

mech-life-2022

Mechanics and thermodynamics of living systems

November 8-10, 2022 — UPEC, Créteil, France

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Organizer : **V. Sansalone**
Université Paris-Est Créteil Val de Marne
MSME UMR 8208 CNRS
E-mail : vittorio.sansalone@u-pec.fr



Foreword

Living systems can adapt to their prevailing mechanical and biochemical environment to meet specific functional requirements. Functional adaptation emerges at different scales—from the subcellular to the whole organism. In particular, at the tissue scale, it may take the form of growth, remodelling, and morphogenesis.

From a mechanical perspective, a thorough understanding of these biological processes requires, in turn, understanding how mechanics and biology interact at different scales. This remains a serious challenge for both experiments and modelling. On the one hand, experiments can hardly probe the actual conditions experienced *in vivo* by living systems. On the other hand, mechanical modelling requires to extend classical approaches to account for the active behaviour of living systems.

There exists a wide spectrum of modelling approaches, ranging from phenomenological to mechanobiological, from mechanistic to statistical approaches. Some models focus on a single scale, whereas others look at several scales aiming to bridge them one another. Some models are concerned with materiomics whereas others with mechanomics. (Mechanics also contributes to the omics era!)

Such an incredible variety of modelling efforts is closely related to the even larger variety, complexity, and specificity of the behaviour shown by living materials. Nevertheless, mechanical modelling alone cannot go very far on this way, and the most significant advances have been made through a synergistic effort with other disciplines—life sciences, biology, physics, mathematics, engineering, just to cite a few.

The main goal of the **mech-life-2022** workshop was to promote this synergy. The format of the workshop was designed so as to leave large room for discussions. Every day focused on a different topic—From cell to tissue (day 1), Hard tissue (day 2), and Soft tissue (day 3)—and included extended seminars given by experts in the fields (morning sessions) and roundtables (afternoon sessions). Every day has been a unique opportunity to foster perspective-taking in mechanical modelling of living systems.

I heartily thank all the speakers for their amazing talks and commitment that made this meeting possible. More than 50 people had the opportunity to listen to them.

I greatly appreciated the extensive, frank, open, and lively discussions that we had during the roundtables, and heartily thank all the people having been there.

I thank all the participants for their interest in this workshop. I hope they appreciated the meeting and that it will be useful for them.

I acknowledge the support of the CNRS “Coss&Vita” IRP and of the MSME laboratory, as well as the AFRAN that sponsored this meeting.

I do think that this meeting has been a great opportunity to remove barriers between mechanics and other disciplines, and very look forward for the next opportunity to follow up with it.

Vittorio Sansalone, PhD

Laboratoire Modélisation et Simulation Multi Echelle

MSME UMR 8208 CNRS

Université Paris-Est Créteil Val de Marne

Créteil, France

E-mail: vittorio.sansalone@u-pec.fr

Program

November 8, 2022

From cell to tissue

9:00-9:50	M. Knothe Tate	The ebb and flow of life - through the lens of mechanics and thermodynamics
10:00-10:50	G. Cappello	Poro-Active biological tissues
11:00-11:50	M. Martin	Modelling peritoneal adhesions: a biomechanical approach
12:00-12:50	P. Pivonka	Mechanistic pharmacokinetic-pharmacodynamic modeling of osteoporosis treatments
<i>Afternoon</i>	<i>Roundtable</i>	

November 9, 2022

Hard tissue

9:00-9:50	D. George	Multiscale bone remodeling: dream or reality?
10:00-10:50	S. Ramtani	Damage Bone Adaptation Following Continuum Thermodynamics
11:00-11:50	N. Mühl-Castoldi	On the modelling of spinal growth
12:00-12:50	F. dell'Isola	An attenuated hyperbolic model for stimulus propagation to describe frequency driven mechano-transduction in bone remodelling
<i>Afternoon</i>	<i>Roundtable</i>	

November 10, 2022

Soft tissue

9:00-9:50	P. Nardinocchi	Passive and active fiber reorientation in anisotropic materials
10:00-10:50	A. Musesti	On the modeling of transversely isotropic active materials
11:00-11:50	D. Ambrosi	A two-rod model for the mitral valve
12:00-12:50	L. Teresi	Self contracting gel
<i>Afternoon</i>	<i>Roundtable</i>	

