



HAL
open science

Influence of periodic diffusive inclusions on the bidomain model

Andjela Davidovic, Yves Coudière, Clair Poignard

► **To cite this version:**

Andjela Davidovic, Yves Coudière, Clair Poignard. Influence of periodic diffusive inclusions on the bidomain model. Workshop Liryc, 2013, Bordeaux, France. hal-00937884

HAL Id: hal-00937884

<https://inria.hal.science/hal-00937884v1>

Submitted on 28 Jan 2014

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

INFLUENCE OF PERIODIC DIFFUSIVE INCLUSIONS ON THE BIDOMAIN MODEL

Andjela Davidović^{1a,2} with advisers: Yves Coudiere^{1a,2,3} and Clair Poignard^{1b,2}

1.Inria Bordeaux Sud-Ouest: 1a.Carmen team, 1b.MC2 team; 2.Institut de Mathematiques de Bordeaux (IBM); 3.IHU-Liryc

Introduction

We present a new mathematical model of the electric activity of the heart. The main **drawback of the standard bidomain model** is that it assumes the existence of excitable cells (myocytes) everywhere in the heart, while it is known that there exist non small regions where non-excitable cells (fibroblasts and collagen) take place. The problems that we are trying to address with our model are:

- The **laminar structure** of the myocardium that shows the presence of collagen, especially between muscle layers [2].

