Molecular Medicine

Podcast Transcript
Episode 38

Hello *Mollie Medcast* listeners and welcome back. *Mollie Medcast* is the podcast for the biomedical journal, *Molecular Medicine*. My name is Margot Puerta. I'm the managing editor here at *Molecular Medicine* and your host for this podcast episode. In this week's podcast we'll be looking at: "Blocking Inflammation In Renal Fibrosis," "PAQR10 In Pancreas," and, "CLL Monoclonal Antibodies [mAbs] Bind Apoptotic And Chemical Oxidation Epitopes."

Let me take a minute to remind you about what our goal here at *Molecular Medicine* is. Our mission is to publish novel work that's concerned with understanding the pathogenesis of disease at the molecular level, which may lead to the design of specific molecular tools for disease diagnosis, treatment, and prevention. If you're interested in submitting a manuscript to the journal, please visit our website for information, www.molmed.org, then click on "Author Center". Okay, so let's get started with the papers in this podcast. The first paper in this Mollie Medcast episode is:

Blocking Inflammation In Renal Fibrosis
Ischemia and reperfusion injury contributes to the development of chronic interstitial fibrosis/tubular atrophy in renal allograft patients. Cyclooxygenase 1, or COX1, and Cyclooxygenase 2, or COX 2, actively participate in acute ischemic injury by activating endothelial cells and inducing oxidative stress. Blockade of COX 1 and 2 is associated with organ improvement after ischemic damage. And, in this work, Dr. Carla Feitoza and her colleagues in Brazil investigate the role of COX 1 and 2 in the development of fibrosis by performing COX 1 and 2 blockade immediately before ischemia and reperfusion injury. The title of the paper is, "Inhibition of COX 1 and 2 prior to Renal Ischemia/Reperfusion Injury Decreases the Development of Fibrosis." Inhibition of COX 1 and 2 resulted in less tissue fibrosis, which was associated with a decrease in proinflammatory cytokines as well as an enhancement of the protective cellular response. The present work demonstrates that COXs play an important role in the development of sustained inflammation. There is no effective treatment for renal fibrosis and COX blockade prior to acute injury may represent a treatment strategy for renal damage associated with fibrosis.

Next up is:
PAQR10 In Pancreas
Steroid hormones have been shown to rapidly modify cell function by binding to cell surface membrane receptors. In a screen for genes differentially expressed in mouse pancreatic β-cells, Dr. Góñez and his colleagues at The Walter and Eliza Institute of Medical Research identified a candidate steroid membrane receptor, the progestin and adipoQ receptor 10, or PAQR 10. The title of the paper is, "Pancreatic Expression and Mitochondrial Localization of the Progestin-AdipoQ Receptor PAQR10." PAQR10 is structurally related to bacterial hemolysins, which are pore-forming virulence factors that target mitochondria and regulate apoptosis. The authors propose PAQR10 may act at the level of the mitochondrion to regulate pancreatic endocrine cell development and survival; further studies are likely to establish a key role of PAQR10 in pancreatic β-cell biology.

And the last paper for this podcast episode is:

CLL mAbs Bind Apoptotic And Chemical Oxidation Epitopes

So, let's start with CLL, that stands for chronic lymphocytic leukemia, and this leukemia is the most prevalent hematologic malignancy affecting Caucasian adults and is one of the four main types of Leukemia.[1] CLL is characterized by a monoclonal expansion of a subset of antigen-experienced human B cells expressing surface membrane CD5. CLL cells likely derive from autoreactive B cells and in this work, Dr. Rosa Catera and colleagues at The Feinstein Institute explored whether apoptosis-associated autoantigens were relevant to the selection and expansion of leukemic cells in CLL. Their findings suggest that CLL arises from a B-cell subset which normally helps clear cellular debris and metabolic byproducts by recognition of ubiquitous, conserved autoantigens. Response to this recognition may drive the clonal expansion of leukemic cells, thereby contributing to clinical outcome.

To find out more about the impact of this CLL paper, you might want to check out our commentary on CLL. This commentary by Dr. Federico Caligaris-Cappio is available on our home page of our website under the "Papers In Press" section and will also appear in an upcoming issue in 2009.

That's it for this week's episode of *Mollie Medcast*. Join us next time for our end of the year episode when we review the top cited Mol Med manuscripts in 2008. You can find the papers from this episode and many more of them on our website, www.m-o-l-m-e-d.org.

Check out our podcast webpage molmed.org/podcast. You can play around with our frappr map and see where other *Molecular Medicine* readers are coming from. I've got my pin up there with a picture too; help us expand our community by adding your very own pin to the map.

This podcast is available on our website and is up in iTunes. *Molecular Medicine* is published bimonthly by The Feinstein Institute for Medical Research. From Long Island, New York, this is margot@molmed.org, thanks for listening!

Written and Produced by Margot Puerta
Managing Editor, *Molecular Medicine*

Production Assistant: Veronica J Davis
Assistant Editor, *Molecular Medicine*

Music: Opuzz.com
Photos: iStock or BigStock, Cover molecule, Wikipedia Commons
References:
1. The Leukemia and Lymphoma Society. http://www.leukemia-lymphoma.org/all_page.adp?item_id=7059 Accessed November 25, 2008.