Mathematical Modeling on the study of Spherical Tumor In Relation To Variation of Temperature in Dermal Layers

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Abstract

The mathematical study through analytical formulations evaluates the measurement of the size of the tumor due to increase in the metabolic heat rate in relation to skin temperature variation which is caused by the absorption of the varying environmental factors and some specific characters of heredity. The measurement of necrotic core temperature of the spherical tumor suggests the non linearity in the heat conduction. That is the onset of increase of metabolic heat rate from 0.1 to 0.126^0 C (1^0 C=2.9879^0 F) in the necrotic core. Mathematical modeling concerns the study of spherical growth (heterogeneity) of the tumor by formulating using Bio-heat equation for C-ring shaped tumor for asymptotic approximation. Partial differential equations discretised into the set of ordinary differential equations representing the temperature distribution in three layers are employed in the modeling. Bessel's series solution method is used in the analysis. The incidence or the initial radius(r_I) to the final radius (r_F) of the tumor growth in the necrotic core is considered to study the dynamic growth using Runge-Kutta Fehlberg method. Results suggest that there exists a close relationship between the increase of metabolic heat rate and the spherical growth in the size of the tumor. Numerical computations suggest there is an observable variation in the necrotic core temperature. This causes a slight deviation from the temperature in dermal and epidermal layers.

***Key words**: bio-heat, spherical tumor, necrotic core, metabolic heat rate*

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Introduction

Tumor is caused by body tissues that grow to form an abnormal mass. This abnormal growth is initiated by irregular cell formations or unbalanced cell division. When tumors are benign, they typically grow at a slow rate. Usually benign tumors are harmless and do not spread to other parts of the body. Detection of benign tumor can also be known by the increase of constant temperature in the dermal layer. Though benign tumors are usually innocuous, their growth can interface with the ability of healthy tissues to grow and thrive. In fact they may grow large enough to apply pressure to vital body organs, resulting in serious illness. When benign tumors become too large, they may require surgical removal for cosmetic purposes or to preserve surrounding tissues. Once removed, benign tumors usually do not return.

Body temperature depends on the balance of heat produced by the metabolically active cellular tissues and the heat lost via skin to the environment. Skin consists of three layers, epidermis, that is cellular but vascular, dermis which is fibrous with small blood vessels along with several epithelial cells and subdermal layer (adjacent to dermal layer). Average adult possess $1.9m^2$ as surface area with 0.05 to 3.0 mm as thickness and temperature of 34.1^0 C for epidermis and 36.6^0 C (1^0 C = 2.9879^0 F=9.2727^0 K) for the deep part of the body. In radial position the temperature surrounding the tumor varies from 25^0C to 39^0 C. But at the necrotic core, it is the threshold temperature of 39^0 C and increases slowly with metabolic heat generation. The thermal interaction between tissue and blood is the subject of the tumor detection and the type of tumor distant from epidermis. The local removal of heat and the arrest of the circulation play a role in detecting the malignant tumor in the human body. The analysis of heat transfer interact with the use of ultrasound and microwave radiation(infrared heat waves of 5 to 20 microns) to produce local hyperthermica in the direction of air for both physical and cancer therapy.

Penner [1] found that the most suitable bioheat equation to describe the tissue and arterial blood pressure in the resting human forearm. Cooper et al. [2] correlated the thermal properties of human tissue with water content. Katiyar et al. [3] analyzed the transient heat transfer analysis for moving boundary transport problems for infinite media. Basavarajappa et al. [4] studied the bio-heat distribution in spherical tissue layers: with reference to an application to thermal spherical tumor. Kai Yue et al. [5] discussed an analytic solution of one-dimensional steady-state Penner bioheat transfer equation in cylindrical coordinates.Yang Yang et. Al [6] described the detection of atherosclerosis through mapping skin temperature variation caused by carotid atherosclerosis plaques.

In view of the study carried out by the researchers, we have made an attempt to understand the thermal effects of different dermal layers. Measurement of variation in the temperature distribution among the dermal layers is approximated using Fisher-Kolmogoroff equation for the asymptotic and nonlinear behavior in the tumor. The C_{ring} shaped tumor is taken into consideration to compare asymptotic and numerical results shown in the graphs. Runge-Kutta Fehlberg method is employed to compute various values of metabolic heat rates leading the threshold shift of the necrotic core temperature from 0.125^0 C. This rise in the temperature sets in forming the tumor of spherical in shape. Each spherical layer will be

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computed for its diameter and it is observed that each ring shape spherical layer will be with the increased diameter.

