Skin Thermal Injury Prediction with Strain Energy *

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Abstract

A three-dimensional model is presented for the quantitative prediction of skin injury resulting from certain thermal exposure on the surface. The model is based on the skin damage equation proposed by Henriques and Moritz for the process of protein denaturation. Different from the standard Arrhenius model for protein damage rate, in which the activation energy includes chemical reaction only, strain energy of tissue due to thermal stress is also considered in the current model. Skin thermal response is modeled using the bioheat transfer equation by including water diffusion on the skin surface, and the corresponding thermal stress is predicted using the modified Duhamel-Neumann equation. Strain energy is then obtained by the stress-strain relation. The extent of burn injury is computed from the transient temperature solution and the effect of strain energy on skin damage is investigated. The time-dependent partial differential equations (PDEs) are discretized using Crank-Nicholson finite difference scheme and the resulting sparse linear systems are solved iteratively.

Key words: skin thermal injury, burn evaluation, heat and mass transfer, finite difference, iterative method.

1 Introduction

Skin performs a vital role in regulating the temperature of human body by serving as the medium of heat transfer between the body and the environment. To be specific, skin is composed of three layers: epidermis, dermis, and subcutaneous. Epidermis is the outermost layer consisting of dead

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cells on the outermost level and deratинocytes on the deepest level. Right beneath epidermis is the dermis layer, which consists of blood vessels, elastic fibers, connective tissues, sweat glands, and nerves [14]. The next layer is subcutaneous that contains larger blood vessels [12]. Blood flow is essential in providing nutrients to dermis and subcutaneous and maintaining energy exchange between skin and the inner parts. However, there are no blood vessels in epidermis layer, and the corresponding heat and mass transport processes are conducted by diffusion between dermis and epidermis.

Under normal conditions, thermal balance between human body and the environment can be maintained and skin is at a comfortable temperature suitable for its metabolic function [14]. Physiological abnormality or changes of environmental conditions could influence the temperature distribution in skin and may even disturb the existing thermal balance in human body. Skin burn is one of such cases in which human body is exposed to high temperature or high heat flux environmental condition for a certain period of time, hence local skin temperature is raised high enough to destroy the organic tissue and to cause injury.

Burn injury is one of the most common accidental injuries in human daily activities. According to some statistics, hundreds of thousands of people suffer burn injuries in the United States every year [18]. In 1999, the U.S. occupational injuries of firefighters alone were as many as 88,500 [15]. The severity of burn injury varies from minor damage in skin superficial layer to severe damage in deeper layers to even fatality. Skin burn injury may be classified based on skin physical structure as first degree with partial damage of epidermis, second degree with total damage of epidermis and partial damage of dermis, and third degree with total damage of epidermis, dermis, and partial damage of subcutaneous. It is important to provide a quantitative connection between the surrounding thermal environmental condition and the degree of burn injury. Conducting such experiments directly to collect data from human beings may not be feasible. On the contrary, computer simulation using data from other biological bodies such as animals may be practical.

Current skin thermal injury model proposed by Henriques and Moritz [6, 12] is based on the chemical process of protein denaturation. Skin endures thermal stress and deformation due to temperature variation. Consequently strain energy may play a role in further damage of skin. The aim of this work is to investigate the effect of strain energy on skin thermal injury. In particular, a three-dimensional (3D) numerical model is developed to predict the transient temperature distribution in skin tissues with the consideration of water evaporation on the skin surface and water diffusion in the tissue. Thermal stress and deformation of skin is modeled using Duhamel-Neuman equation, strain energy is then obtained by the stress-strain relation, and skin thermal injury is evaluated based on the proposed model including strain energy. Finite difference method is used to discretize the 3D PDEs, and the resulting linear systems are solved iteratively.

The rest of the paper is arranged as described in the following. Heat and mass transfer process in skin is introduced in Section 2 and thermal stress and deformation is included in Section 3. Thermal injury with the consideration of strain energy is described in Section 4. Numerical algorithm and solution procedure is given in Section 5. Numerical results are reported and discussed in Section 6. Finally a brief conclusion is given in Section 7.
Table 1: Skin thermal physical properties and constants used in the model.

