

Title: Diabete-mediated IL-17A enhances the progression of diabetic retinopathy

Name: Patricia R. Taylor, PhD
Case Western Reserve University School of Medicine, Cleveland OH, USA

Diabetes is one of the most prevalent and growing health issues worldwide. Further, diabetes leads to progressive complications such as diabetic retinopathy, which is the leading cause of blindness within the working-age population of the Western world. Interleukin (IL)-17A is an important cytokine in the promotion and progression of diabetes. However, the mechanism by which IL-17A contributes to diabetes-mediated capillary non-perfusion has yet to be determined. In the current study, IL-17A was detected in the retina and Th17 cells were adhered to the retinal vasculature in STZ-induced diabetic mice, while the IL-17A receptor was expressed on multiple retina cells. Diabetes-induced retinal endothelial cell death and capillary degeneration were significantly lower in IL-17A$^{-/-}$ mice. Through ex vivo studies, it was determined that retinal endothelial cell death occurs through an IL-17A/IL-17R →Act1/FADD (Fas-associated protein with death domain) signaling cascade, which causes caspase-mediated apoptosis. These findings establish a novel pathologic role for Th17 cells in the early vasoregressive process of retinal capillary degeneration, and also identify an IL-17A-dependent apoptotic mechanism that leads to the early onset of non-proliferative diabetic retinopathy.

Biography

Dr. Patricia R. Taylor is an Assistant Professor of Ophthalmology and Visual Sciences at Case Western Reserve University School of Medicine and a Research Health Scientist at Louis Stokes VA Medical Center in Cleveland, Ohio, USA. Dr. Taylor studies inflammatory components involved in the onset and progression of diabetic retinopathy; prominently IL-17A. She has been very active teaching, speaking in different venues, and writing about different inflammatory pathologies with a focus on diabetes, immune dysfunction, and diabetic retinopathy.

Presenting author details

Full name: Patricia R. Taylor, PhD
Contact number: (216) 368-5821
Email: prt11@case.edu
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