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The anthrax capsule:Target for new vaccines and novel therapeutics

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The polyglutamic acid capsule of *Bacillus anthracis*, the causative agent of anthrax is a well-established virulence factor, conferring antiphagocytic properties on the bacillus. We have shown that the capsule released from the bacillus surface may also contribute to virulence. In our research we have targeted the anthrax capsule for the development of medical countermeasures, first using the capsule as a vaccine similar to successful efforts with other bacteria and secondly, by developing a novel therapeutic against the capsule. Our experiments showed that a capsule vaccine is protective in the mouse model and its efficacy could be enhanced by conjugation to a protein carrier. In initial experiments using high challenge doses, a capsule conjugate vaccine was not protective in the rabbit but did show some protection in the nonhuman primate. Subsequent experiments showed complete protection in the nonhuman primate against lethal aerosol challenge. This suggests the capsule may be useful as an addition to a protective antigen-based vaccine. We are also developing the use of the *B. anthracis* capsule-depolymerizing enzyme, CapD, as a therapeutic. We demonstrated that *in vitro* treatment of the encapsulated anthrax bacillus with CapD enzymatically removed the capsule from the bacterial surface making it susceptible to phagocytic killing. Initial experiments *in vivo* showed that CapD could be used successfully to treat experimental anthrax infections. Such a novel approach to target the capsule virulence factor might be of value in the treatment of infections due to multidrug-resistant strains.

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Fundamental properties of the ultraviolet-induced mutagenesis: A theoretical background

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The usage of ultraviolet (UV) light is an effective measure that prevents spreading of bacterial infection in hospitals and biological laboratories. UV radiation is highly damaging to the wide range of bacterial species and viruses but is relatively safe to humans in low doses. The sensitivity of bacterial cells to this type of exposure is determined mainly by their lower ability to repair UV-induced DNA lesions. However, the limited fraction of bacteria can survive through the successful recovery of introduced DNA lesions with mutagenic or non-mutagenic outcome. In this regard, one of the most important biological mechanisms governing the cell's fate is the SOS response. Upon introducing damage to their genomes, bacteria such as *E. coli* activate the SOS network enabling cells to bypass DNA damage via specifictranslesion synthesis pathway. The current talk discusses the fundamental properties of the UV-induced SOS response on the basis of mathematical model developed by the author. The model summarizes recent findings in the field and provides a background for better understanding of gene regulation of the bacterial UV mutagenesis. It describes a whole sequence of the events leading to the fixation of the primary DNA lesion as a point mutation. The model also shows a possible mechanistic explanation of links between the SOS system and other DNA repair pathways. Finally, the talk review possible implications of recently established fundamental properties of the UV-mutagenesis in treatment of bacterial infections.

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