The Temperatures Reached and the Damage Caused to Hair Follicles by the Normal-Mode Ruby Laser When Used for Depilation

Adam Topping, FRCS(Ed)
David Gault, FRCS
Addie Grobbelaar, FRCS(plast)
Colin Green, PhD
Roy Sanders, FRCS
Claire Linge, PhD

Although it is proposed that heat is the cause of follicular damage leading to depilation, this has never been proved. This study aims to determine the mode by which depilation is effected and, if heat is the mechanism, what temperatures are reached within treated follicles and if sufficient damage is produced therein. Two excised specimens of hair-bearing skin from 5 patients undergoing facelifts were dissected to reveal the hair bulbs/shafts on the deep surface. They were placed on a jig, and one pulse from a normal-mode ruby laser (NMRL) of 15 J per square centimeter was fired on the epidermal surface. A thermal imaging camera recorded dermal temperature changes on the deep surface in real time. Specimens were then examined histologically for the site and extent of cellular damage by immunohistochemical staining for a protein marker of cell damage (p53). The NMRL targeted hair follicles specifically. The most common follicular temperature increase ranged from 5 to 10°C. In specimens from 1 patient the increase was more than 30°C ($p < 0.001$). Heat dissipation into interfollicular tissue in all specimens occurred 2 seconds after exposure. Evidence of laser-induced damage to follicle-lining cells was found only in those follicles with damaged hair shafts. The changes were found to a greater depth (to the bulb) and greater extent (beyond the bulge) in those follicles reaching higher temperatures. These findings suggest that the NMRL should produce permanent depilation. The variability between follicles and between patients explains, perhaps, the uneven outcome regarding depilation using the NMRL. Success appears to depend on peak follicular temperatures achieved during laser exposure, which may result from the follicular characteristics of the individual patient.

Topping A, Gault D, Grobbelaar A, Green C, Sanders R, Linge C. The temperatures reached and the damage caused to hair follicles by the normal-mode ruby laser when used for depilation. Ann Plast Surg 2000; 44:581–590

From the RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, UK.

Received Dec 21, 1999. Accepted for publication Jan 18, 2000.

Address correspondence to Mr Topping, RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, HA6 2RN UK.

The normal-mode ruby laser (NMRL) has now gained acceptance as a method for removing unwanted body hair. Clinical studies dating from 1996 have been published reporting a success rate of as much as 60% hair removal 12 weeks after treatment.[1–4] Nevertheless, the permanency of the technique remains uncertain. Only two studies to date have measured the long-term efficacy of depilation.[5,6] Dierickx and colleagues[5] reported an average of 30% hair removal after 2 years (N = 13), and Liew[6] recorded an average of 14% hair removal at 1 year (N = 88) when all body sites treated were taken into consideration. In the latter study, which alone reached significance ($p < 0.001$), only 44% of patients achieved any long-term depilatory effect, and a mere 9% of patients had more than 50% hair reduction long term. The permanent effect of the currently available techniques, considering the study reported by Liew,[6] therefore appears to be small. In an attempt to improve results, the parameters of the ruby laser such as fluence (Joules per square centimeter) and pulse duration have been adjusted, but so far have produced little immediate improvement in depilation. Some authors have reported the use of different lasers such as the Nd:YAG laser[7] and the Alexandrite laser[8] to determine whether the wavelength of light could improve efficacy. Short-term results (12 weeks) of these studies are similar to those with the ruby laser, but again no long-term follow-up of patients has been performed.

Although there has been little if any studies directly examining the interaction of the NMRL with hair-bearing skin, its potential depilatory role has been supported over the years by the

publication of papers incorporating oversimplified synthetic models[9,10] and mathematical theories[11] regarding the passage of light of differing wavelengths through skinlike constructs. From these papers it was reported that the beam from a ruby laser has a relatively high degree of skin penetrance (approaching 2 to 3 mm) with a 50% reduction in energy of approximately 1.2 mm in depth. In addition, although melanin was known to be a good chromophore over a large range of wavelengths,[12] it was stated that at the ruby wavelength of 694 nm an optical window existed in which absorbance by oxyhemoglobin was relatively little (by a factor of 10) compared with that of melanin.[13] This, in theory, would make the ruby laser selective for hair follicles because the melanin concentration is generally greater than in the epidermis. This is especially true at greater penetrative depths, where follicles are the sole structures containing melanin. These studies, however, by nature of the work, must simplify the circumstances surrounding the interaction between a laser beam and skin. What fails to become apparent are the reasons for the difference in hair response not only between patients of the same hair color and Fitzpatrick skin type but also between individual hair follicles within the same treatment site on a particular patient. No practical demonstration of the theoretical effects of ruby laser irradiation on skin has been performed to date. Discovering what happens in practice could explain how the ruby laser exerts an effect, why there are differential responses, and whether/how it may be possible to improve the permanency of laser-assisted depilation.

