Title:
Immune Function and Infection Risk After Pediatric Cardiac Surgery

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Rising Stars of Research and Scholarship Invited Student Poster Session 1

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References:
Greathouse, K. C. & Hall, M. W. (2016). Critical illness induced immune suppression: Current state of the science. American Journal of Critical Care, 25(1), 85-92. Cornell, T. T. , Sun L., Hall, M. W., Gurney, J. G., Ashbrook, M. J, Ohye, R. G., & Shanley, T.P. (2012). Clinical implications and molecular mechanisms of immunoparalysis after cardiopulmonary bypass. Journal of Thoracic and Cardiovascular Surgery, 143(5), 1160-1166.

Abstract Summary:
Immune function in the context of pediatric critical illness, with an emphasis on those undergoing cardiac surgery will be described. Additionally, specific immune function measures, and techniques to determine changes in immune function and how they relate to important outcomes will be explained.
Learning Activity:

LEARNING OBJECTIVES	EXPANDED CONTENT OUTLINE
The learner will be able to understand the differences between innate and adaptive immune function and how these relate to risk for infection after cardiac surgery.	Description of innate and adaptive immune function, their relevance in the context of pediatric cardiac surgery, and how changes in immune function relate to risk for infection.
The learner will be able to describe how immune function in measured in the context of critical illness, specifically pediatric cardiac surgery.	Description of specific innate and adaptive immune function measures, why they were chosen and relevance to the pediatric cardiac surgery population.

Abstract Text:

Infections occur in up to 20% of pediatric patients who undergo cardiac surgery. Post-operative infections are a significant source of additional morbidity, and can result in re-operations, lengthen hospital stays, and necessitate longer durations of mechanical ventilation and inotropic support. Proper immune function is vital for prevention of post-operative infections. Two arms of the immune system operate together to maintain suitable function. First, the innate immune system serves as the key regulator of the inflammatory response that activates other immune cells and facilities repair of injured tissues. Second,

the adaptive immune response confers specificity to the immune response and their cells typically require presentation of antigen by a member of the innate immune system in order to become activated.

The combination of local trauma, cardiopulmonary bypass as well as pulmonary and myocardial reperfusion results in a significant systemic inflammatory response post-cardiac surgery. The compensatory anti-inflammatory response syndrome is activated to counteract this pro-inflammatory surge. This involves the elaboration of anti-inflammatory mediators resulting in down-regulation of the pro-inflammatory response and inhibition of leukocyte function. If the compensatory anti-inflammatory response is severe and persistent, it represents a form of secondary immune deficiency, which can profoundly affect immune function. This immune suppression has been demonstrated in critically ill children and adults following sepsis, trauma, and severe viral infections, and is also evident post-cardiac surgery.

It is currently unknown how cardiac surgery impacts both innate and adaptive immune function in pediatric patients. Therefore, the purpose of this study is to gather data in pediatric cardiac surgery patients and demonstrate the incidence of cardiac surgery induced immune suppression. In addition, this study will be the first of its kind to evaluate innate and adaptive immune function and determine their relevance to post-surgical outcomes, namely infection. The study described will test the hypotheses that: 1) Cardiac surgery will be associated with a reduction in innate and adaptive immune function in comparison to pre-operative function, with those undergoing cardiopulmonary bypass having the most severe reduction, and 2) Severe, early reductions in innate and adaptive immune function will be associated with increased risk for the development of nosocomial infection.