



ARL-TR-8859 • Nov 2019



A Continuum Mechanical Model of the Lung

by John D Clayton and Alan D Freed

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by John D Clayton and Alan D Freed

Weapons and Materials Research Directorate, CCDC Army Research Laboratory

REPORT DOCUMENTATION PAGE				Form Approved OMB No. 0704-0188	
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1. REPORT DATE (DD-MM-YYYY) November 2019		2. REPORT TYPE Technical Report		3. DATES COVERED (From - To) November 2018–November 2019	
4. TITLE AND SUBTITLE A Continuum Mechanical Model of the Lung				5a. CONTRACT NUMBER	
				5b. GRANT NUMBER	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S) John D Clayton and Alan D Freed				5d. PROJECT NUMBER	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) US Army Combat Capabilities Development Command Army Research Laboratory ATTN: FCDD-RLW-PC Aberdeen Proving Ground, MD 21005-5066				8. PERFORMING ORGANIZATION REPORT NUMBER ARL-TR-8859	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)				10. SPONSOR/MONITOR'S ACRONYM(S)	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION/AVAILABILITY STATEMENT Approved for public release; distribution is unlimited.					
13. SUPPLEMENTARY NOTES primary author's email: <john.d.clayton1.civ@mail.mil>.					
14. ABSTRACT A continuum model for lung parenchyma is constructed. The model describes the thermomechanical response over a range of loading rates—from static to dynamic to shock waves—and a range of stress states, including isotropic expansion, triaxial extension, simple shear, and plane wave compression. Nonlinear elasticity, viscoelasticity, and damage are included. A free energy function is designed for loading at low to moderate rates and tensile pressures, whereby the tissue response, with surface tension, is preeminent. An internal energy function is designed for wave propagation analysis, including shock waves, whereby compressibility of the air inside the alveoli is addressed via a composite stiffness based on a closed-cell assumption. The model accurately represents the response with relatively few measurable parameters, most with an obvious physical interpretation. Longitudinal wave speeds are reasonable for ranges of internal airway pressure and transpulmonary pressure. Airway pressure is shown to strongly affect the response to plane wave compression. Criteria for local injury and damage progression depend on a normalized energy density and its gradient, where the latter is paramount for impact problems involving fast pressure rises.					
15. SUBJECT TERMS nonlinear elasticity, viscoelasticity, damage mechanics, injury mechanisms, soft solids, lung, shock waves					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT UU	18. NUMBER OF PAGES 114	19a. NAME OF RESPONSIBLE PERSON John D Clayton
a. REPORT Unclassified	b. ABSTRACT Unclassified	c. THIS PAGE Unclassified			19b. TELEPHONE NUMBER (Include area code) 410-278-6146

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Acknowledgments

Dr John Clayton acknowledges support of the US Army Combat Capabilities Development Command (CCDC) Army Research Laboratory. Prof Alan Freed acknowledges support of a Joint Faculty Appointment with the CCDC Army Research Laboratory. Informative discussions with Oren Petel of Carleton University and Rohan Banton, Tim Baumer, Michael Kleinberger, and Karin Rafaels of the CCDC Army Research Laboratory on soft tissue biomechanics and ballistic injury mechanisms are also acknowledged.

1. Introduction

The mechanical response of soft biological tissues is described by static and dynamic constitutive models. Models of high fidelity are required for development of better practices or products in the medical, transportation, and defense industries. Novel surgical procedures can be informed, and designs of medical implants with surrogate materials of similar properties can be enhanced, via use of models for the static response of soft tissues. The mechanical response of the body to impacts encountered in sporting activities, vehicular collisions, and ballistic events is described by dynamic tissue models. These models facilitate the design of protective systems in automobiles and personal protective equipment for police, security, and military activities.

Since deformations are very often large, even within normal ranges of everyday biological activity (*e.g.*, muscle functions, circulation, breathing), nonlinear constitutive models are necessary. For very low loading rates, the equilibrium nonlinear elastic response is usually of primary interest, while viscoelasticity becomes crucial to account for rate dependence, hysteresis, and stress relaxation. Models linking degradation of mechanical properties to loss of biological function are sought for traumatic events, wherein damage may be incurred in the tissue. For overviews of this subdiscipline of biomechanics, as well as applications to particular kinds of tissues, the reader is referred to some prominent literature¹⁻⁴ on the subject.

Concurrent work by the authors⁵⁻⁷ seeks to develop a general theoretical framework enabling modeling of the above-mentioned physical phenomena. The framework should be applicable to a host of soft biological tissues (*e.g.*, muscle, skin, internal organs) as well as other viscoelastic solids, such as polymers, capable of sustaining very large deformations. Dynamic behavior and irreversible structure changes associated with damage are also encompassed.

The focus of the present work is application of such a theory to the lung, which is the largest vital organ in the human thorax by volume. Sought eventually are modeling and numerical simulation capabilities of large deformation, stress wave propagation, damage, and injury mechanisms sustained in the lung tissue when the thorax is subjected to traumatic insult by blunt impact at various rates⁸⁻¹⁰ or rapidly rising high-pressure (*i.e.*, blast) waves.¹¹⁻¹⁵

A predictive modeling capability will enable the development of innovative protection systems for vehicular passengers with regard to crashworthiness and Soldiers in military operations.¹³ Pulmonary contusion is the most common thoracic soft tissue injury for blunt trauma, with a mortality rate of 10%–25%.¹⁶ Damage to the lungs with resultant respiratory insufficiency and arterial embolization of air from alveolar pulmonary venous fistulae is the main cause of morbidity and mortality following high-level blast exposure.¹⁷ Lung laceration is also common and debilitating, often accompanied by rib fracture.¹⁸

Perhaps the most popular nonlinear constitutive model for lung mechanics is based on work of Fung, Vawter, and colleagues initiated over four decades ago.^{19–22} Notably, an exponential term in an isotropic strain energy function accounts for the elasticity of the microstructure of the connective tissues in the lung, such as septal walls, assumed to behave similarly to skin¹⁹ and other compliant elastic tissues.²³ A second term in the energy function accounts for the energy of the surface tension of the alveolar membranes.²⁴ Both terms are constructed from the usual invariants of the Green–Lagrange strain tensor. A simplification invoked in this theory is the use of a pseudo-hyperelastic strain energy function giving a different response in loading and unloading paths,^{20,21} rather than the use of a more general viscoelastic treatment.²⁵ Minimally, six material parameters enter the total (elastic + surface) energy function²² for each load path, where only one, the mean alveolar diameter, is physically based and deemed unique. Calibration of the model is accordingly cumbersome, for example, requiring sophisticated optimization algorithms.^{21,26,27} Further hindering calibration are experimental challenges associated with biological specimen variability, moisture and pressure of the testing environment, extreme softness and fragility of the tissues whose properties change over time after extraction, and unwanted inertial effects in the dynamic regime.²⁸ Standardized testing techniques for soft tissue mechanics at very high loading rates are also not established.²⁹

Modern finite-element simulations of lung deformation in the setting of injury mechanics^{10,26,27} adopt a pseudo-hyperelastic theory, with minor variations to account for a different bulk modulus than that derived from hyperelasticity,¹⁰ a viscoelastic deviatoric response,³⁰ or an equation-of-state for the bulk response under blast loading^{15,31} where immobility and compressibility of the gas within become important.^{32,33} Mild anisotropy is likely and may provide better parameterization to data,²¹

but is usually neglected^{20,22,30} to keep the calculations and calibrations tractable.

The present approach seeks a constitutive framework with more physically descriptive, fewer, and easier calibrated parameters for a given soft solid than existing models. In this approach, the parenchyma—including alveoli, connective tissues, and internal fluids (gas and liquid)—is treated as a homogeneous isotropic continuum, following other models used in numerical simulations^{10,15,22,27,30} and a more recent implicit elastic theory.^{4,34} Details of the microstructure^{35,36} and fluid-structure interactions^{37–39} are not resolved explicitly since the length scales and complexities render the use of multiscale or multiphase models for the thorax, with corresponding resolution of the entire organ, computationally intractable.

This work develops a novel and comprehensive nonlinear constitutive model for lung response applicable to static, dynamic, and shock loading. The model is more general than others known to the authors that tend to focus on a single regime (*e.g.*, models designed exclusively for static extension, cyclic expansion as in respiration, blunt impact, or blast). It also is the first known model for lung tissue mechanics that incorporates an evolving internal state variable representative of damage,^{40,41} though different kinds of damage mechanics formulations have been used for other fibrous soft biological tissues.^{42,43} With the entire dynamic constitutive model at hand for the lung, the relationships among local mechanical states and injuries, such as contusion and edema observed in experiments, can be studied. For example, hypotheses for trauma proposed previously^{1,9,12,15,26,27,37,44,45} can be critically evaluated. Notably, the load history-dependent damage variable postulated herein, which depends upon normalized measures of energy density and energy density gradients, is mathematically (*i.e.*, thermodynamically) related to stiffness reduction and, from physical observation, linked to local injury to the tissue microstructure. This variable, when tracked in future finite-element simulations of the thorax, will enable transient monitoring of lung trauma during the course of a blunt impact or a blast event. This contrasts current state-of-the-art methods whereby injury is evaluated a posteriori via calibration,^{15,27} rather than predicted via a model that has been validated a priori as sought in the present work.

The general theoretical basis follows that of several recent works.^{5,7,46} The deformation gradient is decomposed in the format of a Gram–Schmidt (QR) product of a rotation Q and an upper triangular stretch measure R . Strain attributes⁴⁷ enter

the thermodynamic potentials, and their conjugate scalar stress measures enter the constitutive model. Nonlinear viscoelasticity is addressed in a thermodynamically consistent way extending prior research^{48,49} to the QR framework with strain attributes. A composite internal energy formulation accounts for air compressibility, which crucially affects the effective bulk modulus of the overall structure for dynamic compression.^{1,6,32,33,44} The damage variable is linked to changes in mechanical properties (*e.g.*, stiffness) as well as possible functional impairment such as permeability changes and subsequent edema at low loads and hematoma associated with contusion at more intense loads.^{1,44,45,50}

Successful calibration is demonstrated later in this work for the equilibrium elastic response of the lung under isotropic expansion and triaxial extension. Evaluation of the viscoelastic (nonequilibrium) response is performed in the context of pressure relaxation and dynamic simple shear and torsion experiments. Experiments^{44,45} report on edema occurrence after static over-stretching and after dynamic compression via shock tube and projectile impact tests. Damage and injury criteria for dynamic loading are analyzed in this context for plane wave compression. Cyclic response, especially at relatively low load levels pertinent to the normal respiratory cycle, is outside the present scope.

This report is organized as follows. Elements of the general theory,^{5,7,51} notably continuum kinematics and thermodynamics that subsequently enter the application to the lung, are reviewed in Section 2. Nonlinear elasticity, viscoelasticity, damage, and injury mechanisms are considered in the context of both static and dynamic loading, with ample comparison of model and available experimental data. A free energy function most suitable for modeling the lung response at relatively low loading rates is detailed in Section 3. Evaluation of this function and its derivatives entering the governing equations of the constitutive theory are described in the context of experiments from the literature in Section 4. An internal energy function most suitable for modeling the lung response for problems involving wave motion is given in Section 5. Features of this function are evaluated in the context of elastic wave velocities and planar shock compression in Section 6. Conclusions follow in Section 7. The notation applies conventions of modern continuum mechanics, whereby vectors and tensors of higher order are written in boldface type, with scalars and scalar components in italics. When index notation is used for clarity, summation is implied over repeated indices. Unusual notation is defined later as needed.

2. General Continuum Mechanical Theory

General governing equations of the present theory closely follow those of a nonlinear viscoelastic-damage framework based on QR kinematics.^{5,7} A number of specific constitutive formulations under the umbrella of this framework are derived in prior work,^{5,7} without particular application to any single material system. This section summarizes aspects of this framework^{5,7} that are universally applied, irrespective of particular material. Specialization of the model features to the lung follows subsequently in Sections 3 and 5.

2.1 Kinematics of Finite Deformation

Denoted by $\mathbf{X}$ and $\mathbf{x}$ are Lagrangian and Eulerian position vectors of a material particle in the respective reference and spatial configurations, with components of each vector referred to a global Cartesian system with a fixed origin. Let t denote time. Motions between configurations are

$$\mathbf{x} = \boldsymbol{\varphi}(\mathbf{X}, t), \quad \mathbf{X} = \boldsymbol{\Phi}(\mathbf{x}, t). \quad (1)$$

These are presently presumed sufficiently smooth to enable the existence of all subsequent space–time derivatives. Shocks (*i.e.*, surfaces of singularity across which space–time derivatives of motion may jump⁵²) are addressed later in Section 6. The material time derivative, material gradient, and spatial gradient of a generic quantity f are the respective operations

$$\dot{f}(\mathbf{X}, t) = \frac{\partial f(\mathbf{X}, t)}{\partial t}, \quad \nabla_0 f(\mathbf{X}, t) = \frac{\partial f(\mathbf{X}, t)}{\partial \mathbf{X}}, \quad \nabla f(\mathbf{X}(\mathbf{x}, t), t) = \frac{\partial f(\mathbf{X}(\mathbf{x}, t), t)}{\partial \mathbf{x}}. \quad (2)$$

Particle velocity and acceleration are

$$\mathbf{v}(\mathbf{X}, t) = \dot{\mathbf{x}}(\mathbf{X}, t), \quad \mathbf{a}(\mathbf{X}, t) = \dot{\mathbf{v}}(\mathbf{X}, t). \quad (3)$$

Arguments (*e.g.*, $(\mathbf{X}, t)$ in the Lagrangian description) are dropped hereafter unless needed for clarity.

The deformation gradient and its inverse are

$$\mathbf{F} = \nabla_0 \boldsymbol{\varphi} = \partial \mathbf{x} / \partial \mathbf{X}, \quad \mathbf{F}^{-1} = \nabla \boldsymbol{\Phi} = \partial \mathbf{X} / \partial \mathbf{x}. \quad (4)$$

The referential and spatial velocity gradients are

$$\nabla_0 \mathbf{v} = \dot{\mathbf{F}}, \quad \nabla \mathbf{v} = \dot{\mathbf{F}} \mathbf{F}^{-1}. \quad (5)$$

Let dv and dV denote spatial and referential volume elements. Then

$$dv = (\det \mathbf{F}) dV = J dV, \quad \dot{J} = J \nabla \cdot \mathbf{v} = J \text{tr}(\dot{\mathbf{F}} \mathbf{F}^{-1}), \quad (6)$$

with $J > 0$ the Jacobian determinant and $\text{tr}(\cdot)$ the trace.

A fixed global Cartesian frame with spatial basis vectors $\{\mathbf{e}_i\}$, $i = 1, 2, 3$ is used, to which all vector and tensor components are referred unless noted otherwise. Let capital Roman indices $J = 1, 2, 3$ denote Lagrangian components. Then

$$\mathbf{e}_i = \mathbf{e}^i = \delta_j^i \mathbf{e}^J = \delta_j^i \mathbf{e}_J. \quad (7)$$

The deformation gradient in this coordinate frame is

$$\mathbf{F} = F_J^i \mathbf{e}_i \otimes \mathbf{e}^J = F_{ij} \mathbf{e}_i \otimes \mathbf{e}_j. \quad (8)$$

The deformation gradient can be decomposed uniquely into the product of a rotation $\mathbf{Q}$ and upper triangular matrix $\mathbf{R}$, where the latter is known as the Laplace stretch⁵³:

$$\mathbf{F} = \mathbf{Q} \mathbf{R} = Q_\alpha^i R_J^\alpha \mathbf{e}_i \otimes \mathbf{e}^J; \quad \mathbf{Q}^{-1} = \mathbf{Q}^T, \quad \det \mathbf{Q} = +1. \quad (9)$$

Greek indices $\alpha = 1, 2, 3$ correspond to an intermediate configuration whose tangent vectors are pulled back from the spatial configuration by $\mathbf{Q}^{-1}$ or pushed forward from the reference configuration by $\mathbf{R}$. See Fig. 1. The same global Cartesian basis is used for this configuration, called the “physical configuration” in prior work⁴⁶:

$$\mathbf{e}_\alpha = \mathbf{e}^\alpha = \delta_\alpha^i \mathbf{e}_i = \delta_\alpha^J \mathbf{e}_J. \quad (10)$$

In matrix form, with all components referred to the global Cartesian bases of Eqs. 7 and 10,

$$\mathbf{R} = \begin{bmatrix} R_{11} & R_{12} & R_{13} \\ 0 & R_{22} & R_{23} \\ 0 & 0 & R_{33} \end{bmatrix} = \begin{bmatrix} a & a\gamma & a\beta \\ 0 & b & b\alpha \\ 0 & 0 & c \end{bmatrix} = \begin{bmatrix} a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} \begin{bmatrix} 1 & \gamma & \beta \\ 0 & 1 & \alpha \\ 0 & 0 & 1 \end{bmatrix} = \mathbf{\Lambda} \mathbf{\Gamma}. \quad (11)$$

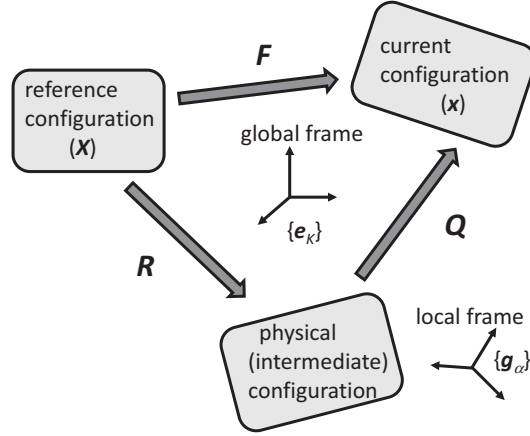


Fig. 1 Configurations, deformations, and coordinate frames^{5,7}

Physically descriptive deformation measures are the three positive elongation ratios a, b, c that enter the extension matrix $\mathbf{\Lambda}$ and the three shear magnitudes α, β, γ that enter the simple shear matrix $\mathbf{\Gamma}$. Volume change is measured by

$$J = \det \mathbf{R} = \det \mathbf{\Lambda} = abc. \quad (12)$$

Parameters $a, b, c, \alpha, \beta, \gamma$ are physical attributes because they can be measured directly by an experimentalist in the physical coordinate system of later Eq. 17, without post analysis.^{46,51,54}

Let

$$\mathbf{C} = \mathbf{F}^T \mathbf{F} = \mathbf{R}^T \mathbf{R} = C_{IJ} \mathbf{e}^I \otimes \mathbf{e}^J = F_I^i \delta_{ij} F_J^j \mathbf{e}^I \otimes \mathbf{e}^J = R_I^\alpha \delta_{\alpha\beta} R_J^\beta \mathbf{e}^I \otimes \mathbf{e}^J \quad (13)$$

denote the symmetric right Cauchy–Green deformation tensor. Nonzero components of $\mathbf{R} = R_{IJ} \mathbf{e}^I \otimes \mathbf{e}^J$ are obtained explicitly from the components C_{IJ} by the recursive formulae⁵⁵

$$\begin{aligned} R_{11} &= \sqrt{C_{11}}, & R_{12} &= C_{12}/R_{11}, & R_{13} &= C_{13}/R_{11}, & R_{22} &= \sqrt{C_{22} - (R_{12})^2}, \\ R_{23} &= (C_{23} - R_{12}R_{13})/R_{22}, & R_{33} &= \sqrt{C_{33} - (R_{13})^2 - (R_{23})^2}. \end{aligned} \quad (14)$$

Elongations and shears are

$$a = R_{11}, \quad b = R_{22}, \quad c = R_{33}; \quad \alpha = \frac{R_{23}}{R_{22}}, \quad \beta = \frac{R_{13}}{R_{11}}, \quad \gamma = \frac{R_{12}}{R_{11}}. \quad (15)$$

The inverse of $\mathbf{R}$ is also upper triangular and can be written explicitly in matrix form:

$$\mathbf{R}^{-1} = \mathbf{\Gamma}^{-1} \mathbf{\Lambda}^{-1} = \begin{bmatrix} 1/a & -\gamma/b & (\alpha\gamma - \beta)/c \\ 0 & 1/b & -\alpha/c \\ 0 & 0 & 1/c \end{bmatrix}. \quad (16)$$

With Eq. 16, $\mathbf{Q} = \mathbf{F}\mathbf{R}^{-1}$ can be calculated by matrix multiplication. Given F_j^i , no eigenvalue analysis is needed for a QR decomposition, in contrast to the more widely used polar decomposition.

2.2 Physical and Convected Bases

Index numbers 1, 2, 3 to the $\{\mathbf{e}_i\}$ should be assigned carefully when the QR decomposition is used. Let the $\{\mathbf{e}_i\}$ be rotated by $\mathbf{Q}$. An ideal choice of bases considers the properties⁵⁶ that the direction aligned with the rotated 1-axis and the direction normal to the rotated 1-2-plane remain invariant under a tangent mapping by upper triangular transformation $\mathbf{R}$. It is assumed henceforth that the $\{\mathbf{e}_i\}$ have been ideally labeled such that the rotated 1-direction aligns with the axis of minimal transverse shear. A pivoting algorithm to achieve the corresponding upper triangular dominance of $\mathbf{R}$ is given in previous work.⁴⁶ The physical basis,⁴⁶ denoted here by $\{\mathbf{g}_\alpha\}$, $\alpha = 1, 2, 3$, corresponds to the aforementioned rotated coordinate system:

$$\mathbf{g}_\alpha = \mathbf{g}^\alpha = Q_\alpha^i \mathbf{e}_i = Q_\alpha^i \delta_i^J \mathbf{e}_J, \quad \mathbf{e}_i = (Q^{-1})_i^\alpha \mathbf{g}_\alpha = (Q^T)_i^\alpha \mathbf{g}_\alpha. \quad (17)$$

In this physical frame, the three elongations a, b, c and three shears α, β, γ (*i.e.*, the physical attributes of the Laplace stretch $\mathbf{R}$) are directly measured. The $\{\mathbf{g}_\alpha\}$ compose a local Cartesian frame (*i.e.*, orthonormal right-handed triad) at each material point $\mathbf{X}$, as shown in Fig. 1. If $\mathbf{Q}_t(\mathbf{X}) = \mathbf{Q}(\mathbf{X}, t)$ is not spatially homogeneous, then obviously the $\mathbf{g}_\alpha$ do not compose a global rectilinear frame.

In the physical coordinate system, introduce a unit cube with edges parallel to the $\{\mathbf{g}_\alpha\}$. The Laplace stretch $\mathbf{R}$ deforms such a cube to a parallelepiped whose edges are the oblique basis vectors $\{\boldsymbol{\gamma}_\alpha\}$, neither always orthogonal nor of unit length.

These are calculated by⁵¹

$$\gamma_\alpha = \Lambda_\alpha^\delta \Gamma_{\beta\delta} \mathbf{g}^\beta \Leftrightarrow \{\gamma_\alpha\} = \mathbf{\Lambda} \mathbf{\Gamma}^T \{\mathbf{g}_\alpha\} \Leftrightarrow \begin{Bmatrix} \gamma_1 \\ \gamma_2 \\ \gamma_3 \end{Bmatrix} = \begin{bmatrix} a & 0 & 0 \\ b\gamma & b & 0 \\ c\beta & c\alpha & c \end{bmatrix} \begin{Bmatrix} \mathbf{g}_1 \\ \mathbf{g}_2 \\ \mathbf{g}_3 \end{Bmatrix}. \quad (18)$$

Though not used subsequently herein, a convected metric with covariant components $\gamma_{\alpha\beta} = \gamma_\alpha \cdot \gamma_\beta$ enables a geometric description of strain in the context of QR kinematics.⁵¹

2.3 Velocity Gradient

The mixed-variant velocity gradient in the second of Eq. 5 obeys

$$\nabla \mathbf{v} = \dot{\mathbf{F}} \mathbf{F}^{-1} = \dot{\mathbf{Q}} \mathbf{Q}^{-1} + \mathbf{Q} \dot{\mathbf{R}} \mathbf{R}^{-1} \mathbf{Q}^{-1} = \mathbf{\Omega} + \mathbf{Q} \boldsymbol{\eta} \mathbf{Q}^{-1}, \quad (19)$$

where $\mathbf{\Omega}$ is skew and the rate contribution from the Laplace stretch is embodied by

$$\boldsymbol{\eta} = \dot{\mathbf{R}} \mathbf{R}^{-1} = \begin{bmatrix} \dot{a}/a & a\dot{\gamma}/b & a(\dot{\beta} - \alpha\dot{\gamma})/c \\ 0 & \dot{b}/b & b\dot{\alpha}/c \\ 0 & 0 & \dot{c}/c \end{bmatrix}. \quad (20)$$

All matrix components in Eq. 20 are referred to the global basis $\{\mathbf{e}_i\}$ of Eqs. 7 and 10. Note that the product of two upper triangular matrices is always upper triangular. Components are also notably equal those of the second term in the last of Eq. 19 when referred to physical basis $\{\mathbf{g}_\alpha\}$:

$$\begin{aligned} \mathbf{Q} \dot{\mathbf{R}} \mathbf{R}^{-1} \mathbf{Q}^{-1} &= Q_\alpha^i \dot{R}_J^\alpha (R^{-1})_J^\beta (Q^{-1})_j^\beta \mathbf{e}_i \otimes \mathbf{e}^j \\ &= \dot{R}_J^\alpha (R^{-1})_J^\beta Q_\alpha^i \mathbf{e}_i \otimes (Q^{-1})_j^\beta \mathbf{e}^j = \eta_\beta^\alpha \mathbf{g}_\alpha \otimes \mathbf{g}^\beta. \end{aligned} \quad (21)$$

Rate terms in Eq. 20 are mostly orthogonal in the terminology of Criscione,⁴⁷ the exception is η_{13} , which contains both $\dot{\beta}$ and $\dot{\gamma}$.

2.4 Strain Attributes

Let a_0, b_0, c_0 and $\alpha_0, \beta_0, \gamma_0$ denote values of a, b, c and α, β, γ in a reference state corresponding to time instant $t = t_0$. A vector consisting of seven strain attributes

$\epsilon_i, i = 0, \dots, 6$ is defined as

$$\{\epsilon_i\} = \begin{Bmatrix} \epsilon_0 \\ \epsilon_1 \\ \epsilon_2 \\ \epsilon_3 \\ \epsilon_4 \\ \epsilon_5 \\ \epsilon_6 \end{Bmatrix} = \begin{Bmatrix} \ln(abc/a_0b_0c_0) \\ \frac{1}{3} \ln(ab_0/a_0b) \\ \frac{1}{3} \ln(bc_0/b_0c) \\ \frac{1}{3} \ln(ca_0/c_0a) \\ \gamma - \gamma_0 \\ \alpha - \alpha_0 \\ \beta - \beta_0 \end{Bmatrix}, \quad \{\dot{\epsilon}_i\} = \begin{Bmatrix} \dot{\epsilon}_0 \\ \dot{\epsilon}_1 \\ \dot{\epsilon}_2 \\ \dot{\epsilon}_3 \\ \dot{\epsilon}_4 \\ \dot{\epsilon}_5 \\ \dot{\epsilon}_6 \end{Bmatrix} = \begin{Bmatrix} \dot{a}/a + \dot{b}/b + \dot{c}/c \\ \frac{1}{3}(\dot{a}/a - \dot{b}/b) \\ \frac{1}{3}(\dot{b}/b - \dot{c}/c) \\ \frac{1}{3}(\dot{c}/c - \dot{a}/a) \\ \dot{\gamma} \\ \dot{\alpha} \\ \dot{\beta} \end{Bmatrix}. \quad (22)$$

Only six of the seven strain attributes in Eq. 22 are independent kinematic variables since

$$\epsilon_1 + \epsilon_2 + \epsilon_3 = 0 \quad \Rightarrow \quad \dot{\epsilon}_1 + \dot{\epsilon}_2 + \dot{\epsilon}_3 = 0. \quad (23)$$

A logarithmic measure of the dilatation is ϵ_0 . When $a_0 = b_0 = c_0 = 1$ is chosen for the datum state, $\epsilon_0 = \ln J$ follows from Eq. 12. This choice is used henceforth, as are $\alpha_0 = \beta_0 = \gamma_0 = 0$. Such choices imply that the initial state is “undeformed”. Logarithmic squeeze modes are $\epsilon_1, \epsilon_2, \epsilon_3$; these are collectively isochoric, though isolated squeeze modes do not preserve volume. Simple shears are $\epsilon_4, \epsilon_5, \epsilon_6$; these individually and collectively preserve volume.

2.5 Stresses and Stress Power

Denote by $\boldsymbol{\sigma}$, $\mathbf{k}$, $\mathbf{s}$, and $\mathbf{P}$ the Cauchy, Kirchhoff, rotated Kirchhoff, and first Piola–Kirchhoff stress tensors, respectively. In the Cartesian frame $\{\mathbf{e}_i\} = \{\mathbf{e}_J \delta_i^J\}$, these stress tensors are related by

$$\begin{aligned} \boldsymbol{\sigma} &= J^{-1} \mathbf{P} \mathbf{F}^T = J^{-1} \mathbf{k} = J^{-1} \mathbf{Q} \mathbf{s} \mathbf{Q}^{-1} \leftrightarrow \\ \sigma_j^i &= J^{-1} P_K^i F_j^K = J^{-1} k_j^i = J^{-1} Q_\alpha^i s_\beta^\alpha (Q^{-1})_j^\beta. \end{aligned} \quad (24)$$

Components of $\mathbf{s}$ are equal to those of the Kirchhoff stress when the latter is referred to local physical basis $\{\mathbf{g}_\alpha\}$:

$$\mathbf{k} = k_j^i \mathbf{e}_i \otimes \mathbf{e}^j = k_j^i (Q^{-1})_i^\alpha \mathbf{g}_\alpha \otimes Q_\beta^j \mathbf{g}^\beta = s_\beta^\alpha \mathbf{g}_\alpha \otimes \mathbf{g}^\beta. \quad (25)$$

Thus, $\mathbf{s}$ is also referred to as a physical stress tensor. The Cauchy pressure is

$$p = -\frac{1}{3}\text{tr}\boldsymbol{\sigma} = -\frac{1}{3J}\text{tr}\mathbf{k} = -\frac{1}{3J}\text{tr}\mathbf{s}. \quad (26)$$

The stress power per unit reference volume is

$$\mathbf{P} : \dot{\mathbf{F}} = J\boldsymbol{\sigma} : \nabla \mathbf{v} = \mathbf{k} : \nabla \mathbf{v} = \mathbf{s} : \boldsymbol{\eta} = \sum_{i=0}^6 \hat{\tau}_i \dot{\epsilon}_i. \quad (27)$$

The rightmost term is the stress power in terms of rates of strain attributes of Eq. 22 and their scalar stress conjugates.^{5,7,46}

Define $\{\hat{\tau}_i\}$, $i = 0, \dots, 6$ in terms of $s_{\alpha\beta}$ as⁴⁶

$$\{\hat{\tau}_i\} = \begin{Bmatrix} \hat{\tau}_0 \\ \hat{\tau}_1 \\ \hat{\tau}_2 \\ \hat{\tau}_3 \\ \hat{\tau}_4 \\ \hat{\tau}_5 \\ \hat{\tau}_6 \end{Bmatrix} = \begin{Bmatrix} \frac{1}{3}(s_{11} + s_{22} + s_{33}) \\ s_{11} - s_{22} \\ s_{22} - s_{33} \\ s_{33} - s_{11} \\ (a/b)s_{21} - \alpha(a/c)s_{31} \\ (b/c)s_{32} \\ (a/c)s_{31} \end{Bmatrix}. \quad (28)$$

Negative pressure per unit reference volume is $\hat{\tau}_0 = -Jp$. Normal stress differences are $\hat{\tau}_1, \hat{\tau}_2, \hat{\tau}_3$. Shear stresses are $\hat{\tau}_4, \hat{\tau}_5, \hat{\tau}_6$. Normal stress differences conjugate to squeeze modes of strain obey

$$\hat{\tau}_1 + \hat{\tau}_2 + \hat{\tau}_3 = 0. \quad (29)$$

The first term in the rightmost sum of Eq. 27 is the rate of pressure–volume working: $\hat{\tau}_0 \dot{\epsilon}_0 = -p\dot{J}$.

