

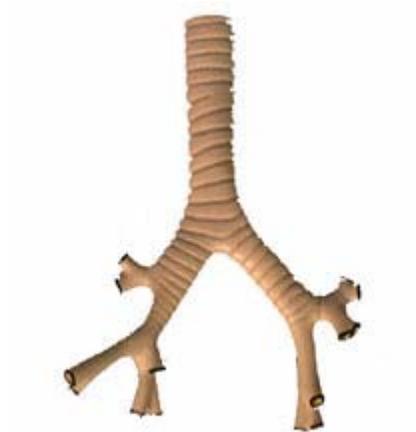
---

---

# TRACHEAL BURNING FROM HOT AIR INHALATION

---

---



Christina Cossell, Jan Ma, Samantha Spindel, Yang Wang

BEE 453: Computer Aided Engineering: Applications to Biomedical Processes  
Professor Ashim Datta  
Cornell University, Ithaca, NY  
May 4, 2007

---

---

## TABLE OF CONTENTS

---

---

Executive Summary.....	3
Introduction.....	3
➤ Background and Importance	
➤ Problem Objective	
➤ Problem Solution	
• Schematic	
• Assumptions	
Results and Discussion.....	7
➤ Results	
• Contour Plots	
• Time Effects	
• Temperature Effects	
• Sensitivity Analysis	
➤ Discussion	
• Comparison	
Conclusion and Design Recommendations.....	13
➤ Conclusions	
➤ Design Recommendations	
➤ Realistic Constraints	
Appendices.....	14
➤ Appendix A: Mathematical Model	
➤ Appendix B: Problem Statement	
➤ Appendix C: References and Acknowledgements	
• References	
• Acknowledgements	

---

---

## EXECUTIVE SUMMARY

---

---

Burns in the trachea from inhaling hot gases are a common occurrence and threaten the recovery of fire victims. Inhalation injury is also one of the most common causes of death, especially among children and the elderly. The thermal injury to the respiratory tract is usually limited to the upper respiratory tract, mainly the trachea. A better understanding of the interplay between transient temperature and injury distribution over the trachea may help to direct treatments in the future. Our goal is to model burns in tracheal tissue as a function of time, inhalation temperature, and inhalation velocity. The objective is to understand how variations in those variables affect tracheal injury. The velocity of air in the trachea varies as a function of time due to inspiration and expiration. As a result, the air temperature fluctuates in a cyclical manner. Since the burn concentration is a function of temperature, the extent of the burn rises as temperature increases with inspiration and remains constant as temperature decreases during expiration. Our model shows burn concentration is limited to the entrance to the trachea and the surface of the trachea.

---

---

## INTRODUCTION

---

---

### **Background and Importance**

In the year 2005, fire killed more people than all natural disasters combined. Over 3,790 deaths and 17,925 injuries occurred in the United States based on statistics from 2006. It is crucial that we study and develop a comprehensive understanding of the circumstances associated with various hazardous conditions and their consequences in order to develop more effective and timely treatments. We aim to comprehend and model the burn process of inhalation of hot air during a fire in the human trachea. We considered several parameters involved in this process including duration of burn and temperature of inhaled air.

The sources of fire vary from natural forest fires to arson to residential accidents, however, the impact on human health and safety are equally significant regardless of the circumstance. The two largest variations of fire are the temperature that the fire reaches depending on the material burnt (air, gasoline, chemical vapors, wood, or other insulation and building materials) and the duration of the exposure. The maximum temperature a human can incur before tissue damage is 45°C. We take into account the varying temperatures of a fire both comparable to and beyond minimal tissue damage temperature. The pattern and extent of the burn is greatly influenced by time.

Tracheal burn is due to two factors. There is heat conduction along the tracheal surface and convection from fluid flow of incoming air along the boundary of the tracheal lumen surface. In addition, there is species or mass transfer to depict actual burns the trachea sustains with the incoming hot air.

We also examined the velocity profile and the flow pattern of the inhaled air. Because the extent of burn is dependent on the velocity profile of the incoming air, we used the stored solution from the fluid flow model to solve for the species equation, which governs the burn

evaluation. The simultaneous modeling of both the air flow and the burn process yields a more accurate model of the inhalation of hot air and its effects on the trachea.

The thermal changes within the tracheal tissue do not merely depend on the inlet air temperature. The tissue is a living entity supplied by blood vessels, which play a role in heat exchange, and is also constantly carrying out metabolism. Thus, other factors taken into consideration when performing calculations are the metabolic rate of tissue, blood perfusion, and various arterial and metabolic effects.

## **Problem Objective**

This study models a normal human trachea with tissue properties derived from published literature. The inlet air temperature and the duration of burn were varied to examine the factors influencing the process. One objective is to determine how temperature varies with time due to the velocity profile of inspiration and expiration. Another goal is to determine the relationship between the extent of the burn and its dependence on the temperature of the incoming air. These objectives were accomplished via the modeling process described below.

First the design of the problem was formulated, creating the schematic and determining parameters. The trachea was modeled with cylindrical symmetry. For governing equations, the Navier Stokes fluid flow was used to model air flow the mass transfer equation was used to model the extent of burn. Initial conditions were set. The material and tissue properties were determined, including the biological tissue properties and the optimal temperature range. Finite element analysis was used to solve the problem. First the fluid flow equation was run, followed by the mass transfer, conduction, and convection equations using the stored solutions from the fluid flow equation. The inlet air temperature was varied and a sensitivity analysis was performed on the deep tissue temperature by measuring its effect on the final burn solution. A mesh convergence was performed in both the horizontal and vertical directions with number of elements plotted against the average tracheal temperature.

