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Impact of *Staphylococcus aureus* Shape and Dimensions on Hemodynamics

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Abstract

One mostly different from other causes of blood and device infections is *Staphylococcus aureus*, which has surface adhesiveness and biofilms forming ability. Using CFD, we investigate in silico the consequences of changes in bacterial size and form on blood flow. Included for simulations to study several parameters wall shear stress (WSS), velocity distribution and vorticity are the blood that is non-Newtonian and different geometries of *S. aureus*. Long forms create strong perturbances in the flow field and can lead to the rise of the flow resistance as well as in the situations of vessel wall obstruction. With much high prediction power on several data sets, neural network analysis successfully confirms the reported association between bacterial form and flow disturbance. These results add fresh understanding of *S.aureus* architecture and haemodynamics, which might guide novel approaches of diagnosis and more effective treatment.

Keywords: Bacterial Morphology, Biofilm Formation, Hemodynamics, Neural Network, Sepsis, *Staphylococcus aureus*.

Introduction

Staphylococcus aureus is a pathogen that causes persisting biofilm-associated nosocomial infections and is recognized as a leading organism involved with catheter-related infections, orthopedic infections, devices, and wound infections. With respect to medical devices, *S. aureus* secretion of surface-associated polymers aids in attachment to devices. *S. aureus* attachment to biomaterials is enhanced by existing proteins that adsorb to the biomaterial surfaces such as plasma vitronectin fibrinogen, and fibronectin, while monoclonal antibodies against surface-associated polymers decreased attachment [1]. Studies have shown that biofilm formation on implanted medical devices is influenced by physical and chemical characteristics of the device surface. Surface roughness and hydrophobicity appear especially important, with spore adherence to hydrophilic smooth surfaces enhanced by flow velocity and impaired by surface roughness [2]. The mechanism by which surface roughness influences attachment of *S. aureus* to smooth surfaces appears to be altered shear stresses resulting in elevated heavy particle concentration near hydrophobic regions of a surface. Performing a computational fluid dynamics analysis, it was determined that under laminar flow conditions, the local Reynolds number at a roughness peak could fall below 1, drastically changing the stable adhesion characteristics of the

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eczema mechano-sensing proteins [3,4]. By quantifying surface roughness and a flow rate parameter for in vivo conditions using ps-1 *S. aureus* biofilm formation in in-vitro flow system, it was possible to determine a roughness range and relative flow rate at which staphylococcal biofilm formation is maximized [5]. This same range has been shown to occur physiologically at roughness peak heights exceeding 05 mm with flow rates exceeding 02 11 s 1. A preliminary model to characterize the parameters influencing flow over a surface of roughness and compare to attachment dynamics of *S. aureus* to pico- or sub-pico-meter surface roughness has been developed [3,6]. In the human body, Staphylococcus aureus regularly resides on epithelial tissues and mucous membranes, among other sites. Approximately 40% of the general population is colonized with *S. aureus*, increasing the risk for infection associated with surgery and intravascular device implants [7]. Fortunately, *S. aureus* can be successfully treated with antibiotics; however, the emergence of antibiotic resistant variants in the last 70 years has rendered this treatment method useless in some cases [8]. Multi-drug resistant strains, notably methicillin-resistant *S. aureus* (MRSA), are of great concern due to their prevalence in hospitals and in the community. Despite heavy observation, MRSA strains continue to proliferate and adapt, avoiding antibiotic treatment in many cases [9]. The lethal reputation of *S. aureus* results from its ubiquity, ability to form biofilms, and ability to produce virulence factors. New strains are able to capitalize on genetic mutations resulting from the mistreatment of infection in previous hosts. Furthermore, biofilms create continued infection environments that are impervious to external stimuli such as the immune system and antibiotic treatment. Mechanisms of biofilm formation by *S. aureus* are similar to that of other bacterial species, including attachment, multiplication, exodus, maturation, and dispersal [10]. The earliest stages of biofilm formation consist of attachment and multiplication on a surface. *S. aureus* is unable to swim toward favorable environmental conditions like some motile bacteria. Rather, initial attachment to surfaces occurs passively by means of random collision. Throughout this process, cells continue to divide and eventually, are joined by other *staphylococci* nearby. At this point, the emergence of microcolonies begins. *S. aureus* microcolonies are characterized by towers that can lack obvious cohesiveness between cells and can change the structure of their surrounding environment by excreting extracellular polymeric substances. Although chain-like aggregates are readily observed, little is known about what induces such aggregates in staphylococci [11]. The objectives of this work are to examine the influence of the shape and size of *S. aureus* bacteria on blood flow patterns (hemodynamics) in blood vessels. We propose to demonstrate how the bacterium morphology influences significantly SS distribution, flow resistance, and possible microcirculatory obstructions that correlate the use of computer modeling and computational fluid dynamic analyses. The long-term goal is to the provide information to help in the diagnosis/ treatment of bloodstream infections and any associated cardiovascular problems caused by *S. aureus*.

