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Theoretical study on the bactericidal nature of nanopatterned surfaces

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HIGHLIGHTS

- A mathematical model was primely used to explain the mechanism of the bactericidal properties of the cicada wing surface.
- The maximum stretching of bacterial layer is at the top of the nanopillars/ridges in region A.
- Optimal antibacterial nanostructures are analyzed and discussed.

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ABSTRACT

A natural biomaterial has been discovered with bactericidal activities, which is mainly attributed to its nanopatterned surface structure. The surface of *Clanger cicada* (*Psaltoda claripennis*) wings has been identified as a natural bactericidal material, which has lead to the emergence of research on the development of novel antibacterial surfaces. From the interactions between bacterial biofilms and nanopatterned surface structures, a new mechanical model is proposed that investigates the rupture of bacterial cells within the framework of the "stretching" theory. The effect of surface nanoroughness on the survival of bacterial cells is evaluated by determining the stretching ability of their cell walls. The results, calculated using Gram-positive and Gram-negative bacteria as examples, show a correlation between the stretching of the cell wall and the geometric parameters of the surface structures. The theoretical results indicate that for a given cell rigidity, the bactericidal nature of the surface is determined by the geometric parameters of the surface structures.

1. Introduction

Some biological materials, including the surfaces of cicadae and lotus (*Nelumbo nucifera*), exhibit contaminant-free natures, even though they are innately surrounded with potential contaminants in their natural environments (Bazaka et al., 2011; Bhushan et al., 2009; Marmur, 2004; Zhang et al., 2006; Zhao et al., 2009). Significant attention has been paid to the contamination-prevention mechanism of these naturally existing surfaces, both experimentally and theoretically, since their self-cleaning properties were discovered. These surfaces have special physical surface structures that significantly increase their hydrophobicity, even to the point of superhydrophobicity, thus, endowing them with self-cleaning qualities (Guo et al., 2011; Su et al., 2010; Watson et al., 2010; Wenzel, 1936; Zheng et al., 2005). Some research groups have tried to establish a relation between the self-cleaning and antibiofouling properties of natural surfaces with those that can prevent the attachment and accumulation of biological material (Carbone, 2004; Decuzzi and Ferrari, 2010; Wenzel, 1936; Zheng et al., 2005). As has been recently reported, superhydrophobic/self-cleaning surfaces are not necessarily antibiofouling in nature. *Pseudomonas aeruginosa* cells that could adhere relatively effectively onto the surface of the wings of the *Clanger cicada* (*Psaltoda claripennis*) were killed with extreme efficiency by the wing surface. The experimental results suggest that there are only physical interactions between bacterial cells and cicada wing surface. Thus, the bactericidal properties of the wings are a result of the mechanical rupture of the bacteria, arising from physical interactions between the cells and nanoscale wing surface structure. A stretching model has been developed by Elena P. Ivanova and colleagues to provide insights on the interactions occurring between bacterial cells and cicada wing surface structures. On the basis of this model, the adsorption of the bacterial cell walls on the pattern of the cicada wing surface could cause a drastic increase in the total area, accompanied by stretching of the cell wall, which could lead to irreversible cell membrane rupture and bacterial death. On the other hand, a very detailed theoretical model has been presented for the prediction of the strength of cellular adhesion to originally

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inert surfaces as a function of the surface structure, accounting for both specific (ligand–receptor) and non-specific interfacial interactions (physical interactions) (Decuzzi and Ferrari, 2010; Campoccia et al., 2013; Hasan et al., 2013a, 2013b; Ivanova et al., 2012, 2013; Nguyen et al., 2013; Pogodin et al., 2013). Results from this model suggest that only physical interactions between bacterial cells and cicada wing surface structures cannot provide sufficient energy to lead to full adhesion and irreversible cell rupture.

In this study, we provide another theoretical model to explain the mechanism of the bactericidal properties of the cicada wing surface. On the basis of the interactions between bacterial cells and nanopatterned surface structures, an elastic mechanical model is proposed that investigates the rupture of bacterial cells within the framework of the stretching theory. The effect of surface nanoroughness on bacterial cells is evaluated by determining the stretching degree of the cell walls. The results for the stretching degree of Gram-positive and Gram-negative bacteria as functions of the geometric parameters of surface structures are obtained and discussed. The theoretical results indicate that given the cell rigidity, the bactericidal nature of the antibacterial surfaces is determined by the geometric parameters of the surface structures.

2. Theoretical model

Two kinds of surfaces, covered either with nanopillar or nanoridge structures, were considered onto which sparsely distributed bacterial cells adhered. As the thickness of bacterial cell walls are an order of magnitude smaller than the dimensions of the nanostructures on these surfaces, the bacterial membranes can be treated as thin elastic layers, whose structural details and composition can be neglected. In other words, we focused our attention on the stretching layer of a bacterial cell interacting with the surface nanostructures, without losing generality. The difference between the stretching layer and free layer of a bacterial cell, i.e., the stretching degree, can be evaluated by calculating the difference between the areas of bacterial cell membranes that could either interact or not with surface nanostructures. Mechanical ruptures would occur on bacterial cell walls if the stretching degree of the elastic layer exceeds its threshold.