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Topic: Soft tissue

A two-rod model for the mitral valve

D. Ambrosi

Dipartimento di Scienze Matematiche, Politecnico di Torino, Italy

E-mail: davide.ambrosi@polito.it

A mitral valve can, in a first approximation, be represented as a mechanical system composed by two rods that bend under the action of a pressure difference; they have one fixed endpoint and are partially in contact. We obtain the balance equations of the mechanical system exploiting the principle of virtual work and the contact point is identified by a jump condition. The problem can be simplified exploiting a first integral. In the case of quadratic energy, another first integral exists: its peculiarity is discussed and a further reduction of the equations is carried out. Numerical integration of the differential system shows

how the shape of the beams and the position of the contact point depend on the applied pressure. For small pressure, an asymptotic expansion in a small parameter allows to find an approximate solutions of polynomial form which is in surprisingly good agreement with the solution of the original system of equations, even beyond the expected range of validity. Finally, the asymptotics predicts a value of the pressure that separates the contact from the no-contact regime of the beams that compares very well with the one numerically evaluated.

The chordae tendineae are then modelled as a force applied to the free endpoint of the flaps. Different possible boundary conditions are investigated at the mitral annulus and, by an asymptotic analysis, we demonstrate that in the pressure regime of interest generic boundary conditions generate a tensional boundary layer. Conversely, a specific choice of the boundary condition inhibits the generation of high tensional gradients in a small layer.

Topic: From cell to tissue

Poro-Active biological tissues

G. Cappello

Université Grenoble Alpes, Laboratoire Interdisciplinaire de Physique, France
E-mail: giovanni.cappello@univ-grenoble-alpes.fr

Biological tissues are inherently composite. They are made of cells, which are virtually impermeable and incompressible but deformable and active, and extracellular matrix, which is permeable to water and about a thousand times more compressible than cells. The whole is permeated by interstitial fluid, which is incompressible but flows according to the mechanical stress suffered or exerted by the tissue.

The rheological features of each of these three components are very different. Consequently, a composite tissue made of cells, extracellular matrix and fluid will exhibit emerging rheological properties that depend both on the structural arrangement and on the volumetric ratio of the three constituents.

In our work, we point the existence of a peculiar feedback between the cells and the extracellular matrix: cells read and react to the deformation of the extracellular matrix imposed by an external perturbation. In response to a gentle compression of the matrix, cells change their proliferation rate, their motility and their contractility. In other words, the cells use the extracellular matrix as an external stress gauge and take advantage of its large compressibility to detect weak (but physiological) mechanical stimuli.

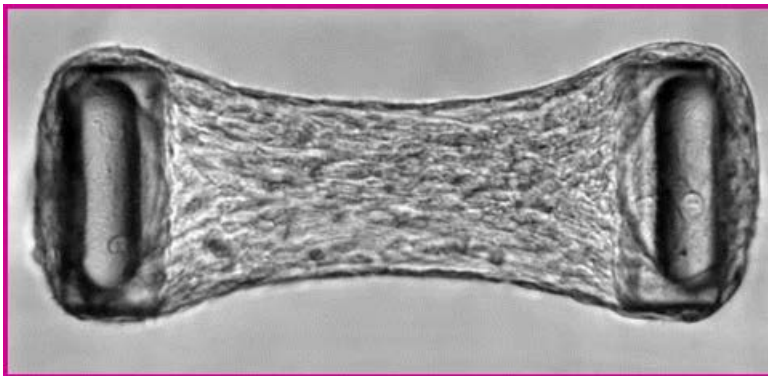


Figure 1: Multicellular composite aggregate, self-assembled between two deformable pillars.

Topic: Hard tissue

An attenuated hyperbolic model for stimulus propagation to describe frequency driven mechano-transduction in bone remodeling

F. dell'Isola

MEMOCS, Università dell'Aquila, Italy

E-mail: francesco.dellisola@univaq.it

*** In Memory of Tomasz LEKSZYCKI ***

The biological signal which drives bone remodelling is produced as a response of the mechanical deformation state of bone tissue.