## **Problem Solution**



## **Report organization**

Our goal is to model the effects of temperature and time on tracheal burn in human subjects. The model development is presented in this section. In the Results and Discussion section, we present data obtained from our model, sensitivity analysis, as well as comparison of our results with those published in literature. In the Conclusion section, we will discuss the realistic constraints of our model and also make design recommendations to further improve the accuracy of the model.

---

---

## **RESULTS AND DISCUSSION**

---

---

# Results

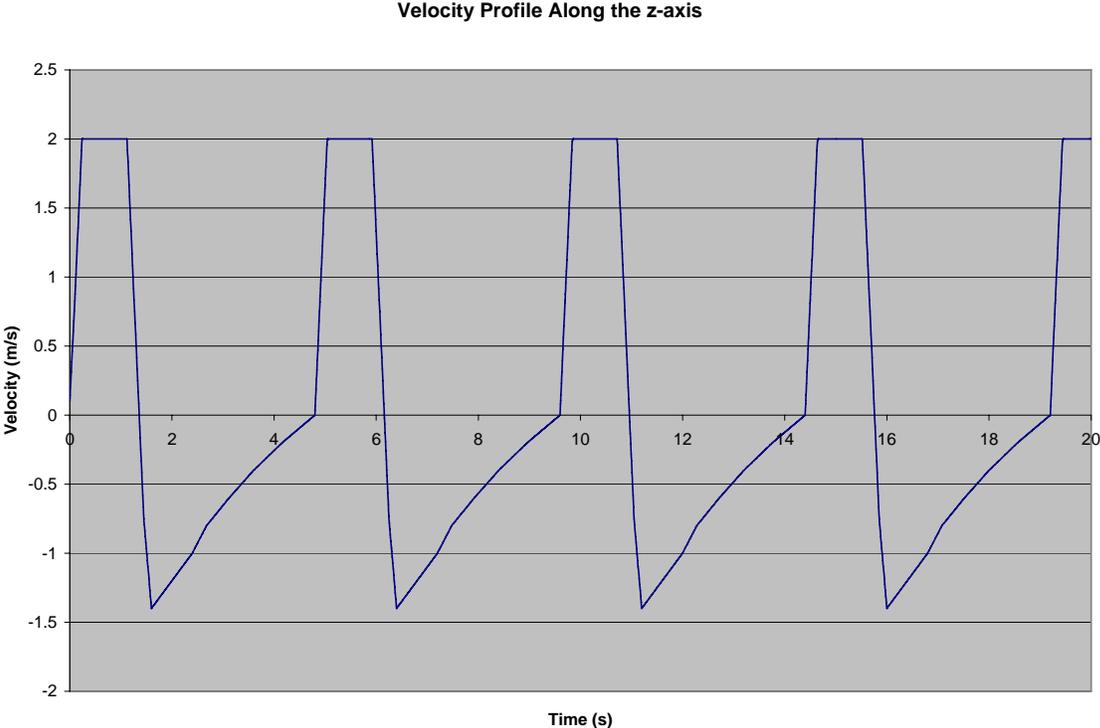
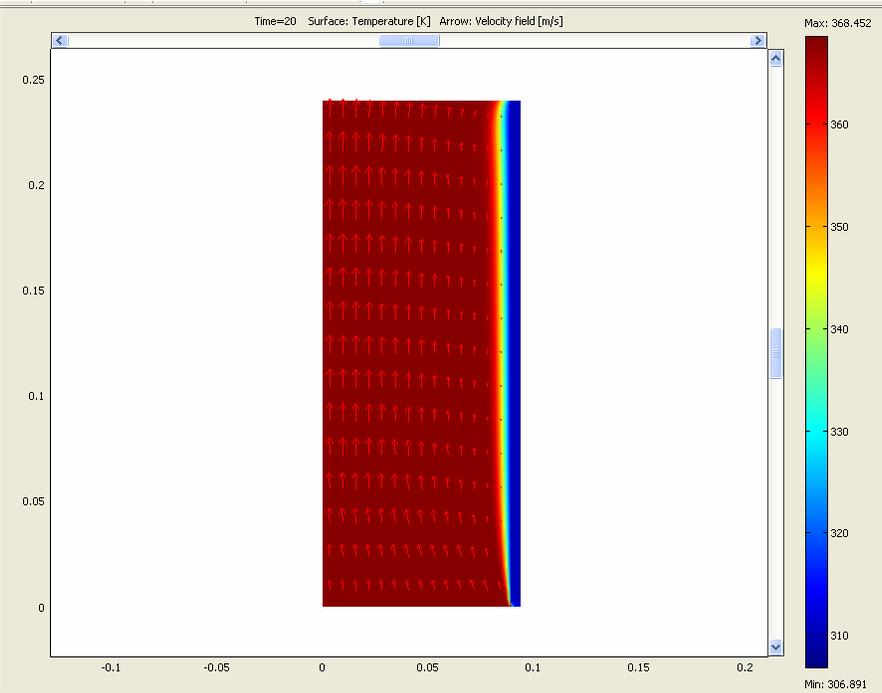
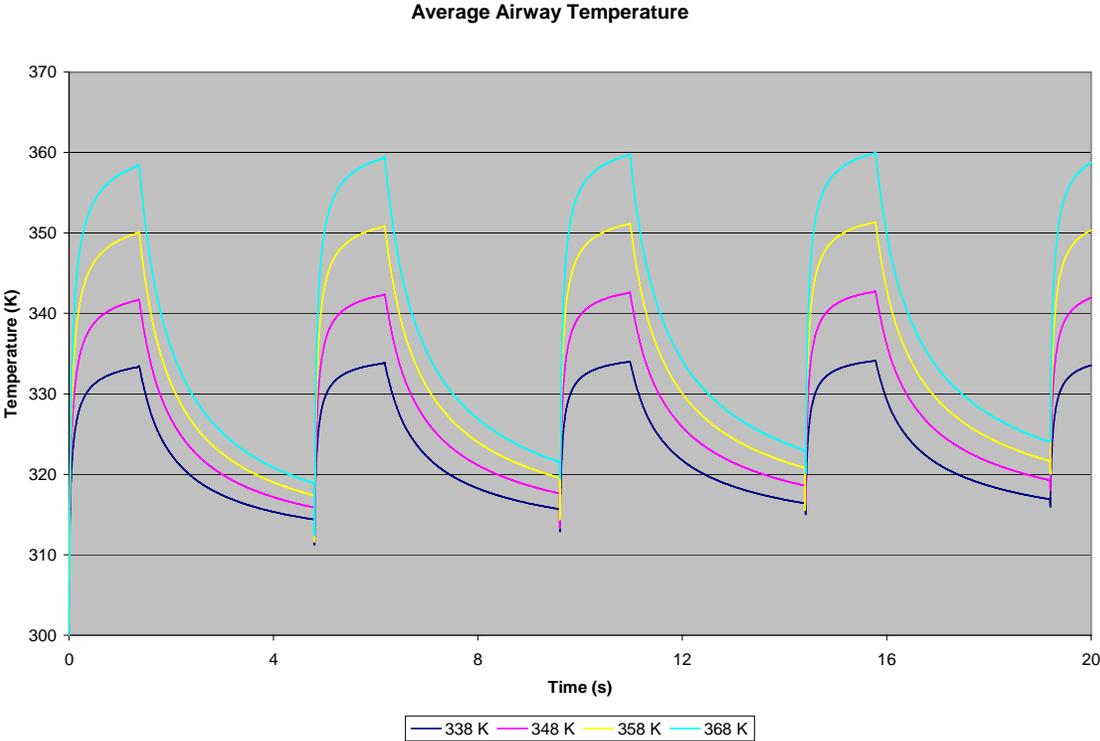


