Obstacle-influenced particulate deposition concentrations in wall-bounded flow

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1 Abstract

This study will contribute to the better understanding of field distributions of LDL particulate deposition around atherosclerotic lesions.
Particle-laden flows are found in many phenomena, both industrial and natural. Of interest to this project is the phenomenon associated with low density lipoprotein particulate transport in blood flow; particularly as influenced by an obstacle such as an atherosclerotic plaque or thrombus.
The particle-laden flow's behaviour will be modelled by coupling the governing Navier-Stokes fluid flow equations with particle-concentration Transport equations, another word convection-diffusion equation. These will then be solved on a computational grid, with the appropriate boundary conditions applied.
The project will comprise of firstly understanding the physics of the problem, then developing the model, and finally developing a parametric study of the underlying variables in the system.
The methodology and general result outcomes may also be employed in the assessment of platelet transport and deposition, as applied to the formation of thrombi in arteries.

2 Introduction

Atherosclerosis is a common form of cardiovascular disease that primarily affects large and medium sized arteries and is the underlying cause of most heart attacks and strokes. Fat, cholesterol, calcium, and other substances form plaque. It has been observed that the cause of atherosclerosis may be due to high concentrations of substances such as

low-density lipoproteins in the artery wall. [1–3] which is the leading cause of death in people over 45 and places a significant financial burden on health care systems. There is evidence that the complex arterial mass transport processes particularly in deposition of highly atherogenic LDL within the arterial wall, play a key role in the development of atherosclerosis.

Also it is observed that the presence of an obstacle in the flow would influence the behaviour of the local viscous fluid. Then it follows that there would be a variation of the local wall shear stress, and hence a consequential variation in deposited particle distribution. Since the particles of interest in this study are the highly atherogenic low-density lipoproteins, it therefore follows that a local increase in their concentration at the obstacle site will greatly influence either the initiation of an atherosclerotic lesion or the subsequent growth of an existing one.

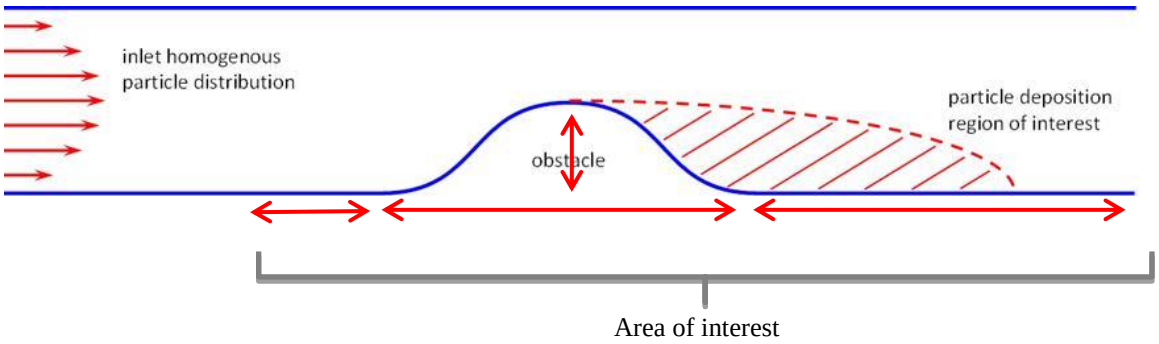

Figure 1. The region of interest of particle deposition within arterial wall.

As depicted in the above figure, from a homogenous inlet particle distribution, it is expected that the variation of wall shear stress caused by the obstacle will cause a subsequent variation in particulate concentrations. Therefore, the size of the obstacle becomes an essential parameter in the mechanics of the system, and thus will be assessed parametrically in this study.

3 Method

3.1 Mathematical Model

Arterial mass transport is coupled with both the bulk blood flow in the lumen and the blood flow in the wall[4]. Therefore our model includes the moving fluid, *Dynamic Fluid* which is the motion of the blood in lumen and also absorption process inside the arterial wall, which is *Solute Dynamic*. The solutes are convected by the blood along the walls.

So in this study our model is concentrating on the flow of fluid within the lumen and through the arterial wall (dynamic fluid) and also deposition of LDL in arterial wall (solute dynamic).

