

The Metabolite Vanillic Acid Regulates *Acinetobacter baumannii* Surface Attachment

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Abstract

The nosocomial bacterium *Acinetobacter baumannii* is protected from antibiotic treatment by acquiring antibiotic resistances and by forming biofilms. Cell attachment, one of the first steps in biofilm formation, is normally induced by environmental metabolites. We hypothesized that vanillic acid, the oxidized form of vanillin, a widely available metabolite with antimicrobial properties, may play a role in *A. baumannii* cell attachment. We first discovered that *A. baumannii* actively breaks down VA through the evolutionarily conserved *vanABKP* genes. These genes are under the control of the repressor VanR, which we show binds directly to VanR binding sites within the *vanABKP* genes bidirectional promoter. VA in turn counteracts VanR inhibition. We identified a VanR binding site and searched for it throughout the genome especially in pili encoding promoter genes. We found a VanR binding site in the pilus encoding *csu* operon promoter and showed that VanR binds specifically to it. As expected, a strain lacking VanR overproduces Csu pili and makes robust biofilms. Our study uncovers the role that VA plays in facilitating the attachment of *A. baumannii* cells to surfaces, a crucial step in biofilm formation. These findings provide valuable insights into a previously obscure catabolic pathway with significant clinical implications.

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