Assessment of the airway alterations by means of a fractal ladder network model

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Abstract: Ladder network models have been successfully employed to assess electrical and mechanical properties in tissue engineering applications, showing phase-locking features of fractal structures. In this study, we simulate an anatomically-based fractal mechanical model of the airways in the 0.016-1.6 Hz frequency range to understand how the phase is affected by structural changes in the airways. With concepts from the non-rigid wall framework, airway geometry and wall structure are included in the model. We found that variations in constriction and heterogeneity led to variations in the stress-strain relation and in the elastic modulus of the airway tissue. The results indicate that constriction plays the dominant role in the stress-strain context, while heterogeneity prevalently affects the elastic modulus. Additionally, we show that a lumped parameter fractional-orders model can capture the frequency dependence of the elastic modulus.

Keywords: fractal; ladder network; impedance; fractional-order model; stress-strain; viscoelasticity; elastic modulus; respiratory system.

1. INTRODUCTION

The ratio of cartilage and soft tissue percent in human airways plays an important role in determining the specific requirements for biomaterials able to reproduce the lost airway wall mechanical properties. The elastic recoil of the lung at normal breathing frequencies is dominated by the stress-strain characteristics of the lung tissue, distinguished in collagen and elastin fibers (Bates [2007], Maksym and Bates [1997]). The amount of collagen and elastin fibers, along with their spatial distribution in the lung, are altered in diseases such as pulmonary emphysema and fibrosis (Barnes [2000]). It is therefore interesting to characterize the lung function in terms of its mechanical properties as stress, strain and viscoelasticity, which are then directly related to changes in airway duct geometry.

In this study, we investigate the mechanical properties of the airways by means of a simulated model of the human lungs. The fractal structure of the lung’s geometry is a useful property to describe such complex systems in a simple, elegant manner. Hence, we employ our previous results to simulate changes in the airway constriction and heterogeneous parameters (Ionescu et al. [2010a,b]). Variations in the airway mechanical properties can be evaluated using a mechanical equivalent model of the respiratory structure. A simplified model of the respiratory duct is employed, taking into account the core details of the human lung morphology: airway radius, length, thickness and cartilage over soft tissue percent. In this paper, we use the symmetric case of morphological values for the airways, which assumes a dichotomously equivalent bifurcation of the airways in subsequent levels (Mandelbrot [1983], Sauret et al. [2002], Weibel [1963, 2005]).

The paper is organized as follows. The respiratory tree and its modeling by the electrical equivalent is briefly explained in the next section, along with the mechanical model derivation for obtaining the stress-strain relation and elastic modulus function. Simulation results and their interpretation are detailed in the third section, while a conclusion section summarizes the main outcome of this investigation.

2. MATERIALS AND METHODS

In this section we introduce the underpinning theoretical principles to obtain the mechanical equivalent of the human respiratory tract. Starting from the respiratory morphology and anatomy, an electrical equivalent model is build, followed by its analogue representation by means of mechanical elements. Next, the stress-strain and elastic modulus relations are determined, allowing observations on the effects of variations in the morphology on their values.

2.1 Viscoelasticity in the lungs

During quiet breathing, energy is used to overcome elastic and resistive forces. The representative model is the
The constant-phase electrical equivalent model from (Hantos et al. [1986]) describing the viscoelastic properties of lung tissues, has been considered superior to the classic spring and dashpot representation, since it contains the \((j\omega)^\beta\) element which combines both these effects:

\[
Z_r(j\omega) = R_r + jL_r(j\omega) + \frac{1}{C_r(j\omega)\beta} \tag{1}
\]

with \(Z_r\) the respiratory impedance (kPa s/l), \(R_r\) the resistance (kPa s/l), \(L_r\) the inertance (kPa s^2/l), \(C_r\) the capacitance (l/kPa) of the combined element and \(\omega\) the angular frequency (rad/s), \(j = \sqrt{-1}\). This representation, \(\beta\) is referred to as the fractional order of the Laplace variable \(s\) expressed in the frequency domain: \(s \rightarrow j\omega\). Although the electrical analogue of viscoelastic processes as well as the phenomenological and mechanical approaches yield good quantitative correspondence with data, they lack anatomic and mechanistic specificity to help relate them to pathology. The model (1) originates from the power-law type of stress relaxation in a rubber balloon (Hantos et al. [1986], Yuan et al. [2000]), which can be written as \(A \times t^{-\alpha} + B\), with \(t\) the time, \(n\) the relaxation exponent and \(A, B\) constants.