Hemodynamic Implications

Hemoneutrality behavior of *Staphylococcus aureus*

The physiological importance of *Staphylococcus aureus* is manifold. As an opportunistic pathogen, it is a leading cause of biomaterial-related infections (e.g., bloodstream, orthopedic devices, and other implanted prosthetics) [5,12]. They can cause cell signaling miscommunications, disrupting healthy vascular hemodynamics and lowering fluidic shear stress in surrounding tissues. They can also induce localized thrombosis through the dysregulation of coagulation and inflammatory cascades. Because *Staphylococcus aureus* species cannot penetrate the endothelial wall, their survival and persistence rely on attachment to the endothelial lumen and adjacent vascularized tissues, thus acquiring a shield from host immune mechanisms. Measurement of the thrombotic potential of *Staphylococcus aureus* species under controlled flow conditions will hopefully illuminate acute pathophysiological processes in vitro.

Use of neural blood plasma and buffer for bacterial thrombo inflammation studies is common practice to ensure compatibility and non-arthritic behavior in vitro. The effects of preservatives also need to be understood, as they threaten to produce artifactual results. Rheology has gained traction in screening the blood compatibility of vascular biomaterials as it describes hemorheological states and activities in computationally and clinically relevant simple in vitro models. These biophysical and biochemical processes are inextricably linked as blood-contacting biomaterials are predominantly long-lasting foreign entities that face altering hemodynamics and often sterile blood. Yet much like physiological observations, consistent end-point measurement is rare and progresses slowly. This is partly due to the difficulty of conducting large numbers of flow studies under consistently representative and relevant experimental conditions.

In vision of this, a rapidly executed and easily miniaturized automated assay has been engineered to screen the blood compatibility of synthetic biomaterials under a physiologically relevant blood product concentration with matrix and background inclusion valves. The yeast polysaccharide beta-glucan is also applied to induce thrombogenic potential in a consistent and representative way. These initial development studies hope to address and elucidate difficult hemorheological questions regarding how matrix composition and blood product particle type affect successful attachment through external shear stress and sudden portal pore gradients.

Figure 1 is a schematic diagram illustrating a research study simulating the presence of *Staphylococcus aureus* bacteria in the bloodstream. The diagram depicts the key elements of the study, including labeled representations of *Staphylococcus aureus* bacteria, red blood cells, white blood cells, and blood vessel walls. The *S. aureus* bacteria are shown in clusters, with labels identifying their main structures. Blood cells are represented with clear distinctions between red and white cells, and the blood vessel walls are depicted with varying levels of detail and distinct textures to highlight