This signal propagates in the bone tissue and, somehow, also in the scaffolds used for reconstructing it.

In this paper it is suggested to adapt the Cattaneo type hyperbolic equation, introduced for resolving the paradox of instantaneous heat signal propagation, also to describe the remodelling biological signal in bone tissues. This modelling choice allows for the description of the effects on bone tissue growth due to the variation of frequency of periodic applied loads.

The initial results obtained via numerical simulations suggest that the novel introduced time delay parameter does effectively describe the capacity of a specific biomechanical resonance to increase the bone growth speed.

Topic: *Hard tissue*

Multiscale bone remodeling: dream or reality?

D. George*, Y. Rémond

ICUBE, CNRS, University of Strasbourg, 300 Boulevard Sébastien Brant, 67400 Illkirch-Graffenstaden, France

E-mail: george@unistra.fr, remond@unistra.fr

* Presenting author

The bone remodeling problem has been at the heart of many biomedical applications for decades both from the surgical or medical diagnostic, and the capacity of scientists to predict its evolution from the mechanical and biological point of view. We will present here some of our latest work at macroscopic and mesoscopic scale together with published works at microscopic and cellular scale in comparison with the current state of the art. We will ask some questions about our capacity to: (i) link these different scales into realistic predictive numerical models and (ii) experimentally measure adequately the model parameters. These are the necessary steps to develop functional predictive in-silico models, but also provide better understanding of the overall problem. Finally, we will highlight some difficulties in reaching these objectives and some specific node locks to solve prior to answer the bone remodeling problem.

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Topic: From cell to tissue

The ebb and flow of life- through the lens of mechanics and thermodynamics

M. L. Knothe Tate

Blue Mountains World Interdisciplinary Innovation Institute, Australia

E-mail: proftate.bmwi3@gmail.com

All life forms change size, shape and material properties throughout their lifespan, and in response to prevailing environmental conditions. The time and length scales relevant to living materials and organisms has not been well characterized, particularly in context of how these temporal and spatial scales relate between species and evolution of species themselves. From a thermodynamics perspective, phase transition diagrams are nonexistent for living materials and their constituents. Similarly, living materials and organisms undergo the equivalent of state changes not dissimilar to those observed during nondestructive mechanical testing of materials; this is akin to a (living) material or system that changes its state (adapts) throughout exposure to shape or volume changing stresses, over relevant length and time scales of the "mechanical test". In this talk, I aim to stimulate discussion and delineation of strategies of to address these grand challenges through the assembly of international and interdisciplinary collaborative teams.

Topic: From cell to tissue

Modelling peritoneal adhesions: a biomechanical approach

M. Martin

CNRS, Univ Paris Est Creteil, Univ Gustave Eiffel, UMR 8208, MSME, F-94010 Créteil, France

E-mail: madge.martin@u-pec.fr

Peritoneal adhesions are pathological fibrotic connections forming between organ surfaces. They typically follow a surgical procedure of the thorax, abdomen or pelvis, or result from inflammatory diseases such as Crohn's or endometriosis. The presence of adhesions is especially common after intra-abdominal surgery (up to 90% of cases (Binneboesel 2008)). They can cause small bowel obstruction, chronic pain or female infertility.

So far, there is very few research around peritoneal adhesions. The initiatives toward the reduction of adhesion formation in the clinical practice have been inconsistent, and the gap between animal models and human trials is still substantial (Hassanabad 2021). Nowadays, despite a lack of awareness of patients and surgeons, adhesions are now identified as a major economic and health burden (ten Broek 2013). However, the strongest obstacle remains the poor understanding of underlying mechanisms of tissue physiological repair and adhesion formation (Brochhausen 2012, Hassanabad 2021).

We will introduce the critical role of biomechanics to better understand, prevent and also provide treatments for peritoneal adhesions, in particular within the spectrum of pain management.

Additionally, a novel and first model for peritoneal adhesions will be presented, which depicts the main underlying biochemical mechanisms associated with adhesion formation and their mechanical consequences. We find that the dissolution of the clot is highly dependent on the protein expression (tPA) of the protective lining on the organ surface, which is a common assumption in the literature (Hassanabad 2021).