The modulus of elasticity in the lung parenchyma can be expressed as a function of frequency:

\[
E(j\omega) = \frac{\text{stress}}{\text{strain}} = E'(\omega) + jE''(\omega) \tag{2}
\]

with \(E'\) the storage modulus, or the component of stress in phase with strain, and \(E''\) the loss modulus or the component of stress in phase with the strain rate (Yuan et al. [2000]). The relation to the \((j\omega)^\beta\) term from (1) is given by:

\[
Z_{\text{tissue}} = \frac{G}{\omega^\beta} - j \times \frac{H}{\omega^\beta} \tag{3}
\]

with \(\beta = \frac{\delta}{2} \tan^{-1} \left( \frac{\nu}{\rho} \right) = 1 - n\), \(G\) denoting the tissue damping and \(H\) the tissue elastance (Yuan et al. [2000]). Structural changes in the distal airways in the respiratory tree will affect the values of the stress-strain relation, hence the complex modulus of elasticity. The effects of constriction and heterogeneity can be investigated using the relation (Nucci et al. [2002]):

\[
r^c = r(1 - \frac{CS_r}{100}) \times \left(1 + \delta \frac{CV}{100}\right) \tag{4}
\]

where \(r\) is the initial airway radius, \(CS_r\) is the constriction percent, \(\delta\) is a random number sampled from a normal distribution with mean 0 and variance 1, and \(CV\) is the coefficient of variation in percent. Changes in the radius imply changes in the airway wall thickness; the latter can be simulated as \(h^c = h \times 100/CS_r\), with \(h\) the initial wall thickness.

### 2.2 Electrical analogy

In this paper we employ the symmetric case of morphological values for the airways, which assumes a dichotomously equivalent bifurcation of the airways in sub-sequent levels (Mandelbrot [1983], Sauret et al. [2002], Weibel [1963, 2005]). Gas enters and leaves the lung through a bifurcating system of tubes that get successively smaller in diameter (i.e. fractal structure) Ionescu et al. [2010b]. The respiratory system consists of two zones: the conductive zone, from level 1 to 15, and the respiratory zone, from level 16 to 24, with level 1 denoting the trachea and 24 the alveoli (Hou et al. [2005]). For the purpose of this study, we investigate changes in the airways within the respiratory zone, which is most affected by pathology. The nominal airway tube parameters are presented in Table 1.

<table>
<thead>
<tr>
<th>Level</th>
<th>Length (cm)</th>
<th>Radius (cm)</th>
<th>Wall thickness (cm)</th>
<th>Cartilage fraction (\kappa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>0.810</td>
<td>0.125</td>
<td>0.0086</td>
<td>0.0229</td>
</tr>
<tr>
<td>17</td>
<td>0.770</td>
<td>0.120</td>
<td>0.0083</td>
<td>0.0308</td>
</tr>
<tr>
<td>18</td>
<td>0.640</td>
<td>0.109</td>
<td>0.0077</td>
<td>0.0262</td>
</tr>
<tr>
<td>19</td>
<td>0.630</td>
<td>0.100</td>
<td>0.0072</td>
<td>0.0224</td>
</tr>
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<td>20</td>
<td>0.517</td>
<td>0.090</td>
<td>0.0066</td>
<td>0.0000</td>
</tr>
<tr>
<td>21</td>
<td>0.480</td>
<td>0.080</td>
<td>0.0060</td>
<td>0.0000</td>
</tr>
<tr>
<td>22</td>
<td>0.420</td>
<td>0.070</td>
<td>0.0055</td>
<td>0.0000</td>
</tr>
<tr>
<td>23</td>
<td>0.360</td>
<td>0.055</td>
<td>0.0047</td>
<td>0.0000</td>
</tr>
<tr>
<td>24</td>
<td>0.310</td>
<td>0.048</td>
<td>0.0043</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

