



## **UNITY Biotechnology Presents UBX1325 Data Demonstrating Improvement in Retinal Vasculature and Function in Preclinical Models of Diabetic Retinopathy Eye Diseases**

May 1, 2021

*UBX1325's selective elimination of senescent cells represents a novel approach for the treatment of diabetic retinopathy and macular edema*

*Preclinical data featured in poster presentations at the ARVO 2021 Annual Meeting*

SOUTH SAN FRANCISCO, Calif., May 01, 2021 (GLOBE NEWSWIRE) -- UNITY Biotechnology, Inc. ("UNITY") [NASDAQ: UBX], a biotechnology company developing therapeutics to slow, halt or reverse diseases of aging, today announced preclinical data demonstrating that UBX1325, a novel senolytic small molecule inhibitor of Bcl-xL, improves retinal vasculature and is differentiated from anti-VEGF agents in preclinical models. Researchers show that inhibition of retinal Bcl-xL by UBX1325 selectively promotes apoptosis of diseased senescent cells of the retina, thereby restoring healthy vasculature and improving retinal function – important distinctions from anti-VEGF treatments. The research was presented in a poster presentation, "UBX1325, a small molecule inhibitor of Bcl-xL, attenuates vascular dysfunction in two animal models of retinopathy" during the Association for Research in Vision and Ophthalmology (ARVO) 2021 Annual Meeting.

"The data presented provide important preclinical support for the potential of UBX1325 as a differentiated treatment for prevalent vascular diseases of the eye," said Przemyslaw (Mike) Sapielha, Ph.D., chief scientific advisor of UNITY. "Retinal vascular diseases, namely diabetic retinopathy and macular edema, are primary causes of blindness in the United States, but current treatments are less than ideal. By targeting the senescent cells that promote inflammation and compromise vascular integrity in the eye, we aim to provide an effective therapeutic option that selectively eliminates diseased and leaky cells within blood vessels while potentially stimulating vascular repair. We are excited that this molecule is now in clinical studies in patients with DME."

Dr. Mike Sapielha will also participate in a panel discussion at ARVO 2021 focused on neglected pathways in vision loss. The consequences of retinal hypoxia and ischemia and how it triggers apoptosis, cellular senescence and ultimately production of factors that precipitate diabetic retinopathy will be discussed.

Bcl-xL is a molecular target that is highly expressed in diseased blood vessels during retinopathy, which have been shown to engage pathways of cellular senescence. In the study described, the novel senolytic candidate, UBX1325, selectively inhibits Bcl-xL, promoting apoptosis in oxygen induced retinopathy models but not normoxic controls. As demonstrated in models of retinopathy, vascular leakage and retinal function improved following a single injection of UBX1325 as measured by reduced retinal vascular permeability and improvements of retinal neovascularization and avascular area.

Featured in an oral presentation at ARVO 2021, preclinical research of UBX1967, a selective Bcl-xL inhibitor that is molecularly distinct to UBX1325, demonstrated that the inhibition of Bcl-xL suppresses the formation of diseased blood vessels and targets Col1a1-positive endothelial cells that are associated with disease. The data included in the paper, "Inhibition of Bcl-xL with the small molecule UBX1967 targets Col1a1-positive endothelial cells in ischemic retinopathy" further supports the use of senolytic therapies to potentially target pathological vasculature without off-target effects on healthy blood vessels, as seen with anti-VEGF therapeutics.

UNITY's ARVO 2021 poster presentations will be available on UNITY's website [here](#) after May 5, 2021.

### **About Diabetic Macular Edema**

Diabetic macular edema (DME) is characterized by leaky blood vessels in the eye, contributing to swelling of the retina and vision loss. The National Eye Institute identifies DME as the most prominent complication of diabetes and is the leading cause of blindness in working age individuals, impacting more than 20 million people worldwide. However, nearly half of people with DME are undiagnosed and those that do get treated with the current standard of care, a significant proportion of patients fail to respond to treatment.

### **About UBX1325**

UBX1325, currently in Phase 1 clinical development, is a novel senolytic small molecule inhibitor of Bcl-xL, a member of the Bcl-2 family of apoptosis regulatory proteins. Bcl-xL is highly expressed in pathogenic blood vessels in the retina, and UBX1325 has been shown to result in the selective elimination of senescent cells, reduction in vascular leak and restoration of healthy blood vessels in preclinical models. UBX1325 is currently being evaluated in clinical trials for the treatment of age-related diseases of the eye – including diabetic macular edema, diabetic retinopathy, and age-related macular degeneration.

### **About UNITY**

UNITY is developing a new class of therapeutics to slow, halt or reverse diseases of aging. UNITY's current focus is on creating medicines to selectively eliminate or modulate senescent cells and thereby provide transformative benefit in age-related ophthalmologic and neurologic diseases. More information is available at [www.unitybiotechnology.com](http://www.unitybiotechnology.com) or follow us on [Twitter](#) and [LinkedIn](#).

### **Forward-Looking Statements**

This press release contains forward-looking statements including statements related to UNITY's understanding of cellular senescence and the role it plays in diseases of aging, the potential for UNITY to develop therapeutics to slow, halt, or reverse diseases of aging, including for ophthalmologic and

neurologic diseases, the potential for UNITY to successfully commence and complete clinical studies of UBX1325 for DME, AMD, and other ophthalmologic diseases, the expected timing of initial results of the Phase 1 study of UBX1325, the timing of the expected commencement of the Phase 2a study of UBX1325, and UNITY's expectations regarding the sufficiency of its cash runway. These statements involve substantial known and unknown risks, uncertainties, and other factors that may cause our actual results, levels of activity, performance, or achievements to be materially different from the information expressed or implied by these forward-looking statements, including the risk that the COVID-19 worldwide pandemic may continue to negatively impact the development of preclinical and clinical drug candidates, including delaying or disrupting the enrollment of patients in clinical trials, risks relating to the uncertainties inherent in the drug development process, and risks relating to UNITY's understanding of senescence biology. We may not actually achieve the plans, intentions, or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. The forward-looking statements in this press release represent our views as of the date of this release. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements at some point in the future, we have no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this release. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of the Company in general, see UNITY's most recent Annual Report on Form 10-K for the year ended December 31, 2020, filed with the Securities and Exchange Commission on March 23, 2021, as well as other documents that may be filed by UNITY from time to time with the Securities and Exchange Commission.

Media

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