<table>
<thead>
<tr>
<th>Properties</th>
<th>Epidermis</th>
<th>Dermis</th>
<th>Subcutaneous</th>
<th>Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thickness $H$ (m)</td>
<td>$8.0 \times 10^{-5}$</td>
<td>0.0020</td>
<td>0.010</td>
<td></td>
</tr>
<tr>
<td>Thermal conductivity $k$ (W/(mK))</td>
<td>0.25</td>
<td>0.50</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td>Density $\rho$ (W/(mK))</td>
<td>1200</td>
<td>1200</td>
<td>1000</td>
<td>1060</td>
</tr>
<tr>
<td>Specific heat $C$ (J/(kgK))</td>
<td>3600</td>
<td>3400</td>
<td>3000</td>
<td>3770</td>
</tr>
<tr>
<td>Blood perfusion rate $u_b$ (m$^3$/s/m$^3$ tissue)</td>
<td>0</td>
<td>0.00125</td>
<td>0.00125</td>
<td></td>
</tr>
<tr>
<td>Absorption coefficient $\mu_e$ (mm$^{-1}$)</td>
<td>80</td>
<td>2.4</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Initial water content $W_0$ (Kg/m$^3$)</td>
<td>780</td>
<td>780</td>
<td>780</td>
<td></td>
</tr>
</tbody>
</table>

2 Heat and Mass Transfer in Skin

Heat and mass transfer in skin is complicated and involves water evaporation and diffusion. External heating source may increase skin temperature, and the increase of skin temperature will accelerate the rate of molecular water evaporation, hence tends to decrease the temperature on skin surface. Skin temperature variation due to spatial heating is modeled by the following bioheat transfer equation [4, 10, 11],

$$\rho C \frac{\partial T}{\partial t} = k \left( \frac{\partial^2 T}{\partial x^2} + \frac{\partial^2 T}{\partial y^2} + \frac{\partial^2 T}{\partial z^2} \right) + u_b C_b (T_a - T) + Q,$$

(1)

where $T$ is tissue temperature ($^\circ$C), $T_a$ the arterial temperature, and $Q$ the heat source. Note that the metabolic heat generation rate is neglected. The physical properties of the model related to Eq. (1) are summarized in Table 1.

The water transport in skin may be simulated by the Fick’s law of diffusion as [26]

$$\frac{\partial \rho_w}{\partial t} = D \left( \frac{\partial^2 \rho_w}{\partial x^2} + \frac{\partial^2 \rho_w}{\partial y^2} + \frac{\partial^2 \rho_w}{\partial z^2} \right),$$

(2)

in which $D$ is the coefficient of water diffusion in tissue (m$^2$/s), taken as $D = 5 \times 10^{-10}$ [11], and $\rho_w$ the corresponding water content (g/m$^3$).

Heat transfer from water evaporation and diffusion is considered by including them in Eq. (1), such that the heat source $Q$ in Eq. (1) may include the regional heat source $Q_r$, the volumetric heat loss due to water evaporation $Q_e$, and the volumetric heat loss due to water diffusion $Q_d$, as shown below,

$$Q = Q_r + Q_e + Q_d.$$

The heat loss due to evaporation $Q_e$ can then be expressed as [11]

$$Q_e = \dot{m} \Delta H_{vap},$$

where $\dot{m}$ is the rate of water vaporization from the skin surface (g/(m$^2$s)), $\Delta H_{vap}$ the enthalpy of vaporization of water (J/kg), a function of temperature as tabulated in [9]. The rate of water vaporization can be written as [7, 11]

$$\dot{m} = \dot{D}_a \frac{M_w}{R} \left( \frac{P_a}{\rho_w} \right)_a - \left( \frac{P_a}{\rho_w} \right)_a \times H_r \times \delta_c.$$
Here $\tilde{D}_a$ is the average water vapor diffusivity in the boundary layer (m$^2$/s), $H_r$ the relative humidity of the air, $M_w$ the molecular weight of water (g/mol), $R$ the universal gas constant (J/(molK)), $P_w$ the vapor pressure of water (Pa), and $T_w$ the temperature of the water vapor (K). $\delta_c$ is the average thickness of the boundary layer over which a water vapor concentration gradient occurs. The average thickness of momentum boundary layer over the distance of $L$ is found to be