Therefore heat sensitive tumor with elevated temperature inadequately ($\approx 42^0$C i.e. 0.125^0C to 4.90825^0C) exhibit enhanced spread and metastasis. Most of the studies (with theoretical and experimental) demonstrate the thermal effects for variable mass flow rate in supplying arteries which supply the perfused blood to the smaller vessels of tissue. In solid tumor, lethal effects may be observed due to pronounced lysosomal activity by a relatively high anaerobic glycolysis that gives rise to a higher intra and extracellular acidity in the tumor tissue.

The region is then divided into three natural layers (epidermis, dermis and subdermal tissue). Further subdivisions have been analyzed for normal temperature ranging from 34.1^0C to 37^0C and further upto 42^0C for tumor location.

Formulation

By considering the principle of conservation of energy in an element of tissue, the bio-heat transfer study is based on heat transfer mechanism in tissue which must not only account for conduction but also heat addition or removal by blood supply and the metabolic heat generation.

The blood circulation around the tumor through capillaries is considered to be pulsatile flow and the flow is assumed as non linear, the physiological time in dimensionless form for the blood flow in the micro vessels is employed in the model. Also the variation of the average tumor temperature is evaluated in reference to blood perfusion.

Governing bio heat transfer equation is given by,

$$K\nabla^2 T + m_b C_b (T_b - T) + S = \rho \overline{C}\frac{\partial T}{\partial t} \tag{1}$$

$$K\frac{\partial T}{\partial \eta} = h(T - T_a) + LE \tag{2}$$

Each part of the human body is assumed to be spherical with variable thermal conductivity in biological tissues depending on the blood flow near the skin. Because the biological tissues generate heat uniformly supplied with blood at constant arterial temperature (i.e. convection by blood flow, vascular heat exchange and the tissue metabolism). For normal temperature distribution studies, the parameters T, m, T_b and S are all independent of time. However, for the situations like benign tumor, malignant tumors and some injuries, the parameters may be considered as time dependent to a smaller extent. Metabolic heat generation and blood mass flow rate are zero at epidermis and constant in subdermal layer. In order to understand the variation in the metabolic heat generation and blood mass flow rate of the three different layers, we study the bioheat equation with variable at dermal layers.

Transforming equations (1) and (2) into spherical polar coordinate system, considering in one –dimensional steady state,

$$\frac{1}{r^2}\frac{d}{dr}\left[K_i r^2 \frac{dT_i}{dr}\right] + M_{b_i} C_{b_i}(T_b - T_i) + S_i = 0$$

$$K_1 \frac{dT_1}{dr} = h_i [T_1 - T_a] + L_i E_i \tag{3}$$

$$\frac{1}{r^2}\frac{d}{dr}\left[K_i r^2 \frac{dT_i}{dr}\right] + Q_i(T_b - T_i) + S_i = 0$$

$$(4)$$

$$K_1 \frac{dT_1}{dr} = h[T_1 - T_a] + LE$$

where $Q_i = M_{b_i} C_{b_i}$, $T_3 = T_b$

For i = 1, the temperature is the same as blood temperature. By convection, radiation and evaporation heat loss takes place when epidermis is exposed to atmospheric temperature. Thermal conductivity is constant in d^E (thickness of epidermis), d^S (thickness of sub-dermal). Consider

$$K_3 \frac{dT_3}{dr} = \frac{d_2 k_3}{r}\frac{dT_2}{dr}$$

$$(5)$$

$$[T_1 = T_2]_{r = d^S},\quad [T_2 = T_3]_{r = d^D},\quad [T_3 = T_b]_{r = d^E}$$

$$(6)$$

$d^E = d_4 - d_3$, (epidermis ≈ 0.5cm), $d^D = d_3 - d_2$, (dermis ≈ 1.0cm) and $d^S = d_2 - d_1$, (subdermal ≈ 1.2cm)