2.6 Conservation Laws and Entropy Production

Let ρ and ρ_0 be spatial and initial mass densities of the material. Body force per unit initial volume is $\mathbf{b}$. Local balance equations for mass, linear momentum, and angular momentum are the usual forms from continuum mechanics:

$$\dot{\rho} + \rho \nabla \cdot \mathbf{v} = 0, \quad \nabla_0 \cdot \mathbf{P} + \mathbf{b} = \rho_0 \mathbf{a}, \quad \mathbf{P}\mathbf{F}^T = \mathbf{F}\mathbf{P}^T. \quad (30)$$

Thermodynamic potentials, measured per unit initial volume, include internal energy density U , Helmholtz free energy density Ψ , and entropy density η , related by

$$U = \Psi + T\eta. \quad (31)$$

Absolute temperature is $T > 0$. The heat flux vector in the reference configuration is $\mathbf{q}$, and a scalar point heat source per unit volume is r . The local balance of energy is

$$\dot{U} = \mathbf{P} : \dot{\mathbf{F}} - \nabla_0 \cdot \mathbf{q} + r. \quad (32)$$

Local entropy production inequalities, the latter following from Eqs. 31 and 32, include

$$\dot{\eta} \geq \frac{1}{T} \left(r + \frac{\mathbf{q} \cdot \nabla_0 T}{T} - \nabla_0 \cdot \mathbf{q} \right), \quad \mathbf{P} : \dot{\mathbf{F}} - \frac{\mathbf{q} \cdot \nabla_0 T}{T} \geq \dot{\Psi} + \dot{T}\eta. \quad (33)$$

2.7 Thermodynamic Potentials

Thermodynamic potentials Ψ or U depend on temperature (T) or entropy (η), internal state variables, and strain attributes. Let $\{\chi_i^\alpha\}$ be a set of dimensionless, strain-like scalar variables associated with viscoelasticity, where $i = 0, \dots, 6$ and $\alpha = 1, \dots, m$, with m a discrete number of relaxation times. Denote by $D \in [0, 1]$ an internal state variable linked to damage processes to be assigned a physical interpretation later. Strain attributes and viscoelastic state variables comprise the column vectors

$$\boldsymbol{\epsilon} = [\epsilon_0, \epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4, \epsilon_5, \epsilon_6]^T, \quad \boldsymbol{\chi}^\alpha = [\chi_0^\alpha, \chi_1^\alpha, \chi_2^\alpha, \chi_3^\alpha, \chi_4^\alpha, \chi_5^\alpha, \chi_6^\alpha]^T. \quad (34)$$

Free and internal energy densities are assigned, respectively, the functional forms below for homogeneous solids:

$$\Psi = \Psi(\boldsymbol{\epsilon}, T, \boldsymbol{\chi}^\alpha, D), \quad U = U(\boldsymbol{\epsilon}, \eta, \boldsymbol{\chi}^\alpha, D). \quad (35)$$

2.8 Constitutive Laws

Using Eq. 35 in the second of Eq. 33 gives the following dissipation inequality:

$$\sum_{i=0}^6 \hat{\tau}_i \dot{\epsilon}_i - \frac{\partial \Psi}{\partial \boldsymbol{\epsilon}} \cdot \dot{\boldsymbol{\epsilon}} - \left(\eta + \frac{\partial \Psi}{\partial \eta} \right) \dot{T} - \sum_{\alpha=1}^m \frac{\partial \Psi}{\partial \boldsymbol{\chi}^\alpha} \cdot \dot{\boldsymbol{\chi}}^\alpha - \frac{\partial \Psi}{\partial D} \dot{D} - \frac{\mathbf{q} \cdot \nabla_0 T}{T} \geq 0. \quad (36)$$

Stress power can be written, using Eqs. 23 and 29, in terms of components of $\dot{\boldsymbol{\epsilon}}$:

$$\sum_{i=0}^6 \hat{\tau}_i \dot{\epsilon}_i = \sum_{i=0}^2 \hat{\tau}_i \dot{\epsilon}_i + (\hat{\tau}_1 + \hat{\tau}_2)(\dot{\epsilon}_1 + \dot{\epsilon}_2) + \sum_{i=4}^6 \hat{\tau}_i \dot{\epsilon}_i. \quad (37)$$

Applying standard arguments for continuum thermodynamic processes, the following thermoelastic constitutive equations are inferred from Eq. 36 to maintain admissibility for stresses

$$\begin{aligned} \hat{\tau}_0 &= \frac{\partial \Psi}{\partial \epsilon_0}, & \hat{\tau}_1 &= \frac{\partial \Psi}{\partial \epsilon_1}, & \hat{\tau}_2 &= \frac{\partial \Psi}{\partial \epsilon_2}, & \hat{\tau}_3 &= \frac{\partial \Psi}{\partial \epsilon_3}, \\ \hat{\tau}_4 &= \frac{\partial \Psi}{\partial \epsilon_4}, & \hat{\tau}_5 &= \frac{\partial \Psi}{\partial \epsilon_5}, & \hat{\tau}_6 &= \frac{\partial \Psi}{\partial \epsilon_6}; \end{aligned} \quad (38)$$

and entropy

$$\eta = -\frac{\partial \Psi}{\partial T}. \quad (39)$$

The first of Eq. 38 corresponds to $p = -J^{-1} \hat{\tau}_0 = -\frac{\partial \Psi}{\partial J}$. Constraints of Eqs. 23 and 29 have not been imposed in the derivation of Eq. 38. Thus, for Eq. 38 to always apply under such constraints, free energy Ψ must be specified consistently with

$$\frac{\partial \Psi}{\partial \epsilon_1} + \frac{\partial \Psi}{\partial \epsilon_2} + \frac{\partial \Psi}{\partial \epsilon_3} = 0. \quad (40)$$

Partial differentiation with respect to ϵ_i is performed with ϵ_j fixed for all $i \neq j$, regardless of Eq. 23. The residual dissipation inequality is

$$-\sum_{\alpha=1}^m \frac{\partial \Psi}{\partial \boldsymbol{\chi}^\alpha} \cdot \dot{\boldsymbol{\chi}}^\alpha - \frac{\partial \Psi}{\partial D} \dot{D} - \frac{\mathbf{q} \cdot \nabla_0 T}{T} \geq 0. \quad (41)$$

The seven-element column vector of stress variables conjugate to strain attributes is

$$\hat{\boldsymbol{\tau}} = [\hat{\tau}_0, \hat{\tau}_1, \hat{\tau}_2, \hat{\tau}_3, \hat{\tau}_4, \hat{\tau}_5, \hat{\tau}_6]^T. \quad (42)$$

Concisely, this is related to strain derivatives of free energy according to

$$\hat{\boldsymbol{\tau}} = \partial_{\boldsymbol{\epsilon}} \Psi = \frac{\partial \Psi}{\partial \boldsymbol{\epsilon}}. \quad (43)$$

If seven components in Eq. 38 are known, then the physical stress tensor $\mathbf{s}$ is, from Eq. 28,

$$\begin{aligned} s_{11} &= \hat{\tau}_0 + \frac{1}{3}(\hat{\tau}_1 - \hat{\tau}_3), & s_{22} &= \hat{\tau}_0 + \frac{1}{3}(\hat{\tau}_2 - \hat{\tau}_1), & s_{33} &= \hat{\tau}_0 + \frac{1}{3}(\hat{\tau}_3 - \hat{\tau}_2), \\ s_{12} &= s_{21} = \frac{b}{a}(\hat{\tau}_4 + \alpha \hat{\tau}_6), & s_{23} &= s_{32} = \frac{c}{b} \hat{\tau}_5, & s_{13} &= s_{31} = \frac{c}{a} \hat{\tau}_6. \end{aligned} \quad (44)$$

Other stress tensors can be obtained from $\mathbf{s}$ using Eq. 24. Thermoelastic constitutive equations in terms of internal energy are derived similarly^{5,7}:

$$\begin{aligned} \hat{\tau}_0 &= \frac{\partial U}{\partial \epsilon_0}, & \hat{\tau}_1 &= \frac{\partial U}{\partial \epsilon_1}, & \hat{\tau}_2 &= \frac{\partial U}{\partial \epsilon_2}, & \hat{\tau}_3 &= \frac{\partial U}{\partial \epsilon_3}, \\ \hat{\tau}_4 &= \frac{\partial U}{\partial \epsilon_4}, & \hat{\tau}_5 &= \frac{\partial U}{\partial \epsilon_5}, & \hat{\tau}_6 &= \frac{\partial U}{\partial \epsilon_6}; & T &= \frac{\partial U}{\partial \eta}. \end{aligned} \quad (45)$$

Constraints analogous to Eq. 40 are imposed on the strain derivatives of internal energy U : $\sum_{i=1,2,3} \partial U / \partial \epsilon_i = 0$.

2.9 Thermoelasticity and Dissipation

Let $c(\boldsymbol{\epsilon}, T, \boldsymbol{\chi}^\alpha, D)$ denote specific heat per unit reference volume at constant deformation, where

$$c = \frac{\partial U}{\partial T} = T \frac{\partial \eta}{\partial T} = -T \frac{\partial^2 \Psi}{\partial T^2}. \quad (46)$$

The following temperature evolution equation can be derived from the local balance of energy:

$$\begin{aligned} c\dot{T} &= - \sum_{\alpha=1}^m (\partial \Psi / \partial \boldsymbol{\chi}^\alpha) \cdot \dot{\boldsymbol{\chi}}^\alpha - (\partial \Psi / \partial D) \dot{D} - \nabla_0 \cdot \mathbf{q} + r \\ &\quad + T \left[(\partial^2 \Psi / \partial \boldsymbol{\epsilon} \partial T) : \dot{\boldsymbol{\epsilon}} + \sum_{\alpha=1}^m (\partial^2 \Psi / \partial T \partial \boldsymbol{\chi}^\alpha) \cdot \dot{\boldsymbol{\chi}}^\alpha + (\partial^2 \Psi / \partial T \partial D) \dot{D} \right]. \end{aligned} \quad (47)$$

Thermal stress coefficients $\boldsymbol{\kappa}$ and Grüneisen coefficients $\mathbf{g}$ are defined as

$$\boldsymbol{\kappa} = -\frac{\partial^2 \Psi}{\partial \boldsymbol{\epsilon} \partial T} = c \mathbf{g}. \quad (48)$$

Isothermal and isentropic, respectively, second-order thermodynamic elastic coefficients (not necessarily constants) are

$$\mathbf{C}^T = \frac{\partial^2 \Psi}{\partial \boldsymbol{\epsilon} \partial \boldsymbol{\epsilon}}, \quad \mathbf{C}^\eta = \frac{\partial^2 U}{\partial \boldsymbol{\epsilon} \partial \boldsymbol{\epsilon}}. \quad (49)$$

Denote thermal expansion coefficients by $\mathbf{A} = \partial \boldsymbol{\epsilon} / \partial T$, with partial differentiation taken at constant stress and constant internal state. Denote specific heat at constant stress by c^s . Relationships can be derived by use of Maxwell-type equalities⁵⁷:

$$\boldsymbol{\kappa} = \mathbf{A} : \mathbf{C}^T, \quad c^S - c = T \mathbf{A} : \boldsymbol{\kappa}, \quad \mathbf{C}^\eta = \mathbf{C}^T + \frac{T}{c} \boldsymbol{\kappa} \otimes \boldsymbol{\kappa}. \quad (50)$$

Define conjugate thermodynamic forces to internal state variables as the negative derivatives

$$\bar{\zeta}^\alpha = -\frac{\partial \Psi}{\partial \chi^\alpha}, \quad \bar{F} = -\frac{\partial \Psi}{\partial D}. \quad (51)$$

Substitution of Eqs. 48 and 51 into Eq. 47 gives the energy balance in a more compact form:

$$c \dot{T} = -T \boldsymbol{\kappa} : \dot{\boldsymbol{\epsilon}} + \sum_{\alpha=1}^m [\bar{\zeta}^\alpha - T(\partial \bar{\zeta}^\alpha / \partial T)] \cdot \dot{\chi}^\alpha + [\bar{F} - T(\partial \bar{F} / \partial T)] \dot{D} - \nabla_0 \cdot \mathbf{q} + r. \quad (52)$$

Assume that dissipation from conduction is always non-negative in Eqs. 33 and 41. The internal dissipation inequality and entropy production rate are then

$$\mathfrak{D} = \sum_{\alpha=1}^m \bar{\zeta}^\alpha \cdot \dot{\chi}^\alpha + \bar{F} \dot{D} \geq 0, \quad T \dot{\eta} = \mathfrak{D} - \nabla_0 \cdot \mathbf{q} + r. \quad (53)$$

A complete constitutive model requires specification of functions Ψ or U in Eq. 35 as well as kinetic equations for (rates of) damage D and viscoelastic internal state variables $\{\chi^\alpha\}$, consistent with positive internal dissipation $\mathfrak{D}$ in Eq. 53. These are established for lung tissue subsequently in this work.

3. Free Energy Formulation for Lung Tissue Mechanics

A constitutive formulation for static and dynamic loading of the lung based on the Helmholtz free energy potential Ψ is developed. This model is preferred for tensile stress states and low to moderate strain rates, and is ideal for isothermal problems. It does not account for compressibility of the air inside the lung, and thus is not appropriate for dynamic compression problems including high-frequency wave propagation and shock loading. The internal energy-based model developed in Section 5 addresses the latter scenarios and is ideal for adiabatic problems.

3.1 General Form

The following form of Ψ in Eq. 35 is invoked:

$$\begin{aligned}\Psi(\epsilon, T, \chi^1, D) &= f(D)[\Psi_0(\epsilon, T, \chi^1)] + \psi(T) \\ &= f(D)[\Psi_0^\infty(\epsilon, T) + \Upsilon_0(\epsilon, \chi^1)] + \psi(T).\end{aligned}\tag{54}$$

This function is representative of one of several first introduced in recent works^{5,7} wherein the lung was not addressed specifically. Equilibrium thermoelastic free energy, including strain energy and coupling from thermal expansion, is Ψ_0^∞ . Configurational free energy associated with viscoelasticity is Υ_0 . As is discussed in detail in Section 3.3, attention is limited to consideration of a single viscous relaxation mode corresponding to $\alpha = 1$ in Eq. 35. The damage degradation function is f . The purely thermal free energy accounting for specific heat is ψ ; this is assumed unaffected by damage.

3.2 Thermoelastic Strain Energy

A material element is considered to occupy a stress-free reference state when undeformed and in the absence of surface tension at the air–liquid interface, wherein its mass density is ρ_0 and temperature is T_0 . This definition of reference state is consistent with that of Fung.^{19,20} Surface tension can be effectively eliminated by washing the tissue in saline; naturally moist lung, when expanded to the same volume as a saline-rinsed lung, will require a tensile pressure in the reference state to offset the effect of surface tension and prevent the tissue from contracting.

Equilibrium thermoelastic free energy depends explicitly on three functions of the

strain attributes of Eq. 22, denoted by ξ_i , $i = 1, 2, 3$. The first accounts for volume changes and is equal to the first attribute itself, the second accounts for squeeze deformations, and the third accounts for simple shears:

$$\begin{aligned}\xi_1 &= \epsilon_0 = \ln J, \\ \xi_2 &= (\epsilon_1)^2 + (\epsilon_2)^2 + (\epsilon_3)^2, \\ \xi_3 &= (\epsilon_4)^2 + (\epsilon_5)^2 + (\epsilon_6)^2 = \alpha^2 + \beta^2 + \gamma^2.\end{aligned}\tag{55}$$

This is a three-mode isotropic theory in the sense of Freed,^{5,7,46,54} where the material may respond differently under pure shear (squeeze) and simple shear. As such, the material is not isotropic in the conventional sense of invariants or group theory; its linearization is reminiscent of a classical linear elastic solid with cubic symmetry.^{5,7} It is demonstrated later how this model effectively represents the more compliant response of the lung in simple shear (*i.e.*, torsion) relative to pure shear under bi-axial or triaxial extension. Dependence of strain energy on functions in Eq. 55 is sufficient but neither unique nor necessary, to ensure three-mode isotropic symmetry in the sense defined in prior work.^{5,7,46} A useful property of $\Psi_0^\infty(\xi_1, \xi_2, \xi_3, T)$ is that the thermoelastic free energy analog of Eq. 40 is identically satisfied via Eq. 23.

Thermoelastic strain energy density per unit reference volume is prescribed:

$$\begin{aligned}\Psi_0^\infty(\xi_1, \xi_2, \xi_3, T) &= \frac{1}{2} \frac{B_0^T}{c_1} \{ \exp[c_1(\xi_1)^2] - 1 \} + K_0 \xi_1 - A_0 B_0^T \xi_1 (T - T_0) \\ &\quad + 3 \frac{\mu_0}{c_2} \{ \exp[c_2 \xi_2] - 1 \} \left[1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right] \\ &\quad + \frac{1}{2} \frac{G_0}{c_3} \{ \exp[c_3 \xi_3] - 1 \} \left[1 - \frac{B_0^T}{G_0} G'_0 \xi_1 \right].\end{aligned}\tag{56}$$

Denoted by B_0^T is the isothermal bulk modulus, μ_0 the shear modulus for pure shear, and G_0 the shear modulus for simple shear, all corresponding to tangent moduli at the undeformed reference state for equilibrium quasi-static deformation (no viscoelasticity), in the absence of damage. The isothermal pressure derivatives of the shear moduli at the reference state are μ'_0 and G'_0 :

$$\mu'_0 = \left[\frac{d}{dp} \mu \right] \Big|_0 = \left[\frac{d}{dp} \left(\frac{1}{6} \frac{\partial^2 \Psi}{\partial \epsilon_i^2} \right) \right] \Big|_{T=T_0, \epsilon=0}, \quad (i = 1, 2, 3); \tag{57}$$

$$G'_0 = \left[\frac{d}{dp} G \right] \Big|_0 = \left[\frac{d}{dp} \left(\frac{\partial^2 \Psi}{\partial \epsilon_i^2} \right) \right] \Big|_{T=T_0, \epsilon=0}, \quad (i = 4, 5, 6). \quad (58)$$

Dimensionless higher-order elastic constants corresponding to dilatation, squeeze, and simple shear are c_1 , c_2 , and c_3 . The associated exponential stiffening is reminiscent of Fung's formulation for soft biological tissues,²³ but constants are deemed physical and unique rather than phenomenological.^{46,52} The equilibrium tangent isothermal bulk modulus corresponding to Eq. 56 is

$$B^T = - \left(J \frac{\partial p}{\partial J} \right) \Big|_{T=T_0} = \frac{B_0^T}{J} [1 + \ln J(2c_1 \ln J - 1)] \exp\{c_1(\ln J)^2\}, \quad (59)$$

with pressure derivative at the ambient state computed as^{5,7}

$$\begin{aligned} B_0^T &= \left[\frac{d}{dp} B^T \right] \Big|_0 = \left[\frac{d}{dp} \left(-J \frac{\partial p}{\partial J} \right) \right] \Big|_{T=T_0, \epsilon=0} \\ &= \left[\frac{d}{dp} \left(\frac{\partial}{\partial \ln J} \frac{\partial \Psi}{\partial J} \right) \right] \Big|_{T=T_0, \epsilon=0} \\ &= 2. \end{aligned} \quad (60)$$

The volumetric coefficient of thermal expansion is A_0 , and the lone nonzero thermal stress coefficient in Eq. 50 is $\kappa_0 = A_0 B_0$. Lagrangian surface tension in the reference state is K_0 . Thermal free energy is defined as follows, with c_0 the specific heat at constant volume:

$$\psi(T) = -c_0 [T \ln(T/T_0) - (T - T_0)]. \quad (61)$$

The second term in square braces, often omitted,^{5,7,57} produces the simultaneous conditions that $\partial\psi/\partial T = 0$ and $\psi = 0$ at $T = T_0$. Whether included or not, $c = c_0 = \text{constant}$ follows from Eq. 46.

The general formula Eq. 56 is valid for lung in air and saline. When saline-washed, values of the bulk thermoelastic constants B_0^T and c_1 differ,^{24,35} with the saline-treated lung more compliant, and $K_0 = 0$ by construction, since surface tension vanishes.

In the limit that $c_i \rightarrow 0$ ($i = 1, 2, 3$), the free energy in Eq. 56 becomes quadratic in volumetric and shear strains. When $\mu_0 = G_0$, a two-mode theory^{5,46,54} is obtained,

and the equilibrium elastic compliances of the material in pure shear and simple shear are identical for small deformations. In the limit that deformations (strains and rotations) are small, then the response in this two-mode case reduces to that of classical isotropic linear elasticity.^{5,7}

3.3 Configurational Free Energy

Viscoelasticity is addressed via a configurational energy Υ_0 and evolution equations for viscous stress components that ensure non-negative dissipation (*i.e.*, thermodynamic consistency). The general form derived in recent work,^{5,7} which in turn extends a second Piola–Kirchhoff stress-based formulation of Holzapfel and Simo^{48,49} to QR kinematics and conjugate stress–strain attributes, is

$$\Upsilon_0(\boldsymbol{\epsilon}, T, \boldsymbol{\chi}^\alpha) = \sum_{\alpha=1}^m \left[\frac{1}{2} \mu_\alpha(T) |\boldsymbol{\chi}^\alpha|^2 - \partial_{\boldsymbol{\epsilon}} \Psi_0^\alpha(\boldsymbol{\epsilon}, T) \cdot \boldsymbol{\chi}^\alpha + \Psi_0^\alpha(\boldsymbol{\epsilon}, T) \right]. \quad (62)$$

Denoted by $\Psi_0^\alpha = \Psi_0^\alpha(\boldsymbol{\epsilon}, T)$ is a free energy function that corresponds to relaxation process $\alpha = 1, \dots, m$ with time scale $t_\alpha > 0$. Denoted by $\mu_\alpha \geq 0$ is a temperature-dependent parameter for each α with dimensions of elastic stiffness. Corresponding viscosity for mode α is $\nu_\alpha = \mu_\alpha t_\alpha$.

The kinetic model for viscous stresses ζ^α , motivated by the standard viscoelasticity model of 1) a spring element for equilibrium response in parallel with 2) m Maxwell (*i.e.*, spring-dashpot) elements, is expressed in tensor form in early work^{48,49} and stress–strain attribute form in more recent work.^{5,7} Recall from Eq. 51 that internal stresses $\bar{\zeta}^\alpha = \partial \Psi / \partial \boldsymbol{\chi}^\alpha$ are thermodynamic conjugates to corresponding internal strains $\boldsymbol{\chi}^\alpha$ in the presence of damage function $f(D)$ that affects $\Psi = f \Psi_0$ in Eq. 54. Define conjugate internal stresses for the free energy Ψ_0 not modified by damage function f as^{5,7}

$$\zeta^\alpha = -\frac{\partial \Psi_0}{\partial \boldsymbol{\chi}^\alpha} = -\frac{\partial \Upsilon_0}{\partial \boldsymbol{\chi}^\alpha}. \quad (63)$$

General differential equations for viscous stresses and initial conditions (*i.e.*, instantaneous internal stresses measured when a load is applied at $t = 0$) corresponding to Eq. 62 are

$$\dot{\zeta}^\alpha + \frac{1}{t_\alpha} \zeta^\alpha = \frac{d}{dt} [\partial_{\boldsymbol{\epsilon}} \Psi_0^\alpha] - \boldsymbol{\omega}^\alpha, \quad \zeta^\alpha|_{t=0} = \zeta_0^\alpha = \partial_{\boldsymbol{\epsilon}} \Psi_0^\alpha|_{\boldsymbol{\epsilon}, T \text{ at } t=0}. \quad (64)$$

The coupling function ω^α accounts for temperature dependence of μ_α and is omitted for the application herein. The solution of Eq. 64 can be written in the form of a convolution integral for each α :

$$\zeta^\alpha(t) = \exp\left(\frac{-t}{t_\alpha}\right) \zeta_0^\alpha + \int_{0+}^t \exp\left[\frac{-(t-s)}{t_\alpha}\right] \left(\frac{d}{ds} [\partial_\epsilon \Psi_0^\alpha(s)] - \omega^\alpha(s)\right) ds. \quad (65)$$

Internal strains and their rates obey

$$\chi^\alpha = \frac{1}{\mu_\alpha} \left[\frac{\partial \Psi_0^\alpha}{\partial \epsilon} - \zeta^\alpha \right], \quad \dot{\chi}^\alpha = \frac{1}{\nu_\alpha} \zeta^\alpha, \quad (\nu_\alpha = \mu_\alpha t_\alpha > 0). \quad (66)$$

The initial internal stress is calculated in Eq. 64, from which the internal strain vanishes at $t = 0$ according to the first of Eq. 66:

$$\zeta_0^\alpha = \frac{\partial \Psi_0^\alpha}{\partial \epsilon} \Big|_{t=0}, \quad \chi_0^\alpha = \chi^\alpha|_{t=0} = 0. \quad (67)$$

The condition imposed for thermodynamic equilibrium is that internal stresses vanish at infinite time, which leads to the following limiting thermoelastic relations, from Eq. 66:

$$\zeta_\infty^\alpha = \zeta^\alpha|_{t \rightarrow \infty} = 0 \quad \Rightarrow \quad \chi_\infty^\alpha = \chi^\alpha|_{t \rightarrow \infty} = \frac{1}{\mu_\alpha} \frac{\partial \Psi_0^\alpha}{\partial \epsilon}, \quad \dot{\chi}_\infty^\alpha = 0. \quad (68)$$

Neither configurational energy Υ_0 nor internal variables χ^α need vanish as $t \rightarrow \infty$, and the latter usually do not. Internal dissipation, without modification by $f(D)$, is of the form

$$\sum_{\alpha=1}^m \zeta^\alpha \cdot \dot{\chi}^\alpha = \sum_{\alpha=1}^m \frac{1}{\nu_\alpha} \zeta^\alpha \cdot \zeta^\alpha = \sum_{\alpha=1}^m \frac{1}{\mu_\alpha t_\alpha} |\zeta^\alpha|^2 \geq 0. \quad (69)$$

The energy invoked here for the lung is one of several forms considered in a general framework recently developed by the authors.^{5,7} For simplicity and sufficiency in forthcoming applications, the configurational energy accounts for only a single relaxation process with $\alpha = 1$ and complementary relaxation time t_1 . For similar reasons, temperature coupling addressed in more general frameworks^{5,7,48} is omitted, so now $\mu_1 = \text{constant}$, irrespective of T . The only independent parameters, beyond those introduced already in Section 3.2, entering this model are $\beta_1 = \mu_1/B_0^T$ and t_1 .

The following quadratic form of relaxation energy, which does not contain t_1 that only affects kinetics, is invoked^{5,7,48}:

$$\Psi_0^1(\boldsymbol{\epsilon}) = \frac{1}{2}\mu_1|\boldsymbol{\epsilon}|^2 = \frac{1}{2}\mu_1[(\ln J)^2 + \epsilon_1^2 + \epsilon_2^2 + \epsilon_3^2 + \alpha^2 + \beta^2 + \gamma^2], \quad \frac{\partial \Psi_0^1}{\partial \boldsymbol{\epsilon}} = \mu_1 \boldsymbol{\epsilon}. \quad (70)$$

This viscoelastic energy is approximately proportional to linear hyperelastic strain energy density in the limit of small deformations when bulk and shear elastic constants are similar in magnitude. Then Eq. 62 produces

$$\Upsilon_0[\boldsymbol{\epsilon}, \boldsymbol{\chi}^1] = \frac{1}{2}\mu_1[|\boldsymbol{\chi}^1|^2 - 2\boldsymbol{\epsilon} \cdot \boldsymbol{\chi}^1 + |\boldsymbol{\epsilon}|^2] = \frac{1}{2}\mu_1|\boldsymbol{\chi}^1 - \boldsymbol{\epsilon}|^2. \quad (71)$$

Viscoelastic kinetics obey, for the present specific formulation wherein $\alpha = m = 1$,

$$\dot{\boldsymbol{\zeta}}^1 + \frac{1}{t_1}\boldsymbol{\zeta}^1 = \mu_1\dot{\boldsymbol{\epsilon}}, \quad \boldsymbol{\zeta}^1|_{t=0} = \boldsymbol{\zeta}_0^1 = \partial_{\boldsymbol{\epsilon}}\Psi_0^1|_{\boldsymbol{\epsilon}, T \text{ at } t=0} = \mu_1\boldsymbol{\epsilon}_0; \quad (72)$$

$$\boldsymbol{\zeta}^1(t) = \mu_1\boldsymbol{\epsilon}_0 \exp\left(\frac{-t}{t_1}\right) + \mu_1 \int_{0^+}^t \exp\left[\frac{-(t-s)}{t_1}\right] \left(\frac{d}{ds}[\boldsymbol{\epsilon}(s)]\right) ds. \quad (73)$$

Internal strains and their rates are

$$\boldsymbol{\chi}^1 = \boldsymbol{\epsilon} - \frac{\boldsymbol{\zeta}^1}{\mu_1}, \quad \dot{\boldsymbol{\chi}}^1 = \frac{\dot{\boldsymbol{\zeta}}^1}{\nu_1}, \quad (\nu_1 = \mu_1 t_1 = \beta_1 B_0^T t_1 > 0). \quad (74)$$

The rate of dissipated energy in Eq. 69 reduces to $|\dot{\boldsymbol{\zeta}}^1|^2/\nu_1 = \nu_1|\dot{\boldsymbol{\chi}}^1|^2 \geq 0$. At equilibrium, Eq. 68 with Eq. 70 produces $\boldsymbol{\chi}_\infty^1 = \boldsymbol{\epsilon}$. The constraint below on squeeze terms of internal strain is imposed, analogously to Eq. 23, such that Eq. 40 will be obeyed:

$$\chi_1^\alpha + \chi_2^\alpha + \chi_3^\alpha = 0. \quad (75)$$

Equation 75 has been posited more generally^{5,7} for all α . Here, $\alpha = 1$.