We report a study that incorporates "real-time" thermal imaging with detailed histological analysis in an attempt to demonstrate the interaction of ruby laser light with hair-bearing skin and to dissect any differential responses seen either between hair follicles within the same treatment site or between patients. A thermal imaging camera recorded both the peak temperatures reached and the time taken for the heat to dissipate, which allowed comparison of hair follicles within the same treatment area and between hair follicles of different patients. The same samples were then assessed histologically, using a modified safranin, indigo carmine and picric acid stain (which allows easy identification of damaged shafts), and immunohistochemically for the cell damage marker p53.[14] This latter protein is expressed maximally between 12 and 24 hours after a variety of injurious stimuli such as exposure to heat[15] and radiation.[16] The relevance of the results to clinical findings are discussed.

Methods

Hair-bearing scalp skin was obtained from five consenting white patients undergoing elective facelift procedures. Each specimen was divided into six pieces of approximately 12 mm^2 and, just before use, had the dermal aspects microdissected to reveal the hair bulbs and the lower hair shafts. Sterile isotonic saline was applied regularly to the dermis during the whole procedure to prevent drying. For each specimen, two pieces were treated with the NMRL (Chromos 694 depilation; SLS Biophile, Llanelli, Wales; pulse duration, 900 μsec; spot size, 7 mm), two acted as positive controls, and two were negative controls. Those specimens undergoing laser irradiation were mounted on a cork ring on a sterile jig so that the epidermal aspect faced the laser, whereas the thermal imaging camera system (Thermovision 900; FLIR Systems, Leighton Buzzard, England) was focused on the exposed hair bulbs of the dermal aspect. The dermal aspect was photographed to allow later comparison with the thermal images. The skin was then exposed to a single pulse of 15 J per square centimeter (typical of the clinical range) from the NMRL. Simultaneously, the thermal imaging system (frequency, 15 Hz) recorded the temperature changes from the dermal side over approximately 45 seconds—the time taken for all heat produced to dissipate.

The two skin pieces from each patient that were the positive controls for cellular damage (as demonstrated by expression of p53) were exposed to ultraviolet radiation (ultraviolet light source model HB-10103AF; 100-W bulb; Nikon Ltd, Tokyo, Japan), filtered to give wavelengths $\geq$ 300 nm on their epidermal aspect for 10 minutes. These specimens were maintained throughout on saline-dampened gauze to prevent dehydration. The negative controls were exposed to air mounted on a dry cork ring for the same duration (1 minute 30 seconds) as the laser-irradiated specimens.

After the procedures, all specimens (laser irradiated and controls) were incubated at 37°C for 18 hours in culture media (Dulbecco's Modified Eagles Medium with 10% fetal calf serum, 1% 1-glutamine, and 1% penicillin and streptomycin all supplied by Gibco Ltd) with the epidermis uppermost and at the air–liquid interface to mimic the skin's natural environment. The specimens were then processed for routine paraffin wax histology and sectioned tangentially (plane parallel to the skin surface) throughout their depths. Two consecutive 4-μm-thick sections were taken for staining every fifth section. The first of the pair was stained using the modified SACPIC technique[17] and the second by an immunohistochemical technique for the p53 protein. The modified SACPIC staining technique was promoted initially for its ability to differentiate the different growth phases of hair follicles, but has also been described elsewhere as being able to differentiate damaged from undamaged hair shafts in ruby laser-irradiated specimens.[6] The marker of cellular damage, p53, was detected using a mouse monoclonal primary antibody (Dako Ltd), a biotinylated rabbit antimouse secondary antibody (Dako Ltd), an avidin–biotin horseradish peroxidase complex (Dako Ltd), and a DAB substrate kit (Vector Laboratories). These sections were then counterstained with hematoxylin–eosin stain. Such alternate staining of two consecutive sections throughout the specimens allowed comparison of the two to establish whether a correlation existed between damaged hair shafts (demonstrated by modified SACPIC) and p53 expression within the viable cells of that same follicle. The depth to which damage to the hair shafts and p53 expression extended in each of the specimens was also estimated by knowing the thickness of each section (4 μm) and the number of sections from the epidermis to the level where shaft damage or p53 expression was no longer present.

