TECHNOLOGY

# Prostaglandin D2 metabolites for treatment of optic nerve diseases

## OVERVIEW

Blockage of blood supply to part or all of the optic nerve within the eye can lead to death or dysfunction of optic nerve cells and is termed non-arteritic ischemic optic neuropathy (NAION). NAION is the most common cause of sudden optic nerve related vision loss and affects about 10,000 Americans every year. Currently there is no effective, FDA-approved treatment for NAION. This technology is a treatment option for patients suffering from NAION that can potentially reduce damage to the optic nerve through the intravitreal administration of prostaglandin J2 (PGJ2) alone or in combination with granulocyte-macrophage colony stimulating factor (GM-CSF).

This technology is a novel solution for the treatment or reduction of damage to the optic nerve through the administration of prostaglandin (PG) alone or in combination with GM-CSF. Preliminary safety studies in rodent anterior ischemic optic neuropathy (rAION) models, an in vivo white matter ischemia model, showed no signs of toxic effects on both functional and structural studies for PGJ2 administration. Instead significant neuroprotective effects were observed when PGJ2 was used immediately and after 5 hours post induction of a white matter infarction. After 30 days post-stroke, optic nerves from the treatment group displayed significant preservation of the number of axons, decreased demyelination, and reduction in tissue edema. Studies conducted in nonhuman primates additionally verified that a single intravitreal (IVT) injection of PGJ2 was neuroprotective even when administered 5 hours post-induction of NAION. Additonal studies using concentrations up to 4x the normal dose also showed no permanent toxicity while still displaying the beneficial neuroprotective effects. These proof-of-principle studies demonstrate the potential of PGJ2 as a clinically effective, non-toxic in vivo therapeutic adjunct to reduce damage following an isolated white matter stroke.

## APPLICATIONS

NAION has no proven, effective treatment to ameliorate vision loss or reduce the risk of recurrence. Approximately 1,500 to 8,000 people develop NAION annually in the US making NAION the most common cause of sudden optic nerve-related vision loss of patients aged 55 years and older. There is usually no pain and vision is not completely lost, however the risk of vision loss in the second eye becomes significantly higher within the following few years. Therapeutic attempts over the past three decades have included both surgical and pharmacologic, but the majority of attempts have been unsuccessful leaving very few if any treatment options to alleviate this condition. Current recommendations attempt to reduce symptoms related to hypertension, diabetes, and high cholesterol that contribute to the increased risk for NAION.

## ADVANTAGES

Sustained neuroprotection: Single injection of IVT PGJ2 preserved RGCs and their axons

Good safety profile: Toxicity not observed in functional and structural studies.

Targeted delivery: Intravitreal administration enables high concentration in a low volume, with minimal risks from systemic side effects.

Effective: Increased survival times and regeneration of retinal cells, improved NAION

### STAGE OF DEVELOPMENT

Data obtained on rodent and primate animal models show treatment with PGJ2 improves survival times and regeneration of retinal cells and results in overall improvement from NAION.

### R&D REQUIRED

Toxicity and dosage studies required

(NC- 6/19/17)

### LICENSING POTENTIAL

UM seeks to develop and commercialize by an exclusive or non-exclusive license agreement and/or sponsored research with a company active in the area.

### CONTACT INFO

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## Additional Information

### INSTITUTION

University of Maryland, Baltimore

### PATENT STATUS

US Patent 8,106,096, issued 1/31/2012

### LICENSE STATUS

Available for licensing; Available for sponsored research

### CATEGORIES

- Therapeutics
- Biologics

### INVESTIGATOR(S)

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### ATTACHMENTS

- Download SB-2008-026 market sheet 6-19-17.pdf

### EXTERNAL RESOURCES

- PGJ(2) provides prolonged CNS stroke protection by reducing white matter edema.
- Cellular inflammation in nonarteritic anterior ischemic optic neuropathy and its primate model.
- Nonarteritic anterior ischemic optic neuropathy (NAION) and its experimental models.
- Axonal degeneration, regeneration and ganglion cell death in a rodent model of anterior ischemic optic neuropathy (rAION).

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