyroidism may inhibit fracture healing and impair the mechanical properties of fracture callus, which indicates that thyroid hormone is a critical factor in fracture healing.

Attard[36] investigated the survival of dental implants in hypothyroid patients receiving thyroid hormone replacement therapy. A total of 27 patients with hypothyroidism were matched with a control group by age, sex, location of implants, type of prosthesis and opposing dentition. The results for 82 implants in medically hypothyroid patients were compared with those for 81 implants in the control group (Table 4); the implants had been in place for 1 to 20 years. There was no statistical difference in the rate of implant failure between the 2 groups. However, analysis of marginal bone around the implants revealed that there was more loss of marginal bone in the first year of loading in the hypothyroid group. This bone loss seemed to slow down in subsequent years, approaching that of the control group. The results suggest that medically controlled hypothyroid patients are not at higher risk of implant failure than matched controls.

Smoking

Cigarette smoking impairs soft-tissue wound healing by affecting the circulatory and immune systems and by impairing normal cellular function. Furthermore, it appears that cigarette smoking during adulthood is associated with decreased hip[37] and vertebral[38] bone density later in life among both women and men. In a study of 41 pairs of twins, those who smoked more heavily had bone mineral density values 5% and 10% lower at the femoral neck and lumbar spine, respectively, for each 20 pack-year difference.[39] The exact mechanism by which smoking exerts its negative effect on bone is not yet fully understood. Bone loss occurs if there is an imbalance between the amount of bone resorbed and the amount of bone formed. The evidence available examining whether one or both of these mechanisms contribute to the bone loss associated with smoking is limited. Hopper and Seeman[39] demonstrated that lower bone density at the lumbar spine in smokers was associated with higher serum calcium

Table 5 **Effect of smoking on implant failure rate (in relation to number of implants)[a]**

	Total no. of implants	No. (and %) of failed implants Early failure	Late failure
Stage I surgery			
Smokers	494	6 (1.2)[b]	10 (2.0)
Nonsmokers	1045	32 (3.1)	35 (3.3)
Smoking history			
Positive	860	38 (4.4)	32 (3.7)[b]
Negative	679	20 (2.9)	13 (1.9)

[a]*Table courtesy of Habsha.*[1]
[b]$P < 0.05$.

Table 6 **Effect of smoking on implant failure rate (in relation to number of patients)[a]**

	No. of patients	No. (and %) of patients with failed implants Early failure	Late failure
Stage I surgery			
Smokers	104	18 (17.3)	8 (7.7)
Nonsmokers	285	24 (8.4)	21 (7.4)
Smoking history			
Positive	192	27 (14.1)	19 (9.9)
Negative	197	15 (7.6)	10 (5.1)

[a]*Table courtesy of Habsha.*[1]

and urine pyridinoline levels, which would be consistent with increased bone resorption. Furthermore, it was suggested that increased bone resorption associated with smoking is, in part, due to decreased production and accelerated degradation of estrogen, which leads to early menopause and higher rate of bone loss. However, histomorphometric investigations[40] suggested that a reduction in bone formation is responsible for the deficit in bone volume seen in smokers. In vitro studies using rat bone marrow cell cultures showed that aryl hydrocarbons, environmental contaminants occurring at high levels in cigarette smoke, inhibit osteodifferentiation and osteogenesis.[41] Furthermore, in vivo animal studies have shown that nicotine impairs bone healing.[42]

Habsha[1] studied the survival of dental implants in relation to smoking history in 464 consecutively treated patients who had had their implants for 1 to 20 years. Initially, patients were grouped on the basis of whether they were smokers or nonsmokers. Smokers were defined as those who smoked at the time of implant placement (stage I surgery). Nonsmokers were defined as those who had never smoked or who had quit smoking before implant placement. Smokers had a higher rate of early implant failure than nonsmokers (Table 5). Patients were then grouped on the basis of their smoking history, where smoking history takes into account the quantity of cigarettes consumed and the number of years during which they were consumed. Two groups were compared: a control group of patients with negative smoking history, which included individuals who had never smoked or who had smoked no more than 25 cigarette-years until stage II surgery (where a cigarette-year is the product of the number of cigarettes smoked per day and the number of years of smoking) and patients with a positive smoking history, which encompassed those with a smoking history of more than 25 cigarette-years. Although there were no significant differences in the rate of early implant failure between the groups, those with a positive smoking history had a significant prevalence of late implant failure (Table 5). Relative risks were calculated, and it was concluded that among patients who smoked during the initial healing phase the incidence of implant failure was 1.69 times greater than among those who did not smoke. Furthermore, patients with a significant smoking history (more than 25 years) had 1.91 times the risk of late implant failure than those who did not smoke.