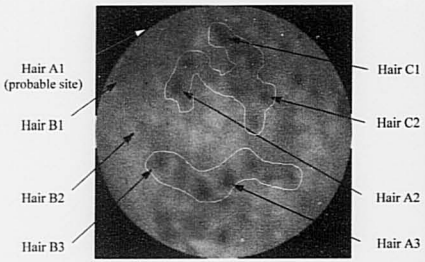
Results

Thermal Imaging

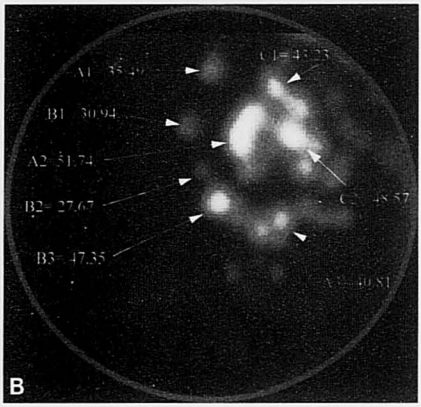
Thermal imaging of ex vivo, hair-bearing skin revealed heat production at the site of the follicles alone when the specimen was exposed to a single pulse of 15 J per square centimeter from the NMRL (Fig 1). Figure 1A is a photograph of the dermal aspect of a representative specimen of hair-bearing skin and Figure 1B is the thermal image taken of the specimen immediately on ruby laser irradiation (as defined by the line visible at the top of the image, showing that the camera was scanning at the moment of irradiation). Figure 1C is the third image taken approximately 0.75 second after laser exposure. The temperatures of the hair follicles recorded by the camera varied between 27°C and 52°C, which is a temperature increase of 7 to 32°C above the background temperature of 20°C. This heterogeneous rise in temperature seen between hair follicles within the same treatment area was observed in all samples treated. This phenomenon, from a representative skin specimen, is illustrated in the form of a graph in Figure 2B, where the temperatures recorded by the pixels along a line drawn across the thermal image (Fig 2A) are shown over time. The range of temperature increase for all hairs from all patient specimens (N = 80 measured) was between 2°C and 32°C, with the most common being between 5°C and 10°C (Fig 3). When the temperature increases were grouped according to patient (Fig 4), 4 of the 5 patients had a mean temperature increase of 9°C, whereas the remaining patient had a mean temperature increase of 18°C. Statistical analysis using one-way analysis of variance (ANOVA) revealed that differences in the median values of the follicular temperature increases between the patients were significantly different ($p < 0.001$).

To establish whether the starting temperature had any effect on the temperature increase after laser irradiation, three samples of hair-bearing skin from the same patient were warmed to different background temperatures and exposed to laser irradiation. All three samples underwent the same range of temperature increase on ruby laser exposure as the specimens from the same patient originally kept at room temperature (data not shown).

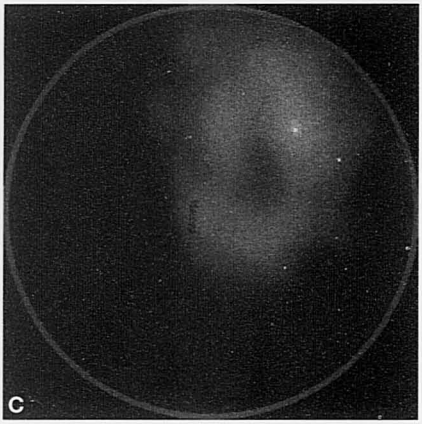
Thermal imaging revealed that the heat produced at the hair follicles reduced over time (see Figs 1B, C) so that most hairs had virtually lost the temperature gain by 2 seconds after exposure. In comparison, the temperatures recorded at the intervening skin between follicles rose over time so that the greatest temperature measured at these sites was maximal when the heat had almost



A



B



C

Fig 1. (A) Dermal aspect of a specimen from Patient 3 after microdissection revealing the hair bulbs and lower hair shafts (original magnification ×10 before 66% reduction). The outlined areas identify the groups of hairs shown in the thermal image in view B. (B) The first thermal image of the specimen in Figure 3 taken approximately 0.15 second after ruby laser exposure at 15 J per square centimeter, showing the peak temperatures (in Centigrade) obtained by the identified hairs in Figure 3 (original magnification ×8.5 before 90% reduction). (C) The third thermal image approximately 0.75 second after ruby laser exposure showing a reduction in temperature at the sites of the hair follicles but an increase in temperatures within the intervening skin (original magnification ×8.5 before 94% reduction).

completely dissipated from the follicles (see Fig 2B). This temperature was often then maintained for the remainder of the recording (approximately 45 seconds) and was noted to be higher than the original starting temperature by 1 to 5°C for all specimens examined.

