Compound for the treatment and/or prevention of sepsis

The Spanish Research Council (CSIC), the Health Research Institute “Fundación Jimenez Diaz” and Autonomous University of Madrid have patented the use of a protein that comprises the catalytic domain of the cytotoxic necrotic factor of Yersinia pseudotuberculosis (CNFY), for the treatment and/or prevention of septicemia and/or edema associated with septicemia.

Pharmaceutical companies interested in patent licensing are being sought for the development of a pharmaceutical composition for the treatment of sepsis.

An offer for Patent Licensing

Prevention of sepsis

Sepsis is a life-threatening disease caused by a body reaction to bacterial blood infection, although it can also be caused by other pathogens, such as fungi or viruses. Usually, the body releases inflammatory stimuli in the bloodstream to fight infections, but during sepsis, the infection becomes systemic and the response to the inflammatory stimuli very strong and out of control, which can damage organs. One of the most devastating effects of sepsis is the acute increase in vascular permeability that results in massive tissue edema that can lead to organic failure. The component of the bacterial cell wall known as endotoxin or lipopolysaccharide (LPS) is one of the main bacterial molecules that trigger this systemic inflammatory response.

The endothelium is a barrier of cells that lies in the inner side of vessels and the main regulator of vascular permeability. The current therapies for treating sepsis include antibacterials, antibodies, small molecules and peptides, protein C, supportive oxygen therapy, intravenous fluids and drugs that increase blood pressure; however, none of these treatments target endothelial barrier dysfunction to prevent organ edema and failure. Our compound, however, can reduce edema associated with this pathology.

Main innovations and advantages of the compound

- Activates the master regulators of the endothelial cytoskeleton, RhoA, RhoB and RhoC, in the proximity of endothelial cell-to-cell contacts, stabilizes adherens junctions and improves barrier function of human endothelium in vitro.
- Reduces the permeability increase in response to inflammatory mediators in vivo.
- Reduces edema in liver, lung and kidney in response to intraperitoneal injection of LPS as an experimental model of sepsis.
- Reduces vascular collapse in an experimental model of sepsis and decreases inflammatory cytokines in blood.

Patent Status

Priority patent application suitable for international extension

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