The Impact of Arterial Plaque Tissue Elasticity on In-Body Molecular Communication Scenarios

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***Abstract*—*Atherosclerosis*, the plaque formation in human arteries, has recently been targeted within molecular communication (MC) research by proposing a joint communication and sensing approach to detect the growing plaque as an influence on the in-body MC channel. While previous work was based on simplifying assumptions, more realistic models are crucial to accurately characterize the potential of this technique. Expanding upon existing work that incorporates the non-Newtonian properties and the time-varying flow speed of blood, we now tackle the influence of tissue elasticity on the system. Using an advanced fluid-solid interaction (FSI) solver, we characterize the plaque and examine the displacement for different FSI models and plaque sizes. Our results show differences between experimental elasticity models from literature but only very small changes to the plaque geometry within fractions of a millimeter across all options. This is an important step to legitimize the assumption of rigid tissue geometry in future investigations of this scenario under realistic conditions.**

***Index Terms*—Fluid-Solid-Interaction, Internet of Bio-Nano Things, Molecular Communication, Plaque Formations**

I. Introduction

MOLECULAR communication (MC) relies on molecules or particles for information exchange and is envisioned to play a role in medical applications for a future Internet of Bio-Nano Things (IoBNT), where diagnosis and treatment are made easier and more effective. MC research has long proposed the human body, specifically its blood vessels, as a potential channel for communication [1]. As the human body changes, the communication channel also changes, which could be used as a detection mechanism similar to joint communication and sensing approaches in classical networks. One of the most significant issues for human health worldwide is plaque formation in arteries known as *atherosclerosis*, the most common cause of cardiac death [2]. Therefore, *atherosclerosis* has been the focus of research in the MC field, including extensive modeling by Felicetti *et al.* of *atherogenesis*, i.e., the starting phase of *atherosclerosis*, as an MC system to improve the understanding of the drivers behind the disease [3].

Recently, we have proposed to interpret plaque formation as a physical change of the MC channel and have found that plaques of different sizes significantly influence characteristics such as the channel impulse response (CIR) [4], [5]. Our goal is to realistically model a blood vessel with a developing plaque, which includes a number of important aspects, such as non-Newtonian fluid dynamics, the pulsatility of the blood flow, the plaque geometry, or the material properties of the tissue. So far, we have specifically addressed all but the last point in the list [5], showing each time the channel effects remain significant, and the concept therefore valid. In this work-in-progress paper, we want to challenge the common modeling assumption that human tissue is rigid. We will use an advanced fluid-solid interaction (FSI) solver to answer the question: How does tissue elasticity impact the MC channel within blood vessels with plaque build-up? This is an important step to assess the validity of our assumptions. Initially, we will focus on the scenario within a human carotid outlined in [4], [5]. Significant work exists based on experimental medical data characterizing the elasticity of plaque tissue. From a biomechanical perspective, researchers have experimentally measured plaque elasticity [6] or employed imaging-based methods to classify tissue types and elasticities [7]. While these studies highlight the importance of these factors in plaque progression and health risk, our work provides an MC viewpoint, focusing on whether FSIs, particularly plaque elasticity under pulsatile blood flow, could significantly alter channel conditions.

II. System Model and Fluid-Solid Interaction

Building upon our previous work in [4], [5], we consider a three-dimensional system model of a plaque formation in a human carotid artery and the corresponding geometry, cf. Fig. 1. We assume a circular transmitter releasing the particles uniformly distributed over the cross-section of the channel. Upon release, the particles propagate to a circular observing receiver, which counts the number of particles passing through the surface. The radial expansion of the plaque is denoted as r_p. For this work, we consider $r_p = \{0.5, 0.75\} \times r_c$.

