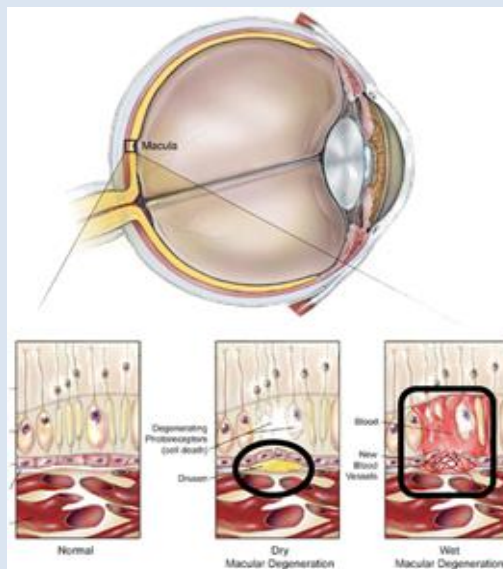


# New Approach for Age-Related Macular Degeneration



## APPLICATIONS

- Potential therapeutic application
  - Wet AMD
  - Dry AMD

## ADVANTAGES

- Unique class of compounds
- Complementary to anti-VEGF therapies
- Proof-of-Principle completed *in vivo*

## INTELLECTUAL PROPERTY

2 PCT patent application filed in 2009

**BACKGROUND** Age-related macular degeneration (AMD) is a disease associated with aging that gradually destroys vision in the center of the visual field. AMD is a leading cause of visual impairment in adults over 60 years of age. The prevalence of this disorder is expected to double in the next decades due to aging of the population.

Macular degeneration is divided into two forms: dry (non-neovascular) and wet (neovascular). The wet form occurs in about 10% of cases and is characterized by the proliferation of blood vessels that are prone to leakage. Wet AMD can rapidly lead to severe loss of central vision. The underlying pathogenesis and sequence of events that lead to AMD are partly due to the accumulation of oxidized lipoproteins and the subsequent macrophage infiltration that act as triggers. Recently, CD36 has emerged as a new biological target involved in lipid accumulation and its modulation may be of interest in wet and dry AMD treatment.

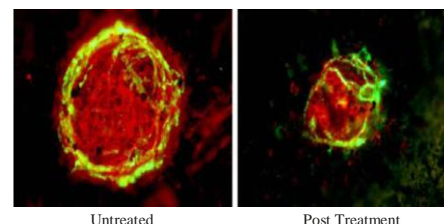
**TECHNOLOGY** The technology was developed by internationally renowned scientists Drs. Ong and Lubell from Université de Montréal, and Dr. Chemtob from CHU Ste-Justine Research Centre, and consists of a unique class of CD36

modulators that inhibits choroidal neovascularization in a murine model of AMD. These small synthetic azapeptides of 6 to 9 residues are specific ligands of the scavenger receptor CD36.

**RESULTS** A structure-activity relationship study was performed that identified lead compounds.

### Proof of concept:

- *In vitro*: Binding studies demonstrated specificity towards CD36
- *Ex vivo*: Inhibition of angiogenesis in the rat aortic ring model
- *In vivo*: Inhibition of choroidal neovascularization in the laser injury murine model.



**Figure above:** Flat mounts of the RPE/choroid/sclera showing the reduction of choroid vessels detected using FITC-conjugated dextran following the treatment with DBG178 (right panel) as compared to the untreated control (left panel). The endothelial cells were visualized in red using lectin (*bandeiraea simplicifolia*).

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