Wavelengths for Laser Treatment of Port Wine Stains and Telangiectasia

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Background and Objective: This report presents analytical modelling of the influence of wavelength on the amount of volumetric rate of heat produced in dermal blood vessels by millisecond laser radiation.

Study design/Materials and Methods: A new anatomical model is proposed that represents port wine stains as well as telangiectatic lesions. It consists of a target blood vessel, representing the deepest dermal blood vessel that requires irreversible injury, and a layer of whole blood, representing all other dermal blood vessels above the target vessel. The laser light that interacts with the blood vessels is assumed to be diffuse. Selective photothermolysis is the basis for the analysis. We consider wavelengths between 577 nm and 600 nm, the argon laser wavelengths at 488/515 nm, and the frequency doubled Nd:YAG laser wavelength at 532 nm.

Results: The rate of volumetric heat production of absorbed laser light in the target blood vessel is expressed analytically as a function of blood absorption, the concentration of additional dermal blood, and the depth of the target vessel.

Conclusion: The model explains why 585 nm is a good compromise for treating port wine stains that vary widely in number of dermal blood vessels. It predicts that wavelengths between 577 nm and 582 nm are excellent for the treatment of port wine stains in young children, and it suggests a possible explanation as to why the argon laser is sometimes said to be capable of treating dark mature port wine stains. The copper vapour laser wavelength at 578 nm, and the frequency doubled Nd:YAG laser wavelength at 532 nm, are predicted to be suitable for the treatment of port wine stains that contain, respectively, a small to moderate and a moderate number of dermal blood vessels. When laser beam spotsize becomes smaller, the best wavelength for producing maximal rate of heat in the target vessel is predicted to shift to 577 nm.

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Many laser systems have been judged suitable, at different times and by different groups, for removing the abnormally ectatic blood vessels in port wine stains (PWS). These include the ruby, argon, argon pumped dye, flashlamp pulsed dye, copper vapour, Nd:YAG, frequency doubled Nd:YAG and CO₂ lasers. Each of these systems has its own unique properties of wavelength, exposure time, and power [1,2]. It is not surprising that such a wide range of wavelengths (488-10,600 nm), exposure times (40 ns to continuous wave), powers (1 Watt to several kWatt), and spot sizes (0.1-7 mm) have resulted in confusion as to what the “ideal” combination of laser parameters might be that would selectively and effectively destroy the abnormal blood vessels in these birthmarks without adversely altering adjacent non-vascular tissues.

One set of laser parameters consisting of a combination of wavelength (585 nm), pulse duration (0.45 ms), fluence (5-8 J/cm²), and spotsize (3-5 mm) has been shown to produce selective vascular injury using the flashlamp pulsed dye laser [3]. The question might then be: Are all of the laser parameters used in this combination equally important to achieve this vascular selectivity or are certain of these parameters more important than others? Because of the complexity of PWS pathology [4] and, until recently, the paucity of histologic data documenting the sequence of pathological events occurring following exposure of the skin to laser irradiation, it has been difficult to assess the response of PWS blood vessels to the different laser systems and to compare the effectiveness of these systems.

The model is simple, analytically solvable, and depends only on the coefficient of blood absorption, the dermal depth of the deepest vessel that requires injury (the target vessel), and the concentration of dermal blood above the target vessel. It provides guidelines for the choice of laser parameters that might suitably be used for conducting comparative clinical studies on PWS and telangiectasia.

The Model

Consider Figure 1A where one (target) blood vessel of a certain diameter located parallel to the air-tissue interface is embedded in the dermis at a certain depth. This model has been widely used to represent the vasculature of a PWS [8-10] and it is obviously an excellent representation of a telangiectatic lesion. The photons, a-d, represent four out of a huge number of photons within the laser beam spotsize that will reach the target vessel.

A more realistic model of a PWS vasculature is shown in Figure 1B. Other blood vessels are added in addition to the target vessel. Again, the blood vessels are chosen parallel to the air-tissue interface. The same photons, a-d, as in Figure 1A are shown, but these photons now cross blood vessels numbered 1 and 3, respectively. As absorption of photons by a chromophore has a certain probability, represented by the absorption coefficient of the chromophore (blood), it is assumed here that photon a is absorbed by blood vessel 1 but that photon d is not absorbed by blood vessel 3 and that its direction of propagation is unchanged. The point of Figure 1B is to suggest that all blood vessels in the irradiated volume of the dermis have a certain probability of absorbing scattered photons that cross the blood vessels and that would otherwise reach the target vessel. It is obvious that the larger the absorption coefficient of blood, the more these scattered photons are ab-
sorbed, and the fewer of them will reach the target vessel.

