Living Biofouling-Resistant Membranes

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Membrane systems are being increasingly used for water treatment, recycling water from wastewater, during food processing, and energy production. They are thus a key technology to ensure water, energy and food sustainability. However, biofouling, the build-up of microbes and their polymeric matrix, clogs these systems and reduces their efficiency. Realizing that a microbial film is inevitable, we engineered a beneficial biofilm that prevents membrane biofouling by limiting its own thickness by sensing the number of its cells that are present via a quorum-sensing circuit (**Figure 1**). The beneficial biofilm also prevents biofilm formation by deleterious bacteria by secreting nitric oxide, a general biofilm dispersal agent as demonstrated by both short-term dead-end filtration and long-term crossflow filtration tests. In addition, the beneficial biofilm was engineered to produce an epoxide hydrolase so that it efficiently removes the environmental pollutant epichlorohydrin. Thus, we have created a living biofouling-resistant membrane system that simultaneously reduces biofouling while providing a platform for biodegradation of persistent organic pollutants.

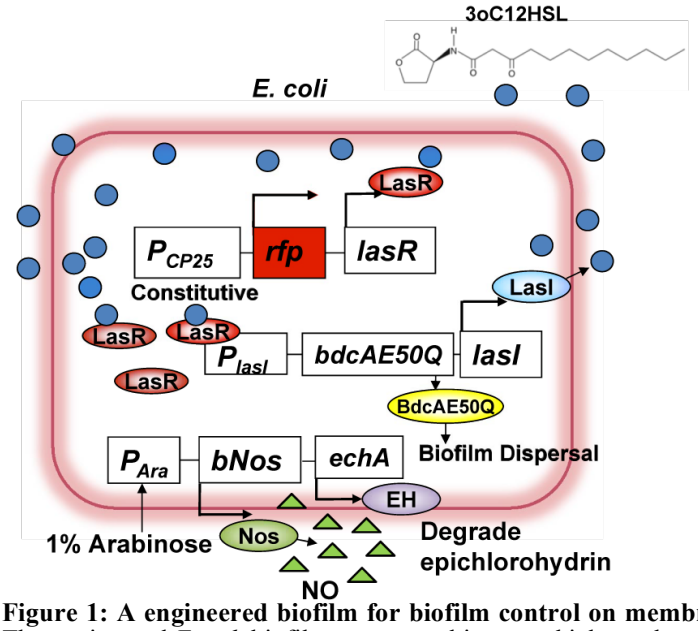


Figure 1: A engineered biofilm for biofilm control on membranes. The engineered *E. coli* biofilm can control its own thickness by using a novel "self-control" circuit while releasing quorum sensing molecules (nitric oxide using the enzyme nitric oxide synthase) that prevent colonization of membranes by other species. It was also engineered to release an enzyme (epoxide hydrolase) that can degrade the micropollutant epichlorohydrin. The self-control circuit was based on the LasI/LasR QS module of *P. aeruginosa*. The LasI protein and the engineered biofilm dispersal protein BdcA E50Q are controlled by the lasI promoter. When LasI is produced, it synthesizes the QS signal 3oC12HSL, and upon reaching a threshold value based on increasing cell density, the QS signal binds to LasR (. The 3oC12HSL+LasR complex activates the lasI promoter which leads to increasing production of dispersal protein BdcA E50Q as cell density increases.