By means of electro-mechanical analogy, the voltage represents the force and the current represent the velocity of the tissue motion. From the geometrical and morphological characteristics of the airway tube, and from the air properties, one can express the electrical parameters for one airway tube (Ionescu et al. [2010b]):

\[
R_m = \ell_m \frac{\rho_\ell^2}{4\pi \ell_m^4 M_{10}} \sin(\xi_{10}), \quad C_m = \ell_m \frac{2\pi \ell_m^3 (1 - \nu_P^2)}{E_m h_m} \tag{5}
\]

with \(\ell\) the length, \(r\) the radius, \(h\) the thickness, \(\nu_P = 0.45\) the Poisson coefficient, \(\mu = 1.86 \times 10^{-5}\) (kg/m s) the viscosity of air and \(\delta = r \sqrt{\frac{\mu}{\rho}}\) the Womersley parameter (Womersley [1957]), where \(\rho = 1.075\) (kg/m^3) is the density of air, \(\omega = 2\pi f\) and \(f\) is the frequency in Hz. \(M_{10}\) and \(\xi_{10}\) are respectively the modulus and phase angle of Bessel functions \(J_0\) and \(J_1\) of the first kind and order 0, respectively 1 (Abramowitz and Stegun [1972]), as in:

\[
M_{10} \exp^{i \xi_{10}} = 1 - \frac{2J_1(\delta^{3/2})}{J_0(\delta^{3/2})} \tag{6}
\]

The absolute elastic modulus \(E\) is considered in function of the airway tissue structure. We take into account the fraction amount \(\kappa\) of corresponding cartilage tissue (index \(c\)) and soft tissue (index \(s\)) for each level (see Table 1), with \(E_c = 400\) (kPa) and \(E_s = 60\) (kPa) (Ionescu et al. [2010b]):

\[
E_m = \kappa_m E_c + (1 - \kappa_m) E_s \tag{7}
\]

Using (5) and with \(e\) the voltage and \(i\) the current represented as in Figure 2, the equations for the electrical model are given by:
with $P$ the pressure (Pa), $Q$ the flow ($m^3/s$), $V$ the volume ($m^3$), $A_{Pm}$ and $A_{Qm}$ areas, $r$ the radius of a tube, $x$ the axial displacement and $\nu_P = 0.45$ the Poisson coefficient.

The force $f$ can be expressed as the pressure $P$ times the surface $A_p$. The velocity $v$ equals the flow $Q$ divided by the surface $A_Q$. Intuitively, each of $A_P$ and $A_Q$ will equal the cross-surface $\pi r^2 m$, while the displacement $x$ equals the volume $V$ divided by the same surface.

In a similar manner as the electrical impedance, one may obtain $H(s)$, which defines the relation from velocity (input) to force (output) $f(s)/v(s)$, with $s$ the Laplace operator. For one damper and one spring in parallel, we have that:

$$H(s) = B + \frac{K}{s}$$

### 2.3 Mechanical Model

Using the electro-mechanical analogy from Table 2, we can derive an equivalent mechanical model. This can be done starting from the electrical model equations (8)-(9). The electrical element (resistance in series with capacitor) corresponds to the mechanical Kelvin-Voigt element (dashpot in parallel with spring):

$$f_0 = B_1 v_1 + f_1; f_1 = \frac{B_2}{2} v_2 + f_2$$

$$v_1 = v_2 + \frac{1}{K_1} f_1; v_2 = \frac{2}{K_2} f_2$$

The resistance and capacitance are calculated with the nominal values in (5): $R_1 = 0.2$ (kPa s/l) and $C_1 = 0.25$ (l/kPa). The total parameter values for each level $m$ are then given by $R^*_m = R_m/2^{m-1}$ and $C^*_m = 2^{m-1} C_m$, with $R_m$ and $C_m$ from (5). From these values one can calculate the equivalent dashpot $B^*_m$ and spring $K^*_m$:

$$B^*_m = \frac{f_m}{v_m} = \frac{P_m A_{Pm} A_{Qm}}{Q_m} = R^*_m 4\pi^2 r^4_m (1 - \nu_P^2)$$

$$K^*_m = \frac{f_m}{x_m} = \frac{P_m}{V_m} A_{Pm} A_{Qm} = \frac{1}{C_m} 4\pi^2 r^4_m (1 - \nu_P^2)$$

Fig. 3. A schematic representation of the mechanical model for the lung parenchymal tissue (levels 16–24).

