# INFLUENCE OF WALL SHEAR AND MECHANICAL STRESS ON ATHEROSCLEROTIC ARTERY DISEASE IN HUMAN CORONARIES

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## Introduction

Atherosclerosis in coronary arteries is the main cause of fatal and disabling coronary events [1]. It is well established that atherosclerotic plaque initiation and growth are affected by a local biomechanical factor; the blood flow induced wall shear stress (WSS) [2, 3]. However, the potential role of another biomechanical factor, the blood pressure induced wall mechanical stress (WMS) has been mainly overlooked. In this study, we investigated the individual and combined effects of WMS and WSS in atherosclerosis progression in coronary arteries.

## Methods

Forty non-stented, non-culprit coronary arteries were imaged with near-infrared spectroscopy intravascular ultrasound (NIRS-IVUS) and optical coherence tomography (OCT) at the baseline and after 12 months (follow-up) [3]. The composition and geometry of the coronaries and plagues were reconstructed based on the co-registration of the segmented NIRS-IVUS and OCT data. WMS in the coronaries was computed with finite element models, using hyperelastic material models and incorporating initial stresses via the backward incremental method [4]. Max principal stress on the peri-luminal region at systolic pressure of 120 mmHg was reported. WSS was computed with Computational Fluid Dynamics and reported previously [3]. Vessel wall and plaque growth was quantified as plaque burden change ($\Delta$PB) over time. For local analysis, the arteries were divided into sectors of 1.5 mm thickness and of 45° circumferential angle. The sectors were categorized as "diseased with lipid rich necrotic core (LRNC)", "diseased w/o LRNC", or "healthy". The individual and combined effect of WSS and WMS on plaque progression was evaluated using Linear Mixed Model in SPSS by dividing the stress metrics in tertiles.

## Results

There was an inverse correlation between the WSS and $\Delta$PB for all three arterial sector types (Fig.1, top panel). The sectors exposed to lower WSS showed higher $\Delta$PB. This correlation was strongest for the diseased sectors with LRNC. The correlation between WMS and $\Delta$PB; however, depended on the sector type (Fig.1, bottom panel). The healthy sectors had a positive correlation whereas the diseased ones with LRNC showed a negative correlation. The analysis of the combined effect of WSS and WMS revealed that the highest $\Delta$PB was associated with low WSS and high WMS for healthy sectors. For the diseased sectors with LRNC, both high and low WMS combined with low WSS were associated with higher $\Delta$PB.

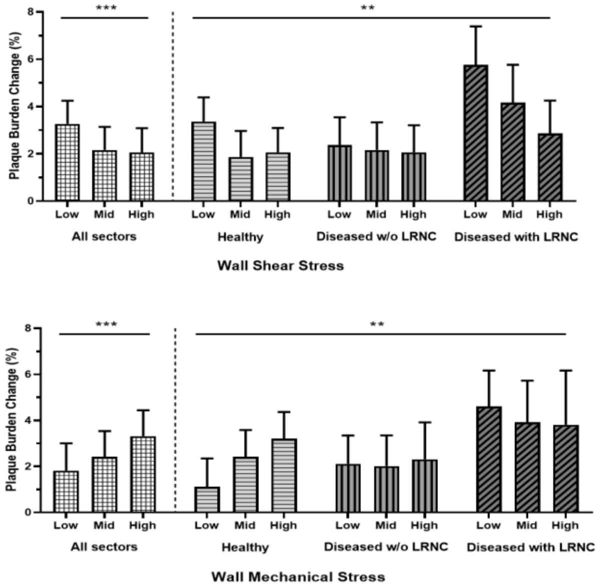


*Figure 1: Plaque burden change distribution based on WSS (top) and WMS (bottom)*

## Discussion

Our findings suggest that the local atherosclerotic disease progression in coronary arteries is not only associated with the well-established WSS, but also with WMS, which has not received enough attention yet. Moreover, our results show that the association of the disease progression with WMS depends on the existing local structural composition. Our findings indicate the importance of biomechanical analysis and local plaque composition assessment for local atherosclerotic disease progression prediction. In the future, studies with larger cohort and multiple follow-up time points are warranted in order to fully understand the association between shear and structural stress with atherosclerosis development over time.

## References

1. Shah, P et al., Thrombosis, 2015:634983, 2015.
2. Costopoulos, C et al., Eur Heart J., 40:1411-1422, 2019.
3. Hartman, E et al., J. of Cardiovasc. Trans. Res., 14:416–425, 2021.
4. Akyildiz, A C et al., Computer. Methods in Biomech. and Biomed. Eng., 19: 771–779, 2016.