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Topic: *Hard tissue*

On the modelling of spinal growth

N. Mühl-Castoldi^{a,b*}, M. Antico^b, P. Pivonka^b, V. Sansalone^a

^a Univ Paris Est Creteil, Univ Gustave Eiffel, CNRS, UMR 8208, MSME, F-94010, France

^b School of Mechanical, Medical and Process Engineering, Queensland University of Technology, Australia

E-mail: natalia.muhl-castoldi@u-pec.fr, Maria.Antico@csiro.au, peter.pivonka@qut.edu.au, vittorio.sansalone@u-pec.fr

* Presenting author

Spinal growth responds to mechanical and biochemical effects, resulting in healthy vertebrae and adequate spinal curvature. By contrast, abnormal growth may lead to pathologies, e.g. scoliosis. Thus, understanding the mechanisms of spinal growth and modelling this phenomenon can help to understand the effects of certain pathologies, and provide insights to counter them. Therefore, this work presents a bulk growth model set up in the framework of generalized continuum mechanics, aiming to better understand the mechanisms linked to spinal growth.

In this context, this formulation introduces a new kinematic configuration, which describes an irreversible growth deformation. The (total) deformation of a material element is thus multiplicatively decomposed into a growth deformation (i.e. a transformation of the local zero-stress reference state, the so-called relaxed state) accompanied by elastic deformation. As the growth deformation is generally incompatible, the compatibility of the final configuration is recovered by the elastic part of the deformation, which explains the presence of residual stresses in tissues even in the absence of mechanical loads. The model introduces mechanical and biochemical stimuli, that enter generalized statements of the virtual power and dissipation principles. The growth law of a material element is then obtained by relying on the dissipation principle, which allows an a priori satisfaction of the thermodynamic compatibility.

The general theory is refined to address the physiological and pathological growth of the spine, e.g. the spinal deformation in patients with adolescent idiopathic scoliosis. Thus, using finite element simulations, we aim to study the growth of the spine under both physiological and pathological conditions to propose avenues of reflection to clinicians working in this field.

Topic: Soft tissue

On the modeling of transversely isotropic active materials

A. Musesti

Università Cattolica del Sacro Cuore, Brescia, Italy
E-mail: alessandro.musesti@unicatt.it

Guided by the important example of skeletal muscle tissue, in the talk I will compare the two main methods used to model activation in biological materials, namely active stress and active strain. Considering an incompressible and transversely isotropic material in a hyperelastic setting, constitutive relations will be designed so that the two approaches produce the same result for a uniaxial deformation along the principal material direction. Then it will be shown that a simple shear produces different stresses in the two approaches. Hence, active stress and active strain can produce contrasting results in shear, even if they both fit uniaxial data. Our results show that collecting experimental data on uniaxial deformations alone is not enough to establish which activation approach better capture the activation mechanics.

Topic: Soft tissue

Passive and active fiber reorientation in anisotropic materials

P. Nardinocchi

Sapienza Università di Roma, Italy

E-mail: paola.nardinocchi@uniroma1.it

Fiber orientation in active and fiber reinforced material may evolve with time, under the influence of appropriate external stimuli. In this talk, I will present a continuum model to describe the reorientation of an anisotropic material structure, characterized by one or two fiber families able to modify their orientations following different evolution dynamics.

Kinematics of the body is described by both a position vector and one or two remodelling tensors that represents the reorientation process of the anisotropic material structure. By using suitable thermodynamical restrictions on the constitutive equations, the appropriate evolution equations of the remodelling tensors governed by Eshelby torques are obtained. A study of the stationary solutions is presented and discussed, in absence of any external source terms.

Topic: From cell to tissue

Mechanistic pharmacokinetic-pharmacodynamic modeling of osteoporosis treatments

P. Pivonka

School of Mechanical, Medical and Process Engineering, Queensland University of Technology, Brisbane, QLD 4000, Australia

E-mail: peter.pivonka@qut.edu.au

Osteoporosis (OP) is a chronic progressive bone disease which affects a large portion of the elderly population worldwide. OP is characterized by a slow reduction of bone matrix and changes in the bone matrix properties which ultimately leads to whole (organ) bone fractures [1].

