



Present Developments in Antimicrobial Resistance and its Clinical Impact

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DESCRIPTION

Antimicrobial Resistance (AMR) has emerged as one of the most irresistible global health challenges of the 21st century [1]. It refers to the ability of microorganisms such as bacteria, viruses, fungi and parasites to survive the effects of medications that once effectively treated infections affected by the of particular concern is bacterial resistance to antibiotics, which has been accelerated by overuse, misuse and a slowing pipeline of new drug development [2]. AMR has serious implications not only for the treatment of infections but also for modern medical practices that rely on effective antibiotics, such as surgery, chemotherapy and organ transplantation [3].

Recent developments in AMR have revealed a troubling increase in resistance across several key pathogens [4]. Strains of bacteria such as *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Acinetobacter baumannii* have developed resistance to multiple antibiotic classes, including last-resort drugs like carbapenems and colistin. The emergence of Carbapenem-Resistant Enterobacteriaceae (CRE) has been especially concerning due to their limited treatment options and high mortality rates. The discovery of the *mcr-1* gene, which confers plasmid-mediated resistance to colistin, has heightened fears, as it can be transferred between different bacterial species and potentially renders infections untreatable [5]. The World Health Organization (WHO) and other health agencies have classified AMR as a global public health threat, emphasizing the need for urgent action. One of the driving forces behind resistance is the inappropriate use of antibiotics in both humans and animals. In many regions, antibiotics are available without prescription and patients often use them improperly, such as not completing prescribed courses or using them for viral infections [6]. In agriculture, antibiotics are commonly used to promote growth and prevent disease in livestock, contributing to the development of resistant strains that can be transmitted to humans through food or direct contact [7].

In clinical settings, AMR is already having a significant impact. Infections caused by resistant bacteria are associated with longer

hospital stays, increased medical costs and higher rates of morbidity and mortality. Common procedures such as cesarean sections, hip replacements and cancer treatments become riskier when the threat of untreatable infections looms. Moreover, the effectiveness of first-line antibiotics is declining, forcing clinicians to use more toxic or expensive alternatives, which may have severe side effects and limited availability [8].

In response to this growing crisis, researchers and healthcare professionals are exploring new strategies to manage and combat AMR. One promising area is the development of rapid diagnostic tools that can quickly identify the causative organism and its resistance profile, allowing for more targeted therapy and reducing unnecessary antibiotic use. Additionally, there has been renewed interest in bacteriophage therapy, which uses viruses that infect bacteria to treat infections. Phages can be highly specific and may serve as a complementary or alternative option to antibiotics [9].

Another important avenue is antimicrobial stewardship, which involves coordinated interventions to improve and measure the appropriate use of antimicrobials. Hospitals and healthcare systems are implementing stewardship programs to ensure antibiotics are used only when necessary and with the correct drug, dose and duration. Public awareness campaigns are also being launched to educate patients on the dangers of misuse and the importance of following medical advice [10].

CONCLUSION

Antimicrobial resistance is an evolving and multifaceted threat that demands a coordinated global response. Its impact on clinical outcomes is already being felt and without immediate and sustained efforts, the effectiveness of modern medicine could be severely compromised. Innovation, education and responsible antibiotic use are key pillars in the fight against AMR and ongoing collaboration between governments, healthcare providers, researchers and the public is essential to safeguarding the future of infection treatment.

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REFERENCES

1. Pellegrino S, Ronda L, Annoni C, Contini A, Erba E, Gelmi ML, et al. Molecular insights into dimerization inhibition of c-Maf transcription factor. *Biochim Biophys Acta*. 2014;1844(12):2108-2115.
2. Ciechanover A. The ubiquitin-proteasome pathway: On protein death and cell life. *EMBO J* 1998;17(24):7151-7160.
3. Hershko A, Ciechanover A. The ubiquitin system. *Annu Rev Biochem*. 1998;67:425-479.
4. Zheng N, Schulman BA, Song L, Miller JJ, Jeffrey PD, Wang P, et al. Structure of the Cul1-Rbx1-Skp1-F boxSkp2 SCF ubiquitin ligase complex. *Nature*. 2002;416(6882):703-709.
5. Chen RH. Cullin 3 and its role in tumorigenesis. *Adv Exp Med Biol*. 2020;1217:187-210.
6. Cullinan SB, Gordan JD, Jin J, Harper JW, Diehl JA. The Keap1-BTB protein is an adaptor that bridges Nrf2 to a Cul3-based E3 ligase: Oxidative stress sensing by a Cul3-Keap1 ligase. *Mol Cell Biol*. 2004;24(19):8477-8486.
7. Lee DF, Kuo HP, Liu M, Chou CK, Xia W, Du Y, et al. KEAP1 E3 ligase-mediated downregulation of NF-kappaB signaling by targeting IKK β . *Mol Cell*. 2009;36(1):131-140.
8. Lee Y, Chou TF, Pittman SK, Keith AL, Razani B, Wehl CC. Keap1/Cullin3 modulates p62/SQSTM1 activity via UBA domain ubiquitination. *Cell Rep*. 2017;19(1):188-202.
9. Wu JT, Lin HC, Hu YC, Chien CT. Neddylation and deneddylation regulate Cul1 and Cul3 protein accumulation. *Nat Cell Biol*. 2005;7(10):1014-1020.
10. Zhao M, Quan Y, Zeng J, Lyu X, Wang H, Lei JH, et al. Cullin3 deficiency shapes tumor microenvironment and promotes cholangiocarcinoma in liver-specific Smad4/Pten mutant mice. *Int J Biol Sci*. 2021;17(15):4176-4191.