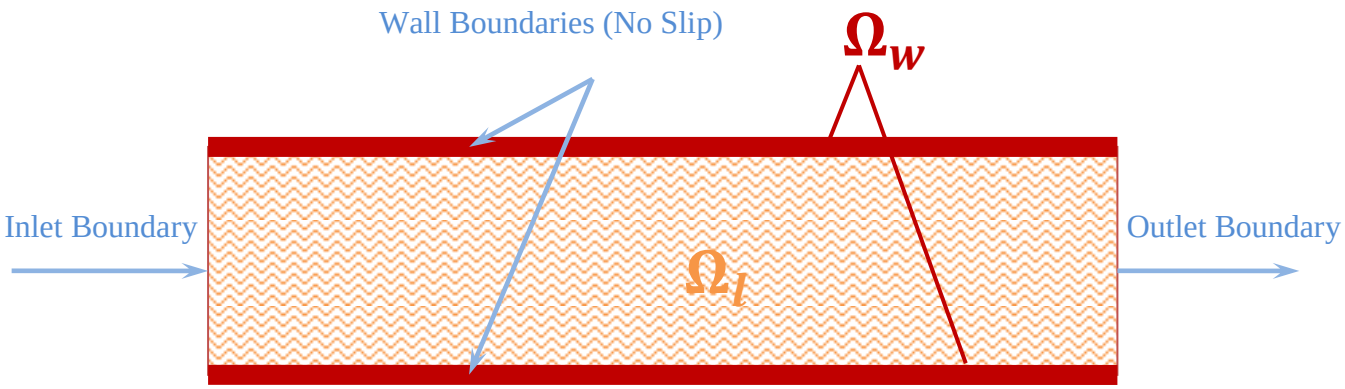

Figure 2. Domains and Boundaries of the single-layered 2D model.

Considering our 2D model which makes the mathematical process to be easier, we use Navier-Stokes equations to model bulk blood flow in the lumen (Ω_l), and Darcy's Law for blood flow in the arterial wall (Ω_w). Also we use the Convection-Diffusion equation in the lumen (Ω_l).

3.2 The Physics of Blood Flow

Blood fluid has many important physical properties. In our study the blood flow consider to be incompressible, with a Newtonian behaviour as its density is $\rho = 1.06 \times 10^3$ kg/m3 and constant. Also blood flow has a low velocity and all particles moving in straight lines parallel to the arterial wall with no disruption between the layer which makes a laminar flow.
The viscosity of blood at 37 ° C is $\mu = 3.5 \times 10^{-3}$ s.Pa which is the resistance to flow and makes a Newtonian behavioural for the blood flow.
These considerations of blood properties help us to have an easy computational process.

3.3 Governing Equation

3.3.1 Blood flow in the lumen and along the arterial wall

We have governed these equations based on the physical properties of blood flow as above description therefore the Navier-Stokes equations [5] hold as:

Momentum equation in blood lumen:

$$\rho\left(\frac{\partial U_l}{\partial t} + U_l.\nabla U_l\right) = -\nabla p_l + \mu.\nabla^2 U_l)$$

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$$\underbrace{\rho \frac{\partial U_l}{\partial t}}_{\text{I}} + \underbrace{\rho\, U_l . \nabla U}_{\text{II}} = \underbrace{-\nabla p_l}_{\text{III}} + \underbrace{\mu . \nabla^2 U_l}_{\text{IV}} \quad (1)$$

I. Local change with time
II. Momentum convection
III. Pressure Gradient
IV. Molecular-dependent momentum exchange (diffusion) – Viscose Terms

In the fluid domain, where U_l is blood velocity in the lumen, p_l is pressure, μ is dynamic viscosity of the blood, and ρ is density of the blood.

Continuity equation in blood lumen:

$$\frac{D\rho}{Dt} + \rho\, . \nabla U_l = 0 \quad (2)$$

Incompressible fluid: $\nabla U_l = 0$

The flow in the arterial wall was modelled by Darcy's Law:

$$U_w - \nabla(\frac{K_w}{\mu}\, p_w) = 0 \quad (3)$$

$$\nabla U_w = 0 \quad (4)$$

In the wall, where U_w is the velocity of the flow in the wall, p_w is pressure in the wall, μ is viscosity of the blood, and K_w is the permeability coefficient of the wall.