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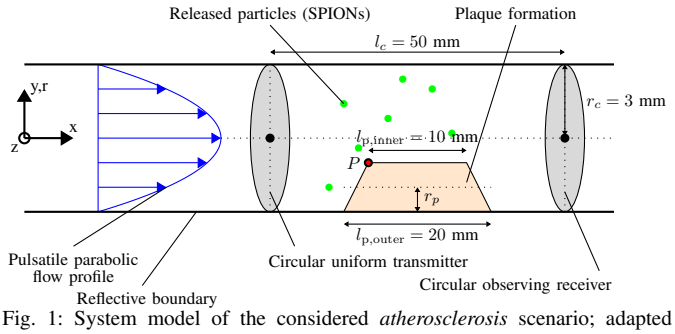

Fig. 1: System model of the considered *atherosclerosis* scenario; adapted from [4], [5]. P marks the observation point for tracking the displacement.

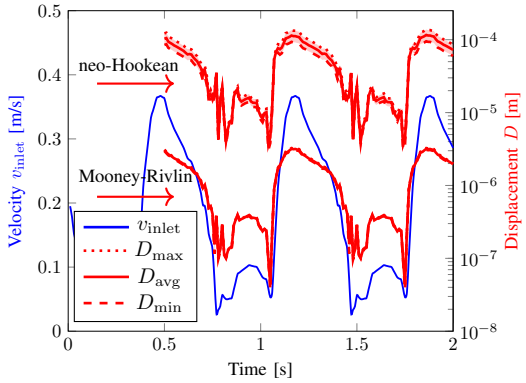

Fig. 2: Fluid velocity v_{inlet} at the inlet and the average, minimum, and maximum displacement magnitudes D_{avg}, $D_{\min}$, and $D_{\max}$ over time. Results are shown for two different elasticity models. The displacement here refers to the observation point P.

Furthermore, as introduced in [5], we consider a time-varying inlet flow boundary condition, resembling pulsatile blood flow [8, Fig. 2]. In this work-in-progress, we only consider a two-dimensional slice of the three-dimensional model for our simulations. This results in a reduced computational domain and faster iteration time steps.

In contrast to previous work, we consider FSI, i.e., we also simulate the behavior of the solid, in our case, the plaque formation. As the material law, we consider two alternative approaches from literature based on experimental measurements and representing the most and least elastic data we could find: the Mooney-Rivlin law (MR) with coefficients $c_{10} = 0.19$ MPa, $c_{01} = 0.026$ MPa, and $c_{11} = 1.377$ MPa [9] and the neo-Hookean (nH) hyperelastic model with Young's modulus $E = 32$ kPa [10]. For the density of the plaque formation, we assume $\rho \approx 1200\,\mathrm{kg/m^3}$ [11] and Poisson ratio $\nu \approx 0.49$ [7]. As the toolbox for implementing FSI in our system model, we utilize the open-source *solids4foam* [12], [13] toolbox. solids4foam performs solid mechanics and FSI simulations in OpenFOAM; the latter was already utilized in [4], [5]. The Aitken and Dirichlet-Neumann coupling approaches are used for numerical relaxation and passing stress between the solid and fluid interfaces, respectively [13], [14].

III. Results and Conclusion

Due to the two-phase solver for fluid and solid, it becomes more challenging to include particles as a third phase. For this initial work, instead of utilizing the received number of particles, we therefore tracked the displacement of a specific plaque cell, marked as observation point P in Fig. 1, after letting the velocity field settle, i.e., starting the FSI after $0.5\,\mathrm{s}$, as shown in Figure 2. The largest deformation consistently coincided with peak systole, underscoring the role of inlet velocity. Comparing different experimental parameter models revealed significant variation in maximum displacement: For the MR model, we observed $D_{\max} \lesssim 2 \times 10^{-3}\,\mathrm{mm}$, whereas the nH hyperelastic model exhibited $D_{\max} \lesssim 0.1\,\mathrm{mm}$. Even at this reasonable upper bound of elasticity, the displacement is too small to meaningfully alter the propagation of particles in an MC channel. We used $r_p = 0.75 \times r_c$ for the plot. For smaller plaques, we found deformations were even further reduced. In conclusion, even with plaques as obstacles, regular MC scenarios in blood vessels such as the carotid do not need to consider FSIs for realistic results. An extension of this work towards other sizes of blood vessels and a three-phase FSI-particle solver will be crucial. Going forward, we will also investigate the effects of surface roughness and aim to propose a coherent plaque detection approach using a realistic model.