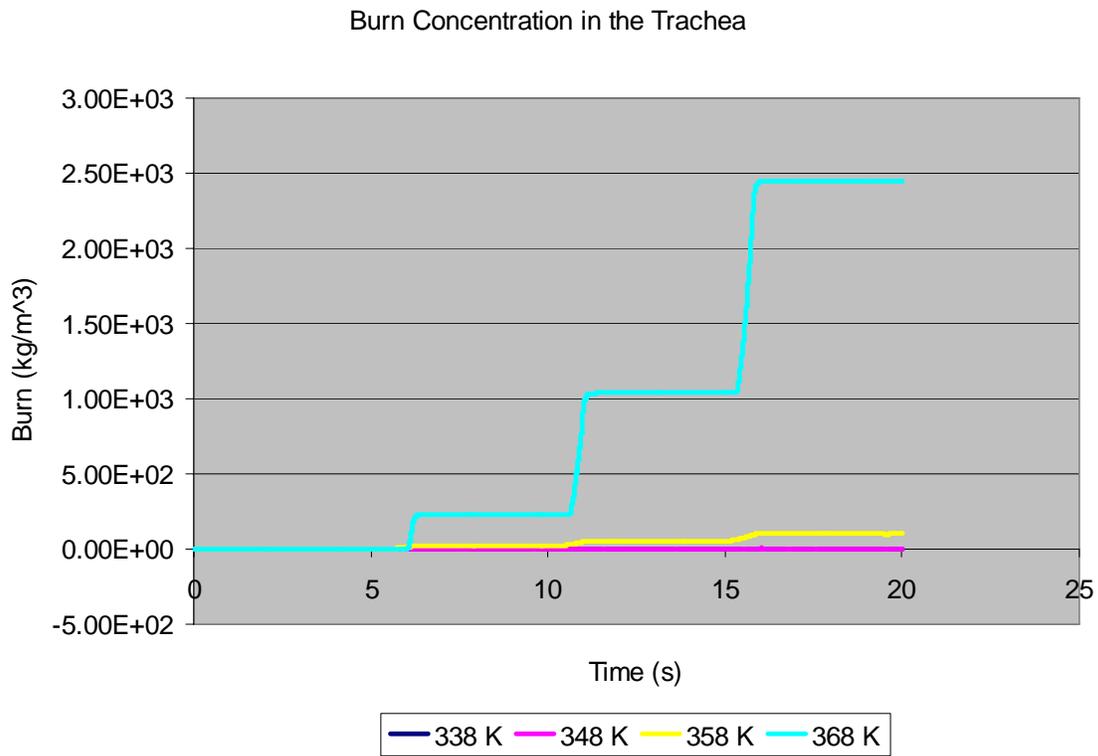
Fig. 2 Velocity profile in the airway.



