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Temperature changes in dental implants following exposure to hot substances in an *ex vivo* model

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Key words: bone, implant, hot beverages, hot foods, temperature

Abstract

Objectives: The habitual consumption of extremely hot foods and beverages may affect implant treatment modality. Our objectives were to: (i) establish the maximum temperature produced intra-orally while consuming very hot substances and (ii) use these values in an *ex vivo* model to assess the temperature changes along the implant–bone interface.

Materials and methods: Temperatures were measured using thermocouples linked to a computer. The thermocouple electrodes were attached to the tooth–gum interface of the interproximal areas in 14 volunteers during consumption of extremely hot foods and beverages. The *in vivo* measured temperature values obtained were used in an *ex vivo* model of a bovine mandible block with an implant and with an assembled abutment. Temperatures were measured by thermocouple electrodes attached to five locations, three of them along the implant–bone interface.

Results: During consumption of a hot beverage, a maximum temperature of up to 76.3°C was recorded, and a calculated extreme intra-oral temperature of 61.4°C was established. The *ex vivo* model showed a high correlation between the temperature measured at the abutment and that measured at the abutment–implant interface and inside the implant, reaching maximum temperatures close to 60°C. At the mid-implant–bone and apical implant–bone interfaces, the maximum temperatures measured were 43.3 and 42°C, respectively.

Conclusions: The maximum temperatures measured at the implant–bone interfaces reached the temperature threshold of transient changes in bone (42°C). The results of this study support the notion that intra-oral temperatures, developed during the consumption of very hot substances, may be capable of damaging peri-implant tissues.

High temperatures may cause irreversible damage to tissues and organs. A small number of patients have the habit of consuming extremely hot foods and beverages. The impact of such a habit on implant treatment modality is poorly characterized. Hot beverages typically anesthetize the oral tissues (Moritz & Henriques 1947). A link between oral tissue loss and exposure to hot beverages, foods, and smoking was suggested (Cullen 1998). Oral-burn syndrome was the proposed term for describing previously unexplained changes in intra-oral tissues, including tissue loss associated with dental implant due to possible exposure to high temperatures (Cullen 1998).

A temperature ranging between 50 and 60°C, simulating a high-end intra-oral temperature, is often used in thermocycling for testing *in vitro* dental materials (Ben-Amar et al. 1986; Kanca 1988). However, there is a wide variation in the

regimens used, the high temperature ranging from 40 to 100°C (Gale & Darvell 1999). Several studies on the range of intra-oral temperatures obtained upon ingestion of food or liquid suggested that the thermo-cycling regimens in the *in vitro* studies are excessive (Longman & Pearson 1987; Michailesco et al. 1995; Youngson & Barclay 2000). Others showed that the temperature produced intra-orally during hot water consumption may reach 67°C (Palmer et al. 1992) and even 77°C (Barclay et al. 2005).

Heat generation in bone during implant insertion has been well characterized (Matthews & Hirsch 1972; Eriksson & Adell 1986; Benington et al. 1996). Temperature changes at the cervical implant during performance of clinical procedures *in vitro* were also studied (Gross et al. 1995; Ormianer et al. 2000). However, there is no information on the temperature developed at the implant–bone interface during consumption of hot substances. The threshold level for heat-induced cortical bone tissue necrosis is 47°C for 1 min (Eriksson & Albrektsson 1983). Heat shock at 42°C induced transient changes in osteoblasts (Li et al. 1999).

We hypothesize that heat generated by excessive exposure to hot substances may exceed the threshold levels for peri-implant tissue damage. The objectives of this study were to: (i) establish the maximum temperature produced intra-orally while consuming very hot substances and (ii) use these values in an *ex vivo* bovine mandible model to assess the temperature changes along the implant–bone interface.

Materials and methods

Maximum temperature measurement *in vivo* during consumption of a hot food and beverage

Fourteen students and staff members of the Hadassah-Hebrew University Dental School, aged 20–50 years, volunteered to participate in the first part of this study. Inclusion criteria included healthy individuals with natural dentition. The procedure, possible discomfort, and risks were fully explained and approved by all volunteers.

