AMMA
A Multiscale Model of Atherosclerosis

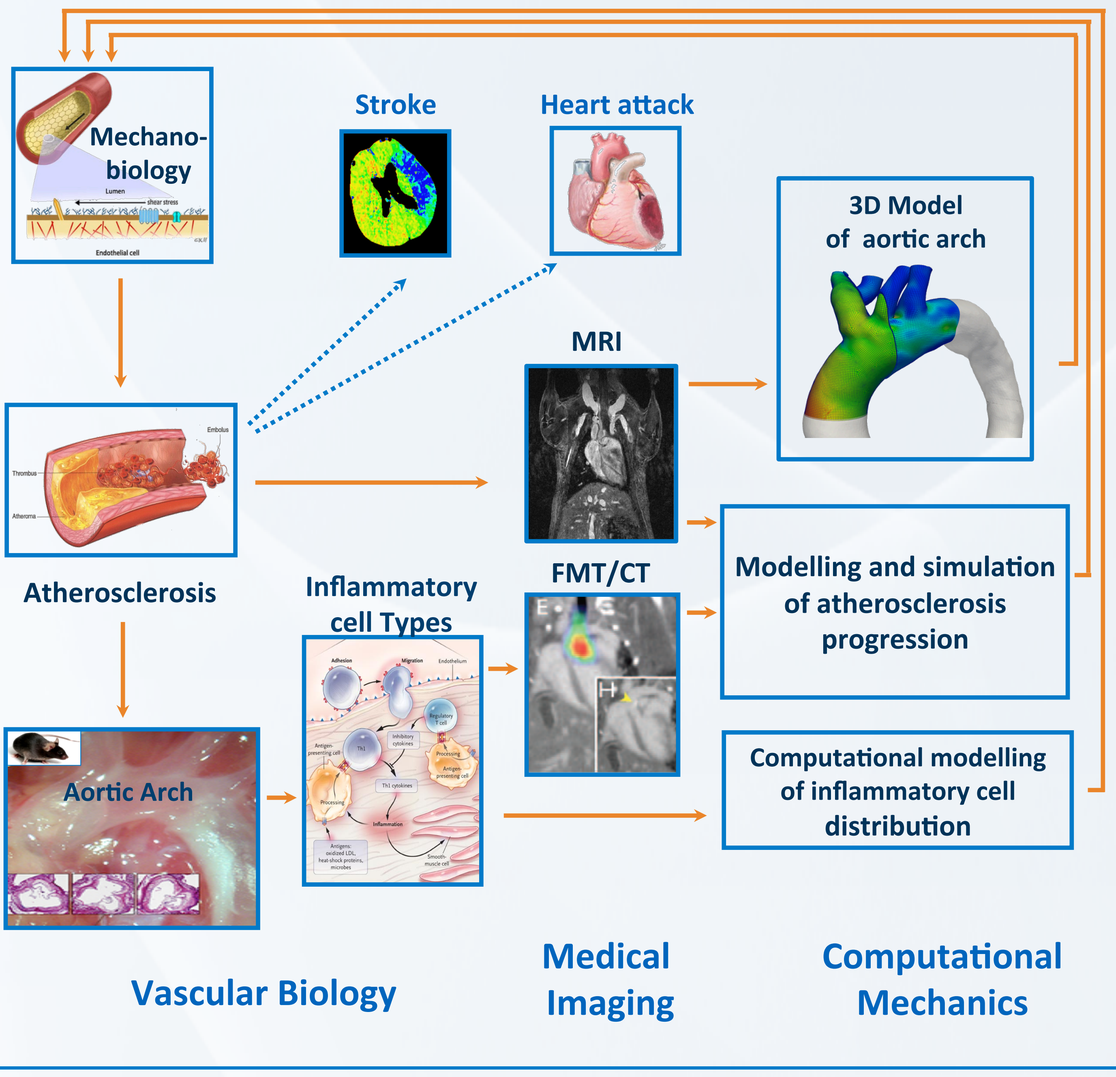
Almut Glinzer[1 & 2], Moritz Thon[3], Moritz Wildgruber[2], Alma Zernecke[1 & 4] and Michael W. Gee[3]

