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INTRODUCTION

In-stent restenosis (ISR) is one of the main drawbacks stenting [1]. Among the several factors:

- **systemic inflammation** cause by arterial wall damage
- **hemodynamic alteration** due to stent presence

AIM

To develop a **patient-specific multiscale model of ISR** integrating the effects of **hemodynamics** and **monocyte gene expression (GE)** on the **cellular dynamics**.

- Coupling of:**
- Computational fluid dynamics (**CFD**)
 - Agent-based model (**ABM**) of cell dynamics

METHODS

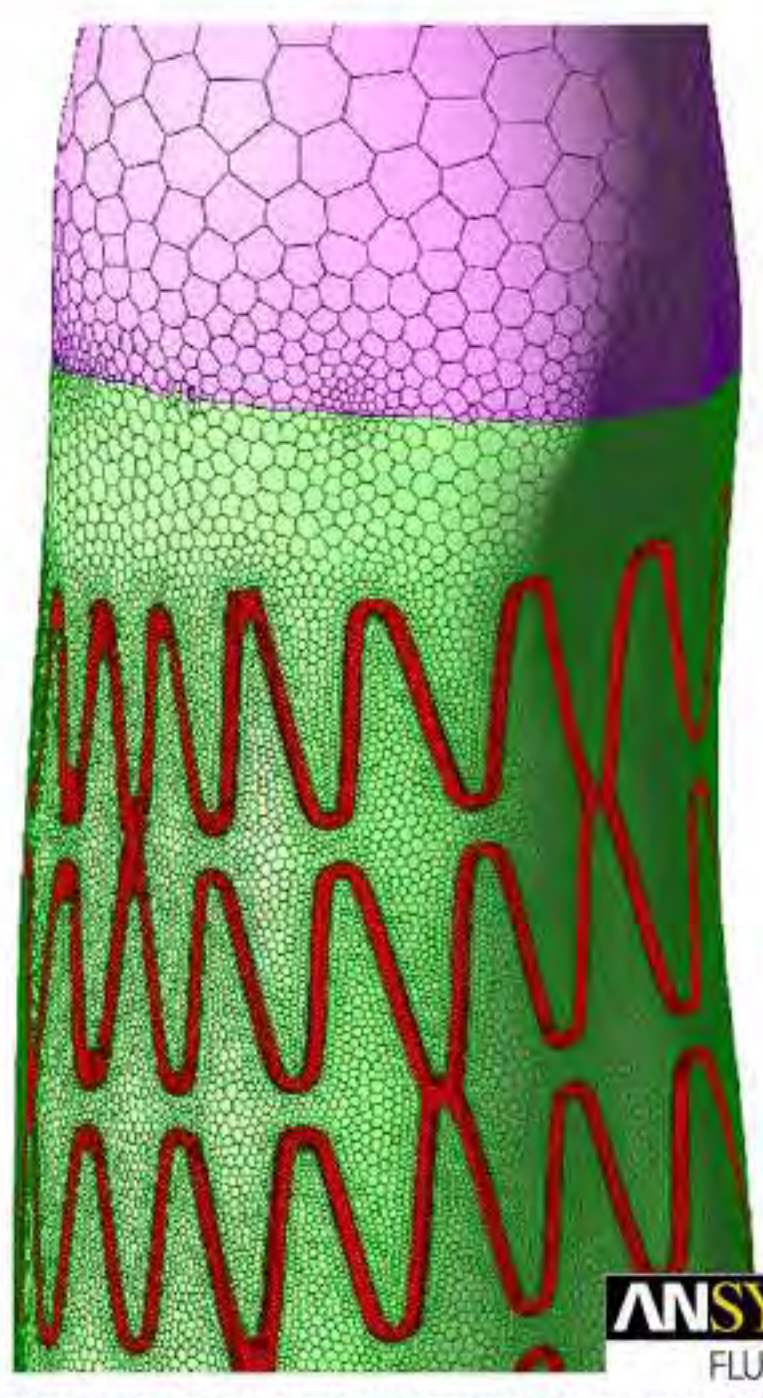
3D model of stented SFA

- **Patient-specific geometry** of the vessel reconstructed from **CT images**
- **Stent deployment** through morphing procedure