Thermocouple electrodes with an accuracy of ± 0.5°C were used to record temperature changes *in vivo*. The thermocouples were linked to a computer with data recording and analysis software (Almemo, Holzkirchen, Germany). Four thermocouple electrodes (0.2 mm diameter) were attached to the tooth–gum interface by a dental toothpick passed through the embrasures between the following teeth: upper incisors, lower incisors, upper pre-molars, and lower molars. Tea or a potato was heated to 90°C. Each volunteer was asked to start eating or drinking only when the temperature was comfortable enough not to cause pain or injury. Temperatures were measured and recorded every 5 s. Maximum temperatures were recorded by each electrode for each volunteer during consumption of the hot beverage or food. According to Palmer et al. (1992), the calculated extreme temperature is obtained by adjusting for error tolerance, adding two standard deviations to the mean maximum temperatures measured *in vivo*, to best guarantee adequate range (95% probability that the measured maximum temperature lies within this range). This value was then used in the *ex vivo* model.

Temperature measurements in different implant locations in the *ex vivo* model

The model consisted of blocks of fresh bovine mandible into which implants were inserted. In the first experiment, two blocks, 1.5 × 4 × 5 cm each, were cut from the bovine mandible, using a diamond low-speed sectioning machine (Buehler, Dusseldorf, Germany). A total of eight titanium-alloy Tapered Screw-Vent dental implants (Zimmer Dental, Carlsbad, CA, USA) were inserted into the bone blocks: four had a 3.5 mm diameter, of which two were coated with hydroxyapetite, and four had a 4.5 mm diameter, of which two were coated with hydroxyapetite. An appropriate titanium-alloy abutment was assembled with each implant. Two compartments separated by a plastic layer sealed with silicon were formed (Fig. 1), the upper compartment containing the abutment, simulating the oral cavity, and the lower compartment containing the bone block with the inserted implant simulating the host tissues. The lower compartment was maintained at 37°C throughout all the experiments, using a Thermoelectric Table (Dental Iceberg, Duisburg, Germany). The upper compartment contained heated water that was allowed to cool spontaneously to 37°C. During this period of time, temperatures were recorded continuously every 5 s along the implant at the following locations: abutment (T1), abutment–implant interface (T2), inside the implant cavity (T3), mid-implant–bone interface (T4), and apical implant–bone interface (T5). Thermocouples were linked to a computer with data recording and analysis software (Almemo). In a second experiment, eight bone blocks from a bovine mandible were prepared, and eight titanium-alloy Taper-Lock™ Screw dental implants (Zimmer Dental), 4 mm in diameter, were inserted into the blocks. The experiment was repeated as described above, and temperature measurements were recorded three times for each implant ($n = 24$).

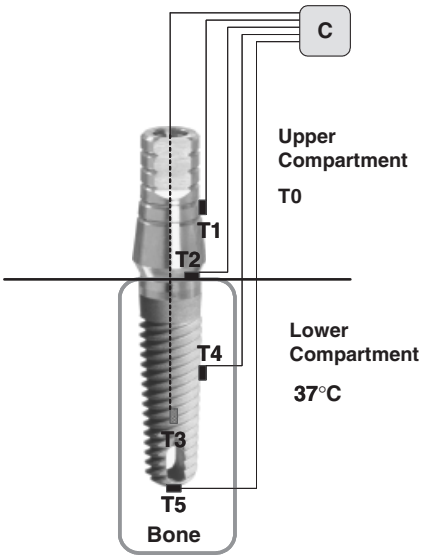

Fig. 1. Scheme of an *ex vivo* two-compartment model for temperature measurements in different implant locations. A titanium-alloy implant was inserted into a bovine mandible block immersed in water heated to 37°C in the lower compartment. An appropriate titanium-alloy abutment was assembled with the implant and placed in the upper compartment, which contained water heated to the temperature that was assessed from the *in vivo* experiments (T0). Five thermocouple electrodes were attached to the: abutment (T1), abutment–implant interface (T2), inside the implant cavity (T3), mid-implant–bone interface (T4), and apical implant–bone interface (T5). Thermocouples were linked to a computer (C) with data recording and analysis software.

Statistical analysis

The Pearson correlation coefficient (r) was calculated and the linear regression model

