

Blood Flow Simulation in an Atherosclerotic Coronary Artery

Manuel Salinas¹ and Joshua Bennett²

¹ Nova Southeastern University

Received: 9 December 2019 Accepted: 2 January 2020 Published: 15 January 2020

Abstract

Introduction: Atherosclerosis highly linked to heart attacks and strokes which are two leading cause of death in the United States according to the Center of Disease Control and Prevention (CDC). The increasing severity of atherosclerosis comes from the instance that symptoms usually appear at late stages of the disease. In addition, the mechanisms by which atherosclerosis forms, develops, and triggers the onset of myocardial infarction is not fully understood. In this study, we simulated the effect that atheromas have on the blood flow physics and the correlation that these flow disturbances may have on the onset of myocardial infarction. **Materials and Methods:** A 3D virtual geometry of an idealized coronary artery with a hemispherical obstruction was created using human anatomical dimensions. All Ansys simulations performed in this study used laminar flow conditions with density = 1060 kg/m³, viscosity = 3.5 centipoise. We applied a physiological velocity waveform at the inlet and a zero relative-pressure condition at the outlet. No slip boundary conditions were prescribed to the coronary artery walls. **Materials and Methods:** A 3D virtual geometry of an idealized coronary artery with a hemispherical obstruction was created using human anatomical dimensions. All Ansys simulations performed in this study used laminar flow conditions with density = 1060 kg/m³, viscosity = 3.5 centipoise. We applied a physiological velocity waveform at the inlet and a zero relative-pressure condition at the outlet. No slip boundary conditions were prescribed to the coronary artery walls.

Index terms— atherosclerosis; coronary artery disease; vascular erosion; computational fluid dynamics; heart attack; stroke.

1 Introduction

Atherosclerosis is highly linked to heart attacks and strokes which are two leading cause of death and morbidity worldwide [1][2][3] [4] [5] [6] [7] [8] [9] [10] [11] [12] [13] [14] [15] [16] [17] . In the United States alone, 8.917 million people worldwide died in 2015 due to atherosclerosis complications. The annual burden on the United States Healthcare system is estimated to be \$351.2 billion in 2014-2015, with \$213.8 billion in direct costs [18] . There is no cure for atherosclerosis and the underlying mechanisms that cause it have not been fully and exhaustibly delineated [1, [19], [20], [21]] .

There are numerous studies dedicated to understanding how atherosclerosis develops. Some studies state that atherosclerosis is correlated to the infiltration of the artery by lipids and proteins [19] . Efforts elsewhere state that viral infections may play a role in the activation of atherosclerosis [21, 22] . Other experiments suggest that blood flow disturbance aids in the onset and progress of the disease [10, 21, 23] .

Choi et al. [24] screened 217 patients who each had a chronic coronary total occlusion (CTO) due to atheromas. They reported varying degrees of myocardial scarring in most myocardial tissue downstream from the atheroma. In another study by Franck et al. [21] , a cuff was used to constrict a rat's aorta. They also reported increased number of inflammatory cells and vessel erosion at zones of oscillatory shear stress.

43 A 3D virtual geometry of an idealized coronary artery with hemispherical atheroma was created using Ansys
44 Design modeler as depicted in Fig. 1 (Ansys Inc., Canonsburg, The vessel diameter equaled 3.1 mm with a length
45 of 30 mm and a hemispherical obstruction of radius 1.55 mm centered 10 mm from the inlet. A standard mesh
46 with 77,220 elements was constructed. Computational fluid dynamics (CFD) simulations were conducted in CFX
47 (Ansys Inc., Canonsburg, PA). All simulations performed used constants and methods previously validated in
48 other studies [25][26][27][28] . Briefly, we prescribed laminar flow conditions with density = 1060 kg/m³ and
49 viscosity = 3.5 centipoise. A zero relative pressure condition was also applied at the outlet. At the

50 2 II. Mesh Construction, Simulation

51 Set-up Despite these and many other studies, the underlying mechanisms by which atheromas contribute to
52 the onset of heart attacks is not well understood and pose a major challenge to the cardiovascular scientific
53 community [21] . In this study, we investigated the effect that atheromas have on the flow physics of blood and
54 the possible correlation that these flow disturbances may have on the onset of myocardial infarction.