References

[1] I. F. Akyildiz, M. Pierobon, S. Balasubramaniam, and Y. Koucheryavy, "The Internet of Bio-Nano Things," *IEEE Commun. Mag.*, vol. 53, no. 3, Mar. 2015.

[2] V. R. Preedy and R. R. Watson, Eds., *Handbook of Disease Burdens and Quality of Life Measures*. New York, NY: Springer, 2010.

[3] L. Felicetti, M. Femminella, G. Reali, P. Gresele, M. Malvestiti, and J. N. Daigle, "Modeling CD40-Based Molecular Communications in Blood Vessels," *IEEE Trans. on NanoBioscience*, vol. 13, no. 3, pp. 230–243, Sep. 2014.

[4] P. Hofmann, S. Schmidt, A. Wietfeld, et al., "A Molecular Communication Perspective on Detecting Arterial Plaque Formation," *IEEE Trans. Mol. Biol. Multi-Scale Commun.*, vol. 10, no. 3, pp. 458–463, Sep. 2024.

[5] A. Wietfeld, P. Hofmann, J. Fuchtmann, et al., "Advanced Plaque Modeling for Atherosclerosis Detection Using Molecular Communication," in *Proc. of the IEEE Int. Conf. on Communications (ICC)*, accepted for publication, Jun. 2025.

[6] S. R. H. Barrett, M. P. F. Sutcliffe, S. Howarth, Z.-.-Y. Li, and J. H. Gillard, "Experimental Measurement of the Mechanical Properties of Carotid Atherothrombotic Plaque Fibrous Cap," *J. Biomech.*, vol. 42, no. 11, pp. 1650–1655, Aug. 2009.

[7] M. C. Moraes, F. M. Cardoso, and S. S. Furuie, "Atherosclerotic Plaque Characterization Using Plaque Area Variation in IVUS Images during Compression: A Computational Investigation," *Rev. Bras. Eng. Bioméd.*, vol. 30, pp. 159–172, Jun. 2014.

[8] S. Sen, R. Petraco, J. Mayet, and J. Davies, "Wave Intensity Analysis in the Human Coronary Circulation in Health and Disease," *Curr. Cardiol. Rev.*, vol. 10, no. 1, pp. 17–23, Mar. 2014.

[9] I. Oliveira, P. Cardiff, C. Baccin, and J. Gasche, "A Numerical Investigation of the Mechanics of Intracranial Aneurysms Walls: Assessing the Influence of Tissue Hyperelastic Laws and Heterogeneous Properties on the Stress and Stretch Fields," *J. Mech. Behav. Biomed. Mater.*, vol. 136, Art. no. 105498, Dec. 2022.

[10] S. Barrett, M. Sutcliffe, S. Howarth, Z.-Y. Li, and J. Gillard, "Experimental Measurement of the Mechanical Properties of Carotid Atherothrombotic Plaque Fibrous Cap," *J. Biomech.*, vol. 42, no. 11, pp. 1650–1655, Aug. 2009.

[11] D. A. Rahdert, W. L. Sweet, F. O. Tio, C. Janicki, and D. M. Duggan, "Measurement of Density and Calcium in Human Atherosclerotic Plaque and Implications for Arterial Brachytherapy," *Cardiovasc. Radiat. Med.*, vol. 1, no. 4, pp. 358–367, Oct. 1999.

[12] P. Cardiff, A. Karac, P. D. Jaeger, et al., "An Open-Source Finite Volume Toolbox for Solid Mechanics and Fluid-Solid Interaction Simulations," *arXiv: Numerical Analysis*, pp. 1–45, Sep. 2018.

[13] Ž. Tuković, A. Karač, P. Cardiff, H. Jasak, and A. Ivanković, "OpenFOAM Finite Volume Solver for Fluid-Solid Interaction," *Trans. of FAMENA*, vol. 42, no. 3, pp. 1–31, Oct. 2018.

[14] P. Cardiff, "solids4foam Overview and Future Directions," in *2nd CCP-WSI Hackathon Streatham Campus*, Jul. 2022.