



Inflammasome Therapeutics' CEO to Be Featured Speaker at Upcoming Virtual Summit on Inflammasomes

Newton, MA (November 12, 2021) -- Paul Ashton, president and CEO of Inflammasome Therapeutics (<https://infram.com>), a private company developing therapies for prevalent, degenerative diseases, will be one of the featured speakers at the *Inflammasome Therapeutics Summit*. This digital event, November 16-18, marks its third year as the only industry-dedicated forum that focuses specifically on inflammasome regulation and novel inflammasome therapeutics.

Dr. Ashton will present "Insights and Learnings from Clinical Investigations of Inflammasome Activation in the Ophthalmic Setting," at 12:30 p.m. ET on Thursday, November 18. Prior to that he will join Steve Glover, co-founder, CEO and chairman of Zyversa Therapeutics and Anil K. Goyal, CEO and chairperson of IMMvention Therapeutix, Inc. to lead the first panel of the conference that will review and focus discussion on the current state of play of the inflammasome therapeutics space.

During his talk, Dr. Ashton will discuss the role that inflammasome activation plays in ophthalmic diseases, highlighting:

- Inflammasome activation as a key pathway in macular degeneration, which affects millions of older individuals worldwide and is a leading cause of blindness among this population.
- Blocking inflammasome activation prevents macular degeneration *in vivo*.
- Clinical data that supports the efficacy of this strategy.

Dr. Jayakrishna Ambati, co-founder of Inflammasome Therapeutics, has been leading the way in research regarding the role inflammasomes play in serious eye disease, specifically Geographic Atrophy (GA) the most severe form of dry Age-related Macular Edema (AMD).

In September, new research published in *Science Advances* and conducted by a team led by Dr. Ambati, DuPont Guerry, III Professor and Founding Director of the Center for Advanced Vision Science at the University of Virginia, described the 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). This research showed that this reverse transcription was confined to rim of lesions in human GA, a new finding that indicates potential to prevent activation of inflammasome activity and halt subsequent progression of this blinding eye disease.

This research also continued to support prior studies published in *Nature's Signal Transduction and Targeted Therapy*; *Science*; *Nature Communications* and *Proceedings of the National Academy of Sciences (PNAS)* that have demonstrated consistently that AIDS drugs, nucleoside reverse transcriptase inhibitors (NRTIs), are highly effective at inhibiting inflammasome activation and that a derivative class of NRTIs, called Kamuvudines, have the potential to provide the same benefit without the significant side-effects and toxicity that comes with long-term use of current NRTI drugs.

Inflammasome Therapeutics is commercially developing Kamuvudines, and the company has said that it plans to bring one or more of its Kamuvudines into the clinic next year. “Our preclinical research points to Kamuvudines as being a class of drug that can halt inflammasome activation, which continues to be recognized as the driver of several chronic, debilitating diseases. We look forward to exploring Kamuvudines in ophthalmic conditions as well as other inflammasome-mediated diseases such as MS and Alzheimer’s Disease,” said Dr. Ashton.

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

blj@bevlynconsulting.com

312-943-1123

773-350-5793 (cell)