A microscale model of biofilm formation in porous media
R.H. Dillon*, L. Fauci** & D. Meng**
*Department of Pure and Applied Mathematics
Washington State University, Pullman, WA 99164, USA
E-mail: dillon@math.wsu.edu
**Department of Mathematics
Tulane University, New Orleans, LA 70118, USA
E-mail: lif@math.tulane.edu, meng@math.tulane.edu

Abstract
In this paper we describe a microscale model of biofilm formation in an idealized two-dimensional porous media. This model includes the flow, transport and bioreaction of nutrients, electron acceptors and microbial cells in a saturated granular porous media. Bacterial cells are predominantly attached to the soil particles but may be also found in the fluid phase. Attachment of bacterial cells to the soil matrix and growth of bacteria can change the local permeability. The coupling of mass transport and bioreaction results in spatial gradients of nutrients and electron acceptor concentrations. We describe a numerical method for the microscale model, show results of a convergence study and present simulations of the model system.

1 Introduction
Microbial processes are an important aspect of transport and reaction phenomena in porous media. These processes are difficult to incorporate into field scale models and the phenomena are not well understood on a microscale level. An important aspect of microbial processes is the propensity of cells to aggregate and adhere to each
other and bind to a surface. The adsorbed cells may produce a matrix of extracellular polymer substance (EPS) binding the cells together. A biofilm consists of the adsorbed cells in association with EPS. The biofilm itself forms an essential part of the porous media – altering its microscale geometry and chemical environment. At the same time, substrates are transported from the bulk fluid to the biofilm by diffusion and advection processes and are consumed by the cells. Substrates may also be embedded within the soil matrix itself and diffuse into the bulk fluid or into the biofilm.

Mathematical models addressing biofilm processes have been developed for saturated soils and aquifers, porous media and closed conduits. A two-dimensional model of microscale transport and biotransformation that couples the Navier-Stokes equations, advection and diffusion of a nonreacting chemical substrate, as well as cell-cell and cell-substratum adhesion is developed in Chen et al. The microscale model described in this article represents the microbes in the biofilm system as discrete entities. Several aspects of this model have been described elsewhere in detail. The model includes the coupling of hydrodynamics, substrate reaction, diffusion and convection within an idealized model of porous media. Cell-cell aggregation and cell-substratum adhesion are modeled by generating binding forces between cells or between cells and the substratum. By representing cells discretely it is possible to more accurately model changes in the local geometry brought about by the biofilm within the porous matrix. This is important because it influences local flow and, this in turn, influences the transport of cells and substrate.

2 Mathematical framework

We present a system of coupled nonlinear equations which describes a model biofilm system. The mathematical framework and numerical method is based in part on the immersed boundary method. This method was introduced by Peskin to model blood flow in the heart and has subsequently been used in a variety of applications including the study of aquatic locomotion, platelet aggregation in the blood, and three-dimensional blood flow in the heart. We assume that the fluid dynamics are governed by the Navier-Stokes equations:

\[ \rho(u_t + (u \cdot \nabla)u) = -\nabla p + \mu \nabla^2 u + F, \]
and

\[ \nabla \cdot \mathbf{u} = 0. \]  

(2)

which describe the balance of momentum and conservation of mass in a viscous incompressible fluid. Here, \( \rho \) is the fluid density, \( \mathbf{u} \) is the fluid velocity vector, \( p \) is pressure, and \( \mu \) is the fluid viscosity. The term \( \mathbf{F} \) is the force density (force per unit area in two dimensions) that the microbes and pore walls exert on the fluid.

The cell body of each microorganism is modeled (in two dimensions) as an elastic ring, whose configuration is defined by the function \( \mathbf{X}_i(s, t) \), where \( s \) is a Lagrangian label (e.g. arclength with respect to an equilibrium configuration), \( t \) is time and \( i \) denotes the \( i^{th} \) microbe. The boundary force per unit length \( f_{\text{cell}(i)}(s, t) \) at each point on the ring is a function of the current ring configuration and consists of a tangential elastic spring force and a normal bending-resistant force. These are designed to preserve the size and the shape, respectively, of the ring. In our simulations, the stiffness constants associated with the cell ring are chosen to reflect the stiffness properties of a bacterial cell wall. This “immersed boundary force” is transmitted directly to the fluid and gives a contribution to \( \mathbf{F} \) which we call \( F_{\text{cell}(i)} \):

\[
F_{\text{cell}(i)}(\mathbf{x}, t) = \int_{\text{microbe}} f_{\text{cell}(i)}(s, t) \delta(\mathbf{x} - \mathbf{X}_i(s, t)) ds. \]  

(3)

Here, the integration is over the points of the ring and \( \delta \) is the two-dimensional Dirac delta function.

