Non-Fourier based thermal-mechanical tissue damage prediction for thermal ablation

Xin Li, Yongmin Zhong, Julian Smith, and Chengfan Gu

ABSTRACT
Prediction of tissue damage under thermal loads plays important role for thermal ablation planning. A new methodology is presented in this paper by combing non-Fourier bio-heat transfer, constitutive elastic mechanics as well as non-rigid motion of dynamics to predict and analyze thermal distribution, thermal-induced mechanical deformation and thermal-mechanical damage of soft tissues under thermal loads. Simulations and comparison analysis demonstrate that the proposed methodology based on the non-Fourier bio-heat transfer can account for the thermal-induced mechanical behaviors of soft tissues and predict tissue thermal damage more accurately than classical Fourier bio-heat transfer based model.

Introduction
Thermal ablation is a minimally invasive thermal treatment for curing unrespectable tumors located inside human organs such as the liver and stomach. Currently, the thermal ablation process is controlled using temperature elevation to form tissue coagulation zones. This method is inaccurate, as an optimized temperature field does not necessarily imply optimized tissue damage. It also ignores the effect of thermal-mechanical deformation during the thermal therapy. In fact, even a small variation in thermal-induced mechanical deformation can lead to various effects such as protein denaturation, altered production of hormones, and suppressed immune response. Therefore, it is an absolute necessity to study tissue response under thermal and mechanical loads to provide an effective guidance for precise control of thermal ablation tasks.

Several authors have studied thermal-mechanical behaviors of soft tissues under thermal therapy. Xu et al. treated human skin as a kind of composite material, laminate, where heat-induced mechanical response (thermal stress) in different layers of skin was calculated using traditional composite material mechanics. However, this method is only limited to layered soft tissues and how thermal-mechanical response is incorporated into existed damage model to qualify thermal-mechanical damage was not discussed. In order to identify the influence of thermally induced mechanical deformation on thermal damage prediction, Shen et al. developed a tissue damage model using Fourier bio-heat transfer equation. It shows that thermally induced mechanical deformation decreases the activation energy for protein denaturation, making soft tissues more easily to be damaged. Recently, the author’s group developed a model to predict thermally induced mechanical deformation and thermal damage of soft tissues by combining the Fourier bio-heat transfer equation with the theory of linear thermo-elasticity. In general, most of the existing methods are mainly dominated by the classical Fourier bio-heat transfer equation, which assumes infinite speed of heat propagation in soft tissues. In fact, a change temperature in a local area does not instantaneously cause a perturbation on the temperature distribution in the rest area of the medium. The heat transfer in a tissue like non-homogenous medium needs a characteristic time to propagate to the nearest point. This non-Fourier thermal behavior has been observed in biological tissues via physical experiments.

CONTACT Yongmin Zhong yongmin.zhong@rmit.edu.au School of Engineering, RMIT University, PO Box 71, Bundoora 3083, Australia. Color versions of one or more of the figures in this article can be found online at www.tandfonline.com/kbie.
This paper gives a new methodology for modeling of thermal-mechanical behaviors and associated damage of soft tissues during thermal ablation, where the modeling process combines non-Fourier bio-heat transfer, continuum mechanics, as well as non-rigid motion of dynamics to predict and analyze temperature distribution and thermal-induced mechanical deformations of soft tissues. An improved tissue damage model is developed by accounting for thermal-induced mechanical damage. The finite difference scheme and numerical algorithms are utilized to construct the proposed thermal-mechanical tissue damage model. Simulations and comparison analysis with the classical Fourier bio-heat transfer have been performed to evaluate the performance of the proposed methodology.

