Giulio Maria Pasinetti
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Biography

Giulio Maria Pasinetti is The Saunders Family Chair and Professor of Neurology, received an MD from the Milan University School of Medicine and a PhD from the University of Milan. He is currently the Program Director of the NIH funded Mount Sinai Centre for Molecular Integrative Neuroresilience and the Chief of the Brain Institute Centre of Excellence for Novel Approaches to Neurodiagnostics and Neurotherapeutics. He is also a Professor of Psychiatry, of Neuroscience, and of Geriatrics and Adult Development. He is the recipient of several academic awards including the prestigious Zenith and Temple awards from the Alzheimer's Association. Most recently, also he was awarded with The Faculty Council Award" for academic excellence at Mount Sinai School of Medicine and "The Charles Dana Alliance for Brain Research Award" from Dana Foundation, recognizing productivity and worldwide leadership in his field of expertise, which further emphasizes his standing as an academic role model.

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Note:

PRINCIPLES OF INFLAMMASOME PRIMING AND INHIBITION: IMPLICATIONS FOR PSYCHIATRIC DISORDERS

The production of inflammatory proteins by the innate immune system is a tightly orchestrated procedure that allows the body to efficiently respond to exogenous and endogenous threats. In the talk the author will first discuss accumulating evidence suggesting that disturbances in the inflammatory response system not only provoke autoimmune disorders, but also can have deleterious effects on neuronal function and mental health. As inflammation in the brain is primarily mediated by microglia, the immune inflammatory cells of the brain, there has been an expanding focus on the mechanisms through which these cells initiate and propagate neuroinflammation. Based on this evidence the author will debate novel concepts about how microglia can enter persistently active states upon their initial recognition of an environmental stressor and are thereafter prone to elicit amplified and persistent inflammatory responses following subsequent exposures to stressors. In view of the recent evidence suggesting that primed microglia may be respond to environmental insults through mechanisms involving the NLRP3 inflammasome; in the presentation the author will then discuss new concepts supporting the activation of NLRP3 inflammasome mechanisms responsible for the generation of inflammatory interleukins into functional forms that elicit several consequential effects in the local neuronal environment. This evidence supports the principle that within primed neuroimmune systems a lowered threshold for NLRP3 activation can cause persistent neuroinflammation or the amplified production of inflammatory cytokines. Collectively, the take home message of my presentation will provide novel evidence suggesting that targeting the NLRP3 inflammasome complex may represent an innovative approach to limit neuroinflammatory states in psychiatric disorders.