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CFD Modeling of Turbulent Flow and Particle Deposition in Human Lungs

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Abstract— **Understanding transport and deposition of inhaled particles in the human airways plays a crucial role in the targeted therapy of pulmonary diseases, and the administration of inhaled medicines. Numerous researchers have studied the inhalation of particles using experiments or computer models. Even though experiments have shown that the airflow in the trachea and the upper branches of the lung is turbulent, the flow is taken to be laminar in most computer models. Only few recently published papers have looked at the turbulent transport of air in the human airways. Even fewer results have been published on the effect of the upper airway structures on the turbulent airflow in the lungs or on the effect of the turbulence on particle deposition. The previously published turbulent models have also mainly used RANS methods to predict the flow. To study the unsteady flow and particle deposition in a human lung, an LES model with a dynamic Smagorinsky subgrid scale model was used. The model equations were solved to study steady inspirational flow at different flow rates for different particle sizes. Results indicate that the upper airway geometry produces turbulence in the flow and the deposition of particles is mainly affected by the particle size and Stokes number.**

I. Introduction

Solid particles like pollen, dust, and particulate pollutants, and liquid particles like aerosolized fuel are constantly inhaled and deposited in the airways. Some medicines like corticosteroids used to control asthma are also nebulized and administered as inhaled particles. Usmani et al. [1] have shown that only 10 to 20% of the inhaled drug reaches the lungs. Experimental studies by Schlesinger et al. [2] using casts of the human airways show a high correlation between regions of high particle deposition and primary origin sites of bronchial carcinoma. Therefore, it is crucial to understand the factors that affect particle deposition in the lungs in order to improve drug delivery mechanisms for targeted therapy of pulmonary diseases. Studying the transport and deposition of inhaled particles in the human airways plays a crucial role in the administration of nebulized medicines [1], and understanding the development of diseases caused by inhaled particles [2], [3]. It is a current research field of interest to numerous researchers who have investigated the inhalation of particles in the human airways using experiments or numerical simulations.

Cheng et al. [4] used casts of human airways to study flow and particle deposition. Instead, Caro et al. [5] constructed ideal models of the human lungs for their experiments. In both cases, experiments revealed that the airflow in the trachea and upper bronchi is turbulent. Most computer models have so far assumed the airflow to be laminar. The analysis of the airflow is restricted to the lower branches below the third generation where the flow is laminar because of their small diameters [6], [7] to justify this assumption. A few recent papers have looked at turbulent transport of air and particles in the human airways.

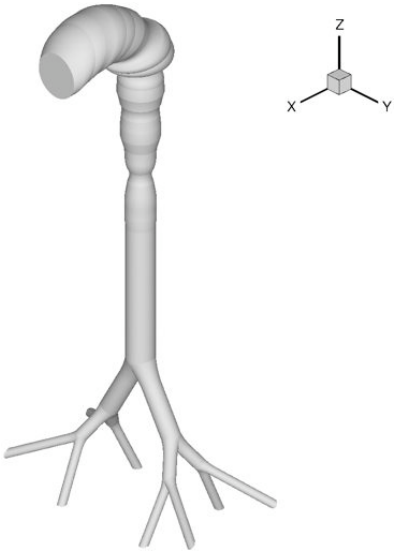

Fig. 1. Model of the human mouth, throat, and airways used to study particle deposition.

The k-ω RANS model has been recenly used by Zhang et al. [8] and other researchers to study turbulent airflow and particle deposition in the airways. Recently, LES has been used by a few researchers [9], [10] to study turbulent flow and particle deposition in the lungs, but they did not consider the effect of the upper airway features. Lin et al. [11] have recently used DNS to study steady inspirational flow in the airways but they did not consider particle deposition in the airways. A parallel finite-volume method-based solver based on LES is used to study turbulent flow and particle deposition in the upper airways and lungs in the work reported here.

