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Antibiotic Residue, Resistance and Novel Strategy to Develop Antimicrobial Agents

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Different drugs use in livestock production has created a build-up of chemicals in the food chain and the environment. Among different drugs, the use of low levels of antibiotics as growth promoters in animal feeds and indiscriminate use of antibiotics to treat human or animal infections are thought to be the cause of an alarming increase in antibiotic resistance among bacteria. Antibiotic residue in meat, milk and its different processed products and resistant of different microorganism have been posing increasingly serious concern to all involve in veterinary and medical science. These problems are now in high alarming state and scientists across the world are now focusing on alternative antimicrobial agents.

Antimicrobial peptides (AMP) are prevalent throughout the nature as a part of the intrinsic defenses of most organisms and provide innate and adaptive immunity. AMP can be used as blueprint for developing novel antimicrobial agents. In order to design the antimicrobial peptides, the most common approach is either to retrieve the required genomic sequences from different databases or to sequence the novel antimicrobial peptide gene. After that prediction of peptide is done from all these sequences to find out the consensus region, specific pattern of amino acid distribution and trace out the mature peptide for synthesis. On the basis of amino acid sequence of AMPs, various analogues can also be prepared by replacing with desired potent amino acid. In the present experiment a number of antimicrobial peptide has been designed on the basis of predicted peptide from the genomic sequences of buffalo (*Bubalus bubalis*) and synthesized using solid phase Fmoc chemistry. Peptides were evaluated for its antimicrobial activity and minimum inhibitory concentration (MIC). Cyto-toxicity and structural analysis of the synthesized peptides were done using Fluorescent Activated Cell Shorter (FACS) and Circular Dichroism (CD) Spectropolarimeter respectively. Designing and synthesis of antimicrobial peptides represents a promising strategy for the development of a novel antimicrobial agents.

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Osteopontin is increased in cystic fibrosis and can skew the functional balance between ELR-positive and ELR-negative CXC-chemokines

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Cystic fibrosis (CF) is characterized by severe and longstanding bacterial infections of the airways. In addition, there is a chronic and dysregulated inflammation characterized by a massive influx of neutrophils to the lungs. Even though the cause of the disease is known (i.e. mutations in the CFTR gene), the mechanisms resulting in the compromised host defense are not fully understood. The glycoprotein osteopontin (OPN) plays a key role in several states of disease associated with inflammation, for example by recruitment of neutrophils but its expression and possible roles in CF have hitherto not been investigated. In this study, upregulation of OPN was detected in bronchial epithelial cells and in sputum of CF patients. In vitro, OPN bound to the ELR-negative antibacterial chemokines MIG/CXCL9, IP-10/CXCL10 and I-TAC/CXCL11 through electrostatic interactions. In contrast, no binding was found to the neutrophil-recruiting ELR-positive chemokines GRO-β/CXCL2, GCP-2/CXCL6 and IL-8/CXCL8. Functional consequences of the binding were that OPN inhibited the bactericidal activity of MIG/CXCL9, IP-10/CXCL10 and I-TAC/CXCL11 and also reduced their activation of the CXCR3-receptor that is expressed on NK cells and cytotoxic T cells. In contrast, OPN did not reduce the receptor-activating properties of GRO-β/CXCL2, GCP-2/CXCL6 and IL-8/CXCL8 against neutrophils. Taken together, upregulation of the neutrophil-recruiting glycoprotein OPN in CF may contribute to the dysregulated inflammation seen in CF, resulting in impaired host defense activities of ELR-negative chemokines (that serve as innate antibiotics and recruit NK cells and cytotoxic T cells), instead promoting an excessive influx of neutrophils, contributing to progress of the disease.

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