# EXPLORING THE LINK BETWEEN WALL SHEAR STRESS TOPOLOGICAL SKELETON AND NEAR-WALL MASS TRANSPORT IN CARDIOVASCULAR FLOWS USING A EULERIAN-BASED METHOD

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## INTRODUCTION

Mass transport plays a key role in vascular disease. Several studies have suggested that, e.g., high plasma levels of low-density lipoproteins (LDL) are involved in the atherosclerosis process [1]. In this context, in the last decade, computational fluid dynamics (CFD) has been adopted to elucidate the links (if any) among disturbed shear, atherogenesis and mass transport in human arteries [2][3]. However, modelling mass transfer in cardiovascular flows requires the detailed, computationally expensive solution of the advection-diffusion equations [4]. To overcome this limitation, a marked interest has recently emerged on the Lagrangian-based features of the WSS topological skeleton, which have demonstrated to provide a reliable template for near-wall transport [5]. Briefly, the WSS topological skeleton is composed by fixed points, points where WSS vanishes, and stable/unstable connection lines (manifolds), identifying WSS expansion/contraction regions. Moving from the proven effectiveness of WSS Lagrangian Coherent Structures as a template of near-wall mass transport in cardiovascular flows, here a Eulerian-based method [6] recently proposed to analyze the WSS topological skeleton, whose features recently emerged as clear indicator of wall degradation in aortic aneurysm [7] and carotid restenosis risk [8], is applied. The final aim is to test its capability to provide a reliable template of the near-wall mass transport in patient-specific computational hemodynamic models of three distinct arterial district, i.e. the aorta, the carotid bifurcation and the right coronary artery.

## METHODS

A scheme of the methods applied in this study is provided in Fig. 1. The 3D geometries of a human thoracic aorta, a carotid bifurcation, and a right coronary artery were reconstructed from medical images as extensively described elsewhere [4,9,10].
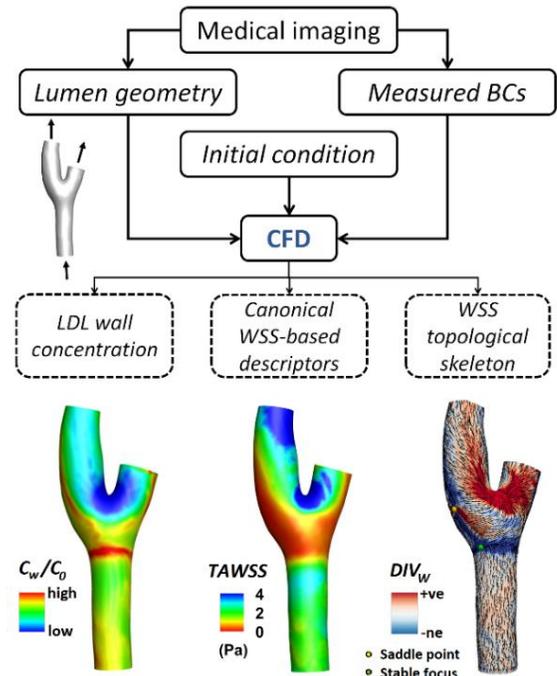


**Figure 1: Overview of the proposed approach.**

**Computational hemodynamics.** To model the transport of low-density lipoprotein (LDL), the finite volume method was applied to solve the coupled Navier-Stokes (NS) and advection-diffusion (AD) equations in their discretized form, using Fluent (ANSYS Inc., USA) on computational mesh-grids with 30 boundary layers of high-quality

prismatic cells near the wall [4]. Subject-specific boundary conditions (BCs) were prescribed to solve the NS equations [4,9,10]. A constant LDL concentration $C_0$ (equal to the average LDL concentration in whole blood) was applied at the inflow section, and the stress-free condition at the outflow sections, to solve AD equation [4]. The LDL blood-to-wall transfer was modelled as follows:

$$C_W v_W - D_{LDL} \left.\frac{\partial C}{\partial n}\right|_W = 0 \qquad (1)$$

where $C_W$ is the LDL concentration at the vessel wall, $v_W$ the water filtration velocity through the wall, $D_{LDL}$ is the diffusivity of LDL in blood, $\frac{\partial C}{\partial n}$ is the concentration gradient normal to the wall. Details on the applied numerical schemes are extensively provided elsewhere [4,9,10].

**WSS topological skeleton.** A recently proposed Eulerian method to analyze the WSS topological skeleton [6] was here considered. Briefly, based on Volume Contraction theory, it was demonstrated that the divergence of the WSS unit vector field $\boldsymbol{\tau}_u$, defined as:

$$DIV_W = \nabla \cdot \left(\frac{\boldsymbol{\tau}}{\|\boldsymbol{\tau}\|_2}\right) = \nabla \cdot \boldsymbol{\tau}_u \qquad (2)$$

represents a template of the WSS vector field manifolds, identifying the WSS expansion/contraction regions on the arterial luminal surface. To complete the WSS topological skeleton analysis, the Poincarè index and the Jacobian analysis were carried out to identify and classify fixed points, according to the scheme proposed in [6]. Here, the Eulerian-based WSS topological skeleton analysis was applied to the cycle-average WSS vector field.