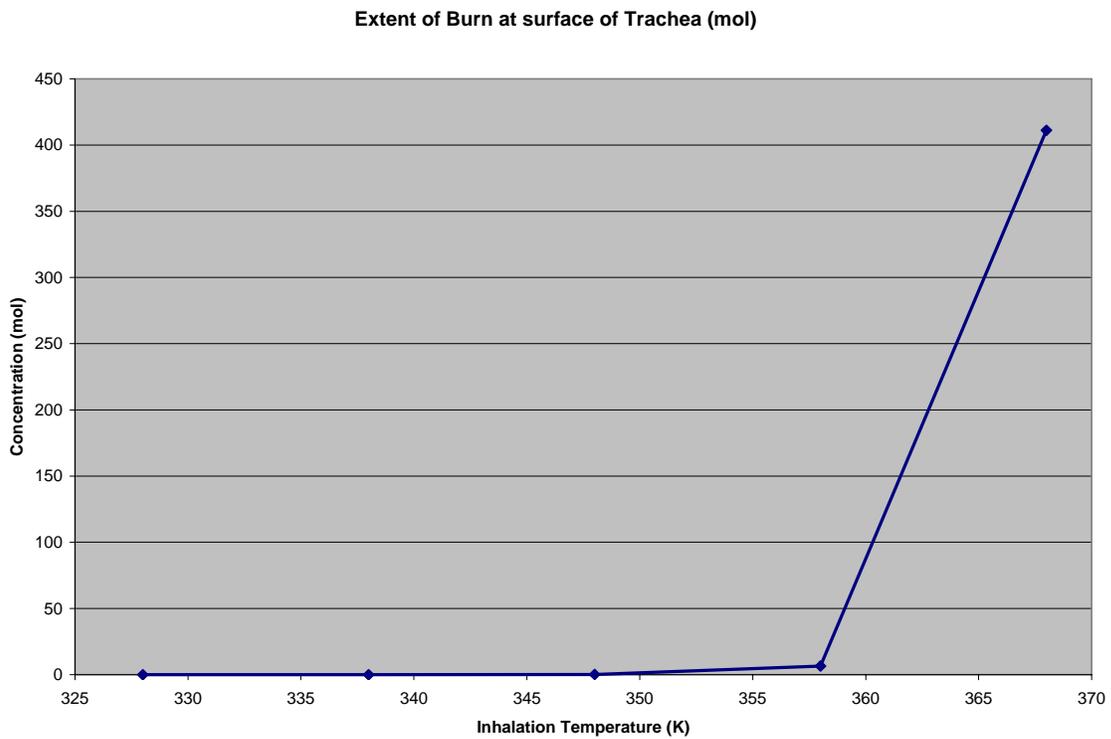
**Fig. 3** Contour plot at time 20s of surface temperature (K) throughout both the airway and trachea and the velocity field (m/s) in the airway.



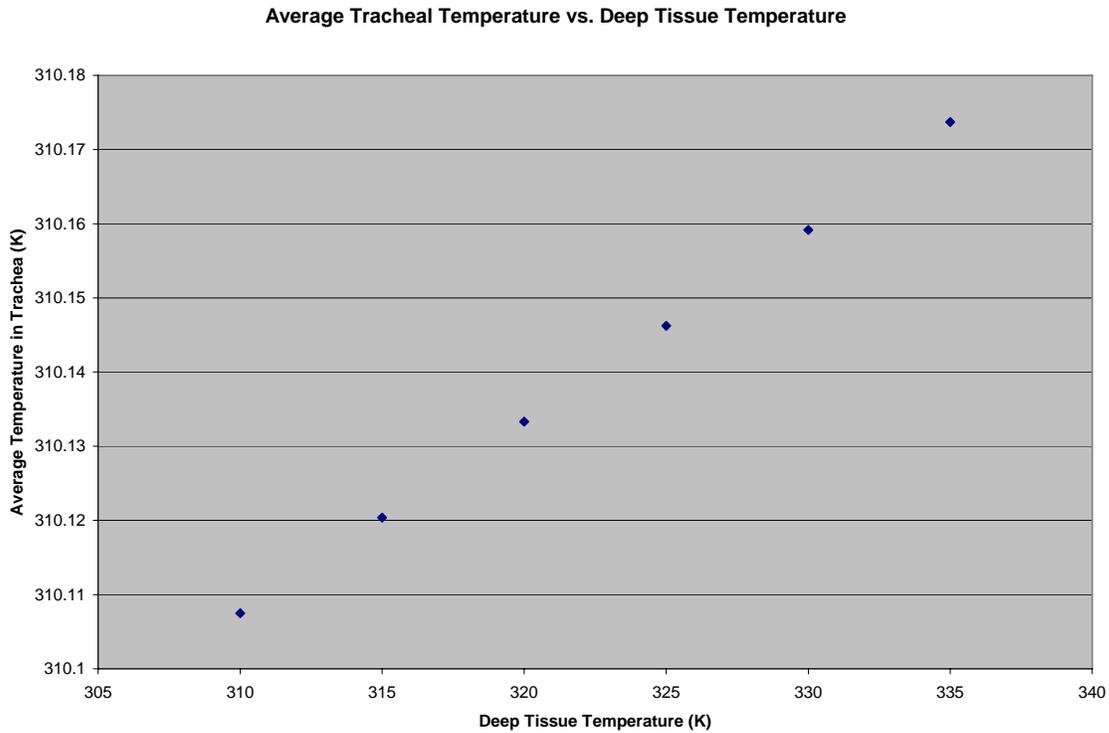
**Fig. 4** Average airway temperature versus time over a range of inhaled ambient hot air temperatures.