In general, $T_i = T_b[1 - \xi_i]$ is taken as dimensionless quantity

i = 1, 2, 3 (epidermis, dermis, subdermis), we get

$$\frac{d}{dr}\left[K_1 r^2 \frac{d\xi_1}{dr}\right] = 0 \quad (\text{For } i = 1)$$

$$(7)$$

$$r^2 \frac{d^2\xi_2}{dr^2} + r\frac{d\xi_2}{dr} - r^2\left(\frac{m+s}{2K_1}\right)(\xi_2) = 0 \quad (\text{For } i = 2)$$

$$(8)$$

$$r\frac{d^2\xi_3}{dr^2} + 2\frac{d\xi_3}{dr} - r\left(\frac{m+s}{2K_1}\right)(\xi_3) = 0 \quad (\text{For } i = 3)$$

$$(9)$$

New boundary conditions are,

$$\frac{d\xi_1}{dr} = \frac{h\xi_1}{k_1} + \frac{h}{k_1}\xi_a + \frac{LE}{K_1 T_b} \quad (\text{subdermal region}, \ i = 3)$$

$$(10)$$

where $\xi_a = \dfrac{T_b - T_a}{T_b}$ (atmospheric temperature 15^{0}C to 33^{0}C)

$$K_1 \frac{d\xi_1}{dr} = \frac{d^D K_3}{r}\frac{d\xi_2}{dr} \quad (\text{dermis interface, } i = 2)$$

$$(11)$$

$$\frac{d^D K_3}{r}\frac{d\xi_2}{dr} = K_3 \frac{d\xi_3}{dr} \quad (\text{epidermis interface, } i = 1)$$

$$(12)$$

$$[\xi_1 = \xi_2]_{r = d^S},\ [\xi_2 = \xi_3]_{r = d^D},\ [\xi_3 = 0]_{r = d^E}$$

$$(13)$$

Analysis

Solving the differential equations (7), (8) and (9) using the conditions (10), (11), (12) and (13) by Bessel modified function, we obtain the solutions for ξ_1, ξ_2, ξ_3 (independent of time) as,

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$$\xi_1 = A_1\left(-\frac{1}{r}\right) + A_2 \tag{14}$$

$$\xi_2 = A_3 I_0(r\varepsilon) + A_4 K_0(r\varepsilon) \tag{15}$$

and

$$\xi_3 = A_5\left(\frac{e^{r\varepsilon}}{r}\right) + A_6\left(e^{-r\varepsilon}\right) \tag{16}$$

where,

$\varepsilon = \left[\dfrac{m+S}{SK_1}\right]^{\frac{1}{2}}$ and A_1, A_2, $A_{3,}$ A_4, A_5 and A_6 are the constants due to the solutions of second order differential equations (7), (8) and (9)

$I_0(r\varepsilon)$ and $I_1(r\varepsilon)$ are the modified Bessel's functions of the first kind, $K_0(r\varepsilon)$ and $K_1(r\varepsilon)$ are the modified Bessel's functions of the second kind. The constant in the Bessel's modified series is taken as 0.57721 for numerical computations of $K_0(r\varepsilon)$ and $K_1(r\varepsilon)$.

For epidermis, i = 1

$$\xi_1 = \frac{\gamma_1}{r}[\{\delta_1 K_0(r\varepsilon) + \delta_2 K_1(r\varepsilon)\}I_1(r\varepsilon) + K_1(r\varepsilon)\{\delta_2 K_1(r\varepsilon) + I_0(r\varepsilon)\}\delta_1 + \gamma_2\{\delta_3 + d_3\delta_4\}] \tag{17}$$

For dermis, i = 2

$$\xi_2 = \gamma_2[\{\delta_1 K_0(r\varepsilon) + \delta_2 K_1(r\varepsilon)\}I_0(r\varepsilon) - \{\delta_1 I_0(r\varepsilon) - \delta_2 K_1(r\varepsilon)\}K_0(r\varepsilon) \tag{18}$$

For sub dermal, i = 3

$$\xi_3 = \gamma_3[\{\delta_1\{K_0(r\varepsilon)I_1(r\varepsilon) + I_0(r\varepsilon)K_1(r\varepsilon)\} + \delta_2\{I_1(r\varepsilon)K_1(r\varepsilon) + (K_1(r\varepsilon))^2\}]\frac{e^{r\varepsilon}}{r}$$

$$+ \gamma_4[\delta_1\{K_0(r\varepsilon)I_1(r\varepsilon) + I_0(r\varepsilon)K_1(r\varepsilon)\} + \delta_2\{I_1(r\varepsilon)K_1(r\varepsilon) + (K_1(r\varepsilon))^2\}]\frac{e^{-r\varepsilon}}{r} \tag{19}$$