3.4 Damage

A continuum damage mechanics model is used, following those introduced in a more generic finite deformation setting.^{5,7,40,43} Recall that the internal state variable representative of deleterious processes that affect mechanical and/or biological functions is denoted by the internal state variable $D \in [0, 1]$. The present theory requires specification of the degradation function $f(D)$ in Eq. 54 and a constitutive equation or evolution law specifying how $D(\mathbf{X}, t)$ may change during the history

of loading at a material point $\mathbf{X}$. The thermodynamic driving force for damage of Eq. 51 is, from Eq. 54,

$$\bar{F} = -\frac{df}{dD}\Psi_0 = -f'(\Psi_0^\infty + \Upsilon_0). \quad (76)$$

The following restrictions are imposed to ensure damage evolution is physically and thermodynamically irreversible:

$$\left(\dot{D} \geq 0, \Psi_0 \dot{D} \geq 0, f' \leq 0\right) \Rightarrow \bar{F} \dot{D} \geq 0. \quad (77)$$

The present model applies the standard linear degradation function of continuum damage mechanics^{40,41,57,58}:

$$f = 1 - D, \quad f' = df/dD = -1 \quad \Rightarrow \quad \bar{F} = \Psi_0. \quad (78)$$

Free energy of the undamaged material Ψ_0 is thermodynamically conjugate to the rate of damage growth. A canonical constitutive assumption is that D , or its rate, should be influenced by Ψ_0 . The following dimensionless transformation of Ψ_0 is defined:

$$Z(\Psi_0) = \begin{cases} \left[2 \left| \frac{\Psi_0^{V\infty} + \Upsilon_0^V}{B_0^T} + \frac{\Psi_0^{\mu\infty} + \Upsilon_0^\mu}{\mu_0} + \frac{\Psi_0^{G\infty} + \Upsilon_0^G}{G_0} \right| \right]^{1/2} & (\text{if } \Psi_0 \geq 0), \\ 0 & (\text{if } \Psi_0 < 0). \end{cases} \quad (79)$$

Strictly, the argument of Z is a function of a partitioning and normalization of Ψ_0 , rather Ψ_0 itself that is used only suggestively for meaning in the notation on the left side of Eq. 79. Thermoelastic free energy is partitioned in contributions from volume change, squeeze, and simple shear as

$$\Psi_0^\infty = \Psi_0^{V\infty} + \Psi_0^{\mu\infty} + \Psi_0^{G\infty}; \quad (80)$$

$$\begin{aligned} \Psi_0^{V\infty} &= \frac{1}{2} \frac{B_0^T}{c_1} \{ \exp[c_1(\xi_1)^2] - 1 \} + K_0 \xi_1 - A_0 B_0^T \xi_1 (T - T_0), \\ \Psi_0^{\mu\infty} &= 3 \frac{\mu_0}{c_2} \{ \exp[c_2 \xi_2] - 1 \} \left[1 - \frac{B_0^T}{\mu_0} \mu_0' \xi_1 \right], \\ \Psi_0^{G\infty} &= \frac{1}{2} \frac{G_0}{c_3} \{ \exp[c_3 \xi_3] - 1 \} \left[1 - \frac{B_0^T}{G_0} G_0' \xi_1 \right]. \end{aligned} \quad (81)$$

Analogously, configurational energy is partitioned as

$$\Upsilon_0 = \Upsilon_0^V + \Upsilon_0^\mu + \Upsilon_0^G; \quad (82)$$

$$\Upsilon_0^V = \frac{\mu_1}{2}(\epsilon_0 - \chi_0^1)^2, \quad \Upsilon_0^\mu = \frac{\mu_1}{2} \sum_{i=1,2,3} (\epsilon_i - \chi_i^1)^2, \quad \Upsilon_0^G = \frac{\mu_1}{2} \sum_{i=4,5,6} (\epsilon_i - \chi_i^1)^2. \quad (83)$$

The maximum value of normalized free energy attained at a material point $\mathbf{X}$ over a time history $s \in (-\infty, t]$ is used subsequently in the damage evolution law:

$$Z_m(\mathbf{X}, t) = \max_{s \in (-\infty, t]} Z(\mathbf{X}, s). \quad (84)$$

Similar quantities were introduced for the same purpose (*i.e.*, to drive evolution of damage variables) in continuum damage mechanics models for polymers and biological fibers.^{40,42}

Two options are noted for specification of damage kinetics. The first, as enacted previously,^{5,7,40,42,43} invokes a rate equation (*i.e.*, an ordinary differential equation for $\dot{D}$) based on the appropriate driving force and consistent with requirements in Eq. 77. Time integration then yields the function $D(\mathbf{X}, t)$ for $t \geq 0$. An alternative, and perhaps simpler, approach proves more effective for the present model of damage in the lung, whereby transient data on time scales of mechanical damage evolution necessary for adequate parameterization of rate laws are scarce, if not nonexistent. The following two-parameter damage function is invoked:

$$D(\mathbf{X}, t) = \begin{cases} \exp \left[\frac{\alpha_D \{Z_m(\mathbf{X}, t) - Z_0\}}{Z_0} \right] - 1 & (\text{if } \frac{Z_m}{Z_0} \in [1, 1 + \frac{\ln 2}{\alpha_D}]), \\ 0 & (\text{if } \frac{Z_m}{Z_0} < 1), \\ 1 & (\text{if } \frac{Z_m}{Z_0} > 1 + \frac{\ln 2}{\alpha_D}). \end{cases} \quad (85)$$

Dimensionless scalar parameters are the threshold thermodynamic force for damage initiation $Z_0 > 0$ and $\alpha_D \geq 0$ that controls the extent of damage progression with force exceeding this threshold. The larger the value of α_D , the greater the reduction in mechanical stiffness with increasing normalized strain energy exceeding the initiation threshold. Constitutive equation Eq. 85 obeys $D \in [0, 1]$ with $D \rightarrow 1$

as $Z_m \rightarrow \infty$. The material time derivative of Eq. 85 is

$$\dot{D} = \begin{cases} \frac{\alpha_D}{Z_0}(1 + D)\frac{dZ_m}{dt} & (\text{if } \frac{Z_m}{Z_0} \in \left[1, 1 + \frac{\ln 2}{\alpha_D}\right]), \\ 0 & (\text{otherwise}). \end{cases} \quad (86)$$

Since Z_m can only remain constant or increase with time according definition Eq. 84 and since α_D is non-negative, $\dot{D} \geq 0$. Furthermore, $\dot{Z}_m > 0$ only in the domain $\Psi_0 \geq 0$ according to the definition of Z in Eq. 79. Thus, $\Psi_0 \dot{D} \geq 0$. The model obeys all restrictions imposed in Eq. 77 and non-negative dissipation is ensured.

3.5 Stresses, Entropy, and Temperature

Derivatives of the free energy function comprising the terms for thermoelasticity, viscoelasticity, and damage set forth in Sections 3.2, 3.3, and 3.4, respectively, produce stress components and entropy according to Eqs. 38 and 39. Equilibrium free energy Ψ_0^∞ of Eq. 56, thermal energy ψ of Eq. 61, configurational energy of Υ_0 of Eq. 71, and damage degradation function f of Eq. 78 are substituted into Eq. 54 to yield the following total free energy function:

$$\begin{aligned} \Psi(\xi_1, \xi_2, \xi_3, T, \chi^1, D) = (1 - D) & \left[\frac{1}{2} \frac{B_0^T}{c_1} \{ \exp[c_1(\xi_1)^2] - 1 \} \right. \\ & + K_0 \xi_1 - A_0 B_0^T \xi_1 (T - T_0) \\ & + 3 \frac{\mu_0}{c_2} \{ \exp[c_2 \xi_2] - 1 \} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right) \\ & + \frac{1}{2} \frac{G_0}{c_3} \{ \exp[c_3 \xi_3] - 1 \} \left(1 - \frac{B_0^T}{G_0} G'_0 \xi_1 \right) \\ & \left. + \frac{1}{2} \mu_1 |\epsilon - \chi^1|^2 \right] - c_0 [T \ln(T/T_0) - (T - T_0)]. \end{aligned} \quad (87)$$

Stress components conjugate to strain attributes obtained from Eqs. 38 and 87 are

$$\begin{aligned}
\hat{\tau}_0 &= (1 - D)B_0^T [\epsilon_0 \exp(c_1 \xi_1^2) - 3\mu'_0 \xi_2 - \frac{1}{2}G'_0 \xi_3 \\
&\quad - A_0(T - T_0) + \beta_1(\epsilon_0 - \chi_0^1) + K_0/B_0^T], \\
\hat{\tau}_1 &= 6(1 - D)\mu_0 [\epsilon_1 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2(\epsilon_1 - \chi_1^1)], \\
\hat{\tau}_2 &= 6(1 - D)\mu_0 [\epsilon_2 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2(\epsilon_2 - \chi_2^1)], \\
\hat{\tau}_3 &= 6(1 - D)\mu_0 [\epsilon_3 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2(\epsilon_3 - \chi_3^1)], \\
\hat{\tau}_4 &= (1 - D)G_0 [\epsilon_4 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3(\epsilon_4 - \chi_4^1)], \\
\hat{\tau}_5 &= (1 - D)G_0 [\epsilon_5 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3(\epsilon_5 - \chi_5^1)], \\
\hat{\tau}_6 &= (1 - D)G_0 [\epsilon_6 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3(\epsilon_6 - \chi_6^1)].
\end{aligned} \tag{88}$$

Three dimensionless stiffness ratios β_i ($i = 1, 2, 3$) have been introduced in Eq. 88:

$$\beta_1 = \frac{\mu_1}{B_0^T}, \quad \beta_2 = \frac{\mu_1}{6\mu_0} = \beta_1 \frac{B_0^T}{6\mu_0}, \quad \beta_3 = \frac{\mu_1}{G_0} = \beta_1 \frac{B_0^T}{G_0}. \tag{89}$$

None are independent parameters if μ_1 and the thermoelastic constants are known; alternatively, prescription of any one of the β_i constrains the other two along with μ_1 . Instantaneous dynamic moduli at the reference state ($\xi_i = 0$, $D = 0$, $\chi = \mathbf{0}$) are $B_0^T \cdot (1 + \beta_1)$, $\mu_0 \cdot (1 + \beta_2)$, and $G_0 \cdot (1 + \beta_3)$. Cauchy stress σ can be obtained from $\hat{\tau}_i$ via sequential substitution into Eq. 44 for s and then s into Eq. 24 for σ , presuming $\mathbf{F} = \mathbf{Q}\mathbf{R}$ is known. Verification is straightforward that, with Eq. 75 imposed, the constraints $\sum_{i=1,2,3} \hat{\tau}_i = 0$ of Eqs. 29 and 40 hold.

Entropy density per unit reference volume is determined from Eq. 39 as follows, noting that Υ_0 does not depend on T :

$$\eta = -\frac{\partial}{\partial T} [(1 - D)\Psi_0^\infty + \psi] = (1 - D)A_0 B_0^T \epsilon_0 + c_0 \ln(T/T_0). \tag{90}$$

Total dissipation in Eq. 53 becomes

$$\mathfrak{D} = (1 - D)\beta_1 B_0^T t_1 |\dot{\chi}^1|^2 + \bar{F} \dot{D} \geq 0, \tag{91}$$

where the (in)equality is verified by the combination of Eqs. 69 and 77 with $\nu_1 = \beta_1 B_0^T t_1 > 0$. The energy balance Eq. 52 in the form of a temperature rate equation

reduces to

$$c_0 \dot{T} = \mathfrak{D} + A_0 B_0 T (\epsilon_0 \dot{D} - \dot{\epsilon}_0) - \nabla_0 \cdot \mathbf{q} + r. \quad (92)$$

Summarizing, thermodynamic potentials and kinetic equations have been formulated consistently with laws of continuum mechanics and thermodynamics, notably those for energy conservation and entropy production. All parameters entering the foregoing theory of Section 3 are obtained for the lung in the following Section 4.

4. Static and Dynamic Loading

The constitutive model of Section 3 is applied to describe several experimental data sets from different loading protocols. These include static triaxial extension in Section 4.1, spherical stress relaxation in Section 4.2, and dynamic torsion and simple shear in Section 4.3. Results demonstrate how the model is able to represent experimental data with relatively few, physically meaningful, parameters. Loadings involve tensile and/or shear deformations that do not require consideration of air compressibility within the lung that is addressed later for rapid compression problems via the theory in Section 5.

Most of the calculations are applied toward describing the response of dog lung, for which data appear most abundant.^{1,19–21,34,59–61} Unfortunately, a comprehensive data set for all loading protocols needed to fully parameterize the model is unavailable in a single published study on a particular animal, or for any single species. Hence, for some cases, available data or observations on sheep, rabbit, and pig lung (*i.e.*, porcine data) are substituted when the need arises, in the absence of data on dog lung. Differences among certain lung tissue properties (*e.g.*, linearized elastic moduli valid for small deformations) for many mammals are not excessive,^{1,35,61–66} perhaps because alveolar dimensions, including thickness of septal walls and membranes, are comparable for animals of similar mass. Humans, however, tend to exhibit a stiffer mechanical response.⁶⁷ The procedures presented in what follows can be reapplied to a consistent data set on a single animal or species when such experimental data become available.

Recall that a three-mode theory based on QR kinematics is proposed in Section 3. The mechanical response (*e.g.*, stress–strain) is anisotropic in the sense that it may differ in pure shear (*i.e.*, squeeze) versus simple shear. Anisotropic (*e.g.*, orthotropic)

models have been used elsewhere²¹ to fit static biaxial extension data. However, the parenchyma itself is thought most realistically viewed as an isotropic material in the sense that no preferred directions correlate with its microstructure, which consists primarily of alveoli of approximately dodecahedral or 14-hedral shape.^{1,68,69}

Application of the three-mode theory requires appropriate selection of the global coordinate basis vectors $\mathbf{e}_i = \delta_i^J \mathbf{e}_J$ ($i, J = 1, 2, 3$) such that the response is uniquely determined, since different bases result in different partitioning of deformation into squeeze and simple shear for general imposed deformations. Proper pivoting of the coordinate axes seeks to achieve this, as explained by Freed.⁴⁶ Let the $\{\mathbf{e}_i\}$ be rotated by an amount $\mathbf{Q}$. Then for the proper choice of basis, the direction aligned with the rotated 1-axis and the direction normal to the rotated 1-2-plane remain invariant under the transformation $\mathbf{R}$. Given a deformation gradient $\mathbf{F}$ in an arbitrary global coordinate system, the pivoting algorithm essentially relabels the coordinate axes at each $\mathbf{X}$ such that $\mathbf{F}$ in its new coordinate system has minimal lower triangular component(s).

Under small deformation, lung tissue responds reasonably isotropically, as indicated by static and wave propagation experiments.^{32,33,35,61,66,69,70} However, some anisotropy may arise under large deformations due to rearrangement (*e.g.*, rotation) of microstructure features.^{21,69} In the present model, which invokes a three-mode response^{46,54} in the QR kinematic framework consisting of dilatation, squeeze, and simple shear, isotropy under small deformations is imposed by setting the simple shear modulus equal to the squeeze modulus: $G_0 = \mu_0$.^{5,7} This ensures that all directions are pure mode directions for linear elastic waves.⁵² However, different stress–strain responses are possible under finite pure shear and simple shear; accordingly, $c_2 \neq c_3$ in the present thermoelastic theory. The pressure derivative of the squeeze modulus is assumed equal to that of the shear modulus ($\mu'_0 = G'_0$) since only the latter appears to have been measured directly from shear wave propagation tests. Values are $G'_0 \approx -0.7$,^{35,65,70} where the negative sign indicates that shear stiffness increases with increasing tension. This value is listed in Table 1 along with others for the free energy-based model, where all parameters besides G'_0 are calibrated to experimental data as explained in the remainder of Section 4.

Table 1 Parameters for free energy-based response: elasticity, viscoelasticity, and damage

Property [units]	Value	Description
B_0^T, G_0 [kPa]	2.5, 1.0	isothermal bulk and shear modulus* in air
	2.0, 1.0	isothermal bulk and shear modulus in saline
c_1, c_2, c_3	3, 10, 0.5	nonlinear bulk, squeeze, simple shear constants in air
	2, 10, 0.5	nonlinear bulk, squeeze, simple shear constants, saline
G'_0	-0.7	initial pressure derivative of shear modulus**
K_0 [kPa]	0.2	reference surface tension in air
Z_0, α_D	0.64, 0.0167	normalized injury threshold, damage growth factor
β_1, t_1 [-,s]	0.6, 0.5	viscoelastic stress multiplier, relaxation time constant
* $\mu_0 = G_0$		
** $\mu'_0 = G'_0$		

4.1 Triaxial Extension

Cauchy pressure is defined as positive for compressive loading. Under triaxial extension, of which spherical extension is a subset, $p < 0$ and $J > 1$. The definition of the bulk modulus in Eq. 59 is consistent with this sign convention. Often the opposite convention is used in the lung mechanics literature, where pressure is defined as positive when the tissue is under tensile stress, and whereby the negative sign would be removed from the definition of the always non-negative bulk modulus. In the present report, following Fung and Vawter,^{1,19,20,22} pressure p supported by a material element is presumed equal to the negative of the transpulmonary pressure p_{tp} , where the latter is positive under tensile loading. By definition,^{1,19,20,22} transpulmonary pressure is the difference between the internal airway pressure in the lung (or inside an internally pressurized element of lung tissue) denoted by p_a and the pleural pressure p_{pl} , which is the external pressure acting on the outer surface of the lung or an element of it:

$$-p = p_{tp} = p_a - p_{pl}. \quad (93)$$

Effects of tension and curvature in the pleural membrane^{19,20} are embedded in p_{pl} and not are resolved explicitly here. Both p_a and p_{pl} are positive in compression. In vivo, pressure in the intrapleural space is less than airway pressure (*i.e.*, negative relative to atmosphere) to keep the lung from collapsing due to its intrinsic surface tension. Under normal breathing cycles, lung tissue remains perpetually in a tensile stress state,⁷¹ whereby the tissue pressure (elastic + surface tension contributions) $p < 0$ and $p_{tp} > 0$. Typically, $p_a \approx 1 \text{ atm} = 101.3 \text{ kPa} = 1.033 \times 10^3 \text{ cm H}_2\text{O}$.

Only isothermal problems are considered in this section. Thermal properties such as specific heat c_0 and thermal expansion coefficient A_0 are defined later in Section 6 where they are first used in dynamic problems. The low strain-rate response is considered here, consistent with experimental data analyzed, and for which a hyperelastic or pseudoelastic model suffices.^{19,20,60} In air, hysteresis may be evident leading to differences in loading and unloading branches of the pressure–volume cycle.⁵⁹ Such hysteresis is minimal in saline-filled specimens, demonstrating that much of the viscoelastic response is linked to changes in surface tension. In forthcoming analysis, test data and model results correspond to the more compliant unloading branch.

4.1.1 Spherical Extension

When the deformation state is spherical, $\mathbf{F} = J^{1/3}\mathbf{1}$, the stress state is hydrostatic according to Eq. 88: $\boldsymbol{\sigma} = (\hat{\tau}_0/J)\mathbf{1} = -p\mathbf{1}$. Notably, $\epsilon_0 = \xi_1 = \ln J$, $\xi_2 = \xi_3 = 0$, and $\epsilon_i = 0$ for $i \geq 1$. Pressure is related to volume change J and potential damage D under isothermal ($T = T_0$) static deformations as follows from the first of Eq. 88 in the infinite time limit ($\chi_0^1 \rightarrow \epsilon_0$):

$$p(J, D) = -\frac{\hat{\tau}_0(J, D)}{J} = -(1 - D) \left[B_0^T \frac{\ln J}{J} \exp\{c_1(\ln J)^2\} + K_0 \right]. \quad (94)$$

The tangent bulk modulus is, extending Eq. 59,

$$B^T(J, D) = (1 - D) \frac{B_0^T}{J} [1 + \ln J(2c_1 \ln J - 1)] \exp\{c_1(\ln J)^2\}. \quad (95)$$

Damage obeys Eq. 85 in terms of $Z_m(\mathbf{X}, t) = \max_{s \in (-\infty, t]} Z(\mathbf{X}, s)$; for $D \in (0, 1)$,

$$D(\mathbf{X}, t) = \exp \left[\frac{\alpha_D \{Z_m(\mathbf{X}, t) - Z_0\}}{Z_0} \right] - 1. \quad (96)$$

The instantaneous potential driving force for $\Psi_0 = \Psi_0^{V\infty} \geq 0$ is, with $J = J(\mathbf{X}, t) \geq 1$,

$$Z(J) = \left[2 \left| \frac{\Psi_0^{V\infty}(J)}{B_0^T} \right| \right]^{1/2} = \left[\frac{1}{c_1} \{ \exp[c_1(\ln J)^2] - 1 \} + 2 \frac{K_0}{B_0^T} \ln J \right]^{1/2}. \quad (97)$$

Five parameters affect the spherical isothermal static response: the isothermal bulk modulus B_0^T , the dimensionless nonlinear elastic constant c_1 that controls bulk stiffening for volume changes, the surface tension K_0 in the undeformed state from air–

tissue interfaces, the threshold normalized energy for damage initiation Z_0 , and the damage growth parameter α_D . For lung tissue in saline for which surface tension presumably vanishes, $K_0 = 0$ and four parameters suffice. For volumetric cycling in regimes within the damage threshold, as in normal breathing activities, $Z < Z_0$ and α_D is not needed.

Shown in Fig. 2 are model results for pressure and tangent bulk modulus obtained using parameters given in Table 1. Experimental data for dog lung are extracted from figures of Hoppin and Lee.^{59,60} Data only address the tensile regime, for which $J \geq 1$. The compressive response is an extrapolation. The same model results are compared versus experimental data for lung tissue in air^{35,59,70} in Fig. 3.

Note that the tangent bulk modulus is minimal near zero pressure. Air-filled lung has a stiffer pressure–volume response due to a higher bulk modulus and surface tension.⁷¹ Effects of surface tension are represented in the present framework by a single parameter K_0 . Lung in saline is stress-free at the reference state $J = 1$, following aforementioned conventions.^{19,60} Lung in air at $J = 1$ is under tension ($p < 0$) due to K_0 . The stress-free state for lung in air according to the present model corresponds to $J = 0.9245$, meaning surface tension causes some contraction in the absence of tensile pleural pressure relative to airway pressure. More elaborate models of surface tension are available,^{1,19,20,22,24} but these are not justified by the fidelity of the presently used experimental data.^{59,60}

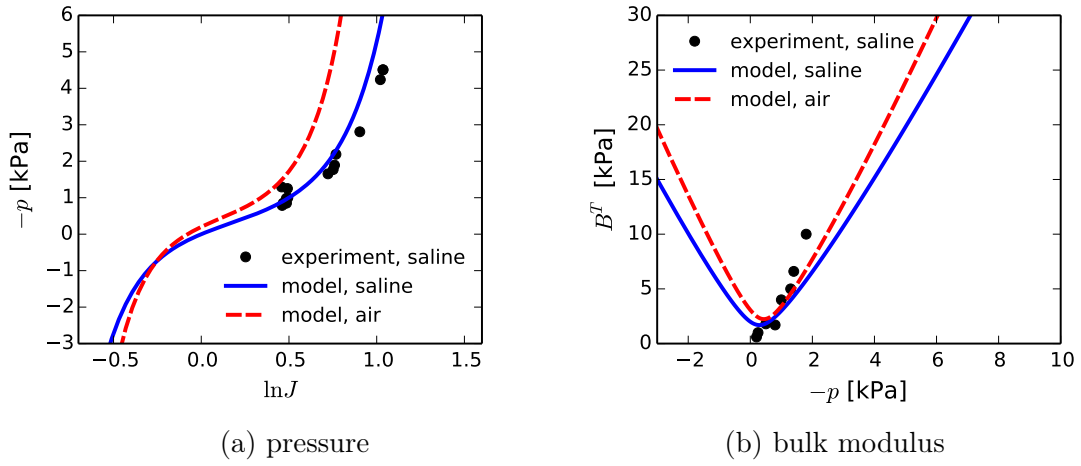


Fig. 2 Results for static isothermal spherical deformation, experimental data for dog lung in saline^{59,60}: a) Cauchy pressure p computed from Eq. 94 and b) tangent isothermal bulk modulus B^T computed from Eq. 95

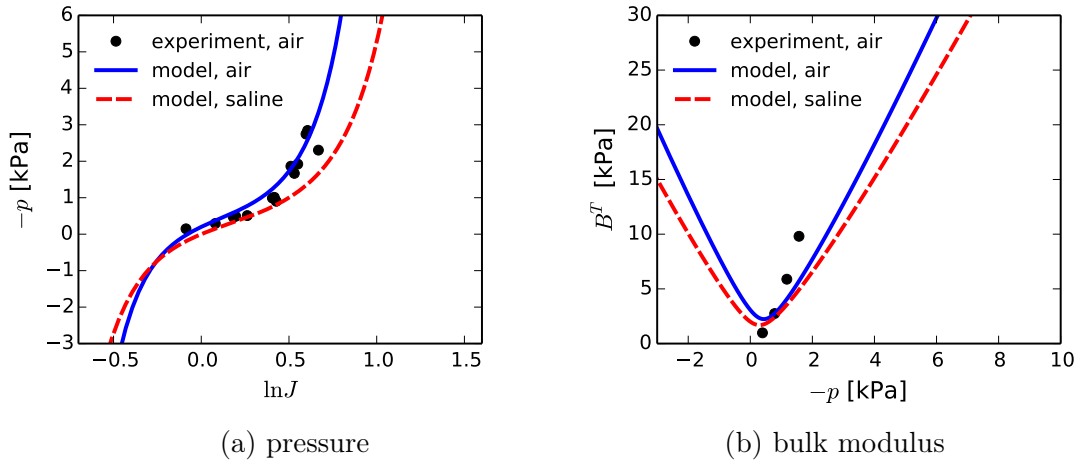


Fig. 3 Results for static isothermal spherical deformation, experimental data for dog lung in air^{35,59,70}: a) Cauchy pressure p computed from Eq. 94 and b) tangent isothermal bulk modulus B^T computed from Eq. 95

4.1.2 Damage Criteria

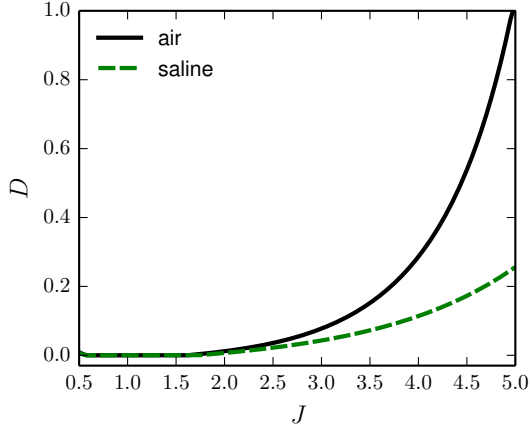
In this framework, damage D is presumed nonzero when either the biological function or mechanical stiffness is compromised. Little quantitative experimental data relating stress–strain state to visibly observable damage mechanisms exist. Cyclic expansion of lung by ventilator at airway pressures exceeding those witnessed in normal breathing causes injury (functional degradation), reduction in shear modulus,⁷² and changes in bulk modulus.^{73,74} Bulk modulus may increase or decrease for cycling in air, where decrease is associated with edema as the air–liquid interface responsible for surface tension is compromised. Longer-term changes in stiffness due to biological adaptation (*e.g.*, stiffness increase due to scarring or inflammation⁷⁵) are not addressed herein.

The present static damage model is calibrated following experiments on saline-ventilated rabbit lung (in the absence of similar data on dog lung) deformed under spherical compression and expansion.^{1,44} In these experiments, alveolar edema and commensurate weight increase due to fluid accumulation occurred in lung stretched beyond a critical magnitude. The rate of edema increased with increasing stretch beyond this threshold. It was concluded in analysis by Fung⁴⁴ that permeability of tissue membranes was compromised at such stretch levels, which were below those necessary to impart observable local tearing or fracture of blood vessels and other structures. Threshold transpulmonary pressures for edema were recorded as 20 to 25 cm H₂O, or approximately 2 kPa. This value was used to obtain $Z_0 = 0.64$ entering damage criterion Eq. 96. In saline, J at this tensile pressure is 1.73. The same value used for air in model calculations, whereby J is 1.62 at $Z = 0.64$.

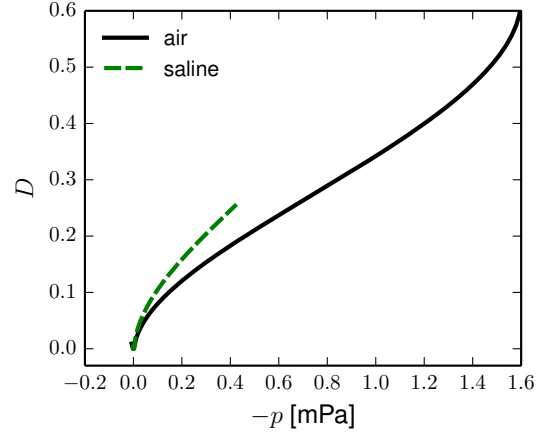
Available data do not enable a precise determination of damage kinetic parameter α_D . Strain softening commensurate with extreme damage ($D \rightarrow 1$) is not evident in experimental data on saline-treated dog lung for $J \leq 3.5$.^{59,60} Nor is any marked reduction in stiffness evident at lower strain levels upon unloading from triaxial stretch ratios exceeding 1.4 in any or all directions.⁶⁰ However, stiffening associated with elastic nonlinearity (manifested by positive c_1, c_2, c_3 in the present model) might mask any decremental contributions from local tearing or damage incurred under large stretch. Eventually, the lung will rupture when stretched beyond an extreme value of J , which should exceed 3.5 according to experimental observations. In the present model, it is assumed that rupture corresponding to $D = 1$ will occur in air-filled lung when $J \geq 5$ (*i.e.*, expansion to five times or more of the nominal

volume). This assumption furnishes the value of $\alpha_D = 0.0167$ listed in Table 1. The same value is used for saline-treated lung.

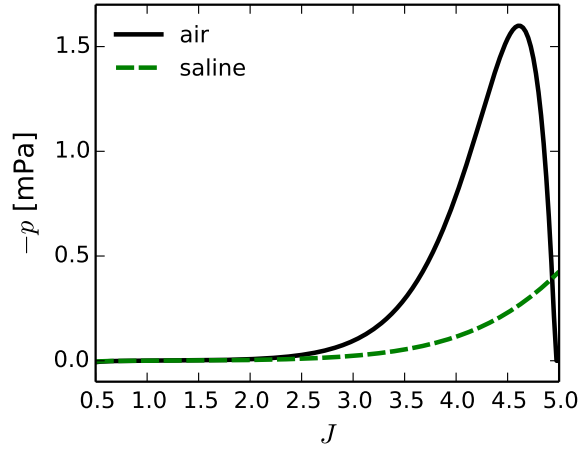
Predictions of Eq. 96 are given in Fig. 4. Damage initiates at a slightly lower volume ratio but slightly higher transpulmonary pressure in air than saline. Damage accumulates more rapidly with increasing volume in air, and more rapidly with increasing tensile pressure in saline, where the different trends manifest from different bulk moduli in the two environments. Pressure–volume curves for lung in air and saline under the extreme loading range $0.5 \leq J \leq 5$ are reported in Fig. 4c. Peak tensile pressure in air-filled lung is attained at $J \approx 4.57$. At larger expansions, tensile pressure decreases with increasing volume as damage accumulates, softening the tangent bulk modulus faster than its increase due to the exponential factor c_1 in the strain energy function.