**Fig. 5** Burn concentration at the tracheal entrance over time with various initial hot air temperature



**Fig. 6** Burn Concentration at the tracheal surface for varying initial inhalation temperatures.



**Fig. 7** Average tracheal temperature plotted against various deep tissue temperatures.

### *Velocity Profile*

The airflow velocity is a continuous and steady state cycle of inspiration and expiration with a four-second cycle duration. The respiration cycle was included in the model to more accurately represent the physiological impacts during the burn (Fig. 2).

### *Contour Plots*

The temperature of the air in the airway cools as the air passes over the trachea and heat is transferred to the tracheal tissue (Fig. 3). Thus, when evaluating the burn concentration of the trachea, the burn was evaluated near the tracheal surface and by the entrance to the trachea rather than deep within the trachea. With a four second inspiration/expiration cycle, the air flow is directed inwards for inspiration at 20 seconds as shown by the arrows.

### *Time Effects*

The average airway temperature fluctuates in a cyclical manner corresponding to the inspiration/expiration cycle (Fig. 4). Initially, inspiration of hot air occurs, and the airway temperature rises rapidly. Then the temperature of the airway gradually decreases as the heat

is dissipated into the tracheal tissue and the lungs. This cycle repeats over time resulting in a steady increase in peak temperature in the airway. The cycle is similar with all of the various initial hot air temperatures (338 K, 348 K, 358 K, 368 K).

### *Temperature Effects*

As the temperature of the airway rises, the burn concentration also increases. Each inspiration/expiration cycle raises the burn concentration by a greater increment. The initial hot air temperatures of 338 K and 348 K caused no significant burn in the trachea (Fig. 6). The initial hot air temperature of 358 K caused some measurable burn concentration. An initial temperature of 368 K caused the largest cyclical increase in burn concentration corresponding to the inspiration/expiration cycles.

The most direct comparison we made was the effects of inhaled temperature on tracheal surface burn. The results show that there is a significant increase in the extent of burn starting at 358K. From 348K to 358K, we see the initial increase in tracheal surface burn. From 358K to 368K, the slope (indicating increase of burn per degree K) increases by a factor of 40.5 mol/K (Fig.7).

### *Sensitivity Analysis*

The choice of deep tissue temperature was not a parameter found through experiment or literature. Therefore, a sensitivity analysis was performed by varying the constant deep tissue temperature to see the overall effect on the model. With deep tissue temperatures ranging from 310 K to 335 K, there was less than 0.08 K change in the average tracheal temperature which is only a 0.0213% difference (Fig. 8). Therefore, the model is not sensitive to the deep tissue temperature parameter and a temperature of 320 K was chosen.

## **Discussion**

### *Comparison*

The results obtained by computer modeling in COMSOL matched those found in the literature. Comparison of exact numerical values to the literature is difficult because breathing cycles used in such papers vary from 3 to 4 seconds and the burn duration our model was capable of was a brief 20 seconds. However, the general trends obtained by modeling are in accordance with papers that have been published. Due to the periodic motion of air into and out of the trachea, the average temperature fluctuates in a cyclical manner. The temperature of the air in the airway cools as it enters the trachea due to the absorption of heat by the tissue. As a result, the greatest degree of burning occurs at the entrance of the trachea. The computer model shows the extent of the burn to be closely correlated with temperature. Consequently, degree of the burn experiences a periodic pattern; with each inspiration/expiration cycle, the burn concentration rises by a greater increment. These observations are consistent with published papers.

One unexpected result is that the initial hot air temperatures of 338 K and 348 K caused no significant burn in the trachea although these temperatures are above the temperature at which cell death occurs (318K). Perhaps because modeling was limited to such a short time period (20 s), the degree of burn appeared insignificant. Since the magnitude of a burn depends on both temperature and time, a burn of the same degree could occur at a hotter temperature and shorter time or at a lower temperature and longer time. This presumed result could not be proved with the limited capacity of the model, which is further discussed in the Realistic Constraints section.

---

---

## CONCLUSION AND DESIGN RECOMMENDATIONS

---

---

### **Conclusions**

We were able to obtain the results that were addressed in the objectives of this study. The goals of this project were to model the effects and extent of burn in a human trachea by varying the time and the initial inhalation temperature (as a function of fire temperature). We concluded that significant burn starts at around an inhalation air temperature of 358K (85°C) over a period of 20 seconds. A person can ideally inhale air under 358K for 20 seconds without sustaining significant tracheal tissue damage. From 358K to 368K, the burn increases dramatically at 40.464 mol/K. For every degree K increase, there is a 40.464 mol increase in the extent of burn of the tracheal tissue. A person inhaling temperatures over 358K over a period of at least 20 seconds will sustain tracheal tissue damage

### **Design Recommendations**

The trachea was simplified to a cylindrical shape, but more complex geometrical and physiological parameters could be incorporated for a more comprehensive prediction. For example, the trachea is made up of different tissues like smooth muscle and cartilage, which have different material properties. A better computer model would incorporate different material properties and also take into account the long exposure periods and environmental conditions.