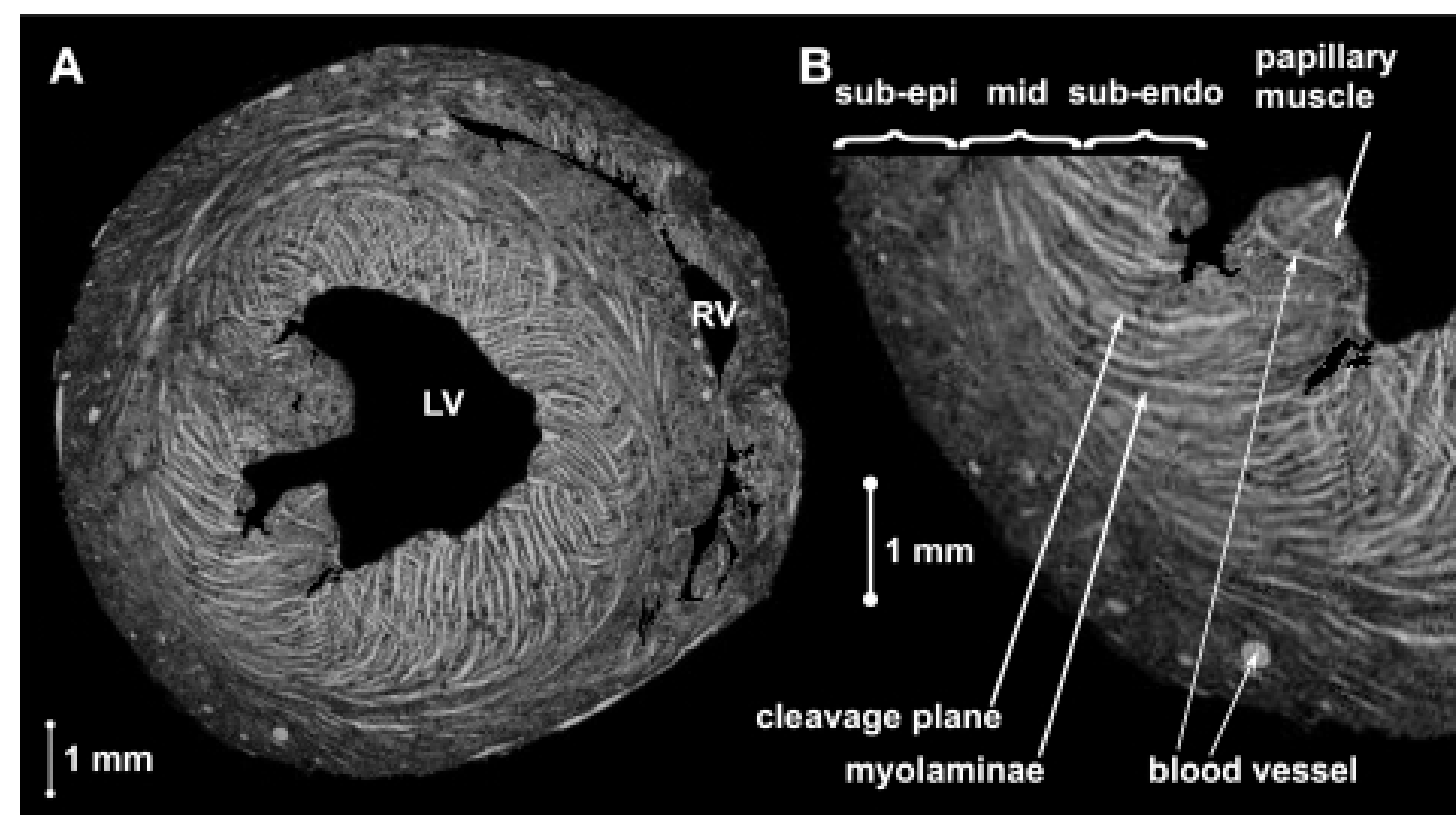


Figure 1: The laminar structure of myocardium. [3]

- The **infarct border zone**. After infraction in the heart some number of myocytes die and they are replaced by collagen and a few cells of fibroblasts. In our current model we consider as if there are no cells of fibroblasts in the extracellular space.

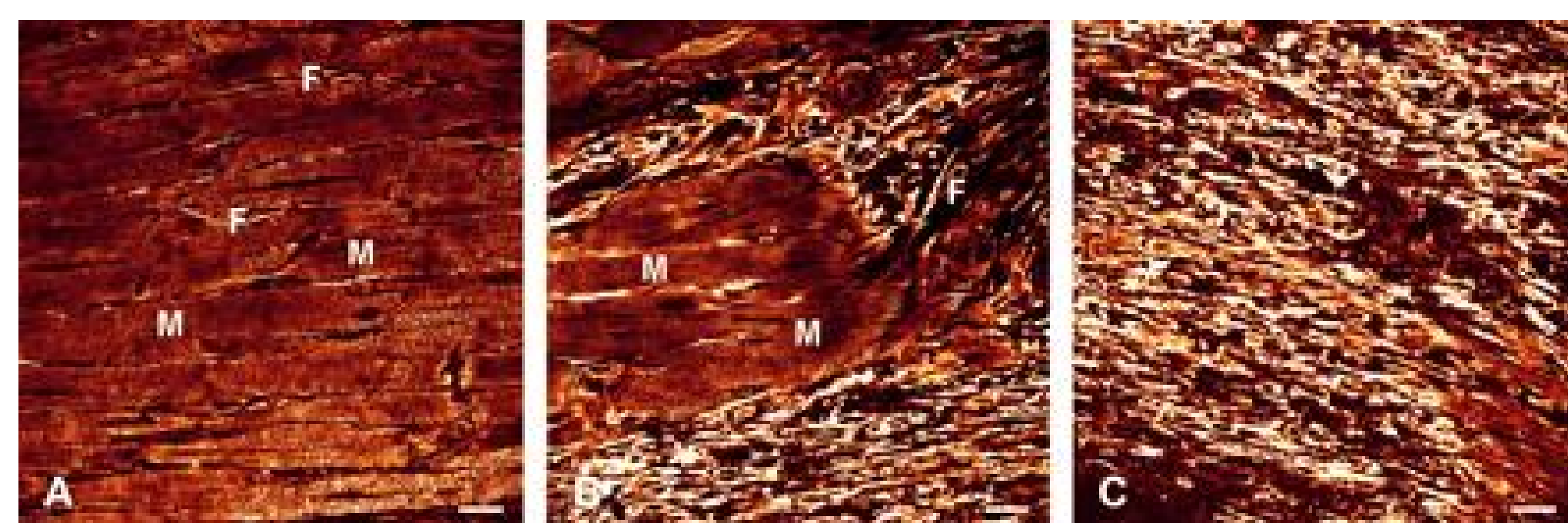


Figure 2: Fibroblast organization in sheep normal ventricular myocardium (A), infarct border zone (B) and centre (C), 1 week after infarction. [4]

I - Mesoscopic model

Modelling assumptions:

- Periodic distribution of added extracellular space
- Extracellular space is a passive conductor

Work on the domain $\Omega = B_\varepsilon \cup D_\varepsilon$

- B_ε represent the bidomain layer.
 - given intra- and extracellular conductivities σ_ε^i and σ_ε^e
 - unknown potentials u_ε^i and u_ε^e
 - define $v_\varepsilon := u_\varepsilon^i - u_\varepsilon^e$
- D_ε represent the diffusive inclusions.
 - conductivity σ_ε^d
 - unknown potential u_ε^d
- $\Sigma_\varepsilon = \partial B_\varepsilon \cap \partial D_\varepsilon$ is the interface.

