Confined Nematic Defects As Active Particles

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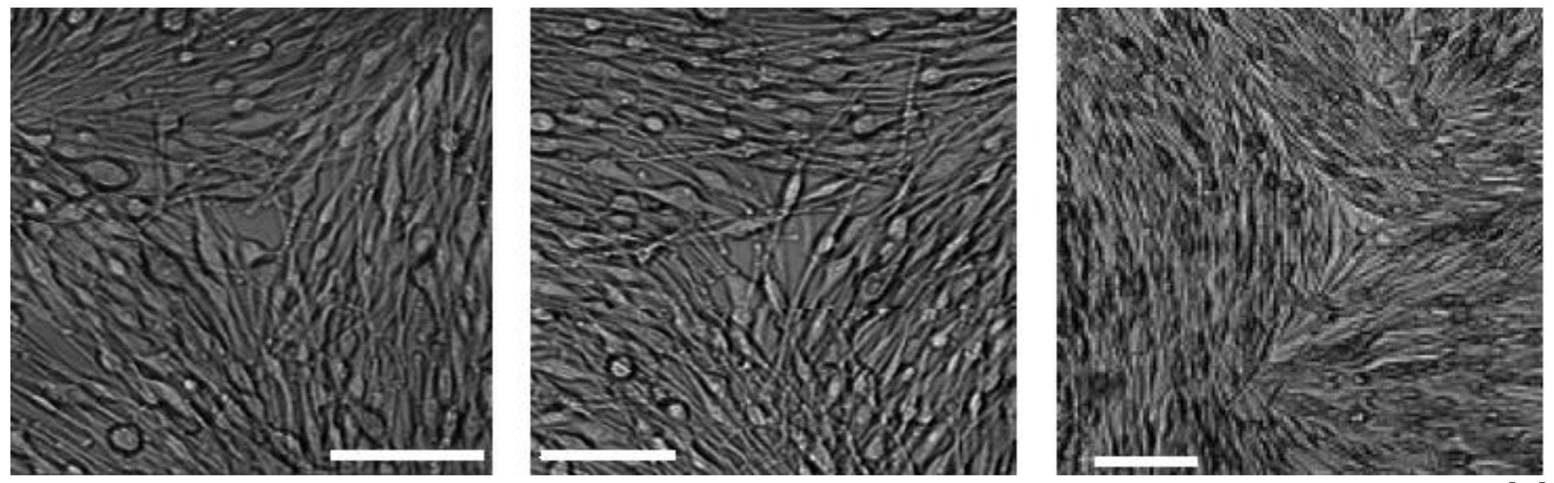
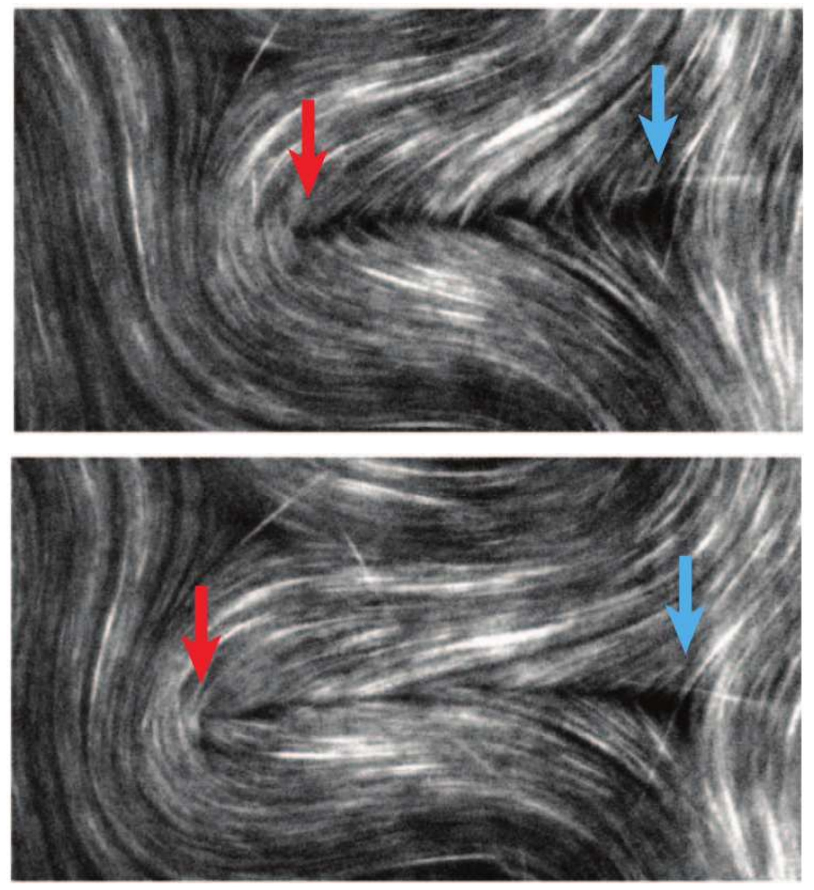
MOTIVATION