55 Abstract-Introduction: Atherosclerosis is highly linked to heart attacks and strokes which are two leading
56 cause of death in the United States according to the Center of Disease Control and Prevention (CDC). The
57 increasing severity of atherosclerosis comes from the instance that symptoms usually appear at late stages of
58 the disease. In addition, the mechanisms by which atherosclerosis forms, develops, and triggers the onset of
59 myocardial infarction is not fully understood. In this study, we simulated the effect that atheromas have on
60 the blood flow physics and the correlation that these flow disturbances may have on the onset of myocardial
61 infarction.

62 Materials and Methods: A 3D virtual geometry of an idealized coronary artery with a hemispherical obstruction
63 was created using human anatomical dimensions. All Ansys simulations performed in this study used laminar flow
64 conditions with density = 1060 kg/m³, viscosity = 3.5 centipoise. We applied a physiological velocity waveform
65 at the inlet and a zero relative-pressure condition at the outlet. No slip boundary conditions were prescribed to
66 the coronary artery walls.

67 Results and Discussion: We observed that the obstruction in the blood flow caused severe flow disturbance
68 downstream from the atheroma. These time dependent cyclical flow profiles cause oscillatory velocities, pressures
69 and shear stresses. These flow alterations have been linked to vessel erosion and may be a key factor on the onset
70 of heart attacks.

71 Conclusions: In this study, we coupled an anatomically relevant time dependent velocity waveform with a
72 segment of a coronary artery blocked by an atheroma. We have demonstrated that coronary arteries afflicted
73 with atherosclerosis causes recirculation areas immediately downstream from the occlusion which are areas linked
74 to vessel erosion and thrombus formation. Our focus in our future work will be to incorporate vessel elasticity
75 and movement. In addition, we hope to be able to correlate our results with tissue culture and small animal
76 studies.

77 3 Keywords: atherosclerosis; coronary artery disease; vascular 78 erosion

79 4 Results and Discussion

80 Fig. ?? : Contour of velocity magnitudes at specified time intervals along a longitudinal central cross-sectional
81 plane. Notice the higher velocities at the apex of the occlusion, and lower velocities near the vessel wall.

82 The velocity magnitudes of blood flow were plotted on a longitudinal cross-sectional plane down the geometry's
83 center as seen in Fig. ?? . In the obstructed and unobstructed regions of the coronary artery, velocity of blood
84 decreased near the vessel walls. Also, fluid velocity increased at the apex of the occlusion. The maximum velocity
85 occurred at 0.16 seconds.

86 A longitudinal cross-sectional plane at the same location was used to measure flow pressure as seen in Fig. 3.
87 Highest blood pressure surrounding the obstruction was reported when velocity was at its maximum corresponding
88 to a time of 0.14 seconds. On the other hand, the lowest pressure corresponded to times of 0.16 seconds in the
89 regions next to the occlusion.

90 Axial velocity vectors were plotted on the same plane as velocity as seen in Fig. ?? . Recirculation was
91 observed downstream from the occlusion particularly at 1 second. This results agree with findings in other
92 studies that suggest atheromas cause abnormal blood flow [20,21,30] . These findings also agree with larger
93 scale animal models developed by Frank et al. [20] where they reported an increase flow disturbance activity
94 downstream from an artificial occlusion in a rat artery and reported increased number of inflammatory cells and
95 vessel erosion. IV. One of the principle achievements of this paper is the coupling of an anatomically relevant time
96 dependent velocity waveform with a segment of a coronary artery blocked by an atheroma. We have demonstrated
97 that coronary artery afflicted with atherosclerosis causes recirculation areas immediately downstream from the
98 occlusion. Our focus in our future work will be to incorporate vessel elasticity and movement. In addition, we
99 hope to be able to correlate our results with tissue culture and small animal studies.