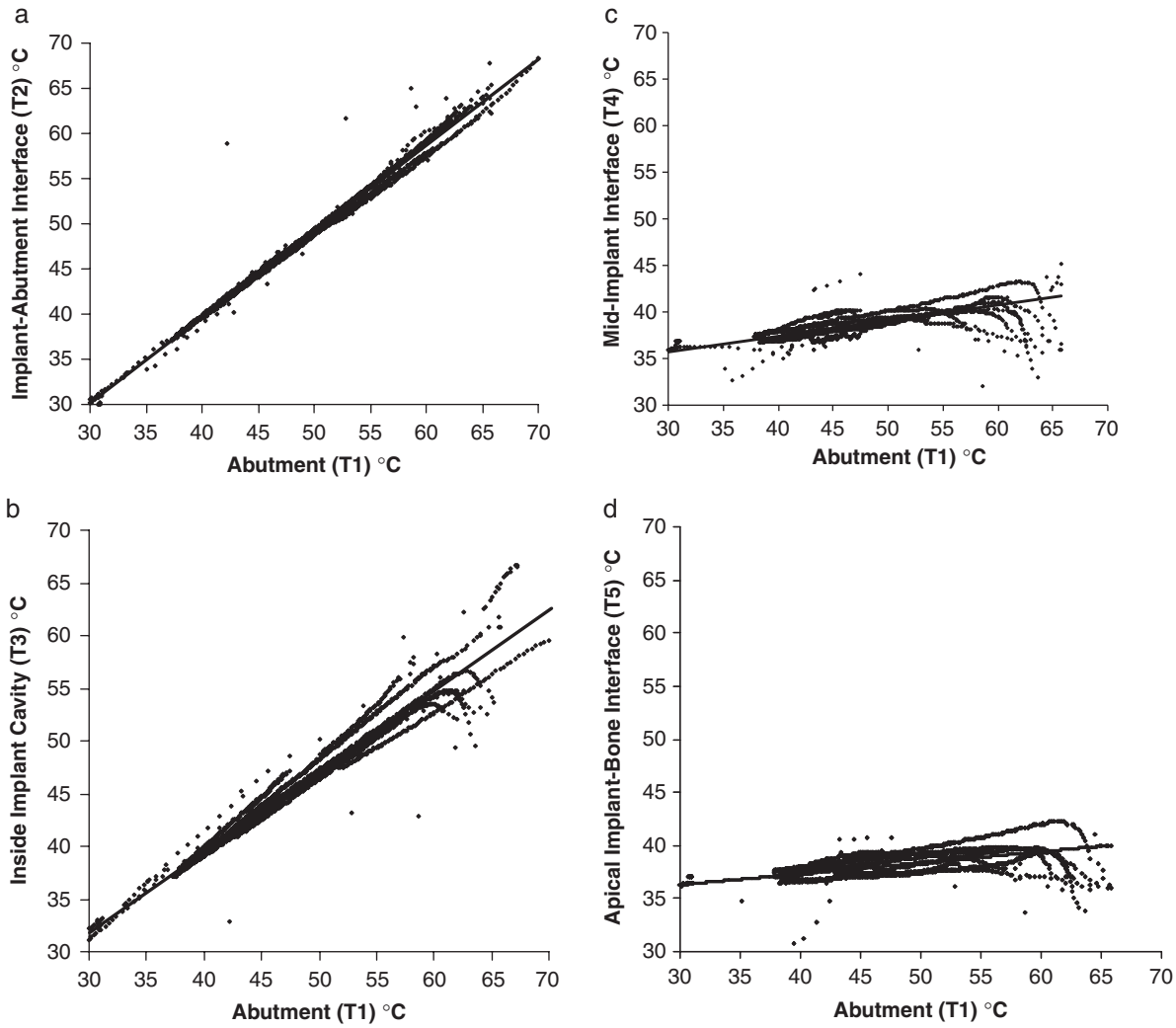

Fig. 2. Linear regression model depicting the correlation between the temperature measured at the abutment (T1) and that measured in (a) the implant–abutment interface (T2) ($r^2 = 0.99$), (b) inside the implant cavity (T3) ($r^2 = 0.97$), (c) the mid-implant interface (T4) ($r^2 = 0.57$), and (d) at the apical implant–bone interface (T5) ($r^2 = 0.35$).

Table 1. **Highest, mean, maximum, and calculated extreme temperatures (°C), measured *in vivo* during consumption of hot beverage and food**

Temperature (°C)	Hot beverage	Hot food
Highest*	76.3	53.6
Mean maximum†	46.4 ± 7.5	41.6 ± 4.3
Calculated extreme‡	61.4	50.2

*The highest temperature measured in one volunteer between the lower incisors.
†The mean maximum temperatures ± standard deviation recorded by each electrode for all volunteers.
‡The calculated extreme temperature obtained by adding two standard deviations to the mean maximum temperatures measured *in vivo*.

was applied to describe the association between the temperature measured at the abutment and that measured in different implant locations. The significance of the differences in temperature developed along the implants between implant sizes or between coated and uncoated implants was assessed using the Mann–Whitney nonparametric test. A P value of 5% or less was considered to be statistically significant.