The laser-irradiated hair follicles showed a rate of heat dissipation that appeared to be different for each individual follicle within the same treatment site on each patient specimen, although the significance of this could not be proved. The plots of the rate of heat loss for each follicle within each specimen were adjusted to account for the time difference between the thermal imaging camera scanning each follicle in the same treatment field. The curve representing that follicle was then moved the appropriate distance along the time axis so that its rate of heat loss could be compared in real time with others in the same image. The time taken for each follicle within each patient specimen to lose half its recorded peak temperature (T50) was calculated from the graphs. A best-fit curve representing the rate of heat dissipation of all the measured follicles from both laser specimens for each patient was then plotted (Fig 5). The median values of T50 calculated for each follicle within both laser specimens from each patient were grouped according to patient and compared statistically with one-way ANOVA. This revealed no significant difference in the rate of heat loss from the follicles between specimens from different patients after ruby laser irradiation.

Histology

Tangential sectioning of specimens enabled the whole of the specimen and its contained follicles

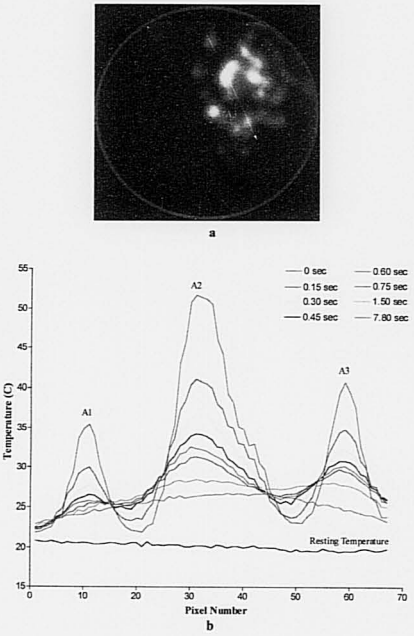


Fig 2. (A) The first thermal image as shown in Figure 1B, but with an arrow across three hairs (A1, A2, and A3), depicting the pixels along which the temperature changes were recorded over time. (B) Graphical representation of the changes in temperature over time recorded by each of the pixels along the arrow shown in view A. Hairs A1, A2, and A3 have been identified, and the increase in the pixel number follows the direction of the arrow.

to be viewed at the same time. Damage to the hair shafts was found to occur only in those specimens that had undergone laser irradiation. The percentage of hairs that were damaged was assessed for each of the patient specimens and is presented in Table 1. The greatest percentage of hair shafts that were damaged (78%) was seen to occur in specimens from Patient 3, whose hair follicles had undergone the greatest temperature increases. This was, however, closely followed by specimens from Patient 4, in whom 75% of the hair shafts exhibited damage.

Although the greatest mean and range of depth to which hair shaft damage extended appeared to occur in specimens from Patient 3 (see Table 1), this difference was not significant from the other patients. Nevertheless, 18% of hairs in specimens from Patient 3 had hair shaft damage extending to the hair bulb, whereas this occurred in only 4% of hair follicles within specimens from Patient 4 and in none of the specimens examined from the remaining patients.

Immunohistochemical staining for p53 expression was performed on all specimens. All positive controls from each patient revealed p53 expression throughout the epidermis in the majority of the keratinocytes and also in dermal cells, which was not seen in sections of the negative controls. Nevertheless, expression of p53 was also seen within cells of the deep dermis of both positive and negative controls, which is thought to have resulted from slight dehydration occurring during the experimental procedure, or as a result of trauma during dissection.

The epidermal keratinocytes of the epidermis of specimens exposed to a single pulse of 15 J per square centimeter from the ruby laser expressed p53 protein after 18 hours of incubation. Unlike the positive control, this was limited to the epidermis and the viable cells of the hair follicles that contained hair shafts showing damage. The follicular cells of those follicles that did not show damage to the hair did not express p53 protein. The percentage of damaged shaft hair follicles with cells that expressed p53 protein is shown in Table 2. This was consistently high throughout all patient specimens (85–93%), revealing a good correlation, and was greatest in specimens from Patient 3, which achieved the greatest follicular temperature increases. The relationship between the median depth of hair follicle damage and the median depth of p53 expression within the viable cells of those same follicles was analyzed using Spearman's rank correlation coefficient (SRC) for the two specimens from each patient. Those from Patient 1 had an SRC value of 0.66 ($p < 0.05$); from Patient 2, an SRC value of 0.87 ($p < 0.05$); from Patient 3, an SRC value of 0.66 ($p < 0.05$); from patient 4, an SRC value of 0.76 ($p < 0.05$); and from Patient 5, an SRC value of 0.77 ($p < 0.05$). A significant difference was seen between the patient specimens when comparing the median values of the depths to which p53 expression was noted within the viable cells