(a) damage versus volume



(b) damage versus pressure



(c) pressure versus volume

Fig. 4 Results for transpulmonary pressure $-p$ of Eq. 94 and damage D from Eq. 96 in dog lung under static isothermal spherical deformation: a) damage versus volume ratio J , b) damage versus Cauchy pressure p , and c) pressure versus volume ratio over extreme loading regime

Shown in Fig. 5 are pressure–volume data extracted from Figure 2 (“fig. data”) and Table II (“tab. data”) of experiments⁶⁰ on saline-treated dog lung. Model results with and without damage enabled are compared. Differences due to damage are minimal for Lagrangian tensile pressures $\hat{\tau}_0 \leq 20$ kPa. At larger extensions, the model with damage enabled provides slightly better agreement with test data. Under compressive loading, damage initiates at $J \lesssim 0.58$, which is thought to be physically realistic. Collapse and air trapping followed by reopening and air release have been observed during cyclic loading to very large compression ($J \lesssim 0.5$).⁷⁶

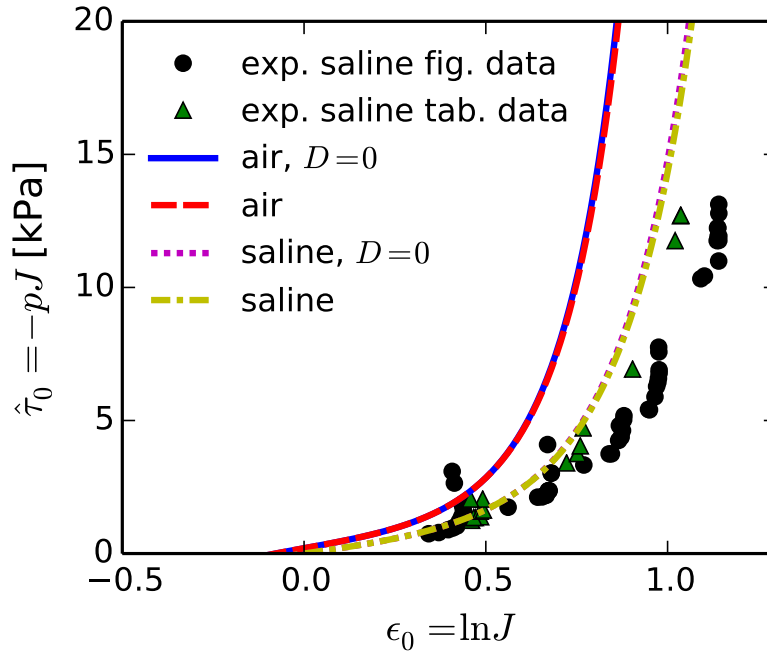


Fig. 5 Pressure–volume results of lung model under spherical extension in air and saline, with ($D \geq 0$) and without ($D = 0$) damage activated, versus experimental pressure–volume data in saline measured under triaxial extension.⁶⁰ See text for explanation of figure versus tabular experimental data.

4.1.3 Biaxial and Triaxial Loading

A coordinate system is chosen with basis vectors $\{\mathbf{e}_K\}$ aligned parallel to the principal directions of deformation. In complementary experiments,^{21,59} cube-shaped samples of lung tissue are sequentially stretched and unloaded parallel to each direction, whereby cube faces are orthogonal to directions of stretch. In the experiments of Hoppin,⁵⁹ further analyzed by Lee and others,^{60,69} the X_1 -direction is that of primary stretch ratio λ_1 corresponding to Lagrangian traction component t_1 . Stretches in orthogonal directions are nearly equal ($\lambda_2 \approx \lambda_3$), as are tractions $t_2 \approx t_3$. The latter orthogonal tractions are held constant in a particular experiment while t_1 is varied. In practice, equality of lateral tractions is enforced mechanically, while all three stretch ratios are measured, confirming that $\lambda_2 \approx \lambda_3$ and isotropic response under pure shear.

Let the origin of the coordinate frame be located at the center of the undeformed cube of material. Then the motion is $x_k = \lambda_k X_k$ (no sum on $k = 1, 2, 3$). The deformation gradient is diagonal, and hence upper triangular by default:

$$\mathbf{F} = \mathbf{R} = \begin{bmatrix} a & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & c \end{bmatrix} = \begin{bmatrix} \lambda_1 & 0 & 0 \\ 0 & \lambda_2 & 0 \\ 0 & 0 & \lambda_3 \end{bmatrix} = \begin{bmatrix} \lambda_1 & 0 & 0 \\ 0 & \lambda_2 & 0 \\ 0 & 0 & \lambda_2 \end{bmatrix}, \quad (98)$$

with Jacobian determinant

$$J = abc = ab^2 = \lambda_1(\lambda_2)^2. \quad (99)$$

Strain attributes are

$$\begin{aligned} \boldsymbol{\epsilon}(\lambda_1, \lambda_2) &= [\epsilon_0, \epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4, \epsilon_5, \epsilon_6]^T \\ &= [\ln\{\lambda_1(\lambda_2)^2\}, \frac{1}{3} \ln\{\frac{\lambda_1}{\lambda_2}\}, 0, -\frac{1}{3} \ln\{\frac{\lambda_1}{\lambda_2}\}, 0, 0, 0]^T. \end{aligned} \quad (100)$$

Combinations entering the free energy function are $\xi_1 = \epsilon_0$ and $\xi_2 = (\epsilon_1)^2 + (\epsilon_3)^2$, noting $\xi_3 = 0$ in the absence of simple shearing modes. The first Piola–Kirchhoff stress tensor (force per unit reference area) is $\mathbf{P} = \text{diag}(t_1, t_2, t_3)$ where $t_2 = t_3$ for symmetric lateral loading. Cauchy stress $\boldsymbol{\sigma} = J^{-1}\mathbf{s}$, with the physical stress tensor

s equal to the Kirchhoff stress since $\mathbf{Q} = \mathbf{1}$, is

$$\boldsymbol{\sigma} = \begin{bmatrix} \frac{t_1}{\lambda_2 \lambda_3} & 0 & 0 \\ 0 & \frac{t_2}{\lambda_1 \lambda_3} & 0 \\ 0 & 0 & \frac{t_3}{\lambda_1 \lambda_2} \end{bmatrix} = \begin{bmatrix} \frac{t_1}{(\lambda_2)^2} & 0 & 0 \\ 0 & \frac{t_2}{\lambda_1 \lambda_2} & 0 \\ 0 & 0 & \frac{t_2}{\lambda_1 \lambda_2} \end{bmatrix}, \quad p = -\frac{1}{3\lambda_2} \left(\frac{t_1}{\lambda_2} + 2\frac{t_2}{\lambda_1} \right). \quad (101)$$

Denoted by p is the Cauchy pressure. The stress attribute vector is

$$\hat{\boldsymbol{\tau}}(\boldsymbol{\sigma}(t_1, t_2, \lambda_1, \lambda_2)) = \begin{Bmatrix} \hat{\tau}_0 \\ \hat{\tau}_1 \\ \hat{\tau}_2 \\ \hat{\tau}_3 \\ \hat{\tau}_4 \\ \hat{\tau}_5 \\ \hat{\tau}_6 \end{Bmatrix} = \lambda_1 (\lambda_2)^2 \begin{Bmatrix} \frac{1}{3}(\sigma_{11} + 2\sigma_{22}) \\ \sigma_{11} - \sigma_{22} \\ 0 \\ \sigma_{22} - \sigma_{11} \\ 0 \\ 0 \\ 0 \end{Bmatrix}. \quad (102)$$

From Eq. 103, nonzero components are obtained from strain attributes, again in the absence of temperature changes or viscous effects, as

$$\begin{aligned} \hat{\tau}_0 &= (1 - D)B_0^T [\epsilon_0 \exp(c_1 \xi_1^2) - 3\mu'_0 \xi_2 + K_0/B_0^T], \\ \hat{\tau}_1 &= -\hat{\tau}_3 = 6(1 - D)\mu_0 [\epsilon_1 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1)]. \end{aligned} \quad (103)$$

Free energy of Eq. 87 reduces to

$$\begin{aligned} \Psi = \Psi^\infty &= (1 - D) \left[\frac{1}{2} \frac{B_0^T}{c_1} \{\exp[c_1(\xi_1)^2] - 1\} + K_0 \xi_1 \right. \\ &\quad \left. + 3 \frac{\mu_0}{c_2} \{\exp[c_2 \xi_2] - 1\} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right) \right]. \end{aligned} \quad (104)$$

Contributions, not reduced by damage, from dilatation and squeeze are, respectively,

$$\begin{aligned} \Psi_0^{V\infty} &= \frac{1}{2} \frac{B_0^T}{c_1} \{\exp[c_1(\xi_1)^2] - 1\} + K_0 \xi_1, \\ \Psi_0^{\mu\infty} &= 3 \frac{\mu_0}{c_2} \{\exp[c_2 \xi_2] - 1\} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right). \end{aligned} \quad (105)$$

The driving force for damage, whose evolution again obeys Eq. 96, is

$$Z = \begin{cases} \left[2 \left| \frac{\Psi_0^{V\infty}}{B_0^T} + \frac{\Psi_0^{\mu\infty}}{\mu_0} \right| \right]^{1/2} & (\text{if } \Psi_0 \geq 0), \\ 0 & (\text{if } \Psi_0 < 0). \end{cases} \quad (106)$$

Material parameters entering the static isothermal triaxial response are those that have been discussed and calibrated already for the spherical response (B_0^T , c_1 , K_0 , Z_0 , α_D) along with the elastic squeeze/shear modulus μ_0 , its ambient pressure derivative μ'_0 , and dimensionless nonlinear squeeze parameter c_2 . The value of μ'_0 is obtained directly from experimental references,^{35,65,70} as explained already. Parameters μ_0 and c_2 are obtained via calibration to experimental data for saline-treated dog lung,^{59,60} wherein $K_0 = 0$ is irrelevant. In these calculations, λ_1 is imposed and t_1 is the model output. Transverse stretch components $\lambda_2 = \lambda_3$ are adjusted until Lagrangian tractions $t_2 = t_3$ match discrete, experimentally prescribed values.

Shown in Fig. 6 are model results and experimental data for all five values of imposed lateral traction $\sqrt{t_2 t_3} \approx t_2$. The squeeze strain is ϵ_3 and its conjugate shear stress is $\hat{\tau}_3$. The sigmoidal response curve is a result of the exponential stiffening of the tangent shear modulus via $c_2 > 0$. Damage is enabled in these results, though as explained, it has a near-negligible effect on the mechanical response. Complementary pressure–volume data from these same experiments has already been compared with model results in Fig. 5.

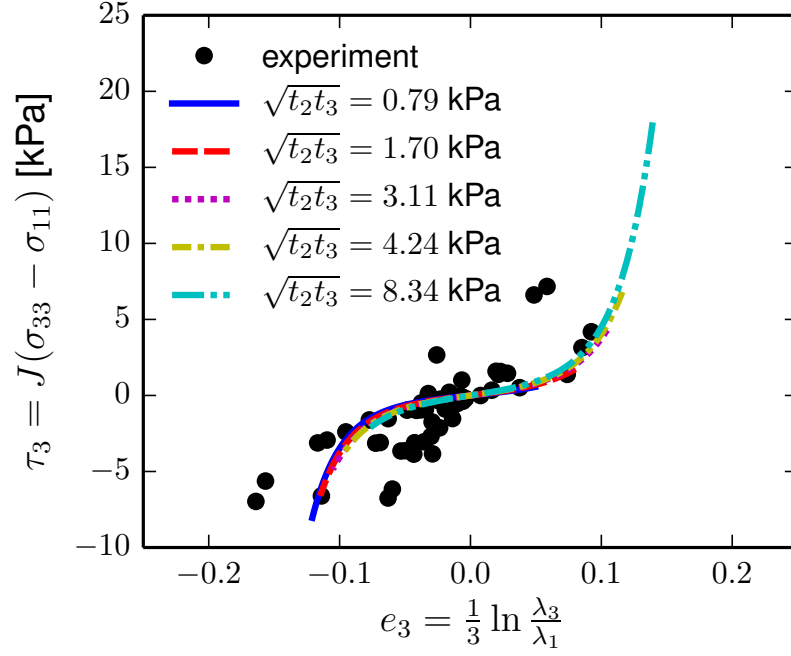


Fig. 6 Squeeze mode stress–strain results of lung model under triaxial extension in saline versus experimental data^{59,60}

Shown in Fig. 7 are Lagrangian traction t_1 versus stretch λ_1 for each discrete value of transverse traction. Model results with and without damage enabled are given for comparison. In each case, effects of D on mechanical response are very small, but thresholds for affecting biological function ($Z_m > Z_0$) are attained at sufficient stretch levels. Evolution of damage versus extension λ_1 for all five cases is shown in Fig. 7f. Damage is most severe for larger lateral tensile loads, as physically expected. Interestingly, for large lateral stress levels, increasing λ_1 above unity does not notably increase D , since the deviatoric strain energy is reduced as dilatational energy increases. The model provides excellent fits to the data at small transverse tractions in Fig. 7a and b. Quality of representation of experimental data degrades at larger values of $\sqrt{t_2 t_3}$ for $\lambda_1 \lesssim 1.3$. Even so, performance of this exponential model under triaxial extension, which requires calibration of only four elastic parameters for lung in saline, is deemed very respectable considering that similar quality fits invoking a polynomial strain energy function required calibration of nine distinct parameters.⁶⁰

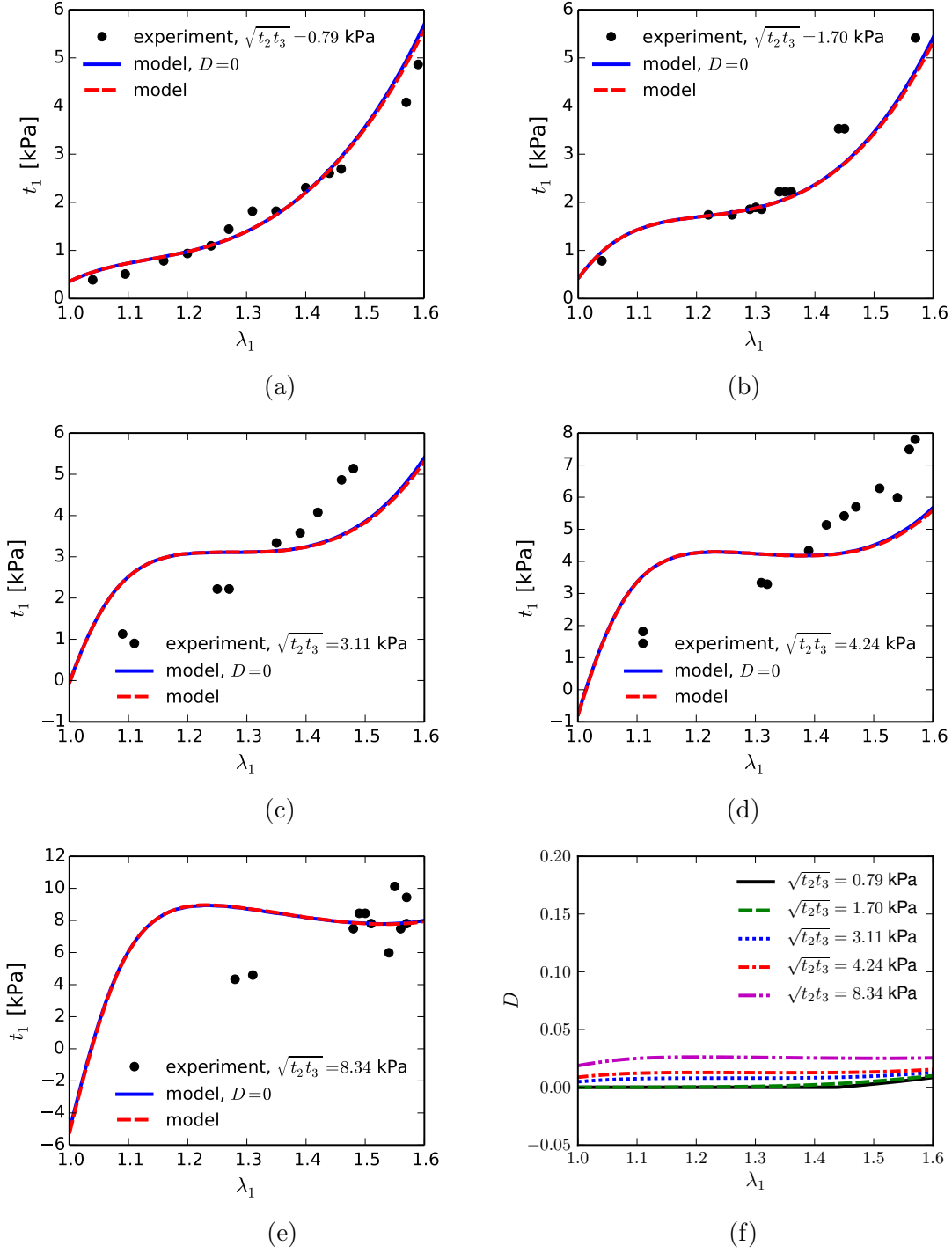


Fig. 7 Principal traction versus stretch for saline-treated dog lung under static isothermal triaxial deformation a)–e) different lateral traction values with experimental data⁶⁰ and f) damage D for all five lateral traction values. Model curves for t_1 with and without ($D = 0$) damage enabled are nearly indistinguishable since $D \lesssim 0.03$ over the prescribed ranges of λ_1 and $\lambda_2 = \lambda_3$.

4.2 Dynamic Pressure Relaxation

Viscoelastic response parameters are obtained by calibration of the model to pressure relaxation experiments on rabbit lung in air,⁷⁷ in the absence of analogous data for dog lung. The two parameters of interest in Table 1 are β_1 and t_1 , the dynamic bulk modulus amplification factor (dimensionless) and the time scale for relaxation. These are related to other constants entering the viscoelastic theory via the dynamic moduli $\mu_1 = \beta_1 B_0^T = 6\beta_2\mu_0 = \beta_3 G_0$ and the dynamic viscosity $\nu_1 = \mu_1 t_1$.

The general nonlinear viscoelastic framework reported recently,^{5,7} as well as those on which the former is based,^{48,49} admits any number of additional relaxation times t_α with corresponding additional dynamic moduli μ_α to allow for description of the response spectrum over a range of loading frequencies. Here, a simple model with only a single relaxation time is implemented to demonstrate basic features and calibrate the instantaneous dynamic response, with the understanding that more viscous modes must be included to address more sophisticated loading protocols (*e.g.*, cycling at disparate frequencies) and the resultant nonlinear viscoelastic response.^{25,75,78–80} The ratio of instantaneous dynamic modulus to equilibrium modulus observed in rabbit lung for spherical deflation⁷⁷ is comparable in magnitude to values observed in pig lung under indentation⁸¹ and human lung under tensile relaxation.⁶⁷

In the present analysis, the viscous stress component ζ_0^1 associated with non-equilibrium pressure is generally nonzero. All six other components of ζ^1 vanish by construction, so $\zeta_i^1 = 0 \Rightarrow \chi_i^1 = \epsilon_i$ for $i \geq 1$. The homogeneous deformation state is spherical: $\mathbf{F} = J^{1/3}\mathbf{1}$. The stress state is hydrostatic: $\boldsymbol{\sigma} = (\hat{\tau}_0/J)\mathbf{1} = -p\mathbf{1}$. Elastic strain variables are $\epsilon_0 = \xi_1 = \ln J$, $\xi_2 = \xi_3 = 0$, and $\epsilon_i = 0$ for $i \geq 1$, leading to $\chi_i^1 = 0$ for $i \geq 1$.

As is verified later, the tensile deformation state under which relaxation is measured is not severe enough to induce damage: $D(t) = 0 \forall t \geq 0$. Viscous heating is omitted under the time scales involved, on the order of seconds.

Pressure is related to volume change J and potential damage D under presumed

isothermal ($T = T_0$) deformations as follows from the first of Eq. 88 ($D = 0$):

$$p(J, \chi_0^1) = -\frac{\hat{\tau}_0(J, \chi_0^1)}{J} = -\frac{B_0^T}{J} \left[(\ln J) \exp\{c_1(\ln J)^2\} + \frac{K_0}{B_0^T} + \beta_1(\epsilon_0 - \chi_0^1) \right]. \quad (107)$$

Free energy is

$$\begin{aligned} \Psi(J, \chi_0^1) &= \Psi_0^{V\infty}(J) + \Upsilon_0^V(J, \chi_0^1) \\ &= \frac{1}{2}B_0^T \left[\frac{1}{c_1} \{ \exp[c_1(\ln J)^2] - 1 \} + \frac{2K_0}{B_0^T} \ln J \right] + \frac{1}{2}\mu_1(\ln J - \chi_0^1)^2. \end{aligned} \quad (108)$$

Under pressure relaxation at fixed volume, $J = J_0 = \text{constant}$. At equilibrium, as $t \rightarrow \infty$, $\chi_0^1 \rightarrow \epsilon_0 = \ln J = \ln J_0$. The value of J_0 was prescribed such that $p(t \rightarrow \infty)$ matches the experimental value:⁷⁷ $J_0 = 1.313$. Damage obeys Eq. 85 but is not important here. Specifically, for $J = 1.313$, the maximum driving force at $t = 0$ is $Z_m = 0.414 < Z_0 = 1$ at $p_{tp} = -p = 1.11$ kPa, so damage never ensues.

The internal strain χ_0^1 evolves with time according to the viscoelastic kinetic law for its internal stress conjugate, ζ_0^1 . From Eqs. 73 and 74 with $J = J_0 = \text{constant}$,

$$\chi_0^1(t) = \ln J_0 - \frac{1}{\beta_1 B_0^T} \zeta_0^1(t) = \ln J_0 \left[1 - \exp\left(\frac{-t}{t_1}\right) \right]. \quad (109)$$

Substituting Eq. 109 into Eq. 107 at $J = J_0$ produces the relaxation function

$$p(t) = -\frac{B_0^T}{J_0} \left[(\ln J_0) \exp\{c_1(\ln J_0)^2\} + \frac{K_0}{B_0^T} + \beta_1(\ln J_0) \exp\left(\frac{-t}{t_1}\right) \right]. \quad (110)$$

Viscoelastic parameters β_1 and t_1 are obtained by matching the relaxation response of p from Eq. 110 versus decay time t to experimental data.⁷⁷ Results are reported in Fig. 8, demonstrating suitability of this two-parameter fit.

The present implementation of theory in Section 3.3—essentially an extension of the standard linear solid model to finite strains and nonlinear “springs”⁴⁸ and with a single discrete relaxation function—is too simplistic to address cyclic viscoelasticity of the lung within a wide range of frequencies. More internal variables χ^α could be added accounting for additional relaxation times, as noted previously, albeit at the expense of two more parameters (t_α, μ_α) for each additional viscous

mode α . More sophisticated nonlinear theories also exist, e.g., those based on nonlinear viscoelastic kernels²⁵ or fractional derivatives⁷⁹ may prove more reliable for capturing the frequency response. However, their incorporation in a nonlinear 3-D thermoelastic framework with damage compatible with all laws of thermodynamics is theoretically challenging. Continuous relaxation spectrum models have been recommended for soft biological tissues, but their use for modeling the creep response of lung is suspect.² The present model requires only two parameters, is thermodynamically consistent and computationally efficient, and is adequate for subsequent applications herein, where either the loading is very slow such that equilibrium static moduli suffice, or very fast, such that instantaneous dynamic moduli suffice.

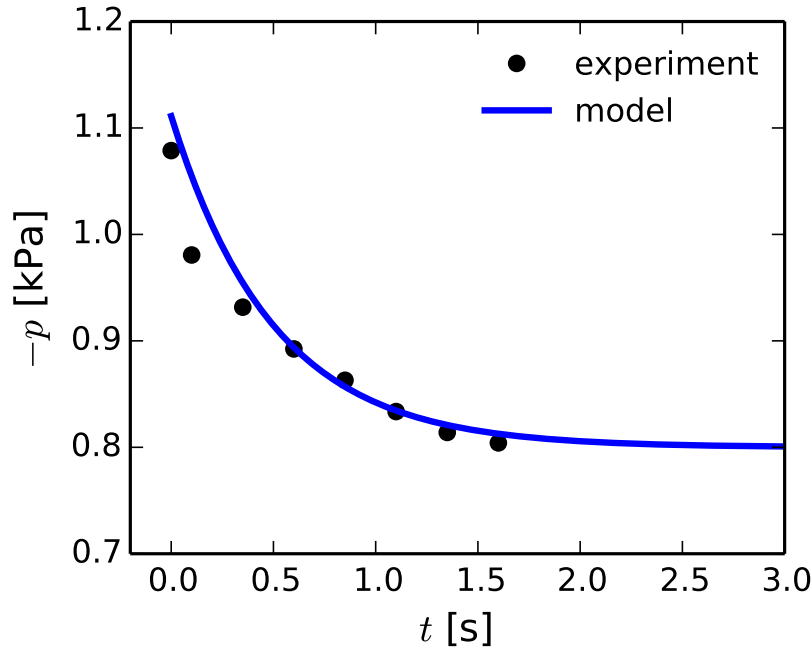


Fig. 8 Pressure versus relaxation time for constant-volume stress relaxation in air: model calibration versus experimental data on rabbit lung⁷⁷

4.3 Simple Shear

The final problem examined for the free-energy based theory of Section 3 is dynamic simple shear. In the absence of comparable data on dog lung, results are compared to dynamic torsion data on porcine lung²⁸ and dynamic simple shear data on human lung.²⁹ The lone material parameter to be identified in Table 1 is c_3 ,

which dictates the change in tangent shear modulus under large simple shearing deformation.

A coordinate system is chosen with basis vector $\mathbf{e}_2$ aligned normal to the plane of shear and $\mathbf{e}_1$ parallel to the direction of shear. In a torsion test, $\mathbf{e}_2$ would be parallel to the axis of twist along a cylindrical specimen, and $\mathbf{e}_1$ would be aligned in an orthogonal circumferential direction. In the latter case, a material element of study would be located near the outer radius of the specimen, where shear stress is maximal. The local deformation gradient at a material element, in these preferred Cartesian coordinates, is presumed one of simple shear and is upper triangular by construction:

$$\mathbf{F} = \mathbf{R} = \begin{bmatrix} 1 & \gamma & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}; \quad J = 1. \quad (111)$$

The only non-vanishing strain attribute, strain combination, and viscous internal state variable entering the free energy function are

$$\epsilon_4 = \gamma, \quad \xi_3 = \gamma^2, \quad \chi^1 = [0, 0, 0, \chi, 0, 0]^T, \quad (\chi = \chi_4^1). \quad (112)$$

Nonzero stress attributes in terms of Cauchy pressure p and shear stress τ conjugate to γ are

$$\begin{aligned} p(\gamma, T) &= -\hat{\tau}_0 = (1 - D)B_0^T \left[\frac{1}{2}G_0'\gamma^2 + A_0(T - T_0) - K_0/B_0^T \right], \\ \tau(\gamma, \chi) &= \hat{\tau}_4 = (1 - D)G_0 \left[\gamma \exp(c_3\gamma^2) + \beta_3(\gamma - \chi) \right], \quad (\beta_3 = \beta_1 B_0^T / G_0). \end{aligned} \quad (113)$$

Nonzero pressure may result from nonlinear coupling with shear, temperature change, and surface tension. Free energy, not including the damage prefactor, is

$$\begin{aligned} \Psi_0(\gamma, T, \chi) &= \Psi_0^{G\infty}(\gamma) + \Upsilon_0^G(\gamma, \chi) + \psi(T) \\ &= \frac{1}{2} \frac{G_0}{c_3} \{ \exp[c_3\gamma^2] - 1 \} + \frac{1}{2} \beta_3 G_0 (\gamma - \chi)^2 \\ &\quad - c_0 [T \ln(T/T_0) - (T - T_0)]. \end{aligned} \quad (114)$$

The lone scalar viscous stress component is $\zeta = \zeta_4^1 = -\partial_\chi \Psi_0 = -\partial_\chi \Upsilon_0^G$. Vis-

coelastic kinetics of Eq. 73 produce the convolution integral

$$\zeta(t) = \beta_3 G_0 \left\{ \gamma \exp\left(\frac{-t}{t_1}\right) + \int_{0+}^t \exp\left[\frac{-(t-s)}{t_1}\right] \left(\frac{d}{ds} [\gamma(s)]\right) ds \right\}. \quad (115)$$

For constant strain rate loading, $\gamma = \dot{\gamma}t$ where $\frac{d}{dt}\gamma = \dot{\gamma} = \text{constant}$, this reduces to

$$\zeta(t) = \beta_3 G_0 \dot{\gamma} \{ t \exp(-t/t_1) + t_1 [1 - \exp(-t/t_1)] \}. \quad (116)$$

The internal strain is, from Eq. 74,

$$\chi(t) = \gamma(t) - \frac{\zeta(t)}{\beta_3 G_0} = \dot{\gamma}t - \dot{\gamma} \{ t \exp(-t/t_1) + t_1 [1 - \exp(-t/t_1)] \}. \quad (117)$$

The minimum strain rate considered in what follows is $\dot{\gamma} = 550 \text{ s}^{-1}$, for which $\min[\exp(-t/t_1)] = 0.9964$ at $\gamma = 1$ and $t = \frac{1}{550} \text{ s}$. Therefore, for such rapid loading, viscous relaxation is insignificant, and $\chi(t) = 0 \forall t \geq 0$ is a very accurate approximation. Damage accrues according to Eq. 85, where $Z_m = \max_{s \in [0, t]} Z(s)$ with

$$Z = \begin{cases} \left[2 \left| \frac{\Psi_0^G + \Upsilon_0^G}{G_0} \right| \right]^{1/2} & (\text{if } \Psi_0 \geq 0), \\ 0 & (\text{if } \Psi_0 < 0). \end{cases} \quad (118)$$

Results of the model for an imposed strain rate of $\dot{\gamma} = 550 \text{ s}^{-1}$ are shown in Fig. 9, with and without damage enabled. Damage only ensues for $\gamma \gtrsim 0.4$, and even then, its effect on τ is minimal for $\gamma \leq 1$. Damage increases with shear strain as indicated in Fig. 9b; shown for comparison are evolution of D for the present dynamic application along with evolution for a similar problem under static conditions for which viscoelasticity is omitted and $\Upsilon_0 = 0$. Damage evolves more severely with γ in the dynamic case due to contributions from viscous configurational energy to the driving force Z . Although loss of biological function may arise at the onset of nonzero D at relatively low stress ($Z > Z_0$ for $\tau \gtrsim 1.05 \text{ kPa}$) the lung can sustain very large simple shear deformations before total rupture: $D \rightarrow 1$ only when $\gamma \gtrsim 3.4$. This prediction is consistent with experimental data for spherical expansion⁴⁴ discussed already in Section 4.1.2.