Bacterial motility is modeled in a simplified manner by means of a discrete set of forces applied to the fluid at points behind the cell body. These point forces are designed to represent the flagellar forces of a swimming bacteria. A detailed description of this mechanism as well as an algorithm for simulating the sequence of runs and tumbles characteristic of bacterial motility is shown in Dillon et al.\(^4\)

The pore walls are modeled in a manner similar to the microbe rings, that is as neutrally buoyant elastic filaments immersed within the fluid. However, these walls cannot move freely since they are tethered to fixed points in space by stiff elastic spring forces. We define \( f_{\text{wall}(i)}(s, t) \) to be the boundary force per unit length on the wall and define the fluid force density contribution from it in a manner analogous to Eq. (3):
\[ F_{\text{wall}(i)}(x, t) = \int_{wall} f_{\text{wall}(i)}(s, t) \delta(x - X_i(s, t)) ds. \] (4)

We note that this representation of the soil matrix makes it easy to change the geometry of the pores.

Cell-cell and cell-wall adhesion are modeled by the creation of elastic springs or 'links' between points on each of the adherent entities. The model for link formation is similar to the model for platelet adhesion and aggregation described in Fogelson and Fauci and Fogelson. If the distance between the centroids of any given pair of cells is less than a prescribed cohesion distance, an elastic spring may be created to link the two cells. The mechanical properties of each spring and the cohesion distance are chosen to reflect biological and physicochemical properties of the system. Cell-wall links are formed in a similar manner. Detachment of cells from the biofilm is modeled by allowing the links to break when they are stretched beyond a prescribed length. Thus, the force term \( F \) includes several contributions arising from the cell bodies, micropore walls, cell-cell and cell-substratum adhesion, and cell motility.

The immersed boundaries (microbes and pore walls) influence the fluid motion through the forces we have just described. In turn, the fluid motion and continuity of the fluid velocity field give equations of motion for the points \( X(s, t) \) on the immersed boundaries, namely:

\[ \frac{dX(s, t)}{dt} = u(X(s, t), t) = \int u(x, t) \delta(x - X(s, t), t) dx. \] (5)

Here the integration is over the entire domain. This can be interpreted as the usual no-slip boundary condition at a fluid-material interface.

The presence of microorganisms in the bulk fluid influences both the flow dynamics and the substrate field. The equation which describes the advection, diffusion, and consumption of a single chemical species within the fluid-filled pore is

\[ c_t + (u \cdot \nabla) c = D \nabla^2 c - R(c)c, \] (6)

where \( c \) is substrate concentration, \( D \) is the molecular diffusivity, and \( R \) is a consumption rate that is nonzero only near the site of each of the microbes.
3 Convergence studies

To show convergence of our immersed boundary representation of porous media, we consider the situation of an obstacle placed midway between two channel walls. Four smaller immersed boundary particles that are free to move within the domain are introduced at the upstream edge of the channel (see Figure 1). For a fixed pressure gradient, we measure the time it takes each particle to progress from its original position to a rectangular buffer region at the right of the channel. This is measured as a function of the ratio of obstacle diameter to channel width $\Gamma$. These ‘breakthrough times’ should increase as the channel becomes more occluded. Figure 2 shows the breakthrough times as a function of $\Gamma$ for one of the particles on successively refined finite difference grids. The convergence is evident. Note that for the coarse grid of $32 \times 32$, the particle never did reach the buffer region for $\Gamma \geq .4$. There is not enough accuracy to allow a nonzero flow rate for this gap width.

![Figure 1: Setup for the breakthrough study.](image1)

![Figure 2: Breakthrough time as a function of dimensionless gap width.](image2)
In this section we show two simulations of the model system. In the first, shown in Figure 3, we simulate the flow of fluid and bacterial cells or particles through a periodic domain with many cylindrical obstacles. The fluid flow is produced by a uniform pressure drop from left to right. The cylinders are modeled as immersed boundaries and tethered to fixed points in the domain. Large elastic spring constants make these structures relatively inelastic. Because of this, the cylinders remain stationary and impose a zero fluid velocity at the cylinder walls. Bacterial cells can be introduced on the left. These cells are motile unless they attach to a cylindrical wall. The mechanism for the bacterial motility is described elsewhere\(^4\). Once the bacterial cells attach, they become a part of the micropore geometry and have an impact on the fluid flow through the domain. In this simulation, the cells are approximately 1\(\mu m\) in diameter. The domain dimensions are approximately 80\(\mu m\) on each side.

In the second simulation, shown in Figure 4, we include the ad-
vection, diffusion and reaction of a substrate that is consumed by the discrete bacterial cells. In this simulation, we show a single tethered cylindrical object in the left center of a micropore. The pore itself is contained within two parallel immersed boundary walls. As in the previous example, a fluid flow is produced by a uniform pressure gradient within the micropore. Motile cells are introduced from the left and these cells can attach to the micropore walls or to the cylindrical obstacle. Initially, the substrate concentration is uniform throughout the micropore. Dirichlet boundary conditions at the micropore and cylinder walls hold the substrate concentration at the initial level at these immersed boundaries for the duration of the simulation. Since the individual bacterial cells consume the substrate, a nonuniform concentration field emerges. Details of the solution of the reaction-advection-diffusion equations are shown in Dillon et al.\textsuperscript{12}

5 Discussion

The simulations shown here are preliminary in nature, but suggest the possibilities inherent in this method of modeling microscale processes in porous media. This model can be used to predict the effect of microbial growth on permeability, to investigate the role of microscale inhomogeneity on transport and bioavailability of chemical species, and the phenomena of the filtering of biological cells that can impede the expansion of bioremediation zones.

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