100 5 Conclusion

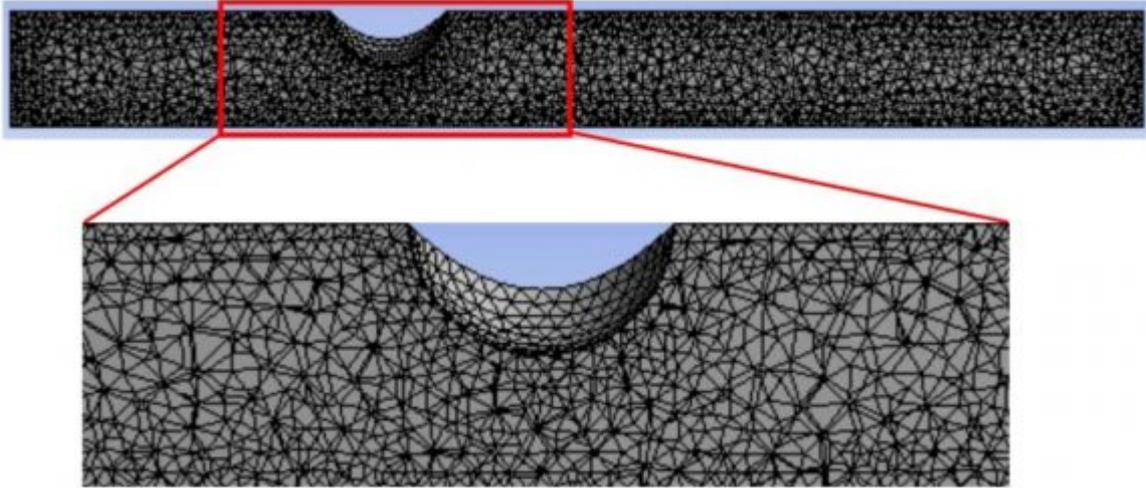


Figure 1:

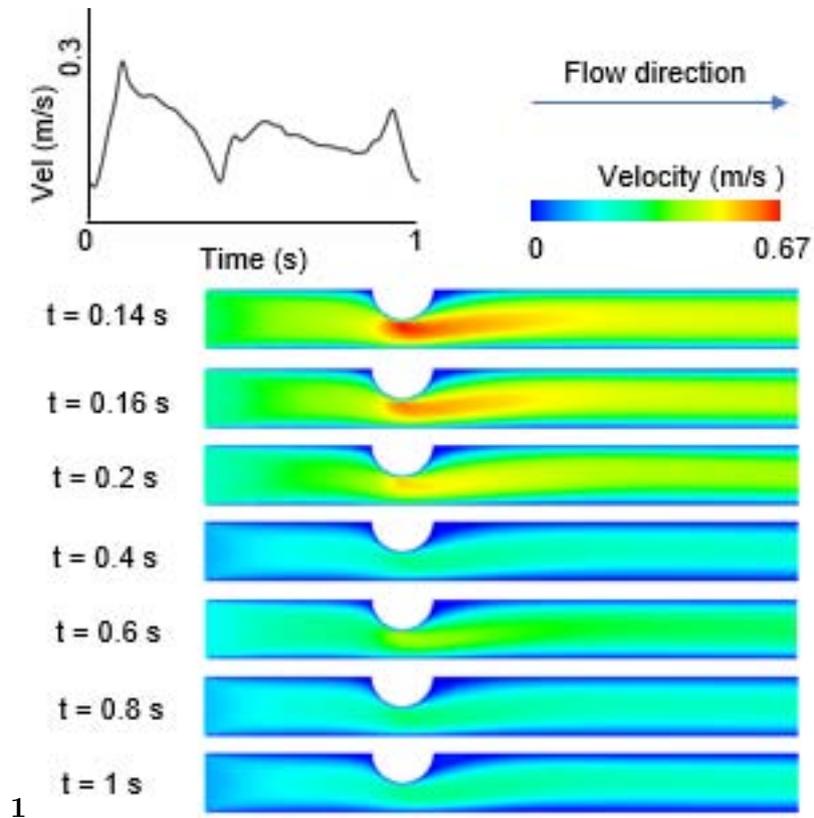
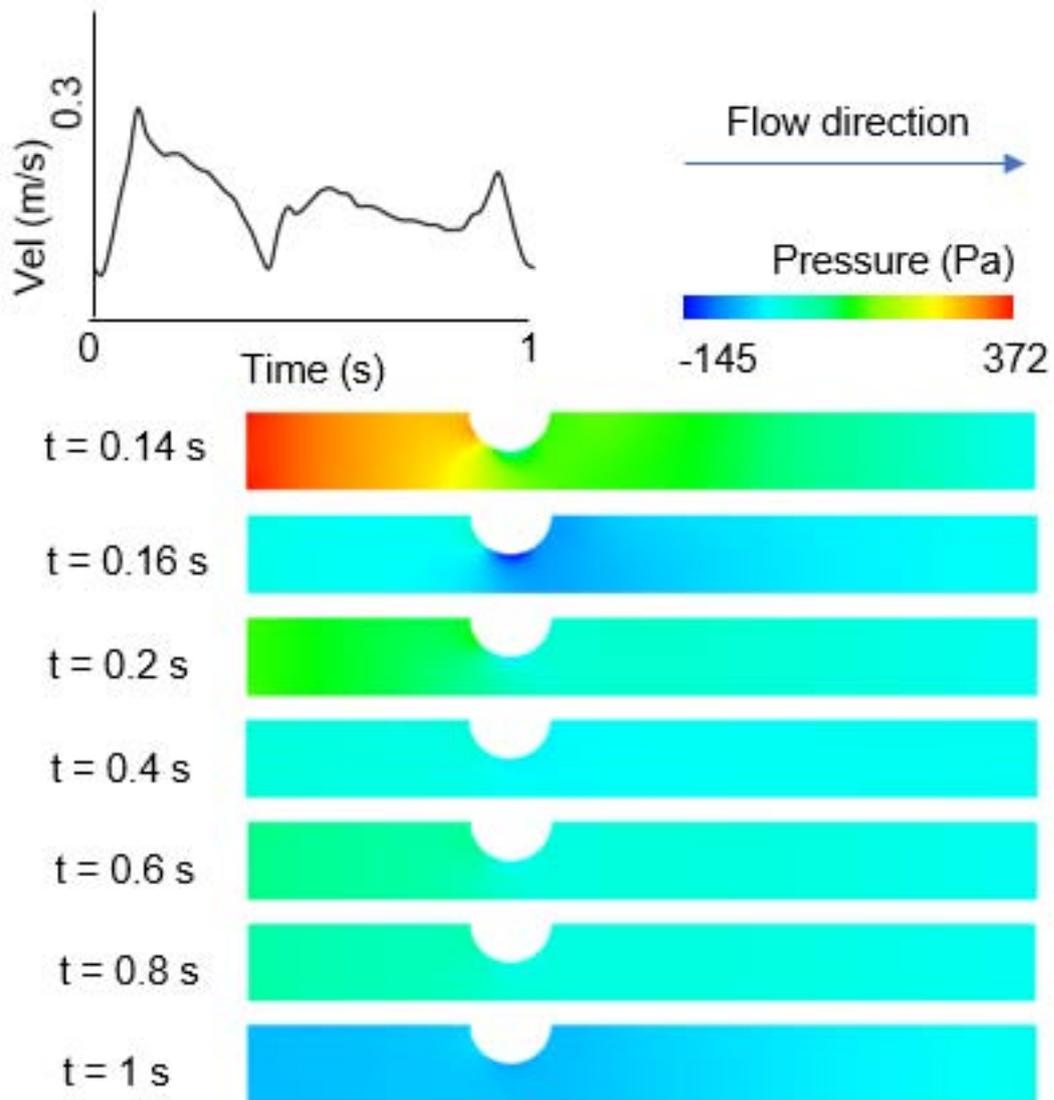


Figure 2: Fig. 1 :



3

Figure 3: Fig. 3 :