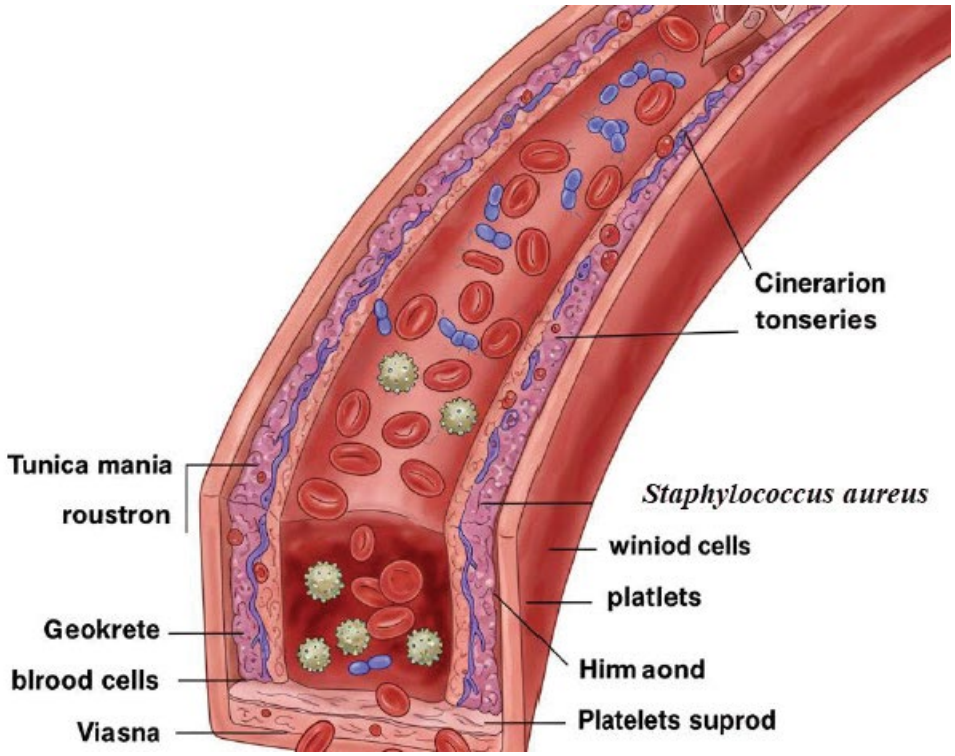

Figure 1: This illustration shows a cross-section of a blood vessel during blood poisoning caused by *Staphylococcus aureus*. The bacteria can be seen alongside normal blood components such as red and white blood cells and platelets, highlighting the spread of the infection in the bloodstream.

their role in the simulation. The schematic may also include labeled elements such as immune cells, cytokines, and any other relevant components of the study.

Material and Methods

Computational modeling (CFD Simulation)

A computational fluid dynamics (CFD) model was developed to evaluate the effects of Staphylococcus aureus morphology on blood flow characteristics, particularly focusing on wall shear stress (WSS) and local velocity perturbations. Simulations were conducted using COMSOL Multiphysics v6.1, solving the steady-state Navier–Stokes equations for incompressible laminar flow.

Geometry and model Setup

A two-dimensional rectangular microchannel (10 mm × 1 mm) was modeled to represent a capillary vessel segment. Different bacterial geometries were placed centrally within the domain (Table 1).

Blood rheology model

Blood was treated as a non-Newtonian fluid, modeled using the Carreau–Yasuda equation (Table 2) [13]:

$$\mu(\dot{\gamma}) = \mu_\infty + (\mu_0 - \mu_\infty)\left[1 + (\lambda\dot{\gamma})^a\right]^{\frac{n-1}{a}}$$

Boundary conditions

The following boundary conditions were applied to simulate blood flow through the microchannel (Table 3):

Meshing and solver settings

A structured quadrilateral mesh with boundary refinement was generated. Mesh independence was ensured through refinement testing (Table 4).

Output parameters

The CFD simulations generated several key hemodynamic parameters for each bacterial geometry (Table 5):

Results and Discussion

Taken overall, these results imply a complicated link between haemodynamic circumstances and the form/size of *S. aureus.* More research is required to understand precisely how these factors interact to define the risk of *S. aureus* sickness and, hence, how best people might be kept from this condition. Overall, this work helps to clarify the link between *S. aureus* and the vascular endothelium and keeps efforts to create a quick screening tool for at-risk dextral geometries and to find fresh therapy targets to more successfully prevent or treat *S. aureus* infection [14].