$$\delta = \frac{10}{3} \sqrt{\frac{vL}{v_\infty}}.$$

Here $v_\infty$ is the free stream air velocity taken as 1cm/s. In the simulation, the dynamic viscosity of air $\nu$ and the diffusivity of water vapor in air at the temperature of 25°C are taken to be $1.57 \times 10^{-5}$ m$^2$/s and $2.6 \times 10^{-5}$ m$^2$/s [26] respectively. The volumetric heat transfer rate due to water diffusion is then calculated as

$$Q_d = \frac{Dc_p(\rho_s - \rho_c)(T - T_c)}{(\Delta x)^2},$$

where $c_p$ is the specific heat of water, $\rho_s$ the water content on the skin surface, $\rho_c$ the water content in the body core, $T$ the tissue temperature, $T_c$ the temperature in the body core. A 3D model of thermal response of skin subject to laser heating is proposed and studied in [20], in which water evaporation and diffusion is seen to move a considerable amount of heat energy from the skin in the simulation. If cryogen spray cooling in laser surgery is modeled, however, the influence of water evaporation and diffusion is negligible.

### 3 Thermal Stress and Deformation

One of the destructive mechanisms for thermal damage of soft tissue is thermal induced high mechanical stress. To study the thermal induced deformation in soft tissues at high temperature, the thermoporoelasticity model is used in which stress, strain, and temperature are related. The constitutive relation describing the elastic deformation of the soft tissue may be expressed as [27]

$$\begin{bmatrix}
\sigma_{xx} \\
\sigma_{yy} \\
\sigma_{zz} \\
\tau_{xy} \\
\tau_{xz} \\
\tau_{yz}
\end{bmatrix} =
\begin{bmatrix}
c_{11} & c_{12} & c_{13} & 0 & 0 & 0 \\
c_{21} & c_{22} & c_{23} & 0 & 0 & 0 \\
c_{31} & c_{32} & c_{33} & 0 & 0 & 0 \\
0 & 0 & 0 & c_{44} & 0 & 0 \\
0 & 0 & 0 & 0 & c_{55} & 0 \\
0 & 0 & 0 & 0 & 0 & c_{66}
\end{bmatrix}
\begin{bmatrix}
\varepsilon_{xx} \\
\varepsilon_{yy} \\
\varepsilon_{zz} \\
\gamma_{xy} \\
\gamma_{xz} \\
\gamma_{yz}
\end{bmatrix} -
\begin{bmatrix}
\beta \\
\beta \\
\beta \\
0 \\
0 \\
0
\end{bmatrix}
\begin{bmatrix}
\theta
\end{bmatrix},$$

where $\sigma$ is the normal stress, $\tau$ the shear stress, $\varepsilon$ the normal strain, $\gamma$ the shear strain, and $\theta$ the temperature difference. The coefficients in Eq. (3) are [2]

$$c_{11} = c_{22} = c_{33} = \frac{1 - \nu}{(1 - 2\nu)(1 + \nu)},$$

$$c_{12} = c_{21} = c_{13} = c_{31} = c_{23} = c_{32} = \frac{\nu}{(1 - 2\nu)(1 + \nu)},$$

$$c_{44} = c_{55} = c_{66} = \frac{\nu}{2(1 + \nu)}.$$
and

\[ \beta = \frac{E\alpha}{1 - 2\nu}. \]

Other symbols in Eq. (3) are \( E \) Young’s modulus, \( \nu \) Poisson’s ratio, and \( \alpha \) thermal expansion coefficient. Strain is related to deformation by the following expression,