The target vessel as well as the other vessels are chosen sufficiently deep inside the dermis that only scattered photons can reach them. We emphasize that for the wavelengths of interest, between 488 nm and 600 nm, scattering dominates over absorption in skin tissue [11]. Hence the average pathlength that a photon can propagate in the (bloodless) dermis between two scattering events (~0.1 mm) is much shorter than the average pathlength between two absorption events (~10 mm). As a consequence, the laser light that reaches the blood vessels is diffuse (photons have scattered several times).

We hypothesize that the dermal blood vessels above the target vessel can be represented as a layer of blood located in the dermis between the air–skin interface and the target vessel (Fig. 1C). The dermal depth of this layer is arbitrary as it is assumed to be deep enough to have only interaction with diffuse light. However, it is highly likely that our model of Figure 1C is not an exact representation of that of Figure 1B.

The thickness of the layer D in Figure 1C is directly proportional to the amount of dermal blood above the target vessel. That is, it is directly proportional to the volumetric concentration of blood $C_b$ (volume of blood per volume of dermis) and to the dermal depth over which these additional vessels occur, which is assumed here to be the depth of the target vessel $z_v$. Hence,

$$D = C_b z_v \quad (1)$$

Assuming values of $z_v$ vary between 0.5 mm for young children and 1 mm for adults [12], and values of $C_b$ between 0 and 10% [13], D varies between 0 and 0.1 mm.

We emphasize that lesion colour correlates with the total amount of dermal blood present [13,14] and, hence, with D (Fig. 1C). Unfortunately, a quantitative correlation between colour and D is unknown, although we assume that small values of D will represent pink lesions.

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Fig. 1. A. Model of a vascular skin lesion that consists of one ectatic target blood vessel located in the dermis at dermal depth $z_v$ and directed parallel to the air–epidermis interface. Four photons are shown, labelled a, b, c, and d, out of a huge number of photons incident on the epidermis that reach the target vessel. B. A more realistic model of a PWS, consisting of the target vessel (as in A, but representing here the deepest ectatic dermal blood vessel that requires irreversible injury) and, e.g., three additional ectatic blood vessels, numbered 1, 2, and 3, which are located elsewhere in the dermis and which are all directed parallel to the target vessel. For simplicity, the additional vessels are assumed to have the same diameter as the target vessel. Photon a is assumed to cross blood vessel 1 and to be absorbed by the blood in that vessel. Photon d is assumed to cross blood vessel 3 without any interaction, reaching the target vessel. C. The final model used here, consisting of a layer of whole blood representing all dermal blood vessels anterior to the target vessel, and the target vessel located below the layer of blood at dermal depth $z_v$. The thickness D of the blood layer is such that it contains the same amount of red blood cells per volume of skin as the anterior vessels. This requires Eq. (1) to be valid.
path length $L$ in blood layer $D$, where $L(D)$ depends on $D$. It is assumed to follow Beer’s law as

$$A_b(\lambda,D) = \exp[-\mu_{a,b}(\lambda) L(D)] \quad (3a)$$

When the light is collimated and normal to the layer $D$, then $L = D$, and $A_b(\lambda,D) = \exp[-\mu_{a,b}(\lambda) D]$. However, if the light is diffuse, the average path-length is equal to $L = 2D$ [15]. Even more complicated is the resulting angular distribution of diffuse light that propagated through an absorbing layer of thickness $D$. The average pathlength that results under such circumstances represents an interesting question that we do not consider here as it does not affect the argument of this report. Here, we assume that the average path length for diffuse light, $L$, is twice the geometrical path-length $D$ [15], so $L = 2D$, and hence

$$A_b(\lambda,D) = \exp[-\mu_{a,b}(\lambda) 2D] \quad (3b)$$

The rate of volumetric heat production (Watt/cm$^3$) at the top of the target vessel lumen at dermal depth $z_\nu$, $Q(\lambda,z_\nu,D)$, is well known to be the product of fluence rate and blood absorption coefficient

$$Q(\lambda,z_\nu,D) = \phi(\lambda,z_\nu,D) \mu_{a,b}(\lambda) \quad (4)$$

For the short pulse durations considered, of $\sim 1$ ms, the temperature rise achieved at the end of the laser pulse is in good approximation proportional to $Q$. Using Eqs. (2) and (3b), Eq. (4) becomes

$$Q(\lambda,z_\nu,D) = E A_{e,d} \exp[-\mu_{a,b}(\lambda) 2D] \mu_{a,b}(\lambda) \quad (5a)$$