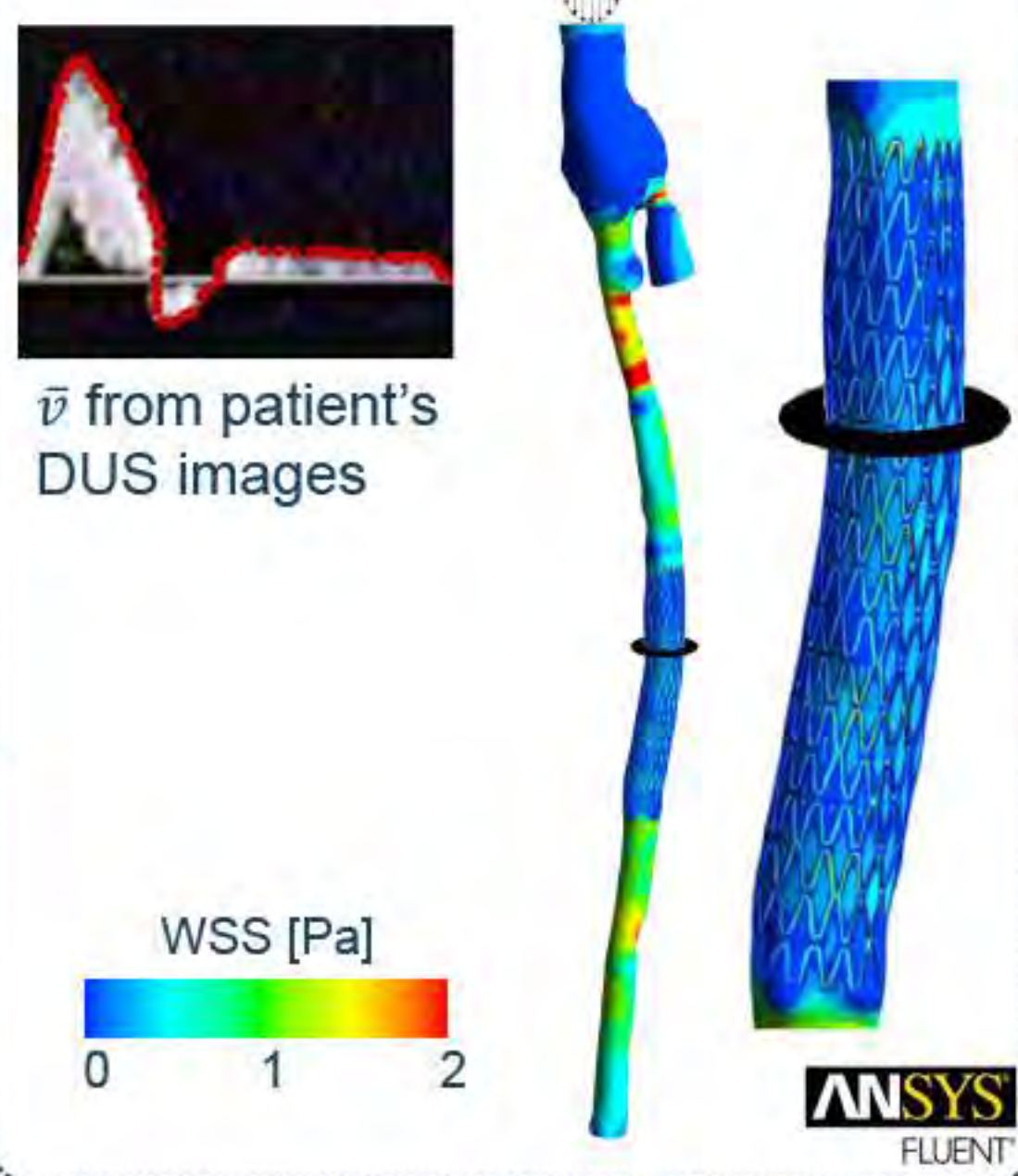
Meshing

- **Polyhedral mesh**
- **5 layers of prism elements**
- **~3 millions** of elements