The study of C_{ring} shaped spherical tumor by quantifying the role of heat conduction, convection and metabolism is analyzed with reference to heat transfer distribution in tumors under the cases of elevated temperature in the dermal layers. Temperature rise in tumors is always due to variation in metabolism as a function of tumor weight (between 2 gm to 20 gm). The corresponding metabolic heat rate (between 0.0 to 5.2) and the temperature difference between (0.0185^0C to 0.24^0C) have been employed in the computations. By physiological point of view the temperature in the periphery of the necrotic tumor is higher than that in the central necrotic core. Taking the C_{ring} spherical shape of the tumor growth initially with radius 'a' and with time't' (constant time periods), the varying radius of the tumor is given by,

r = R (t)   (20)

Clearly

R (0) = a   (21)

The equations for the tumor consisting of pressure P and the nutrient concentration σ (by anaerobic)are,

$\nabla \rho^2 = S_v$ [Inside the surface of the tumor layer]   (22)

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Where S_v is the rate of volume loss (model does not include the motivated cell loss mechanism and shrinkage necrosis)

$$\nabla^2 \sigma = 0 \text{ [Outside the surface of the tumor layer]} \tag{23}$$

Taking the constant nutrient initially σ and attains maximum σ_m at r = R(t).

$$\sigma = \sigma_{max} \qquad at \mid R(t) \mid = \mid R \mid_{(max)} = \mid r \mid \tag{24}$$

$$\hat{n}.\nabla\sigma = \mu\sqrt{\sigma - \sigma_1} \quad at\, r = R(t) \tag{25}$$

Transforming equation (31) into spherical polar form

$$\frac{1}{r^2}\frac{\partial}{\partial r}\Big[r^2 \frac{\partial\sigma}{\partial r}\ \Big] \leq 0, \quad r \geq r(t) \tag{26}$$

Solving equation (28) for radius at initial I and at final F using equations (22), (23), (26), (27), we obtain

$$r_I = 2(D\rho)^{1/2}t\left[1 - \frac{1}{\rho t}\left(\log\left(4\pi Dt\frac{\partial}{N_0}\right)\right)\right]^{1/2} \tag{27}$$

$$r_F = 2(D\rho)^{1/2}t\left[1 - \frac{1}{\rho t}\left(\log\left(4\pi Dt\frac{\partial}{N_0}\right)\right)\right]^{1/2} \tag{28}$$

Since the tumor growth is considered to be spherical form from the necrotic core towards the outer layer (subdermal d^S to epidermis d^E), we define the tumor profile as

$$r_A = \frac{1}{2}[r_I + r_F]$$

Where r_A is the mean radius of r_I and r_F

$$r_A = \frac{1}{2}\Bigg\{\bigg\{2(D\rho)^{1/2}t\left[1 - \frac{1}{\rho t}\left(\log\left(4\pi Dt\frac{\partial}{N_0}\right)\right)\right]^{1/2}\bigg\}_I$$

$$+ \bigg\{2(D\rho)^{1/2}t\left[1 - \frac{1}{\rho t}\left(\log\left(4\pi Dt\frac{\partial}{N_0}\right)\right)\right]^{1/2}\bigg\}_F\Bigg\} \tag{29}$$

Then the mean radial distance of tumor formation from the necrotic core (origin) to the rupture size is modeled using Fisher-Kolmogoroff equation as,

$$r_m = \frac{r_I + r_F}{2} = \frac{\int_0^\infty r^2 c\,(r,t)dr}{\int_0^\infty r\,c\,(r,t)dr} = \int_0^\infty r^2 c\,(r,t)dr \tag{30}$$