\[
\begin{bmatrix}
\varepsilon_{xx} \\
\varepsilon_{yy} \\
\varepsilon_{zz} \\
\gamma_{xy} \\
\gamma_{xz} \\
\gamma_{yz}
\end{bmatrix} =
\begin{bmatrix}
\frac{\partial}{\partial x} & 0 & 0 & \frac{\partial}{\partial y} & 0 & \frac{\partial}{\partial z} \\
0 & \frac{\partial}{\partial y} & 0 & 0 & \frac{\partial}{\partial x} & 0 \\
0 & 0 & \frac{\partial}{\partial z} & 0 & 0 & \frac{\partial}{\partial y} \\
\frac{\partial}{\partial y} & \frac{\partial}{\partial x} & 0 & 0 & \frac{\partial}{\partial z} & 0 \\
\frac{\partial}{\partial z} & \frac{\partial}{\partial x} & 0 & \frac{\partial}{\partial y} & 0 & \frac{\partial}{\partial z} \\
0 & \frac{\partial}{\partial z} & \frac{\partial}{\partial x} & 0 & \frac{\partial}{\partial y} & 0
\end{bmatrix}
\begin{bmatrix}
u_1 \\
u_2 \\
u_3
\end{bmatrix}, \tag{4}
\]

where, \( u_1, u_2, \) and \( u_3 \) are displacements in \( x, y, \) and \( z \) directions respectively. From Eqs. (3) and (4), the following equilibrium equation is obtained,

\[ G \sum_{j=1}^{3} \left( \frac{\partial^2 u_i}{\partial x_j^2} + \frac{1}{1-2\nu} \frac{\partial^2 u_j}{\partial x_j \partial x_i} \right) + \beta \nabla \theta_i = 0, \tag{5}\]

where \( u \) is the displacement vector, and \( G \) is the shear modulus given by

\[ G = \frac{E}{2(1+\nu)}. \]

The thermal induced stress has been investigated and discussed in [21], here we focus on the deformation energy and its effect on skin thermal injury. After obtaining the stress and strain tensors, the unit strain energy can be computed according to the following expression,

\[
E_{mech} = \frac{1}{2} V \sigma_{xx} \sigma_{yy} \sigma_{zz} \sigma_{xy} \sigma_{xz} \sigma_{yz}, \tag{6}\]

in which \( V \) is the molar volume of skin tissue.

## 4 Thermal Injury Prediction

A quantitative description of skin thermal damage due to high temperature exposure, suggested by Henriques and Moritz [6, 12], is the following

\[ \frac{\partial \Omega}{\partial t} = \zeta \exp \left( - \frac{\Delta E}{RT(x,t)} \right), \tag{7}\]

where \( \zeta \) is the pre-exponential factor, \( \Delta E \) is the activation energy for the reaction, and \( R \) is the gas constant. The temperature is determined by Eq. (1) and the environmental conditions. The constants and parameters used in this model are: at the epidermis layer, \( \zeta = 3.1 \times 10^{98} \) (s\(^{-1}\)), \( \Delta E = 627.9 \) (kJ/mol); at the dermis layer, \( \zeta = 4.32 \times 10^{84} \) (s\(^{-1}\)), \( \Delta E = 418.6 \) (kJ/mol) for temperature \( 44 \leq T \leq 55 \) °C, and \( \zeta = 9.39 \times 10^{104} \) (s\(^{-1}\)), \( \Delta E = 669.8 \) (kJ/mol) for temperature
\[ T \geq 55 \, ^\circ\text{C} \] [24, 25]. Integrating Eq. (7) over the period of burn, the total accrual of thermal injury as a function of position within the tissue is

\[
\Omega = \zeta \int_0^t \exp \left( -\frac{\Delta E}{RT(x, t)} \right) \, dt. \tag{8}
\]

In the case of skin burn, soft tissues are subject to thermal stress, which may cause possible tissue deformation. Intuitively, tissue may be more easily damaged under such a condition. That is, tissue protein denaturation may happen at relatively small activation energy \( E \) in Eq. (8). Therefore we propose the following integral equation to estimate the local accrual of thermal injury

\[
\Omega = \zeta \int_0^t \exp \left( -\frac{\Delta E - E_{\text{mech}}}{RT(x, t)} \right) \, dt, \tag{9}
\]

where \( E_{\text{mech}} \) is the mechanical energy defined by Eq. (6).

5 Numerical Algorithm and Solution Procedure

According to the proposed model for predicting skin burn injury, Eqs. (1), (2), (5), and (9) have to be solved at the same time. The corresponding algorithm is listed below.

