Inflammasome Therapeutics' Kamuvudines Continue to Show Promise for Future Treatment – and Potentially a Cure – for Blindness in Seniors

- *New research published today in Science Advances describes precise mechanism of how Alu reverse transcription occurs from RNA to cDNA, and its subsequent triggering of inflammasome activation and consequent cell death in Geographic Atrophy (GA) – the most severe form of dry age-related macular degeneration.*

- *Research shows Alu RNA/cDNA reverse transcription confined to rim of lesions in human GA; new finding indicates potential to prevent activation of inflammasome activity and halt subsequent progression of GA.*

- *Further confirms NRTIs and Kamuvudines block inflammasome activity and point to development of drugs to treat GA – potentially offering a cure for this eye condition where no therapeutic treatments are available*

Newton, MA (September 30, 2021) – Inflammasome Therapeutics (https://inflam.com), a private company developing therapies for prevalent, degenerative diseases, reported new research conducted by a team of scientists led by Inflammasome's co-founder Dr. Jayakrishna Ambati, and published today in Science Advances https://www.science.org/doi/10.1126/sciadv.abj3658 on Geographic Atrophy (GA), the most severe form of dry Age-related Macular Degeneration (AMD). In GA an area of the retinal pigmented epithelium (RPE, a layer of cells in the retina) becomes atrophied and the overlying sensory retina dies. Over time this area slowly spreads leading to blindness. It has been previously reported that Alu RNA is over expressed in the retinas of individuals with this condition. The paper confirms that in human eyes Alu RNA/cDNA conversion triggers inflammasome activity and resulting retinal cell death. Further, the paper shows that this process is confined to the rim of the lesion, indicating a potential for treatments to halt inflammasome activation and the progression of this disease to otherwise normal retina.

Additionally, the group showed that nucleoside reverse transcriptase inhibitors (NRTIs) and their low toxicity derivatives, Kamuvudines, halt inflammasome activation and disease progression in pre-clinical models– thereby offering a potential treatment for dry AMD in humans.

"Our research found that in human eyes with GA, Alu cDNA is almost entirely located at the border of healthy and dying RPE and is barely detectable in RPE of healthy eyes or the still healthy regions of eyes with GA," said Dr. Ambati, who is also the DuPont Guerry, III Professor and Founding Director of the Center for Advanced Vision Science at the University of Virginia. "Additionally, we continue to strengthen the body of peer-reviewed science that demonstrates NRTIs' and Kamuvudines' ability to inhibit inflammasome activation. This points to a strong potential for therapeutic treatments to be developed that could arrest the process that causes GA while most of the RPE and retina is still healthy. It would be as close to a functional cure for GA as one could hope for in a condition that has no approved therapeutic treatment at the present time."

Dr. Paul Ashton, president and CEO of Inflammasome, said, “Age-related Macular Degeneration affects approximately 20% of the population over 70 years of age and 50% of the population over 80. It comes in two forms, wet AMD, which accounts for 15% of cases and can be treated with monthly intravitreal injections, and dry AMD which accounts for the rest and is untreatable. The most severe form of dry-AMD is geographic atrophy affecting more than one million people in the US. Unfortunately, dry AMD has been a difficult target. Over the past 15 years more than 100 clinical trials for GA treatments have failed; most of these drugs under development tried to inhibit specific toxic processes, but this disease is multifactorial so targeting one element may not work. Dr. Ambati’s ongoing research in this area indicates that targeting the disease process, rather than a specific element may offer the best way forward. The work continues strengthen the science showing the role of inflammasome activation as the common factor in GA. And, if we can block this activation, we believe we have potential to treat – and perhaps even cure – one of the main causes of AMD – and blindness in an aging population.”

Dr. Ashton said that Inflammasome plans to bring one or more of its Kamuvudines into the clinic in 2022. “All of our preclinical research points to our Kamuvudines as being a class of drug that can halt inflammasome activation, which in addition to GA, is becoming recognized by scientists as the driver of several chronic, debilitating, diseases. We look forward to exploring Kamuvudines in GA as well as other inflammasome-mediated diseases such as MS and Alzheimer’s Disease.”

Inflammasome Therapeutics (www.inflam.com) was founded by Jayakrishna Ambati, M.D. and Paul Ashton, Ph.D. in 2016 to develop therapies for prevalent, degenerative diseases. The company combines scientific excellence with proven development expertise and works to develop products via a mixture of licensing agreements and internal development. The company currently has collaboration agreements with Boehringer Ingelheim and the Bill & Melinda Gates Foundation.

Contact:
Beverly Jedynak
312-943-1123
773-350-5793 (cell)
blj@bevlynconsulting.com