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Thrombin flux and wall shear rate regulate fibrin fiber deposition state during polymerization under flow DAMIAN ILLING, KEITH NEEVES, Chemical Engineering, Colorado School of Mines — Thrombin is released as a soluble enzyme from the platelet surface to trigger fibrin polymerization during thrombosis under flow conditions. While isotropic fibrin polymerization under static conditions involves protofibril extension and lateral aggregation leading to a gel, factors regulating fiber diameter and orientation are poorly quantified under hemodynamic flow due to the difficulty of setting thrombin fluxes. A membrane microfluidic device allowed combined control of both thrombin wall flux $(10^{-13} \text{ to } 10^{-11} \text{ nmol}/\mu \text{ m}^2$ s) and the wall shear rate (10 to 100 s⁻¹) of a flowing fibringen solution. At the thrombin flux of 10^{-12} nmol/ μ m² s, both fibrin deposition and fiber thickness decreased as the wall shear rate increased from 10 to 100 s⁻¹. Direct measurement and transport-reaction simulations at 12 different thrombin flux-wall shear rate conditions demonstrated that two dimensionless numbers, the Peclet number (Pe) and the Damkohler number (Da), defined a phase diagram to predict fibrin morphology. For Da<10, we only observed thin films at all Pe. For 10<Da<100, we observed either mats of surface fibers or gels depending on the Pe. For Da>900 and Pe<100, we observed three-dimensional gels. These results indicate that increase wall shear rate first quenches lateral aggregation and then protofibril extension.

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