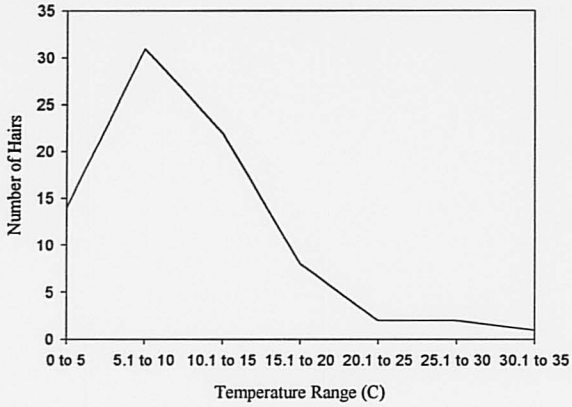


Fig 3. Graph showing the distribution of maximum temperature increases of all hairs measured from all patients (N = 80). The most common temperature increase was from 5.1 to 10°C.

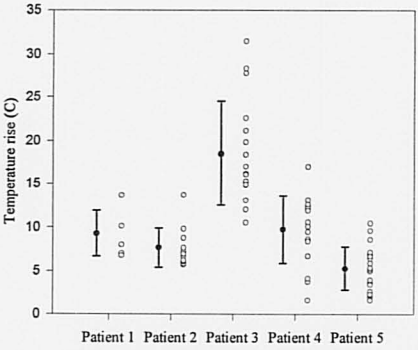


Fig 4. Graph showing the distribution of maximum temperature increases by patient of all hairs measured. The range (hollow dots), and the mean and standard deviation (line with a filled dot) are all shown.

of follicles containing damaged hair shafts on one-way ANOVA ($p < 0.05$).

The extent to which p53 expression was seen to occur radially from the damaged follicle was usually limited to the cells of the follicle alone (specifically those of the outer root sheath), with surrounding fibroblasts, endothelial cells, and glandular tissues appearing unaffected. However, the specimens from the patient whose follicles reached the greatest temperatures, Patient 3, contained follicles in which positive p53 expression was often seen within the adjoining sebaceous gland as well (Fig 6).

Discussion

This study has shown that on exposure of specimens of hair-bearing skin to a single pulse from an NMRL, heat is produced preferentially in the hair follicles, presumably from the conversion of photon energy. The ruby laser does indeed appear to target hair follicles specifically, and this is presumably because of the greater quantity of chromophores within these specific skin appendages.

Heterogeneous temperature increases were achieved in hair follicles within the same treatment site in all patient specimens measured. This suggests that a difference inherent to the hair follicles exists between them, resulting in a variance in heat production. This could be a result of interfollicular differences in hair pigmentation. There are two types of melanin: the red-yellow pheomelanin and the brown-black eumelanin. Relative phenotypic expression of both by an individual is governed by genetic inheritance. Work by Liew and colleagues[18] has shown that the darker eumelanin is the chromophore associated with successful ruby laser-assisted depilation. Therefore, the quantity of this particular chromophore may be important in determining the amount of heat produced and thus the relative success of laser treatment.

A significant difference in temperature increase was also noted between follicles from different patient specimens ($p < 0.001$). This does not

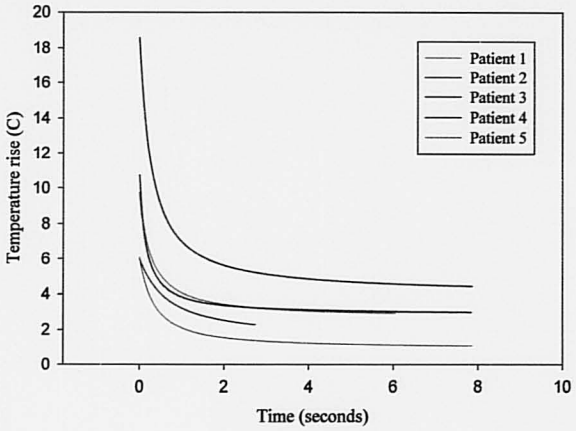


Fig 5. Graph showing the best-fit curves for the rate of heat loss from all follicles measured by patient.

