

RUNX1 may play role in proliferative diabetic retinopathy

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RUNX1 inhibition. Using immunohistochemical staining for *RUNX1*, reactivity was seen in vessels of patient-derived FVM and angiogenic tufts in the retina of mice with oxygen-induced retinopathy. Use of the Ro5-3335 small molecule to inhibit *RUNX1* activity resulted in significant reduction of neovascular tufts in oxygen-induced [retinopathy](#).

"These findings, including the high glucose-dependent expression of *RUNX1*, identify a novel pathway of potential therapeutic interest, and implicate *RUNX1* in aberrant angiogenesis in multiple conditions," the authors write.

Several authors have filed a provisional patent application on *RUNX1* regulation for the treatment of aberrant angiogenesis and [proliferative diabetic retinopathy](#).

More information: [Abstract/Full Text \(subscription or payment may be required\)](#)

(HealthDay)—The Runt-related transcription factor 1 (*RUNX1*) gene may play a role in human proliferative diabetic retinopathy (PDR), and upregulation may be a marker of aberrant retinal angiogenesis, according to a study published online April 11 in *Diabetes*.

Noting that PDR affects those with type 1 and 2 diabetes, and is a common cause of blindness in the developed world, Jonathan D. Lam, M.D., from the Schepens Eye Research Institute in Boston, and colleagues examined the role of *RUNX1* in PDR.

The researchers identified *RUNX1* as a gene that was upregulated in CD31+ vascular [endothelial cells](#) obtained via transcriptomic analysis from human PDR fibrovascular membranes (FVM). Increased *RUNX1* RNA and protein expression were seen in response to high glucose in in-vitro studies using human retinal microvascular endothelial cells (HRMECs); HRMEC migration, proliferation, and tube formation were reduced with

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