The lung parenchyma consists of interwoven collagen (infinitely stiff) and elastin (elastic) fibers. Each level in the respiratory tree has a specific balance between these two components. In our model we take this balance into account in (7), in function of the cartilage percent (Table 1). Following this reasoning, a similar representation of the mechanical model is given in Figure 3. Here, the cylinders represent the airway branches which are interconnected with inextensible unstressed strings. Once a string is taut, any further increases in strain will cause its associated airway branch to become strained. Only those levels with taut strings bear stress. As the tissue is stressed progressively more of the strings become taut and the stiffness of the entire model increases accordingly. The lung elasticity is determined by elastin fibers, while collagen, which is virtually inextensible, limits the maximum lung dimensions. This representation varies from that of Bates in that it represents the total collagen-elastin distribution in a level and not in a single tissue strip (Bates [2007]).
2.4 Stress-strain derivation

The elastic modulus is defined as the ratio between stress and strain properties. The Kelvin-Voigt body is the simplest viscoelastic model that can store and dissipate energy, consisting of a perfectly elastic element (i.e. spring) arranged in parallel with a purely viscous element (i.e. dashpot). The corresponding differential equation is given by:

\[ \sigma(t) = \frac{K}{A_{cross}} \epsilon(t) + \frac{B}{A_{cross}} \frac{de(t)}{dt} \]  

with \( \sigma \) the stress, \( \epsilon \) the strain, \( \ell \) the length, \( A_{cross} = 2\pi rh \) the cross section of the tube, with \( r \) the radius and \( h \) the thickness, \( K \) and \( B \) are the constants of respectively the spring and dashpot (Craiem and Armentano [2007]). The stress can be defined as pressure, whereas the latter is given by force distribution over the area. The strain \( \epsilon \) is defined as the ratio of the change in length over the initial length: \( \Delta \ell/\ell \). Thus, the strain increases in steps of 10% until it reaches 100%. The new length can be calculated as:

\[ \ell_{new} = (1 + \epsilon) \ell_{old} \]  

with the subscript old denoting the unstrained properties. Assuming a constant tissue volume \( V_t \), the radius will decrease:

\[ r_{new} = \frac{V_t}{2\pi \ell_{new} h} = \frac{r_{old} \ell_{old}}{\ell_{new}} \]  

We neglect the changes in the thickness \( h \) of the tube wall with changes in the strain. Applying an oscillatory flow \( Q \) of constant amplitude 0.5 (l/s) and a frequency of 0.25 (Hz), the velocity \( v \) can be calculated as:

\[ v_{new} = 5 \cdot 10^{-4} \frac{A_{Qnew}}{A_{Pnew}} \]  

Since the \( B \)'s and \( K \)'s are time-invariant material properties, the transfer function \( H \) will be independent of the strain. This mechanical impedance \( H \) is defined as force over velocity. The new pressure is then given by:

\[ P_{new} = \frac{f_{new}}{A_{Pnew}} = \frac{H \cdot v_{new} H}{A_{Pnew}} = \frac{H \cdot 5 \cdot 10^{-4}}{A_{Pnew} A_{Qnew}} \]  

with the multiplication of the areas \( A_{Pnew} A_{Qnew} = 4\pi r_{new}^4 (1 - \nu^2_p) \). The elongation of the tube can be expressed as (Ionescu et al. [2010a]):

\[ P + \frac{h}{r (1 - \nu^2_p)} \left( \frac{K \ell}{A_{cross}} \epsilon + \frac{B \ell}{A_{cross}} \frac{de(t)}{dt} \right) = 0 \]  

The stress \( \sigma \) are then given by:

\[ \sigma_{new} = -P_{new} \frac{r_{new} (1 - \nu^2_p)}{h} \]  

Now the stress and strain properties can be evaluated using equations (16)-(21). For a viscoelastic material the mechanical impedance \( H(s) \) of this material leads to the following relation for the complex modulus:

\[ E^*(s) = \frac{\ell}{A_{cross}} \cdot s \cdot H(s) \]  

with \( s \) the Laplace variable and \( H(s) \) from (14). We can then observe how the changes in the airways alter the elastic modulus of the tissue and its frequency response.