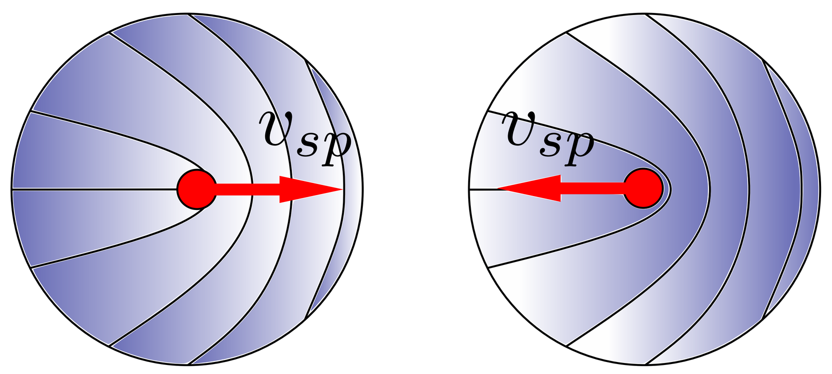
A $-1/2$ disclination in a nematoid layer of Human *Melanocytes*[1]

- Active nematics display topological defects at high density
- The $+1/2$ disclinations move by virtue of active stresses



Streaming $+1/2$ disclination in an active microtubule network[2]

$+1/2$ defects can be described as **Self-Propelled Particles** (SPP) in 2D. The defect velocity (v_{sp}) is controlled by the viscous flows from the director distortions. The velocity is proportional to activity (α), so defects in extensile ($\alpha < 0$) systems move opposite to contractile ($\alpha > 0$) systems.



(a) Extensile (b) Contractile

But $v_{sp} = \dfrac{\alpha R}{\mu}$

diverges as the system size (R)![3]

(μ is the fluid viscosity)

THE MODEL

(Hydrodynamics of Active Nematic Defects in 2D Confined Fluids)

Bulk Fluid 1 (μ_{b1})

$$\mu_{b2}\nabla^2 \boldsymbol{u}_2 - \nabla p_2 = \boldsymbol{0}$$

Fluid Interface (μ_0) at $z = 0$

$$\mu_0 \nabla_\perp^2 \boldsymbol{u}_{0\perp} - \nabla_\perp p_0 + \hat{\boldsymbol{n}} \cdot (\boldsymbol{\tau}_1 - \boldsymbol{\tau}_2)_\perp|_{z=0} + \boldsymbol{f}_\perp = \boldsymbol{0}$$

d_1

d_2

Bulk Fluid 2 (μ_{b2})

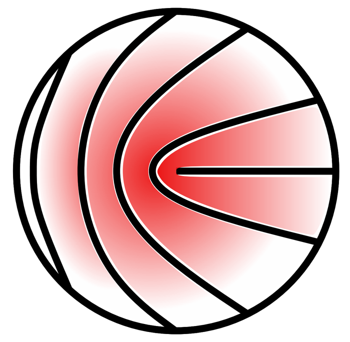
$$\mu_{b1}\nabla^2 \boldsymbol{u}_1 - \nabla p_1 = \boldsymbol{0}$$

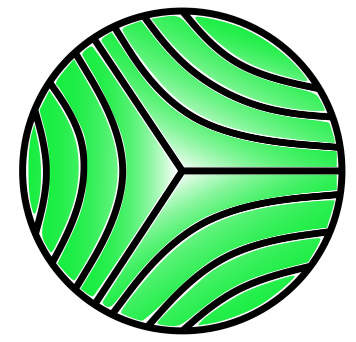
τ_1, τ_2: Viscous stress on the Interface from the Bulk Fluids

ρ: Scalar Nematic Order Parameter

Force due to the distortion of the nematic field

$$\boldsymbol{f}_\perp = \begin{cases} \alpha\left(\rho_r + \dfrac{\rho}{r}\right)\hat{\boldsymbol{e}}_x & \text{for a } +1/2 \text{ disclination} \\ \alpha\left(\rho_r - \dfrac{\rho}{r}\right)(\cos 2\phi \hat{\boldsymbol{e}}_x - \sin 2\phi \hat{\boldsymbol{e}}_y) & \text{for a } -1/2 \text{ disclination} \end{cases}$$





THE SOLUTION

Eliminating the bulk fluids to get an effective hydrodynamic propagator for the in-plane flow at the interface, we get (in fourier space)

$$\hat{\boldsymbol{u}}_{0\perp}(\boldsymbol{k}) = \hat{G}(k)\mathcal{P}\hat{\boldsymbol{f}}_\perp(\boldsymbol{k})$$

where $\mathcal{P}(= \boldsymbol{I} - \boldsymbol{k}\boldsymbol{k}/k^2)$ is the transverse projector (as the flow is incompressible)

$$\hat{G}(k) = \frac{1}{\mu_0 k^2 + k(\mu_{b1}\tanh(kd_1) + \mu_{b2}\coth(kd_2))}$$

