

Abstract Directions for the 2025 WV-INBRE Summer Research Symposium

ABSTRACT DEADLINE Friday, July 11, 2025. Send your abstract to Sheila Anderson by e-mail at the following e-mail address: sdanderson@hsc.wvu.edu. Please note that if your abstract is not received by the deadline, it will not appear in the Symposium booklet.

Specific format checklist: (for the sake of consistency it is VERY IMPORTANT that you follow the format in the sample abstract shown below EXACTLY AS SHOWN); please pay attention specifically to the following points:

1. Use Arial, 12 pt. font for all text – including title, authors & affiliations, and body of the text.
2. Title – put the title in **boldface** type
3. Authors and affiliations– put in regular, italicized type
4. Text of abstract – the text should be written in a **SINGLE paragraph**; in regular (not boldface or italicized) type; there is a **250 word limit** for the body of text.
5. At the end of the abstract add the following statement in parentheses (as shown): **(Supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence)**

IMPORTANT – YOUR ABSTRACT MUST BE APPROVED BY YOUR MENTOR before sending it in –please indicate this approval and cc your mentor in your abstract submission e-mail). (MENTORS: Please be certain to approve your intern's abstract before it is submitted.)

SAMPLE ABSTRACT format for the 2025 WV-INBRE Summer Program:

Disparate Primary and Secondary Allospecific CD8+ T Cell Cytolytic Effector Function in the Presence or Absence of Host CD4+ T Cells. *Phillip H. Horne†, Mitchel A. Koester*, Kartika Jayashankar*, Keri E. Lunsford†, Heather L. Dziema*, Ginny L. Bumgardner*†.* *Department of Surgery, Comprehensive Transplant Center, The Ohio State University Medical Center, Columbus, OH; †Integrated Biomedical Science Graduate Program, College of Medicine, The Ohio State University, Columbus, OH

The role of CD4+ T cells in promoting CD8+ T cell effector activity in response to transplant antigens in vivo has not been reported. We utilized a hepatocellular allograft model known to initiate both CD4-dependent and CD4-independent rejection responses to investigate the contribution of CD4+ T cells to the development, function, and persistence of allospecific CD8+ T cell effectors in vivo. Complete MHC mismatched hepatocellular allografts were transplanted into C57BL/6 (CD4-sufficient) or CD4 KO (CD4-deficient) hosts. The development of in vivo allospecific cytotoxicity was determined by clearance of CFSE-labeled target cells. CD8+ T cell cytotoxic effector activity was enhanced in response to allogeneic hepatocellular grafts with a greater magnitude of allocytotoxicity and a prolonged persistence of CTL effector activity in CD4-sufficient hosts compared to CD4-deficient hosts. Cytolytic activity was mediated by CD8+ T cells in both recipient groups. In response to a second hepatocyte transplant, rejection kinetics were enhanced in both CD4-sufficient and CD4-deficient hepatocyte recipients. However, only CD4-sufficient hosts developed recall CTL responses with an augmented magnitude and persistence of allocytotoxicity in comparison to primary CTL responses. These studies show important functional differences between alloreactive CD8+ T cell cytolytic effectors which mature in vivo in the presence or absence of CD4+ T cells. **(Supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence)**