Results

Maximum temperature measurement *in vivo* during consumption of a hot food and beverage

The highest temperature for hot beverage consumption was measured between the lower incisors, reaching a maximum value of 76.3°C (Table 1). The mean of the maximum temperatures recorded by each electrode for all volunteers was 46.4 ± 7.5°C (Table 1). For hot food, the highest temperature was measured in the upper incisor, reaching a value of 53.6°C, and the mean maximum temperature was 41.6 ± 4.3°C (Table 1). The extreme intraoral temperature during hot food and beverage consumption was calculated by adding two standard deviations to the mean of the maximum temperatures. The calculated extreme temperature was 61.4°C for

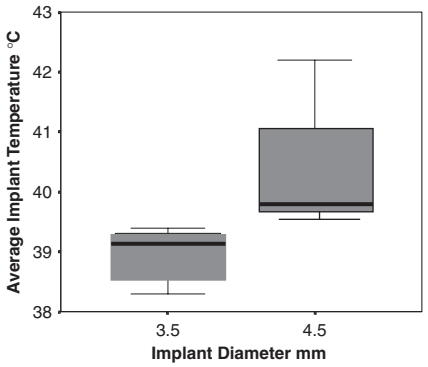

Fig. 3. Box plots of the average temperature along the bone–implant interface (T2, T4, and T5) measured in 3.5 and 4.5 mm diameter implants. Mann–Whitney Test, $P = 0.02$.

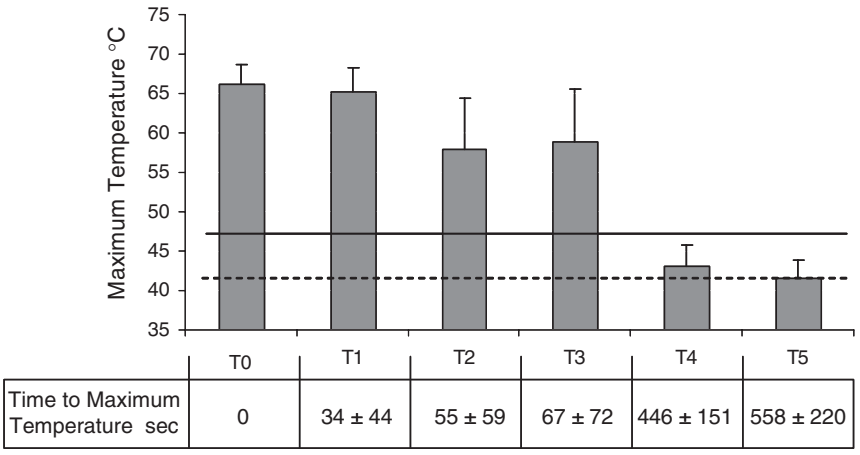

Fig. 4. Maximum temperature measurements (mean ± standard deviation; $n = 24$) for the implants at the upper compartment (input temperature – T0) and at different implant locations: T1, abutment; T2, abutment–implant interface; T3, inside the implant cavity; T4, mid-implant–bone interface; and T5, apical implant–bone interface. Error bars represent standard deviations. Time [mean (s) ± standard deviation] between exposure to T0 and the maximum temperature measured at the respective implant locations are shown below the *X* axis. The broken line indicates the temperature threshold of transient changes in osteoblasts (42°C) (Li et al. 1999). The continuous line indicates the threshold level of bone tissue necrosis (47°C) (Eriksson & Albrektsson 1983).

hot beverages and 50.2°C for hot food (Table 1). A temperature above 42°C was sustained for the longest period of time in the lower molars; this was observed for 13 min and for 4 min during consumption of hot beverage and hot food, respectively (data not shown).

Temperature measurements in different implant locations in the *ex vivo* model

The input temperature in the upper compartment, measured by the electrode attached to the abutment (T1), and the temperatures measured at the same time in the lower compartment by electrodes attached to all implants are shown in Figs 2 and 3.

The linear regression model and the r^2 were used as descriptive measurements. The linear regression model showed a high correlation between the temperature measured at the abutment (T1) and that measured in the abutment–implant interface (T2) (Fig. 2a, $r = 0.99$), and inside the implant (T3) (Fig. 2b, $r = 0.98$). A lower correlation was found between the temperature measured at the abutment (T1) and that measured in the mid-implant–bone interface (T4) (Fig. 2c, $r = 0.75$), and in the apical implant–bone interface (T5) (Fig. 2d, $r = 0.59$).

The measured temperature at the bone–implant interface along the 3.5 mm diameter implants was about 1°C lower than that of the 4.5 mm diameter implants ($P = 0.02$) (Fig. 3). The temperature measured along the hydroxyapatite-coated implants was not significantly different from the temperature measured along the non-coated implants ($P = 0.83$) (not shown).