**Material model and numerical modeling**

**Non-Fourier bio-heat transfer equation**

The classical Fourier based bio-heat transfer equation considering coupled thermo-elastic effect can be described as,

\[
\rho C \frac{\partial T}{\partial t} + \beta T_0 \frac{\partial (\nabla \mathbf{u})}{\partial t} = k \nabla^2 T + C_b \omega_b \rho_b (T_b - T) + Q_m + Q_{\text{ext}} \quad (2)
\]

where \( \rho \) is the density of soft tissues, \( C \) the specific heat, \( \beta \) the coefficient of thermal expansion, \( \mathbf{u} \) the displacement vector, \( k \) the thermal conductivity, \( T \) the temperature at time \( t \), \( \omega_b \) the blood perfusion, \( \rho_b \) the density of blood, \( C_b \) the specific heat of blood, \( T_b \) the temperature of blood vessel, \( T_0 \) the initial temperature, \( Q_{\text{ext}} \) the external heat source, and \( Q_m \) the generated metabolic heat.

Applying the concept of finite heat propagation velocity, a modified unsteady heat transfer equation can be obtained

\[
q(r, t + \tau_q) = -k \nabla T(r, t) \quad (2)
\]

where \( \tau_q \) is the characteristic time of biological soft tissues.

Using first-order Taylor expansion, the above equation becomes

\[
q(r, t + \tau_q) = q(r, t) + \tau_q \frac{\partial q(r, t)}{\partial t} \quad (3)
\]

Substituting (3) into (1), the non-Fourier bio-heat transfer equation can be obtained,

\[
\tau_p \rho C \frac{\partial^2 T}{\partial t^2} + (\tau_p C_b \omega_b \rho_b + \rho C) \frac{\partial T}{\partial t} + \tau_p \beta T_0 \frac{\partial^2 (\nabla \mathbf{u})}{\partial t^2} \]

\[
+ \beta T_0 \frac{\partial (\nabla \mathbf{u})}{\partial t} = k \nabla^2 T + \tau_p \frac{\partial Q_m}{\partial t} + \tau_p \frac{\partial Q_{\text{ext}}}{\partial t} + C_b \omega_b \rho_b (T_b - T) + Q_m + Q_{\text{ext}} \quad (4)
\]

**Linear elastodynamics**

Biologically, soft tissues are complex in terms of material compositions and structural formations. While soft tissue structure shows time-dependent, nonlinear and anisotropic behaviors, in terms of small deformations caused by thermal load, soft tissues can be investigated by linear thermo-elastic models to a high precision.

From the constitutive elastic material law under thermal loads, the stress tensor is related to the strain tensor and temperature change \( \theta \), which equals \( T - T_0 \)

\[
\sigma_{ij} = \mu (u_{ij,i} + u_{ji,i}) + \lambda u_{kk,i} \delta_{ij} - \beta \theta \delta_{ij} \quad (5)
\]

where \( u_{ij} \) is the displacement components, and \( \delta_{ij} \) is the Kronecker’s symbol defined as

\[
\delta_{ij} = \begin{cases} 
1 & \text{for } i = j , \\
0 & \text{for } i \neq j ,
\end{cases}
\]

Rewriting the governing equation of non-rigid mechanics of motion as

\[
\rho \ddot{\mathbf{u}}_i = \sigma_{ij,i} + F_i \quad (6)
\]

Rearranging the motion equation as,

\[
\rho \ddot{\mathbf{u}}_i = (\mu (u_{ij,i} + u_{ji,i}) + \lambda u_{kk,i} \delta_{ij} - \beta \theta \delta_{ij}) + F_i \quad (7)
\]

where \( \lambda \) and \( \mu \) are the Lame constants, \( F \) is the exerted external force and,

\[
2\mu = \frac{E}{1 + \nu} , \quad \lambda = \frac{v E}{(1 + \nu)(1 - 2\nu)} ,
\]

\[
\beta = \frac{\alpha E}{1 - 2\nu} = \alpha (3\lambda + 2\mu) , \quad \mu = G
\]

Constructing the proposed model on a regular cubic mesh straightforwardly using a finite difference
scheme, gives the following global characteristic equation for all mesh points:

\[ M \ddot{u} + C \dot{u} + K u = F, \quad u = \begin{bmatrix} U \\ \theta \end{bmatrix} \quad (8) \]

To solve Eq. (8), it is necessary to specify the boundary conditions under concern. Here, the boundary condition for solving Eq. (8) is realized by Dirichlet boundary condition, in which specified temperature and displacement are enforced to the related points on the solution domain boundaries at all times.