### **Realistic Constraints**

One economic constraint is that computers with greater memory capacity were unavailable for use and too expensive to obtain. We looked at tracheal burns for fairly brief time periods (20 s) since our model reached the maximum memory capacity of the computers. Although we compensated for the shorter time period by increasing inhalation temperatures to slightly above normal range, we could not perfectly model what occurs in the trachea during a fire. We would have preferred to run the simulation for a longer time period, reflective of how long a person might be trapped in a building in a real fire (several minutes). However, due to the limited storage capability of available computers, modeling beyond this limit was not possible. Although a reasonable assumption is that the extent of the burn will increase with increasing time, a definitive conclusion about the trend of the burn beyond 20 s could not be obtained.

Another constraint is due to ethical and moral considerations associated with health and safety. Experiments cannot be conducted to determine the extent of tracheal burns on real human subjects. Consequently, the model could not be checked against experimental data for accuracy.

Environmental constraints of the model include not taking into account the presence of particulate matter in the air. In addition, the hot gas in the model was assumed to be air (a combination of oxygen and nitrogen), but in a real fire, additional gasses will form depending upon what type of material is burning and what carcinogens are released. Certain gasses may burn the trachea quicker than others and some gasses may react with itself or upon contact with the trachea, causing further deterioration of tissue. Also, the model does not include the effects of densely hot wet air, such as water vapor, where phase change heat transfer must be considered.



**Heat Equation:**

$$\frac{1}{\alpha} \frac{\partial T}{\partial t} = \frac{\partial^2 T}{\partial r^2} + \frac{1}{r} \frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} + \frac{W_b C_b}{K} (T_a - T) + \frac{Q_m}{K}$$

The last two terms represent the heat source.

**Burn Evaluation (modeled with Transient Diffusion Equation):**

$$\Omega = \int P \exp\left(\frac{\Delta E}{RT}\right) dt$$

**Incompressible Navier Stokes Equation:**

$$\rho \frac{D\mathbf{v}}{Dt} = \nabla \cdot \mathbb{P} + \rho \mathbf{f}$$

*Boundary Conditions*

As applied to the heat equation, boundary 1 has axial symmetry. Boundary 2 has the temperature function of  $368*(v \geq 0) + T*(v < 0)$  with 368 representing the hot air temperature during inhalation with air flow velocity greater than zero. Boundary 3 has the temperature function of  $T*(v \geq 0) + 320*(v < 0)$ , which indicates that at the end of the trachea (assumed to be deep tissue), the temperature remains constant at 320K throughout the burn process. Heat flux is zero at boundaries 5, 6, 7.

As applied to the species equation, there is only mass transfer at boundaries 4, 5, 6, and 7. At the air-trachea interface, the flux is zero at boundary 4; the flux is also zero at boundaries 5, 6, and 7. The burn is limited to within the tracheal tissue.

As applied to the incompressible Navier Stokes equation, boundary 1 has axial symmetry. Boundary 2 has inflow velocity of  $u=0$  and  $v$ =function that models the respiration cycle. Boundary 3 has velocity of  $u$  and  $v$ , which are solved in the model. At boundary 4, a no-slip condition is applied.

**Transient Convection and Conduction:**

*In the air:*

- 1- Axial Symmetry
- 2- Temperature:  $368*(v \geq 0) + T*(v < 0)$
- 3- Temperature:  $T*(v \geq 0) + 320*(v < 0)$

*In the trachea:*

- 5- Heat Flux:  $Q = 0$
- 6- Heat Flux:  $Q = 0$
- 7- Heat Flux:  $Q = 0$

**Transient Diffusion Equation:**

*At the air/trachea interface:*

4- Flux:  $Q=0$

*In the trachea:*

5- Flux:  $Q=0$

6- Flux:  $Q=0$

7- Flux:  $Q=0$

**Incompressible Navier Stokes Equation:**

*In the air:*

1- Axial Symmetry

2- Inflow Velocity:  $u = 0$ ,  $v = \text{function}$

3- Inflow/Outflow Velocity:  $u = u$ ,  $v = v$

*Boundary between air and trachea:*

4- No-slip Condition

**Initial Conditions**

The initial trachea temperature was assumed to be constant at 310K (body temperature) with no initial burn:  $\Omega=0$ . All velocities in were assumed to be zero:  $u = 0$ ,  $v = 0$ .