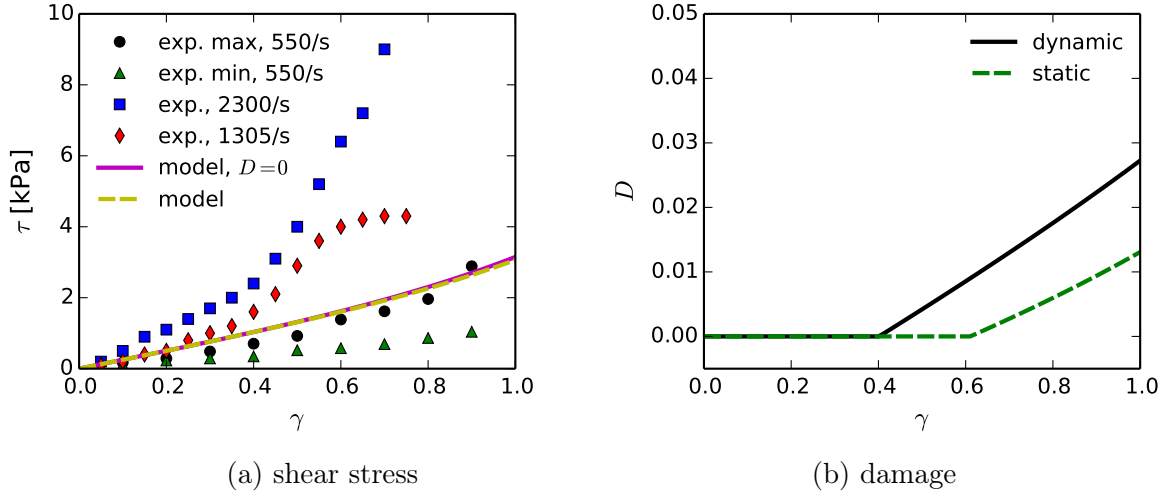


Fig. 9 Model response in dynamic simple shear: a) shear stress τ versus simple shear strain γ , compared to experimental max/min data on porcine lung²⁸ tested at $\dot{\gamma} = 550 \text{ s}^{-1}$ and human lung²⁹ tested at $\dot{\gamma} = 1305 \text{ s}^{-1}$ and $\dot{\gamma} = 2300 \text{ s}^{-1}$ and b) damage variable D versus shear γ for dynamic (with viscosity) and static (without viscosity) loading

Shear stress τ is compared with data obtained at the same strain rate from dynamic torsion experiments on porcine lung undertaken in a modified Kolsky bar.²⁸ Data presented in that work demonstrate significant test-to-test variability; maximum and minimum stress–strain curves are reproduced here in Fig. 9b. Also shown are experimental data obtained from direct, dynamic simple shear experiments on human lung at two higher strain rates.²⁹ Predictions of the present model are identical if a strain rate $\dot{\gamma} > 550 \text{ s}^{-1}$ is imposed.

Shear stress data for human lung are notably stiffer than data for porcine lung. As a compromise, the present model, with $c_3 = 0.5$, produces τ - γ response curves falling between the two species for $\gamma \lesssim 0.4$ and results closer to the maximal porcine data for $\gamma \gtrsim 0.4$. Dog data are expected to more closely match porcine than human data, as also suggested elsewhere⁶⁷ where human lung tissue was reported to be stiffer than dog lung tissue in biaxial tension. Differences may also arise from sample preparation and different experimental techniques used at the different laboratories. Shear elastic constants can be adjusted in the future, if warranted, should consistent data on tissue samples conditioned uniformly from a single species be obtained. It is noted setting c_3 equal to the nonlinear elastic squeeze constant (*i.e.*, $c_3 \rightarrow c_2 \gg 0.5$) produces a simple shear stress–strain response that is overly stiff with regard to

both human and porcine data for $\gamma \gtrsim 0.4$. This distinction demonstrates utility of the presently invoked three-mode theory (distinct dilatation, squeeze, and simple shear) over a traditional, two-mode (distinct dilatation and deviatoric deformation) isotropic elasticity theory.

5. Internal Energy Formulation for Air-Tissue Response

A constitutive formulation for static and dynamic loading of the lung based on the internal energy potential U is now developed. This model is preferred for compressive stress states and high strain rates, and it is ideal for isentropic problems such as elastic wave propagation. Unlike the formulation of Section 3, it does account for compressibility of the air inside the lung, and thus can be used to model adiabatic shock loading processes in which air does not have time to redistribute among or escape from the alveoli.

5.1 General Form

The following form of U in the second of Eq. 35 is invoked:

$$\begin{aligned} U(\boldsymbol{\epsilon}, \eta, \boldsymbol{\chi}^1, D) &= \bar{f}(D)[U_0^V(\boldsymbol{\epsilon}, \eta, \boldsymbol{\chi}^1)] + f(D)[U_0^\mu(\boldsymbol{\epsilon}, \boldsymbol{\chi}^1) + U_0^G(\boldsymbol{\epsilon}, \boldsymbol{\chi}^1)] + u(\eta) \\ &= \bar{f}(D)[U_0^{V\infty}(\epsilon_0, \eta) + \Upsilon_0^V(\epsilon_0, \chi_0^1)] \\ &\quad + f(D)[U_0^{\mu\infty}(\boldsymbol{\epsilon}) + U_0^{G\infty}(\boldsymbol{\epsilon}) + \Upsilon_0^\mu(\boldsymbol{\epsilon}, \boldsymbol{\chi}^1) + \Upsilon_0^G(\boldsymbol{\epsilon}, \boldsymbol{\chi}^1)] + u(\eta). \end{aligned} \quad (119)$$

This function mirrors that of free energy Eq. 54, where temperature T is replaced with entropy density η . Equilibrium thermoelastic internal energy, including strain energy and coupling from thermal expansion, is $U_0^\infty = U_0^{V\infty} + U_0^{\mu\infty} + U_0^{G\infty}$, partitioned into contributions from dilatation, squeeze, and simple shear. Configurational internal energy associated with viscoelasticity, partitioned similarly, is $\Upsilon_0 = \Upsilon_0^V + \Upsilon_0^\mu + \Upsilon_0^G$. Thermoelastic and viscous contributions also simply sum as can be inferred from Eq. 119:

$$\begin{aligned} U_0 &= U_0^V + U_0^\mu + U_0^G; \\ U_0^V &= U_0^{V\infty} + \Upsilon_0^V, \quad U_0^\mu = U_0^{\mu\infty} + \Upsilon_0^\mu, \quad U_0^G = U_0^{G\infty} + \Upsilon_0^G. \end{aligned} \quad (120)$$

Functions entering Υ_0 are assigned identical forms to those used already in Section 3. As implemented already in Section 3.3, attention is limited to consideration

of a single viscous relaxation mode corresponding to $\alpha = 1$ in Eq. 35. Two damage degradation functions are $\bar{f}$ and f , where the latter are prescribed identically to that in Eq. 54. Function f degrades the stiffness in pure and simple shear. Function $\bar{f}$ degrades the bulk stiffness, and $\bar{f}(D) \geq f(D)$ is imposed conditionally later such that under rapid compression, the tangent bulk modulus cannot fall below that of air alone in the initial state. The purely entropic internal energy accounting for specific heat is u ; this is assumed unaffected by damage.

5.2 Thermoelastic Strain Energy

A material element consists of dense lung tissue, surface liquid, and air inside alveoli. Under slow deformation processes, air has time to move throughout the microstructure—or when the entire lung is considered, enter and exit through open trachea—and only the tissue network and surface liquid affect the stiffness. Under very rapid loading (or if the trachea is artificially sealed), air does not have time to redistribute or escape, and compressibility of the air affects the response in conjunction with the dense tissue and liquid phase.³² As described in detail later, effects of trapped air on stiffness measured during wave propagation have been widely documented.^{1,32,33,44,66} The loading rate or frequency at which a transition from open tissue-dominated to air-dominated stiffness affects wave speeds is not known precisely, and an intermediate region that falls between the two regimes likely exists.³³ The present model addresses only the two extreme regimes: slow and fast loading corresponding to an open-cell and closed-cell model for the effective bulk modulus, respectively.³³

For the open-cell model, the reference state is identical to that of the free energy-based theory of Section 3.2. The stress supported by the continuum material element is that sustained by the tissue and its surface air–liquid interface, if not saline-washed. A material element is considered to occupy a stress-free reference state when undeformed ($\mathbf{F} = \mathbf{1}$ and $J = 1$) and in the absence of surface tension at the air–liquid interface, wherein its mass density is ρ_0 and entropy is η_0 . This definition of reference state is consistent with those in the lung mechanics literature.^{19,20} Surface tension will produce tensile pressure at $J = 1$. Transpulmonary pressure p_{tp} is equal to the negative of pressure supported by continuum element, related to internal airway pressure p_a and pleural/external pressure p_{pl} via Eq. 93. Neither p_a nor p_{pl} need be known explicitly to use the open-cell model or the framework of

Sections 3 and 4. When one is given (*e.g.*, $p_a = 1 \text{ atm}$), then the other (*e.g.*, p_{pl}) can be computed a posteriori once p is obtained from the constitutive model. All pressures entering Eq. 93 are referred in this work to a datum of absolute zero (vacuum). Accordingly, $p_a \geq 0$ since air alone cannot sustain tensile pressure, at least under conventional models such as those for ideal gases.⁸²

The closed-cell model is more complicated. A continuum material element consists of the dense tissue phase (solid + liquid) as well as the internal air phase (gas). Volume fractions occupied by each phase in the reference state are denoted by v_t and v_a for dense tissue and air, respectively, related by $v_t + v_a = 1$. The reference state of the effective continuum element for which $\mathbf{F} = \mathbf{1}$, $J = 1$, $\rho = \rho_0$, and $\eta = \eta_0$ is defined as follows. The continuum stress vanishes, and thus $p = 0$, for this element at the reference state in the absence of surface tension. The internal airway pressure at the reference state is imposed a priori as $p_a = p_{a0} > 0$, where a vacuum airway state is not considered to avoid singularities later. Thus, from Eq. 93, the pleural pressure, positive in compression, at the reference state at $J = 1$ is $p_{pl} = p + p_a = -K_0 + p_{a0}$, where K_0 is the surface tension. In the absence of surface tension, pleural pressure and internal airway pressure balance to ensure the reference state is stress-free. Material coefficients entering the internal energy function depend on v_t and p_{a0} , which are regarded as fixed initial values during solution of any boundary value problem. This does not imply that internal airway pressure p_a remains constant during the time history of deformation. However, pressures and other state functions (*e.g.*, other stress components, temperatures, entropies) are not resolved explicitly within each phase by the present composite model during the deformation process. This would require a multi-phase analysis^{37–39} outside the present scope. Rather, only the effective values of state variables for the homogenized continuum element are resolved herein.

Equilibrium thermoelastic internal energy depends explicitly on three functions of the strain attributes of Eq. 22, denoted by ξ_i , $i = 1, 2, 3$, defined identically to Eq. 55:

$$\begin{aligned}\xi_1 &= \epsilon_0 = \ln J, \\ \xi_2 &= (\epsilon_1)^2 + (\epsilon_2)^2 + (\epsilon_3)^2, \\ \xi_3 &= (\epsilon_4)^2 + (\epsilon_5)^2 + (\epsilon_6)^2 = \alpha^2 + \beta^2 + \gamma^2.\end{aligned}\tag{121}$$

This choice represents, like the free energy-based formulation in Section 3, a three-mode isotropic theory in the sense of Freed.^{5,46,54} A convenient property of the form $U_0^\infty(\xi_1, \xi_2, \xi_3, \eta)$ is that the thermoelastic internal energy analog of Eq. 40 is identically satisfied through Eq. 23.

Thermoelastic strain energy density per unit reference volume is prescribed as follows:

$$\begin{aligned} U_0^\infty(\xi_1, \xi_2, \xi_3, \eta) = & \frac{1}{2} \frac{\bar{B}_0^\eta}{c_1} \{\exp[c_1(\xi_1)^2] - 1\} - g_0 T_0 \eta \xi_1 + K_0 \xi_1 \\ & + 3 \frac{\mu_0}{c_2} \{\exp[c_2 \xi_2] - 1\} \left[1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right] \\ & + \frac{1}{2} \frac{G_0}{c_3} \{\exp[c_3 \xi_3] - 1\} \left[1 - \frac{B_0^T}{G_0} G'_0 \xi_1 \right]. \end{aligned} \quad (122)$$

Thermoelastic strain energy is partitioned into contributions from dilatation, squeeze, and simple shear analogously to Eqs. 80 and 81:

$$U_0^\infty = U_0^{V\infty} + U_0^{\mu\infty} + U_0^{G\infty}; \quad (123)$$

$$\begin{aligned} U_0^{V\infty} &= \frac{1}{2} \frac{\bar{B}_0^\eta}{c_1} \{\exp[c_1(\xi_1)^2] - 1\} + K_0 \xi_1 - g_0 T_0 \xi_1 \eta, \\ U_0^{\mu\infty} &= \Psi_0^{\mu\infty} = 3 \frac{\mu_0}{c_2} \{\exp[c_2 \xi_2] - 1\} \left[1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right], \\ U_0^{G\infty} &= \Psi_0^{G\infty} = \frac{1}{2} \frac{G_0}{c_3} \{\exp[c_3 \xi_3] - 1\} \left[1 - \frac{B_0^T}{G_0} G'_0 \xi_1 \right]. \end{aligned} \quad (124)$$

Denoted by $\bar{B}_0^\eta$ is the effective isentropic bulk modulus, μ_0 the shear modulus for pure shear, and G_0 the shear modulus for simple shear, all corresponding to tangent moduli at the undeformed reference state for equilibrium quasi-static deformation (no viscoelasticity), in the absence of damage. The latter two are identical in value and definition to μ_0 and G_0 introduced in Eq. 56, since only the bulk modulus, and not squeeze and shear moduli, differ under isothermal and isentropic deformation in isotropic thermoelastic solids, as readily verified via Eqs. 49 and 50. The datum value of entropy at the reference state is chosen as $\eta_0 = 0$ for simplicity. Temperature at the reference state is the constant T_0 . The isentropic pressure derivatives of the shear moduli at the reference state are μ'_0 and G'_0 , numerically equivalent to those obtained from definitions Eq. 57 and Eq. 58. The isothermal bulk modulus is

B_0^T , identical in value to that introduced in Section 3.2. Dimensionless higher-order elastic constants corresponding to dilatation, squeeze, and simple shear are c_1 , c_2 , and c_3 , also identical to those introduced already in Eq. 56, with values in Table 1, enabling Fung-type exponential stiffening in soft biological tissues.^{1,2,23}

Energetic contributions from volume-preserving squeeze and shear modes and from surface tension (third, fourth, and fifth terms) in Eq. 122 are all identical to those introduced already in the free energy-based theory of Eq. 56 in Section 3.2. Shear-related terms are identical since ambient shear moduli are identical for isothermal and isentropic response in isotropic solids, and since the reference internal airway pressure is thought to not affect the shear modulus when transpulmonary pressure is held fixed.⁶⁶ The simple one-parameter model of surface tension ($K_0 = \text{constant}$) does not distinguish between open- and closed-cell regimes or between isothermal and isentropic loading.

The first term in Eq. 122 accounts for the isentropic pressure–volume response and contains the isentropic bulk modulus at the reference state, $\bar{B}_0^\eta$. The second term in Eq. 122 accounts for thermoelastic coupling and contains the Grüneisen parameter at the reference state, g_0 . These two coefficients are defined shortly in the text. The purely entropic contribution to internal energy in Eq. 119 is defined in terms of specific heat at constant volume c_0 , also specified fully later, as follows:

$$u(\eta) = T_0 \eta \left[1 + \frac{1}{2} \frac{\eta}{c_0} \right]. \quad (125)$$

Denote by ρ_t the constant mass density of the condensed tissue phase at the reference state. Denote by ρ_{a0} the mass density of the internal air at the reference state. Treating air as an ideal gas, $\rho_{a0} = (p_{a0}/p_1)\rho_{a1}$, where $\rho_{a1} = 1.2 \times 10^{-3} \text{ g/cm}^3$ is the mass density of air at room temperature T_0 and $p_1 = 1 \text{ atm}$. The reference mass density of the composite lung (condensed tissue + gas phases) is

$$\rho_0(v_t, p_{a0}) = v_t \cdot \rho_t + (1 - v_t) \cdot \rho_{a0}(p_{a0}). \quad (126)$$

In practice, the mass density of the composite lung ρ_{00} is measured in air at a given internal pressure $p_{a0} = p_{pl}$ (*i.e.*, at zero transpulmonary pressure). The reference state mass density ρ_0 is then found from mass conservation as $\rho_0 J_0 = \rho_{00} J_{00}$, where J_{00} is the calculated contraction due to surface tension K_0 computed using the static

spherical response of Section 4.1.1. For properties given in Table 1, $J_{00} = 0.9245$ is obtained from implicit solution of Eq. 94 with $D = 0$. Note that $J_0 = 1$ by definition in the undeformed reference state. Finally, the volume fraction of tissue v_t is obtained by an implicit solution of Eq. 126. In subsequent analysis, ρ_0 and v_t are then treated as constants for a given lung sample and set of initial conditions.

Mixture theory is used to estimate the coefficient of thermal expansion for the composite lung, denoted by A_0 , and the specific heat per unit reference *volume* at constant volume, denoted by c_0 . Denote by subscripts $(\cdot)_t$ and $(\cdot)_a$ quantities for dense tissue and air phases at the reference state at temperature T_0 , and denote by C the specific heat per unit *mass* at constant volume. Then,

$$A_0 = v_t \cdot A_t + (1 - v_t) \cdot A_a, \quad c_0 = v_t \rho_t C_t + (1 - v_t) \rho_{a0} \cdot C_a. \quad (127)$$

For an ideal gas, $A_a = 1/T_0$, and specific heat per unit mass is reasonably approximated as constant at moderate excursions from room temperature,⁸² here $C_a = 718$ J/kg·K. Equations 127 can be formally derived under the condition that temperature T is equal in each phase; otherwise, these relations are viewed as basic modeling assumptions.

The definition of the effective isentropic bulk modulus at the undeformed reference state depends on the loading regime, where an open-cell model is used for static to moderate volumetric strain rates, and a closed-cell model for high rates wherein air does not have time to flow through or exit the alveolar microstructure. Specifically, following Rice and Butler^{32,33},

$$\bar{B}_0^\eta = \begin{cases} B_0^\eta = B_0^T (1 + g_0 A_0 T_0) & \text{(open-cell model),} \\ \left[\frac{v_t}{B_t^\eta} + \frac{1-v_t}{\Gamma p_{a0}} \right]^{-1} & \text{(closed-cell model).} \end{cases} \quad (128)$$

The definition of the open-cell modulus is a standard thermodynamic identity.^{52,82} Recall that B_0^T is the open-cell isothermal bulk modulus introduced already in Eq. 56. The closed-cell modulus results from a referential volume average of the compliance of each of the constituent phases. Results later confirm that this approximation is more accurate than a volume average of the bulk moduli of each phase, where the latter (Voigt approximation) produces a composite bulk modulus that is much too stiff relative to observed wave velocities in the inflated lung. The

closed-cell equation for $\bar{B}_0^\eta$ can be formally derived under the condition that pressure changes in each phase are identical for small average volume changes from the reference state or can be treated as a basic modeling assumption. The latter is more amenable to the present application since pressures and volume changes presumably both vary heterogeneously among phases during passage of a stress wave through the material. For example, absolute tensile pressure can only be supported by the dense tissue phase. The isentropic bulk modulus of the dense phase at the reference state is B_t^η . The isentropic bulk modulus of air under the ideal gas model is $B_a^\eta = \Gamma B_a^T = \Gamma p_{a0}$, with $\Gamma = 1.4$ the ratio of specific heat at constant pressure to that at constant volume of air.

The Grüneisen parameter is obtained from the thermodynamic identity^{52,82}

$$g_0 = A_0 \bar{B}_0^T / c_0, \quad (129)$$

where A_0 and c_0 are found from Eq. 127. The effective isothermal bulk modulus $\bar{B}_0^T$ at the reference state is defined by regime similarly to Eq. 128:

$$\bar{B}_0^T = \begin{cases} B_0^T & \text{(open-cell model),} \\ \left[\frac{v_t}{B_t^T} + \frac{1-v_t}{p_{a0}} \right]^{-1} & \text{(closed-cell model).} \end{cases} \quad (130)$$

Summarizing, thermoelastic coefficients A_0 , c_0 , and g_0 always depend on v_t and p_{a0} . Bulk modulus $\bar{B}_0^T$ depends on v_t and p_{a0} under the closed-cell assumption but is independent of phase volume fraction and airway pressure in the open-cell model, where it becomes the constant B_0^T . Bulk modulus $\bar{B}_0^\eta$ always depends on v_t and p_{a0} , though its dependence in the open-cell model arises only indirectly via g_0 and A_0 . Surface tension coefficient K_0 , shear moduli μ_0 and G_0 and their ambient pressure derivatives, and higher-order elastic constants c_1 , c_2 , and c_3 are all assumed to be constant, irrespective of tissue volume fraction and internal airway pressure. Recall also from Section 3 that K_0 vanishes and B_0^T and c_1 are reduced in value if the lung has been treated to remove surface tension by saline immersion, for example.

5.3 Configurational Free Energy

Viscoelasticity is addressed by an identical theoretical framework as that used for the open-cell, free energy-based treatment of Section 3.3. The viscoelastic response

is the same for closed- and open-cell models because air in the closed-cell model is treated as an inviscid compressible fluid (*e.g.*, ideal gas) that does not contribute to dynamic viscosity. Thermal and entropic effects are omitted in the configurational free and internal energies associated with viscoelasticity, in part because moderate temperature variations do not strongly affect stiffness or hysteresis,⁸³ and because sufficient data do not exist for the lung to enable parameterization of a dynamic viscosity function $\mu_1(T)$ or $\mu_1(\eta)$. The former behavior is contradictory to that of typical polymers whose viscosity is temperature dependent.

Derivations in Eqs. 62–69 still apply for the general framework. The specific relaxation internal energy U_0^1 is equal to the free energy function of Eq. 70:

$$U_0^1(\boldsymbol{\epsilon}) = \Psi_0^1(\boldsymbol{\epsilon}) = \frac{1}{2}\mu_1|\boldsymbol{\epsilon}|^2 = \frac{1}{2}\mu_1 [(\ln J)^2 + \epsilon_1^2 + \epsilon_2^2 + \epsilon_3^2 + \alpha^2 + \beta^2 + \gamma^2]. \quad (131)$$

The total configurational internal energy is then identical to Eq. 71:

$$\Upsilon_0[\boldsymbol{\epsilon}, \boldsymbol{\chi}^1] = \frac{1}{2}\mu_1 [|\boldsymbol{\chi}^1|^2 - 2\boldsymbol{\epsilon} \cdot \boldsymbol{\chi}^1 + |\boldsymbol{\epsilon}|^2] = \frac{1}{2}\mu_1 |\boldsymbol{\chi}^1 - \boldsymbol{\epsilon}|^2. \quad (132)$$

This is partitioned into dilatational, squeeze, and shear contributions, identically to Eqs. 82 and 83:

$$\begin{aligned} \Upsilon_0 &= \Upsilon_0^V + \Upsilon_0^\mu + \Upsilon_0^G; \\ \Upsilon_0^V &= \frac{1}{2}\mu_1(\epsilon_0 - \chi_0^1)^2, \\ \Upsilon_0^\mu &= \frac{1}{2}\mu_1 \sum_{i=1,2,3} (\epsilon_i - \chi_i^1)^2, \\ \Upsilon_0^G &= \frac{1}{2}\mu_1 \sum_{i=4,5,6} (\epsilon_i - \chi_i^1)^2. \end{aligned} \quad (134)$$

The viscous internal stress of Eq. 63 now also obeys $\boldsymbol{\zeta}^1 = -\partial_{\boldsymbol{\chi}^1} U_0 = -\partial_{\boldsymbol{\chi}^1} \Upsilon_0$. Its time evolution is given by the convolution integral in Eq. 73. Internal strains $\boldsymbol{\chi}^\alpha$ and their rates obey Eq. 74.

The squeeze terms in the internal strain vector sum to zero, in analogy with the volume-preserving property of squeeze strain attributes in Eq. 23: $\sum_{i=1,2,3} \chi_i^1 = 0$. This is the same requirement used in Eq. 75 that produces $\sum_{i=1,2,3} \hat{\tau}_i = 0$ in accordance with thermodynamic restriction Eq. 29. The rate of dissipated energy is unconditionally non-negative: $|\boldsymbol{\zeta}^1|^2/\nu_1 = \nu_1|\dot{\boldsymbol{\chi}}^1|^2 \geq 0$. At equilibrium as $t \rightarrow \infty$,

internal strain equals thermoelastic strain: $\chi_\infty^1 = \epsilon$. Two independent parameters (material constants) enter the specific theory for the lung: the dimensionless dynamic bulk modulus amplification factor β_1 and the relaxation time t_1 . These acquire the same values here as proposed in Section 3 and calibrated in Section 4 via analysis of stress relaxation in air-filled lung, listed in Table 1. Dynamic viscous stiffness is $\mu_1 = \beta_1 B_0^T$ and dynamic viscosity is $\nu_1 = \mu_1 t_1$.

5.4 Damage

The internal state variable representative of deleterious processes that affect mechanical or biological functions is universally denoted by the internal state variable $D \in [0, 1]$. The internal energy-based theory requires prescription of the degradation functions $\bar{f}(D)$ and $f(D)$ in Eq. 119 and specification of how $D(\mathbf{X}, t)$ may change during the history of loading at a material point $\mathbf{X}$.

The thermodynamic driving force for damage in Eq. 51 is found in terms of thermoelastic strain, viscous internal strain, and entropy by equating partial derivatives of free and internal energies with respect to D :

$$\bar{F} = - \left. \frac{\partial \Psi}{\partial D} \right|_{\epsilon, \chi^\alpha, T} = - \left. \frac{\partial U}{\partial D} \right|_{\epsilon, \chi^\alpha, \eta}. \quad (135)$$

This derivation is analogous to that followed for obtaining stress components as partial strain derivatives of internal energy in Eq. 45.^{5,7} The thermodynamic driving force for damage of Eq. 135 is then, from differentiation of Eq. 119,

$$\begin{aligned} \bar{F} &= - \left[\frac{d\bar{f}}{dD} U_0^V + \frac{df}{dD} (U_0^\mu + U_0^G) \right] \\ &= -\bar{f}' [U_0^{V\infty} + \Upsilon_0^V] - f' [U_0^{\mu\infty} + \Upsilon_0^\mu + U_0^{G\infty} + \Upsilon_0^G]. \end{aligned} \quad (136)$$

The linear degradation function of classical damage mechanics introduced in Section 3.4 is retained for f , albeit here in the context of internal energy rather than free energy:

$$f(D) = 1 - D, \quad f' = df/dD = -1. \quad (137)$$

This function is contrasted with quadratic functions used in phase field and other generalized continuum theories,^{84–87} which are all admissible in the generic thermodynamic framework of recent publications.^{5,7} The degradation function $\bar{f}$ is defined

as

$$\begin{aligned}\bar{f}(D) &= 1 - \bar{D}(D); \\ \bar{D} &= \begin{cases} D \cdot \left(1 - \frac{B_a^\eta}{B_0^\eta}\right) = D \cdot \left(1 - \frac{\Gamma p_{a0}}{B_0^\eta}\right) = D \cdot (1 - \bar{\gamma}) & \text{(closed-cell model),} \\ D & \text{(open-cell model).} \end{cases}\end{aligned}\tag{138}$$

Denoted by $\bar{\gamma} \leq 1$ is the ratio of the bulk modulus of the gas phase to that of the composite material for the closed-cell theory of Section 5.2. Accordingly, as $D \rightarrow 1$, $\bar{f} \rightarrow \bar{\gamma}$, and the bulk stiffness of the composite at null applied strain reduces to that of the air contained within: $\bar{B}_0^\eta \rightarrow B_{a0}^\eta = \Gamma p_{a0}$. From Eq. 138, the derivative with respect to damage is

$$\bar{f}'(D) = \begin{cases} \bar{\gamma} - 1 & \text{(closed-cell model),} \\ -1 & \text{(open-cell model).} \end{cases}\tag{139}$$

It follows from insertion of Eqs. 137, 138, and 139 into Eq. 135 that, in order to ensure damage evolution is physically and thermodynamically irreversible,

$$\dot{D} \geq 0, \quad \bar{F}\dot{D} = \bar{U}_0\dot{D} \geq 0.\tag{140}$$

Conditional internal energy density per unit reference volume $\bar{U}_0 = \bar{F}$ is defined as

$$\bar{U}_0 = \begin{cases} (1 - \bar{\gamma})U_0^V + U_0^\mu + U_0^G & \text{(closed-cell model),} \\ U_0 & \text{(open-cell model).} \end{cases}\tag{141}$$

Internal energy $\bar{U}_0$ of the undamaged material, with the bulk energy factored by $(1 - \bar{\gamma})$ in the closed-cell case, is thermodynamically conjugate to the rate of damage growth in the dissipation product $\bar{F}\dot{D}$ entering total dissipation rate $\mathfrak{D}$ of Eq. 53.

A canonical constitutive assumption is that D , or its rate, should be affected by U_0 or $\bar{U}_0$. The following dimensionless transformation of U_0 is defined for use subse-

quently as a driving force or indicator of damage:

$$Z(U_0, \nabla_0 U_0) = \begin{cases} \left[2 \left| \frac{U_0^{V\infty}}{B_0^\eta} + \frac{\Upsilon_0^V}{B_0^\eta} + \frac{U_0^{\mu\infty} + \Upsilon_0^\mu}{\mu_0} + \frac{U_0^{G\infty} + \Upsilon_0^G}{G_0} \right| \right]^{1/2} \cdot [1 + \omega(|\nabla U_0|)] & (\text{if } \bar{U}_0 \geq 0), \\ 0 & (\text{if } \bar{U}_0 < 0). \end{cases} \quad (142)$$

This function matches that of the free energy-based damage model of Eq. 79, intended for low to moderate strain rates in Section 3.4, with two exceptions. First, internal energy density normalized by the isentropic bulk modulus in Eq. 142 replaces free energy density normalized by the isothermal bulk modulus in Eq. 79. Second, the factor $1 + \omega$ appears only in the present internal energy-based model intended for moderate to high strain rates, most notably those involving wave propagation. Arguments of Z are indicated only implicitly by those in parenthesis on the left of Eq. 142. Rather, Z depends explicitly on partitioned and normalized contributions from U_0 and its reference gradient ∇U_0 , where the latter only affects Z through ω .

The function ω is defined as follows, with ω_D a dimensionless non-negative scalar constant and l a length parameter later associated with alveolar diameter and used for normalization along with the open-cell bulk modulus B_0^η :

$$\omega = \omega_D \sqrt{\frac{l}{B_0^\eta}} |2\nabla_0 U_0|^{1/2} = \omega_D \sqrt{\frac{2l}{B_0^\eta}} \left[\frac{\partial U_0}{\partial X_K} \frac{\partial U_0}{\partial X_K} \right]^{1/4} \geq 0. \quad (143)$$

The function ω is included in the driving force Z to account for deleterious effects of pressure gradients on the lung when subjected to blast loading:^{9,12,13} steep pressure rises have been experimentally and numerically associated with more severe injuries than those incurred by gradual loading to similar overall stress and strain magnitudes.

