Molecular Medicine

Podcast Transcript
Episode 28

Hello fellow scientists, and welcome back to "Mollie Medcast," the podcast for the biomedical journal, *Molecular Medicine*. This is Margot Puerta, I'm the Associate Editor here at *Molecular Medicine* and your host for this podcast episode. In this week's podcast: "Colorectal Cancer Prognostic Factor", "Targeting LPA In Sepsis", and our review and assess paper this week, "'Alarmin' The Host Of Danger."

Our goal here at *Molecular Medicine* 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 diagnosis, treatment and prevention. If you are interested in submitting a manuscript to *Molecular Medicine*, please visit our website for information, www.molmed.org. Alright, so let's get started with the papers in this podcast. The first paper in this "Mollie Medcast" episode is:

Colorectal Cancer Prognostic Factor
Colorectal cancer or CRC is the third leading cause of cancer deaths in Western countries and there are an estimated 140,000 new cases each year.[1,2] Development of distant metastases by tumor cells spread from the primary tumor site is a major cause of death. Using biomarkers to identify colorectal cancer patients who would benefit from adjuvant treatment may decrease the risk of recurrence. Low folate levels are seen in colorectal cancer patients with poor survival. The folate levels affect gene-specific hypermethylation, and in this work Dr. Yvonne Wettergren and her colleagues from Sweden investigated whether hypermethylation of the p16 promoter in mucosa could be detected and related to survival of colorectal cancer patients. The manuscript title is, "p16(INK4a) Gene Promoter Hypermethylation in Mucosa as a Prognostic Factor for Patients with Colorectal Cancer." Patients with p16 hypermethylation in the mucosa had an increased risk of cancer-related death and shorter disease-free survival. Hypermethylation of p16 was identified as an independent prognostic parameter for cancer-specific survival and an independent predictor of disease free survival.

Targeting LPA In Sepsis
Overactivity of the innate immune system results in systemic inflammatory response syndrome or SIRS, as well as in septic shock. A lack of the Gi protein results in augmented inflammatory responses to lipopolysaccharide or LPS. LPA, which is lysophosphatidic acid, activates Gi proteins, and because of this, Dr. Fan and colleagues from the Medical University of South Carolina and the Cincinnati Children's Hospital Medical Center in Ohio hypothesized that LPA could inhibit LPS-induced inflammatory responses through activation of the Gi-coupled anti-inflammatory signaling pathways. The title of the manuscript is, "Lysophosphatidic Acid Inhibits Bacterial Endotoxin-Induced Pro-Inflammatory Response: Potential Anti-Inflammatory Signaling Pathways." Results demonstrate that LPA has an anti-inflammatory effect on LPS-induced systemic inflammation. This occurs through ERK1/2, serine/threonine phosphatases and P13 kinase signaling pathways. Targeting LPA and the corresponding signaling pathways may result in potential therapeutic treatments for sepsis.

If you're interested in finding out some more information about sepsis, check out the Sepsis Alliance website. It's located at www.sepsisalliance.org. You can find out more about what sepsis is, and read real life stories of sepsis survivors.

And last up for this week's podcast is our RNA article, or "Review and Assess", it's called:

'Alarmin' The Host Of Danger
Recent advances in understanding the mechanisms of innate immune system activation have pointed to certain pattern recognition receptors as a common pathway for immune identification of both microbial invasion and tissue injury. By sensing either pathogens or endogenous danger signals released upon cellular stress or damage, these pattern recognition receptors, or alarmins, are capable of alerting the host to danger by activating the innate immune system. Dr. John Klune and his colleagues describe the role of an archetypical alarmin, HMGB1, and its potential therapeutic role in various disease states. The paper title is, "HMGB1: Endogenous Danger Signaling" and is available free on our website for downloading.

That's it for this week's episode of "Mollie Medcast". Join us next time when we find an "Ace In The Hole" and learn that female sex steroids do the heavy lifting in bone metabolism. You can find all these papers and many more of them on our website, www.molmed.org, that's www.m-o-l-m-e-d.org. For questions or comments regarding this podcast, please send me an email at: margot@molmed.org.

Check out our podcast webpage www.molmed.org/podcast. You can play around with our frappr map and see where other *Molecular Medicine* readers are coming from. July is Mollie Medcast's one year birthday and at the end of July we're giving away a free iPod shuffle to one of our lucky frappr map members. So hurry up and get your name on the map to be included in the raffle.

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From Long Island, New York, this is margot@molmed.org, thanks for listening!

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