TITLE: MAM7: An Inhibitor of Bacterial Infection With a Novel Mechanism of Action
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TECHNOLOGY: Biologic
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SUMMARY:

This technology describes a bacterial adhesion inhibitor (MAM7) that blocks host tissue infection by a broad spectrum of gram-positive (gram+) and gram-negative (gram-) bacterial pathogens without interfering with the natural wound healing process.

In over half a century, antibiotic use caused a sharp decrease in infectious disease related deaths and increased life expectancy by about 30 years. Unfortunately, their mechanisms of action (kill or inhibit bacterial growth), are strong selective-pressure resulting in the emergence of resistance. This problem is amplified by antibiotic misuse and by the common practice of using antibiotics as growth promoters for animals farmed for human consumption.

To start an infection, bacteria need to bind tightly to host tissues. For this purpose, a wide range of gram-negative pathogenic bacteria use Multivalent Adhesion Molecule (MAM) 7.

Chronic wounds including diabetic ulcers, venous ulcers, pressure ulcers, and burns, are typically infected by biofilm-producing bacteria and these infections usually induce a low grade persistent inflammatory response; together they contribute to impede wound healing.

At least 2 million people become infected with antibiotic-resistant bacteria every year in the US, and at least 23,000 people die each year as a direct result of these infections. Also, there are more than 1 million burn injuries causing more than 100,000 hospitalizations every year in the US, and ~75% of the burn-patient mortality is associated with infections.

Few new anti-bacterials have been approved since 2000, and bacteria develop resistance at alarming speeds. This has led experts to warn of an upcoming ghastly “post-antibiotic era.” Therapeutic approaches with new mechanisms of action are needed.

The present technology describes the use of recombinant MAM7 as an efficacious broad-spectrum drug to prevent bacterial adhesion to host tissue, including drug-resistant and fastidious pathogens such as MRSA (gram+) and P. aeruginosa (gram-).

As proof of concept in a burn-infection mouse model, MAM7 was shown to prevent colonization, to not interfere with the wound healing process, and to not induce inflammatory or immune responses. Also, since MAM7 does not affect pathogen survival, it is less likely to induce a strong selective-pressure leading to development of resistance.

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