

*Industry Insider* is a timely chat with an ophthalmic industry thought leader.

# Aviceda Therapeutics Mohamad Genead

*Aviceda Therapeutics creates therapies that rely on immunological “switches” that can turn on or off pathologic mechanisms within the body. Its lead product is AVD-104, an optimized biodegradable nanoparticle that targets dry AMD. A Phase 2 clinical trial for AVD-104 is scheduled for later this year.*

**Mohamad Genead, MD**, co-founded Aviceda and serves as its CEO and president. He has more than 20 years of experience in ophthalmology research and development.



Mohamad Genead, MD

**Ophthalmology Management:** Could you explain “immunological switches,” and how they can be used to treat dry AMD?

**Mohamad Genead, MD:** Using this therapy, we target a part of biology called the SIGLEC receptors family (sialic acid-binding immunoglobulin-type lectins), which tend to be more present in the eyes of dry AMD patients. SIGLECs are checkpoint receptors on our immune “defense” cells that act as a first line of defense and react whenever they detect foreign material. All immune cells like this have “switches” we can activate to turn these responses on and off depending on a patient’s needs. For instance, in cancer patients this therapy is used to “switch on” defensive cells to protect the body. With AVD-104, we use it to “switch off” cells that create the chronic inflammation response of dry AMD.

**OM:** What makes AVD-104 unique from other treatments?

**MG:** AVD-104 is a novel biodegradable nanoparticle that targets the biology of the SIGLECs. It is the first therapy to treat the macrophages, the

root cellular cause of dry AMD; most dry AMD treatments only target the complement inhibitor, whereas we target both. The macrophages are cells that act as “housekeepers” of the body, clearing it of waste products and defending it against damage. However, they can become “hyperactivated,” and react even without signs of damage or waste and cause harm to the cells. This is what causes the retinal degeneration in dry AMD. With AVD-104, we instruct the macrophages’ SIGLECs to “switch off” this response and restore the macrophages to their resolution state, in addition to deactivating the complement “non-cellular” pathway.

**OM:** Do you have any other products in your pipeline?

**MG:** We are planning to apply for a second indication of AVD-104 for patients with macular atrophy secondary to wet AMD. What we’ve seen over years of working in the retina space is that some patients will still develop macular atrophy, even after using anti-VEGFs on a monthly basis. Based on our preclinical data, AVD-104 could be a very good adjunctive

treatment for macular atrophy patients receiving anti-VEGFs.

We are also developing two other assets, both in preclinical phase. AVD-201 is for treatment of retinal fibrosis, and AVD-302, for treatment of diabetic retinopathy, is close to receiving an Investigational New Drug Application from the FDA.

**OM:** How has your experience in ophthalmic R&D prepared you for your role at Aviceda?

**MG:** My background in biopharmaceutical companies has always paid dividends. When you go through all the phases of drug development and work in a big company, you learn a lot about discipline and standard operating procedures. I was able to apply the skills I learned to move our assets as fast as possible. In 2 years at Aviceda, we went from a discovery in a lab to a clinical trial initiation, scheduled for this year. That is an extremely aggressive timeline.

While your background plays a role, the team you bring to the table is also a huge factor in your success. Most of my team have 20+ years in drug development in ophthalmic space. That’s invaluable, too. **OM**