Perspective

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The Mechanism of How Cell Wall Demolishing Antibiotics Kill

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Introduction

New research led by experts from the University of Sheffield in the United Kingdom looks at how bacteria maintain their unique cell walls and how antibiotics can disrupt these maintenance systems to kill bacteria.

Most bacteria have a cell wall, which is a strong and dynamic polymeric net of sugars and amino acids (peptidoglycans) that surrounds them and is necessary for their development, division, and survival. Antibiotics such as beta-lactams—penicillin, methicillin, and cephalosporin—as well as non-beta-lactams like vancomycin—attack the peptidoglycan cell wall to kill bacteria. However, it is unknown how antibiotics accomplish this.

Antibiotics induce two bactericidal mechanisms, one linked to bacterial growth and the other to bacterial division, according to the research team, which included experts from Xiamen University in China, Masaryk University in the Czech Republic, and McMaster University in Canada. These processes rely on two peptidoglycan disrupting enzymes (peptidoglycan hydrolases), which both generate holes in the cell wall that cover the full thickness. As the bacteria multiply, the perforations grow larger, finally killing them [1].

Cecil George Paine, a member of the University's pathology department, performed the first documented penicillin treatment in Sheffield in 1930. Paine used a crude filtrate from a penicillin-producing mould provided by his instructor to cure an eye ailment. "For over 80 years, penicillin and other antibiotics in its class have been a cornerstone of human healthcare, saving nearly 200 million lives. "However, the global rise of antimicrobial resistance puts their usage in jeopardy," said Simon Foster, PhD, of the University of Sheffield's School of Biosciences. "Our research on the superbug MRSA [methicillin-resistant *S. aureus* found that drugs cause microscopic holes in the cell wall to develop, which gradually enlarge as part of growth-related processes, eventually killing the bacteria." We also discovered several of the enzymes involved in the formation of the holes."

Using atomic force microscopy, the researchers defined the peptidoglycan mesh's molecular architecture as an extended hydrogel whose exterior surface is a porous open network but whose inside surface is smoother and denser. In their current investigation, the scientists used this design to identify the significance of coordinated peptidoglycan synthesis and hydrolysis in *S. aureus* growth, division, and antibiotic-induced bactericidal action.

"Peptidoglycan hydrolases cause perforating holes across the cell wall in both methicillin and vancomycin therapy," says the researcher [2].

Methicillin causes plasmolysis, which is a process in which the protoplasm shrinks away from the cell wall. During bacterial cell division, the septum, an expansion of the cell wall, forms down the centre of the cell, with each subsequent partition resulting in a separate cell. Vancomycin prevents inflated and deformed septa, which is caused by methicillin.

The researchers discovered that while inhibiting peptidoglycan hydrolase activity in the presence of cell wall antibiotics reduces killing, hydrolase activity deregulation caused by the loss of teichoic acids—a copolymer that strengthens bacterial cell walls—increases death [3,4]. The scientists also demonstrated the efficiency of a novel combination therapy against *S. aureus* based on these findings and how the enzymes involved in generating holes in the cell wall are controlled. "Our discoveries go to the heart of understanding how existing antibiotics work and open up new paths for therapy development in the face of the global pandemic of antimicrobial resistance," Foster said.

The researchers discovered that in *S. aureus*, autonomous management of cell wall production and hydrolysis results in growth, equilibrium, or death, pointing to new ways to combat this deadly infection [5].

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