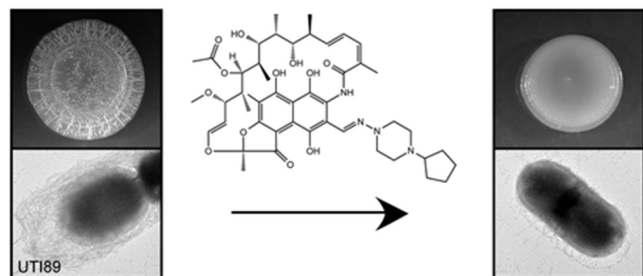
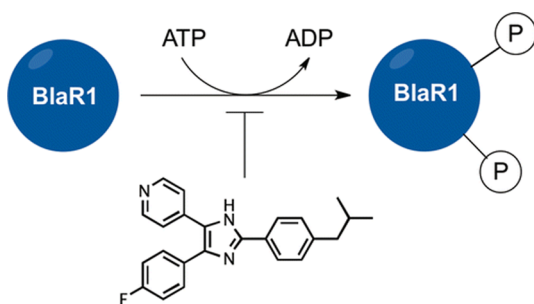


■ BIOFILMS UNSTUCK



Uropathogenic *Escherichia coli* (UPEC) forms biofilms both in vivo and on the surfaces of medical devices such as implants and in-dwelling catheters. Biofilm formation presents a significant challenge for the treatment of UPEC infection as the biofilm serves as a protective environment against antibiotic treatment. UPEC biofilm formation requires adhesion mediated by amyloid fibers termed curli.

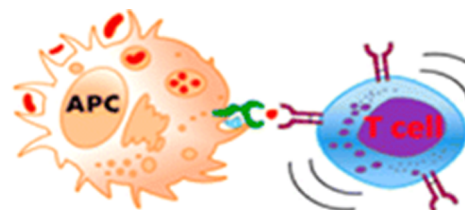
In this issue, Maher et al. (DOI: 10.1021/acsinfecdis.5b00055) develop a high-throughput screen for compounds that inhibit curli-mediated adhesion and biofilm formation of UPEC. The authors screen a compound library to identify a single compound, rifampine, which selectively inhibits biofilm formation via inhibition of the transcriptional regulator CsgD, which mediates expression of curli genes. The work presented here identifies a novel strategy for inhibiting biofilm formation in UPEC.

■ HELPING β -LACTAMS FIGHT MRSA

β -Lactams were once the antibiotics of choice for treatment of *Staphylococcus aureus* infections. Inducible resistance to this class of antibiotics in methicillin-resistant *S. aureus* (MRSA) has rendered the use of all β -lactams obsolete for the treatment of MRSA. Few treatment options now exist for this multidrug-resistant pathogen, the causative agent of approximately 19000 deaths annually in the United States.

In this issue, Boudreau et al. (DOI: 10.1021/acsinfecdis.5b00086) elucidate the molecular mechanisms involved in the initiation of inducible resistance to β -lactams in MRSA. The authors identify the phosphorylation event at the root of the manifestation of antibiotic resistance in MRSA and develop a small molecule inhibitor targeting this phosphorylation event, which restores susceptibility to β -lactams.

■ INCREASING VACCINE SHELF LIFE



Neisseria meningitidis is the primary cause of human bacterial meningitis worldwide. In a region of Africa known as the meningitis belt, seasonal epidemics of *N. meningitidis* serogroup A (MenA) result in case fatality rates of up to 75% in children and adolescents. In 2010, a MenA–tetanus toxoid conjugate vaccine was introduced to the region; however, poor chemical stability in water solution and high sensitivity to temperature prevent the widespread use of this vaccine.

Here, Fallarini et al. (DOI: 10.1021/acsinfecdis.5b00071) develop and synthesize new stable analogues of MenA capsular polysaccharide. The authors conjugate the analogues to a carrier protein and demonstrate their ability to effectively induce immune responses both in vitro and in vivo. These findings demonstrate the potential feasibility for the development of new stable vaccines for prevention of MenA.

Received: September 21, 2015

Published: October 9, 2015