In an analysis of implant failure according to the number of patients, it was found that a higher proportion of smokers sustained implant failure than nonsmokers (Table 6). This indicates that the failures were not clustered within individuals, as had previously been suggested.[2]

Conclusions and Future Research Directions

Data from patients treated in the IPU at the University of Toronto suggest that patients at risk for osteoporosis and those with cardiovascular diseases, controlled diabetes and hypothyroidism are not at greater risk of implant failure. However, patients who smoke are at greater risk. Although these results appear reassuring, it should be emphasized that the strength of these studies was limited by their retrospective design and small sample sizes. The effects of systemic conditions on bone changes in the jaws are not fully understood. Clearly, animal models are convenient research tools to investigate these changes and the impact of systemic diseases on the healing behaviour of osseointegrated implants. Although several attempts to address these concerns have been reported, important considerations such as appropriateness of the models have been overlooked. Furthermore, as more evidence is presented to support the notion of heterogeneity of the skeleton, it is important that healing of implants be investigated in relevant sites, specifically the mandible and maxilla. The subsequent loaded and time-dependent integrity of the osseointegrated response must also be investigated to avoid inadequate interpretations of the pathogenesis of implant failure. A lack of scientific rigour in the reporting of the long-term outcomes of implant prosthodontic treatment has led to simplistic and possibly incorrect comparisons of implant failure to processes resembling periodontal disease. The fundamental difference between the developmental nature of a periodontal ligament and the healing response elicited in the osseointegration protocol is all too frequently overlooked, and much clinical confusion has resulted.[43] A basic understanding of the nature of the interfacial healing response of osseointegration, particularly in

the context of relative or adverse occlusal overloading, is more likely to affect the dental profession's collective understanding of why osseointegration sometimes fails.

At this stage of our understanding of the nature of the osseointegration response, it appears prudent to conclude that implant failure is most likely multifactorial. The studies reviewed here attempted to control for confounding factors such as age, sex, time since implantation, implant location, bone quality, opposing dentition, medical conditions and smoking habits. Even when such variables are controlled for, studies may document the presence or absence of associations but do not prove causality. Proving causality usually requires a randomized intervention study, the design and performance of which are usually formidable. Experimental studies, which are easier to design and which allow for better control of confounding factors, as well as larger multicentre clinical studies, are clearly needed to further elucidate the causes and mechanisms of implant failure. ◆

Le Dr Elsubeihi est un étudiant de troisième cycle à la faculté de médecine dentaire, Université de Toronto, Toronto (Ontario).

Le Dr Zarb est professeur et directeur, prosthodontie, au département des sciences cliniques, faculté de médecine dentaire, Université de Toronto.

Écrire au : Dr Emad S. Elsubeihi, Département des sciences cliniques, Faculté de médecine dentaire, Université de Toronto, 124, rue Edward, Toronto (Ontario) M5G 1G6. Courriel : emad.elsubeihi@utoronto.ca.

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Références

1. Habsha E. Survival of osseointegrated dental implants in smokers and non-smokers [MS thesis]. Toronto (ON): University of Toronto; 2000.

2. Weyant RJ, Burt BA. An assessment of survival rates and within-patient clustering of failures for endosseous oral implants. *J Dent Res* 1993; 72(1):2-8.

3. Cox JF, Zarb GA. The longitudinal clinical efficacy of osseointegrated dental implants: a 3-year report. *Int J Oral Maxillofac Implants* 1987; 2(2):91-100.

4. Zarb GA, Schmitt A. The longitudinal clinical effectiveness of osseointegrated dental implants: the Toronto study. Part I: Surgical results. *J Prosthet Dent* 1990; 63(4):451-7.