Here $c(r,t) = 2\pi \int_0^\lambda C_{ring}\,(r,t,r_0)r_0 dr_0$ \hfill (31)

$$c(r,t) = e^{-r^2/4t}\left[1 - e^{-\lambda^2/4t}\right] + \frac{r^2}{4t}e^{-r^2/4t}\left[1 - e^{-\lambda^2/4t}\left(1 + \frac{\lambda^2}{4t}\right)\right] + \cdots \tag{32}$$

which is called the asymptotic approximation to various temperature distribution values.
By employing the expression for cell density in equation (30),

$$r_m = \left(1 - e^{-A\lambda^2}\right)\left(\frac{\sqrt{\pi}}{4A^{3/2}}\right) + \left(A - Ae^{-A\lambda^2} - A^2\lambda^2 e^{-A\lambda^2}\right)\left(\frac{3\sqrt{\pi}}{8A^{5/2}}\right) + \cdots \tag{33}$$

Neglecting second and higher terms,

$$r_m = \left(1 - e^{-A\lambda^2}\right)\left(\frac{\sqrt{\pi}}{4A^{3/2}}\right) \tag{34}$$

The temperature rise in the distribution among the three layers is due to metabolism as a function of tumor size, the tumor grows larger and it develops a central necrosis, (the temperature is maximum). Tumor cells are more sensitive to heat than normal cells. Introducing equations (27) and (28) into (19) under the assumption that the spherical tumor can develop adequate nourishment in the necrotic core , then for various values of ξ_3 in the radius range 0.25 - 1.05cms we obtain,

$$\xi_3(r) = \begin{cases} \xi_3(r_I), & r = 2.5cm \\ \xi_3(r_F), & r = 0.5cm \end{cases} \qquad (35)$$

$$\xi_2(r) = \begin{cases} \xi_2(r_I), & r = 2.5cm \\ \xi_2(r_F), & r = 0.5cm \end{cases} \qquad (36)$$

Where r_I modeled as

$$r_I = r_I^1 C_E \quad \text{Where} \quad C_E = h_E A_E (T_E - \overline{T}_{AE})$$

Then replacing r_I and r_F in ξ_3 , we compute the values of $\xi_3(r_I)$ and $\xi_3(r_F)$. The rise of temperature (nonlinearly in the distribution) in any location describes the progression of the tumor with certain initial radius. This helps in detecting the tumor in the field of thermography using hyperthermia for the minimum size of the tumor (initial stage) when associates with the rise of temperature of the core (i.e. in the subdermal region taken as ξ_3) .

Results and Discussion

Mathematical model with C_{ring} shaped spherical tumor predicts the relative metabolic heat generation in the three spherical layers. Numerical results influence the factors for the improvement on the detection of abnormalities in the temperature pattern in the dermal region. Asymptotic approximation and Numerical scheme suggest the growth of tumor in spherical form due to an increase of metabolic heat rate leads to threshold shift of 0.125^0 C. Results have shown that the irregularities between the temperature distributions in the range 1.0 to 1.6 cm at the skin surface will cause the elevation in the temperature of surrounding tumor. The study indicates the variation of C_{ring} shaped tumor due to the variation of temperature.

From the figures we observe that the temperature distribution for metabolic heat rate is at 0.029 cal/cm^3-min (Fig 1), 0.018 cal/cm^3-min (Fig 2), 0.018 cal/cm^3-min (Fig 3). Also Fig. (4) shows the increasing of metabolic heat rate in tumor. The difference in the temperature of the necrotic core in the tumor along the spherical layers (towards the decreasing radii of tumor layers) and the subdermal layers is between 0.127^0C – 0.288^0 C. Therefore the increasing temperature at the necrotic center may indicate the viable tumor in the human mammary epitheliomas and in the surrounding normal tissues.