II. Method

A. Lung Model

The geometry of the airways was modeled using two sources. The upper airway geometry was modeled using the cross-sectional areas measured from a cast of the human thorax [4]. The cross-sectional areas were recast as diameters

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which were used to model the geometry of the oral cavity and the upper airway up to the trachea as a pipe with circular cross-sections of varying diameter. The trachea and the branches were modeled as a symmetrically bifurcating series of pipes based on the regular dichotomy model, Weibel's [12] Model A. The axis of the daughter branches were offset by 30° from the axis of the originating branch, and each branch was rotated from the plane of the previous branch by 90° to generate an out-of-plane symmetric lung model. The last level of branches were terminated in a common reservoir that is used to represent the lower bronchi, alveoli, and other structures in the lung not considered in the model. The solutions of the flow model in this reservoir are ignored during the analysis of results. The final geometry used in the LES computations is shown in Fig. 1. This geometry was meshed using approximately three million control volumes. To test the resolution of the mesh, the LES solutions were compared with DNS solutions computed on the same mesh. The maximum difference in the turbulent kinetic energy estimates was less than 7 % which indicates that the mesh was fine enough for well-resolved LES.

B. Flow Solver Details

The flow of air in the lungs is described by the Navier-Stokes equation and the mass continuity equation:

$$\frac{\partial \mathbf{v}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v} = -\frac{1}{\rho}\nabla p + \mathbf{g} + \nu \nabla \cdot (\nabla \mathbf{v} + \nabla \mathbf{v}^T) \quad (1)$$

$$\nabla \cdot \mathbf{v} = 0 \quad (2)$$

where $\mathbf{v}$ is the air velocity, p is the local pressure, ρ is the density of air, taken to be 1.2 kg/m^3, and ν is the kinematic viscosity of air taken to be 1.5×10^{-5}m^2/s. An LES model with a dynamic Smagorinsky sub-grid scale (SGS) model is used to build a finite-volume method based solver which is used to solve the model equations in the lung geometry to study steady inspirational flow at flow rates of 30 l/min and 60 l/min. The dynamic Smagorinsky SGS model was chosen because it does not produce spurious results even when the flow becomes transitional or laminar. This is very critical in the study of flow in the lungs where the flow transitions from laminar to turbulent not only in time with inhalation and exhalation but also in space as the bronchi become smaller at the lower levels.

C. Particle Transport Model Details

The particles in the lung are assumed to be point particles, i.e. it has a zero radius. One-way coupling between the flow and the particles is also assumed, i.e. the particles are aware of the flow and the particle properties are affected by the fluid properties but the fluid properties are not affected by particle properties. The particle diameters studied are 1 μm, 5 μm, and 10 μm.

D. Boundary Conditions

At the mouth, a uniform velocity profile was used as the inlet boundary condition. At the outlet of the reservoir, a penalty boundary condition is applied to ensure that the net mass within the geometry is conserved. The incompressibility condition is enforced using a pressure-Poisson equation. The time integration is done using a constant time-step size of 10^{-5} seconds. Ten particles are introduced at the mouth with zero velocity at every time-step which results in a million particles per second. The solutions of the LES model for the volumetric flow rate of 60 l/min and 1 μm, 5 μm, and 10 μm diameter particles are reported below.

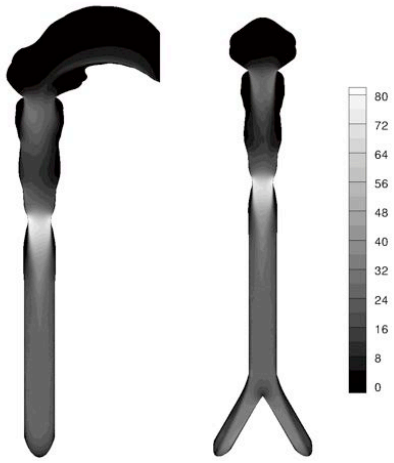

Fig. 2. Distribution of mean kinetic energy within the upper parts of the airways.

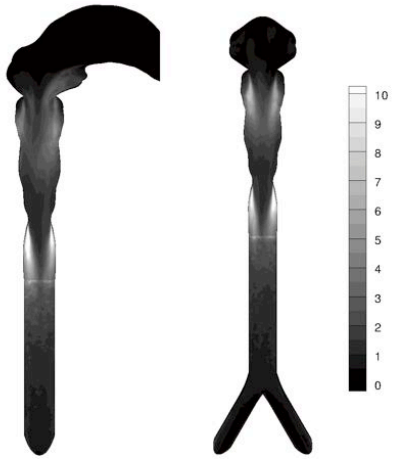

Fig. 3. Distribution of turbulent kinetic energy within the upper parts of the airways.