2.5 Relating the fractional order with frequency

The Laplace transform for integral and derivative order \( n \) are, respectively (Oustaloup [1995]):

\[ L \{ D^n f(t) \} = s^n F(s) \]  

\[ L \{ D^\nu f(t) \} = s^\nu F(s) \]  

where \( F(s) = L \{ f(t) \} \) and \( s \) is the Laplace complex variable. The Fourier transform can be obtained by replacing \( s \) by \( j\omega \) in the Laplace transform and the equivalent frequency-domain expressions are:

\[ (j\omega)^\pm n = \omega^{\pm n} \left( \frac{\cos \frac{n\pi}{2} \pm j \sin \frac{n\pi}{2}}{2} \right) \]  

Thus, the modulus and the argument of the FO terms are given by:

\[ Modulus(dB) = 20 \log |(j\omega)^\pm n| = \mp 20n \log |\omega| \]  

\[ Phase(rad) = \arg((j\omega)^\pm n) = \mp n \frac{\pi}{2} \]  

resulting in:

- Magnitude (dB vs log-frequency): straight line with a slope of \( \mp 20n \) passing through 0dB for \( \omega = 1 \);
- Phase (rad vs log-frequency): horizontal line, thus independent with frequency, with value \( \mp n \frac{\pi}{2} \).

The corresponding sketches can be seen in figure 4. Notice that the phase constancy is a feature of fractional order systems, originated by fractal structures (Mandelbrot [1983], Oustaloup [1995], Weibel [2005]). In short, models with fractional-orders can be used to characterize the frequency-dependent magnitude of a system by means of a minimum number of model parameters.

3. RESULTS AND DISCUSSION

The effect of variations in the constriction percent from nominal to 20%, 40% and 60% respectively, upon the airway parameters calculated in single airways, is depicted in figure 5-left. This implies that constriction affects mainly the elasticity of the tissue, which decreases as constriction increases. The effect of variations in the heterogeneity percent from nominal to 10%, 20% and 40% respectively, upon the airway parameters calculated in single airways, is depicted in figure 5-right. In both tests, no significant influence occurs in the \( B_m \) parameters, while visible changes occur in the \( K_m \) parameters. A threshold value.
of 40% heterogeneity denotes a critical state for the tissue elasticity. If heterogeneity is significant, such as in emphysema, the empty spaces between the healthy airways are increased, leading to the 'fake'-effect of an increased elasticity. However, due to the fact that elastin links are broken in the lung parenchyma in this case, the overall tissue elasticity is much decreased, making difficult both in-take as well as out-take of air into the lungs. The net effects of variations in the constriction percent and in the heterogeneity percent, in the overall airway parameters are depicted in Figure 6.

Fig. 5. LEFT: influence of constriction in single airways; RIGHT: influence of heterogeneity in single airways.

Fig. 6. LEFT: influence of constriction in overall airways; RIGHT: influence of heterogeneity in overall airways.

Fig. 7. Bode plot for $H(s)$ for constriction (left) and for heterogeneity (right) effects.

Fig. 8. The stress-strain curves with variations in constriction (left) and in heterogeneity (right).

Similarly to the single airways case, no significant influence occurs in the overall $K_m$ parameters. Consequently, we conclude that constriction affects mainly the elasticity of the tissue, which decreases as constriction increases in the overall airway structure. Heterogeneity seems not to play an important role when referred to the overall airway structure, but it might become significant in parts of the lungs structure (e.g. lung cancer, carcinoma, etc).

It is important now to look at the force-velocity component in function of the low frequencies. Constriction and heterogeneity effects upon the transfer function from force to velocity of tissue are given in Figure 7. The transfer function from (14) represents the force-velocity relation of the tissue in motion during oscillations applied at different frequencies ($\omega = 2\pi f$, with $f$ the frequency). This is usually represented by the Bode plot, namely magnitude and phase in function of the angular frequency $\omega$. The electrical equivalent of the relation $H(s)$ is the electric impedance, whereas the force is denoted by the voltage (physically corresponding to air pressure) and the velocity is denoted by the current (physically corresponding to air-flow). We can observe that both constriction and heterogeneity affects the magnitude as well as the phase (dynamics) of the system.