The **Boundary Conditions** are:

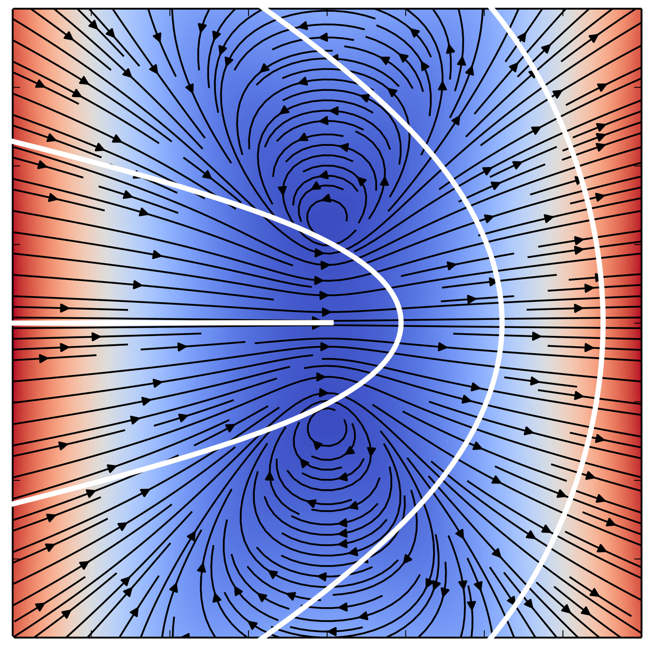
- Continuity of tangential velocity at $z = 0$
- Vanishing normal velocity at $z = 0$
- Continuity of normal stress at $z = 0$
- Vanishing velocity and pressure at infinity
- No-slip at the bottom wall
- Upper bulk fluid surface is free

At large distances ($k \to 0$), the flow is frictionally screened:

$$\hat{G}(k) \sim \frac{1}{\mu_R(k^2 + \xi^{-2})}$$

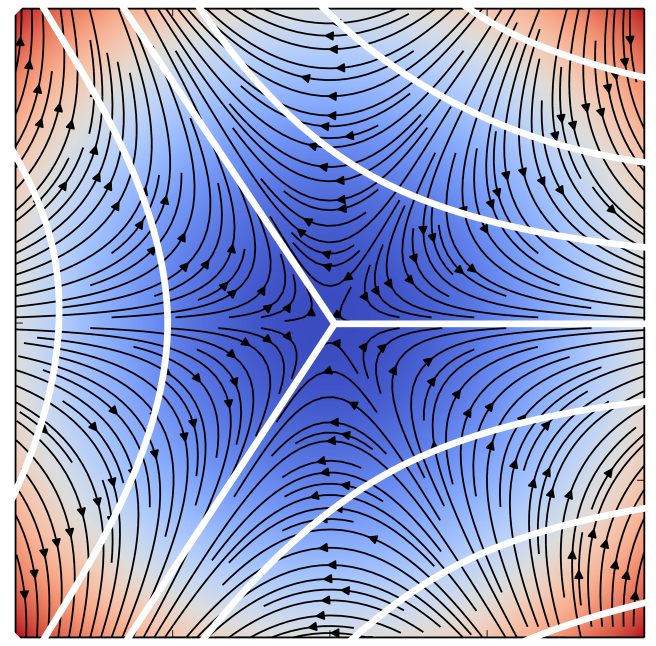
with $\xi = \sqrt{d_2\mu_R/\mu_{b2}}$ as the frictional screening length and a finite renormalized interfacial viscosity $\mu_R = \mu_0 + \mu_{b1}d_1 + \mu_{b2}d_2/3$!

RESULTS



$+1/2$ defect velocity streamlines

Angular dependence of the flow field is **dipolar** for the $+1/2$ disclination and **sextuplet** for the $-1/2$ disclination (by virtue of symmetry).



$-1/2$ defect velocity streamlines

The core velocity (for the $+1/2$ defect) is non-vanishing -

$$v_{sp} \sim \frac{\alpha}{2(\mu_{b_1} + \mu_{b2})}\ln\left(\frac{\xi}{\ell}\right)$$

for large ξ/ℓ, ℓ being the defect core radius

The divergence of v_{sp} is now controlled by ξ (logarithmically).

References:
1. Kemkemer, R., et al. EPJ E 1.2-3 (2000): 215-225.
2. Sanchez, Tim, et al. Nature 491.7424 (2012): 431-434.
3. Mishra, Prashant, et al. Bulletin of the APS 59 (2014).