**Thermal-mechanical damage prediction model**

To predict thermal damage with consideration of thermal induced mechanical deformation effect, the relevant governing equation is expressed in Eq. (9). Basically, it assumes that soft tissues subject to thermal stress cause tissue deformation. Thus, tissues are more easily being damaged under such a condition, because tissue protein denaturation may occur at relatively small activation energy.

\[ \Omega = \xi \int_{0}^{t} \exp \left( -\frac{\Delta E - E_{\text{mechanical}}}{RT(x,t)} \right) dt \quad (9) \]

where \( E_{\text{mechanical}} \) is the strain energy per mole, which is represented by Eq. (10). \( V_{\text{mol}} \) stands for molar volume of target tissue.

\[ E_{\text{mechanical}} = \frac{1}{2} V_{\text{mol}} \sum_{i,j=1}^{3} \sigma_{ij} \epsilon_{ij} \quad (10) \]

**Results and discussion**

Several simulations were conducted to test the performance of proposed thermal-mechanical model based on non-Fourier bio-heat transfer. Consider a cubic volumetric tissue model, which contains 1000 elements with 1331 nodes, with a needle inserted inside (see Fig. 1). The needle has 5 evenly spaced point sources at the front to generate heat energy and they are positioned in the Y direction at the center of the middle horizontal plane inside the cubic tissue. The needle generates thermal energy at the 5 points at \( 4 \times 10^8 \) W to heat up the tissue model. Table 1 shows the thermal and mechanical parameters used for the cubic tissue under thermal loads.\(^{13-18}\) The boundary of the cubic model is fixed and its temperature is set to 310 K. The cubic model has the initial temperature of 310 K at the rest state.

**Temperature distributions**

To study the thermal behavior of the cubic tissue, 3 observation points \( P_1 \) (0.005, 0.005, 0.005), \( P_2 \) (0.005, 0.005, 0.006) and \( P_3 \) (0.005, 0.005, 0.007) (blue points in Fig. 1), were chosen to observe the temperature variation inside the cubic tumor during the heating

**Table 1. Tissue parameters and constants.**

| Parameters                      | Value       |
|---------------------------------|-------------|
| Density \( \rho \); kg/m\(^3\) | 1060        |
| Specific heat \( C_{s} \); J/kgK | 4192        |
| Thermal conductivity \( k \); W/mK | 0.613       |
| Thermal expansion coefficient \( \alpha \); 1/°C | \( 1 \times 10^{-4} \) |
| Young’s modulus \( E \); Pa       | 1.1 \times 10^6 |
| Poisson’s ratio \( \nu \)          | 0.48        |
| Blood perfusion rate \( \omega_{b} \); kg/(m\(^3\)s) | 0.5        |
| Arterial temperature \( T_{a} \); K  | 310         |
| Metabolic heat generation rate \( Q_{m} \); W/m\(^3\) | 33800      |
| Blood specific heat \( C_{b} \); J/kgK | 3600       |
| Characteristic Time \( \tau_{c} \); s | 16         |
| Activation energy \( \Delta E / \text{mol} \) | 667000     |
| Frequency factor \( \gamma \); 1/s  | \( 1.98 \times 10^{10} \) |
| Gas constant \( R_{f} \); J/(molK) | 8.314      |
process. Figure 2 illustrates the temperature distributions at these 3 points within a heating period of 120 s for both models. As we can see, at the initial stage of heating (within about 40 s), the non-Fourier model involves oscillations in the temperature rise. After the initial stage of heating, with the increase of the heating time, the temperature rise for the non-Fourier model is larger than Fourier model, but the difference between both models gradually becomes smaller and finally the temperature curves of both models are converged to each other. This demonstrates that although the Fourier and non-Fourier models have similar accuracy for temperature prediction after a sufficient time of heating, the non-Fourier bio-heat model accounts for the transition state of bio-heat transfer, thus more suitable for the prediction of tissue’s instantaneous thermal-mechanical behavior.