To simplify and reduce the variables as shown in Figure 2 we are constructing our model in 2D with a steady state condition as velocity does not change over the time. Therefore the velocity vector $U(u, v)$ in our 2D Cartesian coordinate (x, y)will simplify all the equations above as follows:

Note: y in Cartesian coordinate system denoted by r which is the radius of the artery.

Momentum equation:

$$\rho \left(u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y}\right) = \frac{-dp}{dx} + \rho v \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) \quad (5)$$

$$\rho \left(u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y}\right) = \frac{-dp}{dy} + \rho v \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\right) \quad (6)$$

Continuity equation:

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0 \tag{7}$$

Darcy's law:

$$u = \frac{K_w}{\mu} \cdot \frac{dp}{dx} \tag{8}$$

$$v = \frac{K_w}{\mu} \cdot \frac{dp}{dy} \tag{9}$$

Therefore, the velocity in the wall is proportional to pressure gradient.

3.3.2 LDL deposition within arterial Wall

LDL transfer in the blood lumen was modelled by the Transport equation [4].

$$\frac{\partial C}{\partial t} + u \frac{\partial C}{\partial x} = D \frac{\partial^2 C}{\partial x^2}$$

Considering steady state condition,
Transport equation:

$$u \frac{\partial C}{\partial x} = D \frac{\partial^2 C}{\partial x^2} \tag{10}$$

Where C is concentration of LDL and D is the diffusivity of LDL and the velocity u is the convective term solved by Navier-Stokes equation.

As LDLs are such a small spherical molecules with a very small diameters of 21-26 nm and density of 1.063*10-3 kg/m3 transported both *passively* which LDLs are carried by the flowing blood itself and also *voluntarily* which they carried by molecular diffusion.[6]

3.4 Boundary Conditions

For the blood flow, a constant pressure and a given velocity at the inlet of the lumen is placed. A zero velocity normal to the side boundaries is placed as a result of no slip condition. Also an average static pressure is given at the outer wall boundary [7].

Inlet Boundary

$$P = P_0 \ , \qquad u = 2\,u_0 \left[1 - \left(\frac{r}{R_0}\right)^2\right] \qquad \text{at} \qquad x = 0$$

u_0 Mean velocity of blood at the inlet
r Reference distance from the central axis of the vessel
R_0 Radius of the vessel at the inlet.

Wall Boundary $u = 0$ at R= r

Outlet Boundary

$$P_{spec} = \frac{1}{A} \int P_{ip}\, dA$$

P_{ip} Imposed pressure at the integration point
A Whole area
P_{spec} Constant pressure over the whole outlet

For the LDL deposition and its related Transport equation (10), a flat concentration is set at the inlet, the concentration at the wall is a proportion of the change of flux and a zero flux condition is imposed at the outlet. [7]

Inlet Boundary

$$\boldsymbol{C = C_0} \quad \text{at} \quad n = 0$$

C_0 Concentration at the inlet
n Unit vector normal to the surface

Wall Boundary

$$-\boldsymbol{D}\,\frac{\partial \boldsymbol{C}}{\partial \boldsymbol{n}} = \left(\boldsymbol{K} - \boldsymbol{V_w}\right)\boldsymbol{C_w} \quad \text{at} \quad r = R$$

C_w Wall concentration
V_w Water filtration velocity $4 \times 10^{-08}\ m/s$
K LDL permeability $2 \times 10^{-10}\ m/s$
D LDL diffusivity $5 \times 10^{-12}\ m^2/s$

Outlet Boundary

$$\frac{\partial \boldsymbol{C}}{\partial \boldsymbol{n}} = \boldsymbol{0} \quad \text{at} \quad l = L$$

n Unit vector normal to the surface

3.5 Governing Non-Dimensional Equations and Parameters Value

We use nondimensionalisation method to convert the Transport equation to a easier form to use with just one non dimensional parameter, Peclet number (Pe)[8].

$$\frac{\partial C_i}{\partial t} + u\,\frac{\partial C_i}{\partial x} = D_i\,\frac{\partial^2 C_i}{\partial x^2}$$

Non-dimensionalise:

$$\frac{\partial C_i^{*}}{\partial t^{*}} + \vec{u}^{*}\,\frac{\partial C_i^{*}}{\partial x^{*}} = D_i\,\frac{\partial^2 C_i^{*}}{\partial x^{2*}}$$

$$\mathrm{Pe} = \frac{\mathrm{u}\,.\,\mathrm{l}}{\mathrm{D}}$$

Where l is the characteristic length, u the velocity, D the mass diffusion coefficient.