Using the relations introduced in section 2.4, one obtains the stress-strain curves depicted in Figure 8. The strain is increased in steps of 10% from 10 to 100%. Starting from level 24, one can then calculate the stress-strain curve at the input of each level. This then will give rheological information in the context of all airway levels interconnected. The overall stress-strain relationship becomes quasi-linear with the overall constriction variations. As expected from physiology, variation in heterogeneity does not affect the stress-strain relation in the lung parenchyma.

Since (22) is the derivative of (14), it can be concluded that the complex modulus will pose a similar absolute variation with frequency. Hence, if one would identify a lumped model in a limited frequency range, one would need a fractional-order model to account for the frequency dependent modulus ([Ionescu et al., 2010a, Craiem and Armentano, 2007, Bates, 2007]). It has been shown in (Craiem and Armentano [2007]) that elastic modulus needs to be characterized by two separate fractional order derivatives, in order to accurately capture the power-law response which varies with frequency:

$$E^*(j\omega) = \frac{1}{C_E(j\omega)^{\alpha_E}} + L_E(j\omega)^{\beta_E}$$  (28)

with $\frac{1}{C_E(j\omega)^{\alpha_E}}$ the model from (3). Our modelling approach justifies further the necessity of having such fractional order by its intrinsic relation to the airway morphology. Nonlinear least squares identification in the 0.016-1.6 Hz (up to the resonance peak) with the model from (3) could not capture the frequency dependence of (22) in a minimum error sense. Our findings suggest that the lung parenchyma exhibits different dynamic behavior of viscoelastic effects, for different frequency intervals. Our model identification results for the constriction and heterogeneity are given in Table 3.

From Table 3 we observe that $a_E$ decreases with increasing constriction, suggesting a stiffening of the tissue. The same stiffening in tissue is also observed by the increasing values
Table 3. Identified model parameters; cstr - constriction; heter - heterogeneity.

<table>
<thead>
<tr>
<th></th>
<th>1/CE</th>
<th>aE</th>
<th>LE</th>
<th>bE</th>
</tr>
</thead>
<tbody>
<tr>
<td>nominal</td>
<td>3.44</td>
<td>0.0191</td>
<td>1.18</td>
<td>1.11</td>
</tr>
<tr>
<td>20% cstr</td>
<td>4.73</td>
<td>0.0139</td>
<td>1.73</td>
<td>1.23</td>
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<tr>
<td>40% cstr</td>
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<td>2.23</td>
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<tr>
<td>60% cstr</td>
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<td>0.0022</td>
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<tr>
<td>10% heter</td>
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<td>0.0039</td>
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<td>1.32</td>
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<td>3.06</td>
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<tr>
<td>40% heter</td>
<td>11.31</td>
<td>0.023</td>
<td>4.98</td>
<td>1.59</td>
</tr>
</tbody>
</table>

Fig. 9. Evaluation of the parameters given in Table 3 at frequency 1 Hz, with \( D_E = 1/CE \).

of \( b_E \) with constriction. This is shown in figure 9 and validated by the direction of increasing values in the terms of (28) for frequency 1 Hz, with changing constriction (top) and heterogeneity (bottom) percent, respectively. The fact that the absolute rate of variations in the values of \( a_E \) and \( b_E \) are different may originate from the changes in the slope in the modulus and phase of the dynamic elastic modulus with frequency. A similar reasoning was found for models in two fractional-orders for the arterial elastic modulus with frequency. A similar reasoning was found for models in two fractional-orders for the arterial elastic modulus with frequency.

4. CONCLUSIONS

In this study, we simulate an anatomically based fractal electro-mechanical model of the airways in the 0.016-1.6 Hz frequency range to understand how the phase is affected by alterations in the airway properties. Airway geometry and wall structure is taken into account, using non-rigid wall considerations. Variations in constriction and heterogeneity lead to variations in the stress-strain relation and in the elastic modulus. The results indicate that overall variations in airway constriction are more significant than those in airway heterogeneity in stress-strain relation, but that heterogeneity prevalently affects the elastic modulus.

The model presented in this paper provides an initial framework to help understanding the relation between variations in the airway structure and consequent effects in the overall tissue viscoelasticity and corresponding fractional-order model parameters. Furthermore, it can also serve as a basis for electro-mechanical models for other branching systems, e.g. the circulatory system.

REFERENCES