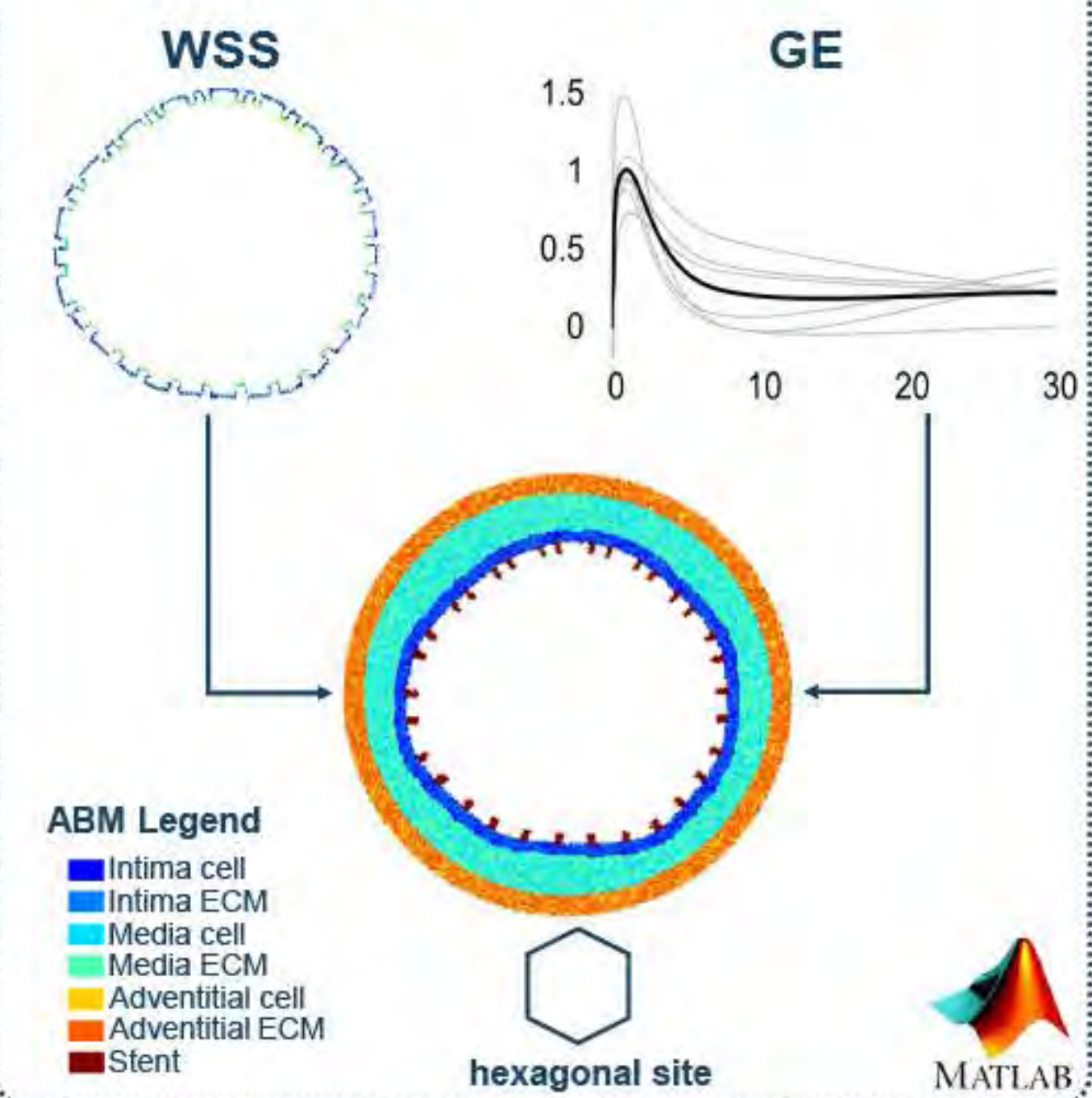
CFD simulation

- **Steady state**
- **Boundary conditions**
- ☐ **Inlet:** parabolic velocity profile
- ☐ **Outlet:** flow-split



