last time: poroelastic → electrophysiology

\[ \frac{\partial u_i}{\partial t} = \nabla \cdot \left( k \nabla u_i \right) + U_0 + \frac{k_{12}}{k_{22}} J_0 \]

- neglect inertia: solid \( V_A \approx 1 \)
- fluid: \( R e \ll 1 \)

\[ k = k_n - k_{22} > 0 \text{ positive diffusivity} \]

\[ \dot{u}_i = \frac{\partial u_i}{\partial t} + U_0 \]

\[ u_2 = k_n \frac{\partial p}{\partial x_2} + k_{12} \frac{\partial V}{\partial x_1} \]

\[ J_2 = k_{22} \frac{\partial p}{\partial x_1} - k_{21} \frac{\partial V}{\partial x_1} \]

\[ \frac{\partial J_0}{\partial x_1} = 0 \]

new effects: electrosomotive flows \( \Delta V \) generated by \( \nabla V \)
streaming potential \( \Delta V \) generated by flow

electrokinetics \( \rightarrow \) fixed charge density (cartilage, and others...)
e.g. \( [NH_4^+] \text{, } [COO^-] \text{, } [SO_4^{2-}] \) from GAGs

not covered:
- osmotic pressure, dependence of \( k_n, \lambda, \beta \) on concentration

Today: transition to cell mechanics

- molecules (nm, pN)
- cell mechanics
- tissue mechanics
- single molecules
- porous / viscoelastic materials

- cells
- molecular motors
- ion channels
- cytoskeletal filaments
- continuum mechanics (CSK as a continuum)

- why cells?
- cartilage: cell content doesn't really affect eddy diffusion, its mechanical behavior...but cells respond to mechanical deformation...
- arterial wall: mechanotransduction (response of endothelial cells to fluid shear stress → atherosclerosis)
- smooth muscle cells (contractile, can redistribute the flow)
- muscle: cardiac / skeletal muscle cells, smooth muscle cells
- airway wall: mechanotransduction (epithelial cells)

> Force / biology interaction

Prototypical example: muscle

- anatomy / macroscopic behavior
- activation / contraction
- cross-bridge dynamics (Huxley)

see slides
- Maxon: maximum contracted state
- Exponentially stiffening behavior \( \frac{d\sigma}{dx} = a(x + \beta) \Rightarrow \sigma = C \exp(x \beta) + \beta \)

- How much force can be generated depending on the velocity of force development?
  - Strictly speaking, Hill's equations \( \frac{v}{V_{max}} = 1 - \frac{F}{F_{max}} \) are purely empirical, true for all muscles normalized efficiency \( \eta = \frac{\text{mechanical work}}{\text{chemical energy input}} \approx 25\% \) (comparable to car)
  - \( \Delta G = 25 k_B T \) per molecule
  - Step size (myosin + actin) \( \Delta x = 5 \text{ nm} \) and \( F \approx 3-4 \text{ pN} \)

- Activation / contraction
  - Depolarization (by nervous cell) conducted transversely \( \Rightarrow \text{Ca}^{2+} \) released \( \Rightarrow \text{contraction} \)
  - After contraction is completed, \( \text{Ca}^{2+} \) sequestration in SR
  - Sliding filament model: thick filament = myosin, thin filament = actin
  - Dark = overlap (A band) + light = actin only (I band)
  - A. Huxley and Niedergerke
  - H. Huxley and Hanson simultaneously in Nature in 1954
  - ATP-dependent conformational change \( \Rightarrow \) power stroke and displacement toward the (+) end of actin
  - \( \text{Ca}^{2+} \) removes the (tropomyosin) barrier \( \Rightarrow \) walk possible

Model by Jonathan Howard (following article by Pate, 1993)

\[ \begin{align*}
\text{myosin head} & \quad \Delta \text{actin binding site} \\
\end{align*} \]

\[ \begin{align*}
\text{binding} & \quad \text{unbinding} \\
\end{align*} \]

\[ \begin{align*}
\text{binding} & \quad \text{unbinding} \\
\end{align*} \]

\( n(x, t) \) probability of binding

\[ \begin{align*}
\frac{dn}{dt} = \frac{\partial n}{\partial x} + \frac{\partial n}{\partial x} = -v \frac{\partial n}{\partial x} \text{ for different } x \text{ regions} \\
\end{align*} \]