TREATING LIFE-THREATENING PURPURA FULMINANS

Novel approach to treat purpura fulminans: small molecules inhibiting bacterial adhesion to blood vessels

ERGANEO
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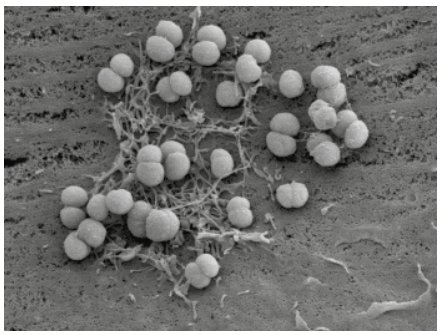
PRESENTATION

Purpura fulminans (PF) is an uncommon but fatal disorder characterized by rapidly progressive thrombosis with hemorrhagic infarction, disseminated intravascular coagulation and multi-organ failure, which leads to death in 30% of patients and often severe sequelae in survivors. PF is a feature of severe sepsis in response to infection by a wide range of bacteria species, among which *Neisseria meningitides, Streptococcus pneumoniae and Escherichia coli*. Bacterial colonization of human blood vessel cells is a hallmark of the PF. Rapid administration of antibiotics effectively treats sepsis, but it has only moderate effect on thrombosis development.
The present offer relates to a small molecules family to treat PF. *In vitro* evaluation of these molecules revealed that they quickly disaggregate bacterial colonies on human endothelial cells, acting on the bacteria type IV pili. The molecules efficacy has been confirmed *in vivo* using a human skin xenograft in a mouse model, where a drastic reduction of the bacteria colonization of the vascular system is shown. A novel therapeutic strategy can be considered, which would use these molecules to inhibit bacterial virulence without killing the bacteria. The lack of selective pressure should therefore prevent the emerging of resistance towards these small molecules.



Purpura fulminans - Neisseria meningitidis - Bacterial adhesion - Type IV pili

COMPETITIVE ADVANTAGES

- Novel therapeutic strategy through bacterial virulence inhibition
- The lack of selective pressure should prevent the emerging of resistance towards the small molecules
- Can increase efficacy of current antibiotic treatment

INTELLECTUAL PROPERTY

Priority patent application, filed on November 5th 2016

PUBLICATIONS

Bernard *et al.*, Nature Med, 2014

APPLICATIONS

Treating purpura fulminans

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DEVELOPMENT PHASE

- PoC *in vivo* on human skin xenograft in immunodeficient mouse model