In this report, we do not specify $A_{e,d}$ any further as we want only to study the wavelength dependence of the rate of heat production in the target vessel at a fixed, although unspecified value of $z_\nu$. We therefore divide $Q$ in Eq. (5) by $Q(\lambda = 577\text{nm},z_\nu,D = 0)$, which represents the maximum possible rate of heat production in the target vessel.

$$Q(\lambda,z_\nu,D)/Q(577\text{nm},z_\nu,D = 0) = \exp[-\mu_{a,b}(\lambda) 2D]\mu_{a,b}(\lambda)/35.4 \quad (5b)$$

Equation (5b) is the final result of our model. It represents the rate of volumetric heat produced in blood at the top of the target vessel lumen and relates this with laser wavelength, dermal location of the target vessel, and amount of dermal...
blood. Blood absorption coefficient $\mu_{a,b}(\lambda)$ is the factor that varies with wavelength $\lambda$, Figure 2, and $D$ is the factor that varies with $z_\nu$ and $C_b$, Eq. (1). The model does not explicitly account for factors such as epidermal pigmentation or laser beam spotsize. This would require computation of $A_{e,d}$ in Eqs. (2) and (5a) by using photon propagation models (e.g., Monte Carlo numerical methods).

RESULTS

A plot of $Q(\lambda,z_\nu,D)$, divided by $Q(\lambda = 577\text{ nm},z_\nu, D = 0)$, Eq. (5b), is given in Figure 3 as a function of wavelength $\lambda$, and for various values of blood layer thickness $D$. The values of $Q$ at $D = 0$ represent the rate of volumetric heat production at the top of a telangiectatic blood vessel lumen at dermal depth $z_\nu$, which are proportional to the blood absorption coefficient. Note that the maximum value of $Q(\lambda,z_\nu,D = 0)$ is proportional to the maximum value of the blood absorption coefficient $\mu_{a,b}(\lambda)$, which is at $\lambda = 577$ nm. This implies that a single isolated dermal ectatic vessel at (any) dermal depth $z_\nu$ can best be injured by a laser pulse at 577 nm, in accordance with the concept of selective photothermolysis [10] that we adopted here. Figure 3 shows that addition of a small amount of dermal blood (values of $D$ up to 0.0141 mm, obtained from $\mu_{a,b}(577\text{ nm})2D = 1$, see Appendix) lowers the amount of heat that can be produced in the target vessel, but retains 577 nm as the best wavelength for irreversible injury (see Appendix). Further addition of dermal blood vessels is predicted not only to lower the amount of heat that can be deposited in the target vessel, but also to shift the “best” wavelength for heat production to larger values than 577 nm. Note, e.g., that for $D = 0.04$ mm, there is a 2.2 times greater rate of heat production at 586.9 nm (wavelength of maximum heat production) than at 577 nm.

The “optimal” wavelength, $\lambda_0$, where the volumetric rate of heat production in the target vessel is maximal, see Figure 3 and Eq. (7) of Appendix, and those wavelengths $>577$ nm where 80% and 90% of the maximum heat is produced, are plotted in Figure 4 as a function of blood layer thickness $D$. This figure shows that the “best” wavelength is 577 nm when $D \leq 0.0141$ mm; it changes abruptly to wavelengths longer than 577 nm when $D > 0.0141$ mm. Figure 4 predicts that when 80% of the maximum rate of heat production is considered adequate, wavelengths longer than 582 nm can handle more and more dermal blood (larger $D$ values). Figure 4 also predicts that wavelengths between 577 nm and 582 nm produce virtually the same rate of heat in the target vessel for values of $D < 0.03$ mm. This prediction correlates surprisingly well with results by Tan et al. [16] who showed in albino pigs virtually identical dermal vascular injury for $\lambda = 572$ nm (with identical blood absorption as 581 nm, Fig. 2), 577 nm and 580 nm, for incident energy densities between 5 and 10 $\text{J/cm}^2$.

