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Memory T cell protection of reproductive health following influenza infection

Project Number Contact PI/Project Leader 5R21HD093948-02 STRUTT, TARA MARLENE

Awardee Organization
UNIVERSITY OF CENTRAL
FLORIDA

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Abstract Text

Project Summary Respiratory tract infections are a leading cause of death worldwide. Compromised cell-mediated immune responses, low vaccine efficacy, and poor vaccine uptake all increase the likelihood that serious infection will ensue in pregnant women following exposure to respiratory viruses such as Influenza A virus (IAV); approximately 80% of pregnant women are at high risk for influenza-associated complications. Our best defense is vaccination. Vaccine-induced protection is mediated by immunological memory. The majority of immunological memory studies to date have focused on the differentiation, survival, and delineation of the antipathogen defense mechanisms employed by pathogen-specific T cells under normal physiological circumstances. The impact of pregnancy on the persistence and functional recall potential of memory T cells is surprisingly unknown. The overarching hypothesis of this proposal is that IAV-specific T cell memory generated prior to conception will mediate potent universal, heterosubtypic protection throughout pregnancy. This hypothesis arose from our observations that memory effector CD4 T cells isolated from influenza infected lungs express low levels of estrogen and progesterone hormone receptors in comparison to primary effector CD4 T cells. Pregnancy hormones are well known to have detrimental impacts on the generation and function of primary anti-viral immune responses. Preliminary findings herein show that heterosubtypic recall responses are indeed protective in gravid animals primed prior to conception. In the first aim, the persistence of IAV-specific CD4 and CD8 memory T cells during pregnancy, as well as their ability to differentiate into the many protective subsets that mediate diverse and potent anti-viral effector functions will be determined. Our prior work has shown that memory CD4 T cells regulate both local and systemic anti-viral inflammatory responses, and, additionally, mobilize innate lymphoid cells during pathogen infection. Innate lymphoid cells, which function in tissue repair, have recently been shown to be present in placental tissue. In the second aim, we will thus elucidate the role of IAV-specific memory T cells in preserving both healthy placental and fetal development through the regulation of systemic innate inflammatory and cellular responses following IAV infection. The insight gained from the proposed, 'reverse-mechanism', studies will advance our ability to develop improved vaccines that elicit memory T cells capable of orchestrating protective anti-viral defenses during pregnancy. This research may also lead to the development of novel strategies to treat expectant mothers suffering from serious influenza for which current therapeutic options are lacking. Respiratory tract infection with Influenza A virus remains a serious public health concern, particularly for pregnant women, who have suffered disproportionally high morbidity and mortality in past epidemic and pandemic seasons. The proposed study has the potential to make a broad and significant translational impact on human reproductive health.

Public Health Relevance Statement

Relevance Pregnant women and their developing children are amongst those at the highest risk for serious influenza infection-associated complications and death. The best defense against viral pathogens, such as Influenza A virus, is vaccination-induced immunological memory. The insight gained from the proposed studies will advance our ability to develop improved vaccines that elicit immunity capable of preventing the adverse maternal and fetal outcomes of viral infection during pregnancy.

NIH Spending Category

Contraception/Reproduction Emerging Infectious Diseases Immunization Immunotherapy

Infectious Diseases Influenza Pediatric Pneumonia & Influenza Pregnancy Prevention

Vaccine Related Women's Health

Project Terms

Activities of Daily Living Animals Antiviral Agents B-Lymphocytes CD4 Positive T Lymphocytes **Cause of Death** CD8B1 gene **Cell Survival Cell physiology** Cells **Cellular Immunity Cessation of life** Child Conceptions Coupled Cytoprotection **Data Defense Mechanisms Development Epidemic Estrogen Receptors Female Estrogens Exposure to Fetal Development Fetal health Generations Genes Gravid High Risk Woman Hormone Receptor Immunity** Histopathology **Hormones** Human Immune response Infection **Immunologic Memory Immunosuppressive Agents Impairment** Infant Inflammation **Inflammatory Response** Inflammatory Influenza Influenza A virus Lead Lung

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Contact PI/Project Leader

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01-August-2018

31-July-2021

01-August-2019

Contact

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Organization

Project Number

5R21HD093948-02

Department Type State Code Name **OTHER BASIC SCIENCES** UNIVERSITY OF CENTRAL FLORIDA FL

Organization Type City **Congressional District ORLANDO SCHOOLS OF MEDICINE** 07

Country **UNITED STATES (US)**

Other Information

FOA Administering Institutes or Centers **Project Start EUNICE KENNEDY SHRIVER** PA-16-444 Date NATIONAL INSTITUTE OF CHILD Study Section **HEALTH & HUMAN DEVELOPMENT Project End Date**

Immunity and Host Defense Study **DUNS Number** CFDA Code Section[IHD] **Budget Start** 150805653 865

Award Notice Date Fiscal Year Date 12-July-2019 2019 **Budget End Date**

31-July-2021

Project Funding Information for 2019

Indirect Costs Total Funding Direct Costs \$186,250 \$125,000 \$61,250

FY Total Cost b Year **Funding IC**

2019 EUNICE KENNEDY SHRIVER NATIONAL INSTITUTE OF CHILD HEALTH & HUMAN DEVELOPMENT \$186,250

NIH Categorical Spending

Funding IC

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FY Total Cost by IC

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EUNICE KENNEDY SHRIVER NATIONAL INSTITUTE OF CHILD HEALTH & HUMAN DEVELOPMENT \$186,250 Contrac Emergir Disease Immuno Diseas€ Pediatri Influenz Prevent Women

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