Table 1. Mean Percentage of Hair Follicles From Both Patient Specimens Exhibiting Histological Damage to the Hair Shaft

Parameter	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Hairs exhibiting damaged shafts (of total examined), %	52 (N = 86)	55 (N = 56)	78 (N = 90)	75 (N = 122)	30 (N = 135)
Depth to which damage extended, mm; mean (range)	0.37 (0–0.74)	0.33 (0–0.66)	0.39[a] (0–1.64)	0.29[a] (0–1.17)	0.27 (0–0.54)
Hairs in telogen phase, %	14 (N = 12)	5 (N = 3)	7 (N = 7)	14 (N = 17)	6 (N = 9)

[a]Those patient specimens in which damage to the follicular hair shafts was noted to extend to the hair bulb.

Table 2. Percentage of Follicles Containing Histologically Damaged Hair Shafts With Follicular Cells Expressing p53 Protein

Parameter	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Damaged hairs expressing p53, %	91 (N = 38)	90 (N = 26)	91 (N = 63)	93 (N = 80)	85 (N = 36)
Depth to which p53 expression reached in all damaged hairs, mm; mean (range)	0.08 (0–0.2)	0.06 (0–0.14)	0.12 (0–0.54)	0.092 (0–0.37)	0.06 (0–0.13)

appear to correlate with the percentage of hairs in the growing phase (see Table 1). It would be more likely that the chromophore differed between the patients, even though all specimens were taken from whites with Fitzpatrick skin type 2 or 3, with similar colored dark hair. Another possibility would be that the hairs from different patients acted differently to the heat produced, with those in the specimens achieving greater temperatures having a greater combustive nature.

Currently, it is still unclear exactly which parts of the hair follicle need to be destroyed to prevent regrowth. It is thought that the stem cells reside at the hair bulge region,[19] which is approximately two thirds of the way down a hair follicle,[20] and also within the hair bulb during the anagen or active growth phase. During the involution phase, the hair bulb increases to the level of the bulge, and it is believed that stem cells with interspersed melanocytes migrate from the bulge to the site of the old bulb. This "new" bulb then descends to the original site and a new anagen phase commences. These two sites have been described as containing melanocytes and melanin pigment to a variable extent and is cycle dependent.[21] Whether the chromophore is

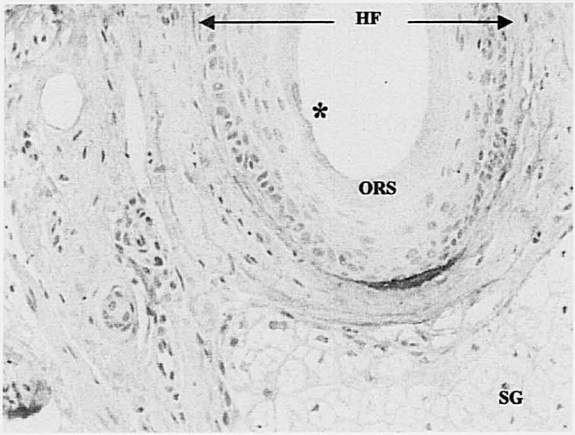


Fig 6. p53 Immunostaining of a tangential section of a specimen from Patient 3 at the level of the sebaceous gland (SG). Damage has occurred to the outer root sheath (ORS), shown by the asterisk. The cells lying adjacent to this area in both the follicle and the SG expressed p53 protein. Hematoxilyn counterstain, original magnification ×200 before 99% reduction.

present in sufficient quantities to produce enough heat at these sites cannot be answered. It is perhaps more likely that the majority of heat is produced within the highly pigmented hair shaft, and dissipation of this heat to the adjoining viable cells is the main cause of cellular damage. Heat could be seen to transfer from follicle to skin, which would lend support to this theory. Nevertheless, whether the photon energy, which eventually reaches these depths, is great enough to produce a sufficient heating effect to damage those cells is also unknown.

Heat-induced damage to the viable stem cells of the hair follicles depends on the peak temperature achieved and the time taken for it to dissipate. The rate of heat loss appeared variable between follicles within the same treatment sites, but because of the nature of the experiment, statistical testing was not possible. The rate of heat loss, when compared between patients, was found not to be different significantly. In addition, for all specimens the heat produced had almost completely dissipated 2 seconds after irradiation. Cultured human epidermal keratinocytes have been found not to survive a 1-second exposure to heating more than 58°C,[22] which might imply that this factor would have a minimal influence on successful depilation. It could also suggest that differences in thermal relaxation times may not be important in determining the extent of damage caused by laser treatment. The peak temperature, however, did vary significantly ($p < 0.001$); between patients, with one achieving markedly greater temperatures in hair follicles during irradiation. The greatest temperature increase in this experiment was 32°C, suggesting that, in vivo, the follicle would be approaching a temperature of 69°C. Cellular damage was assessed by p53 protein expression, which has been stated to undergo increased cellular expression after exposure to a thermal insult.[15,23] p53 Protein is able to halt cell cycling to allow the cell to repair itself or, if the damage is deemed to be too great, can instigate apoptosis. From 58 to 72°C, it has been stated that the cell undergoes a mixture of accidental cell death and apoptotic cell death, with the latter alone involving p53.[23]