Novel drug treatments are developed to more effectively reduce the risk of bone fractures. Assessing the effects of novel and existing treatments on OP is challenging due to the complexity of the bone remodeling process, its effects on the bone matrix and the different spatial and temporal scales involved. Identification and characterization of various bone biomarkers has significantly improved our understanding of OP pathophysiology. The bone matrix and its constituents are specific bone biomarkers measured at a particular bone site. On the other hand, biochemical ligands released during bone remodeling and measured in blood or urine are non-specific bone biomarkers. These biomarkers can be used to characterize the underlying bone mechanobiological system and drug treatment effects [1].

Recently, disease system analysis (DSA) has been proposed as a novel approach to quantitatively characterize drug effects on disease progression [1]. DSA integrates physiology, disease progression and drug treatment in a comprehensive mechanism-based modelling framework using a large amount of complementary biomarker data. In this summer school, I will present latest mechanistic pharmacokinetic-pharmacodynamic (PK/PD) models of osteoporosis treatment. Examples of currently used drug interventions including denosumab [2,3] romozosumab [4], and PTH [5] treatments will serve as discussion points on which mechanisms are essential for accurate bone remodeling simulations. Bone matrix mineralization turns out to be an essential model feature that is required to predict BV/TV changes for the case of anti-catabolic drug treatments of OP [3].

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Topic: Hard tissue

Damage Bone Adaptation Following Continuum Thermodynamics

S. Ramtani

Université Sorbonne Paris Nord, CNRS, UMR 7244, CSPBAT, Laboratoire de Chimie, Structures et Propriétés de Biomatériaux Et D'Agents Thérapeutiques Université Paris 13, Sorbonne Paris Cité, Bobigny, France

E-mail: ramtani@univ-paris13.fr

Bone as a smart and living material is based on a particularly elaborate architectural organization of the bone tissue, and on the existence of an adaptability of its structure (architectural adaptation to changes in mechanical conditions) due to the mechanism of modelling and remodeling, which are themselves linked to the coupled activity of specialized bone cell populations. The first hypothesis about the dependence between the form of bones and the load they carry was argued by Galileo as long ago as 1638. As early as 1960, Frost demonstrated, from a clinical point of view, the influence of damage, identified as micro cracks, on the bone remodeling process. These micro cracks involved in fractures are known as: (a) dispersed (diffuse) throughout the deformed bone volume, (b) very close to each other, but (c) do not show any apparent interaction and are mutually unaware. It is also showed that the adaptation process due to damageable loading (outside of physiological limits) is preceded by a dramatic increase in the frequency of micro cracking in the vicinity of the patterning and remodeling sites, consistent with the idea of stimulation of the adaptation process by damage. Following adaptive elasticity within the framework of continuum thermodynamics, due to Cowin & coworkers, adaptive bone damage law has been derived as well as behavior law, damage-remodeling energy release rate, damage dissipation, ... Some illustrative examples are presented in order to appreciate both the potential and the limits of the proposed damage bone adaptation model.

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Topic: *Soft tissue*

Self Contracting Gel

A. Bernheim-Groswasser^a, P. Nardinocchi^b, F. Recrosi^b, L. Teresi^{c*}

^a Dept of Chemical Engineering, Ilse Kats Institute for Nanoscale Science and Technology, Ben Gurion University of the Negev, Beer-Sheva, Israel

^b Dept. Structural Engineering and Geotechnic, Sapienza Università di Roma, Rome, Italy

^c Dept. Mathematics and Physics, University Roma Tre, Rome, Italy

E-mail: teresi@uniroma3.it

* Presenting author

We present the study of the transient behaviour of self-contractile disc-shaped biopolymers based on a mathematical model of active gels, numerically implemented within a finite element code. The study has been inspired by the experimental tests presented in Ideses, 2018. The analysis of liquid fluxes and gel boundary velocities evidences as the model is able to qualitatively reproduce a few key characteristics of those experiments. Moreover, the model also allows to discuss the impact of the aspect ratio of the disc on the elastic state, stresses and strains, and on the liquid diffusion driven by the self-contraction.

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