2.1. Nanoridge-covered surfaces

The side-elevation sketch map of a bacterial cell adhered onto nanoridges is shown in Fig. 1, where the shaded region represents the bacterial cell. Due to the interaction between nanoridges and gravity, deformations occur along the elastic bacterial membrane, as shown in Fig. 1. Assume that the side elevation of each periodically arranged nanoridge is parabolic, height of each nanoridge is H, width at the bottom of each parabola is $2R$, contact area between the bacterial membrane and nanoridge is S_A, and area of the suspended membrane is S_B. For simplification, each bacterial cell is treated as a cylinder with a diameter of A and density of ρ. The length of each cylinder is greater than its diameter.

Let α denote the stretching of the bacterial membrane due to its surface interaction, where k denotes the coefficient of stiffness that is related to the intensity of the bacterial membrane and θ denotes the angle between the tangent to the bacterial membrane and horizontal line. When the stretching of the bacterial membrane is within the range of elastic deformation, these stresses are proportional to the stretching, and $T/S=E\alpha$, where S is crossection area and E is Young's modulus of the bacterial cell walls.

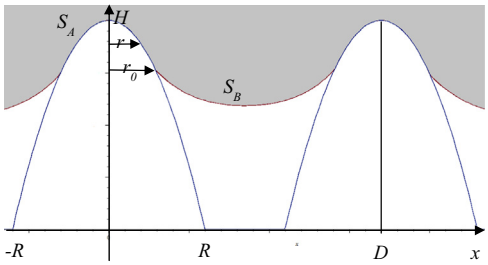

Fig. 1. Side-elevation sketch map of a bacterial membrane adsorbing onto two neighboring nanoridges, where H is the height of the nanoridge, $2R$ is the bottom width of the nanoridge, S_A denotes the part of the bacterial membrane covering the nanoridge, S_B denotes the suspended membrane surface, r_0 is the distance from the dividing line to the x-axis, and D is the distance between two adjacent nanopillars.

2.1.1. For the area of S_A

Set a rectangular coordinate system, as shown in Fig. 1, where the parabola intersects with the x-axis at points $(R, 0)$ and $(-R, 0)$, α_A is local stretching of the layer in region A, and α_B is the local stretching of the layer in region B. Then, the curve equation of the parabola can be obtained as

$$g(r) = H(1 - r^2/R^2).$$

Hence, we can obtain the slope of the bacterial membrane: $g'(r) = -\tan\theta$.

The distance from the x-axis to the dividing line is r_0, and distance from the x-axis to an arbitrary point in the curve is r. External forces on the cell membrane comprise G, which is the local gravity of the bacterial cell, and T_1 and T_2, are the pulling forces over and under the infinitesimal area, respectively (Fig. 2a). Define γ as the thickness of the cell wall of the bacterium, $\lambda=\rho gA$ as the gravity of unit bacterial cell walls. Set an infinitesimal membrane, taking the length as 1 nm, and the width (dr) in the direction perpendicular and parallel to the tension, then we can get the infinitesimal area $ds=1\times dr$. Let r' be the radius of the curvature and $d\theta$ be the field angle. Section stress of the infinitesimal area can be written as follows (Gerstmayr et al., 2013; Such et al., 2009):

$$T_1 \cos\frac{d\theta}{2} = T_2 \cos\frac{d\theta}{2} + G_A \sin\theta \tag{1}$$

$\because d\theta \to 0$, $\therefore \cos(d\theta/2) \to 1$. Substituting $G_A=\lambda\, ds$ into Eq. (1), we can obtain

$$T_1 - T_2 = \lambda \sin\theta\, ds. \tag{2}$$

Set T_0 to be the strain of the vertices and T_A to be the strain at any position in region (A); thus

$$T_0 - T_A = \int_A \frac{-g'(r)\lambda ds}{\sqrt{1+(g'(r))^2}} = \frac{\lambda R^2}{2H}\sqrt{1+\left(\frac{2H}{R^2}r\right)^2}. \tag{3}$$

The strain at the dividing line can be regarded as

$$T_{r_0} = T_0 - \frac{\lambda R^2}{2H}\sqrt{1+\left(\frac{2H}{R^2}r_0\right)^2}. \tag{4}$$

2.1.2. For the area of S_B

To determine the portion of the cell membrane suspended between nanoridges (Fig. 2b), set its curvilinear equation as $f(r)$; hence, the slope is $f'(r) = -\tan\theta$. The external forces can be described through the catenary equation.