The bidomain model

$$\begin{aligned} \partial_t v_\varepsilon + cv_\varepsilon &= \nabla \cdot (\sigma_\varepsilon^i \nabla u_\varepsilon^i), & B_\varepsilon, \\ \partial_t v_\varepsilon + cv_\varepsilon &= -\nabla \cdot (\sigma_\varepsilon^e \nabla u_\varepsilon^e), & B_\varepsilon, \end{aligned}$$

The diffusive inclusion

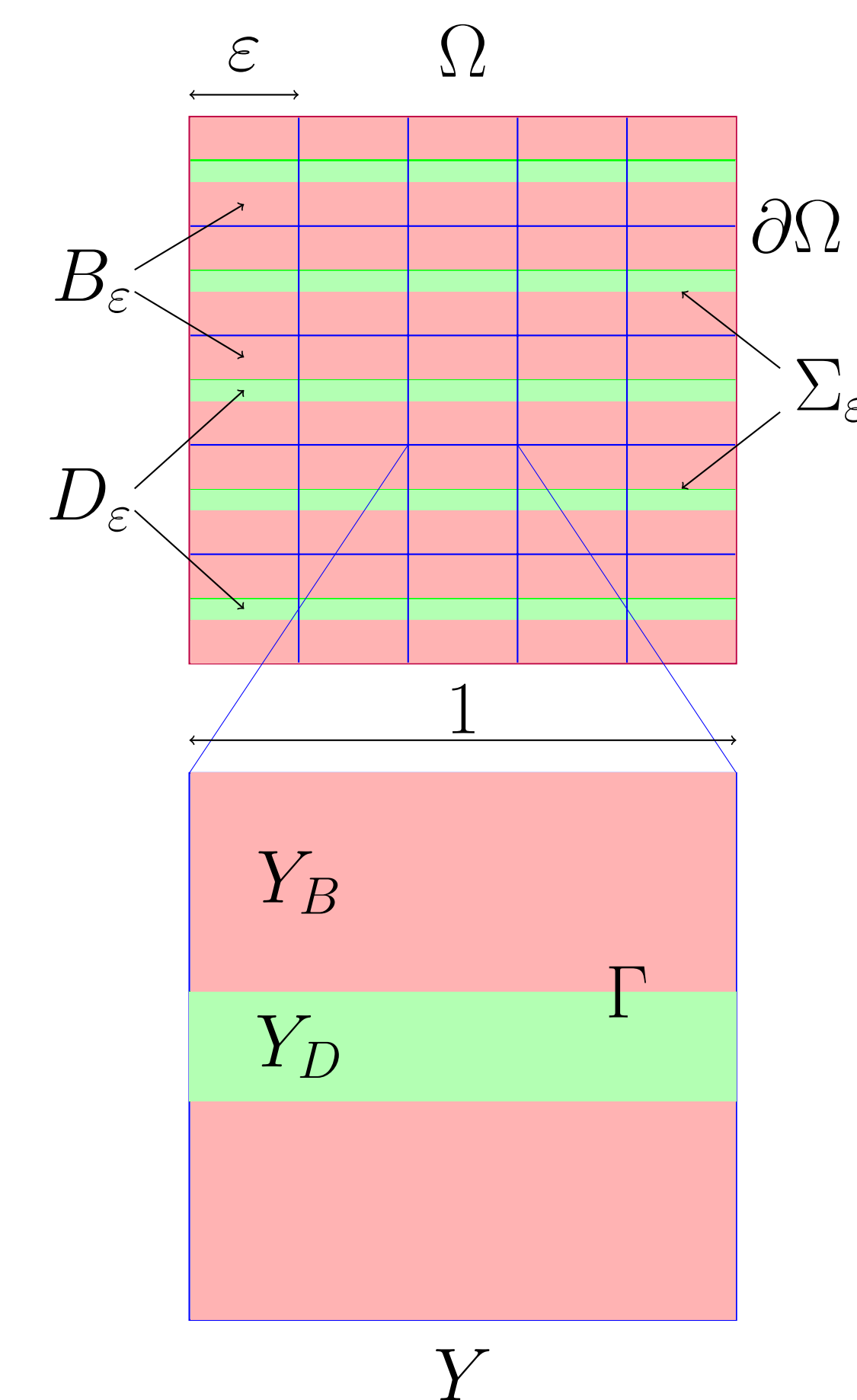
$$0 = -\nabla \cdot (\sigma_\varepsilon^d \nabla u_\varepsilon^d), \quad D_\varepsilon,$$

The transmission conditions

$$\left. \begin{aligned} \sigma_\varepsilon^i \nabla u_\varepsilon^i \cdot n_{\Sigma_\varepsilon} &= 0, \\ \sigma_\varepsilon^e \nabla u_\varepsilon^e \cdot n_{\Sigma_\varepsilon} &= \sigma_\varepsilon^d \nabla u_\varepsilon^d \cdot n_{\Sigma_\varepsilon}, \\ u_\varepsilon^e &= u_\varepsilon^d, \end{aligned} \right\} \quad \Sigma_\varepsilon,$$

The initial and the boundary conditions given.