1. Set \( t := 0 \), \( n := 0 \)
2. Assign initial values to \( T^{(0)} \), \( w^{(0)} \), \( u^{(0)} \), \( \Omega^{(0)} \)
3. Initialize values at level \( n \): \( T^{(n)} \leftarrow T^{(0)} \), \( w^{(n)} \leftarrow w^{(0)} \), \( u^{(n)} \leftarrow u^{(0)} \), \( \Omega^{(n)} \leftarrow \Omega^{(0)} \)
4. \textbf{while} \( t < t_{\text{end}} \) \textbf{do}
5. \text{Solve Eq. (1) iteratively for} \( T^{(n+1)} \)
6. \text{Solve Eq. (2) iteratively for} \( w^{(n+1)} \)
7. \text{Solve Eq. (5) iteratively for} \( \sigma^{(n+1)} \)
8. Compute stress and strain tensors from Eqs. (3) and (4)
9. Compute unit strain energy from Eq. (6)
10. Compute the accrual of thermal injury from Eq. (9)
11. Assign values as: \( T^{(n)} \leftarrow T^{(n+1)} \), \( w^{(n)} \leftarrow w^{(n+1)} \), \( \Omega^{(n)} \leftarrow \Omega^{(n+1)} \), \( t := t + \Delta t \)
12. \textbf{end while}

In the above algorithm both Eqs. (1) and (2) are discretized using Crank-Nicholson finite difference scheme. Eq. (5) is not time dependent and is discretized using a second order 19 point finite difference scheme. Using Eq. (1) as an example, the following expression can be derived,

\[
\frac{T^{n+1} - T^n}{\Delta t} = \frac{1}{2} \alpha A T^{n+1} + \frac{1}{2} \alpha A T^n + S^{n+1/2}. \tag{10}
\]

Here \( \Delta t \) is the time step, \( A \) the finite difference approximation of the elliptic operator, and \( S \) the source term. The truncation error of the scheme is of order \( O(\Delta t^2 + h^2) \). Eq. (10) may be written in a matrix form as,

\[
\left( I - \frac{\alpha \Delta t}{2} A \right) T^{n+1} = \left( I + \frac{\alpha \Delta t}{2} A \right) T^n + \alpha \Delta t S^{n+1/2}. \tag{11}
\]

For each of Eqs. (1), (2) and (5), a sparse linear system, denoted by \( Ax = b \), can be obtained from the discretization. They are solved iteratively using the fast iterative solver GMRES, accompanied
with an ILUT preconditioner [16]. Note, in the GMRES method, the required storage grows quadratically with the number of iterations. So the number of iterations should be confined to not a big one. Practically, A restarted version of GMRES is employed as GMRES(10), where 10 is the dimension of the Krylov subspace, so that GMRES is restarted after 10 iterations. An incomplete LU factorization with threshold (ILUT) is used to speed up the convergence rate of GMRES. The ILUT preconditioner makes use of a dual dropping strategy that is represented by two parameters $p$ and $\tau$, where $p$ is the number of fill-in elements, and $\tau$ is the dropping tolerance. In our computation, we used $p = 40$ and $\tau = 10^{-4}$. Instead of the original linear system $Ax = b$, an equivalent one $M^{-1}Ax = M^{-1}b$ is solved in the preconditioned iterations, where $M$ is the ILUT preconditioner. More information about the GMRES solver and the ILUT preconditioner can be found in [17].