**Transient Convection and Conduction:**

*In the air:*

Temperature = 310K

*In the trachea:*

Temperature = 310K

**Transient Diffusion Equation:**

$\Omega=0$

**Incompressible Navier Stokes Equation:**

*In the air:*

$u = 0$

$v = 0$

### Input Parameters

<i>Variable</i>	<i>Value</i>	<i>Description</i>
$C_b$	4000 J/kg°C	Specific Heat of blood <sub>1</sub>
$C_a$	1005	Specific Heat of air <sub>1</sub>
$T_a$	310 °C	Artery Temperature <sub>1</sub>
$Q_m$	420 W/m <sup>3</sup>	Metabolic Rate of Tissue <sub>1</sub>
$K_t$	0.5 W/m°C	Thermal Conductivity of Tissue <sub>1</sub>
$K_a$	0.025 W/m°C	Thermal Conductivity of Air <sub>4</sub>
$W_b$	0.5 kg/m <sup>3</sup> s	Tissue Blood Perfusion <sub>1</sub>
$\rho_a$	1.205 kg/m <sup>3</sup>	Density of air <sub>4</sub>
$\rho$	1000 kg/m <sup>3</sup>	Density of tissue <sub>1</sub>
$P$	$3.1 \times 10^{98}$ 1/s	Henriques' Constant <sub>1</sub>
$\Delta E$	$6.27 \times 10^5$ J/mol	Activation Energy <sub>1</sub>
$R$	8.31447 kJ/kmolK	Universal Gas Constant <sub>3</sub>
$\eta$	0.00002 Pas	Dynamic Viscosity <sub>4</sub>
$D$	$1 \times 10^{-18}$ m <sup>2</sup> /s	Diffusion Coefficient <sub>2</sub>

**Table 1** Input parameters

Variable sources:

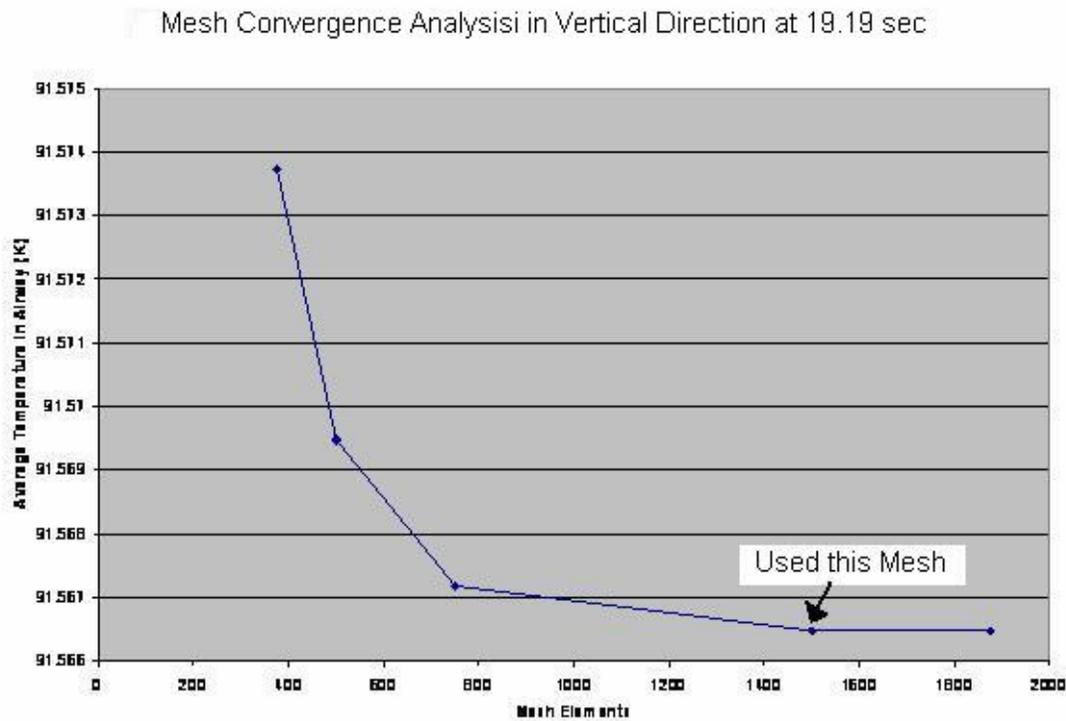
1 - Yong-Gang Lv *et al.* Theoretical evaluation of burns to the human respiratory tract due to inhalation of hot gas in the early stage of fires. *Burns*, 32 (2006) 436-446.

2 - Advised by Vineet Rakesh to choose this value.

3 - Common knowledge

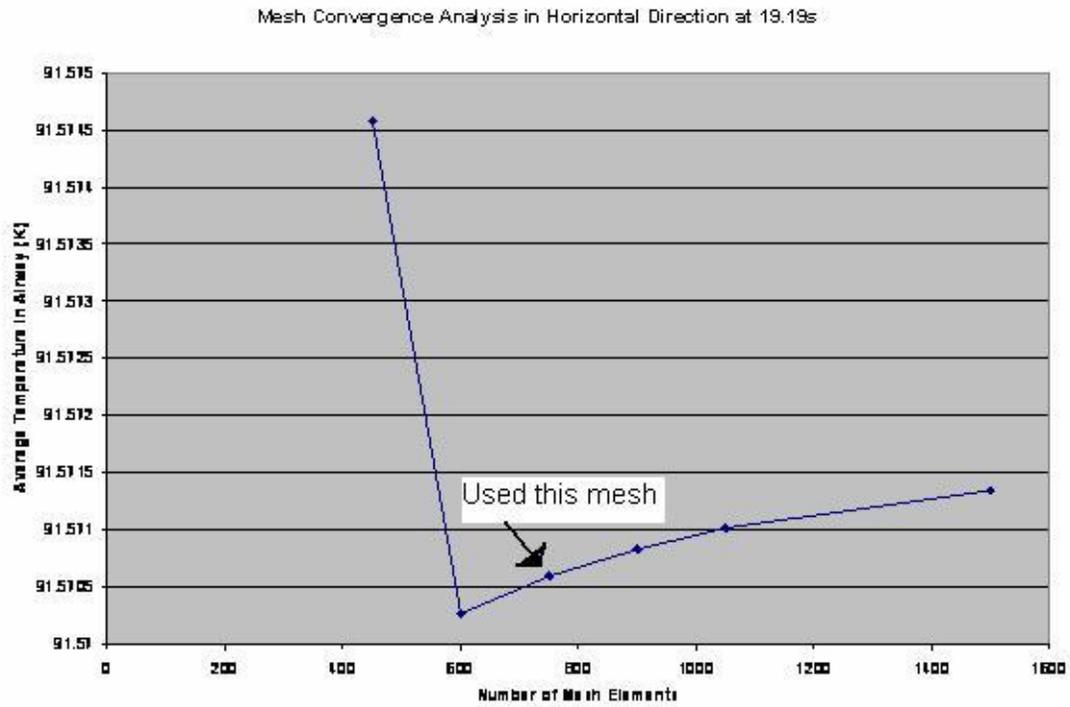
4 - Young, D.F., *et al.* A Brief Introduction to Fluid Mechanics. 3<sup>rd</sup> Edition. (2004).