The damage evolution law makes use of the maximum value of instantaneous driving force Z defined in Eq. 142 over the time history of deformation at material point $\mathbf{X}$, analogously to Eq. 84:

$$Z_m(\mathbf{X}, t) = \max_{s \in (-\infty, t]} Z(\mathbf{X}, s). \quad (144)$$

Damage then obeys the constitutive equation set forth in Eq. 85, where now Z_m accounts for normalized energy and its material gradient:

$$D(\mathbf{X}, t) = \begin{cases} \exp \left[\frac{\alpha_D \{Z_m(\mathbf{X}, t) - Z_0\}}{Z_0} \right] - 1 & (\text{if } \frac{Z_m}{Z_0} \in \left[1, 1 + \frac{\ln 2}{\alpha_D} \right]), \\ 0 & (\text{if } \frac{Z_m}{Z_0} < 1), \\ 1 & (\text{if } \frac{Z_m}{Z_0} > 1 + \frac{\ln 2}{\alpha_D}). \end{cases} \quad (145)$$

Parameters $Z_0 > 0$ and $\alpha_D \geq 0$ denoting the threshold for damage initiation and quantifying its rate of accumulation, respectively, are unchanged in physical meaning from those already used in Sections 3 and 4, with values given in Table 1. The material time derivative of Eq. 145 is calculated as

$$\dot{D} = \begin{cases} \frac{\alpha_D}{Z_0} (1 + D) \frac{dZ_m}{dt} & (\text{if } \frac{Z_m}{Z_0} \in \left[1, 1 + \frac{\ln 2}{\alpha_D} \right]), \\ 0 & (\text{otherwise}). \end{cases} \quad (146)$$

Since Z_m can only remain constant or increase with time via its definition in Eq. 144, and because $\alpha_D \geq 0$ and $Z_0 > 0$, $\dot{D} \geq 0$. Furthermore, $\dot{Z}_m > 0$ only in the domain $\bar{U}_0 \geq 0$ according to the definition of Z in Eq. 142. Therefore, $\bar{U}_0 \dot{D} = \bar{F} \dot{D} \geq 0$, ensuring positive dissipation from damage kinetics according to Eq. 140. This model requires only three parameters— Z_0 , α_D , and ω_D —where the third is the only additional parameter over the model calibrated already for static loading of the lung in Section 4.

5.5 Stresses, Entropy, and Temperature

Derivatives of the internal energy function consisting of terms for thermoelasticity, viscoelasticity, and damage set forth in Sections 5.2, 5.3, and 5.4, respectively, produce stress components and temperature according to Eq. 45. Equilibrium internal energy U_0^∞ of Eq. 122, thermal energy u of Eq. 125, configurational energy of Υ_0 of Eq. 132, and damage degradation functions f of Eq. 137 and $\bar{f}$ of Eq. 138 are

substituted into Eq. 119 to yield the following total internal energy function:

$$\begin{aligned}
U(\xi_1, \xi_2, \xi_3, \eta, \chi^1, D) &= (1 - \bar{D}) \left[\frac{1}{2} \frac{\bar{B}_0^\eta}{c_1} \{ \exp[c_1(\xi_1)^2] - 1 \} + K_0 \xi_1 - g_0 T_0 \eta \xi_1 + \frac{1}{2} \mu_1 (\epsilon_0 - \chi_0^1)^2 \right] \\
&+ (1 - D) \left[3 \frac{\mu_0}{c_2} \{ \exp[c_2 \xi_2] - 1 \} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0 \xi_1 \right) \right] \\
&+ (1 - D) \left[\frac{1}{2} \frac{G_0}{c_3} \{ \exp[c_3 \xi_3] - 1 \} \left(1 - \frac{B_0^T}{G_0} G'_0 \xi_1 \right) \right] \\
&+ (1 - D) \left[\frac{1}{2} \sum_{i=1}^6 \mu_1 (\epsilon_i - \chi_i^1)^2 \right] + T_0 \eta \left(1 + \frac{1}{2} \frac{\eta}{c_0} \right).
\end{aligned} \tag{147}$$

Stress components conjugate to strain attributes obtained from Eqs. 45 and 147 are

$$\begin{aligned}
\hat{\tau}_0 &= (1 - \bar{D}) \{ \bar{B}_0^\eta [\epsilon_0 \exp(c_1 \xi_1^2)] - g_0 T_0 \eta + B_0^T \beta_1 (\epsilon_0 - \chi_0^1) + K_0 \} \\
&\quad - (1 - D) B_0^T \{ 3 \mu'_0 \xi_2 + \frac{1}{2} G'_0 \xi_3 \}, \\
\hat{\tau}_1 &= 6(1 - D) \mu_0 [\epsilon_1 \exp(c_2 \xi_2) (1 - \{ B_0^T / \mu_0 \} \mu'_0 \xi_1) + \beta_2 (\epsilon_1 - \chi_1^1)], \\
\hat{\tau}_2 &= 6(1 - D) \mu_0 [\epsilon_2 \exp(c_2 \xi_2) (1 - \{ B_0^T / \mu_0 \} \mu'_0 \xi_1) + \beta_2 (\epsilon_2 - \chi_2^1)], \\
\hat{\tau}_3 &= 6(1 - D) \mu_0 [\epsilon_3 \exp(c_2 \xi_2) (1 - \{ B_0^T / \mu_0 \} \mu'_0 \xi_1) + \beta_2 (\epsilon_3 - \chi_3^1)], \\
\hat{\tau}_4 &= (1 - D) G_0 [\epsilon_4 \exp(c_3 \xi_3) (1 - \{ B_0^T / G_0 \} G'_0 \xi_1) + \beta_3 (\epsilon_4 - \chi_4^1)], \\
\hat{\tau}_5 &= (1 - D) G_0 [\epsilon_5 \exp(c_3 \xi_3) (1 - \{ B_0^T / G_0 \} G'_0 \xi_1) + \beta_3 (\epsilon_5 - \chi_5^1)], \\
\hat{\tau}_6 &= (1 - D) G_0 [\epsilon_6 \exp(c_3 \xi_3) (1 - \{ B_0^T / G_0 \} G'_0 \xi_1) + \beta_3 (\epsilon_6 - \chi_6^1)].
\end{aligned} \tag{148}$$

Three dimensionless stiffness ratios β_i ($i = 1, 2, 3$) are identical to those defined already in Eq. 89. Only one of μ_1 and the three β_i is independent. Instantaneous dynamic moduli at the reference state ($\xi_i = 0$, $D = 0$, $\chi^1 = \mathbf{0}$) are $\bar{B}_0^\eta + \beta_1 B_0^T$, $\mu_0 \cdot (1 + \beta_2)$, and $G_0 \cdot (1 + \beta_3)$. Cauchy stress $\boldsymbol{\sigma}$ can be obtained from $\hat{\tau}_i$ and Eqs. 44 and 24 when $\mathbf{F} = \mathbf{Q}\mathbf{R}$ is imposed at a material point. Verification is straightforward that, when Eq. 75 is invoked, the constraints $\sum_{i=1,2,3} \hat{\tau}_i = 0$ of Eqs. 29 and 40 are obeyed automatically for squeeze components of stress.

Absolute temperature is determined from Eq. 45 as follows, recalling that Υ_0 , U_0^μ ,

and U_0^G do not depend on η :

$$T = \frac{\partial}{\partial \eta} [\bar{f}U_0^{V\infty} + u] = T_0 \left[1 - (1 - \bar{D})g_0 \ln J + \frac{\eta}{c_0} \right]. \quad (149)$$

Accordingly, temperature tends to increase with decreasing volume and increasing entropy. Total dissipation in Eq. 53 is, similarly to Eq. 91,

$$\mathfrak{D} = \beta_1 B_0^T t_1 \left[(1 - \bar{D})(\dot{\chi}_0^1)^2 + (1 - D) \sum_{i=1}^6 (\dot{\chi}_i^1)^2 \right] + \bar{U}_0 \dot{D} \geq 0, \quad (150)$$

where the (in)equality is verified by Eqs. 69 and 140 with dynamic viscosity $\nu_1 = \beta_1 B_0^T t_1 > 0$ in the former. An energy balance similar to Eq. 92 in the form of a temperature rate equation still holds, but this is not needed since Eq. 149 can be used to obtain the temperature when deformation and entropy are known. For continuous processes, the latter is obtained from integration of the differential equation in Eq. 53:

$$\dot{\eta} = \frac{1}{T} [\mathfrak{D} - \nabla_0 \cdot \mathbf{q} + r], \quad (151)$$

where heat flux $\mathbf{q}$ and heat source r vanish by definition only for adiabatic problems.

6. Low-Amplitude Waves and Shock Waves

The constitutive model of Section 5 is applied to problems of wave propagation in the lung. Introductory comments of Section 4 continue to hold here. Physical properties and parameter values derived in the static analysis in the context of the free energy-based theory of Section 3 and Table 1 also remain valid. However, several additional parameters are needed for application of the internal energy-based model of Section 5, namely, those accounting for air compressibility in the closed-cell formulation and amplification effects of energy gradients on damage and injury criteria as applicable in the context of shock and blast waves.

6.1 Composite Properties and Wave Speeds

Addressed first is the response of lung tissue under low-amplitude isentropic wave propagation. Elastic longitudinal and shear waves are associated with dynamic displacement gradients of infinitesimal magnitude, per the usual assumptions of linear elastodynamics.^{88,89} The initial state of the lung may be statically pressurized and

finitely spherically deformed from an unstressed state, but wave motion is restricted to small deformations and small deformation gradients superposed on the initial state. Large-amplitude waves, specifically planar shock waves of finite compressive strain, are addressed later in Section 6.2.

An upper triangular form of the deformation gradient matrix is sufficiently general to address deformations considered in what follows next. As such, a global coordinate system is chosen such that $\mathbf{Q} = \mathbf{1}$, following McClellan.⁹⁰ In this context, $\mathbf{F} = \mathbf{R}$ cannot be arbitrary, but rather the Laplace stretch must be integrable to a vector potential.^{5,7} Let $\mathbf{u}(\mathbf{X}, t)$ denote the displacement field. An upper triangular deformation gradient, measured relative to the initial state, can be written

$$\begin{aligned}
 \mathbf{F} = \mathbf{1} + \nabla_0 \mathbf{u} = \mathbf{R} &= \begin{bmatrix} 1 + \partial u_1 / \partial X_1 & \partial u_1 / \partial X_2 & \partial u_1 / \partial X_3 \\ 0 & 1 + \partial u_2 / \partial X_2 & \partial u_2 / \partial X_3 \\ 0 & 0 & 1 + \partial u_3 / \partial X_3 \end{bmatrix} \\
 &= \begin{bmatrix} 1 + e_{11} & 2e_{12} & 2e_{13} \\ 0 & 1 + e_{22} & 2e_{23} \\ 0 & 0 & 1 + e_{33} \end{bmatrix} \\
 &= \begin{bmatrix} 1 + e_1 & e_4 & e_6 \\ 0 & 1 + e_2 & e_5 \\ 0 & 0 & 1 + e_3 \end{bmatrix}.
 \end{aligned} \tag{152}$$

Components of the linear strain tensor are $e_{ij} = \frac{1}{2}(\partial u_i / \partial X_j + \partial u_j / \partial X_i)$, and e_α ($\alpha = 1, 2, \dots, 6$) are components of strain in Voigt notation. This useful form of $\mathbf{F}$ was first derived and emphasized by McClellan.⁹⁰ Under the condition that $|e_\alpha| \ll 1$, the following relationships to strain attributes of Eq. 22 are derived:

$$\begin{aligned}
 \epsilon_0 &= \ln(abc) = \ln J = \ln(1 + \partial u_i / \partial X_i + \dots) \approx \partial u_i / \partial X_i = e_1 + e_2 + e_3, \\
 \epsilon_1 &= \frac{1}{3} \ln(a/b) = \frac{1}{3} [\ln(1 + e_1) - \ln(1 + e_2)] \approx \frac{1}{3}(e_1 - e_2), \\
 \epsilon_2 &= \frac{1}{3} \ln(b/c) = \frac{1}{3} [\ln(1 + e_2) - \ln(1 + e_3)] \approx \frac{1}{3}(e_2 - e_3), \\
 \epsilon_3 &= \frac{1}{3} \ln(c/a) = \frac{1}{3} [\ln(1 + e_3) - \ln(1 + e_1)] \approx \frac{1}{3}(e_3 - e_1), \\
 \epsilon_4 &= \gamma \approx (1 + e_1)\gamma = a\gamma = e_4, \\
 \epsilon_5 &= \alpha \approx (1 + e_2)\alpha = b\alpha = e_5, \\
 \epsilon_6 &= \beta \approx (1 + e_1)\beta = a\beta = e_6.
 \end{aligned} \tag{153}$$

Succinctly, $\epsilon_0 \approx \text{tre}$, and $\epsilon_\alpha \approx e_\alpha$ for $\alpha = 4, \dots, 6$.

Next the stress–strain equations of Eq. 148 are linearized about a reference state at pressure p_0 under isentropic conditions pertinent to elastic wave propagation, in the absence of damage ($D = 0$). For instantaneous response corresponding to high-frequency waves, no viscoelastic relaxation occurs, and thus $\chi_i^1 = 0$. Terms of $O(|e|^2)$ and higher are truncated under linearization, leaving

$$\begin{aligned}
\hat{\tau}_0 &= \bar{B}_0^\eta \left[\epsilon_0 \exp(c_1 \xi_1^2) - 3\mu'_0 \xi_2 - \frac{1}{2} G'_0 \xi_3 + \bar{\beta}_1 \epsilon_0 \right] - p_0 \\
&\approx \bar{B}_0^\eta \cdot (1 + \bar{\beta}_1)(e_1 + e_2 + e_3) - p_0, \\
\hat{\tau}_1 &= 6\mu_0 \left[\epsilon_1 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2 \epsilon_1 \right] \approx 2\mu_0 \cdot (1 + \beta_2)(e_1 - e_2), \\
\hat{\tau}_2 &= 6\mu_0 \left[\epsilon_2 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2 \epsilon_2 \right] \approx 2\mu_0 \cdot (1 + \beta_2)(e_2 - e_3), \\
\hat{\tau}_3 &= 6\mu_0 \left[\epsilon_3 \exp(c_2 \xi_2)(1 - \{B_0^T/\mu_0\}\mu'_0 \xi_1) + \beta_2 \epsilon_3 \right] \approx 2\mu_0 \cdot (1 + \beta_2)(e_3 - e_1), \\
\hat{\tau}_4 &= G_0 \left[\epsilon_4 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3 \epsilon_4 \right] \approx G_0 \cdot (1 + \beta_3)e_4, \\
\hat{\tau}_5 &= G_0 \left[\epsilon_5 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3 \epsilon_5 \right] \approx G_0 \cdot (1 + \beta_3)e_5, \\
\hat{\tau}_6 &= G_0 \left[\epsilon_6 \exp(c_3 \xi_3)(1 - \{B_0^T/G_0\}G'_0 \xi_1) + \beta_3 \epsilon_6 \right] \approx G_0 \cdot (1 + \beta_3)e_6.
\end{aligned} \tag{154}$$

Letting $\bar{\beta}_1 = \beta_1 \cdot B_0^T / \bar{B}_0^\eta$, dynamic thermoelastic moduli are $\bar{B}_D^\eta = \bar{B}_0^\eta \cdot (1 + \bar{\beta}_1) = \bar{B}_0^\eta + \beta_1 B_0^T$, $\mu_D = (1 + \beta_2) \cdot \mu_0$, and $G_D = (1 + \beta_3) \cdot G_0$. Then, from Eq. 28, linearized constitutive equations for Kirchhoff stress components $s_{ij} = k_{ij}$ are

$$\begin{aligned}
s_{11} + s_{22} + s_{33} &\approx 3\bar{B}_D^\eta \cdot (e_1 + e_2 + e_3) - 3p_0, \\
s_{11} - s_{22} &\approx 2\mu_D \cdot (e_1 - e_2), \\
s_{22} - s_{33} &\approx 2\mu_D \cdot (e_2 - e_3), \\
s_{33} - s_{11} &\approx 2\mu_D \cdot (e_3 - e_1), \\
s_{12} &\approx G_D \cdot e_4, \\
s_{23} &\approx G_D \cdot e_5, \\
s_{31} &\approx G_D \cdot e_6.
\end{aligned} \tag{155}$$

These equations are identical to those of linear anisotropic elasticity for solids with cubic symmetry and an initial homogeneous pressure p_0 , positive in compression. When $\mu_D = G_D$, they reduce to the constitutive equations for linear isotropic elasticity. For low-frequency waves ($\chi_i^1 = \epsilon_i$), or in the absence of viscoelasticity (e.g., saline-washed lung, $\beta_i = 0$), the linearized stress–strain equations are of

identical form, but with static moduli replacing dynamic moduli. Strain components e_α are those measured relative to the initial state wherein $\mathbf{F} = J_0^{1/3} \mathbf{1}$. The initial pressure from surface tension, $-K_0$, is assumed included in p_0 , and thus is not explicitly written in Eqs. 154 or 155. Strictly, some elastic coefficients entering Eq. 155 as reported in Table 1 have been obtained relative to an undeformed state wherein $J = J_0 = 1$, and where $p_0 = -K_0$ in air or $p_0 = 0$ in saline, and thus their values could differ under large initial deformations with $|J_0 - 1| \gg 0$. If the material is homogeneously damaged in the initial state with a uniform field $D = D_0$, then Eq. 155 still applies, with each of the dynamic moduli reduced by a factor of $1 - D_0$.

Under the foregoing small-deformation assumptions for geometrically linear mechanics, the equations of motion in the absence of body forces are^{88,89}

$$\frac{\partial s_{ij}}{\partial X_j} = \rho_0 \frac{\partial^2 u_i}{\partial t^2}, \quad (156)$$

where ρ_0 is the mass density in the initial state upon which wave motion is superposed. Addition of a homogeneous initial stress field (*e.g.*, p_0) does not affect the left side of Eq. 156.

For the rectilinear Cartesian coordinate system associated with Eq. 152, each of the three coordinate axes is a pure mode direction for plane wave motion. Each axis permits a longitudinal plane wave (displacement parallel to propagation direction) propagating at velocity c_L and two shear plane waves (polarization orthogonal to propagation direction) propagating at velocity c_S . Considering Eqs. 155 and 156, wave speeds are deduced to be of the standard form^{91,92}

$$c_L = \sqrt{\frac{\bar{B}_D^\eta + \frac{4}{3}\mu_D}{\rho_0}}, \quad c_S = \sqrt{\frac{G_D}{\rho_0}}, \quad (157)$$

where in general, now admitting a homogeneous initial damage field D_0 , the tangent moduli in the initial state are

$$\begin{aligned} \bar{B}_D^\eta &= \bar{B}_0^\eta \cdot (1 + \bar{\beta}_1)[1 - (1 - \bar{\gamma})D_0], \\ \mu_D &= \mu_0 \cdot (1 + \beta_2)(1 - D_0), \\ G_D &= G_0 \cdot (1 + \beta_3)(1 - D_0). \end{aligned} \quad (158)$$

For the case of isotropic properties (*i.e.*, identical squeeze and simple shear moduli $\mu_D = G_D$) all directions are pure mode directions. In the absence of viscoelastic effects, $\bar{\beta}_1 = \beta_2 = \beta_3 = 0$ in Eq. 158.

Properties of each of the two phases composing the composite lung, and thus affecting its composite thermoelastic properties and wave speeds through Eq. 157, are listed below the horizontal demarcation line in Table 2. Notably, the mass density of the dense tissue phase ρ_t and its isentropic bulk modulus B_t^η are obtained from the closed-cell treatment of sound speeds in parenchyma of Rice.³² These two values are realistically unaffected by any initial static (transpulmonary) pressure p_0 and any corresponding static volume change J_0 of a composite material element. Since the bulk modulus of the dense phase is six orders of magnitude larger than that of B_0^T and four orders of magnitude larger than B_{a0}^η , the magnitude of initial pressure required to enact any measurable change of B_t^η , assuming a pressure derivative $B_t' \approx 5.3$ comparable to water,⁹³ cannot be attained for physically realistic initial deformations. Mass density and specific heat of air are standard and universally known; specific heats and thermal expansion coefficients for the dense tissue phase are estimated from soft tissue-relevant properties for collagen and elastin.^{94–97}

Table 2 Parameters for closed-cell internal energy-based response, supplementing Table 1

Property [units]	Value	Description
ρ_0 [g/cm ³]	0.337	composite lung mass density at $J = 1$
v_t	0.336	solid-liquid tissue volume fraction
$\bar{B}_0^\eta$ [kPa]	213	composite isentropic bulk modulus ($p_{a0} = 1$ atm)
c_0 [kPa/K]	741	composite specific heat capacity ($p_{a0} = 1$ atm)
A_0 [1/K]	2.39×10^{-3}	composite thermal expansion ($T_0 = 310$ K)
g_0	4.91×10^{-4}	composite Grüneisen parameter ($p_{a0} = 1$ atm)
Ω_D	8	energy/pressure gradient contribution to damage
l [μm]	60.1	mean alveolar diameter
ρ_{a0} [g/cm ³]	1.2×10^{-3}	air density at 1 atm
ρ_t [g/cm ³]	1.003	reference solid-liquid tissue density at $J_0 = 1$
B_t^η [kPa]	2.3×10^6	solid-liquid isentropic bulk modulus
C_t, C_g [J/kg·K]	2200, 718	specific heats of solid-liquid and air phases

Like those of Table 1, composite properties above the demarcation line correspond to dog lung. Composite mass density ρ_0 is obtained from the stress-free mass density $\rho_{00} = 3.65$ g/cm³ reported by Vawter²² using the analysis procedure described in Section 5.2. Densities of each phase then enter Eq. 126, which is solved itera-

tively for the value of v_t listed in Table 2. The composite bulk modulus $\bar{B}_0^\eta$, composite specific heat c_0 , composite thermal expansion coefficient A_0 , and composite Grüneisen parameter g_0 are obtained using relations in Eqs. 127, 128, and 129, where the closed-cell model equations are invoked in the context of Table 2. The numerical value listed for damage amplification parameter Ω_D is justified later in Section 6.2, wherein the alveolar diameter l (value for dog lung from Fung¹) first enters the description in the context of minimum shock front width.

Several prominent features are of note regarding values in Table 2. Firstly, the closed-cell bulk modulus $\bar{B}_0^\eta$ is on the order of 200 kPa for an internal airway pressure of 1 atm, compared to the value of static isothermal bulk modulus B_0^T in Table 1 of 2.5 kPa in air and 2.0 kPa in saline. The open-cell isentropic bulk modulus computed from Eq. 128 is nearly indistinguishable, essentially also 2.5 kPa in air and 2.0 kPa in saline. Thus, the closed-cell modulus is two orders of magnitude larger than the open-cell modulus, demonstrating the dominant effect of air compressibility. Effects of viscoelasticity on $\bar{B}_D^\eta$ are effectively negligible since $\beta_1 = 0.6$ from Table 1 and $\bar{\beta}_1 = \beta_1 B_0^T / \bar{B}_0^\eta \approx 0.007$. In the absence of viscosity, $G_D = G_0 = \mu_0 = \mu_D = 1$ kPa, and low-amplitude elastic wave propagation is fully isotropic. When viscoelasticity is incorporated, then from Eq. 89, $\beta_3 = 6\beta_2 = \frac{5}{2}\beta_1 = \frac{3}{2}$, so wave propagation becomes mildly anisotropic, notably shear wave speed depends on directions of propagation and polarization. However, $\mu_D \approx G_D \ll \bar{B}_D^\eta$, since dynamic shear moduli are two orders of magnitude smaller than the composite closed-cell bulk modulus for an airway pressure of 1 atm. Therefore, for the range of parameters considered here and an internal air pressure $p_{a0} \approx 1$ atm, longitudinal wave propagation is effectively isotropic with a velocity essentially equal to the bulk sound speed:

$$c_L \approx \sqrt{\bar{B}_D^\eta / \rho_0} \approx \sqrt{\bar{B}_0^\eta / \rho_0}. \quad (159)$$

The initial closed-cell bulk modulus $\bar{B}_D^\eta \approx \bar{B}_0^\eta$ increases in magnitude when initial airway pressure p_{a0} increases according to Eq. 128. This in turn results in an increase in longitudinal wave propagation speed c_L via Eq. 157.

Results of Eqs. 128 and 157 for the closed-cell model are reported in Fig. 10. Shown in Fig. 10a versus p_{a0} are this closed-cell dynamic bulk modulus normalized by the open-cell modulus $B_0^\eta \simeq B_0^T$ for lung in air and saline, with respective values of 2.5

and 2.0 kPa. The condition $J_0 = 1$ is imposed for calculations reported in Fig. 10a, meaning that the initial transpulmonary pressures are $p_0 = -K_0 = 0.2$ kPa in air and $p_0 = 0$ in saline. As internal air pressure increases from 0.1 to 10 atm, the ratio of dynamic to static (*i.e.*, closed- to open-cell) bulk modulus increases from around 10 to around 1000.

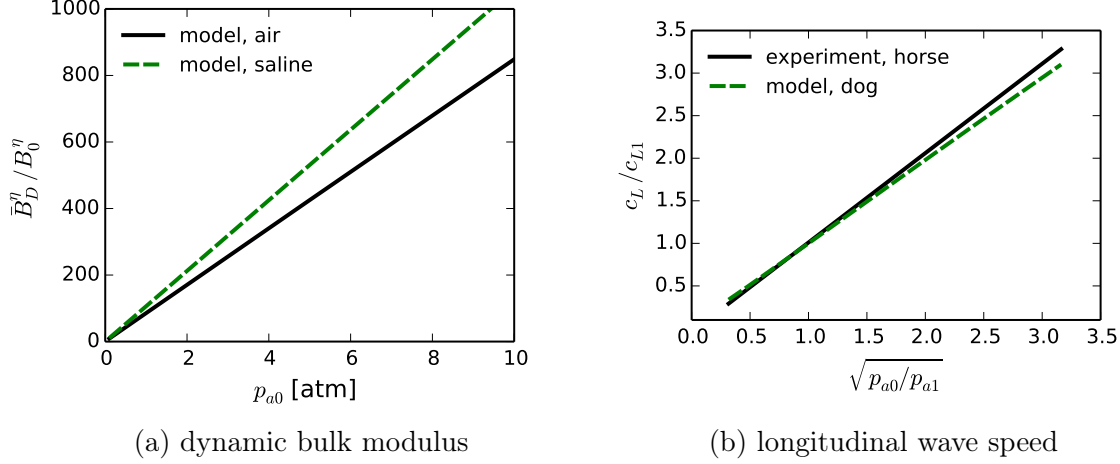


Fig. 10 Predictions for isentropic dynamic bulk modulus and longitudinal wave speed versus initial airway pressure normalized by $p_{a1} = 1$ atm, with experimental data for horse lung in air³²: a) bulk modulus $\bar{B}_D^\eta$ computed from Eqs. 128 and 158 at reference state with $J_0 = 1$ and $D_0 = 0$ and b) wave speed c_L computed from Eq. 157 at reference state of $p_{tp0} = 5$ cm H₂O

Shown in Fig. 10b versus initial airway pressure are longitudinal wave speeds computed from the first of Eq. 157, normalized by wave speed $c_{L1} = 27.1$ m/s, where the latter is a reference value computed at a fixed internal airway pressure of $p_{a1} = 1$ atm. Here, transpulmonary pressure is fixed at $p_{tp0} = -p_0 = 5$ cm H₂O = 0.49 kPa. Complementary experimental ratios of wave velocity versus internal airway pressure, measured at the same fixed transpulmonary pressure of 5 cm H₂O in horse lung,³² are shown for comparison. The current model predicts trends for dog lung that very closely match the data for horse lung, providing confidence in the present methodology for computing the effective dynamic bulk modulus and longitudinal sound speed for high-frequency wave motion. A Voigt-averaged bulk modulus of the lung would be on the order of 773 MPa, yielding wave speeds too large by a factor on the order of 50.

Finally, shown in Fig. 11 are predictions of c_L given by the present model for dog lung in Eq. 157 for a fixed external (pleural) pressure of $p_{pl} = 1$ atm and a vari-

able initial transpulmonary pressure $p_{tp0} = -p_0$ up to a value of approximately 2 kPa. According to Eq. 93, initial airway pressure p_{a0} is close to atmospheric since $|p_0| \lesssim 2 \text{ kPa} \ll 101 \text{ kPa} = 1 \text{ atm}$. Since B_t^η and $B_a^\eta = \Gamma p_{a0}$ do not depend on transpulmonary pressure, effects of p_{tp0} on c_L in the present model emerge primarily from effects of transpulmonary pressure on J_0 , and thus on initial mass densities ρ_t , ρ_{a0} , and finally ρ_0 according to Eq. 126. Specifically, $\rho_t \rightarrow \rho_t/J_0$, where the reference value at $J_0 = 1$ is given in Table 2. Shear moduli are affected by initial transpulmonary pressure associated with expansion J_0 , *e.g.*, $\mu_0 \rightarrow \mu_0 - \mu'_0 B_0^T \ln J_0$, where values of $\mu_0 = G_0 = 1 \text{ kPa}$ and $\mu'_0 = G'_0 = -0.7$ are listed in Table 1. However, the value of the shear modulus trivially affects c_L since $\bar{B}_D^\eta$ exceeds μ_D by two orders of magnitude. As p_{tp0} increases, the lung expands, and mass density ρ_0 decreases, leading to an increase in wave speed. In reality, a change in $\bar{B}_D^\eta$ might be expected with initial expansion $J_0 > 1$, but this effect is deemed of secondary importance relative to density changes as indicated by data of Yen⁶⁶ and is omitted in the present application of the model.

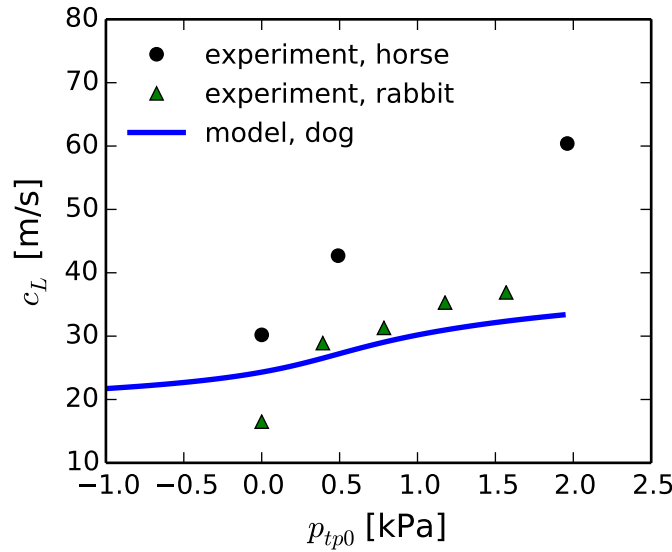


Fig. 11 Predictions for longitudinal wave speed c_L computed from Eq. 157 at ambient external pressure ($p_{pl} = 1 \text{ atm}$) versus initial transpulmonary pressure p_{tp0} : experimental data for horse lung in air³² and rabbit lung in air⁶⁶

Predictions are compared with high-frequency response data on horse lung³² as well as data on rabbit lung measured under sealed trachea conditions,⁶⁶ where in both references a closed-cell model has been stated as appropriate. Maximum ini-

tial transpulmonary pressures probed in the model and these experiments are on the order of 2 kPa or 20 cm H₂O, just beneath those required to cause edema in saline-treated rabbit lung.⁴⁴ At the upper end of this pressure range, according to the present theory, minimal damage D_0 and stiffness reduction incurred in the initial state due to tensile transpulmonary pressure p_{tp0} do not significantly affect tangent moduli or wave speeds. Supporting calculations for damage effects under spherical expansion are explained in Section 4.1.2 and quantified by results in Fig. 4b. Wave motion itself is presumed infinitesimal and thus incapable of inducing further damage. Model predictions of wave speed c_L in dog lung reasonably match those for rabbit lung⁶⁶ at similar transpulmonary pressures ranging from approximately 0.4 to 1.6 kPa. Agreement with data on horse lung is reasonable at null transpulmonary pressure. Significant differences emerge among experimental data sets at low and high transpulmonary pressures, notably the much higher wave speed reported for horse lung at $p_{tp0} \approx 2$ kPa. Since mass densities reported for rabbit and horse are comparable at this pressure, the difference in wave speeds is thought primarily due to a larger tangent bulk stiffness in the horse lung compared to rabbit and dog lung.