In Figure 5, the maximum rate of volumetric heat production is shown at optimal wavelength $\lambda_0$ (see Appendix), and at a fixed albeit unspecified depth $z_\nu$, relative to the maximum possible rate of heat production at $z_\nu$ (which is at 577 nm and for $D = 0$ mm) as a function of $D$. From Eq. (5b) this is given by

$$Q(\lambda_0,z_\nu,D)/Q(577\text{ nm},z_\nu,D = 0) = \exp[-\mu_{a,b}(\lambda_0)2D] \mu_{a,b}(\lambda_0)/35.4$$ (6)
Fig. 4. Wavelength of maximum heat production, \( \lambda_0 \), and wavelengths at which 80% and 90% of the maximum heat is produced, as a function of D. (For details, see Appendix.) The wavelengths longer than 577 nm that have equal blood absorption as the argon and frequency doubled Nd:YAG laser wavelengths are also indicated (the dashed lines at 586.6/587.4 nm and 583 nm, respectively). We assume that values of D that are very small correspond to pink PWS and values of D that are large correspond to purple PWS.

Figure 5 shows that a substantial reduction in rate of heat production occurs at the target vessel when the dermal blood concentration increases, even for irradiation with optimal wavelength \( \lambda_0 \). In the Appendix it is derived that Eq. (6) shows a rapidly decreasing behaviour as \( \exp(-35.4 \cdot 2 \cdot D) = \exp(-70.8 \cdot D) \) for D between 0 and 0.0141 mm, and a much slower decrease as \( \mu_{\text{a,b}}(\lambda)/35.4 \cdot e \) for D > 0.0141 mm. As an example, for D = 0.035 mm, only 15% of the maximum possible heat is produced in the target vessel at dermal depth \( z_r \) compared to a single telangiectatic vessel (D = 0 mm) at the same depth.

**DISCUSSION**

The Model

The model is based upon three assumptions. First, selective photothermolysis [10] is the basis for the analysis. This implies (1) 577 nm is the best wavelength for injury of an (isolated) blood vessel, irrespective of the size of the vessel, and (2) ms pulse durations, implying that influences of cooling the target by heat conduction, and by blood flow of the dermal vessels [17] can be neglected. Second, the light surrounding all blood vessels is diffuse. The influence of the additional blood vessels, in terms of attenuating the diffuse photons that are bound to reach the target vessel, is represented by one single layer of whole blood. The thickness D of that layer is directly related to the product of the dermal concentration of additional blood (vessels) and to the dermal depth over which these vessels occur (which is here assumed to be the depth \( z_r \) of the target vessel), Eq. (1). Attenuation of diffuse light by the layer of blood is assumed to follow Beer’s law. The use of 2D as the effective thickness of the layer is an approximation, most likely valid only for small values of D, that is for \( \mu_{\text{a,b}}(\lambda)2D << 1 \). For large values of D, nonzero transmission of the diffuse light through the layer occurs mainly at (near) perpendicular incidence. Under such conditions of large D values, the factor \( \mu_{\text{a,b}}(\lambda)L \) in Eq. (3a) should most likely become close to \( \mu_{\text{a,b}}(\lambda)D \). In this work, we use the factor of 2 in Eq. (3b) for convenience, but it must be kept in mind that this factor is expected to be between 2 and 1, depending upon whether D is small or large. Third, it is tacitly assumed that once the target vessel is injured all other anterior vessels are injured, too. It is also assumed that the absorption coefficient of blood remains constant during the laser pulse. The use of one layer of blood to represent volumetrically distributed dermal vessels may not be exact, but, unfortunately, its verification requires an optical model that contains a distribution of blood vessels comparable to “real” PWS skin. Nevertheless, the advantage of the present model is its simplicity, as expressed by Eq. (5b). Obviously, such an approach can only be useful when relative behaviour is studied, such as shown in Figures 3–5. Another advantage of the present model is that it has been made independent of the actual absorption and scattering behaviour of skin tissues by normalizing the rate of heat production to the maximum possible rate of heat production in the target vessel (at 577 nm, and D = 0), Eq. (5b). It
Laser Wavelengths for Port Wine Stains

only depends on the absorption coefficient of whole blood. Fortunately, this latter parameter is well known, whereas the optical parameters for epidermis and dermis are not yet available with great accuracy.

