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YOU ARE INVITED TO ATTEND THE DEFENSE OF THE DOCTORAL DISSERTATION

"DYSREGULATION OF MONOCYTES PRIOR TO LIVER TRANSPLANT IS ASSOCIATED WITH INCREASED RISK OF POST-TRANSPLANT MORTALITY"

By

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Thursday, May, 21 2026
Medical Science Building, B610
9:00 A.M.

Join Zoom presentation
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Meeting ID: 913 9462 6441
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ABSTRACT

Liver transplantation (LT) is the only definitive cure for end-stage liver disease; however, due to the shortage of available donors, only a small portion of patients receive donor livers; therefore, liver allocation is prioritized for patients with the greatest need (i.e., the “sickest first”), who might derive the greatest benefit from transplant. Unfortunately, the sickest patients also tend to be at the highest risk for sepsis and death following transplant due to their illness severity at transplant. We have identified that *pre*-transplant immune dysfunction is a major risk factor in *post*-transplant morbidity and mortality. This risk can be predicted using a validated *pre*-transplant biomarker panel, the Liver Immune Frailty Index (LIFI), which stratifies patients into risk tertiles for *post*-LT mortality based on plasma MMP3 and Fractalkine levels. One-year *post*-LT mortality rates are 63% for LIFI-high versus 1.8% for LIFI-low (c-statistic 0.83). Although LIFI demonstrates strong predictive performance, the mechanisms of cirrhosis-associated immune dysfunction that increase the risk of *post*-liver transplantation mortality remain undefined. To investigate immune dysfunction at LT, plasma and peripheral blood mononuclear cells (PBMCs) were collected immediately prior to LT (T0), and patients were stratified based on LIFI risk tertile. Transcriptomic analysis demonstrated differentially expressed gene profiles, with cell death, cell migration, and extracellular matrix degradation enriched in LIFI-high. Further assessment with scRNAseq demonstrates significant dysregulation in the monocyte compartment with impaired activation and enhanced immunosuppression. In particular, LIFI-high is associated with decreased expression and downstream signaling of pro-inflammatory IL-6 and elevated anti-inflammatory IL-10, indicating an inappropriate shift toward an anti-inflammatory immune response. Dysregulation of the inflammatory response is suggested by increased TNFR1-mediated cell death in monocytes, as well as diminished autophagy activity. Persistence of monocyte dysregulation early after LT could contribute to ongoing immune dysfunction resulting in increased susceptibility to infection and death early *post*-transplant.