6 Numerical Experiments and Discussions

A 3D view of the skin structure is depicted in Fig. 1, where from the top to the bottom, each layer is shown as the epidermis, the dermis, and the subcutaneous in order. As can be seen in Table 1, the thickness of each layer is epidermis $8.0 \times 10^{-5}$ m, dermis 0.002 m, and subcutaneous 0.01 m [24]. For the convenience of simulation, the area of interest on the skin surface is assumed to be a square with a dimension of $L = W = 0.015$ m. One of our major interests for conducting this research is to investigate the instant thermal mechanical response in each of the three skin physical layers and evaluate the degree of burn injury. Due to the extreme thin thickness of the epidermis layer, uniform grid may not be appropriate. The grid points are hence distributed as 10 intervals in the epidermis layer, 40 intervals in each of the dermis and subcutaneous layers, which gives a mesh size in the $x$ direction as $\Delta x = 8 \times 10^{-6}$ m in the epidermis layer, $\Delta x = 5 \times 10^{-5}$ m in the dermis layer, and $\Delta x = 2.5 \times 10^{-4}$ m in the subcutaneous layer, as listed in Table 1. Along the $y$ and $z$ directions, equally spaced 50 intervals are assigned. That gives a mesh size of $\Delta y = \Delta z = 0.0003$ m. Such a small $\Delta x$ puts a constraint on the determination of time step $\Delta t$, which is chosen as $\Delta t = 0.001$ s. The criterion for determining $\Delta t$ is presented in our recent paper [21]. With this arrangement, a total of $90 \times 50 \times 50$ grid intervals are resulted. The number of unknowns resulting from discretizing Eqs. (1) and (2) is therefore 213,689 each, and the corresponding number of nonzeroes in the coefficient matrix is 1,473,577. Since there are 3 components at each grid points, a discretization of Eq. (5) gives 641,067 unknowns.

This work differs from others [3, 13, 24, 25] in predicting skin thermal injury by including the effect of mechanical energy resulting from thermal induced stress and deformation, and interpreting the tissue damage as stages of mechanical response and chemical reaction. In the stage of mechanical response, soft tissue is under thermal stress and has deformation energy, which can further affect the ongoing chemical reaction process by possibly lowering the activation energy. The model is general in simulating various thermal burning processes of biological tissue by easily modifying the heat source term in Eq. (1). Using this skin burn model, a few numerical experiments have been conducted, including laser therapy, flash light, and steam burn, which are discussed below.

**Laser Therapy** As a means of treatment to dermatological patients, laser therapy has been widely
used due to its apparent advantages over conventional surgeries [23]. As an example, dye lasers can be used to selectively damage some microvasculature in human skin [1]. Laser therapy, however, may cause possible side effects and complications because of unintentional heating of the adjacent good tissues. This example studies the tissue damage of laser type heat source exposure. The heat transfer due to laser heating may be approximated by a Gaussian type heat source with the following expression,

$$Q_r = \mu_a \phi(x, y, z) f(t),$$

where $\mu_a$ is the tissue absorption coefficient (1/m), $f(t)$ is the time function [21], and $\phi(x, y, z)$ is the local fluency rate of the laser light (W/m²) [4],

$$\phi(x, y, z) = E \exp(-2(y^2 + z^2)/W^2) \exp(-\mu_a x),$$

in which $E$ is defined as $E = 2P/(\pi W^2)$, $P$ the laser power (W), $W$ the 1/e² waist (m). The time function $f(t)$ is assumed to be periodic and may be expressed as [19]

$$f(t) = \begin{cases} 
1 & (i - 1)P < t < [(i - 1) + \kappa]P, \\
0 & [(i - 1) + \kappa]P < t < iP 
\end{cases} \quad i = 1, 2, 3, \ldots,$$

where $i$ is the number of periods, $P$ the period (s), $\kappa$ the fraction of period in which the skin is exposed to laser source. It is assumed that skin under investigation is subject to laser heating every 0.1 s with a duration of 0.005 s, and the time step is chosen as $\Delta t = 0.001$ s. The parameters of the Gaussian shaped laser beam corresponding to Eq. (12) are $P = 12$ W, $W = 0.0025$ m, and $\mu_a = 80, 2.4$, and 1 mm⁻¹ in the epidermis, dermis and subcutaneous regions respectively.