5. Zarb GA, Schmitt A. The longitudinal clinical effectiveness of osseointegrated dental implants: the Toronto study. Part II: The prosthetic results. *J Prosthet Dent* 1990; 64(1):53-61.

6. Avivi-Arber L, Zarb GA. Clinical effectiveness of implant-supported single-tooth replacement: the Toronto study. *Int J Oral Maxillofac Implants* 1996; 11(3):311-21.

7. Wyatt CC, Zarb GA. Treatment outcomes of patients with implant-supported fixed partial prostheses. *Int J Oral Maxillofac Implants* 1998; 13(2):204-11.

8. Davey RA, Hahn CN, May BK, Morris HA. Osteoblast gene expression in rat long bones: effects of ovariectomy and dihydrotestosterone on mRNA levels. *Calcif Tissue Int* 2000; 67(1):75-9.

9. Jilka RL, Takahashi K, Munshi K, Williams DC, Roberson PK, Manolagas SC. Loss of estrogen upregulates osteoblastogenesis in the murine bone marrow. Evidence for autonomy from factors released during bone resorption. *J Clin Invest* 1998; 101(9):1942-50.

10. Wronski TJ, Dann LM, Horner SL. Time course of vertebral osteopenia in ovariectomized rats. *Bone* 1989; 10(4):295-301.

11. Yokose S, Ishizuya T, Ikeda T, Nakamura T, Tsurukami H, Kawasaki K, and others. An estrogen deficiency caused by ovariectomy increases plasma levels of systemic factors that stimulate proliferation and differentiation of osteoblasts in rats. *Endocrinology* 1996; 137(2):469-78.

12. Elsubeihi ES, Grynpas MD, Cheung A, Zarb GA, Heersche JNM. The effect of ovariectomy on bone loss in the edentulous and dentate mandible. *Bone* 2001; 28(5 Suppl):Abstract P525S:S217.

13. Bresin A, Kiliaridis S, Strid KG. Effect of masticatory function on the internal bone structure in the mandible of growing rat. *Eur J Oral Sci* 1999; 107(1):35-44.

14. Yamada K, Kimmel DB. The effect of dietary consistency on bone mass and turnover in the growing rat mandible. *Arch Oral Biol* 1991; 36(2):129-38.

15. Minaire P, Meunier P, Edouard C, Bernard J, Courpron P, Bourret J. Quantitative histological data on disuse osteoporosis: comparison with biological data. *Calcif Tissue Res* 1974; 17(1):57-73.

16. Bailey AJ, Knott L. Molecular changes in bone collagen in osteoporosis and osteoarthritis in the elderly. *Exp Gerontol* 1999; 34(3):337-51.

17. Walsh WR, Sherman P, Howlett CR, Sonnabend DH, Ehrlich MG. Fracture healing in a rat osteopenia model. *Clin Orthop Rel Res* 1997; (342):218-27.

18. Bolander ME, Sabbagh RC, Jeng C, Vivianno D, Boden SD. Estrogen treatment during fracture repair strengthens healing callus in an experimental model. *Trans Orthop Res Soc* 1992; 17:138.

19. Dao TT, Anderson JD, Zarb GA. Is osteoporosis a risk factor for osseointegration of dental implants? *Int J Oral Maxillofac Implants* 1993; 8(2):137-44.

20. Smith DE, Zarb GA. Criteria for success of osseointegrated endosseous implants. *J Prosthet Dent* 1989; 62(5):567-72.

21. Minsk L, Polson AM. Dental implant outcomes in postmenopausal women undergoing hormone replacement. *Compend Contin Educ Dent* 1998; 19(9):859-62, 864.

22. Becker W, Hujoel PP, Becker BE, Willingham H. Osteoporosis and implant failure: an exploratory case-control study. *J Periodontol* 2000; 71(4):625-31.

23. Khadivi V, Anderson J, Zarb GA. Cardiovascular disease and treatment outcomes with osseointegration surgery. *J Prosthet Dent* 1999; 81(5):533-6.

24. Tan H, MacLean DR. Epidemiology of diabetes mellitus in Canada. *Clin Invest Med* 1995; 18(4):240-6.

25. Devlin H, Garland H, Sloan P. Healing of tooth extraction sockets in experimental diabetes mellitus. *J Oral Maxillofac Surg* 1996; 54(9):1087-91.