Referring to Fig 5, we notice that C_{ring} shaped spherical tumor growth becomes nonlinear form of the layers in the necrotic core. Metabolic heat rate is slightly higher at the nonlinear locations by 0.128^0C to 0.287^0C. This is because of the development of the central necrotic core. Therefore due to glycolysis the vascular heat exchange appear to rise the temperature from 0.05^0C to 0.26^0C. This helps to detect the tumor by thermography using hyperthermia.
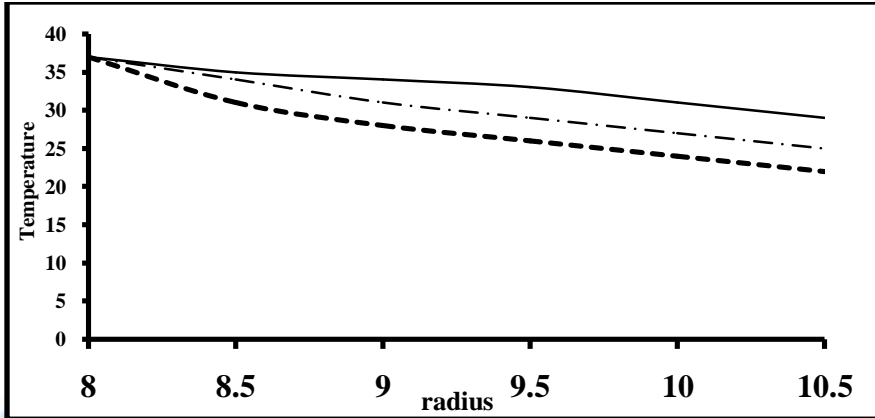
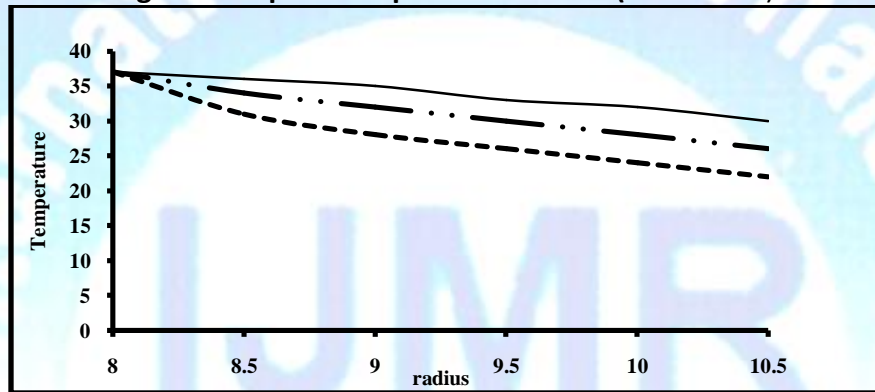**Fig.1   Temperature profile Vs radius (at T = 16^{0}C)**

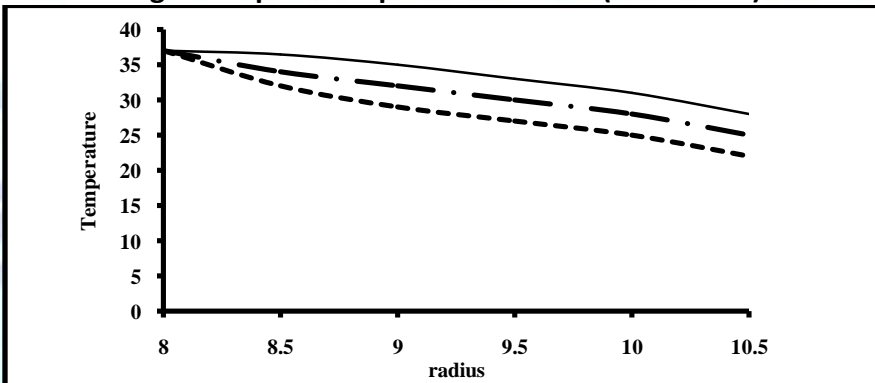**Fig.2 Temperature profile Vs radius (at T = 24^{0}C)**

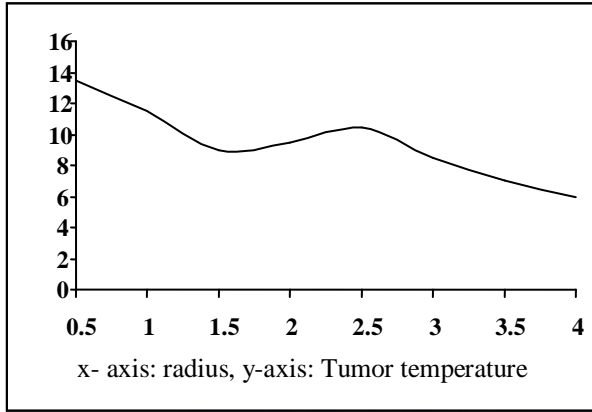**Fig 3   Temperature profile Vs radius (at T = 34^{0}C)**