We can use the Peclet number and apply the non-dimensionalisation to Naiver-Stokes equation, therefore having another non dimensional parameter, Reynolds number (Re).

$$\mathrm{Re} = \frac{\rho\,.\,\mathrm{u}.\,\mathrm{l}}{\mu} = \frac{\mathrm{u}\,.\,\mathrm{l}}{\frac{\mu}{\rho}}$$

Where, l is the characteristic length, u the velocity and $\mathrm{v} = \frac{\mu}{\rho}$ is the kinematic viscosity.

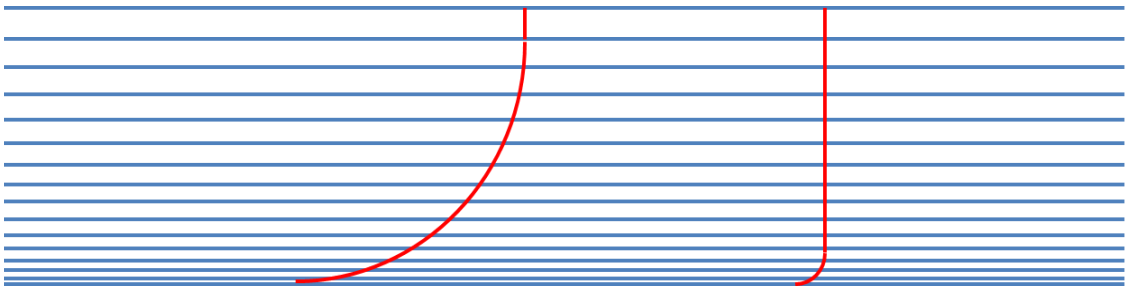
Now we can govern our non-dimensional equations with these two parameters and it is clear that our flow is Peclet number dependant.

4 Results

By governing those non-dimensional equations and solving the coupled equations for physiological conditions:

- we have found that large peclet numbers are very characteristic
- we have a small mass boundary layer

Therefore high density surface grid is required for capturing large gradients.



velocity boundary layer >> mass boundary layer

Figure 3. High density surface and large gradients

Having these conditions and in order to more accurately model, we varied each of these variables; filtration velocity (V_w), freestream velocity (U) and diffusion coefficient (D) and noted the changes of mass boundary layer in Height and Concentration plan.

We compared the results of each variation over the plan while $y\ axis$ is distance from the wall and $x\ axis$ is concentration surface.

First we varied the filtration velocity V_w while had D and U as constants:

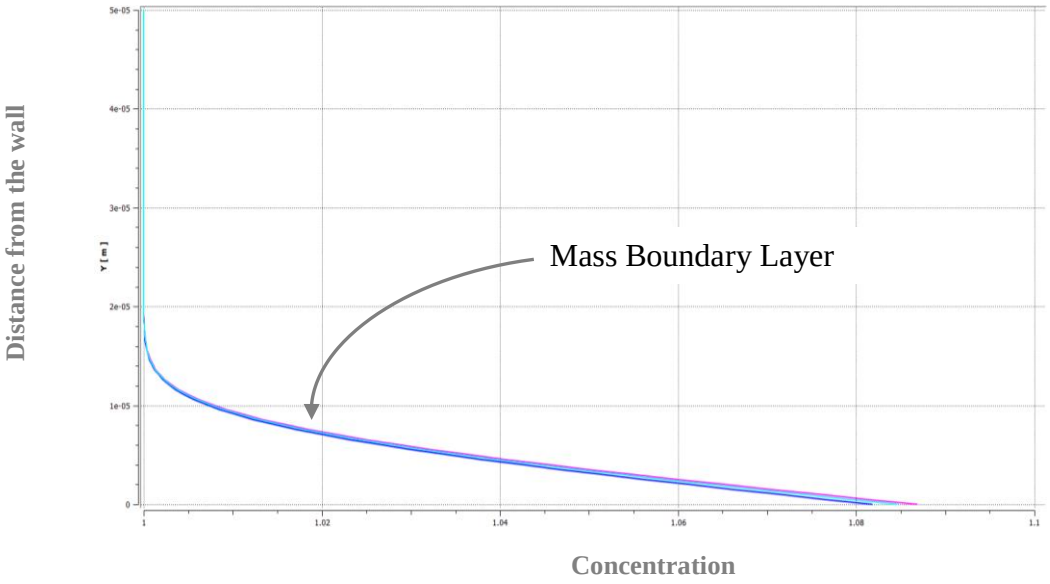


Figure 4. Mass boundary layer while filtration velocity is varied .

As we can see changing filtration velocity at the wall has very small effect on the mass boundary layer therefore the surface concentration.

Then we varied the freestream velocity $\boldsymbol{U}$ and had $\boldsymbol{D}$ and $\boldsymbol{V_w}$ are constant:

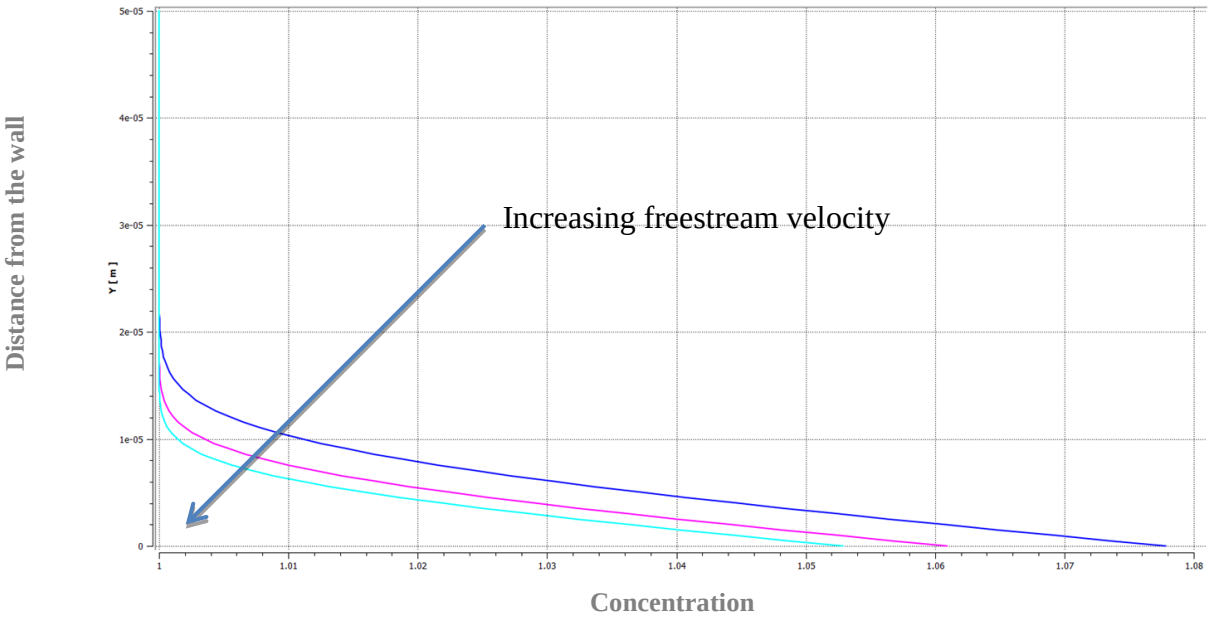


Figure 5. Mass boundary layer while freestream velocity is varied.

As it shows in the above graph by increasing $\boldsymbol{U}$, the boundary of height is decreasing and also surface concentration is decreasing too.