p53 Expression was found to occur within the positive controls and the laser-exposed specimens in all patient specimens. In the positive controls, expression was noted within the epidermis whereas in the laser specimens, in addition to the epidermis, expression was also seen in cells lining the hair follicles, but only in those follicles with hair shafts that were damaged. p53 Expression was noted to extend to a greater depth in the cells of follicles from the specimens with follicles that achieved greater temperatures. A good correlation was noted between the depth of p53 expression in follicular cells lining the hair canals and the depth of hair shaft damage by

Spearman's rank correlation coefficient of median values. Damage to hair shafts was also noted to occur in laser-irradiated specimens alone, and this was noted to extend to the hair bulb to a greater extent in specimens from the patient whose follicular temperatures were greatest.

p53 Expression within cells was found to extend in a radial fashion within the hair follicle. It is likely that some of the more severely damaged cells are incapable of expressing p53, and thus the stain would probably underestimate the extent of cell death resulting from heat production. Nevertheless, the outer boundary of cells with heat damage should express p53, because the temperature should decrease in a radial fashion away from the source, so delineating the extent to which detectable heat-induced damage had reached. p53 Expression was found to occur within the sebaceous gland of specimens from Patient 3 alone, whose follicles underwent the greatest temperature increases, suggesting that damage could and had extended beyond the limits of the follicle itself. Figure 6 is a section from one of the two specimens from Patient 3, and has been cut at the level of the sebaceous gland. Damage occurred to the outer root sheath and the viable cells lying adjacent to this site both within the hair follicle and the sebaceous gland, which have stained positive for p53 protein. Sufficient heat could have passed from the hair shaft outward, resulting in damage being inflicted on these cells. It could also imply that damage could have occurred to the bulge region, which resides just below the sebaceous gland but in the outer root sheath, to produce possible permanent depilation.

The thermal imaging camera showed that the skin between hair follicles in all specimens used did not heat directly on ruby laser irradiation, but did so afterward as a result of dissipation of heat from the hair follicles. Apart from the hair follicles, melanin within skin is confined to the epidermis. However, any heat produced at this site would not be seen by the thermal imaging camera, which was focused on the dermis. This may be a result of the heat production by such small concentrations of melanin being undetectable or the thermal imaging camera cannot detect it until it has diffused through to the deep dermis. It is possible that the prolonged temperature increase seen in skin after laser exposure (up to 5°C for approximately 45 seconds) could cause the side effect of damage to nonfollicular skin structures. However, it must be remembered that these experiments were performed on skin biopsies, and therefore in the absence of the effects of uninterrupted circulation. The circulation of blood undoubtedly aids the dissipation of heat from the skin considerably.[24] However, it would seem unlikely that blood flow would affect the peak temperatures recorded at the follicles because the flow rate, when compared with the pulse duration of the laser, is much slower. It is more likely that any side effects of skin damage associated with laser depilation are mainly a direct result of the presence of melanin within the epidermis and the consequent localized production of heat. This is in agreement with the common observation that side effects are more prevalent in patients with darker skin and ultrastructural findings of damage to the melanosomes within the keratinocytes of the epidermis.[6]

In summary, this experiment reflects the current clinical scenario. The ruby laser does appear to interact specifically at the site of the hair follicle, where heat is produced to an extent in some patients that could result in permanent damage being inflicted on the viable hair-producing cells of the hair follicles, preventing hair regrowth. p53 Expression was noted to occur radially beyond the extent of hair follicles in specimens from 1 patient, which suggests by implication that damage to the bulge region could have occurred as the heat traversed this region to reach the sebaceous gland.

A significant ($p < 0.001$) difference was noted between the median follicular temperature increases between patient specimens. This suggests that something intrinsic to the hairs of this patient affects the maximum temperature achieved by laser treatment. A higher temperature could be reached by either increased photon energy conversion and/or combustibility of the hair shaft, which accounts for the temperatures achieved by a particular follicle and the subsequent damage incurred. Additional work is required to assess whether a change in the laser parameters (not possible with this laser), particularly the pulse duration, would achieve greater temperature increases or greater histological damage to the sites,

where it is required to do so to achieve a greater depilatory success rate.