It is emphasized that all of the properties listed in Table 2, with the exception of Ω_D that does not enter the present treatment of low-amplitude elastic waves, are obtained directly from fundamental measurements and the basic homogenization equations of Section 5.2. No parameters have been adjusted or calibrated to obtain results in Figs. 10 or 11, demonstrating validation of the model predictions in this context.

A few remarks on applicability of the model in the context of cited experiments are in order. Experiments⁶⁶ induce wave motion via localized impact of a water jet to exposed lung tissue at a prescribed initial transpulmonary pressure. As noted by Yen,⁶⁶ the water jet may impart sufficient pressures to induce injury, but the area at which such injury occurs is thought limited to a very small localized region relative to the dimensions of the sample over which wave transit times are measured. The dominant frequency for sound speed measurements reported by Rice³² is on the order of 300 Hz, which appears high enough to justify use of a closed-cell model. For lower-rate applications, wherein measured sound frequencies do not exceed 100 Hz, other experiments on dog, sheep, and pig lung suggest an open-cell model may be appropriate since wave speeds appear about an order of magnitude smaller.^{33,63,65} Also noted by Rice³² is that for lower frequencies amenable to the open-cell range

(*e.g.*, $\lesssim 100$ Hz), compression of air inside the lung tends more toward isothermal than isentropic, which would lead to a further reduction in bulk modulus and sound speed.

Arguments of Rice³² suggest that, for effective homogeneous elastic properties to be used in analysis of harmonic waves, a closed-cell continuum model is most valid for signal wavelengths exceeding around 10 alveolar diameters. With $c_L = 24$ m/s and $l = 100 \mu\text{m}$ for horse lung, the maximum frequency for such validity is 24 kHz.³² Repeating the calculation using the predicted value $c_{L1} = 27.1$ m/s for dog with $l = 60.1 \mu\text{m}$ from Table 2, a corresponding maximum frequency in this context is 45 kHz. At much higher frequencies, with wavelengths approaching the order of the alveolar size, waveforms become highly distorted since a snapshot in time of any single wave may not encompass materials of each phase of the microstructure, and since compressibilities of the dense tissue phase and air are drastically different. Analysis of Grimal⁹⁸ suggests that phase velocities of high-frequency waves are frequency dependent, and that cut-off frequencies on the order of several hundred kHz exist above which harmonic waves do not propagate.

6.2 Shock Compression

The final problem set considered in this report addresses propagation of a planar longitudinal compressive shock wave through the lung at various initial airway pressures. This analysis fulfills two main purposes. Firstly, it enables assessment of effects of initial pressure, surface tension, and dynamic viscosity on shock velocity and the shocked thermo-mechanical state of the lung: dynamic pressure–volume and shear response, temperature rise, and entropy produced. Secondly, it enables quantification of criteria for onset of injury and severity of damage with increasing magnitude of shock stress or volume/density change. This correspondence is achieved by quantitative comparison of model results with experimental data for thresholds on edema^{1,45} and numerical or simulation data^{10,99} for thresholds on contusion. Qualitative comparison with physical and numerical observations^{9,12,13,15,50,98} on high-frequency lung injuries provides further insight into physical meaning of damage variable D afforded by the theory. The treatment presented here is of much greater scope than the preliminary study by the authors⁶ that did not address damage, injury, or distinctions between normal and saline-infused lung for shock compression.

The shock is analyzed in the framework of the Rankine–Hugoniot jump conditions^{52,82,91,100} rather than the continuum balance laws for mass, momentum, and energy conservation given in Section 2.6. Material in the vicinity of the shock, both upstream and downstream, is treated as a homogeneous continuum with effective properties such as mass density, internal energy, and bulk stiffness. The shock front (*i.e.*, the region across which density, pressure, and particle velocity rapidly increase) is not resolved explicitly. Validity of application of the Rankine–Hugoniot jump conditions for mass, momentum, and energy requires that either the thickness of this front, called the “shock width”, be infinitesimal (*i.e.*, a limiting singular surface) or the entire waveform analyzed, even if the front is of finite width, moves at constant Lagrangian velocity.^{52,82} The latter interpretation is more physically valid in the present context, as the minimum width w of the shock front should be constrained by the microstructure, here the diameter l of an alveolus. In other words, $w \gtrsim l$ is presumed, since the entire pressure rise cannot be contained within a single alveolus as it would only sample material of the gas phase, rather than the effective continuum. The shock is initiated by a step loading (impact) in particle velocity or pressure on the lung surface. Presumably, after a transit period, a steady shock structure will be attained beyond some distance from the impact surface, where w is ultimately dictated by the microstructure as well as dissipation that tends to spread the front.

Even though the lung is a two-phase material with gas and relatively stiff dense tissue, a continuum mechanical treatment of shock propagation is deemed valid. Similar equations are widely used to analyze highly porous solids⁸² including metallic foams, particulates, and powders, wherein, like the lung, a large volume fraction of the medium consists of trapped air. The homogeneous continuum approach naturally is unable to address local fluctuations in response due to microstructure heterogeneities, and behaviors at material points of either phase within the wavefront itself are not explicitly resolved. Multiphase models^{37–39} or models in which the domain is partitioned into distinct entities of each phase,⁹⁸ either of which require advanced numerical implementation, are necessary to address such fine-scale microstructure effects. Computational cost of these advanced numerical methods exceed those permissible in contemporary finite-element studies of the torso under impact and blast loading,^{10,15} which is the intended domain of ultimate use for the presently developed continuum theory.

6.2.1 Governing Equations

The planar shock problem is idealized via standard protocols of shock physics research.^{52,58,82,91,100,101} The same rectilinear Cartesian coordinate frame for $\{X_K\}$ used in Section 6.1 is invoked here. An infinitely column of lung material consisting of dense tissue and air phases is subjected to a planar longitudinal compressive shock wave traveling at constant Lagrangian velocity v_s in the $X = X_1$ -direction in material coordinates. States of the material on either side of the wavefront, which may be of small but finite width w , are uniform. Let superscripts $(\cdot)^+$ and $(\cdot)^-$ denote quantities or conditions measured ahead of and behind the shock front, respectively, corresponding to respective un-shocked (upstream) and shocked (downstream) states.

First consider the upstream state. Material ahead of the shock is undeformed ($J^+ = 1$) and at rest. The upstream airway pressure is $p_a^+ = p_{a0}$, which varies among calculations later. The upstream stress in the lung tissue is hydrostatic. The transpulmonary pressure in the tissue upstream is the initial surface tension $p^+ = -p_{tp}^+ = -K_0$ for standard lung, and $p^+ = -p_{tp}^+ = 0$ for saline-treated lung. The external (e.g., pleural) pressure necessary to impose both conditions is then found via Eq. 93 as $p_{pl}^+ = p^+ + p_a^+$. The upstream entropy density is taken as the zero datum, $\eta^+ = 0$, and the upstream temperature is chosen as $T_0 = 310$ K. The following abbreviated notations are then used:

$$\eta^+ = 0, \quad \eta^- = \eta; \quad T^+ = T_0, \quad T^- = T. \quad (160)$$

The shocked material in the downstream state is deformed under uniaxial strain conditions corresponding to plane wave compression. Accordingly, transverse dimensions of the sample of lung material are sufficiently large such that release from lateral edges does not occur within the column analyzed for time scales of interest. The downstream pressure p^- is defined relative to the datum initial state in the absence of surface tension. In other words, $p^- = p^+$ in the limit of zero imposed compression in the shock wave. A total downstream pressure can be defined as $p_{tot} = p^- + p_{a0}$, which thus is defined relative to a datum state close to vacuum because initial surface tension is much smaller in magnitude than atmospheric pressure. Thus, p_{tot} includes the initial airway pressure, the initial surface tension, and the pressure rise due to the shock, while $p^- \leq p_{tot}$ does not include the initial

airway pressure. The balance equations provide the same solutions regardless of which constant datum is used for pressure (*e.g.*, 1 atm or vacuum for saline-treated lung) since only the jumps in stress and internal energy are used.

The downstream Cauchy stress state $\boldsymbol{\sigma}^-$ in the composite lung material is determined from simultaneous solution of the Rankine–Hugoniot jump conditions and closed-cell constitutive model of Section 5 for the homogenized effective medium. This stress tensor is diagonal due to symmetries of the material and loading mode, but it is not necessarily spherical. The downstream pressure is $p^- = -\frac{1}{3}\text{tr}\boldsymbol{\sigma}^-$. The shock stress is defined as the longitudinal component of Cauchy stress, positive in compression: $P = -\sigma_{11}^-$. Lateral stress components are $\sigma_{22}^- = \sigma_{33}^-$, and a measure of Cauchy shear (squeeze) stress is the difference $\tau = -\frac{1}{2}(\sigma_{11}^- - \sigma_{22}^-)$. When the shocked state is perfectly spherical, then $\tau = 0$ and $P = p^-$. Stresses in the dense tissue phase and the gas phase are not resolved individually by the model in the downstream state. Only one stress tensor $\boldsymbol{\sigma}^-$ in the composite material is obtained in the analysis, whereby the effective properties (bulk modulus, Grüneisen parameter, etc.) are implemented for the homogenized two-phase medium. As noted already, resolution of local differences in stress, strain, and other state variables within components of the microstructure is outside the present scope, requiring a much more sophisticated treatment. The stress tensor $\boldsymbol{\sigma}^-$ is measured with respect to the same datum state corresponding to p^- , following the convention used throughout this work and that of Fung^{1,19,20} that $p^- = 0$ and $\boldsymbol{\sigma}^- = \mathbf{0}$ in the undeformed state with null surface tension. A total downstream stress tensor that also includes initial airway pressure is defined consistently as $\boldsymbol{\sigma}_{tot} = \boldsymbol{\sigma}^- - p_{a0}\mathbf{1}$. Neither the downstream airway pressure nor the isolated pressure in the dense tissue phase are calculable in the shocked state.

Summarizing these textual definitions, downstream and upstream deformation gradients are, respectively,

$$\mathbf{F}^- = \mathbf{R}^- = \begin{bmatrix} a & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}, \quad J^- = a = \frac{\rho^+}{\rho^-}; \quad \mathbf{F}^+ = \mathbf{1}, \quad J^+ = 1. \quad (161)$$

Cauchy stresses in the shocked and initial states are the following:

$$\begin{aligned}\boldsymbol{\sigma}^- &= \begin{bmatrix} -P & 0 & 0 \\ 0 & 2\tau - P & 0 \\ 0 & 0 & 2\tau - P \end{bmatrix}, & p^- &= P - \frac{4}{3}\tau; \\ \boldsymbol{\sigma}^+ &= -p_0 \mathbf{1}, & p_0 = p^+ &= \begin{cases} -K_0 & \text{in air,} \\ 0 & \text{in saline.} \end{cases}\end{aligned}\quad (162)$$

Upstream pressure p^+ has been relabeled as p_0 . Composite mass densities are, from Eqs. 126 and 161,

$$\rho^- = \frac{1}{J^-} \rho^+ = \frac{1}{a} \rho^+, \quad \rho^+ = v_t \cdot \rho_t + (1 - v_t) \cdot \rho_{a0}, \quad (163)$$

where the value of v_t in Table 2 is maintained. Since $J^+ = 1$ and $\rho_t = \text{constant}$, ρ^+ is nearly constant in forthcoming calculations, with only a slight variation incurred from ρ_{a0} as initial airway pressure changes. Denoted by $\rho_0 = \rho^+$ is the constant reference density at $p_{a0} = 1 \text{ atm}$, with a numeric value given in Table 2.

As was the case in Section 6.1, the deformation process is deemed too rapid for viscoelastic stress relaxation to commence. As justification, consider a shock front of maximum width $w = 10l \approx 600 \mu\text{m}$ and minimum propagation velocity $v_s = c_L \approx 20 \text{ m/s}$, giving a maximum rise time of $t_R = w/v_s \approx 30 \mu\text{s}$. Considering the relaxation time scale $t_1 = 5 \times 10^5 \mu\text{s}$ from Table 1, $t_R/t_1 \ll 1$, viscous relaxation is negligible, and thus internal strain vanishes (*i.e.*, $\chi_1 = \mathbf{0}$ in the constitutive framework of Section 5).

According to Eq. 161, all strain attributes of Eq. 22 vanish identically in the upstream state: $\epsilon_i^+ = 0$ and $\boldsymbol{\epsilon}^+ = \mathbf{0}$. Furthermore, the initial condition $D^+ = D_0 = 0$ is imposed, consistent with the low initial transpulmonary pressure due only to surface tension at $J^+ = 1$, which does not impart sufficient driving force $Z^+ \geq Z_0$ according to Eqs. 79 and 85 to instigate any damage in the reference state. Since $\eta^+ = 0$ by construction, the internal energy density of Eq. 147 in the upstream state also vanishes: $U^+ = 0$. The succinct notation $\boldsymbol{\epsilon}^- = \boldsymbol{\epsilon}$, $U^- = U$, and $D^- = D$ is thus adopted for strain, internal energy density, and damage in the downstream state, consistently with Eq. 160.

Define the jump and average of a quantity across the shock front as follows:

$$[[(\cdot)]] = (\cdot)^- - (\cdot)^+, \quad \langle\langle(\cdot)\rangle\rangle = \frac{1}{2}[(\cdot)^- + (\cdot)^+]. \quad (164)$$

Denote by $v_p = v_1(X, t)$ the uniaxial particle velocity field. Since the upstream material is quiescent, $v_p^+ = 0$, and the abbreviated notation $v_p^- = v_p$ is invoked. Recall that v_s is defined as the steady Lagrangian shock velocity; this is equal to the Eulerian shock velocity since $v_p^+ = 0$ and $J^+ = 1$.

Rankine–Hugoniot jump conditions for mass, linear momentum, and energy conservation across the shock front are expressed in Lagrangian form as^{52,82}

$$[[v_p]] = -v_s \cdot [[J]] \quad \Rightarrow \quad v_p = v_s \cdot (1 - a), \quad (165)$$

$$[[\sigma_{11}]] = -\rho^+ v_s^2 [[J]] \quad \Rightarrow \quad v_s = \left[\frac{P - p_0}{\rho^+ \cdot (1 - a)} \right]^{1/2}, \quad (166)$$

$$[[U]] = \langle\langle\sigma_{11}\rangle\rangle [[J]] \quad \Rightarrow \quad U = \frac{1}{2}(P + p_0)(1 - a). \quad (167)$$

For compressive shocks, $0 < a < 1$ and $v_s > v_p > 0$. Under the trivial case of null deformation, $a = 1$, $P = p_0$, and $v_s = v_p = 0$. Assume that the deformation induced by the shock, a , is imposed as the loading parameter. Assume that the upstream pressure and density are known according to Eqs. 162 and 163. Then Eqs. 165–167 are three scalar equations with five unknowns in the set of variables (v_p, v_s, P, U, η) . The system becomes closed when augmented by constitutive equations for stress and internal energy that both depend on a and η . Analogous arguments apply if particle velocity v , shock velocity v_s , or shock stress P is imposed as the load parameter and $a = J^- = \rho^+/\rho^-$ is rendered an unknown. Shock compression is presumed adiabatic as in the literature,^{52,82,100,102} with time scales too brief for heat conduction, but it is strictly neither isothermal nor isentropic. The jump form of the entropy production inequality in the present case requires that $\eta \geq 0$ (*i.e.*, entropy must not decrease across the shock front).

From Eq. 161, strain attributes of Eq. 22 in the downstream state are

$$\epsilon_0 = \ln a, \quad \epsilon_1 = -\epsilon_3 = \frac{1}{3} \ln a, \quad \epsilon_2 = \epsilon_4 = \epsilon_5 = \epsilon_6 = 0. \quad (168)$$

State variables of strain in Eq. 121 are

$$\xi_1 = \epsilon_0 = \ln a, \quad \xi_2 = \frac{2}{9}(\epsilon_0)^2 = \frac{2}{9}(\ln a)^2, \quad \xi_3 = 0. \quad (169)$$

Although internal viscoelastic strain χ_1^- vanishes according to these arguments, at least in the material region of interest closely trailing the shock front, damage $D^- = D$ may be nonzero due to driving force Z induced by shock compression that may exceed Z_0 for shock stresses of sufficient magnitude.^{1,45} Stress components conjugate to rates of Eq. 168 are, from Eq. 148, dropping the $(\cdot)^-$ superscript for brevity on the left and with $\epsilon_0 = \epsilon_0(a)$ on the right,

$$\begin{aligned} \hat{\tau}_0(a, \eta, D) &= [1 - (1 - \bar{\gamma})D] \{ \bar{B}_0^\eta [\epsilon_0 \exp(c_1 \epsilon_0^2)] - g_0 T_0 \eta + B_0^T \beta_1 \epsilon_0 + K_0 \} \\ &\quad - \frac{2}{3}(1 - D) B_0^T \mu'_0 (\epsilon_0)^2, \\ \hat{\tau}_1(a, \eta, D) &= 2(1 - D) \mu_0 [\epsilon_0 \exp(\frac{2}{9} c_2 \epsilon_0^2) (1 - \{B_0^T / \mu_0\} \mu'_0 \epsilon_0) + \beta_2 \epsilon_0], \\ \hat{\tau}_3(a, \eta, D) &= -\hat{\tau}_1(a, \eta, D); \\ \hat{\tau}_2 &= \hat{\tau}_4 = \hat{\tau}_5 = \hat{\tau}_6 = 0. \end{aligned} \quad (170)$$

Nonzero Cauchy stress components $\sigma_{ij} = \frac{1}{J} s_{ij}$ are, from Eq. 44,

$$\sigma_{11} = \frac{1}{a} \left(\hat{\tau}_0 + \frac{2}{3} \hat{\tau}_1 \right), \quad \sigma_{22} = \sigma_{33} = \frac{1}{a} \left(\hat{\tau}_0 - \frac{1}{3} \hat{\tau}_1 \right). \quad (171)$$

Shock stress, pressure, and shear stress of Eq. 162 are then

$$P = -\frac{1}{a} \left(\hat{\tau}_0 + \frac{2}{3} \hat{\tau}_1 \right), \quad p^- = -\frac{1}{a} \hat{\tau}_0, \quad \tau = -\frac{1}{2a} \hat{\tau}_1. \quad (172)$$

Normally, $P \geq p^- \geq 0$ and $\tau \geq 0$, so $\hat{\tau}_0 \leq 0$ and $\hat{\tau}_1 = -\hat{\tau}_3 \leq 0$.

Internal energy density in the shocked state is, from Eq. 147,

$$\begin{aligned}
U(a(\epsilon_0), \eta, D) &= [1 - (1 - \bar{\gamma})D] \left[\frac{1}{2} \frac{\bar{B}_0^\eta}{c_1} \{\exp[c_1(\epsilon_0)^2] - 1\} + K_0\epsilon_0 - g_0T_0\eta\epsilon_0 \right] \\
&\quad + (1 - D) \left[3 \frac{\mu_0}{c_2} \{\exp(\frac{2}{9}c_2\epsilon_0^2) - 1\} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0\epsilon_0 \right) + \frac{11}{18} \beta_1 B_0^T (\epsilon_0)^2 \right] \\
&\quad + T_0\eta \left(1 + \frac{1}{2} \frac{\eta}{c_0} \right).
\end{aligned} \tag{173}$$

Downstream temperature is, from Eq. 149,

$$T(a, \eta, D) = T_0 \left[1 - \{1 - (1 - \bar{\gamma})D\} g_0 \ln a + \frac{\eta}{c_0} \right]. \tag{174}$$

Damage obeys Eq. 145, where for non-trivial evolution in regime $\frac{Z_m}{Z_0} \in \left[1, 1 + \frac{\ln 2}{\alpha_D} \right]$,

$$D(\mathbf{X}, t) = \exp \left[\frac{\alpha_D \{Z_m(\mathbf{X}, t) - Z_0\}}{Z_0} \right] - 1, \quad Z_m(\mathbf{X}, t) = \max_{s \in (-\infty, t]} Z(\mathbf{X}, s). \tag{175}$$

Internal energy density of Eq. 173 is partitioned according to Eqs. 123 and 133 as

$$U_0 = U_0^{V\infty} + \Upsilon_0^V + U_0^{\mu\infty} + \Upsilon_0^\mu; \tag{176}$$

$$\begin{aligned}
U_0^{V\infty} &= \frac{1}{2} \frac{\bar{B}_0^\eta}{c_1} \{\exp[c_1(\epsilon_0)^2] - 1\} + K_0\epsilon_0 - g_0T_0\eta\epsilon_0, \\
U_0^{\mu\infty} &= 3 \frac{\mu_0}{c_2} \{\exp(\frac{2}{9}c_2\epsilon_0^2) - 1\} \left(1 - \frac{B_0^T}{\mu_0} \mu'_0\epsilon_0 \right), \quad (U_0^{G\infty} = 0);
\end{aligned} \tag{177}$$

$$\Upsilon_0^V = \frac{1}{2} B_0^T \beta_1 (\epsilon_0)^2, \quad \Upsilon_0^\mu = \frac{2}{3} \mu_0 \beta_2 (\epsilon_0)^2, \quad (\Upsilon_0^G = 0). \tag{178}$$

Substitution into Eqs. 142 and 143 gives the driving force for damage kinetics for non-negative energy density $\bar{U}_0 = (1 - \bar{\gamma})(U_0^{V\infty} + \Upsilon_0^V) + U_0^{\mu\infty} + \Upsilon_0^\mu \geq 0$ with

$B_0^\eta \simeq B_0^T$:

$$\begin{aligned}
Z &= \left[2 \left| \frac{U_0^{V\infty}}{B_0^\eta} + \frac{\Upsilon_0^V}{B_0^T} + \frac{U_0^{\mu\infty} + \Upsilon_0^\mu}{\mu_0} \right| \right]^{1/2} \cdot \left[1 + \omega_D \left| 2 \frac{l}{B_0^\eta} \nabla_0 U_0 \right|^{1/2} \right] \\
&= \left[\frac{1}{c_1} \{ \exp[c_1(\epsilon_0)^2] - 1 \} + 2 \frac{K_0}{B_0^\eta} \epsilon_0 - 2 \frac{g_0 T_0}{B_0^\eta} \eta \epsilon_0 + \beta_1(\epsilon_0)^2 \right. \\
&\quad \left. + \frac{6}{c_2} \{ \exp(\frac{2}{9} c_2 \epsilon_0^2) - 1 \} \left(1 - \frac{B_0^T}{\mu_0} \mu_0' \epsilon_0 \right) + \frac{4}{3} \beta_2(\epsilon_0)^2 \right]^{1/2} \\
&\quad \times \left[1 + \omega_D \left| 2 \frac{l}{B_0^\eta} \nabla_0 U_0 \right|^{1/2} \right].
\end{aligned} \tag{179}$$

The internal energy gradient across the shock front is estimated by assuming a linear profile over the shock width w ,¹⁰³ recalling that $U_0^+ = 0$ in the initial upstream state and $U_0 = U_0^-$:

$$\begin{aligned}
-\nabla_0 U_0 &\approx \frac{[[U_0]]}{w} = \frac{U_0}{w} \Rightarrow \\
\omega &= \omega_D \left| \frac{l}{(B_0^\eta)} \right|^{1/2} |2 \nabla_0 U_0|^{1/2} \approx \sqrt{2} \Omega_D \left| \frac{U_0}{B_0^T} \right|^{1/2},
\end{aligned} \tag{180}$$

where

$$\Omega_D = \omega_D \sqrt{\frac{l}{w}}. \tag{181}$$

All parameters entering the constitutive model and foregoing analysis have been determined already with the exception of dimensionless term Ω_D . The value of Ω_D listed in Table 2 is justified in Section 6.2.4, noting that the present estimate of $|\nabla_0 U_0|$ depends on w . However, the shock width w need not be known explicitly here since only the combined form of ω enters the damage criterion of Eq. 145 and w is assumed constant in the present application to shock tube loading.

6.2.2 Method of Solution

Hugoniot response curves are generated for lung in air and treated with saline, at initial airway pressures ranging from $p_{a0} = 0.5$ atm to $p_{a0} = 3$ atm. The dense tissue phase has initial volume fraction v_t given in Table 2, regarded as a fixed material property for dog lung. The overlying equations in Section 6.2.1 are solved using a numerical procedure whose algorithm is outlined here:

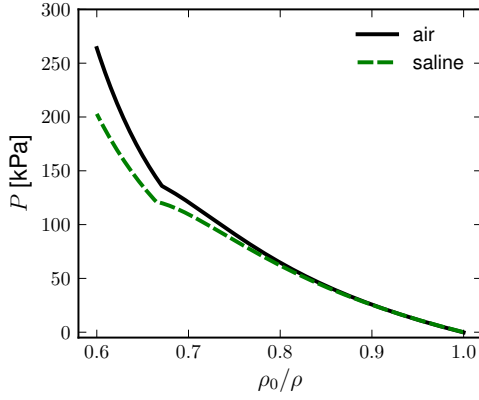
1. Initial airway pressure p_{a0} is assigned. Composite properties that depend on

p_{a0} such as mass density ρ^+ , closed-cell bulk modulus $\bar{B}_0^\eta$, specific heat c_0 , and Grüneisen parameter g_0 are computed using Eqs. 126–129.

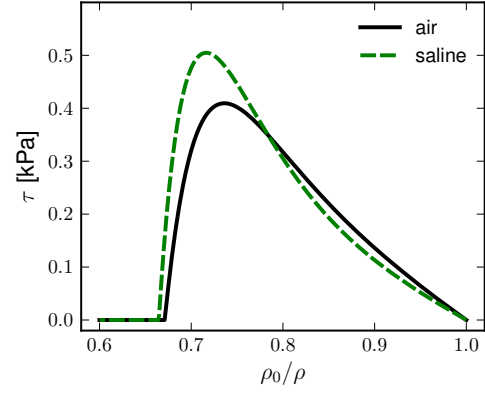
2. The upstream state is specified: $T_0 = 310 \text{ K}$, $U^+ = \eta^+ = 0$, $p^+ = p_0$, $\sigma^+ = -p_0 \mathbf{1}$, and $D^+ = 0$.
3. Volume change $J^- = a = \exp(\epsilon_0)$ is decremented by a small amount corresponding to compressive uniaxial strain in the plane shock wave, as indicated by Eq. 161.
4. Energy balance Eq. 167 and constitutive equations for the downstream (shocked) state in Eqs. 170–180 are solved iteratively for internal energy $U = U^-$, longitudinal stress $P = -\sigma_{11}^-$, shear stress τ , entropy $\eta = \eta^-$, temperature $T = T^-$ and damage variable $D = D^-$.
5. Momentum balance Eq. 166 is solved for shock velocity v_s , followed by solution of mass balance Eq. 165 for particle velocity v_p .
6. Return to step 3 until a minimum imposed volume ratio J^- is attained, corresponding to a maximum induced shock stress P .
7. Return to step 1 for the next initial value of internal airway pressure p_{a0} until all intended values have been probed.

6.2.3 Results: Shock Wave Mechanics

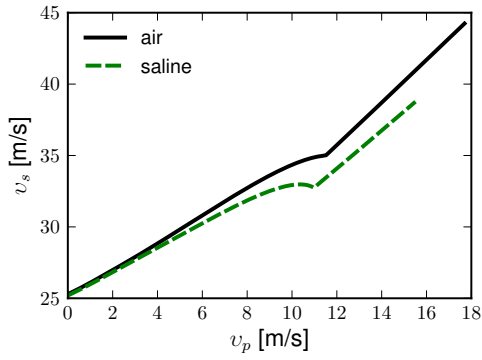
Results for compressive shocks on lung at standard internal airway pressure of $p_{a0} = 1 \text{ atm}$ are discussed first. These are presented in Fig. 12 for both normal lung tissue in air and saline-treated lung tissue. Hugoniot curves in Fig. 12 and subsequent figures should not be interpreted as paths taken by the material through the shock front in any one impact event. Rather, each curve represents the locus of end shocked states attained in a series of impact events. Each such event corresponds to a different compression ratio ρ_0/ρ , different shock stress P , and so forth.^{52,82,100} Recall that in saline-washed tissue, the bulk stiffness is reduced (see Table 1), surface tension $K_0 = 0$, and viscoelastic stiffening vanishes via $\beta_1 = \beta_2 = \beta_3 = 0$. Mass density in the shocked state is $\rho^- = \rho$ and in the initial state is $\rho^+ = \rho_0$. Calculations span $0.6 \leq \rho_0/\rho \leq 1$, where the maximum compressive strain is 40%. Curves in Fig. 12, with the exception of Fig. 12c, should be read from right to left with increasing compressive strain in successive shock states.



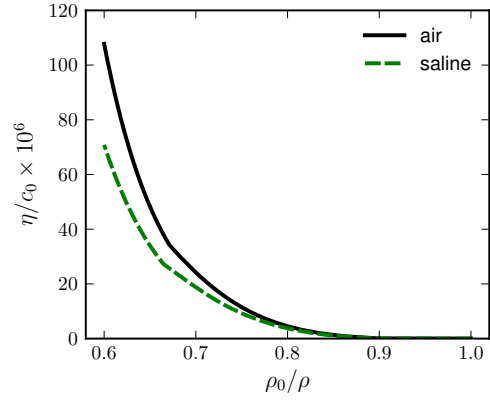
(a) axial stress



(b) shear stress



(c) shock velocity



(d) entropy

Fig. 12 Predicted Hugoniot response for air or saline-treated lung under planar shock compression at initial air pressure $p_{a0} = 1$ atm with mass density in the shocked state denoted by $\rho = \rho^-$ and that of the undeformed lung in the upstream state by $\rho^+ = \rho_0$: a) axial stress (*i.e.*, shock pressure) P , b) shear stress τ , c) shock velocity v_s versus particle velocity v_p , and d) entropy η normalized by composite specific heat at constant volume c_0

Shown in Fig. 12a is longitudinal shock stress $P = -\sigma_{11}^-$ of Eq. 172. Axial stress is very similar in both air and saline up to moderate compression ratios, with the former becoming larger than the latter for $\rho_0/\rho \lesssim 0.75$. At low shock stress magnitudes, the stiffening effect of surface tension on the bulk modulus is offset by the initial tensile transpulmonary pressure K_0 . An inflection occurs for $\rho_0/\rho \approx 0.75$ as damage affects the tangent bulk modulus. Strong stiffening resumes for $\rho_0/\rho \lesssim 0.68$ as the highly compressed material behaves like a densified compressible fluid. Note that even at 40% compression, the mass density of lung in air is $\rho = 0.561 \text{ g/cm}^3$, compared to $\rho_t = 1.003 \text{ g/cm}^3$ for the tissue phase. Thus, a significant amount of compressed air remains in the composite material even for the strongest shocks considered here. At much larger compressive pressures, effects of air would lessen relative to the stiffness of the dense phase, and the two curves should approach each other as surface tension effects become negligible. The present model is not intended to address such extreme, near fully collapsed, regimes.