III. Results and Discussion

A. Flow Characterstics

Computational results showed that the constrictions, expansions, and other structures in the upper airway produced turbulence in the airflow in the lungs. Eddies and structures produced in the upper airway are carried into the lower branches of the lungs.

The mean kinetic energy (MKE) of the flow is defined as $\sqrt{\bar{u}^2 + \bar{v}^2 + \bar{w}^2}$ where $\bar{u}$, $\bar{v}$, and $\bar{w}$ are the time-averaged x-, y-, and z-directional velocity components. The turbulent kinetic energy (TKE) of the flow is defined as

$\sqrt{u'^2 + v'^2 + w'^2}$ where u', v', and w' are the root mean square of the velocity components along the three cartesian directions. The MKE of the flow in the upper airway is Fig. 2. Regions with high MKE in Figure 2 are the regions with the fastest flow, in this case, the epiglottis and larynx which are the first and second constrictions in the figure. In Fig. 2, regions with low MKE close to the bronchi walls near the bifurcation point indicate that the flow is separating from the walls there. A stagnation point is also visible at the ridge of the bifurcation point, as indicated by the low MKE there. Fig. 3 plots the turbulent kinetic energy of the flow. Regions with high TKE are regions where the velocity is fluctuating rapidly, and the turbulence is the highest. The regions with the highest TKE are the regions immediately downstream of the epiglottis and the larynx.

Fig. 4(a) shows the iso-surface where the average velocity magnitude is 37.5% of the maximum, and Fig. 4(b) shows the iso-surface where the average velocity magnitude is 25% of the maximum. The iso-surface in Fig. 4(a) shows the tracheal jet which starts at the larynx and breaks up as it nears the first bifurcation. This jet was also seen in direct numerical simulations of airflow in the lungs [11]. The iso-surface in Fig. 4(b) shows the jet that starts from the epiglottis and then strikes the dorsal wall of the glottis, the area between the epiglottis and the larynx. This epiglottal jet was also seen in previously reported experimental results [13]. The epiglottal jet can also be seen in the MKE distribution in Fig. 2.

B. Particle Deposition

The distribution of 1 μm, 5 μm, and 10 μm diameter particles in the mouth and throat are shown in Fig. 5. The 1 μm particles which have the least inertia are sensitive to even the smallest scales of turbulence and are distributed almost uniformly, as seen in Fig. 5(a). The 10 μm particles have the most inertia and tend to follow the mean flow, as seen in Fig. 5(c). The 10 μm particles are seen to be concentrated close to the dorsal wall of the glottis because of the epiglottal jet seen in Figure 4(b). The 5 μm particles in Fig. 5(b) are dispersed more evenly than the 10 μm particles but not as evenly as the 1 μm particles.

Figure 6 shows the deposition pattern of the 1 μm, 5 μm, and 10 μm diameter particles at the first three bifurcation levels. Because most deposition occurs by inertial impaction, the particles are concentrated near the bifurcations. Larger quantities of the bigger particles are deposited compared to the smaller particles, which is also observed in experimental and other numerical results. Most of the smaller particles are swept in to the lower levels of the lungs where they get deposited. A small number of particles get trapped in the recirculation downstream of the larynx, and deposited on the tracheal wall close to the larynx. As seen in Fig. 6, more 1 μm particles are deposited relative to 5 μm and 10 μm particles because the smaller particles are the most sensitive to the secondary flows.

IV. Conclusions and Future Work

A. Conclusions

A finite volume solver using an LES model with dynamic Smagorinksy SGS was used to study turbulent airflow and particle deposition in the human lungs. Results show that structures in the upper airway geometry trigger turbulence in the flow, and this turbulence affects the particle distribution and deposition. Because the deposition is mainly by inertial impaction, the secondary flows reduce the particle deposition at the ridges compared to the laminar flow. The secondary flows are also responsible for deposition on tracheal walls not seen in laminar models. The distribution and deposition patterns are strongly dependent on the particle size and Stokes number.