And finally we varied diffusion coefficient $\boldsymbol{D}$ and keep $\boldsymbol{U}$ and $\boldsymbol{V_w}$ are constant:

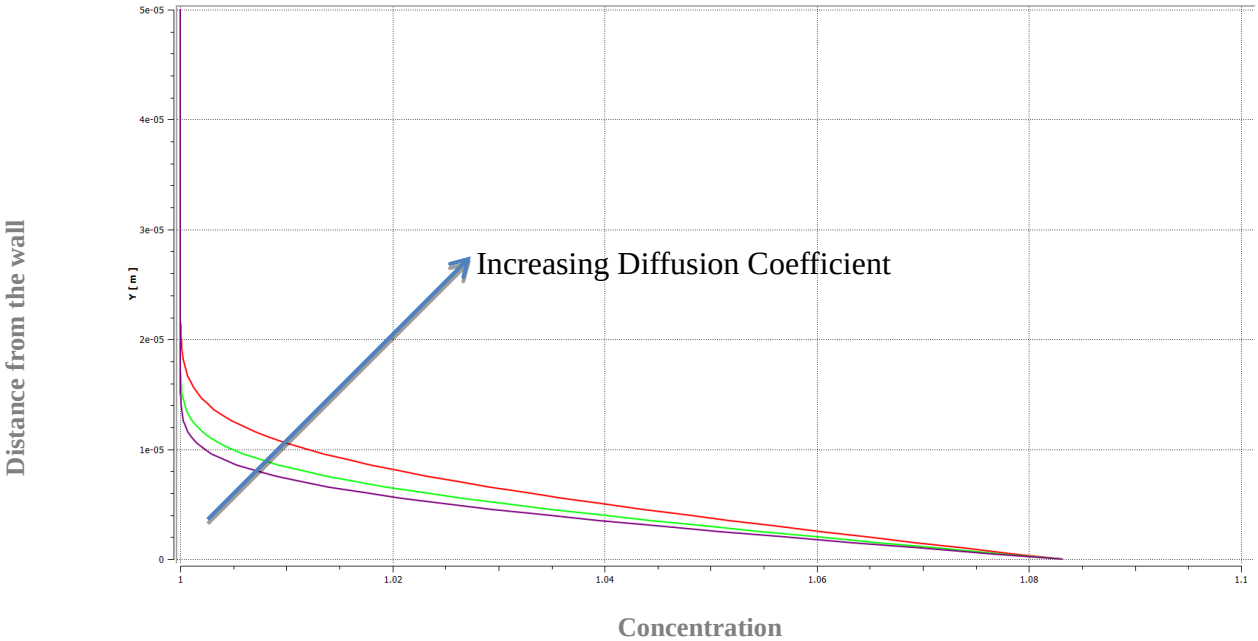


Figure 6. Mass boundary layer while diffusion coefficient is varied.

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It is shown that by increasing diffusion coefficient, the boundary layer of height is increased too but it does not affect the surface of concentration.

Naiver-Stokes equation and Transport equation as described above were solved using a finite volume method.
It seems the problem is most strongly affected by free stream velocity **U** and diffusion coefficient ***D***.

5 Discussion

This study shows that: a decrease in free stream velocity in the plaque region will increase the mass boundary layer and hence further elevate the LDL concentration within endothelium. Our results suggest a complex between velocity of flow, LDL diffusivity and LDL concentration at the wall.
This study has observed mechanisms of LDL deposition in the artery wall within specific condition of blood flow and is not proven.

6 Conclusion

In the present study, a mathematical model was developed to simulate the distributions of LDL particulate deposition around atherosclerotic lesions using Fluent CFD to integrate steady state transport processes in the endothelium and arterial wall. Also Finite Volume Method is placed as the method of numerical procedures solution.
The mathematical model and parameters were tested in idealised arterial geometry (2D). The results of the numerical solution show that, the surface concentration has no change due to increasing diffusion coefficient and has a very small effect by change of filtration velocity at the wall.
But the concentration is increasing noticeably due to decreasing velocity.

Previous computational studies in arterial mass transport have revealed that assumptions made regarding the behaviour of the fluid and species at the wall-bounded flow interface have a significant impact on the resulting interface concentration profiles [9].

As per this study, concentration of LDL in the arterial wall expects to be altered by changing velocity of blood flow.
At the present time this must be considered as an interesting theoretical finding. If this study was confirmed by experimental results, it would give us opportunities for more results regarding contribution of LDL deposition around the neighbourhood of plague.

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