The present anatomical model combines two previously used PWS models: the layer of blood [18,19], and the target vessel [9,10]. The layer of blood accounts here for the attenuation of diffuse light by all blood vessels that are present in the dermis (in addition to the target vessel) instead of representing the vascular target itself [18,19]. In this way our model accounts for the competition that occurs between absorption of photons by the additional dermal blood vessels and absorption of photons for producing heat in the target vessel: a large (small) blood absorption coefficient produces correspondingly a large (small) attenuation coefficient. At 577 nm the model predicts that for \( D = 0.0141 \) mm of additional blood content (e.g., \( z_v = 0.5 \) mm and 2.82\% blood concentration, or \( z_v = 1 \) mm and 1.41\% blood concentration) attenuation by the other blood vessels becomes the limiting factor in the production of heat by preventing too many photons from reaching the target and producing heat. A reduction in blood absorption, by shifting the wavelength to larger values than 577 nm, results in so much more light fluence rate at the top of the target vessel lumen that the rate of heat production increases again despite the reduction in (target) blood absorption.

Wavelengths

From Figures 3 and 4, it can be assessed that 585 nm produces at least 80\% of the maximum possible volumetric rate of heat in the target vessel for values of \( D \) between 0.012 mm and 0.049 mm, with a maximum at \( D = 0.026 \) mm. At \( D = 0 \) mm, 585 nm still produces 54\% of the maximal possible rate of heat (maximum at \( \lambda_0 = 577 \) nm), whereas at \( D = 0.07 \) mm, this is 50\% (maximum at \( \lambda_0 = 589.8 \) nm). Our model clearly predicts 585 nm to be a good compromise in producing optimal volumetric rate of heat in the target vessel for a wide range of dermal blood layer thicknesses and hence for a wide range of PWS anatomies and colours. However, our model also predicts that 585 nm is not the single best wavelength for treating PWS, but that other wavelengths can perform equally well, or even better, depending upon the amount of dermal blood present [7,8]. An interesting speculation is based on the observation in Figure 4 that wavelengths between 577 nm and 582 nm are excellent to treat PWS with an amount of dermal blood equivalent to about \( D < 0.03 \) mm (at least 80\% of the maximum rate of heat production). For children having a dermis of \( \sim 0.5-0.7 \) mm thickness [12], \( D < 0.03 \) mm corresponds to dermal blood concentrations \( C_b < 6\% \). The present analysis, therefore, predicts 577–582 nm to be excellent laser wavelengths for the treatment of PWS in very young children. Although this speculation must still be tested clinically, it is supported by an earlier publication of Tan et al. [20], who described excellent clinical results of PWS in children using 577 nm.

A quantitative comparison between the argon, frequency doubled Nd:YAG, and copper vapour laser wavelengths and the flashlamp pulsed dye laser is difficult to make as the former lasers can produce only a fraction of the power of the flashlamp pulsed dye laser. Consequently, these lasers operate with longer irradiation times and/or smaller beam spot sizes and are theoretically less optimal for PWS treatment [2,8] because they easily cause nonspecific dermal injury. In addition, blue-green and green wavelengths are expected to have smaller skin penetration depths than yellow wavelengths due to larger epidermal and dermal absorption and scattering (smaller \( A_{\text{eq}} \) values). Furthermore, copper vapour and the new frequency doubled Nd:YAG lasers have short pulses, high peak powers per pulse, but such a high repetition rate that these lasers are considered to operate as “quasi continuous wave.” In this report, a qualitative comparison is made by considering wavelengths between 577 nm and 600 nm that have equal blood absorption coefficients as the argon, frequency doubled Nd:YAG, and copper vapour laser wavelengths (Fig. 2), but that have otherwise identical pulse durations and beam spot sizes as the flashlamp pulsed dye laser (typically \( \sim 1 \) ms and 3 to 5 mm, respectively).

Consider, first, the argon blue-green laser lines at 488/515 nm. The wavelengths of equal blood absorption are 586.6/587.4 nm and our model predicts that they produce maximum volumetric heating of the target vessel when \( D = 0.038/0.045 \) mm. The range of \( D \) values where at least 80\% of the maximal possible rate of heat is produced is between 0.017 mm and 0.081 mm. Assuming for adults the deepest target vessel is at \( z_v = 1 \) mm, such values of \( D \) represent a dermal concentration of blood vessels that correspond to the darker types of PWS [13,14]. Although blue-green light from an argon laser has a well-documented limited depth of vascular injury [21], usually non-
specific, it is a noteworthy coincidence that the argon laser is claimed to do well in dark, mature PWS [2,22]. Second, the 532 nm green wavelength of a frequency doubled Nd:YAG laser has a blood absorption that is equal to that at 583 nm (Fig. 2). This latter wavelength is predicted to produce maximum heating for \( D = 0.019 \) mm, and at least 80% of the maximum for \( D \) between 0.005 and 0.035 mm corresponding, respectively, to light to darker PWS (for a baby, \( z_r = 0.5 \) mm and \( C_b \) is between 1% and 7%; for an adult, \( z_r = 1 \) mm and \( C_b \) is between 0.5% and 3.5%). Third, our model predicts that the 578 nm copper vapour laser wavelength is optimal (producing at least 80% of the maximum possible rate of heat at the target vessel) for \( D \) between 0 and 0.026 mm, with a maximum for \( D = 0.014 \) mm which corresponds to a lighter PWS.