To further study the thermal behavior of the cubic tumor, the plane at \( Y = 0.005 \) m, which is perpendicular to the heating line, was selected to observe the temperature variation during the heating process. Figure 3 shows the temperature distributions on this plane at the initial time of 10 s for both Fourier and non-Fourier models. It can be seen that the temperature distributions of both models have a similar behavior with the highest temperature at the center of the selected plane, which is also the center of the heating sources. However, the non-Fourier model has smaller temperature rise than the Fourier model. This is because the non-Fourier model involves a process of time relaxation, leading to the smaller temperature rise at the initial heating stage.

**Thermal-induced mechanical deformations**

Trials were also conducted to study the thermal-induced deformation behaviors of the cubic tumor. Figure 4 shows the strain distributions at the above plane (\( Y = 0.005 \) m) at the heating time of 10 s for both Fourier and non-Fourier based methods. It can be seen that following the temperature distributions shown in Fig. 3, the strain in the \( Y \) direction of the non-Fourier based method is also smaller than that of the Fourier based method. It should be noted that as the selected observation plane is perpendicular to the \( Y \) axis, the strains in the \( X \) and \( Z \) directions are of a similar behavior, but are different from that of the strain in the \( Y \) direction. The above similar phenomenon can also be observed for the stresses in the 3 axis directions shown in Fig. 5. Figure 6 shows the strain energy distributions for both models. It can be seen that the non-Fourier model has smaller strain energy than the Fourier model at the initial heating time of 10 s. This proves that the strain energy distribution also follows the temperature distributions.
shown in Fig. 3. From the above, it is evident that the temperature distribution has a significant effect on thermal-induced strain and stress as well as strain energy.

**Damage distribution**

Simulations were also conducted to test the performance of the proposed thermal-mechanical tissue damage model. Figure 7 shows the damage comparison between the Fourier and non-Fourier models at the 3 selected points $P_1$-$P_3$. As the non-Fourier based method involves the transition state at the initial heating stage, at the points (e.g. $P_1$ and $P_2$) nearer to the center of the heat sources (i.e. the center of the selected observation plane), it needs more time to reach the threshold of irreversible tissue thermal damage ($\Omega = 1$) than the Fourier based method. This phenomenon basically follows the above analysis at initial heating time of 10 s for points $P_1$ and $P_2$, where both temperature and strain energy prediction values were larger for

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**Figure 4.** Comparison of strain distributions between the Fourier (top) and non-Fourier (bottom) based methods.

**Figure 5.** Comparison of stress distributions between the Fourier (top) and non-Fourier (bottom) based methods.
attributed to larger temperature prediction values toward the time point where the damage threshold was reached (around 60 s) for the non-Fourier model at point P3, as shown in Fig. 2. Moreover, Fig. 8 shows the damage distributions at the above plane (Y = 0.005 m) at the end heating time of 120 s. It can be seen that the final tissue damage zone for the non-Fourier based method (in green) is clearly larger for that of the Fourier based method (in Bangladesh green).

**Conclusion**

This paper presents a new methodology to predict thermal-induced mechanical deformation and associated tissue damage of soft tissues under thermal load. This methodology establishes a thermal-mechanical model and tissue damage model based on non-Fourier bio-heat transfer and continuum mechanics. Simulations and comparison analysis demonstrate that the proposed methodology based on non-Fourier bio-heat transfer accounts for thermal-induced mechanical deformation of soft tissues more accurately, leading to more accurate prediction of tissue thermal damage. Future work will focus on the construction of the proposed methodology on irregular human organ models using finite element method.

**Disclosure of potential conflicts of interest**

No potential conflicts of interest were disclosed.

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