- We have a well posed problem up to a constant.
- Numerical simulations expensive.



II - Homogenisation

Principle: Redefine the problem in two scales and obtain the limit problem that depends only on a large scale. It gives a global, macroscopic behaviour of the unknown functions.

Assumption

$$\sigma_\varepsilon^i(x) = \sigma_i \left(\frac{x}{\varepsilon} \right), \quad \sigma_\varepsilon^e(x) = \sigma_e \left(\frac{x}{\varepsilon} \right), \quad \sigma_\varepsilon^d(x) = \sigma_d \left(\frac{x}{\varepsilon} \right).$$

Having apriori estimates on the norms of the unknown functions and their derivatives, and following the work in [1], there is a limit for $\varepsilon \rightarrow 0$

$$u_\varepsilon^i \rightarrow u_0^i, \quad u_\varepsilon^e \rightarrow u_0^e, \quad u_\varepsilon^d \rightarrow u_0^d.$$

III - Macroscopic equations

$$\text{Define} \quad \sigma_i^* := \sigma_i / |Y_B|, \quad \sigma_e^* := \sigma_e / |Y_B|, \quad \sigma_d^* := \sigma_d / |Y_D|, \quad \rho = |Y_D| / |Y_B|.$$

$$\text{In the limit} \quad u_0^e(t, x) = u_0^d(t, x) =: u_0(t, x).$$

And the limit problem is again **the bidomain model** with updated conductivities

$$\begin{aligned} \nabla_x \cdot ((\sigma_i^* + Id) \nabla_x u_0^i) &= \partial_t v_0 + cv_0, \quad \text{in } \Omega, \\ \nabla_x \cdot ((\sigma_e^* + \rho \sigma_d^* + (\sigma_e - \sigma_d) (A_e + \rho A_d)) \nabla_x u_0) &= -(\partial_t v_0 + cv_0), \quad \text{in } \Omega, \end{aligned}$$

Where A_e and A_d are constant matrices that depend only on the geometry of the unit cell.

Remark:

- The modified conductivities depend on the volume fraction of the diffusive part and on the geometry of the unit cell.
- If $\sigma_e = \sigma_d$ the problem simplifies and the modified conductivities depend only on the volume fraction.

IV - Numerical verifications

We work on the numerical simulations

- On simple geometries.
- Using FEM method.
- Values of parameters obtained from the literature.
- Using *Gmsh* mesh generator ([6]) and *FreeFem++* PDE solver ([5]).

We expect to observe the convergence of the mesoscopic model to the macroscopic model.

Perspectives

We can develop our work in several directions.

- Perform numerical experiments on different geometries, simulating only the macroscopic model.
- Use real data obtained by clinicians. For example, the late enhancement MRI provides the volume fraction of the extracellular space, ρ .
- Apply the ionic current model.
- Develop a model for the infarct scar transiting from the classical bidomain, via our new model, to the purely diffusive centre of the scar.

References

- [1] Allaire, *Homogenisation and two scale convergence*, SIAM J. MATH. ANAL. Vol. 23, No. 6, pp. 1482-1518, November 1992.
- [2] Hooks et al, *Laminar Arrangement of Ventricular Myocytes Influences Electrical Behavior of the Heart*
- [3] Gilbert et al, *Visualization and quantification of whole rat heart laminar structure using high-spatial resolution contrast-enhanced MRI*, Am J Physiol Heart Circ Physiol. 2012 January; 302(1): H287-H298
- [4] Camelliti et al, *Structural and functional characterisation of cardiac fibroblasts*, Cardiovasc Res (2005) 65 (1): 40-51.
- [5] Pironneau et al, *FreeFem++* <http://www.freefem.org/>
- [6] Geuzaine, Remacle *Gmsh* <http://geuz.org/gmsh/>

Contact: andjela.davidovic@inria.fr, yves.coudiere@inria.fr, clair.poignard@inria.fr