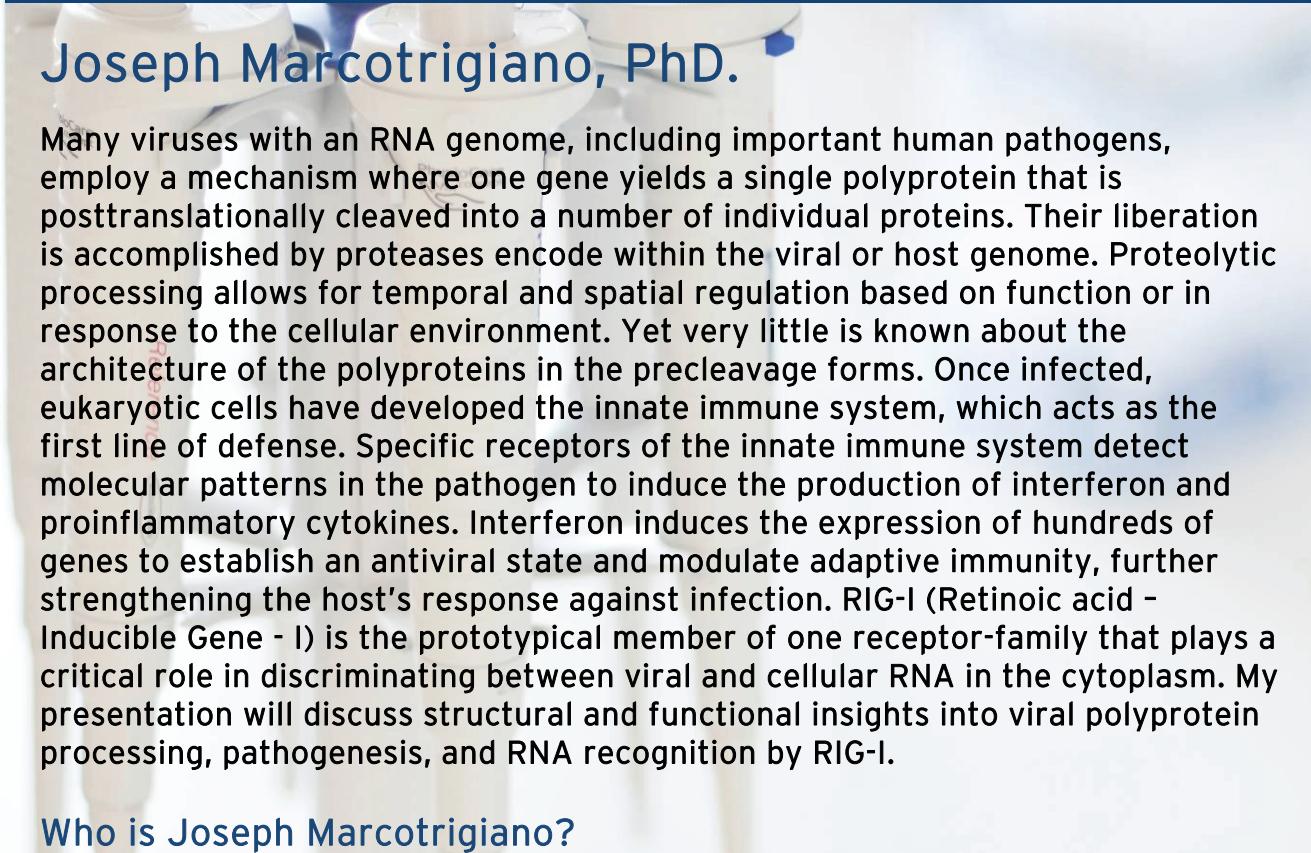


TWINCORE - Seminar

Thursday March 22nd, 2012, 12.30 p.m.
TWINCORE Lecture Hall

"Replication of RNA viruses and the Innate Immune Response"



Joseph Marcotrigiano, PhD.

Many viruses with an RNA genome, including important human pathogens, employ a mechanism where one gene yields a single polyprotein that is posttranslationally cleaved into a number of individual proteins. Their liberation is accomplished by proteases encoded within the viral or host genome. Proteolytic processing allows for temporal and spatial regulation based on function or in response to the cellular environment. Yet very little is known about the architecture of the polyproteins in the precleavage forms. Once infected, eukaryotic cells have developed the innate immune system, which acts as the first line of defense. Specific receptors of the innate immune system detect molecular patterns in the pathogen to induce the production of interferon and proinflammatory cytokines. Interferon induces the expression of hundreds of genes to establish an antiviral state and modulate adaptive immunity, further strengthening the host's response against infection. RIG-I (Retinoic acid - Inducible Gene - I) is the prototypical member of one receptor-family that plays a critical role in discriminating between viral and cellular RNA in the cytoplasm. My presentation will discuss structural and functional insights into viral polyprotein processing, pathogenesis, and RNA recognition by RIG-I.

Who is Joseph Marcotrigiano?

Dr. Marcotrigiano is a structural biologist and his lab has provided major contributions to our understanding of RNA virus replication and their interaction with factors of the innate immune response. For a reference please see the following articles:

- Jiang F, Ramanathan A, Miller MT, Tang G-Q, Gale M Jr., Patel SS, Marcotrigiano J. Structural basis of RNA recognition and activation by innate immune receptor RIG-I. *Nature* 2011 Sep 25;479(7373):423-7.
- Saito T, Owen DM, Jiang F, Marcotrigiano J, Gale M Jr. Innate immunity induced by composition dependent RIG-I recognition of hepatitis C virus RNA. *Nature* 454(7203):523-7, 2008.

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