The maximum temperature measured at the abutment (T1) was virtually equal to that of the heated water in the upper compartment. The maximum temperature measured at the abutment–implant interface (T2) and inside the implant (T3) reached a temperature above 57°C, and that measured at the mid (T4) and apical implant–bone (T5) interfaces reached 43 and 41.6°C, respectively (Fig. 4).

A time delay between exposure to input temperature (in the upper compartment) and the maximum temperature measured at the implant was observed: at the abutment (T1) a delay of 34 s, at the abutment–implant interface (T2) and inside the implant cavity (T3) a delay of approximately 60 s, and at the mid-implant–bone interface (T4) and at the apical implant–bone interface (T5) delays of 446 and 558 s, respectively (Fig. 4).

Discussion

The established maximum temperature values measured intra-orally were 76.3°C for hot beverages and 53.6°C for hot foods. These temperatures may represent the situation in the small number of patients who habitually consume extremely hot foods and beverages. To date, the impact of such a habit on implant treatment modality has not been characterized.

The high-temperature volunteers did not report any damage or discomfort of the oral tissues, nor was any damage observed clinically. The highest tolerable temperature is subjective and thus varies considerably among the population (Palmer et al. 1992; Barclay et al. 2005), and is probably affected by factors such as the degree of keratinization of the oral mucosa and age. Plant et al. (1974) determined that coffee in the cup was too hot to sip above 68°C, but subjects could sip it with discomfort between 60 and 68°C. Mouth temperature was limited by sipping and simultaneous intake of air (Plant et al. 1974), or by the protection of soft tissues (Lloyd et al. 1978) afforded to certain oral sites. The buccal aspect of the lower incisors and the palatal aspect of the upper incisors, which receive less protection from the oral soft tissues, recorded the greatest temperature fluctuations during drinking from a cup (Barclay et al. 2005).

In our bovine *ex vivo* model, the simulation of high-temperature conditions was based on the calculated extreme temperature measured *in vivo*. An immediate increase in temperature at the abutment–implant interface (T2), which was high above the 47°C threshold level of bone tissue necrosis (Eriksson & Albrektsson 1983), was observed. At the mid-implant–bone (T4) and at the apical implant–bone (T5) interfaces, similar simulation of high-temperature conditions led to a delayed increase in temperature that reached the 42°C threshold of transient changes in osteoblasts (Li et al. 1999) (see Fig. 4).

The delayed and lower temperature values measured at T4 and T5 as compared with T2 were probably due to the distance from the input temperature and the heat transfer to the surrounding bone tissue.

In a theoretical model using computed simulation, Wong et al. (2001) reported that intra-oral exposure to 60°C may cause an increase in temperature of up to 47°C along the surface of an implant embedded in the bone. In our study, the temperatures measured were lower than the calculated one. However, both temperatures were above the temperature threshold of transient changes in osteoblasts (Li et al. 1999).

Little is known about the relationship between *in vivo* thermal exposure (temperature and time of exposure) and thermal damage, especially upon prolonged exposure and at lower temperatures, e.g., 39–42°C (Dewhirst et al. 2003). Although most foods have heat transfer coefficients lower than those of beverages (Jacobs et al. 1973), their repeated consumption could lead to cumulative intra-oral thermal damage. In our study, although less extreme temperatures were measured along the posterior teeth, they were sustained longer than in the incisors, which is in agreement with other studies (Palmer et al. 1992; Youngson & Barclay 2000). The potential damage could be worsened by other intra-oral environmental factors, such as patients with oral-burn syndrome or a low salivary flow rate, who might be more susceptible to bacterial infection (Cullen 1998).

The results of our study support the notion that intra-oral temperatures developed during the consumption of very hot substances may exceed the threshold levels for damaging peri-implant tissues. Highly conductive metal implants could conduct extreme temperatures to the osseointegration interface. Furthermore, extremely high temperatures might be a risk factor during the healing process following implant insertion. Discussing the thermal–mechanical effect of high temperature on the implant–bone interface, Wang et al. (2007) suggested that thermal stress should not be ignored in evaluating the performance of dental implants. The identification of a possible risk factor is initiated by clinical observation, backed by biological and physical accumulated knowledge and by basic research. The next step would be the development of a risk assessment model, followed by an assessment step, in which new populations are screened for the factors included in the model, and the targeting step, in which the effectiveness of prevention or intervention treatment to modify the exposure of the individual is evaluated (Beck 1994).

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