Blood flow velocities cause shading from low speed in blue to high in yellow. Figure 2 shows a blood artery in cross-section. The lumen of the vascular bundle consists on the rod, sometimes known as spherical or ellipsoidal or helical form microorganism. A cusp cap is Laminar flow causes flow rate to be a parabola. {It exists and has maximum Velocity between inhibitory rates}. Shear pressure resulting from blood flow affects bacterial dispersion in the blood artery seen in figure 2. The several strains of bacteria produce different drag effects and behaviour in the flow. Examining

Shape	Length (µm)	Width (µm)	Notes
Spherical	0.8	0.8	Perfectly circular
Rod-shaped	1.5	0.5	Rectangular capsule-like
Ellipsoidal	1.2	0.6	Smooth oval, symmetric
Helical	~2.0	~0.3	Sinusoidal approximation

Table 1: Bacterial Geometry Specifications.

Parameter	Value	Unit	Description
μ_0	0.056	Pa·s	Zero-shear viscosity
μ_∞	0.0035	Pa·s	Infinite-shear viscosity
λ	3.313	s	Time constant
n	0.3568	-	Power-law index
a	2.0	-	Sharpness of transition

Table 2: Carreau–Yasuda Model Parameters.

Boundary	Condition	Description
Inlet	Parabolic velocity profile	Maximum velocity = 5 mm/s
Outlet	Pressure = 0 Pa	Reference pressure
Vessel walls	No-slip	Zero velocity at solid walls
Bacterial surface	No-slip	Treated as static rigid inclusions

Table 3: CFD Boundary Conditions.

Setting	Value / Method
Mesh type	Quadrilateral (structured)
Refinement	Fine around bacteria
Solver	PARDISO (direct solver)
Tolerance	1e-6 (relative)
Model type	Steady-state, laminar flow

Table 4: Mesh and Solver Parameters.

Output Parameter	Description
Wall Shear Stress (WSS)	Along vessel walls, compared to bacteria-free case
Velocity streamlines	Visualized recirculation and disturbance zones
Shear rate ($\dot{\gamma}$)	Evaluated near vessel walls and bacteria
Vorticity	Detected eddies and vortical structures
Pressure drop	Across entire domain

Table 5: Key Simulation Outputs.

such connection in the sense that the flow in a fluid drive is the bacterium motion, the interaction between the bacteria and the white blood cells, and the adhesion to the vessel wall depends on an awareness of such process. Being septicaemia is a blood circulation process.

The using of neural network

Applied on the result of the neural network regression model. Figures 3 presents an error histogram. It evaluates the variation in the ground truth target from the prediction on training, validation, and test sets. The most of the mistakes are found close to 0, implying that the model actually fits the fundamental structure in the data.

The bell-shaped distribution captures the reality of typically distributed errors. Most of the projections get really close to zero error. Moreover, not quite distinct from one another were the widths of the errors discovered in the blue, green, and red phases of the test. We therefore were able to observe the positive generalisation of the model free from overfitting occurrences. Since the network generalises effectively in all phases of the main activity, it is reliable when data is being obtained [11,12,15].

Table 4 shows the overall MSE values for every method. Figure 4 shows, for a neural network (NN), the mean ± SD accuracy of 3 folds throughout the training, validation, and test sets during 12 iterations. Epoch number six has the lowest validation error 0.24229 so this is the point of maximum generalisation. The fact that the validation and test errors seem to have stabilised after this period suggests a less overfitting model. Training and validation curves near proximity guarantees consistent learning and suitable generalising of models. This fits the best early stopping strategies and neural network learning approaches covered in [16] and [17].

