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Endothelial colony-forming cells regulates neurovascular trophic effect via paracrine mechanism

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Abstract
Vascular abnormalities are a common component of eye diseases that often lead to vision loss. Vaso-obliteration is associated with inherited retinal degenerations, since photoreceptor atrophy lowers local metabolic demands and vascular support to those regions is no longer required. Given the degree of neurovascular crosstalk in the retina, it may be possible to use one cell type to rescue another cell type in the face of severe stress, such as hypoxia or genetically encoded cell-specific degenerations. Here, we show that intravitreally injected human endothelial colony-forming cells (ECFCs) that can be isolated and differentiated from cord blood in xeno-free media collect in the vitreous cavity and rescue vaso-obliteration and neurodegeneration in animal models of retinal disease. Furthermore, we determined that a subset of the ECFCs was more effective at anatomically and functionally preventing retinopathy; these cells expressed high levels of CD44, the hyaluronic acid receptor, and IGFBPs (insulin-like growth factor-binding proteins). Injection of cultured media from ECFCs or only recombinant human IGFBPs also rescued the ischemia phenotype. These results help us to understand the mechanism of ECFC-based therapies for ischemic insults and retinal neurodegenerative diseases.