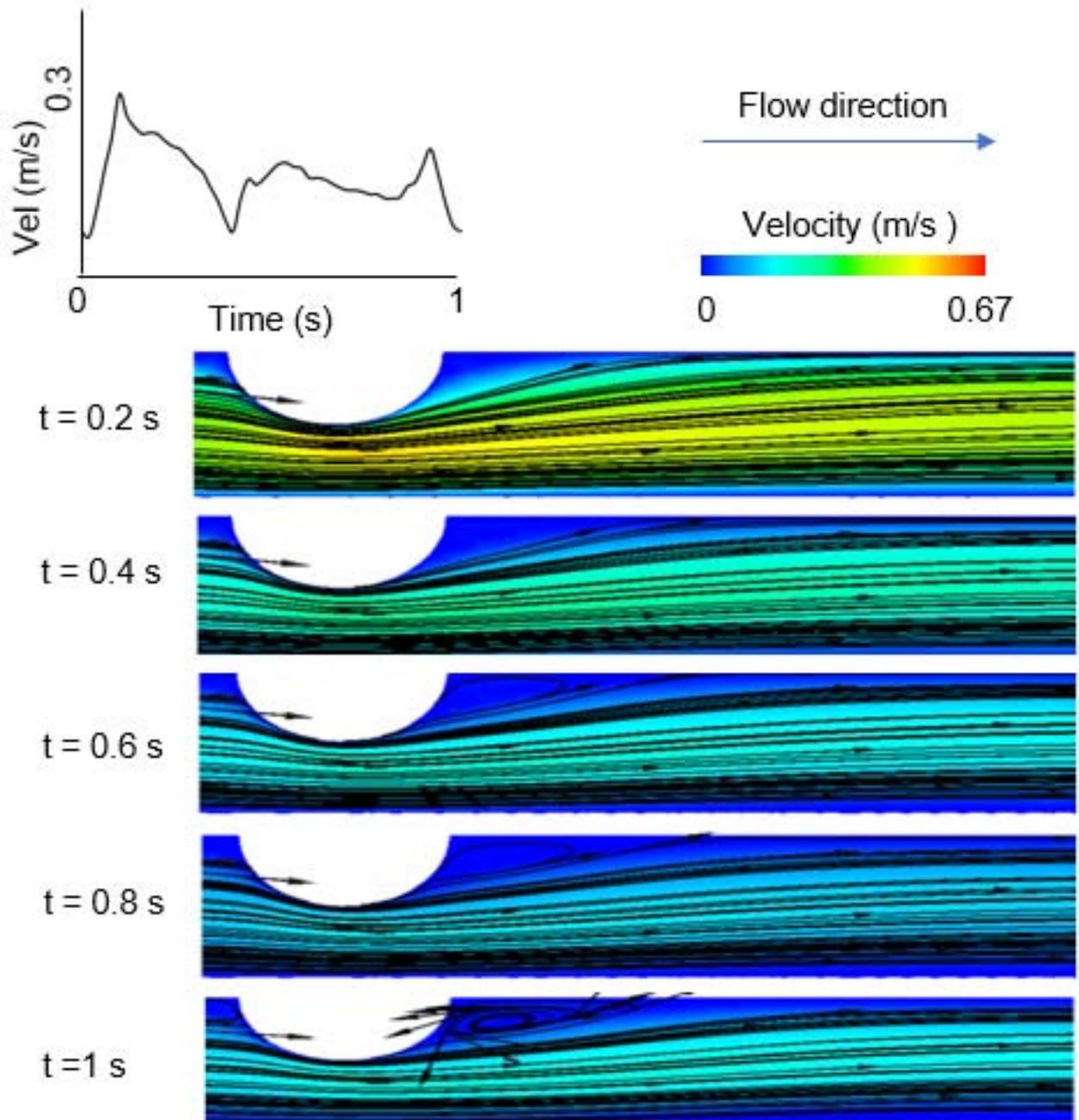


Figure 4:

.1 Acknowledgements

We would like to thank Dr. Alexander Soloviev for sharing Ansys with us.

[Nahrendorf et al. ()] ‘18F-4V for PET-CT imaging of VCAM-1 expression in atherosclerosis’. M Nahrendorf , E Kelihier , P Panizzi . *JACC: Cardiovascular Imaging* 2009. 2 (10) p. .