Effect of Shape and Dimensions of Staphylococcus aureus on Hemodynamics Interpretation [14]. One could understand figure five as follows. With the dimensions and forms of Staphylococcus aureus, the figure 5 shows that the model forecasts the outputs precisely. The strong R values of more than 0.90 for all training, validation, and test

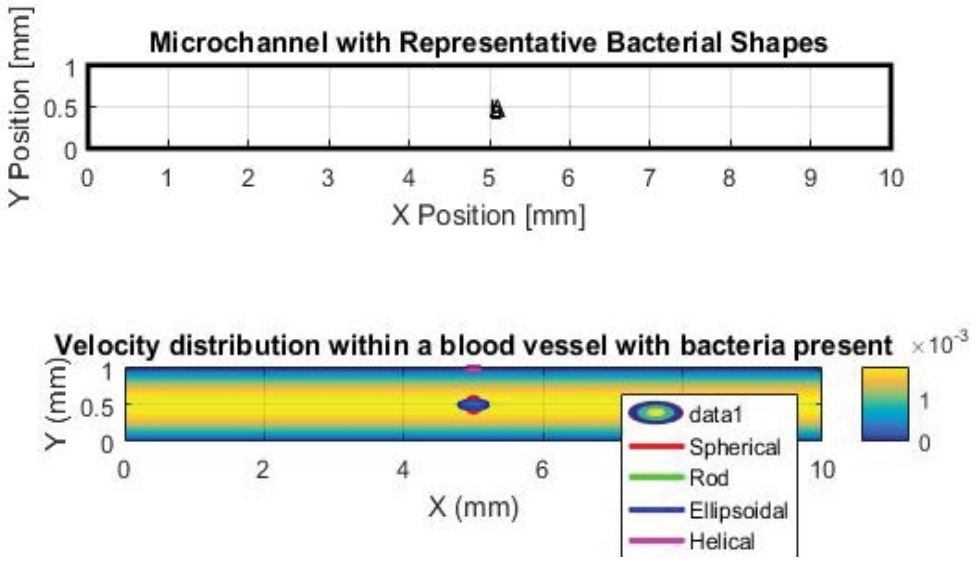

Figure 2: *S. aureus* in a blood artery, velocity distribution.

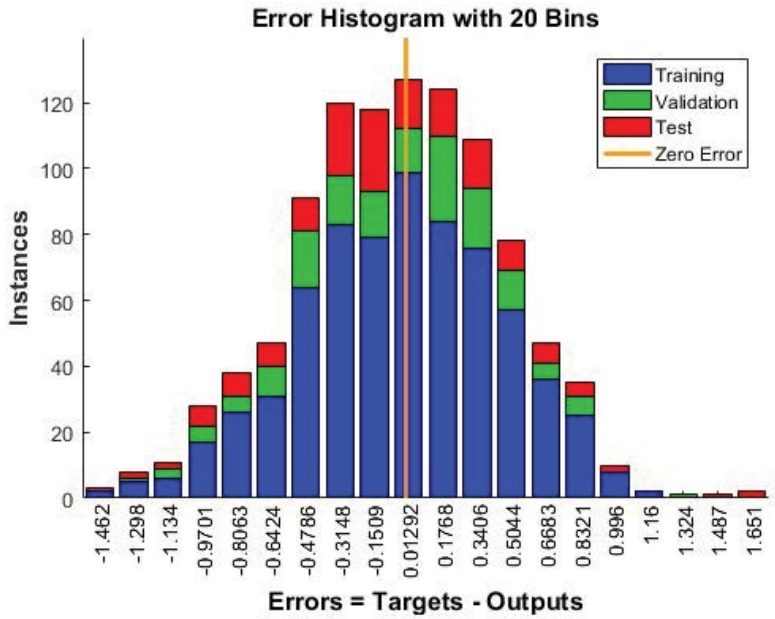

Figure 3: Training (blue), validation (green), and test (red) set mistakes of the 20-bin error histogram of the learnt neural network model. Most errors hint to almost perfect generalizing accuracy and strong predictive capability. Best zero-error performance is displayed by the orange line.

data show that the morphology of the bacteria is a distinct and quantifiable component in modifying the blood flow characteristics. This makes it abundantly evident that among other factors, the size and geometry influence the shear stress, the velocity, among others. The linearity between the prediction and actual values indicates that the model was reasonable; hence, the hypothesis regarding the effect of *S. aureus* physical picture is valid.

