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| NEWS RELEASE |

*FOR IMMEDIATE RELEASE*

## **MIGENIX Presents Data Supporting Efficacy of MX-4565 in Retinitis Pigmentosa**

**Vancouver, BC, CANADA & San Diego, CA, USA – November 9, 2004** – MIGENIX Inc. (TSX: MGI; OTC: MGIFF), a clinical-stage developer of drugs for infectious and degenerative diseases, recently presented evidence of efficacy of MX-4565, a small molecule with potent neuroprotective activity, in a transgenic model for Retinitis Pigmentosa (RP) at the First International Symposium on Translational Clinical Research for Inherited and Orphan Retinal Diseases in Washington, D.C.

The presentation, entitled "Photoreceptor Preservation in the S334ter Model of Retinitis Pigmentosa by a Novel Estradiol Analog" reports that a single intraocular injection of MX-4565 moderates programmed cell death of the retinal cells at risk in patients. In this model, almost all the retinal cells responsible for converting light into neuronal signals are dead by day 21 of development, whereas almost 40% are still present after a single treatment with MX-4565. MX-4565 appears to function by preserving the integrity of retinal mitochondria, the parts of the cell that generate almost all of the cell's energy. These data have been accepted for publication in the journal "Biochemical Pharmacology". The transgenic animal experiments were conducted with support from the Foundation Fighting Blindness, while the mechanistic studies at MIGENIX were supported by a grant from the National Eye Institute of the National Institutes of Health (NIH).

"With the clear evidence of neuroprotective activity in this rigorous model of RP, MX-4565 has the potential to become a treatment option for people suffering from RP who have few options today to treat their condition," stated Tim Schoen, Ph.D., Director of Medical Therapy Program at the Foundation Fighting Blindness.

### **About MX-4565**

MX-4565 is one of a series of novel, non-feminizing, phenolic steroid analogs that MIGENIX is developing. It is a small molecule drug candidate that exerts potent neuroprotective actions without activating classical feminizing hormone pathways. MX-4565 represents an important preclinical program for MIGENIX with additional studies currently being conducted in RP and Parkinson's disease.

### **About Retinitis Pigmentosa**

Retinitis pigmentosa (RP) is the name given to a group of inherited eye diseases that affect the retina. RP causes the degeneration of photoreceptor cells in the retina. Photoreceptor cells capture and process light helping us to see. As these cells degenerate and die, patients experience loss of night and peripheral vision and progressive vision loss. In North America the incidence of primary RP is approximately 1 in 4000.

### **About the Foundation Fighting Blindness**

The Foundation Fighting Blindness (FFB) is the largest non-governmental source of funding for retinal degenerative disease research in the world. Diseases such as macular degeneration, retinitis pigmentosa and Usher syndrome affect more than nine million Americans. The FFB is ranked as a "Top-Rated" charity by the American Institute of Philanthropy. In 2002, Worth Magazine designated FFB as one of the Best 100 Charities in the country.

## About MIGENIX

MIGENIX is committed to advancing therapy, improving health, and enriching life by developing and commercializing drugs in the areas of infectious and degenerative diseases. With multiple product opportunities in various stages of clinical and preclinical development, the Company's most advanced clinical programs include drug candidates for the treatment of chronic Hepatitis C infections, the prevention of catheter-related infections, the treatment of Alzheimer's disease and the treatment of acne. MIGENIX is headquartered in Vancouver, British Columbia, Canada with US operations in San Diego, California. Additional information can be found at [www.migenix.com](http://www.migenix.com).

"Jim DeMesa"

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The Toronto Stock Exchange has not reviewed and does not accept responsibility for the adequacy or accuracy of this release.