[1] Department of Vascular Biology, Klinikum Rechts der Isar, Technische Universität München, [2] Department of Radiology, Klinikum Rechts der Isar, Technische Universität München,
[3] Mechanics and High Performance Computing Group, Technische Universität München, [4] Institute for Clinical Biochemistry and Pathobiochemistry, Universitätsklinikum Würzburg

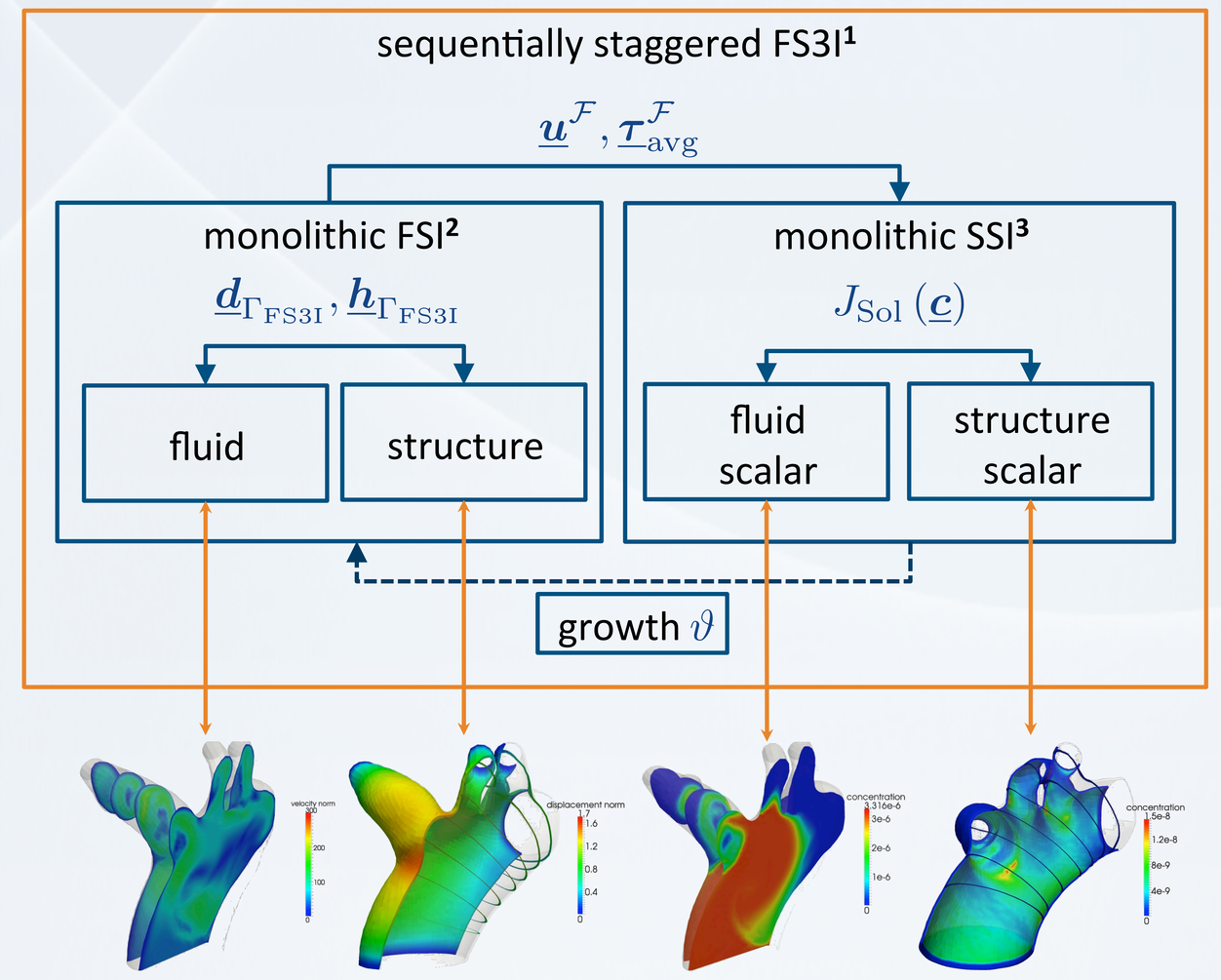
1 Background

Atherosclerosis plays a central role in the pathogenesis of cardiovascular diseases and is commonly found in the population of developed countries. It is characterized by an accumulation of inflammatory cells and lipids in the intima and media of the arterial wall resulting in arterial wall thickening and narrowing of the vessel lumen. Atherosclerosis causes sequelae such as angina, heart attack and stroke. So far the processes of disease progression, especially mechanically relevant aspects that promote arterial wall thickening, are not fully understood. Within the AMMA-Project we develop a mathematical multiscale model of atherosclerosis to establish a better understanding of its pathogenesis and especially its mechanobiology.
Therefore, three disciplines join forces: **Vascular Biology, Medical Imaging and Computational Mechanics.**