26. Fahey TJ 3rd, Sadaty A, Jones WG 2nd, Barber A, Smoller B, Shires GT. Diabetes impairs the late inflammatory response to wound healing. *J Surg Res* 1991; 50(4):308-13.

27. Mathiassen B, Nielsen S, Ditzel J, Rodbro P. Long-term bone loss in insulin-dependent diabetes mellitus. *J Intern Med* 1990; 227(5):325-7.

28. Verhaeghe J, van Herck F, Visser WJ, Suiker AM, Thomasset M, Einhorn TA, and others. Bone and mineral metabolism in BB rats with long-term diabetes. Decreased bone turnover and osteoporosis. *Diabetes* 1990; 39(4):477-82.

29. Sasaki T, Kaneko H, Ramamurthy NS, Golub LM. Tetracycline administration restores osteoblast structure and function during experimental diabetes. *Anat Rec* 1991; 231(1):25-34.

30. Accursi GE. Treatment outcomes with osseointegrated Branemark implants in diabetic patients: a retrospective study [thesis]. Toronto (ON): University of Toronto; 2000.

31. Zarb GA, Albrektsson T. Consensus report: towards optimized treatment outcomes for dental implants. *Int J Prosthodont* 1998; 11(5):389.

32. Mosekilde L, Eriksen EF, Charles P. Effects of thyroid hormones on bone and mineral metabolism. *Endocrinol Metab Clin North Am* 1990; 19(1):35-63.

33. Schmid C, Schlapfer I, Futo E, Waldvogel M, Schwander J, Zapf J, and other. Triiodothyronine (T3) stimulates insulin-like growth factor (IGF)-1 and IGF binding protein (IGFBP)-2 production by rat osteoblasts in vitro. *Acta Endocrinol* 1992; 126(5):467-73.

34. Valimaki M, Liewendahl, Karonen SL, Helenius T, Suikkari AM. Concentrations of somatomedin-C and triiodothyronine in patients with thyroid dysfunction and nonthyroid illnesses. *J Endocrinol Invest* 1990; 13(2):155-9.

35. Urabe K, Hotokebuchi T, Oles KJ, Bronk JT, Jingushi S, Iwamoto Y, and other. Inhibition of endochondral ossification during fracture repair in experimental hypothyroid rats. *J Orthop Res* 1999; 17(6):920-5.

36. Attard N. Implant prosthodontic management of medically treated hypothyroid patients [MS thesis]. Toronto (ON): University of Toronto; 2001.

37. Hollenbach KA, Barrett-Connor E, Edelstein SL, Holbrook T. Cigarette smoking and bone mineral density in older men and women. *Am J Public Health* 1993; 83(9):1265-70.

38. Egger P, Duggleby S, Hobbs R, Fall C, Cooper C. Cigarette smoking and bone mineral density in the elderly. *J Epidemiol Community Health* 1996; 50(1):47-50.

39. Hopper JL, Seeman E. The bone density of female twins discordant for tobacco use. *N Engl J Med* 1994; 330(6):387-92.

40. de Vernejoul MC, Bielakoff J, Herve M, Gueris J, Hott M, Modrowski D, and others. Evidence for defective osteoblastic function. A role for alcohol and tobacco consumption in osteoporosis in middle-aged men. *Clin Orthop* 1983; (179):107-15.

41. Ferrara GE, Sukhu B, Casper RF, Savouret JF, D'Agata R, Tenenbaum H. Inhibition of osteoblast function in vitro by benzo[a]pyrene and dimethylbenz[a]anthracene and the preventative effect of resvertrol. *J Bone Miner Res* 2000; 15(Suppl 1):Abstract M192:S502.

42. Hollinger JO, Schmitt JM, Hwang K, Soleymani P, Buck D. Impact of nicotine on bone healing. *J Biomed Mater Res* 1999; 45(4):294-301.

43. Zarb GA, Albrektsson T. Osseointegration: a requiem for the periodontal ligament? Guest editorial *Int J Periodontics Restorative Dent* 1991; 11:88.

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