Reported in Fig. 12b is Cauchy shear stress τ of Eq. 172, proportional to squeeze stress component $\hat{\tau}_1$. Initially, for low compressive strain, the shear stress in air exceeds that of saline because of viscoelastic stiffening of the tangent shear modulus. At larger compressions $\rho_0/\rho \lesssim 0.8$, the shear stress for saline-treated lung becomes larger than standard lung tissue in air. The distinction is later attributed to damage-induced softening of the tangent squeeze modulus, which becomes more severe for lung in air than saline-washed tissue. Comparing results in Figs. 12a and b, $P \gg \tau$ for any non-negligible compressive strain, since the tangent bulk modulus for the closed-cell model is two orders of magnitude larger than the tangent shear modulus. For $\rho_0/\rho \lesssim 0.68$, $\tau \rightarrow 0$ as $D \rightarrow 1$.

Presented in Fig. 12c is a standard plot of shock physics research, the steady Lagrangian shock velocity v_s versus downstream particle velocity v_p , related via Eq. 165. Shock velocity is higher in air than saline-treated lung since its stiffness is greater. The inflection in the saline curve corresponds to a change in curvature of the $P - \rho$ Hugoniot in Fig. 12a. This implies a small range of instability ($0.663 \lesssim \rho_0/\rho \lesssim 0.688$) wherein spreading of the shock structure, unaccounted for in the present calculations, would be expected for the saline-treated lung, though the predicted stress-volume and damage responses should remain accurate regardless due to the low entropy produced. Computed results yield $v_s \rightarrow c_L$ in the limit of small deformations, as $v_p \rightarrow 0$, providing cross-validation of model results for longitudinal

sound speed and shock velocity. In other words, the shock velocity reduces to the linear isentropic longitudinal wave speed of Eq. 157 for very small imposed P , here on the order of 25 m/s. The value is slightly less than $c_{L1} = 27.1$ m/s reported in the context of Fig. 10 since initial transpulmonary pressures and initial mass densities differ for cases studied in Figs. 10 and 12c.

Shown in Fig. 12d is downstream entropy density per unit reference volume η normalized by specific heat c_0 of Eq. 127. Entropy is always positive, as required by thermodynamics, and it increases with increasing shock stress P and compressive strain. Entropy production is lower in saline-treated lung than standard lung in air, with larger values of the latter due to surface tension, viscoelasticity, and damage effects. Entropy produced is too small to impart a significant temperature change $\Delta T > 1$ K according to the present closed-cell constitutive theory, however. Composite specific heat $c_0 = 741$ kPa/K in Table 2 is close to that of water at $c_0 = 10^3$ kPa/K, so significantly more dissipation than that generated here is required to raise T substantially, which in this case would thus appear to be closely tied to the temperature in the dense tissue phase. Temperature of enclosed air might be expected to increase more substantially due to its much lower specific heat per unit volume, on the order of 1 kPa/K, but the present composite theory does not provide explicit resolution of temperatures in each phase.

Discussed next are model predictions for standard (*i.e.*, with surface tension and viscoelasticity, not saline-washed) lung at initial airway pressures ranging from 0.5 to 3 atm. In each case, the initial transpulmonary pressure due to surface tension is fixed at $p_{tp0} = -p_0 = K_0 = 0.2$ kPa. Mass density in the shocked state is $\rho^- = \rho$. In the initial state at 1 atm airway pressure, density is written as ρ_0 as used in Fig. 13. Since density of the gas phase depends on initial pressure, the upstream composite density of Eq. 126 varies slightly with p_{a0} . When $p_{tp0} < 1$ atm, $\rho^+ < \rho_0$; conversely, when $p_{tp0} > 1$ atm, $\rho^+ > \rho_0$, though differences among all values of ρ^+ do not exceed 1% for $p_{a0} \in [0, 3]$ atm. Shock-compressed density ratios probed in Fig. 13 are $0.8 \leq \rho_0/\rho \leq 1$, corresponding to a maximum compressive strain of around 20%.

Reported in Fig. 13a are shock stress, normalized by $B_0^T = 2.5$ kPa, versus density ratio at different initial airway pressures. Shock stress P increases substantially with increasing airway pressure since p_{a0} dominates the closed-cell bulk modulus

of Eq. 128. Given in Fig. 13b are shear stress, normalized by $G_0 = \mu_0 = 1$ kPa, versus density ratio for the same internal pressure range. Origins of each curve at the bottom right are offset slightly since ρ increases with increasing initial air pressure. As is discussed in detail in Section 6.2.4, damage increases more rapidly with decreasing ρ_0/ρ as initial airway pressure increases since the corresponding internal energy jump across the shock front increases with airway pressure. The increased damage in turn leads to a reduction in tangent shear modulus that causes lower shear strength for shocks at higher initial airway pressure.

Shown in Fig. 13c are shock velocity versus particle velocity profiles. Shock velocity increases with particle velocity in all cases shown. Shock velocity increases substantially with increasing p_{a0} , consistent with increasing longitudinal elastic wave speeds reported in Fig. 10b. The reason for this is the increasing bulk stiffness with initial airway pressure. Effects of shear stiffness, orders of magnitude smaller than bulk stiffness, on shock velocity are negligible.

Finally, Fig. 13d presents entropy density, again normalized by specific heat, for $p_{a0} \in [0.5, 3]$ kPa. Dissipation is non-negative and increases with increasing shock strength and with increasing p_{a0} . However, even for the most extreme case considered of $p_{a0} = 3$ atm, η is too small to impart significant increase in temperature of the lung tissue, which has a relatively large specific heat, as discussed already in the context of Fig. 12d.

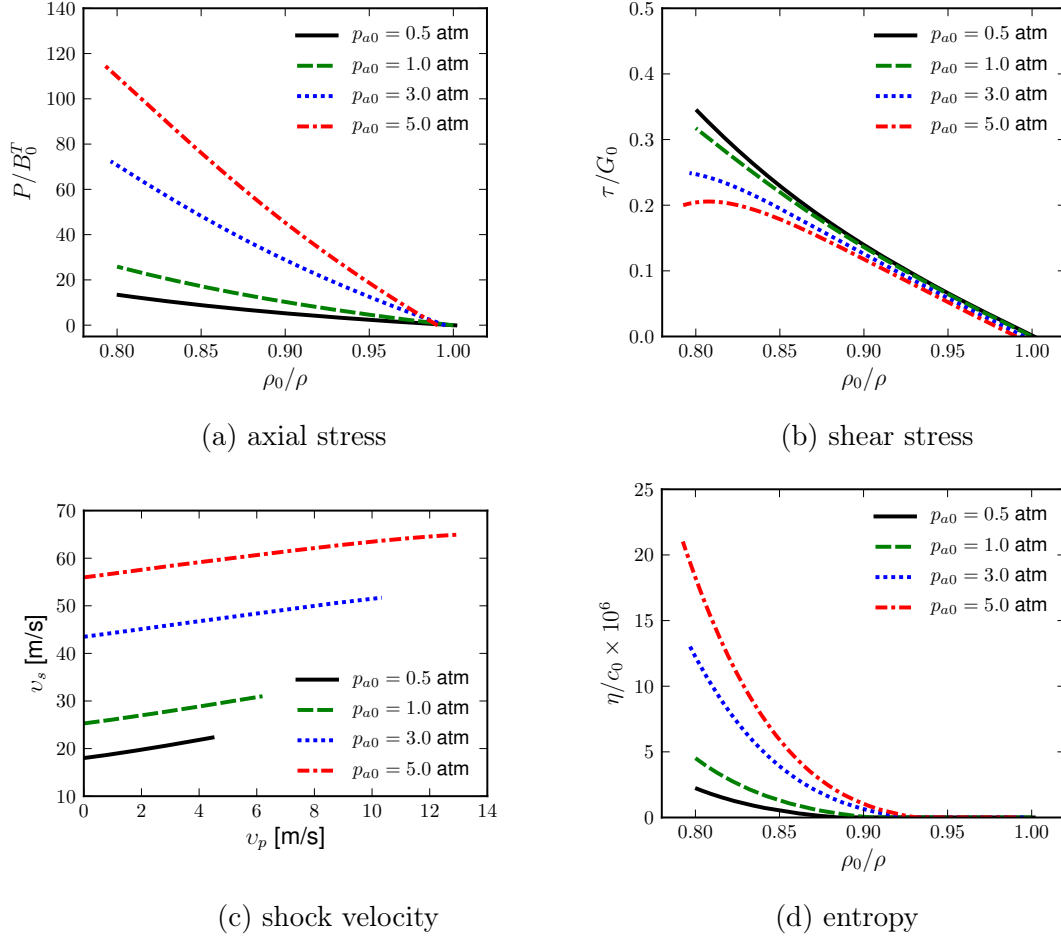


Fig. 13 Predicted Hugoniot response of lung in air at four values of initial air pressure p_{a0} , with mass density in the shocked state denoted by $\rho = \rho^-$ and that of the undeformed lung at $p_{a0} = 1$ atm by ρ_0 : a) axial stress P normalized by isothermal bulk modulus B_0^T , b) shear stress τ normalized by static shear modulus $G_0 = \mu_0$, c) shock velocity v_s versus particle velocity v_p , and d) entropy η normalized by composite specific heat at constant volume c_0

6.2.4 Results: Injury and Damage Criteria

Internal state variable D , indicative of severity of injury as well as mechanical stiffness degradation due to local tearing or fracturing of microstructure elements, is now analyzed in depth. Numerical results predicted by Eq. 145 are reported first, followed by a more thorough discussion relating D to physics of trauma and local material failure.

Results for compressive shocks on lung at $p_{a0} = 1$ atm are discussed first. These predictions are shown in Fig. 14 for both normal lung tissue in air and saline-treated lung tissue. Differences between the tissue types arise in the model from surface tension and viscous stiffening that are omitted for saline-washed lung. The stress-free mass density of saline-treated lung is lower than that of lung with surface tension since the latter causes contraction in the absence of transpulmonary pressure. Recall from Eqs. 179 and 180 that $\Omega_D = \omega_D \sqrt{l/w}$ scales the contribution of internal energy gradient $|\nabla U_0| \approx [[U_0]]/w$ to the driving force for damage initiation and growth, with l and w the alveolar size and shock width, respectively.

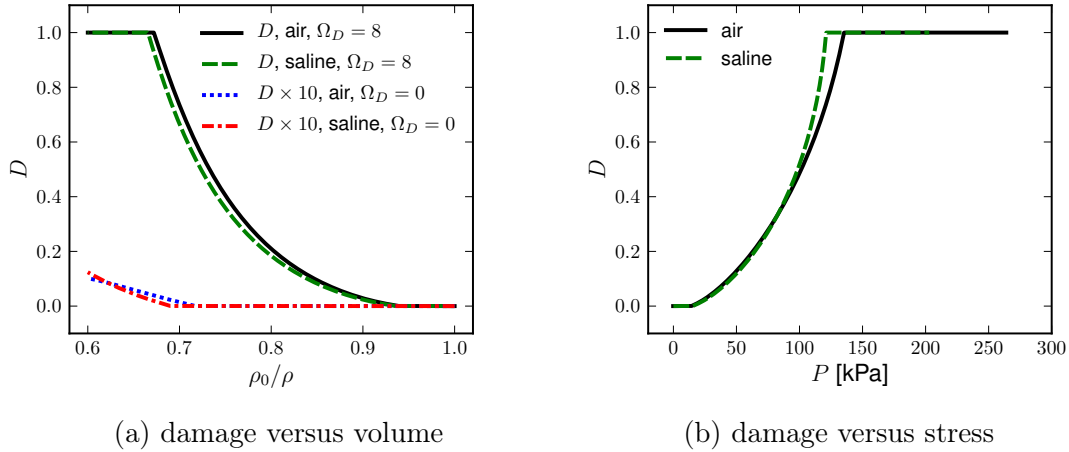


Fig. 14 Predicted damage response for air or saline-treated lung under planar shock compression at initial air pressure $p_{a0} = 1$ atm with mass density in the shocked state denoted by $\rho = \rho^-$ and in the upstream state by $\rho^+ = \rho_0$. $\Omega_D = 0$ when energy/pressure gradient effects are unrealistically omitted from damage kinetics. Damage shown a) versus volume ratio $\rho_0/\rho = J^-$ and b) versus shock stress P .

Shown in Fig. 14a are damage variable D versus density ratio for the usual, realistic case where $\Omega_D = 8$ from Table 2 is invoked as well as for the case when the gradient amplification is omitted via $\Omega_D = 0$. The value of D is magnified by a factor of 10

in the latter case to enable visualization. Mass density in the shocked state is $\rho^- = \rho$ and in the initial state is $\rho^+ = \rho_0$. The value of $\Omega_D = 8$ is chosen such that, in saline-treated lung, the threshold shock stress P for the onset of injury is $P = 15$ kPa. This corresponds to the threshold pressure for injury imparted by a shock tube to exposed, saline-treated rabbit lung in experiments described by Yen and Fung.^{1,45} In these experiments, $P \lesssim 14$ kPa produced no evidence of injury, while $P \gtrsim 16.5$ kPa produced edema as evidenced by increased lung weight with time after the impact event. Initial transpulmonary pressure (tension) was 10 cm H₂O ($p_{tp0} = -p_0 \approx 1$ kPa) in these experiments, and the shape of the shock front moving inside the material was unknown. Results in Fig. 14a show that, with increasing compressive strain, damage increases slightly more rapidly in air than in saline-treated lung. Also, when the deleterious effect of energy gradients (*i.e.*, steepness of the pressure rise in the shock front) is omitted via $\Omega_D = 0$, damage criterion Eq. 145 requires compressive strains exceeding 30% and shock stresses exceeding 110 kPa to impart the onset of injury via $Z > Z_0 \Rightarrow D > 0$.

Presented in Fig. 14b is D versus shock stress P of Eq. 172, where only results with $\Omega_D = 8$ are reported here and subsequently in Fig. 15. Initially with increasing P , damage accumulates more quickly in air than saline. The threshold pressure P for $D > 0$ is 15.3 kPa in saline and 14.4 kPa in air. After a cross-over at $P \approx 100$ kPa, damage accumulates more rapidly in saline-treated lung. Note, however, that stiffness is lower in saline-treated lung so more compressive strain is required to achieve the same shock pressure.

Threshold impact pressures for mild injury are low, on the order of 15 kPa or 2 psi, in accordance with experimental observations on edema in bare exposed lung tissue,^{1,45} which is very delicate. Local lung over-pressures required for hemorrhage and contusion are thought to be in the 70 to 100 kPa range.^{10,99} According to the results in Fig. 14b, this level of injury correlates to damage variable values in the range of $0.25 \lesssim D \lesssim 0.5$, which is physically realistic considering the description afforded by the model as discussed later in the context of Fig. 16. For lung tissue in air, according to the present results, local damage reaches its maximum possible value of $D = 1$ for shock pressures P exceeding 135 kPa. Lethal global over-pressures can be on the order several hundred kPa^{11,14,31} depending on pulse shape and duration; however, in-vivo the lung is encased by the ribcage, skin, muscle, and other soft tissue that alter the localized pressure waveforms ultimately experienced

by the lung tissue. Transmission and reflection within the body during the event also affect maximum local pressures, strains, and strain rates achieved. Refinements to the present theory and/or its kinetic parameters may emerge later in applications to such more complicated problems requiring finite-element analysis.³⁰

Damage and local injury predictions for lung in air (no saline treatment) at $p_{a0} = 0.5, 1, 2,$ and 3 atm are given in Fig. 15. The density range probed is $0.8 \leq \rho_0/\rho \leq 1$ (*i.e.*, a maximum compressive strain of 20%). Shown in Figs. 15a and 15b are damage state variable D versus density ratio ρ_0/ρ and versus shock stress P , respectively, with ρ_0 a constant value of composite mass density from Eq. 126 at a gas pressure of 1 atm. Damage increases more rapidly with increasing compressive strain, *i.e.*, with decreasing ρ_0/ρ , as p_{a0} increases since more energy is imparted by a shock wave of corresponding strain as airway pressure increases.

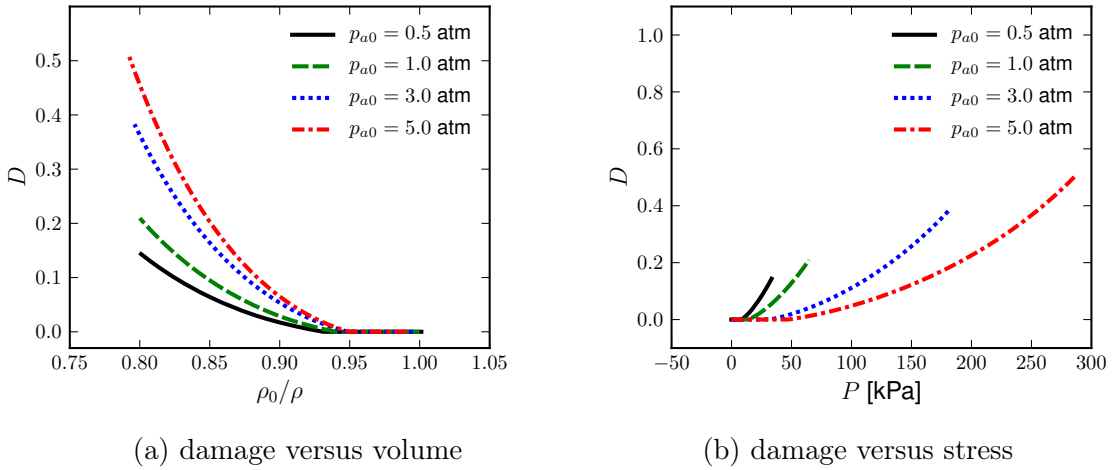


Fig. 15 Predicted damage response for lung in air at four values of initial air pressure p_{a0} , with mass density in the shocked state denoted by $\rho = \rho^-$ and density of undeformed lung at $p_{a0} = 1$ atm by ρ_0 . Damage shown a) versus density ratio ρ_0/ρ and b) versus shock stress P .

The threshold for onset of injury in terms of compressive strain attained in the shock front decreases with increasing initial air pressure. However, as seen in Fig. 15b, the shock stress P required to impart a given level of damage D increases with increasing p_{a0} since bulk stiffness increases significantly with increasing airway pressure. The threshold shock stress $P = P_0$ for initiation of injury (edema) increases accordingly with p_{a0} . Summarizing results in Fig. 15, the damage resistance of the lung increases with increasing initial air pressure in terms of imposed stress since its

stiffness increases, but injury resistance decreases in terms of imposed deformation since the driving force (energy or pressure) increases with air pressure.

A physical description of the micromechanical and biological changes associated with numerical ranges of internal state variable $D \in [0, 1]$ is afforded by the schematic of Fig. 16. The horizontal axis corresponds to driving force Z_0 , which essentially depends on normalized internal energy U_0 and its gradient $\nabla_0 U_0$ through Eqs. 142, 143, and 145. The energy gradient, in turn, is related to the jump in internal energy across the shock front as indicated in Eq. 180. The power of $\frac{1}{2}$ in the driving force effectively leads to a dependence of damage on axial strain or shock pressure, closely coupled in Figs. 12a and 13a, for example. The amplification of injury due to the rate of pressure rise, or the magnitude of pressure jump, is discussed by Cooper,¹² wherein the correlation of thoracic wall acceleration to injury severity is quantified experimentally. As explained in the lung trauma literature,^{9,12,98} as a longitudinal wave pulse propagates through the lung, pressure differentials emerge in the microstructure across capillary–alveolar interfaces, where larger local differentials are linked to shorter rise times of the pulse. Failure of the interfaces and hemorrhages will occur above a threshold that depends, in macroscopic experiments and models, on both the total normalized work or energy⁹⁹ and its rate of deposition.

A local injury criterion, similar to the damage model developed here, considers the maximum product of strain and strain rate experienced by an element of lung tissue over the history of a blast event,¹⁵ which is found to correlate with contusion in sheep lung subjected to blast loading. Overextension, bulging, and ultimately tearing and rupture of alveolar membranes have likewise been observed in histology studies on blast lung in human cadavers,⁵⁰ wherein the cause of death was explosion-related.

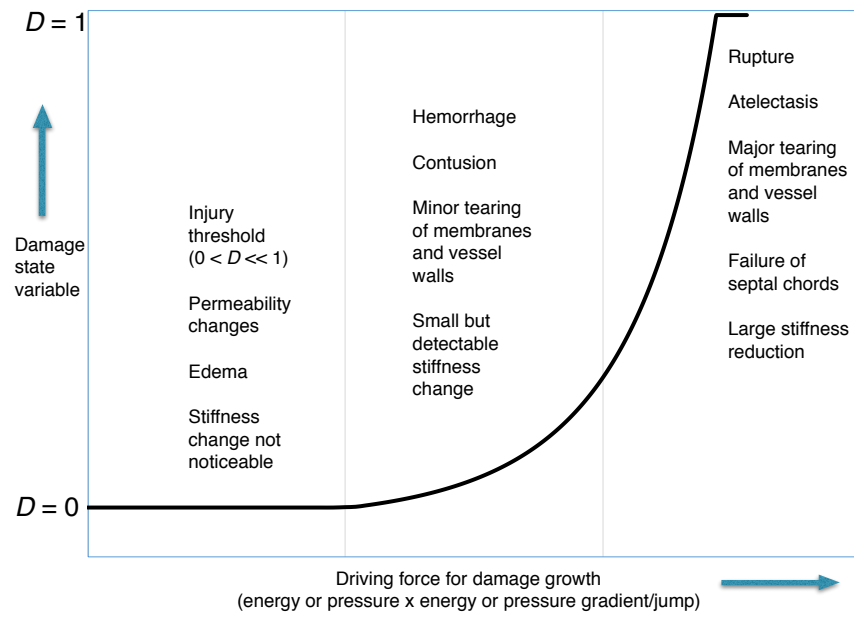


Fig. 16 Physical processes related to injury and microstructure degradation occurring in lung tissue in conjunction with evolution of damage state variable D . Exponential growth versus driving force is consistent with kinetic laws in Eqs. 85 and 145.

Summarizing static and dynamic experiments of Fung and colleagues,^{1,44,45} permeability changes to tissue membranes are induced at relatively low thresholds of imparted pressure, strain, or energy. These result in edema (*i.e.*, pooling of liquid) after sufficient time for fluid transport to occur. Threshold stresses and strains for the onset of injury are exceeded, by substantial margins in some cases, in static and dynamic experiments,^{1,19,21,59,60,67} but these experiments show no significant reduction of elastic stiffness upon unloading.

The present model reflects this behavior, as indicated collectively in results of Sections 4 and 6. Accordingly, in the exponential kinetic laws of Eqs. 85 and 145 and their resultant rate forms in Eqs. 86 and 146, damage increases slowly at first with increasing driving force Z just exceeding the threshold Z_0 , followed by more severe growth at much higher driving forces. Significant loss of mechanical stiffness is not realized until injury is severe (*e.g.*, tearing of membranes in shear, rupture of septal chords in tensile loading, or atelectasis (alveolar collapse) under compressive loading). Bulk stiffness is compromised by damage even under dynamic compressive loading since local holes or tears in membranes facilitate gas exchange over gas compression in the closed-cell microstructure.

Assume now as in Eq. 180 that, to first order, internal energy and other state variables change relative to their initial or upstream values linearly within a steady shock wave of width w , Lagrangian velocity v_s , and rise time t_R . The latter three are simply related, leading to several expressions for the material energy gradient $\nabla_0 U_0$:

$$w = v_s \cdot t_R \quad \Rightarrow \quad |\nabla_0 U_0| = \left| \frac{\partial U_0}{\partial X} \right| = \left| \frac{\partial U_0}{\partial t} \left(\frac{DX}{Dt} \right)^{-1} \right| = \frac{|\dot{U}_0|}{v_s} = |\dot{U}_0| \frac{t_R}{w} \approx \frac{[[U_0]]}{w} = \frac{[[U_0]]}{v_s t_R}. \quad (182)$$

The next-to-rightmost approximation in terms of internal energy jump is used in Eq. 180 and the present calculations. However, in numerical simulations involving finite elements,^{10,15,30} calculation of energy gradients may be cumbersome, so the approximation of the gradient or jump in terms of the product of material time derivative and wave speed may be a pragmatic choice as a driving force for injury, as also suggested by Grimal.⁹⁸ In that case, to avoid damage magnification during rapid unloading, $|\dot{U}_0| \rightarrow \langle \dot{U}_0 \rangle$ could be used such that only positive energy rates are applied to ω , though further study is needed to identify whether or not this constraint

is warranted. The approximation $v_s \approx c_L$ can further be invoked in the weak shock regime. According to the model of Eq. 145 and experimental evidence quoted in the literature,^{9,12,98} likelihood and severity of traumatic injury increases with increasing $|\nabla_0 U_0|$. Thus, according to the rightmost expression in Eq. 182, for a fixed amount of energy density U_0 supplied by the wave pulse at a fixed wave speed $v_s \geq c_L$, injury severity increases with decreasing rise time t_R .

Calculations conducted here invoke a value of $\Omega_D = \omega_D \sqrt{l/w}$ of 8 as calibrated to match experimental data of Yen.⁴⁵ The alveolar size is known a priori ($l = 60.1 \mu\text{m}$ in dog lung from Table 2 and Fung¹), but neither the material *constant* ω_D nor the *variable* shock width w is known or obtainable from presently available data. Assume for illustration that $w = w_1$ in the experiments of Yen,⁴⁵ leading to $\Omega_{D1} = \omega_D \sqrt{l/w_1}$. Then when the lung is subjected to shock pulses of different width or rise time propagating at the same speed $v_s \gtrsim c_L$, the value of Ω_D entering the driving force through Eqs. 145 and 180 would vary as

$$\Omega_D = \Omega_{D1} \sqrt{\frac{w_1}{w}} = 8 \sqrt{\frac{t_{R1}}{t_R}}. \quad (183)$$

Thus, as intended, Ω_D decreases as rise time of the shock front t_R increases relative to t_{R1} . This in turn results in less damage accrued through Eq. 145 for the same imparted internal energy U_0 . Shown in Fig. 17 are predicted effects of rise time of the shock pulse on damage progression and the onset of injury corresponding to edema. Damage accrues more slowly in Fig. 17a with increasing shock stress as the rise time increases, wherein trauma to standard lung in air is depicted. The threshold shock stress for initiation of damage also increases with rise time as shown in Fig. 17b, from around 15 kPa to 25 or 27 kPa for air or saline-treated tissue, respectively, as the rise time t_R increases by a factor of 10.

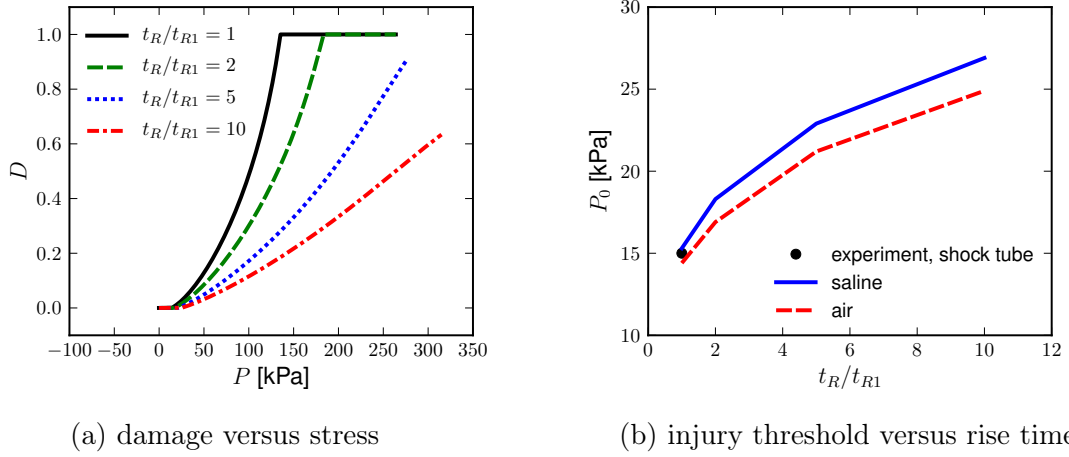


Fig. 17 Predicted damage response and injury threshold for exposed lung at different pulse rise times t_R , where t_{R1} is the minimum rise time for shock compression corresponding to experimental data⁴⁵ : a) damage variable D versus shock stress P for lung in air and b) threshold compressive stress for injury P_0 versus normalized pulse rise time t_R/t_{R1}

7. Conclusions

A geometrically and materially nonlinear model of lung mechanics has been presented. The model accounts for thermoelasticity, viscoelasticity, and damage phenomena, where the latter encompasses both injury and mechanical stiffness degradation. Kinematics and thermodynamics are based on a QR decomposition of the deformation gradient, strain attributes, and conjugate scalar stress measures. Viscoelasticity and damage are treated by internal state variables in a thermodynamically consistent manner, leading to non-negative dissipation.

Two variations of the model have been developed: a free energy-based theory pragmatic for static and isothermal loading, and an internal energy-based theory more amenable to dynamic problems involving wave propagation. The latter theory accounts for effects of enclosed air on the overall thermodynamic properties, most importantly the dynamic bulk modulus that becomes two orders of magnitude larger than the static tensile modulus when air compressibility is included. The present work is thought to represent the most complete continuum theory for lung mechanics in terms of loading regimes and physical mechanisms addressed.

The model has been demonstrated to provide reasonably close agreement with experimental data using relatively few calibrated parameters. These parameters have

a physical origin and have been determined readily and uniquely, without recourse to numerical optimization routines. Experimental protocols addressed here for the free energy-based theory include spherical static expansion, triaxial extension, dynamic pressure relaxation, and dynamic torsion. Satisfactory representation of data collected on mammals of small to moderate size, primarily dog, has been achieved. Accuracy of the model would benefit, however, from consistent data from single source, including a fixed species and means of specimen preparation.

Quantitative agreement with experimental data on low-amplitude, high-frequency wave speeds predicted by the internal energy-based theory has also been obtained. Analysis of planar shock waves has produced agreement with these validated wave speeds in the low-amplitude limit, but detailed experimental data are presently lacking for validation of the predicted shock compression response at larger impact pressures.

Damage and injury have been addressed to the extent possible given existing limits on quantitative experimental data relating mechanics (*e.g.*, stress–strain histories) to biology (*e.g.*, traumatic functional impairment) at the local tissue scale. The model does incorporate realistic thresholds for the onset of injury, corresponding to local membrane permeability changes and edema, based on experiments. Threshold stresses, strains, or deposited energies for such injury are far below those that impart noticeable stiffness deterioration (*e.g.*, substantial tears or rupturing of alveolar membranes and septal chords composing the microstructure). The importance of inclusion of the gradient of applied energy/pressure or its rate of application on injury criteria has been verified, with shorter pulse rise times associated with more imminent injury and more severe damage progression for the same pressure or energy density imparted to the shocked state. Moderate values of the damage variable correlate with local pressures for hemorrhage and lung contusion reported elsewhere.

Future work should address the following topics. Implementation of the theory in a dynamic finite-element code is underway. Dynamic impact experiments on isolated lung, in conjunction with numerical simulations, will be used to validate the theoretical model at high compressive loading rates. Effects of internal airway pressures and externally imposed stresses and their rates will be explored. A more quantitative linkage of damage progression to observed injury mechanisms above known threshold levels will be sought via comparison with results of ongoing live animal

testing. Finally, predictive computer simulations of trauma in the thorax, both animal and human, when subjected to blunt impact and blast events are foreseen.

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