Quorum sensing based membrane biofouling control for water treatment: A review

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ABSTRACT

Exploring novel biological strategies to mitigate membrane biofouling is of significant value in order to allow sustainable performance of membrane systems for water and wastewater treatment. Quorum sensing (QS) is a bacterial communication process that involves small diffusible signalling molecules, which activate the expression of myriad genes that control a diverse array of phenotypes such as bioluminescence, virulence, biofilm formation and sporulation. Since QS is often associated with biofilm formation, inhibition of QS should be a promising strategy to control membrane biofouling. Recently, a revolutionary application of bacterial QS has been as a novel strategy for the mitigation of biofouling in membrane systems. In this review an attempt is made to correlate membrane biofouling with QS activity. Moreover, recent trends in membrane biofouling control based on QS are presented and the mechanisms by which different agents mitigate membrane biofouling based on QS are discussed. The potential impact of QS-based methods of biofilm control is assessed. Lastly, brief conclusions and future research challenges in membrane biofouling control based on QS are highlighted.

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1. Introduction

Over the course of the last 25 years, membrane systems have become a favoured technological innovation for water and wastewater treatment [1]. Membrane systems are extensively used for wastewater treatment because they ensure improved effluent quality [2,3]. However, fouling is still a major limitation to the application of membrane bioreactor (MBR) as well as reverse osmosis and nanofiltration systems. Fouling is of various types, e.g., organic, inorganic, and biofouling [4]. Of these, "biofouling", resulting from extracellular polymeric substances (EPS) and microbial cells, presents a particular operation challenge [5,6]. Membrane biofouling decreases filtration performance owing to increased retention time caused by the deposition and growth of bacterial biofilms onto and into the membrane [7]. This major hindrance and limitation of the process has been under analysis since the early stages of membrane system development, and it is one of the most demanding obstacles to further application and enhancement of membrane technology [8].

Membrane biofouling is the adhesion, metabolism, and growth of microbial cells as a biofilm on the surface of a membrane, which is a main cause of loss of membrane permeability, and therefore, membrane flux and efficiency [9]. Biofilm formation on membrane surface is a complex process. For example, the initial adsorption of organics and suspended particles on the membrane surface form a conditioning film. This enables attachment of planktonic cells to the membrane surface, followed by the formation of microcolonies and biofilm maturation, where bacterial cells are embedded in a self-produced matrix of extracellular polymeric substances (EPS). Various biofouling control strategies have been developed through engineering and chemistry; all of these approaches have limitations [1]. Various antimicrobial compounds have been used to mitigate membrane biofouling such as silver salts, nitrofurazone, ammonium surfactants and antibacterial peptides etc. [10]. However, some anti-biofilm compounds also pollute the aquatic environment and are toxic to non-specific organisms. Moreover, killing the cells using disinfectants, as practiced in industry for example, does not always work, because it is not possible to kill 100% of the cells, leaving some viable cells to attach to solid surfaces and form a biofilm [11]. As a consequence of these limitations, there is a clear need to identify new strategies to control microbial fouling of membranes, and such strategies may be derived from an understanding of the biological process of biofilm formation. One regulatory system that has been linked to the control of biofilm formation in bacteria is the quorum sensing (QS) regulatory system [12–14]. Efforts to disrupt biofilms have enabled the identification of molecules produced by prokaryotes and eukaryotes with abilities to quench the QS system [15–19]. Thus, interfering with QS represents a 'non-disinfectant' biological alternative approach to control membrane biofouling.

Here, we review QS in membrane biofouling and an attempt is made to correlate membrane biofouling with QS activity. Recent trends in membrane biofouling control based on QS are presented and mechanisms by which different agents mitigate membrane biofouling based on QS are discussed. The potential impact of QS-based methods of biofilm control is assessed. Lastly, brief conclusions and future research challenges in membrane biofouling control based on QS are highlighted. It is expected that this review may serve as a stepping stone for further development and application of QS toward effective control of membrane biofouling. While the emphasis in this review is on biofouling control in membrane bioreactors (MBRs) the concepts are applicable to other membrane applications in the water domain.

1.1. Quorum sensing

Originally discovered in the 1970's, quorum sensing was first described as a mechanism for the coordinated expression of a phenotype, e.g., bioluminescence, at the population level [20,21]. Quorum sensing (QS) is a mechanism of cell to cell communication that is used by microbial cells to assess their local densities or diffusion gradients and control gene expression [22–25]. The mechanism of QS is based on the production, secretion and sensing of signalling molecules which, when they accumulate to a threshold concentration, trigger a change in gene expression in the population (Fig. 1) [22,26–28]. When the population density is low or when diffuse rates are high, acylhomoserine lactone (AHL) are present at low concentrations and the LuxR receptor (it is a transcriptional activator of the Lux operon that is activated when bacterial cell density is high) is quickly degraded (Fig. 1A). When the AHL concentration reaches a specific concentration, the AHL signalling molecules binds LuxR to make an AHL/LuxR complex, hence activating the receptor. AHL based signalling is predominantly found only in approximately 10% of proteobacteria (Gram-negative), although there are some exceptions. The QS systems of Gram-positive function in an analogous fashion, although the specific signal is an autoinducer peptide (AIP) (Fig. 1B). In this system, the AIP precursors are produced, are modified post-transcriptionally and secreted via specific transporters. When mature AIPs are in a high concentration, they bind to a transmembrane histidine kinase (HK) and the HK receptor is activated, which activates the downstream response regulator (DRR). This activated RR initiates transcription of specific genes.

There are a diverse array of phenotypes that are regulated by QS, either AHL or AIP, including luminescence, virulence, motility, competence and biofilm formation. While QS is important for the expression of these phenotypes, loss of QS does not appear to be lethal to the cells. Hence, QS has been proposed to be an ideal target for microbial control since inhibition of QS does not exert a strong selection pressure. As a consequence, it has been hypothesised that bacteria are less likely to develop resistance to QS inhibitors [29–33]. Interestingly, recent publications are suggesting that despite the low apparent selection pressure, some resistance can be evolved in the laboratory [34]. None the less, it remains an interesting target to control bacteria, especially biofilm formation, which is especially relevant to the fouling of water purification membranes. QS signalling molecules are produced in a very small quantity, so these molecules cannot be commonly detected, identified and characterized via conventional techniques. A brief summary of the approaches used for detection quantification, identification and characterization are presented in Table 1.

The process of QS can be disrupted by different mechanisms [34,46]: (a) inhibiting the production of QS signalling molecules [47,48], (b) degradation of AHL [49–51], (c) reducing the activity of AHL cognate receptor protein or AHL synthase [52,53], and (d) mimicking the signal molecules primarily by synthetic compounds as analogues of signal molecules [47,48]. Given that QS plays an important role in biofilm formation by a range of bacteria as well as virulence factor expression (Table 2), there have been a