B. Future Work

The present work was restricted to steady inspirational flow, and its effects on particle deposition. It is currently being expanded to study the differences between flow and deposition in adult and juvenile airways, and healthy and constricted airways due to COPD. Statistical analysis of the particle deposition is also being performed and will be reported in a future paper.

In the future, the effect of cyclical flow, i.e. inhalation and exhalation, on particle deposition and flow will be investigated. Currently, the deposited particles are assumed to be firmly attached to the lung walls. The effect of wall shear stress on dislodging the deposited particles and redispersing them, which is of particular significance when considering exhalation, will also be studied in the future.

V. Acknowledgments

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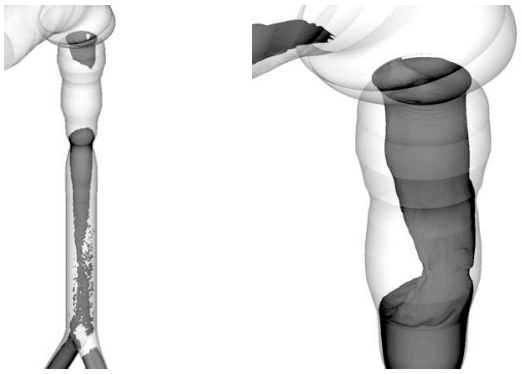
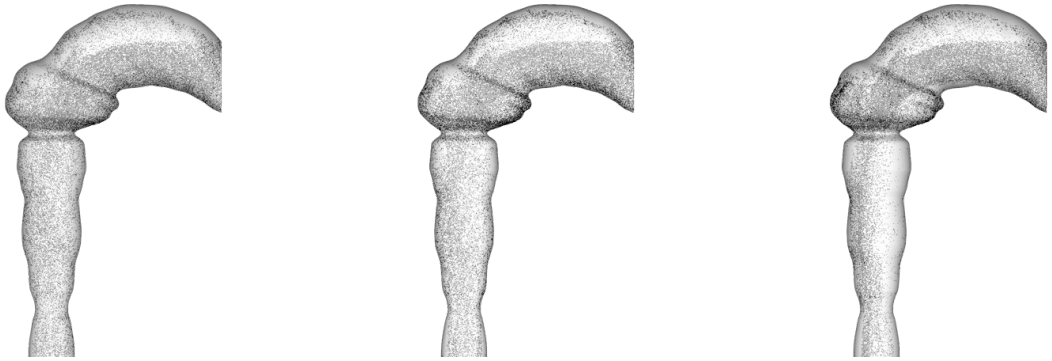


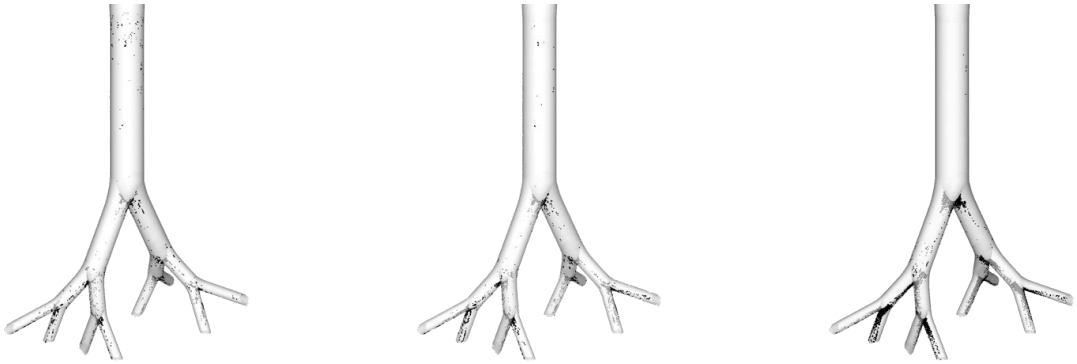
(a) $|\mathbf{v}| = 0.375$ (b) $|\mathbf{v}| = 0.25$

Fig. 4. Jets arising from the epiglottis and larynx are seen by plotting iso-surfaces of normalized mean velocity magnitude.



(a) 1 μm particles (b) 5 μm particles (c) 10 μm particles

Fig. 5. Distribution of particles in the mouth and throat.



(a) 1 μm particles (b) 5 μm particles (c) 10 μm particles

Fig. 6. Distribution of deposited particles in the branches of the lung.

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