Skin damage occurs when the temperature at the base of the epidermis layer reaches above 44°C. The rate of burn injury is said to be second degree if the accumulated integral of $\Omega$ is greater than 1. A burn injury is third degree if the temperature at the base of the dermis layer reaches above 44°C and the accumulated integral of $\Omega$ is more than 1 [6, 24]. The thermal response of skin subject to laser exposure is shown in Fig. 2. Fig. 2(a) is the temperature distribution at the
base of epidermis layer for a period of 50 s. Fig. 2(b) is the consequent strain energy due to tissue deformation. Figs. 2(c) and 2(d) are the corresponding results of the accrual damage without and with the consideration of the mechanical energy. It can be seen that after 50 s the damage contour of $\Omega$ with level 1 is at $r \leq 0.0021$ m when the mechanical energy is not considered and at $r \leq 0.0022$ m when the mechanical energy is considered. That is to say that for the same laser power and exposure time, more area is subject to the second degree burn when the mechanical energy is considered. In other words, less time is needed for the second degree burn. Fig. 3(a) is the contour plot of the accrual damage at the base of the dermis layer for an exposure time of 200 s. The threshold of the third degree burn, the damage contour with level 1, is at the location of $r_0 = 0.001$ m when the mechanical energy is not considered. It is at the location of $r_0 = 0.0012$ m when the mechanical energy is considered. Any place inside the circle of $r = r_0$ is considered to have a third degree burn. It can be found by a comparison between Figs. 2 and 3 that an inclusion of the mechanical damage affects more for the third degree burn prediction than for the second degree one. This may be partially due to the relatively small value of the activation energy $\Delta E$ in Eq. (9) in calculating the accrual damage of the dermis layer for temperatures of $44 \leq T \leq 55 \, ^\circ$C.

It can be seen from Fig 2(b) that in this particular case the calculated strain energy is less than 1,000 J/cm$^2$, which is small compared with the activation energy $\Delta E$ in Eq. (9). The strain energy can be large, however, in certain circumstances. It is observed from Eq. (1) that the tissue temperature is related to the parameters of $\alpha_1 = k/(\rho C), \alpha_2 = \omega_b C_b (T_0 - T)/(\rho C), \text{ and } \alpha_3 = Q/(\rho C)$, where $\alpha_1, \alpha_2, \text{ and } \alpha_3$ denote the effects of thermal properties, blood flow, and external heat source on temperature variation respectively. In most skin burn injury cases, the influence of $\alpha_2$ may be negligible due to its relative small quantity compared with $\alpha_3$. Since the skin thermal properties are assumed to be constant, the temperature distribution is solely determined by the external heat flux. It is assumed that mechanical properties are constant as well, then tissue displacement is solely determined by the temperature derivatives, as shown in Eq. (5). Thus if the external heat source $\alpha_3$ is extremely large, a large temperature derivative will result, which leads to large tissue displacement, stress and strain tensors, and mechanical energy. Consequently, the influence of mechanical deformation on tissue damage may be significant.

Note, in order to display the results more clearly, only part of them, i.e., $0 \leq y \leq 0.005$ m and $0 \leq z \leq 0.005$ m, is shown in Figs. 2 and 3(a), and the accumulated damage $\Omega$ is scaled to $\Omega'$ by $\Omega' = 1 + \log \Omega$.

**Flash Fire** Flash fire can be simulated using the current model by modifying Eq. (1). The body exposure to flash fire is assumed to be uniformly distributed, so the problem can be treated as one-dimensional. This can be achieved by specifying symmetric boundary conditions along the border of the region under investigation, i.e., $y$ and $z$ directions, and uniform heat flux on the skin surface. The current model is used to predict the time to second degree burn for a number of exposures with the intense heat flux ranging from 4.186 kW/m$^2$ to 4.86 kW/m$^2$. This can be achieved easily by modifying the local heat source in Eq. (1) as $Q_r = C$, where $C$ is a constant corresponding to the actual heat fluxes being tested. It can be seen from Fig. 4 that the present results agree well with the published ones [24]. The predicted time to the second degree burn falls between the experimental data given by Stoll and Greene [22] and the closed form solution provided in [5].
Figure 2: Two-dimensional temperature and accumulated damage plots for laser heating with a Gaussian profile. (a) Temperature (°C) distribution at the base of the epidermis layer after 50 s. (b) The contour of mechanical energy (J/cm³) at the base of the epidermis layer after 50 s. (c) The contour of accumulated damage at the base of the epidermis layer after 50 s without the consideration of mechanical energy. (d) The contour of accumulated damage at the base of the epidermis layer after 50 s with the consideration of mechanical energy. Note: in Figs. 2(c) and 2(d) the accumulated damage is scaled to \( \Omega' = 1 + \lg \Omega \).
the same exposure intensity, our results predict more time than the finite element model by Torvi and Dale [24]. This may be due to the fact that water transport is considered in our model, which removes some of the incoming thermal energy and tends to decrease the degree of thermal damage.