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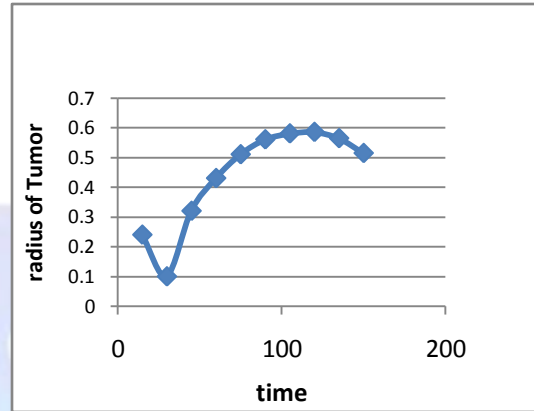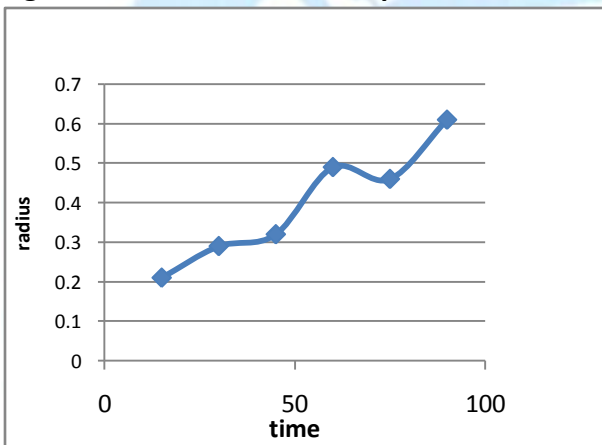**Fig4 radius   Vs Tumor temperature**

Fig 5   Time v/s radius of tumor

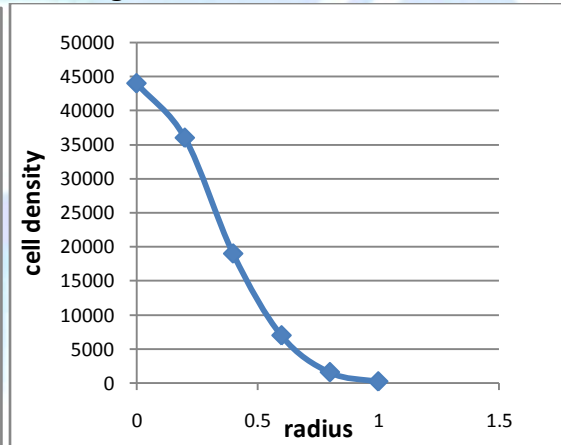**Time v/s mean radius of tumor**

Fig 6

Fig 7   Radius v/s Cell density(for t = 24 hrs)
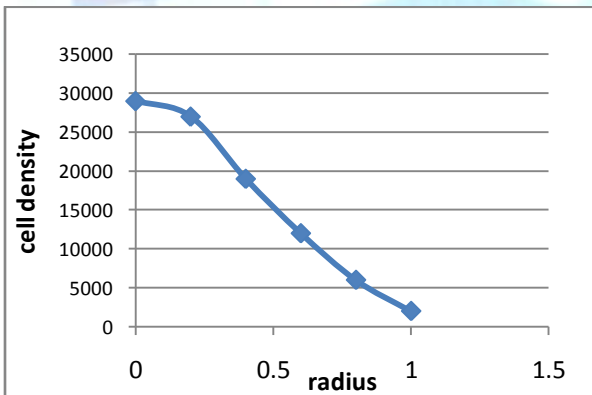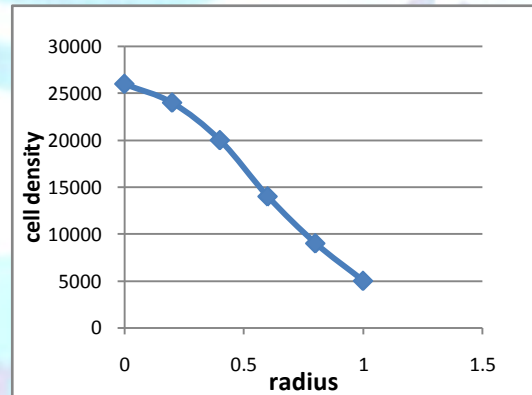

Fig 8 Radius v/s Cell density(for t = 48 hrs) Fig   9
   Radius v/s Cell density(for t =72 hrs)