2 Project Overview

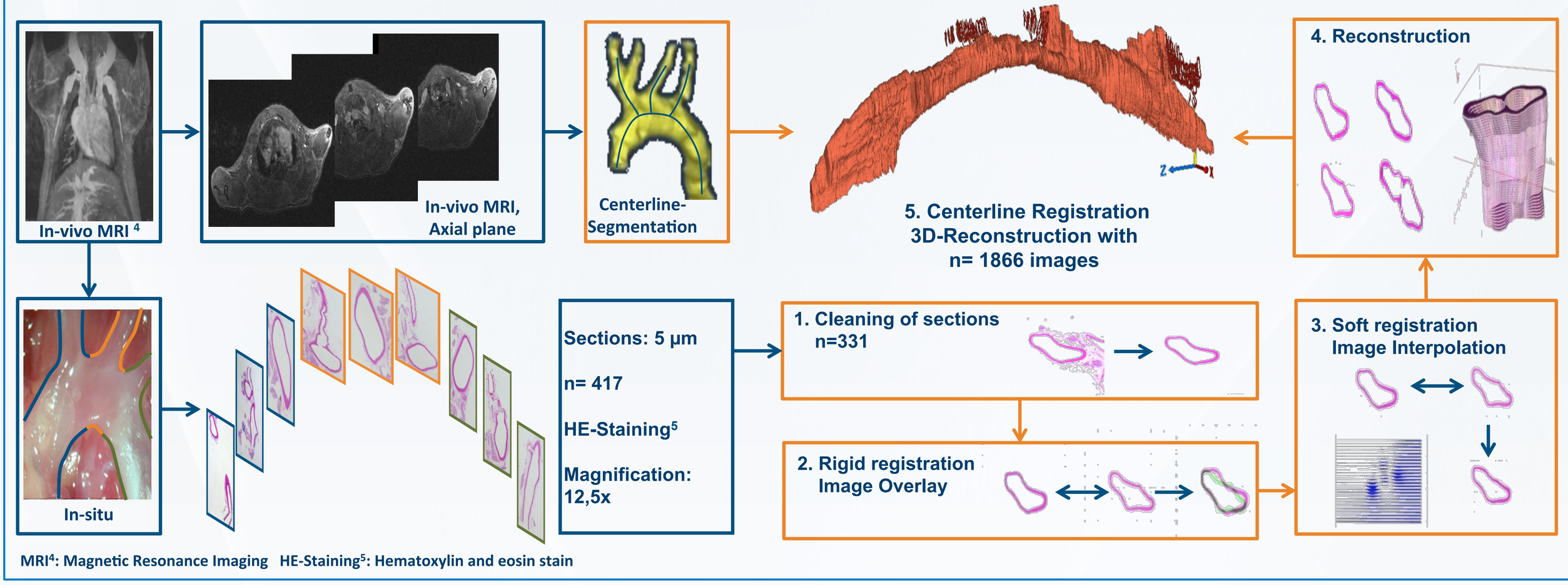


3 Mathematical Model



FS3I[1]: fluid-structure-scalar-scalar interaction FSI[2]: fluid-structure interaction SSI[3]: scalar scalar interaction

4 3D Reconstruction of the Murine Aortic Arch using Histological Sections



MRI[4]: Magnetic Resonance Imaging HE-Staining[5]: Hematoxylin and eosin stain

5 Outlook

We combine the use of cell biology techniques together with medical imaging to visualize and quantify basic pathomechanisms of lesion formation. The acquired data is used to formulate mathematical equations establishing a 3D mechanobiological model of the mouse aortic arch. Simulation of mechanical interactions of blood flow and arterial wall biology and its impact on the pathogenesis of atherosclerosis will be verified at site specific localizations of plaque development and the accumulation of certain immune cells. With this interdisciplinary collaboration it will be possible to gain more knowledge on mechanical and biological interactions involved in atherogenesis.