MS09-2-2 Structures and interactions of anti-CRISPR AcrIF2 and AcrIF7
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Abstract
Clustered regularly interspaced short palindromic repeats (CRISPRs) and CRISPR-associated (Cas) proteins form a microbial adaptive immune system against phage infection. In response, phages evolved anti-CRISPR (Acr) proteins to neutralize the host CRISPR-Cas immune system as a counter-defense mechanism. AcrIF2 and AcrIF7 were discovered in *Pseudomonas aeruginosa* phages and strongly inhibit the type I-F CRISPR-Cas systems. Here we determined the structures of AcrIF2 and AcrIF7 using X-ray crystallography and NMR spectroscopy, respectively. AcrIF2 contains a four-stranded antiparallel β-sheet sandwiched between two pairs of antiparallel α-helices. AcrIF7 features a three-stranded antiparallel β-sheet with flanking two α-helices. Both Acr inhibitors interact strongly with the Cas8f:Cas5f heterodimer, which is a subcomplex of the type I-F CRISPR surveillance complex. They compete for the same binding interface on Cas8f without common structural motifs. Our findings suggest that Acrs of divergent origin may have acquired specificity to a common target through convergent evolution of their surface charge configurations.