## Appendix B: Mesh Convergence

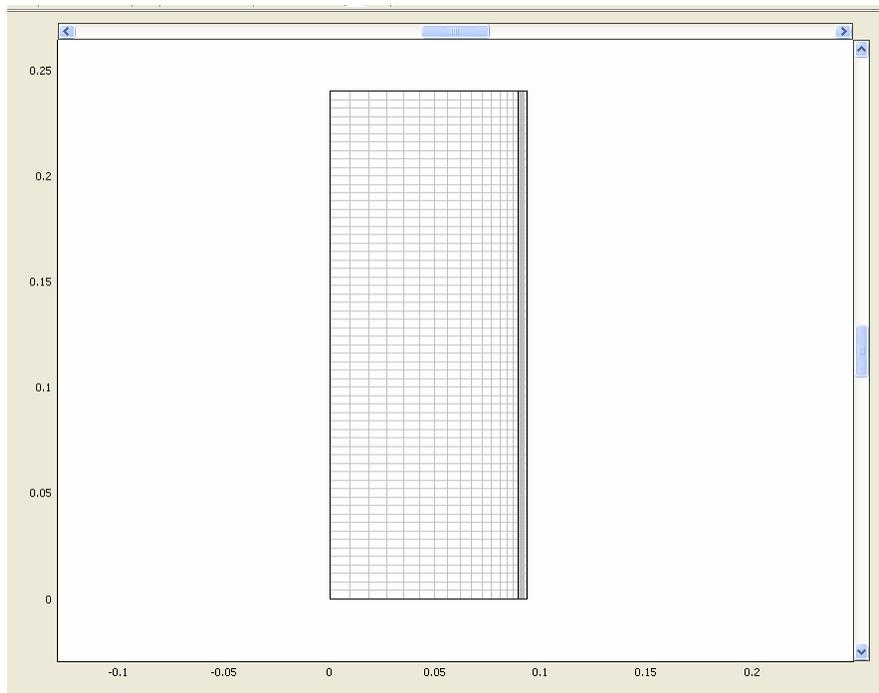


**Fig. 8** Average temperature in the airway plotted against the number of mesh elements at 19.19s for a mesh convergence analysis in the horizontal direction of the model.

The mesh convergence analysis was performed by plotting the average temperature in the airway versus the number of mesh elements. A mesh convergence was performed in both the horizontal direction for the airway only and the vertical direction for the airway and trachea. In the horizontal direction, the average airway temperature did not converge to one temperature but continued to vary slightly as the number of mesh elements increased (Fig. 8). There was only 0.001176% difference between the average temperature at 600 mesh elements and 1500 mesh elements. A total number of 750 mesh elements corresponding to 15 nodes in the horizontal direction of the airway was chosen. In the vertical direction, 1500 mesh elements were chosen, which corresponds to 60 nodes.



**Fig. 9** Average temperature in the airway plotted against the number of mesh elements at 19.19s for a mesh convergence analysis in the vertical direction of the model.



**Fig. 10** Final mesh used for modeling

## **Appendix C: References and Acknowledgements**

### *References*

- Datta, A.K., Computer-aided Enigneering: applications to biomedical processes. BEE/MAE 453 Lectures. Spring 2007.
- Daviskas, E. *et al.* Mathematical modeling of heat and water transport in human respiratory tract. J. Appl. Physiol. 69(1), (1990) 362-372.
- Ferguson, J.C. *et al.* Burn wound evaporation - an evaluation of air diffusion resistances governing heat transfer in a clean air unit. Clin, Phys. Physiol. Meas., Vol. 10, No. 4 (1989) 319-330.
- Gardnert, G.G., *et al.* The mathematical modelling of thermal responses of normal subjects and burned patients. Physiol. Meas. 15 (1994) 381-400.
- National Fire Protection Association Fire Loss in the U.S. During 2005 and USFA's Firefighter Fatalities in the United States in 2005.
- Yong-Gang Lv *et al.* Theoretical evaluation of burns to the human respiratory tract due to inhalation of hot gas in the early stage of fires. Burns, 32 (2006) 436-446.
- Young, D.F., *et al.* A Brief Introduction to Fluid Mechanics. 3<sup>rd</sup> Edition. (2004).

### *Acknowledgments*

Professor Ashim Datta  
Vineet Rakesh