The effect of bacterial shape

For several aspect ratios—0.50 for flattened forms and 3.00 for elongated forms—the figure 6 shows the shape distortion of staphylococcus aureus. These geometries of theorised or observed bacterial body forms can arise in many environmental or biological niche. These variations are critical in haemodynamic circumstances since changes in bacterial size and shape can significantly influence the interaction of the bacteria with the blood flow [18,19]. Longer bacteria (higher aspect ratios) could be the source of more severe vascular blockage or biofilm development as well as possible disturbance in the flow field and shear strains on vessel walls. Subsequent computational investigations aiming at addressing how the various forms produce fluid dynamics and haemodynamic parameters find their subject in this model [20].

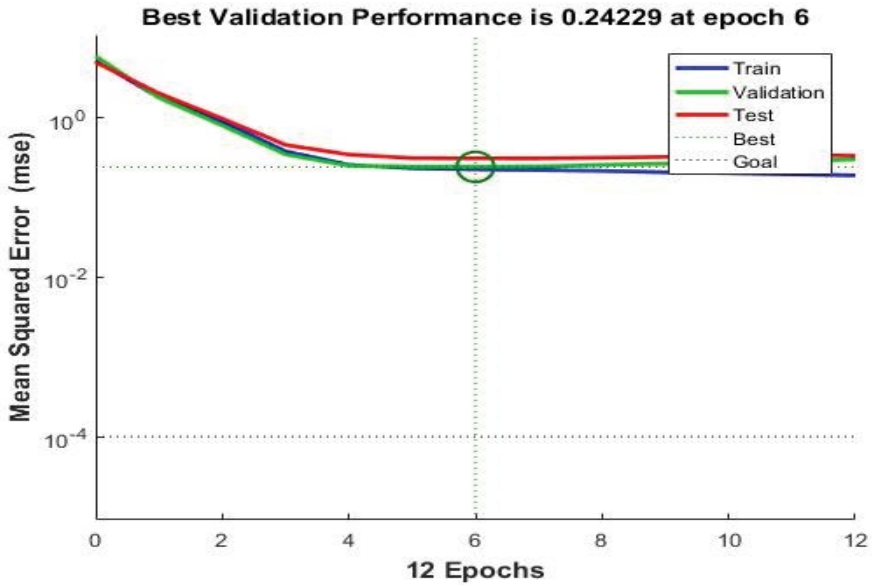

Figure 4: A performance graph of the neural network shows, during 12 epochs, mean squared error of training (in blue), validation (in green), and test (in red). With a validation error of 0.24229 Epoch 6 had the lowest value, implying that six is the optimal time for generalising.

