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Host Response and Immunity to Yersenia pestis Infection

Project Number
5R01AI129996-04

Contact PI/Project Leader
ANDERSON, DEBORAH M

Awardee Organization
UNIVERSITY OF MISSOURI-COLUMBIA

Description

Abstract Text

PI: Anderson, Deborah M Project Summary "Host response and immunity to Yersinia pestis infection" Project Summary Type I interferons are expressed by eukaryotic cells upon intracellular invasion by microbial pathogens and they induce a potent anti-viral response. Yet during bacterial infection, expression of type I IFN often leads to a pathologic response that depletes populations of immune effector cells necessary to mediate clearance. Our laboratory has shown that type I IFN signaling contributes to neutrophil depletion during infection by Yersinia pestis, a Gram-negative bacterium that is the causative agent of the plague. Bubonic plague is a highly **infectious** vector borne **disease** that can be transmitted through the respiratory route and disseminated through the vasculature of its victims. Septicemic and pneumonic plagues involve the rapid development of an uncontrolled systemic inflammatory response that causes the clinical collapse of the patient, even with antibiotic treatment. These three forms of plague have been responsible for three major pandemics and still cause annual cases of human **disease** with a high mortality rate worldwide including a hotspot in the Southwestern United States. To date, little about the host responses that directly or indirectly contribute to the progression of plague. Such responses may present new strategies to approach the post- symptomatic treatment of plague and other acute inflammatory diseases. In this application, we propose to study interactions between phagocytic cells and Y. pestis that are responsible for inducing inflammatory responses that contribute to the progression of infection in a murine model. We have identified the broadly conserved Toll-like receptor 7 (TLR7) as activated during infection by wild type Y. pestis. Activation of TLR7 by Y. pestis triggers a non-canonical signaling pathway that induces the expression of type I IFN and its downstream IFN stimulated genes which subsequently interfere with the neutrophilic response and promote the progression of **disease**. In this project, we aim to understand the molecular signaling events of this novel pathway and their role during infection with Y. pestis. Our long term goal is to use the information gained from this program to better understand innate immune response to bacterial infection and develop host-targeted therapeutics that broadly protect from acutely inflammatory **infectious** diseases such as the infamous pneumonic plague.

Public Health Relevance Statement

PI: Anderson, Deborah M Project Narrative Host response and immunity to Yersinia pestis infection Project Narrative The long term goal of our research is to better understand the human innate immune response to bacterial infection in order to develop therapeutics that target harmful host responses that are promoting disease. To achieve this, we are studying host interactions with Yersinia pestis, the causative agent of plague. Plague is a rapidly progressing lethal infection that is facilitated by the mammalian innate immune response. Our research has discovered a novel pathway for activating expression of type I IFN that contributes to the progression of plague. We hypothesize that inhibition of this pathway could be a novel therapeutic strategy for the treatment of pneumonic plague as well as other bacterial diseases.

NIH Spending Category

Biodefense **Emerging Infectious Diseases** **Infectious Diseases** **Lung**

Pneumonia **Pneumonia & Influenza** **Rare Diseases** **Vector-Borne Diseases**

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Contact PI/Project Leader
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Generations Genes Goals Gram-Negative Bacteria Growth Human IRF3 gene Immune Immune Evasion Immune response Immunity Infection Inflammation Inflammatory Inflammatory Response Inhalation Innate Immune Response Interferon Type I Interferons Investigation

Read More

Details

Contact PI/ Project Leader

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Not Applicable

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City
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Country
UNITED STATES (US)

Department Type
VETERINARY SCIENCES

Organization Type
SCHOOLS OF VETERINARY MEDICINE

State Code
MO

Congressional District
04

Other Information

FOA
PA-16-160

Study Section
Host Interactions with Bacterial Pathogens Study Section[HIBP]

Fiscal Year
2020

Award Notice Date
09-April-2020

Administering Institutes or Centers
NATIONAL INSTITUTE OF ALLERGY AND INFECTIOUS DISEASES

DUNS Number
153890272

CFDA Code
855

Project Start Date
08-May-2017

Project End Date
30-April-2022

Budget Start Date
01-May-2020

Budget End Date
30-April-2021

Project Funding Information for 2020

Total Funding
$365,705

Direct Costs
$250,000

Indirect Costs
$115,705

Year	Funding IC	
2020	NATIONAL INSTITUTE OF ALLERGY AND INFECTIOUS DISEASES	$365,705

NIH Categorical Spending

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Funding IC

Thank you for your feedback!

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Host Response and Immunity to Yersenia pestis Infection

Project Number
5R01AI129996-04

Contact PI/Project Leader
ANDERSON, DEBORAH M

Awardee Organization
UNIVERSITY OF MISSOURI-COLUMBIA

Diseases; Lung; Pneumonia; Pneumonia & Influenza; Rare Diseases; Vector-Borne Diseases;

Sub Projects

No Sub Projects information available for 5R01AI129996-04

Publications

No Publications available for 5R01AI129996-04

Patents

No Patents information available for 5R01AI129996-04

Outcomes

The Project Outcomes shown here are displayed verbatim as submitted by the Principal Investigator (PI) for this award. Any opinions, findings, and conclusions or recommendations expressed are those of the PI and do not necessarily reflect the views of the National Institutes of Health. NIH has not endorsed the content below.

No Outcomes available for 5R01AI129996-04

Clinical Studies

No Clinical Studies information available for 5R01AI129996-04

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Related News Releases

No news release information available for 5R01AI129996-04

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Host Response and Immunity to Yersenia pestis Infection

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5R01AI129996-04	ANDERSON, DEBORAH M	UNIVERSITY OF MISSOURI-COLUMBIA

No Similar Projects information available for 5R01AI129996-04