To complement the analysis, the luminal distribution of three well-established descriptors of flow disturbances, i.e., the time-average WSS (*TAWSS*), the oscillatory shear index (*OSI*), and the relative residence time (*RRT*), were also evaluated.

**Co-localization analysis.** The analysis of the co-localization between WSS manifolds as identified by $DIV_W$, and local LDL uptake, was carried out according to schemes proposed elsewhere [4]. Briefly, (1) the surface area (SA) exposed to normalized LDL concentration values ($C_W/C_0$) higher than the 90th percentile was quantified and denoted as LDL90, and (2) the SA exposed to $DIV_W$ values lower than 10th percentile was identified and denoted as DIV10. Similarly, luminal SAs exposed to altered hemodynamics were identified by the lower 10th percentile for *TAWSS* (TAWSS10), and upper 90th for *OSI* and *RRT* (OSI90 and RRT90, respectively), and their co-localization with LDL90 was investigated.

## RESULTS

The luminal surface distributions of $DIV_W$ and normalized LDL are provided in Fig. 2 for the three analyzed vascular districts (panel A and B, respectively). WSS contraction regions are coloured in blue (negative $DIV_W$), while WSS expansion regions are presented in red colour (positive $DIV_W$, Fig. 2-A). It clearly emerges by visual inspection that contraction regions of the WSS vector field co-localize with LDL concentration polarization on the vessels wall. The observed co-localization is evident: (1) at the inner wall of the aortic arch and in the descending aorta for the thoracic aorta; (2) in the proximal carotid sinus where the bulb expansion starts to occur, and at the outer wall of the internal and external carotid artery; (3) along the inner curvature of the right coronary artery. A direct co-localization analysis between LDL concentration polarization and WSS contraction regions is provided in Fig. 2-C, where LDL90 and DIV10 (black contour lines) luminal SAs are displayed. A marked spatial overlap between LDL90 and DIV10 emerges, with the contour lines of DIV10 mostly encasing luminal regions with high LDL luminal concentration. The co-localization of LDL polarization concentration with WSS contraction regions is less pronounced only locally in the proximal part of the right coronary artery. From a quantitative analysis, it emerged that WSS contraction

regions co-localize with high LDL concentration regions at least the 40% more than canonical WSS-based descriptors, with an increase from 20% to 35% (the 75% more) in the right coronary artery (Table 1).
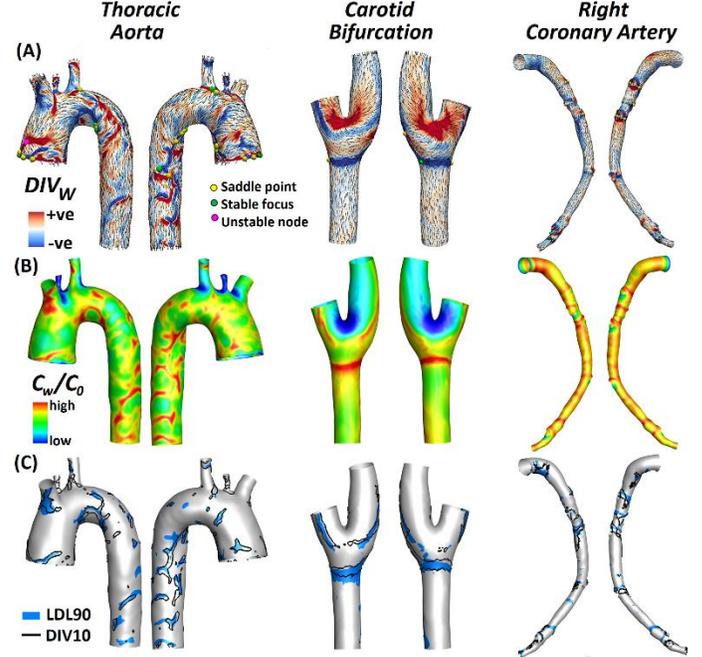


**Figure 2: (A) Cycle-average WSS topological skeleton. (B) LDL wall concentration. (C) Distributions of LDL90 an DIV10.**

**Table 1: Percentage overlap with LDL90**

|  | TAWSS10 | OSI90 | RRT90 | DIV10 |
|---|---|---|---|---|
| *Thoracic Aorta* | 33% | 27% | 34% | 46% |
| *Carotid Bifurcation* | 30% | 28% | 29% | 48% |
| *Right Coronary Artery* | 21% | 19% | 20% | 35% |

## DISCUSSION

The findings of this study: (1) confirm that WSS manifolds can be used as reliable templates of near-wall mass transport in cardiovascular flows [5]; (2) demonstrate that the recently proposed Eulerian-based method for analysing the WSS topological skeleton [6] efficiently provides a template of the LDL blood-to-wall transfer. The methodology, requiring less computational efforts with respect to a fully 3D simulation of mass transport in cardiovascular flows, candidates as an effective tool enriching the analysis of cardiovascular flows, avoiding solving AD equation to identify near-wall regions where mass transport is more pronounced [4]. Moreover, the co-localization of the LDL luminal polarization distribution with the WSS vector field contraction regions is higher than canonical WSS-based descriptors of flow disturbances.

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