Steam Burn As an example, steam burn is also studied. The physical process differs for different type of burns. Laser burn and flash burn have the same nature of relatively high pressure on the skin surface and possible water evaporation and diffusion from skin surface to the environment, while steam burn is different. We model steam burn by adjusting the source term to include condensation. Since condensation is the reverse process of evaporation, we may implement this by changing the sign of $Q_e$ and $Q_d$ in Eq. (1). Due to the lower pressure at skin surface, water molecules moves from hot air toward it. The movement of vapor molecules slows down on skin surface, since temperature on skin is lower than the steam temperature. A thin layer of liquid film is formed on the skin surface. Heat transfer from steam to skin is via this thin film. Assume steam heat transfer to skin can be modeled as film condensation on a vertical plate, the heat transfer to skin surface may be written as [8]

$$Q = \tilde{h}_L A(T_{\text{sat}} - T_s),$$

where $Q$ is the heat transfer rate from steam to skin, $T_{\text{sat}}$ is the temperature of saturated vapor, the convective heat transfer coefficient is

$$\tilde{h}_L = C \left[ \frac{g \rho_l (\rho_l - \rho_v) k_l H}{\mu_l (T_{\text{sat}} - T_s) L} \right]^{1/4},$$

in which $C$ is a constant, $g$ the gravitational acceleration (m/s²), $\rho_l$ the density of liquid water (kg/m³), $\rho_v$ the density of saturated water vapor, $k_l$ the thermal conductivity of liquid water (W/(mK)), $H$ the latent heat of vaporization (J/kg), which is a function of temperature, $\mu_l$ the
viscosity of liquid water (kg/(sm)), and $L$ the characteristic length (m). In applying Eq. (13) to find the temperature distribution in skin, $T_s$ is not known, which has to be solved iteratively with Eq. (1). The latent heat of water condensation is temperature dependent, which is expressed in a polynomial from least-square data fitting based on the tabulated data from [9]. All liquid phase properties are evaluated at the film temperature $T_f = (T_{\text{sat}} + T_s)/2$. Part of the solution is shown in Fig. 5 as compared with that of Ng and Chua’s [13] for the prediction of the third degree burn. For the same steam temperature, our model predicts that more time is needed for the third degree burn. It is worth noting that the threshold for a third degree burn in their model is at temperature above 44 ºC at the base of the dermis layer, while in our model it is the accumulated damage of $\Omega = 1$ for temperatures over 44 ºC.

Figure 5: Predicted time to the third degree burn for various steam temperature.
7 Conclusion

A 3D model is developed to investigate the skin injury caused by thermal exposure with the consideration of deformation, which may introduce strain energy. The model is based on the consideration of Pennes’s bioheat equation for skin thermal response, Duhamel-Neuman equation for thermal stress and strain calculation, and Henrique's suggestion of Arrhenius equation for the skin damage prediction. A numerical algorithm is presented to solve those equations in sequence. It is found that strain energy lowers the activation energy for protein denaturation, and thus tends to give a prediction of shorter time for both the second and the third degree skin burns. But the effect may not be significant in the tested cases, so skin burn prediction without the consideration of deformation energy is still a reasonable approach. However, if the external heating source is extremely large, mechanical energy may play an important role in predicting skin thermal injury.

This model can be easily adapted to simulate burns of various thermal exposure. In particular, a number of numerical experiments have been conducted including laser type heating source, flash fire, and steam exposure, which are typical thermal burns. Our model tends to predict longer time for skin thermal damage. This may be due to the fact that water evaporation is considered in modeling the skin heat transfer process. Water evaporation takes away some of the heat, which cools skin surface.

Finite difference method is used to discretize the PDEs involved in the model, which gives an accuracy of second order in space. For the time dependent problems, Crank-Nicholson scheme is used such that second order accuracy is achieved in time as well. Preconditioned iterative method is used for fast solution of the resulting sparse linear systems.

References