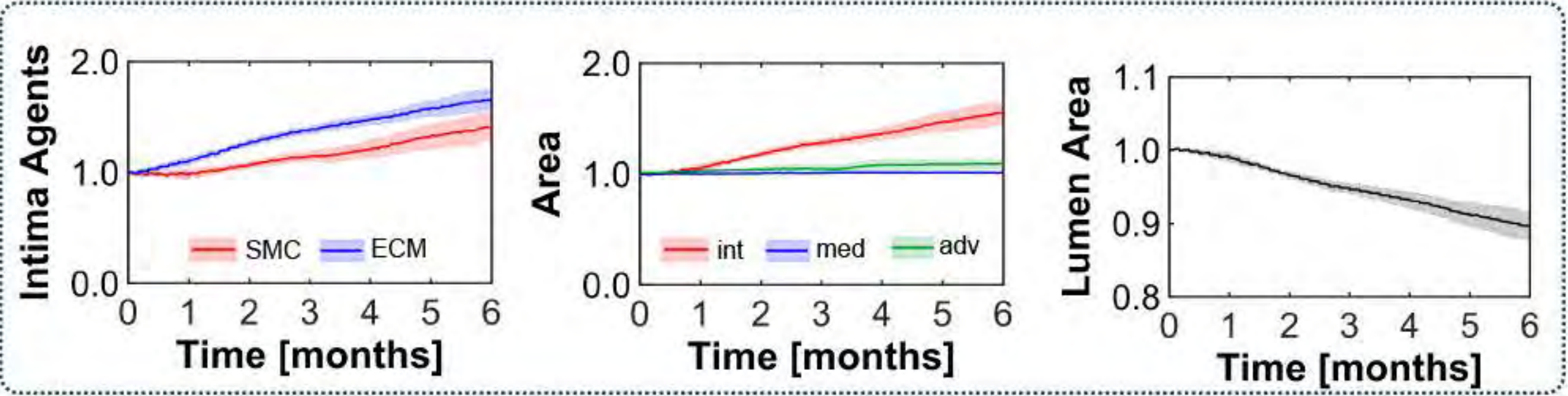
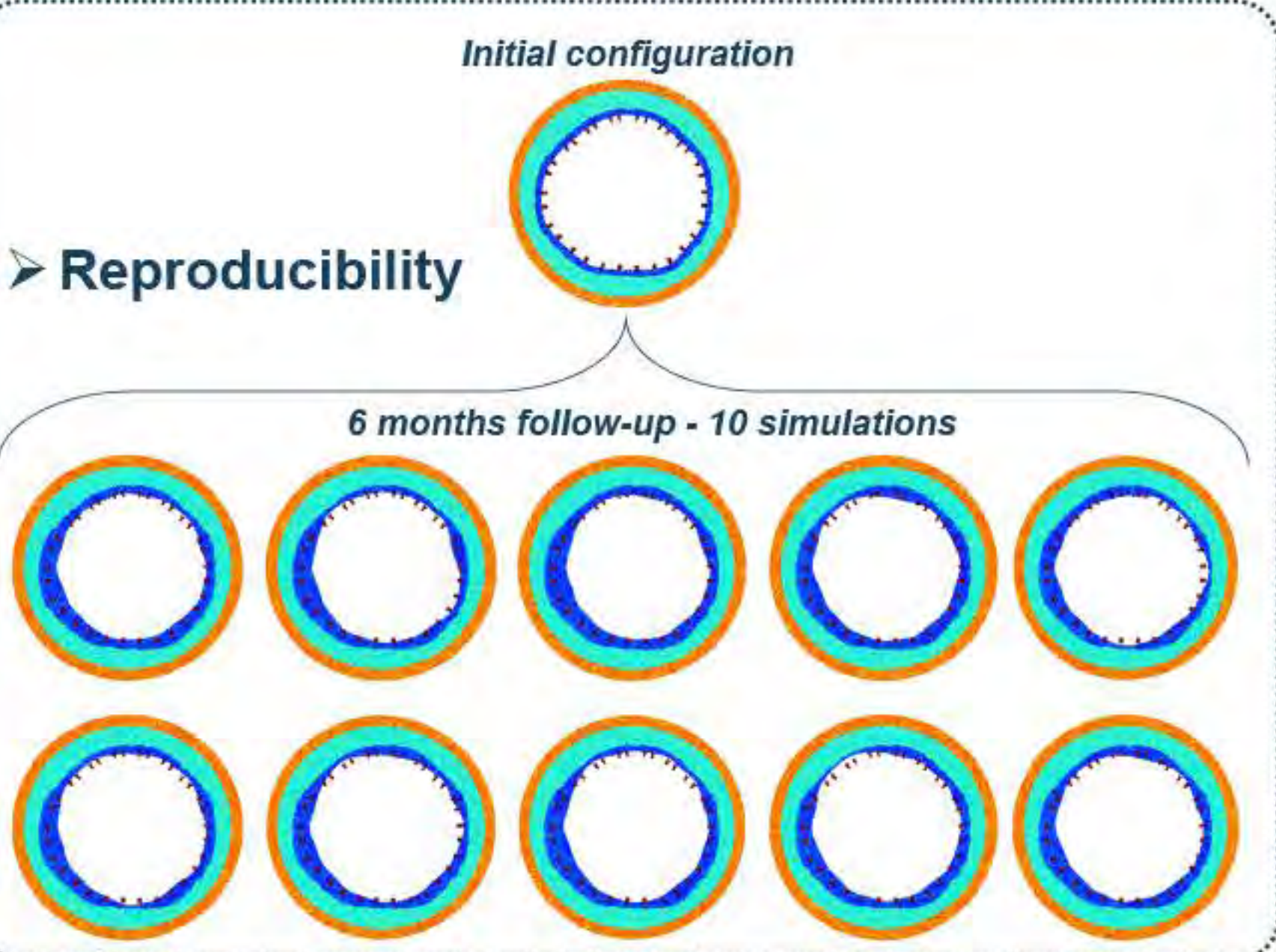
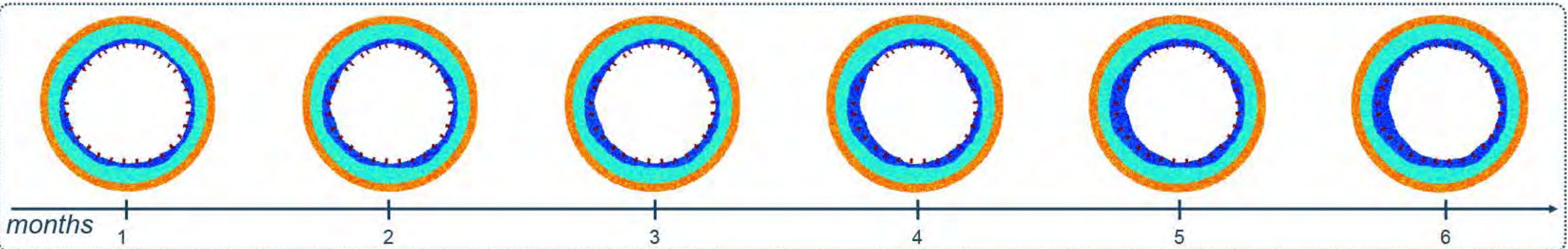
ABM simulation

- Simulation of **cell and ECM dynamics**
- **Cell proliferation and ECM production** are function of the local **WSS** and **GE**
- **Stochastic model** → 10 simulations



Starting from a **3D model of patient-specific stented superficial femoral artery (SFA)**, the hemodynamics was computed and the wall shear stress (**WSS**) profile was extracted at a selected cross-section. An **ABM** of that cross-section was **initialized with both the WSS profile and the monocyte GE** data, accounting for the systemic inflammation. Finally, the ABM simulated the arterial response to the stenting procedure, by replicating **cell and ECM dynamics**.

RESULTS



- **Lumen area decrease** due to **intima growth**
- Higher **growth** at **low WSS** regions
- **Increase** of **intimal SMC** and **ECM** over time
- Most of **stent struts** covered at 6 months
- **Baseline** media and adventitia

CONCLUSIONS

- The framework:
- Replicates the process of **ISR at cell/tissue scale**
 - Integrates **fluid dynamics, gene expression** and **cellular dynamics**
 - Investigates the effects of stent-induced **hemodynamic alteration** and **inflammation** on the outcome of stenting