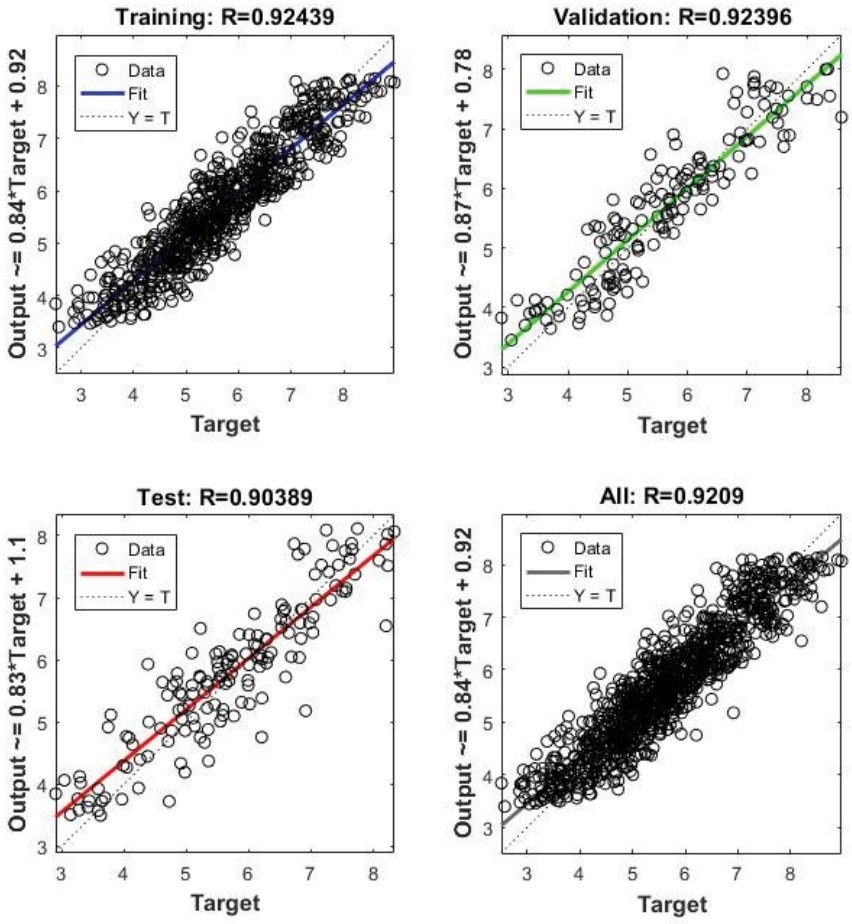

Figure 5: Illustrates that using Staphylococcus aureus' dimensions and forms, the model precisely forecasts the outcomes.

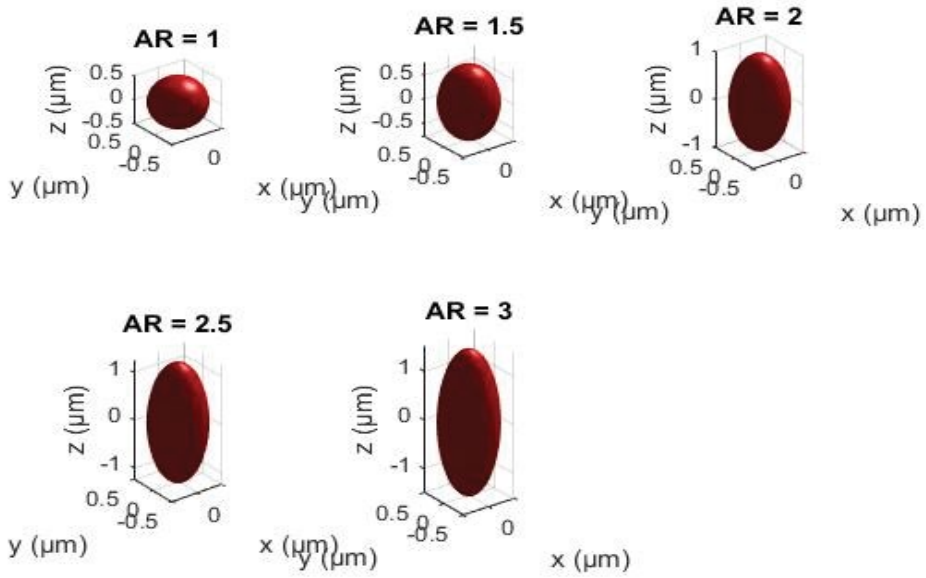

Figure 6: Shows *Staphylococcus aureus's* shape morphing at several aspect ratios (0.50 for flattened forms and 3.00 for elongated forms).

Conclusion

The haemodynamics alter as the form of the bacterium itself changes; consequently, a significant shift in risk from *S. aureus* attachment and consequent colonisation of the valves results from balance between elongation versus volume or aspect ratio of a 1x1 versus 5x1 cuboid. A simple approach to better estimate the displacements between fluid elements will enable a more methodical investigation of how to decide between acting naively by running a single quantity attached to an object and doing the more complex thing by building objects depending on extra intrinsic coordinates. And how could one better see the flow and displacement fields themselves? Although other visualisations using different thresholds and different alpha values have produced different results in this work the fact that main vorticity vectors tend to point to the same location in space over much of the total simulation time as well as only provide view of a few extreme values is not desirable.

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