Laser Beam Diameter

In Eq. (1), it is tacitly assumed that laser spotsize is much larger than the average dermal distance between blood vessels (e.g., a 5 mm spotsize vs. 0.1 mm to 0.4 mm distance between vessels). When the spotsize becomes so small that only a few, or even single, vessels are being irradiated (for spotizes smaller than 0.5 mm, say), \( C_b \) in Eq. (1) becomes a function of spotsize, usually being smaller for smaller spotsizes, and will become zero when only the target vessel is irradiated. Clinically, therefore our model predicts that an isolated (target) blood vessel can be optimally injured by the laser wavelength of maximum absorption in the red blood cells (at 577 nm), irrespective of the size of the vessel. Some modelling suggests vessel size influences the optimum wavelength for injury of that vessel because of the quantity of blood within the vessel [24]. However, no conclusive clinical or histological information is currently available that shows a relationship between optimal wavelength for irreversible injury and vessel size.

In conclusion, the present model is simple, analytically tractable, and, hence, as transparent as possible to understand better the interactions between laser light and benign vascular skin lesions. In particular, the choice of laser wavelength for producing heat in dermal blood vessels is discussed in some detail and is related to the amount of blood present in the dermis and, hence, to the colour of the lesion. This sort of information may be of particular value when, in the near future, comparative trials may be designed in which the influence of different wavelengths on the clinical outcome will be studied. Our analysis also points out the need of characterizing the vascularization of a PWS prior to treatment. Although possible methods for such a characterization are currently not established, several of them have been summarized by Pickering et al. [25]. An interesting and promising new approach, using fast pulsed photothermal thermography imaging [26], was recently presented at the 14th Annual Meeting of the American Society for Laser Medicine and Surgery (Toronto, Ontario, Canada, April 8–10, 1994).

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Laser Wavelengths for Port Wine Stains


APPENDIX

The wavelength of maximum rate of heat production, \( \lambda_0 \), follows from the first derivative of \( Q(\lambda, z_0, D) \), Eq. (5a), with respect to \( \lambda \) and solving \( \lambda \) from equating this derivative to zero. Unfortunately, an analytic relation between \( Q \) and \( \lambda \) is not available. Therefore, we first solve for the optimum blood absorption coefficient \( \mu_{a,b}(\lambda_0) \), by requiring the first derivative of \( Q \) of Eq. (5a) with respect to \( \mu_{a,b}(\lambda) \) to be zero, yielding

\[
\mu_{a,b}(\lambda_0) = 1/(2D) \tag{7}
\]

and using Figure 2 to convert \( \mu_{a,b}(\lambda_0) \) to the corresponding \( \lambda_0 \). As \( \mu_{a,b}(\lambda) \) is at most 35.4 mm\(^{-1} \) (Fig. 2), values of \( D \) that are smaller than \( 1/(2 \times 35.4) = 0.0141 \) mm refer to \( \lambda_0 = 577 \) nm.

The relative maximum volumetric rate of heat production has been expressed as a function of blood absorption coefficient and dermal blood layer thickness in Eq. (6). For \( D \leq 0.0141 \) mm, i.e., when \( \lambda_0 = 577 \) nm and \( \mu_{a,b}(\lambda_0) = 35.4 \) mm\(^{-1} \), the relative maximum rate of heat production equals \( \exp(-35.4 \times 2 \times D) = \exp(-70.8 \) D). For \( D > 0.0141 \) mm, i.e., when \( \mu_{a,b}(\lambda_0) 2 D = 1, \) Eq. (7), the relative maximum rate of heat production equals \( \mu_{a,b}(\lambda_0) = 35.4 \) e = \( \mu_{a,b}(\lambda_0)/96.2 = 1/(96.2 \times 2 \times D) = 1/(192.4 \) D). The two descriptions \( \exp(-70.8 \) D \) and \( 1/(192.4 \) D \) are smoothly continuous at \( D = 0.0141 \) mm. That is, the functions, as well as the derivatives with respect to \( D \), are equal at \( D = 0.0141 \) mm.