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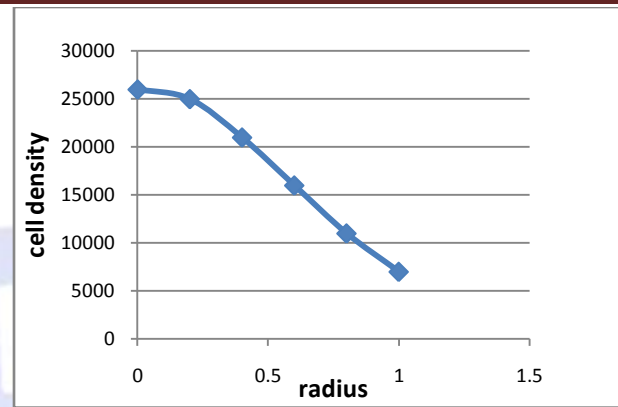http://www.ijmr.net.in email id- irjmss@gmail.com   Page 849

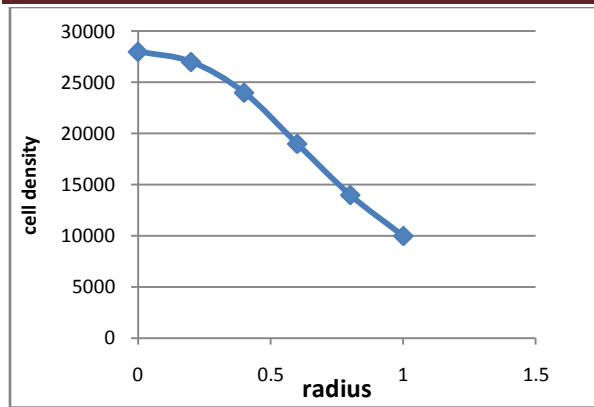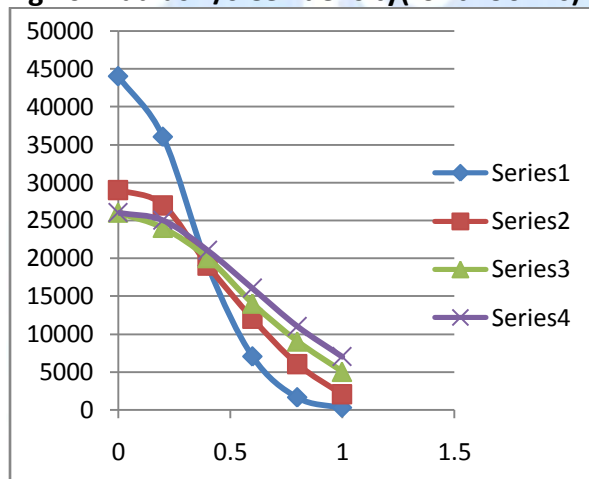**Fig 10   Radius v/s Cell density(for t =96 hrs) Fig 11 Radius v/s Cell density(for t =120 hrs)**

x-axis: radius, y-axis: cell density

Fig 12   Radius v/s Cell density

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NOMENCLATURE

T – temperature

k – thermal conductivity (cal/cm-sec)

m_b –blood flow rate (ml/cm^2-sec)

c_b – specific heat of blood (cal/gm- ^{0} C)

T_b – blood temperature

S – metabolic heat rate (cal/cm^2)

ρ - tissue density (gm/cm^2)

C – specific heat of tissue (cal/gm-^{0}C)

h – heat transfer coefficient

t – time

L – latent heat of evaporation

E – rate of evaporation

T_a – atmospheric temperature (^{0}C)

ξ - non dimensional temperature

d^E – thickness of epidermis layer (d_4-d_3)

d^D – thickness of dermis layer (d_3-d_2)

d^S – thickness of subdermal layer (d_2-d_1)

$Q_i = m_{bi}c_{bi}$

$$\xi_a = \frac{T_b - T_a}{T_b}$$

$$E = \left[\frac{m + S}{2k_1}\right]^{1/2}$$

a – radius of the tumor initially

R(t) = radius of the tumor at time t

δ - thickness of constriction

γ – sensitivity constant

r_I – initial radius

r_F – final radius

$I_0(r\varepsilon), I_1(r\varepsilon)$ - modified Bessel's functions of first kind

$K_0(r\varepsilon), K_1(r\varepsilon)$ - modified Bessel's functions of second kind

D – homogenous dissusion

C – cell density

N_0 – initial number of tumor cells at model time t = 0

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