[Williams et al. ()] ‘A ”sweet-spot” for fluidinduced oscillations in the conditioning of stem cellbased engineered heart valve tissues’. A Williams , S Nasim , M Salinas , A Moshkforoush , N Tsoukias , S Ramaswamy . *J Biomech* 2017. 65 p. .

[Ramaswamy et al. ()] ‘A novel bioreactor for mechanobiological studies of engineered heart valve tissue formation under pulmonary arterial physiological flow conditions’. S Ramaswamy , S Boronyak , T Le , F Sotiropoulos , A Holmes , M S Sacks . *J Biomech Eng* 2014. 136 (12) .

[Engelmayr et al. ()] ‘A novel bioreactor for the dynamic flexural stimulation of tissue engineered heart valve biomaterials’. G C Engelmayr , D K Hildebranda , F W Sutherland , J E Mayer , M S Sacksa . *Biomaterials* 2003. 24 (14) p. .

[Butcher et al. ()] ‘Aortic valve disease and treatment: The need for naturally engineered solutions’. J T Butcher , G J Mahler , L A Hockaday . *Adv Drug Deliv Rev* 2011. 63 (4-5) p. .

[Yang et al. ()] ‘Assessing potential population impact of statin treatment for primary prevention of atherosclerotic cardiovascular diseases in the USA: Population-based modelling atherosclerosis: Roles in lipid permeability, lipid retention, and smooth muscle cell proliferation’. Q Yang , Y Zhong , C Gillespie . *Circ Res* 2008. 103 (1) p. .

[Aikawa and Schoen (ed.)] *Cellular and molecular pathobiology of cardiovascular disease*, E Aikawa , F J Schoen . 10.1016/B978-0-12-405206-2.00009-0. <http://dx.doi.org.ezproxy.fiu> Stone MSWWHR (ed.) San Diego: Academic Press. 2014 p. . (Chapter 9 -calcific and degenerative heart valve disease)

[Sun et al. (ed.) ()] *Computational modelling of mass transport in large arteries*, N Sun , N B Wood , X Y Xu . 10.5772/5965.10.5772/5965. <https://doi.org/10.5772/5965.10.5772/5965> Giuseppe Petrone, Giuliano Cammarata (ed.) 2008. (Modelling and simulation. Rijeka: IntechOpen)

[Franck et al. ()] ‘Flow perturbation mediates neutrophil recruitment and potentiates endothelial injury via TLR2 in mice: Implications for superficial erosion’. G Franck , T Mawson , G Sausen . *Circ Res* 2017. 121 (1) p. .

[Choi et al. ()] ‘Frequency of myocardial infarction and its relationship to angiographic collateral flow in territories supplied by chronically occluded coronary arteries’. J H Choi , S A Chang , J O Choi . *Circulation* 2013. 127 (6) p. .

[Franck et al. ()] ‘Haemodynamic stress-induced breaches of the arterial intima trigger inflammation and drive atherogenesis’. G Franck , G Even , A Gautier . *Eur Heart J* 2019. 40 (11) p. .

[Benjamin et al. ()] ‘Heart disease and stroke statistics-2019 update: A report from the american heart association’. E J Benjamin , P Muntner , A Alonso . *Circulation* 2019. 139 (10) p. .

[Visser and Vercellotti ()] ‘Herpes simplex virus and atherosclerosis’. M R Visser , G M Vercellotti . *Eur Heart J* 1993. 14 p. . (Suppl)

[Quillard et al. ()] ‘Mechanisms of erosion of atherosclerotic plaques’. T Quillard , G Franck , T Mawson , E Folco , P Libby . *Curr Opin Lipidol* 2017. 28 (5) p. .

[Salinas et al. ()] ‘Oscillatory shear stress created by fluid pulsatility versus flexed specimen configurations’. M Salinas , D E Schmidt , M Libera , R R Lange , S Ramaswamy . *Comput Methods Biomech Biomed Engin* 2012.

[Fukuda et al. ()] ‘The role of Dll4-notch signaling in shared mechanisms for atherosclerosis and metabolic disorders’. D Fukuda , T Miyazaki , K Morishige , E Aikawa , M Aikawa . *Cardiovascular Pathology* 2013.