References

1 Grossman MC, Dierickx C, Farinelli W, et al. Damage to hair follicles by normal-mode ruby laser pulses. J Am Acad Dermatol 1996;35(6):889–894
2 Lask G, Elman M, Slatkine M, et al. Laser-assisted hair removal by selective photothermolysis. Preliminary results. Dermatol Surg 1997;23(9):737–739
3 Solomon MP. Hair removal using the long-pulsed ruby laser. Ann Plast Surg 1998;41(1):1–6
4 Bjerring P, Zachariae H, Lybecker H, Clement M. Evaluation of the free-running ruby laser for hair removal. A retrospective study. Acta Derm Venereol (Stockh) 1998; 78(1):48–51
5 Dierickx CC, Grossman MC, Farinelli WA, Anderson RR. Permanent hair removal by normal-mode ruby laser. Arch Dermatol 1998;134(7):837–842 [See comments]
6 Liew S. Ruby laser-assisted hair removal: clinical efficacy and evaluation of cutaneous changes [doctoral thesis]. London: University College; 2000.
7 Goldberg D, Littler C, Wheeland R. Topical suspension-assisted Q-switched Nd:YAG laser hair removal. Dermatol Surg 1997;23:741–745
8 Finkel B, Eliezri Y, Waldman A, Slatkine M. Pulsed Alexandrite laser technology for noninvasive hair removal. J Clin Laser Med Surg 1997;15(5):225–229
9 Flock S, Patterson M, Wilson B, Wyman D. Monte Carlo modeling of light propagation in highly scattering tissues—1: model predictions and comparison with diffusion theory. IEEE Trans Biomed Eng 1989;36(12):1162–1168
10 Flock S, Wilson B, Patterson M. Monte Carlo modeling of light propagation in highly scattering tissues—2: comparison with measurements in phantoms. IEEE Trans Biomed Eng 1989;36(12):1169–1173
11 Gemert MV, Jacques S, Sterenborg H, Star W. Skin optics. IEEE Trans Biomed Eng 1989;36(12):1146–1154
12 Goldman L. Optical radiation hazards to the skin. In: Sliney D, Wolbarsht C, eds. Safety with lasers and other optical sources: a comprehensive handbook. New York: Plenum Press, 1983:167–169
13 Anderson R. Laser tissue interactions. In: Goldman MP, Fitzpatrick RE, eds. Cutaneous laser surgery—the art and science of selective photothermolysis. St. Louis, MO: Mosby Year Book, 1984:1–18
14 Lane D. p53, Guardian of the genome. Nature 1992;358: 15–16
15 Nakazawa K, Sahuc F, Damour O, et al. Regulatory effects of heat on normal human melanocyte growth and melanogenesis: comparative study with UVB. J Invest Dermatol 1998;110:972–977
16 Brash D, Ziegler A, Jonason A, et al. Sunlight and sunburn in human skin cancer: p53, apoptosis and tumor promotion. J Invest Dermatol 1996;1:136–142
17 Nixon A. A method of determining the activity state of hair follicles. Biotech Histochem 1993;68(6):316–325
18 Liew S, Ladhani K, Grobbelaar A, et al. Ruby laser-assisted hair removal: success in relation to anatomical factors and melanin content of hair follicles. Plast Reconstr Surg 2000 [In press]
19 Cotsarelis G, Sun T, Lavker R. Label-retaining cells reside in the bulge area of pilosebaceous unit: implications for follicular stem cells, hair cycle and skin carcinogenesis. Cell 1990;61:1329–1337
20 Rochat A, Kobayashi K, Barrandon Y. Location of stem cells of human hair follicles by clonal analysis. Cell 1994;76(6):1063–1073
21 Narisawa Y, Kohda H, Tanaka T. Three-dimensional demonstration of melanocyte distribution of human hair follicles: special reference to the bulge area. Acta Derm Venereol (Stockh) 1997;77:97–101
22 Bowman P, Schuschereba S, Lawlor D, et al. Survival of human epidermal keratinocytes after short-duration high temperature: synthesis of HSP 70 and IL-8. Am J Physiol 1997;272:C1988–C1994
23 Matylevitch N, Schuschereba S, Mata J, et al. Apoptosis and accidental cell death in cultured human keratinocytes after thermal injury. Am J Pathol 1998;153(2):567–577
24 Welch A, Wissler E, Priebe L. Significance of blood flow in calculations of temperature in laser irradiated tissue. IEEE Trans Biomed Eng 1980;BME-27:164–166